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LOW BIRTHWEIGHT AND THE CONTRIBUTION OF RESIDENTIAL SEGREGATION: NEW YORK CITY, 2000

by

SUE C. GRADY

A dissertation submitted to the Graduate Faculty in Earth and Environmental Sciences in partial fulfillment of the requirements for the degree of Doctor of Philosophy, The City University of New York

2005

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Abstract

LOW BIRTHWEIGHT AND THE CONTRIBUTION OF RESIDENTIAL SEGREGATION: NEW YORK CITY, 2000

by

Sue C. Grady

Advisor: Professor Sara McLafferty

Low birthweight, the percentage of babies weighting less than 2,500 grams at birth, is a major public health problem in the United States, contributing substantially to infant mortality and childhood morbidity. Over the past several decades the rate of low birthweight for African American women has exceeded that of other racial and ethnic groups in the United States. Today, these racial differentials are still largely unexplained. The purpose of this research was to investigate the geography of racial disparities in low birthweight in New York City. This cross-sectional multi-level study focused on racial residential segregation and its contribution to adverse medical conditions in African American mothers and the risk of low birthweight among African American infants. It was hypothesized that exposure to residential segregation would deteriorate the health status of African American women, leading to medical conditions that reduce the quality of the intrauterine environment, increasing fetal vulnerability to low birthweight. The

results indicated that African American women living in segregated neighborhoods were at substantially greater risk of low birthweight than similar women not living in segregated neighborhoods. U.S.-born African American women were at greater risk of low birthweight than foreign-born African American women, also living in segregated areas, suggesting that longer duration of exposure is detrimental to birth outcomes. African American women also had a greater number and more complex array of medical conditions earlier in their reproductive years than white women, evidence of early health deterioration. Medical conditions, specifically chronic hypertension, pregnancy-related hypertension, and preeclampsia were identified as mediators in the residential segregation and low birthweight relationship. In New York City, residential segregation is an important determinant of racial disparities in low birthweight.

This dissertation is dedicated to my mother

Jean Carole Grady

1932 to 2004

&

Beloved Lucas

1988 to 2005

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I would also like to thank my committee members, including David Rindskopf for his guidance on multilevel modeling, Ines Miyares for teaching me the historical context of African American women, and Sean Ahearn for his insight into New York City. Their comments and questions at my defense were also productive and validating.

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CHAPTER 1:

RACIAL DISPARITIES IN LOW BIRTHWEIGHT

1. INTRODUCTION

Low birthweight, the percentage of babies weighting less than 2,500 grams at birth, is a major public health problem in the United States, contributing substantially to infant mortality and childhood morbidity. Low birthweight reflects not only the infant's health, but also the mother's health, and is a useful indicator of women's reproductive health and how it varies among neighborhoods and communities (McLafferty and Tempalski, 1995). In New York City, low birthweight is unevenly distributed at the borough and health center district levels (New York City Department of Health, 2000). These geographic inequalities reflect the combined and interacting effects of differences in income, class, race and ethnicity, and other social dimensions. Of these, the effect of race and how it translates into the social and economic environment to affect birth outcome is poorly understood. In New York City, the low birthweight rate for 'African American' and 'Hispanic' infants exceeds the rate for 'white' and 'Asian' infants and this disparity has persisted for several decades.

Traditionally low birthweight has been studied at the individual level by identifying and eliminating individual-level risk factors. Strategies to reduce the number of low birthweight births have focused on reducing risk factors through health education related to reproduction and family planning, reduction of risky behaviors, increasing the accessibility of early and regular high-quality prenatal care services, and removing barriers to prenatal care. To address racial differentials the National Institute of

Medicine, Committee to Study the Prevention of Low Birthweight (1985) recommended that future research focus on the cumulative effects of poverty and social neglect, and the interaction of these factors with biological parameters. Understanding the social etiology of low birthweight in addition to its behavioral and medical risk factors is of utmost importance.

