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Sociodemographic Correlates of Allostatic Load and Longitudinal Patterns of Obesity among Adolescents in the United States: A Mediating Model of Stressful Life Events

Author

Rainisch, Bethany Kay Wexler

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A dissertation submitted in partial satisfaction of the

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in Public Health

by

Bethany Kay Wexler Rainisch

2012

ABSTRACT OF THE DISSERTATION

Sociodemographic Correlates of Allostatic Load and Longitudinal Patterns of Obesity among Adolescents in the United States: A Mediating Model of Stressful Life Events

by

Bethany Kay Wexler Rainisch
Doctor of Philosophy in Public Health
University of California, Los Angeles, 2012
Professor Dawn M. Upchurch, Chair

The period of adolescence is a transitional developmental stage, critical in shaping health trajectories across the life course. While many individuals traverse the adolescent transition relatively unscathed, a number suffer from significant health problems from adolescence to adulthood. Recent studies have begun to investigate the biological mechanism through which social conditions early in the life course influence health trajectories. Previous research on allostatic load, an indicator of physiological dysregulation resulting from the wear and tear of stress, has focused on adults and the aging, but few have explored allostatic load among adolescents. Additionally, recent literature has begun to explore the mechanisms through which social conditions during adolescence lead to changing obesity patterns during the transition to adulthood, though none have considered the mediating mechanism of stressful life events.

Using data from two nationally representative samples of adolescents age 12 to 19 years, the National Health and Nutrition Examination Survey (NHANES) (N = 8,431) and the National Longitudinal Study of Adolescent Health (Add Health) (N = 9,311), the overarching goals of this study were (1) to investigate sociodemographic correlates of AL across adolescence from age 12 to 19 years, and (2) to determine whether stressful life events (SLE) in adolescence explain the relationship between adolescent sociodemographic factors and longitudinal patterns of obesity from adolescence to young adulthood. The research for this dissertation drew upon life course, social stratification, and stress process theories.

Specific significant differences in AL among adolescents by age and race/ethnicity were found. In particular, higher AL scores, suggestive of greater cumulative physiological dysregulation, were significantly associated with older age and Black race/ethnicity. Select significant mediating pathways of adolescent SLE on becoming obese among males were also found. Mediation analysis revealed that SLE done *to* adolescent males partially explained the relationship between age and low family income on becoming obese over time. Surprisingly, no significant mediating effects of adolescent SLE on longitudinal obesity patterns were found among females.

This study provided the first examination of associations between major sociodemographic factors and AL among a nationally representative sample of adolescents. The concept of AL offers great promise toward expanding our understanding of how social and environmental factors are embodied within our biological regulatory systems, and translated into disease outcomes and health disparities. The present research has significant implications for informing health prevention interventions among younger populations. Additionally, by focusing on the intervening mechanism through which adolescent social conditions may affect obesity,

this research contributes to an understanding of the processes of obesity development during childhood and adolescence. These findings point to possible interventions that can prevent obesity among young males who are from low SES households. Recommendations regarding stress management and coping mechanisms could aid in the fight against the increasing obesity epidemic among the younger population.

This dissertation of Bethany Kay Wexler Rainisch is approved.

Carol S. Aneshensel

Anne R. Pebley

Cameron D. Campbell

Dawn M. Upchurch, Committee Chair

University of California, Los Angeles

2012

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

Page

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Vita

Bethany Kay Wexler Rainisch

Education
MPH, Emory University, Atlanta, GA, 2008
Department of Behavioral Sciences and Health Education, Rollins School of Public Health

BA, University of California, Berkeley, 2003
Major: Molecular and Cell Biology; Minor: Spanish

Professional Positions
Instructor, *Introduction to Public Health*, 4/2010-Present
UCLA Extension, Humanities and Social Sciences, Los Angeles, CA
Project Manager, 5/2011-Present
UCLA Study of Family Health
UCLA Semel Institute, Center for Culture and Health, Los Angeles, CA
Graduate Research Assistant, 11/2008-Present
UCLA Fielding School of Public Health, Community Health Sciences, Los Angeles, CA
Special Reader II, *Community Health Sciences 100*, 1/2009-3/2011
UCLA Fielding School of Public Health, Community Health Sciences, Los Angeles, CA
Assistant Epidemiologist, Summer 2009
Centers for Disease Control and Prevention, Division of Tuberculosis Elimination, Atlanta, GA
Medical Services Representative, 6/2005-6/2006
Planned Parenthood Golden Gate, San Francisco, CA

Awards

2012	Population Association of America Annual Meeting Poster Winner
2011-2012	National Institute on Aging Traineeship
2009-2011	National Institute of General Medical Sciences Bio-demography Traineeship
2010	UCLA Graduate Division, Summer Graduate Research Mentorship Fellowship
2009-2010	UCLA School of Public Health Bixby Award, Reproductive Health
2009-2010	UCLA School of Public Health Judith Blake Award for Research in Demography
2008-2009	UCLA School of Public Health Tuition Fellowship
1999-2000	Women's Sports Foundation Scholarship
1999-2000	Jewish War Veteran's Scholarship

Peer-Reviewed Publications
Upchurch, D. and Wexler Rainisch, B. Racial and Ethnic Profiles of Complementary and Alternative Medicine Use Among Young Adults in the United States: Findings From the

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D.C. Upchurch, D. & Wexler Rainisch, B. 2011. "Racial and Ethnic Differences in Acupuncture and Traditional Chinese Medicine (TCM) Use in the US, 2007." *Poster Presentation at the American Public Health Association Annual Meeting, Washington, D.C.*

Wexler, B. 2011. "Family Structure and Intra-Family Relationship Effects on Depressive Symptoms in Young Adulthood." *Poster Presentation at the Population Association of America Meeting, Washington, D.C.*

Wexler, B. 2011. "Do Parent-child and Sibling Relationships in Adolescence Explain the Effects of Family Structure on Depressive Symptoms in Young Adulthood?" *Poster Presentation at the Annual Meeting and Scientific Sessions of the Society of Behavioral Medicine, Washington, D.C.*

Wexler, B. 2010. "A Multilevel Model: Effects of Adolescent Family Structure, Parent-child, and Sibling Relationship Quality on Young Adults' Mental Health." *Oral Presentation at the American Public Health Association Annual Meeting, Denver, C.O.*

CHAPTER 1

INTRODUCTION AND SPECIFIC AIMS

1.1. Introduction

Throughout the life course, patterns of behavior and health combine to construct health development trajectories over time (Elder, 1994, 1998a, 1998b; Elder, Modell, & Parke, 1999). Such trajectories are formulated through the dynamic interactions between person and circumstance, whereby multiple organizational levels interconnect, including the social and biological, to influence health over the lifespan. Recently, research on health trajectories has considered the period of adolescence as a significant contributor of health patterns that sustain well into adulthood (Elder, 1980; Halfon & Hochstein, 2002; Lerner, 1998; McNeely & Blanchard, 2009; Mulye et al., 2009; Pearlin et al., 2005).

The period of adolescence is a transitional developmental stage, characterized by significant physiological, social, and psychological changes (Cobb, 2010; Gluckman, Beedle, & Hanson, 2009; Santrock, 2010). While many individuals traverse the adolescent transition relatively unscathed, a number suffer from significant physical and psychological health problems, including obesity, emotional distress, and type II diabetes; as well as harmful health behaviors including substance abuse and unprotected sex (Centers for Disease Control and Prevention, 2012a; Knopf, Park, & Mulye, 2008; Mulye et al., 2009). Disparities in health and behavior such as these also continue well into young adulthood, contributing to chronic diseases across the life course (Adler & Rehkopf, 2008; Green & Darity, 2010; Harris et al., 2006; Mulye et al., 2009; Pearlin et al., 2005; Phelan, Link, & Tehranifar, 2010).

Such disparities in behavior and health during adolescence result from numerous factors: individual social conditions that influence exposure to life's experiences; the transitional nature

of adolescence in coping with life's challenges; and the timing with which such challenges occur during adolescence (Braveman et al., 2010; Chen, Martin, & Matthews, 2006; Goodman, 1999). Individual variation of these factors influences one's health trajectory over the lifespan (Datta et al., 2006; Frieden, 2010; Institute of Medicine, Committee on Capitalizing on Social Science and Behavioral Research to Improve the Public's Health, & Division of Health Promotion and Disease Prevention, 2000; Leventhal & Brooks-Gunn, 2000; Lynch, Kaplan, & Salonen, 1997; Marmot et al., 2008; McNeill, Kreuter, & Subramanian, 2006; Villard, Ryden, & Stahle, 2007; Yen & Syme, 1999).

Recent studies have begun to investigate the biological mechanism through which social conditions early in the life course influence health trajectories (Green & Darity, 2010; Taylor, Way, & Seeman, 2011). An indicator of the cumulative wear and tear the body experiences as a result of chronic stress over time, allostatic load (AL) has been used as an appropriate framework for exploring the biological mechanism of how adversity across the life course contributes to aging health outcomes (Phelan et al., 2010; Seeman et al., 2010a; Seeman et al., 2001). AL focuses on the interconnection of physiological systems as regulatory markers become unbalanced, a result from prolonged stress (McEwen & Wingfield, 2003). Over time, regulatory systems become incapable of adapting to life's challenges, eventually leading to physiological dysregulation and subsequent negative health outcomes over time (Goldman et al., 2005; Seeman et al., 2001).

Although numerous studies have examined the effects of individual sociodemographic factors on levels of AL among adults and the aging (Chyu & Upchurch, 2011; Crimmins et al., 2003; Geronimus et al., 2006; Karlamangla et al., 2002; Kubzansky, Kawachi, & Sparrow, 1999; McEwen & Wingfield, 2003; Peek et al., 2010; Seeman et al., 2004; Seeman et al., 2001;

Seeman et al., 2002), few have conducted research among a generally representative sample of adolescents (Evans, 2003; Evans et al., 2007; Goodman et al., 2005). Because of the significant transitional period of adolescence within the life course, and its influence on health trajectories, it is important to examine profiles of AL by individual social and economic factors within this age group.

Additionally, while the original intent of this dissertation was to first examine AL among adolescents, followed by the mediating mechanism of stressful life events on levels of AL in young adulthood, time and data restrictions made this second objective unattainable. Rather than examining AL in young adulthood, this dissertation focuses on a highly salient public health issue, adolescent obesity (Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, & Division of Nutrition Physical Activity and Obesity, 2011e). Obesity prevalence among adolescents has nearly tripled in the last three decades (Ogden et al., 2012), and obesity in adolescence is highly predictive of adult obesity (Guo et al., 1994; Whitaker et al., 1997), suggesting the importance of investigating longitudinal patterns of obesity from adolescence to young adulthood.

Obesity is influenced by both social conditions as well as exposure to life's stressful experiences (Barry & Petry, 2008; Lee, Harris, & Gordon-Larsen, 2009; Wang, 2001). The stress process model posits that the social and environmental contexts of one's life contribute to exposure to life's stressors, subsequently influencing health outcomes (Pearlin, 1989, 1999; Pearlin et al., 1981; Pearlin et al., 2005). As recent literature has begun to explore the mechanisms through which social conditions during adolescence lead to changing obesity patterns during the transition to adulthood (Lee, 2009), none have considered the mediating mechanism of stressful life events. Because sociodemographic factors during adolescence

contribute significantly to variation in stress exposure, more research on stress as a mediator between adolescent sociodemographic factors and health outcomes in young adulthood is warranted.

Using a dynamic life course framework incorporating theories of social conditions and the stress process, this dissertation investigates the sociodemographic correlates of an indicator of cumulative physiological dysregulation (AL) among adolescents and examines the mechanism of adolescent stressful life events through which sociodemographic characteristics influence longitudinal patterns of obesity from adolescence to young adulthood.

1.2. Specific Aims

The present research builds on previous work examining social conditions, stressful life events, and health trajectories across the life course, and intends to expand our understanding of how stress influences biological dysregulation in adolescence and obesity across the transition to adulthood. The overall objectives of this dissertation are (1) to investigate the sociodemographic correlates of AL across adolescence from age 12 to 19 years, and (2) to determine whether stressful life events (SLE) in adolescence explain the relationship between adolescent sociodemographic characteristics and longitudinal patterns of obesity from adolescence to young adulthood. To meet these objectives, this study uses two sets of data: NHANES, a cross-sectional biannual survey that collects rich biomarker data from the U.S. population, and Add Health, a nationally representative longitudinal survey of adolescents with detailed social, economic, psychological, and physical well-being information. The specific aims of the proposed research draw upon life course, social stratification, and stress process theories to investigate the following:

Aim 1: **To use a nationally representative sample of adolescents in NHANES to examine the change in levels of AL by age and race/ethnicity across adolescence from 12 to 19 years.**

Description: AL is operationalized as a summary score of the number of biomarkers falling within a high-risk quartile zone as defined by the sample distribution. AL is comprised of 9 biological indicators of systemic regulation: cardiovascular (diastolic and systolic blood pressure), immune/inflammatory (C-reactive protein and serum albumin), and metabolic (body mass index, waist circumference, total cholesterol, high-density lipoprotein, and glycosylated hemoglobin). Bivariate regressions and adjusted Wald tests are used to examine differences in mean AL by sociodemographic group. A multivariate negative binomial regression model with an interaction between race/ethnicity and age is employed to predict whether AL score is conditional on age and race/ethnicity.

Hypotheses: Levels of AL are expected to increase with age and be significantly higher among Black adolescents than Whites. U.S.-born adolescents are also anticipated to have higher levels of AL than foreign born. Low socioeconomic status (SES) adolescents, as measured by household family income and household representative education level, are expected to have significantly higher levels of AL compared to those with higher SES. Last, AL score is expected to significantly increase with age across adolescence, more so for Blacks than for Whites.

Aim 2: To use Add Health to examine adolescent sociodemographic differences in longitudinal patterns of obesity during the transition to adulthood separately for females and males.

Description: Longitudinal patterns of obesity are operationalized as individual change in body mass index (BMI) from Wave II to Wave III of Add Health including: becoming obese, staying obese, or staying non- or reduce obesity. Sociodemographic characteristics include individual characteristics (age, race/ethnicity, nativity status) and family background characteristics (parental education, household income, and family structure), nativity status, race/ethnicity, and age. Multivariate analysis using multinomial logistic regression is used to estimate the relationship between Wave I sociodemographic characteristics and becoming obese or staying obese relative to staying non- or reduce obesity. Coefficients are exponentiated to obtain estimates of the risk ratio (RR) of each obesity outcome as a function of each Wave I sociodemographic characteristic.

Hypotheses: Prevalence of obesity for females and males is expected to increase from Wave II to Wave III. Black and Hispanic adolescents are hypothesized to have a higher risk of becoming and staying obese compared to Whites. Foreign born adolescents and Asians are hypothesized to have a lower risk of becoming and staying obese. An inverse relationship between factors of SES and becoming or staying obese over time is expected. Adolescents living in non-two-biological parent families are also expected to have a greater risk of becoming and staying obese.

Aim 3: To use Add Health to estimate the mediating effects of adolescent SLE on the relationship between sociodemographic characteristics and longitudinal patterns of obesity from adolescence to adulthood separately for females and males.

Description: SLE are measured as the cumulative number of stressful events experienced within one year of the Wave I interview. Events occurring *to* the adolescent are discerned from those performed *by* the adolescent. Multivariate analyses using ordered and multinomial logistic regression, in addition to a mediation model, are used to examine the effect of SLE on the association between Wave I sociodemographic characteristics and longitudinal patterns of obesity. Three mediation criteria are examined: the relationship between each Wave I sociodemographic characteristic and longitudinal patterns of obesity; the relationship between each Wave I sociodemographic characteristic and SLE done *to* and performed *by* adolescence; and the association between adolescent SLE and longitudinal patterns of obesity. If all three relationships are significant, viable mediation models are examined to determine the indirect effect of SLE as a mediator. All models are stratified by gender. To determine the mediating effects of SLE, I use path analysis within MPlus.

Hypotheses: Older adolescents are expected to experience and perform higher numbers of SLE. Blacks and Hispanics are hypothesized to experience and perform more SLE than Whites during adolescence. Adolescents living in low SES households and non-intact families are also expected to experience and perform greater numbers of SLE. Higher numbers of SLE are hypothesized to be significantly associated with a higher risk of becoming and staying obese among both males and females. Adolescent SLE are also expected to significantly mediate the relationship between Wave I sociodemographic characteristics in adolescence and longitudinal patterns of obesity from adolescence to adulthood. Specifically, a higher number of SLE done *to* and performed *by* the adolescent will explain the sociodemographic variation in longitudinal patterns of obesity more so than lower numbers of SLE experienced during adolescence.

1.3. Dissertation Overview

The goal of this dissertation is to explore how the period of adolescence plays a significant role in the development and trajectory of health across the lifespan, influenced by social conditions and exposure to stress. The main objectives of this dissertation are to examine the sociodemographic correlates of AL among a nationally representative sample of adolescents, and to determine whether adolescent SLE explain the relationship between adolescent sociodemographic characteristics and longitudinal patterns of obesity from adolescence to young

adulthood. The research was conducted as a secondary data analysis of two nationally representative samples of adolescents age 12 to 19 years.

Chapter 2 of this dissertation will describe the background and significance, and conceptual framework for investigating sociodemographic correlates of AL among adolescents, and the mediating mechanism of adolescent stressful life events between adolescent social conditions and longitudinal patterns of obesity from adolescence to young adulthood. Chapter 3 reviews the research design and analytic methods that will be used to examine and describe the associations. Survey data used in the present research come from the NHANES and the Add Health.

Chapter 4 presents NHANES results from multivariate negative binomial regression models used to identify differential AL scores across adolescence by race/ethnicity. Chapter 5 presents Add Health results from multivariate multinomial logistic regression models demonstrating how longitudinal patterns of obesity differ by sociodemographic characteristics separate for females and males. Chapter 6 presents Add Health results of viable mediation models to determine whether adolescent stressful life events explain the association between adolescent sociodemographic characteristics and patterns of obesity from adolescence to young adulthood. Last, Chapter 7 presents the discussion, strengths and limitations, and public health implications of these results.

CHAPTER 2

BACKGROUND AND SIGNIFICANCE, AND CONCEPTUAL FRAMEWORK

2.1. Background and Significance

2.1.1. Overview

This section outlines the background and significance for the present dissertation, and is divided into five sections: The first section describes the life course perspective, focusing on adolescence as a critical developmental period, and the importance of health trajectories from adolescence to young adulthood. The second section discusses the significance of social determinants of health and social/economic and racial/ethnic disparities in health, setting the cornerstone for unhealthy life trajectories. The third section considers the stress process theory, highlighting the effect of stressors on health and differential exposure to stressors. The fourth section provides details on physiological dysregulation and the measure of AL, emphasizing biomarkers and measurement of AL, and limited research of AL among the adolescent population. The fifth and final section explains the association between obesity progression and stress, proposing the mechanism that adolescent SLE explains the sociodemographic variation in longitudinal obesity patterns from adolescence to adulthood.

2.1.2. Transitional Period from Adolescence to Young Adulthood

2.1.2.1. The Life Course Perspective

The proposed research utilizes Elder's life course perspective as a framework for exploring how the developmental period of adolescence is a time where stressful life events occur, leading to unhealthy life trajectories and health disparities across the transition to adulthood. The life course perspective interweaves multiple aspects of life course and

experience, supporting the investigation of human development and the social contexts that influence behavior (Elder, 1994, 1998a, 1998b). The life course perspective also focuses on the interplay between an individual's choices and opportunities, social integration, and historical experiences of time and place; all of which occur across the lifespan to affect individual development and health outcomes (Aneshensel, 1992). The following sections use the life course perspective as a foundation for discussing how health development trajectories are shaped by life transitions, such as adolescence and emerging adulthood, and the timing of events and health development.

2.1.2.2. Health Development Trajectories

The life course perspective defines trajectories as stable patterns of behavior or health across time. Health trajectories are often formulated early in life and reinforced over time, thus affecting health trajectories later in life. An expansion of Elder's life course perspective is the life course health development framework, which conceptualizes how the interaction between biological and environmental factors influences individual behavioral functioning across the lifespan, thereby offering a comprehensive understanding of how diseases occur (Halfon & Hochstein, 2002). The life course health development approach provides a foundation with which the present research investigates how stressful life experiences in adolescence influence physiological systemic functioning, including AL, and later health conditions, including obesity progression.

Health development trajectories are formulated through the interconnection of multiple organizational levels, including the biological (e.g., cellular, genetic) through the psychological and social levels (e.g., families, peers) to the distant cultural and policy levels (e.g., institutional, government)(Bronfenbrenner, 1979; Ford & Lerner, 1992; Gottlieb, 1992; Lerner, 1998, 2002;

Riegel, 1975; Thelen & Smith, 1994). Previous research supports the concept that these multilevel pathways affect everyday life, and influence developmental processes such as growth and transition to puberty, which ultimately affect disease risk, progression, and the aging process (Repetti, Taylor, & Seeman, 2002; Worthman, 1999a). By examining the developmental stage of adolescence, the present research contributes to the understanding of how health trajectories are shaped by significant environments during unique phases in the lifespan.

Health development life trajectories are also defined by the design of cumulative events and experiences within individual and population contexts (Elder, 1980, 1994, 1998b; Hertzman et al., 2001; Moen & Wethington, 1999). Life experiences may be independent, gradually causing change over time, or may occur clustered together, across organizational levels (Ben-Shlomo & Kuh, 2002). During one's younger years, life's circumstances can become biologically embedded, causing an accumulation of adverse events over time (Flouri & Kallis, 2007), steering one's health development down a pathway of positive or negative health; depending on the events and experiences. By examining health development trajectories, and the sociodemographic contexts influencing adverse events during adolescence, this study contributes to the understanding of how early life social conditions significantly contribute to later life health outcomes.

2.1.2.3. Health Development Transitions during Adolescence

Elder's life course perspective also takes into account life transitions, which are defined as changes in social roles or responsibilities across the life course. Additional transitions include biological and developmental changes. Such life transitions create, shift, and accumulate significant life events, contributing to individual health development trajectories. The timeframe with which events and development occur also play a role in health development trajectories.

The unique timing of development can greatly contribute to the creation, shift, and accumulation of significant health behaviors and health development trajectories, playing a significant role in the variation of trajectories over time.

Adolescence is a critical developmental period defined by transition (Dusek, 1991; Santrock, 2010; Steinberg & Morris, 2001). Below I define adolescence from a historical, sociocultural, and biological perspective, followed by major theories of adolescence, to demonstrate how the period of adolescence is a significant transitional stage abundant with events and experiences that influence health trajectories over the lifespan.

2.1.2.3.1. Historical and Sociocultural Perspective of Adolescence

The history of adolescence reflects the fusion of scientific discovery and demographic change during the twentieth century. Prior to the twentieth century, the concept of 'adolescence' was non-existent. Youths made an evolutionary transition into adulthood as did centuries of generations before them; beginning with manual labor and ending with marriage. It was not until Stanley Hall published his book, *Adolescence,* in 1904 that the concept of adolescence bloomed (Cobb, 2010; Furstenberg Jr., 2001; Kett, 1993, 2003; Santrock, 2010). Influenced by Darwin, Hall proposed that adolescence is a turbulent period of "storm-and-stress". Hall postulated that maturing sexual drives during puberty conflicted with a wall of social constraint, and that the behavior of young people was neither childish nor adult (Kett, 1993, 2003). The inventor of a model of adolescent behavior, Hall believed that adolescents had to prepare themselves for maturity through life's experiences.

Hall's writings on adolescence were not the only driving factor in the development of 'adolescence' as a concept. The sociocultural conditions of the turn of the twentieth century stimulated active changes in understanding the meaning of 'adolescence'. During the time period

between 1890 and 1920, known as the "age of adolescence", western societies transitioned from agrarian to industrial economies and numerous changes further stimulated the development of an adolescent age group: urbanization, appearance of youth groups (YMCA, boy scouts), and legislation requiring youth to attend school. By the 1920s, protective laws prohibiting employers from hiring children, and post World War I prosperity made it possible for parents to send their children to school rather than work (Santrock, 2010).

As society changes, so too does society's understanding of the period of adolescence. Expectations, role transitions, and normative beliefs of how an adolescent should act, think, and grow into adulthood naturally describe the transitional period of adolescence. The present study posits that this transitional stage in development plays a critical role in influencing health development trajectories over time.

2.1.2.3.2. Puberty as a Biological Process

Over the decades the "storm-and-stress" hypothesis proved to be an iconic depiction of the transition known as adolescence. The biological process of puberty functions as one mode contributing to this transitional period of adolescence.

Puberty is biologically the most important marker of the beginning of adolescence (Gluckman et al., 2009; Santrock, 2010). Puberty is a period of physical development involving both bodily and hormonal changes, including changes in the endocrine system, weight and height, and physical appearance (Ibanez et al., 2003; Kadlubar et al., 2003; Stavrou et al., 2002; Xita et al., 2005). Puberty is believed to be set to occur on average between 9 to 16 years of age (Kaminski & Palmert, 2008).

Bodily changes during puberty begin with a developmental neuroendocrine "switch", whereby a flood of hormones stimulate physical pubertal development (DiVall & Radovick,

2008; Gluckman et al., 2009; Sisk & Zehr, 2005). The interaction between the hypothalamus, the pituitary gland, and the sex organs (gonads) comprise the endocrine system's role in puberty. The hypothalamus releases gonadotropin-releasing hormone (GnRH), which triggers the pituitary gland to release follicle-stimulating (FSH) and luteinizing hormones (LH) (Herbison et al., 2008; Ojeda et al., 2006). FSH and LH stimulate the growth of testes and ovaries. Additional maturation of sex characteristics, such as pubic hair, genital, and breast development, also occurs (Dorn et al., 2006), along with changes in height, weight, hip (females) and shoulder (males) width. A pubertal growth spurt occurs for girls begins on average at 9 years of age, and 11 years for boys (Cobb, 2010; Gluckman et al., 2009; Santrock, 2010).

The physical transformations associated with puberty clearly demonstrate how puberty plays a significant role in the transitional nature of adolescence. Individual differences in puberty onset and progression also occur (Gluckman et al., 2009). Black females experience earlier stages of breast, pubic hair, and menarche development than white females (Chumlea et al., 2003; Herman-Giddens et al., 1997; Sun et al., 2002); and Black males experience earlier onset genital and pubic hair development than White males (Herman-Giddens, Wang, & Koch, 2001; Karpati et al., 2002). Given the wide age range of puberty, two adolescents of the same age can be at distinctively different stages of puberty development. The variation in pubertal timing by gender and race/ethnicity demonstrates how the unique transitional period of adolescence can contribute to variations in health development trajectories. The present study takes into account these pubertal variations by gender by examining health development trajectories of obesity separately for females and males.

The transitional effects of puberty are subjective to adolescent contextual and social surroundings, triggering hormonal and physical development at ages earlier or later than normal

(Allsworth, Weitzen, & Boardman, 2005; Brooks-Gunn & Duncan, 1997; Ellis et al., 2011b). As numerous social and environmental factors play a role in pubertal timing, health development trajectories are affected. The biological process of puberty also affects behavior, emotional wellbeing, and physical health (Blakemore, Berenbaum, & Liben, 2009; Ge, Conger, & Elder Jr, 2001; Klump et al., 2006; Patton & Viner, 2007; Siegel et al., 1998). The biological process of puberty is therefore a stimulus to adolescent health and development, further demonstrating the importance of examining the life stage of adolescence as a significant contributor to health development trajectories across the life span.

2.1.2.3.3. Theories of Adolescent Development

Theories of adolescent development demonstrate another mode by which the transitional period of adolescence influences health development trajectories. Psychoanalytic (including psychosocial) and cognitive theories of development demonstrate how the period of adolescence is truly a significant transitional stage abundant with unconscious and cognitive changes. The following section briefly discusses these theories of adolescent development.

Psychoanalytic theories consider development as an unconscious emotion, stressing the importance of how early life experiences shape development (Cobb, 2010; Santrock, 2010). Freud emphasized that during adolescence, biological changes and personality transformations bring about tremendous tension, conflict, and vulnerability (Freud, 1917). In order to reduce this tension, adolescents develop psychological coping mechanisms in defense of the anxiety associated with this transitional period. Such defense mechanisms are designed to protect the self, but can also be harmful to the individual if inappropriately or inadequately applied (Frankland, 2000).

Different from Freud, Erikson developed a psychosocial view of adolescent development (Erikson, 1950, 1968) focusing on external events and society as key factors. Erikson proposed specific stages of development across the lifespan with unique developmental tasks at each stage (Mossler, 2011). During adolescence, the stage of 'identity versus identity confusion' was proposed, whereby adolescents explore who they are, what they want, and where they are going in life. The more successful an adolescent is at navigating this stage of development, the healthier one's behavior and development trajectories will be (Hopkins, 2000).

Whereas psychoanalytic theories focused on the unconscious, cognitive theories emphasized the conscious. Piaget's theory of cognitive development proposed that adolescents organize and adapt to their environments during a stage of 'formal operational skills and thinking' (Piaget, 1954). In this stage, adolescents begin to think in more abstract, logical terms. The inability to think logically, abstractly, and develop cognitive skills during adolescence can greatly impede an adolescent's ability to adapt to life's changes, thus influencing health behavior and development trajectories throughout the life course.

The changing social and cultural expectations of adolescence, in addition to the biological process of puberty, confirm the transitional and critical developmental nature of the period of adolescence. Adequate thinking skills and defense processes enable adolescents to positively navigate this transitional period, initiating positive health behaviors and health development trajectories at a young age. Thus, examining the period of adolescence is essential in understanding how health trajectories progress across the lifespan.

2.1.2.4. Adolescent Health and Behavior

As a result of adolescence's tumultuous period of 'storm and stress', many adolescents experience negative physical and psychological health outcomes. In 2007 reports showed that

approximately 18% of adolescents were overweight, and 18% were obese (Centers for Disease Control and Prevention, 2012a). Such prevalence rates have nearly doubled compared to fifteen years ago (Centers for Disease Control and Prevention, 2012b; National Center for Health Statistics, 2011). Diabetes is also one of the most common chronic diseases in children and adolescents; one in 400 adolescents and children under age 20 have type II diabetes, with prevalence rates growing as rates of obesity among young age groups continues to rise (Bloomgarden, 2004).

Other psychological and physical health outcomes, as well as unhealthy health behaviors, are also prevalent among adolescents, including suicide, depressive symptoms, drugs, alcohol, and sexually transmitted infections (Centers for Disease Control and Prevention, 2009, 2012c; Dehne & Riedner, 2001; Knopf et al., 2008; Mulye et al., 2009; Substance Abuse and Mental Health Services Administration, 2012).

Adolescents who begin experimenting with adult behaviors during a period of confusion, instability, and psychological change promote lasting health trajectories. Childhood health is a strong predictor of adult health (Ben-Shlomo & Kuh, 2002; Case, Fertig, & Paxson, 2005; Halfon & Hochstein, 2002), and health trajectories formulated during adolescence progress across the lifespan into emerging adulthood, becoming even more damaging or improved with age. Obesity in adolescence is highly predictive of adult obesity (Guo et al., 1994; Whitaker et al., 1997), more so than childhood obesity; and increases the risk for other diseases including diabetes, heart disease, cancer or stroke (The et al., 2010; Thomson et al., 2004; USDHHS, 2000). Where adolescence initially set the groundwork for health trajectories, young adulthood continues this trend.

2.1.2.5. Health Development Transitions during Young Adulthood

Like adolescence, young adulthood is a period of prolonged transition. Beginning as early as age 18, young adults seek maturity, independence, and responsibility; often with the diminishing support commonly provided to adolescents. Although socialization promotes the independence of young adults at specific ages (the right to vote at age 18), many remain within a state of conflicting roles and responsibilities (Arnett, 2004). Conceptually, young adulthood has been re-written as "emerging adulthood", to embrace the developmental limbo that is the process of becoming an adult. Arnett states that emerging adulthood is characteristic of identity exploration, role experimentation, and the search for freedom and maturity.

During the transition to adulthood, individuals often leave home, enter the workforce, and form romantic relationships and families. Such a period filled with transitions has significant implications for health behavior and health trajectories (Arnett, 2000; Brown, 2004; Settersten, Furstenburg, & Rumbaut, 2005). Young adults report higher levels of perceived stress and mental distress compared to older adults (Mulye et al., 2009), in part because of these transitions. Substance use decline is also linked to transitions such as parenthood, employment, and marriage (Schulenberg et al., 2005), while diminished parental supervision and changing societal norms contribute to delayed marriage and risky sexual behaviors (Arnett, 2000).

Rates of obesity are higher among young adults than adolescents (Gordon-Larsen et al., 2004; Gordon-Larsen, The, & Adair, 2010; Lee et al., 2009), as well as other negative health outcomes and behaviors (Mulye et al., 2009)(Park, 2006). Such health and behavior influenced by emerging adulthood's life transitions suggest that health trajectories initiated in adolescence continue onward to the next developmental phase in life (Ben-Shlomo & Kuh, 2002; Case et al., 2005; Halfon & Hochstein, 2002).

The transitions of adolescence to young adulthood present an important life phase whereby health trajectories are formulated, modified, and extended. The sociocultural, biological, and cognitive transitions of adolescence greatly define the initiation of health behaviors and health trajectories during this life stage. As one transition ends, another begins. Emerging adulthood continues to build upon prior developed health behaviors and trajectories, further promoting or suppressing positive health later in life. The present dissertation integrates the transitional period of adolescence to young adulthood to examine how health development trajectories, in this case obesity, changes from adolescence to young adulthood.

2.1.3. Social Determinants of Health and Health Disparities

In addition to using the life course framework, the proposed research focuses on social determinants of health to examine the sociodemographic influences and variations in health trajectories from adolescence to young adulthood. The following section includes a discussion on social determinants of health as fundamental causes of disease, and health disparities as they emerge over the life course, emphasizing social, economic, and racial/ethnic disparities in health.

2.1.3.1. Social Determinants of Health as Fundamental Causes of Disease

The contexts in which we live, work, and interact with our social and contextual environment influence both the opportunities that become available to us and our ability to make decisions that lead us down a path of positive health (Datta et al., 2006; Frieden, 2010; Institute of Medicine et al., 2000; Leventhal & Brooks-Gunn, 2000; Link & Phelan, 1995; Lynch et al., 1997; Marmot et al., 2008; McNeill et al., 2006; Villard et al., 2007; Yen & Syme, 1999). For example, the physical or social environment may reduce or enhance the availability of resources and opportunities for positive health behavior and change (Chuang et al., 2005; Datta et al., 2006;

Diez-Roux et al., 1999; Lee & Cubbin, 2002; Pickett & Pearl, 2001). Thus, researchers have taken on the larger scheme of fundamental factors that put individuals at risk for poor health.

Link and Phelan theorized that social conditions are the fundamental causes of health inequality, and that individuals who are able to avoid risks for morbidity and mortality are those who have adequate resources (Link & Phelan, 1995). They demonstrated that SES is related to multiple disease outcomes by means of multiple risk factors, and that the utilization of resources plays a critical role in the association between SES and health (Phelan et al., 2010).

In particular, greater educational attainment is associated with better employment opportunities, higher income and wealth, healthy working and living conditions, access to health care and health resources, and long term financial stability (Braveman, Egerter, & Williams, 2011; Link & Phelan, 1995; Phelan et al., 2010). Increased wealth leads to better quality health care, fresh and healthy food, regular child care, and neighborhood environments that support the healthy development of children. In contrast, lower educational attainment and lack of economic security can severely contribute to a life filled with adversity, stress, unhealthy work and home environments, and negative physical and psychological health outcomes.

The present dissertation focuses on upstream social determinants of health, such as education, income, and family structure to examine health trajectories from adolescence to young adulthood. Social contexts that vary by gender, age, race/ethnicity, and SES contribute to the formation and progression of health development trajectories over the lifespan, leading to differential health outcomes.

2.1.3.2. Health Disparities across the Life Course

Health disparities are defined as "differences in the incidence, prevalence, mortality, and burden of disease and other adverse health conditions that exist among specific population

groups" (National Institutes of Health, 2005). In other words, differences in health exist by social group, such as: age, gender, race/ethnicity, and socioeconomic status among others (Adler & Rehkopf, 2008; Braveman, 2006).

From early childhood, through adolescence and adulthood, individuals are differentially exposed to life's conditions, contributing to health behaviors and health inequalities (Thoits, 2010). The present study builds upon prior health disparities literature across the lifespan to explore sociodemographic differences in cumulative physiological health and obesity status from adolescence to young adulthood.

Socioeconomic disparities early on, such as household income and parental education, contribute to life-long negative behavioral and health consequences (Adler & Rehkopf, 2008; Bradley & Corwyn, 2002; Duncan et al., 1998; LaVeist, 2005; McEwen & Wingfield, 2003). Children and adolescents, who are unable to care for themselves, are especially exposed to the negative effects of detrimental social and contextual surroundings, resulting in poorer health in adulthood (Anda et al., 2006; Bauer & Boyce, 2004; Case et al., 2005; Hertzman, 1999; Marmot et al., 2008; Smith et al., 1998).

Socioeconomically advantaged adolescents have greater access to resources that can improve health during adolescence and adulthood (Battin Pearson et al., 2000; Brooks-Gunn & Duncan, 1997; Haveman & Wolfe, 1995; Phelan et al., 2010). Family structure also plays a significant role in health disparities. Intact families with two biological parents are likely to have a higher family income, access to greater resources, greater parental support, monitoring, and necessary attention to children and adolescents than non-intact families.

Economic disparities across income and education groups are prevalent within numerous health conditions, throughout the life span (Adler & Rehkopf, 2008; Braveman et al., 2010;

Williams & Collins, 1995; Williams & Jackson, 2005). Infant mortality rate increases with fewer years of maternal schooling (Mathews & MacDorman, 2007), and children's health status patterns by income and education also show significant disparities (Braveman & Egerter, 2008; Centers for Disease Control and Prevention, 2001-2005).

Such disparities show health inequality not only between the highest and lowest SES groups, but also significant differences across the gradient; suggesting the importance that socioeconomic disparities of health are not solely among the most disadvantaged (Black et al., 1980; Braveman et al., 2010; Lantz et al., 1998; Marmot et al., 1991; Pamuk et al., 1998). The current dissertation further contributes to prior health disparities work by exploring disparities in cumulative physiological health and obesity patterns from adolescence to young adulthood as a result of social conditions during adolescence.

Health disparities from adolescence to young adulthood also exist by race/ethnicity (Harris et al., 2006). Blacks and Hispanics are more likely to grow up in an environment with fewer resources and greater neighborhood disadvantage compared to Whites (Braveman et al., 2005; Pickett & Pearl, 2001; Steptoe & Feldman, 2001; Williams et al., 2005). Thus, income gradients in fair or poor health are seen within each racial/ethnic group, and racial/ethnic gradients in health are seen within each level of income (Centers for Disease Control and Prevention, 2001-2005), suggesting the significance of considering race/ethnicity in addition to SES when examining sociodemographic differences in health trajectories over the life course.

Health disparity trends continue into adulthood, with Black adults having higher morbidity and all-cause mortality rates across the lifespan than their White counterparts (National Center for Health Statistics, 2008; Williams & Collins, 1995). However, the same is not true for Hispanic adults, who fare better health wise. The Hispanic paradox states that

although foreign born Hispanic adults have fewer resources and less education, they have the same if not better health than U.S.-born non-Hispanic White adults (Franzini, Ribble, & Keddie, 2001; National Center for Health Statistics, 2008; Palloni & Arias, 2004). Such findings consider nativity status as an important determinant of health disparities across the lifespan. A health development trajectory framework posits that Hispanic adults are expected to have worse health due to earlier health disparities in adolescence, but that foreign born adolescents would have better health relative to their U.S.-born counterparts. The present research utilizes race/ethnicity and nativity status as determinants of health to explore disparities in obesity status and cumulative physiological health from adolescence to young adulthood.

The weathering hypothesis, one theory behind disproportionately greater health disparities among Black adults, suggests that as a result of sociodemographic adversities experienced over time, physiological deterioration occurs at an earlier age for Blacks compared to Whites, leading to worse health across the life span (Geronimus et al., 2006). The current research builds upon the weathering hypothesis and previous health disparities work to examine whether specific sociodemographic groups, including females, Blacks and Hispanics, and low SES adolescents, experience greater adolescent physiological dysregulation and risk for obesity from adolescence to young adulthood.

2.1.4. Role of Stress in Defining Health Development Trajectories

Thus far, I have highlighted the life course perspective to demonstrate how health trajectories are shaped by the period of adolescence, and how variations in health trajectories are further influenced by social conditions, contributing to disparities in health across the life course. Last, to provide a comprehensive examination of health trajectories across the life course, I discuss the physiological mechanism of how stress affects health.

Building upon multiple nested organizational levels influencing health trajectories, the present dissertation incorporates how external environments influence health through the formation of inner biological pathways, which connect experiences from social or environmental adverse events into the regulation of functioning physiological processes (Green & Darity, 2010; Halfon & Hochstein, 2002; Seeman et al., 2010b; Seeman et al., 2004; Seeman et al., 1997; Taylor, Repetti, & Seeman, 1997; Taylor et al., 2011).

The following section discusses the role of stress in defining health development trajectories. I first highlight the stress process model, to show how the location of an adolescent within the social hierarchy of resources influences exposure to stress and thus vulnerability to health outcomes. I then discuss the physiological stress response, followed by the effect of stressors on physiological regulatory systems, and differential exposure of stressors on health, to demonstrate how the current dissertation uses stressful life events to predict longitudinal patterns of obesity from adolescence to young adulthood.

2.1.4.1. The Stress Process Model

Pearlin's stress process model provides a useful orienting framework in explaining how social factors lead up to and away from stress (Pearlin, 1999; Pearlin et al., 1981; Pearlin et al., 2005; Wheaton, 1999). Its basic premise is that the location of an individual within the social hierarchy of resources influences exposure to stress and thus vulnerability to health outcomes (Pearlin, 1999; Pearlin et al., 1981; Pearlin et al., 2005). Just as social determinants act as fundamental causes of disease, individual characteristic including age, gender, race/ethnicity, and socioeconomic status impact well-being by exposing one to more or less stress.

Among social scientists, a stressor can be a condition (threatening, demanding, or constraining) that challenges the normal adaptive abilities of an individual (Wheaton, 1999).

More specifically, stressors are states, experiences, or socioenvironmental burdens that can threaten the physical, emotional, and psychological wellbeing of an individual, taking a toll on one's ability to adjust to life's challenges (Aneshensel, 1992). For example: economic constrains, work, home, and school demands are all stressors. While stressors are demands exterior to one's physical being, the internal biological response to such demands is known as stress (the biological response to stress is discussed in more detail in the following section). As each person's ability to withstand and/or respond to a stressor differs, each individual stress response to a particular stressor also differs.

Stressors exist in the form of events, states or even continuous experiences. The "universe of stress" includes stressors that exist across a continuum from discrete time events to continuous ongoing strains (Wheaton, 1999). Across the continuum of stressor duration, exists layers of stressor domain, such as stressors within the individual or micro domain, all the way to the macro level, entailing stressors of political or economic nature. Furthermore, the continuum includes stressor severity, from traumas to regular daily hassles (Wheaton, 1999). The current research focuses on stressful life events during adolescence as stressors influencing obesity patterns from adolescence to young adulthood.

