THE SOCIAL STRUCTURAL CONTEXT OF PREGNANCY AND ADVERSE BIRTH OUTCOMES: THE ROLE OF RACE, PLACE, AND TIME

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ABSTRACT

LIANA JANINE RICHARDSON: The Social Context of Pregnancy and Adverse Birth Outcomes: The Role of Race, Place and Time
(Under the direction of Jo Anne Earp (chair), Susan Ennett, Arjumand Siddiqi, Kenneth Bollen, and Kathleen Mullan Harris)

The persistence of racial disparities in low birth weight (LBW) and preterm birth (PTB) is one of the most widely documented problems in public health. Most studies of these birth outcomes have focused primarily on maternal exposure to individual-level risk factors during the prenatal period. That research has resulted in only a partial explanation for the observed disparities. My research, therefore, had two primary aims: (1) to examine the relationship between adverse birth outcomes and maternal exposure to family and neighborhood disadvantage during childhood; and (2) to determine the behavioral, psychosocial, and health pathways through which these exposures affect birth outcomes and translate into racial disparities.

Data for this research came from Waves I and III of the National Longitudinal Study of Adolescent Health (Add Health). The sample consisted of 500 Black and 1,077 White females who gave birth in late adolescence or young adulthood. I used structural equation modeling to test hypotheses associated with the study aims. Latent variables representing four domains of childhood disadvantage (i.e., family disadvantage and neighborhood

structural, social, and physical disadvantage) were used in the analysis, along with measures of childhood health, depression, and substance use.

When the latter measures were not included in the model, the effects of childhood family disadvantage and neighborhood structural disadvantage on infant birth weight were fully mediated by the other neighborhood variables and gestational age. When childhood substance use, depression, and health were entered into the model as mediators, a direct effect of childhood neighborhood physical disadvantage on infant gestational age remained. All relationships between birth weight and other aspects of childhood disadvantage were fully mediated. When the analysis was stratified by race, clear racial differences in overall and component model fit were apparent. The fit for Whites more closely matched the fit of the full sample model than did the fit for Blacks; indeed, no variables were associated with birth outcomes among Blacks.

These findings suggest that disadvantage experienced at multiple levels and in multiple domains during childhood may play a role in subsequent birth outcomes. They also suggest that the role may differ between racial groups.

To my parents, Bruce and Rita Richardson

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TABLE OF CONTENTS

LIST OF TABLES

Table

LIST OF FIGURES

Figure

ABBREVIATIONS

Add Health	National Longitudinal Study of Adolescent Health
CES-D	Center for Epidemiologic Studies Depression Scale
LBW	Low Birthweight
IOM	Institute of Medicine
PTB	Preterm Birth
SEM	Structural Equation Modeling
SES	Socioeconomic Status
U.S.	United States

CHAPTER 1

INTRODUCTION

1.1. Statement of the Problem

The persistence of racial disparities in health is one of the most widely documented problems of contemporary public health in the United States. Among the most challenging disparities are those that pertain to perinatal health. A two- to three-fold higher risk of preterm birth (PTB),[1] low birthweight (LBW),[2] and infant mortality[3] has persisted among African Americans as compared to Whites, Hispanics, and Asian Americans for decades (Singh & Yu, 1995; Guyer et al., 1997; Martin et al., 2002; NCHS, 2003; Hoyert et al., 2006). Much of the racial/ethnic differences in infant mortality rates are due to disparate rates of PTB—a primary cause of LBW and the main determinant of infant mortality among African Americans (Mathews & MacDorman, 2007). Although the rate of PTB among African American women was lower in 2000 than it was in 1990, it has begun to increase again in recent years (Hoyert et al., 2006). Concomitantly, the disparity in mortality between African American and White American preterm infants is widening (Demissie et al., 2001; Mathews & MacDorman, 2007). Among infants who survive the first year of life, PTB and

[1]Preterm birth refers to a live birth prior to the 37th completed week of gestation (Nguyen & Wilcox, 2005).

[2]Low birthweight refers to the birth of an infant (at term or preterm) weighing less than 2500 grams (Nguyen & Wilcox, 2005).

[3]Infant mortality refers to death in the first year of life (Nguyen & Wilcox, 2005).

LBW are associated with a variety of childhood health and developmental problems and adult morbidity (IOM, 2006; Boardman et al., 2002).

Despite decades of research on risk factors for adverse birth outcomes, many cases occur without known cause (Goldenberg et al., 2008; Mattison et al., 2001; Slattery & Morrison, 2002; Kramer, 1995). Known risk factors, such as smoking, inadequate prenatal care, lower socioeconomic status, and chronic or gestational illness, explain fewer than half the cases of adverse birth outcomes and do not account for a large portion of the racial/ethnic variation in rates of these outcomes (Shiono et al., 1997; Hummer et al. 1999; Berg, Wilcox, & d'Almada, 2001; Goldenberg et al., 2008). Furthermore, removal or treatment of modifiable risk factors during prenatal care has not been sufficient to eliminate the racial disparities (Gennaro, 2005).

The epidemiological and intervention literature not only fails to fully explain racial disparities in birth outcomes but also displays several limitations. Chief among them are the frequent use of a single risk factor approach and a focus on decontextualized exposures to biological, behavioral, and psychosocial risks during the prenatal period or the period immediately preceding conception (Wing, 1994; Krieger, 1994; Wise, 1993; Misra, Grason, & Weisman, 2000). Thus, traditional birth outcome studies often are limited in their ability to determine causation and ignore how factors act together, as well as how they are shaped by the social structural context in which they occur, over time (and well before pregnancy). Even social factors, such as socioeconomic status (SES), are decontextualized in traditional birth outcome studies by the frequent reliance on individual-level measures, such as income and education. This practice has led some researchers to suggest that examination of SES-

related *living conditions* is necessary if we are to understand better the influence of social stratification processes on adverse birth outcomes (Kaufman, Cooper, & McGee, 1997; Oliver & Shapiro, 1995; Lynch & Kaplan, 2000; Culhane & Elo, 2005).

Although studies of lifecourse and contextual influences on birth outcomes have begun to be published, these studies are in their infancy and have not examined lifecourse and context simultaneously (Lu & Halfon, 2003). A more integrative approach seems desirable—one that: (1) examines the association between women's exposure *histories* and their risk for adverse birth outcomes; (2) explores the clustering, accumulation, or potential interactions among risk factors at the individual and contextual levels across the lifecourse; and (3) identifies the pathways and mediating mechanisms by which these relationships create racial disparities in adverse birth outcomes (Lu & Halfon, 2003; Hogan et al., 2001a, 2001b; IOM, 2006). Developing a strong theoretical foundation for this approach is a necessary first step (IOM, 2006).

1.2 Purpose and Aims of the Study

In an attempt to address these gaps, I conducted theory-based research on the combined role of race, place, and time in explaining racial disparities in adverse birth outcomes. In doing so, I examined the relationships between early life exposure to social and contextual risk factors and subsequent birth outcomes, as well as the more proximate factors that mediate those relationships, among African American and White mothers. The specific aims of the study were to:

1) Describe the prevalence of adverse birth outcomes by maternal age and race;

2) Examine the relationship between adverse birth outcomes and maternal exposure to family-level and neighborhood-level disadvantage during childhood; and

3) Determine the behavioral, psychosocial, and health pathways through which these exposures affect birth outcomes and translate into racial disparities.

Previous studies have shown that racial disparities in adverse birth outcomes increase with increasing maternal age, beginning in the late teens and early 20s, and that this increase is not due to the over-representation of African American mothers at earlier maternal ages (Geronimus, 1986, 1996; Ventura et al., 2000; Rauh, Andrews, & Garfinkel, 2001; Schempf et al., 2007). Thus, I explored the relationships and pathways described in my study aims using a sample of females in late adolescence and young adulthood from the National Longitudinal Study of Adolescent Health (Add Health). Add Health is a school-based, nationally representative study of the health, health-related attitudes and behaviors, and social contexts of a cohort of individuals from adolescence through their adult years.

1.3 Significance of the Study

This study was conducted at a time when interest in social determinants of health was prevalent in public health. In addition, research on contextual effects and the lifecourse impacts of social determinants on health was growing (Kawachi & Berkman, 2003; Diez-Roux, 2001; Hertzman, 2004; James et al., 2006). The study also followed on the heels of the Institute of Medicine's (2006) report on PTB, which documented progress on the prediction and prevention of PTB and outlined the work remaining to be done. Table 1 below lists specific IOM recommendations to which this study responded.

Table 1. Selected Recommendations from IOM Report on Preterm Birth (2006)

▪ Examine depression in African American women.
▪ Examine the pathways by which racism influences PTB, and whether it acts together with other risk factors to increase PTB risk.
▪ Gather evidence to support the weathering hypothesis.
▪ Study adverse neighborhood conditions and gestational age instead of birth weight to reduce confounding with small-for-gestational age.
▪ Study risk factors at multiple levels across the life course, with regard to disparities in PTB.
▪ Develop strong theoretical models of the pathways from psychosocial factors to PTB.

This research also responded to the Institute of Medicine's report on racial and ethnic differences in health over the lifecourse (Hertzman, 2004), and to the Healthy People 2010 objective to eliminate racial disparities in birth outcomes (USDHHS, 2000). Moreover, it offers policy- and practice-relevant theoretical and methodological contributions to disparities scholarship, including: evidence of the potential utility of combining family- and contextual-level measures of risk with more proximate measures; applying a multilevel and longitudinal conceptual framework, based on social ecological and lifecourse perspectives; and using an analytic strategy involving latent variable modeling and estimation of simultaneous equations in a structural equation modeling framework.

1.4 Overview of the Dissertation

The dissertation has six chapters. Chapter Two presents the findings from a review of the literature on factors associated with racial disparities in birth outcomes, with a critical analysis of the emerging literature on the relationship between race, the level and timing of exposure to risk and protective factors, and adverse birth outcomes. Chapter Three contains the theoretical foundation for the study and the study's conceptual model, and

presents the research questions and the hypotheses that were tested. Chapter Four lays out the methodology used to answer the research questions, including study design, sample construction, key variables and their definitions, and analytic techniques. Chapter Five presents descriptive statistics for the study and the results of the hypothesis tests. Chapter Six synthesizes the key findings, situates them within the context of the extant literature, and makes recommendations for future research and practice.

CHAPTER 2

LITERATURE REVIEW

Adverse birth outcomes, such as low birthweight (LBW) and preterm birth (PTB), are of public health concern because they are associated with an increased risk for subsequent infant mortality and, among survivors, a variety of short- and long-term health and developmental problems (Mathews & MacDorman, 2007; Barker, 2007; Jaddoe & Witteman, 2006; Boardman et al., 2002). Consequently, adverse birth outcomes have implications for numerous public sector services, including public health practice. Despite decades of research on their causes, little is known about how to prevent adverse birth outcomes and eliminate the persistent racial/ethnic disparities in their occurrence (Mattison et al., 2001). In this chapter, I: (1) review and synthesize the findings of this research; (2) identify its limitations and gaps, focusing on those to which the dissertation responds; and (3) argue for further research on the social and temporal context of adverse birth outcomes to assist in identifying appropriate prevention strategies.

2.1 The Epidemiology of Adverse Birth Outcomes

In 2005, a total of approximately 500,000 (13%) live births were classified as preterm and 340,000 (8.2%) were classified as low birthweight in the U.S. (Martin et al., 2007). These rates are considerably higher than the Healthy People 2010 goals of 7.6% and 5.0%,

respectively (USDHHS, 2000). They are also higher than the rates in other developed countries, including countries in East Asia, the Scandinavian or Nordic regions of Europe, the rest of Western Europe, and Latin America (Paneth, 1995; Bale, Stoll, Lucas, 2003; Langhoff-Roos et al., 2006). In 2004, for example, only 6.2% of infants were classified as preterm in Denmark; 5.8% in Sweden; and 6.4% in Norway (Langhoff-Roos et al., 2006; Morken et al., 2005, 2008). In 2003, the U.S. LBW rate ranked 25th among 28 wealthy industrialized OECD (Organization for Economic Cooperation and Development) countries for which these data were available; its infant mortality rate also ranked 25th in 2002 (OECD, 2005).[4]

Although they are conceptually and sometimes etiologically distinct categories (Nguyen & Wilcox, 2005), preterm birth (PTB) and low birthweight (LBW) are not mutually exclusive. Nearly two-thirds of preterm infants are also LBW because PTB is one of its primary causes (Martin et al., 2007). Both are associated with an increased risk of infant mortality. In 2004, for example, 27,860 infant deaths occurred; of those infants, 19,219 were classified as LBW at birth and 18,585 were classified as PTB at birth (Mathews & MacDorman, 2007).

African Americans have consistently experienced higher rates of LBW, PTB, and infant mortality than all other racial/ethnic groups (Singh & Yu, 1995; Guyer et al., 1997; Martin et al., 2002; NCHS, 2003; Hoyert et al., 2006). In 2004 and 2005, non-Hispanic

[4]Higher rankings indicate poorer relative performance.

Blacks[5] were the only racial/ethnic group with rates of these outcomes that were higher than the national rates (Table 2). In addition, non-Hispanic Black rates of LBW, PTB, and infant mortality were 1.9, 1.6, and 2.4 times higher than the rates for non-Hispanic Whites, respectively (NCHS, 2004, 2005).

Table 2. Percentage of preterm births, percentage of low birthweight births, and number of infant deaths per 1,000 live births, by race and Hispanic origin of mother

	Birth Outcome		
Maternal Race and Hispanic Origin	**Low birthweight**	**Preterm**	**Mortality Rate**
Non-Hispanic Black	14.0	18.4	13.60
Non-Hispanic White	7.3	11.7	5.66
Hispanic	6.9	12.1	5.55
American Indian or Alaska Native	7.4	14.1	8.45
Asian or Pacific Islander	8.0	10.8	4.67
All races and origins	*8.2*	*12.7*	*6.78*

DATA SOURCE: National Center for Health Statistics, Final Birth Data, 2005 and Linked Birth/Infant Death Data, 2004

Much of the racial difference in infant mortality rates is due to disparate rates of PTB. In 2004, the infant mortality rate due to preterm-related causes for non-Hispanic Black infants was approximately 3.5 times greater than the rates for non-Hispanic Whites, American Indians, Hispanics, and Asians or Pacific Islanders (Table 3). Nearly one-half of Black infant deaths were due to preterm-related causes as compared to one-third or fewer of infant deaths for other racial and ethnic groups (Mathews & MacDorman, 2007).

[5]The literature on adverse birth outcomes inconsistently uses the terms African American, Black, and non-Hispanic Black to describe this population. Thus, in this dissertation, I use the terms Black and African American interchangeably.

Table 3. Preterm-related infant mortality rates per 1,000 live births, by race and Hispanic origin of mother

Maternal Race and Hispanic Origin	Preterm-related infant mortality rate
Non-Hispanic Black	6.29
Non-Hispanic White	1.82
Hispanic	1.85
American Indian or Alaska Native*	1.89
Asian or Pacific Islander	1.65
All races and origins	*2.48*

DATA SOURCE: National Center for Health Statistics, Linked Birth/Infant Death Data, 2004

In addition to the racial/ethnic disparities, rates of adverse birth outcomes tend to increase as maternal age at the time of birth approaches the extremes of the reproductive age range—i.e., below 15 years of age or over 40 years of age (Eura, Lindsay & Graves, 2002; NCHS, 2005) (Table 4). Researchers also have reported an interaction effect of race/ethnicity and maternal age on adverse birth outcomes. For example, Geronimus (1986) demonstrated that the risk of delivering a LBW infant declined between maternal ages of 15 and 29 for Whites while it increased for Black mothers of the same maternal ages, especially those living in low income areas.[6] Another study showed a U-shaped relationship between maternal age and LBW among Whites, with the youngest and oldest mothers being at higher risk than 25 – 29 year-olds; but Black 15 – 19 year-olds had significantly lower risks of delivering LBW infants than did Black women aged 25 – 29 (Reichman & Pagnini, 1997).

[6]Another study found that the risk of LBW rose more quickly with maternal age for socioeconomically disadvantaged women, regardless of race/ethnicity—a finding which the authors say suggests that the steep increase in risk of LBW with increasing maternal age for Black women may be explained by the high prevalence of disadvantage in this population (Rich-Edwards et al., 2003). Yet other findings discussed in Section 2.2.4.2 refute this assertion.

Other studies have shown that the risk of delivering a LBW infant begins to rise at age 30 for White women but at age 20 for Black women (Ventura et al., 2000; Rich-Edwards et al., 2003; Rauh, Andrews, & Garfinkel, 2001; Schempf et al., 2007; Reichman & Pagnini, 1997). Taken together with recent national prevalence data (Table 4 and Figure 1), these findings demonstrate the widening of racial/ethnic differences in LBW with increasing maternal age, beginning in women's late teens and their early 20s.[7]

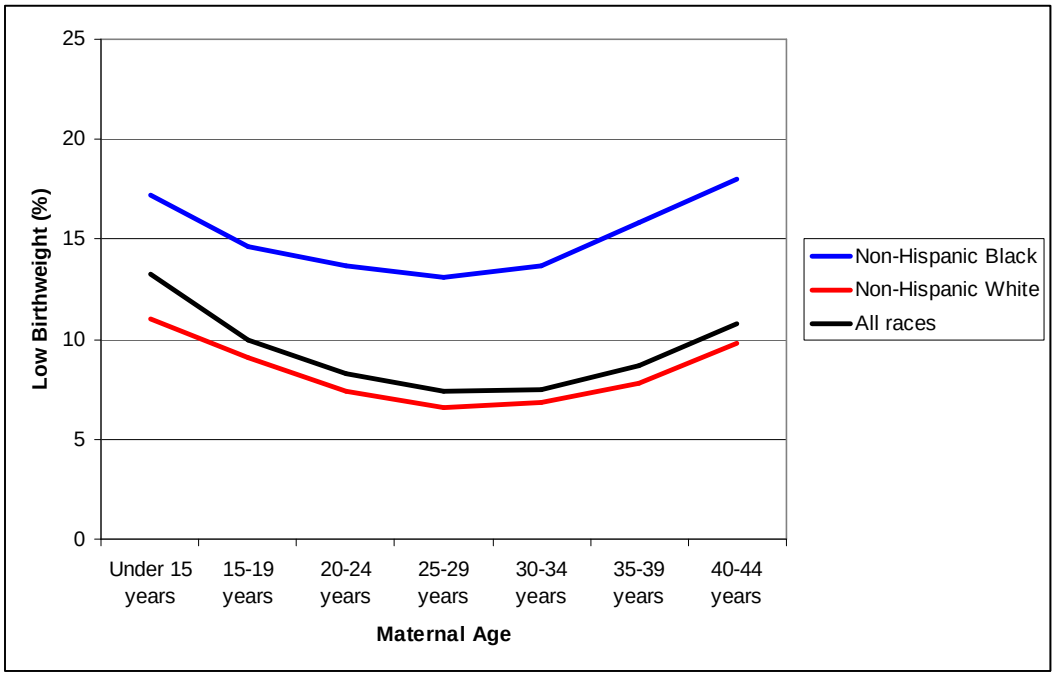
Table 4. Percentage low birthweight, by maternal age and race

	Maternal Race and Hispanic Origin			
Maternal Age	**All races**	**Non-Hispanic White**	**Non-Hispanic Black**	***Black/White Ratio***
Under 15 years	13.3	11.0	17.2	***1.56***
15-19 years	10.0	9.1	14.6	***1.60***
20-24 years	8.3	7.4	13.7	***1.85***
25-29 years	7.4	6.6	13.1	***1.98***
30-34 years	7.5	6.8	13.7	***2.01***
35-39 years	8.7	7.8	15.8	***2.02***
40-44 years	10.8	9.8	18.0	***1.84***
All ages	*8.2*	*7.3*	*14.0*	*1.92*

DATA SOURCE: National Center for Health Statistics, Births: Final Data, 2005

[7]It is important to note that the racial/ethnic disparities in adverse birth outcomes, and the relationship between maternal age and these outcomes, are *not* due to the increased number of births to African American women, in general, and African American teens, in particular (Geronimus, 1986). For example, Geronimus (1986) showed that if no teen pregnancies had occurred in her study, the racial disparity in rates of neonatal mortality (which is mediated by PTB and/or LBW) would have dropped only trivially.

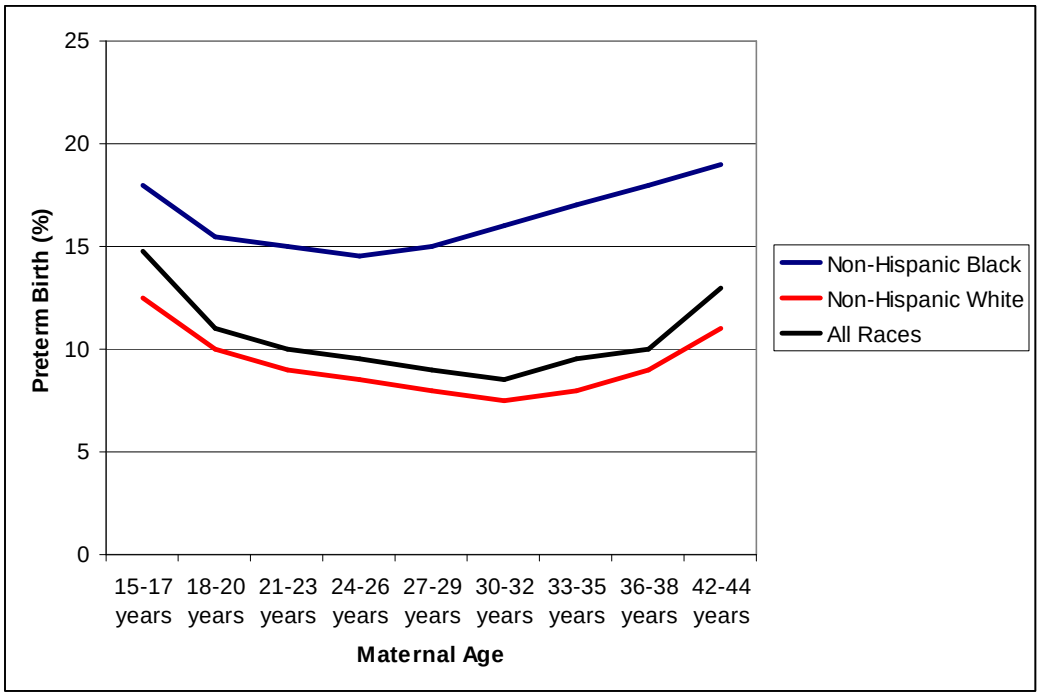
Figure 1. Percentage low birthweight, by maternal age and race



DATA SOURCE: National Center for Health Statistics, Births: Final Data, 2005

Fewer studies of the interaction effect of maternal age and race on PTB have been conducted. However, data from the 1998 to 2000 U.S. birth cohorts showed maternal age-race interactions for PTB to be similar to those for LBW—i.e., that the PTB rate begins to rise at a younger age for non-Hispanic African Americans than for non-Hispanic Whites, and the slope of the increase in PTB with increasing age is greater for African Americans than for Whites (Figure 2). The IOM (2006) has recently acknowledged the need for further studies of the interaction effect of maternal age and race on PTB, especially studies that use longitudinal data to control for potential cohort effects.

Figure 2. Percentage preterm, by maternal age and race



DATA SOURCE: National Center for Health Statistics, Births: Final Data, 2005

2.2 Factors Associated with Adverse Birth Outcomes

The literature on adverse birth outcomes reveals a number of known and suspected risk factors. According to Hogan (2004), in order for a factor to be considered a contributor to racial disparities in adverse birth outcomes, it (1) must be a risk factor for the outcomes or affect the distribution of another known risk factor and (2) should be more prevalent in the vulnerable group. Thus, below I discuss the extent to which the known and suspected risk factors of adverse birth outcomes, ordered from most proximal to most distal, meet Hogan's criteria. Throughout this review, I focus solely on maternal characteristics for two main reasons. First, despite the paternal contribution of genetic material to the developing fetus, the literature on paternal contributions to adverse birth outcomes is sparse. Second, several of the studies that have examined both maternal and paternal contributions to birth

outcomes suggest that maternal characteristics are stronger predictors than paternal characteristics. For example, in two separate, albeit dated, studies, the maternal race of mixed race infants was a stronger predictor of LBW than was the paternal race (Miagone et al., 1991; Collins & David, 1993). Specifically, infants with African American mothers were more likely to be LBW than infants with White mothers, regardless of the father's race.

2.2.1 Biological Factors

For the purposes of this review, biological factors are defined as physiological variables and biomedically-defined illnesses that have potential health and developmental consequences for both mothers and their fetuses. These include: (1) genetics; (2) pregnancy characteristics; and (3) maternal health status.

2.2.1.1 Genetics: Researchers long have hypothesized genetic reasons for racial disparities in adverse birth outcomes based on the observation that these outcomes tend to be repeated in subsequent births to the same women, within and across family generations, and within certain racial/ethnic groups but not others (Dizon-Townson, 2001; DeFranco, Teramo, & Muglia, 2007; Adams & Eschenbach, 2004; Mattison et al., 2001; Porter et al. 1997; Adams et al. 2000; Foster et al., 2000). Race-based genetic hypotheses are further supported by data showing distinct gestational age distributions for African Americans and Whites (Papiernik, Alexander & Paneth, 1990; Patel et al., 2004). However, other studies involving foreign-born and U.S.-born women with the same continental ancestry suggest that racial/ethnic differences in gestational age distributions are not genetic (Kramer et al.,

2006).[8,9] Moreover, studies of possible genetic bases for adverse birth outcomes that involve examining repeat occurrences within and across generations could be indicative of continued or shared exposure to the same social and environmental risks (Fiscella, 2005; Nesin, 2007; Kaufman, Geronimus, & James, 2007)). In addition, at least one study suggests that adverse birth outcomes are less likely to be repeated in subsequent generations of African Americans than in subsequent generations of Whites (Conley & Bennett, 2000). Thus, the contribution of genetics to racial disparities in adverse birth outcomes remains questionable.

2.2.1.2 Pregnancy Characteristics: Coincident with the rise in use of assisted reproductive technology, one of the most widely recognized correlates of adverse birth outcomes in recent literature is multiple gestations, also known as multifetal pregnancies or plural births (Lee, Cleary-Goldman, & d'Alton, 2006; Russell et al., 2002; Mattison et al., 2001). Multifetal pregnancies account for only 3% of all pregnancies but 15-20% of all preterm births (Mattison et al., 2001; Goldenberg et al., 2008). Studies have shown that Blacks have a higher twin birth rate than Whites but that racial disparities in adverse birth outcomes are mimicked in twin and other multifetal pregnancies (Zach, Prahmanik, & Ford, 2007; Luke et al., 2005). For example, in 2004, the infant mortality rate for plural births was 55.35 deaths per 1,000 live births among Blacks as compared to 25.77 among Whites, 28.90 among Hispanics, 37.0 among American Indians, and 23.13 among Asians or Pacific

[8]In addition, it is now widely acknowledged that genetic differences between races are minimal (Cooper & Freeman, 1999; Jorde & Wooding, 2004) and that genetic variation is greater within races than between them (Cooper & David, 1986).

[9]Further discussion of continental or national ancestry and place of birth can be found in Section 2.2.4.4.

Islanders (Mathews & MacDorman, 2007). The disparities between these rates are slightly smaller than the overall infant mortality rate comparisons presented in Section 2.1; however, the racial/ethnic patterns are similar.

Two other characteristics of pregnancies that may lead to adverse birth outcomes are short interpregnancy interval (Smith, Pell, & Dobbie, 2003; DeFranco, Teramo, & Muglia, 2007) and parity (Helsel, Petitti, & Kunstadter, 1992). The contribution of these factors to racial disparities in birth outcomes, however, has not been firmly established.

2.2.1.3 Maternal Health Status: Studies have linked several health conditions to adverse birth outcomes, including: hypertension, diabetes, genitourinary tract infections (e.g., bacterial vaginosis), periodontal infections, and anemia (Vettore et al., 2006; Kieffer et al., 1998; Culhane et al., 2001; Offenbacher, Katz, & Fertik, 1996; Jeffcoat et al., 2001; Rosenberg et al., 2005). Some of these studies, however, have methodological limitations, including unreliable exposure measures (Vettore et al., 2006). And only one study has attempted to determine the extent to which the timing of exposure impacts the birth outcome. Specifically, Haas and colleagues (2005) tested the hypothesis that health risks experienced one month before conception would be associated with a woman's risk of PTB, independent of risk factors that occur during pregnancy, and found some support for this hypothesis.

Nevertheless, studies have consistently shown that pregnant African American women have a higher prevalence of hypertension, anemia, and genitourinary tract infections than pregnant women of other races (Goldenberg et al., 1995; Culhane et al., 2002; D'Angelo et al., 2007). At least one study found that the associations between these

conditions and adverse birth outcomes were stronger among African American women than White women (Meis et al., 1995). We do not know why African American women have higher rates of these conditions during pregnancy than other women do, although researchers speculate that the experience of more psychosocial stress may be one explanation (Culhane et al., 2002; Wadhwa et al., 2001a; Hilmert et al., 2008). Nevertheless, these conditions do not fully account for racial/ethnic disparities in adverse birth outcomes (Kramer, 1995; Berg, Wilcox, & d'Almada, 2001; Mattison et al., 2001).

2.2.2 Behavioral Factors

Known and suspected behavioral risk factors for adverse birth outcomes include engaging in the following behaviors during pregnancy: tobacco use; alcohol consumption; illicit drug use; obtaining late, no, or inadequate prenatal care; poor nutrition, including poor nutrient intake or less than ideal weight gain during pregnancy; and experiencing intimate partner violence (Nothnagle et al., 2000; Berg, Wilcox, & d'Almada, 2001; Siega-Riz et al., 2001; Moore & Zaccaro, 2000; Schieve et al., 2000; Baeton, Bukusi, & Lambe, 2001; Janssen et al., 2003; Ahern et al., 2003; Vintzileos et al., 2002; Cnattingius, 2004; Schempf, 2007). Racial/ethnic differences in these behavioral risk factors vary by factor. For example, White women have a higher prevalence of tobacco and alcohol use during, after, and immediately prior to pregnancy than women of other races, while the prevalence of intimate partner violence prior to pregnancy is higher among African American women than among women of other races (Berg, Wilcox, & d'Almada, 2001; Beck et al., 1999; Lu et al., 2005; D'Angelo et al., 2007). African American women are also more likely to have less than ideal weight gain during pregnancy and less likely to initiate prenatal care during the first

trimester (CDC, 2009; Martin et al., 2007). Yet the behavioral factors that are more prevalent among African American women do not fully explain racial/ethnic disparities in adverse birth outcomes (Berg, Wilcox, & d'Almada, 2001; Goldenberg et al., 1996; Martin et al., 2007).

2.2.3 Psychosocial Factors

Psychosocial factors pertain to one's psychological status in the context of one's social environment and are presumed to have both conscious and unconscious physiological effects (Daniel, Moore, & Kestens, 2008). Three psychosocial factors have been discussed most frequently in the literature on adverse birth outcomes: (1) depression; (2) psychological stress; and (3) stress accumulation.

2.2.3.1. Depression: A recent review of the literature suggests that prenatal depression is associated with an increased risk for adverse birth outcomes (Field, Diego, & Hernandez-Reif, 2006), although a growing number of studies have found no such association (e.g., Dole et al., 2003, 2004; Suri et al., 2007). A small number of studies with samples limited to African American women have found a positive association (Orr, James, & Blackmore-Prince, 2002; Orr & Miller, 1995). Moreover, researchers have found that African American women report higher levels of depression than White women, in general, during pregnancy, and during the prepregnancy period (Rickert, Weimann, & Berenson, 2000; Orr, Blazer, & James, 2006; Dole et al., 2004; D'Angelo et al., 2007; Gavin et al., 2009). Yet, the extent to which racial/ethnic differences in maternal depression explain racial disparities in adverse birth outcomes has not been determined (IOM, 2006). In the only study of its kind, Gavin and colleagues (2009) found that prepregnancy depression was

associated with PTB among Black and White mothers but did not mediate the association between race and PTB.

2.2.3.2. Psychological Stress: Historically, studies of the relationship between psychological stress, social support (a moderator of psychological stress), and adverse birth outcomes have not produced consistent results and, when present, positive associations have been small (e.g., Lu & Chen, 2004; Goldenberg et al., 1996; Copper et al., 1996; Lobel, 1993; Hoffman & Hatch, 1996). The evidence for the influence of psychosocial stress on racial disparities in adverse birth outcomes has been no more consistent or positive (Lu & Chen, 2004).

Disagreement about the conceptualization and operationalization of stress pervades this literature. For example, Austin and Leader (2000) have noted researchers' failure to examine the cumulative effect of the number, severity, and chronicity of stressors on birth outcomes—a problem related to the fact that stress is often assessed at only one time point during pregnancy (O'Campo & Schempf, 2005; Gennaro & Hennessy, 2003). In addition, some researchers argue that traditional conceptualizations of stress are decontextualized—e.g., they neglect to consider how the distribution of stressors is shaped by social, political, and economic conditions (Lu & Chen, 2004; Krieger, 2001; Dressler, 1991). Others note that the common use of single measures of stress or assessment of exposure to only one type of stressor may underestimate exposure (Hogue , Hoffman, & Hatch, 2001). These problems have made it difficult to identify exactly which stressors, experienced at what time, by which women, and under what conditions, are most likely to lead to adverse birth outcomes (Gennaro & Hennessy, 2003).

Although stress has not been established firmly as a risk factor in previous studies of adverse birth outcomes, further research on the relationship between stress and birth outcomes is important because of its biological plausibility. Specifically, the body's response to stress involves the activation of stress hormones, including corticotropin-releasing hormone (CRH), which may initiate PTB (Ruiz, Fullerton, & Dudley, 2003; Hobel, Dunkel-Schetter & Roesch, 1998; Hobel et al., 1999; Rich-Edwards et al., 2001). Chronic exposure to stress also may produce adverse birth outcomes through its immunosuppressant and vascular effects (e.g., increasing susceptibility to infection and likelihood of hypertension) (Wadhwa et al., 2001a, 2001b; Culhane et al., 2002). Chronic stress also leads to increases in allostatic load (i.e., the body's inability to achieve homeostasis in the presence of chronic stress), which causes persistent immune, vascular, and neuroendocrine activation (McEwen & Seeman, 1999). This persistent activation results in physiologic "wear and tear" (Seeman et al., 2001) and may be responsible for racial differences in physiological and metabolic variables that precipitate ill health (Bruner & Marmot, 2001) and adverse birth outcomes (Geronimus et al., 2006).

2.2.3.3. Stress Accumulation: An alternate approach to traditional conceptualizations of point-in-time and acute stress is to consider the accumulation of chronic stress and its effects over time (Hogue & Bremner, 2005). One example is found in the literature on the "weathering" hypothesis (Geronimus, 1992, 1996, 2001) and "stress age" (Hogue & Bremner, 2005). As noted in Section 2.1, studies have shown that birth outcomes begin to gradually worsen among African American women at an earlier age than they do for White women, so that outcomes of African American and White women at the same age

are not comparable. In particular, Geronimus (1986, 2001) found that White teenage mothers, who have the worst birth outcomes among Whites and are disproportionately poor, and Black teenage mothers have better birth outcomes than Black mothers in their 20s and early 30s. These findings led Geronimus (1992, 2001) to propose the "weathering" hypothesis, which suggests that a woman's health reflects the cumulative impact of social environmental stressors to which she is exposed daily. According to this hypothesis, the presence of racial disparities in adverse birth outcomes and the widening of these disparities with increasing age may be due to the fact that African American women accumulate the effects of chronic stressful life circumstances and experiences (such as ongoing racism) earlier, and more over time, than do White women (Geronimus, 1992, 1996; Hogue & Bremner, 2005). As a result of this accumulation, African American women may experience an accelerated aging process (i.e., "weathering" or "stress age") and progressive health decline. Evidence supporting the weathering hypothesis with respect to adverse birth outcomes remains inconclusive, however, as does determination of the mechanisms underlying "weathering"(IOM, 2006). Allostatic load (discussed in 2.3.3.2) is one potential mechanism (Geronimus et al., 2006).

The "weathering" hypothesis can be accommodated by a lifecourse perspective on racial/ethnic disparities in adverse birth outcomes. As I discuss in Chapter 3, a lifecourse perspective refers to the idea that risk and protective factors cluster cross-sectionally and accumulate or interact longitudinally to impact current, future, and intergenerational health (Kuh & Ben-Shlomo, 2004; Blane, 1999; Pollitt, Rose, & Kaufman, 2005). Proponents of the lifecourse perspective posit that exposure to both risk and protective factors over the

lifecourse will vary between individuals, and the differences between these "life exposure trajectories" are what manifest as health disparities (Hertzman, 2004). Recently, Lu and Halfon (2003) reviewed studies of disparities in adverse birth outcomes between African Americans and Whites, and proposed the use of a lifecourse perspective in future studies. To date, only one lifecourse study of racial disparities in adverse birth outcomes has appeared in the U.S. published literature since Lu and Halfon published their 2003 review[10] and it only allows for speculation about the effect of "weathering". Colen and colleagues (2006) estimated the extent to which intergenerational upward socioeconomic mobility might result in a lower probability of giving birth to a LBW baby. They found that race moderated the relationship; upward socioeconomic mobility resulted in a significantly lower probability of LBW for Whites and a non-statistically significant and weaker reduction in the probability of LBW for Blacks—a difference that was not explained by proximate risk factors for LBW.

2.2.4 Social Environmental Conditions

Another approach to conceptualizing stress that should be considered in studies of racial disparities entails considering the systemic form of social environmental stress that is specific to racial group experiences and reflects inequalities in society, rather than the random stress in traditional stress research which operates similarly across groups (Daniel et al., 1999). Below I describe some of the social environmental conditions that may contribute to the differential distribution of systemic stress across racial/ethnic lines and to

[10]In a previous study, it was found that racial disparities in LBW and PTB persisted among women experiencing persistent socioeconomic advantage across the lifecourse (Foster et al., 2000). In a recent Swedish study, Gisselmann (2006) found that maternal childhood and adulthood social class are both independently associated with inequalities in LBW, but that the influence of adult class was greater than that of childhood class.

racial disparities in adverse birth outcomes: (1) racism; (2) socioeconomic status; (3) place; and (4) migration.

2.2.4.1. Racism: Some researchers have argued that previous studies of psychosocial stress failed to explain racial disparities in adverse birth outcomes because the stress scales used in those studies did not adequately capture the life experiences of African American women, especially racism-associated stress (McLean et al., 1993; Hoffman & Hatch, 1996; Jackson et al., 2001; Hogue & Bremner, 2005). These claims resulted in several studies of the relationship between adverse birth outcomes and racism or racial discrimination. All such studies found an association between self-reported experiences of discrimination or perceived racism and adverse birth outcomes (Mustillo et al., 2004; Rosenberg et al., 2002; Collins et al., 2004; Dole et al., 2003, 2004). The association was stronger for women with lower levels of education in one of the studies (Rosenberg et al., 2002), while it was stronger for college-educated women in another one of the studies (Collins et al., 2004).

Instead of focusing on individual-level perceptions, a systemic stress view of racism would involve examining the social processes that cause racial/ethnic groups to end up occupying more or less favorable living conditions, and how the unequal distribution of risks, resources, and opportunities impacts health (Daniel & Linder, 2002). An example of this approach can be found in the qualitative literature on the intersection of race, class, and gender in the lives of pregnant African American women. In an ethnographic study of the social context of reproduction in Harlem, Mullings and Wali (2001) identified many stressful living conditions related to the effects of racism, in general, and gendered racism, in

particular, on the environment, housing, work, and social service delivery. They argued that these conditions, when combined with women's attempts to cope with them and modify their quality of life, could serve as catalysts for adverse birth outcomes. Jackson and colleagues (2001) reported similar findings from a qualitative study of college-educated African American women in Atlanta. To date, however, the extent to which racism or gendered racism explains racial disparities in adverse birth outcomes has not been empirically determined.

2.2.4.2. Socioeconomic Status: As with nearly all other health outcomes, a social gradient in adverse birth outcomes is evident by social class: risk decreases as SES increases (Martin et al., 2007). This is true for women of all racial/ethnic groups. At every level of SES, however, African American women have higher rates of adverse birth outcomes than White women (Pamuk, 1998). In addition, the difference in rates of adverse birth outcomes between African American and White women widens as their education increases (Kleinman & Kessel, 1987; Din-Dzietham & Hertz-Picciotto, 1998; Williams, 2002). Indeed, White women who do not complete high school have lower rates of adverse birth outcomes than African American women who have graduated from college (Table 5), suggesting that increases in SES do not offer the same degree of protection against adverse birth outcomes for African Americans as they do for Whites (CDC, 2005; Williams, 2002; Pamuk, 1998; Schoendorf et al., 1992; McGrady et al., 1992). Controlling for SES in studies of racial disparities in adverse birth outcomes does not eliminate the gap (Krieger et al., 1993). Nor do disparities in SES between African American and White women fully account for the gap between these two groups in their rates of adverse birth outcomes (Hummer, 1993).

Table 5. Infant Mortality Rates by Maternal Education and Race, Mothers Aged 20+

	Maternal Race		
Maternal Education	**White**	**Black**	***Black/White Ratio*****
<12 years	9.0	15.2	***1.69***
12 years	6.6	13.6	***2.06***
>12 years*	4.1	11.9	***2.90***

DATA SOURCE: National Center for Health Statistics, Linked Birth/Infant Death Data, 2002

* Previous studies have divided the ">12 years" category into two categories—13-15 years and 16+ years—and also found an increase in the black/white ratio as education increases (e.g., Pamuk, 1998)

** Data on other adverse birth outcomes, such as PTB, mirror these findings but with slightly less dramatic increases in the black/white ratio with increasing education (e.g., Pamuk, 1998).

On the other hand, a lack of consensus regarding the appropriate conceptual and operational definitions of SES exists. Critics have long argued that income and education indicators do not fully capture SES, and that the actual socioeconomic conditions of African Americans and Whites at the same income or education level are not comparable (Kaufman, Cooper, & McGee, 1997; Wise, 1993; Conley, 1999; Lynch & Kaplan, 2000). This incomparability may be due to the negative impact of racial discrimination on the economic return on education among African Americans (Wise & Pursley, 1992; Krieger et al,. 1993; Williams, 1998). In addition, purchasing power may differ between racial/ethnic groups because Whites have substantially more wealth than African Americans at the same income level (Oliver & Shapiro, 1997; Conley, 1999; Lynch & Kaplan, 2000), resulting in disparate living conditions (Williams, 1998). Thus, traditional measures of SES may underestimate racial/ethnic differences,[11] biasing the results of studies that attempt to examine their association with racial disparities in adverse birth outcomes.

[11]This problem is commonly referred to as "residual confounding" (Kaufman, Cooper, & McGee, 1997).

2.2.6.3. Place: Living in neighborhoods characterized by deprivation, physical disorder, and social disorder has been identified as a chronic stressor, and the literature on the effects of these place-based stressors on health is burgeoning (Culhane & Elo, 2005; Kawachi & Berkman, 2003; Tunstall, Shaw, & Darling, 2004). The effects of neighborhood socioeconomic disadvantage on LBW have been particularly well-documented, demonstrating small to moderate effect sizes (Farley et al., 2006; Ahern et al., 2003; Sellstrom & Bremberg, 2006). Similar findings have been reported in the few studies of neighborhood disadvantage and PTB (Pickett et al., 2002; O'Campo et al., 2008; Messer et al., 2008). Other studies have pointed to the potential association between birth outcomes and other neighborhood-level factors such as lack of social support, high crime rates or low perceived safety, homelessness, and poor air quality (Buka et al., 2003; Culhane et al., 2002; Messer et al, 2006a; Maisonet et al., 2004; Collins & David, 1997; Collins, 1998; Elo, Rodriguez, & Lee, 2001; Morenoff, 2003). Some studies suggest the possibility that these neighborhood-level (or "contextual") factors may explain or mediate the relationship between neighborhood disadvantage and adverse birth outcomes (Masi et al., 2007), although empirical evidence of this assertion with respect to any health outcome is scant (Franzini et al., 2005).

While at least one study has found smaller effect estimates for African American women than Whites (Messer et al., 2008), a number of these studies have linked neighborhood disadvantage to adverse birth outcomes among African Americans but not (or less strongly) among Whites (Buka et al., 2003; O'Campo et al., 1997, 2008; Rauh, Andrews, & Garfinkel, 2001; Pearl, Braveman, & Abrams, 2001). It is well-known that, compared to White women, African American women are more likely to live in

neighborhoods disproportionately affected by adverse conditions (Wilson, 1987; Massey & Denton, 1993; Williams & Collins, 2001). This difference has been attributed to racial residential segregation—i.e., the physical separation of racial/ethnic groups in residential contexts (Williams, 1996; Massey & Denton, 1993). Racial residential segregation is a manifestation of historical, social, economic, and political processes, including institutionalized racism, which has resulted in constrained life chances, differential housing, educational, and employment opportunities, adverse neighborhood conditions,[12] and decreased quality of life for African Americans residing in these areas (Williams & Collins, 2001; Williams, 1998; Wallace, 1999; Grady, 2006; Massey & Denton, 1993). Studies of racial disparities in adverse birth outcomes and residential segregation may have the potential to produce important findings about the role of place.