The purpose of this research is to investigate the geography of racial disparities in low birthweight in New York City. This cross-sectional multilevel study will focus particularly on residential segregation and its effect on the risk of low birthweight among African American infants and mothers. Recent studies suggest that residential segregation contributes to the sharp racial inequalities in health that exist in the United States (Fang et al, 1998; Acevedo-Garcia, 2000; Acevedo-Garcia, 2003). We know that residential segregation makes neighborhoods particularly vulnerable to disinvestments and decay but we do not know if African American women who live in segregated areas have poorer reproductive health outcomes than similar women who do not live in segregated areas. Residential segregation may contribute to racial disparities in low birthweight because it isolates African American women from amenities, opportunities, and resources, and such isolation may result in stress-related conditions and/or detrimental lifestyle behaviors affecting birth outcome.

This research starts from the premise that race is a social construct. Race is a category of difference defined or assigned to individuals by social groups. Racial definitions vary from one society to another and shift with changes in socio-political relations. In the

Untied States four major racial groups are identified by the Census: American Indian or Alaskan Native, Asian, Black or African American, and White [(a)]. When race is socially stratified Whites are considered the majority and all other racial groups are referred to as minorities. Of the minority groups, African Americans face a unique set of social, economic and political disadvantages that are rooted in slavery and maintained by racism. The degree to which these historical perceptions shape society to impact the reproductive health of African American women today is an underlying theme of this dissertation.

Residential segregation is the degree to which racial groups live separated from one another in the urban environment (Kaplan and Holloway, 1988). Residential segregation is most often studied within the context of African Americans because of their unique experience with racism, prejudice, and discrimination (Massey and Denton, 1989). The causal pathway by which residential segregation impacts low birthweight is probably not direct. We cannot assume that low birthweight will be prevalent because an area is highly segregated. In fact it could be argued that residential segregation may be protective of low birthweight because women of the same racial group, who live in the same neighborhood, are able to support each other. Alternatively, residential segregation may be associated with economic, social, and health care disadvantages, which independently or jointly, contributes to higher rates of low birthweight. This dissertation will investigate the scenario that residential segregation creates concentrated areas of poverty (Massey and Fischer, 2000), which in turn deteriorates the health status of

[(a)] In 2000, respondents were allowed to select one or more racial categories when they self identify or they were allowed to select "Some Other Race." Although these changes were made in the 2000 Census surveys they are not reflected in the birth certificate data.

African American women who live in these areas. In pregnant women, medical conditions that result from health deterioration may reduce the quality of the intrauterine environment, increasing fetal vulnerability to low birthweight.

Residential segregation creates concentrated areas of poverty through its interaction with income inequality (Massey and Denton, 1993; Massey and Fischer, 2000). In the 1970s, economic restructuring led to an increase in low wage earners and a momentum toward class isolation (Wilson, 1987). People of low socio-economic class either stayed at their present location or relocated to areas that were more affordable. The low-income areas where people remained or moved into probably lacked employment opportunities and many amenities in general. People had to choose between traveling farther for reasonably good paying jobs, settling for local minimal wage positions, collecting unemployment, and/or looking to the streets for unorthodox work. Massey and Fischer (2000) reported that structural changes had far greater consequences for African Americans living in segregated areas because of their limited mobility options. Some of the barriers to mobility included racism, prejudice and discrimination, few employment options, and/or lack of transportation. Barriers also differed by gender, with African American women experiencing additional difficulties because of single parent status, obtaining day care for children, and/or general fatigue due to poor nutrition.

In this dissertation residential segregation is considered a *hazard* to which African American women are exposed. Exposure may occur prior to or during pregnancy and risk may be acute or cumulative. The amount of exposure will depend on the level of

segregation. The duration of exposure will be estimated and based on nativity or place of birth of the mother. It is assumed that African American women born in the United States will have a longer duration of exposure to residential segregation than immigrating black women. I hypothesize that the effect of exposure to segregation on the risk of low birthweight will vary by the mother's place of birth.