Stressful life events (SLE) are events that occur at a discrete point in time, and usually represent significant life changes. Holmes and Rahe were the first to develop a scale of life events to measure social readjustment of natural, social demands in a person's life (Holmes & Rahe, 1967). Events such as the death of a spouse or the loss of a job would cause high adjustment, while minor events such as a speeding ticket would cause little adjustment. SLE are also assessed according to one's stage in the life course, enabling a measure of stress to provide a

reasonable and balanced representation of events relevant to individuals occupying specific role sets (Cohen, Kessler, & Gordon, 1995).

SLE measures are generally used to examine the cumulative impact of events over a prolonged period (usually ranging from six months to two years) (Cohen et al., 1995), and remain the dominant method use by researchers interested in examining the negative effects of life events on health outcomes (Cohen et al., 1995). Previous research examining the relationship between SLE and negative health outcomes is based on the assumption that increased numbers of negative SLE experienced within a relatively short period of time (12 months) increase the risk for developing psychological and physical health problems (Compas, 1987; Compas & Wagner, 1991; Thoits, 1995, 2010). As SLE represent discrete significant life changes, the present dissertation uses SLE during adolescence to examine the effect of SLE on longitudinal patterns of obesity from adolescence to young adulthood.

SLE literature has also emphasized the importance of the temporal characteristics of stressors relative to disease outcomes (Cohen et al., 1995). Specifically, events occurring within one year of the time of interview are recommended. The one year reference period is long enough to obtain a reasonable estimate of variations in exposure to recent life events, and short enough to prevent considerable recall bias for events that occurred longer than a year prior (Cohen et al., 1995). The number of events needed for associations with health outcomes has also been shown to reach an "upper limit", with a list of 30-50 relevant life events recommended (Cohen et al., 1995). The present study focuses on numerous acute SLE occurring within a twelve month period, enabling the investigation of an association between SLE during adolescence and obesity patterns from adolescence to young adulthood.

Additionally, researchers of stress are also concerned with confounding between measuring stress and the outcome. Often times there is overlap, whereby life event stressors include some form of serious illness, and thus end up measuring the same concept as the outcome. This bias can be minimized in longitudinal studies, with stressors used to predict changes in the outcome over time (Cohen et al., 1995). Using longitudinal data also alleviates the issue of causal confounding, where illness can cause exposure to stressful events. The current dissertation utilizes a longitudinal survey to facilitate the investigation of how early life stressors contribute to health trajectories over time.

Last, the association between SLE and health differs depending on varying levels of individual control over an event (Brown & Birley, 1968; Dohrenwend, 1973; Dohrenwend & Crandell, 1970; Myers et al., 1972). For example, being shot or stabbed are events that an individual does not have control over, whereas shooting someone is an event performed by the individual. The present research incorporates the context of individual control in examining how SLE done *to* and performed *by* an adolescent influence trajectories of obesity from adolescence to young adulthood.

2.1.4.2. The Biological Stress Response

The most common biological definition of stress is that mentioned by Seyle in his 1956 book *The Stress of Life* (Seyle, 1956). Working with animals, Seyle suggested that stress was the consequence of an organism to unsuccessfully adapt to a physical or emotional threat. Seyle proposed a three stage General Adaptation Syndrome. The first stage, alarm reaction, is the body's physiological response to a stressor. The body activates the fight-or-flight response, stimulates a cascade of events within the nervous and endocrine systems, and experiences

numerous bodily changes, such as increased heart rate, blood pressure, and oxygen intake (Seyle, 1956).

The second stage, adaptation, posits that the body will adapt to continuous stressors in order to reduce the effects of stress exposure. The final stage, exhaustion, is when the body's struggle for readjustment disrupts normal physiological mechanisms, eventually exhausting the organism and increasing illness vulnerability (Seyle, 1956). The present dissertation highlights Seyle's adaptation and exhaustion stages to demonstrate how social conditions and stressors during adolescence influence adolescent physiological systemic dysregulation and obesity patterns from adolescence to young adulthood.

2.1.4.3. Effects of Stressors on Health and Physiological Regulatory Systems

Stressful life events are linked prospectively with poorer psychological health across the life course (Ge et al., 2001; Pine et al., 2002), as well as physical illnesses and disorders including coronary disease, multiple sclerosis, the progression of HIV to AIDS, and post-traumatic stress disorder (Carroll et al., 2005; Glaser, 2005; Mohr & Pelletier, 2006; Mol et al., 2005). Early life stressors influence normal adolescent development and continuing adult health (Barker et al., 2005; Bateson et al., 2004; Eriksson et al., 2000; Eriksson et al., 2001; Eriksson et al., 1999; Forsen et al., 2004; Landrigan et al., 2005), demonstrating the importance of examining the effect of adolescent stressors on health across the lifespan.

A number of studies show that adolescents are at greatest risk for negative health outcomes when they simultaneously experience multiple stressors (Dubois et al., 1992; Petersen, Sarigiani, & Kennedy, 1991; Simmons et al., 1987). In fact, the number of SLE, rather than the novelty or type of event, is associated with negative health (Adkins, 2009; Brooks-Gunn & Warren, 1989; Ge, 1994; Ge, Natsuaki, & Conger, 2006). Youth experiencing greater SLE

(uncontrollable life events such as a family member or friend dying, financial trouble in the home, or being the victim of a violent crime) during adolescence also experienced negative physical and psychological health concerns (Cicchetti & Rogosch, 2002; Ge, 1994; Ge et al., 2006; Graham-Bermann & Seng, 2005; Meinlschmidt & Heim, 2005).

Stressful events that occur during critical periods influence health by becoming biologically embedded in the systemic functioning of an individual, causing enduring dysfunction (Barker et al., 2005; Bateson et al., 2004; Ben-Shlomo & Kuh, 2002; Eriksson et al., 1999; Landrigan et al., 2005; Shonkoff, Boyce, & McEwen, 2009). During adolescence, physiological regulatory pathways are constructed, and are particularly susceptible to stressors (Tosevski & Milovancevic, 2006). Stressors during the development of the nervous system can alter neural development and activity (Eisenberg, 1995, 1999; Johnston, 2004), thereby disrupting future neural patterns and causing overall biological disability (Earls & Carlson, 2001; Johnston, 2004). Thus, circumstances that occur at specific ages play a significant role in shaping the growth, development, and functionality of the nervous system.

In addition to the nervous system, adolescent SLE can also contribute to individual dysregulation of biological regulatory markers (Tosevski & Milovancevic, 2006). Adolescents in more hostile environments, such as abusive families, experience increases in daily blood pressure (Southard et al., 1986) and depressed cardiovascular reactivity (Cichetti & Rogosch, 2001; Murali & Chen, 2005). Early life adversity has also been shown to increase metabolic risk factors, and elevated inflammation levels in adulthood (Danese et al., 2009). The present dissertation builds upon prior research to examine how physiological regulatory systems are influenced by adolescent social conditions and obesity trajectories are affected by adolescent stressors.

Recent studies on biological sensitivity to stressors have begun to uncover the curvilinear relationship between early exposure to adversity and stress reactivity. In other words, children with heightened biological sensitivity to stressors are not only more vulnerable to negative risk factors, but also have a greater capacity to benefit from positive environmental factors (Boyce & Chesterman, 1990; Obradović et al., 2010). Therefore, being highly sensitive biologically may be maladaptive to adverse experiences, but also adaptive in the context of supportive and nurturing environments (Ellis & Boyce, 2008; Ellis, Essex, & Boyce, 2005; Ellis, Jackson, & Boyce, 2006).

While stressors influence individual functioning of physiological regulatory systems, the accumulation of stressors can also cause a wide range of physiological dysregulation across multiple interconnected physiological systems. Declines in immune cells can contribute to respiratory infections, delayed healing, and progression of cancer (Glaser, 2005). Stressors can cause an HPA axis response of hormone dysregulation, influencing the sympathetic nervous system response, and subsequent adaptation to future stressors (Sapolsky, Romero, & Munck, 2000).

Since the body relies on multiple interrelated regulatory systems to exist, dysregulation of one or more of these systems over long periods of time leads to cumulative physiological dysregulation (Goldman et al., 2005; Seeman et al., 2010b; Seeman et al., 2004; Seeman et al., 2001; Seeman et al., 1997; Seeman et al., 2002). With time, as the body begins to decline functionally, chronic diseases and illnesses become more prominent, including heart disease, hypertension, cancer, and psychological disorders (Brunner & Marmot, 1999; McEwen, 1998; McEwen & Seeman, 1999; McEwen & Wingfield, 2003). The current dissertation takes a multi-

system approach to understanding how stress exposure during adolescence affects adolescent physiological systemic regulation and obesity progression across the life course.

2.1.4.4. Differential Exposure to Stressors and Health

As discussed in accordance with the stress process model, individuals are exposed to stressors in large part though the social and economic contexts of their lives (Pearlin et al., 1981). Individuals who experience more stressful events and chronic burdens include: members of racial/ethnic minority groups, unmarried or widowed adults, females, and adolescents and young adults among others (Geronimus et al., 2006; Kessler et al., 2005; Mirowsky & Ross, 2003b). Those who experience more stressors are consequently at a higher risk for adverse health outcomes (Pearlin, 1989, 1999; Pearlin et al., 1981; Pearlin et al., 2005; Seplaki et al., 2004).

In fact, differential exposure to stressful experiences is one of the key ways that health inequalities among gender, social class, and race/ethnicity are produced (Thoits, 2010). Studies show that adolescent girls are either exposed to SLE more so than boys (Burke & Weir, 1978; Compas & Wagner, 1991; Dornbusch et al., 1991; Larson & Ham, 1993; Siddique & Darcy, 1984; Wagner & Compas, 1990), or that girls are more distressed when these events occur (Compas & Wagner, 1991; Dornbusch et al., 1991; Siddique & Darcy, 1984; Simmons et al., 1987); contributing to gender health inequalities at a young age.

Previous studies using longitudinal data show that the number of SLE experienced during adolescence differs by age, race/ethnicity, and socioeconomic status (Ge, 1994; Ge et al., 2006). Adkins et al. found that SLE explain a large portion of minority disadvantage in depressive symptoms for Black and Latino adolescents (Adkins, 2009). Similarly, Blacks are more likely than Whites to be exposed to early life stress, and experience more SLE into young adulthood, thus leading to increased risk for depression (Boardman & Alexander, 2011). Such early life SLE

include being the victim of a violent crime, the death of a relative or friend, experiencing the end of a romantic or sexual relationship, and performing acts of violence, delinquency, or harm to oneself (Adkins, 2009; Boardman & Alexander, 2011).

Taking into account the stress process model, the current research focuses on how an adolescent's social conditions influence exposure to stress and thus vulnerability to health outcomes. As the biological response to stress affects the regulatory functioning of physiological systems, differential exposure to stressors contributes to health development trajectories and subsequent health disparities across the life course. The present dissertation incorporates the physiological mechanism of how social conditions and stress affect health to examine adolescent cumulative physiological dysregulation and how stressful life events predict obesity patterns from adolescence to young adulthood.

2.1.5. Allostatic Load

As previously discussed, stress affects health through the functioning of regulatory physiological systems; and as the body's regulatory systems are interconnected, multiple systemic dysfunction can contribute to lasting health outcomes. The present dissertation focuses on such multiple systemic dysfunction through the concept allostatic load (AL). The concept states that chronic stressors, resulting from environmental or social factors, accumulate over time and lead to increasing AL and greater risk for poor health outcomes. The mechanism through which chronic stressors lead to increased AL is cumulative physiological dysregulation, where interconnected physiological systems, such as the cardiovascular, metabolic, immune, and inflammatory systems, become dysregulated (McEwen & Seeman, 1999).

The following section discusses the concept of AL. I first define allostasis and allostatic load, followed by the measurement of AL, and biomarkers of AL. I end with the justification for studying AL among adolescents and a review of the literature on AL and adolescents.

2.1.5.1. Definitions of Allostasis and Allostatic Load

Allostasis and allostatic load are defined best in comparison to homeostasis. The principle of homeostasis refers to the stability of essential physiological systems by holding internal parameters within a narrow set-point range, such as blood pH or internal body temperature (Cannon, 1932). However, constancy is not a fundamental condition for life (Sterling, 2004), and systems of the body must be flexible to adapt to constant changes in the environment. Stability through change is also known as allostasis (Sterling & Eyer, 1988). Allostasis states that bodily systems respond to predictable and unpredictable changes in the environment (Juster, McEwen, & Lupien, 2009). During allostasis, the body adapts by creating broader set-point ranges, such as changes in blood pressure.

When allostasis becomes dysregulated over prolonged periods of time, allostatic load occurs. AL is the wear and tear the body undergoes as it experiences stressful situations over time (McEwen & Stellar, 1993). As the body responds to numerous stressors over time, physiological systems become incapable of adapting to current or future stressors. Since the body relies on multiple interrelated physiological systems to exist, dysregulation of one or more of these systems over long periods of time leads to cumulative physiological dysregulation, and eventually negative health outcomes (Goldman et al., 2005; Seeman et al., 2001).

The concept of AL includes a multi-system approach to understanding how stressors affect cumulative physiological dysregulation. While large changes in the regulation of physiological systems increase risk for health disparities, small, moderate changes can also cause

substantial health effects (McEwen & Stellar, 1993). Thus, AL does not represent poor health, but rather puts individuals at greater risk for poor health outcomes (Gersten, 2008).

Allostatic load measures the collective consequences across multiple interconnected systems (Seplaki et al., 2004) through a series of cause and effect mechanisms. When an individual perceives a threat (be it acute or chronic) the sympathetic-adrenal-medullary (SAM) axis and the hypothalamic-pituitary-adrenal (HPA) axis release primary stress mediators, such as glucocorticoids (cortisol) and catecholamines (epinephrine, norepinephrine), which are chemical messengers that have widespread influence, triggering primary adaptive effects in the body that alter the function and structure of specific cells and tissues (Karlamangla et al., 2002). Secondary outcomes, such as changes in blood pressure, cholesterol, and blood glucose, occur when alterations in cell function and gene expression shift the production of biological parameters and 'set-point' ranges.

When a system becomes dysregulated for a prolonged period of time, other inter-connected systems also become affected. For example, the release of catecholamines causes the fight-or-flight response which induces an increase in heart-rate, blood vessel constriction, and increased oxygen to the body. Eventually allostatic overload occurs and causes damaging effects known as tertiary outcomes (Juster et al., 2009). The present dissertation uses both primary mediators and secondary outcomes to examine sociodemographic differences in AL among adolescents.

2.1.5.2. Measurement of Allostatic Load

As AL is comprised of primary mediators and secondary outcomes, numerous methods of AL measurement exist. To use AL as a tool in measuring subclinical dysregulation, various data reduction techniques have been proposed (Juster et al., 2009). The operationalization of AL was first formulated using ten biological parameters: systolic blood pressure, diastolic blood pressure,

waist-hip ratio, total cholesterol, glycosylated hemoglobin, chlorine, norepinephrine, epinephrine, high-density lipoprotein, and serum dihydroepiandrosterone sulfate (DHEA-S); with set quartiles based on the sample distribution and empirical evidence of health risk (Seeman et al., 1997). Participants that fell into a risky quartile for each biomarker received a score of one for that parameter. A composite AL score was created by summing the number of parameters that fell into a risky quartile, with AL values close to zero signifying few markers of systemic dysregulation.

Researchers continue to experiment with AL operationalization, adding commonly known biomarkers of health risk, such as triglycerides and interleukin-6 (Gruenewald et al., 2006; Seeman et al., 2008; Seplaki et al., 2005). Researchers are also challenged by classifying "risky" zones. While most studies choose extreme quartile values, others suggest using 2-tailed extremes and deciles (Seplaki et al., 2004), which may be advantageous in capturing a more comprehensive picture of physiological dysregulation. Additionally, researchers may consider cut-off values that differ for men and women, or by age, thereby creating an AL score specific to a population subgroup.

Previous studies among adolescents or young adults have differing AL scales based on six or even nine biomarkers (Evans, 2003; Evans et al., 2007; Goodman et al., 2005). Presently, there is no preferred set of biomarkers for use with adolescents, and there has yet to be standardizations in AL measurement among different demographic groups. The present dissertation attempts to formulate a measure of AL among adolescents using biological markers among a national sample, hopefully setting a standard by which future studies can begin to examine AL among adolescents.

Another challenge in AL operationalization is formulating the score. Within the summation AL score each biomarker is allotted an equal weight, suggesting that all markers of regulatory systems are equal. However, some systems may be better represented and weigh more heavily on a system than others. As a result, other techniques attempt to accurately calculate AL, such as the "z-score AL index", which is a summary measure that represents the sum of an individual's z-scores from each biomarker based on the population distribution of biomarker values (Juster et al., 2009). Rather than examining AL as a predictor of health outcomes, the current research explores sociodemographic patterns of AL among adolescents, utilizing the summation AL score to represent adolescent's overall cumulative biological risk profiles and subclinical health status.

2.1.5.3. Biomarkers of Allostatic Load

The current dissertation explores primary mediators and secondary outcomes to determine the level of cumulative physiological dysregulation, and thus, uses AL as an indicator of such dysregulation among adolescents. Below I elaborate on relevant primary mediators and secondary outcomes according to their involvement in specific regulatory systems: immune/inflammatory, metabolic, and cardiovascular; providing a rationale for their use as a biological marker of stress within the measure of AL for the present dissertation.

2.1.5.3.1. Cardiovascular System Biomarkers

The amount of pressure the blood exerts on the vessel walls is indicative of cardiac activity. Correspondingly, diastolic blood pressure (DBP) is a marker of the minimal force exerted by blood against the vessel walls when the left ventricle is relaxed as in diastole, and systolic blood pressure (SBP) represents the maximal force exerted by blood on the vessel walls when the left ventricle is contracting as in systole. Hypertension, also known as high blood pressure, is a major risk factor for heart disease, stroke, and kidney disease (Centers for Disease

Control and Prevention, 2011a). Among adolescents, elevated blood pressure is strongly associated with obesity (Ostchega et al., 2008).

Low childhood SES and adverse family environments are associated with elevated risk for hypertension over time (Karlamangla et al., 2005; Lehman et al., 2009). The present research uses the secondary outcome of blood pressure as a useful indicator of the cardiovascular system. Higher blood pressure is indicative of cardiovascular dysregulation, and contributes to overall cumulative physiological dysregulation.

2.1.5.3.2. Metabolic System Biomarkers

Commonly used metabolic markers in formulating AL score include body mass index (BMI), high-density lipoprotein (HDL), total cholesterol, waist circumference, and glycosylated hemoglobin (HbA1c). Body mass index is a measure of body mass fat percentage. BMI ranges on a spectrum from severely underweight to differing levels of obese. Waist circumference is a measure of adipose fat, and is associated with type II diabetes, hypertension, and heart disease (National Institutes of Health, 1998). The current dissertation uses BMI and waist circumference as secondary outcomes to determine the functioning of metabolic processes. Higher BMI and waist circumference values contribute to metabolic dysfunctioning and overall physiological dysregulation.

Other markers including cholesterol and blood glucose are also secondary outcomes of metabolic dysregulation. Cholesterol is a fat-like substance produced by the liver that functions in providing strength to cell membranes. To mediate transport to and from tissues, cholesterol is joined with lipoprotein. Persistently high levels of contribute to plaque development and eventual heart disease (Libby, 2007; Stamler et al., 2000). HDL promotes good vascular health by clearing excess cholesterol build-up in plaques and delivering it back to the liver. HDL is an

inverse predictor of cardiovascular events (Castelli et al., 1977). The current research uses the secondary outcomes of cholesterol and HDL as measures of metabolic functioning, which contribute to the overall measure of AL. Higher values of cholesterol, and lower values of HDL, are indicative of metabolic dysregulation.

In addition to cholesterol, another biomarker of metabolic functioning is glycosylated hemoglobin (HbA1c). HbA1c is a molecule that reflects the mean blood sugar concentration over several months, not just at the time of measurement (Koenig et al., 1976). Hyperglycemia, known as high blood glucose, can lead to an increased risk for type II diabetes. This dissertation uses the secondary outcome measure of HbA1c as an indicator of metabolic functioning; further contributing to overall cumulative physiological dysregulation and AL score. In sum, the present study uses primary mediators and secondary outcomes such as those listed above to explore the functioning of the immune, cardiovascular, and metabolic systems in order to measure overall cumulative physiological dysregulation among adolescents.

2.1.5.3.3. Immune/Inflammatory System Biomarkers

C-reactive protein (CRP) is a primary mediator that reflects immune system regulation. A well-known marker, CRP, is linked to numerous negative health outcomes later in life such including cardiovascular disease and depression (Miller, Freedland, & Carney, 2005; Ridker, 2001). CRP is a measure of underlying inflammatory response, and is a useful indicator of immune injury to tissue as it amplifies secretion of pro-inflammatory molecules that further contribute to negative health outcomes if levels remain consistently high (Black, Kushner, & Samols, 2004). Low childhood SES is related to increased CRP in adulthood (Taylor et al., 2006), in addition to adolescent stress (Fuligni et al., 2009). The present dissertation uses CRP as a primary mediatory to measure regulation of the immune system.

Assembled in the liver, albumin is the most abundant protein in the blood, and plays a key role in immune system functioning as a secondary outcome. When the body is infected by an agent, the immune system produces more protein-based cells to fight the intruder. As the concentration of immune system proteins goes up, the concentration of albumin declines. Lower levels of albumin are linked with cancer, increased risk for heart disease, and are a predictor of mortality (Goldwasser & Feldman, 1997; Phillips, Gerald Shaper, & Whincup, 1989). The present dissertation uses albumin as a secondary outcome due to its advanced measurement of immune system regulation.

2.1.5.4. Scientific Justification for Studying Allostatic Load among Adolescents

Although many studies have examine AL, much of the prior research has focused on specific groups such as adults, the elderly, and women (Allsworth et al., 2005; Chyu & Upchurch, 2011; Crimmins et al., 2003; Geronimus et al., 2006; Karlamangla et al., 2002; Peek et al., 2010; Seeman et al., 2010a; Seeman et al., 2001; Seeman et al., 2002; Taylor et al., 2011). Such findings showed that physiological systems experience acute and chronic wear and tear, contributing to increasing levels of AL with age, thus reducing one's ability to adapt to future life stressors (Crimmins et al., 2003; Crimmins & Seeman, 2004). Research by Crimmins and colleagues using NHANES found that among adults, mean AL increases with age, supporting the idea that cumulative dysregulation occurs over time (Crimmins et al., 2003).

Prior research also demonstrates how certain demographic groups experience increasing AL more so than others. Black women have the highest AL score among other racial/ethnic and gender groups (Chyu & Upchurch, 2011; Geronimus et al., 2006), and Hispanic adults have AL scores significantly lower than Whites. AL scores are also shown to increase among adults with lower educational status and income; and to increase steadily with age from adulthood into the aging years (Chyu & Upchurch, 2011; Crimmins et al., 2003).

Geronimous et al. (2006) used AL to measure the cumulative wear and tear of prolonged sociodemographic hardship. They found that Blacks age 18-64 had higher AL scores at all ages than Whites; this was true even after controlling for poverty (Geronimus et al., 2006). These results support the concept that AL differs across racial/ethnic groups among adults, and that physiological dysregulation can occur at earlier ages for minority populations such as Blacks. Thus, AL may be valuable in identifying the physiological mechanism through which sociodemographic hardships across the life course contribute to health disparities.

While prior research among adults and women has highlighted disparities in AL by race/ethnicity, SES, nativity status, and age, the need to explore AL among younger populations is emerging. Prior research on AL during the period of adolescence remains scarce. Examining the physiological regulation of adolescents across gender and racial/ethnic groups can further our understanding of health disparities across the life course, help identify adolescents at risk for high AL, and inform healthy adolescent physiological and psychosocial development.

To date, there are no studies examining child or adolescent racial differences in allostatic load. A few noteworthy researchers have begun to uncover associations between adolescent health and behavior and AL. Recent studies have attempted to undertake AL among younger populations by examining relative risk (Evans et al., 2007; Goodman et al., 2005). Evans found that middle school children exposed to greater accumulated psychosocial and physical risk factors (such as crowding, substandard housing, noise, and poverty) have higher levels of AL; these effects remained consistent over time (Evans, 2003). Goodman and colleagues found that social inequalities, such as parental education, are associated with metabolic and cumulative risk among a small sample of suburban Midwestern adolescents (Goodman et al., 2005). Both studies

used small convenience samples, and did not examine sociodemographic profiles of AL during adolescence.

The empirical evidence suggests that studying sociodemographic differences in AL among younger populations is useful in understanding the physiological mechanisms of how early life social conditions contribute to health trajectories over the life course. This study examines AL among a nationally representative sample of adolescents. By demonstrating that differences in AL exist among a young population of adolescents, the present research sets a foundation for exploring adolescent biological markers of health as indicators of future adult health outcomes

2.1.6. Longitudinal Obesity Patterns and Stress

In addition to examining sociodemographic differences in AL among adolescents, the present dissertation sought to extend this relationship one step further by exploring the association between adolescent stressful life events and AL in young adulthood. However, due to the unpredictability of the release of Add Health biomarker data, such objectives could not be met. Thus, the current dissertation examines the relationship between adolescent stressful life events and longitudinal patterns of obesity during the transition to young adulthood.

A significant public health concern (Centers for Disease Control and Prevention et al., 2011e), the prevalence of obesity among youth and adults has reached epidemic proportions (Block, 2004; Flegal et al., 2002; Jolliffe, 2004; Kimm & Obarzanek, 2002; Ogden et al., 2002). Obesity prevalence among adolescents age 12 to 19 years has more than tripled in the past thirty years (Ogden, Carroll, & Flegal, 2008). Recent studies on longitudinal trends in obesity using Add Health also show that a substantial amount of weight is gained during the transition to

adulthood (Gordon-Larsen et al., 2004), with obesity prevalence doubling from adolescence to the early 20s, and again to the early 30s (Gordon-Larsen et al., 2010).

Obesity etiology is based on the imbalance of energy intake relative to energy expenditure. A biological explanation reveals that the body's stress response via the HPA axis stimulates the release of glucocorticoids (cortisol) that may disrupt the regulation of food intake (stimulate appetite) and food reduction in the body (Torres & Nowson, 2007). Thus, a prolonged stress response could result in long-term increased energy intake, dysregulated energy reduction, and overall fat accumulation (Rosmond, Dallman, & Bjorntorp, 1998; Torres & Nowson, 2007). A positive energy balance is therefore responsible for fat accumulation over time, and subsequent obesity (Dallman, Pecoraro, & la Fleur, 2005; Moreno & Rodriguez, 2007).

Numerous forms of stress contribute to obesity. Obesity is associated with work stress (Kivimaki et al., 2006; Kouvonen et al., 2005) and early childhood trauma (Gunstad et al., 2006). Stress created by racial discrimination is also associated with increased obesity (Gee et al., 2008). Recent findings also show that SLE are significantly associated with obesity; specifically, obese and extremely obese individuals experience SLE more so than non-obese individuals (Barry & Petry, 2008).

Highest risk of obesity incidence is found among adolescent males from disadvantaged backgrounds who experience life transitions early, such as entering the work force and marriage. Adolescent females exposed to enduring life challenges also tend to have a high risk of obesity (Scharoun-Lee et al., 2009). The present dissertation builds upon this previous research to examine the relationship between adolescent stressful events and longitudinal patterns of obesity from adolescence to young adulthood.

Significant racial/ethnic, gender, and SES disparities in obesity among adolescents also exist. Among females, Black adolescents are significantly more likely to be obese than Whites (Ogden et al., 2012). Gordon-Larsen and colleagues (2010) found that Black females start off with a higher proportion of obesity, and have higher annual incidence rates of obesity (Gordon-Larsen et al., 2010). Among males, obesity prevalence is significantly higher among Hispanic adolescents compared to Whites.

Rates of obesity are also highest among the most disadvantaged population groups, such as those with the highest poverty rates and the lowest education levels (Drewnowski & Specter, 2004). Such inverse relationships of SES and obesity among adolescents are variable by race and gender (Wang & Zhang, 2006), with a stronger inverse relationship for females compared to males (McLaren, 2007; Scharoun-Lee et al., 2009; Wang, 2001; Zhang & Wang, 2003). The present study further explores the associations of SES and obesity separately for females and males.

Recent work by Lee and colleagues (2009) examined the mediating mechanisms through which low SES in adolescence, such as poverty, may operate on the progression of obesity (Lee et al., 2009). They found that parental monitoring, physical activity, skipping breakfast, and insufficient sleep are significant intervening factors among females. Adolescents in low SES households are less likely to grow up among a stable and supportive parental environment. Poor parents or single parents are less able to monitor their children's dietary patterns, physical activity, and other health behaviors due to stress and extended working hours. Low SES children also have fewer opportunities to exercise outdoors as a result of unsafe neighborhood environments.

Empirical evidence shows that adolescent SLE and sociodemographic factors are significantly associated with obesity in adolescence and adulthood, and vary greatly by gender. However, a model exploring the intervening mechanism of SLE between adolescent sociodemographic characteristics and obesity progression has yet to be proposed. Built upon the life course perspective, social determinants of health, and stress process models, the current dissertation examines whether greater SLE exposure as a result of social conditions in adolescence contributes to an increased risk of obesity from adolescence to adulthood.

2.1.6.1. Adolescent Measurement of Obesity

The most commonly used measure of classifying obesity is through one's body mass index (BMI). BMI is a measure of weight adjusted for height, and is calculated by dividing weight in kilograms (kg) by height in meters squared (m^2). While often used, BMI does not differentiate between individuals that have a high BMI as a result of excessive muscle mass (such as professional athletes) or fat mass. Despite the small percentage of individuals that are misclassified, the majority of individuals with high BMI have excess body fat. Though a number of methods of measuring adipose fat may be more accurate than BMI, such as full body X-ray scans (Sopher, Shen, & Pietrobelli, 2005), they also have limited relevance in obesity screening and large population survey samples. Obtaining height and weight measurements is simple, economical, and non-invasive. BMI is also highly correlated with these more direct fat measures (Pietrobelli et al., 1998; Willett, 1998).

Researchers frequently use BMI to examine obesity status at different stages in the life course. Previous studies on obesity prevalence consider adults separately from children and adolescents (Flegal et al., 2002; Ogden et al., 2012; Ogden et al., 2002). Among adults, BMI cut-points defining obesity (BMI of 30 kg/m^2 or higher) are standard, regardless of age and gender

(Pi-Sunyer et al., 1998). Whereas among children and adolescents (who are developmentally growing), BMI varies by age and gender. For example, a 12-year-old girl with a BMI of 27 kg/m^2 is considered obese, while a 19-year-old girl with a BMI of 27 kg/m^2 has a normal weight.

Thus, for BMI to be a significant indicator of obesity among adolescents, researchers use a reference standard that accounts for child and adolescent gender and age (Must & Anderson, 2006). Age- and gender-specific BMI percentiles are therefore used as cutoffs during childhood and adolescence. In the U.S., BMI-for-age reference standards by gender are based on nationally representative data from the 2000 growth curves of the Centers of Disease Control and Prevention (Dinsdale, Ridler, & Ellis, 2011; Kuczmarski et al., 2000). CDC growth reference charts define children and adolescents as obese if their BMI exceeds the 95^{th} percentile (Centers for Disease Control and Prevention, 2000; Kuczmarski et al., 2000).

Based on prior research, the present dissertation examines obesity patterns over time from adolescence to young adulthood, taking into account differing classifications of obesity for adolescents and adults. By examining obesity classification during two different stages in the life course, the present study provides an accurate investigation of the onset of obesity during the transition to adulthood.

2.1.7. Measurement and Methodological Issues

Although the literature has demonstrated the importance of early life social determinants of health contributing to health trajectories across the lifespan, research is still hindered by the lack of representative measurement of adolescent cumulative physiological dysregulation, and the mechanism through which adolescent stressors influence longitudinal patterns of obesity during the transition to adulthood.

Specifically, research on AL has been limited to adult and aging populations. While useful in demonstrating the link between stress accumulation over the life course and morbidity and mortality at older ages, these studies neglect to consider the formation of dysregulated physiological systems at a younger age. Because the period of adolescence is such a critical developmental stage filled with hormonal, physical, cognitive, and emotional changes significant in formulating health behaviors and health trajectories, a closer examination of biological markers of stress at this period is warranted.

Additionally, previous research of AL among adolescents has been based solely on convenience samples; these findings are not representative of the U.S. adolescent population. The continuous cross-sectional NHANES includes detailed biomarkers among the adolescent population, representative of the regulation of multiple physiological systems. Such rich biomarker information allows investigators to more effectively measure AL among younger populations. One of the first to provide descriptive findings of cumulative physiological dysregulation at such a young age, the current study empirically explores sociodemographic differences in AL among a representative sample of adolescents in order to demonstrate the importance of physiological dysregulation and early life health trajectories across the lifespan.

In addition to examining AL among adolescents, the current study seeks to investigate the relationship between adolescent sociodemographic characteristics and longitudinal patterns of obesity over time. Much of the prior obesity research has examined obesity status at a specific point in time (Ogden et al., 2008; Ogden et al., 2012; Ogden et al., 2002). This approach allows researchers to cross-sectionally study obesity prevalence among a population, but neglects to consider how obesity may change over time.

Select studies have begun to use longitudinal data to investigate the change in obesity patterns over time within the same population (Gordon-Larsen et al., 2004; Gordon-Larsen et al., 2010; Lee et al., 2009), enabling researchers to model the temporal ordering of predictive factors on subsequent obesity. The Add Health includes multiple measures of BMI over time, in addition to thorough social, contextual, and environmental factors. By using multiple measures of BMI over time, the present dissertation examines the dynamics of obesity status from adolescence into young adulthood, and explores pathways of body mass change during a period of increased risk for obesity development. The current study uses obesity data from two points in time, developing measures that represent continuity and change in obesity status from adolescence into young adulthood.

The present dissertation also models the theoretical mechanism through which adolescent sociodemographic characteristics may operate to influence obesity transitions. Earlier literature has shown that patterns of obesity during the transition to adulthood vary by gender, race/ethnicity, and SES (Lee et al., 2009; Scharoun-Lee et al., 2009), but no studies to date have explored the mediating pathway of adolescent SLE on these effects. By using Add Health's diverse measures of stressful life events, the present study explores the role of adolescent SLE as a mediating mechanism to demonstrate how adolescent sociodemographic characteristics influence obesity progression from adolescence to young adulthood. As prior studies indicate sociodemographic differences in obesity by gender, the current research contributes further to this literature by exploring how the mediating mechanism of adolescent SLE operates differently on obesity patterns for females and males.

In summary, this dissertation addresses these measurement and methodological issues and thereby extends the research in this area. Using NHANES, an array of biological markers of

cumulative physiological regulation among adolescents is examined, demonstrating the necessity to study the critical developmental period of adolescence as a significant contributor to health behaviors and health trajectories across the life course. In addition, using Add Health, longitudinal patterns of obesity during the transition to adulthood and the mediating mechanism of adolescent SLE are investigated to assess whether adolescent social conditions lead to greater exposure of stressful life events and subsequent changes in obesity status over time.

2.2. Conceptual Framework

2.2.1. Overview

The current research utilizes key aspects of the life course perspective to provide the overarching framework for conceptualizing the link between adolescent sociodemographic characteristics, biological markers of physiological dysregulation, stressful life events, and longitudinal patterns of obesity. Theories of social stratification and stress process are also incorporated to interpret the key associations. As the present dissertation utilizes two distinct datasets, the following section provides an overview of two separate conceptual models, each of which is followed by a justification for present dissertation hypotheses.

2.2.2. NHANES Conceptual Model

Figure 2.1 illustrates the conceptual approach using NHANES, and underlines the major constructs used in this study. This framework views adolescent sociodemographic characteristics as social determinants of health, significantly influencing physiological systemic regulation during adolescence (this is explored in Aim 1). Adolescents are shaped by the environmental and social conditions around them, cross-cutting multiple organizational levels. In the current study, demographic characteristics including age, gender, race/ethnicity, and nativity status represent an

adolescent's position within society, including social roles and expectations, which influence both the opportunities that become available to adolescents and their ability to make decisions that lead down a path of positive or negative health trajectories.

In addition, an adolescent's family background also act as social determinants of health. Specifically, parental education level and family income reflect an adolescent's access to resources, further contributing to an adolescent's ability to cope with life's barriers and make positive health decisions. In the present dissertation, these sociodemographic characteristics are hypothesized to shape adolescent health development trajectories over the life course.

This framework also emphasizes how health development trajectories are formed through inner biological pathways, representing individual and interconnected physiological regulatory systems. The present research examines levels of specific biomarkers within three physiological regulatory systems: 1) cardiovascular markers including systolic and diastolic blood pressure; 2) metabolic markers including total cholesterol, high-density lipoprotein, waist circumference, body mass index, and glycosylated hemoglobin; and 3) immune/inflammatory markers including C-reactive protein and albumin. Figure 2.1 depicts the interconnection of these physiological systems through multiple double-headed arrows.

As an adolescent's social conditions influence access to resources and the ability to make positive health decisions, individual physiological regulatory systems become affected. Dysregulation of multiple biomarkers within a single system are posited to influence additional regulatory systems. The current research utilizes the measure of AL as an indicator of this network of systemic dysregulation. Individual biomarkers that fall into a risky quartile range are identified as dysregulated. Thus, AL is a count of the number of dysregulated biomarkers.

2.2.2.1. NHANES Hypotheses

Allostatic load is shown to increase with age from adulthood onward, demonstrating the age-effect of dysregulated physiological systems (Seeman et al., 2001; Seeman et al., 1997; Seplaki et al., 2005). As health trajectories are formulated from the variation in adolescent development (Cobb, 2010; Santrock, 2010), the current dissertation expects increasing AL with age across adolescence from 12 to 19 years.

Developmental changes during adolescence also occur differently for females and males, creating further variation in the experiences of adolescence. Previous research has shown that adult females experience higher levels of cumulative dysregulation through the indicator AL, than males (Chyu & Upchurch, 2011; Crimmins et al., 2003; Geronimus et al., 2006), suggesting a similar pattern among adolescents may also exist. In the present research, levels of AL are anticipated to differ by gender, with levels higher for females than males.

Experiences and development during adolescence also vary by race/ethnicity. Black and Hispanic adolescents often experience limited access to resources, increasing the risk for negative health behaviors and health trajectories at a young age (Mulye et al., 2009). AL has been shown to differ significantly by race/ethnicity among adults, with higher levels among Blacks than Whites (Geronimus et al., 2006). The weathering hypothesis also suggests that physiological deterioration occurs at an earlier age among Black adults compared to Whites, leading to worse health across the life span (Geronimus et al., 2006).Similarly, in the current study, Black and Hispanic adolescents are anticipated to have higher levels of AL compared to Whites.

Adolescent nativity status may also affect health trajectories across the life course. Given the Hispanic paradox, a result of selective in-migration patterns of healthy individuals and out-

migration of sick individuals (Crimmins et al., 2005; Palloni & Arias, 2004), no studies to date have examined the effects of adolescent nativity status on physiological markers of systemic regulation during adolescence. A unique strength of the current research is its first look at such a relationship, hypothesizing that foreign born adolescents have lower levels of AL compared to U.S.-born adolescents, indicative of the protective effect of the Hispanic paradox even at a young age.

Socioeconomic status during adolescence significantly contributes to variations in adolescent adverse experiences, which can improve or impede health behavior and health trajectories across the lifespan (Phelan et al., 2010). Lower parental education level and lower family income are also significantly associated with higher AL among both adolescents (Goodman et al., 2005) and adults (Crimmins et al., 2003; Seeman et al., 2004; Seeman et al., 2010a). Thus, the present dissertation hypothesizes that lower parental education and family income is associated with higher levels of AL during adolescence.

Last, a unique strength of the present research using NHANES is that adolescent cumulative physiological dysregulation is hypothesized to increase with age across adolescence, varying by race/ethnicity. As AL is expected to increase with age during adolescence, and race/ethnicity is projected to influence levels of AL during this transitional period, the effect of age on AL is expected to vary by race/ethnicity. Specifically, Black adolescents are anticipated to have higher AL scores across all ages of adolescence than Whites.

2.2.3. Add Health Conceptual Model

Figure 2.2 illustrates the conceptual approach using Add Health, and underlines the construct of health trajectories over the lifespan in combination with stress exposure. Similar to the previous conceptual model, this framework views adolescent sociodemographic

characteristics as social determinants of health, but these factors are hypothesized to significantly influence patterns of obesity from adolescence to young adulthood (this is explored in Aim 2). In the present research, longitudinal patterns of obesity over time are representative of health development trajectories across the life course, and are measured by the change in obesity status across Waves II and III of Add Health, a period of increased risk for obesity development.

Four obesity patterns are recognized: 1) becoming obese (not obese in Wave II and obese in Wave III); 2) staying obese (obese in Waves II and III); 3) reducing obesity (obese in Wave II and non-obese in Wave III); and 4) staying non-obese (not obese in Waves II and III). Demographic characteristics in the current study are indicative of individual social conditions and identity. In Add Health, such factors are similar to those in NHANES, and are represented by age, gender, race/ethnicity, and nativity status, which influence an adolescent's health behavior and health trajectories. However, Add Health provides more detailed family background information, with the present framework incorporating family structure in addition to parental education level and family income.

Figure 2.2 also builds upon the stress process model, which emphasizes that life's stressors are influenced by the social and environmental contexts within our lives (Pearlin, 1989, 1999; Pearlin et al., 1981; Pearlin et al., 2005; Thoits, 1995, 2010). Thus, an adolescent's sociodemographic characteristics are anticipated to expose adolescents to greater or lower levels of stress (this is explored in Aim 3). The current study measures stress through the accumulation of an array of stressful life events that occurred within the past year of Add Health Wave I, representing acute events during a significant transitional period in the life course. Additionally, the present study discerns as stressful events occurring *to* an individual from those performed *by* an individual.

Empirical generalizations from prior research also suggest that differential exposure to stressors during adolescence contribute to health disparities (Aneshensel, 1992; Thoits, 1999; Thoits, 1983, 1995, 2010, 2011). The current framework posits that SLE variation during adolescence influences longitudinal patterns of obesity (this is also explored in Aim 3). The timing of SLE exposure, specifically during the transitional period of adolescence, significantly affects health development trajectories of obesity status across the lifespan.

By fusing the life course perspective, stress process model, and social determinants of health frameworks together, Figure 2.2 also illustrates how adolescent SLE explain the relationship between adolescent sociodemographic factors and longitudinal patterns of obesity during the transition to adulthood (also explored in Aim 3). Adolescent sociodemographic factors are superimposed on the stress process, affecting its components and the relationships among them, encircling stressful life events and varying exposure to stress. This variation in stress exposure leads to positive or negative health trajectory patterns, such as obesity progression and continuity. This exploration of the mediating mechanism of SLE contributes to understanding how sociodemographic characteristics influence obesity in adolescence and its trajectory into young adulthood by examining the role of adolescent SLE.

2.2.3.1. Add Health Hypotheses

Previous research shows that females are more likely to become obese compared to males (Gordon-Larsen et al., 2004; Gordon-Larsen et al., 2010). Black female adolescents are more likely to be obese than Whites (Ogden et al., 2012). Racial/ethnicity disparities in obesity during the transition to adulthood also show that Hispanic males have a higher obesity prevalence than. In the present research, the risk of becoming and staying obese over time is expected to increase with age, more so for females than males. Blacks and Hispanics are anticipated to have a higher

prevalence of becoming obese and staying obese over time compared to Whites for both females and males.