Past studies have found that African American infant mortality and LBW are higher in cities with more residential segregation, independent of the compositional effect of household poverty (Polednak, 1997; LaVeist, 1993; Ellen, 2000). In more recent studies, the isolation of African Americans within predominantly African American communities was associated with lower birth weights and higher rates of PTB (Bell et al., 2006; Grady, 2006). On the other hand, the clustering of adjacent, predominantly African American neighborhoods was associated with better birth outcomes (Bell et al., 2006). Pickett and colleagues (2005) found that higher neighborhood SES also protected against PTB for African American women living in those communities. Another study, however, suggests that this protection may not be as pronounced in less severely segregated environments

[12]It should be noted that the racial difference in neighborhood quality persists at all SES levels. SES mediates part, but not all, of the association between segregation and health (Acevedo-Garcia & Lochner, 2003).

(Vinikoor et al., 2008). These findings are consistent with the assertions made by social scientists that racial residential segregation may offer protection, if, for example, high degrees of social homogeneity leads to greater social cohesion within the community, while it may be detrimental if it isolates communities and limits the availability of social and economic resources that exist in other non-segregated communities.

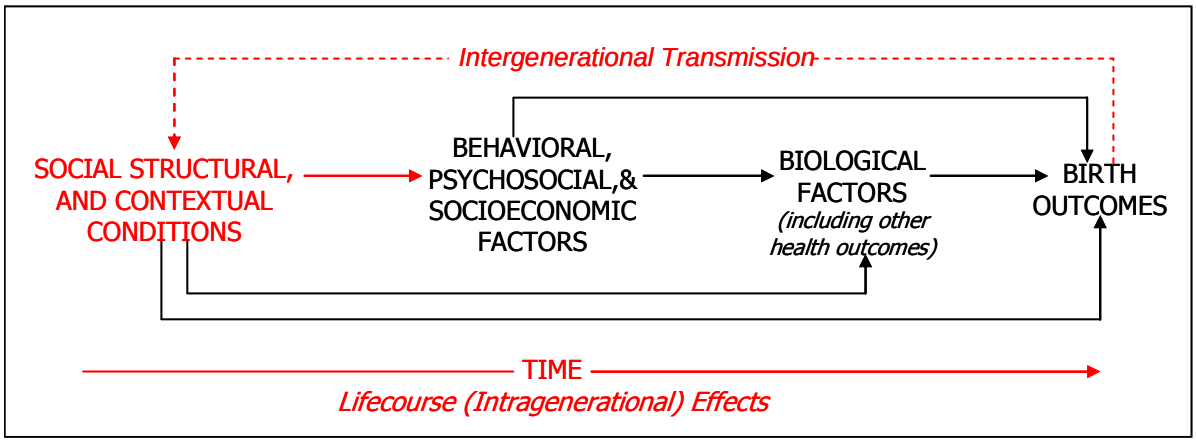
2.2.4.4. Migration: Social environmental explanations for racial disparities in birth outcomes also are implicated in studies involving immigrants (Tunstall, Shaw, & Darling, 2004).[13] Most notably, the Latino infant mortality paradox refers to the long-standing paradoxical finding that despite lower SES, Latinas generally have good birth outcomes. Furthermore, the strength of the association between being Latina and having normal birth outcomes is greater for foreign-born women than for those born in the U.S., despite the fact that U.S.-born Latinas may have fewer sociodemographic risks than their foreign counterparts (Lara et al., 2005; Gould et al., 2003; Landale, Oropesa, & Gorman, 2000; Acevedo-Garcia, Soobader, & Berkman, 2007; Madan et al., 2006). Similarly, studies have found that foreign-born Black women have lower risks for PTB, LBW, and infant mortality than do U.S.-born Black women (Baker & Hellerstedt, 2006; Forna et al., 2003; Howard et al., 2006; Mathews & MacDorman, 2007). For example, one study found that the birth weights of African American infants whose mothers were born in Africa resembled those of White infants born to U.S.-born mothers (David & Collins, 1997). In another study, African American infants with Caribbean-born mothers weighed more than African American infants with U.S. born mothers, regardless of risk status (Pallotto, Collins, & David, 2000).

[13]To the extent that concepts of "race/ethnicity" denote continental or national ancestry, these studies also dispel race-based genetic hypotheses regarding racial disparities in adverse birth outcomes (see Section 2.2.2.1).

2.3 Synthesis of the Literature

Based on the survey of the literature presented in the preceding sections, the need for a multilevel, multifactorial, and longitudinal conceptual framework for understanding racial disparities in birth outcomes is compelling. In addition to accounting for proximate determinants of birth outcomes, such a model would ideally take into account social, structural, and residential arrangements that comprise the context in which individual-level factors in women's lives are embedded. The literature reviewed also suggests the need to consider the multiple ways in which health is shaped by structural and contextual factors over the lifecourse—not just during pregnancy or immediately prior to conception. To be comprehensive, the model also should specify the process by which these factors are translated into racial/ethnic differences in birth outcomes through behavioral, socioeconomic, psychosocial, and biological (non-genetic) pathways. The conceptual framework in Figure 3 depicts this synthesis.

Figure 3. Overarching Conceptual Framework



Note: Red text and arrows denote factors and pathways that are understudied or under-theorized.

2.3.1 Limitations and Gaps

This overarching framework, while not an analytical model, differs from traditional epidemiological models of adverse birth outcomes by: (1) displaying an entire "web of causation", including the "spider" (Krieger, 1994); (2) incorporating upstream social and contextual variables that may influence more proximate determinants of risk for adverse birth outcomes; and (3) considering the influence of these exposures over the lifecourse. As the literature review reveals, the prevailing approach to studying adverse birth outcomes in the epidemiological literature has involved the frequent use of a single risk factor approach and a focus on decontextualized exposures to biological, behavioral, and psychosocial risk factors. These more proximate determinants of adverse birth outcomes are inadequate for understanding racial/ethnic disparities on a population level (Wise, 1993; Rose, 1992), although they may mediate the relationship between longer-term exposures to more distal factors (e.g., social environmental stressors) and adverse birth outcomes (Kramer et al., 2001).

Moreover, data on determinants of adverse birth outcomes have primarily been collected during the prenatal period about that period, or about the period immediately preceding conception (Wise, 1993; Misra, Grason, & Weisman, 2000). This focus on temporally proximate determinants and outcomes limits our ability to determine causation (Savitz, Dole, & Herring, 2006). Such cross-sectional "snapshots" also ignore exposures that may have preceded pregnancy, or even the whole childbearing period, and been crucial in precipitating adverse birth events (Lu & Halfon, 2003; Gisselmann, 2006). A lifecourse perspective instead illuminates the pathways by which exposures to risk and protective

factors over the lifecourse can shape perinatal health, and the potential for intervening at any point in women's lives to prevent adverse birth events (Daniel, Moore, & Kestens, 2008; Lu & Halfon, 2003). One problem, however, is that lifecourse studies of birth outcomes are in their infancy; to date they have been limited to examining the intergenerational correspondence of birth outcomes, and the effects of intergenerational improvements in socioeconomic status on racial disparities in these outcomes (Colen et al., 2006; Foster et al., 2000; Collins, Wu, & David, 2002; Gisselmann, 2006). These studies capture little of women's exposure histories prior to their pregnancies. In particular, they overlook the lifecourse impacts of neighborhood- or community-level factors (Lu & Halfon, 2003), despite the fact that these factors may vary in their nature and intensity at different stages of the life span and may exert their greatest influence on health and development during early childhood and late adolescence (Massey & Denton, 1993).

Another common theme in the literature pertains to the conceptual, operational, and methodological shortcomings of research on some of the determinants that may hold the most promise for furthering our understanding of racial disparities in adverse birth outcomes—e.g., stress and SES. In future studies, stress must be contextualized within the social, political, and economic realities that create differential stress exposures (Krieger, 2001; Dressler, 1991). Using the notion of systemic stress offered by Daniel and his colleagues (1999) may assist this process. To improve our measures of stress, researchers also must take into account the chronicity and duration of stress exposure (Austin & Leader, 2000), and stressors that differentially impact African Americans, such as racism (and gendered racism) and its manifestations (e.g., residential segregation). With regard to SES,

future studies should consider using multiple indicators of social and economic conditions other than income and education (which may be poor indicators of the actual living conditions that distinguish the realities of African Americans' and Whites' lives). These indicators include material deprivation, housing instability, and area-level factors such as neighborhood disadvantage (Misra, O'Campo, & Strobino, 2001; Rich-Edwards, 2002). Including these variables could enhance our measurement of socioeconomic conditions and assist in minimizing the degree to which residual confounding of race/ethnicity by SES is a problem (O'Campo & Schempf, 2005).

Theoretical and methodological uncertainties afflict area-level studies, however. For example, questions remain about the proper scale and boundaries of places to study, the appropriate methods for disentangling context from composition and determining causation, and the best way to model individual-level effects (i.e., as confounders, mediators, or moderators of contextual effects) (Diez-Roux, 2001; Macintyre, Ellaway, & Cummins, 2002; Tunstall, Shaw, & Darling, 2004; Culhane & Elo, 2005; Daniel, Moore, & Kestens, 2008). In addition, researchers continue to search for the relative importance of (and nature of the relationship between) subjective appraisals, direct observation, and objective measures of neighborhood characteristics (Ingoldsby et al., 2006; Weden, Carpiano, & Robert, 2008; Wen, Hawkley, & Cacioppo, 2006; Bowling & Stafford, 2007; Laraia et al., 2006). Finally, the mechanisms by which neighborhood characteristics affect adverse birth and other health outcomes are unknown, although research usually posits or implicates psychosocial, socioeconomic, material, and behavioral risk pathways (e.g., Reagan & Salsberry, 2005; Matheson et al., 2006; Franzini et al., 2005; Wen, Hawkley, &

Cacioppo, 2006; Brewster, 1994; Browning & Cagney, 2002). Another possibility, as suggested by the concept of allostatic load and Geronimus' weathering hypothesis (McEwen & Seeman, 1999; Geronimus, 1992), is that contextual disadvantage experienced over the lifecourse may have physiological effects that lead to progressive health decline. Theory-informed studies combining contextual, social, psychosocial, behavioral, and biological factors into a single unified study are lacking, however.

2.4 Conclusion

While the influence of early life circumstances on adult health disparities has begun receiving more attention (e.g., Graham, 2002; Hertzman et al., 2001; Spencer, 2006; Kroenke, 2008), the relationship between adverse birth outcomes and early life exposure to social and contextual disadvantage remains understudied (Lu & Halfon, 2003). To fill this gap, studies of adverse birth outcomes should contextualize proximate determinants within the context of more distal factors at several levels of causation, and use a longer, earlier timeframe than just the prenatal period to test their hypotheses (Ashton, 2006; Strobino, Grason, & Minkovitz, 2002). Examining disadvantage at several explanatory levels and from a lifecourse perspective, including mediating mechanisms, will allow us to better understand both intergenerational transmission and intragenerational accumulation of disadvantage. Such a layered, longitudinal approach may also help better explain racial/ethnic disparities in adverse birth outcomes, especially in light of the racial/ethnic differences in maternal age trajectories associated with these outcomes (see Section 2.1).

CHAPTER 3

THEORETICAL FOUNDATION

As noted in the previous chapter, some of the understudied factors and pathways in research on adverse birth outcomes include the longitudinal effects of social structural and contextual conditions, and the mechanisms by which these effects are created and maintained. Support for including these factors in my study and the development of their conceptual definitions was derived from social ecological theories, lifecourse perspectives, intersectionality theory, and place- and health- relevant theories. In this chapter, I describe these theories and present the study's conceptual model, drawing from both the theories and the literature review to define each of my conceptual model's constructs and hypothesize the relationships between them. I conclude by outlining the study's research questions, generated from the conceptual model, and positing the hypotheses I tested.

3.1 Theoretical Support

Conducting research on the impact of social and contextual factors on racial disparities in birth outcomes requires us to move from biological, behavioral, and psychosocial explanatory models that do not attend to these upstream phenomena toward social theory-driven models (Frohlich et al., 2004). Many social theories, however, either do not offer specific enough constructs or do not have explicit applications to health.

Therefore, the theories discussed below are presented heuristically to assist in understanding the key constructs of my study and their hypothesized relationships with each other.

3.1.1. Social Ecological Frameworks

Social ecological frameworks, such as Krieger's ecosocial theory (2001), recognize several levels of influence on health that interact with each other reciprocally (Krieger, 2001; McLeroy et al., 1988; Sallis & Owen, 1997). They describe the simultaneous structuring of health by characteristics of the social structure, including social stratification processes, and by individual characteristics (Krieger, 2001). Some social ecological frameworks also consider the interaction of these factors at single points in time, e.g., within particular life stages, or over the entire lifecourse (McMichael, 1999). These frameworks, therefore, lend support to the multilevel and temporal foci of this study.

3.1.2. Lifecourse Perspectives

Lifecourse scholarship can largely be traced to Glen Elder's (1979) work on the changing historical and social contexts of lives and their consequences for human development and aging. Public health researchers recently have drawn on several of his lifecourse principles and concepts to advance a "lifecourse epidemiology" or lifecourse perspective on health. In particular, they have drawn on Elder's idea that transitions, turning points, and durations, embedded in social context, have implications for developmental trajectories (Elder & Johnson, 2003). In addition to Elder's work, they have drawn on the concept of cumulative advantage, which is traceable to Merton's (1973, 1988) work on the "Matthew effect" and has since then been invoked by lifecourse scholars to

explain inequality. Cumulative advantage (or disadvantage) with respect to key resources, such as wealth, is believed to result in diverging life trajectories (DiPrete & Eirich, 2006).

Lifecourse perspectives on health are premised on the idea that the body is a "register" for social experience and in this way tells the story of the past—not just for an individual's own lifetime, but also the lifetimes of the preceding generation (Kuh & Ben-Shlomo, 2004; Nguyen & Preshard, 2003). Specifically, these perspectives posit that risk and protective factors cluster cross-sectionally and accumulate, interact, or combine longitudinally to impact current, future, and intergenerational health (Kuh & Ben-Shlomo, 2004; Blane, 1999; Pollitt, Rose, & Kaufman, 2005). Towards this end, at least three lifecourse causal models have been proposed: (1) a latent effects, "biological chains of risk", or critical period model; (2) a cumulative or "accumulation of risk" model; and (3) a pathway or "social chains of risk" model (Pollitt, Rose, & Kaufman, 2005; Kuh et al., 2003; Hertzman, 2004). These models are not exclusive, but do have different implications for research and practice (Graham, 2002).

A latent effects model suggests that early exposures to risk and protective factors are associated with later health risk, regardless of intervening exposures. A cumulative model posits that exposures to risk and protective factors across the lifecourse combine to influence later health risk, producing a greater effect than would be produced by the same magnitude of exposure at just one point in the lifecourse. A pathway model is one in which early experiences place an individual on a certain 'life trajectory', eventually impacting later health. It also suggests that changes in the trajectory at any point in time (i.e., intervening exposures) may modify the effect (Pollitt, Rose, & Kaufman, 2005; Hertzman, 2004).

These lifecourse causal models are helpful for considering mechanisms by which both beneficial and harmful exposures over the lifecourse and across generations may affect the *development* of differential risk for adverse birth outcomes across racial/ethnic groups. To date, researchers have not determined which of these developmental processes underlies racial disparities in birth outcomes. I believed that testing at least one of these lifecourse models in my study was an important task.

3.1.3. Intersectionality Theory

The overarching framework in Figure 3 (Chapter 2) also points to the need for theorizing about social, structural, and contextual conditions that may influence racial disparities in birth outcomes. Among these conditions are the creation and maintenance of social inequalities due to race, class, and gender ideologies. Intersectionality theory may be useful for understanding the role these inequalities play in racial disparities in birth outcomes.

Intersectionality, a concept coined by Crenshaw (1989) and featured in the theoretical frameworks of Critical Race Theory (Delgado & Stefancic, 2001) and Black Feminist Thought (Collins, 2000), suggests that dimensions of social inequality—such as race and gender—vary as a function of each other, and are interconnected. With regard to African American women, the concept refers to this group's *simultaneous* positioning at the losing end of race, gender, and class hierarchies (Davis, 1981; King, 1998; Crenshaw, 1994).[14] This positioning is thought to operate at the level of institutions, and to structure lived

[14]While it can be argued that African American men fare worse in this country than any other group, data have shown that the combined effects of race and gender on class place African American women at the bottom of the social hierarchy. For example, African American women receive the lowest economic return on education compared to White women, White men, and African American men (Williams, 2002; King, 1998).

experiences by constraining resources, opportunities, and life chances at the individual level (Collins, 2000; Weber, 2006). Intersectionality theory, therefore, may help explain why African American women are more likely than White women to experience socioeconomic and material disadvantage.

Interest in applying intersectionality theory to the understanding of racial disparities in health is growing (e.g., Schulz & Mullings, 2006). Recently, the theory was invoked to explain why African American women inhabiting higher socioeconomic strata have a greater risk for stress-induced adverse birth outcomes than do White women in any other stratum. As Mullings and Wali (2001) observe:

> First, the consequences of race and gender—of being a black woman—contribute to the instability of class status…. Furthermore, race dilutes the protections of class. For example, middle stratum black women may have attained the achievements necessary for middle-class status, but they continue to suffer job and occupational discrimination; they are less likely to marry and more likely to become single heads of households because they too are subject to the shortage of "marriageable men" as a consequence of disproportionate unemployment and the prison-industrial complex. (p. 164)

My study followed from this line of reasoning by hypothesizing that the intersection between race and individual-level as well as contextual measures of disadvantage manifests itself in increased risk for adverse birth outcomes among African American women more so than it does for White women.

3.1.4. Place- and Health-Relevant Theories

The impact of social inequalities on birth outcomes also may be understood using theories that focus on the relevance of "place" to health disparities. Noting the paucity of such theories, Curtis and Rees Jones (1998) offer three place-relevant theoretical

frameworks. The first refers to the spatial patterning and differential distribution of environmental risk factors, such as air quality, crime, distribution of alcohol outlets, and sanitation of public spaces. The second framework treats place as a vehicle through which social structural and material factors (e.g., quality and affordability of housing, employment opportunities, access to health and social services, social welfare policies, and police protection) shape social relations. The third framework refers to the affective attachments to places that people who reside in them develop, or the symbolic meaning of places to its residents.

These three frameworks converge when one considers the spatial, social, and symbolic effects of racial residential segregation on the individuals who live in racially segregated neighborhoods. As noted in Chapter 2, the clustering, concentration, and isolation of African Americans produced by racial residential segregation often lead to social exclusion, constrained opportunities, limited resources, and neighborhood disadvantage, including neighborhood poverty and poor neighborhood quality (Massey & Denton, 1993; Williams & Collins, 2001). My study drew on the theoretical frameworks proposed by Curtis and Rees Jones (1998), viewing their assertions through the lens of racial residential segregation. The first and second of their frameworks—i.e., the spatial patterning of health-relevant risks, resources, and social relationships—supported the inclusion of social and contextual conditions in the study and assisted in conceptually defining those conditions. The third framework—i.e., the symbolic meanings of place—supported the use of both subjective and objective measures.

3.2. Conceptual Model

The theoretical frameworks presented in this chapter helped me expand upon the overarching framework in Figure 3 (Chapter 2) to develop a conceptual model from which research questions and associated hypotheses for my study could be generated. This conceptual model is presented in Figure 4 (page 42). Consistent with social ecological frameworks, early life and young adult circumstances are represented by variables at different levels of aggregation (i.e., neighborhood, family, and individual). Among them are factors that may result from gendered racism and other factors that can lead to the spatial patterning of risks, resources, and social relations, as suggested by intersectionality and place theories. Drawing from lifecourse theories, the model also depicts the potential for latent, pathway, and cumulative effects of childhood exposures to individual, social, and contextual risks on subsequent birth outcomes. In particular, the model in Figure 4 suggests that birth outcomes may be shaped by one of the following eight pathways:

Pathway 1: Maternal childhood circumstances directly influence infant birth outcomes *(path a)*

Pathway 2: Maternal childhood circumstances influence maternal childhood health and development, which in turn influence infant birth outcomes *(paths b – j)*

Pathway 3: Maternal childhood circumstances influence maternal childhood health and development, which in turn influence maternal young adult circumstances that affect infant birth outcomes *(paths b-i-f)*

Pathway 4: Maternal childhood circumstances indirectly influence infant birth outcomes through their influence on maternal young adult circumstances *(paths e – f)*

Pathway 5: Maternal childhood circumstances influence maternal childhood health and development, which in turn influence maternal young

adult health and developmental characteristics that impact infant birth outcomes *(paths b – c – d)*

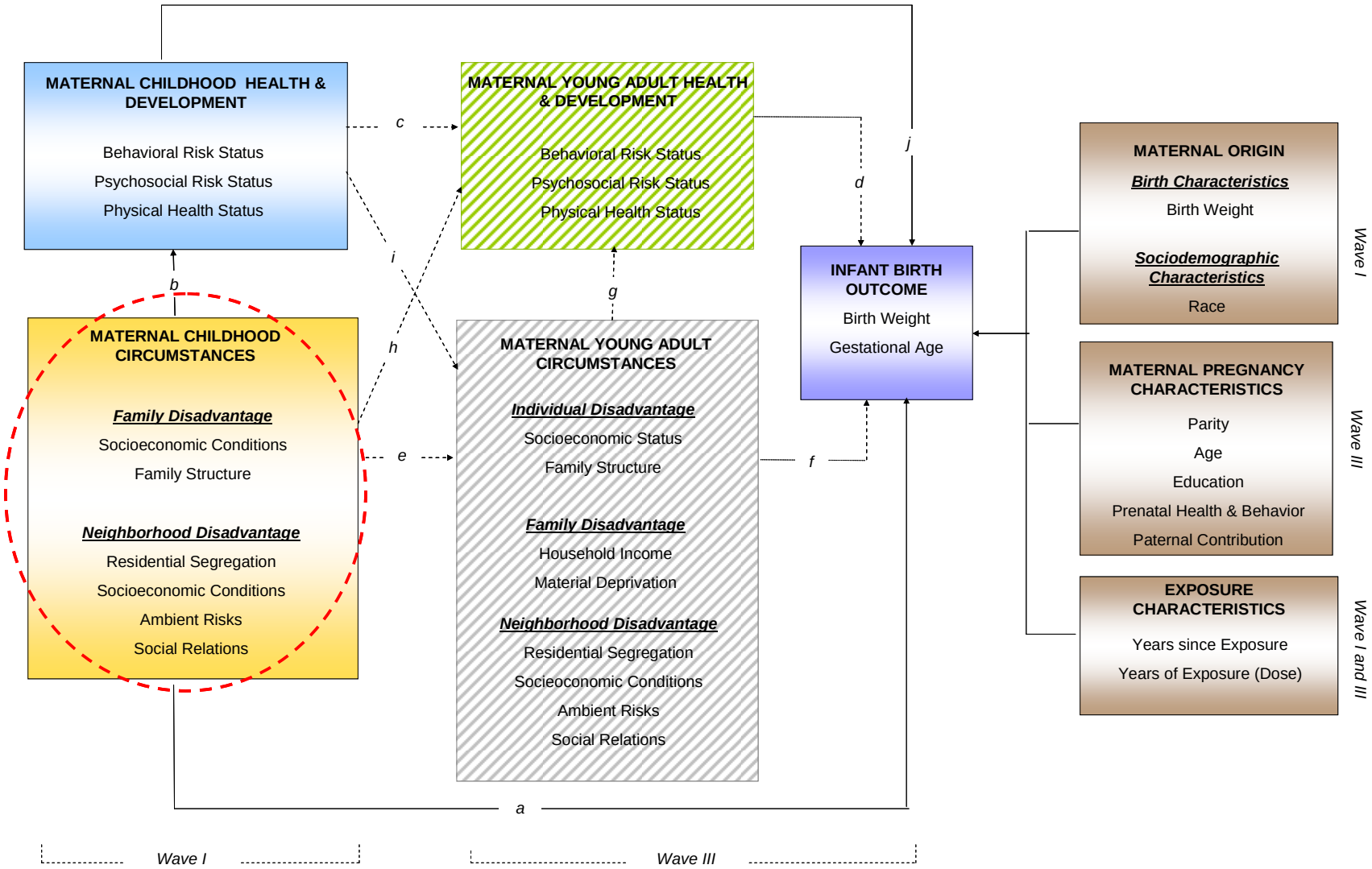
Pathway 6: Maternal childhood circumstances influence maternal childhood health and development, which in turn influence maternal young adult circumstances that then affect maternal young adult health and development, and thereafter infant birth outcomes *(paths b – i – g– d)*

Pathway 7: Maternal childhood circumstances affect maternal young adult health and development, which in turn influence infant birth outcomes *(paths h – d)*

Pathway 8: Maternal childhood circumstances influence young adult circumstances, which in turn influence young adult health and developmental characteristics that impact birth outcomes *(paths e – g – d)*

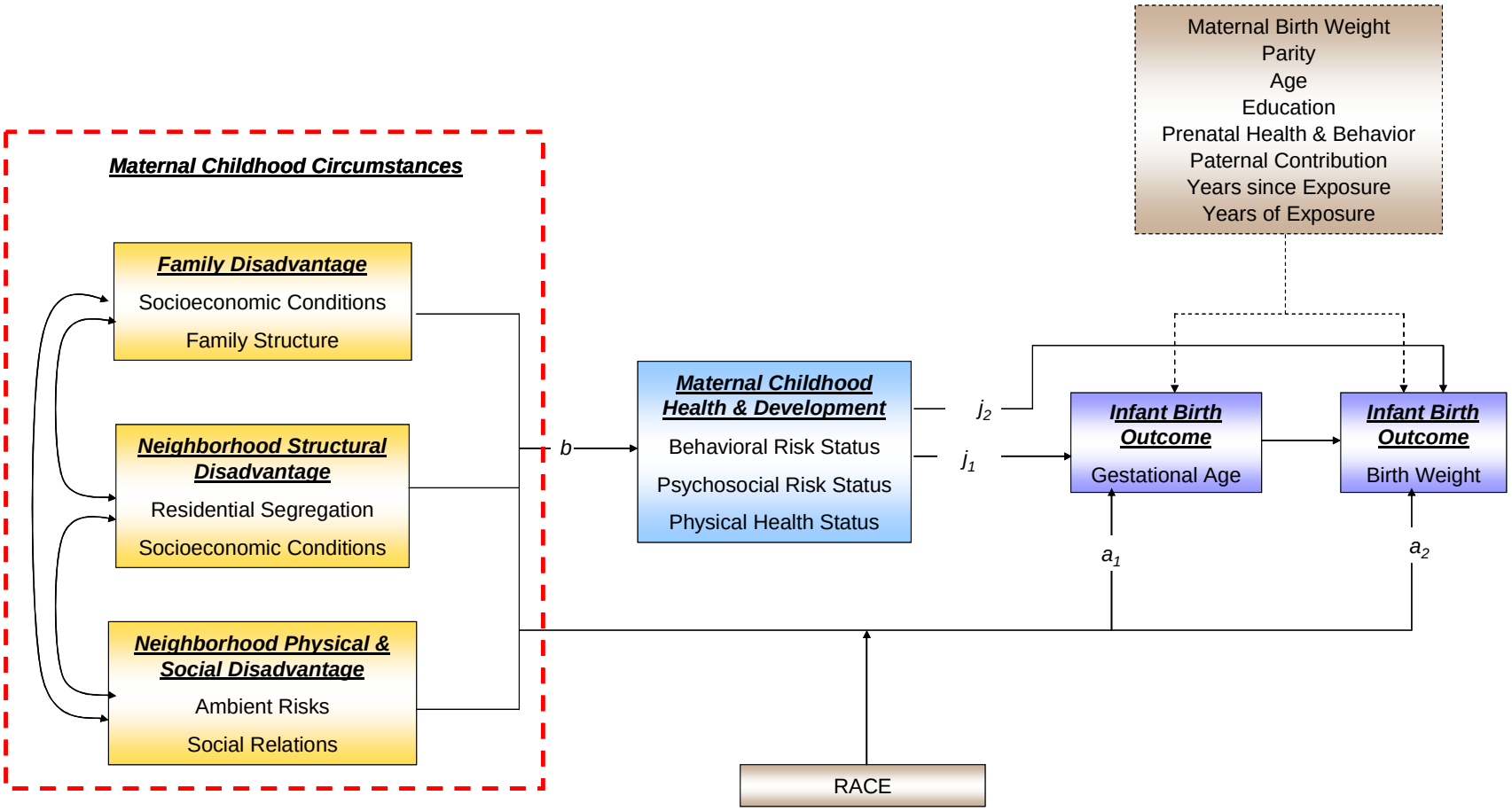
In other words, the model in Figure 4 posits direct effects (i.e., pathway 1) and indirect effects (i.e., pathways 2 – 8) of maternal childhood circumstances on birth outcomes. Due to data limitations (not conceptual reasons), maternal young adult factors (and, therefore, pathways 3 - 8) were not examined in the current study (see Section 6.3 for a discussion of the data limitations). Thus, Figure 5 (page 43) shows the final reduced conceptual model I used, with young adult circumstances, health, and development omitted. Although this omission removed the possibility of examining pathway or cumulative effects, the remaining pathways still allowed the testing of latent effects. Both Figure 4 and Figure 5 include controls for maternal birth weight, parity, age, education, and prenatal health and behavioral risks, as well as paternal contribution and "dose" of exposure to early life factors.

Figure 4. Study Conceptual Model[15]



[15]Some labels for the categories and their arrangement in Figure 4 were adapted from Graham & Power (2004).

Figure 5. Reduced Conceptual Model



NOTES: In both Figure 4 and Figure 5, path labels are denoted by italicized letters on each arrow, and are used to label the relationships between key constructs in the models. Colors and path labels in Figure 4 directly correspond to the colors and path labels in Figure 5. In cases where paths from Figure 4 have been further explicated in Figure 5, a numeric subscript is added to the path label (e.g., path j in Figure 4 corresponds to paths $j_1 - j_2$ in Figure 5).

3.3. Key Constructs

Below I conceptually define the key constructs in Figure 5.

Maternal Childhood Circumstances: Maternal childhood circumstances are the properties of the mother's familial and residential contexts that reflect and expose her to individual, social, or contextual risks. These properties include: (1) "family disadvantage", such as the socioeconomic conditions and family structure of the mother's family of origin in childhood which can result in resource limitations; and (2) "neighborhood disadvantage", which refers to the simultaneous absence of economic, material, and social resources in the neighborhood of the mother's family of origin when she was a child, resulting from the spatial patterning of those resources (Ross & Mirowsky, 2001; Curtis & Rees Jones, 1998). As suggested by the literature review, this residential adversity can be characterized by *neighborhood structural disadvantage* produced by residential segregation and adverse socioeconomic conditions. It also can be characterized by *neighborhood physical and social disadvantage* produced by: (1) ambient risks, such as poor neighborhood and housing quality; and (2) social disorder, such as a lack of social cohesion or ties, and a lack of safety (Culhane & Elo, 2005; Wheaton & Clarke, 2003).

Maternal Childhood Health and Development: Maternal childhood health and development refers to the impact of the mother's status during childhood on indicators of physical, behavioral, and psychosocial health that have a known or suspected relationship to adverse birth outcomes that occur during her pregnancy. As outlined in the literature review, key physical health risks include infection, diabetes, and hypertension. Key

behavioral risks include smoking, alcohol use, drug use, and experiencing intimate partner violence. A potentially important psychosocial risk is depression. While the literature review also notes perceived stress and racism among the suspected psychosocial risk factors, my conceptual model moves beyond individual-level perceptions to the more systemic view of stress discussed in Section 2.2.4. It does so by examining the social environmental stressors that produce more or less favorable conditions of living for different racial groups, and attempting to explicate how this unequal distribution of stressors impacts health (Daniel et al., 1999; Daniel & Linder, 2002). From this perspective, I believe that "Maternal Childhood Circumstances" comes closer to capturing the meaning of stress and racism than individual-level perceptions do.

Infant Birth Outcome: Infant birth outcome refers to the birth weight and gestational age of the infant born to the mother.

3.4. Research Questions and Hypotheses

As noted in the introduction, there are three specific aims of the proposed study. These aims are reiterated below, along with the research questions and hypotheses that will be asked, and tested, in order to satisfy the aims. The hypothesized relationships for Aims 2 and 3 are also depicted diagrammatically to assist the reader in linking them to the conceptual models in Figures 4 and 5 presented earlier in this chapter (Section 3.2).

AIM 1: To describe the distributions of birth weight and gestational age, and the prevalence of adverse birth outcomes, overall and by maternal age and race.

RQ1.1: What are the mean birth weight and mean gestational age among infants born to women in the study?

RQ1.2: What is the prevalence of LBW and PTB among infants born to women in the study?

RQ1.3: Do mean birth weight and mean gestational age among infants born to women in the study vary by maternal age or race?

H1.3.1: Mean birth weight and mean gestational age will decrease with maternal age among the study sample.

H1.3.1: Mean birth weight and mean gestational age will be lower among Blacks than among Whites.

RQ1.4: Does the prevalence of LBW and PTB among infants born to women in the study vary by maternal age or race?

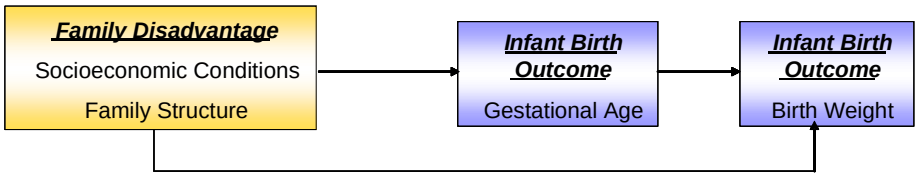
H1.4.1: The prevalence of LBW and PTB will decrease with increasing maternal age among the study sample.

H1.4.2: The prevalence of LBW and PTB will be higher among Blacks than among Whites.

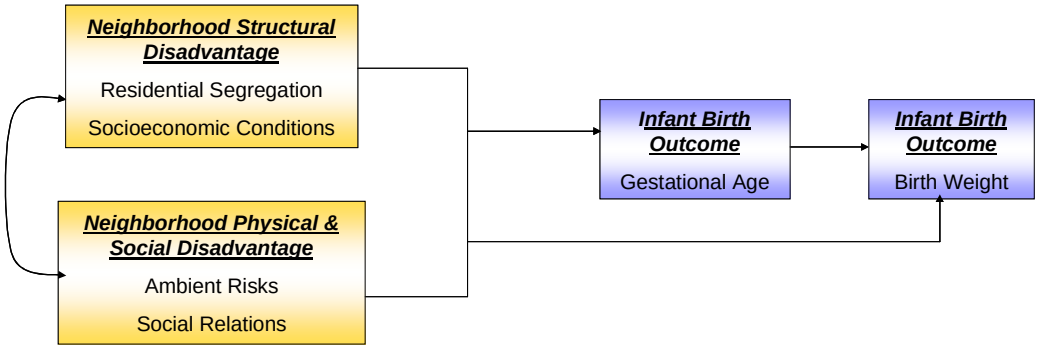
AIM 2: To examine the relationship between adverse birth outcomes and maternal exposure to family-level and neighborhood-level disadvantage during childhood, overall and by race.

RQ2.1: Is there an association between adverse birth outcomes and maternal exposure to disadvantage during childhood?

H2.1.1: Exposure to family-level disadvantage during childhood will be associated with decreases in birth weight and gestational age and, therefore, increased risks for LBW and PTB.

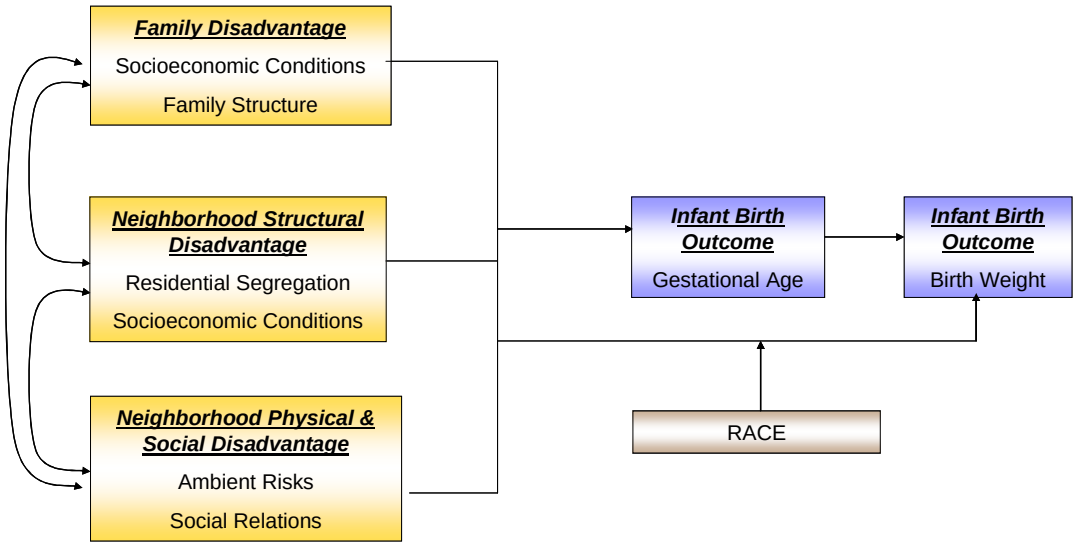


H2.1.2: Exposure to neighborhood-level disadvantage during childhood will be associated with decreases in birth weight and gestational age and, therefore, increased risks for LBW and PTB.



RQ2.2: Do the relationships between childhood exposure to disadvantage and adverse birth outcomes vary by race?

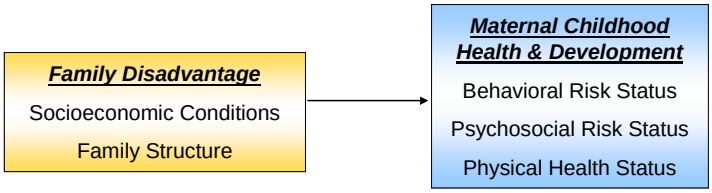
H2.2.1: At the family and neighborhood level, the effect of exposure to disadvantage during childhood on subsequent birth outcomes will be greater for Blacks than for Whites.



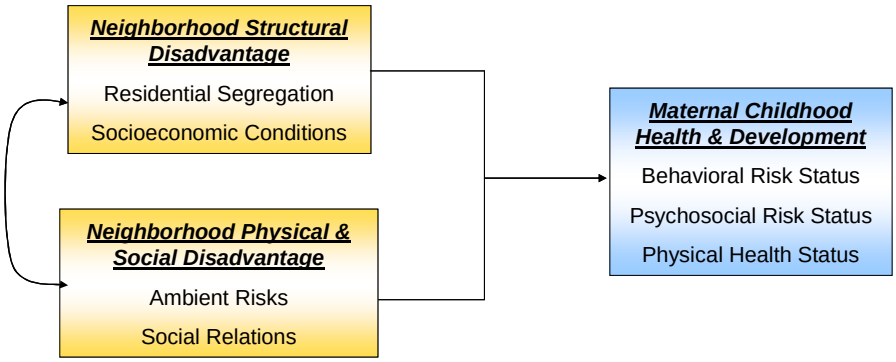
AIM 3: To explore the behavioral, psychosocial, and health pathways through which exposures to disadvantage during childhood affect subsequent birth outcomes and translate into racial disparities in adverse birth outcomes.

RQ3.1: Is the relationship between childhood exposure to disadvantage and subsequent birth outcomes mediated by childhood behavioral risk status, childhood psychosocial risk status, or childhood physical health status?

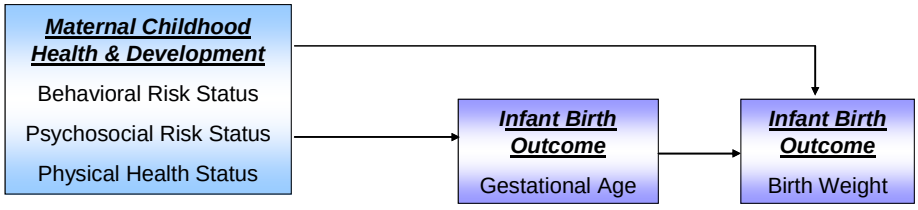
H3.1.1: Women who were exposed to family disadvantage in childhood will demonstrate more childhood behavioral, psychosocial, and physical health risks than women who were not exposed to family and neighborhood disadvantage in childhood.



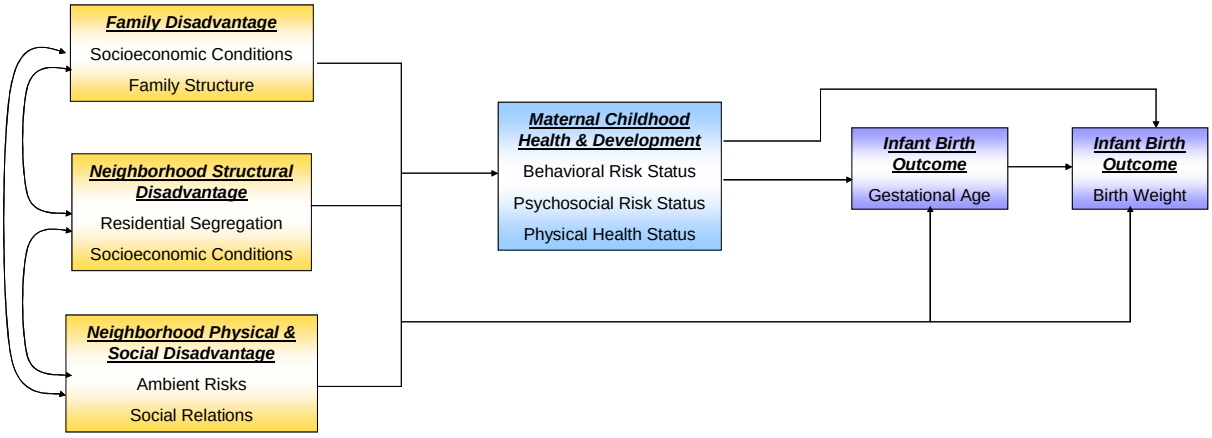
H3.1.2: Women who were exposed to family and neighborhood disadvantage in childhood will demonstrate more childhood behavioral, psychosocial, and physical health risks than women who were not exposed to family and neighborhood disadvantage in childhood.



H3.1.3: Exposure to behavioral, psychosocial, and physical health risks during childhood will be associated with decreases in birth weight and gestational age and, therefore, increased risks for LBW and PTB.

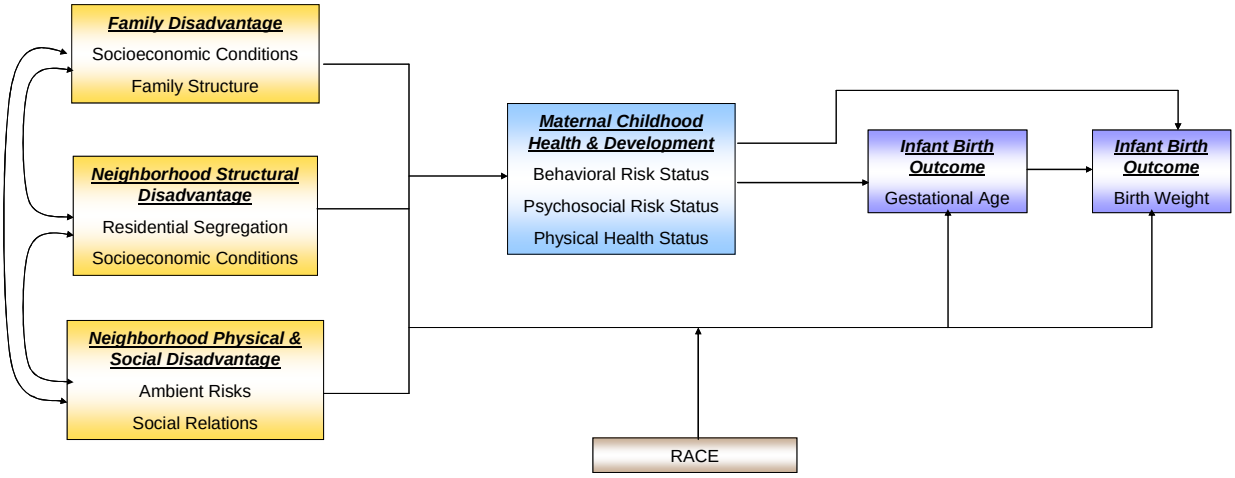


H3.1.4: The relationship between exposure to disadvantage in childhood and subsequent adverse birth outcomes will not be fully mediated by behavioral, psychosocial, and physical health risk.



RQ3.2: Do behavioral, psychosocial, and physical health risk factors explain the overall effect of race on the relationship between adverse birth outcomes and maternal exposure to disadvantage during childhood?

H3.2.1: Exposure to family and neighborhood disadvantage during childhood will produce divergent pathways of behavioral, psychosocial, and physical risk for Black and White women that lead to later disparities in adverse birth outcomes.



CHAPTER 4

METHODOLOGY

My study involved secondary analysis of data from Waves I and III of the National Longitudinal Study of Adolescent Health (Add Health) contractual dataset (Udry, 2003). In this chapter, I describe: (1) the Add Health study design; (2) the sample for my study; (3) the study measures; and (4) the analysis strategy for each study aim.