In health statistics the term 'Black' encompasses Caribbean and African-born immigrants and native-born women. Previous studies of immigrant black mothers have demonstrated favorable birth outcomes compared to U.S.-born black mothers, despite their living in similar disadvantaged areas. These studies contradict the large body of research that observes higher morbidity and mortality in poor neighborhoods and in poor populations. This phenomenon is referred to in the literature as an *epidemiological paradox* (Markidas and Coreil, 1986). Improved birth outcomes among immigrant populations have been attributed to cultural factors, a higher standard of living during childhood, higher educational levels, extended family and community support networks, not smoking or participating in detrimental lifestyle behaviors, improved nutrition, and overall hardiness as a result of who emigrates (also referred to as selection bias) (Rumbaut, 1997; Shiono, 1997; Cervants et al, 1999).

While the *epidemiological paradox* has been observed among first generation immigrants, there is evidence that by the second generation, the incidence of adverse birth outcomes begins to increase and migrate toward that of the receiving population. For example, Landale (1996) found that first generation Mexican American women

living in predominately African American neighborhoods had birth outcomes similar to those of U.S.-born whites, but by the second generation their birth outcomes were more similar to those of their African American neighbors. These findings and others (Emanuel et al, 2004) suggest that place can exert a powerful influence on human reproduction.

Crane (1991) reported that living in segregated and impoverished neighborhoods without role models can lead to the adoption of values, attitudes, and behaviors in opposition to mainstream society, through peer influence. Initially these values and attitudes emerge because of an inability to accomplish mainstream ideals. Later it becomes a protective mechanism for self esteem (Wilson, 1987). Massey and Denton (1993) and Wilson (1987) explain that concentrated poverty in racially isolated areas has created an underclass of individuals with distinct behaviors and norms. One facet of these norms is that victims of segregation learn to accommodate their victimized status (Clark, 1965). Feelings of stress, anger, distrust, powerlessness, subordination, collective and enduring oppression (Massey and Denton, 1993) and some degree of weariness (Kozol, 1995) are common.

Stress can impinge on women through biological pathways that cause premature birth, a leading cause of low birthweight among African American women. From a biological perspective, stress is any challenge, psychological or physical, that threatens or is perceived to threaten homeostasis (Lazarus and Folkman, 1984; Wadhwa et al, 2001). Chronic stress is related to chronic or acute states of hypertension and may make women

more vulnerable to infection by weakening their immune systems. Both of these medical conditions are prevalent among African American women, and important causes of low birthweight.

Stress may also leave women more vulnerable to lifestyle behaviors that can negatively affect birth outcome. The most important of these is smoking. Smoking can impede intrauterine growth by restricting blood flow, oxygen, and nutrients, and can also cause premature rupture of fetal membranes. Young African American women are less likely to smoke than young white women but this trend reverses with age (Geronimus, 1993). Other unhealthy behaviors may include alcohol usage, illicit drug usage, poor nutritional habits, and/or delayed prenatal care. Interventions in the field of maternal and child health have focused on persuading women to relinquish unhealthy behaviors for healthier behaviors while pregnant.

In addition to focusing on risk factors while the mother is pregnant some researchers believe that an African American woman's life experience in itself can negatively impact reproductive outcome. Geronimus (1986, 1996, and 2001) explains that African American women at all socioeconomic levels are at risk of early health deterioration due to cumulative exposure to social, economic, and/or political exclusion. This health disadvantage, also called the *weathering hypothesis*, is particularly evident among African American women living in high-poverty urban areas. Racial differences in health trends begin at about 25 years of age and become most pronounced among 35 to 64 year olds. The concept of an accelerated aging process is supported by the lower life

expectancy observed among African American women compared to White women. The primary causes of early mortality are chronic disease deaths. Chronic diseases may also lead to early disabilities that can negatively impact reproductive outcomes. In addition, a worsening of health over the reproductive years means that the peak of childbearing may occur at a younger age. The research of Rauh et al (2001) also supports the findings of *weathering* in African American women.