Immigrant adolescents have also been shown to have a lower obesity prevalence compared to their U.S.-born counterparts (Singh, Kogan, & Yu, 2009), thus the current study expects foreign born adolescents will be less likely to become and stay obese relative to U.S.-born adolescents. An inverse relationship of SES and obesity among adolescents also exists (Wang & Zhang, 2006), more so for females than males (Scharoun-Lee et al., 2009). Similarly, this study expects an inverse relationship between SES and obesity patterns. Lower parental education and family income are also hypothesized to increase the risk of becoming and staying obese, more so for females than males.

The present research also takes into account adolescent family composition, as family structure plays a significant role in family socioeconomic status and thus health trajectories across the life course. Intact families with two biological parents are likely to have a higher family income, access to greater resources, and provide more support, monitoring, and necessary attention to children and adolescents than non-intact families. Thus, it is anticipated that adolescents in non-two-biological-parent families, such as step-families, single parent households, or other situations will have a higher risk of becoming and staying obese over time.

The location of an individual within the social hierarchy influences the probability of being exposed to certain types of stressors, with variation in stress exposure consequently contributing to disparities in health outcomes (Pearlin, 1989, 1999; Pearlin et al., 1981; Pearlin et al., 2005; Seplaki et al., 2004). Therefore, the present dissertation hypothesizes that adolescent females, those who are Black or Hispanic, and who live in low SES and non-two-biological-

parent households will experience higher numbers of SLE done *to* them and performed *by* them during adolescence.

Prior research has shown that obese individuals experience SLE more so than non-obese individuals (Barry & Petry, 2008); and adolescent females exposed to continuing life challenges also have an increased risk for obesity (Scharoun-Lee et al., 2009). The present study hypothesizes that greater numbers of SLE done *to* and performed *by* adolescents will be significantly associated with a greater risk for becoming and staying obese over time.

Last, adolescents are exposed to greater numbers of stressful events as a result of the socioeconomic and demographic factors that shape their lives. And given the transitional period of adolescence, the timing with which these stressful events occur contributes to the variation in health development trajectories across the life course. Thus, varying patterns of obesity by adolescent sociodemographic characteristics during the transition to adulthood are expected to be explained through the mechanism of stress exposure and SLE during adolescence. The risk of becoming and staying obese from adolescence to young adulthood is therefore anticipated to increase with greater numbers of stressful events experienced or performed, as a result of specific sociodemographic characteristics increasing exposure to stress during such a transitional developmental period in the life course.

2.3. Overview of Present Study

The present study builds on previous work of early life adversity and physiological dysregulation, and adolescent stressful life events and obesity, but its focus differs in some essential ways. First, this dissertation focuses on the adolescent population to examine sociodemographic differences in AL across adolescence from age 12 to 19 years; a population

yet to be explored within the literature of cumulative physiological dysregulation. Second, I use a longitudinal design to examine the relationship between SLE and obesity transitions beginning in adolescence through the transition to young adulthood. Previous research has examined the intervening mechanisms between adolescent SES and progression to obesity from adolescence to adulthood without considering stressful life events in adolescence. This study is the first to investigate such a mechanism.

Figure 2.1. NHANES Conceptual Model

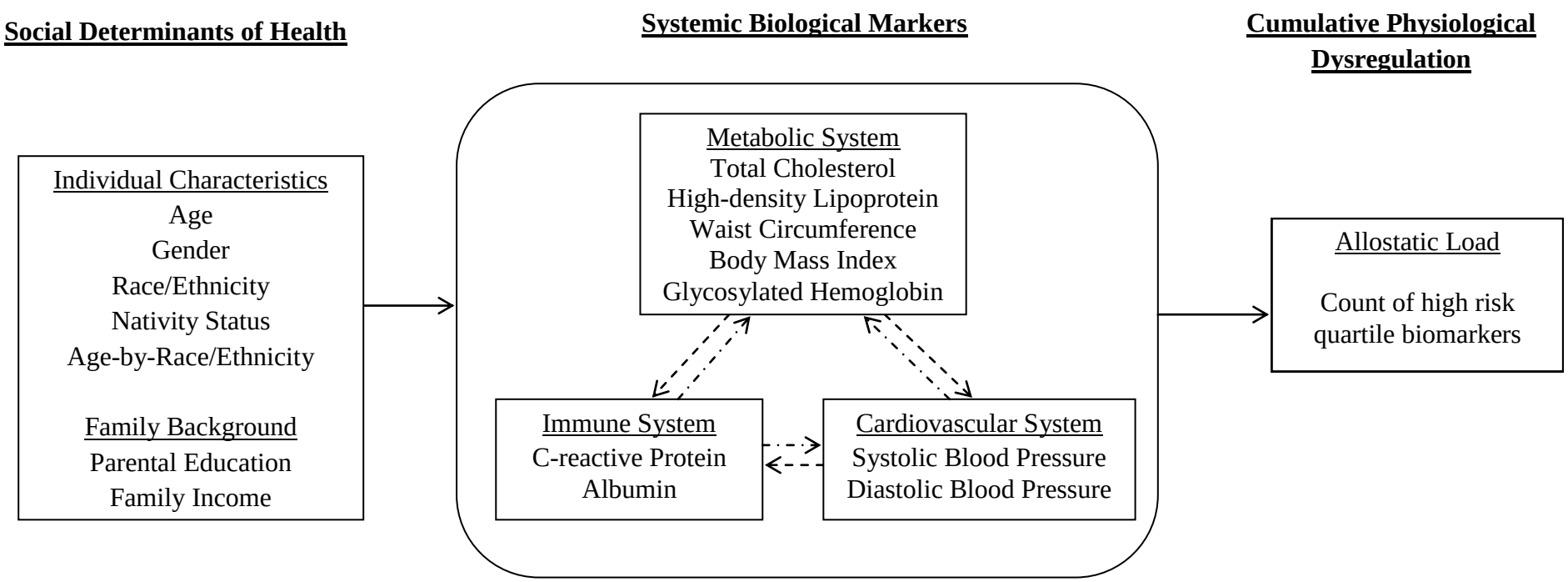
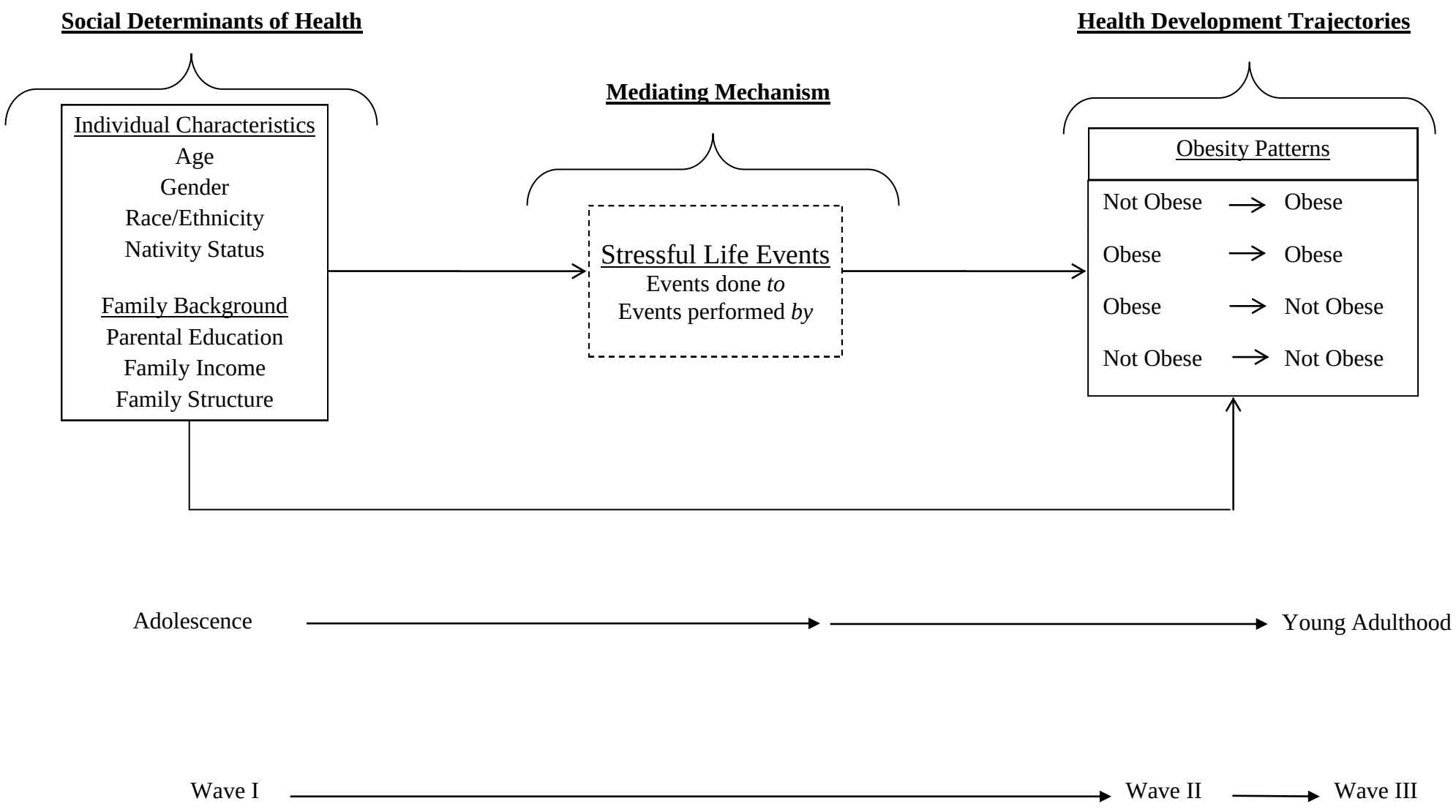


Figure 2.2. Add Health Conceptual Model



CHAPTER 3

RESEARCH DESIGN AND METHODS

3.1. Overview

The purpose of this chapter is to elaborate the specific study designs, survey methods, data description, and techniques used in the present dissertation. The following chapter is divided into three major components: 1) a description of the National Health and Nutrition Examination Survey (NHANES) study design and sampling strategy, followed by details on data collection and protocol, response rates, operationalization of study variables, the analytic sample, and selection analysis; 2) a similar discussion of the National Longitudinal Study of Adolescent Health (Add Health); and 3) a detailed explanation of the analytical techniques and strategies used to achieve each study aim.

3.2. National Health and Nutrition Examination Survey (NHANES)

Aim 1 of this dissertation focuses on the change in levels of AL by age and race/ethnicity across adolescence among a nationally representative sample of adolescents age 12 to 19 years. The data for this first aim come from NHANES. NHANES is administered by the National Center for Health Statistics (NCHS) and is a bi-annual cross-sectional survey that monitors the health status of the U.S. civilian, non-institutionalized population of all ages, from infants to the elderly. The goal of NHANES is to monitor trends in health status, risk behaviors, environmental exposures, and risk factors to explore emerging public health issues. The survey includes in-home interviews of sociodemographic information and physical examinations conducted in fully equipped medical examination centers providing detailed measurements of physiological systemic regulation. This dissertation uses data from NHANES in-home interviews and

biological measures from physical examinations. All NHANES protocol follow NCHS guidelines for personal safety and received approval from the institutional review board. Due to public access of NHANES data, the present dissertation did not require approval from the University of California institutional review board.

3.2.1. Study Design and Sampling

The continuous NHANES beginning in 1999 used a complex, multistage, probability sampling design comprised of 4 stages. Stage 1 selected primary sampling units (PSUs). These units were mostly single counties, or in a number of cases, groups of neighboring counties with probability proportional to a measure of size (PPS). The second stage divided the PSUs into sections by city blocks; sample segments were also selected according to PPS. Stage 3 listed households within each section, and a random sample of households was then drawn. Geographic areas where the proportion of age, ethnic, and income groups were a minority were oversampled, such as counties with high proportions of non-Hispanic Blacks, Mexican Americans, low income non-Hispanic Whites, adolescents aged 12 to 19 years, and persons over the age of 60.

In the final stage, individuals of any age (from infants to the elderly) were chosen to participate in the study from a list of all persons living in the household. Individuals were randomly selected within specified gender, race/ethnicity, and age sub-categories (Centers for Disease Control and Prevention, 1999-2008a). Selected individuals were then screened to participate in the study, consenting to an in-home interview component followed by a physical examination (discussed in more detail in the following section). As NHANES is a bi-annual survey, every two year cycle the complex, multistage sampling design occurred.

Approximately 12,000 respondents are screened to participate in NHANES during a cycle. The sample design for NHANES makes it possible to combine two or more survey cycles to increase the sample size and analytic power. Each two-year cycle and any combination of two-year cycles are nationally representative. As recommended by NCHS to increase statistical stability, the proposed study utilizes data from five NHANES cross-sectional cycles from 1999-2008 (Centers for Disease Control and Prevention, 1999-2008a).

3.2.2. Data Components

The interview and physical examination components of the NHANES survey combined to collect data pertaining to the following health-related areas: cardiovascular disease, vision, hearing, mental illness, infectious disease, obesity, dietary behavior, nutrition, disability, physical fitness, and other related health topics. Below I discuss the interview and physical examination components, highlighting the details of specific biomarker collection and protocol used for the present study.

Randomly selected individuals were first interviewed in their homes. Household interview data were collected via computer-assisted personal interviewing (CAPI) and included questions on demographics, socioeconomic status, dietary, and health-related questions. Participants under the age of 18 had a proxy adult complete household socioeconomic information (Centers for Disease Control and Prevention, 1999-2008a). English-Spanish bilingual interviews were also conducted by trained bilingual staff. The present research uses sociodemographic information collected from adolescent participants and their proxy adult during the in-home interview of NHANES as independent variables to examine the sociodemographic differences in adolescent allostatic load.

Individuals consented to participate in the interview and physical examination components separately. The physical examination occurred within one to two weeks after the in-home interview. Those who refused to consent to the physical exam were excluded from the examination phase of the survey. Physical examinations were conducted in a specially equipped and CDC designed medical examination centers (MEC) that traveled to survey locations throughout the nation (Centers for Disease Control and Prevention, 1999-2008c). The MEC operated five days a week, and included a variation of weekday, evening, and weekend sessions. The MEC contained state-of-the-art exam equipment, and was divided into private rooms for individual examinations. The full exam lasted approximately three-and-a-half hours, and depended on the study participant's age. The examination included a physical and dental exam conducted by a physician and dentist, laboratory tests, and a variety of physical measurements (Centers for Disease Control and Prevention, 1999-2008c). The current study uses biological markers collected during the examination component to formulate the outcome measure of allostatic load.

During the MEC exam, participants also completed additional survey questions including a dietary questionnaire, and other special topics conducted by highly trained medical personnel. Standardized procedures and protocols were developed and validated by NCHS for all clinical exams and laboratory tests (Centers for Disease Control and Prevention, 1999-2008b, 1999-2008c). To ensure quality control, roughly 5% of study participants aged 12-69 were asked to return for a second MEC exam.

3.2.2.1. Biomarker Collection and Protocol

As the present research uses biological markers of physiological systemic regulation to create the measure of AL, a detailed description of the collection, protocol, and analysis of each

biological marker is warranted. The examination component of NHANES, performed in the MEC, incorporated the collection and recording of anthropometric measures, as well as the processing and shipment of participant biological blood and urine samples to analytic laboratories. All anthropometric readings were done on routinely calibrated instruments by trained examination staff according to NCHS protocol (Centers for Disease Control and Prevention, 1999-2008b).

Anthropometric measures collected during the MEC exam were managed by the NCHS Integrated Survey Information System (ISIS) to accurately store and record participant examination and laboratory results (Centers for Disease Control and Prevention, 1999-2008c). The ISIS database also allowed examiners to record MEC component data electronically using complex instrumentation. The ISIS database used built-in quality control checks to flag unusual entries for verification. After each examination session, the ISIS database sent all participant data to a central survey database that was then forwarded to the NHANES headquarters office (Centers for Disease Control and Prevention, 1999-2008c). ISIS provided protection of participant information, data collection, and management.

Below I discuss anthropometric measurements collected within the MEC, including the cardiovascular markers of systolic and diastolic blood pressure, and the metabolic markers of body mass index (BMI) and waist circumference. This discussion is followed by a description of the analysis and measurement of biomarkers at offsite laboratories through participant blood samples.

Systolic and diastolic blood pressure recordings were taken from participants age eight years and older, and followed the latest recommendations of the American Heart Association Human Blood Pressure Determination by sphygmomanometers (Centers for Disease Control and

Prevention, 1999-2008b, 1999-2008c). Participants with any of the following on both arms were excluded from blood pressure measurements: rashes, gauze dressings, casts, edema, paralysis, tubes, open sores or wounds, withered arms, a-v shunts, radical mastectomy or if the blood pressure cuff did not fit on the arm. After resting quietly in a sitting position for 5 minutes, three consecutive blood pressure readings were obtained. If a blood pressure measurement was interrupted or incomplete, a fourth attempt was made (Centers for Disease Control and Prevention, 1999-2008c).

All blood pressure measurements were in millimeters of mercury (mmHG). Final blood pressure readings were established according to the following protocol: if only one reading was obtained, that reading was considered the participant's average blood pressure. If there were two or more readings, the average of the readings was calculated. A diastolic pressure of zero was recorded if all diastolic readings were zero. If a reading of zero was obtained but additional reading(s) were not zero, the non-zero reading(s) were used to calculate the average (Centers for Disease Control and Prevention, 1999-2008c).

The metabolic markers of BMI and waist circumference were also measured within the MEC using anthropometric techniques. BMI was measured using weight and height measurements. Weight was taken for participants of all ages using a Toledo digital scale built into the floor of the MEC, measured in pounds and converted to kilograms in the automated ISIS system (Centers for Disease Control and Prevention, 1999-2008c). The maximum weight capacity of the scale was 440 pounds. Portable scales were also available in the event of scale malfunction. Also, if a participant exceeded scale capacity, two portable scales were used, with each participant's foot on a scale to determine weight (Centers for Disease Control and Prevention, 2007). Participants were instructed to wear the proper MEC exam gown while being

weighed. Anyone with a cast or prosthesis, or who wore their street clothes instead of the MEC gown, was coded as such by the examiner. Respondents refusing to remove their shoes prior to stepping on the scale were coded as having an invalid weight.

Standing height was measured from respondents age two years and older who could stand unassisted, using a fixed stadiometer (measured in meters) with a vertical backboard and moveable headboard (Centers for Disease Control and Prevention, 2007). Respondents were instructed to remove any hair ornaments and stand up straight against the backboard. Weight evenly distributed with both feet flat on the floor, heels together and toes apart. Respondents were asked to take a deep breath and hold their position while their height was measured. This procedure helps straighten the spine to yield a more consistent height measurement. The data management system also adjusted height measurements to account for respondents who refused to remove their shoes (Centers for Disease Control and Prevention, 2007). NHANES then calculated BMI as weight in kilograms divided by height in meters squared (kg/m^2).

Waist (abdominal) circumference was measured on participants age two years and older. Waist circumference was recorded in centimeters (cm) and determined using a measuring tape fit snuggly around the trunk in a horizontal plane parallel to the floor (Centers for Disease Control and Prevention, 1999-2008c). Respondents were instructed to pull their gown or shirt above their waist. Standing on the participant's right side, the examiner palpated the hip area to locate the pelvis. Using a cosmetic pencil, the examiner drew a horizontal line just above the pelvis, and proceeded to measure and record the circumference. Measurements were taken to the nearest 0.1 cm.

Blood Samples. In the present study, select biomarkers were measured from participant blood samples (Centers for Disease Control and Prevention, 1999-2008b): including

immune/inflammatory markers such as C-reactive-protein (CRP) and serum albumin, and metabolic markers such as total cholesterol, high-density lipoprotein (HDL), and glycosylated hemoglobin (HbA1c). Participant blood collection procedure consisted of administering a questionnaire to screen for conditions that excluded participants from the blood draw such as: hemophilia, recent chemotherapy, and the presence of numerous skin allergies and rashes. All MEC phlebotomists completed comprehensive training in standardized laboratory procedures to ensure participant safety (Centers for Disease Control and Prevention, 1999-2008b).

Biomarkers measured through participant blood samples were processed and analyzed offsite at federal, private, and university-based contract labs. Contract laboratory analysis methods were based on scientifically reliable and valid procedures commonly used to analyze biological specimens (Centers for Disease Control and Prevention, 1999-2008a, 1999-2008b). Laboratory performance was monitored through repeat testing on two percent of all specimens. Contract laboratories also used progress reports to detail information regarding shipment and receipt of specimens, calibration, and quality control instrument problems (Centers for Disease Control and Prevention, 1999-2008b).

C-Reactive Protein (CRP) was measured from respondents age three years and older, and analyzed at the University of Washington laboratory, Seattle, Washington. CRP analysis was done using the Behring Nephelometer, a latex-enhanced nephelometry (Centers for Disease Control and Prevention, 1999-2008b, 1999-2008c). This technique used a particle-enhanced assay based on the reaction between a soluble analyte and its corresponding antibody. To quantify CRP, the bound antibody was mixed with latex particles. If CRP was present, it formed an antigen-antibody complex with the latex particles. A calibration curve was used to determine

CRP concentrations (Centers for Disease Control and Prevention, 1999-2008b). Values of CRP were measured in milligrams per decileter (mg/dL).

Serum albumin was measured from participants age six years and older, and was analyzed using solid-phase fluorescent immunoassay by multiple laboratories: the Coulston Foundation in Alamagordon, New Mexico (from 1999-2001); the Collaborative Laboratories, LLC in Ottawum, Iowa (from 2002-2004); and the University of Minnesota, Minneapolis, MN (from 2005-2008) (Centers for Disease Control and Prevention, 1999-2008b, 1999-2008c). The fluorescent immunoassay is a non-competitive, double-antibody method for determining albumin in serum blood. Albumin values were recorded in grams per decileter (g/dL). Although the laboratories used different analyzers, NCHS determined that serum albumin values from all labs were not significantly different (Centers for Disease Control and Prevention, 1999-2008b).

Total cholesterol and HDL were measured from participants age six years and older and analyzed by the Johns Hopkins Hospital, Baltimore, MD (Centers for Disease Control and Prevention, 1999-2008b). Depending on the year, cholesterol was measured on a Hitachi Model 407, 717, or 912 analyzer (no adjustment was necessary for the change in instrumentation), which measured the sample enzymatically through a series of coupled reactions that hydrolyzed cholesteryl esters and oxidized the alcohol group of cholesterol (Centers for Disease Control and Prevention, 1999-2008b). A reaction byproduct, hydrogen peroxide, was measured using a peroxidase-catalyzed reaction that produced color emission; the intensity of the emission was proportional to the concentration of cholesterol found in the sample. HDL was measured using the Heparin-Mn Precipitation Method. This method precipitates the removal of lipoproteins with the compounds heparin sulfate and Manganese Dicholoride. HDL was then measured in the

remaining supernatant (Centers for Disease Control and Prevention, 1999-2008b). All cholesterol markers were measured in mg/dL.

Glycosylated hemoglobin (HbA1c) was measured from blood samples of participants aged 12 years and older. Prior to 2005, a Primus instrument was used to test for HbA1c by the University of Missouri-Columbia, Columbia, Missouri. The Primus method used a fully automated glycohemoglobin analyzer (Centers for Disease Control and Prevention, 1999-2008b); which utilized boronate affinity high performance liquid chromatography (HPLC). From 2005-2008, samples were analyzed at the University of Minnesota using to Tosoh method. The Tosoh method separated the blood specimen using ionic interactions, which also utilized HPLC. A crossover study was performed to compare the Primus with the Tosoh method, and little difference was found between the two methods (Centers for Disease Control and Prevention, 1999-2008b). Values of HbA1c were measured as a percent (%).

3.2.3. NHANES Adolescents Screened, Interviewed, and Examined

The eligible sub-sample for this dissertation is composed of adolescents age 12 to 19 from NHANES 1999-2008 who completed both the in-home interview component and the MEC examination (Centers for Disease Control and Prevention, 1999-2008a). A more detailed discussion of the final analytic sample can be found in section 3.2.8. Table 3.1 illustrates selected participants and response rates for adolescents interviewed and examined from each cross-sectional cycle of NHANES.

Data from the 1999-2000 NHANES consisted of 2,732 adolescents age 12 to 19 years screened to participate; 2,415 of them participated in the interview (representing an 88.4% response rate), and 2,314 also agreed to have a physical exam (84.7% response rate) (Centers for

Disease Control and Prevention, 1999-2008). In the 2001-2002 NHANES 2,799 adolescents were screened, 89% were interviewed and 80% completed the MEC exam. The 2003-2004 NHANES screened 2,649 adolescents, interviewed 2,303 respondents (86.9% response rate), and examined 2,248 (85% response rate) (Centers for Disease Control and Prevention, 1999-2008). In 2005-2006 approximately 2,689 adolescents were screened, 85% were interviewed, and 82% completed the MEC exam. Last, in 2007-2008, 1,448 adolescents were screened, 85.5% were interviewed, and approximately 84% were examined. In total, 12,317 adolescents age 12 to 19 were screened from 1999-2008. Of these, 87% were interviewed, and 84% participated in the MEC exam (Centers for Disease Control and Prevention, 1999-2008).

Table 3.1. Selected Participants and Response Rates for Adolescents Age 12-19 Years Interviewed and Examined, NHANES 1999-2008

NHANES Cycle	Screened N	Interviewed N (Response Rate)	Examined N (Response Rate)
1999-2000	2,732	2,415 (88.4)	2,314 (84.7)
2001-2002	2,799	2,487 (88.9)	2,418 (86.4)
2003-2004	2,649	2,303 (86.9)	2,248 (84.9)
2005-2006	2,689	2,288 (85.1)	2,207 (82.1)
2007-2008	1,448	1,238 (85.5)	1,210 (83.6)
Total	12,317	10,731 (87.1)	10,397 (84.4)

3.2.4. NHANES Sample Weights

Because this dissertation uses a survey with a complex, multistage, sampling design, sample weights developed by NHANES are used. NHANES assigned a sample weight to each sample individual, indicative of the measure of the number of people in the population represented by that sample person. The sample weight reflects the unequal probability of selection, nonresponse adjustment, and adjustment to independent population controls (Centers

for Disease Control and Prevention, 1999-2008a).When unequal selection probability is applied, the sample weights are used to create an unbiased estimate of the national population.

As the present research combines five cross-sectional cycles of NHANES, and focuses on participants who completed the MEC component, a five-cycle weight was constructed using NHANES MEC weights. NHANES provided specific two-year sample weights according to the Bureau of the Census, with weights for the 1999-2000 cycle based on the 2000 census (Centers for Disease Control and Prevention, 1999-2008a). However, different population bases were used for subsequent cycles after 2000, making weights between cycles not directly comparable. Therefore, it was necessary to use a NHANES pre-constructed four-year sample weight from 1999-2002 in conjunction with two-year NHANES weights for each subsequent cycle thereafter (Centers for Disease Control and Prevention, 1999-2008a). Thus, over the five cycles, the four-year weight variable from 1999-2002 received two-fifths of the weight, while each following two-year cycle received one-fifth of the weight.

3.2.5. Operationalization of Variables

This section summarizes the variables used in Aim 1 of this study and how they are operationalized as variables. I first discuss the main outcome variable, allostatic load, as a sum of the number of biological markers identified as risky according to empirical quartile cut-points. I then describe the sociodemographic characteristics used as independent variables in my analysis.

3.2.5.1. Allostatic Load

AL was comprised of nine biomarkers. The biomarkers used for this study were selected for their high public health relevance, significant association with biological systemic functioning, and their availability in the data (Centers for Disease Control and Prevention, 1999-2008a). The nine biomarkers used were indicative of regulation among three systems: 1)

cardiovascular markers including diastolic and systolic blood pressure; 2) metabolic functioning markers including body mass index (BMI), waist-circumference, total cholesterol, high-density lipoprotein (HDL), and glycosylated hemoglobin (HbA1c); and 3) inflammatory markers including serum albumin and C-reactive protein (CRP).

The operationalization of AL in this study was a summation score. This AL score uses empirical cut-points based on the sample distribution rather than clinical thresholds, because no clinical cut-points exist for a number of biomarkers, among younger age groups, and because distribution-based cut-points better capture sub-clinical dysregulation. Empirically-based AL scores are often used to capture cumulative physiological dysregulation at more conservative, pre-disease levels and represent overall subclinical health conditions (Crimmins et al., 2003; Crimmins et al., 2007). For each of seven indicators, empirical cut-points were determined by the 75^{th} percentile value, identified as high risk, and for the remaining two biomarkers HDL and albumin, high risk cut-points were defined as below the 25^{th} percentile (Seeman et al., 1997; Seplaki et al., 2005).

The decision to use quartile cut-points was based on previous studies, and is considered the preferred approach (Seeman et al., 2004; Seeman et al., 2010a; Seeman et al., 2001). Adolescents who exhibited high risk levels of biological markers received a score of one for that parameter. Therefore, a composite AL index was created by summing the number of parameters that fell into the high risk quartile. For a composite score with nine biomarkers, the range of AL scores was 0-9, with higher values signifying greater systemic dysregulation.

3.2.5.2. Sociodemographic Characteristics

Independent variables for Aim 1 in this study include age, gender, race/ethnicity, nativity status, family income, and parental education. Age was calculated by NHANES using the date of

the screening interview and the respondent's birth date, measured in single years, and was included in the present study as a continuous variable from 12 to 19. Initially, age was stratified into early (12-15 years) and late (16-19 years) adolescence to allow for a more informative exploration and description of AL. Similar distributions of AL among both age groups warranted a more useful interpretation of AL by combining all ages of adolescence (as can be seen in Chapter 3). By examining age continuously, this study was able to distinguish the change in AL from one year to the next across adolescence, allowing for the identification of demographic age groups that may be at higher risk for cumulative physiological dysregulation.

Gender was coded as female (1) and male (0). Race/ethnicity was coded into four categories, giving priority to Hispanic ethnicity: non-Hispanic White (reference category), non-Hispanic Black, and Hispanic. Hispanic adolescents were comprised of self-identified Mexican American and Other Hispanic adolescents. Approximately 11% of all Hispanics were Hispanics other than Mexican Americans. Adolescents who stated they were more than one race and then identified a main race as non-Hispanic white or non-Hispanic Black were coded as such. An interaction variable for race/ethnicity and mean-centered age was also created to enable the interpretation of AL across adolescence by race/ethnicity. Nativity status was asked of respondents through the question, "In what country were you born?" coded as born in the 50 U.S. states or Washington, D.C.; born in Mexico; or born elsewhere. A dichotomous variable was then created of foreign born (1) and U.S. born (0).

Socioeconomic status (SES) was measured using two of the most commonly used variables, household family income and educational attainment of the household representative (proxy adult) (LaVeist, 2005; Williams & Collins, 1995). Family income was ascertained by asking respondents the total combined income for all family members living in the household in

the past 12 months. Eleven income categories separated by $5,000 increments from $0 to $75,000 and over were provided. For the present study, these groups were collapsed into five main categories: <$20,000, $20,000-$44,999, $45,000-$74,999, and ≥$75,000. The highest income category was selected as the reference category, as levels of AL are anticipated to increase as a result of lower SES.

Educational attainment was based on the response of the household representative, and was categorized for this study as less than high school, high school graduate or GED, and more than high school. Greater than a higher school degree was selected as the reference category as levels of AL are expected to increase with lower parental education status.

3.2.6. Analytic Sample Derivation

As the first aim of the present dissertation focuses on the change in levels of AL by age and race/ethnicity across adolescence from 12 to 19 years, the current analytic sample is comprised of adolescents who completed both the interview and physical examination components, and have complete biomarker measurements. Of 10,731 adolescents age 12 to 19 years who were interviewed, approximately 97% (N=10,397) also completed the examination component.

AL in this study is the summation of 9 biological markers, thus any adolescent who was missing one or more biomarkers used in the summation of AL was excluded. A total of 8.6% of those who were interviewed and examined (N=1,461) were missing one or more biomarkers. Approximately 876 adolescents were excluded from the venipuncture portion of the physical examination due to exclusion criteria mentioned earlier. An additional 283 adolescents were

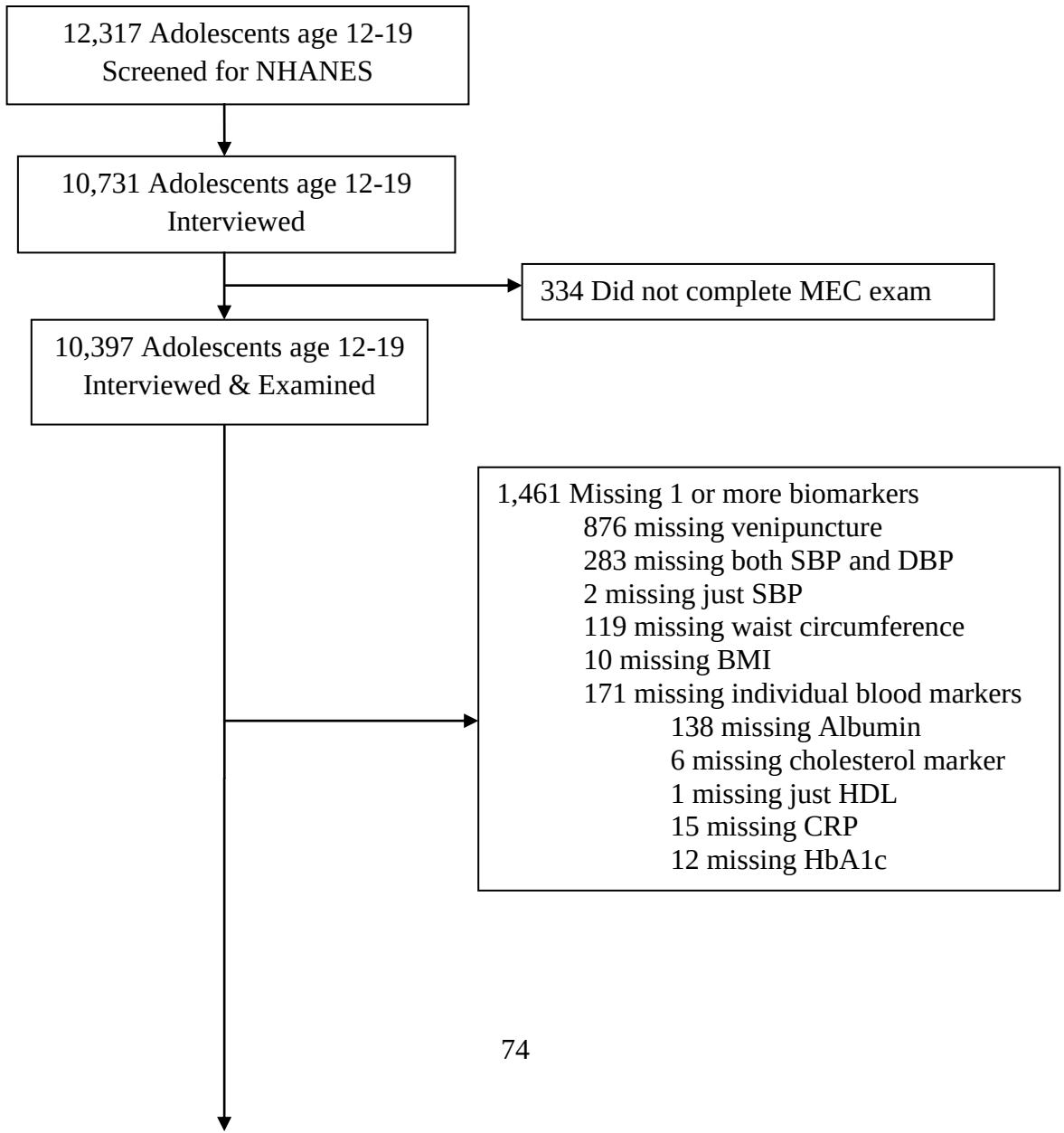
missing both systolic and diastolic blood pressure measurements[1]. Two adolescents also had a systolic blood pressure reading of zero, which NHANES identified as an invalid measure. A number of adolescents were also missing anthropometric measurements: 119 had invalid waist circumference measurements[2], and 10 were missing BMI measurements[3]. An additional 171 adolescents were missing individual biomarkers obtained from blood samples, including 138 of albumin, 6 cholesterol-related markers, 15 of C-reactive protein, and 12 of glycosylated hemoglobin. NHANES did not indicate reasons for such missing individual blood markers.

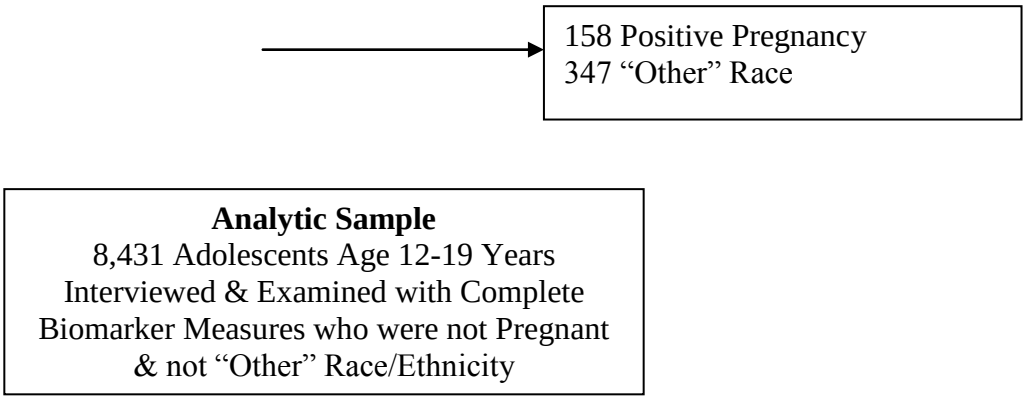
Pregnant women (N=158) were also excluded from the study, as fluctuating and/or elevated biomarker levels are considered normal during pregnancy and not indicative of physiological dysregulation (Petraglia et al., 1996). Last, NHANES identified "Other" race (N=347), including multiracial, as a racial category, yet did not further specify racial/ethnic grouping. Due to the difficulty in interpreting results of AL among a non-specific group of "Other" race/ethnicity, these adolescents were excluded from the sample.

In sum, the analytic sample used for Aim 1 included adolescents age 12 to 19 who completed the interview and examination elements, had valid data on all biomarkers used to create the allostatic load score, were not pregnant, and did not identify as "Other" race/ethnicity. The final sample included 8,431 adolescents. A description of the exclusions required to arrive at this sample is presented in Figure 3.1.

[1] Of the 283 missing: 24 refused, 18 reported there was no time, 17 had a physical condition, 13 reported to be ill or had an emergency, 1 came late, 77 reported some other reason, 2 had safety exclusions, and 9 were missing for no known reason.
[2] Of the 119 missing: 3 could not be obtained, 28 had clothing obstruction, and 88 were missing for no known reason.
[3] Of the 10 missing: 1 could not be obtained, 6 had clothing obstruction, 2 reported a medical appliance, and 1 was missing for no known reason.

Figure 3.1. Criteria for Selection of Analytic Sample, Adolescents 12 to 19 Years, NHANES 1999-2008 (N=8,431)





3.2.7. Missing Data

Of the 8,341 adolescents included in the sample, there were no missing cases of gender or race/ethnicity, but there existed limited missing data for education, nativity status, and annual family income. Missing values (N=17) for nativity status numbered less than one percent, and were thus coded into the modal category of U.S. born. Results did not differ whether missing cases were coded to the modal category or dropped. Approximately 164 household proxy respondents (1.9% of the sample) reported a family income of >$20,000, but no further information was provided to determine which income category they identified with. An additional 215 proxy respondents (2.6%) were missing family income data, along with 271 (3.2%) missing household education information. Eighty (0.9%) were missing both education and income information.

To retain these missing cases of family income and representative education level in the final sample (N=730, 8.7%), multiple imputation using chained equations (ICE) was employed. This method assumes data are missing at random, and utilizes an iterative Monte Carlo Markov Chain (MCMC) algorithm to approximate imputation of missing values (Heeringa, West, & Berglund, 2010). Each iteration of the algorithm moves one by one through the series of

variables in the imputation model. Imputed values are generated from a series of univariate models, whereby a single variable is imputed based on a group of variables. The present study's imputation model included information of the household proxy representative: gender, age, education level, and marital status, along with household size, family income, and poverty income ratio. Imputations were weighted to account for sample design.

A total of five datasets were imputed to estimate accurate standard errors. Comparisons found slightly smaller standard errors and unchanged point estimates of imputed representative education and family income than those from the sample if the cases had been excluded using list-wise deletion. By using the imputed values of representative education and family income, I increased the size of the sample and reduced selection bias. Also, smaller standard errors from ICE reflect that the imputation recovered some additional statistical information from the incomplete cases (though be it very small).

3.2.8. Selection Analysis

As depicted in Figure 3.1, 2,300 adolescents age 12 to 19 were excluded from the analytic sample. The majority of these respondents were excluded because they did not have complete AL biomarker measurements (N=1,461); the remaining respondents were excluded because they did not complete the physical examination (N= 334), were pregnant (N=158), or identified as "other" race (N=347). In order to better understand the extent to which these respondents differ from those in the analytic sample, bivariate analyses with a design-based Wald test were first employed, followed by a logistic regression model predicting being excluded from the analytic sample as a function of sociodemographic characteristics. This analysis was weighted using constructed NHANES weights from 1999-2008 and accounted for the complex study design using the survey estimation commands available in Stata 12.0 (StataCorp, 2011).

Table 3.2 presents bivariate results of respondents included and excluded from the analytic sample. By design, the excluded group contained a significantly larger proportion of females to males due to the exclusion of pregnant females, as well as fewer Whites and a greater number of "Other" race individuals. The proportion of those born outside of the U.S. was greater among the excluded sample, likely a result of excluding "Other" race individuals. Indicators of socioeconomic status also differed for those who were excluded from the sample. Specifically, in the excluded sample, there existed a slightly lower proportion of those reporting higher family incomes.

Table 3.2. Weighted Sample Characteristics of those Included and Excluded from the Analytic Sample, NHANES 1999-2008

Sociodemographics	Study Sample (N=8,431)	Excluded Sample (N=2,300)
	% or Mean (SE)	
Individual Characteristics		
Age	15.4 (.04)	15.6 (.08)
Gender**		
Male	52.3	46.0
Female	47.7	54.0
Race/ethnicity***		
NH White	65.8	46.3
NH Black	15.3	13.8
Hispanic	18.9	13.5
Other	0.0	26.4
Nativity status***		
U.S.-born	92.0	86.2
Foreign born	8.0	13.8
Family Background		
Household Representative Education*		
Less than high school	21.0	24.2
High school/GED	26.3	28.3
More than high school	52.7	47.5
Family income**		
<$20,000	23.9	29.2
$20,000-44,999	25.2	26.1
$45,000-74,999	21.1	20.8

≥$75,000	29.9	24.0

Note: Bivariate analyses with a design-based Wald test; NH= non-Hispanic.
*p≤.05; **p≤.01; ***p≤.001

Table 3.3 presents results of the selection analysis using logistic regression. By design, respondents who were female were significantly more likely to be excluded from the analytic sample. Additionally, non-Hispanic Black females were also more likely to be excluded. These logistic regression results suggest that there is modest differential selection, where Black females appear to be under-represented in the analytic sample. This under-representation implies that race effects on allostatic load found in the present study may not be wholly representative.