4.1. Parent Data Source

4.1.1. Study Design and Purpose

The National Longitudinal Study of Adolescent Health (Add Health) is a school-based, nationally representative study of the health, health-related attitudes and behaviors, and social contexts of a cohort of individuals from adolescence through their young adult years. It was started by researchers at UNC's Carolina Population Center in 1994 and is reportedly the most comprehensive study of adolescents ever conducted (Harris et al., 2003). Three waves of data have been collected and released since 1994, and a fourth wave of data will be released this year. Together, Waves I, II, and III provide longitudinal data on a variety of measures, including sociodemographic characteristics, risk behaviors, physical and mental health indicators, and family and neighborhood conditions. The fourth wave repeated the collection of these kinds of data, and also collected new biospecimens

that will allow for the assessment of metabolic, neuroendocrine, and inflammatory processes that may be involved in stress physiology (Harris, 2007).

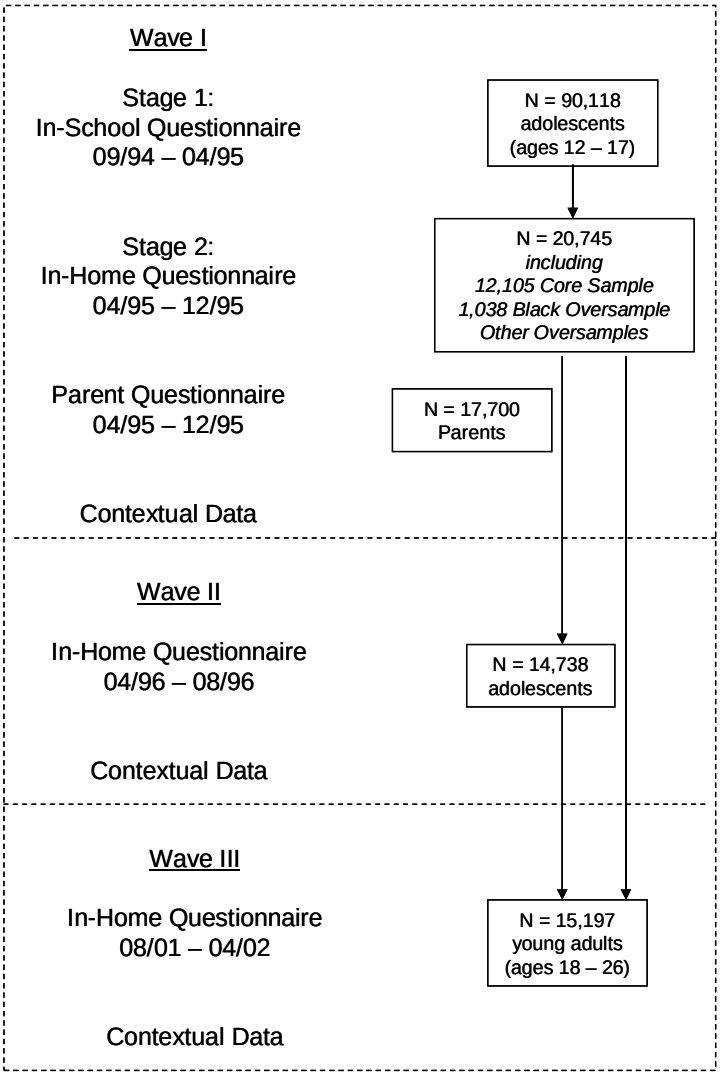
4.1.2. Sampling Strategy[16]

Stratified random sampling was used to ensure that high schools selected for inclusion in the study were representative of U.S. schools in terms of region of the country, level of urbanization, size, type, and race/ethnicity (i.e., percent White) (Chantala, 2006). High schools were stratified into 80 clusters based on these characteristics. Eligible high schools must have had an 11th grade and enrolled more than 30 students. Each participating high school helped identify a pool of feeder schools in the same community that included a 7th grade and sent at least five graduates to that high school. From that feeder pool, one was selected with probability proportional to the number of students it contributed to the high school. In total, adolescents from 132 schools were included in the core study. An initial questionnaire (discussed in 4.1.3 below) was administered in the schools, and of the 90,118 adolescents who completed it, a sample of 27,000 adolescents was drawn for participation in the remainder of the study. This sample consisted of a core sample from each community plus special oversamples, including disabled adolescents, Blacks with a college-educated parent, Chinese, Cubans, Puerto Ricans, and siblings (Harris et al., 2003).[17] Figure 6 depicts the timing and sizes of the samples for each wave of data collection.

[16]The sampling strategy and data collection activities have been previously described in more detail elsewhere (Harris et al., 2003).

[17]Eligibility for oversamples was determined based on adolescent responses to the questionnaire. Adolescents could qualify for more than one sample (Harris et al., 2003).

Figure 6. Add Health Data Collection and Sample Size Flowchart



4.1.3. Data Collection

The in-school, self-administered questionnaire was given to a nationally representative sample of students in grades 7 through 12 between 1994 and 1995 (Wave I). A series of in-home, computer-assisted personal interviews of a smaller sample of these respondents were conducted approximately one (Wave I), two (Wave II), and six (Wave III) years later (Figure 6). (The response rates for each of the three waves were 78.9%, 88.2%,

and 77.4%, respectively) (http://www.cpc/unc.edu/projects/addhealth/faqs)). In addition, data were collected from parents at Wave I to obtain information about family, neighborhood, and relationship characteristics, and from preexisting databases at all three waves to obtain information about neighborhoods and communities based on the spatial (GPS) data collected for respondents' households at each wave of data collection. At Wave III, a small set of biomarkers were collected from respondents as well (Harris et al., 2003). Information about the occurrence of pregnancies and the outcome of the pregnancies (i.e., whether live birth or not) was collected at all three waves; however, the outcomes of the live births (e.g., birth weight and gestational age) were not collected until Wave III.

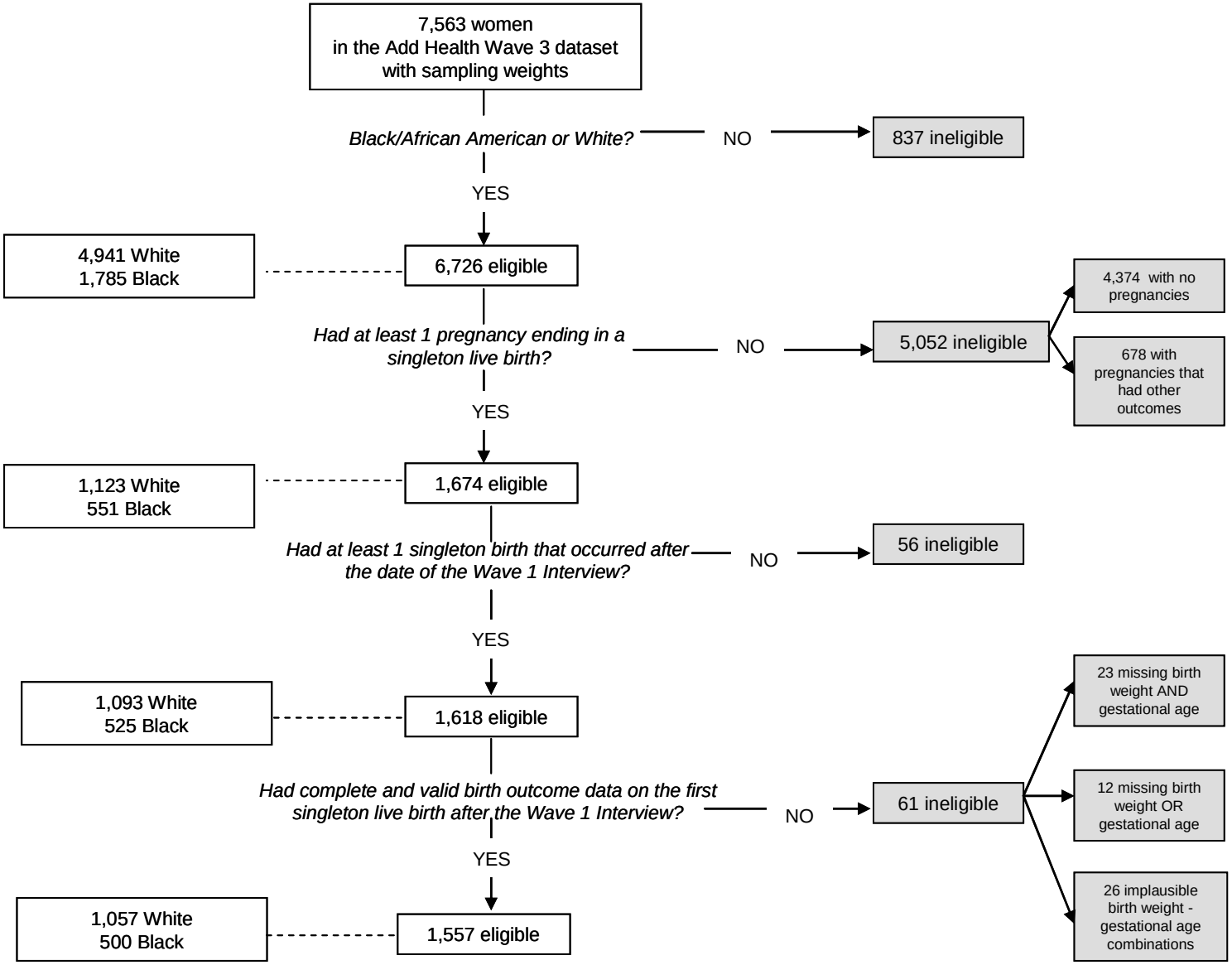
4.2 Current Study Sample

The sampling frame for my study consisted of the 8,030 women in the Add Health Wave III dataset because it is the wave in which live birth information was reported and because of my intention to treat mothers—not births—as the unit of analysis. From this pool, women were included in the study if they met the following criteria:

(a) Had sampling weights (n=7,563);
(b) Identified as White or Black/African American (n = 6,726);
(c) Reported at least one pregnancy ending in a singleton live birth (n = 1,674);[18]
(d) Reported at least one singleton live birth that occurred after the date of the Wave I interview (n = 1,618); and
(e) Had complete and plausible data on gestational age and birth weight for the first singleton live birth after the date of the Wave I interview (n = 1,557).

[18]According to another section of the survey in which women were asked to report the number of biological children in the household, the study sample size may underestimate the number of live births. Specifically, the difference between the number of women who reported biological children in the household and the number who reported live births was 246. This difference appears to be random based on the results of limited analyses comparing women with biological children in the household to women with live births by race.

Figure 7. Sample Selection Flowchart



This sampling strategy resulted in a final sample of 1,577 births to 1,577 women. Figure 7 visually summarizes the effects of restrictions 'a' – 'e' on the sample size, by respondent race, and the reasons for exclusions. Justifications for the five restrictions follow.

Sample Restriction (a): As I discuss in Section 4.3, complex survey data like Add Health include sampling weights that are used during design-based analysis to reflect the unequal probabilities of selection of the sample elements. Respondents in Add Health who were selected outside the sampling frame do not have sampling weights and, therefore, were removed prior to analysis.

Sample Restriction (b): Only Black and White respondents were included in the study because of the predominant focus in the published literature on perinatal health disparities between Blacks and Whites. In addition, Add Health has an insufficient number of female respondents in the other racial groups that would have met my eligibility criteria to ensure appropriate testing of the study hypotheses with those groups.

Sample Restriction (c): As is conventional in birth outcome studies, only women who had singleton live births were selected for inclusion in the study because multifetal pregnancies have lower birth weights and shorter lengths of gestation than singletons.

Sample Restriction (d): The sample also was limited to women who had at least one singleton live birth after the Wave I interview to ensure that all independent variables (which were derived from the Wave I interview) preceded the pregnancy.

Sample Restriction (e): I further restricted my sample to women who did not have missing or outlier data on birth weight and gestational age for their *first* singleton live birth after Wave I. I restricted birth outcomes to only the first singleton live birth after Wave I for

several reasons. First, this strategy is consistent with one of the few longitudinal investigations of birth outcomes in the published literature (e.g., Gavin et al., 2009). Second, the majority of women (>90%) who met restrictions 'a' through 'd' were nulliparous prior to Wave I, meaning that the birth order of most births included in the analysis was comparable across women (i.e., mostly first births). Third, relatively few women in the Add Health study at Wave III reported more than one singleton live birth,[19] thereby limiting the value of linking sequential births to each woman in the study. In addition, the findings of studies about the effect of birth order on subsequent birth outcomes are mixed. For example, the findings differ depending on whether the study is cross-sectional or longitudinal. In cross-sectional studies, second births appear to have worse outcomes, while in longitudinal studies second births to the same women appear to have better outcomes than do their first births (Klerman, 2006). Studies of the likelihood of repeat PTB or LBW also have been inconsistent—ranging from 15 to 50% depending on the causes, gestational ages, maternal ages, and interpregnancy intervals (Esplin, 2006; Mercer et al., 1999; Adams et al., 2000).

Thus, after identifying the records for the women's first singleton live births after Wave I, I excluded women with missing or outlier data on infant birth weight and gestational age using birth weight distributions developed at the CDC and reported in Adams et al. (1997). I considered outliers to be records in which the birth-weight-gestational-age combination fell outside of a "normal" range defined by the race- and gestational-age-specific mean birth weight plus or minus 2.5 times its standard deviation.

[19] To be exact, 280 White women and 135 Black women reported having more than one singleton live birth after Wave I. The intracluster correlation coefficient was 0.599 for birth weight and 0.415 for gestational age. Using analytic techniques that can account for non-random clustering, therefore, would be worthwhile when analyzing data from future waves of Add Health that will have a larger sample of repeat (i.e., multiparous) mothers.

4.3 Measures

Explanatory, outcome, and control variables were selected based on theoretical or empirical evidence of their utility and appropriateness. Table 6 summarizes these variables in relation to study constructs and items. (A more detailed list of study measures, as well as the Add Health items used in their creation, variable names and values, is included in Appendix A). Following the table, I describe the key study measures in detail. Throughout the description, I use the term "respondent" to refer to women in my sample (during adolescence or at the time of their infant's birth), and refer to their interviews as the "Waves I and III In-Home Interviews". The term "maternal" also refers to the respondent and is used to distinguish the characteristics that pertain to her from those of her infant. "Parent" refers to the person in the respondent's home who completed the "Wave I Parent Interview" while she was an adolescent. "Resident mother or father" is used to denote the person in the respondents' home to whom the respondent referred as such during her interviews. Figure 8 depicts the relationships between these individuals, the terms I use to refer to them, and the data sources used to obtain information from or about them.

Figure 8. Relationships, Terminology, and Data Sources

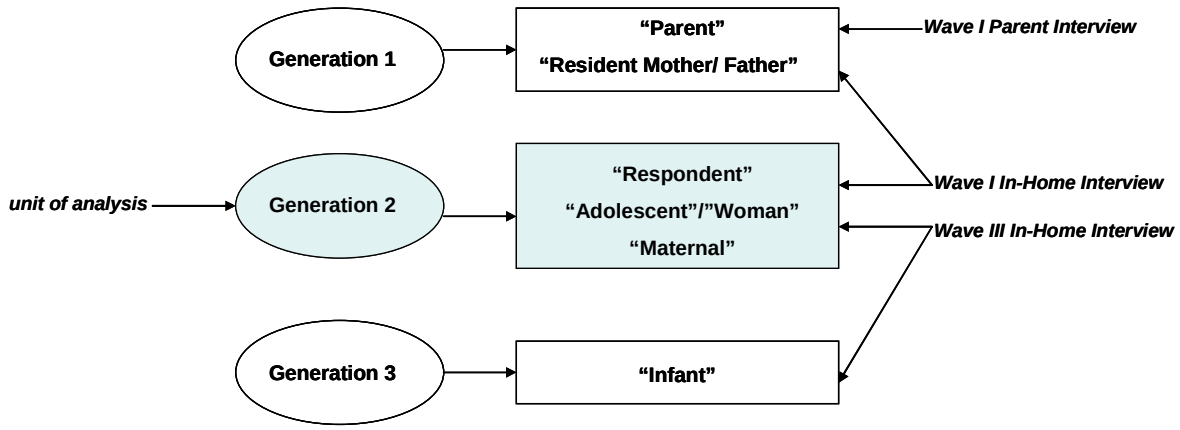


Table 6. Summary of Key Study Measures

	Construct	Indicators	Items Used*
Infant Birth Outcome	Birth Weight	Calculated birth weight (grams)	▪ What was the baby's birth weight, in pounds? ▪ What was the baby's birth weight, in ounces?
	Gestational Age	Calculated gestational age (weeks)	▪ Was the baby born too early – that is, after a pregnancy of less than 40 weeks? ▪ How many weeks early?
Maternal Race	Race	Race	▪ What is your race? ▪ Which one category best describes your racial background? ▪ What is your race? *(parent)* ▪ Which one category best describes your racial background? *(parent)* ▪ Observation of race *(interviewer)*
Maternal Childhood Neighborhood Disadvantage	Structural Disadvantage	Poverty	▪ Proportion of families with dependents with income in 1989 below poverty level, tract *(census)*
		Public Assistance	▪ Proportion households with public assistance income, tract *(census)*
		Unemployment	▪ Total unemployment rate *(census)*
		Education	▪ Proportion aged 25 years and over with no HS diploma or equivalency, tract *(census)*
		Family Structure	▪ Proportion households that are female headed, no husband, with dependents *(census)*
		Residential Segregation	▪ Proportion black *(census)*
	Social and Physical Disadvantage	Social Ties	▪ People in the neighborhood look out for each other
		Safety	▪ Do you usually feel safe in your neighborhood?
		Safety	▪ Total crime rate per 100,000 population – county *(uniform crime reports)*
		Safety	▪ Did you feel concerned for your safety? *(interviewer)*
		Housing Quality	▪ How well kept are buildings on street or, if rural, the building/house in which respondent lives? *(interviewer)*
		Housing & N'hood Quality	▪ Proportion vacant housing units, tract *(census)*

* Except where noted (in italics following the item), responses to the items were given by the respondent. Census items are from the Census of Population and Housing, 1990: Summary Tape File 3A. Uniform crime reports are from the U.S. FBI.

Table 6 continues on next page

Table 6. Summary of Key Study Measures *(continued)*

	Construct	Indicators	Items Used*
Maternal Childhood Family Disadvantage	Family Disadvantage	Education	▪ How far did (resident mom/dad) go in school?
			▪ How far did you go in school? *(parent)*
		Poverty Status	▪ About how much total income, before taxes, did your family receive in 1994 (incl. own income, other HH members' income, and other sources, e.g., welfare)?
			▪ Are you receiving public assistance? *(parent)* ▪ Last month, did you or any member of the household receive AFDC? Food stamps? Housing subsidy or public housing? *(parent)*
			▪ Does [resident mother or father] receive public assistance, such as welfare?
		Family Structure	▪ What is [NAME]'s relationship to you? ▪ Which description best fits [NAME] relationship to you?
Maternal Childhood Health and Development	Behavioral Risk Status	Substance Use Score *(count of number used)*	▪ During the past 30 days, on how many days did you smoke cigarettes? ▪ During the past 12 months, on how many days did you drink alcohol? ▪ During the past 30 days, how many times have you used marijuana, cocaine, or any other types of illegal drugs?
	Physical Health Risk Status	Self-Rated Health	▪ In general, how is your health?
	Psychosocial Risk Status	Unable to Shake Blues	▪ During the past week, how often did you feel like you could not shake off the blues?
		Felt Depressed	▪ During the past week, how often did you feel depressed?
		Felt Happy	▪ During the past week, how often did you feel happy? (reverse scored)
		Felt Sad	▪ During the past week, how often did you feel sad?
		Felt Life Not Worth Living	▪ During the past week, how often did you feel that life was not worth living?

* Except where noted (in italics following the item), responses to the items were given by the respondent. Census items are from the Census of Population and Housing, 1990: Summary Tape File 3A. Uniform crime reports are from the U.S. FBI.

4.3.1. Outcome Variables

Both gestational age (PTB) and birth weight (LBW) were included as outcome variables[20] in the study because, despite the overlap between them (as discussed in Chapter 2), they are suspected of having distinct etiologies. In addition, most previous studies of the relationship between neighborhoods and birth outcomes examined LBW, which is confounded with small-for-gestational age. Thus, researchers have recommended that future studies explicitly examine the relationship between neighborhoods and PTB (IOM, 2006).

In the majority of perinatal epidemiology studies, gestational age and birth weight variables are dichotomized (e.g., PTB or not, and LBW or not). This practice is perhaps due to the clinical and public health significance of PTB and LBW categories (Pickett et al., 2005) and/or the desire for comparability across studies. However, several recent neighborhood studies have modeled gestational age and birth weight as continuous variables, with investigators offering a variety of reasons for this practice. First, it may allow for the detection of subtle associations that might not be apparent using dichotomies (Bell et al., 2006; Masi et al., 2007). Second, continuous variables can assist in understanding not only racial/ethnic disparities in rates of LBW and PTB, but also racial/ethnic differences in the entire birth weight and gestational age distributions (discussed in Section 2.2.1.1; Farley et al., 2006). In addition, socioeconomic indicators are associated with birth weight all along

[20]Technically, birth weight is the outcome variable and gestational age is presumed to be antecedent to birth weight (i.e., a mediator) in my study conceptual model. Because of the overlap between these birth outcomes, however, my model also assumes that all explanatory variables may be associated with both of them (thereby suggesting that the association between the explanatory variables and birth weight is partially mediated by gestational age). For this reason, I discuss them here as if they are both outcome variables while recognizing that I have modeled a relational pathway between them that suggests birth weight is the only true outcome variable in my model.

the birth weight continuum (Pearl, Braveman, & Abrams, 2001). Third, treating gestational age and birth weight as continuous variables increases statistical power to estimate covariate effects with precision, which in turn facilitates uncovering true relationships when they exist (Pearl, Braveman, & Abrams, 2001; Sastry & Hussey, 2003).[21] Based on these arguments, I treated gestational age and birth weight as continuous variables.

Unlike the majority of perinatal epidemiology studies that use medical records to assess gestational age and birth weight, my study obtained these measures from respondent self-report. Studies have shown, however, that mother's recall of her infant's birth outcome is generally reliable (McCormick & Brooks-Gunn, 1999; Buka et al., 2004; Tomeo et al, 1999).[22]

Infant Gestational Age:

Two items in the Wave III dataset (i.e., "Was the baby born after a pregnancy of less than 40 weeks?" and, "How many weeks early?") were used to determine infant gestational age. Responses were recoded to form a continuous variable—i.e., 40 minus the number of weeks early for respondents who reported that the birth occurred after a pregnancy of less than 40 weeks; 40 for respondents who said "No" in response to this question. As noted in Chapter 2, preterm birth (PTB) refers to a birth that occurs less than 37 weeks of gestation (Nguyen & Wilcox, 2005). In its dichotomous form, therefore, preterm birth was defined in this study as a birth that reportedly occurred more than three weeks early.

[21]It should be noted that several of these studies also modeled categorical (dichotomous) versions of these variables in sensitivity analyses, and found similar results to those obtained from analyses using continuous outcomes (Farley et al., 2006; Pearl, Braveman, & Abrams, 2001).

[22]In addition, the validity of gestational age data reported on birth certificates has been questioned because errors in the recording of gestational age vary systematically by race (Pickett et al., 2005).

Infant Birth Weight:

Respondents were asked to report the weight of their infant at birth, in pounds and ounces. To be consistent with previous literature that treated birth weight as a continuous variable (e.g., Bell et al., 2006; Masi et al., 2007; Sastry & Hussey, 2003) and with clinical measurements of birth weight (Nguyen & Wilcox, 2005), I converted the responses for pounds and ounces to grams and then added them. The following formulas were used in the conversion: 1 pound = 453.59 grams, and 1 ounce = 28.35 grams. Clinically, LBW refers to any birth weight less than 2500 grams (Nguyen & Wilcox, 2005), which is roughly equivalent to a weight of 5 pounds, 8 ounces (or 5 ½ pounds). Thus, in its dichotomous form, LBW was defined in this study as a birth reportedly weighing less than 2500 grams.

4.3.2. Explanatory Variables

As the conceptual model in Figure 5 (Chapter 3) implies, the explanatory variables in my study included maternal race, several variables to reflect exposure to neighborhood- and family-level disadvantage during childhood, and childhood behavioral, psychosocial, and physical health risk status. The reader will notice that similar variables from different sources were used for some constructs in the study. While the possibility of discordant responses across data sources would ordinarily make this practice inadvisable, the analytic approach used in this study (which I discuss in Section 4.4) rendered it possible and created the potential for measuring constructs with greater reliability than using single items from a single source.

Maternal Race: At Waves I and III, respondents were asked to self-identify their race/ethnicity and could choose more than one racial category. They were also asked to select the category that best described their racial/ethnic background. Parents also reported their race/ethnicity, and interviewers were asked to observe the race/ethnicity of respondents. In my study, self-identified race was used for respondents who selected a single racial category. Parent and interviewer reports were used to confirm race/ethnicity for respondents who selected more than one race/ethnicity or to assign race/ethnicity for respondents who did not report it. Only respondents coded as non-Hispanic White or Black/African American were included in my study.

Childhood Neighborhood Disadvantage: Three key constructs were assumed to represent neighborhood disadvantage: neighborhood structural disadvantage, neighborhood social disadvantage, and neighborhood physical disadvantage. To measure such constructs, especially neighborhood structural conditions, the majority of contextual studies of adverse birth outcomes use census-based measures of neighborhood conditions. This practice has been criticized because census boundaries may not accurately specify neighborhood boundaries as well as subjective assessments can (Culhane & Elo, 2005). In addition, while some researchers have found that census-based measures of neighborhood conditions are highly predictive of subjective perceptions of those conditions (Sampson & Raudenbusch, 1999; Weden, Carpiano, & Robert, 2008), others have asserted that subjective assessments or direct observation may offer specific insights into the factors contributing to neighborhood conditions that cannot be captured by census data (Laraia et al., 2006;

Sampson, Morenoff, & Gannon-Rowley, 2002; Ingoldsby et al., 2006). On the other hand, objective census-based measures may capture important structural features of the neighborhood that residents may not or cannot perceive (Weden, Carpiano, & Robert, 2008). Choosing objective over subjective measures (or vice versa) may, therefore, result in the over- or under-estimation of neighborhood effects (Mykyta et al., 2007; Kawachi & Berkman, 2003). Thus, as I discuss below, I measured the three constructs of neighborhood disadvantage in my study using census-based (objective), observational, and self-reported (subjective) data from the Wave I Contextual Data, Wave I In-Home Interview, and Wave I Parent Interview.

For the census-based items, I used the tract-level rather than the block group-level versions of the variables in order to be consistent with recent investigations of neighborhood disadvantage and birth outcomes (e.g., Messer et al., 2006b, 2006c, 2008). Moreover, while block groups may better approximate neighborhoods than census tracts, recent contextual studies of adverse birth outcomes have shown that the geographic unit chosen to approximate the neighborhood did not influence the observed association between neighborhood-level variables and adverse birth outcomes (e.g., Mason et al., 2008).

Neighborhood Structural Disadvantage. To assess neighborhood structural disadvantage, I used five census-tract level measures of neighborhood socioeconomic conditions that also have been used in other studies of structural or neighborhood influences on health (e.g., Culhane & Elo, 2005; Cubbin et al., 2005; Pickett et al., 2002; Schieman, 2005; Messer et al., 2006b, 2006c, 2008). These

measures were: proportion of families with dependents with income in 1989 below poverty level, proportion of households with public assistance income, unemployment rate, proportion of residents age 25 years and older with no high school diploma or equivalency, and proportion of households that are female-headed, with dependents and no husband. Another indicator of neighborhood structural disadvantage I used is residential segregation. However, conventional measures of residential segregation, such as the dissimilarity index (Massey & Denton, 1988), are not available in Add Health. Thus, I used a census-based measure of the proportion of Black residents in the census tract as a proxy for residential segregation. While this measure is probably better thought of as a measure of racial composition, some researchers have argued that it is an appropriate spatial measure of residential segregation (Mason et al., 2009).

Neighborhood Social Disadvantage. To assess neighborhood social relations indicative of disadvantage, I used several measures of problematic social ties and lack of safety, which I assumed to be a reflection of social disorder (Sampson & Raudenbush, 1999). I used one item from the Wave I In-Home Interview that asked respondents to indicate whether or not neighbors look out for each other. Responses to this item were coded 0 if Yes and 1 if No. I used three indicators of neighborhood safety. Respondents reported during the Wave I interview whether or not they felt safe in their neighborhoods. Interviewers also were asked to report whether they had safety concerns when they went to the respondent's home for the interview. I coded the

responses 0 if the respondent or interviewer reported feeling safe and 1 if they did not. While previous studies have found that self-reported neighborhood characteristics are reliable (Echeverria, Diez-Roux, & Link, 2004), I also used a measure of the crime rate per 100,000 residents in the county from the U.S. Federal Bureau of Investigations (FBI) Uniform Crime Statistics.

Neighborhood Physical Disadvantage. Based on previous literature distinguishing neighborhood social disorder from physical disorder (e.g., Sampson & Raudenbush, 1999), two ambient risk conditions were chosen to represent neighborhood physical disadvantage—i.e., how well kept the neighborhoods and homes in the neighborhood were. To assess these conditions, I used data reported by interviewers during the in-home interviews, as well as a census-based measure. Interviewers were asked to indicate, on a scale from 1 (very well) to 4 (very poorly), how well kept the buildings or homes on the respondent's street were. They skipped this item, however, if the home was in a rural area; in those cases, I used the interviewer rating of how well kept the home in which the respondent lived was (which used the same rating scale as the skipped item). Because few interviewers rated the housing quality as very poorly kept, the categories for poorly kept (3) and very poorly kept (4) were combined prior to the analysis. I also used a census-based measure of housing and neighborhood quality: proportion of vacant housing units in the census tract.

Childhood Family Disadvantage: Five indicators of childhood family disadvantage were drawn from the Wave I In-Home and Parent Interviews to represent family resource limitations due to: (1) family structure and (2) household socioeconomic conditions.

Family Structure was measured using responses to questions in the Wave I In-Home Interview about residents in the house and their relationship to the respondent. I categorized the responses into two groups: (1) two-parent families, which included families with two biological parents, two adoptive parents, or one biological parent and one non-biological parent; and (2) single or surrogate families, which consisted of single parent families or other parenting/guardianship arrangements with no biological or adoptive parents in the household, such as foster parents, stepparents, grandparents, aunts, uncles, siblings, or other adults who acted as parent figures. A code of 0 was assigned to respondents in two-parent families and a code of 1 was assigned to respondents in single or surrogate families.

Family Socioeconomic Conditions were measured by two indicators of socioeconomic status: family poverty status and parent education. For family poverty status, I used a combination of items from: (1) the Wave I Parent Interview that asked parents to report before-tax family income from all sources in 1994 and whether he/she or any member of the household received public assistance, and (2) items from the Wave I In-Home Interview that asked respondents to indicate whether either of their resident parents or guardians received public assistance. A respondent was classified as having lived in an impoverished family if their family income was

below the 1994 poverty level for a 4-person household (approximately $15,000) OR if someone in the household received public assistance of any kind. Respondents who met at least one of these criteria were assigned a code of 1; those who did not meet either of these criteria were assigned a code of 0. For highest parental education, I chose the highest education level attained by either resident parent, as reported by the respondent or, if missing, by the parent. Responses were coded on a 4-point scale, where 1 = completed college or higher, 2 = completed some college, 3 = completed high school or GED, and 4 = did not complete high school.[23]

Childhood Behavioral Risk: To assess childhood behavioral risk, I used several items from the Wave I In-Home Interview to measure substance use. The items were: "During the past 30 days, on how many days did you smoke cigarettes?", "During the past 12 months, on how many days did you drink alcohol?", and "During the past 30 days, how many times have you used marijuana, cocaine, or any other types of illegal drugs?" Consistent with the literature on cumulative risk scoring approaches (Appleyard et al., 2005;

[23]Parent interviews were not conducted for 3,076 of the Wave I respondents. In addition, up to 10% of parents or guardians who completed an interview refused to answer the income question, although they were somewhat more likely to respond to the question about receipt of public assistance. I made the decision to combine parent and adolescent reports of socioeconomic indicators because of this missing data problem, but only after determining that the responses across parents and adolescents were highly concordant in cases where both were available. For the public assistance items, approximately 75% of the responses were the same. After combining the respondent public assistance items with the parent reported public assistance and income items to create the poverty status indicator, it was clear that only 15% of the values would have differed if I had chosen only one of the items. (In other words, there was concordance across the income and public assistance item responses when both were available, as well). For the education items, a lower percentage (~67%) of the responses were the same across adolescents and parents but this was to be expected, given that respondents were asked about both of their resident parents or guardians (when they had more than one) while the person who responded to the parent interview just reported his/her own education. (And this person could have been an adult other than the resident parent to whom the adolescent referred). For this reason, the adolescent report was given preference for the parental education indicator when responses were available from both sources, and "parent" reports were only used when the adolescent report was missing. Dummy variables also were created to reflect the data source (i.e., adolescent only, parent only, both adolescent and parent, or neither) to enable determination of whether source made a difference.

Newcomb, Maddahian, & Bentler, 1986; Ostaszewski & Zimmerman, 2006), the responses to these items were first recoded to 0 or 1, where 0 corresponded to not using the substance at all during the reference time period and 1 corresponded to using the substance at least once during that period. These values were then added to create a summary substance use score that reflected the extent to which respondents had engaged in substance use. Scores ranged from 0 (no risk) to 3 (high risk).

Childhood Psychosocial Risk: To assess psychosocial risk, I used five indicators of depression because of previous research that has pointed to the potential role played by this condition in racial/ethnic disparities in birth outcomes (discussed in Chapter 2). The five items were part of a 19-item version of the Center for Epidemiologic Studies Depression Scale (CES-D) (Radloff, 1977) that was included in the Add Health Wave I interview. The original CES-D consists of 20 items that are presumed to represent all major components of depressive symptomatology (e.g., depressed mood, feelings of helplessness and hopelessness, loss of appetite, sleep disturbance, and psychomotor retardation). The items are rated on 4-point scales indicating the degree of their occurrence during the past week (ranging from 0 = never or rarely to 3 = always). The Add Health research team retained this scoring approach but dropped two items, worded two items differently, and added one item because of findings of earlier studies with adolescents and the wording used for children in those studies (e.g., Garrison et al., 1991; Perreira et al., 2005).

I used only five of the 19 items in Add Health—i.e., feeling that *life* was not worth living, feeling *sad*, feeling *depressed*, being unable to shake the *blues*, and feeling *happy*

(reverse-scored)—because of the findings of a study by Perreira and colleagues (2005) that assessed the measurement equivalence of the 19-item scale across race/ethnicity and immigrant generation using Add Health Wave I data. They found that the five-item index was structurally invariant across groups and that there were no substantively large differences in the parameter estimates for all indicators considered together. Thus, they concluded that the 5-item index is appropriate for intergroup comparisons, but recommended that researchers include a measurement model in estimations instead of using a composite score because the reliability of the five-item CES-D was only .81. And indeed it was .80 in my study; thus, I included all five items as indicators of a psychosocial risk latent variable.

None of the other relevant psychosocial factors identified in the literature review (Section 2.2.3)—that is, racism and stress—were available at the individual-level in the Add Health dataset. As I suggest in Section 3.3., the neighborhood-level measures of social structural conditions that I included in the study, such as residential segregation, may better capture these constructs for the purposes of my study.

Childhood Physical Health Risk: To assess childhood physical health risk, I used a measure of self-rated health obtained from the Wave I interview. Specifically, respondents were asked to rate their health on a 5-point scale, ranging from excellent (1) to poor (5). Because few respondents rated their health as poor, the categories for fair health and poor health were combined prior to the analysis. Support for using this item comes from numerous studies that have consistently found self-rated health to be a valid measure of

current physical health status and predictor of mortality among adults (Benyamini & Idler, 1999; Idler & Benyamini, 1997), and from one study that found self-rated health to be a moderately stable and reliable spontaneous health assessment among adolescents in Add Health (Boardman, 2006).

4.3.3. Control Variables

To address potential confounding, additional variables were included in the study based on their theoretical or empirical relationship to birth outcomes and to the measures of social and contextual disadvantage. Specifically, the following respondent characteristics were considered potential control variables:

Maternal Age: Maternal age referred to the respondent's age when her infant was born. This variable was calculated by subtracting her birth date from the infant's birth date, which was obtained from the Wave III In-Home Interview.[24]

Maternal Education: In my study, maternal education was defined as whether or not the respondent met the level of education that would be expected of her given her maternal age. To create this variable, I first estimated the respondent's education at the time of her infant's birth by using the respondent's reported educational attainment at Wave I ("Wave I education"), the difference between her age at Wave I and her age at the time of her pregnancy ("age difference"), and her reported educational attainment at Wave III ("Wave III education"). If "Wave I education" and "Wave III education" were equal,

[24]Due to the sampling strategy and the requirement that infant birth date be non-missing in order to identify it as a birth that occurred after the Wave I interview (see Section 4.2), this variable was able to be computed for all women in the sample.

then her maternal education level was assumed to be the same value as those variables. If they differed, however, then I compared the sum of "Wave I education" and "age difference" to "Wave III education". If the sum was equal to or greater than "Wave III education", then I assumed her maternal education level to be the value of Wave III education; otherwise, I assumed her maternal education level to be the value of the sum. Because of differences in maternal age throughout the sample, comparisons between women using the absolute value of my estimates of maternal education would not be appropriate. Therefore, I followed the approach of Hertz-Picciotto and colleagues (2000), which involved classifying my maternal education estimates into three categories: 1 = more than a high school education, 2 = either completed high school or age less than 20 years and did not complete high school, and 3 = did not complete high school and age 20 or older. Based on my original definition of Maternal Education, I assumed these categories to also represent the degree to which the respondent had met educational expectations for her age at the time of her infant's birth, with the middle category treated as the expected level.

Parity: Parity was defined as the number of births experienced by the respondent, regardless of whether it was a live birth or a still birth, counting a multiple birth pregnancy as one birth (Nguyen & Wilcox, 2005). Thus, I computed parity based on respondent reports of the number of times she had been pregnant and the outcomes of those pregnancies. The counts were divided into three variables – parity prior to the Wave I interview, parity after the Wave I interview, and overall parity. For "parity prior to the Wave 1 interview", the counts were originally categorized as nulliparous (i.e., no births),

uniparous (i.e., one birth), and multiparous (i.e., two or more births). However, because few respondents were multiparous prior to the Wave I interview, the uniparous and multiparous categories were combined prior to the analysis. For "parity after the Wave I interview", the counts were categorized as uniparous or multiparous. I retained "overall parity" as a count variable instead of categorizing it.

Prenatal Behavioral Risk: To measure prenatal behavioral risk, I created two indicators. The first measure of prenatal behavioral risk was the total number of substances respondents reported using during pregnancy. This prenatal substance use score was created using the cumulative risk scoring approach described previously (see "Childhood Behavioral Risk Status" above) to first code whether or not the respondent reported using cigarettes, alcohol, or illegal drugs during pregnancy (using 0 to represent No and 1 to represent Yes) and then to sum the responses across those three items. Scores, therefore, ranged from 0 (no risk) to 3 (high risk). The individual items for each of the three substances were also retained to see if any one of them was more influential to the score than others.

The second measure of prenatal behavioral risk was the adequacy of prenatal care. This measure was created from two items to capture both initiation and timing of prenatal care: (1) Did you visit a doctor or nurse during your pregnancy?, and (2) In what month did you first visit the doctor or nurse during your pregnancy? Responses were combined and coded on a scale from 1 to 4, where 1 was considered very adequate (i.e., received prenatal care during the first trimester), 2 was considered somewhat adequate (i.e., received prenatal

care during the second trimester), 3 was considered somewhat inadequate (i.e., received prenatal care during the third trimester), and 4 was considered inadequate (i.e., did not receive prenatal care). Because preliminary analyses revealed that very few respondents did not receive prenatal care at all, categories 3 and 4 were combined prior to the analysis.

Prenatal Health Risk: To assess prenatal health risk, I created a prenatal health risk score based on the presence or absence of chronic health conditions that have a known or suspected relationship with adverse birth outcomes (see Section 2.2.1.3). These health conditions were presumed to be chronic and, therefore, present during the prenatal period if they were present at both Wave I and Wave III. Thus, I used four items from the Wave I In-Home Interview and six items from the Wave III In-Home Interview to create three variables representing the presence or absence of bacterial vaginosis, diabetes, and obesity at Wave I and Wave III. For diabetes, I used the items "Have you ever been diagnosed with diabetes?" and "How old were you when you were told you had diabetes?" from the Wave III Interview, and compared the latter to the respondent's age at the time of the Wave I interview in order to determine if the respondent had diabetes at Wave I because it was not asked during that interview. For obesity, I computed BMI from self-reported height and weight at Wave I and Wave III using the formula: BMI = 703 x [weight in pounds / (height in inches)^2]. BMI greater than 30 was assumed to represent obesity. For bacterial vaginosis, I used the item "Have you ever been told by a doctor or nurse that you have bacterial vaginosis?" from the Waves I and III interviews. Using the same cumulative risk scoring approach described above (see "Childhood Behavioral Risk"), I assigned a value of 0 to each

of the three health condition variables if the condition was not present at both Wave I and Wave III and a value of 1 if the condition was present at both time points. The responses to these three items were then summed to create the health risk score. Thus, scores ranged from 0 (no risk) to 3 (high risk). The individual items for each of the three health conditions were also retained to see if any one of them was more influential to the prenatal health risk score than the other two.

Paternal Contribution: I used two variables to indicate the father's contribution of social support to the respondent during her pregnancy: prenatal relationship status and prenatal care participation. Prenatal relationship status was created from three items that captured whether or not the respondent was married, cohabitating, dating, or having any contact with the father during her pregnancy. This variable was coded on a 4-point scale, where 1 was married, 2 was cohabitating but not married, 3 was steady relationship, and 4 was no relationship. Prenatal care participation was an item that asked whether or not the baby's father attended prenatal care. Responses were coded 0 for Yes and 1 for No. Add Health did not include measures of the father's contribution of economic resources to the respondent during her pregnancy.

Time to Birth: To control for differences across respondents in the time between exposure to childhood disadvantage and the birth event, I created a variable to reflect the "years since exposure" by computing the number of years between the infant's birth date and the date of the Wave I interview.

Dose: To control for differences across respondents in the length of time they were exposed to neighborhood disadvantage, I created a variable to reflect the dose or "years of exposure" based on the number of years the respondent lived in her childhood residence prior to the Wave I interview. Respondents were asked to report how old they were when they moved to the residence in which they lived during the Wave I interview. Dose was computed by subtracting their response to this item from their age at the time of the Wave I interview.

4.4. Data Analysis

I conducted analyses for the three study aims in two stages: first, descriptive and bivariate analyses; and second, tests of hypotheses for each study aim using structural equation modeling (SEM). Prior to performing these analyses, the dataset was prepared and preliminary analyses were performed to identify any issues that might impact the analysis. Throughout the analyses, I performed sensitivity analysis (discussed in Section 4.4.4) to determine whether the results were robust to variation in variable definition or model specification.

It is typical for analyses of complex survey data like Add Health to account for the complex sampling design (e.g., clustering, stratification, and unequal probability of selection) in order to produce unbiased estimates of standard errors, avoid underestimating the variance, and reduce the likelihood of false-positive statistical test results, i.e., rejecting the null hypothesis when in fact it is true (Chantala, 2003; Wang, Yu, & Lin, 1997). This approach involved several steps. First, I accounted for both the clustering of respondents

within schools and the stratification of schools by region at Wave I by including both strata and cluster variables in the analyses (Chantala, 2006). Second, I retained all Wave III respondents in the dataset and created a subpopulation variable in order to identify respondents who met my eligibility criteria (see Section 4.2), while all others were identified as observations outside the analytic domain and assigned a weight of zero. This strategy guaranteed statistically valid subpopulation analyses (Lee & Forthofer, 2006).

Another important step in the accounting process for studies involving complex survey data is to apply sampling weights to correct for unequal probability of selection and eliminate selection bias (Asparouhov, 2005). In my study, weighting the data would have entailed using the weights that were computed by the Add Health research team for analyses involving participants interviewed at both Wave I and Wave III since my data were drawn from those two waves. For reasons discussed in Section 4.4.4.1, however, I did not apply sample weights. Nevertheless, I performed all analyses using the SAS System, Version 9.2 (SAS Institute Inc., 2008) and Mplus, Version 5.2 (Muthèn & Muthèn, 2008a)—both of which are capable of adjusting estimates for clustering and stratification when computing point estimates and standard errors (Asparouhov, 2005). In analyses performed in SAS, I used the Taylor series (linearization) method for variance estimation because it assumes first stage sampling was done with replacement, an assumption that Add Health can be considered to meet because the first stage sampling fraction was small enough (Chantala, 2003). I used weighted least squares mean and variance adjusted (WLMSV) estimation during the SEM analysis (see Section 4.4.1 for more details) and a significance level of $p \leq 0.05$ for all hypothesis tests. However, I applied a more conservative probability

value of 0.20 or less to the results of bivariate analyses when making decisions about which control variables to include in the multivariate SEM models.[25]

4.4.1. Data Preparation

The measures needed for the analyses were derived from variables that were located in eight different Add Health datasets: the original Wave I and Wave III questionnaire datasets, the Wave I contextual dataset, three datasets containing pregnancy and live birth data, the dataset containing sample design information, and a dataset containing variables that were constructed by the Add Health research team (such as race, family structure, and calculated ages) to assist researchers in the use of the data. All datasets except the pregnancy and live birth datasets consisted of one record per respondent; the pregnancy and live birth datasets consisted of one record per pregnancy so that each respondent could have more than one record. As is clear in Section 4.3, I created the variables I needed for the analysis from existing variables in each of the Add Health datasets based on theoretical and empirical considerations, as well as prior research. I also created dummy variables for all categorical variables with greater than two categories in preparation for the analysis. Then, I merged the eight data sets together in stages using SAS 9.2 (SAS Institute, 2008).