In summary, African American women appear to have a very unique life experience from other racial and ethnic groups in the United States that researchers have not yet fully realized. While the long-term health effects of racial prejudice and discrimination are becoming more apparent at the individual level, we still do not know to what extent residential segregation, a manifestation of racism, contributes to racial disparities on a population level. Residential segregation is a tangible place-based outcome of racism that probably increases the risk of low birthweight in African American women through cumulative exposure and biological mechanisms that deteriorate health status during the reproductive years. This study on the geography of racial disparities in low birthweight in New York City will test this hypothesis using hierarchical modeling and spatial analytical techniques.

The specific objectives of this research include:

1. Measure the extent of racial disparity in low birthweight in New York City and how it varies for U.S.-born and foreign-born women;

2. Measure the association between residential segregation and low birthweight, controlling for individual-level risk factors;
3. Determine if African American women have a greater number of medical conditions or a more complex array of medical conditions during pregnancy at a relatively young age;
4. Determine if the relationship between residential segregation and low birthweight is mediated through medical conditions, and if so, which specific medical conditions are important mediators.

Research Significance

This research will contribute to our understanding of racial disparities in low birthweight in New York City by providing insight into the direct and indirect effects of residential segregation. We will also gain additional knowledge about the *epidemiological paradox* and answer the question of whether or not a paradox exists in locally segregated areas. In addition, this research will dispute or validate the *weathering hypothesis* by investigating the types of medical conditions that African American women experience over their reproductive years, and the degree to which these medical conditions mediate the local segregation and low birthweight relationship. Finally the findings of this research will provide future direction for public health program and policy initiatives.

Organization of Dissertation

This research is presented in the upcoming chapters. Chapter 2 reviews the literature and is divided into four sections (a) health geography and studies on geographic variation in

low birthweight, (b) pregnancy outcomes and known and suspected risk factors for low birthweight, (c) the history and significance of residential segregation, and (d) study hypotheses. Chapter 3 describes the data and methodology, including birth weight measures, segregation indices, and hierarchical models. The results are divided into two chapters, with the descriptive results in Chapter 4, and the analytical results in Chapter 5. Chapter 6 summarizes the findings, provides concluding remarks, and offers public health intervention and policy recommendations.

CHAPTER 2:

INDIVIDUAL AND GEOGRAPHIC VARIATIONS IN LOW BIRTHWEIGHT

2. BACKGROUND

This chapter contains a literature review of topics related to the geographic and social variation of low birthweight. The first section focuses on the discipline of health geography and how health geographers analyze relationships between health and place in space and time. Specific studies on geographic variations in low birthweight are examined. The second section reviews the known and suspected individual-level risk factors for low birthweight for African American women. It also provides an overview of pre-pregnancy and pregnancy-related medical conditions and describes how these conditions may potentially cause *weathering* and/or impact birth outcomes. The last section dwells into the history of residential segregation in the United States and provides evidence of why this social phenomenon persists today. It also discusses how the context of this place environment may affect African American women's reproductive success. Finally, research hypotheses are generated about how individual-level risk factors and residential segregation act independently or jointly to increase the risk of low birthweight in African American women and contribute to racial disparities in New York City.

2.A. HEALTH GEOGRAPHY

The link between place and health is a primary topic in the field of health geography. Place is defined as the relationship between an individual's place in the world, as well as their experience of the world they live in. For example, a woman's socioeconomic status

helps to shape her experience of the local environment just as the local setting influences her opportunity for health-related activities and experience (Eyles, 1985). Traditionally medical geographers have focused on disease ecology or understanding the context of disease from a bio-medical geographic perspective. Their primary focus was on disease etiology, how diseases spread, and how they could be eliminated. Over the last two decades, a paradigm shift occurred in the discipline and health geography emerged as a primary focus of research. Health geographers investigate health and ill- health from a socio-ecological geographic perspective and are interested in the situated relationships between a population and their social, cultural, and physical environments (Kearns, 1993). This shift in paradigm from medical to health geography compliments the goals of the World Health Organization, whose definition of health is "a state of complete physical, social and mental well-being and not merely the absence of disease or infirmity." This shift in paradigm also compliments the national health objectives "Health People 2010" at the Centers for Disease Control and Prevention. Kearns (1993) reports that while public health specialists and politicians sketch out a framework for health promotion, health geographers are able to complete this picture with their knowledge of how to incorporate elements of place in space and time. This dissertation will investigate the geography of low birthweight in New York City by considering residential segregation an important characteristic of places in which African American women reside. The degree to which these place characteristics directly or indirectly, impact birth outcomes, will be explored.