Table 3.3. Weighted Logistic Regression Results of Being Excluded from the Analytic Sample as a Function of Sociodemographic Characteristics, NHANES 1999-2008 (N=10,731)

Sociodemographics (reference)	b	95% CI
Individual Characteristics		
Age	-0.02	-0.18, 0.15
Gender (Male)		
Female	0.29**	0.10, 0.48
Race/Ethnicity (NH White)		
NH Black	0.25*	0.05, 0.45
Hispanic	-0.10	-0.39, 0.20
Nativity status (U.S.-born)		
Foreign-born	0.27	-0.09, 0.63
Family Background		
Household Representative Education (Less than high school)		
High school/GED	0.12	-0.15, 0.38
More than high school	-0.04	-0.31, 0.23
Family income (<$20,000)		
$20,000-44,999	-0.04	-0.34, 0.26
$45,000-74,999	0.04	-0.28, 0.36
≥$75,000	-0.12	-0.43, 0.19

Note: NH = non-Hispanic; CI = Confidence intervals.
*p≤.05; **p≤.01; ***p≤.001

3.2.9. Descriptive Statistics of Sociodemographic Characteristics

Table 3.4 presents weighted descriptive statistics and unweighted Ns of adolescent sociodemographic characteristics in the analytic sample. The first column shows weighted univariate results; the second column presents the matching unweighted sample size. The mean age of the analytic sample was 15.4 years old. The majority of adolescents were White (66%), with 15% of the sample Black, and almost 19% Hispanic. Only 8% of adolescents were foreign-born. Over 50% of adolescents had a household representative with more than a higher school education, and roughly a quarter of adolescents lived in a home with a family income below $20,000 or between $20,000 and $45,000.

Table 3.4. Weighted Descriptive Statistics of Independent Variables, NHANES 1999-2008 (N=8,431)

Sociodemographics	% or Mean Weighted	N Unweighted
Individual Characteristics		
Age	15.4	--
Gender		
Male	52.3	4,409
Female	47.7	4,202
Race/ethnicity		
NH White	65.8	5,548
NH Black	15.3	1,290
Hispanic	18.9	1,593
Nativity status		
U.S.-born	92.0	7,756
Foreign born	8.0	675
Family Background		
Household Representative Education		
Less than high school	21.0	1,771
High school/GED	26.3	2,217
More than high school	52.7	4,443
Family income		
<$20,000	23.9	2,015
$20,000-44,999	25.2	2,125
$45,000-74,999	21.1	1,779
≥$75,000	29.9	2,520

Note: NH= non-Hispanic.

3.3 National Longitudinal Study of Adolescent Health (Add Health)

The second and third aims of this dissertation focus on the longitudinal patterns of obesity from adolescence to young adulthood to investigate the possible mediating mechanism of adolescent stressful life events. The data for these aims come from Add Health. Add Health is a prospective observational study that uses in-school and in-home surveys to obtain data from a nationally representative sample of U.S. adolescents enrolled in grades 7 to 12 in 1994-1995 as they transition to young adulthood. The Add Health survey was funded by the National Institute of Child Health and Development (NICHD) to the University of North Carolina at Chapel Hill (UNC) Carolina Population Center (Harris, 2008). The longitudinal study examined the social factors that influence adolescent and young adult relationships, behavior, and social interactions (Harris et al., 2008). Special attention was given to personal characteristics, family structure, peer networks, neighborhood structure, and academic achievement. This dissertation uses data from three of four waves of Add Health. Wave I, which is the source of data collected from adolescents during 1994-1995; Wave II, which collected data one year later in 1996; and Wave III, which collected data from Wave I participants during 2001-2002 when they were young adults. All Add Health protocols received approval from the institutional review board. This dissertation research has been approved by the University of California Office for the Protection of Human Subjects.

3.3.1. Study Design and Sampling Procedures

The complex study design and sampling procedure of Add Health is shown in Figure 3.2. The initial wave of Add Health used a sampling frame of 26,666 U.S. high schools (Harris et al.,

2008). From the sampling frame, 80 high schools and 52 feeder schools were selected by stratified random sampling to ensure the sample was representative of U.S. schools with respect to region of country, urbanicity, school size, school type, and ethnicity (Udry, 2001). The final sample included 132 schools, with school sizes varying from fewer than 100 to over 3,000 students.

Approximately 90,000 eligible students in grades 7-12 from the selected schools completed an in-school questionnaire. Of those who completed the in-school questionnaire, a random sample of 15,243 students, stratified by gender and grade, were chosen to participate in an in-home Add Health interview (Harris et al., 2008). Those who completed the in-home interview (N=12,105) represent a core sample of students in grades 7-12 in the United States from 1994-1995.

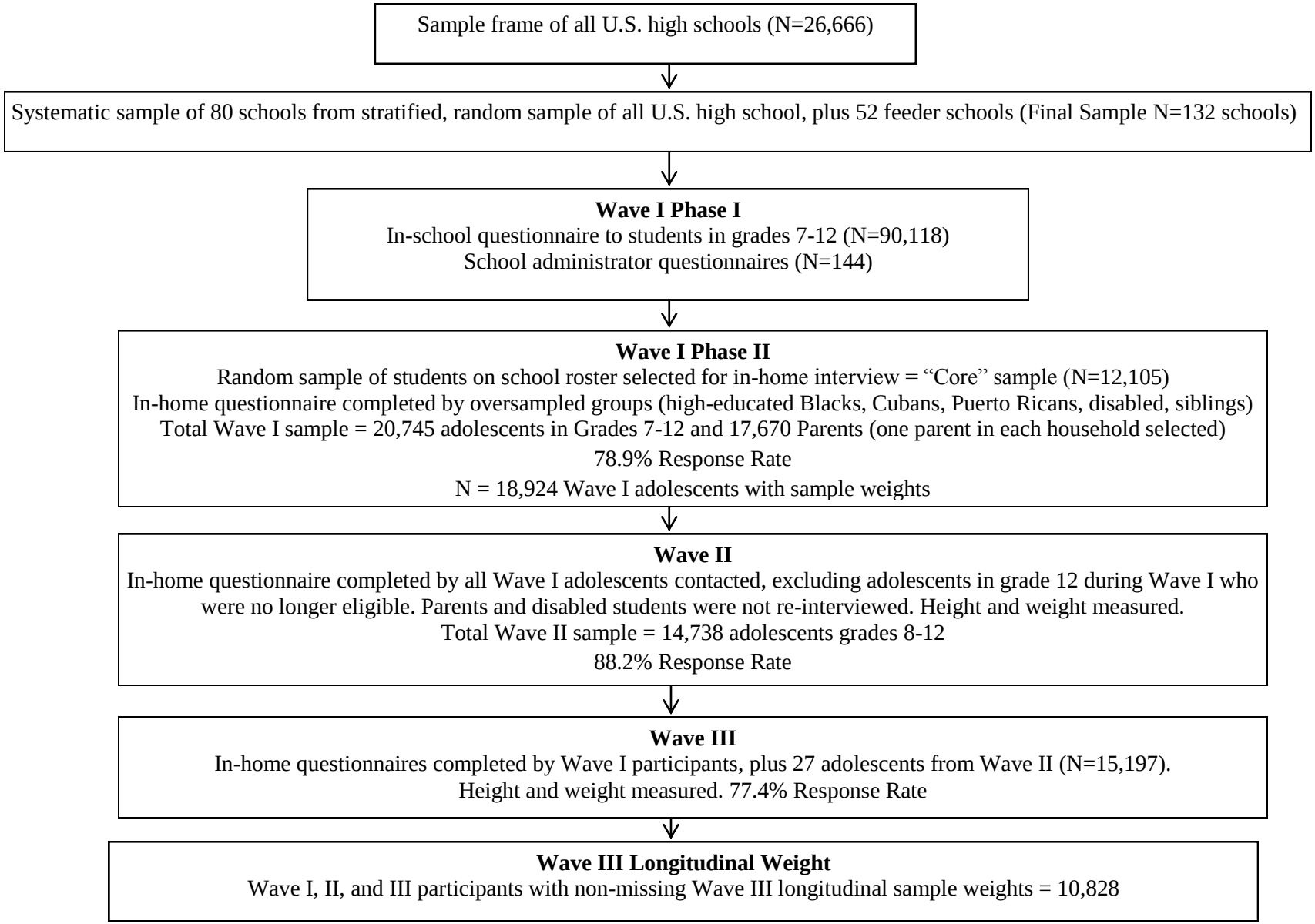
In addition to this core sample, Add Health oversampled certain adolescent populations, including Black adolescents with college-educated parents, and specific Hispanic subgroups including Cuban and Puerto Rican adolescents. A sample of siblings, including twins, full-, half-, and non-related adolescent siblings were also included. In sum, the total Wave I sample included 20,745 adolescents in grades 7-12. Wave I respondents were 11 to 21 years old (Harris et al., 2008).

Wave II was collected one year later from 1995-1996, and included Wave I respondents still enrolled in high school, including dropouts (Harris et al., 2008). Students who exceeded the grade eligibility requirement were excluded. Approximately 14,738 adolescents completed the survey (88.2% response rate); adolescents were now 12-22 years old. In Wave III, all Wave I respondents were followed regardless of Wave II participation. Of the 20,745 Wave I

respondents, 5,575 were not re-interviewed or were excluded from the Wave III interview[4]. An additional 27 respondents who were "genetic samples" added in Wave II were also re-interviewed at Wave III. Wave III was conducted from 2001-2002, and included 15,197 young adults (77.4% response rate) age18-27 years old (Harris et al., 2008).

[4] Of the 5,575: 687 were not part of the probability sample or the sibling sample at Wave I, and were thus considered ineligible for Wave III (Chantala, Kalsbeek, & Andraca, 2004) An additional 96 respondents from Wave I were reported as deceased at the time of the Wave III interview. The Add Health team was unable to make contact with 2,330 respondents from Wave I. Of those contacted, 1,293 were willing to participate but unable (i.e., language barrier, active military duty, institutionalized, etc.), and 1,160 refused to participate during Wave III. Last, 9 cases were excluded from the Wave III interview due to lost or unusable data, or the respondent was younger than 18 years of age (Chantala et al., 2004)

Figure 3.2. Add Health Study Design, Waves I – III (1994-2002)



3.3.2. Data Collection: In-Home Component

Add Health administered in-home questionnaires during all three Waves. The in-home survey during Wave I was completed by core adolescents and those oversampled, as well as one parent or guardian living in the home (N=17,760). The parent survey included questions about family socioeconomic status, household composition, and information pertaining to the adolescent (Harris et al., 2008). Wave II of Add Health administered in-home surveys to Wave I adolescents. At Wave III, in-home surveys were conducted to Wave I adolescents (Harris et al., 2008). Pertinent to the current study, height and weight measurements were also taken during the in-home interview of Waves II and III (Harris et al., 2008).

Add Health in-home interviews were conducted using CAPI. Trained interviewers would ask respondents questions, and respondents would use the CAPI to electronically report their answers. Sensitive information, such as sexual and romantic relationships and pregnancy information, was administered using audio computer-assisted self-interviewing (ACASI) devices, whereby respondents confidentially reported electronic answers based on headphone administered audio questions (Harris et al., 2008).

Height and weight were measured in Waves II and III during in-home surveys according to standardized procedures. All participants removed their shoes and stood up against a wall (mainly on a non-carpeted surface). A level and tape measure supplied by the interviewer was used to accurately measure height. In Wave II, height was measured in feet and inches. In Wave III, height was measured to the nearest 1/8 inch (Entzel et al., 2008).

Weight measurements were taken using a digital bathroom scale calibrated and supplied by the interviewer, measuring to the nearest 1/2 pound. The scale was placed on a flat, even surface and the participant removed his or her shoes or extra layers of clothing. In Wave III, if a

respondent weighed more than the scale maximum, or 330 pounds, a separate code was used to indicate this event (Harris et al., 2008).

3.3.3. Add Health Sample Weights

This dissertation uses weights developed by Add Health to account for the survey's complex sampling design and attrition across waves. It is important to account for the sampling design to obtain unbiased estimates of model parameters and standard errors. Technical advisors of the Add Health recommend longitudinal research from Wave I to Wave III be done using Wave III longitudinal grand sample weights (Chantala & Tabor, 1999). Therefore, to apply grand sample weights at Wave III to adjust for longitudinal analysis, respondents without a Wave III grand sample weight must be excluded (Chantala & Tabor, 1999). While 15,197 Wave I respondents completed the interview at Wave III, only 10,828 cases were also respondents in Wave II and had non-missing weights at Wave I.

3.3.4. Operationalization of Variables

The operationalization of variables used in Aims 2 and 3 of the current study are discussed below. I first discuss the main outcome variable of longitudinal obesity patterns from adolescence to young adulthood, focusing on the differences in adolescent and adult obesity classification. I then describe the mediating variable of adolescent stressful life events (SLE) as the sum of the number of events within one year of the Wave I interview done *to* the adolescent separately from those performed *by* the adolescent. Last, I describe the independent variables of Wave I sociodemographic characteristics.

3.3.4.1. Longitudinal Patterns of Obesity from Wave II to Wave III

I use standard conventions for comparing Body Mass Index (BMI) across adolescents (who are still developmentally growing) and young adults (who have reached their maximum

adult height) (Centers for Disease Control and Prevention, 2000, 2011c; Gordon-Larsen et al., 2010; Ogden et al., 2012; Ogden et al., 2002). Thus, different assessments of obesity for adolescents and young adults were used. BMI is calculated from an individual's weight and height (kg/m^2) as an indicator of obesity. The CDC recommends using gender- and age-specific BMI percentiles (Centers for Disease Control and Prevention, 2000) as a growth reference during adolescence to determine obesity. As such, an adolescent who is above the 95^{th} BMI percentile curve for age and gender is classified as obese. This is because BMI changes at different rates by age and gender during normal adolescent development. However, among adults (older than 19 years), a BMI of 30 or more is classified as obese regardless of age and gender.

To appropriately use the gender- and age-specific growth curves, CDC suggests transforming adolescent BMI into BMI z-scores. BMI z-scores, also known as BMI standard deviation scores, are measures of relative weight adjusted for child and adolescent age and gender (Must & Anderson, 2006), indicating how many units (of the standard deviation) an adolescent's BMI is above or below the average BMI value for their age group and gender (according to the entire population).

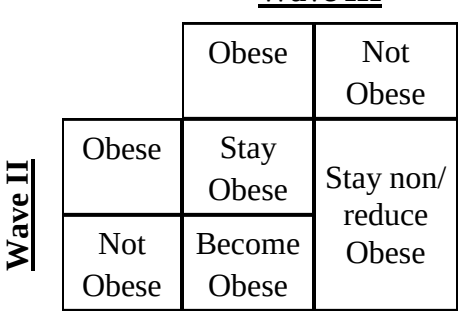
In the present study, I use the Stata program *zanthro* (Vidmar et al., 2004), to automatically calculate BMI z-scores from height and weight measured at Wave II, variable by age and gender, with an external reference of the CDC growth curves. This procedure follows that 5% of the child and adolescent population is above the 95^{th} percentile of BMI for their age. Prior work on adolescent obesity using Stata and the CDC growth curves have also utilized zanthro (Knutson & Lauderdale, 2007; Sanigorski et al., 2007; Snell, Adam, & Duncan, 2007).

Using BMI z-scores and a z-table of the normal distribution, adolescents falling at the 95^{th} percentile cutoff or above were identified as obese. For young adults at Wave III, obesity

was classified as a BMI of 30kg/m^2 or higher, regardless of age and gender. A select number of cases (<1%) at Wave III had a weight that exceeded scale capacity. These individuals were coded as obese.

Similar to previous Add Health literature on longitudinal obesity (Gordon-Larsen et al., 2004; Gordon-Larsen et al., 2010; Lee et al., 2009; Scharoun-Lee et al., 2009), a two-by-two table of obesity status at Wave II and Wave III was used to create a categorical variable of longitudinal obesity patterns. The illustration below demonstrates the longitudinal patterns created: *Becoming obese* (not obese at Wave II but obese at Wave III); *Staying Obese* (obese at both Waves II and III); *Reduce Obesity* (obese at Wave II but not obese at Wave III); and *Staying Non-Obese* (not obese at both Waves II and III). Following convention, a small proportion of respondents identified with the *reduce obesity* category, and were thus combined with the *stay non-obese* group.

Figure 3.3. Categorization of Longitudinal Obesity Patterns from Wave II to Wave III, Add Health



3.3.4.2. Wave I Mediating Variable- Adolescent Stressful Life Events

Consistent with prior research (Adkins, 2009; Boardman & Alexander, 2011; Ge et al., 1994; Ge et al., 2006; Ge et al., 2009), the present dissertation operationalizes SLE as the cumulative number of events occurring during adolescence at Wave I. The measure of SLE was

based on those life events experienced prior and reported at Wave I of Add Health by Adkins and colleagues (Adkins, 2009), originally derived from an SLE index developed by Ge et al (Ge, 1994). Adkins and colleagues expanded and modified the original measure for Add Health, examining only acute events that occurred within the past 12 months of the interview. They included nearly 50 items from numerous life domains, such as family, romantic conflicts, academic problems, violence exposure, and death of family members or friends.

As the measure of SLE provides a relatively comprehensive grouping of major life events, a weight of one was given if an event occurred, and zero if not. Additionally, the current study takes into account individual control over whether an event occurred. Thus, distinct SLE indices were created according to events that occurred *to* or were performed *by* the adolescent (Table 3.5). A detailed descriptive table of SLE can be found in section 3.3.8.

The empirical distribution of SLE done *to* and performed *by* adolescents showed a large proportion of adolescents experiencing one or fewer events, suggesting a categorical variable of SLE would best represent the true nature of stress exposure than a continuous index (Cohen et al., 1995). Due to the number of events (range from 0-16), and a wider distribution of SLE done *to* adolescents, a four category variable was created: 0 SLE, 1 SLE, 2 SLE, and 3 or more SLE. For the index of SLE performed *by* the adolescent (range from 0-7), a three category variable was created: 0 SLE, 1 SLE, and 2 or more SLE. Correlation between these two SLE categorical variables was 0.35.

The higher the number of SLE experienced, the greater the stress exposure. Thus, adolescents falling into the 3 or more category for SLE done *to* the adolescent (or 2 or more category among SLE performed *by* the adolescent) were identified as having "high stress" relative to an adolescent who reported experiencing zero SLE, or who had "no stress". No stress

was set as the reference category, corresponding with no stress exposure and thus better health, to enable the comparison of higher stress categories when examining obesity progression or continuity from adolescence to adulthood.

Table 3.5. List of Stressful Life Events According to Event Control, Add Health Wave I (Adkins, 2009; Boardman & Alexander, 2011)

SLE Done *to* the Adolescent	SLE Performed *by* the Adolescent
Death of mother	Suicide attempt resulting in injury
Death of father	Threatened someone with knife/gun
Friend committed suicide	Shot/stabbed someone
Relative committed suicide	Hurt someone in a physical fight
Saw violence	Had sex for money
Threatened by a knife or gun	Ran away from home
Was shot	Raped Someone
Was stabbed	
Was jumped	
Was injured in a physical fight	
Suffered a serious injury	
Romantic Relationship Ended	
Non-romantic Sexual Relationship Ended	
Was raped	
Contracted an STD	
Was expelled from school	

3.3.4.3. Wave I Independent Variables- Sociodemographic Characteristics

Adolescent Individual characteristics include age, gender, race/ethnicity, nativity status, and age. The present study measured age as a continuous variable, calculated from the adolescent's date of birth and the date of the Wave I interview. The purpose for including age at Wave I was two-fold. First, age at which the stressful life event occurred corresponds with an event during the transitional period of adolescence. Second, age at Wave I is one year prior to

obesity status identified during Wave II, and may act as a predictor of change in obesity status over time.

Gender was coded as female (1) and male (0). Race/ethnicity was coded into four categories, giving priority to Hispanic ethnicity: non-Hispanic White (reference category), non-Hispanic Black, Hispanic, and non-Hispanic Asian. A small percent (4%) of adolescents reported a multiracial/ethnic identity. Add Health interviewers assigned one racial identity for adolescents who reported multiple backgrounds (Harris et al., 2008). Adolescents identified as Native American or Other race were excluded from the analysis due to limited cases. A dummy variable for nativity status (1 = foreign born, 0 =U.S.-born) was created using a single item, "Were you born in the U.S.?"

Adolescent family background characteristics include family structure, parental education level, and family income, and are used to assess conditions during adolescence. Family structure was created according to previous studies (Lee et al., 2009; Upchurch et al., 2002), and was constructed using a household roster in the Wave I in-home interview. Each adolescent was asked about his or her relationship to each household member. Respondents were asked, "Please tell me the first names of all the people, other than yourself, who live in your household. If someone usually lives with you but is away for a short time, include him or her." Adolescents were then asked to further identify the gender and relationship of each member.

From these questions, a detailed four-category family structure variable was created: 1) two biological parents (reference category), 2) step family, including biological mother and stepfather, and biological father and stepmother, 3) single biological parent household including single biological mother or single biological father, and 4) all other situations (no biological parent in the household) including foster parents, step parents, grandparents, aunts, uncles,

siblings, or other parental figures (Lee et al., 2009). Two-biological parent families were identified as the reference category to indicate higher SES.

Parental education was determined according to parental reports from the Wave I parent interview (N=17,670) of the highest level of education completed (in years) dependent on family structure. The biological mother's report of education was used for adolescents living in a household with a biological mother. The biological father's report was used for those living with a biological father. For adolescents living with two biological parents, the parent with the highest education level was used. Consistent with prior research (Upchurch et al., 2002), missing values were imputed according to the adolescent's report of parental education. In the rare instances where neither the parent nor the adolescent reported parental education, weighted means were used according to racial categories to impute these values. Parental education was then categorized into three groups: less than high school, high school diploma or GED earned, and more than high school. Greater than a higher school degree was set as the reference category, as lower SES is expected to significantly predict progression of obesity over time.

Family income was only available from information obtained from the Wave I parent questionnaire. Consistent with prior research (Upchurch et al., 2002), family income in the present study was determined by the parent's total family income according to the family composition, with missing values imputed from a number of family background characteristics as reported by the adolescent using OLS regression[5]. Family income in the present study was further coded into a five category variable: <$20,000, $20,000-$44,999, $45,000-$74,999, and ≥$75,000. The highest income category was set as the reference category as progression of obesity is anticipated to be more greatly associated with lower SES.

[5] The family background characteristics used in the imputation model included: family structure, mother's and father's working hours per week, parental public assistance, parental presence in the household, and the education level of the mother and father.

3.3.5. Attrition at Wave III

Prior work by technical staff at the University of North Carolina, Chapel Hill (UNC) constructed weights to adjust for attrition bias. Non-response at Wave III analysis performed by UNC technical staff found that interviewed Wave III respondents were more likely to be female, non-black, and enrolled in earlier grades when at Wave I than non-respondents (Chantala et al., 2004). Although sampling weights have been constructed to adjust for attrition (Chantala & Tabor, 1999), I also performed an analysis to examine the extent to which there were significant differentials based on the above characteristics.

To examine differences between respondents that were followed up at Wave III, and those who were not re-interviewed, I use Wave III longitudinal sample weights. Therefore, respondents who were missing a Wave I sample weight (N=1,821), were excluded per Add Health's recommendations for data analysis (see Figure 3.3 for more details). Additionally, as the present study examines adolescents age 12 to 19 years, those who were outside this age range at the time of Wave I were also excluded (N=100)[6]. Thus, the attrition analysis compares selected data on 18,824 Wave I adolescents age 12 to 19 years.

Of these, approximately 10,801 Wave I adolescents age 12 to 19 years were interviewed at Waves II and III (had valid Wave III longitudinal sample weights). A logistic regression model predicting attrition at Wave III as a function of Wave I characteristics was conducted in order to examine the degree to which adolescents age 12 to 19 years interviewed during Waves I, II, and III (N=10,801) differ from those not re-interviewed at Wave III (N=8,023). Table 3.6 shows attrition analysis results.

[6] Of 100 outside the age range: 13 respondents were 11 years old, 80 were older than age 19, and 7 refused to disclose their age during the interview.

Age was significantly and positively associated with attrition; older respondents were more likely to have been lost to follow-up. Females had significantly lower odds of being lost to follow-up compared to males. There was no significant association between race/ethnicity and loss to follow-up. Nativity status, parental education level, and family income were not significantly associated with attrition. Respondents living in non-two-biological parent households had significantly higher odds of attrition at Wave III relative to those living in two-biological parent homes.

In sum, men, older teens, and those living in non-intact family environments were more likely to have attrited by Wave III. However, the longitudinally constructed sampling weights allow for some adjustment of this differentiated attrition, thus biases are probably minimal.

Table 3.6. Weighted Logistic Regression of Attrition of Adolescents Age 12-19 Years at Wave III, Add Health, Waves I-III (N=18,824)

Wave I Sociodemographics (reference)	b	95% CI
Individual Characteristics		
Age	0.36***	0.31, 0.42
Gender (Male)		
Female	-0.25***	-0.35, -0.16
Race/Ethnicity (NH White)		
NH Black	0.13	-0.05, 0.31
Hispanic	0.10	-0.14, 0.33
NH Asian	-0.22	-0.56, 0.11
Nativity status (U.S.-born)		
Foreign-born	0.07	-0.11, 0.25
Family Background		
Parental Education Level (Less than high school)		
High school/GED	-0.06	-0.24, 0.11
More than high school	-0.15	-0.32, 0.02
Family income (<$20,000)		
$20,000-44,999	0.04	-0.11, 0.19
$45,000-74,999	0.01	-0.16, 0.17
≥$75,000	-0.07	-0.31, 0.17
Family Structure (2 biological parents)		
Step-Family	0.23***	0.08, 0.38
1 Biological Parent	0.28***	0.16, 0.41
Other Situations	0.51***	0.32, 0.70
Constant	-6.16***	-7.05, -5.28

Note: NH = non-Hispanic; CI = Confidence intervals.
*p≤.05; **p≤.01; ***p≤.001

3.3.6. Analytic Sample Derivation

The current analytic sample is comprised of Wave I adolescents age 12 to 19 years who were also interviewed at Waves II and III, who have non-missing data for BMI at Waves II and III, and who had valid longitudinal sample weights. Obesity status over time is the main outcome for Aims 2 and 3, thus any respondent without a valid BMI measurement during the transition to adulthood was excluded. During Wave II, approximately 262 adolescents had invalid height or

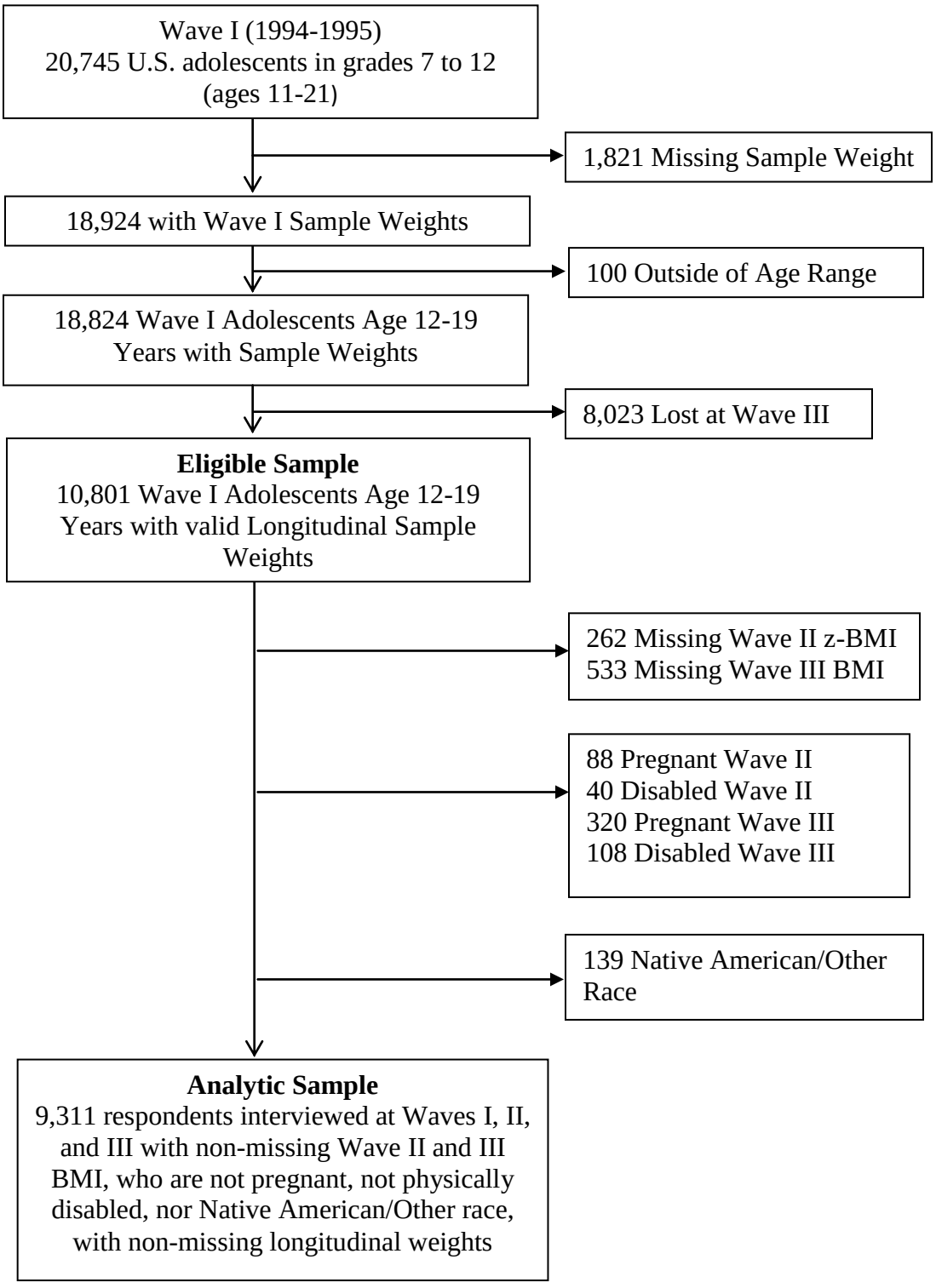
weight measurements used to determine a BMI z-score[7]. Additionally, in Wave III, 533 young adults were missing height and weight measurements to determine a valid BMI score[8]. Add Health did not indicate reasons for missing height and weight measurements.

Additionally, pregnant or seriously disabled respondents in Waves II and III were also excluded, due to the impact of these conditions on height and weight, and subsequent obesity status. In Wave II, 88 females identified as pregnant, and another 40 were classified as physically disabled. By Wave III in young adulthood, 320 females were pregnant at the time of the interview, and 108 were identified as physically disabled. Last, due to the small sample size of those identified as Native American or "Other" race/ethnicity (N=139), these adolescents were also excluded from the sample.

In sum, the analytic sample used for Aims 2 and 3 included adolescents age 12 to 19 interviewed at Waves I, II, and III, with valid adolescent and young adult BMI scores, who were not pregnant or physically disabled, were not Native American or Other race/ethnicity, and who had valid Wave III longitudinal weights. The final sample included 9,311 respondents. A description of the exclusions required to arrive at this sample is presented in Figure 3.3.

[7] Of the 262, 116 were missing only weight measurements, only 2 missing height, and 43 were missing both weight and height. The remaining 101 missing z-BMI scores were as a result of extended age-range conflicting with CDC 2000 growth curve age-specific charts.
[8] Of the 533, 183 were missing only weight, 124 missing only height, and 226 were missing both weight and height.

Figure 3.4. Criteria for Selection of Analytic Sample, Add Health (N=9,311)



3.3.7. Selection Analysis

As depicted in Figure 3.3, 1,490 of the 10,801 adolescents age 12 to 19 with a sample weight at Wave I who were not lost to follow-up were excluded from the analytic sample. In order to better understand the extent to which these respondents differ from those in the analytic sample, a logistic regression model predicting being excluded from the analytic sample as a function of Wave I sociodemographic characteristics was done. This analysis was weighted using Add Health Wave III longitudinal weights and accounted for the complex study design using the survey estimation commands available in Stata 12.0 (StataCorp, 2011).

Table 3.7 presents results of this selection analysis. Older teens and females were significantly more likely to be excluded from the analytic sample. Those who were foreign born and adolescents who lived in a household with a higher family income compared to those with a family income of less than $20,000 were significantly less likely to be excluded from the analytic sample. These logistic regression results suggest that there is differential selection, and that higher risk (female, older) adolescents may have been excluded from the analytic sample.

Table 3.7. Logistic Regression Results of Being Excluded from the Analytic Sample as a Function of Wave I Sociodemographic Characteristics, Add Health Waves I-III (N=10,801)

Wave I Sociodemographics (reference)	b	95% CI
Individual Characteristics		
Age	0.17***	0.11, 0.24
Gender (Male)		
Female	0.49***	0.34, 0.64
Race/Ethnicity (NH White)		
NH Black	-0.01	-0.29, 0.27
Hispanic	-0.05	-0.37, 0.27
NH Asian	-0.36	-0.96, 0.24
Nativity status (U.S.-born)		
Foreign born	-0.47*	-0.88, -0.06
Family Background		
Parental Education Level (Less than high school)		
High school/GED	-0.18	-0.48, 0.13
More than high school	-0.23	-0.57, 0.11
Family income (<$20,000)		
$20,000-44,999	-0.29**	-0.50, -0.07
$45,000-74,999	-0.47***	-0.73, -0.21
≥$75,000	-0.69***	-1.06, -0.32
Family Structure (2 Biological Parents)		
Step-Family	0.08	-0.17, 0.33
1 Biological Parent	-0.001	-0.18, 0.18
Other Situations	0.30	-0.03, 0.62

Note: Longitudinal weight applied; NH = non-Hispanic; CI = Confidence intervals.
*p≤.05, **p≤.01, ***p≤.001

3.3.8. Descriptive Statistics of Add Health Wave I Sociodemographic Characteristics and Stressful Life Events

Table 3.8 presents weighted descriptive statistics and unweighted Ns of Add Health sociodemographic characteristics at Wave I for the analytic sample. As Aims 2 and 3 of the present dissertation examine longitudinal patterns of obesity from adolescence to young adulthood for females and males separately, Table 3.8 is stratified by gender.

Females and males demonstrated virtually identical distributions. Mean age at Wave I was 15.5 years. Approximately 69% of adolescents were White, 15% Black, 12% Hispanic, and close to 4% Asian. The majority of teens were U.S.-born (95%), with only 5% of adolescents born outside of the U.S. Approximately 12% of adolescents had a parent with an education level less than a high school degree, and nearly three-quarters of parents reported more than a high school degree. Nineteen percent of teens had a family income of less than $20,000, 40% with an income between $20,000 and $45,000, and close to 13% had a family income greater than $75,000. Nearly 60% of adolescents reported living in a two-biological parent household, 10% in a step-family, 27% with one biological parent, and 4% in some other family situation.

Table 3.8. Analytic Sample: Weighted Descriptive Statistics and Unweighted Ns of Wave I Sociodemographic Characteristics, by Gender, Add Health Waves I-III (N= 9,311)

	Females (N=4,813)		Males (N=4,498)	
Wave I Sociodemographics	% or Mean (SE) Weighted	N Unweighted	% or Mean (SE) Weighted	N Unweighted
Individual Characteristics				
Age (Years)	15.4 (.11)	-	15.6 (.11)	-
Race/Ethnicity				
NH White	69.1	2,685	68.9	2,519
NH Black	15.6	1,052	14.6	842
Hispanic	11.6	754	12.1	769
NH Asian	3.7	322	4.5	368
Nativity Status				
U.S. born	95.0	4,491	94.8	4,175
Foreign born	5.0	321	5.2	319
Family Background				
Parental Education Level				
Less than high school	12.5	698	12.1	607
High school grad/GED	11.6	569	10.6	493
More than high school	76.0	3,546	77.3	3,398
Family Income				
<$20,000	19.4	918	18.8	825
$20,000-44,999	39.8	1,976	39.9	1,815
$45,000-74,999	27.8	1,312	28.8	1,267
≥$75,000	13.0	607	12.4	591
Family Structure				
2 Biological parents	59.0	2,740	59.0	2,639
Step-Family	9.4	467	10.3	470
1 Biological Parent	27.3	1,354	26.4	1,200
Other Situations	4.3	252	4.4	189

Note: NH = Non-Hispanic; Longitudinal sample weights used.

Table 3.9 presents weighted descriptive statistics and unweighted Ns of experiencing stressful life events at Wave I for the analytic sample. As Aim 3 of the present dissertation examines adolescent SLE for females and males separately, Table 3.9 is stratified by gender.

Of stressful life events done *to* adolescents, females and males reported the highest percentages of seeing violence (7.9% and 11.5%, respectively), being threatened by a knife or gun (5.8% and 16.4%, respectively), suffering a serious injury (10.7% and 17.8%, respectively), and experiencing the end of a romantic relationship (33.1% and 31.7%, respectively). Males also reported a high percentage of being jumped (15.6%) and being injured in a physical fight (10.9%). Of stressful life events performed *by* adolescents, females and males reported the highest percentages of hurting someone in a physical fight (9.1% and 19.5%, respectively) and running away from home (7.8% and 6.7%, respectively).

Table 3.9. Weighted Descriptive Statistics and Unweighted Ns of Experiencing Wave I Stressful Life Events, by Gender, Add Health (N= 9,311)

	Females (N=4,813)		Males (N=4,498)	
Wave I Stressful Life Events	% Weighted	N Unweighted	% Weighted	N Unweighted
SLE To				
Death of mother	0.40	26	0.36	21
Death of father	1.2	69	0.63	56
Friend suicide	3.4	138	2.7	113
Relative suicide	1.1	53	0.71	33
Saw violence	7.9	435	11.5	621
Threatened by knife/gun	5.8	285	16.4	789
Was shot	0.41	18	1.9	87
Was stabbed	2.7	137	6.1	271
Was jumped	4.6	231	15.6	725
Was injured fight	4.7	256	10.9	479
Suffered serious injury	10.7	514	17.8	820
Romantic Rel Ended	33.1	1,534	31.7	1,385
Non-romantic Sexual Rel Ended	2.1	100	2.7	129
Was raped	5.7	278	0.0	0
Contracted an STD	2.9	134	1.1	51
Expelled from school	2.5	114	5.4	232
SLE By				
Suicide attempt injury	1.3	56	0.38	17
Threatened with knife/gun	2.0	101	5.8	287
Shot/stabbed someone	0.66	32	2.1	111
Hurt someone in fight	9.1	446	19.5	857
Had sex for money	0.6	29	1.3	52
Raped Someone	0.0	0	0.98	52
Ran away from home	7.8	389	6.7	275

Note: NH = non-Hispanic; Longitudinal sample weights used.

3.4. Analytic Techniques

The primary objectives of the dissertation are to: 1) examine sociodemographic correlations of AL across adolescence from age 12 to 19 years using NHANES, and 2) determine whether stressful life events in adolescence explain the relationship between adolescent

sociodemographic factors and longitudinal patterns of obesity from adolescence to young adulthood using Add Health. Because each objective has a different outcome and different functional forms, multiple multivariate techniques were employed. For the investigation of allostatic load, negative binomial regression was used. For examining mediation effects of adolescent SLE on change in obesity from adolescence to young adulthood, a portion of the elaboration technique, known as MacKinnon's mediation model, was used employing multinomial and ordinal logistic regression.

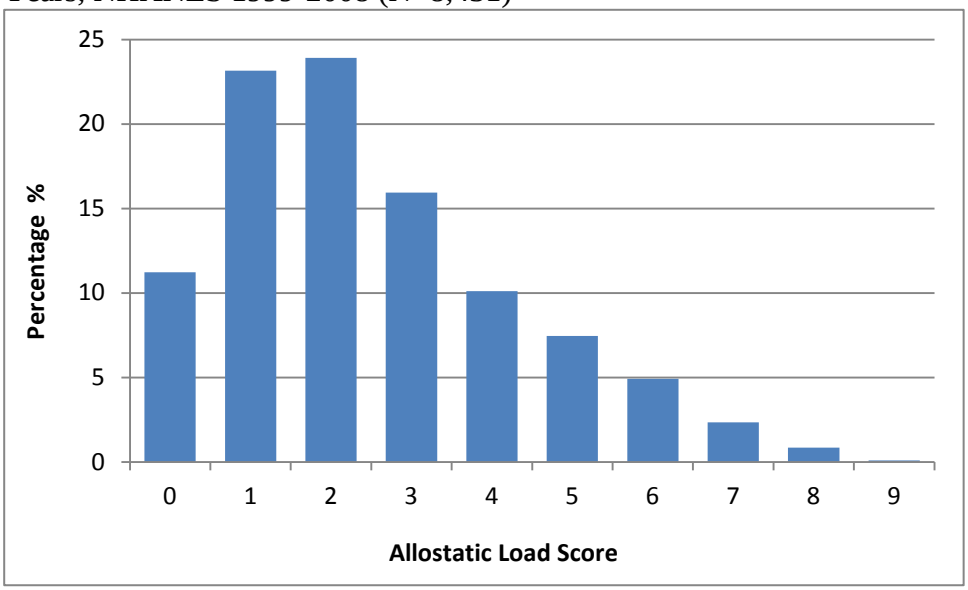
All analytic techniques used for this dissertation employ survey procedures in Stata using appropriate NHANES and Add Health weights discussed earlier. Analyses for the first and second aims were conducted using Stata 12 (StataCorp, 2011), and MPlus Version 6.12 (Muthén & Muthén, 1998-2011) for mediation within the third aim.

3.4.1. Aim 1: To use a nationally representative sample of adolescents in NHANES to examine the change in levels of AL by age and race/ethnicity across adolescence from 12 to 19 years.

3.4.1.1. Negative Binomial Regression

The measure of AL is a non-negative integer count-based summation, which is often conceptualized as the collective occurrence of binomial or multinomial trials of an event. Figure 3.5 presents the percentage distribution of AL among the adolescent sample, and illustrates the non-normal distribution of AL. This distribution is positively skewed where the mean is close to zero. Of nine possible dysregulated biomarkers, the mean of the present sample was 2.50, confirming the positively skewed distribution.

Figure 3.5. Percentage Distribution of Allostatic Load Score among Adolescents Age 12-19 Years, NHANES 1999-2008 (N=8,431)



Given this distribution, if a regular OLS function were used, the estimates would be biased and inconsistent. A Poisson distribution model is more appropriate, although still problematic because of distributional assumptions which may not be in accordance with the empirical distribution of allostatic load. Specifically, Poisson probabilities can be used to create a Poisson distribution defined by parameter λ.

(Eq. 1.)

$$\Pr(y|\lambda) = \frac{e^{-\lambda}\lambda^{y}}{y!} \qquad \text{for y events} = 0,1,2\ldots$$

The Poisson model assumes that the conditional mean and conditional variance are equal to λ (Cameron & Trivedi, 1998; Long & Freese, 2001), or that the variability of counts is equal to the mean (the probability of an event occurring is the same for every individual). In reality, as in the case with AL, the conditional variance often exceeds the conditional mean, also known as overdispersion. When overdispersion occurs, the Poisson model estimates biased standard errors,

overestimated z-values and unwarranted small p-values (Cameron & Trivedi, 1998; Long & Freese, 2001).