I then screened the data for plausibility, univariate outliers, and non-normality (e.g., skewness and kurtosis) using univariate descriptive statistics (e.g., frequencies, distributions, means, dispersion, etc.). I computed Cronbach's alpha for sets of items to determine reliability, and kappa statistics for similar items obtained from different data

[25]For each categorical indicator with more than two categories, this criterion was applied to the relationship between the outcome variable and each of the dummy variables for the indicator, and to the overall chi-square for the relationship between the outcome variable and all dummy variables for the indicator. Wherever appropriate, both *p* values are reported in Chapter 5.

sources to assess the degree of concordance between them. I also examined a matrix of the correlations between the predictors to check for collinearity.

To further prepare the data for analysis, I also evaluated the presence of missing data in order to determine the most appropriate method for handling it. As stated in Section 4.2, I deleted cases that were missing weights (i.e., respondents who were selected outside the sampling frame), and verified that the strata and cluster variables were non-missing. I also deleted 35 cases that were missing data on gestational age and/or birth weight. The cases excluded based on the study's eligibility criteria were not considered as missing data, however. Instead missing data were defined as missing values for the study's explanatory variables. I assessed the extent of missing data by conducting univariate analyses, and also by examining the rate of missing for each set of variables that I considered representative of a single construct (for example, the percentage of cases missing on all five indicators of depression). The missing data rate for all variables and sets of variables in my study was less than 5%, and the rate of missing across all indicators in the study was approximately 6.5%. I assumed this pattern of missing data to be random and did not pursue the use of missing data routines, such as multiple imputation, to address it.

4.4.2. Analysis for Aim 1

The first aim of the study was to describe the distribution of birth outcomes and the prevalence of adverse birth outcomes for the sample overall and by maternal age and race. To meet this aim, I used univariate and bivariate descriptive statistics. I also performed standard multiple regression to assess the degree to which maternal age and race predicted

birth outcomes.[26] The equation for this analysis took the form: (BIRTH OUTCOME)′ = A + B_1(RACE or AGE) + ERROR, where (BIRTH OUTCOME)′ was the predicted value of BIRTH OUTCOME (i.e., gestational age or birth weight), A was the value of (BIRTH OUTCOME)′ when RACE or AGE was zero, and B_1 represented the regression coefficient for RACE or AGE. A two-way interaction effect of race and age was not included in the analysis because of the data presented in Section 2.1 indicating that no such effect should be expected for this study sample's age group. I also assessed bivariate associations between planned control variables and birth outcomes to determine which of them to retain for inclusion in multivariate regression models. (As a reminder, although the significance level for all hypothesis tests was set at $p \leq 0.05$ for all hypothesis tests, I applied a more conservative probability value of 0.20 or less to the results of bivariate analyses when making decisions about which variables to include in the multivariate models). The regression analyses were then repeated with controls for the retained variables in the model.

4.4.3. Analysis for Aims 2 and 3

The second aim of the study was to examine the relationship between adverse birth outcomes and maternal exposure to family- and neighborhood-level disadvantage in childhood, as well as racial differences in these relationships. Expanding on this examination of direct effects, the third study aim was to explore the behavioral and psychosocial pathways through which exposures to disadvantage in childhood affect birth outcomes and translate into racial disparities in adverse birth outcomes. A typical analytic

[26]Consistent with footnote 21, although gestational age was presumed to be antecedent to birth weight (i.e., a mediator) in my study conceptual model and in the full structural equation model, both gestational age and birth weight were assumed to be associated with race and all other explanatory variables in the model because of the overlap between these two outcomes. They were assessed separately from each other.

approach to these aims would involve a series of race-specific multilevel logistic regression analyses. However, explicit multilevel modeling techniques were not used because, as described in previous studies using Add Health data (e.g., Cubbin et al., 2005), a median of two adolescents were sampled per census tract. This means that data were not sufficiently nested for multilevel modeling. I also did not use logistic regression analyses because I opted to model gestational age and birth weight as continuous variables (see Section 4.3.1 for a discussion of this choice). In addition, I contend that structural equation modeling is a more appropriate analytic technique for the analyses of these two study aims.

4.4.3.1. Overview of Structural Equation Modeling: Structural equation modeling (SEM) facilitates the simultaneous estimation of multiple pathways (and assessment of their relative strength) by accounting for correlated errors among related variables and controlling for measurement error (Bollen, 1989; Kline, 2005). Its use allows for the estimation of direct, indirect, and total effects, making it an ideal method for testing mediation. It is often used when the variables of interest cannot be measured perfectly (Bollen, 1989)—i.e., when they are measured by a set of items or instruments with measurement error or when latent variables are presumed to underlie the measured items and produce dependence among them (Skrondal & Rabe-Hasketh, 2004). I expected this to be the case with, at a minimum, the indicators of neighborhood social processes and psychosocial risk factors included in the study.

4.4.3.2. Rationale for using Structural Equation Modeling: The overwhelming majority of studies of the relationship between adverse birth outcomes and lifecourse, social, or contextual factors cited in the literature review (see Chapter 2) use logistic

regression and do not explore potential pathways between these factors and birth outcomes. A few studies have attempted to examine mediation or indirect effects, yet often they do not analyze all paths in the model[27] (O'Campo & Schempf, 2005; Reagan & Salsberry, 2005). Those that do often rely on multiple logistic regression models—an approach adopted frequently in the epidemiological literature (Misra, O'Campo, & Strobino, 2001). Several researchers have suggested that other techniques, such as SEM, may be more appropriate for these types of analyses (Misra, O'Campo & Strobino, 2001; Kupek, 2006; Kramer et al., 2001). For example, they have noted that traditional approaches to mediation analysis in epidemiology do not take potential measurement error into account, which can lead to residual confounding or incorrect conclusions about direct and indirect effects (Misra, O'Campo, & Strobino, 2001). The modeling of a common cause of observed risk factors and its influence on the outcomes of interest has been considered impossible outside an SEM framework (Kupek, 2006). As some reproductive epidemiologists also have noted, the ability for SEM to model all regression equations simultaneously—i.e., to test all possible relationships between the variables in the model, including mediating effects and possible latent variables—is one major advantage of SEM over separate logistic regression models (Kupek, 2006; DeStavola et al., 2005; O'Campo & Schempf, 2005) and all other analytic techniques, for that matter (Ullman, 2007).

Despite these observations, use of SEM in birth outcomes literature appears to be limited to a handful of studies, most of which appeared in the published literature roughly

[27]For example, they merely analyze the path between the social factor and a more proximate risk factor for adverse birth outcomes, such as behavior or infection (e.g., Culhane et al., 2002), but do not examine the full pathway between the social factor and adverse birth outcomes.

a decade ago (Lobel et al., 1992, 2000; Rini et al., 1999; Zambrana et al., 1999; Sheehan, 1998; Feldman et al., 2000). None of those studies incorporated contextual factors or applied a lifecourse perspective. SEM has been used in lifecourse and neighborhood studies of other health outcomes, however. For example, one study used SEM to examine the relative importance of lifecourse socioeconomic position to self-reported health, while another used SEM to examine the relationships between neighborhood economic indicators, neighborhood social and physical characteristics, and individual health outcomes (Singh-Manoux, Richards, & Marmot, 2005; Franzini et al., 2005). The similarities of these studies to my study, coupled with the work of reproductive health researchers who acknowledge the need for SEM in birth outcomes research, lent conceptual and empirical support to my use of SEM.

4.4.3.3 Model Specification: The first step in SEM is to specify path diagrams of the measurement and structural models based on theory, logic, and prior research on the relationships between key variables (Bollen, 1989). The measurement model is the part of the model that relates the observed variables to latent variables. The latent variable model is the part of the model that relates the latent variables to each other. In my study, these models expanded on Figure 5 (Chapter 3) to include latent variables and the observed variables that are presumed to be associated with them. The original combined model with all latent and observed outcome and explanatory variables is presented in Appendix B. The relationships in the model were directly translated into equations to facilitate model estimation. The steps described in Sections 4.4.3.4 and 4.4.3.7 below resulted in various modifications to the model. These modifications are discussed and illustrated in Chapter 5.

4.4.3.4 Model Identification: Prior to model estimation, I followed several rules and procedures to establish the identification status of the measurement and latent variable portions of the model (Kline, 2005; Bollen, 1989). These included applying the t-rule, scaling rule, and 2-indicator rule to all measurement models. They also included checking for empirical evidence of under-identification in the latent variable models, such as correlations between factors that were close to zero or one and standard errors of the factor loadings that had large variations from one part of the model to other. (See also Section 4.4.3.6).

4.4.3.5 Measurement Model Fit and Re-Specification: I used confirmatory factor analysis (CFA) to test measurement models containing effect indicators. For each model tested, I evaluated overall model fit (i.e., the fit between the sample covariance matrix and the estimated population covariance matrix) by using multiple indices that were robust to model misspecification (Bollen, 1989). These indices included chi-square (χ^2), the comparative fit index (CFI), the Tucker-Lewis index (TLI), the root mean squared error of approximation (RMSEA), the standardized mean residual (SRMR), and the weighted root mean squared residual (WRMR).[28] Lower and non-significant chi-square values indicate better model fit. However, when the sample size is small, or the assumptions underlying the chi-square test statistic are violated, the probability levels may be inaccurate (Hu & Bentler, 1995). Consequently, the other measures of overall model fit listed above were emphasized. I considered CFI's closer to 1 than 0, RMSEA's less than or equal to 0.06, TLI's and CFI's greater than or equal to .95, SRMR's less than 0.05, and WRMR's less than or equal to 1.00 to be evidence of good model fit (Hu & Bentler, 1995, 1999).

[28]The WRMR is used when estimating measurement models with categorical indicators, while the SRMR is computed when estimating models with continuous indicators (Muthèn & Muthèn, 2007).

I also evaluated the component fit of the models, in which case good component fit was suggested by having parameter estimates that were the "right" sign, were statistically significant, and accounted for a high proportion of the variance , i.e., R^2 (Bollen, 2006). Thus, indicators with poor face validity, low loadings (i.e., < 0.40), or low R-squares were considered for elimination. I also examined the correlation residuals or residual covariances, which are small, centered around zero, and symmetrically distributed if the model has good fit (Bollen, 1989). These assessments of model fit, as well as theoretical considerations, were used to make decisions about model re-specification, if needed to produce a better fit[29] or to test alternate hypotheses about relationships in the model. If deemed appropriate, models were re-specified after removing poor items or making other model adjustments and results were re-examined for evidence of improved or more parsimonious fit. Such evidence came from chi-square difference tests that I performed to compare the less restrictive (i.e., unconstrained or less parsimonious) and more restrictive (i.e., nested, constrained, or more parsimonious) models; non-significant chi-square statistics produced by these tests indicated that the more restrictive model provided a better fit to the data, while significant chi-square values indicated that the less restrictive model provided a better fit to the data.

4.4.3.6 Treatment of Causal Indicators in the Analysis: It is important to point out that some of the indicators of family-level and neighborhood level socioeconomic conditions in my original structural equation model were modeled as causal or formative

[29]It should be noted, however, that SEM is a confirmatory technique; thus, few modifications of the model were tested for the purpose of improving fit so as to avoid moving into exploratory data analysis and inflating Type I error levels (Ullman, 2007).

indicators, rather than effect indicators, of the latent variables. Although not often found in research, the appropriate specification of SES as a causal indicator model has been widely discussed in the SEM literature (e.g., MacCallum & Browne, 1993; Weston & Gore, 2006). In particular, it has been observed that socioeconomic status is a reflection of one's income and education, unlike other latent variables such as depression which underlie (or cause) responses to questions about depressive symptoms.

When a latent variable only has causal indicators, it results in an endogenous construct that is a linear combination of those indicators, with a disturbance term, and without error terms associated with the indicators (MacCallum and Browne, 1993). This construct cannot be estimated using CFA, as effect indicator models can be, because it is not identified.[30] In order to identify it (i.e., to satisfy the t rule and scaling rule for identification), I fixed the path from one of the causal indicators to the construct or fixed a path from the construct to another variable and fixed its residual variance to zero (Bollen & Davis, 1994; MacCallum & Browne, 1993). In the overall structural equation model, I ensured that two paths were emitted from the causal indicator latent variable in order to identify it and to enable freeing the disturbance term to be estimated (MacCallum & Browne, 1993; Bollen & Davis, forthcoming).

When a latent variable has both causal and effect indicators, it is referred to as a MIMIC (Multiple Indicators Multiple Causes) model (Hauser & Goldberger, 1971; Kline, 2005). Unlike causal measurement models, MIMIC models can be tested using a variant of

[30]Procedures for confirming or refuting the causal indicator model specification do exist (e.g., confirmatory tetrad analysis (Bollen & Ting, 2000), but I did not deem it necessary to use them because of the widespread acceptance of the causal indicator model specification for SES in the SEM literature.

the CFA procedures I discuss in Section 4.4.3.5 above. For these models, I first tested the effect indicator portion of the model (if identified). Then I added the causal indicators, re-estimated the model, and checked for changes in the factor structure and whether direct effects between the causal indicators and effect indicators made theoretical sense. (MIMIC models have been referred to as "CFA plus covariates" models (Muthèn & Muthèn, 2008b)).

Multicollinearity in Causal Measurement Models. As other researchers have suggested (Culhane & Elo, 2005; Land, McCall, & Cohen, 1990), some of my causal indicators of neighborhood socioeconomic conditions were highly correlated. For example, the correlation between proportion in poverty and proportion receiving public assistance, unemployment rate, and proportion of female-headed households were 0.83, 0.78, and 0.76, respectively. When all neighborhood variables were entered into a regression model, these variables had the highest variance inflation factors (VIFs), although they were less than five, suggesting that concerns about multicollinearity may be unwarranted. Nevertheless, multicollinearity is a concern in SEM because it can result in empirical under-identification and unstable estimates for the factor loadings, inflate the size of error terms, or make it difficult to identify the distinct influences of each indicator on the construct (Bollen, 1989; Bollen & Lennox, 1991). Typical solutions for dealing with multicollinearity, such as factor analysis and principal components analysis (PCA), assume that indicators are effect indicators and often result in the elimination or combination of indicators (Tabachnick & Fidell, 2007). Although used in recent research on neighborhood deprivation and adverse birth outcomes (Messer et al., 2006b), both solutions also are problematic because they could alter the meaning of the construct (Bollen & Lennox, 1991) or make interpreting parameter

estimates difficult (Diamantopoulos, Riefler, & Roth, 2007). As an alternative, I retained all of the indicators and specified highly correlated indicators with and without equality constraints imposed on them during the SEM analysis, thereby testing the assumption that the indicators represent the construct equally well. I considered multicollinearity to be absent if I obtained separate estimates for each of the indicators (and if all statistical operations functioned properly) when the equality constraints were lifted.

4.4.3.7. Structural Model Estimation: After satisfactory measurement models were established, structural models representing the study hypotheses were specified and model parameters estimated. Final judgments about support for or rejection of hypotheses were only made after adjusting for control variables. It seems customary among SEM studies reported in the published public health and sociology literature, including those involving birth outcomes or neighborhood context, for researchers to trim all non-significant paths from the structural model after control variables are entered and then to draw conclusions from the trimmed model (e.g., Ross & Mirowsky, 2009; Lobel, Dunkel-Schetter, & Scrimshaw, 1992; Lobel, DeVincent, Kaminer, & Meyer, 2000). I departed somewhat from this strategy because dropping theoretically important indicators from the model would weaken my ability to interpret the remaining variables within the context of the larger theory-driven model. In addition, this practice presumably can alter model fit to the data. Thus, I followed a procedure similar to the one described by Thrasher (2005) which involved first estimating a baseline adjusted model with the outcome variable simultaneously regressed on all potential control variables (including those retained from Aim 1). Then, I performed a nested comparison with a reduced model in which only the

non-statistically significant paths originating from the control variables in the baseline model were constrained to zero. A chi-square difference test was performed between the baseline (i.e., unconstrained) and reduced (i.e., constrained) models; a non-significant chi-square value indicated that the reduced model provided a better fit to the data, while a significant chi-square value indicated that the baseline model provided a better fit to the data. Path estimates representing the key study hypotheses were then examined only for the best-fitting adjusted model. These estimated path coefficients were interpreted as indirect and direct effects, which were summed to determine total effects. A pattern of significant indirect effects but non-significant direct effects was considered to be strong evidence for mediation (Kline, 2005).

4.4.3.8 Hypothesis Tests for Moderation: A typical approach for testing the hypothesis that the relationship between childhood exposure to disadvantage and adverse birth outcomes varies by race in structural equation modeling would be to assess measurement invariance (MI)—i.e., the degree to which measurements across the two racial groups yield equivalent measures of the same attributes (Horn & McArdle, 1992)—using multiple group analysis (MGA). While Mplus has a built in command for conducting MGA, it cannot be used simultaneously with the command for conducting subpopulation analysis of complex survey data. To my knowledge, only three alternatives to MGA with subpopulations exist: (1) subsetting the data (i.e., removing all cases outside the analytic domain from the dataset) so that the subpopulation command is not required; (2) adding interaction terms between race and the latent variables to the model; or (3) performing stratified analysis. The first option is only appropriate if subsetting the data does not result

in a loss of primary sampling units (i.e., clusters) and strata. (This is why I state earlier that retaining all cases in the dataset—even those outside the subpopulation of interest—is the only way to guarantee statistically valid subpopulation analyses of complex survey data). Unfortunately, my analyses necessarily resulted in the loss of at least one primary sampling unit because one of the schools sampled in Add Health was an all-boys school.

The other two alternatives also have limitations. Adding interaction terms into measurement models assumes that indicator measurement error does not differ across race so that only differences in factor loadings can be examined at the construct level (S. Christ, personal communication, 2009). Stratified analysis, which is similar to the first stage of MGA in which both groups are tested together with all parameters free across groups, only allows a researcher to observe differences between parameter estimates across groups. Neither stratified analysis nor adding interaction terms allows for equality constraints to be placed on all parameters in the two separate models in order to test for measurement invariance between them, as one would do in MGA.

I opted to perform stratified analysis instead of using interaction terms because of the sheer complexity of my model. In addition, stratified analysis has been used to assess moderation in other birth outcome studies involving SEM (e.g., Rini et al., 1999), as well as SEM studies of other health issues (e.g., Thrasher, 2005). In preparation for this analysis, I created two new subpopulation variables—one to identify the Black sample, and one to identify the White sample (for the same reasons discussed on page 77 with regard to the full sample). Then, as the aforementioned studies did, I ran separate models for each racial group and examined differences in primary structural parameters between them and in

comparison to the full sample. For each group, a baseline adjusted model was estimated. If warranted by the results, I performed nested comparisons to determine whether constraining non-statistically significant paths to zero resulted in substantial reductions in model fit. Differences in the non-statistically significant paths and consistent increases or decreases in the strength of the coefficients across the race-specific models were considered evidence that the relationships in the hypothesized model varied by race (Thrasher, 2005).

4.4.4. Sensitivity Analysis

In research, sensitivity analyses are often conducted to determine if results are sensitive (or robust) to variations in parameters, model specification, and/or variable definitions (e.g., Colen et al., 2006). In my study, I performed sensitivity analysis for two main reasons: (1) to assess the appropriateness of the original full sample weights for my subpopulation and alternatives to using them, and (2) to assess alternate operationalizations of key study variables to determine if conclusions drawn from the results using the alternatives would differ from the original results.

4.4.4.1 Appropriateness of the Sampling Weights: During preliminary design-based regression analyses (i.e., analyses that incorporated the weight, stratification, and cluster variables), I noticed that I obtained some results that were contrary to findings that have been found widely throughout the literature. Furthermore, the design effects[31] were sometimes quite large. Given that my sample represents only 10% of the overall Add Health Wave III sample and only 20% of female participants, questions can be raised about

[31]Design effects are the factors by which the variance of an estimated mean increases after weighting the data. Larger design effects may be an indication that the data are less reliable—the larger the effects are, the greater the variances, the more difficult it is for statistical tests to be significant, and the wider the confidence intervals (Lee & Forthofer, 2006; Kish, 1992).

the appropriateness of the sampling weights for my sample. The Add Health sampling weights are full sample weights. Thus, I examined box plots (see Appendix C) and univariate statistics for the sampling weight variable and discovered what has been referred to in the literature as "extreme variation" in the values among my sample (Lee & Forthofer, 2006; Izrael, Battaglia, & Frankel, 2009). In particular, values of the sampling weights ranged from 28.36 to 5634.59 among Blacks (*coefficient of variation (CV)* = 88.4),[32] and from 20.77 to 6177.67 among Whites (*CV* = 81.4). A considerable number of outliers also were present in both groups.

Such variation can serve to increase the variance substantially and perhaps render the weighted analysis inefficient when compared with the unweighted analysis (Lee & Forthofer, 2006). In addition, extreme weights contribute to design effects (and, correspondingly, to the true sample variance) and result in some cases becoming disproportionately important (called "influential cases") in estimating overall statistics simply because of the magnitude of their weight and not because of their value on any given variable (Botman, 1993). For this reason, I performed sensitivity analysis to determine whether alternate analytic procedures would result in the same or different results when compared to the weighted and unweighted results. Two alternate procedures were used for this comparison: (1) weighted analysis after weight trimming, and (2) model-based analysis.

Weight trimming procedures are used to reduce the impact of extreme values on the variances and increase the precision of the estimates (and, therefore, the design effects)

[32]Coefficient of variation (CV) refers to the ratio of the standard deviation to the mean multiplied by 100. The higher the CV, the greater the dispersion of the variable. It has been suggested that a CV of no more than three is desirable (C. Weisen, personal communication, 2009).

during weighted (design-based) analysis (Izrael, Battaglia, & Frankel, 2009; Botman, 1993). To my knowledge, there are no rules-of-thumb to guide researchers in defining extreme weights and no gold standard (or even standard practice) for trimming weights; thus, an infinite number of weight trimming procedures exist (C. Weisen, personal communication, 2009). Several approaches seem to be more common: (1) adjusting any weight larger than 3 to 5 times the mean or median weight to be equal to that limit, (2) adjusting any weight larger than the median weight plus 5 or 6 times the interquartile range of the weights to be equal to that limit, or (3) adjusting weights above a certain percentile (e.g., 95th) in the distribution of weights to be equal to that percentile (Izrael, Battaglia, & Frankel, 2009). Of these strategies, I chose the strategy that would result in the greatest degree of trimming—i.e., truncating weights above the 95th percentile to the value of the 95th percentile—because of the high level of variation in the sampling weights. I implemented this strategy both for the sample overall and for each racial group individually because of the key role of race in this study and because the distributions of the sampling weights varied within the groups. Figures C.1 to C.3 in Appendix C compare the distributions of the weights following each of these trimming procedures. It is clear that neither of the trimming methods resulted in a substantial difference in the variability of the weights (e.g., in the race-specific trimming, the coefficient of variation changed from 81.4 to 76.7 for Whites, and 88.4 to 77.8 for Blacks). Nevertheless, after trimming the weights, I ran portions of my planned analyses and compared the results to the weighted (untrimmed) and unweighted results. The findings of this comparison are presented in Appendix C.

I also ran the same portions of my planned analyses using (unweighted) model-based analysis (Lee & Forthofer, 2007). Model-based analysis, which involves augmenting the model with survey design variables (or the variables that were used to create them) by including them among the independent variables or as control variables, reduces the bias created by performing unweighted analysis and is more efficient (i.e., yields smaller standard errors) than weighted design-based analysis if the model is correctly specified (Lee & Forthofer, 2007; Winship & Radbill, 1994). This procedure required me to include dummy variables provided by the Add Health research team that were used to flag respondents in the core sample as well as eight of the special over-samples: disabled, blacks with a college educated parent, twin, full sibling, half sibling, non-related adolescent, PAIR school, and sibling of twins.[33] Bivariate analyses of these sample flags revealed that few respondents in my sample were simultaneously flagged by two different sample flags. (The highest proportion was 2.8%). Thus, no interactions between the sample flags were included in the model-based sensitivity analysis. The results of this analysis are presented alongside the weighted, unweighted, and trimmed-weight weighted results in Appendix C.

4.4.4.2. Appropriateness of Operational Definitions of Variables: I also performed post hoc sensitivity analysis to determine whether the results of the study differed (or whether conclusions drawn from the results would differ) depending on the operational definitions I used. First, because of concerns that cumulative risk scores may mask the true effects of the items that comprise the score, I compared the results of some analyses using

[33]Add Health over-sampled 11 groups but I excluded three – Cuban, Puerto Rican, and Chinese – because I did not include these racial/ethnic groups in my study.

the cumulative risk scores with those using the individual items. The results of these comparisons are discussed wherever appropriate in Chapter 5.

Second, to determine whether the results of my analyses would be consistent if I used categorical versions of the outcome variables (i.e., LBW and PTB) instead of continuous versions (i.e., birth weight and gestational age), I compared some of the analyses of racial differences using both versions of the variables. Because the conclusions were the same regardless of this difference in operational definition, I performed the majority of the analyses as originally planned.

Finally, the values of birth weight (measured in grams) were so much larger than all other variable values in the study that it raised concerns about the possibility of computational errors. (The values of the unstandardized residual variances for birth weight also were so large that they exceeded the space allotted in the Mplus output and, therefore, could not be viewed). In addition, birth weight demonstrated some skewness and kurtosis in this study (i.e., -0.74 and 2.68, respectively). Thus, I performed a natural logarithm transformation of the variable. Doing so, however, resulted in values that were more skewed and kurtotic (i.e., -2.79 and 16.72, respectively) than the original values of birth weight. So I instead re-scaled the variable in some analyses by a factor of 1/100 or 1/1000. This re-scaling did not change the results obtained in analyses (i.e., *p* values remained the same, coefficients and standard errors were the same, albeit scaled down by a factor of 1/100 or 1/1000). Thus, the unstandardized results could still be interpreted in the original units (i.e., in grams).

4.5 Power Analysis

An important task for any study is to determine the level of statistical power produced by a given sample size to detect an effect if one is present. To my knowledge, no absolute standards, or rules of thumb, for determining the sample size needed to achieve sufficient statistical power can be found in the SEM literature. Suggestions have ranged from having 10 to 20 times as many cases as variables to having five cases per parameter estimate (Garson, 2008; Bentler & Chou, 1987)—none with any empirical support. On the other hand, some researchers have developed procedures for determining sample size requirements in SEM studies. For example, MacCallum, Browne, and Sugawara (1996) describe an interval-halving procedure for determining sample size in covariance structure modeling research based on upper and lower limits of the RMSEA.

With a sample size of 1,557, degrees of freedom equal to 43,[34] a desired alpha (α) level of 0.05, and null and alternate values for the RMSEA of 0.05 and 0.08, respectively,[35] I used the interval-halving procedure to determine the power of the test of the null hypothesis H_0: RMSEA $\leq$ 0.05 (i.e., a close fit), if the true value of RMSEA was 0.08 (i.e., not a close fit). I found that my sample size afforded me over 80% power to reject the null hypothesis that the model was a close fit if it really was not a close fit. I reached the same conclusion when I switched the alternate value of RMSEA to 0.01 in order to determine the

[34]The WLSMV estimator used during the SEM analysis computes degrees of freedom in a manner that produces results other than what would be produced if hand-calculated. Thus, I hand calculated the degrees of freedom by subtracting the number of unknowns (i.e., parameters to be estimated) from the number of knowns (i.e., variances and covariances among the observed variables). This hand calculation resulted in a value of 137; WLSMV produced a value of 43. Since the WLSMV estimate is the more stringent of the two with respect to the statistical power and minimum sample size calculation, I used that value in the interval-halving procedure.

[35]These values were recommended by the authors and, as discussed previously, RMSEA's < 0.06 are indicative of good fit. A value of zero indicates exact fit.

likelihood of rejecting the null hypothesis that the fit was not close (i.e., H_0: RMSEA ≥ 0.05), if the model was actually a very close fit (i.e., RMSEA = 0.01). In fact, both tests suggested that the minimum sample size needed to achieve 80% power in my study (with degrees of freedom, alpha, null, and alternate RMSEA values as listed above) was less than 300. This meant that I also had adequate power to detect an effect during the stratified analysis I performed to determine whether race moderated the relationships in the model.

It should be noted that the other guidelines mentioned earlier in this section—i.e., 10 to 20 times the number of variables or five times the number of parameters to be estimated—would have resulted in somewhat similar, although not exact, conclusions to those generated from the interval-halving procedure. Using the variable "method" with 24 observed variables, my overall sample size as well as my race-specific subsamples exceed 20 times that number. Using the parameter "method" with 137 parameters to be estimated, my overall sample size exceeds five times that number but the size of my subsample of Black women does not.

CHAPTER 5

RESULTS

This chapter describes the study sample and reports the study findings by study aim. I conclude the chapter with a summary of the support for the study hypotheses.

5.1. Sample Description

Tables 7, 8, 9, and 10 present descriptive statistics for the study sample. Approximately 68% of women in the sample were White and 32% were Black (Table 7). The mean age of the women when they were interviewed at Wave III was roughly 22, with most of them (78%) having at least a 12th grade education. The parents of only 72% of the sample reported their family income at Wave I; of those, less than a quarter (21%) lived in households where the income was greater than the 1994 poverty level (~$15,000). Approximately 40% of the respondents' parents had more than a high school education, with 18% having at least a college education. Sixty percent of the women in the sample were part of families with two biological or adoptive parents at the time of the Wave I interview. Women in the sample were significantly different from women who were excluded from the study on all these characteristics. Compared to excluded women, women in the study were slightly older and more likely to have completed only the 12th grade at Wave III. They also were more likely to have lived in impoverished households and

neighborhoods, in a single-parent or surrogate family, and with a parent(s)/guardian(s) who had no more than a high school education at Wave I.

Table 7. Comparison between Women Included in the Study and Excluded Women (*N* = 7,563)

Variables	Range/ Values	Total (*n* = 7,563) % or Mean (SE)[a]	Included (*n* = 1,557) % or Mean (SE)	Excluded (*n* = 6,006) % or Mean (SE)	*p* value[b]
Race	0 – 2				n/a
White		65.3	67.9	64.7	
Black		23.6	32.1	21.4	
Other		11.1	0.0	13.9	
Age *(at Wave III)*	18 – 27	21.9 (.14)	22.3 (.10)	21.8 (.16)	<.0001
Highest Education *(at Wave III)*	1 – 3				<.0001
More than 12th grade		58.0	33.8	64.2	
12th grade		31.2	44.3	27.8	
Less than 12th grade		10.9	21.8	8.0	
Family In Poverty *(at Wave I)*	0 – 1				<.0001
Yes		18.3	26.8	16.2	
No		80.5	71.4	82.9	
Parental Education *(at Wave I)*	1 – 4				<.0001
College grad or more		34.3	18.3	38.4	
More than high school		21.3	22.4	21.0	
High school grad or GED		28.5	36.2	26.5	
Less than high school		14.3	20.4	12.8	
Family Structure *(at Wave I)*	0 – 1				<.0001
Two parent		70.0	59.5	72.8	
Single/Surrogate parent		30.0	40.5	27.2	
Neighborhood Poverty *(at Wave I)* *(Proportion below poverty level)*	0 – 0.90	0.16 (.01)	0.20 (.02)	0.15 (.01)	<.0001

Abbreviations: *SE*, standard error; %, percent

[a] % (unweighted) reported for categorical variables; mean (SE) reported for continuous variables; percentages for each variable do not add up to 100% due to missing values

[b] *p* determined from Rao-Scott χ^2 test for categorical variables and *t*-test for continuous variables

These differences are mirrored in the comparison of Black and White women in the study to those who were not in the study. Table 8 shows that both Black and White women

in the study were older and more likely to have completed only the 12th grade at Wave III than their excluded counterparts. They also were more likely to have lived in impoverished households and neighborhoods, in a single-parent or surrogate family, and with a parent(s)/guardian(s) who had no more than a high school education at Wave I.

Table 8. Comparison between Included and Excluded Women, by Race (*N* = 7,563)

Variables	Range/ Values	Black % or Mean (SE)[a]		White % or Mean (SE)	
		Included	Excluded	Included	Excluded
Age *(Wave III)*	18 - 27	22.1 (.17)***	21.7 (.20)	22.3 (.10)***	21.7 (.13)
Highest Education *(at Wave III)*	1 – 3				
More than 12th grade		42.2***	58.8	29.9***	66.7
12th grade		41.4	32.3	45.7	25.8
Less than 12th grade		16.4	8.9	24.4	7.4
Family in Poverty *(at Wave I)*	0 – 1				
Yes		21.0*	17.0	12.3***	7.6
No		47.2	55.1	63.0	71.9
Parental Education *(at Wave I)*	1 – 4				
College grad or more		24.2***	36.3	15.5***	38.9
More than high school		20.6	21.5	23.2	21.4
High school grad or GED		33.0	26.1	37.7	27.8
Less than high school		19.0	14.5	21.0	11.0
Family Structure *(at Wave I)*	0 – 1				
Two parent		42.6*	49.0	67.5***	80.1
Single/Surrogate parent		57.4	51.0	32.5	19.9
Neighborhood Poverty *(at Wave I)* *(Proportion below poverty)*	0 – 0.90	0.29 (.02)**	0.26 (.02)	0.16 (.01)***	0.12 (.01)

Abbreviations: *SE*, standard error; *%*, percent

[a] % (unweighted) reported for categorical variables; mean (SE) reported for continuous variables; percentages for each variable do not add up to 100% due to missing values

[b] Significance levels reported for the difference between sampled Black women and non-sampled Black women, and between sampled White women and non-sampled White women; *p* determined from Rao-Scott χ^2 test for categorical variables and *t*-test for continuous variables;

‡ p <.10; * p <.05; ** p <.01; *** p <.001

Tables 9 and 10 below focus exclusively on women in the sample and the characteristics intended as control variables. These tables are stratified by race because of

the study's emphasis on racial disparities. (Tables comparing Black and White women on key study variables are provided in Appendix D).

Table 9. Characteristics of the Sample: Demographic and Prenatal Characteristics, by Race

Variables	Range/ Values	Total (N = 1,557) % or Mean (SE)[a]	Maternal Race: Black (n = 500) % or Mean (SE)	Maternal Race: White (n = 1,077) % or Mean (SE)	p value[b]
Age (years) on Infant's Birth Date	14.5 – 25.8	20.0 (.08)	19.7 (.14)	20.1 (.09)	0.0080
18 Years Old or Older (Yes)		*82.7*	*81.0*	*85.4*	*0.0422*
Education on Infant's Birth Date	1 – 3				0.0030
More than High School		26.1	31.2	23.7	
High School or <20 and No HS		64.0	62.2	64.8	
≥20 and No High School		6.7	4.6	7.8	
Prenatal Substance Use Score	0 – 3	0.3 (.02)	0.1 (.02)	0.3 (.03)	<0.0001
Smoked During Pregnancy		*18.0*	*4.0*	*24.6*	*<0.0001*
Used Alcohol During Pregnancy		*4.1*	*1.8*	*5.2*	*<0.0001*
Used Drugs During Pregnancy		*3.1*	*2.4*	*3.3*	*0.3951*
Adequacy of Prenatal Care	1 – 4				0.0010
Very Adequate		80.0	74.2	82.7	
Somewhat Adequate		11.9	16.2	9.8	
Inadequate		4.7	4.8	4.6	
Prenatal Health Risk Score	0 – 3	0.10 (.01)	0.11 (.02)	0.10 (.01)	0.6205
Relationship Status during Pregnancy	1 – 4				<0.0001
Married		28.7	9.2	38.0	
Cohabitating		29.4	23.8	32.0	
Steady Relationship		16.6	29.4	10.6	
No relationship		23.1	34.6	17.7	
Father Attended Prenatal Care	0 – 1				<0.0001
Yes		68.5	60.6	72.2	
No		27.5	35.0	23.9	

Abbreviations: *SE,* standard error; *%,* percent

[a] % (unweighted) reported for categorical variables; mean (SE) reported for continuous variables; percentages for each variable do not add up to 100% due to missing values

[b] p determined from Rao-Scott χ^2 test for categorical variables and t-test for continuous variables

According to Table 9, Black and White women in the sample had a mean maternal age (for the birth included in the analysis) of approximately 20 years old. In addition, most women in each group were 18 years old or older (i.e., 81.0% Black, 85.4% White). Mean prenatal health risk scores were negligible in both groups of women, reflecting their low rates of diabetes (0.4%), bacterial vaginosis (2%), and obesity (7.4%) histories. However, the Black sample (n = 500) differed significantly from the White sample (n = 1,077) on most other sociodemographic and pregnancy characteristics. A greater proportion of Black women had obtained more than a high school education or beyond than had White women, although the majority of both groups had met or exceeded educational expectations for their age (as defined in Section 4.3). Compared to Whites, Blacks were less likely to: be married at the time of their pregnancy; have initiated prenatal care during the first trimester of their pregnancy; or have had the baby's father present during their prenatal care visits. Black women also had a lower mean prenatal substance use score than White women—a difference largely driven by the racial difference in use of cigarettes. Specifically, Black women were nearly six times less likely than White women to smoke cigarettes during their pregnancies (4.0% versus 24.6%, respectively).

Some racial differences in the pregnancy histories of women in the sample were present as well (Table 10). The mean number of births prior to the Wave I interview was slightly higher among Black women than White women and Black women were significantly less likely than White women to have been nulliparous prior to Wave I, although nearly all Black and White women in the sample were nulliparous prior to the Wave I interview. Three quarters of both groups only had one birth (i.e., the birth included

in the analysis) after the Wave I interview. As a result, no significant difference between Blacks and Whites was found in the mean number of births after the Wave I interview nor in the overall number of births reported at Wave III. The mean lag time between the Wave I interview and first birth after that date was only about 2-1/2 months shorter for Blacks than Whites.

Table 10. Characteristics of the Sample: Pregnancy History and Other Control Variables, by Race

Variables	Range/ Values	Total (N = 1,557) % or Mean (SE)[a]	Maternal Race: Black (n = 500) % or Mean (SE)	Maternal Race: White (n = 1,077) % or Mean (SE)	p value[b]
Time to Birth (years)	0.01 – 6.30	3.6 (.05)	3.4 (.10)	3.6 (.06)	0.0348
Live births before Wave I	0 – 2	0.04 (.01)	0.06 (.01)	0.03 (.01)	0.0184
Nulliparous (Yes)		*95.8*	*93.8*	*96.7*	*0.0051*
Live births after Wave I	1 – 4	1.3 (.02)	1.3 (.03)	1.3 (.02)	0.6043
Uniparous (Yes)		*75.1*	*77.0*	*74.3*	*0.3149*
Total number of live births	1 – 6	1.4 (.02)	1.4 (.04)	1.3 (.03)	*0.3587*
Length of residence in neighborhood before Wave I (years)	0 – 19	6.3 (.16)	6.2 (.28)	6.4 (.20)	0.5802
Age moved to the neighborhood	0 – 19	9.2 (.22)	9.9 (.33)	9.6 (.20)	0.9591
Before Age 12 (Yes)		*50.7*	*47.8*	*52.1*	*0.1330*

Abbreviations: *SE*, standard error; %, percent

[a] % (unweighted) reported for categorical variables; mean (SE) reported for continuous variables; percentages for each variable do not add up to 100% due to missing values

[b] p value for race determined from Rao-Scott χ^2 test for categorical variables and t-test for continuous variables

No significant racial differences emerged in the other control variables listed in Table 10. For both groups of women, the mean length of residence in the neighborhood in which their Wave I interview occurred was about six years; the mean age at which they moved

there was just under 10 years old. Approximately half the women in the study moved to the residence in which their Wave I interview took place before becoming an adolescent.

5.2. Results for Aim 1

Aim 1: To describe the distributions of birth weight and gestational age, and the prevalence of adverse birth outcomes, overall and by maternal age and race

5.2.1. Distribution and Prevalence of Birth Outcomes, Overall

Among the reference births in the sample, birth weights ranged from 567.0 grams to 4848.9 grams (M = 3289.74, SE = 12.71),[36] and gestational ages ranged from 28 weeks to 40 weeks (M = 39.3, SE = 0.04). When dichotomized, these data translated into a LBW rate of 6.2% and PTB rate of 6.9%. The prevalence of both outcomes in this sample was lower than the overall prevalence for all women of childbearing age in other national data. On the other hand, about two-thirds (64%) of infants whose birth weights qualified as LBW also had gestational ages that qualified as PTB, demonstrating the same degree of co-occurrence found in other national data (as discussed in Section 2.1).

Table 11 shows unadjusted bivariate associations between birth weight, gestational age, and the factors treated as control variables because of their known or suspected relationship to birth outcomes. Being unmarried or not cohabitating with the baby's father during pregnancy, and not having the baby's father present during prenatal care, were correlated with significantly lower birth weight but not with gestational age.

[36]Recalling the formulas presented in Section 4.3.1 (i.e., 1 pound = 453.59 grams, and 1 ounce = 28.35 grams), this translates into a birth weight range of 1.25 pounds to 10.69 pounds (M = 7.3 pounds, SE = 0.03). To aid interpretation of birth weights and regression coefficients from this point forward, the following reference points can be used: 113.4 grams = 4 ounces = ¼ pound; 226.8 grams = 8 ounces = ½ pound.

Table 11. Bivariate Associations between Birth Outcomes and Sociodemographic and Prenatal Characteristics (*N* = 1,557)

	Gestational Age			Birth Weight		
Variables	***B***[a]	**(95% CI)**	***p***	***B***[a]	**(95% CI)**	***P***
Age (years) at Infant's Birth	-0.03	(-0.07, 0.03)	0.207	8.75	(-5.64, 23.13)	0.233
Education at Infant's Birth						
College Education or More	-0.06	(-0.29, 0.16)	0.588	30.53	(-36.44, 97.50)	0.372
High School or <20 & No HS[b]	----	----	----	----	----	----
≥20 and No High School	-0.13	(-0.58, 0.32)	0.587	-23.16	(-135.08, 88.77)	0.685
Prenatal Health Risk Score	-0.20	(0.51, 0.12)	0.228	-48.12	(-120.19, 23.95)	0.191
Prenatal Substance Use Score	0.08	(-0.05, 0.22)	0.224	-30.59	(-74.21, 13.04)	0.169
Smoked During Pregnancy						
Yes	*0.04*	*(-0.16, 0.23)*	*0.710*	*-62.79*	*(-129.44, 3.86)*	*0.065*
No[b]	----	----	----	----	----	----
Drank During Pregnancy						
Yes	*0.21*	*(-0.10, 0.52)*	*0.186*	*62.63*	*(-41.63, 166.88)*	*0.239*
No[b]	---	----	----	----	----	----
Used Drugs During Pregnancy						
Yes	*0.21*	*(-0.01, 0.74)*	*0.058*	*-41.43*	*(-168.54, 85.68)*	*0.523*
No[b]	----	----	----	----	----	----
Adequacy of Prenatal Care						
Very Adequate[b]	----	----	----	----	----	----
Somewhat Adequate	0.04	(-0.05, 0.06)	0.773	-28.16	(-116.45, 60.12)	0.532
Inadequate	0.23	(-0.01, 0.06)	0.096	-54.97	(-176.26, 66.31)	0.374
Relationship Status						
Married[b]	----	----	----	----	----	----
Cohabitating	0.08	(-0.13, 0.29)	0.463	-8.57	(-72.49, 55.35)	0.793
Steady Relationship	-0.05	-0.33, 0.24)	0.755	-106.46	(-181.90, -31.02)	0.006
No relationship	-0.07	(-0.33, 0.19)	0.622	-101.46	(-179.91, -23.00)	0.011
Father Attended Prenatal Care						
Yes[b]	----	----	----	----	----	----
No	-0.09	(-0.08, 0.03)	0.396	-78.12	(-146.15, -10.09)	0.024
Time to Birth (years)	-0.02	(-0.07, 0.04)	0.519	8.21	(-7.28, 23.69)	0.299
Nulliparous prior to Wave I						
Yes[b]	----	----	----	----	----	----
No	0.20	(-0.12, 0.52)	0.227	84.63	(-51.89, 221.13)	0.224
Total number of births	-0.01	(-0.16, 0.14)	0.854	-21.13	(-67.00, 24.75)	0.367

[a] Regression coefficients are unstandardized and based on unweighted data

[b] Reference category

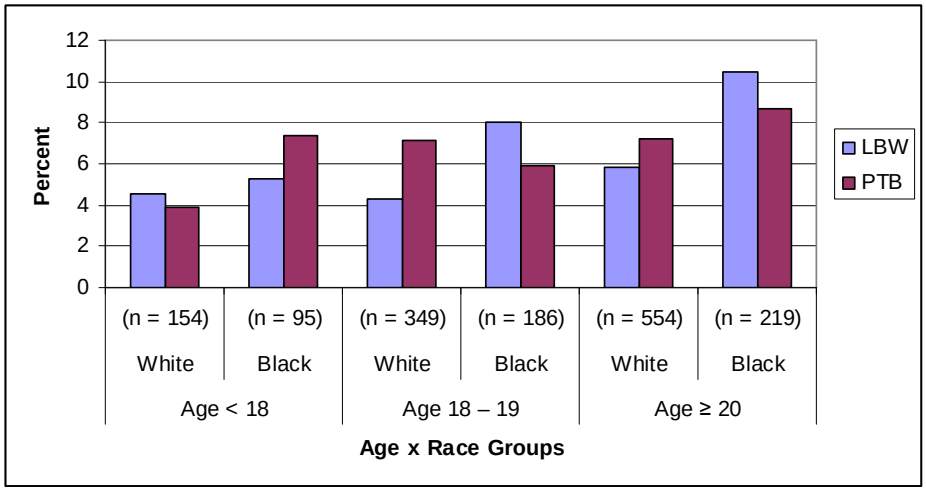
Cigarette use during pregnancy had a borderline significant relationship with lower birth weight, while drug use during pregnancy and having inadequate or no prenatal care had borderline significant relationships with gestational age. Other control variables that met the cutoff criterion of $p \leq .20$ were prenatal health risk and substance use scores. Both were associated with lower birth weights but not gestational age.