Studies on geographic variations in low birthweight consistently support the hypothesis that low birthweight is strongly correlated with poverty. O'Regan and Wiseman (1990) monitored the changing incidence of low birthweight at the census tract level in Oakland, California and from that were able to evaluate changing neighborhood poverty and social stress. This study supported other research using historical birth weight data showing birth weight was a sensitive indicator of short-term changes in living conditions (Vangen, 2004).

McLafferty and Tempalski (1995) examined the impact of political and economic restructuring on the changing context of women's reproductive health in New York City at the Zip code level by analyzing spatial and temporal trends in the late 1970s and 1980s. These authors measured the statistical significance of observed change with a Z score statistic. They found that in high status neighborhoods, increases in low birthweight were associated with an increase in non-white births and more households headed by women. In low status neighborhoods (i.e., Central Harlem, Central Brooklyn, and South Bronx) increases in low birthweight were related to a decline in women's ability to find employment, decreased utilization of prenatal care, and an increased number of households headed by women. Finally, in areas with a high percentage of female-headed households and a low percentage of households below poverty, there was still an increase in low birthweight. These important findings were attributed to women's vulnerability to economic restructuring, shifts in insurance coverage, and the reconfiguration of health services.

Roberts (1997) investigated neighborhood and social environments and the distribution of low birthweight at the community level in Chicago, Illinois. Using logistic regression, the most important finding was that economic hardship, as defined by the percentage of unemployed adults and the percentage of families living in poverty, was positively associated with low birthweight.

O'Campo et al (1997) used multilevel modeling to study the effect of macro-level social factors (i.e., community empowerment, unemployment, per-capita income, and household wealth) on the risk of low birthweight at the census tract level in Baltimore, Maryland. Social 'risk' was defined as "environmental stressors that shape individual vulnerability, and resistance to risk factors for health." These authors found that residents with a per-capita income of $8,000 or less had a significantly higher risk of low birthweight than did women with a higher income. Furthermore, per-capita income modified the relationship between health insurance status and low birthweight. For example, although the risk of low birthweight was greater for women on Medicaid, that risk was lower for women living in neighborhoods with a per-capita income of $8,000 or less than for women living in neighborhoods with a per-capita income of $8,000 or more. In other words, Medicaid reduced the risk of low birthweight in high-risk areas more than in low-risk areas. These authors also found that as unemployment increased the protective effect of early prenatal care initiation diminished. In fact, trimester of prenatal care was weakly associated with low birthweight in neighborhoods with high unemployment. These research findings demonstrated the need for policy initiatives that consider both individual- and community-level characteristic risk factors to reduce the

incidence of low birthweight. The authors also suggested that future research on low birthweight include macro-level social factors such as residential segregation to explain the racial and ethnic gap in low birthweight.

Gorman (1999) used multi-level modeling to investigate racial and ethnic variation in low birthweight at the county level in the United States. This author found that black, Puerto Rican, and Cuban women were at higher risk of low birthweight compared to white women, after controlling for individual-level risk factors. Mexican-Americans were the only group that had lower odds of low birthweight compared to white women, supporting previous research of more favorable birth outcomes in this population group. When contextual covariates were added to the models, the percentage of female- headed families had a positive effect on low birthweight for all groups, with the strongest effects among Cuban and Puerto Rican infants. The number of doctors per 100,000 population had no effect on low birthweight for any group. Median household income was negatively associated with low birthweight for Mexican- and White-Americans. Finally, as the percentage of the unemployed population increased the risk of low birthweight for black, Puerto Ricans and whites also increased. Interestingly, this effect was the opposite for Mexican- and Cuban-Americans.