When this occurs, the negative binomial function allows for a more flexible Poisson distribution, and for the variance to be greater than or less than the mean (Long, 1997). In the negative binomial function, the outcome is generated through a similar process as the Poisson, but overdispersion is accounted for, as well as estimates of the possible deviation of the variance (unobserved heterogeneity) from that expected under the Poisson distribution.

(Eq. 2)

$$\lambda(\mathrm{X}_1, \mathrm{X}_2, \ldots \mathrm{X}_\mathrm{p}) = \exp(\beta_0 + \beta_1 X_1 + \beta_2 X_2 + \beta_3 X_3 + \beta_4 X_4 \cdots + \beta_p X_p) \exp(\varepsilon_i)$$

where λ = the mean or expected value of the distribution, and ε = unobserved heterogeneity.

Where in ordinary least squares regression, beta represents the change in the outcome variable given a 1-unit change in the predictor, in negative binomial regression the parameter estimates are log counts. Estimated count ratios are determined after exponentiating the log count.

3.4.1.2. Aim 1 Analysis Plan

First, descriptive statistics including the range, mean, quartiles, and empirical cut-points of each of the 9 individual biomarkers used to determine AL score were explored according to the unweighted empirical sample to develop an AL index specific to the population (Crimmins et al., 2003; Crimmins et al., 2007; Geronimus et al., 2006). Next, adjusted Wald tests and bivariate regressions were used to examine differences in mean AL by demographic group. Then, multivariate negative binomial regression models were estimated to examine AL variations by sociodemographic group, specifically highlighting interaction effects of age-by-race/ethnicity. The final interaction negative binomial regression model is presented below:

(Eq. 3)

$$\ln(\text{Allostatic Load Score}) = \beta_0 + \beta_1(\text{Mean-centered Age}) + \beta_2(\text{Gender}) + \beta_3(\text{Race/Ethnicity}) + \beta_4(\text{Nativity Status}) + \beta_5(\text{Household Representative Education}) + \beta_6(\text{Family Income}) + \beta_7(\text{Mean-centered Age} * \text{Race/Ethnicity})$$

Levels of allostatic load were expected to increase with age more so for Black adolescents than White. U.S. born adolescents, those with a parent reporting less than a high school education, and living in low income households were also expected to have higher levels of AL during adolescence.

3.4.2. Aims 2 and 3

Aim 2: To use Add Health to explore adolescent sociodemographic differences in longitudinal patterns of obesity during the transition to adulthood separately for females and males.

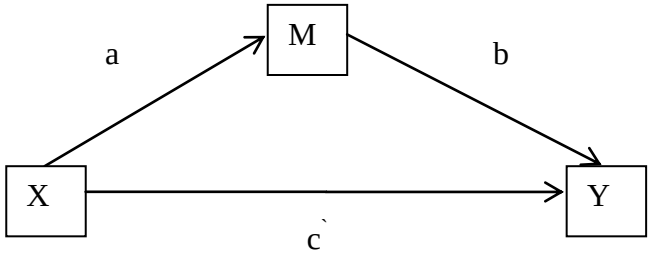
Aim 3: To use Add Health to estimate the effects of adolescent SLE on the relationship between sociodemographic characteristics and longitudinal patterns of obesity from adolescence to adulthood separately for females and males.

3.4.2.1. MacKinnon's Mediation Model

Aims 2 and 3 of the present dissertation use a portion of the elaboration model, known as MacKinnon's mediation model, to estimate the mediating effects of adolescent SLE on the relationship between adolescent sociodemographic characteristics and longitudinal patterns of obesity from adolescence to young adulthood. Below is an illustration of MacKinnon's mediation model: X represents the independent variable of each Wave I adolescent

sociodemographic characteristic, Y is the dependent variable of longitudinal obesity patterns from Waves II to III, and M is the mediating factor of Wave I adolescent SLE.

Figure 3.6. MacKinnon's Mediation Model



The MacKinnon's mediation model is an approach that enables the discovery of relationships between variables in order to better understand the potential mechanism by which variables are related to one another (Aneshensel, 2002). The first phase in the mediation model is identifying whether a relationship exists between each independent variable and the dependent variable (pathway c`) as predicted by theory. In the proposed research, this step is accomplished by examining the relationship between each Wave I sociodemographic characteristic and the progression of obesity from adolescence to young adulthood using multinomial logistic regression (Aim 2).

The second phase is elaborating on the relationship between the independent and dependent variable. This is done by investigating other relationships that are possible components of the focal relationship, otherwise known as mediating variables. In the present research, the associations between the Wave I sociodemographic characteristics and adolescent SLE (pathway a) are examined, as well as that between the mediating variable and the dependent variable (pathway b), or the relationship between adolescent SLE and the obesity patterns from adolescence to young adulthood (Aim 3).

Upon adding the mediating variable, some of the association between the independent and outcome variable should be accounted for. Mediating variables identify how the independent variables affect the dependent variable (Aneshensel, 2002). In the proposed research, the third aim examines the magnitude of the focal relationship upon the addition of the mediating variable. In sum, the mediating variable is the most important part of understanding the cause and effect relationship, because it directly examines the causal mechanism proposed by the theoretical model that links the independent variables to the outcome variable (Aneshensel, 2002).

3.4.2.2. Multinomial Logistic Regression

Aims 2 and 3 of this study use multinomial logistic regression to estimate pathways 'c`' and 'b' within the mediation model. Multinomial regression is suitable when the dependent variable is an unordered nominal variable with j categories, as in the present dissertation with three categories of longitudinal obesity patterns. The regression model estimates the log of the ratio of the probability of being a member of the *j*th category (in this case, become and staying obese) relative to a reference category (stay non-obese/reduce obesity). For *j* categories, this requires *j*-1 equations, one for each category relative to the reference category. The underlying model is:

(Eq. 4)

$$\text{logit}\left(P_j\right) = \log\left[\frac{P_j}{P_0}\right] = \beta_{j0} + \beta_{j1}\text{X}_1 + \beta_{j2}\text{X}_2 + \beta_{j3}\text{X}_3 + \beta_{j4}\text{X}_4 \cdots + \beta_{jk}\text{X}_k$$

Where P is the probability of being in the *j*th longitudinal obesity category relative to the reference category of remaining non- or reduce obesity (P_0). In this equation, β_j is the coefficient for the jth category, and k represents the kth independent variable. In multinomial logistic regression the parameter estimates are log odds. While exponentiating the log odds normally

presents odds ratios, in the context of multinomial logistic regression with a longitudinal component, as presented here, where one category is relative to the reference category, and the other category is held constant, the ratio of the two probabilities is equivalent to the risk ratio (RR) (Long & Freese, 2001).

Interpretation of the RR are as follows: variables that are significant and have a RR greater than 1.0 have an increased risk of becoming obese or staying obese compared to remaining non-obese for that particular sociodemographic category relative to the sociodemographic reference category; variables that are significant and have a RR less than 1.0 have a decreased risk of becoming obese or staying obese compared to remaining non-obese for that particular sociodemographic category relative to the sociodemographic reference category.

3.4.2.3. Aim 2 Analysis Plan

First, distributions of obesity status and longitudinal patterns of obesity by Wave I sociodemographic characteristics were explored separately for females and males. Adjusted Wald tests were employed to test for significance. Next, multivariate analysis using multinomial logistic regression was used to estimate the relationship between each Wave I adolescent sociodemographic factor and obesity change from Wave II to Wave III. Stay non-obese/reduce obesity was set as the base category. Coefficients were then exponentiated to obtain the RR of each obesity outcome as a function of each adolescent sociodemographic characteristic.

A final adjusted model of all Wave I sociodemographic factors on longitudinal patterns of obesity over time was also examined. This adjusted model is presented below by gender:

(Eq. 5)

$$\text{Females: } \ln\left[\frac{P_j}{P_{\text{stay non obese}}}\right] = \beta_{j0} + \beta_{j1}(\text{Age}) + \beta_{j2}(\text{Race}) + \beta_{j3}(\text{Nativity Status}) + \beta_{j4}(\text{Parental Education}) + \beta_{j5}(\text{Family Income}) + \beta_{j6}(\text{Famliy Structure})$$

$$\text{Males: } \ln\left[\frac{P_j}{P_{\text{stay non obese}}}\right] = \beta_{j0} + \beta_{j1}(\text{Age}) + \beta_{j2}(\text{Race}) + \beta_{j3}(\text{Nativity Status}) + \beta_{j4}(\text{Famiy Income}) + \beta_{j5}(\text{Parental Edcation}) + \beta_{j6}(\text{Family Structure })$$

Note: j represents the two categories of obesity progression (become obese and stay obese) compared to the base category (stay non- or reduce obesity).

Black and Hispanic adolescents, compared to White, were expected to have a higher risk of becoming and staying obese relative to remaining non- or reduce obesity. An inverse relationship between factors of SES and becoming or staying obese over time was also expected. U.S.-born adolescents and those living in non-two-biological parent families were also expected to have a greater risk of becoming obese and staying obese from adolescence to young adulthood.

3.4.2.4. Ordinal Logistic Regression

Aim 3 of this dissertation uses ordinal logistic regression to estimate pathway 'a' of the mediation model. Ordinal logistic regression is employed when the dependent variable is an ordered categorical variable. In the present case of pathway 'a', adolescent SLE is an ordinal variable (e.g., 0 SLE, 1 SLE, 2 SLE, and 3+ SLE). An assumption of ordered logistic regression is that the relationship between each pair of outcome groups is the same. Thus, ordered logistic regression assumes that the coefficients describing the relationship between the highest SLE category versus all lower SLE categories are the same as those that describe the relationship between the next lowest SLE category and all higher SLE categories. This is known as the

proportional odds assumption (Long & Freese, 2001). As the relationship between all pairs of SLE groups is the same, only one model is estimated.

Similar to logistic regression, odds ratios are obtained from ordinal logistic regression. Interpretation of such odds ratios is as follows: given a 1-unit increase in an independent variable, the odds of experiencing or performing high SLE versus combined fewer SLE is 'X' greater. Additionally, with only a single estimated model, ordinal logistic regression also generates a latent continuous variable to identify threshold cutpoints that are observed within the distribution of the outcome. These threshold cutpoints are not generally interpretable.

3.4.2.5. Mediation

Aim 3 of this study estimates mediating effects using multinomial logistic regression. While mediation is most commonly done using OLS, it should not be done when working with a logistic function (Aneshensel, 2012; Mood, 2010). The problem stems from changes in logit coefficients (and exponentiated values) from one model to the next, which shows the effect of the added mediator, and the rescaling of the logistic regression equation with the addition of the mediating variable. Unlike OLS, once the mediator is included, the explained variance of the dependent variable increases, ultimately increasing the total variance and thus the scale with which the dependent variable is measured on. By nature of the scale changing so too do the coefficients. In general, rescaling increases the magnitude of the logit coefficient, which may interfere with the mediating effect of the intervening variable, causing biased interpretation.

In sum, logistic regression scaling changes from one model to the next with the inclusion of explanatory variables. The magnitude of the regression coefficient indicates not only the effect of the added mediating variable, but also the rescaling. Thus, the change in the estimate of the

coefficient from one model to the next is really a function of both the effect of the added intervening variable and the rescaling of the model.

To address this concern, a number of approaches have been proposed. One solution using Stata is known as Y* standardization, which allows equivalent scales across models by standardizing the logit coefficients across equations (Aneshensel, 2012; Winship & Mare, 1983). Specifically, Y* standardized coefficients are calculated by dividing the coefficient of the focal independent variable by the estimated standard deviation of Y* within each model[9]. By using Y* standardization, it becomes possible to compare coefficients across nested models (Aneshensel, 2012; Mood, 2010). However, a major limitation of Y*standardization is that it cannot be used in combination with adjustments for complex sample designs. The Add Health, as earlier described, has such as design.

A second solution within Stata is the Karlson/Holm/Breen (KHB) method, which rescales a reduced logistic regression equation of the focal relationship by adding the residuals of the mediating variable to it, formulating a full model (Karlson, Holm, & Breen, 2010; Kohler, Karlson, & Holm, 2011). The KHB method holds constant the scale of the reduced and full model, allowing comparison of the logit coefficients between the two models. While KHB can be used with complex data utilizing sample weights and clustering, it is not capable of stratification, which can be significantly limiting when analyzing survey data such as Add Health.

A third solution to comparing coefficients across logistic regression models is to use path analysis. Path analysis is used to estimate a system of equations among observed variables, or structural relationships, thereby allowing an accurate mediation model (Muthén & Muthén,

[9] Y* is an unobserved continuous variable corresponding to some observed dichotomous form of variable Y, such that Y=0 below some threshold value of Y*, and Y=1at higher values of Y*.

1998-2011). A benefit of path analysis is the ability to estimate mediating effects. To estimate the mediating effect of Wave I adolescent SLE on the relationship between Wave sociodemographic characteristics and longitudinal obesity patterns from Wave II to Wave III, the product of coefficients test is used (MacKinnon et al., 2007b), where I multiply the coefficients of pathway 'a' by pathway 'b'. This is also known as the indirect effect, or how much a 1-unit change in the dependent variable changes the outcome through its effect on the mediator. Thus, I am able to calculate how much of the focal relationship is explained by the mediator. To determine the total effect of each Wave I sociodemographic characteristic on the patterns of obesity over time, I calculate the sum of the indirect (pathway ab) and direct effects (pathway c`), or [c` + ab]. The proportion of total effect mediated is thus equal to the indirect effect divided by the total effect [$\frac{ab}{(c`+ab)}$].

In order to perform path analysis to simultaneously estimate the regression pathways posited in the mediation model, I use MPlus. To approximate accurate standard errors, MPlus uses the Delta Method, an expansion of the calculus known Taylor series to derive a linear function that approximates a more complicated function (more details on the Delta method can be found elsewhere (Hosmer, Lemeshow, & May, 2008)). MPlus also enables a multinomial logistic function (Muthén & Muthén, 1998-2011), as with the present study's multinomial outcome of obesity patterns (Powers & Xie, 2000). Additionally, MPlus allows the use of categorical variables in mediation, whereby indirect effects are determined for each category of the mediator, and total indirect effects can be determined by summing each categorical pathway of indirect effects (Feinberg, 2012; Iacobucci, 2012; MacKinnon & Cox, 2012).

Compared to the KHB method and Y* standardization, path analysis using MPlus offers a relatively uncomplicated and clear approach to determining successful mediation without biased estimates.

3.4.2.6. Aim 3 Analysis Plan

First, distributions of obesity status and longitudinal patterns of obesity by adolescent SLE were explored separately for females and males. Adjusted Wald tests were employed to test for significance. Bivariate and multivariate ordinal logistic regression was also used to estimate pathway 'a'. Next, bivariate and multivariate analysis using multinomial logistic regression was used to estimate the relationship between adolescent SLE and longitudinal patterns of obesity (pathway b). Stay non-obese/reduce obesity was set as the base category. Coefficients were then exponentiated to obtain estimates of the RR of each obesity outcome as a function of adolescent SLE. Given gender differences in obesity and experiences of SLE, all models were gender stratified.

To test for mediation, each of the three mediation model pathways must show significant associations: 1) Wave I sociodemographic characteristics examined must be significantly associated with becoming and/or staying obese over time (Aim 2); 2) a significant relationship between Wave I sociodemographic characteristics and the mediator of adolescent SLE done *to* and performed *by* the adolescent must exist; and 3) the mediator of adolescent SLE must significantly predict the outcome. If these three criteria were satisfied, mediation using path analysis was done. All models were estimated separately for females and males.

Using MPlus, I set "remain non-obese or reduce obese" as the reference category, employing multinomial logistic regression for "become obese" and "stay obese" compared to the reference category to examine mediation. Then using the product of coefficients method, I

calculated the indirect effect. Decomposition of mediation was also determined by calculating the percent of the total effect mediated (MacKinnon, Fairchild, & Fritz, 2007a). An adjusted model of mediation between all Wave I sociodemographic characteristics and longitudinal patterns of obesity over time is presented below by gender.

(Eq. 6)

$$\text{Females: } \ln\left[\frac{P_j}{P_{\text{stay non obese}}}\right] = \beta_{j0} + \beta_{j1}(\text{SLE}_{\text{to/by}}) + \beta_{j2}(\text{Age}) + \beta_{j3}(\text{Race}) + \beta_{j4}(\text{Nativity Status}) + \beta_{j5}(\text{Famly Income}) + \beta_{j6}(\text{Parental Education}) + \beta_{j7}(\text{Family Structure})$$

$$\text{Males: } \ln\left[\frac{P_j}{P_{\text{stay non obese}}}\right] = \beta_{j0} + \beta_{j1}(\text{SLE}_{\text{to/by}}) + \beta_{j2}(\text{Age}) + \beta_{j3}(\text{Race}) + \beta_{j4}(\text{Nativity Status}) + \beta_{j5}(\text{Parental Education}) + \beta_{j6}(\text{Famliy Income}) + \beta_{j7}(\text{Family Structure})$$

Note: j represents the two categories of obesity progression (become obese and stay obese) compared to the base category (stay non- or reduce obesity).

Blacks and Hispanics, and those living in low SES households and non-intact families were expected to experience greater numbers of SLE during adolescence. Higher numbers of SLE were expected to be significantly associated with a higher risk of becoming and staying obese over time. Higher numbers of SLE done *to* and performed *by* adolescents were expected to partially explain the sociodemographic variation in longitudinal patterns of obesity more so than lower numbers of SLE experienced during adolescence.

Chapter 4

AIM 1 RESULTS

4.1. Overview

Aim 1: **To use a nationally representative sample of adolescents in NHANES to examine the change in levels of AL by age and race/ethnicity across adolescence from 12 to 19 years.**

Previous research shows that AL score among adults increases with age, and differs significantly by race/ethnicity, nativity status, and socioeconomic status. During the developmental period of adolescence, AL score is hypothesized to follow similar trends, increasing with age across adolescence and among sociodemographically disadvantaged adolescents. Specifically, levels of AL are expected to be significantly higher among Black adolescents than White, among U.S.-born compared to foreign born adolescents, and among low SES compared to higher SES adolescents. AL score is also expected to significantly increase with age across adolescence, conditional on race/ethnicity.

This chapter is divided into three sections. First, bivariate analyses are used to describe single biomarker cutpoints and summary AL by age group (12 to15 years, 16 to 19 years, and all

ages 12 to 19 years). As the transitional period of adolescence often occurs uniquely among early adolescents (12 to 15 years) and late adolescents (16 to 19 years), it is expected that biomarker cutpoints will increase with age. The second section presents descriptive distributions and mean AL summary scores by sociodemographic characteristics for each age group to further explore changes in AL by age. The third and final section presents a multivariate negative binomial regression model of AL score by sociodemographic characteristics, specifically highlighting the interaction between mean-centered age and race/ethnicity to predict estimated count ratios of AL score conditional on age and race/ethnicity.

4.2. Biomarker and Allostatic Load Distribution by Age

Table 4.1 presents unweighted descriptive statistics for the 9 individual biomarkers and AL summary scores for early adolescents age 12 to 15 years. The first three columns show the range, mean, and standard deviation for each biomarker. The last three columns present quartiles used as cutpoints for determining AL summary scores among the empirical sample of adolescents age 12 to 15 years. Individual biomarker values ≥ 75% were defined as high risk, with the exception of albumin and HDL, for which values ≤ 25% were defined as high risk. AL scores were counts of the number of biomarkers for which an adolescent was considered high risk. The bottom row of Table 4.1 shows the AL summary score. Among adolescents age 12 to 15 years, the range for AL score was 0-9, and the mean AL score was 2.43, with the majority of adolescents also having an AL score of 4 or less.

Table 4.1. Unweighted Descriptive Statistics of Individual Biomarkers among Early Adolescents Ages 12-15, NHANES 1999-2008 (N=4,217)

			Standard	Quartiles		
Biomarker	Range	Mean	Deviation	25%	50%	75%

Cardiovascular Markers						
Diastolic Blood Pressure (mmHg)	15.0-93.0	59.6	11.5	101.0	108.0	114.0
Systolic Blood Pressure (mmHg)	78.0-144.0	107.9	9.8	53.0	60.0	68.0
Metabolic Markers						
Total Cholesterol (mg/dL)	68.0-309.0	159.4	28.5	139.0	157.0	176.0
High-density Lipoprotein (mg/dL)	20.0-122.0	51.6	12.1	43.0	50.0	59.0
Glycosylated Hemoglobin (%)	3.5-15.1	5.18	0.44	5.0	5.2	5.4
Body Mass Index (kg/m²)	13.1-57.2	22.8	5.5	18.9	21.5	25.5
Waist Circumference (cm)	51.4-155	78.7	13.8	68.7	75.3	86.0
Immune/Inflammatory Markers						
C-reactive Protein (mg/dL)	0.01-11.2	0.16	0.45	0.01	0.04	0.12
Albumin (g/dL)	3.1-5.4	4.5	0.30	4.3	4.4	4.6
Allostatic Load Score[a]	0-9	2.43	1.80	1.0	2.0	4.0

[a] Allostatic Load score is determined by counting the number of biomarkers where the respondent has a value ≥75% on the corresponding biomarker, or high risk. Albumin and HDL have a high risk of ≤25%.

Table 4.2 presents similar unweighted descriptive statistics, but for late adolescents age 16 to 19 years. As expected, cutpoints of nearly all biomarkers among late adolescents were higher than those for early adolescents. Mean AL score based on the empirical sample of late adolescents was approximately 2.50, suggesting an increase in mean AL summary score with age.

Table 4.2. Unweighted Descriptive Statistics of Individual Biomarkers among Late Adolescents Ages 16-19, NHANES 1999-2008 (N=4,314)

			Standard	Quartiles		
Biomarker	Range	Mean	Deviation	25%	50%	75%
Cardiovascular Markers						
Diastolic Blood Pressure (mmHg)	10.0-104.0	62.6	11.2	56.0	63.0	70.0
Systolic Blood Pressure (mmHg)	78.0-158.0	111.9	10.6	105.0	112.0	118.0
Metabolic Markers						
Total Cholesterol (mg/dL)	75.0-548.0	163.5	32.7	141.0	159.0	181.0
High-density Lipoprotein (mg/dL)	15.0-121.0	50.8	12.5	42.0	49.0	58.0
Glycosylated Hemoglobin (%)	2.5-14.5	5.15	0.48	5.0	5.1	5.3
Body Mass Index (kg/m²)	14.1-60.9	24.9	5.9	20.8	23.3	27.6
Waist Circumference (cm)	47.1-165.5	84.4	14.9	73.7	80.5	91.5

Immune/Inflammatory Markers						
C-reactive Protein (mg/dL)	0.01-9.7	0.22	0.54	0.03	0.06	0.20
Albumin (g/dL)	1.2-5.7	4.5	0.3	4.3	4.5	4.7
Allostatic Load Score[a]	0-9	2.49	1.87	1.0	2.0	4.0

[a] Allostatic Load score is determined by counting the number of biomarkers where the respondent has a value ≥75% on the corresponding biomarker, or high risk. Albumin and HDL have a high risk of ≤25%.

Upon combining early and late adolescents, Table 4.3 shows descriptive results of individual biomarkers and AL summary scores according to the empirical sample for all ages of adolescents (12 to 19 years). Mean allostatic load score was 2.50, with the majority of adolescents also having an AL score of four or less. Suggesting that although some adolescents could have a score as high as 9, most showed physiological signs of dysregulation for only 4 of the 9 biomarkers. Figure 4.2 shows the percentage distribution of AL score among adolescents age 12 to 19 years, with a peak from 1-2 high risk markers, and a steady decline thereafter.

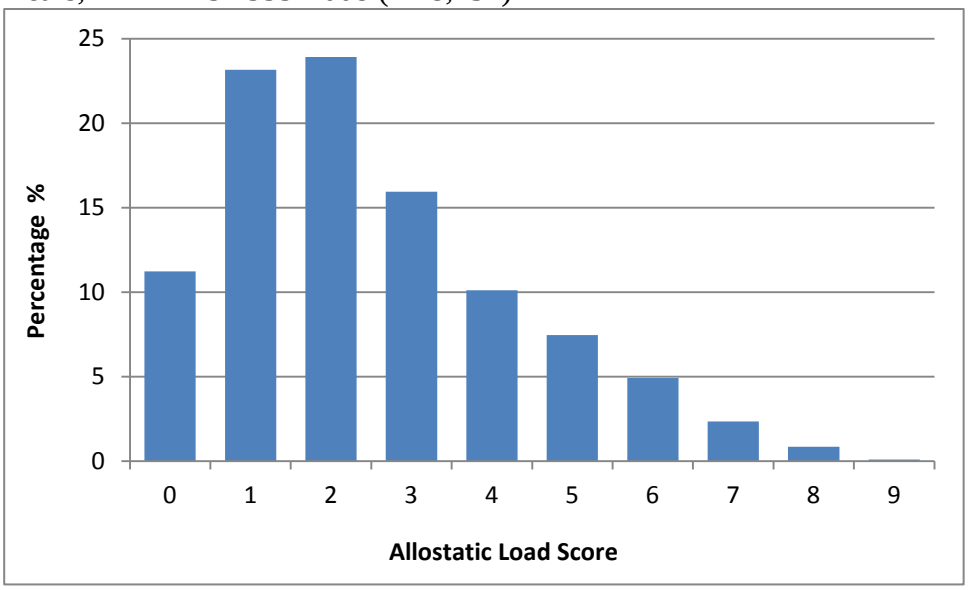
Table 4.3. Descriptive Statistics of Individual Biomarkers among All Adolescents Ages 12-19, NHANES 1999-2008 (N=8,431)

			Standard	Quartiles		
Biomarker	Range	Mean	Deviation	25%	50%	75%
Cardiovascular Markers						
Diastolic Blood Pressure (mmHg)	10.0-104.0	61.10	11.4	54.0	62.0	69.0
Systolic Blood Pressure (mmHg)	78.0-158.0	109.9	10.4	103.0	110.0	116.0
Metabolic Markers						
Total Cholesterol (mg/dL)	68.0-548.0	161.3	30.7	140.0	158.0	179.0
High-density Lipoprotein (mg/dL)	15.0-122.0	51.3	12.3	42.0	50.0	58.0
Glycosylated Hemoglobin (%)	2.50-15.1	5.2	0.5	5.0	5.1	5.3
Body Mass Index (kg/m²)	13.1-60.9	23.9	5.8	19.8	22.4	26.6
Waist Circumference (cm)	47.1-165.6	81.5	14.6	71.2	77.9	88.8
Immune/Inflammatory Markers						
C-reactive Protein (mg/dL)	0.01-11.2	0.19	0.50	0.02	0.05	0.16
Albumin (g/dL)	1.2-5.7	4.46	0.32	4.30	4.50	4.70

Allostatic Load Score[a]	0-9	2.50	1.84	1.00	2.00	4.00

[a] Allostatic Load score is determined by counting the number of biomarkers where the respondent has a value ≥75% on the corresponding biomarker, or high risk. Albumin and HDL have a high risk of ≤25%.

Figure 4.1. Percentage Distribution of Allostatic Load score Among Adolescents Age 12-19 Years, NHANES 1999-2008 (N=8,431)



4.3. Sociodemographics and Allostatic Load

As shown above, examination of individual biomarker and AL summary score distributions separately for early and late adolescents revealed differences in biomarker cutpoints. However, similar AL descriptives were also found according to each empirical sample of ages. To further explore AL by age, Table 4.4 presents weighted mean empirical AL summary scores for all ages, early, and late adolescents by each sociodemographic variable. Adjusted Wald tests were done separately for all ages (12 to 19 years), early (12 to 15 years) and late adolescents (16 to 19 years).

Virtually identical mean AL distributions by sociodemographic characteristics were found among all ages, early, and late adolescents. Mean AL scores did not differ significantly by gender. Non-Hispanic Black adolescents had the highest mean AL, followed by Hispanics and

Whites. U.S.-born adolescents had significantly higher mean AL scores than foreign born adolescents. Mean AL scores also increased monotonically (indicating worse health) with lower household representative education level and family income.

Table 4.4. Weighted Mean Allostatic Load Score among All Ages, Early, and Late Adolescents by Sociodemographics, NHANES 1999-2008 (N=8,431)

Sociodemographics	Mean AL Ages 12-19[a]	Mean AL Ages 12-15[b]	Mean AL Ages 16-19[c]
Individual Characteristics			
Age (Years)	2.50	2.43	2.49
Gender			
Male	2.51	2.43	2.49
Female	2.49	2.44	2.50
Race/ethnicity			
NH White	2.30***	2.20***	2.31***
NH Black	2.79	2.72	2.79
Hispanic	2.41	2.35	2.38
Nativity status			
U.S.-born	2.54***	2.46	2.57***
Foreign born	2.22	2.25	2.13
Family Background			
Household Represent Education			
Less than high school	2.62***	2.53***	2.65***
High school/GED	2.51	2.47	2.48
More than high school	2.38	2.33	2.38
Family income			
<$20,000	2.64***	2.52***	2.65***
$20,000-44,999	2.53	5.49	2.53
$45,000-74,999	2.49	2.48	2.47
≥$75,000	2.17	2.10	2.17

Note: Analysis weighted; NH= non-Hispanic.
*p≤.05, **p≤.01, *** p≤ .001.
[a] Wald test for all ages 12-19 years.
[b] Wald test for early adolescents age 12-15 years.
[c] Wald test for late adolescents age 16-19 years.

Given that Aim 1 of this study focuses on racial/ethnic differences in AL across adolescence from 12 to 19 years, and mean AL distributions by sociodemographic characteristics

were similar for early and late adolescents, there was justification to compute AL cutpoints based on the sample of all adolescents age 12 to 19 years. Therefore, the present dissertation collapsed early and late adolescents and created AL summary scores from this sample of all ages to perform negative binomial regression.

4.4. Multivariate Negative Binomial Regression Model of AL Score

Table 4.5 presents results from the negative binomial regression model of AL score among adolescents age 12 to 19 years, and includes the mean-centered age by race/ethnicity interaction. There were no significant gender differences in level of AL. AL significantly increased with age and Blacks had higher AL scores than Whites. However, the rate of increase in AL over age for Blacks was significantly lower than Whites ($p=0.019$), as demonstrated by the convergence of the race-specific trajectories in Figure 4.2. In other words, the trajectory of predicted AL scores among Whites had the greatest positive slope; beginning at the lowest score relative to Blacks and Hispanics, then at around age 16 surpassing Hispanics and approaching the AL score of Blacks. There was no difference between Whites and Hispanics by age.

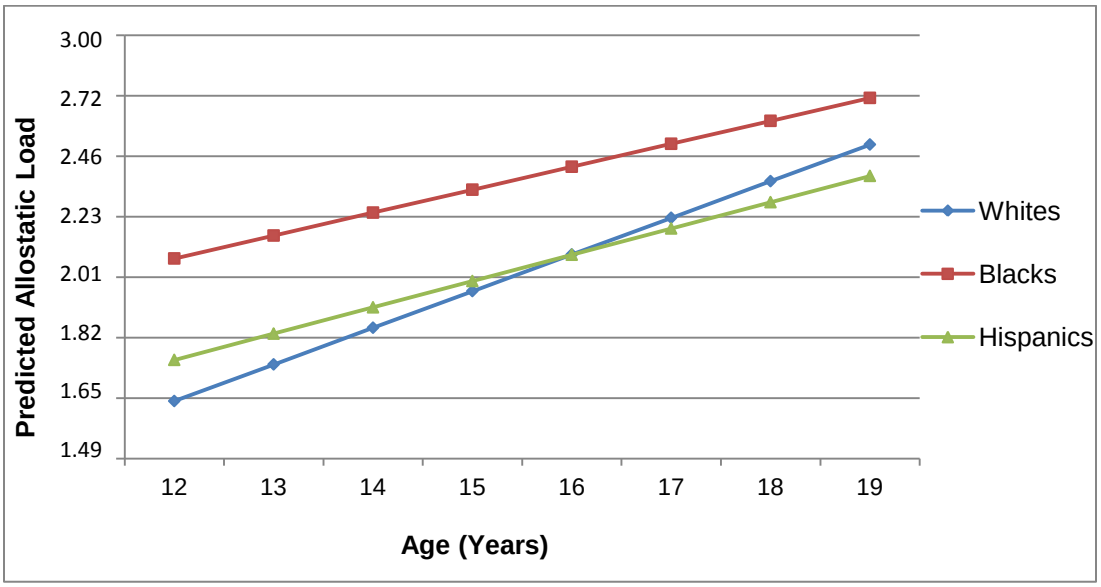
As anticipated, those who were foreign born had significantly lower AL than U.S.-born. An inverse relationship between family background measures of socioeconomic status and AL score were also found. Adolescents whose household representative had less than a high school education had significantly higher AL scores than those whose representative had more than a high school education. Adolescents living in lower income households also had significantly higher levels of AL compared to those with the highest family income.

Table 4.5. Negative Binomial Regression Results of Age by Race/Ethnicity and Other Sociodemographic Characteristics for Allostatic Load Score among Adolescents Age 12-19, NHANES 1999-2008 (N=8,431)

Sociodemographics (reference)	Coefficient	Adjusted Count Ratio[a]	95% CI
Individual Characteristics			
Gender (Male)			
Female	-0.02	0.98	0.94, 1.03
Mean-centered Age (15.4 Years)	0.06***	1.06	1.05, 1.08
Race/Ethnicity (NH White)			
NH Black	0.50***	1.66	1.23, 2.33
Hispanic	0.27	1.31	0.97, 1.76
Mean-centered Age by Race/Ethnicity (Mean-centered Age x NH White)			
Age x NH Black	-0.02*	0.98	0.96, 0.99
Age x Hispanic	-0.02	0.98	0.97, 1.01
Nativity Status (U.S.-born)			
Foreign born	-0.23***	0.80	0.74, 0.86
Family Background			
Household Representative Education (More than high school)			
Less than high school	0.14***	1.15	1.07, 1.23
High school grad/GED	0.07*	1.07	1.01, 1.14
Family Income (≥$75,000)			
<$20,000	0.16***	1.18	1.10, 1.25
$20,000-$44,999	0.15***	1.16	1.09, 1.24
$45,000-$74,999	0.10**	1.11	1.03, 1.19
Intercept	-0.23***	0.79	0.62, 1.01

Note: Analysis weighted; NH= non-Hispanic; CI= Confidence intervals.
*p≤ .05, **p≤ .01, ***p ≤ .001.
[a] Adjusted Count Ratios are interpreted as: Holding all other variables in the model constant, each 1-unit change in the predictor variable is expected to change the estimated AL score by a factor of the respective count ratio relative to the reference category.

Figure 4.2. Predicted Allostatic Load by Mean-Centered Age (15.4 Years) and Race/Ethnicity, NHANES (1999-2008)



Note: Age 12 to 19 years; Analysis weighted; Predicted values of AL are based on coefficients within the negative binomial regression model of the interaction between age and race/ethnicity. The interaction is interpreted as predicted AL score by race/ethnicity conditional on age, with covariates set equal to the mean or the reference category.

4.5. Chapter Summary

The purpose of this chapter was to examine sociodemographic differences in AL across adolescence from age 12 to 19 years. Although there was concern regarding separate analysis for early and late adolescents, bivariate analysis of the covariates revealed similar patterns across age groups. Therefore, there was justification to compute AL cutpoints based on the sample of all adolescents age 12 to 19 years. As expected, the empirical values of the summary AL scores were highly skewed, supporting the utilization of the negative binomial functional form for regression modeling.

Multivariate analysis revealed significant findings for nativity status, education, and income. I also found a significant age-by-race/ethnicity interaction. Although AL significantly increased with age and was higher for non-Hispanic Blacks compared to Whites, the rate at which AL increased for Blacks was significantly lower than Whites. This indicates that Whites (and Hispanics) approach the level of AL of Blacks as they age.

The present results support my theoretical model, which uses the life course approach of stressor accumulation over time, and greater cumulative biological dysregulation with age. These findings are also consistent with Link and Phelan's (1995; 2010) theory of social conditions and fundamental causes of health differences. The increased stress of lower SES, coupled with the developmental period of adolescence, potentially contributes to higher levels of AL during adolescence, which may continue into in adulthood (Karlamangla et al., 2002; Karlamangla, Singer, & Seeman, 2006; Seeman et al., 2004). Research on AL provides important insight into the linkage between early life stressors and adverse outcomes over the life course, and can also help inform health interventions at earlier life stages aimed at eliminating health disparities.

Chapter 5

AIM 2 RESULTS

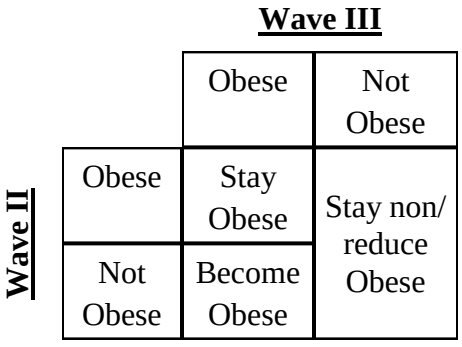
5.1. Overview

Aim 2: To use Add Health to examine adolescent sociodemographic differences in longitudinal patterns of obesity during the transition to adulthood separately for females and males.

To achieve my second aim, I of examine the relationship between the independent variables of Wave I sociodemographics and the outcome of change in obesity status over time. Specifically, bivariate distributions and multivariate analyses using multinomial logistic regression were done. This analysis aligns with my proposed mediation model of pathway 'c``.

Longitudinal patterns of obesity from adolescence to young adulthood were defined according to measures of body mass index from Wave II to Wave III of Add Health, and are consistent with previous research (Gordon-Larsen et al., 2004; Gordon-Larsen et al., 2010; Lee et al., 2009). Three main obesity pattern categories were determined (Figure 5.1): *Becoming obese* (adolescent not obese at Wave II but obese at Wave III); *Staying Obese* (obese at both Waves II and III); and *Staying Non- or Reduce Obese* (not obese at both Waves II and III, or obese at Wave II but not obese at Wave III).

Figure 5.1. Categorization of Longitudinal Obesity Patterns from Wave II to Wave III, Add Health



Previous research has shown that racial/ethnic disparities in obesity exist, and that they differ by gender. Thus, I hypothesized that among females, Blacks would be more likely to have a higher risk of becoming and staying obese relative to Whites. Among males, Hispanics would be more likely to transition to obesity and remain obese compared to Whites. Additionally, foreign born adolescents would be less likely to become or stay obese over time compared to U.S.-born adolescents.

Guided by my conceptual framework that shapes social conditions as fundamental causes of health conditions (Link & Phelan, 1995; Phelan et al., 2010), I also investigated the impact of Wave I family background characteristics on subsequent patterns of obesity across the transition to adulthood. I hypothesized that disadvantaged adolescents, including those with parents who have less than a high school education, in lower income households, and non-two-biological parent family situations, would have a higher risk of becoming and staying obese over time compared to higher SES adolescents.

This chapter is organized into two sections. First, weighted distributions of obesity status and longitudinal patterns of obesity by each Wave I sociodemographic characteristic are presented, separately for females and males. Adjusted Wald tests are employed to test for significance. Second, multivariate analysis using multinomial logistic regression to estimate the relationship between Wave I sociodemographics and longitudinal patterns of obesity are shown, highlighting exponentiated coefficients to obtain estimates of the risk ratio (RR) of each obesity outcome as a function of each Wave I sociodemographic characteristic.

5.2. Distribution of Obesity Status and Longitudinal Patterns of Obesity by Wave I Sociodemographic Characteristics

5.2.1. Distributions among Females

Table 5.1 presents bivariate relationships for obesity at Wave II, III, and longitudinal obesity patterns for females. Column 1 shows the patterns for Wave II; overall, 11.1% were obese. There were significant differences in obesity status at Wave II according to race/ethnicity with the highest percentages reported among Blacks and the lowest among Asians. U.S.-born girls had significantly higher percentages of obesity than foreign born. Parental and household indicators measured at Wave I were less significant. There was no relationship between daughters' obesity status and parental education or family structure. However, there was a significant relationship according to family income, with girls in lower income families having higher percentages of obesity. By Wave III, obesity prevalence had increased to 22.3% ($p<.001$ for adjusted Wald test between Wave II and Wave III obesity, results not shown). Overall, the sociodemographic effects remained the same with regards to significance, except family income and structure became significant, with girls who have lower educated parents and who live in one biological or other family situations having the highest obesity prevalence. It should be noted that the prevalence levels of obesity increased in virtually all categories of every variable.

The last three columns of Table 5.1 show the longitudinal patterns of obesity from Wave II to Wave III. Overall, 12.7% became obese between the two time periods, 9.7% stayed obese, and 77.7% remained non- or reduce obesity. Every sociodemographic characteristic measured at Wave I was associated with the longitudinal measure of obesity. Black females had the highest

rates of becoming and staying obese between the two waves. This pattern was also true for U.S.-born females. Similarly girls whose parents had the lowest levels of education and income had the highest rates of becoming obese and staying obese. Last, females living in one biological parent or other family situations had the highest levels of becoming and staying obese, although the differences in staying obese across all of the family structure categories was less pronounced than in other sociodemographic characteristics.

Table 5.1. Weighted Percents and Standard Errors of Obesity Status and Patterns of Obesity by Wave I Sociodemographic Characteristics, Females, Add Health Waves I-III (N=4,813)

	Cross-Sectional Obesity[a] % (SE)		Longitudinal Obesity Patterns[b] % (SE)		
Wave I Sociodemographics	Wave II	Wave III	Become Obese	Stay Obese	Stay Non/Reduce Obese
Individual Characteristics					
Age (12-19 Years)	11.1 (.008)	22.3 (.011)	12.7 (.008)	9.7 (.008)	77.7 (.001)
Race/Ethnicity					
NH White	9.7 (.009)***	20.2 (.013)***	11.6 (.009)***	8.5 (.008)	79.8 (.013)
NH Black	17.0 (.015)	32.7 (.025)	17.3 (.018)	15.4 (.013)	67.3 (.025)
Hispanic	12.8 (.020)	25.8 (.022)	14.5 (.021)	11.3 (.018)	74.2 (.022)
NH Asian	2.8 (.012)	8.5 (.028)	7.3 (.027)	1.2 (.006)	91.5 (.028)
Nativity Status					
U.S.-born	11.3 (.008)*	22.9 (.012)**	13.0 (.008)**	9.9 (.007)	77.1 (.012)
Foreign born	6.6 (.018)	11.7 (.027)	6.8 (.018)	4.8 (.018)	88.3 (.027)
Family Background					
Parental Education Level					
Less than High School	13.9 (.019)	30.2 (.023)**	17.7 (.020)**	12.6 (.018)	69.8 (.023)
High School Grad	10.7 (.015)	25.2 (.028)	15.4 (.025)	9.8 (.014)	74.8 (.028)
More than High School	10.6 (.009)	20.6 (.013)	11.5 (.008)	9.2 (.008)	79.4 (.013)
Family Income					
<$20,000	15.1 (.016)***	30.1 (.028)***	16.5 (.019)***	13.6 (.015)	69.9 (.028)
$20,000-44,999	12.3 (.010)	24.8 (.015)	14.2 (.011)	10.6 (.009)	75.2 (.014)
$45,000-74,999	7.7 (.010)	17.3 (.015)	10.7 (.014)	6.6 (.009)	82.7 (.015)
≥$75,000	8.3 (.018)	13.8 (.002)	6.5 (.015)	7.3 (.017)	86.2 (.023)
Family Structure					
2 Biological Parents	10.5 (.009)	20.5 (.013)**	11.2 (.009)*	9.3 (.009)	79.5 (.012)
Step-Family	9.5 (.016)	18.5 (.024)	10.5 (.019)	8.0 (.015)	81.6 (.024)
1 Biological Parent	12.6 (.011)	26.9 (.020)	16.0 (.016)	10.9 (.010)	73.0 (.020)
Other Situations	12.5 (.033)	27.3 (.042)	16.8 (.033)	10.5 (.029)	72.7 (.042)

Note: Design-based Wald test for bivariate analysis; NH= non-Hispanic.
* p≤0.05, **p≤0.01, ***p≤0.001.
[a] Wald test conducted separately for Wave II and Wave III.
[b] Wald test compared differences across the 3 categories of longitudinal obesity outcome.