5.2.2. Distribution and Prevalence of Birth Outcomes, by Maternal Age

As noted in Table 11, differences in birth weight by maternal age were non-significant ($\beta = 8.75$, $SE = 7.34$, $p = 0.23$), as were differences in gestational age by maternal age ($\beta = -.03$, $SE = 0.021$, $p = 0.21$). Likewise, when birth weight and gestational age were dichotomized, the odds of these outcomes occurring did not differ by maternal age (*OR* = 1.004, *95% CI* = [0.926, 1.088], $p = 0.93$ for LBW; *OR* = 1.011, *95% CI* = [0.924, 1.107], $p = 0.81$ for PTB). Thus, I found no support for my hypothesis that the prevalence of adverse birth outcomes would decrease with increasing maternal age.

Figure 9 helps explain these findings. It shows the general relationship between maternal age, LBW, and PTB for Black and White women in the sample, using age categories similar to those that are commonly found in the published literature for this relationship (e.g., Rich-Edwards et al., 2003). According to the figure, the prevalence of LBW increases with increasing maternal age for Blacks and Whites; the same is true for Whites with respect to PTB, while Blacks demonstrate a U-shaped curve for PTB. (In the 18 – 19 year old age group, Blacks are 17% less likely to report a preterm birth than Whites). These patterns are not statistically significant, and are contrary to those found in national data for this age group (see Table 4 and Figures 1 and 2 in Section 2.1).

Figure 9. Percent* LBW and PTB by Age and Race* (*N* = 1,557)



* Percentages are unweighted

5.2.3. Distribution and Prevalence of Birth Outcomes, by Maternal Race

As Table 12 below shows, infants born to White women had a mean birth weight of 3343.7 grams (i.e., 7 pounds, 6 ounces) and an average gestational age of 39.3 weeks, while infants born to Black women in the sample averaged 3175.2 grams (i.e., 7 pounds, 0 ounces) although they also averaged 39.3 weeks of gestation. The correlations between birth weight and gestational age were 0.60 and 0.57 for Whites and Blacks, respectively. Of the White infants whose birth weights qualified as LBW, 58% also had gestational ages that qualified as PTB; this pattern was true for 68% of Black LBW infants. Despite this overlap, I found no difference in mean gestational age between the groups, although the difference in mean birth weight between them was statistically significant.

The magnitude of the racial difference in mean birth weight seems somewhat modest (i.e., 167.99 grams or 5.9 ounces), but it translates into a more noticeable difference in the rates of LBW. Specifically, the LBW rate was 1.7 times higher for Blacks than Whites (8.6%

and 5.1%, respectively). Thus, although the rates of LBW are lower than other national estimates, the Black-White ratio in this sample is similar to the ratio for women in this maternal age group reported in other national data (see Table 4 in Section 2.1). Taken together, these findings support the hypothesized relationship between race and birth weight, but not between race and gestational age.

Table 12. Summary Birth Weight and Gestational Age Statistics, by Race (N = 1,557)

Variables	Range/ Values	Total (n = 1,557) % or Mean (SE) [a]	Maternal Race: Black (n = 500) % or Mean (SE)	Maternal Race: White (n = 1,077) % or Mean (SE)	*p* value [b]
Birth Weight (gms)	567.0 – 4848.9	3289.7 (12.71)	3175.7 (32.75)	3343.7 (16.29)	<0.0001
LBW (Yes)	*n/a*	*6.2*	*8.6*	*5.1*	*0.0115*
Gestational Age (wks)	28 – 40	39.3 (.04)	39.3 (.04)	39.3 (.06)	0.8739
PTB (Yes)	*n/a*	*6.9*	*7.4*	*6.7*	*0.5770*

Abbreviations: *SE*, standard error; %, percent

[a] % (unweighted) reported for categorical variables; mean (SE) reported for continuous variables

[b] *p* determined from Rao-Scott χ^2 test for categorical variables and *t*-test for continuous variables

Further analyses of the relationship between race and birth weight, controlling for the variables retained from Section 5.2.1, are presented in Table 13 below. Unstandardized coefficients are reported to aid interpretability. The coefficients for race can be interpreted as the average increment or difference in birth weight (in grams) between Blacks and Whites, when all other variables in the model are held constant. Model 1 is the unadjusted model with birth weight regressed on race. Consistent with the findings presented in Table 12, the regression coefficient for race indicates that, on average, Blacks had birth weights that were 167.99 grams (i.e., 5.9 ounces) lower than those of Whites.

Table 13. Regression Coefficients and Standard Errors for Four Models of Birth Weight on Race, Other Sociodemographics, Pregnancy Characteristics, and Prenatal Substance Use (*N* = 1,557)

Variable	Model 1 [a]	Model 2	Model 3	Model 4
Race				
White [b]	----	----	----	----
Black	-167.99 (32.75)***	-169.30 (37.15)***	-160.50 (26.20)***	-166.57 (25.96)***
Relationship Status				
Married[b]	-----	-----	-----	-----
Cohabitating	-----	29.00 (33.35)	12.29 (29.09)	10.33 (28.70)
Steady Relationship	-----	-29.50 (39.99)	-28.67 (31.01)	-28.01 (31.05)
No Relationship	-----	-11.96 (51.62)	-9.04 (38.49)	-8.86 (38.63)
Prenatal Care				
Very Adequate[b]	-----	-----	-----	-----
Somewhat Adequate	-----	6.32 (45.89)	-4.66 (37.81)	-5.43 (37.26)
Inadequate	-----	-40.39 (61.23)‡	-82.89 (48.27)‡	-85.51 (47.51)‡
Father At Prenatal Care				
Yes [b]	-----	-----	-----	-----
No	-----	-41.98 (40.41)	-28.63 (29.45)	-29.67 (28.83)
Prenatal Health Risk	-----	-42.34 (38.08)	-6.28 (30.67)	-8.90 (30.31)
Prenatal Substance Use Score	-----	-65.64 (22.41)***	-76.34 (18.72)***	-----
Prenatal Smoking				
Yes	-----	-----	-----	-120.76 (27.85)***
No [b]	-----	-----	-----	-----
Gestational Age		-----	176.62 (5.75)***	176.24 (5.78)***

[a] Regression coefficients are unstandardized and based on unweighted data; standard errors are in parentheses

[b] Reference category

‡ p <.10; * p <.05; ** p <.01; *** p <.001

Model 2 shows that the magnitude and significance of the relationship between race and birth weight is attenuated only slightly after controlling for differences in relationship status during pregnancy, prenatal care adequacy, father attendance at prenatal care, and prenatal health risk and substance use. The same conclusion can be drawn from the results

for Model 3, which adds gestational age to Model 2.[37] Because of the earlier finding that prenatal substance use was largely driven by cigarette use, I disaggregated the score and entered cigarette use alone into Model 4. The results for the effect of race in this model match the results in Model 3, although the magnitude of the effect of cigarette use on birth weight was greater than the effect of the prenatal substance use score.

Infant gestational age had a significant positive relationship with birth weight in all adjusted models. In Model 4, for example, each additional week of gestation was associated with an average increase of approximately 176 grams (i.e., 6.2 ounces) in birth weight. Conversely, inadequate prenatal care and use of substances during pregnancy had negative relationships with birth weight. In Model 4, having inadequate prenatal care (i.e., initiating prenatal care during the third trimester or never) was associated with a borderline significant decrease of approximately 85 grams (i.e., 3.0 ounces) in birth weight when compared to initiating care during the first trimester. In the same model, smoking during pregnancy was associated with an average decrease of 120.8 grams (i.e., 4.3 ounces) in birth weight when compared to not smoking during pregnancy.[38]

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[37]As noted in footnote 21, my conceptual and structural equation models specified gestational age as a key variable in the model, antecedent to birth outcomes, and mediating the relationship between exogenous variables, other endogenous variables, and birth weight. This placement, however, results in gestational age being controlled for in analyses of the relationships between birth weight and all other study variables.

[38]Another potentially important covariate—respondent's own birth weight—was not included in the model due to the large number of missing values (>25%). The use of OLS estimation in SAS resulted in listwise deletion of cases with any missing values, thereby substantially impacting the outcomes. ML estimation—the gold standard for analyzing data with missing values—in Mplus also produced undesirable results when own birth weight was entered into the model. This is because minimum covariance coverage (i.e., 10% of cases in any given cell) was not fulfilled for all groups in the analysis. In both cases, respondent LBW was significantly associated with a decrease in infant birth weight (as we might expect given the discussion in Section 2.2.1.1); however, the impact on the analysis of the missing values was deemed of greater negative consequence to the study than omitting the variable. It also should be noted that no racial difference in this variable was found (i.e., a difference of less than 2.0% in the rates of LBW among Blacks and Whites whose parents reported their birth weights [p = .09]).

5.3. Results for Aim 2

Aim 2: To examine the relationship between adverse birth outcomes and maternal exposure to family-level and neighborhood-level disadvantage during childhood

For Aim 2 of the study, I used structural equation modeling to examine the relationship between birth outcomes and maternal exposure to family and neighborhood disadvantage during childhood.[39] Prior to hypothesis testing for Aim 2, I first examined unadjusted bivariate associations between birth outcomes and indicators of childhood disadvantage to get a preliminary sense of whether or not the data were consistent with expectations. The results of these bivariate analyses are discussed briefly below. (See Appendix E for tables). I then present the results of the analyses that established the measurement models, followed by the results of hypothesis tests regarding the structural portion of the structural equation model.

5.3.1. Bivariate Associations between Birth Outcomes and Indicators of Childhood Disadvantage

Unadjusted bivariate analyses revealed that only two of the three indicators of family disadvantage were significantly associated with birth weight. Being raised in a single-parent or surrogate family was associated with a decrease of approximately 60 grams (i.e., ~2 ounces) in birth weight when compared to being raised in a two-parent family. On the other hand, having a resident parent with educational training beyond high school was associated with an increase of roughly 78 grams (~2.8 ounces) in infant birth weight when

[39] A corollary question for this aim was whether or not the relationship varied by race; however, this question was not answered until the full structural equation model (with mediating paths) for Aim 3 was estimated. Thus, I reserve the presentation of those results for the end of Section 5.4.

compared to women who had parents with only a high school education. No indicators of family disadvantage were significantly associated with gestational age.

All but two indicators of neighborhood structural disadvantage were significantly associated with birth weight. The two exceptions were: the proportion of residents over age 25 without a high school diploma or GED and the proportion of households receiving public assistance. Infant birth weight decreased with increasing proportions of: families in poverty; unemployment rates; female-headed households; and Black residents. The magnitude of the decrease was especially large for tract-level unemployment rate (734.47 grams or 1.62 pounds) and proportion of female-headed households (705.95 grams or 1.56 pounds).

Two indicators of neighborhood physical disadvantage were also negatively associated with birth outcomes in unadjusted bivariate analyses. Specifically, the proportion of vacant housing units was negatively associated with both birth weight and gestational age: on average, a 1% increase in the proportion of vacant housing units resulted in average decreases of 453.40 grams (i.e., ~1 pound) of birth weight and 1.7 weeks of gestation. Interviewer ratings of housing as being of very or somewhat poor quality were significantly associated with a decrease in birth weight—but not gestational age.

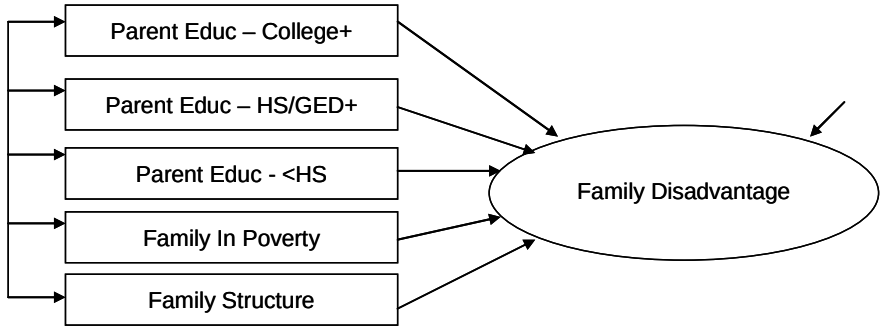
None of the indicators of neighborhood social disadvantage were significantly associated with birth weight or gestational age in these unadjusted bivariate analyses. And, with the exception of the proportion of vacant housing units, none of the other indicators of neighborhood disadvantage were significantly associated with gestational age.

5.3.2. *Measurement Models*

The hypothesized measurement models of disadvantage, as they were specified in the original structural equation model discussed in Section 4.4.3, are illustrated in Appendix B. Below I discuss the original specification of the measurement models, reasons for and results of model modifications, and the final model specifications.

5.3.2.1 Measurement Model for Family Disadvantage. Family disadvantage was originally specified as being caused by three indicators: one indicator of parent education, one indicator of family poverty, and an indicator of family structure. As discussed in Section 4.4.3.6, I estimated this causal model by setting the latent variable's residual variance to zero, scaling the latent variable by fixing a path to or from it to one, and regressing other variables in the structural model on the latent variable. Regardless of the other variables selected, model fit statistics (e.g., chi-square, CFI, TLI, RMSEA, and WRMR) suggested that the model had adequate fit. In addition, the path coefficients for the causal indicators were generally of the right sign and most were statistically significant. Family structure, which is generally not among the three indicators of socioeconomic status (i.e., the third is usually occupation), had less stable estimates across the different tests. Because causal models must be evaluated within the context of other variables in the model, however, further modifications of this measurement model were not pursued until structural model estimation (Section 5.3.4). Thus, the original model illustrated in Figure 10 was retained.

Figure 10. Measurement Model for Family Disadvantage



KEY: Circles represent constructs; squares represent indicators; double-headed arrows between indicators represent covariances; no regression coefficients are shown because it is not possible to estimate this model without regressing other variables on it

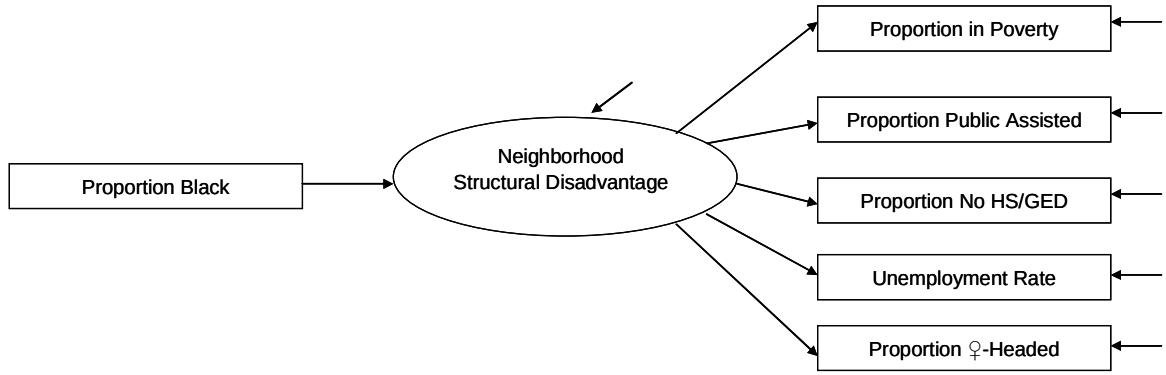
5.3.2.2. Measurement Model for Neighborhood Structural Disadvantage.

Neighborhood structural disadvantage was originally assumed to be influenced by six causal indicators: proportion of black residents, unemployment rate, proportion of residents without a high school diploma or GED, proportion of female-headed households, proportion of families living in poverty, and proportion of families receiving public assistance. As I did with the causal model of family disadvantage, I estimated the causal model of neighborhood structural disadvantage by setting the latent variable's residual variance to zero, scaling it by setting a path to or from the latent variable to one, and regressing other variables in the structural model on it. Regardless of the other variables selected, the regression coefficients were non-significant and/or in the wrong direction. Although I modeled them as causal to be consistent with the SEM literature on the proper specification of socioeconomic status, literature and theory discussed in Chapters 2 and 3

actually suggest that neighborhood structural disadvantage could operate differently than the individual-level measures.

In those chapters, I argued that residential segregation creates neighborhood disadvantage in the form of concentrated poverty, and differential educational and employment opportunities. Thus, I re-specified the model of neighborhood structural disadvantage as a MIMIC[40] model, retaining one indicator as a causal indicator (i.e., proportion of Black residents), while all others were treated as effect indicators (Figure 11). In addition to its consistency with notions about the manifestations of residential segregation, this re-specification was more consistent with the idea of contextual effects (despite the use of compositional measures) than was the original specification which modeled neighborhood structural disadvantage as purely a consequence of its residents.

Figure 11. Alternate Specification of Measurement Model for Neighborhood Structural Disadvantage



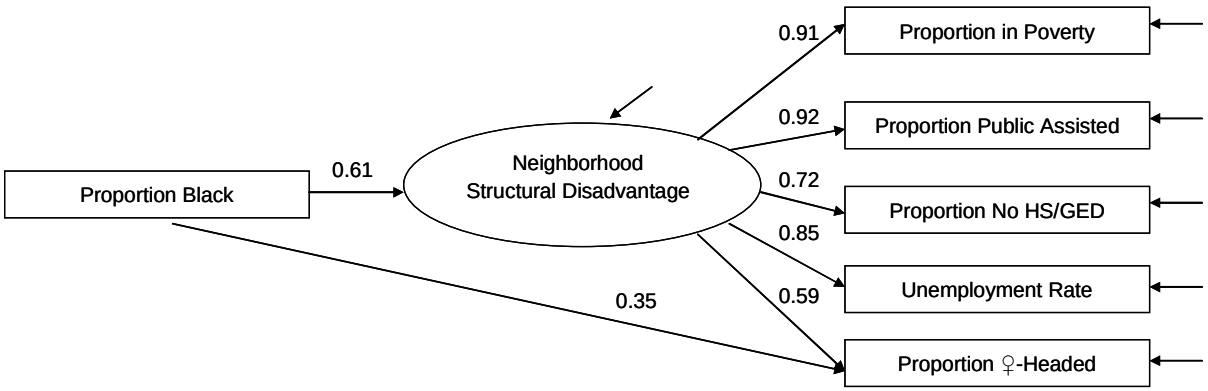
KEY: Circles represent constructs; squares represent indicators.

[40]As discussed in Chapter 4, MIMIC (Multiple Indicator Multiple Causes) models have both causal and effect indicators, the analysis of which can crudely be thought of as CFA with covariates.

Some of the fit statistics for this alternate model suggested good model fit. For example, all standardized factor loadings were greater than or equal to 0.80 and statistically significant. On the other hand, I obtained a highly significant chi-square statistic, and unacceptably low CFI and TLI values (0.90 and 0.83, respectively) for this model. It seemed logical, however, to include a direct effect from proportion of Black residents to proportion of female-headed households, reflecting the assumption that the proportion of female-headed households would not be measurement invariant at different levels of residential segregation because of the multi-generational caregiving that may occur in predominantly Black environments. This change resulted in some improvement in model fit with respect to the values of the CFI and TLI (i.e., 0.95, 0.91), and the RMSEA and SRMR were in the range of good fit (0.057 and 0.04, respectively). Moreover, the residual covariances were small and centered around zero, as they should be when model fit is good. On the other hand, the chi-square value remained statistically significant ($\chi^2[8] = 47.978$, $p < 0.0001$). Nevertheless, a Satorra-Bentler scaled chi-square difference test confirmed that the model with the direct effect provided improved fit ($\chi^2_{diff}[1] = 1114.95$, $p < .0001$).

This final model of neighborhood structural disadvantage is presented in Figure 12 with standardized regression coefficients. (Other model fit statistics are included in Table 14 at the end of this section). All factor loadings for the model were statistically significant at the $p \leq .05$ level. The same is true of the regression coefficient for the relationship between the latent variable and the causal indicator. In addition, the magnitude of the factor loadings were moderately to very high for all indicators (i.e., ≥ 0.60), as were the R^2 values for the indicators (i.e., ≥ 0.50).

Figure 12. Final Standardized Measurement Model for Neighborhood Structural Disadvantage



KEY: Circles represent constructs; squares represent indicators; regression coefficients are standardized to aid comparison; all regression coefficients are significant at $p \leq .05$.

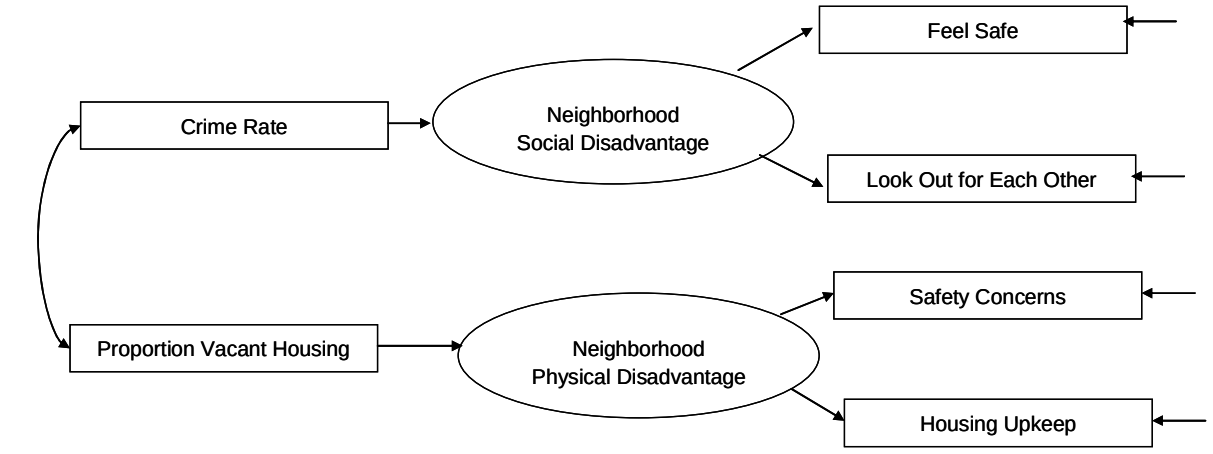
5.3.2.3. Measurement Models for Neighborhood Social and Physical Disadvantage.

Neighborhood social and physical disadvantage were originally conceptualized as a single latent variable with six indicators (i.e., feeling safe, perceiving that neighbors look out for each other, county crime rate, having the interviewer report that the housing in the neighborhood was not well kept or that the appearance of the neighborhood created safety concerns for him/her, and the proportion of vacant housing units in the census tract). When I estimated this model,[41] I obtained a highly significant chi-square statistic ($\chi^2[7] = 43.64$, p <.0001) and lower than ideal values of CFI and TLI (0.78 and 0.66, respectively). The R^2 values for several of the indicators were less than 0.25, and especially low (i.e., < 0.05) for the two objective measures (i.e., county crime rate and proportion of vacant housing units in the census tract).

[41]The scale for county crime rate was so much larger than the other indicators included in the model that I re-scaled it by a factor of 1/10,000 before model estimation to avoid possible computational errors.

It seemed logical to shift the role of the two objective measures from effect to causal indicators, reflecting the reasonable assumption that crime rate and housing quality are the conditions that underlay (and cause variation in) the respondent and interviewer perceptions captured by the other indicators. In addition, my description of this construct in Chapter 4 suggested that it should be two-dimensional. This reasoning resulted in the two MIMIC models depicted in Figure 13 below. A nested comparison (i.e., a single latent variable model with two causal indicators nested within the two latent variable model) confirmed that the two-factor model was an improvement over the single-factor model ($\chi^2_{diff}[2] = 31.48$, $p < .0001$).

Figure 13. Alternate Specifications of Neighborhood Social and Physical Disadvantage Measurement Models

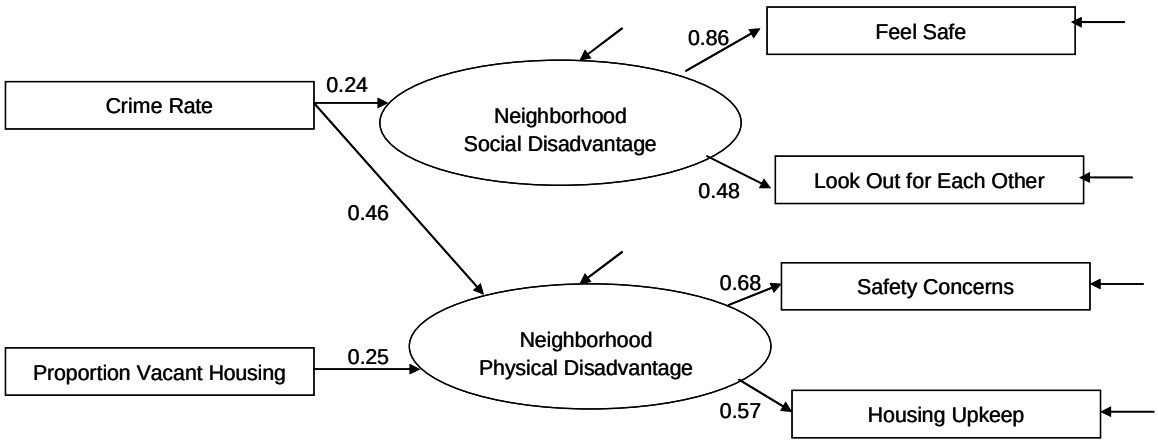


KEY: Circles represent constructs; squares represent indicators.

Based on literature presented in Chapters 2 and 4, I also expected that crime rate would not only influence neighborhood social disadvantage but also neighborhood physical disadvantage. Thus, I added a path between the two. According to the model fit statistics,

this modification resulted in substantial improvement ($\chi^2[6] = 10.716$, p = 0.0975, CFI = 0.971, TLI = 0.951, WRMR = 0.729, and RMSEA = 0.008). A chi-square difference test comparing this model to the model without the path from crime rate to the neighborhood physical disadvantage latent variable confirmed this conclusion ($\chi^2_{diff}[1] = 39.67$, p <.0001). The R^2 for "neighbors look out for each other" remained low (i.e., ~0.20) throughout these modifications. Yet it could not be removed without resulting in model under-identification. Plus, the value of its standardized regression weight was above the 0.40 cutoff. Thus, no further modifications were made to these measurement models prior to structural model estimation. The final measurement models of neighborhood social disadvantage and neighborhood physical disadvantage are presented in Figure 14 with standardized regression coefficients.

Figure 14. Final Standardized Measurement Models for Neighborhood Social and Physical Disadvantage



KEY: Circles represent constructs; squares represent indicators; regression coefficients are standardized to aid comparison; all regression coefficients are significant at $p \leq .05$.

Table 14 below summarizes the fit statistics for all the measurement models of disadvantage, except for family disadvantage. Here we see that, with few exceptions, all models had fit statistics at the high end of the acceptable range.

Table 14. Fit Statistics for the Final Measurement Models of Disadvantage, except for Family Disadvantage

Factors	Observed Indicators	Regression Weights* (Factor Loading Estimates)		R^2
		Unstandardized	Standardized	
Neighborhood Structural Disadvantage				
$\chi^2(8) = 47.978$	Proportion Public Assisted	1.000**	0.919	0.845
$p = 0.0000$	Unemployment Rate	0.587	0.853	0.727
CFI = 0.95	Proportion in Poverty	1.962	0.913	0.833
TLI = 0.91	Proportion No NS/GED	1.349	0.722	0.521
SRMR = 0.04	Proportion ♀-Headed HH	0.459	0.592	0.728
RMSEA = 0.06	∞*ON Proportion Black Residents*	*0.150*	*0.613*	*n/a*
	∞*♀-Headed ON Black Residents*	*0.066*	*0.350*	*n/a*
Neighborhood Social and Physical Disadvantage				
$\chi^2(6) = 10.716$	Feel Safe	1.000**	0.862	0.743
$p = 0.0975$	Neighbors Look out for Each Other	0.553	0.483	0.234
CFI = 0.97	Concern about Safety *(interviewer)*	1.000	0.573	0.465
TLI = 0.95	Housing Upkeep *(interviewer)*	1.197	0.682	0.328
WRMR = 0.73	∞*Physical ON Vacant Housing*	*2.034*	*0.245*	*n/a*
RMSEA = 0.01	∞*Social ON Crime Rate*	*0.717*	*0.235*	*n/a*
	∞*Physical ON Crime Rate*	*0.302*	*0.458*	*n/a*

Abbreviations: *CFI* = comparative fit index; *TLI* = Tucker-Lewis index; *RMSEA* = root mean squared error of approximation; *SRMR* = standardized mean residual; *WRMR* = weighted root mean squared residual; R^2 = multiple squared correlation

* All factor loadings or path coefficients were significant at $p \leq .05$;

** Parameter constrained to 1.00 to scale the construct; constrained parameters were not tested for statistical significance

∞ Denotes causal indicator

5.3.3. Structural Model

Using the final measurement models developed in the previous section, I then estimated a structural equation model linking neighborhood- and family-level disadvantage to birth weight and gestational age. The original model that I set out to test is illustrated in Figure 15 (page 123). This model reflects the assumption, discussed in Chapter 3, that exposure to any form of disadvantage during childhood has long-term consequences for subsequent birth outcomes. By all the goodness of fit statistics except for the chi-square statistic, the original model was a very poor fit for the data (χ^2 [26] = 183.10, p = 0.000, CFI = 0.683, TLI = 0.659, WRMR = 1.204). These results suggested that the model needed modification in order to improve the fit. Consistent with confirmatory rather than exploratory factor analysis, modifications were made primarily based on theoretical or substantive (with respect to SEM) considerations. Below I describe the modifications, while Table 15 (page 124) summarizes them along with the fit indices for the model after each modification. Figure 16 on page 125 visually depicts the modifications.

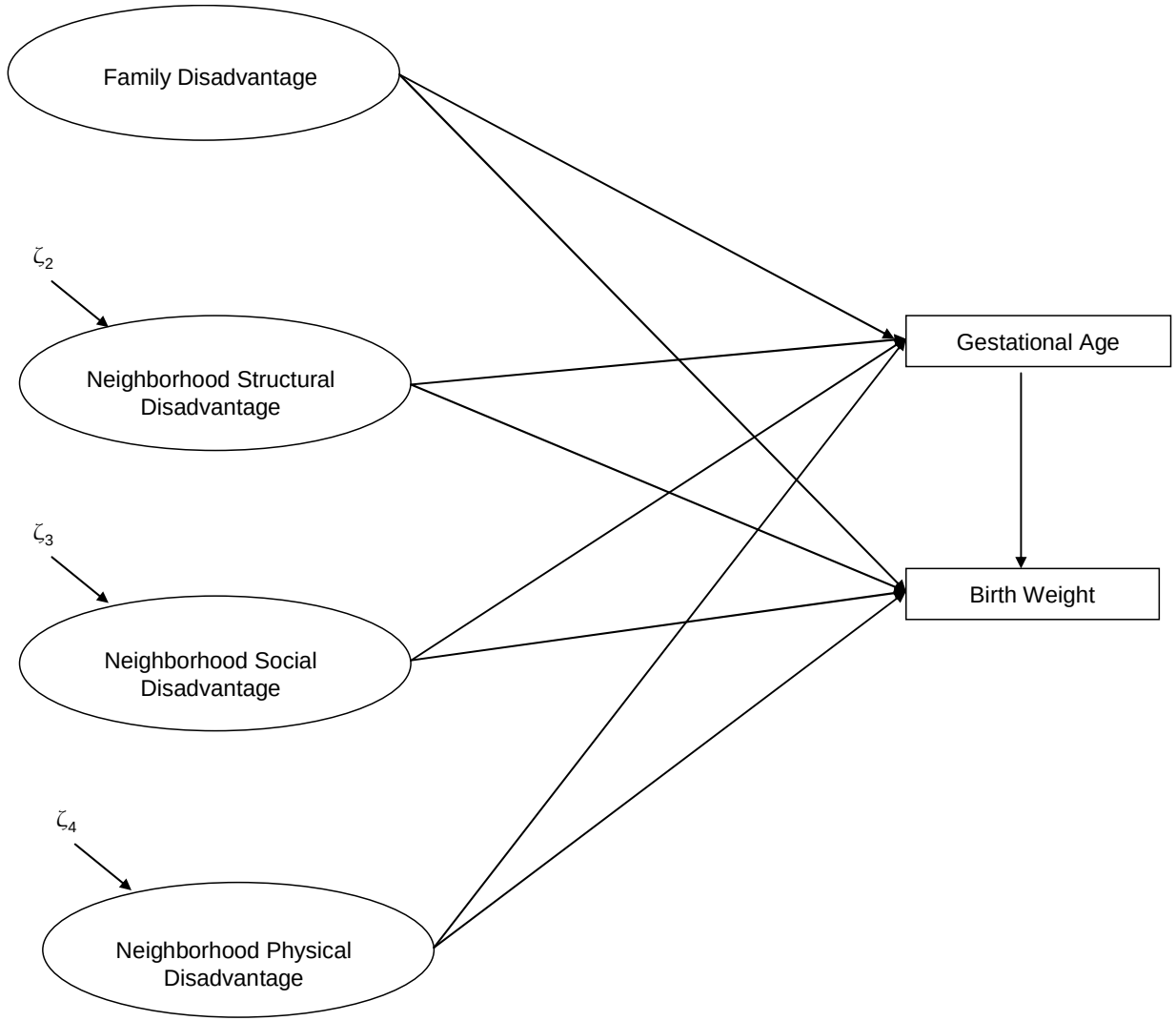
Modification 1: The original model depicted no relationships among the latent variables of disadvantage, although the literature and theory reviewed in Chapters 2 and 3 suggested that it should. Thus, the first modification I made involved explicating those relationships. In particular, I drew on literature and theory presented in Chapters 2 through 4 which suggested that: (1) neighborhood social and physical disadvantage (or disorder) may mediate the relationship between neighborhood structural disadvantage and health (e.g., Masi et al., 2007), and (2) lower rates of social disorder explain lower rates of physical

disorder when neighborhood structural disadvantage is controlled (e.g., Sampson & Raudenbush, 1997). These ideas led me to add direct paths from neighborhood structural disadvantage to neighborhood social disadvantage, and from neighborhood social disadvantage to neighborhood physical disadvantage. I further hypothesized that individual poverty and low educational attainment give rise to neighborhood social disorder (e.g., petty criminal activity) and contribute to neighborhood structural disadvantage, particularly as reflected by census tract-level measures of socioeconomic status (e.g., proportion of persons living in poverty). This hypothesis resulted in direct paths from family disadvantage to neighborhood structural and social disadvantage. Adding these direct paths improved the fit of the model substantially, as indicated by improved CFI , TLI, and WRMR values (i.e., 0.91, 0.90, and 1.17, respectively) and a lower chi-square.

Modification 2: I also added two correlations among the measurement errors of the four indicators of neighborhood social and physical disadvantage.[42] This change reflected my assumption that the similar content and same data source for the responses with respect to each latent variable would result in over- or under-estimation to the same extent across the items. As a result of these correlated measurement errors, the chi-square dropped even lower, and the CFI, TLI, and WRMR values reached the criterion for good fit (i.e., 0.95, 0.95, and 0.99, respectively).

[42]When I attempted to make this change during the CFA stage (discussed in Section 5.3.2.3), I was unable to do so for identification reasons.

Figure 15. Original Structural Equation Model Linking Childhood Disadvantage and Subsequent Birth Outcomes



Modification 3: As a result of the additional paths among the latent variables of disadvantage, I was able to lift the zero constraint I had placed on the residual variance of family disadvantage during earlier stages of the estimation process in order to identify that latent variable. This change meant that its disturbance could now be freely estimated just as could the disturbances for the other endogenous variables in the model. While doing this resulted in a larger chi-square statistic, I deemed this change appropriate on statistical grounds (Bollen &Davis, forthcoming). No theoretical or substantive reason warranted further model modifications. And the final unadjusted model had good fit according to all fit indices (e.g., CFI = 0.95, TLI = 0.94, RMSEA = 0.010, WRMR = 1.00).

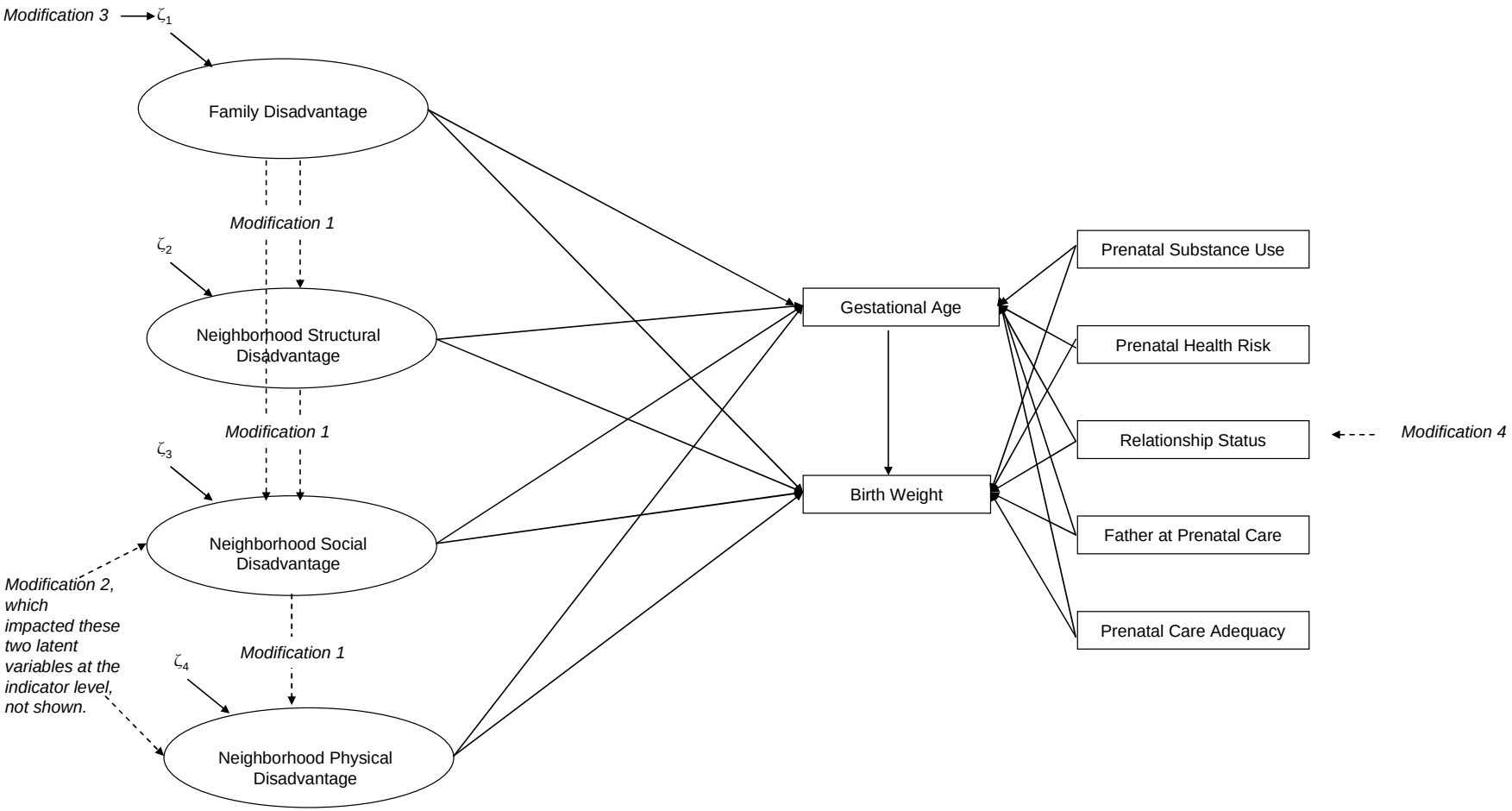
Table 15. Measures of Overall Model Fit for Original and Modified Structural Models of the Relationship between Childhood Disadvantage and Birth Outcomes

Models	X^{2*}	df	CFI	TLI	RMSEA	WRMR
Original Unadjusted Model	183.10	26	0.68	0.66	0.02	2.10
Modification 1: Explicated relationships among disadvantage latent variables	73.78	27	0.91	0.90	0.01	1.17
Modification 2: Added correlated errors among indicators of neighborhood disadvantage	53.26	27	0.95	0.95	0.01	0.99
Modification 3: Removed zero constraint on disturbance of family disadvantage	54.45	27	0.95	0.94	0.01	1.00
Modification 4: Added control variables to final unadjusted model	48.763	33	0.95	0.95	0.02	0.95
Final Adjusted Model: Dropped non-significant paths from control variables and added zero constraint on disturbance of family disadvantage	54.47	29	0.95	0.95	0.02	1.00

Abbreviations: *df* = degrees of freedom; *CFI* = comparative fit index; *TLI* = Tucker-Lewis index; *RMSEA* = root mean squared error of approximation; *WRMR* = weighted root mean squared residual.

* Chi-square and degrees of freedom generated from WLSMV estimation cannot be used for chi-square difference testing

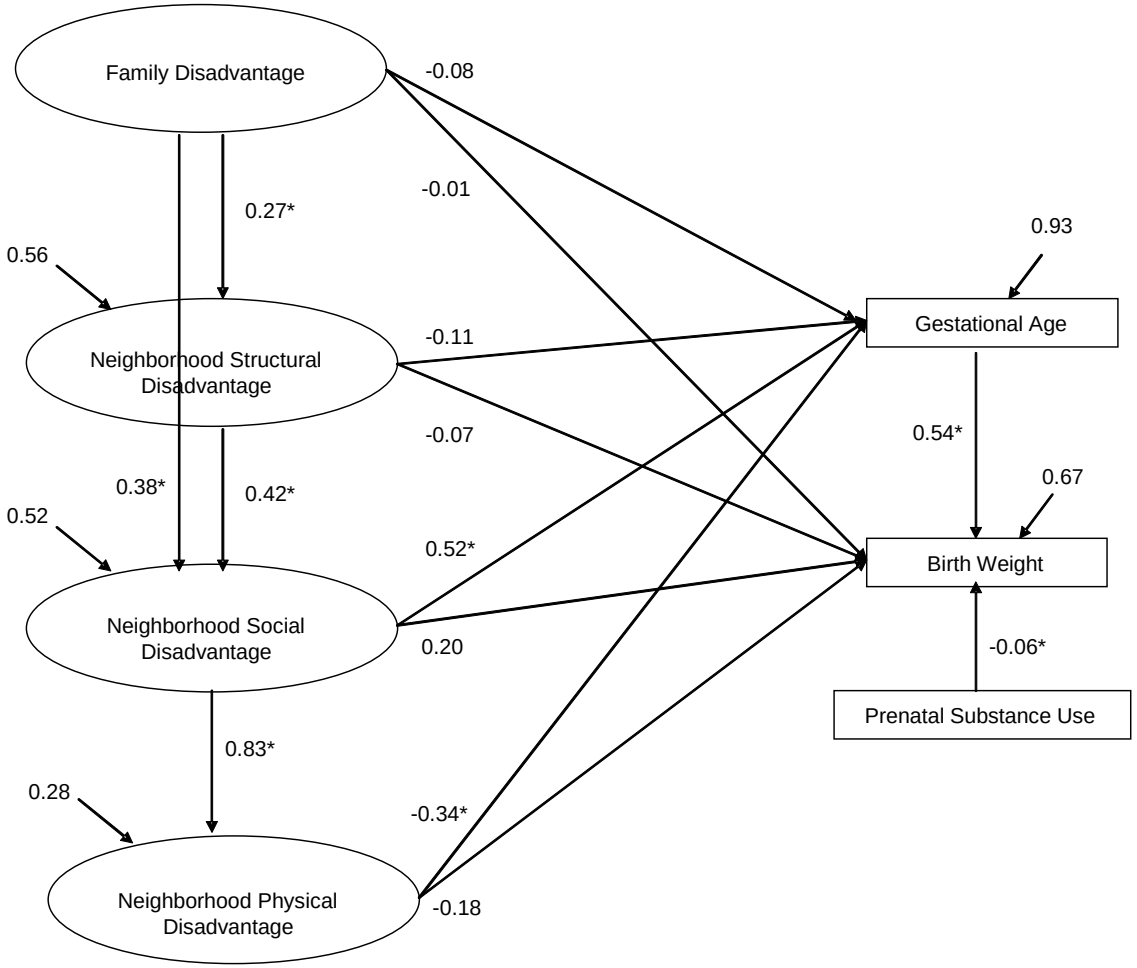
Figure 16. Modifications to the Structural Equation Model Linking Childhood Disadvantage and Subsequent Birth Outcomes



Modification 4: Adding potential control variables to the model was the next major change. As discussed in Section 4.4.3.7, I added the control variables retained from Aim 1 to produce a baseline adjusted model in which birth weight was simultaneously regressed on all the control variables. By all goodness of fit statistics, it seemed this model better fit the data than the unadjusted model (χ^2 [33] = 48.76, p = 0.038, CFI = 0.953, TLI = 0.951, RMSEA = 0.018, WRMR = 0.950). However, only one of the control variables—prenatal substance use score—was significantly associated with birth weight and none were significantly associated with gestational age. I constrained all non-significant paths stemming from the control variables to zero, performed a chi-square difference test, and obtained a non-significant chi-square statistic (χ^2_{diff} [8] = 3.953, p = 0.8613). The more parsimonious model seemed to provide no worse a fit than the unconstrained model. As a result, I dropped the four non-significant control variables from the model. This change resulted in the empirical under-identification of family disadvantage, however, so I reapplied the zero constraint on its disturbance term prior to re-estimation.