Rauh et al (2001) explored whether women with similar risk profiles but living in different communities (poverty versus non-poverty) had the same probability of delivering a low birthweight infant, or whether the probability varied as a function of poverty. These authors found that after controlling for other individual-level risk factors,

there was an increased risk in low birthweight for older women and African American women. In addition, individual poverty (as measured by Medicaid status) could exacerbate the effect of maternal aging on moderately low birthweight births. There were not significant racial differences in the strength of the maternal age effect between African American and white women not receiving Medicaid. These findings suggested that racial disparities were mostly explained by community poverty. To test this hypothesis the authors used race-specific multilevel models to examine the main and cross-level effects of maternal age and community poverty. They found community poverty was associated with moderately low birthweight births among African American women only. However, community poverty did not exacerbate the effect of maternal aging on moderately low birthweight births, after controlling for individual-level risk factors. The authors reported on the need to explore residential history patterns of exposure to various community conditions to learn how these cumulative exposures relate to reproductive health outcomes over the reproductive years.

Pearl et al (2001) examined the relationship between neighborhood socioeconomic characteristics and birth weight, accounting for individual socioeconomic characteristics, among five ethnic groups in Los Angeles, California. Overall neighborhood poverty and unemployment were associated with decreasing birth weight. When stratified by ethnicity and birthplace, neighborhoods with high unemployment and poverty were related to decreasing birth weight among blacks and Asians only. Paradoxically, among foreign-born Latinas, living in the neighborhoods with the highest rates of unemployment

and poverty was associated with higher mean birth weights and lower risk of low birthweight. There was no neighborhood association with birth weight among whites.

Reader (2001) used an exploratory spatial data analysis approach to identify areas of high and low risk of low birthweight by race across the state of Florida. After spatial clusters were identified, selected socio-demographic characteristics of the census tracts underlying these clusters were compared. High rates of low birthweight for African American women were located in Miami/Dade/Broward/Palm counties which were predominately African American in population mix, whereas the African American high rate clusters in Central Florida were associated with census tracts there were still predominately white. High rate census tracts in general were associated with lower income levels, more diverse population mix, and lower levels of home ownership. In Central Florida, high rate clusters were associated with census tracts that were losing population or had very low population growth.

Sims and Rainge (2002) investigated the extent of infant health disparities between poor and non-poor African Americans and white mothers in Milwaukee. Three indicators of neighborhood risk were analyzed (a) economic, demographic, health and physical conditions, (b) concentrated poverty, or the extent to which poor mothers lived in high-poverty areas with other poor families, and (c) level of residential segregation between poor African-American/white mothers and affluent families. These authors found the average low birthweight rate for poor African American women was twice that for poor white women. African American mothers lived in less desirable areas that were

concentrated with poverty. They also reported that African Americans had to cope not only with their own poverty but that of those around them, a condition which made it even harder to escape distressed areas. In addition, they lived apart from affluent families, whose resources could provide a buffer against growing economic and health disparities. The authors concluded that economic conditions of poor neighborhoods, as well as a lack of prenatal care for African American mothers, might impact infant health disparities.

Finally, English et al (2002) overlayed a 0.5 mile grid on a map of San Diego County, California (n = 1,112 grid points) and measured changes in rates of low birthweight, premature birth, and intrauterine growth retardation, between 1980 and 1990, using the Z score test statistic similar to that used by McLafferty and Tempalski (1995). Using this continuous surface, these authors found that "hot spots" of low birthweight were associated with neighborhoods containing older and poorer housing, high rents, and a high density of childhood lead poisoning. Changes in maternal race and ethnicity composition and a measure of stability were significantly associated with intrauterine growth retardation. Measures of affluence significantly predicted premature birth.

In summary, previous studies have shown that African Americans are at higher risk of low birthweight than other racial and ethnic groups. Macro-structural changes account for their elevated risk as defined by economic hardship, high unemployment, a greater number of female-headed households, a lower percentage of homeowners, a lack of available prenatal care, and a decreased utilization of prenatal care services. Importantly,

however, is that Medicaid reduces the risk of low birthweight in poor areas. Also important to this study is that while community poverty increases the risk of low birthweight for African American women, it does not exacerbate the effect of maternal aging. These results suggest that health deterioration may not be due to community poverty. This research will reevaluate this finding by measuring the effect of maternal aging on low birthweight in the context of residential segregation.