5.2.2. Distributions among Males

Table 5.2 shows bivariate relationships for obesity at Wave II, III, and longitudinal obesity patterns for males. Overall, there were few significant relationships between males' obesity and Wave I sociodemographic characteristics. Column 1 shows the patterns for Wave II; overall, 13.6% of boys were obese. There were no significant relationships for obesity measured at Wave II. By Wave III, 19.9% of boys were obese ($p<.001$ for adjusted Wald test between Wave II and Wave III obesity, results not shown); the only significant relationship was for family income. Males from lower income families had significantly higher percentages of obesity than those from more affluent families. The last three columns show the longitudinal pattern of obesity between Waves II and III. Among males 9.3% became obese and 10.6% remained obese across the two waves. There were no significant associations between Wave I sociodemographic characteristics and longitudinal obesity patterns except, again, for family income. Here, the real distinctions in obesity rates were in the highest income category compared to all others. For example, among males who became obese, the percentages are very similar for the first three income categories and substantially lower in the highest ($\geq$\$75,000).

Table 5.2. Weighted Percents and Standard Errors of Obesity Status and Patterns of Obesity by Wave I Sociodemographic Characteristics, Males, Add Health Waves I-III (N=4,498)

	Cross-Sectional Obesity[a] % (SE)		Longitudinal Obesity Patterns[b] % (SE)		
Wave I Sociodemographics	Wave II	Wave III	Become Obese	Stay Obese	Stay Non/Reduce Obese
Individual Characteristics					
Age (12-19 Years)	13.6 (.008)	19.9 (.009)	9.3 (.007)	10.6 (.008)	80.1 (.009)
Race/Ethnicity					
NH White	13.8 (.011)	19.2 (.012)	8.8 (.007)	10.5 (.010)	80.8 (.012)
NH Black	13.8 (.018)	22.7 (.023)	11.5 (.020)	11.3 (.018)	77.3 (.023)
Hispanic	14.0 (.018)	20.6 (.021)	9.7 (.015)	10.9 (.015)	79.4 (.021)
NH Asian	9.5 (.023)	18.3 (.037)	9.3 (.027)	9.1 (.023)	81.7 (.037)
Nativity Status					
U.S.-born	13.9 (.009)	20.0 (.010)	9.3 (.007)	10.6 (.008)	80.0 (.010)
Foreign born	9.5 (.024)	18.0 (.030)	8.7 (.022)	9.4 (.024)	82.0 (.030)
Family Background					
Parental Education Level					
Less than High School	13.1 (.021)	18.9 (.024)	9.9 (.017)	9.0 (.017)	81.1 (.024)
High School Grad	16.8 (.022)	20.3 (.026)	7.5 (.016)	12.8 (.019)	79.7 (.026)
More than High School	13.3 (.010)	19.9 (.011)	9.5 (.007)	10.5 (.009)	80.1 (.011)
Family Income					
<$20,000	16.9 (.021)	21.2 (.022)***	9.4 (.013)*	11.8 (.019)	78.8 (.022)
$20,000-44,999	14.3 (.013)	21.9 (.014)	10.3 (.011)	11.7 (.012)	78.1 (.014)
$45,000-74,999	12.0 (.014)	19.3 (.016)	9.8 (.011)	9.5 (.013)	80.7 (.016)
≥$75,000	10.3 (.017)	12.5 (.020)	4.8 (.012)	7.6 (.015)	87.6 (.020)
Family Structure					
2 Biological Parents	13.7 (.011)	19.0 (.012)	8.6 (.007)	10.4 (.010)	81.0 (.012)
Step-Family	12.2 (.020)	20.5 (.026)	9.8 (.018)	10.7 (.020)	79.5 (.026)
1 Biological Parent	13.9 (.015)	20.1 (.015)	9.4 (.012)	10.6 (.013)	80.0 (.015)
Other Situations	15.2 (.033)	27.9 (.039)	16.4 (.035)	11.5 (.030)	72.2 (.039)

Note: Design-based Wald test for bivariate analysis; NH= non-Hispanic.
* p≤0.05, **p≤0.01, ***p≤0.001.
[a] Wald test conducted separately for Wave II and Wave III.
[b] Wald test compared differences across the 3 categories of longitudinal obesity outcome.

5.3. Multinomial Logistic Regression Effects of Wave I Sociodemographic Characteristics on Longitudinal Patterns of Obesity by Gender

Tables 5.3-5.8 show separate bivariate and final full multivariate multinomial logistic regression models of Wave I sociodemographic characteristics on longitudinal obesity patterns. All models are gender stratified. Risk ratios (RR) and their 95% confidence intervals are presented. Interpretation of the risk ratio is as follows: variables that are significant and have a risk ratio greater than 1.0 have an increased risk of becoming obese or staying obese compared to remaining non-obese or reducing obesity for that particular sociodemographic category relative to the sociodemographic reference category; variables that are significant and have a risk ratio less than 1.0 have a decreased risk of becoming obese or staying obese compared to remaining non-obese or reducing obesity for that particular sociodemographic category relative to the sociodemographic reference category.

5.3.1. Multinomial Logistic Regression Results among Females

Unadjusted results, individual characteristics, Wave I. Table 5.3 shows the unadjusted risk ratios and the 95% confidence intervals of age, race/ethnicity, and nativity status on becoming obese and staying obese compared to the reference group (non-obese/becoming non-obese). With each year increase in adolescence, females are significantly more likely to become obese relative to the reference group (panel a). Compared to Whites, Black females were significantly more likely to become obese relative to the reference group between Waves II and III. There were no differences between White, Hispanic, and Asian females (panel b). Foreign born females were significantly less likely than U.S.-born to become obese relative to the reference group (panel c). Moreover, older females were significantly more likely to stay obese

over time, as well as Black females compared to Whites. Asians were significantly less likely to stay obese compared to Whites. Similarly, foreign born females were significantly less likely than U.S.-born to stay obese relative to the reference group.

Table 5.3. Bivariate Multinomial Logistic Regression of Longitudinal Obesity Patterns by Wave I Individual Characteristics, Females, Add Health Waves I-III (N=4,813)

Wave I Individual Characteristics (reference)	Become Obese		Stay Obese	
	RR	95% CI	RR	95% CI
(a)				
Age (Years)	1.12*	1.02, 1.22	1.10*	1.01, 1.20
(b)				
Race/Ethnicity (NH White)				
NH Black	1.77***	1.27, 2.46	2.14***	1.58, 2.90
Hispanic	1.35	0.94, 1.93	1.43	0.95, 2.14
NH Asian	0.55	0.25, 1.20	0.12***	0.04, 0.35
(c)				
Nativity Status (U.S.-born)				
Foreign born	0.46*	0.25, 0.84	0.43*	0.19, 0.94

Note: Analysis weighted; NH= non-Hispanic; RR= Risk ratios; CI= Confidence intervals.
* p≤0.05, **p≤0.01, ***p≤0.001

Unadjusted results, family background characteristics, Wave I. Table 5.5 shows the unadjusted risk ratios and 95% confidence intervals of parental education (panel a), family income (panel b), and family structure (panel c) on becoming and staying obese compared to staying non-obese or reduce obesity. Compared to adolescents whose parent had the highest level of education, those with the lowest were significantly more likely to become obese relative to the reference group. Similarly, compared to adolescents whose parents reported the highest level of income, those with the two lowest had significantly higher risks of becoming obese. The effects were stronger for the lowest income (<$20,000, RR=3.12) than the next lowest income ($20,000-

$44,999, RR=2.50). Compared to young adults who came from two biological parent families, those from single biological parent families were significantly more likely to become obese relative to the reference.

The results were somewhat different for staying obese. The effects of parental education were similar to becoming obese, with adolescents whose parent had the lowest level of education, compared to those with the highest education level, significantly more likely to stay obese (although the RR was somewhat smaller). For income, compared to those with the highest income, only those with the lowest were significantly more likely to stay obese relative to the reference. Family structure was not significant.

Table 5.4. Bivariate Multinomial Logistic Regression of Longitudinal Obesity Patterns by Wave I Family Background Characteristics, Females, Add Health Waves I-III (N=4,813)

Wave I Family Background Characteristics (reference)	Become Obese		Stay Obese	
	RR	95% CI	RR	95% CI
(a)				
Parental Education Level (More than High School)				
Less than High School	1.75***	1.31, 2.34	1.56*	1.07, 2.29
High School Grad/GED	1.43	0.92, 2.22	1.13	0.78, 1.64
(b)				
Family Income (≥$75,000)				
<$20,000	3.12***	1.78, 5.47	2.28**	1.31, 3.98
$20,000-44,999	2.50***	1.53, 4.09	1.67	0.99, 2.80
$45,000-74,999	1.71	0.95, 3.09	0.94	0.58, 1.53
(c)				
Family Structure (2 Biological Parents)				
Step-Family	0.91	0.58, 1.43	0.84	0.54, 1.29
1 Biological Parent	1.55**	1.15, 2.08	1.28	0.99, 1.67
Other Situations	1.64	0.99, 2.72	1.24	0.63, 2.42

Note: Analysis weighted; NH= non-Hispanic; RR= Risk ratios; CI= Confidence intervals.
* p≤0.05, **p≤0.01, ***p≤0.001

Adjusted full model. Table 5.5 shows the combined results for individual and family background characteristics on becoming and staying obese among females. Age at Wave I remained positively and significantly associated with becoming obese relative to the reference. Race effects were no longer significant when family background characteristics were also considered. Compared to U.S.-born, foreign born were significantly less likely to become obese relative to the reference. Parental education was no longer significant when individual characteristics were taken into account. The effects of income were the same as in the bivariate model, but the effects were reduced. Family structure was no longer associated with becoming obese.

The results for staying obese were a bit more consistent across the bivariate and multivariate models than those for becoming obese. Compared to Whites, Blacks were significantly more likely to stay obese, and Asians were significantly less likely to stay obese relative to the reference category. The effects of nativity status remained the same as in the unadjusted model. However the effects of parent education were no longer significant. Family income remained significant with those in the lowest income category compared to the highest category having a higher risk of staying obese relative to reference. Family structure effects remained non-significant.

Table 5.5. Multivariate Multinomial Logistic Regression of Longitudinal Obesity Patterns by Wave I Sociodemographic Characteristics, Females, Add Health Waves I-III (N=4,813)

Wave I Sociodemographics (reference)	Become Obese		Stay Obese	
	RR	95% CI	RR	95% CI
Individual Characteristics				
Age (Years)	1.11*	1.02, 1.21	1.10*	1.01, 1.20
Race/Ethnicity (NH White)				
NH Black	1.38	0.97, 1.97	1.83***	1.38, 2.44
Hispanic	1.17	0.74, 1.84	1.32	0.84, 2.08
NH Asian	0.74	0.30, 1.84	0.16***	0.05, 0.49
Nativity Status (U.S.-born)				
Foreign born	0.38*	0.18, 0.80	0.42*	0.18, 0.98
Family Background				
Parental Education Level (More than High School)				
Less than High School	1.41	0.98, 2.04	1.14	0.71, 1.81
High School Grad/GED	1.19	0.75, 1.89	0.92	0.60, 1.43
Family Income (≥$75,000)				
<$20,000	2.52**	1.42, 4.47	2.14*	1.19, 3.82
$20,000-44,999	2.28**	1.37, 3.81	1.65	0.98, 2.80
$45,000-74,999	1.73	0.95, 3.14	0.94	0.57, 1.53
Family Structure (2 Biological Parents)				
Step-Family	0.79	0.51, 1.24	0.75	0.47, 1.22
1 Biological Parent	1.05	0.78, 1.43	0.82	0.59, 1.13
Other Situations	1.12	0.68, 1.85	0.71	0.36, 1.39

Note: Analysis weighted; NH= non-Hispanic; RR= Risk ratios; CI= Confidence intervals.
* p≤0.05, **p≤0.01, ***p≤0.001

5.3.2. Multinomial Logistic Regression Results among Males

Unadjusted results, individual characteristics, Wave I. Table 5.6 shows the unadjusted risk ratios and the 95% confidence intervals of age, race/ethnicity, and nativity status on becoming obese and staying obese compared to the reference group (non-obese/becoming non-obese) among males. With each year increase in adolescence, males were significantly more

likely to become obese relative to the reference group (panel a). Race/ethnicity and nativity status were not significantly associated with becoming obese relative to the reference group. Similarly, individual characteristics were not significantly associated with staying obese among males.

Table 5.6. Bivariate Multinomial Logistic Regression of Longitudinal Obesity Patterns by Wave I Individual Characteristics, Males, Add Health Waves I-III (N=4,498)

Wave I Individual Characteristics (reference)	Become Obese		Stay Obese	
	RR	95% CI	RR	95% CI
(a)				
Age (12-19 years)	1.17***	1.08, 1.27	1.00	0.90, 1.11
(b)				
Race/Ethnicity (NH White)				
NH Black	1.37	0.88, 2.13	1.13	0.75, 1.69
Hispanic	1.13	0.77, 1.64	1.06	0.74, 1.53
NH Asian	1.05	0.54, 2.04	0.86	0.48, 1.55
(c)				
Nativity Status (U.S.-born)				
Foreign born	0.91	0.52, 1.56	0.86	0.48, 1.55

Note: Analysis weighted; NH= non-Hispanic; RR= Risk ratios; CI= Confidence intervals.
* $p \leq 0.05$, ** $p \leq 0.01$, *** $p \leq 0.001$

Unadjusted results, family background characteristics, Wave I. Table 5.7 shows the unadjusted risk ratios and 95% confidence intervals of parental education, family income, and family structure on becoming and staying obese compared to being non-obese/becoming non-obese among males. Parental education was not significantly associated with becoming obese relative to the reference group for males. Compared to boys living in families with the highest income, those in every other income category were significantly more likely to become obese relative to the reference. Compared to boys who grew up with two biological parents, those who

lived in other situations during adolescence were significantly more likely to become obese relative to the reference. Parental education was also not associated with staying obese among males.

The results of family income and family structure were somewhat different for staying obese. Compared to the highest income families, boys in the two lowest categories were significantly more likely to stay obese relative to the reference. However, the magnitude of the effects was smaller than for becoming obese. Family structure was not associated with staying obese.

Table 5.7. Bivariate Multinomial Logistic Regression of Longitudinal Obesity Patterns by Wave I Family Background Characteristics, Males, Add Health Waves I-III (N=4,498)

Wave I Family Background Characteristics (reference)	Become Obese		Stay Obese	
	RR	95% CI	RR	95% CI
(a)				
Parental Education Level (More than High School)				
Less than High School	1.03	0.67, 1.55	0.84	0.54, 1.32
High School Grad/GED	0.80	0.50, 1.28	1.23	0.81, 1.86
(b)				
Family Income (≥$75,000)				
<$20,000	2.15*	1.16, 3.97	1.72*	1.05, 2.83
$20,000-44,999	2.38**	1.30, 4.34	1.72*	1.09, 2.73
$45,000-74,999	2.20**	1.23, 3.91	1.36	0.80, 2.31
(c)				
Family Structure (2 Biological Parents)				
Step-Family	1.16	0.77, 1.74	1.05	0.64, 1.70
1 Biological Parent	1.11	0.80, 1.55	1.03	0.77, 1.39
Other Situations	2.14**	1.24, 3.68	1.23	0.68, 2.22

Note: Analysis weighted; NH= non-Hispanic; RR= Risk ratios; CI= Confidence intervals.
* p≤0.05, **p≤0.01, ***p≤0.001

Adjusted full model. Table 5.8 shows the combined results for individual and family background characteristics on becoming and staying obese. Age at Wave I remained significantly and positively associated with becoming obese in the full model. There were no significant racial or ethnic differences. Parental education was also not significantly associated with becoming obese. The effects of family income were similar as those in the unadjusted model. Namely, compared to boys living in families with the highest income, those living in every other income category were more likely to become obese. However, in contrast to the unadjusted model, family structure was no longer significant.

There were few significant results for the staying obese outcome. Age, race/ethnicity, nativity status, parental education, and family structure were not significant. Only family income was significant. Compared to boys living in families with the highest income, those living in the two lowest categories were significantly more likely to stay obese relative to the reference.

Table 5.8. Multivariate Multinomial Logistic Regression of Longitudinal Obesity Patterns by Wave I Sociodemographic Characteristics, Males, Add Health Waves I-III (N=4,498)

Wave I Sociodemographics (reference)	Become Obese		Stay Obese	
	RR	95% CI	RR	95% CI
Individual Characteristics				
Age (Years)	1.17***	1.07, 1.27	1.00	0.90, 1.12
Race/Ethnicity (NH White)				
NH Black	1.22	0.80, 1.86	1.04	0.66, 1.64
Hispanic	1.18	0.76, 1.83	1.18	0.77, 1.81
NH Asian	1.13	0.57, 2.23	0.81	0.47, 1.76
Nativity Status (U.S.-born)				
Foreign born	0.76	0.43, 1.33	0.85	0.73, 1.69
Family Background				
Parental Education Level (More than High School)				
Less than High School	0.89	0.52, 1.51	0.67	0.41, 1.17
High School Grad/GED	0.67	0.10, 1.17	1.08	0.68, 1.72
Family Income (≥$75,000)				
<$20,000	2.08*	1.04, 4.15	1.91*	1.06, 3.44
$20,000-44,999	2.39**	1.27, 4.49	1.80*	1.10, 2.97
$45,000-74,999	2.23**	1.24, 4.03	1.38	0.80, 2.38
Family Structure (2 Biological Parents)				
Step-Family	1.17	0.78, 1.77	0.99	0.61, 1.65
1 Biological Parent	1.04	0.73, 1.49	0.90	0.64, 1.26
Other Situations	1.60	0.88, 2.93	1.02	0.54, 1.93

Note: Analysis weighted; NH= non-Hispanic; RR= Risk ratios; CI= Confidence intervals.
* p≤0.05, **p≤0.01, ***p≤0.001

5.4. Chapter Summary

The purpose of this chapter was to investigate the effects of adolescent sociodemographic factors on longitudinal patterns of obesity during the transition to adulthood separately for females and males. As hypothesized, bivariate distributions among females revealed significant differences in obesity status at Waves II and III, and longitudinal patterns of obesity by Wave I sociodemographic characteristics. Significant bivariate and multivariate findings of longitudinal

obesity patterns by Wave I sociodemographics were also found, mainly by age, race/ethnicity, and family income.

However, contrary to my hypotheses, no significant differences in obesity status or obesity patterns among males were found, except by family income. Despite these findings, bivariate and multivariate findings of longitudinal obesity patterns by Wave I sociodemographics among males revealed that age and lower income status at Wave I remained significantly and positively associated with becoming obese when accounting for family and individual characteristics.

Overall, the empirical evidence presented in this chapter support the first criteria of the mediation model (pathway c`), that the independent variables of Wave I sociodemographic characteristics significantly predict the outcome of longitudinal patterns of obesity. Such associations indicate that the transition between adolescence and young adulthood is a period of increased risk of development of obesity (Gordon-Larsen et al., 2004; Gordon-Larsen et al., 2010; Lee et al., 2009). This upward trend was found especially among older adolescents, non-Hispanic Black females, U.S.-born females, and adolescents of low SES, suggesting the critical nature of focusing on obesity prevention before adulthood and enhancing mechanisms of promoting healthy behaviors during the transition to adulthood.

The current results also support the life course approach of health development trajectories over time (Elder, 1998b), and Link and Phelan's (1995; 2010) theory of social conditions and fundamental causes of health differences. The adverse conditions of low SES, coupled with the developmental period of adolescence, contributes to higher risk of becoming and staying obese over time among females and males (Gordon-Larsen et al., 2004; Gordon-Larsen et al., 2010; Lee et al., 2009).

Chapter 6

AIM 3 RESULTS

6.1. Overview

Aim 3: To use Add Health to estimate the mediating effects of adolescent SLE on the relationship between sociodemographic characteristics and longitudinal patterns of obesity from adolescence to adulthood separately for females and males.

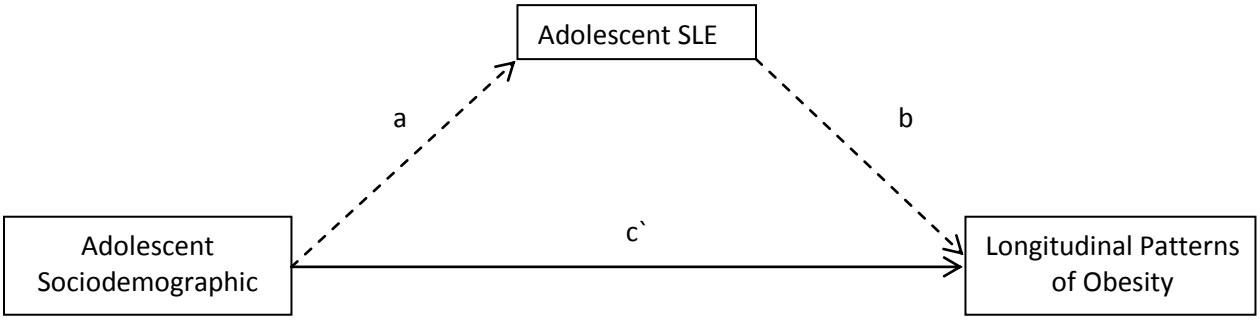
To my third aim, mediating models examining the influence of adolescent SLE on becoming and staying obese during the transition to adulthood separately for females and males were done. Using path analysis within MPlus, I determined the unadjusted and adjusted indirect effects of adolescent SLE on the relationship between Wave I sociodemographic characteristics and longitudinal patterns of obesity.

Adolescent SLE was defined by two distinct indices of stressful events according to whether events occurred *to* the adolescent or were performed *by* the adolescent (see section 3.3.4.2 in Chapter 3). Adolescent SLE reflect the amount of stress an adolescent is exposed to, which in turn contributes to fat accumulation and subsequent obesity. Individual control over an event may also contribute to differences in obesity patterns over time. I hypothesized that the greater the number of SLE done *to* adolescents, the higher the risk of becoming and staying obese among both males and females. Differences in obesity patterns were also expected among those who performed higher numbers of SLE during adolescence.

In order for adolescent SLE to act as a mediator between Wave I sociodemographic characteristics and longitudinal patterns of obesity from adolescence to adulthood, certain criteria must be met (see section 3.4.2.6 in Chapter 3). Each of three relationships within the mediation model must be significant. The diagram below depicts these relationships. First, the adolescent sociodemographic factor must significantly predict the two outcomes of interest: 1) becoming obese and 2) staying obese (pathway c`). Second, the sociodemographic factor must also predict

the mediator (pathway a). Last, the mediator must be significantly associated with the outcome (pathway b). If all three criteria were met, mediation analysis was done.

Figure 6.1. Mediation Model Criteria



This chapter is organized into four sections. The first section presents results of the relationship between the independent variables (Wave I sociodemographic characteristics) and the categorical mediator (adolescent SLE done *to* and performed *by*). Weighted distributions of SLE by each Wave I sociodemographic characteristic are presented, stratified by gender. Adjusted Wald tests were also employed to test for significance. The second section shows ordered logistic regression analyses to estimate the relationship between each Wave I sociodemographic characteristic and each measure of SLE. All models are gender stratified. Interpretation of odds ratios within ordered logistic regression is as follows: given a 1-unit increase in the independent variable (or a given independent category relative to the reference category), the odds of experiencing or performing high SLE versus combined fewer SLE is 'X' greater.

The third section provides details on the relationship between each SLE mediator (adolescent SLE done *to* and performed *by*) and the outcome (longitudinal patterns of obesity). Weighted distributions of obesity status and longitudinal patterns of obesity by SLE are

presented separately for females and males. Adjusted Wald tests were also employed to test for significance. Multivariate analysis using multinomial logistic regression to estimate the relationship between adolescent SLE and longitudinal patterns of obesity are shown, highlighting exponentiated coefficients to obtain estimates of the risk ratio (RR) of each obesity outcome as a function of adolescent SLE.

The fourth and final section shows the mediation analysis using the procedures available in MPlus that include path analysis, which decomposes the total effects into direct and indirect components, thereby calculating the indirect effect of each SLE measure on the relationship between Wave I sociodemographic characteristics and longitudinal patterns of obesity from adolescence to young adulthood. Given gender differences in obesity and experiences of SLE, all models are gender stratified.

6.2. Relationship between Wave I Sociodemographic Characteristics and Wave I Stressful Life Events

6.2.1. Distribution of Wave I SLE by Wave I Sociodemographic Characteristics

6.2.1.1. Distribution of SLE among Females

Tables 6.1 presents weighted descriptive sample statistics of adolescent SLE done *to* females by Wave I sociodemographic characteristics. There were significant associations for all of the sociodemographic characteristics and the number of SLE done *to* adolescent girls. Among adolescent females of all ages, 45.9% reported experiencing 0 SLE, 33.4% experienced 1 SLE, 12.0% experienced 2 SLE, and 8.7% experienced 3 or more SLE. There was a significant association between race/ethnicity and number of SLE done *to* adolescent girls. Although over three-quarters of girls across all race/ethnic categories reported experiencing 0 or 1 SLE, higher percentages of Black and Hispanic girls reported experiencing 3 or more. U.S.-born adolescent

girls reported a greater number of SLE done *to* them than foreign-born. Girls whose parent had lower education also reported a greater number of SLE, as did girls in families with lower incomes. Girls living in other situations reported substantially higher percentages of experiencing 3 or more SLE (21.6%) than girls living in two biological parent households (4.8%).

Table 6.1. Weighted Percent and Standard Errors of Adolescent SLE by Wave I Sociodemographic Characteristics, Females, Add Health Waves I-III (N=4,813)

	SLE Done *To* % (SE)			
Wave I Sociodemographics	0 SLE	1 SLE	2 SLE	3+ SLE
Individual Characteristics				
Age (12-19 Years)	45.9 (.009)	33.4 (.007)	12.0 (.007)	8.7 (.007)
Race/Ethnicity***				
NH White	46.1 (.014)	34.7 (.011)	12.2 (.009)	7.0 (.007)
NH Black	42.5 (.025)	30.3 (.018)	13.9 (.012)	13.3 (.019)
Hispanic	45.6 (.029)	30.4 (.024)	11.4 (.016)	12.6 (.017)
NH Asian	58.2 (.032)	30.8 (.049)	3.9 (.020)	7.1 (.024)
Nativity Status*				
U.S.-born	45.3 (.012)	33.4 (.009)	12.5 (.007)	8.8 (.008)
Foreign born	57.8 (.053)	33.1 (.047)	3.2 (.017)	6.0 (.019)
Parental Education Level**				
Less than High School	40.2 (.034)	32.3 (.026)	12.9 (.021)	15.6 (.017)
High School Grad	40.0 (.024)	34.4 (.023)	13.9 (.019)	11.8 (.020)
More than High School	47.7 (.013)	33.4 (.010)	11.6 (.007)	7.3 (.007)
Family Income***				
<$20,000	41.1 (.023)	32.5 (.021)	11.9 (.013)	14.4 (.015)
$20,000-44,999	46.5 (.015)	32.3 (.013)	12.4 (.011)	8.9 (.010)
$45,000-74,999	46.1 (.018)	35.6 (.016)	12.4 (.013)	5.9 (.008)
≥$75,000	51.1 (.027)	33.0 (.024)	10.4 (.014)	5.6 (.013)
Family Structure***				
2 Biological Parents	51.9 (.014)	33.1 (.011)	10.2 (.008)	4.8 (.006)
Step-Family	37.6 (.024)	37.0 (.030)	15.3 (.022)	10.1 (.018)
1 Biological Parent	37.9 (.020)	33.4 (.019)	14.1 (.014)	14.6 (.015)
Other Situations	32.8 (.010)	29.2 (.038)	16.4 (.030)	21.6 (.041)

Note: Design-based Wald test for bivariate analysis; NH= non-Hispanic.
* p≤0.05, **p≤0.01, ***p≤0.001.

Table 6.2 presents weighted descriptive sample statistics and adjusted Wald tests of adolescent SLE performed *by* females by Wave I sociodemographic characteristics. Among females of all ages, 82.9% reported performing 0 SLE, 13.6% performed 1 SLE, and 3.5% performed 2 or more SLE. There were significant associations between race/ethnicity, parental education, family income, and family structure and the number of SLE performed by females. In this case, nativity status was not significant. However, overall the vast majority of girls, regardless of sociodemographic characteristic, reported performing 0 SLE (ranging from 71.0% to 88.3%). However, some groups of girls did report performing 2 or more SLE. In particular, Black (4.5%) and Hispanic girls (5.8%), those whose parent had less than a high school education (6.4%), an income less than $20,000 (5.3%), and girls who lived in other family situations (9.0%).

Table 6.2. Weighted Percent and Standard Errors of Adolescent SLE by Wave I Sociodemographic Characteristics, Females, Add Health Waves I-III (N=4,813)

	SLE Performed *By* % (SE)		
Wave I Sociodemographics	0 SLE	1 SLE	2+ SLE
Individual Characteristics			
Age (12-19 Years)	82.9 (.009)	13.6 (.007)	3.5 (.003)
Race/Ethnicity*			
NH White	84.3 (.010)	12.9 (.009)	2.8 (.004)
NH Black	78.7 (.020)	16.8 (.018)	4.5 (.009)
Hispanic	79.2 (.025)	15.0 (.021)	5.8 (.013)
NH Asian	85.1 (.031)	11.9 (.028)	3.0 (.012)
Nativity Status			
U.S.-born	82.6 (.009)	13.9 (.007)	3.5 (.004)
Foreign born	86.9 (.024)	10.1 (.026)	3.0 (.012)
Family Background			
Parental Education Level**			
Less than High School	78.1 (.023)	15.5 (.019)	6.4 (.014)
High School Grad	79.7 (.026)	15.9 (.022)	4.4 (.012)
More than High School	84.1 (.009)	13.1 (.009)	2.8 (.003)
Family Income***			
<$20,000	77.4 (.019)	17.3 (.017)	5.3 (.010)
$20,000-44,999	81.9 (.045)	14.1 (.013)	4.0 (.005)
$45,000-74,999	85.4 (.014)	12.6 (.012)	2.0 (.005)
≥$75,000	88.3 (.017)	9.7 (.016)	2.0 (.009)
Family Structure***			
2 Biological Parents	86.8 (.009)	11.5 (.009)	1.7 (.003)
Step-Family	80.6 (.024)	14.9 (.023)	4.4 (.011)
1 Biological Parent	76.9 (.016)	17.0 (.013)	6.0 (.008)
Other Situations	71.0 (.038)	20.1 (.035)	9.0 (.023)

Note: Design-based Wald test for bivariate analysis; NH= non-Hispanic.
* $p \leq 0.05$, ** $p \leq 0.01$, *** $p \leq 0.001$.

6.2.1.2. Distribution of SLE among Males

Table 6.3 reports weighted distributions and adjusted Wald tests of SLE done *to* males by Wave I sociodemographic characteristics. There were significant associations for all the sociodemographic characteristics and the number of SLE done *to* adolescent boys. Among adolescent males of all ages, 37.6% reported experiencing 0 SLE, 30.5% experienced 1 SLE, 15.8% experienced 2 SLE, and 16.1% experienced 3 or more SLE. There was a significant association between race/ethnicity and number of SLE done *to* adolescent boys. While approximately one-third or more of White, Black, and Hispanic males experienced 0 SLE, higher percentages of Black and Hispanic males reported experiencing 3 or more. Asian boys reported the lowest percentage of 3 or more SLE (9.5%). U.S.-born adolescent males reported a greater number of SLE done *to* them than foreign born. Males whose parent had lower education also reported experiencing a greater number of SLE, as did boys in families with lower incomes. Adolescent males living in other situations reported substantially higher percentages of experiencing 3 or more SLE (29.2%) than boys living in two biological parent homes (11.6%).

Table 6.3. Weighted Percent and Standard Errors of Adolescent SLE by Wave I Sociodemographic Characteristics, Males, Add Health Waves I-III (N=4,498)

	SLE Done *To* % (SE)			
Wave I Sociodemographics	0 SLE	1 SLE	2 SLE	3+ SLE
Individual Characteristics				
Age (12 to 19 Years)	37.6 (.013)	30.5 (.010)	15.8 (.008)	16.1 (.009)
Race/Ethnicity***				
NH White	38.9 (.015)	31.6 (.011)	15.6 (.010)	13.9 (.009)
NH Black	31.3 (.028)	26.2 (.021)	19.5 (.019)	23.0 (.021)
Hispanic	30.3 (.028)	31.6 (.024)	15.4 (.017)	22.7 (.026)
NH Asian	58.8 (.028)	22.9 (.041)	8.8 (.026)	9.5 (.026)
Nativity Status**				
U.S.-born	36.8 (.013)	30.5 (.010)	16.3 (.008)	16.4 (.010)
Foreign born	52.0 (.064)	29.4 (.046)	7.8 (.022)	10.9 (.028)
Family Background				
Parental Education Level***				
Less than High School	29.0 (.027)	30.5 (.023)	18.8 (.022)	21.8 (.023)
High School Grad	32.0 (.036)	30.8 (.031)	13.9 (.019)	23.3 (.033)
More than High School	39.7 (.014)	30.4 (.010)	15.6 (.009)	14.3 (.009)
Family Income***				
<$20,000	30.3 (.022)	30.2 (.021)	17.5 (.017)	22.0 (.021)
$20,000-44,999	36.6 (.019)	29.6 (.016)	15.5 (.012)	18.4 (.014)
$45,000-74,999	41.5 (.019)	32.8 (.016)	13.6 (.013)	12.1 (.011)
≥$75,000	42.9 (.028)	28.1 (.024)	19.7 (.019)	9.4 (.016)
Family Structure***				
2 Biological Parents	43.8 (.016)	30.3 (.012)	14.4 (.009)	11.6 (.008)
Step-Family	32.4 (.029)	33.1 (.033)	16.8 (.024)	17.7 (.023)
1 Biological Parent	28.4 (.018)	29.4 (.019)	18.7 (.015)	23.6 (.020)
Other Situations	22.6 (.043)	32.9 (.049)	15.4 (.036)	29.2 (.043)

Note: Design-based Wald test for bivariate analysis; NH= non-Hispanic.
* p≤0.05, **p≤0.01, ***p≤0.001.

Table 6.4 reports weighted distributions and adjusted Wald tests of SLE performed *by* males by Wave I sociodemographic characteristics. Among adolescent males of all ages, 70.6% reported performing 0 SLE, 23.2 performed 1 SLE, and 6.2% performed 2 or more SLE. There were significant associations for all the sociodemographic characteristics and the number of SLE performed *by* adolescent males. Overall, the majority of adolescent boys, regardless of sociodemographic characteristics, reported performing 0 SLE (ranging from 59.3% to 79.1%). However, some groups of males did report performing 2 or more SLE. In particular, Black (12.6%) and Hispanic boys (8.1%), those who were U.S.-born (6.4%), whose parent had a high school education or GED (10.1%), an income less than $20,000 (11.4%), and adolescent boys who lived in other family situations (14.6%).

Table 6.4. Weighted Percent and Standard Errors of Adolescent SLE by Wave I Sociodemographic Characteristics, Males, Add Health Waves I-III (N=4,498)

	SLE Performed *By* % (SE)		
Wave I Sociodemographics	0 SLE	1 SLE	2+ SLE
Individual Characteristics			
Age (12-19 Years)	70.6 (.011)	23.2 (.009)	6.2 (.062)
Race/Ethnicity***			
NH White	72.4 (.013)	22.9 (.012)	4.7 (.006)
NH Black	61.7 (.022)	25.7 (.021)	12.6 (.017)
Hispanic	68.9 (.021)	23.0 (.019)	8.1 (.016)
NH Asian	77.0 (.036)	20.9 (.031)	2.1 (.010)
Nativity Status**			
U.S.-born	70.2 (.012)	23.5 (.010)	6.4 (.006)
Foreign born	79.1 (.027)	18.6 (.025)	2.3 (.010)
Family Background			
Parental Education Level***			
Less than High School	67.1 (.025)	23.7 (.022)	9.2 (.017)
High School Grad	59.6 (.039)	30.4 (.037)	10.1 (.020)
More than High School	72.7 (.011)	22.2 (.009)	5.2 (.005)
Family Income***			
<$20,000	62.3 (.027)	26.3 (.024)	11.4 (.014)
$20,000-44,999	67.8 (.017)	25.5 (.016)	6.7 (.009)
$45,000-74,999	77.3 (.015)	19.3 (.014)	3.4 (.007)
≥$75,000	76.6 (.025)	20.5 (.023)	2.9 (.009)
Family Structure***			
2 Biological Parents	75.8 (.011)	20.9 (.010)	3.3 (.005)
Step-Family	69.1 (.030)	26.6 (.030)	4.3 (.013)
1 Biological Parent	61.5 (.019)	26.6 (.015)	11.9 (.013)
Other Situations	59.3 (.047)	26.1 (.043)	14.6 (.033)

Note: Design-based Wald test for bivariate analysis; NH= non-Hispanic.
* p≤0.05, **p≤0.01, ***p≤0.001.

6.2.2. Ordered Logistic Regression Analysis of Adolescent SLE by Wave I Sociodemographic Characteristics

Tables 6.5-6.10 show bivariate and multivariate ordered logistic regression results of Wave I adolescent SLE by Wave I sociodemographic characteristics separately for females and males. Proportional odds ratios (OR) are presented, based on the assumption that the relationship between each pair of outcome groups is the same. Thus, the relationship between the highest SLE category versus all lower SLE categories are the same as those that describe the relationship between the next lowest SLE category and all higher SLE categories (see section 3.4.2.4).

6.2.2.1. Regression Results among Females

Unadjusted results, individual characteristics, Wave I. Table 6.5 presents the unadjusted odds ratios and 95% confidence intervals of age, race/ethnicity, and nativity status on adolescent SLE done *to* and performed *by* females. Older adolescents were significantly more likely to experience greater numbers of SLE done *to* them (panel a). Compared to White females, Black females were significantly more likely to have a greater number of SLE done to them (panel b). Compared to U.S.-born girls, those who were foreign-born were significantly less likely to have a greater number of SLE done to them (panel c). The results for SLE performed *by* girls were somewhat different. Age effects on SLE performed *by* females were similar to those on SLE done *to* females. Compared to White adolescent females, both Black and Hispanic females were significantly more likely to report they had performed a higher number of SLE. Nativity status was not significant.

Table 6.5. Bivariate Ordered Logistic Regression of Adolescent SLE by Wave I Individual Characteristics, Females, Add Health Waves I-III (N=4,813)

Wave I Individual Characteristics (reference)	SLE Done *to*		SLE Performed *by*	
	OR	95% CI	OR	95% CI
(a)				
Age (Years)	1.27***	1.21, 1.33	1.08*	1.01, 1.15
(b)				
Race/Ethnicity (NH White)				
NH Black	1.32*	1.03, 1.67	1.45**	1.12, 1.87
Hispanic	1.14	0.90, 1.45	1.43*	1.03, 1.98
NH Asian	0.62	0.37, 1.02	0.94	0.56, 1.56
(c)				
Nativity Status (U.S.-born)				
Foreign born	0.57***	0.38, 0.86	0.72	0.47, 1.10

Note: Analysis weighted; NH= non-Hispanic; OR = Odds ratio; CI = Confidence intervals.
* p≤0.05, **p≤0.01, ***p≤0.001.

Unadjusted results, family background characteristics, Wave I. Table 6.6 presents the unadjusted odds ratios and 95% confidence intervals of parental education, family income, and family structure on adolescent SLE done *to* and performed *by* females. Compared to girls whose parent had the highest level of education, both categories of lower parental education were significantly associated with a greater number of SLE done *to* adolescent females (panel a). Compared to girls in families with the highest income, those in the lowest two categories of income were more likely to have a greater number of SLE done *to* them (panel b). Compared to girls living with both biological parents, all other family structure situations were significantly associated with a greater number of SLE done *to* them (panel c). This was especially true for girls living in other family situations (OR=2.88, p<.001).

The results for SLE performed *by* adolescent females were similar. Females whose parent had less than a high school education were significantly associated with performing a greater

number of SLE compared to girls whose parent had the highest level of education. Compared to girls in families with the highest income, those in the lowest two categories of income were also more likely to perform greater numbers of SLE. Females living in non-two-biological parent homes were significantly associated with performing a greater number of SLE compared to girls living with both biological parents.

Table 6.6. Bivariate Ordered Logistic Regression of Adolescent SLE by Wave I Family Background Characteristics, Females, Add Health Waves I-III (N=4,813)

	SLE Done *to*		SLE Performed *by*	
Wave I Family Background Characteristics (reference)	OR	95% CI	OR	95% CI
(a)				
Parental Education Level (More than High School)				
Less than High School	1.50**	1.12, 1.99	1.52**	1.12, 2.08
High School Grad/GED	1.42***	1.16, 1.74	1.35	0.98, 1.86
(b)				
Family Income (≥$75,000)				
<$20,000	1.66***	1.28, 2.15	2.22***	1.51, 3.26
$20,000-44,999	1.26*	1.01, 1.58	1.68**	1.20, 2.34
$45,000-74,999	1.19	0.97, 1.48	1.28	0.87, 1.88
(c)				
Family Structure (2 Biological Parents)				
Step-Family	1.81***	1.45, 2.25	1.60**	1.14, 2.24
1 Biological Parent	1.98***	1.66, 2.38	2.01***	1.62, 2.50
Other Situations	2.88***	1.99, 4.15	2.80***	1.90, 4.13

Note: Analysis weighted; NH= non-Hispanic; OR = Odds ratios; CI = Confidence intervals.
* p≤0.05, **p≤0.01, ***p≤0.001.

Adjusted full model. Table 6.7 presents the combined results for individual and family background characteristics on adolescent SLE done *to* and performed *by* females. Adjusted odds ratios (AOR) and 95% confidence intervals are shown. Age at Wave I remained positively and

significantly associated with experiencing greater numbers of SLE done *to* females. Race effects were no longer significantly associated with SLE done *to* females when family background characteristics were also considered. Compared to U.S.-born, foreign born were significantly less likely to have SLE done *to* them. Parental education was no longer significant when individual characteristics were taken into account. The effects of lower income were no longer significant. However, those with an income of $45,000 to $74,999 were more likely to have a greater number of SLE done *to* them than females with the highest income. Females living in non-two-biological parent homes remained significantly associated with experiencing greater numbers of SLE done *to* them.