The final adjusted model had model fit statistics in the acceptable range (χ^2 [29] = 54.47, p = 0.0029, CFI = 0.947, TLI = 0.945, RMSEA = 0.019, WRMR = 1.003). As expected, the component fit statistics for each of the latent variables were good (i.e., all paths were statistically significant, and nearly all factor loadings met or exceeded the 0.40 cutoff). Figure 17 provides the standardized estimates to aid comparison of the path estimates. Below I discuss these results, as well as the unstandardized coefficients for birth weight and gestational age because these variables can be more easily interpreted in terms of grams and weeks.

Figure 17. Final Structural Equation Model Linking Control Variables, Childhood Disadvantage, and Subsequent Birth Outcomes with Adjusted Standardized Parameter Estimates**



* $p \leq 0.05$, determined from the unstandardized solution

** Controlling for prenatal substance use

As Figure 17 and Table 16 show, statistically significant relationships existed among the disadvantage latent variables. Only two of them—neighborhood social and physical disadvantage—were significantly associated with gestational age, when prenatal substance use was held constant. In particular, gestational age increased by half a standard deviation as neighborhood social disadvantage increased by one standard deviation, and it decreased by 0.34 standard deviations as neighborhood physical disadvantage increased by one standard deviation. In the unstandardized solution, these reductions corresponded to about 12 and 6 days, respectively. For a one standard deviation increase in gestational age, there was an increase of 0.54 standard deviations in birth weight—i.e., about 164 grams (or 5.8 ounces) for every additional week of gestation.

Table 16. Mean and Variance Adjusted Weighted Least Squares Regression Weights for the Final Structural Model of the Relationship between Childhood Disadvantage and Birth Outcomes

Path	Unstandardized Estimate*	Standard Error	Standardized Estimate*	*Z***
FAMDIS → STRUC	0.045	0.008	0.270	5.459
FAMDIS → SOC	0.241	0.057	0.378	4.261
STRUC → SOC	1.630	0.351	0.422	4.646
SOC → PHYS	1.095	0.190	0.829	5.748
FAM → GEST	-0.164	0.122	-0.078	-1.343
STRUC → GEST	-0.786	0.839	-0.062	-0.936
SOC → GEST	1.720	0.630	0.521	2.730
PHYS → GEST	-0.843	0.381	-0.337	-2.211

Abbreviations: *FAM* = family disadvantage, *STRUC* = neighborhood structural disadvantage, *SOC* = neighborhood social disadvantage, *PHYS* = neighborhood physical disadvantage, *GEST* = gestational age

* Controlling for prenatal substance use

** z score reported for unstandardized estimate

Table 16 continues on next page

Table 16. Mean and Variance Adjusted Weighted Least Squares Regression Weights for the Final Structural Model of the Relationship between Childhood Disadvantage and Birth Outcomes *(continued)*

Path	Unstandardized Estimate*	Standard Error	Standardized Estimate*	*Z***
FAM → BWT***	-0.077	0.234	-0.012	-0.328
STRUC → BWT***	-2.629	1.990	-0.068	0.186
SOC → BWT***	1.999	1.823	0.199	1.097
PHYS → BWT***	-1.399	1.096	-0.184	-1.277
GESTAGE → BWT***	1.642	0.086	0.538	19.091

Abbreviations: *FAM* = family disadvantage, *STRUC* = neighborhood structural disadvantage, *SOC* = neighborhood social disadvantage, *PHYS* = neighborhood physical disadvantage, *GEST* = gestational age, *BWT* = birth weight

* Controlling for prenatal substance use

** z score reported for unstandardized estimate

*** Birth weight was re-scaled by 1/100 to avoid computational or convergence issues; all unstandardized values for parameters estimated when birth weight is the dependent variable should be multiplied by 100 before interpreting.

Together these findings suggest that the relationship between family disadvantage and birth weight was fully mediated by neighborhood structural, social, and physical disadvantage and gestational age. Likewise, they suggested that the relationship between neighborhood structural disadvantage and birth weight was fully mediated by neighborhood social and physical disadvantage and gestational age. Table 17 below provides some support for these conclusions. According to the table, the total indirect effects of family disadvantage on gestational age were small but statistically significant, while the direct effect was non-significant. The total indirect effects of neighborhood structural disadvantage on gestational age also were small but borderline significant. Although not listed in the table, the specific indirect paths that were statistically significant involved the paths between the disadvantage variables, and the paths from neighborhood social and physical disadvantage to gestational age (Appendix F). In addition, the effects of

neighborhood social and physical disadvantage on gestational age were larger than the effects of the other disadvantage variables on that outcome.

Table 17. Direct, Total, and Indirect Effects of Childhood Disadvantage on Birth Outcomes*

Path	Unstandardized Estimate**	Standard Error	*Z****	Standardized Estimate**
Total Effect of Family Disadvantage on Gestational Age	0.051	0.066	0.773	0.024
Total Indirect Effect	0.215	0.103	2.094	0.102
Direct Effect	-0.164	0.122	0.179	-0.078
Total Effect of Family Disadvantage on Birth Weight****	0.113	0.228	0.160	0.006
Total Indirect Effect	0.036	0.245	0.463	0.018
Direct Effect	-0.077	0.234	-0.328	-0.012
Total Effect of Neighborhood Structural Disadvantage on Gestational Age	0.514	0.369	1.394	0.040
Total Indirect Effect	1.300	0.684	1.899	0.102
Direct Effect	-0.786	0.839	-0.936	-0.062
Total Effect of Neighborhood Structural Disadvantage on Birth Weight****	-1.023	1.436	-0.713	-0.026
Total Indirect Effect	1.606	1.385	1.159	0.041
Direct Effect	-2.629	1.990	-1.321	-0.068
Total Effect of Neighborhood Social Disadvantage on Gestational Age	0.797	0.359	2.222	0.242
Total Indirect Effect	-0.922	0.452	-2.039	-0.279
Direct Effect	1.720	0.630	2.730	0.521

* Specific indirect effects not shown

** Controlling for prenatal substance use

*** z score reported for unstandardized estimate

**** Birth weight was re-scaled by 1/100 to avoid computational or convergence issues; all unstandardized values for parameters estimated when birth weight is the dependent variable should be multiplied by 100 before interpreting.

Table 17 continues on next page

Table 17. Direct, Total, and Indirect Effects of Childhood Disadvantage on Birth Outcomes* *(continued)*

Path	Unstandardized Estimate**	Standard Error	*Z****	Standardized Estimate**
Total Effect of Neighborhood Social Disadvantage on Birth Weight****	1.777	1.178	0.131	0.177
Total Indirect Effect	-0.222	1.410	0.875	-0.022
Direct Effect	1.999	1.823	1.097	-0.199
Total Effect of Neighborhood Physical Disadvantage on Gestational Age	-0.843	0.381	-2.211	-0.337
Total Indirect Effect	0.000	0.000	n/a	0.000
Direct Effect	-0.843	0.381	-2.211	-0.337
Total Effect of Neighborhood Physical Disadvantage on Birth Weight****	-2.783	1.354	-2.055	-0.365
Total Indirect Effect	-1.384	0.611	-2.266	-0.182
Direct Effect	-1.399	1.096	0.202	-0.184

* Specific indirect effects not shown

** Controlling for prenatal substance use

*** z score reported for unstandardized estimate

**** Birth weight was re-scaled by 1/100 to avoid computational or convergence issues; all unstandardized values for parameters estimated when birth weight is the dependent variable should be multiplied by 100 before interpreting.

It should be noted, however, that only 7% of the variance in gestational age is accounted for by these factors, and much of the 33% variance in birth weight is likely attributable to its relationship with gestational age. In addition, a consistent finding in these analyses was that family structure had a non-statistically significant relationship with the family disadvantage latent variable—a finding that is perhaps no surprise given the instability of this indicator during earlier estimations of the causal model (see Section 5.3.2.1). Constraining the path to zero did little to alter the family disadvantage findings in this model, however.

To explore the possibility that family disadvantage and neighborhood structural disadvantage did not exhibit direct effects because they were both in the model, I re-ran the model omitting the latent variable of family disadvantage. The results of that analysis were identical to the full model analyses—i.e., neighborhood structural disadvantage was fully mediated by the other neighborhood-level latent variables. When neighborhood structural disadvantage was entered into the model without other disadvantage variables, it resulted in a statistically significant decrease in birth weight of 301.60 grams (10.6 ounces), but no decrease in gestational age.

5.4. Results for Aim 3

Aim 3: To explore the behavioral, psychosocial, and health pathways through which exposures to disadvantage during childhood affect subsequent birth outcomes and translate into racial disparities in adverse birth outcomes

For Aim 3 of the study, I used structural equation modeling to examine the extent to which the relationship between birth outcomes and maternal exposure to family-level and neighborhood-level disadvantage during childhood was mediated by behavioral, psychosocial, and health risks experienced during the intervening years. In addition, I sought to determine the extent to which these relationships varied by race. Prior to hypothesis testing, I first examined unadjusted bivariate associations between birth outcomes and indicators of childhood behavioral, psychosocial, and physical health risks to get a preliminary sense of whether or not the data were consistent with expectations. The results of these bivariate analyses are discussed briefly below. (See Appendix E for tables). I then present the results of the analyses that established the final measurement model

needed for this study aim, followed by the results of hypothesis tests regarding the structural model and the hypothesized role of race.

5.4.1. *Bivariate Associations between Birth Outcomes and Indicators of Childhood Behavioral, Psychosocial, and Physical Health Risks*

Statistically significant relationships were found between childhood substance use and both birth weight and gestational age, but they were not in the expected direction. Increases in substance use scores were associated with small but statistically significant *increases* in both gestational age and birth weight. Feeling that life was not worth living "a lot of the time" during the week before the Wave I interview was associated with an average decrease of 175.8 grams (i.e., 6.2 ounces) in birth weight when compared to never or rarely feeling that way. Yet none of the other indicators of depression were significantly associated with birth outcomes. Neither were the dummy variables for self-rated health, although the overall chi-square for the association of that indicator with gestational age reached borderline significance ($\chi^2[3, 1557] = 7.32$, $p = .06$). Despite these findings, I retained all the indicators for the next stage of analysis due to their conceptual importance, as well as the possibility of a relationship between them and the indicators of disadvantage.

5.4.2. *Measurement Models*

As discussed in Chapter 4, childhood behavioral, psychosocial, and health risks were measured by indicators of substance use, depression, and self-rated health. Only depression was modeled as a measurement model, while the other two were each measured by a single observed variable. The hypothesized measurement model of depression, as it was specified

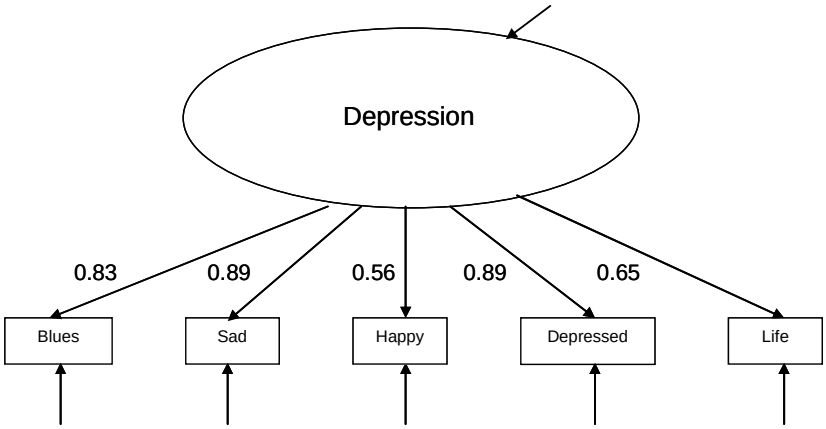
in the original structural equation model discussed in Section 4.4.3, is illustrated in Appendix B. Below I discuss the original specification of the measurement model, reasons for and results of model modifications, and the final model specifications.

5.4.2.1 Measurement Model for Depression. For reasons discussed in Chapter 4, depression was originally specified as a latent variable with 5 indicators—i.e., being unable to shake the blues, feeling depressed, feeling happy (reverse-scored), feeling sad, and feeling that life was not worth living. This 5-indicator model produced a non-statistically significant chi-square statistic ($\chi^2[4] = 8.393$, $p = .0782$), CFI and TLI of 0.999, and RMSEA of 0.01—all suggesting almost exact model fit. Some of the other measures of model fit led to the same conclusion. For example, the R^2 values of three of the indicators (i.e., blues, depressed, and sad) were high in magnitude (i.e., > 0.60). However, although the factor loading estimates for Happy and Life were high (0.58 and 0.65, respectively) and statistically significant, their R^2 values were less than 0.45. For this reason, I performed a nested comparison of the 5-indicator model with a model constraining the factor loadings for Happy and Life to zero. A statistically significant chi-square difference statistic was obtained, suggesting that the unconstrained model provided better fit ($\chi^2_{diff}[2] = 1518.15$, $p = <0.0001$).

Due to the similar wording of the Depressed and Sad items, I assumed that the measurement errors of those indicators could be correlated. However, when added to the model, the correlations were non-significant. Thus, I retained the original 5-indicator model, without correlated measurement errors between the indicators. The final

measurement model for depression is depicted with standardized regression coefficients in Figure 18, and other fit statistics for the model are reported in Table 18 below. As the table shows, this measurement model had fit statistics at the high end of the acceptable range.

Figure 18. Final Standardized Measurement Model for Depression



KEY: Circles represent constructs; squares represent indicators; regression coefficients are standardized to aid comparison; all regression coefficients are significant at $p \leq .05$.

Table 18. Fit Statistics for the Final Measurement Model of Depression

Factor	Observed Indicators	Regression Weights* (Factor Loading Estimates)		R^2
		Unstandardized	Standardized	
$\chi^2(4) = 8.393$ $p = 0.0782$ $CFI = 0.999$ $TLI = 0.999$ $RMSEA = 0.01$ $WRMR = 0.44$	Unable to Shake Blues	1.000**	0.831	0.690
	Feel Depressed	1.074	0.893	0.797
	Feel Happy *(reverse-scored)*	0.696	0.578	0.334
	Feel Sad	1.025	0.852	0.725
	Life Not Worth Living	0.780	0.648	0.419

Abbreviations: *CFI* = comparative fit index; *TLI* = Tucker-Lewis index; *RMSEA* = root mean squared error of approximation; *WRMR* = weighted root mean squared residual; R^2 = multiple squared correlation

* All factor loadings or path coefficients were significant at $p \leq .05$;

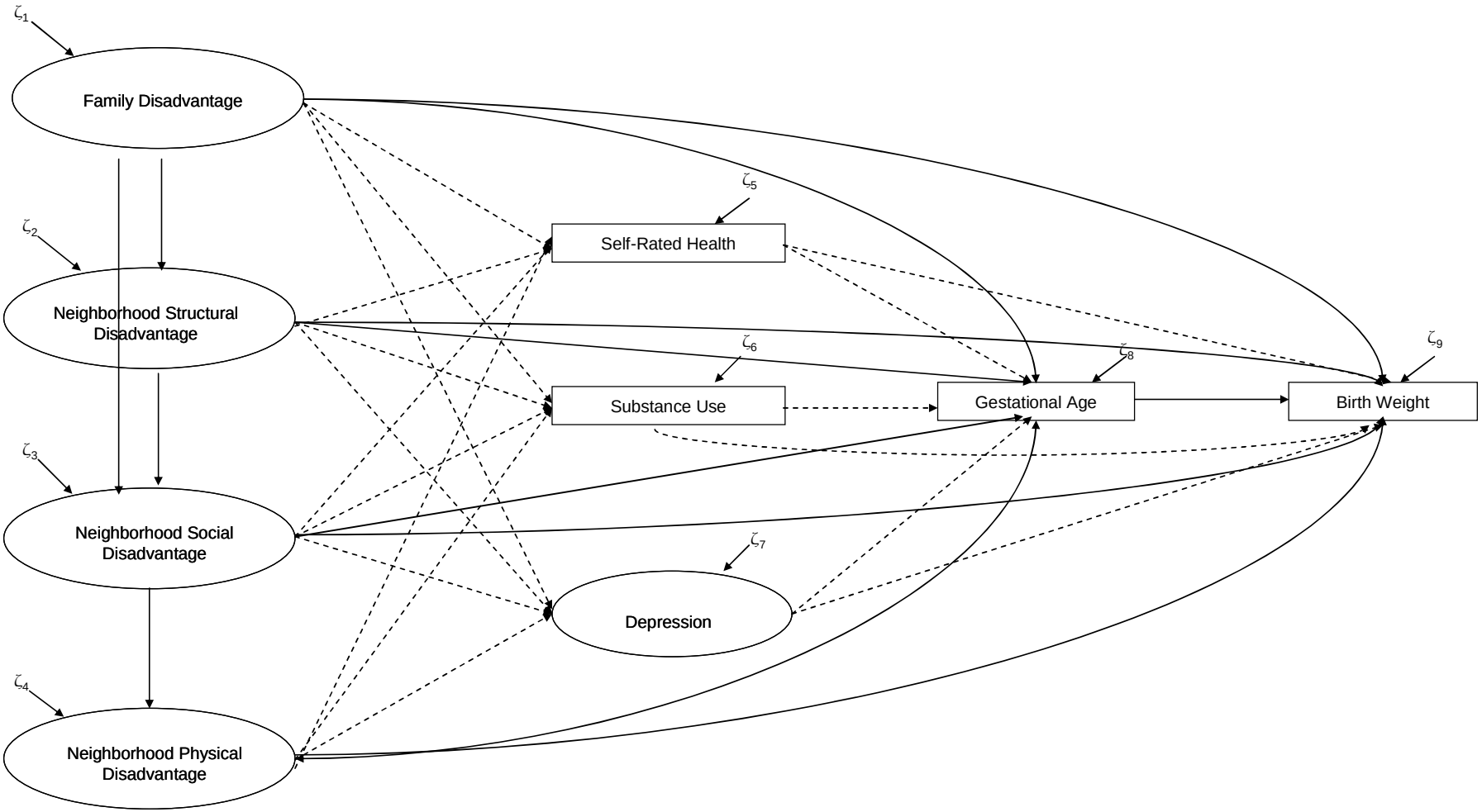
** Parameter constrained to 1.00 to scale the construct; constrained parameters were not tested for statistical significance

5.4.3. Structural Model

Using the final measurement model of depression, as well as the measurement models developed for Aim 2, I estimated a structural equation model linking neighborhood- and family-level disadvantage to birth weight, with physical, behavioral, and psychosocial risk and gestational age as mediators of the relationship. The model I set out to test for this study aim is illustrated in Figure 19 below. It reflects the following underlying assumptions discussed in Chapter 3: (1) exposure to any form of disadvantage during childhood has a negative impact on childhood health, and increases the likelihood of engaging in substance use and experiencing depressive symptoms during childhood, and (2) childhood health, substance use, and depression have long-term consequences for subsequent birth outcomes. As the figure shows, I retained the modifications made to the latent disadvantage variables during Aim 2 (i.e., explication of the relationships among them) and lifted the zero constraint on the disturbance for family disadvantage.

By all the goodness of fit statistics except for the chi-square statistic, the original model was well-fitted (χ^2 [38] = 72.61, p = 0.000, CFI = 0.979, TLI = 0.979, WRMR = 1.019). Although these results were good enough to accept the model without modification, I had already hypothesized that the disturbances among the mediators may be correlated. (Modifications to the model are depicted in Figure 20 on page 139 and their impacts on the overall model fit statistics are summarized in Table 19 on page 138). Although lacking information to support this speculation, it seemed logical to expect some shared sources of variability underlying self-reported health, substance use, and depressive symptoms other than the factors in the model.

Figure 19. Original Structural Equation Model Linking Childhood Disadvantage, Mediators, and Subsequent Birth Outcomes



Thus, I allowed the disturbances of the mediators to intercorrelate *(Modification 5*). The correlation between the disturbances of self-rated health and depression was not statistically significant so I removed it. I then performed a chi-square difference test to compare the model with the remaining correlations between disturbances to the model without those correlations. The chi-square difference statistic was slightly higher and borderline significant ($\chi^2_{diff}[2] = 5.953$, $p = 0.0510$), but because the correlations were statistically significant, I retained them. The impact of these maneuvers on the overall model fit statistics was very minor (Table 19).

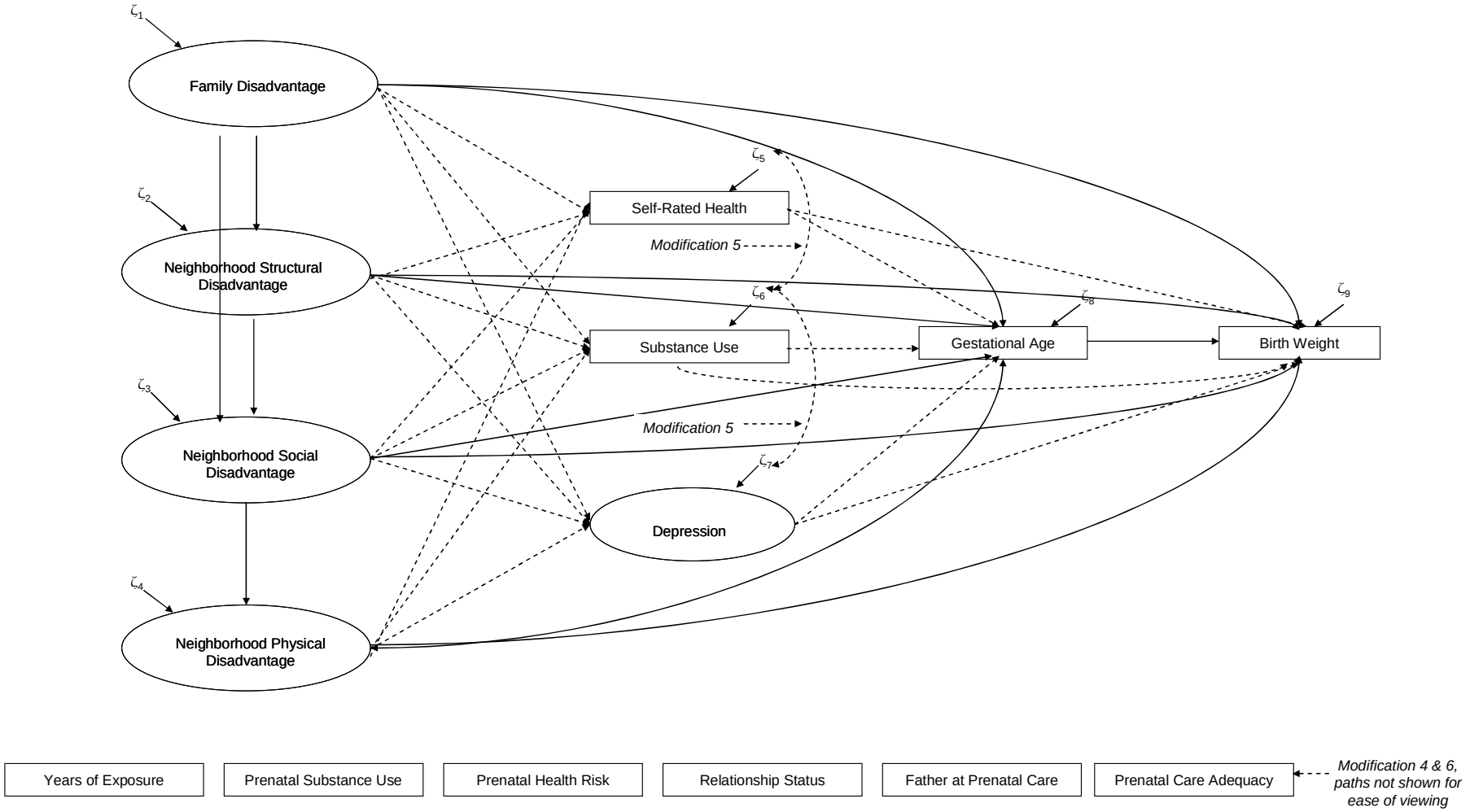
Next, I added the control variables retained from Aim 1 to produce a baseline adjusted model with birth weight simultaneously regressed on all the control variables. It also seemed appropriate to control for the prenatal behavioral risk score with respect to childhood substance use *(Modification 6)*. The adjusted model fit statistics were indicative of good model fit ($\chi^2[43] = 70.46$, $p = 0.000$, CFI/TLI = 0.98, RMSEA = 0.021, WRMR = 1.04).

Table 19. Measures of Overall Model Fit for Original and Modified Structural Models of the Relationship between Childhood Disadvantage, Mediators, and Birth Outcomes

Models	X^{2*}	df	CFI	TLI	RMSEA	WRMR
Model Carried Over from Aim 2 with Mediators Added	72.61	38	0.98	0.98	0.01	1.02
Modification 5: Added correlated errors among the mediators	71.89	38	0.98	0.98	0.01	1.01
Modification 6: Added control variables to final unadjusted model	70.46	43	0.98	0.98	0.02	1.04
Final Adjusted Model: Dropped non-significant control variables	77.13	39	0.98	0.98	0.02	1.05

Abbreviations: *df* = degrees of freedom; *CFI* = comparative fit index; *TLI* = Tucker-Lewis index; *RMSEA* = root mean squared error of approximation; *WRMR* = weighted root mean squared residual.
* Chi-square values generated from WLSMV estimation cannot be used for chi-square difference testing

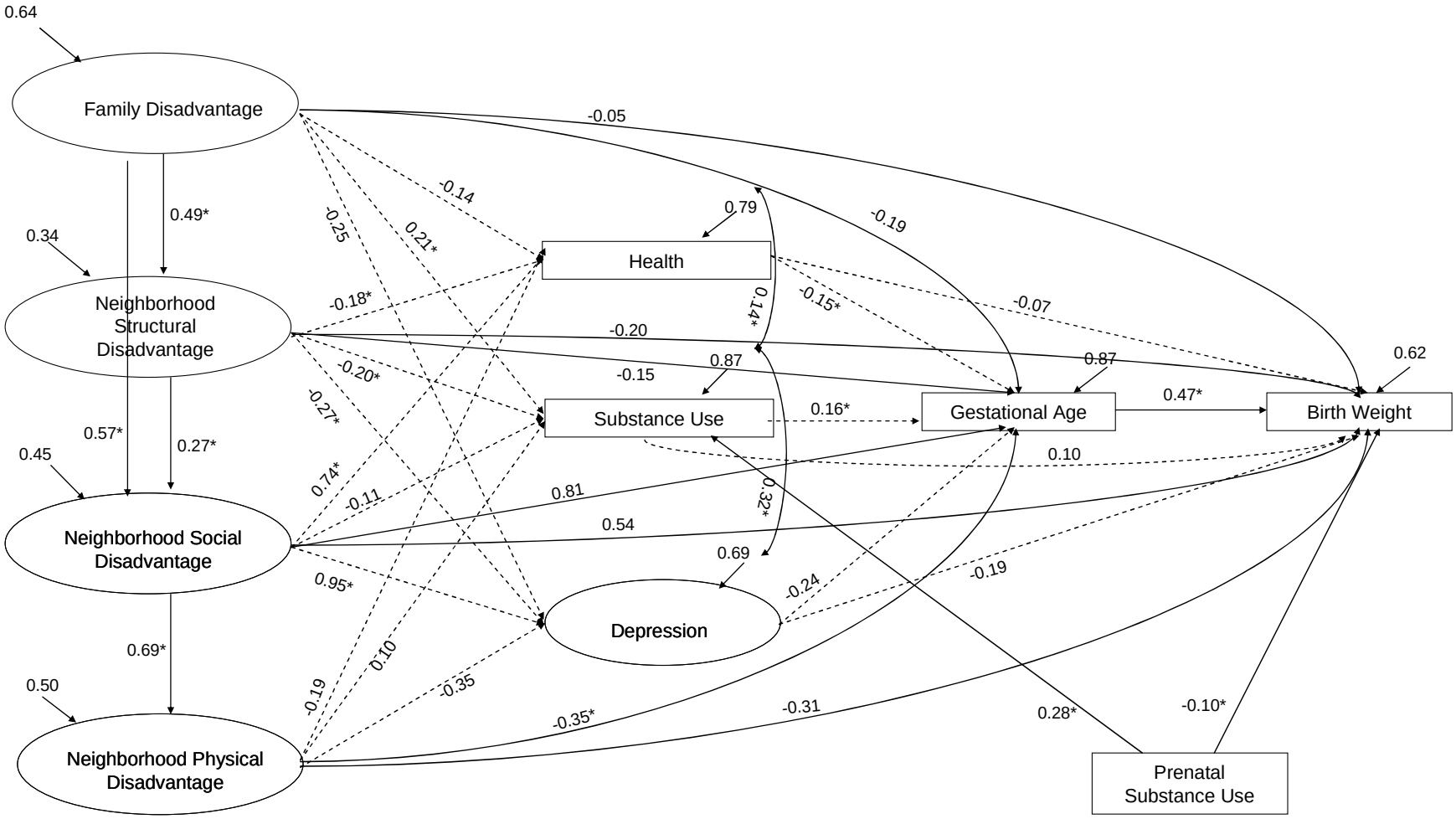
Figure 20. Modifications to the Structural Equation Model Linking Childhood Disadvantage, Mediators, and Subsequent Birth Outcomes



Consistent with my analysis plan, I then constrained all non-significant paths from the control variables to zero and tested the extent to which doing so resulted in improved or worsened model fit. The chi-square difference test produced a non-statistically significant chi-square (χ^2_{diff} [5] = 2.922, p = 0.719), suggesting that the constrained model provided no better or worse fit to the data than the unconstrained model. Thus, I dropped the control variables that were not significantly associated with other variables in the model, leaving only prenatal substance use in the model. The fit statistics for this final model are presented in the last row of Table 19. Although the values of the chi-square statistic and the WRMR increased somewhat from the previous modification, the other fit indices were unchanged.

As Figure 21 below and Table 20 (page 142) show, the relationships among the latent disadvantage variables remained statistically significant when the mediators were introduced into the model. Of the two variables significantly associated with gestational age in Aim 2 (i.e., neighborhood social and physical disadvantage), only neighborhood physical disadvantage retained its relationship with similar magnitude. No direct effects between the other disadvantage variables and gestational age were present. Instead two of them had direct effects on the mediators. In particular, neighborhood social disadvantage and neighborhood structural disadvantage had statistically significant direct effects on self-rated health, substance use, and depression. Family disadvantage had a borderline significant positive relationship with substance use. In turn, substance use and self-rated health were significantly associated with gestational age.

Figure 21. Final Structural Equation Model Linking Control Variables, Childhood Disadvantage, Mediators and Subsequent Birth Outcomes with Adjusted Standardized Parameter Estimates**



* $p \leq 0.05$, determined from the unstandardized solution

** controlling for prenatal substance use

Table 20. Mean and Variance Adjusted Weighted Least Squares Regression Weights for the Final Structural Model of the Relationship between Childhood Disadvantage, Mediators, and Birth Outcomes

Path	Unstandardized Estimate*	Standard Error	Standardized Estimate*	*z***
FAM → STRUC	0.047	0.008	0.489	5.809
FAM → SOC	0.187	0.047	0.567	3.978
STRUC → SOC	0.905	0.447	0.266	2.025
SOC → PHYS	0.776	0.245	0.693	3.175
FAM → HEALTH	-0.106	0.124	-0.138	-0.850
FAM → SUBUSE	0.162	0.083	0.208	1.950
FAM → DEPRESS	-0.155	0.112	-0.246	-1.391
STRUC → HEALTH	-1.379	0.662	-0.175	-2.083
STRUC → SUBUSE	-1.584	0.497	-0.197	-3.188
STRUC → DEPRESS	-1.738	0.598	-0.267	-2.907
SOC → HEALTH	1.709	0.472	0.737	3.620
SOC → SUBUSE	-0.268	0.485	-0.114	-0.553
SOC → DEPRESS	1.822	0.564	0.952	3.232
PHYS → HEALTH	-0.397	0.298	-0.192	-1.331
PHYS → SUBUSE	0.204	0.267	0.097	0.766
PHYS → DEPRESS	-0.599	0.345	-0.351	-1.737
FAM → GEST	-0.245	0.301	-0.190	-0.814
STRUC → GEST	-1.992	1.638	-0.150	-1.216
SOC → GEST	3.179	1.728	0.813	1.839
PHYS → GEST	-1.234	0.610	-0.353	-2.024
HEALTH → GEST	-0.247	0.112	-0.146	-2.209
SUBUSE → GEST	0.261	0.120	0.157	2.170
DEPRESS → GEST	-0.495	0.297	-0.242	-1.669

Abbreviations: *FAM* = family disadvantage, *STRUC* = neighborhood structural disadvantage, *SOC* = neighborhood social disadvantage, *PHYS* = neighborhood physical disadvantage, *HEALTH* = self-rated health, *SUBUSE* = substance use, *DEPRESS* = depression, *GEST* = gestational age

* Controlling for prenatal substance use

** z score reported for unstandardized estimate

*** Birth weight was re-scaled by 1/100 to avoid computational or convergence issues; all unstandardized values for parameters estimated when birth weight is the dependent variable should be multiplied by 100 before interpreting.

Table 21 continues on next page

Table 20. Mean and Variance Adjusted Weighted Least Squares Regression Weights for the Final Structural Model of the Relationship between Childhood Disadvantage, Mediators, and Birth Outcomes *(continued)*

Path	Unstandardized Estimate*	Standard Error	Standardized Estimate*	*z***
FAM → BWT***	-0.178	0.636	-0.045	-0.280
STRUC → BWT***	-8.266	4.829	-0.203	-1.712
SOC → BWT***	6.499	5.773	0.543	1.126
PHYS → BWT***	-3.271	2.493	-0.306	-1.312
HEALTH → BWT***	-0.380	0.373	-0.074	-1.018
SUBUSE → BWT***	0.486	0.401	0.096	1.214
DEPRESS → BWT***	-1.196	0.983	-0.191	-1.216
GESTAGE → BWT***	1.442	0.269	0.472	5.367

Abbreviations: *FAM* = family disadvantage, *STRUC* = neighborhood structural disadvantage, *SOC* = neighborhood social disadvantage, *PHYS* = neighborhood physical disadvantage, *HEALTH* = self-rated health, *SUBUSE* = substance use, *DEPRESS* = depression, *GEST* = gestational age, *BWT* = birth weight

* Controlling for prenatal substance use

** z score reported for unstandardized estimate

*** Birth weight was re-scaled by 1/100 to avoid computational or convergence issues; all unstandardized values for parameters estimated when birth weight is the dependent variable should be multiplied by 100 before interpreting.

Table 21 summarizes the direct, indirect, and total effects of all of the variables in the model on birth outcomes. It shows that neighborhood physical disadvantage had a statistically significant direct effect on gestational age with non-significant indirect effects, confirming that it was not mediated by other variables in the model. Despite relationships among the other variables in the model, the direct and indirect effects of these relationships on the birth outcomes were not statistically significant. The total effects of neighborhood structural disadvantage and neighborhood physical disadvantage on birth weight were statistically significant, as were the total effects of family disadvantage and neighborhood physical disadvantage on gestational age. The total effects of family disadvantage and neighborhood structural disadvantage were relatively small, however.

Table 21. Direct, Total, and Indirect Effects of Childhood Disadvantage on Birth Outcomes with Mediators in the Model*

Mediated Path	Unstandardized Estimate**	Standard Error	*Z****	Standardized Estimate**
Total Effect of Family Disadvantage on Gestational Age	0.114	0.055	2.084	0.088
Total Indirect Effect	0.359	0.286	1.256	0.278
Direct Effect	-0.245	0.301	-0.814	-0.190
Total Effect of Family Disadvantage on Birth Weight*****	0.389	0.207	1.876	0.098
Total Indirect Effect	0.567	0.631	0.899	0.144
Direct Effect	-0.178	0.636	-0.280	-0.045
Total Effect of Neighborhood Structural Disadvantage on Gestational Age	-0.140	0.705	-0.198	-0.011
Total Indirect Effect	1.852	1.586	1.167	-0.139
Direct Effect	-1.992	1.638	-1.216	-0.150
Total Effect of Neighborhood Structural Disadvantage on Birth Weight*****	-5.050	2.471	-2.043	-0.124
Total Indirect Effect	3.216	4.046	0.795	0.079
Direct Effect	-8.266	4.829	-1.712	-0.203
Total Effect of Neighborhood Social Disadvantage on Gestational Age	1.175	0.870	1.351	0.301
Total Indirect Effect	-2.004	1.013	-1.978	-0.512
Direct Effect	3.179	1.728	1.839	0.813
Total Effect of Neighborhood Social Disadvantage on Birth Weight*****	3.446	2.659	0.195	0.288
Total Indirect Effect	-3.053	4.102	-0.744	-0.255
Direct Effect	6.499	5.773	1.126	0.543
Total Effect of Neighborhood Physical Disadvantage on Gestational Age	-0.786	0.395	-1.992	-0.225
Total Indirect Effect**	0.448	0.294	1.524	0.128
Direct Effect	-1.234	0.610	-2.024	-0.353

* Specific indirect effects not shown

** Controlling for prenatal substance use

*** z score reported for unstandardized estimate

**** Birth weight was re-scaled by 1/100 to avoid computational or convergence issues; all unstandardized values for parameters estimated when birth weight is the dependent variable should be multiplied by 100 before interpreting.

Table 21 continues on next page

Table 21. Direct, Total, and Indirect Effects of Childhood Disadvantage on Birth Outcomes with Mediators in the Model* *(continued)*

Mediated Path	Unstandardized Estimate**	Standard Error	*Z****	Standardized Estimate**
Total Effect of Neighborhood Physical Disadvantage on Birth Weight*****	-3.438	1.702	-2.020	-0.322
Total Indirect Effect	-0.167	1.129	-0.148	-0.016
Direct Effect	-0.245	0.301	-0.814	-0.306

* Specific indirect effects not shown

** Controlling for prenatal substance use

*** z score reported for unstandardized estimate

**** Birth weight was re-scaled by 1/100 to avoid computational or convergence issues; all unstandardized values for parameters estimated when birth weight is the dependent variable should be multiplied by 100 before interpreting.

Although not listed in Table 21, fewer specific indirect pathways were found to be statistically significant at the $p \leq 0.05$ level when compared to the model without the mediators in Aim 2 (Appendix F). The relationships between the latent disadvantage variables were retained in the mediation model, but the presence of the mediators eliminated the statistically significant indirect effects on birth outcomes found in Aim 2 that operated solely through those relationships. The only exceptions were the pathways from neighborhood social and physical disadvantage to birth weight through gestational age; they remained statistically significant and had the highest regression coefficients. Finally, although the individual paths from neighborhood structural disadvantage to self-rated health and from self-rated health to gestational age were statistically significant, the overall indirect effect of this pathway was not statistically significant.

A considerable amount of the variance in each of the model variables remained unexplained by other variables in the model, even after adding the mediators. This was especially true for gestational age, self-rated health, and substance use—all three of which

had multiple squared correlations (R^2's) of less than 0.40. The mediators resulted in only a four percent increase in the variance of gestational age explained by the model, and only a slightly higher increase in the explained variance of birth weight. Again, much of the variance in birth weight was likely attributable to its relationship with gestational age.

5.4.4. Results of Analyses Stratified by Race

The final study questions were whether the relationship between childhood disadvantage and subsequent birth outcomes varied by race and whether the moderated relationship was mediated by childhood behavioral, psychosocial, and health risk. In Section 5.2, I discussed the presence of a statistically significant racial difference in birth weight; the tables in Appendix D show that racial differences also were present with regard to many indicators of childhood neighborhood and family disadvantage, behavioral risk, psychosocial risk, and physical health risk. As we saw in the previous sections, however, unadjusted bivariate relationships may tell us little about how the overall structural equation model operates.

Thus, I estimated the final adjusted model depicted in Figure 21 separately for each racial group to identify the existence of differences in path estimates, magnitudes of the coefficients, variance explained in the key study variables, factor loadings at the construct level, and statistical significance of the paths. These baseline adjusted models differed in the following ways: (1) the model fit was better for Whites than for Blacks; (2) the findings for Whites more closely matched the findings for the overall sample than the findings for Blacks; and (3) only the associations between family disadvantage and neighborhood structural and social disadvantage, between depression and neighborhood social

disadvantage, and between gestational age and birth weight were statistically significant for the Black sample. (Table 22 provides the comparison of all path estimates for the race-specific models, which can be compared to the full sample estimates in Table 20 on page 142. Diagrams illustrating the statistically significant paths for the full sample, White subsample, and Black subsample models are provided in Appendix G).

Table 22. Adjusted Influence of Disadvantage and Mediators on Birth Outcomes, by Race

Paths	Parameter Estimates, by Race					
	Black			White*		
	Unstd	Z**	Std	Unstd	Z**	Std
FAM → STRUC	0.026	2.573	0.392	0.049	4.321	0.493
FAM → SOC	0.179	2.280	0.827	0.293	3.029	0.738
STRUC → SOC	0.676	1.588	0.208	0.135	0.189	0.034
SOC → PHYS	0.470	1.858	0.590	0.951	2.878	0.751
FAM → HEALTH	-0.704	-0.611	-0.645	-0.379	-1.338	-0.410
FAM → SUBUSE	-0.134	-0.140	-0.106	-0.121	-0.748	-0.122
FAM → DEPRESS	-0.145	-0.767	-0.432	-0.171	-1.103	-0.216
STRUC → HEALTH	-2.294	-0.615	-0.358	0.094	0.106	0.010
STRUC → SUBUSE	-0.656	-0.692	-0.134	-0.731	-1.311	-0.073
STRUC → DEPRESS	-1.265	-1.882	-0.251	-0.988	-1.878	-0.124
SOC → HEALTH	1.529	0.779	0.291	1.974	2.319	0.849
SOC → SUBUSE	0.345	0.247	0.229	0.340	0.617	0.136
SOC → DEPRESS	1.568	2.089	0.889	1.657	2.576	0.835
PHYS → HEALTH	-0.558	-0.904	-0.225	-0.329	-0.922	-0.179
PHYS → SUBUSE	0.089	0.247	0.047	0.004	0.016	0.002
PHYS → DEPRESS	-0.296	-0.770	-0.152	-0.579	-1.482	-0.370

Abbreviations: *FAM* = family disadvantage, *STRUC* = neighborhood structural disadvantage, *SOC* = neighborhood social disadvantage, *PHYS* = neighborhood physical disadvantage, *HEALTH* = self-rated health, *SUBUSE* = substance use, *DEPRESS* = depression

* White model adjusted for prenatal substance use

** z score reported for unstandardized estimate

*** Birth weight was re-scaled by 1/100 to avoid computational or convergence issues; all unstandardized values for parameters estimated when birth weight is the dependent variable should be multiplied by 100 before interpreting.