The results for SLE performed *by* adolescent females were a bit more consistent across the bivariate and multivariate models than those for SLE done *to* females. Age effects were no longer significant when family background characteristics were also considered. Compared to White females, Hispanics were significantly more likely to perform higher numbers of SLE during adolescence. Being Black was no longer significantly associated with performing higher numbers of SLE once family background characteristics were taken into account. Compared to U.S.-born, foreign born were significantly less likely to perform SLE. However, the effects of parent education were no longer significant. Family income remained significant, with those in the lowest income categories compared to the highest category being more likely to perform greater numbers of SLE. Additionally, family structure effects remained significant. Females living in non-two-biological parent homes were significantly more likely to perform greater numbers of SLE.

Table 6.7. Multivariate Ordered Logistic Regression of Adolescent SLE by Wave I Sociodemographic Characteristics, Females, Add Health Waves I-III (N=4,813)

Wave I Sociodemographic Characteristics (reference)	SLE Done *to*		SLE Performed *by*	
	AOR	95% CI	AOR	95% CI
Individual Characteristics				
Age (Years)	1.26***	1.20, 1.32	1.06	0.99, 1.14
Race/Ethnicity (NH White)				
NH Black	1.00	0.78, 1.29	1.09	0.84, 1.42
Hispanic	1.18	0.89, 1.57	1.49*	1.01, 2.19
NH Asian	0.88	0.53, 1.47	1.26	0.75, 2.12
Nativity Status (U.S.-born)				
Foreign born	0.46**	0.28, 0.76	0.52*	0.32, 0.86
Family Background				
Parental Education Level (More than High School)				
Less than High School	1.16	0.84, 1.60	1.00	0.68, 1.48
High School Grad/GED	1.09	0.85, 1.39	0.95	0.68, 1.32
Family Income (≥$75,000)				
<$20,000	1.20	0.89, 1.63	1.58*	1.02, 2.42
$20,000-44,999	1.10	0.88, 1.39	1.42*	1.01, 2.01
$45,000-74,999	1.24*	1.00, 1.53	1.27	0.86, 1.87
Family Structure (2 Biological Parents)				
Step-Family	1.71***	1.36, 2.13	1.60**	1.12, 2.28
1 Biological Parent	1.82***	1.49, 2.23	1.80***	1.39, 2.31
Other Situations	2.32***	1.63, 3.30	2.35***	1.59, 3.50

Note: Analysis weighted; NH= non-Hispanic; AOR = Adjusted odds ratios; CI = Confidence intervals.
* p≤0.05, **p≤0.01, ***p≤0.001.

6.2.2.2. Regression Results among Males

Unadjusted results, individual characteristics, Wave I. Table 6.8 presents the unadjusted odds ratios and 95% confidence intervals of age, race/ethnicity, and nativity status on adolescent SLE done *to* and performed by males. Older adolescents were significantly more likely to experience greater numbers of SLE done *to* them (panel a). Compared to White males, Black and Hispanic males were significantly more likely, and Asian males significantly less likely, to have

a greater number of SLE done to them (panel b). Compared to U.S.-born males, those who were foreign-born were significantly less likely to have a greater number of SLE done to them (panel c). The results for SLE performed *by* boys were slightly different. Age effects on SLE performed *by* males were slightly less significant than those on SLE done *to* males. Compared to White adolescent males, only Black males were significantly more likely to report they had performed a higher number of SLE. Nativity status effects were similar to those on SLE done *to* adolescent males.

Table 6.8. Bivariate Ordered Logistic Regression of Adolescent SLE by Wave I Individual Characteristics, Males, Add Health Waves I-III (N=4,498)

	SLE Done *to*		SLE Performed *by*	
Wave I Individual Characteristics (reference)	OR	95% CI	OR	95% CI
(a)				
Age (Years)	1.23***	1.17, 1.30	1.07*	1.01, 1.13
(b)				
Race/Ethnicity (NH White)				
NH Black	1.61***	1.25, 2.07	1.75***	1.40, 2.18
Hispanic	1.52***	1.20, 1.94	1.23	0.97, 1.55
NH Asian	0.48**	0.30, 0.77	0.77	0.51, 1.17
(c)				
Nativity Status (U.S.-born)				
Foreign born	0.53**	0.34, 0.83	0.61**	0.44, 0.85

Note: Analysis weighted; NH= non-Hispanic; OR = Odds ratios; CI = Confidence intervals.
* $p \leq 0.05$, ** $p \leq 0.01$, *** $p \leq 0.001$.

Unadjusted results, family background characteristics, Wave I. Table 6.9 presents the unadjusted odds ratios and 95% confidence intervals of parental education, family income, and family structure on adolescent SLE done *to* and performed *by* males. Compared to boys whose parent had the highest level of education, both categories of lower parental education were significantly associated with a greater number of SLE done *to* adolescent males (panel a).

Compared to males in families with the highest income, those in the lowest two categories of income were more likely to have a greater number of SLE done *to* them (panel b). Compared to boys living with both biological parents, all other family structure situations were significantly associated with a greater number of SLE done *to* them (panel c). This was especially true for boys living in other family situations (OR=2.60, p<.001).

The results for SLE performed *by* adolescent males were similar. Males whose parent had a high school degree or lower level of education were significantly associated with performing a greater number of SLE compared to boys whose parent had the highest level of education. Compared to males in families with the highest income, those in the lowest two categories of income were also more likely to perform greater numbers of SLE. Males living in non-two-biological parent homes were also significantly associated with performing a greater number of SLE compared to those living with both biological parents.

Table 6.9. Bivariate Ordered Logistic Regression of Adolescent SLE by Wave I Family Background Characteristics, Males, Add Health Waves I-III (N=4,498)

	SLE Done *to*		SLE Performed *by*	
Wave I Family Background Characteristics (reference)	OR	95% CI	OR	95% CI
(a)				
Parental Education Level (More than High School)				
Less than High School	1.62***	1.27, 2.06	1.35*	1.06, 1.71
High School Grad/GED	1.47*	1.07, 2.03	1.83***	1.31, 2.54
(b)				
Family Income (≥$75,000)				
<$20,000	1.77***	1.36, 2.31	2.12***	1.52, 2.96
$20,000-44,999	1.36**	1.08, 1.71	1.58**	1.14, 2.19
$45,000-74,999	1.01	0.82, 1.23	0.97	0.74, 1.27
(c)				
Family Structure (2 Biological Parents)				
Step-Family	1.57***	1.25, 1.98	1.38*	1.05, 1.81
1 Biological Parent	2.07***	1.71, 2.52	2.10***	1.74, 2.55
Other Situations	2.60***	1.79, 3.77	2.38***	1.57, 3.60

Note: Analysis weighted; NH= non-Hispanic; OR = Odds ratios; CI = Confidence intervals.
* p≤0.05, **p≤0.01, ***p≤0.001.

Adjusted full model. Table 6.10 presents the combined results for individual and family background characteristics on adolescent SLE done *to* and performed *by* males. Adjusted odds ratios and 95% confidence intervals are shown. Age at Wave I remained positively and significantly associated with experiencing greater numbers of SLE done *to* males. Hispanic males compared to White males remained significant more likely to experience greater numbers of SLE done *to* them. Race effects of Black and Asian males were no longer significantly associated with SLE done *to* males when family background characteristics were also considered. Compared to U.S.-born, foreign born were significantly less likely to have SLE done *to* them. Parental education and family income were no longer significant when individual

characteristics were taken into account. Males living in non-two-biological parent homes remained significantly associated with experiencing greater numbers of SLE done *to* them.

The combined results for SLE performed *by* adolescent males revealed somewhat similar results. Age effects were no longer significant when family background characteristics were also considered. Compared to White males, Blacks remained significantly more likely to perform higher numbers of SLE during adolescence. Compared to U.S.-born, foreign born were significantly less likely to perform SLE. However, the effects of parent education were no longer significant. Males in the lowest income category compared to the highest category remained significantly more likely to perform greater numbers of SLE. Other income categories among males were no longer significant when individual characteristics were accounted for. Additionally, the effects of living in a single biological parent or other family situations, compared to two biological parent homes, remained significantly associated with performing greater numbers of SLE.

Table 6.10. Multivariate Ordered Logistic Regression of Adolescent SLE by Wave I Sociodemographic Characteristics, Males, Add Health Waves I-III (N=4,498)

	SLE Done *to*		SLE Performed *by*	
Wave I Sociodemographics (reference)	AOR	95% CI	AOR	95% CI
Individual Characteristics				
Age (Years)	1.23***	1.17, 1.29	1.05	1.00, 1.11
Race/Ethnicity (NH White)				
NH Black	1.15	0.91, 1.46	1.27*	1.02, 1.58
Hispanic	1.53**	1.15, 2.04	1.17	0.86, 1.60
NH Asian	0.68	0.45, 1.03	1.03	0.67, 1.59
Nativity Status (U.S.-born)				
Foreign born	0.39***	0.25, 0.63	0.53***	0.36, 0.77
Family Background				
Parental Education Level (More than High School)				
Less than High School	1.15	0.84, 1.57	1.02	0.75, 1.37
High School Grad/GED	1.02	0.74, 1.41	1.31	0.94, 1.83
Family Income (≥$75,000)				
<$20,000	1.26	0.96, 1.65	1.50*	1.06, 2.11
$20,000-44,999	1.17	0.93, 1.46	1.35	0.98, 1.85
$45,000-74,999	1.02	0.83, 1.24	0.96	0.73, 1.25
Family Structure (2 Biological Parents)				
Step-Family	1.47***	1.16, 1.86	1.21	0.93, 1.58
1 Biological Parent	1.76***	1.45, 2.13	1.63***	1.36, 1.96
Other Situations	1.82**	1.24, 2.69	1.78**	1.15, 2.75

Note: Analysis weighted; NH= non-Hispanic; AOR = Adjusted odds ratios; CI = Confidence Intervals.
* p≤0.05, **p≤0.01, ***p≤0.001.

6.3. Distribution of Obesity Status and Longitudinal Patterns of Obesity by Wave I SLE

Tables 6.11 presents bivariate relationships for obesity at Wave II, III, and longitudinal obesity patterns by Wave I SLE for females. Column 1 shows the patterns for Wave II. There were significant differences in obesity status at Wave II, with the highest percentages reported among females who experienced 3 or more SLE done *to* them (17.1%). There was no relationship between SLE performed by females and obesity status at Wave II. By Wave III,

percentages of obesity were higher among all categories of SLE done *to* females, with the highest among those who experienced 3 or more SLE (34.3%). SLE performed by females were not significantly associated with obesity in Wave III.

The last three columns of Table 6.11 show the longitudinal patterns of obesity from Wave II to Wave III. Females who experienced 3 or more SLE done *to* them had the highest rates of becoming and staying obese between the two waves (20.3% and 14.0%, respectively). Females who performed 2 or more SLE had the highest percentage of becoming obese (21.4%); however, those who performed 1 or more SLE had a higher percentage of staying obese (11.6%).

Table 6.11. Weighted Percents and Standard Errors of Obesity Status and Patterns of Obesity by Wave I SLE, Females, Add Health Waves I-III (N=4,813)

	Cross-Sectional Obesity[a] % (SE)		Longitudinal Obesity Patterns[b] % (SE)		
Wave I SLE Characteristic	Wave II	Wave III	Become Obese	Stay Obese	Stay Non/Reduce Obese
SLE Done *To*					
0 SLE	10.8 (.010)*	22.2 (.013)***	12.6 (.009)***	9.6 (.010)	77.8 (.013)
1 SLE	9.7 (.011)	18.9 (.015)	10.7 (.011)	8.2 (.009)	81.1 (.015)
2 SLE	11.5 (.115)	23.7 (.022)	13.0 (.020)	10.8 (.016)	76.3 (.022)
3+ SLE	17.1 (.025)	34.3 (.033)	20.3 (.030)	14.0 (.023)	65.7 (.657)
SLE Performed *By*					
0 SLE	10.9 (.008)	21.5 (.012)	12.1 (.007)*	9.5 (.008)	78.5 (.012)
1 SLE	12.9 (.017)	25.9 (.026)	14.3 (.019)	11.6 (.017)	74.1 (.026)
2+ SLE	8.2 (.024)	27.4 (.048)	21.4 (.045)	6.0 (.021)	72.7 (.048)

Note: Design-based Wald test for bivariate analysis; NH= non-Hispanic.
* $p \leq 0.05$, ** $p \leq 0.01$, *** $p \leq 0.001$.
[a] Wald test conducted separately for Wave II and Wave III.
[b] Wald test compared differences across the 3 categories of longitudinal obesity outcome.

Tables 6.12 presents bivariate relationships for obesity at Wave II, III, and longitudinal obesity patterns by Wave I SLE for males. There were no significant differences in obesity status at Wave II or Wave III by adolescent SLE done *to* or performed *by* males. Longitudinal patterns of obesity from Wave II to Wave III by SLE revealed that males who experienced 3 or more SLE done *to* them had the highest percentage of becoming obese (14.2%), while those who experienced 0 SLE and 1 SLE had the highest percentages of staying obese (11.5% and 11.1%, respectively). There was no significant association between SLE performed *by* males and longitudinal patterns of obesity.

Table 6.12. Weighted Percents and Standard Errors of Obesity Status and Patterns of Obesity by Wave I SLE, Males, Add Health Waves I-III (N=4,498)

	Cross-Sectional Obesity[a] % (SE)		Longitudinal Obesity Patterns[b] % (SE)		
Wave I SLE	Wave II	Wave III	Become Obese	Stay Obese	Stay Non/Reduce Obese
SLE Done *To*					
0 SLE	15.2 (.014)	18.1 (.014)	6.6 (.007)**	11.5 (.013)	81.9 (.014)
1 SLE	12.3 (.013)	19.9 (.015)	10.6 (.012)	9.3 (.012)	80.1 (.015)
2 SLE	14.1 (.019)	19.4 (.021)	8.3 (.014)	11.1 (.017)	80.6 (.021)
3+ SLE	12.3 (.018)	24.4 (.022)	14.2 (.018)	10.2 (.018)	75.6 (.022)
SLE Performed *By*					
0 SLE	13.6 (.009)	19.7 (.010)	8.9 (.007)	10.8 (.008)	80.3 (.010)
1 SLE	14.2 (.017)	19.8 (.018)	9.6 (.012)	10.3 (.016)	80.2 (.018)
2+ SLE	11.6 (.023)	22.0 (.036)	12.5 (.029)	9.4 (.021)	78.1 (.036)

Note: Design-based Wald test for bivariate analysis; NH= non-Hispanic.
* $p \leq 0.05$, ** $p \leq 0.01$, *** $p \leq 0.001$.
[a] Wald test conducted separately for Wave II and Wave III.
[b] Wald test compared differences across the 3 categories of longitudinal obesity outcome.

6.4. Multinomial Logistic Regression Effects of Wave I SLE on Longitudinal Patterns of Obesity by Gender

Tables 6.13-6.14 show separate bivariate multinomial logistic regression models of Wave I SLE on longitudinal obesity patterns separately for females and males. Risk ratios and their 95% confidence intervals are presented. Table 6.13 shows that compared to females who experienced 0 SLE done *to* them, those who experienced 3 or more SLE were significantly more likely to become obese relative to the reference group of remaining non-obese or reduce obesity (panel a). Similarly, females who performed 2 or more SLE, compared to those who performed 0 SLE, were significantly more likely to become obese from Wave II to Wave III (panel b). The effects of experiencing 3 or more SLE done *to* females on staying obese were similar to becoming obese, although slightly lower. There were no significant effects of SLE performed *by* females on staying obese.

Table 6.13. Bivariate Multinomial Logistic Regression of Longitudinal Obesity Patterns by Wave I SLE, Females, Add Health Waves I-III (N=4,813)

	Become Obese		Stay Obese	
Wave I SLE (reference)	RR	95% CI	RR	95% CI
(a)				
SLE *to* (0 SLE)				
1 SLE	0.81	0.63, 1.04	0.82	0.62, 1.10
2 SLE	1.05	0.72, 1.53	1.15	0.81, 1.63
3+ SLE	1.91**	1.27, 2.86	1.73*	1.13, 2.63
(b)				
SLE *by* (0 SLE)				
1 SLE	1.26	0.90, 1.75	1.29	0.87, 1.92
2+ SLE	1.91*	1.12, 3.27	0.68	0.32, 1.46

Note: Analysis weighted; NH= non-Hispanic; RR = Risk ratios; CI = Confidence intervals.
* $p \leq 0.05$, ** $p \leq 0.01$, *** $p \leq 0.001$.

Table 6.14 presents separate bivariate multinomial logistic regression results of Wave I SLE on longitudinal obesity patterns among males. Compared to males who experienced 0 SLE done *to* them, those who experienced 1 SLE and 3 or more SLE were significantly more likely to become obese relative to the reference group of stay non-obese or reduce obesity (panel a). SLE performed by males were not significantly associated with becoming obese. The effects of Wave I SLE done *to* or performed *by* males on staying obese were not significant.

Table 6.14. Bivariate Multinomial Logistic Regression of Longitudinal Obesity Patterns by Wave I SLE, Males, Add Health Waves I-III (N=4,498)

	Become Obese		Stay Obese	
Wave I SLE (reference)	RR	95% CI	RR	95% CI
(a)				
SLE *to* (0 SLE)				
1 SLE	1.65**	1.21, 2.26	0.83	0.58, 1.19
2 SLE	1.28	0.85, 1.94	0.98	0.64, 1.50
3+ SLE	2.35***	1.61, 3.43	0.96	0.61, 1.52
(b)				
SLE *by* (0 SLE)				
1 SLE	1.07	0.77, 1.49	0.95	0.68, 1.33
2+ SLE	1.45	0.84, 2.50	0.90	0.53, 1.52

Note: Analysis weighted; NH= non-Hispanic; RR = Risk ratios; CI = Confidence intervals.
* $p \leq 0.05$, ** $p \leq 0.01$, *** $p \leq 0.001$.

6.5. Criteria for Viable Mediation

As discussed in section 3.4.2.6, three criteria must be met for a mediation model to be viable. Table 6.15 shows unadjusted viable mediation models for becoming and staying obese for both females and males. The first two columns designate SLE done *to* and performed *by* the adolescent as the mediator on becoming obese; the last two columns indicate SLE done *to* and performed *by* the adolescent as the mediator on staying obese. A viable mediation model is

represented by a female or male symbol. For example, the first row of Table 6.15 shows viable unadjusted mediation models whereby SLE done *to* adolescents possibly explains the relationship between age and becoming obese for both females and males. Of 52 possible mediation models for females, 17 viable models were found: 7 of SLE done *to* as a mediator on becoming obese, 5 of SLE performed *by* as a mediator on becoming obese, and 5 of SLE done *to* as a mediator on staying obese. Of 52 possible mediation models for males, only 4 of SLE done *to* as a mediator on becoming obese were found.

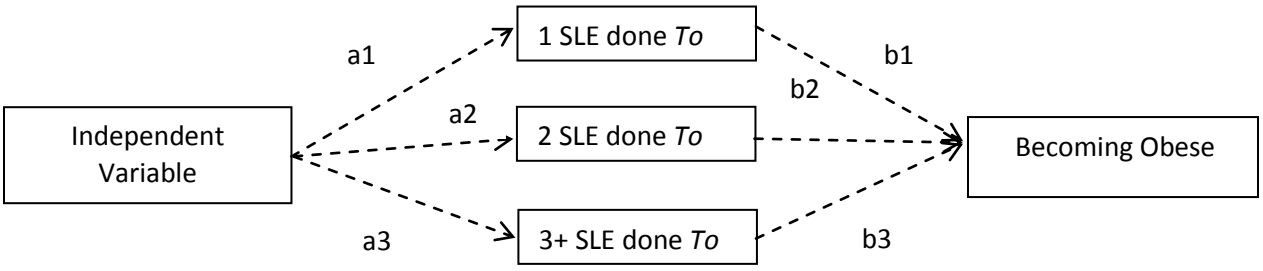
Table 6.15. Viable Unadjusted Mediation Models of SLE Done *To* and Performed *By* on Becoming and Staying Obese, Add Health Waves I-III (N=9,311)

	Become Obese		Stay Obese	
Model Variables (reference)	SLE *to*	SLE *by*	SLE *to*	SLE *by*
Age (Years)	♀♂		♀	
Race/Ethnicity (NH White)				
NH Black	♀	♀	♀	
Hispanic				
NH Asian				
Nativity Status (U.S.-born)				
Foreign born	♀		♀	
Parental Education Level (More than High School)				
Less than High School	♀	♀	♀	
High School Grad/GED				
Family Income (≥$75,000)				
<$20,000	♀ ♂	♀	♀	
$20,000-44,999	♀ ♂	♀		
$45,000-74,999				
Family Structure (2 Biological Parents)				
Step-Family				
1 Biological Parent	♀	♀		
Other Situations	♂			

Note: ♀ represents viable mediation for females; ♂ represents viable mediation for males; NH= non-Hispanic.

As discussed in Chapter 2, traditional mediation is done with a continuous mediator. However, in the current dissertation, the mediator is a categorical variable (categories of SLE done *to* and performed *by* the adolescent). Therefore, determining the indirect effect is a bit more complex. The illustration below depicts the complex nature of the indirect effect among a categorical mediator (Figure 6.2). As the mediator of SLE done *to* the adolescent has three categories (excluding the reference of 0 SLE), there are three pathways from the independent variable to each category of SLE, and three pathways from SLE to the outcome. Therefore, an indirect effect can be determined based on only a single category of SLE, or a total indirect effect through all categories of SLE greater than 0 SLE. For example, the indirect effect of the independent variable through 3 or more SLE is equal to (a3*b3). But that of the independent variable through all three SLE categories greater than 0 SLE is the sum of each individual indirect pathway: (a1*b1)+(a2*b2)+(a3*b3). The following mediation results section presents individual indirect and total indirect effects for viable mediation models presented in Table 6.15.

Figure 6.2. Complex Nature of Categorical Mediation



6.6. Mediation Models of Wave I SLE on Becoming Obese

Using the procedures available in MPlus that include path analysis, which decomposes the total effects into direct and indirect components, multinomial regression was done to examine the potential role of SLE done *to* and performed *by* the adolescent as a mediator between Wave I sociodemographic characteristics and becoming obese. Viable unadjusted and adjusted mediation models were analyzed separately for females and males.

6.6.1. Mediator: Stressful Life Events Done To Adolescent Females and Males

Table 6.16 shows mediation results of Wave I age through SLE done *to* adolescents on becoming obese over time among females and males. Age and experiencing 3 or more SLE during adolescence, compared to experiencing 0 SLE, were significantly associated with becoming obese among females. Among males, age and experiencing 1 SLE and 3 or more SLE were significantly associated with becoming obese.

Table 6.16. Multinomial Regression Model of Becoming Obese by Age and SLE Done *To* Adolescents, Add Health Waves I-III

	Females (N=4,813)		Males (N=4,498)	
Wave I Characteristics (reference)	b	SE	b	SE
Age (Years)	0.097*	0.043	0.134***	0.039
SLE done *to* (0 SLE)				
1 SLE	-0.240	0.127	0.465**	0.159
2 SLE	-0.023	0.187	0.182	0.207
3+ SLE	0.564***	0.198	0.756***	0.039

Note: Analysis Weighted.
* p≤0.05, **p≤0.01, ***p≤0.001

Table 6.17 presents the decomposition of mediated effects for females and males, including each indirect pathway through varying degrees of SLE (a*b), the total indirect pathway (Σab), the direct effect (c`), and the total effect (c`+ Σab). Among females, the significant indirect effect of age through 3 or more SLE on becoming obese was found. The proportion of the total effect of age mediated by 3 or more SLE among females was 65.6% (0.160/0.244). The total indirect effect of age through all SLE greater than 0 SLE done *to* females on becoming obese was not significant. Among males, significant indirect effects of age through 3 or more SLE and all SLE greater than 0 SLE on becoming obese were found. The proportion of the total effect of age mediated by 3 or more SLE among males was 53.9% (0.179/0.332), and that by all SLE greater than 0 SLE was 59.6% (0.198/0.332).

Table 6.17. Decomposition of Mediated SLE Effects of Wave I Age on Becoming Obese, Add Health Wave I-III

	Females (N=4,813)		Males (N=4,498)	
Wave I Characteristics (reference)	b	SE	b	SE
Age				
1 SLE (a1*b1)	-0.008	0.008	0.000	0.012
2 SLE (a2*b2)	-0.005	0.040	0.019	0.023
3+ SLE (a3*b3)	0.160**	0.057	0.179***	0.051
Total Indirect Effect (Σab)	0.147	0.076	0.198**	0.063
Direct Effect (c`)	0.097*	0.043	0.134***	0.039
Total Effect (c`+ Σab)	0.244**	0.093	0.332***	0.075

Note: Analysis weighted.
* p≤0.05, **p≤0.01, ***p≤0.001

6.6.1.1. Summary of Viable Unadjusted and Adjusted Mediation of SLE Done To Females on Becoming Obese

Remaining mediation and decomposition tables similar to those presented above for viable individual Wave I sociodemographic characteristics are found in the Appendix (Tables A1-A32). Below is a substantive summary of those results.

Unadjusted Results. Table 6.18 presents a summary of the 7 viable unadjusted mediation models of 3 or more SLE and all SLE greater than 0 SLE done t*o* females on the relationship between select Wave I sociodemographics and becoming obese. As discussed above, the indirect effects of 3 or more SLE done *to* females on age and becoming obese were significant. For Black females, in comparison to Whites, the proportion of the total effect mediated by 3 or more SLE was 10.9%. No significant indirect effects of 3 or more SLE were found between nativity status and becoming obese over time among females. However, significant mediation was found between less than a high school educated parent and becoming obese over time. For these females, in comparison to those whose parent had more than a high school degree, the proportion of the total effect mediated by 3 or more SLE was 46.0%.

Among females living in a home with the lowest income, in comparison to those with the highest income, the proportion of the total effect mediated by 3 or more SLE was 34.5%. No significant mediation effects for females living with a family income of $20,000 to $44,999 were found. However, SLE done *to* females significantly mediated the relationship between living in a single biological parent home and becoming obese. Among these females, compared to those in two biological parent homes, 3 or more SLE done *to* them significantly mediated 63.4% of the total effect.

Mediation results of all SLE greater than 0 SLE done *to* females were slightly different to those of 3 or more SLE. As discussed earlier, the total indirect effect of age through all SLE greater than 0 SLE done *to* females on becoming obese was not significant. For Black females, in comparison to Whites, the proportion of the total effect mediated by all SLE greater than 0 SLE was 46.2%, over 30% higher than that mediated by 3 or more SLE alone. No significant indirect effects of all SLE greater than 0 SLE were found between nativity status and becoming obese over time. Unadjusted mediation effects of all SLE greater than 0 SLE on family background characteristics and becoming obese were nearly identical to those of 3 or more SLE, with the percent of the total effect mediated remaining relatively equal to that of 3 or more SLE (47.4% and 46.0%, respectively); suggesting that the vast majority of the effect of individual family background characteristics is mediated through 3 or more SLE done *to* females.

Adjusted Results. Adjusted mediation of SLE done *to* females on combined Wave I individual and family background characteristics and becoming obese over time (Appendix Table A11) were not significant, suggesting that experiencing SLE does not act to mediate the relationship between combined Wave I sociodemographics and becoming obese over time among females.

Table 6.18. Summary of Unadjusted and Adjusted Mediation of 3 or More SLE and All SLE Done *To* Females on the Relationship between Select Wave I Sociodemographics and Becoming Obese (N=4,813)

	3+ SLE Done *To*		All SLE Done *To*	
Unadjusted Wav e I Sociodemographics (reference)	a*b (SE)	% Total Effect Mediated	Σab (SE)	% Total Effect Mediated
Individual Characteristics				
Age (Years)	0.160** (0.057)	65.6	0.147 (0.076)	--
Race/Ethnicity (NH White)				
NH Black	0.106* (0.178)	10.9	0.450* (0.189)	46.2
Nativity Status (U.S.-born)				
Foreign born	-0.268 (0.222)	--	-0.288 (0.354)	--
Family Background				
Parental Education Level (More than High School)				
Less than High School	0.450* (0.193)	46.0	0.464* (0.200)	47.4
Family Income (≥$75,000)				
<$20,000	0.576* (0.268)	34.5	0.584* (0.278)	35.0
$20,000-44,999	0.276 (0.184)	--	0.287 (0.196)	--
Family Structure (2 Biological Parents)				
1 Biological Parent	0.653* (0.285)	63.4	0.650* (0.317)	63.1
Adjusted Wave I Sociodemographics[a] (reference)				
None Significant				

Note: Analysis weighted; NH= non-Hispanic.
* p≤0.05, **p≤0.01, ***p≤0.001.
[a] Model adjusted for other sociodemographic characteristics: age, race/ethnicity, nativity status, parental education level, family income, and family structure.

6.6.1.2. Summary of Viable Unadjusted and Adjusted Mediation of SLE Done To Males on Becoming Obese

Unadjusted results. Table 6.19 presents a summary of the 4 viable unadjusted mediation models of 3 or more SLE and all SLE greater than 0 SLE done *to* males on the relationship between select Wave I sociodemographics and becoming obese. With only 4 viable mediation models of 52, it is possible that the findings below represent type I error (false positive), whereby a null hypothesis that there is no significant mediating effect of SLE done *to* males on the relationship between Wave I sociodemographic characteristics and becoming obese over time is erroneously rejected.

As discussed above, the indirect effects of 3 or more SLE done to males on age and becoming obese were significant. Significant mediation was also found between males who lived in the two lowest income categories and becoming obese over time. For these males, in comparison to those with the highest income, the proportion of the total effect mediated by 3 or more SLE was 55.3% and 45.7%. Additionally, SLE done to males significantly mediated the relationship between living in other family situations and becoming obese. Among these males, compared to those in two biological parent homes, 3 or more SLE done to them significantly mediated 58.0% of the total effect.

Mediation results of all SLE greater than 0 SLE done to males were similar to those of 3 or more SLE. As discussed earlier, the proportion of the total effect of age mediated by all SLE greater than 0 SLE was 59.6%. Unadjusted mediation effects of all SLE greater than 0 SLE on family background characteristics and becoming obese were nearly identical to those of 3 or more SLE, with the percent of the total effect mediated remaining similar to that of 3 or more SLE. The proportion of the total effect of living in other family situations compared to those

living in two biological parent homes mediated by all SLE greater than 0 SLE on becoming obese was 62.7%, slightly higher than that mediated by 3 or more SLE (58.0%); suggesting that the vast majority of the effect of individual family background characteristics is mediated through 3 or more SLE done to males.

Adjusted results. Significant mediation of SLE done *to* males on select Wave I sociodemographics and becoming obese over time were also found once individual and family background characteristics were combined. Higher numbers of SLE done *to* adolescent males significantly mediated the relationship between Wave I age and becoming obese over time. With each year increase in age during adolescence, the proportion of the total effect mediated by 3 or more SLE on becoming obese was 51.3%. Among males living in a home with $20,000-$44,999, compared to those with the highest income, the proportion of the total effect mediated by 3 or more SLE was 33.3%. Mediating effects of males living with the lowest income and those in other family situations through 3 or more SLE were not significant once Wave I individual characteristics were taken into account.

Adjusted mediation results of all SLE greater than 0 SLE done *to* males were similar to those of 3 or more SLE. The proportion of the total effect of age mediated by all SLE greater than 0 SLE on becoming obese was 57.7%. All SLE greater than 0 SLE done *to* males significantly mediated 31.5% of the total effect of males with a family income of $20,000-$44,999 on becoming obese. A family income of less than $20,000 and a family structure of other family situations were not significantly mediated by all SLE greater than 0 SLE once Wave I individual characteristics were taken into account.

Table 6.19. Summary of Unadjusted and Adjusted Mediation of 3 or More SLE and All SLE Done *To* Males on the Relationship between Select Wave I Sociodemographics and Becoming Obese (N=4,498)

	3+ SLE Done *To*		All SLE Done *To*	
Unadjusted Wave I Sociodemographics (reference)	a*b (SE)	% Total Effect Mediated	Σab (SE)	% Total Effect Mediated
Individual Characteristics				
Age (Years)	0.179*** (0.051)	53.9	0.198** (0.063)	59.6
Family Background				
Family Income (≥$75,000)				
<$20,000	0.822*** (0.236)	55.3	0.834*** (0.236)	56.1
$20,000-44,999	0.634** (0.212)	45.7	0.593** (0.203)	42.8
Family Structure (2 Biological Parents)				
Other Situations	0.937*** (0.293)	58.0	1.013*** (0.287)	62.7
Adjusted Characteristics (reference)[a]				
Age (Years)	0.159*** (0.023)	51.3	0.179** (0.059)	57.7
Family Income (≥$75,000)				
$20,000-44,999	0.403* (0.170)	33.3	0.381* (0.169)	31.5

Note: Analysis weighted; NH= non-Hispanic.
* p≤0.05, **p≤0.01, ***p≤0.001.
[a] Model adjusted for other sociodemographic characteristics: race/ethnicity, nativity status, parental education level, family structure.

6.6.2. Mediator: Stressful Life Events Performed By Adolescent Females

As discussed earlier, Table 6.15 indicated that 5 viable mediation models of SLE performed *by* females on becoming obese existed. No viable mediation models of SLE performed *by* males and becoming obese were found. Among females, mediation results showed that neither the individual indirect effect of 2 or more SLE, nor the total indirect effect of all SLE compared to 0 SLE performed *by* females significantly mediated the relationship between select Wave I sociodemographic characteristics and becoming obese over time. Similar results were found upon examining adjusted mediation (see Appendix Tables A13-A21 for detailed mediation decomposition).

6.7. Mediation Models of Wave I SLE on Staying Obese

Multinomial regression was done to examine the potential role of SLE done *to* and performed *by* the adolescent as a mediator between Wave I sociodemographic characteristics and staying obese. Table 6.15 previously showed that 5 viable unadjusted models whereby SLE done *to* females on staying obese existed. However, no viable unadjusted models of SLE performed *by* females on staying obese were found. Similarly, no viable unadjusted mediation models of SLE done *to* and performed *by* males on staying obese were found.

6.7.1. Mediator: Stressful Life Events Done To Adolescent Females

Unadjusted results. Table 6.20 presents a summary of the 5 viable unadjusted mediation models (detailed mediation results found in Appendix Tables A22-A31). With each year increase in age, the proportion of the total effect mediated by 3 or more SLE on staying obese was 59.2%. No significant mediating effects of race/ethnicity and nativity status through 3 or more SLE done *to* females on staying obese were found. However, significant mediation was found between less than a high school educated parent and staying obese over time. For these females, in comparison to those whose parent had more than a high school degree, the proportion of the total effect mediated by 3 or more SLE on staying obese was 47.6%. SLE done *to* females did not significantly mediate the effect of family income on staying obese over time.

Mediation results of all SLE greater than 0 SLE done to females revealed no significant mediating effects between age and staying obese. However, among Black females, compared to Whites, the proportion of the total effect mediated by all SLE greater than 0 SLE on staying obese was 33.5%. No significant unadjusted mediation results were found indicating that all SLE done *to* adolescent females acts as a mediator between nativity status and staying obese over time. However, significant mediation results showed that for females with less than a high school

educated parent, in comparison to those whose parent had more than a high school degree, the proportion of the total effect mediated by all SLE greater than 0 SLE was 50.6%. Mediation results of all SLE between family income and staying obese among females were not significant.

Adjusted results. Adjusted mediation of SLE done to females on Wave I sociodemographics and staying obese over time (Appendix Table A32) were not significant, suggesting that experiencing SLE does not act to mediate the relationship between combined Wave I sociodemographics and staying obese over time among females.

Table 6.20. Summary of Unadjusted and Adjusted Mediation of 3 or More SLE and All SLE Done *To* Females on the Relationship between Select Wave I Sociodemographics and Staying Obese (N=4,813)

	3+ SLE Done *To*		All SLE Done *To*	
Wave I Sociodemographics (reference)	a*b (SE)	% Total Effect Mediated	Σab (SE)	% Total Effect Mediated
Individual Characteristics				
Age (Years)	0.135* (0.066)	59.2	0.144 (0.086)	--
Race/Ethnicity (NH White)				
NH Black	0.315 (0.171)	--	0.367* (0.178)	33.5
Nativity Status (U.S.-born)				
Foreign born	-0.225 (0.204)	--	-0.375 (0.350)	--
Family Background				
Parental Education Level (More than High School)				
Less than High School	0.391* (0.184)	47.6	0.416* (0.195)	50.6
Family Income (≥$75,000)				
<$20,000	0.455 (0.249)	--	0.478 (0.258)	--
Adjusted Wave I Sociodemographics[a] (reference)				
None Significant				

Note: Analysis weighted; NH= non-Hispanic.
* p≤0.05, **p≤0.01, ***p≤0.001.
[a] Model adjusted for other sociodemographic characteristics: age, race/ethnicity, nativity status, parental education level, family income, and family structure.

6.8. Chapter Summary

The purpose of this chapter was to investigate the mediating effect of adolescent stressful life events (SLE) done *to* and performed *by* adolescent females and males on the relationship between Wave I sociodemographic characteristics and longitudinal patterns of obesity during the transition to adulthood using Add Health. To determine viable mediation models, bivariate and multivariate multinomial and ordinal logistic regression analyses were employed to examine the relationship between Wave I sociodemographics and Wave I SLE, and between Wave I SLE and longitudinal obesity patterns. Viable mediation models were then examined using path analysis.

Overall, my theoretical model found mixed support in the empirical evidence presented in this chapter and in Chapter 5. Consistent with the stress process model, the associations between select Wave I sociodemographic characteristics and Wave I SLE were significant (mediation pathway a), as well as the associations between Wave I SLE and longitudinal obesity patterns among females and males (mediation pathway b). As hypothesized, bivariate distributions and multivariate ordered logistic regression of Wave I SLE by Wave I sociodemographics revealed significant associations for females and males. Additionally, SLE done *to* adolescents played a more significant role in contributing to longitudinal obesity patterns than SLE performed *by* adolescents.

However, significant mediating effects of select Wave I sociodemographic characteristics (age and family income) were only found for Wave I SLE done *to* males on becoming obese, and such results were tenuous given a possible type I error. These findings suggest that the further examination of stress as a mediator are needed to explain sociodemographic differences in becoming and staying obese from adolescence to young adulthood, and that other factors, such as

physical activity, eating behavior, and other psychosocial factors may also play a significant role (Lee et al., 2009).

CHAPTER 7

DISCUSSION, LIMITATIONS, AND PUBLIC HEALTH IMPLICATIONS

7.1. Summary of the Study

The overall objectives of the current study were to examine the sociodemographic correlates of allostatic load (AL) among a nationally representative sample of adolescents age 12 to 19 years, and to explore the mediating mechanism of adolescent stressful life events (SLE) on the relationship between social conditions and longitudinal patterns of obesity during the transition to adulthood. This study used two nationally representative datasets: five cross-sectional cycles (1999-2008) of adolescents within NHANES, and three longitudinal waves of adolescents across Add Health. Specific significant differences in AL among adolescents by age and race/ethnicity were found, as well as select significant mediating pathways of adolescent SLE on becoming obese among males.

The findings of this dissertation are noteworthy in building upon the life course health development framework by indicating that the transitional period of adolescence plays an essential role in the variation of early life social conditions contributing to physiological dysregulation at a young age, which thereby play a role in health trajectories across the life course. Additionally, this study is among the first to demonstrate using the stress process framework that adolescent stressful life events may partially explain how adolescent males become obese during the transition to adulthood.

This section will review the major findings of the dissertation by specific aim. I will then describe strengths and limitations of the study that may influence interpretation of those findings, followed by a discussion of public health implications and suggestions for future research.

7.2. Discussion of Major Findings

7.2.1. Aim 1: To use a nationally representative sample of adolescents in NHANES to examine the change in levels of AL by age and race/ethnicity across adolescence from 12 to 19 years.

Overall, the main hypotheses of this aim were supported. Results indicated that AL was significantly associated with a number of sociodemographic factors. Specifically, higher AL scores, suggestive of greater cumulative physiological dysregulation, were significantly associated with older age, NH Black race/ethnicity, and being U.S.-born. As predicted, levels of AL score increased with age across adolescence, indicative of the accumulation of stress biomarkers across the life course and the inability of the body's regulatory systems to adapt to stressors over time.

Non-Hispanic Black adolescents had higher AL scores compared to Whites, net of other covariates, a finding consistent with previous AL research among adults and the aging (Chyu & Upchurch, 2011; Geronimus et al., 2006) suggesting that Blacks may experience greater stress during adolescence than other racial/ethnic groups. However, the present findings are also contrary to prior work on AL among a convenience sample of Midwestern adolescents, which found that AL score did not differ significantly by race/ethnicity or age (Goodman et al., 2005).

The healthier biological risk profile of foreign born adolescents was uniform with previous research assessing AL among adult women (Chyu & Upchurch, 2011). Possible explanations for healthier AL profiles among foreign born adolescents are protective cultural practices and health behaviors, or even selective migration of healthier children or infants migrating with healthy parents from the country of origin (Crimmins et al., 2005; Palloni & Arias, 2004; Vega et al., 2011). Socioeconomic status also had a significant impact on AL score; adolescents who had a household representative with less than a high school education, and had a

family income of less than $20,000, had higher AL scores than their higher SES counterparts; similar to previous findings among adolescents and adults (Goodman et al., 2005; Seeman et al., 2010a). Such findings on SES and AL score support positive linkages between SES and "downstream" physiological outcomes, which state that individuals with fewer socioeconomic resources are at risk for poor health due to limited access to health care, greater exposure to environmental and psychosocial stressors, and less support (LaVeist, 2005; Taylor et al., 1997; Thoits, 1995; Williams & Collins, 1995).

Age by race/ethnicity interaction terms in multivariate negative binomial regression models revealed significant racial/ethnic differences in AL scores across adolescence from 12 to 19 years. As expected, for all racial/ethnic groups, AL score increased with age. However, non-Hispanic Black adolescents had higher AL scores relative to other racial/ethnic groups across all ages of adolescence. Explanations for this may be greater exposure to environmental and psychosocial stressors during adolescence, or differences in the interpretation or response to stressful stimuli among Black adolescents compared to Whites. Additional findings showed that White adolescents appeared to lose their advantage over time, with the disparity in AL score between Blacks and Whites declining after age 15. Further exploration of AL between White and Black adolescents is needed to explain this trend.

These findings are unique, in that they are the first to show that cumulative physiological dysregulation increases significantly during adolescence, and such increase differs by race/ethnicity. Previous research on AL among youth and young adolescents has not examined specific trends in AL by age. Specifically, Evans and colleagues showed that cumulative risk elevated AL during childhood (Evans, 2003; Evans et al., 2007; Gersten, 2008), while Goodman et al. (2005) found no significant difference in AL by age; likely a result of small sample size.

The present results support the life course approach of stressor accumulation over time, the body's inability to adapt to life's social conditions, and greater cumulative biological dysregulation with age.

Additionally, the current study also examines Hispanic adolescents, a demographic group that had yet to be investigated. The present study found that across all ages of adolescence, Hispanics had similar cumulative biological risk profiles as did their non-Hispanic White counterparts; however, post-hoc interactions revealed no difference in AL score for foreign born Hispanics by age compared to U.S. born Hispanics (or for any other racial category), a finding seen in prior research on Mexicans and nativity status (Adler & Rehkopf, 2008; Chyu & Upchurch, 2011; Crimmins et al., 2007; Peek et al., 2010).

Chyu and Upchurch (2011) found that U.S.-born Mexican women had higher AL than foreign-born Mexican women, and that foreign-born Mexican women had AL scores comparable to or lower than AL scores for White women (Chyu & Upchurch, 2011). Reasons for this discrepancy may be that Hispanic adolescents react differently to stressors, or that they have protective coping resources. Resources such as ethnic enclaves and family support systems are consistent with the Hispanic paradox. The present results are largely consistent with the broader literature on the Hispanic paradox, and that even after considering nativity, there may be unmeasured protective factors that account for the relatively lower AL scores. With Mexicans and other Hispanics among the fastest growing demographic groups in the U.S. (Adler & Rehkopf, 2008), future research on acculturation, migration experiences, and coping resources, may help uncover why nativity status and Hispanic ethnicity contribute to decreased AL scores among adults but not adolescents.

The present research is consistent with Link and Phelan's (1995; 2010) theory of social conditions and fundamental causes of health differences. Results showed that adolescents from lower SES households had higher levels of AL. The increased stress of lower SES, coupled with the developmental period of adolescence, potentially contributes to higher levels of AL and ultimately, poorer health in adulthood (Karlamangla et al., 2002; Karlamangla et al., 2006; Seeman et al., 2004).

7.2.2. Aim 2: To use Add Health to examine adolescent sociodemographic differences in longitudinal patterns of obesity during the transition to adulthood separately for females and males.

Overall, the main hypotheses of this aim were supported. Using the longitudinally diverse Add Health data, the present dissertation found that over the 5-year period between Waves II and III considerable differences in longitudinal patterns of obesity were observed for females and males. On the basis of the pediatric cutoff for adolescents (95^{th} percentile, 2000 CDC growth charts), 11% of females were obese. By young adulthood, according to the adult cutoff (BMI $\leq$ 30), 22% of females were obese. Longitudinal patterns of obesity showed that 13% of females became obese over time, while only 10% stayed obese. Among males, 14% of males were obese in adolescence, and 20% were obese in young adulthood. Nine percent of males became obese over time, while only 10% stayed obese.

Findings also showed that Black females were more likely to start off with a higher proportion of obesity in adolescence compared to Whites, and subsequently have a higher incidence of becoming obese in young adulthood. U.S.-born females, those in low SES households, and living in single biological parent homes were also more likely to become obese over time. Asian females were significantly less likely to stay obese over time compared to their

White counterparts. U.S.-born females, those in low SES households were also significantly more likely to stay obese. Among males, those living in lower family income and other family situations were more likely to become obese. No significant differences in becoming over time were found by race/ethnicity or nativity status. Males with a lower family income were significantly more likely to stay obese over time.

Consistent with previous research, these findings indicate a high incidence and prevalence of obesity during the transition from adolescence to young adulthood (Gordon-Larsen et al., 2004; Gordon-Larsen et al., 2010; Lee et al., 2009). Despite the rapid height growth of males after puberty and the equally profound hormonal and physical changes in females during this time, nearly a quarter of females and males were obese in young adulthood, double that from adolescence. This suggests the critical nature of focusing on obesity prevention before adulthood and enhancing mechanisms of promoting healthy behaviors during the transition to adulthood.

The effect of these findings on the population was substantial. This Add Health analysis sample represented ~15.9 million 12 to 19 year-old students at public and private schools in the U.S., and suggests that nearly 2 million adolescents became obese during the transition to adulthood, and another 1.6 million remained obese during this period. These findings reflect similar studies that show significant trends in childhood overweight and obesity continuing well into adulthood (Gordon-Larsen et al., 2004; Lee et al., 2009; Power & Hertzman, 1997; Serdula et al., 1993; Srinivasan et al., 1996). Adolescent obesity is highly predictive of adult obesity, more so than childhood obesity (Guo et al., 1994; Whitaker et al., 1997). The present findings indicate that the transition between adolescence and young adulthood is a period of increased risk of development of obesity. Such an upward trend was found especially among non-Hispanic

Black females, U.S.-born females, and males and females of low SES. Older adolescents were also at high risk of becoming and staying obese over time.

7.2.3. Aim 3: To use Add Health to estimate the mediating effects of adolescent SLE on the relationship between sociodemographic characteristics and longitudinal patterns of obesity from adolescence to adulthood separately for females and males.

In addition to confirming the increasing prevalence of obesity from adolescence to young adulthood, the present dissertation used nationally representative data to examine the mediating mechanism of adolescent stressful life events (SLE) on the relationship between adolescent sociodemographic characteristics and longitudinal patterns of obesity from adolescence to young adulthood. This research is one of the few studies to examine such a mechanism; for previous research on mechanisms of obesity progression have focused on the mediating measures of physical activity, parental monitoring, and sleep (Lee et al., 2009).

As hypothesized, Black females were more likely to experience and perform higher numbers of SLE during adolescence compared to Whites, in addition to lower SES females and those living in non-two biological parent homes. Similar findings were also found among males. These results are consistent with prior work showing that Black adolescents experience higher numbers of SLE than White (Boardman & Alexander, 2011). Research using longitudinal data confirms these findings, and shows that the number of SLE experienced during adolescence differs by age, race/ethnicity, and socioeconomic status (Ge, 1994; Ge et al., 2006).

Disadvantaged adolescents are likely to experience added burdens of caring and supporting for family members, entering the workforce, and suffering from limited resources at a young age, demonstrating how life's social conditions, per the stress process model, expose individuals to varying levels of stress. Such stressors, as found in the current dissertation, include

life events of violence, suicide, relationship termination, and sexually transmitted infections. Present findings showed that males reported experiencing higher numbers of SLE done *to* and performed *by* them than females. This is consistent with Adkins and colleagues (2009) findings that males experienced greater exposure to SLE than females during early life, likely due to males being greater risk takers, performing violent SLE, or experiencing events more so than females as a result of hormones during puberty.

Stressful life events experienced or performed during the period of adolescence are also significantly likely to contribute to health outcomes across the lifespan. As hypothesized, the present research found that SLE experienced and performed by adolescent females were significantly associated with becoming obese over time. However, among males, only SLE experienced were significantly associated with becoming obese. The female-male difference in the relationship between adolescent SLE and longitudinal obesity patterns over time is an important finding. A few explanations of this gender difference exist. Females are more likely to report SLE during adolescence than males, and are also more likely to be obese during both adolescence and young adulthood. Other possible explanations include female coping mechanisms to stress, such as eating behavior or emotional distress, which may be more closely linked with obesity progression among females than males (Boardman & Alexander, 2011; Ge, 1994; Rolls, Fedoroff, & Guthrie, 1991). Additionally, hormonal and physical changes among females during puberty may also contribute to differences in how females internalize stress compared to males (Santrock, 2010; Steinberg & Morris, 2001).

Upon examining the potential adjusted mediating mechanism of adolescent SLE through which adolescent social conditions may operate on longitudinal patterns of obesity, I found important intervening effects of adolescent SLE done *to* males on the relationship between age

and low family income on becoming obese over time. However, these findings may represent a type I error and should be treated with caution considering that only four of 52 possible mediation models were significant.

In accordance with the stress process model, male adolescents experienced greater SLE with age, as well as males in low income families, which subsequently contributed to greater risk for becoming obese during the transition to adulthood. Such mediation is consistent with prior research that males experience greater SLE with age (Boardman & Alexander, 2011), that low SES adolescent males are significantly more likely to experience SLE (Ge, 1994; Ge et al., 2006), and that those who have experienced greater numbers of SLE are more likely to suffer from negative health outcomes (Adkins, 2009; Boardman & Alexander, 2011; Dallman, Akana, et al., 2003; Dallman, Pecoraro, et al., 2003; Dallman et al., 2005; Ge, 1994). However, no viable mediation models of SLE on staying obese over time existed, as there were no significant associations in staying obese among males; a finding similar to those of Lee and colleagues (2009).

Surprisingly, no significant adjusted mediation was found among females. Possible reasons for this could be that other factors, such as physical activity, parental monitoring, and sleep quality act to explain this relationship instead of stressful life events (Lee et al., 2009). Prior literature shows that physical activity is a significant mediator of poverty on obesity, whereby adolescents who grow up in low income households, especially girls, partake is significantly less physical activity, placing them at risk for obesity later in life.

In sum, the limited mediation found among males and lack of mediation among females suggests that the sociodemographic differences in becoming and staying obese from adolescence to young adulthood are likely due to other factors. For example, other possible stressors not

examined herein may explain these differences, such as more chronic long-term stressors or traumas; or daily hassles, which may contribute to harmful eating behaviors and patterns over time. Further research on stress exposure, eating behavior, physical activity, and emotional support during adolescence are needed to help explain why differences in longitudinal obesity patterns among female and male adolescents exist.

7.3. Strengths and Limitations of the Study

7.3.1. NHANES

The data used in this study from NHANES are cross-sectional, limiting any causal associations or linkages over time. And as each cycle of NHANES data represent a cohort of adolescents, this study is unable to longitudinally examine how AL for each individual adolescent changes with age. Additionally, there may exist selection bias within the cross-sectional data; however, as the sample age is so young, the likelihood of selectivity becoming greater with increasing age is very small. The second limitation of this study is the use of select biomarkers available in the data for the adolescent age group. Such biomarkers did not include indicators of neuroendocrine system functioning, such as cortisol, epinephrine or norepinephrine. These indicators have been identified as other important biomarkers of regulatory systems that contribute to a reliable measure of AL.

Given the variability in biomarkers used to create AL, and the limited research on AL among adolescents, there has yet to be a preferred set of biomarkers for use with younger populations. The present research is the first to present a comprehensive multisystem AL score among a representative sample of adolescents. Perhaps future AL studies will inform standardizations in AL measurement among different demographic groups, building on the

present findings. Hopefully future research collecting biological markers of systemic functioning will integrate a range of biomarkers that will enable development of a more inclusive and replicable measure of AL across all ages.

The third limitation of this study using NHANES is the use of the summation AL score, or a summary measure of the number of biomarkers falling within a high risk quartile based on the sample's distribution. Although this is the traditional and most often used method of AL score creation, it allots each biomarker an equal weight in the AL score due to the dichotomization of biomarkers depending on pre-set cut-points. The possible issue with such a method is that it is unlikely that each biomarker contributes equally to AL, and that particular risk factors may contribute to certain health outcomes more so than others for different sociodemographic groups and at different ages (McDade, 2008). For example, resting pulse rate is often used in AL indices calculated from NHANES data. However, among adolescents pulse rate declines from early to late adolescence, suggesting that if pulse rate were included in the AL score, early teens would automatically have a higher AL score due to a higher pulse rate, even though a higher pulse rate among younger teens is physiologically appropriate. Thus, the current study did not include pulse as a representative biomarker within the AL score.

Additionally, evidence suggests that although the biomarkers are equally weighted in the construction of AL score, and may not reflect physiological reality, they may act as a conservative estimate of the association between AL and health outcomes (Karlamangla et al., 2002). The fourth limitation of this study is that AL scores were calculated using cut-points according to the sample's empirical distribution. Thus, the criteria of AL composition and scores apply only to the study sample of adolescents, and are not generalizable to other populations, such as adults or the aging. Moreover, this study created AL scores using cut-points indicative of

traditional high-risk quartiles (75^{th} or 25^{th} percentile); and reports have shown that using upper and lower bound extremes may be advantageous in capturing a more comprehensive picture of physiological dysregulation. For example, risky values of BMI can be those above 25 or under 18.5, while measures of blood pressure and cholesterol can also be dangerous if at very low or high levels.

Last, this dissertation did not look at psychosocial and behavioral factors, which play an important role in determining how adolescent social conditions are processed and impact physiological functioning. Further research on psychosocial and behavioral factors as mediating and moderating variables is needed. Despite these limitations, this study is one of the first to examine descriptive profiles of allostatic load among a nationally representative sample of U.S. adolescents age 12 to 19 years. It is also the first to develop an AL score representative of the adolescent population indicative of multisystem physiological functioning.

7.3.2. Add Health

A great strength of the present research is its use of the stress process framework to define the mediating mechanism of adolescent SLE. By using a longitudinal design, the current dissertation enables the temporal ordering of the affect by sociodemographic conditions in adolescence, the mediating mechanism through which these social conditions may operate during adolescence, and longitudinal obesity patterns as measures of change and continuity during the transition to adulthood. Finally, this research uses distinct measures of SLE based on individual control to account for the difference in stress exposure and experience during adolescence.

The accepted and standard classification of obesity among adolescents is based on age- and gender-specific percentiles of the 2000 CDC growth charts (Centers for Disease Control and Prevention, 2000), which indicate statistical rather than risk-based definitions of obesity.

However, adult obesity classifications are based on recommendations from expert panels, such as the National Heart, Lung, and Blood Institute (National Heart Lung and Blood Institute & Expert Panel on the Identification Evaluation and Treatment of Overweighte and Obesity in Adults, 1998), and indicate health risk as well as statistical distribution. Therefore, a major limitation of this study is its comparison of obesity classification from adolescence to young adulthood.

The approach used in this study attempted to address this concern in differences in obesity definition 2-fold. First, this analysis focused on obesity rather that overweight, which reduced the chance of misclassifying weight status as a result of changes in lean body mass rather than adiposity. Second, this dissertation used the CDC reference curves of adolescent obesity classification, which provide a clear comparison during the transition to adulthood.

While the data used in this study from Add Health are longitudinal, they also include a number of limitations. The Add Health survey does not include a comprehensive measure of stress. The current research conceptualizes stress as limited to SLE within the data, and other known sources of stress and strain that very well could be obtained from Add Health were not included in the present study. For example, there is no inclusion of stressors related to physical or built environment, factors that are known to be linked to well-being (Cohen & Weinstein, 1981; Downey & Van Willigen, 2005). Additionally, the stress measure used in the current study did not differentiate between different sources of stress, such as social, residential, work- or family-related issues that may have distinctly different consequences on mediating longitudinal patterns of obesity.

Agreement exists that stress is composed of both acute and cumulative stress (Pearlin, 1999; Pearlin et al., 2005), and measurement strategies typically involve summing the total number of stressors during a period of time. While the present study used this method of

measuring SLE, future research would benefit from more descriptive constructs of SLE, such as stress trajectories (Boardman & Alexander, 2011), as well as more longitudinal measure of SLE across waves (Adkins, 2009; Boardman & Alexander, 2011). Longitudinal measures would differentiate not only between low and high stress, but also between individuals who face high stress at younger ages to those who had minimal stress through adolescence.

Other aspects of the stress process, such as chronic stressors and buffering resources, may also be important components of the stress-obesity relationship (Pearlin, 1989; Pearlin et al., 1981; Pearlin et al., 2005). Future research should improve upon the present analysis with more exhaustive models integrating chronic stressors and buffering psychosocial resources as predictors and mediators in the stress process. This study was also limited in that earlier waves of Add Health begin at adolescence, and there is a fair amount of pre-existing overweight or obesity prior to adolescence. With earlier waves of Add Health before adolescence, this dissertation would be able to determine exactly when social conditions and SLE may have begun to influence obesity status. However, the longitudinal design of this dissertation does capture patterns of obesity and allow an investigatory look at time-varying influences of social conditions and SLE during adolescence.

Despite these limitations, the current dissertation constructs path analysis models within MPlus to accurately determine mediation effects and detailed decomposition of such mediation. This analysis enables the appropriate examination of mediation, avoiding the limitation of comparing logistic regression coefficients across models, which as a result of rescaling may be misinterpreted as mediation.

7.4. Directions for Future Research and Public Health Implications

7.4.1. Allostatic Load and Adolescence

Addressing health disparities among adolescents is a new goal for Healthy People 2020 (U.S. Department of Health and Human Services & Office of Disease Prevention and Health Promotion, 2011). Research on AL not only provides important insight into the linkage between early life stressors and adverse outcomes over the life course, but can also help inform health interventions at earlier life stages aimed at eliminating health disparities. Assessing AL during adolescence may also help illuminate why early life adversity is associated with increased morbidity and premature mortality later in life. Elevated AL early in life could be a possible mechanism for how early life exposure to stressors can lead to morbidity later in life. Future research examining AL over time, among a longitudinal sample, may help directly test the role of AL in linking early life adversity to morbidity.

With the incorporation of biological information in large population surveys, biodemographers can better observe trends in population health and mortality, and increase our understanding of how environmental and psychosocial factors "get under the skin" to affect health. This research, in combination with future research among adolescents taking into account other factors that may contribute to differing rates of AL accumulation, such as genetic influences, lifestyle and behavioral influences, psychological influences, inter-personal or social influences, and experience to chronic stressors (Seeman et al., 2010a), may play a critical role in the understanding of health disparities and health trajectories across the life course.

Methodologically, expanding on current AL algorithms to consider differential weighting and subscales of biomarkers may help clarify whether individual regulatory systems play a larger role in overall cumulative physiological dysregulation (Seeman et al., 2010b). Similarly, more

work is needed in developing criteria for creating group-specific AL indices, such as those particular to adolescents. Little research exists on clinical cut-points among adolescents for a number of biological risk factors, making it difficult to identify subclinical risky zones.

This study provides the first examination of associations between major sociodemographic factors and AL among a nationally representative sample of adolescents, focusing on racial/ethnic differences in age patterns of AL. Much research has yet to be done on AL, and even more so among younger populations. However, the concept of AL offers great promise toward expanding our understanding of how social and environmental factors are embodied within our biological regulatory systems, and translated into disease outcomes and health disparities.

The present research has significant implications for informing health prevention interventions among younger populations as well as clinical applications and policymaking aimed at reducing health disparities at earlier ages. Preventive health interventions at early stages in life, such as childhood and adolescence, targeted at improvements in family cohesion, parental monitoring, support, healthy eating habits, physical activity, and other health behaviors to buffer stressful or adverse conditions, may make a difference in an individual's health over the lifespan. The social determinants of health, such as the social gradient of health and racial inequality, which interventions and policies fail to eliminate, must also continue to be addressed. Research on AL among younger populations can also help inform clinical practice by framing the developmental period of adolescence as an integral stage in the life course susceptible to adverse experiences that can contribute to morbidity later in life. Pediatricians can apply preventive health interventions at young ages and use measures of biological systemic regulation to inform such interventions, thus inhibiting health deterioration at an early age.

7.4.2. Longitudinal Obesity Patterns from Adolescence to Young Adulthood

The substantial upward trend in the prevalence of obesity during the transition to adulthood has significant public health implications. The trend foreshadows higher rates of nutrition related chronic diseases, such as type II diabetes and metabolic syndrome, emerging at younger ages (Freedman et al., 2004; Li et al., 2006; Mokdad et al., 2001; Must et al., 1992; Nieto, Szklo, & Comstock, 1992). Moreover, life expectancy is greatly reduced by obesity, especially among young adults (Fontaine et al., 2003). Increasing obesity trends among young adults are also seen well into midlife, significantly associated with a reduction in physical activity during the transition to adulthood (Armstrong, Welsman, & Kirby, 2000; Kelder et al., 1994; Sallis, Prochaska, & Taylor, 2000). The need for adolescent-focused obesity prevention among disadvantaged groups, such as non-Hispanic Black females and low SES adolescents, is also of great importance.

Furthermore, the present study also contributes to current literature on ethnic and gender differences in exposure to adolescent SLE, and how these SLE mediate the relationship between adolescent social conditions and longitudinal patterns of obesity over time. These present findings demonstrate the utility of combining large population-based data with a stress process framework to investigate mediating models of becoming and staying obese during the transition to adulthood. As shown, this combination enabled the exploration of variation in adolescent SLE, and their role in longitudinal patterns of obesity across the life course. Future research will hopefully continue to employ the use of adolescent SLE to investigate the mediating mechanisms explaining obesity progression and continuity.

By focusing on the intervening mechanism through which adolescent social conditions may affect obesity, this research contributes to an understanding of the processes of obesity

development during childhood and adolescence. Results of the present dissertation point to possible interventions that can prevent obesity among young males who are from low SES households. Findings of SLE experienced during adolescence as an explanatory pathway to becoming obese are new, and recommendations regarding stress management and coping mechanisms could aid in the fight against the increasing obesity epidemic among the younger population. Neighborhood and school programs could engage in relaxation or meditation techniques among youth, as well as additional coping mechanisms to buffer stress exposure such as enhanced family relationships. Additionally, healthy physical activity and eating habits formed during adolescence can be further reinforced throughout adulthood, altering risk for future obesity, chronic disease, and type II diabetes.

APPENDIX

Table A1. Multinomial Regression Model of Becoming Obese by Race/Ethnicity and Adolescent SLE Done *To* Females, Add Health Waves I-III (N=4,813)

Wave I Sociodemographic (reference)	b	SE
Race/Ethnicity (NH White)		
NH Black	0.524**	0.167
Hispanic	0.254	0.185
NH Asian	-0.622	0.398
SLE done *to* (0 SLE)		
1 SLE	-0.214	0.127
2 SLE	0.010	0.191
3+ SLE	0.581**	0.213

Note: Analysis weighted; NH = non-Hispanic.
* p≤0.05, **p≤0.01, ***p≤0.001

Table A2. Decomposition of Mediated SLE Effects of Wave I Race/Ethnicity on Becoming Obese, Females, Add Health Waves I-III (N=4,813)

Wave I Sociodemographic (reference)	b	SE
NH Black		
1 SLE	0.043	0.036
2 SLE	0.002	0.029
3+ SLE	0.106*	0.178
Total Indirect Effect (Σab)	0.450*	0.189
Direct Effect (c`)	0.524**	0.167
Total Effect (c`+ Σab)	0.974***	0.250

Note: Analysis weighted; NH = non-Hispanic; Model adjusted for other racial/ethnic categories, reference to NH White.
* p≤0.05, **p≤0.01, ***p≤0.001;

Table A3. Multinomial Regression Model of Becoming Obese by Nativity Status and Adolescent SLE Done *To* Females, Add Health Waves I-III (N=4,813)

Wave I Sociodemographic (reference)	b	SE
Nativity Status (U.S.-born)		
Foreign born	-0.760*	0.304
SLE done *to* (0 SLE)		
1 SLE	-0.218	0.126
2 SLE	0.016	0.193
3+ SLE	0.630**	0.206

Note: Analysis weighted.
* p≤0.05, **p≤0.01, ***p≤0.001

Table A4. Decomposition of Mediated SLE Effects of Wave I Nativity Status on Becoming Obese, Females, Add Health Waves I-III (N=4,813)

Wave I Sociodemographic (reference)	b	SE
Foreign		
1 SLE	0.003	0.047
2 SLE	-0.023	0.280
3+ SLE	-0.268	0.222
Total Indirect Effect (Σab)	-0.288	0.354
Direct Effect (c`)	-0.760*	0.304
Total Effect (c`+ Σab)	-1.048*	0.429

Note: Analysis weighted.
* p≤0.05, **p≤0.01, ***p≤0.001

Table A5. Multinomial Regression Model of Becoming Obese by Wave I Parental Education Level and Adolescent SLE Done *To* Females, Add Health Waves I-III (N=4,813)

Wave I Sociodemographic (reference)	b	SE
Parental Education Level (More than HS)		
Less than HS	0.515***	0.149
HS Grad/GED	0.332	0.227
SLE done *to* (0 SLE)		
1 SLE	-0.223	0.128
2 SLE	0.021	0.196
3+ SLE	0.577**	0.149

Note: Analysis weighted; HS = High School.
* p≤0.05, **p≤0.01, ***p≤0.001

Table A6. Decomposition of Mediated SLE Effects of Wave I Parental Education on Becoming Obese, Females, Add Health Waves I-III (N=4,813)

Wave I Sociodemographic (reference)	b	SE
Less than HS		
1 SLE	0.011	0.027
2 SLE	0.003	0.023
3+ SLE	0.450*	0.193
Total Indirect Effect (Σab)	0.464*	0.200
Direct Effect (c`)	0.515***	0.149
Total Effect (c`+ Σab)	0.979***	0.237

Note: Analysis weighted; HS = High School; Model adjusted for other parental education categories, reference to More than HS.
* p≤0.05, **p≤0.01, ***p≤0.001

Table A7. Multinomial Regression Model of Becoming Obese by Wave I Family Income and SLE Done *To* Adolescents, Add Health Waves I-III

	Females (N=4,813)		Males (N=4,498)	
Wave I Sociodemographic (reference)	b	SE	b	SE
Family Income (≥$75,000)				
<$20,000	1.087***	0.279	0.652*	0.302
$20,000-$44,999	0.896***	0.249	0.793**	0.297
$45,000-$74,999	0.543	0.300	0.758**	0.288
SLE done *to* (0 SLE)				
1 SLE	-0.221	0.129	0.490**	0.159
2 SLE	0.022	0.190	0.265	0.206
3+ SLE	0.548**	0.202	0.824***	0.188

Note: Analysis weighted.
* p≤0.05, **p≤0.01, ***p≤0.001

Table A8. Decomposition of Mediated SLE Effects of Wave I Family Income on Becoming Obese, Add Health Waves I-III

	Females (N=4,813)		Males (4,498)	
Wave I Sociodemographic (reference)	b	SE	b	SE
<$20,000				
1 SLE	0.005	0.030	0.050	0.078
2 SLE	0.003	0.030	-0.037	0.051
3+ SLE	0.576*	0.268	0.822***	0.236
Total Indirect Effect (Σab)	0.584*	0.278	0.834***	0.236
Direct Effect (c`)	1.087***	0.279	0.652*	0.302
Total Effect (c`+ Σab)	1.671***	0.434	1.486***	0.433
$20,000-$44,999				
1 SLE	0.007	0.028	0.037	0.075
2 SLE	0.004	0.038	-0.077	0.067
3+ SLE	0.276	0.184	0.634**	0.212
Total Indirect Effect (Σab)	0.287	0.196	0.593**	0.203
Direct Effect (c`)	0.896***	0.249	0.793**	0.297
Total Effect (c`+ Σab)	1.183***	0.326	1.386***	0.407

Note: Analysis weighted; Model adjusted for other family income categories, reference to ≥$75,000.
* p≤0.05, **p≤0.01, ***p≤0.001

Table A9. Multinomial Regression Model of Becoming Obese by Waves I Family Structure and SLE Done *To* Adolescents, Add Health Waves I-III

	Females (N=4,813)		Males (N=4,498)	
Wave I Sociodemographic (reference)	b	SE	b	SE
Family Structure (2 biological parents)				
Step-family	-0.117	0.226	0.078	0.205
1 biological parent	0.381*	0.154	-0.002	0.164
Other situations	0.387	0.270	0.604*	0.273
SLE done *to* (0 SLE)				
1 SLE	-0.229	0.126	0.485**	0.158
2 SLE	0.002	0.198	0.237	0.202
3+ SLE	0.532**	0.207	0.817***	0.187

Note: Analysis weighted.
* p≤0.05, **p≤0.01, ***p≤0.001

Table A10. Decomposition of Mediated SLE Effects of Wave I Family Structure on Becoming Obese, Add Health Waves I-III

	Females (N=4,813)	
Wave I Sociodemographic (reference)	b	SE
1 biological parent		
1 SLE	-0.003	0.024
2 SLE	0.001	0.072
3+ SLE	0.653*	0.285
Total Indirect Effect (Σab)	0.650*	0.317
Direct Effect (c`)	0.381*	0.154
Total Effect (c`+ Σab)	1.030**	0.332
	Males (N=4,498)	
Other situations		
1 SLE	0.059	0.116
2 SLE	0.018	0.070
3+ SLE	0.937***	0.293
Total Indirect Effect (Σab)	1.013***	0.287
Direct Effect (c`)	0.604*	0.273
Total Effect (c`+ Σab)	1.617***	0.386

Note: Analysis weighted; Model adjusted for other family structure categories, reference to two biological parent homes.
* p≤0.05, **p≤0.01, ***p≤0.001

Table A11. Multinomial Regression Model of Becoming Obese by Wave I Sociodemographic Characteristics and SLE Done *To* Adolescents, Add Health Waves I-III

	Females (N=4,813)		Males (N=4,498)	
Wave I Sociodemographic (reference)	b	SE	b	SE
SLE *to* (0 SLE)				
1 SLE	-0.274	0.130	0.446**	0.159
2 SLE	-0.119	0.200	0.177	0.204
3+ SLE	0.382	0.206	0.709***	0.185
Age (Years)	0.104*	0.043	0.131**	0.043
Race/Ethnicity (NH White)				
NH Black	0.309	0.178	0.189	0.212
Hispanic	0.120	0.236	0.099	0.219
NH Asian	-0.340	0.462	0.145	0.340
Nativity Status (U.S.-born)				
Foreign born	-0.943*	0.384	-0.183	0.294
Parental Education Level (More than High School)				
Less than High School	0.344	0.191	-0.129	0.268
High School Grad/GED	0.184	0.238	-0.398	0.271
Family Income (≥$75,000)				
<$20,000	0.913**	0.289	0.689*	0.343
$20,000-44,999	0.825***	0.259	0.828**	0.315
$45,000-74,999	0.559	0.303	0.774**	0.295
Family Structure (2 Biological Parents)				
Step-Family	-0.244	0.224	0.118	0.043
1 Biological Parent	0.026	0.157	-0.030	0.181
Other Situations	0.059	0.268	0.376	0.303

Note: Analysis weighted; NH = non-Hispanic.
* p≤0.05, **p≤0.01, ***p≤0.001

Table A12. Decomposition of Mediated SLE Effects by Wave I Sociodemographic Characteristics on Becoming Obese, Males, Add Health Waves I-III (N=4,498)

Wave I Sociodemographic (reference)	b	SE
Age (Years)		
1 SLE	0.000	0.012
2 SLE	0.019	0.023
3+ SLE	0.159***	0.023
Total Indirect Effect (Σab)	0.179**	0.059
Direct Effect (c`)	0.131**	0.043
Total Effect (c`+ Σab)	0.310***	0.072
$20,000-$44,999		
1 SLE	0.038	0.069
2 SLE	-0.060	0.072
3+ SLE	0.403*	0.170
Total Indirect Effect (Σab)	0.381*	0.169
Direct Effect (c`)	0.828**	0.315
Total Effect (c`+ Σab)	1.209**	0.074

Note: Analysis weighted; Model adjusted for other sociodemographic characteristics: race/ethnicity, nativity status, parental education level, family structure.
* p≤0.05, **p≤0.01, ***p≤0.001

Table A13. Multinomial Regression Model of Becoming Obese by Wave I Race/Ethnicity and Adolescent SLE Performed *By* Females, Add Health Waves I-III (N=4,813)

Wave I Sociodemographic (reference)	b	SE
Race/Ethnicity (NH White)		
NH Black	0.549***	0.171
Hispanic	0.272	0.185
NH Asian	-0.608	0.398
SLE performed *by* (0 SLE)		
1 SLE	0.200	0.173
2+ SLE	0.593*	0.279

Note: Analysis weighted; NH = non-Hispanic.
* p≤0.05, **p≤0.01, ***p≤0.001

Table A14. Decomposition of Mediated SLE Effects of Wave I Race/Ethnicity on Becoming Obese, Females, Add Health Waves I-III (N=4,813)

Wave I Sociodemographic (reference)	b	SE
NH Black		
1 SLE	0.061	0.058
2+ SLE	0.285	0.195
Total Indirect Effect (Σab)	0.346	0.210
Direct Effect (c`)	0.549***	0.171
Total Effect (c`+ Σab)	0.895***	0.223

Note: Analysis weighted; NH = non-Hispanic; Model adjusted for other racial/ethnic categories, reference to NH White.
* p≤0.05, **p≤0.01, ***p≤0.001; NH = non-Hispanic.

Table A15. Multinomial Regression Model of Becoming Obese by Wave I Parental Education Level and Adolescent SLE Performed *By* Females, Add Health Waves I-III (N=4,813)

Wave I Sociodemographic (reference)	b	SE
Parental Education Level (More than HS)		
Less than HS	0.532***	0.146
HS Grad/GED	0.342	0.224
SLE performed *by* (0 SLE)		
1 SLE	0.209	0.168
2+ SLE	0.575*	0.262

Note: Analysis weighted; HS = High School.
* p≤0.05, **p≤0.01, ***p≤0.001

Table A16. Decomposition of Mediated SLE Effects of Wave I Parental Education on Becoming Obese, Females, Add Health Waves I-III (N=4,813)

Wave I Sociodemographic (reference)	b	SE
Less than HS		
1 SLE	0.041	0.049
2+ SLE	0.495	0.326
Total Indirect Effect (Σab)	0.536	0.332
Direct Effect (c`)	0.532	0.146
Total Effect (c`+ Σab)	1.068**	0.361

Note: Analysis weighted; HS = High school; Model adjusted for other parental education categories, reference to More than HS.
* p≤0.05, **p≤0.01, ***p≤0.001

Table A17. Multinomial Regression Model of Becoming Obese by Wave I Family Income and SLE Performed *By* Females, Add Health Waves I-III (N=4,813)

Wave I Sociodemographic (reference)	b	SE
Family Income (≥$75,000)		
<$20,000	1.104***	0.284
$20,000-$44,999	0.895***	0.249
$45,000-$74,999	0.534	0.298
SLE performed *by* (0 SLE)		
1 SLE	0.173	0.172
2+ SLE	0.535*	0.269

Note: Analysis weighted.
* p≤0.05, **p≤0.01, ***p≤0.001

Table A18. Decomposition of Mediated SLE Effects of Wave I Family Income on Becoming Obese, Females, Add Health Waves I-III (N=4,813)

Wave I Sociodemographic (reference)	b	SE
<$20,000		
1 SLE	0.114	0.114
2+ SLE	0.547	0.387
Total Indirect Effect (Σab)	0.661	0.424
Direct Effect (c`)	1.104***	0.284
Total Effect (c`+ Σab)	1.765***	0.522
$20,000-$44,999		
1 SLE	0.072	0.077
2+ SLE	0.391	0.297
Total Indirect Effect (Σab)	0.463	0.321
Direct Effect (c`)	0.895***	0.249
Total Effect (c`+ Σab)	1.358***	0.418

Note: Analysis weighted; Model adjusted for other family income categories, reference to ≥$75,000.
* p≤0.05, **p≤0.01, ***p≤0.001

Table A19. Multinomial Regression Model of Becoming Obese by Wave I Family Structure and SLE Done Performed *By* Females, Add Health Waves I-III (N=4,813)

Wave I Sociodemographic (reference)	b	SE
Family Structure (2 biological parents)		
Step-family	-0.116	0.226
1 biological parent	0.400**	0.150
Other situations	0.433	0.262
SLE performed *by* (0 SLE)		
1 SLE	0.184	0.174
2+ SLE	0.531*	0.268

Note: Analysis weighted.
* p≤0.05, **p≤0.01, ***p≤0.001

Table A20. Decomposition of Mediated SLE Effects of Wave I Family Structure on Becoming Obese, Females, Add Health Waves I-III (N=4,813)

Wave I Sociodemographic (reference)	b	SE
1 biological parent		
1 SLE	0.084	0.078
2+ SLE	0.696	0.385
Total Indirect Effect (Σab)	0.780	0.408
Direct Effect (c`)	0.400**	0.150
Total Effect (c`+ Σab)	1.180**	0.434

Note: Analysis weighted; Model adjusted for other family structure categories, reference to two biological parent homes.
* p≤0.05, **p≤0.01, ***p≤0.001

Table A21. Multinomial Regression Model of Becoming Obese by Wave I Sociodemographic Characteristics and SLE Performed *By* Females, Add Health Waves I-III (N=4,813)

Wave I Sociodemographic (reference)	b	SE
SLE *by* (0 SLE)		
1 SLE	0.126	0.178
2+ SLE	0.461	0.264
Age (Years)	0.107*	0.044
Race/Ethnicity (NH White)		
NH Black	0.322	0.181
Hispanic	0.137	0.232
NH Asian	-0.305	0.460
Nativity Status (U.S.-born)		
Foreign born	-0.947*	0.374
Parental Education Level (More than High School)		
Less than High School	0.340	0.187
High School Grad/GED	0.186	0.234
Family Income (≥$75,000)		
<$20,000	0.912**	0.289
$20,000-44,999	0.817***	0.257
$45,000-74,999	0.544	0.302
Family Structure (2 Biological Parents)		
Step-Family	-0.252	0.222
1 Biological Parent	0.024	0.153
Other Situations	0.071	0.257

Note: Analysis weighted; NH= non-Hispanic.
* p≤0.05, **p≤0.01, ***p≤0.001

Table A22. Multinomial Regression Model of Staying Obese by Wave I Age and SLE Done *To* Females, Add Health Waves I-III (N=4,813)

Wave I Sociodemographic (reference)	b	SE
Age (Years)	0.084	0.044
SLE done *to* (0 SLE)		
1 SLE	-0.221	0.148
2 SLE	0.076	0.179
3+ SLE	0.474*	0.215

Note: Analysis weighted.
* p≤0.05, **p≤0.01, ***p≤0.001

Table A23. Decomposition of Mediated SLE Effects of Wave I Age on Staying Obese, Females, Add Health Waves I-III (N=4,813)

Wave I Sociodemographic (reference)	b	SE
Age		
1 SLE	-0.007	0.008
2 SLE	0.016	0.039
3+ SLE	0.135*	0.066
Total Indirect Effect (Σab)	0.144	0.086
Direct Effect (c`)	0.084	0.044
Total Effect (c`+ Σab)	0.228**	0.089

Note: Analysis weighted.
* p≤0.05, **p≤0.01, ***p≤0.001

Table A24. Multinomial Regression Model of Staying Obese by Wave I Race/Ethnicity and Adolescent SLE Done *To* Females, Add Health Waves I-III (N=4,813)

Wave I Sociodemographic (reference)	b	SE
Race/Ethnicity (NH White)		
NH Black	0.728***	0.155
Hispanic	0.332	0.205
NH Asian	-2.105***	0.527
SLE done *to* (0 SLE)		
1 SLE	-0.203	0.145
2 SLE	0.077	0.178
3+ SLE	0.450*	0.213

Note: Analysis weighted; NH = non-Hispanic.
* p≤0.05, **p≤0.01, ***p≤0.001

Table A25. Decomposition of Mediated SLE Effects of Wave I Race/Ethnicity on Staying Obese, Females, Add Health Waves I-III (N=4,813)

Wave I Sociodemographic (reference)	b	SE
NH Black		
1 SLE	0.041	0.037
2 SLE	0.012	0.029
3+ SLE	0.315	0.171
Total Indirect Effect (Σab)	0.367*	0.178
Direct Effect (c`)	0.728***	0.155
Total Effect (c`+ Σab)	1.095***	0.232

Note: Analysis weighted; NH = non-Hispanic; Model adjusted for other racial/ethnic categories, reference to NH White.
* p≤0.05, **p≤0.01, ***p≤0.001

Table A26. Multinomial Regression Model of Staying Obese by Wave I Nativity Status and Adolescent SLE Done *To* Females, Add Health Waves I-III (N=4,813)

Wave I Sociodemographic (reference)	b	SE
Nativity Status (U.S.-born)		
Foreign born	-0.828*	0.401
SLE done *to* (0 SLE)		
1 SLE	-0.203	0.145
2 SLE	0.104	0.177
3+ SLE	0.529*	0.213

Note: Analysis weighted.
* p≤0.05, **p≤0.01, ***p≤0.001

Table A27. Decomposition of Mediated SLE Effects of Wave I Nativity Status on Staying Obese, Females, Add Health Waves I-III (N=4,813)

Wave I Sociodemographic (reference)	b	SE
Foreign		
1 SLE	0.003	0.044
2 SLE	-0.152	0.270
3+ SLE	-0.225	0.204
Total Indirect Effect (Σab)	-0.375	0.350
Direct Effect (c`)	-0.828*	0.401
Total Effect (c`+ Σab)	-1.203*	0.484

Note: Analysis weighted.
* p≤0.05, **p≤0.01, ***p≤0.001

Table A28. Multinomial Regression Model of Staying Obese by Wave I Parental Education Level and Adolescent SLE Done *To* Females, Add Health Waves I-III (N=4,813)

Wave I Sociodemographic (reference)	b	SE
Parental Education Level (More than HS)		
Less than HS	0.406*	0.192
HS Grad/GED	0.101	0.189
SLE done *to* (0 SLE)		
1 SLE	-0.201	0.145
2 SLE	0.123	0.173
3+ SLE	0.501*	0.215

Note: Analysis weighted; HS = High School.
* p≤0.05, **p≤0.01, ***p≤0.001

Table A29. Decomposition of Mediated SLE Effects of Wave I Parental Education on Staying Obese, Females, Add Health Waves I-III (N=4,813)

Wave I Sociodemographic (reference)	b	SE
Less than HS		
1 SLE	0.101	0.025
2 SLE	0.015	0.035
3+ SLE	0.391*	0.184
Total Indirect Effect (Σab)	0.416*	0.195
Direct Effect (c`)	0.406*	0.192
Total Effect (c`+ Σab)	0.822**	0.283

Note: Analysis weighted; HS = High School; Model adjusted for other parental education categories, reference to More than HS.
* p≤0.05, **p≤0.01, ***p≤0.001

Table A30. Multinomial Regression Model of Staying Obese by Wave I Family Income and SLE Done *To* Females, Add Health Waves I-III (N=4,813)

Wave I Sociodemographic (reference)	b	SE
Family Income (≥$75,000)		
<$20,000	0.787**	0.282
$20,000-$44,999	0.495	0.262
$45,000-$74,999	-0.063	0.248
SLE done *to* (0 SLE)		
1 SLE	-0.201	0.148
2 SLE	0.120	0.179
3+ SLE	0.433**	0.217

Note: Analysis weighted.
* p≤0.05, **p≤0.01, ***p≤0.001

Table A31. Decomposition of Mediated SLE Effects of Wave I Family Income on Staying Obese, Females, Add Health Waves I-III (N=4,813)

Wave I Sociodemographic (reference)	b	SE
<$20,000		
1 SLE	0.004	0.027
2 SLE	0.019	0.036
3+ SLE	0.455	0.249
Total Indirect Effect (Σab)	0.478	0.258
Direct Effect (c`)	0.787**	0.282
Total Effect (c`+ Σab)	1.265***	0.366

Note: Analysis weighted; Model adjusted for other family income categories, reference to ≥$75,000.
* p≤0.05, **p≤0.01, ***p≤0.001

Table A32. Multinomial Regression Model of Staying Obese by Wave I Sociodemographic Characteristics and SLE Done *To* Females, Add Health Waves I-III (N=4,813)

Wave I Sociodemographic (reference)	b	SE
SLE *to* (0 SLE)		
1 SLE	-0.226	0.154
2 SLE	0.022	0.183
3+ SLE	0.353	0.215
Age (Years)	0.092*	0.044
Race/Ethnicity (NH White)		
NH Black	0.590***	0.147
Hispanic	0.253	0.229
NH Asian	-1.859***	0.558
Nativity Status (U.S.-born)		
Foreign born	-0.822	0.425
Parental Education Level (More than High School)		
Less than High School	0.124	0.237
High School Grad/GED	-0.076	0.222
Family Income (≥$75,000)		
<$20,000	0.752*	0.294
$20,000-44,999	0.499	0.265
$45,000-74,999	-0.062	0.251
Family Structure (2 Biological Parents)		
Step-Family	-0.303	0.253
1 Biological Parent	-0.231	0.165
Other Situations	-0.405	0.339

Note: Analysis weighted; NH= non-Hispanic.
* p≤0.05, **p≤0.01, ***p≤0.001

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