Table 22 continues on next page

Table 22. Adjusted Influence of Disadvantage and Mediators on Birth Outcomes, by Race *(continued)*

Paths	Parameter Estimates, by Race					
	Black			White*		
	Unstd	Z**	Std	Unstd	Z**	Std
FAM → GEST	0.082	0.080	0.096	-1.175	-1.209	-0.840
STRUC → GEST	0.285	0.079	0.022	-0.477	-0.167	-0.034
SOC → GEST	-0.051	-0.008	-0.013	6.725	2.189	0.912
PHYS → GEST	-0.013	-0.015	-0.003	-2.274	-2.457	-0.819
HEALTH → GEST	-1.059	-0.122	-0.029	-0.484	-2.088	-0.320
SUBUSE → GEST	0.221	1.302	0.085	0.163	-0.970	0.115
DEPRESS → GEST	-0.128	-0.366	-0.051	-0.660	-1.586	-0.372
FAM → BWT***	0.261	0.100	0.109	0.377	0.137	0.084
STRUC → BWT***	0.243	0.025	0.007	-3.446	-1.291	-0.076
SOC → BWT***	0.243	0.015	0.022	0.100	0.006	0.009
PHYS → BWT***	-2.093	-0.773	-0.151	-0.750	-0.141	-0.084
HEALTH → BWT***	0.108	0.085	0.019	0.035	0.031	0.007
SUBUSE → BWT***	0.373	0.852	0.051	-0.009	-0.024	-0.002
DEPRESS → BWT***	-0.143	-0.156	-0.020	-0.226	-0.144	-0.040
GESTAGE → BWT***	1.759	21.876	0.627	1.653	2.189	0.513

Abbreviations: *FAM* = family disadvantage, *STRUC* = neighborhood structural disadvantage, *SOC* = neighborhood social disadvantage, *PHYS* = neighborhood physical disadvantage, *HEALTH* = self-rated health, *SUBUSE* = substance use, *DEPRESS* = depression, *GEST* = gestational age, *BWT* = birth weight

* White model adjusted for prenatal substance use

** z score reported for unstandardized estimate

*** Birth weight was re-scaled by 1/100 to avoid computational or convergence issues; all unstandardized values for parameters estimated when birth weight is the dependent variable should be multiplied by 100 before interpreting.

Wondering whether the findings for the Black sample were due to my inclusion of the proportion of Black residents in the census tract as a causal indicator of the model for neighborhood structural disadvantage, I constrained this path to zero. Although the relationships between neighborhood structural disadvantage, neighborhood social disadvantage, and depression became statistically significant when I did so, chi-square difference tests indicated that the reduced model did not provide a better fit to the data.

Thus, I removed the zero constraint. Because no paths to the birth outcomes were statistically significant for the Black sample, I did not reduce the models by constraining non-statistically significant paths to zero within each racial group in order to make further comparisons.

5.5. Summary of Results

Table 23 below summarizes the extent of the support for each of the study hypotheses based on the findings reported in this chapter. This table shows that my findings are mixed with respect to the hypotheses.

5.5.1. Summary of Results for Aim 1

My hypotheses regarding the prevalence of adverse birth outcomes overall and by maternal age were generally not supported. While the prevalences of LBW and PTB for the study sample were lower than the overall prevalence for all women of childbearing age, these prevalences were not the same as other national data for women of the same maternal ages. In fact, they were considerably lower. Moreover, instead of demonstrating a decrease in adverse birth outcomes with increasing maternal age as we would expect from national data for this age group, my sample exhibited the opposite pattern.

My hypotheses regarding the effect of maternal race on adverse birth outcomes were only partially supported as well. In particular, I found significant racial differences in mean birth weight and the rate of LBW—differences that remained even after controlling for sociodemographic and pregnancy characteristics—but no racial difference in mean

gestational age and a non-significant racial difference in PTB. Given the degree of correlation between the two outcomes, these mixed results were unexpected.

Table 23. Summary of Support for Study Hypotheses

Hypothesis	Supported		
	Yes	Partial	No
H1.3.1: Mean birth weight and mean gestational age will decrease with maternal age among the study sample.			✓
H1.3.2: Mean birth weight and mean gestational age will be lower among Blacks than among Whites.		✓	
H1.4.1: The prevalence of LBW and PTB will decrease with increasing maternal age among the study sample.			✓
H1.4.2: The prevalence of LBW and PTB will be higher among Blacks than among Whites.		✓	
H2.1.1: Exposure to family-level disadvantage during childhood will be associated with decreases in birth weight and gestational age and, therefore, increased risks for LBW and PTB.		✓	
H2.1.2: Exposure to neighborhood-level disadvantage during childhood will be associated with decreases in birth weight and gestational age and, therefore, increased risks for LBW and PTB.		✓	
H2.2.1: At the family and neighborhood level, the effect of exposure to disadvantage during childhood on subsequent birth outcomes will be greater for Blacks than for Whites.			✓
H3.1.1: Women who were exposed to family disadvantage in childhood will demonstrate more childhood behavioral, psychosocial, and physical health risks than women who were not exposed to family and neighborhood disadvantage in childhood.		✓	
H3.1.2: Women who were exposed to neighborhood disadvantage in childhood will demonstrate more childhood behavioral, psychosocial, and physical health risks than women who were not exposed to family and neighborhood disadvantage in childhood.		✓	

Table 23 continues on next page

Table 23. Summary of Support for Study Hypotheses *(continued)*

Hypothesis	Supported		
	Yes	Partial	No
H3.1.3: Exposure to behavioral, psychosocial, and physical health risks during childhood will be associated with decreases in birth weight and gestational age and, therefore, increased risks for LBW and PTB.		✓	
H3.1.4: The relationship between exposure to disadvantage in childhood and subsequent adverse birth outcomes will not be fully mediated by behavioral, psychosocial, and physical health risk.			✓
H3.2.1: Exposure to family and neighborhood disadvantage during childhood will produce divergent pathways of behavioral, psychosocial, and physical risk for Black and White women that lead to later disparities in adverse birth outcomes.		✓	

5.5.2. Summary of Results for Aim 2

My hypotheses that all family-level and neighborhood-level disadvantage variables would be directly associated with decreases in birth weight and gestational age were not supported. The findings of the analysis for Aim 2 demonstrate direct effects on gestational age only for neighborhood physical and social disadvantage. On the other hand, they suggest that the effects of family disadvantage and neighborhood structural disadvantage on birth weight were fully mediated by those other neighborhood processes and gestational age. These findings contrast with the unadjusted bivariate associations that showed large, statistically significant effects of each indicator of neighborhood structural disadvantage on birth weight, and the relatively weaker and non-statistically significant bivariate associations between other neighborhood-level measures and birth weight. It is possible

that the effect of neighborhood structural disadvantage may have been attenuated by the adjustment for family-level disadvantage.

Likewise, because gestational age was a mediator of all relationships between birth weight and the latent variables in the model, it was controlled for in the analyses of those relationships, thereby attenuating the effects of the other latent variables on birth weight. Despite the statistically significant direct effects of neighborhood social and physical disadvantage on gestational age, however, they accounted for only a small proportion of its variance. And gestational age probably accounts for nearly all of the variance (33%) in birth weight in this model because no other direct effects were present for birth weight, except for the effect of prenatal substance use (a control variable).

5.5.3. Summary of Results for Aim 3

My hypothesis that the relationship between birth outcomes and family-level and neighborhood-level disadvantage would be partially mediated by more proximal risk factors was not supported. Other than neighborhood physical disadvantage which only had a direct effect on gestational age, all other relationships between disadvantage and birth outcomes in the model could be characterized as full mediation. The addition of the mediators to the model did not, however, result in substantial increases in the amount of variance in the birth outcomes accounted for by the model. The existence of a considerable amount of unexplained variance, therefore, limits the extent to which we can deem any of the factors in the model important to understanding adverse birth outcomes, at least in this sample. It is clear, however, that childhood depression did not contribute to these outcomes.

My hypotheses regarding the role of race with respect to the final adjusted model were not fully supported. In particular, I could not identify or investigate a difference in the magnitudes of the effects of model factors on birth outcomes due to the poor overall fit of the model and the non-significance of the path estimates for the Black sample. The findings suggest that the pathways through which adverse birth outcomes occur in Blacks and Whites may differ.

CHAPTER 6

DISCUSSION

6.1. Synthesis of Findings

The implications of perinatal health disparities for population health and the health care system in this country are tremendous. As a result, both the Healthy People 2000 and 2010 Reports established the reduction of perinatal health disparities as one of their primary goals (USDHHS, 2000). Achieving this goal requires that we understand the contributors to racial disparities in adverse birth outcomes, and particularly those factors that place African American women at increased risk. In addition, as Link and Phelan (1995) argue, it is important to contextualize proximal risk factors in order to create more effective interventions. Unfortunately, as discussed in Chapter 2, traditional research to identify risk factors for adverse birth outcomes often has been reductionist, decontextualized, and biased toward biological, behavioral, and psychosocial explanations. Not only have these explanations failed to account fully for the incidence of adverse birth outcomes, but they have also been unable to explain the difference in rates of adverse birth outcomes between African American and White women.

Thus, the purpose of this dissertation was to place proximal risk factors for adverse birth outcomes within the context of neighborhood- and family-level measures of

disadvantage experienced prior to the prenatal period. In particular, I sought to understand the impact of mothers' exposure to disadvantage during childhood/adolescence on subsequent birth outcomes, while taking into account the proximal behavioral, psychosocial, and physical health risk factors that mothers experienced during the intervening years. In addition, I examined the extent to which these relationships varied by race.

My results suggest that studying pre-pregnancy factors may offer some added value to studies of adverse birth outcomes, although insufficient control variables related to the prenatal period limit our ability to determine exactly how much these factors add. The fact that a relationship between childhood substance use and gestational age remained in the presence of a control for prenatal substance use (which was correlated with childhood substance use) is just one indication. My findings also suggest that disadvantage experienced during childhood/adolescence is among the prepregnancy factors that may play a role—albeit an indirect one—in subsequent birth outcomes. With the exception of neighborhood physical disadvantage, the relationships between other measures of disadvantage and birth outcomes were fully mediated. This finding is consistent with the findings of Sheehan's (1998) study of the relationship between economic stress, family stress, and birth weight.

The mediation pathways among the disadvantage variables were somewhat consistent with other studies that have found neighborhood social and physical disorder mediates the relationship between neighborhood impoverishment and health outcomes (e.g., Franzini et al., 2005). On the other hand, my model suggests the possibility of self-

selection of individuals into neighborhoods or, at a minimum, confounding of the relationship between neighborhood-level disadvantage and birth outcomes by family disadvantage—a possibility that contradicts the hypothesis advanced by others that individual-level disadvantage is a mediator or moderator of contextual effects (Diez-Roux, 2001; Macintyre, Ellaway, & Cummins, 2002; Kobetz, Daniel, & Earp, 2004). Nevertheless, neighborhood-level variables demonstrated a more consistent relationship to other variables in the model than did family disadvantage; in doing so, they lend credence to the assertion made in Chapter 2 that multiple indicators of social and economic conditions other than income and education (which may be poor indicators of actual living conditions) could enhance our measurement of socioeconomic conditions (O'Campo & Schempf, 2005, Misra, O'Campo, & Strobino, 2001; Rich-Edwards, 2002). In addition, my study speaks to the question about the relative importance and nature of the relationship between subjective appraisals, direct observation, and objective measures of neighborhood characteristics mentioned in Chapter 2 (Ingoldsby et al, 2006; Weden, Carpiano, & Robert, 2008; Wen, Hawkley, & Cacioppo, 2006; Bowling & Stafford, 2007). It demonstrates how all three of these measures can be used simultaneously to create latent variables that capture neighborhood characteristics meaningfully.

The mediation pathways involving proximal risk factors in my study have been widely speculated to underlay the mechanisms by which neighborhood characteristics affect adverse birth and other outcomes (e.g., Reagan & Salsberry, 2005; Matheson et al., 2006; Franzini et al., 2005; Wen, Hawkley, & Cacioppo, 2006; Brewster, 1994; Browning & Cagney, 2002). My study offers support for behavioral and health pathways, but not psychosocial

pathways. Specifically, childhood depression was not associated with birth weight or gestational age, whether examined alone or in the full mediation model. This finding is inconsistent with a recent study—the only one of its kind—that found a small but statistically significant association between prepregnancy depression and preterm birth among Black and White women in the CARDIA study (Gavin et al., 2009).

My findings with regard to race are perhaps even more interesting. First, a Black-White disparity existed for birth weight in this subsample of Add Health Wave III respondents. Contrary to expectations, however, no such disparity appeared to exist for gestational age. In fact, mean gestational age was equal for Blacks and Whites and the difference in rates of PTB between the two groups was not significant nor in the expected direction. This could mean that among LBW infants in the study, more of the Black infants were small-for-gestational age than preterm. As noted in Chapter 4, the confounding of LBW with small-for-gestational age is among the reasons why it has often been recommended that research focus on PTB instead of LBW (IOM, 2006). This potential confounding was minimized in my study by placing gestational age along all pathways leading to birth weight and including it in all equations involving birth weight. In doing so, gestational age was controlled for in all analyses and, as a result, birth weight in my study actually represented fetal growth (or fetal growth restriction).

Other explanations for the inconsistent findings for birth weight and gestational age with respect to race may exist, however. As discussed in Chapter 2, some researchers have observed distinct gestational age distributions (Papiernik, Alexander, & Paneth, 1990; Patel et al., 2004), and others have shown distinct birth weight—gestational age combinations

(Adams et al., 1997), for Blacks and Whites. My study is not positioned to support or refute those findings, although it does suggest that other factors may play a role in the atypical distribution of gestational age that I found. For instance, about 20% of the sample reported that they did not receive prenatal care during the first trimester of their pregnancies. For these women, gestational age estimations—whether determined by ultrasound or the date of their last menstrual period—may have been less precise than women who initiated prenatal care during the first trimester. The accuracy of ultrasound in determining gestational age is known to decline as pregnancies advance; by the third trimester, it predicts gestational age within confidence intervals of up to ±3 weeks (Merz, 2005). In addition, these women may not have been as capable of remembering the date of their last menstrual period by the time they initiated prenatal care (if they initiated it at all). Another possible source of measurement error was the wording of the gestational age item itself. Having respondents engage in a calculation, rather than merely asking them the month or the week in which they delivered, introduced the possibility of miscalculation and under- or over-estimation on the part of some respondents. Finally, the distributions of birth outcomes by race (and overall) may differ from those found in other studies and other national data because of the non-response discussed in footnote 18.

The stratified analysis showed that the structural equation model for the full sample fit well for the White sample but far less well for the Black sample, even after the proportion of Black residents was removed from the model. Indeed the model did not offer any explanation for birth outcomes in the Black sample. The findings for the White sample were nearly identical to the findings for the full sample, and the same change occurred when the

proportion of Black residents was removed from the model as it did for the Black sample. On the one hand, this phenomenon suggests that the mechanisms underlying adverse birth outcomes differ across racial groups and that separate conceptual models are needed to guide studies of this problem. Sastry and Hussey (2003) arrived at the same conclusion when they found differences in covariate effects for Blacks and Whites in stratified analyses of birth weight in Chicago neighborhoods. On the other hand, the null results for the Black sample may be largely due to the fact that Black race is confounded with family-level and neighborhood-level disadvantage—the effects of which may be enduring. In fact, one study that examined the influence of social mobility (i.e., moving from disadvantage during childhood to a more advantaged social position in adulthood) on birth outcomes found that it reduced the risk of LBW for Whites but not for Blacks (Colen et al., 2006).

6.2. Strengths of the Study

This study built on previous studies of lifecourse and intergenerational effects of SES on birth outcomes by incorporating neighborhood-level measures of disadvantage, beyond traditional individual-level measures of SES. To date, such integration of lifecourse and contextual perspectives has only been undertaken with respect to health outcomes other than LBW and PTB (e.g., Wheaton & Clarke, 2003). In addition, only two other studies were found that examined the association between prepregnancy conditions other than childhood SES and adverse birth outcomes (Haas et al., 2005; Gavin et al., 2009), and only one of those two used prospective reports of prepregnancy conditions (i.e., depressive mood) that occurred earlier than the 12-month period immediately preceding pregnancy

(Gavin et al., 2009)). Unlike traditional birth outcome studies, therefore, the perspective that shaped my dissertation was a multilevel, intergenerational and contextual one, far upstream from—and much earlier than—the more traditional emphasis on the prenatal period or the 12-month period immediately preceding pregnancy. This shift positioned my study to be better able to help identify the actions that public health leaders need to take, and for which groups of women, in what contexts, and at what age across their life spans, in order to more effectively prevent adverse birth outcomes and enhance infant, childhood, and young adult health (Wang, 2006). In these ways, my study responded to some of the recommendations made in the Institute of Medicine's (2006) report on PTB listed in Chapter 1. It also responded to the Institute of Medicine's report on racial and ethnic differences in health over the lifecourse (Hertzman, 2004), and to the Healthy People 2010 objective to eliminate racial disparities in birth outcomes (USDHHS, 2000).

The study used data from the National Longitudinal Study of Adolescent Health (Add Health), a dataset that offers a unique opportunity to include in the analysis of the adult risk of adverse birth outcomes the effects of childhood disadvantage on the lives of women before they reach adulthood and/or begin childbearing. Although these effects are often under-studied (Attree, 2004), evidence is mounting for their importance (e.g., Boardman & Onge, 2005). My study examined psychosocial, behavioral, and physiological effects as a function of the social, contextual, and temporal environment in which they occurred. It is one of the few studies that attempted to understand the pathways by which social structural and contextual factors lead to adverse birth outcomes. Add Health is in many ways well-suited to such research, not only because of its longitudinality, but also

because of the sheer number and comprehensiveness of its health, psychosocial, behavioral, and contextual variables.

The study also used structural equation modeling (SEM), a technique that has been used in other neighborhood and lifecourse studies (e.g., Singh-Manoux, Richards, & Marmot, 2005; Franzini et al., 2005) but seems limited to only a handful of reproductive epidemiology studies, most of which were conducted a decade or more ago. For example, Lobel and colleagues (1992, 2000) tested two different models of the relationships among prenatal maternal stress (i.e., perceived stress, anxiety, and life events), birth weight, and gestational age. Similarly, Rini and colleagues (1999) tested a structural equation model of the relationship between prenatal maternal stress and birth outcomes, adding maternal personal resources (e.g., mastery, optimism, self-esteem) to their model. Zambrana and her colleagues (1999) used SEM to test a hypothesized model in which differences in birth weight were expected to be mediated by ethnic differences in substance use, psychosocial factors, and medical risk. Feldman and colleagues (2000) used SEM to test a hypothesized model of the relationship between maternal social support, infant birth weight, and fetal growth. Finally, Sheehan (1998) used SEM to test alternative models of how economic stress, family stress, and social support influence each other and LBW—i.e., whether their influence on LBW was simple and direct, or mediated by addictive behaviors. None of these studies incorporated contextual or prepregnancy factors as my study did.

SEM allowed me to simultaneously estimate the direct and indirect effects of early life exposure to neighborhood disadvantage on later birth outcomes, while accounting for potential measurement error all along the pathway. Thus, it is ideal for studies like mine

that seek to avoid replicating the single risk factor approaches common in epidemiological studies while accounting for the fact that some factors are measured imperfectly. SEM also allowed me to include multiple indicators of socioeconomic conditions, and correctly specify them as causal indicators of a single construct even in the presence of collinearity, rather than using other procedures such as factor analysis and principal components analysis which would have necessitated treating them as effect indicators, dropping items, or combining them into a single index. Using these latter practices could have led to misspecification bias (Perreira et al., 2005), altered the meaning of the construct (Bollen & Lennox, 1991), or made it difficult to interpret parameter estimates (Diamantopoulous, Riefler, & Roth, 2007). Being able to include multiple indicators for a single latent variable allowed me to have more information in my model—and to measure the underlying constructs with greater reliability—than in typical analyses of individual-level and neighborhood-level socioeconomic conditions and adverse birth outcomes. I also was able to include indicators of constructs from multiple data sources, even when the indicators tapped the same issue. For example, I used multiple sources (i.e., census, interviewer, and respondent) for indicators of parental education, and neighborhood social and physical disadvantage. SEM, therefore, offered a unique way to incorporate both subjective and objective indicators of neighborhood disadvantage in my study.

Another strength of the study was its consideration of race as a moderator rather than as a predictor in the model. Doing the latter reflects an assumption that the processes by which the other factors in the model impact birth outcomes are the same across racial groups, and results in what some have called "one size fits all" solutions (Sastry & Hussey,

2003). Treating race instead as a moderator of the relationship relaxes this assumption and allows for the possibility of identifying differences in the magnitude of the overall effect of disadvantage on birth outcomes across racial groups, as well as the process by which this overall moderated effect was produced (Muller, Judd, & Yzerbyt, 2005). In my case, the race-specific models were incomparable due to the lack of statistical significance throughout the model for the Black sample.

This study departed from the prevailing approach to studying adverse birth outcomes in other ways as well. In general, single risk factor studies are common, resulting in the development of separate interpretations for the relationships of risk factors and birth outcomes, and interventions targeted toward a relatively small group of high-risk individuals. Often times these studies focus on proximate determinants of adverse birth outcomes, which are inadequate for reducing population differences (Wise, 1993; Rose, 1992). My study simultaneously assessed multiple risk factors—from proximal to distal—at multiple levels to explore the relationships between and among them, determine their relative importance, and develop an interpretation of the findings for each variable within the context of a larger web of factors. This strategy has been widely recommended (e.g., Krieger, 1994) but too infrequently followed.

6.3. Limitations of the Study

Despite the significance and strengths of the study, several noteworthy limitations exist. From a conceptual standpoint, focusing solely on maternal characteristics reflects assumptions that birth outcomes are a result of maternal health and well-being and that

paternal factors are negligible. The latter assumption may be inaccurate. For example, recent evidence of an association between paternal age and birth outcomes has been found and, in studies where maternal characteristics were more predictive of birth outcomes than paternal characteristics, the latter were not completely unrelated (e.g., Miagone et al, 1991; Collins & David, 1993).

Several methodological limitations also are apparent. The most important limitations pertain to the age range of respondents in the Add Health dataset. In the Wave I dataset, childhood and adolescence are not equivalent; thus, capturing the living conditions of youth between the ages of 12 and 17 may not capture the same conditions as those experienced before age 12 (i.e., in childhood). On the other hand, my computation of the age at which respondents first moved to the home in which the Wave I interview took place revealed that half of all respondents were younger than age 12 when the move occurred. For those respondents, my neighborhood-level measures did capture childhood exposures as I had hoped to do. In either case, my study was positioned to assist in identifying earlier targets for policy and intervention than typical birth outcome studies afford.

Second, several limitations stem from the fact that the maximum age of respondents in the Wave III dataset was 26, thereby limiting the number of parous women available for study. Among other things, the low sample size (particularly for Black women in the study), combined with the large number of parameters estimated in the study, may have reduced the precision and power of the stratified analyses. In addition, correlations and covariances—the basis for SEM analysis—are less stable when estimated from small

samples; parameter estimates and chi-square tests of fit also are very sensitive to sample size (Ullman, 2007). Modeling the birth outcomes as continuous variables instead of categorical variables provided one source of protection against reducing power in the study. But doing so still may have been insufficient, despite the results of the power analysis performed prior to the study. The interval-halving procedure I used to calculate power and minimum required sample size indicated that the overall sample size and the race-specific sample sizes would have at least 80% power to detect an effect if one were present. However, the procedure is based on the RMSEA—a statistic that was consistently lower than the cutoff for good fit throughout my study, even when other fit statistics suggested less adequate fit.

The number of multiparous women in Add Health was also limited. Although this ensured that birth order was comparable across women, it reduced my ability to link changing exposures to sequential births over time. The study also was unable to examine changing exposures over time for several other reasons. First, exposure data from Wave III follows all births reported during that interview in time, thereby preventing the use of that dataset for repeated measures data on exposures. Second, even if I could have used Wave III data, the age range (18 – 26) between Wave I and Wave III truncated the lifecourse to a period that may have been insufficiently long enough to reveal noticeable effects of cumulative disadvantage or for measurable changes in social and contextual conditions to occur. At Wave III, some respondents had not moved from their adolescent residence, some were in college and, in any case, younger women are likely to be of lower SES than women at the other end of the reproductive age range.

The inclusion of Wave II data in the present study would not have solved these sample-related issues because the Wave II interview occurred just one year after the Wave I interview; previous studies have found moderate levels of agreement and intraclass correlations for some of the measures included in my study between the two waves (e.g., Boardman, 2006). For these reasons, I did not include one aspect of the full conceptual model in Figure 4 that depicts all possible lifecourse pathways (i.e., maternal young adult characteristics). As a result, cumulative and pathway models discussed in the lifecourse epidemiology literature (Section 3.1.2) were not tested. In addition, the independent variables and mediators in this study were collected at the same time point (Wave I), interfering with my ability to establish causality in the mediation analysis. Although other datasets with more births, older women, and more data collection points (such as the National Survey of Family Growth (NSFG) and National Longitudinal Survey of Youth (NLSY)) exist, Add Health is superior to them with respect to the number and comprehensiveness of its health, psychosocial, behavioral, and contextual variables. Moreover, findings from previous studies suggest that differences in pregnancy outcomes and evidence of weathering among African American women emerge in their early 20's (Geronimus, 1992). I conclude, therefore, that testing the latent effects of early exposure to disadvantage on the birth outcomes of this sample of younger women remained a worthwhile endeavor.

The sampling strategy I used may have introduced additional limitations. In particular, focusing on the first birth after the Wave I interview limited the length of time between the interview and the birth for those women who had more than one birth. This

suggests that a better strategy may have been to use the last birth or all births in the analysis. Because only 25% of the sample had more than one birth, however, both of these strategies were of questionable value in this research. In addition, I treated the respondent—not the birth–as the unit of analysis. An alternate sampling strategy would have been to use all births, treat the birth as the unit of analysis, and adjust calculated standard errors for clustering at the level of the respondent (Colen et al., 2006). This strategy, however, would have produced findings representative of the populations sampled in Add Health rather than generalizable to a national cohort. Using the first birth after the Wave I interview also subjected the prenatal variables collected during the Wave III interview that I used as control variables to the possibility of recall bias. On the other hand, the prospective assessment of the key independent variables in the study reduced the likelihood of recall bias among them.

Limitations associated with some of my measurement strategies may also exist. For example, the cumulative risk scoring approach used to develop the health and behavioral risk scores for the study assume that all risks included in the scores are weighted equally. This assumption may not be true. For example, research is beginning to suggest that the presence of bacterial vaginosis may be more strongly associated with the risk of adverse birth outcomes than other factors (Culhane et al., 2002), although I did not find this to be the case in my study. In addition, research discussed in Chapter 2 suggests that smoking during pregnancy may be associated with LBW among Whites but not among Blacks (Berg, Wilcox, & d'Almada, 2001). In my study, the prevalence of smoking prior to pregnancy (i.e., at Wave I) was nearly four times greater among Whites than Blacks, while alcohol use

and drug use were almost twice as high. During pregnancy, the prevalences for smoking, alcohol use, and drug use were approximately six, three, and one and half times higher among Whites than Blacks. And, in the stratified analyses, the relationship between prenatal substance use score and birth weight was statistically significant for the White sample but not for the Black sample. Cumulative risk scores like the ones used in my study could artificially mask some of the relationships that may exist between specific risk factors, such as smoking, and adverse birth outcomes—not only in terms of which relationships but also to what degree.

Maternal education may be another measure of questionable utility. My intention was to create a variable to reflect respondents' achievement of age-appropriate education by adjusting their reported education at Wave III according to their age at the time of the infant's birth and their Wave I education. I still believe such an approach is more desirable than using absolute education levels given their incomparability across women of different ages, although doing so confounds the measure with maternal age. In addition, the middle response category of my maternal education measure—i.e., completed high school, or less than 20 years old and did not complete high school—lacks specificity. Post hoc analysis revealed that some respondents who were under 20 years old and did not complete high school may not have reached age-appropriate education (e.g., being 19 but in the 9th grade at the time of their infant's birth). It is possible that results may have differed if these cases (as well as drop outs, when they could be identified) had been placed in a category separate from those who were under 20 years and did not complete high school because they were still in high school. The fact that age-appropriate education could vary

depending on whether respondents started school late, were left back, or stopped school temporarily while pregnant also could have had an effect on the quality of the maternal education variable. My inability to be more specific, and the necessity of artificially lumping dissimilar respondents into a single category, may explain why maternal education was not significantly related to birth weight in these analyses. It is also possible that the procedure I used to estimate maternal education (based on the levels reported at Wave I and Wave III education) did not produce reliable or valid estimates.

The prenatal measures I used as control variables in the study were derived from the Wave III interview which followed the referent birth by up to six years. This fact increased the likelihood of recall bias and made those items of questionable reliability. On the other hand, as noted earlier, it would have been helpful to include more prenatal measures that corresponded to the Wave I measures in order to distinguish the unique contributions of early life and contemporaneous exposures to adverse birth outcomes. However, spontaneous assessments of health and depression during pregnancy are likely impossible in longitudinal studies like Add Health, and it is not logical to ask about prenatal depression years after the pregnancy has ended.

Software limitations introduced another set of challenges for this study. Mplus is one of only two software programs that allow for structural equation modeling with complex survey data.[43] However, subpopulation analysis and multiple group analysis—two strategies warranted by my sampling and analysis plans—cannot be combined in Mplus. This hindered my ability to engage in multiple group analysis, which would have

[43]The current version of LISREL (8.8) includes statistical methods for conducting structural equation modeling with complex survey data, but it was released after this study already began.

allowed me to better test my moderation hypotheses by identifying differences in levels of effect across the racial groups and exactly which parameter (i.e., factor loadings, regression coefficients, disturbances, and measurement error) differed between them. As a result, the conclusions drawn from the stratified analysis are tentative in a SEM context.

Finally, limitations inherent in any study involving secondary analysis of existing data related to the availability and operationalization of the measures were present in my study. For example, youth were sampled for Add Health from schools, not neighborhoods, and the neighborhood clusters for my sample, which amounted to only 10% of the Wave III sample, did not contain a sufficient number of participants to warrant multilevel modeling. As a result I elected to treat the contextual variables as individual-level covariates. In addition, the vast majority of the neighborhood-level measures I used were compositional measures rather than contextual measures. The latter would have indicated group differences in outcomes due to group variables rather than group composition. Indeed this restriction prevents me from fully characterizing my study as a contextual one, and limits my ability to contribute to the ongoing debate about whether the effects of place on health are attributable to the places or to the people in them.

The classic downside of using a dataset not specifically designed for the purposes of the study is that it results in several tradeoffs. The largest tradeoff pertained to my sampling strategy and the sampling weights. My study had clear conceptual and methodological reasons for deviating from typical birth outcome studies, which use samples derived from prenatal clinic patient populations or hospital medical records. Although using a cohort study design instead reduced the bias that drawing samples from

clinics or hospitals can introduce (Holzman et al., 2001), doing so with a set of variables and a sample not specifically selected for a study of birth outcomes resulted in a large number of women being ineligible for inclusion in my study. Ending up with such a small subsample of the original Add Health sample that was not derived by simple random sampling had several negative consequences for my study. Chief among them was that my sample demonstrated what has been referred to in the published literature as "extreme variation" (Lee & Forthofer, 2006) in the sampling weights. Because this variation could have served to increase the variance substantially and perhaps rendered the weighted analysis inefficient when compared with the unweighted analysis, I performed preliminary analyses involving both weight-trimming and model-based analysis to see how the results compared to the weighted and unweighted results in order to determine the appropriateness of using the full sampling weights for my subpopulation.

Weight trimming and model-based analysis are not without their own limitations, however. For example, while weight trimming procedures lower sampling variability and variances of estimates, they can also increase bias (Izrael, Battaglia, & Frankel, 2009). Model-based analyses can reduce the bias introduced by unweighted analysis or weight-trimmed weighted analyses and yield smaller standard errors than the weighted design-based analysis (although I did not always find this to be the case in my sensitivity analysis). However, model-based analysis is very sensitive to model misspecification—a possibility that the use of sample weights in design-based analysis protects against (Lee & Forthofer, 2008). Also, some design information, such as that needed to modify the sample weights for non-response and post-stratification adjustment were not available from Add Health for

inclusion in my model during the sensitivity analysis. Besides that, these and other variables that would need to be included in the model-based analysis were not necessarily consistent with the purpose of my analysis so that conditioning on them could have interfered with hypothesized relationships. Despite the existence of tradeoffs with every single approach tried in the sensitivity analysis, the consistency of results across unweighted and model-based analyses suggest that confidence in my results is not misplaced.

6.4. Implications for Future Research

Many of the limitations discussed in the previous section can be resolved with Add Health Wave IV in-home interview data and Wave III contextual data, which will be released later this year. In light of this, my study may be viewed as a pilot or feasibility study that allowed me to hone in on the most appropriate measurement and analytic strategies to be used in future secondary analyses of the new data when they become available.

In the Wave IV dataset, respondents will be older (~25 – 33 years old), have reported more pregnancies and births, and may have provided biospecimens to allow for the examination of biomarkers of stress. These additional data, along with the Wave III contextual data, will facilitate more fully testing the "weathering" hypothesis because of the availability of multiple observations of the same women over time and the ability to link changing exposures to sequential pregnancy outcomes. They will also allow me to examine the relative importance of, as well as the relationship between, current versus past contexts

(e.g., mothers' current neighborhood or family conditions versus those of her family of origin) in explaining racial disparities in adverse birth outcomes. Cumulative and interaction effects, as well as the lagged effects included in my dissertation, can be studied. This opportunity will provide a more complete understanding of the effect of duration and timing of exposure to disadvantage on birth outcomes. It should also help us better understand which aspects of women's lives or which critical periods of their lifespans we should target with our interventions and policies to halt the accumulation of risk and consequent "weathering" among African American women (Blane, 1999; Graham, 2002). To date, the effect of the timing of risk exposure on birth outcomes has been undertaken only once and then only with respect to proximal risk factors in the prenatal period and the period immediately prior to pregnancy (Haas et al., 2005).

The new data also will allow me to properly examine causality in the mediation pathways, as well as interaction effects of maternal age and race on adverse birth outcomes. As the Institute of Medicine (2006) has recommended, using all available longitudinal data, including the interaction of maternal age and race in the analytical model, and controlling for potential cohort effects would make it possible to examine "weathering" with respect to birth outcomes. The new biospecimen data and, in my research especially, the biomarkers of stress may enable assessments of allostatic load—which, as stated in Chapter 2, could be an important mechanism by which weathering occurs. As Massey (2004) and others suggest, long-term exposure to segregation and stratification arising from racism may be one cause of higher allostatic load among African Americans.

The aforementioned tasks are important for future research whether the data analyzed comes from Add Health or not. In addition, several other questions remain unanswered by my study. For example, the relationships among distal independent variables (i.e., neighborhood and family disadvantage) remain to be further explicated in future research on adverse birth outcomes. Two assumptions of my original conceptual model were that all exogenous variables were correlated, and that none of those variables should be controlled if we were to identify the unique contribution (i.e., attributable risk) to adverse birth outcomes made by the other variables. These are reasonable assumptions that, among other things, avoid concerns about statistical over-control of individual-level factors in order to distinguish the effects of area-level factors from them (Diez-Roux, 2001). These assumptions also help alleviate concerns about the direction of causality created by the possibility of self-selection into neighborhoods (Culhane & Elo, 2005), or questions raised by others about the impact of artificially separating people from their neighborhoods in research (Tunstall, Shaw, & Darling, 2004). Nevertheless, these assumptions led to a poor-fitting model that required me to draw on theory to specify relationships among the disadvantage variables. The way I modeled those relationships is perhaps just one way of doing so. Determining the nature of the association—i.e., whether interactive, bidirectional, or causal—will render research on the social structural determinants of adverse birth outcomes better able to specify relationships and identify more precisely targets for policy and intervention. For example, my study calls attention to neighborhood physical disadvantage as a potential target or point of intervention. It is important, however, that future research of this type do a better job of incorporating more contextual measures

instead of relying as heavily as I did on compositional measures of neighborhood conditions. Doing so will not only assist in identifying which specific dimensions of neighborhoods matter for pregnancy and birth outcomes but also contribute insight to the ongoing debate about the explanatory power of compositional versus contextual effects of place (Macintyre & Ellaway, 2000).

Several relationships among social factors have already been posited and should be tested in future research on adverse birth outcomes. Residential segregation, for example, is presumed to create concentrated poverty, poor housing quality, increased physical hazards, and lack of access to resources and services (Krieger, 2000; Massey & Denton, 1993; Wilson, 1987). Thus, an alternative to my model might have been to specify "proportion of Black residents" as a causal indicator not only of neighborhood structural disadvantage but also neighborhood social and physical disadvantage. However, my model was consistent with research suggesting that neighborhood social factors are the key link between structural disadvantage and health risk behavior among adolescents (Leventhal & Brooks-Gunn, 2000; Sampson, Morenoff, & Gannon-Rowley, 2002; Sampson, Morenoff, & Earls, 1999). On the other hand, it was inconsistent with the suggestion that individual SES may interact with or lie along the pathway between neighborhood SES and health outcomes (Diez-Roux, 2001; Kobetz, Daniel, & Earp, 2003).

Another question remaining to be answered is how best to model social factors in biopsychosocial research on adverse birth outcomes. Modeling the social factors as antecedents to proximal biological, behavioral, or psychosocial risk factors, as I did, is just one way to model them. Other models might view social factors as modifiers of, or tightly

intertwined with, proximal risk factors (Diez-Roux, 2007). For those models, partitioning out the contributions of distal and proximal factors would be futile. Thus, while my study sought to establish the contribution of both distal and proximal factors simultaneously, future research can do a better job of exploring the interconnections between them.

Finally, despite the racial disparity in LBW I found and others have consistently found (see Chapter 2), most women in the study did not experience adverse birth outcomes. An important task for future research, rather than focusing on risk factors and conditions that place Black women at increased risk, may be to identify protective factors that offset or interact with risk factors among women who have them but do not experience adverse birth outcomes (IOM, 2006). Another future focus should include within-group studies to identify those factors that distinguish Black women who experience adverse birth outcomes from those who do not. The stratified analyses conducted in this study and others (e.g., Sastry & Hussey, 2003) lend some credence to the need for such studies.

6.5. Implications for Policy and Practice

Although the findings of this study are tentative at best, they suggest that the problem of adverse birth outcomes is multilevel and multifactorial. Likewise, the solution to the problem must involve addressing many pathways simultaneously (e.g., biological, behavioral, social, structural), via multiple targets (e.g., women and neighborhoods), during multiple time periods (e.g., before and during pregnancy). Taken together with the lifecourse perspective on health, this means that the first and most important change that can be made in adverse birth outcomes prevention strategies is to broaden them beyond an

exclusive focus on the prenatal period to a focus on the overall health of women, regardless of their pregnancy status or childbearing plans (Hughes & Simpson, 1995; Misra, Grason, & Weisman, 2000). As Wise (1993) points out, "we must recognize that, in some large measure, problems with infant ill health are a legacy of women's ill health generally" (p. 14).

For many years, it was thought that improving access to and utilization of prenatal care would improve birth outcomes. Such improvements, as well as improvements in prenatal care itself, have not produced significant reductions in the prevalence and incidence of adverse birth outcomes or the racial disparities in them (Misra, Guyer, & Allston, 2003). In my study, adequacy of prenatal care was not associated with birth weight or gestational age. And, while prenatal substance use was, Haas and her colleagues (2005) observe that the prenatal period is likely too late to modify these and other risk factors for adverse birth outcomes. A recent publication by the Centers for Disease Control and Prevention (CDC) suggested that providing "preconception" care to women may help reduce adverse birth outcomes (Johnson et al., 2006). To the extent that self-rated health during childhood is both a spontaneous and enduring health assessment as Boardman (2006) has suggested, and is a valid and reliable measure of actual health, my study and the lifecourse perspective that guided it supports the CDC recommendations.

This study does not resolve the question of whether health policies and interventions should be aimed towards particular areas or the people in them. This is the case because of my need to rely more heavily than would ideally be desirable on compositional measures of neighborhood context and also because of the presence of statistically significant indirect effects and the absence of direct effects on birth outcomes for

all disadvantage variables except for neighborhood physical disadvantage. The answer to the question, however, is probably "both" rather than "either/or". Thus, in addition to increasing the time frame (i.e., earlier than the period immediately preceding conception), public health practitioners most likely must introduce a wider range of interventions that target both place and people.

Factors such as neighborhood physical disadvantage, which this study found to be one determinant of birth outcomes, are not addressed by health care and not fully addressed in public health practice because the health sector has little or no direct control over them. However, CDC's Task Force on Community Preventive Services recently recommended housing subsidy programs for low-income groups to improve neighborhood safety and reduce family exposure to violence (Anderson et al., 2002). The Moving to Opportunity and Gautreaux Residential Mobility housing voucher programs (Acevedo-Garcia et al., 2004; Leventhal & Brooks-Gunn, 2003) offer examples of the benefits that such "upstream" interventions can provide for health. Such strategies will require collaboration between multiple entities involved in shaping or setting health and social policy (Braveman & Gruskin, 2003; Woolf, 2009). And, as with care provided to women before pregnancy, these types of holistic strategies should be implemented early enough to have a good chance of repairing the damage caused by past disadvantage and potentially moving disadvantaged women into a more advantaged trajectory (Blane, 1999).

In conclusion, it is important to note that the overarching framework in Figure 3 and the conceptual model for my study (Figures 4 and 5) suggest the potential for future studies to make contributions to disparities scholarship beyond those studies that focus solely on

adverse birth outcomes. This potential is derived from the plausibility of a fundamental cause argument implied by the framework (Link & Phelan, 1995). Consistent with fundamental cause theory, the social risk conditions depicted in my model could manifest as other adverse health outcomes that either precede or occur independently of adverse birth outcomes, despite changes in particular risk and protective factors. In other words, the framework implies that distal social factors may be related to multiple health outcomes through either common or clustered pathways (Diez-Roux, 2007). In this way, it may be broadly applicable to the health of women, regardless of their childbearing status or plans.

APPENDIX A
DETAILED MEASURES TABLES

Table A.1. Outcome Variables

	Construct	Items	Old Values	New Values
Birth Outcome (Infant)	Birth Weight	▪ What was the baby's birth weight, in pounds?	1 – 10 pounds	567.0 – 4848.90 grams
		▪ What was the baby's birth weight, in ounces?	0 – 15 ounces	
	Gestational Age	▪ Was the baby born too early – that is, after a pregnancy of less than 40 weeks?	0 = No 1 = Yes	28 – 40 weeks
		▪ *If Yes*, how many weeks early?	1 – 12 weeks	

Table A.2. Explanatory Variables

	Construct	Items	Old Values	New Values
Maternal Childhood Neighborhood Disadvantage	Structural Disadvantage	▪ Proportion of families with dependents with income in 1989 below poverty level, tract	n/a	0.00 – 0.81
		▪ Proportion households with public assistance income, tract	n/a	0.00 – 0.46
		▪ Unemployment rate, total, tract	n/a	0.00 – 0.35
		▪ Proportion aged 25 years and over with no HS diploma or equivalency, total, tract	n/a	0.02 – 0.78
		▪ Proportion households that are female headed, no husband, with dependents, tract	n/a	0.00 – 0.37
		▪ Proportion black, tract	n/a	0.00 – 1.00

Table A.2 continues on next page

Table A.2. Explanatory Variables *(continued)*

	Construct	Items	Old Values	Values
Maternal Childhood Neighborhood Disadvantage	Social Disadvantage	**Social Ties**		
		▪ People in the neighborhood look out for each other	1 = True 2 = False	0 = Yes 1 = No
		Safety:		
		▪ Do you usually feel safe in your neighborhood?	0 = No 1 = Yes	0 = Yes 1 = No
		▪ Total crime rate per 100,000 population in county	n/a	522.2 – 16855.3
	Physical Disadvantage	**Housing and Neighborhood Quality:**		
		▪ How well kept are buildings on street (H1IR14) or, if rural, the building/house in which respondent lives (H1IR11)?	1 = very well kept 4 = very poorly kept	1 = very well kept 3 = poorly kept
		▪ Proportion vacant housing units, tract (block)	n/a	0 – 0.68
		▪ Did you feel concerned for your safety?	0 = No 1 = Yes	0 = No 1 = Yes
Maternal Childhood Family Disadvantage	Family Structure	▪ What is [NAME]'s relationship to you? ▪ Which description best fits [NAME]'s relationship to you?	1 = 2 Biological 2 = 2 Parents 3 = Single Mom 4 = Single Dad 5 = Other	0 = 2 parent family 1 = single/surrogate family

Table A.2 continues on next page

Table A.2. Explanatory Variables *(continued)*

	Construct	Items	Old Values	New Values
Maternal Childhood Family Disadvantage *(continued)*	Socioeconomic Status	**<u>Parent Educational Attainment:</u>** ▪ How far did (resident mom/dad) go in school? *(adolescent)* ▪ How far did you go in school? *(parent report)*	1 = ≤ 8th grade 2 = >8th but <HS 3 = biz, trade, or voc'l school, not HS 4 = HS graduate 5 = completed a GED 6 = biz, trade, or voc'l school after HS 7 = college, no grad 8 = college grad 9 = >college 10 = never schooled 11 = DK level 12 = DK if went	1 = college or more 2 = some college 3 = high school 4 = no high school
		<u>Family Poverty Status:</u> ▪ About how much total income, before taxes, did your family receive in 1994 *(parent)* ▪ Are you receiving public assistance, e.g. welfare? *(parent)* ▪ Last month, did you or any member of household receive AFDC? Food stamps? Housing subsidy or public housing? *(parent)* ▪ Does [resident mother or father] receive public assistance, such as welfare? *(adolescent)*	0 – 999 thousand 0 = No, 1 = Yes 0 = No, 1 = Yes 0 = No, 1 = Yes	0 = Not in Poverty 1 = In Poverty

Table A.2 continues on next page

Table A.2. Explanatory Variables *(continued)*

	Construct	Items	Old Values	New Values
Maternal Childhood Health and Development	Behavioral Risk Status	▪ During the past 30 days, on how many days did you smoke cigarettes? ▪ During the past 12 months, on how many days did you drink alcohol? ▪ During the past 30 days, how many times have you used marijuana? ….cocaine? ….any of these types of illegal drugs?	n/a	0 (no risk) – 3 (high risk)
	Psychosocial Risk Status	▪ How often was each of the following things true during past wk? (blues, depressed, happy, sad, life)	0 = never or rarely 1 = sometimes 2 = a lot of the time 3 = most or all time	Same
	Physical Health Status	▪ In general, how is your health?	1 = Excellent 2 = Very Good 3 = Good 4 = Fair 5 = Poor	1 = Excellent 2 = Very Good 3 = Good 4 = Fair or Poor

Table A.3. Planned Control Variables

	Construct	Items	Old Values	New Values
Exposure	″Dose″	▪ How old were you when you moved here to current residence?	0 = Since <1 1 – 19 years old	0 – 19
Maternal Sociodemo-graphics	Maternal Age	▪ Please indicate the month and year the pregnancy ended ▪ What is your birth date?	n/a	14.5 – 25.8

Table A.3 continues on next page

Table A.3. Planned Control Variables *(continued)*

	Construct	Items	Old Values	New Values
Maternal Sociodemo-graphics (continued)	Maternal Education	▪ What is the highest grade or year of regular school you completed?	6 = 6th grade 12 = 12th grade 13 = 1 year college 16 = 4 years college 17 = 5+ years college 18 = 1 yr grad school 22 = 5+ yrs grad schl	1 = More than HS 2 = HS or <20 and no HS 3 = ≥ 20 and no HS
		▪ Are you presently in school/were you in school last year? ▪ What grade are/were you in? ▪ Why aren't/weren't you in school?	0 = No, 1 = Yes 7 – 12	
	Paternal Support during Pregnancy	**<u>Prenatal Contact/Support:</u>** ▪ Did [the father] go along with you for any of these [prenatal care] checkups??	0 = No 1 = Yes	0 = Yes 1 = No
		<u>Relationship Status During Pregnancy:</u> ▪ Were you married to each other at the time of the birth? Living together at the time of the birth?	0 = No 1 = Yes	1 = Married 2 = Cohabitating 3 = Steady Relationship 4 = None of the above
		▪ If no, which of the following statements best describes relationship at time of birth?	1 = No Contact 5 = Steady, Romantic	
Prenatal Health	Prenatal Health Risk	▪ Have you ever been told by a doctor or nurse that you have bacterial vaginosis? ▪ Have you ever been diagnosed with diabetes? ▪ How old were you when you were told you had diabetes?	0 = No, 1 = Yes 0 = No 1 = Yes	0 = no risk – 2 = high risk
		▪ Self-reported height (feet and inches) and weight		

Table A.3 continues on next page

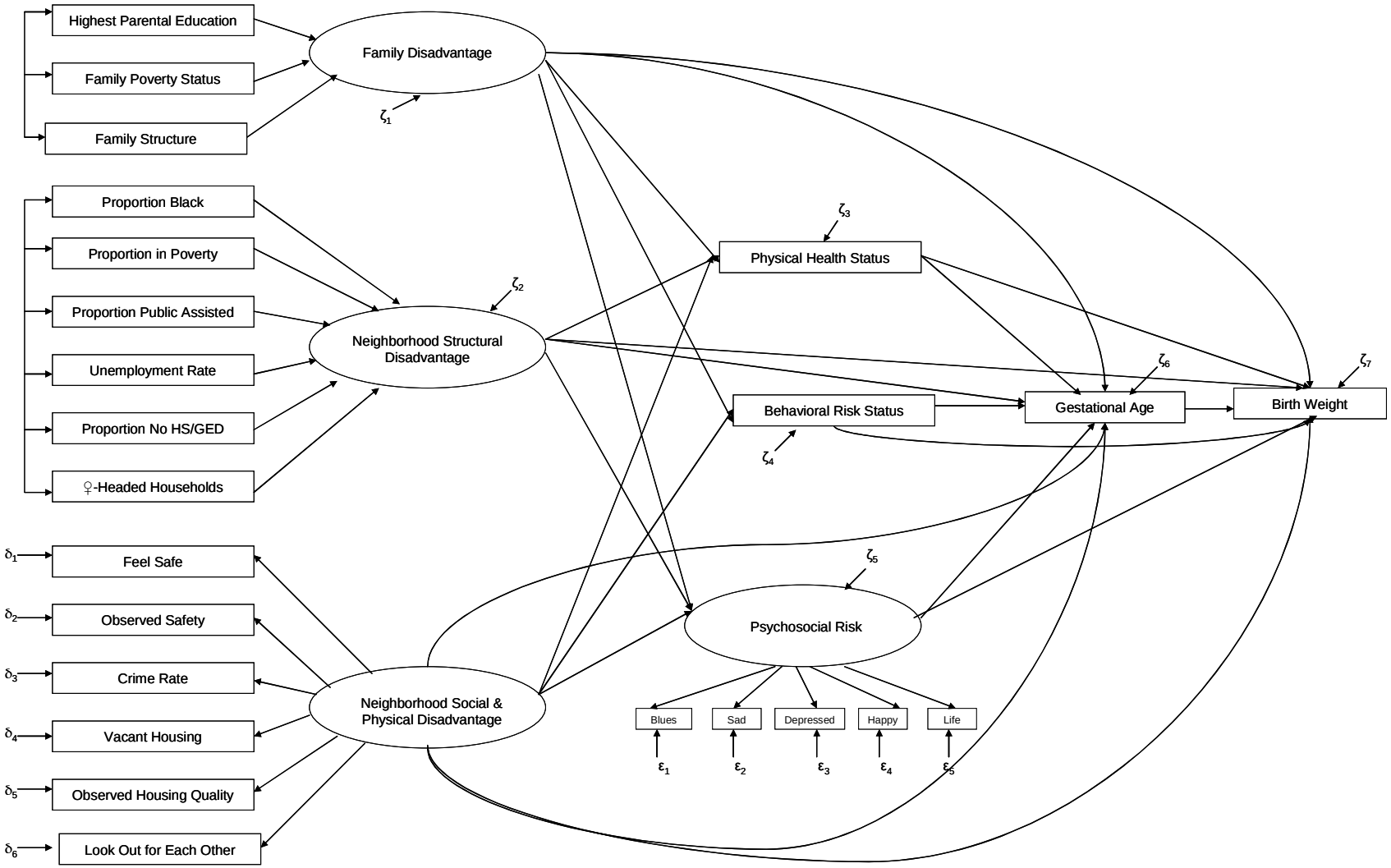
Table A.3. Planned Control Variables *(continued)*

	Construct	Items	Old Values	Values
Prenatal Behavior	Prenatal Behavioral Risk	**<u>Prenatal Substance Use:</u>** ▪ During this pregnancy, how often did you drink alcoholic beverages? ▪ How often did you use drugs such as marijuana, crack cocaine, or heroin? ▪ How many cigarettes did you smoke?	0 = Never 1 = < Once/Mo. 2 = Several X/Mo. 3 = Several X/Wk. 4 = Almost Every Day 0 = None, 3 = ≥2 pks/day	0 = no risk – 3 = high risk
		<u>Prenatal Care:</u> ▪ While you were pregnant, did you visit a doctor or nurse-midwife for prenatal care or pregnancy check-ups? ▪ In which month, did you first visit a doctor or nurse-midwife for prenatal care or pregnancy check-ups?	0 = No 1 = Yes 1 – 90	1 = Very adequate 2 = Somewhat adequate 3 = Inadequate
Pregnancy History	Parity and Birth Timing	▪ Date WI interview completed ▪ Please indicate the month and year the pregnancy ended ▪ Please indicate the outcome of this pregnancy ▪ Please indicate how many babies were born alive for each pregnancy	n/a n/a 4 = Live Birth 7 = Multiple, Live + Another Outcome 1 – 6	0 = Nulliparous 1 = Uniparous 2 = Multiparous

APPENDIX B

ORIGINAL SEM SPECIFICATIONS

Figure B.1. Original Structural Equation Model



APPENDIX C

SENSITIVITY ANALYSIS RESULTS

Comparison of Distributions of Final Weight and Trimmed Weights

Figure C.1. Original Weight (GSWGT3_2)

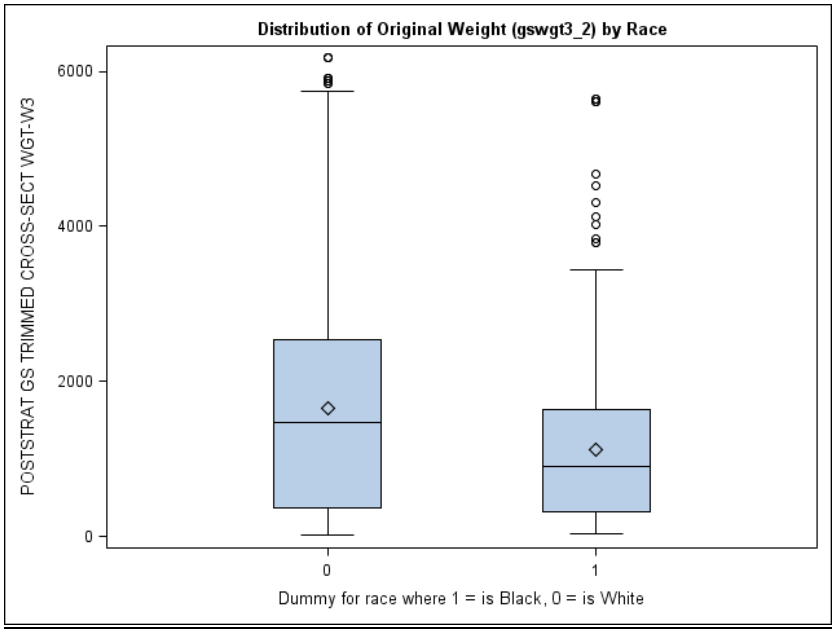


Figure C.2. Trimmed Extreme Weights to 95th Percentile of Original Weight within Race

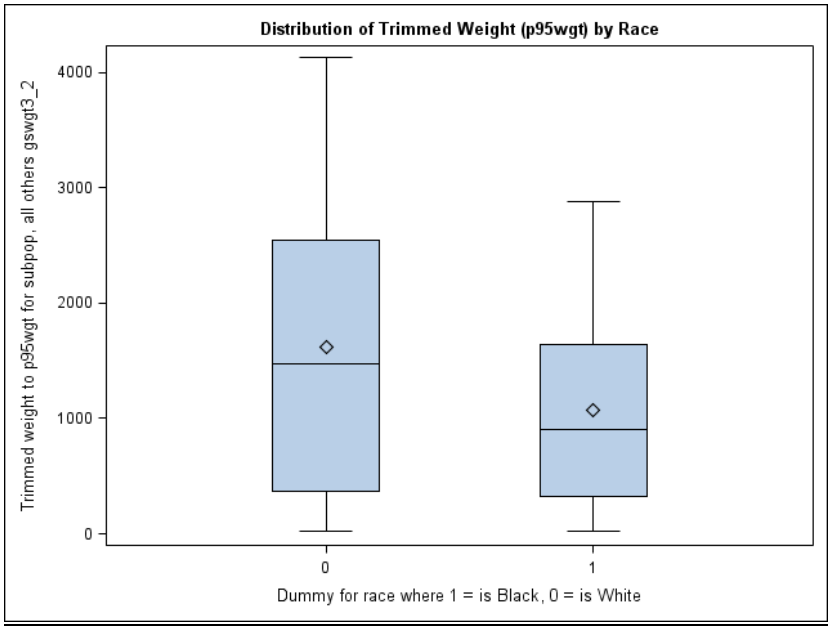
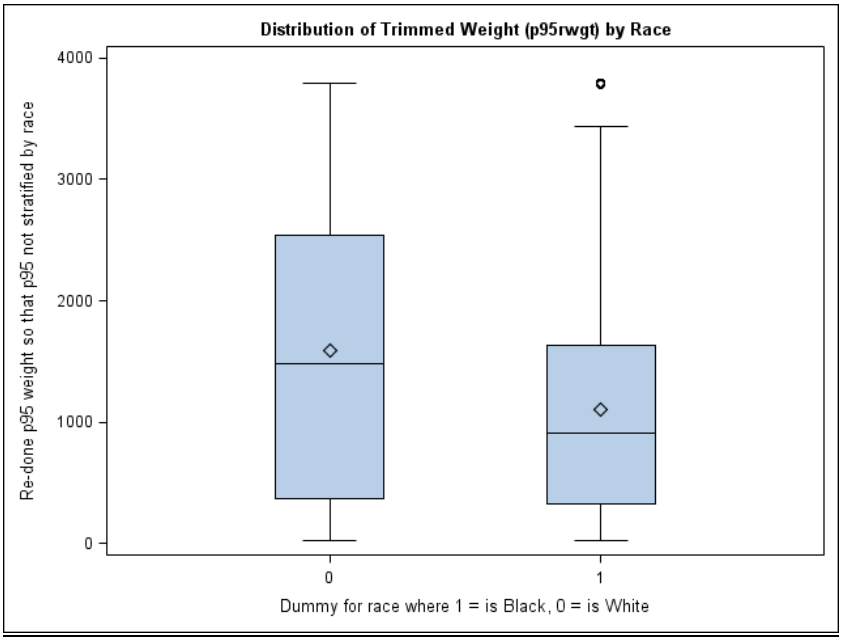


Figure C.3. Trimmed Extreme Weights to 95[h] Percentile of Original Weight



Comparison between Weighted, Weight-Trimmed, Model-Based, and Unweighted Statistics on Birth Weight and Gestational Age, Percent LBW and PTB, Total and By Race

Table C.1. Summary Birth Weight and Gestational Age Statistics, by Race and Analysis Strategy

Variables	Range	Total (n = 1,557)	Black (n = 500)	White (n = 1,077)	p value[c]
WEIGHTED WITH ORIGINAL WEIGHT					
Birth Weight (grams) Mean (SE)[a]	567.0 – 4848.9	3296.3 (16.57)	3199.0 (34.21)	3327.6 (20.11)	0.0021
LBW (<2500 grams) Percent[b]	n/a	5.8	7.8	5.1	0.1081
Gestational Age (wks) Mean (SE)[a]	28 – 40	39.3 (.05)	39.3 (.09)	39.3 (.06)	0.8739
PTB (<37 weeks) Percent[b]	n/a	6.5	6.0	6.7	0.6473
WEIGHTED WITH WEIGHTS TRIMMED TO 95TH PERCENTILE					
Birth Weight (grams) Mean (SE)[a]	567.0 – 4848.9	3293.1	3195.5 (32.34)	3325.0 (19.32)	0.0011
LBW (<2500 grams) Percent[b]	n/a	6.0	8.0	5.4	0.1169
Gestational Age (wks) Mean (SE)[a]	28 – 40	39.3 (.05)	39.3 (.10)	39.3 (.06)	0.8366
PTB (<37 weeks) Percent[b]	n/a	6.6	6.2	6.7	0.6798

Table C.1 continues on next page

Table C.1. Summary Birth Weight and Gestational Age Statistics, by Race and Analysis Strategy *(continued)*

Variables	Range	Total (n = 1,557)	Black (n = 500)	White (n = 1,077)	p value[c]
MODEL-BASED*					
Birth Weight (grams) Mean (SE)[a]	567.0 – 4848.9	n/a	n/a	n/a	<0.0001
LBW (<2500 grams) Percent[b]	n/a	n/a	n/a	n/a	0.1461*
Gestational Age (wks) Mean (SE)[a]	28 – 40	n/a	n/a	n/a	0.7812
PTB (<37 weeks) Percent[b]	n/a	n/a	n/a	n/a	0.6121
UNWEIGHTED					
Birth Weight (grams) Mean (SE)[a]	567.0 – 4848.9	3289.7 (12.71)	3175.7 (32.75)	3343.7 (16.29)	<0.0001
LBW (<2500 grams) Percent[b]	n/a	6.2	8.6	5.1	0.0115
Gestational Age (wks) Mean (SE)[a]	28 – 40	39.3 (.04)	39.3 (.07)	39.3 (.06)	0.8739
PTB (<37 weeks) Percent[b]	n/a	6.9	7.4	6.7	0.5770

Abbreviations: *SE*, standard error; %, percent

[a] mean (SE) reported for continuous variables

[b] percent (unweighted) reported for categorical variables

[c] p value for race determined from Rao-Scott χ^2 test for categorical variables and t-test for continuous variables

[d] % (unweighted) reported for categorical variables

[e] estimates derived from logistic and linear regression with more than one independent variable are not interpretable as percentages and means

[f] p value for race determined from logistic regression for categorical variables and linear regression for continuous variables

* In the model-based analysis, race is likely confounded by the "black with a college-educated parent" sample flag that was included in the model (which, it should be noted was significantly associated with both birth weight and LBW). When this sample flag is removed from the model, the racial difference in LBW has a p-value of 0.206.

Comparison between Weighted, Weight-Trimmed, Model-Based, and Unweighted Fit Statistics for Selected Measurement Models

Table C.2. Overall Model Fit Statistics for Depression

Modeling Approach	χ^2	df	*p*	χ^2/df	CFI	TLI	RMSEA	WRMR
Weighted	12.782*	4	.012	3.20	0.997	0.997	0.012	0.509
Weight-Trimmed to 95th Percentile	12.023	4	.017	3.01	0.998	0.997	0.012	0.501
Model-Based	17.169	14	.247	1.23	0.999	0.999	0.004	0.636
Unweighted	8.393	4	.078	2.10	0.999	0.999	0.009	0.433

Abbreviations: *df* = degrees of freedom *CFI* = comparative fit index; *TLI* = Tucker-Lewis index; *RMSEA* = root mean squared error of approximation; *WRMR* = weighted root mean squared residual

Table C.3. Component Fit Statistics for Depression

Observed Indicators	Factor Loading Estimates**		R^2
	Unstandardized	Standardized	
WEIGHTED COMPONENT FIT FOR STATISTICS			
Blues*	1.000	0.861	0.741
Depressed	1.020	0.878	0.771
Happy	0.669	0.576	0.332
Sad	0.992	0.854	0.730
Life	0.730	0.628	0.394
WEIGHT-TRIMMED COMPONENT FIT STATISTICS			
Blues*	1.000	0.857	0.735
Depressed	1.024	0.878	0.770
Happy	0.678	0.581	0.338
Sad	0.996	0.854	0.729
Life	0.741	0.635	0.404
MODEL-BASED COMPONENT FIT STATISTICS			
Blues*	1.000	0.833	0.694
Depressed	1.075	0.895	0.802
Happy	0.694	0.580	0.336
Sad	1.024	0.853	0.727
Life	0.783	0.654	0.428
UNWEIGHTED COMPONENT FIT STATISTICS			
Blues*	1.000	0.831	0.690
Depressed	1.074	0.893	0.797
Happy	0.696	0.578	0.334
Sad	1.025	0.852	0.725
Life	0.780	0.648	0.419

* Parameter constrained to 1.00 to scale the construct.

** All of the factor loading estimates were statistically significant at $p < .05$; however, constrained parameters were not tested.

Table C.4. Path Estimates for Birth Weight Regressed on Depression

Modeling Approach	β	SE	*p*
Weighted	-10.57	6.68	0.114
Weight-Trimmed to 95th Percentile	-10.63	6.62	0.108
Model-Based	-5.36	5.96	0.368
Unweighted	-4.09	6.19	0.509

Abbreviations: *SE*, standard error

APPENDIX D

DESCRIPTIVE STATISTICS FOR KEY STUDY VARIABLES, BY RACE

Table D.1. Selected Characteristics of the Sample: Childhood Disadvantage by Race

Variables	Range/ Values	Total (*n* = 1,577) % or Mean (SE)[a]	Maternal Race: Black (*n* = 500) % or Mean (SE)	Maternal Race: White (*n* = 1,077) % or Mean (SE)	*p* value[b]
Neighborhood SES					
Proportion below poverty	0.00 – 0.81	0.20 (.016)	0.29 (.024)	0.16 (.014)	<0.0001
Proportion with public assistance	0.00 – 0.46	0.11 (.007)	0.16 (.010)	0.09 (.007)	<0.0001
Unemployment rate	0.00 – 0.35	0.09 (.004)	0.11 (.005)	0.08 (.004)	<0.0001
Proportion ≥25 with no HS/GED	0.02 – 0.78	0.32 (.011)	0.35 (.017)	0.30 (.015)	0.0228
Proportion ♀-headed households	0.00 – 0.37	0.09 (.004)	0.13 (.007)	0.06 (.003)	<0.0001
Residential Segregation					
Proportion Black	0.00 – 1.00	0.22 (.029)	0.54 (.035)	0.07 (.009)	<0.0001
Neighborhood Social Relations					
Neighbors look out for each other	0 – 1				0.0297
Yes		67.6	64.2	69.3	
No		30.0	34.6	27.8	
Total crime rate per 100,000	522 – 16855	5820 (376.2)	7280 (383.1)	5105 (407.5)	<0.0001
Perceived safety	0 – 1				<0.0001
Yes		85.5	78.2	89.0	
No		13.8	21.6	10.1	
Perceived safety (interviewer)	0 – 1				0.0027
Yes		92.7	88.8	94.5	
No		6.2	10.0	4.4	
Ambient Risks					
Proportion of vacant housing units	0.00 – 0.68	0.09 (.007)	0.10 (.008)	0.09 (.007)	0.2582
Housing quality (interviewer)	0 – 2				0.0024
Very Well Kept		39.6	35.0	41.8	
Somewhat Well Kept		39.3	39.2	39.4	
Very or Somewhat poorly kept		20.1	25.2	17.7	

Abbreviations: *SE*, standard error

[a] % (weighted) reported for categorical variables; mean (SE) reported for continuous variables; percents for each variable do not add up to 100% due to missing values

[b] *p* value for race determined from Rao-Scott χ^2 test for categorical variables and *t*-test for continuous variables

Table D.1 continues on next page

Table D.1. Selected Characteristics of the Sample: Disadvantage Indicators by Race *(continued)*

Variables	Range/ Values	Total (n = 1,577) % or Mean (SE)[a]	Maternal Race: Black (n = 500) % or Mean (SE)	Maternal Race: White (n = 1,077) % or Mean (SE)	p value[b]
Family Socioeconomic Status					
Family Income < Poverty (parent)	0 – 1				<0.0001
No		57.9	47.2	63.0	
Yes		15.1	21.0	12.3	
Public Assistance (parent)	0 – 1				<0.0001
No		63.8	50.6	70.00	
Yes		18.9	28.8	14.3	
Public Assistance	0 – 1				<0.0001
No		80.3	74.8	82.9	
Yes		15.2	21.4	12.2	
Highest Parental Education	1 – 4				0.0167
College grad or more		18.2	24.2	15.4	
More than high school		21.3	19.6	22.1	
High school		34.4	31.8	35.7	
Less than high school		18.0	15.4	19.2	
Highest Parental Education (parent)	1 – 4				0.0304
College grad or more		9.2	13.0	7.5	
More than high school		23.8	24.4	23.5	
High school		29.8	25.0	32.1	
Less than high school		20.5	18.0	21.6	
Family Structure					
Parenting Arrangements	0 - 1				<0.0001
Two parent		59.5	42.6	67.5	
Single/surrogate parent		40.5	57.4	32.5	

Abbreviations: *SE*, standard error; %, percent

[a] % (weighted) reported for categorical variables; mean (SE) reported for continuous variables; percents for each variable do not add up to 100% due to missing values

[b] p value for race determined from Rao-Scott χ^2 test for categorical variables and t-test for continuous variables

Table D.2. Selected Characteristics of the Sample: Mediators by Race

Variables	Range/ Values	Total (n = 1,577) % or Mean (SE)[a]	Maternal Race: Black (n = 500) % or Mean (SE)	Maternal Race: White (n = 1,077) % or Mean (SE)	p value[b]
Behavioral Risk Status					
Behavioral Risk Score	0 – 3	0.8 (.05)	0.5 (0.05)	1.1 (.04)	<0.0001
Smoked (Yes)		*34.8*	*13.0*	*45.1*	*<0.0001*
Drank (Yes)		*34.9*	*22.2*	*40.9*	*<0.0001*
Used Drugs (Yes)		*18.3*	*11.0*	*21.8*	*0.0040*
Psychosocial Risk Status					
Depression Score	0 – 15	3.52 (.09)	3.65 (.15)	3.46 (.13)	0.3572
Blues (mean)		0.64 (.023)	0.67 (.037)	0.62 (.031)	0.3549
Depressed (mean)		0.80 (.028)	0.79 (.040)	0.81 (.037)	0.7180
Happy (mean)		1.02 (.025)	1.06 (.040)	1.00 (.032)	0.2550
Sad (mean)		0.81 (.027)	0.83 (.042)	0.80 (.031)	0.5498
Life (mean)		0.25 (.014)	0.30 (.031)	0.23 (.018)	0.0638
Physical Health Status					
Self-Rated Health	1 – 4				0.0026
Excellent		19.0	25.6	15.9	
Very Good		36.0	36.4	35.8	
Good		33.7	28.2	36.2	
Fair or Poor		11.4	9.8	12.1	

Abbreviations: *SE,* standard error; *%,* percent

[a] % (weighted) reported for categorical variables; mean (SE) reported for continuous variables

[b] p value for race determined from Rao-Scott χ^2 test for categorical variables and t-test for continuous variables

APPENDIX E

UNADJUSTED BIVARIATE ASSOCIATIONS BETWEEN BIRTH OUTCOMES AND KEY STUDY VARIABLES

Table E.1. Bivariate Associations between Indicators of Childhood Family Disadvantage and Subsequent Birth Outcomes (*N* = 1,557)

	Gestational Age			Birth Weight		
Variables	***B*** [a]	**(95% CI)**	***p***	***B*** [a]	**(95% CI)**	***p***
Family In Poverty						
Yes	0.022	(-0.23, 0.27)	0.859	-63.53	(-132.59, 5.53)	0.071
No [b]	-----	-----	-----	-----	-----	-----
Highest Parental Education						
College grad or more	-0.040	(-0.29, 0.21)	0.755	-10.25	(-77.02, 56.51)	0.763
More than high school	0.147	(-0.06, 0.36)	0.170	78.33	(6.11, 150.54)	0.034
High school [b]	-----	-----	-----	-----	-----	-----
Less than high school	0.072	(-0.19, 0.33)	0.587	72.51	(-16.30, 161.32)	0.110
Family Structure						
Two parent [b]	-----	-----	-----	-----	-----	-----
Single/surrogate parent	0.006	(-0.19, 0.20)	0.953	-60.59	(-118.60, -2.57)	0.041

[a] Regression coefficients are unstandardized and based on unweighted data

[b] Reference category

Table E.2. Bivariate Associations between Indicators of Childhood Neighborhood Disadvantage and Subsequent Birth Outcomes (*N* = 1,557)

	Gestational Age			Birth Weight		
Variables	***B*** [a]	**(95% CI)**	***p***	***B*** [a]	**(95% CI)**	***p***
Proportion of families < poverty	-0.166	(-0.598, 0.366)	0.541	-262.36	(-450.15, -74.54)	0.006
Proportion with public assistance	0.619	(-0.401, 1.640)	0.235	-397.33	(-795.06, 0.19)	0.050
Unemployment rate	-0.036	(-1.662, 1.589)	0.965	-734.47	(-1389.98, -79.39)	0.028
Proportion ≥25 with no HS or GED	-0.136	(-0.71, 0.44)	0.641	-188.26	(-423.87, 47.33)	0.117
Proportion ♀-headed households	0.342	(-1.294, 1.978)	0.682	-705.95	(-1321.82, -8.99)	0.025
Proportion Black	-0.123	(-0.41, 0.16)	0.393	-192.92	(-297.97, -87.84)	<0.001
Neighbors look out for each other						
Yes [b]	----	----	----	----	----	----
No	0.029	(-0.17, 0.23)	0.774	43.02	(-12.79, 98.81)	0.131
Total crime rate per 100,000	0.001	(-0.002, 0.004)	0.537	0.00	(-0.008, 0.007)	0.899
Perceived safety						
Yes [b]	----	----	----	----	----	----
No	0.069	(-0.149, 0.287)	0.537	14.39	(-57.41, 86.19)	0.694

Variables	Gestational Age			Birth Weight		
	B [a]	(95% CI)	*p*	*B* [a]	(95% CI)	*p*
Perceived safety *(interviewer)*						
Yes [b]	----	----	----	----	----	----
No	0.160	(-0.140, 0.460)	0.296	-55.41	(-171.70, 60.89)	0.350
Proportion of vacant housing units	-1.73	(-2.76, -0.71)	0.001	-453.40	(-779.00, -127.79)	0.006
Housing quality *(interviewer)*						
Very Well Kept [b]	----	----		----	----	----
Somewhat Well Kept	0.03	(-0.15, 0.21)	----	-19.29	(-82.61, 44.04)	0.551
Very or Somewhat poorly kept	0.09	(-0.24, 0.33)	0.751	-76.64	(-153.10, -0.18)	0.049
			0.490			

[a] Regression coefficients are unstandardized and based on unweighted data

[b] Reference category

Table E.3. Bivariate Associations between Childhood Psychosocial, Behavioral, and Physical Health Risks and Subsequent Birth Outcomes (*N* =1,557)

Variables	Gestational Age			Birth Weight		
	B [a]	(95% CI)	*p*	*B* [a]	(95% CI)	*p*
Self-Rated Health						
Excellent [b]	-----	-----	-----	-----	-----	-----
Very Good	-0.08	(-0.32, 0.17)	0.536	62.54	(-4.81, 129.88)	0.069
Good	0.09	(-0.17, 0.35)	0.500	43.57	(-34.54, 121.67)	0.274
Fair or Poor	-0.33	(-0.70, 0.05)	0.085	-11.06	(-102.95, 80.84)	0.814
Behavioral Risk Score	0.12	(0.055, 0.187)	<0.0001	30.53	(5.61, 55.45)	0.016
Unable to Shake Blues						
Never or rarely [b]	-----	-----	-----	-----	-----	-----
Sometimes	0.043	(-0.16, 0.25)	0.688	10.17	(-58.57, 78.92)	0.772
A lot	0.028	(-0.26, 0.32)	0.846	-26.63	(-97.33, 44.07)	0.460
Most or all time	-0.147	(-0.70, 0.41)	0.605	-62.44	(-225.18, 100.30)	0.452
Felt Depressed						
Never or rarely [b]	-----	-----	-----	-----	-----	-----
Sometimes	0.034	(-0.13, 0.20)	0.690	1.93	(-0.05, 0.05)	0.945
A lot	-0.138	(-0.46, 0.18)	0.395	-13.85	(-0.07, 0.05)	0.767
Most or all time	0.022	(-0.47, 0.51)	0.930	-87.69	(-0.09, 0.02)	0.180
Felt Happy						
Most or all time [b]	-----	-----	-----	-----	-----	-----
A lot	-0.06	(-0.26, 0.15)	0.579	-7.432	(-64.21, 49.35)	0.798
Sometimes	0.11	(-0.08, 0.29)	0.269	36.44	(-24.14, 97.03)	0.238
Never or rarely	-0.25	(-0.89, 0.40)	0.457	-45.33	(-241.17, 150.51)	0.650
Felt Sad						
Never or rarely [b]	-----	-----	-----	-----	-----	-----
Sometimes	-0.035	(-0.19, 0.12)	0.652	15.74	(-35.37, 66.85)	0.546
A lot	0.200	(-0.07, 0.47)	0.147	-2.68	(-90.63, 85.28)	0.952
Most or all time	-0.388	(-1.17, 0.39)	0.328	-105.45	(-272.59, 61.69)	0.216
Life Not Worth						
Never or rarely [b]	-----	-----	-----	-----	-----	-----
Sometimes	-0.03	(-0.18, 0.14)	0.822	5.42	(-74.37, 85.20)	0.894
A lot	0.09	(-0.23, 0.33)	0.718	-175.83	(-340.68, -10.97)	0.037
Most or all time	0.32	(-0.04, 0.40)	0.103	-9.82	(-191.50, 171.87)	0.916

[a] Regression coefficients are unstandardized and based on unweighted data

[b] Reference category

APPENDIX F

STATISTICALLY SIGNIFICANT INDIRECT PATHWAYS
BETWEEN KEY STUDY VARIABLES AND BIRTH WEIGHT

AIM 2	AIM 3
• FAM → STRUC → SOC → PHYS → GEST → BWT • FAM → SOC → PHYS → GEST → BWT • FAM → STRUC → SOC → GEST → BWT • FAM → SOC → GEST → BWT • STRUC → SOC → PHYS → GEST → BWT • STRUC → SOC → GEST → BWT • SOC → PHYS → GEST → BWT • SOC → GEST → BWT • PHYS → GEST → BWT	• FAM → STRUC → BEHAVE → GEST → BWT • STRUC → BEHAVE → GEST → BWT • SOC → GEST → BWT • PHYS → GEST → BWT

Abbreviations: *FAM* = family disadvantage, *STRUC* = neighborhood structural disadvantage, *SOC* = neighborhood social disadvantage, *PHYS* = neighborhood physical disadvantage, *HEALTH* = self-rated health, *SUBUSE* = substance use, *DEPRESS* = depression, *GEST* = gestational age, and BWT = birth weight

APPENDIX G

COMPARISON OF STATISTICALLY SIGNIFICANT PATHS DERIVED FROM ESTIMATION OF THE FINAL ADJUSTED MODEL FOR THE FULL SAMPLE, WHITE SUBSAMPLE, AND BLACK SUBSAMPLE

Figure G.1. Statistically Significant Paths in Full Sample Model

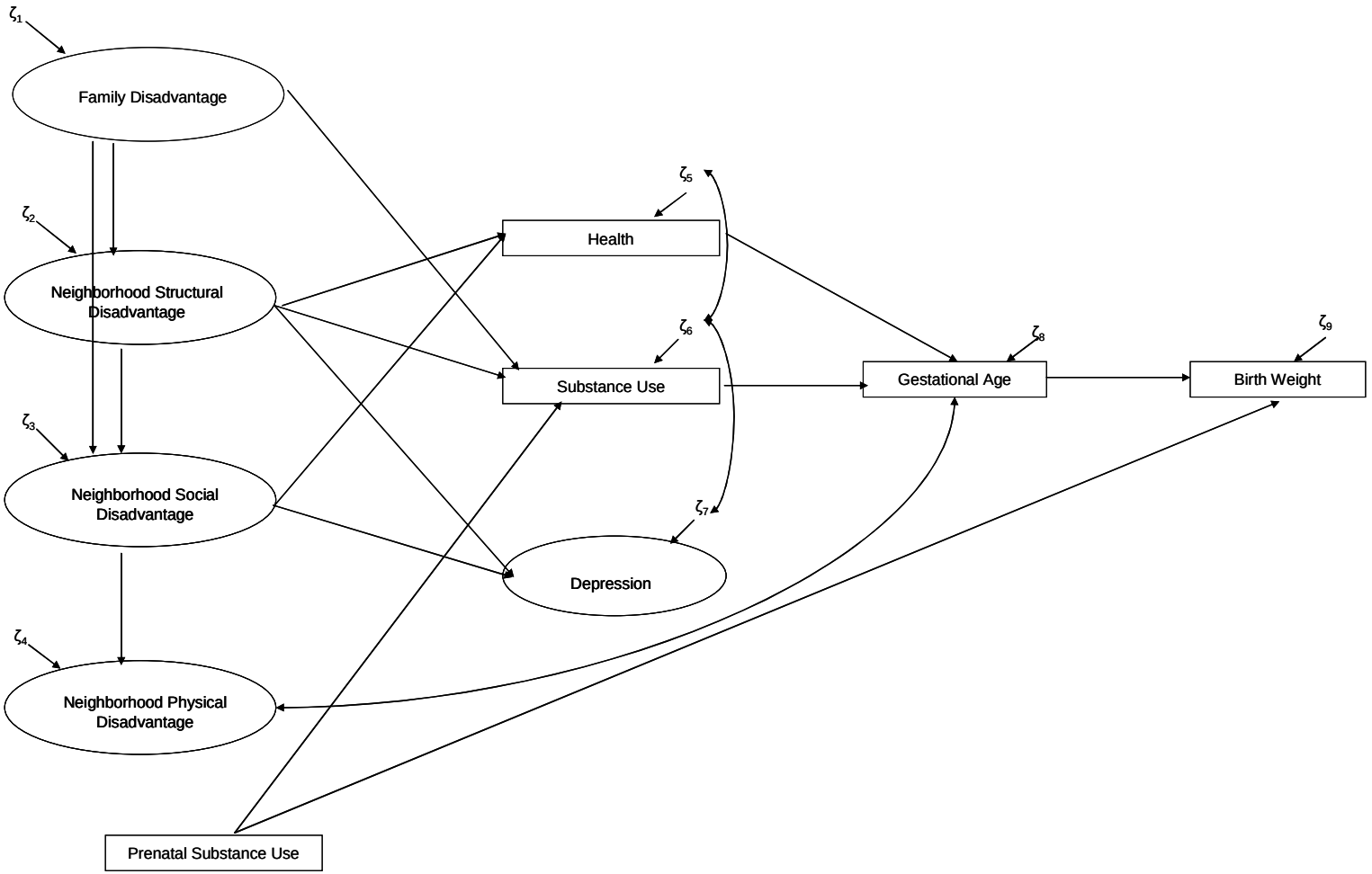


Figure G.2. Statistically Significant Paths in White Subsample Model

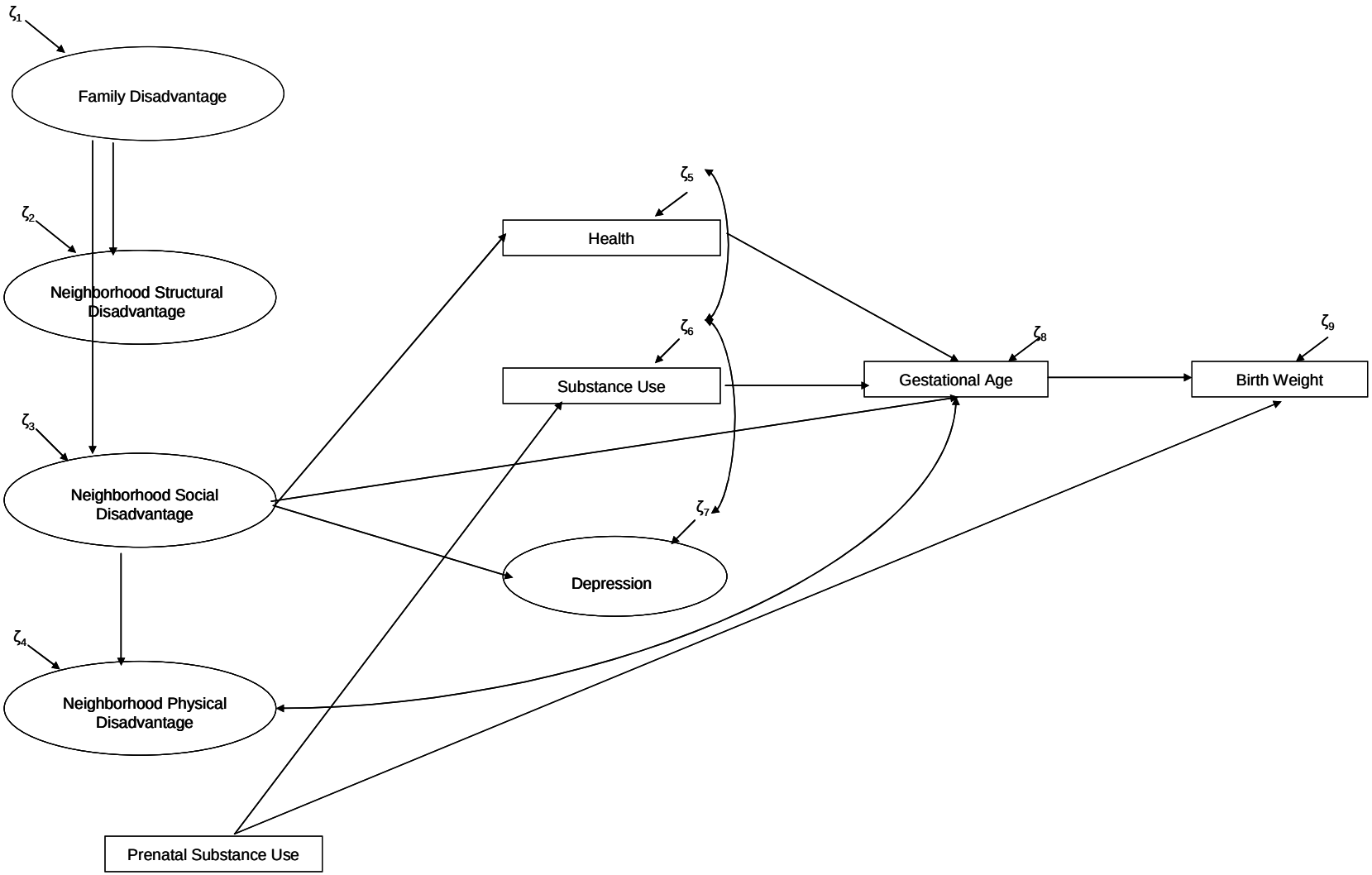
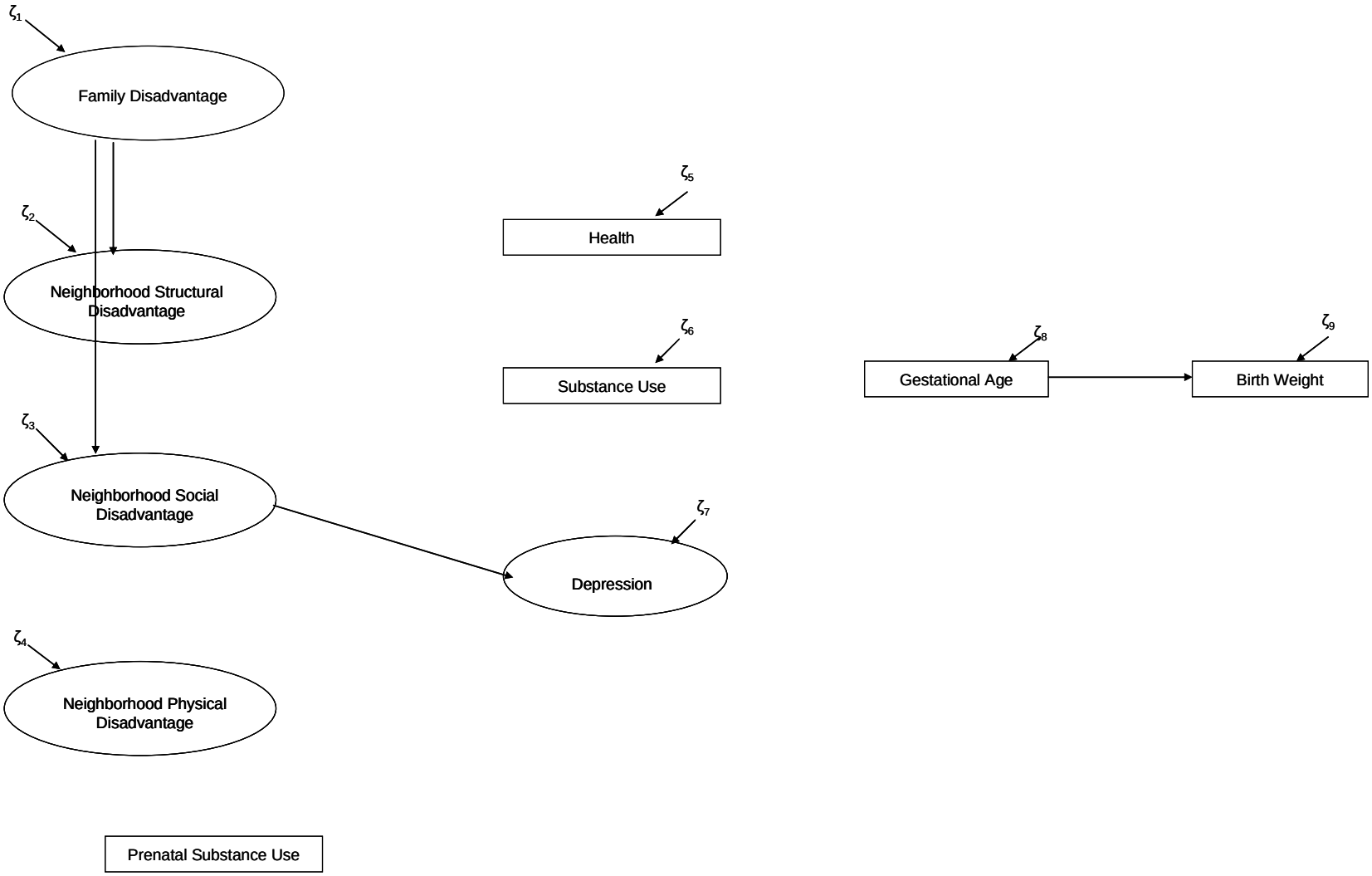


Figure G.3. Statistically Significant Paths in Black Subsample Model



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