2.B. PREGNANCY AND LOW BIRTHWEIGHT

Pregnancy is a critical period for the health of an infant. Successful birth requires that the mother provide her fetus with nutrients and oxygen essential for growth. She must create and maintain an intrauterine environment suitable for the fetus to develop. In normal pregnancy the fetus grows as a result of vital organ system development and function until it reaches the 'optimal' size for birth to occur. Optimizing the fetus's potential for growth takes approximately 42 weeks or 295 days of gestation. Although there is no single optimal birth weight, full-term infants in the United States typically weight between 3,285 and 3,487 grams (50^{th} percentile) (Alexander et al, 1999).

Low Birthweight

In 1950, the World Health Organization adopted the figure of 2,500 grams (5 pounds, 8 ounces) as a universal definition of low birthweight and 1,500 grams (3 pounds, 5 ounces) for very low birthweight. Infants are considered premature if they are born less than 37 weeks of gestation and weight less than 2,500 grams. Very low birthweight infants are almost always premature. Infants born at 37 weeks or greater gestation but

weight less than 2,500 grams are considered to have intrauterine growth retardation (IUGR).

In population studies birth weight is generally not normally distributed. Instead there is a large lower tail reflecting an excess of small babies. Wilcox and Russell (1986) suggests that a normal birth weight curve in any population is really a mixture of two distributions, one of the healthy population, and the other of infants (referred to as the "residual" distribution) in whom small size is a reflection of unhealthy maternal or fetal conditions. Low birthweight infants predominate in the residual distribution. The pathological group of infants is the excess of small babies in the residual distribution. A challenge to the study of low birthweight is to distinguish between those infants who are small but healthy from those who are small because of a pathological condition.

Premature Births

As mentioned earlier, low birthweight can occur from two general circumstances – prematurity and IUGR. Of these two, premature birth is the primary cause of low birthweight (Paneth, 1995) and is classified according to: births occurring from spontaneous premature labor, related to premature contractions (50 percent of cases), births related to the spontaneous rupture of the fetal membranes (30 percent of cases), and indicated delivery of a premature infant for the benefit of either the infant or mother (20 percent of cases) (Tucker et al, 1991; Goldenberg and Rouse, 1998). The causes of spontaneous premature birth related to premature contractions are still under investigation. It is known that hypertension during pregnancy reduces blood flow to the

fetus, resulting in premature contractures and premature labor. Infection can weaken membranes causing them to rupture, resulting in premature delivery. Other causes of premature birth include placenta previa (a condition in which the placenta is implanted over or nearly over the cervical os instead of in the proper position higher on the uterine wall), multi-parity (due to progressive damage to the cervix following successive deliveries), maternal obesity, and stand-up working conditions (Collaborative Perinatal Project, 1972).

Intrauterine Growth Retardation

Intrauterine growth retardation (IUGR) is associated with conditions that interfere with the circulation to the placenta, with the development or growth of the fetus, or with the general health and nutrition of the pregnant woman. Certain medical conditions associated with IUGR include chronic diabetes, chronic hypertension, and chronic renal disease. Other behavioral mechanisms such as smoking, alcohol, or drug use may lead to hypoxia and IUGR. The duration and severity of these medical conditions and hypoxic events are roughly related to the severity of the IUGR (National Institute of Medicine, 1985).

Development Disorders

Low birthweight infants can experience an array of development disorders after birth and later on in life. These disorders include: neurological handicaps (McCormick, 1985) such as cerebral palsy (Ellenberg and Nelson, 1979), hearing impairment (Daghistani et al, 2002; Nafstad et al, 2002), deafness (Bergman et al, 1985), blindness (Gallo and

Lennerstrand, 1991), epilepsy (Dunn et al, 1986), chronic lung disease (Kraybill et al, 1987), metabolic disorders (Feusner and Plaschkes, 2002; Tenhola et al, 2002), autism (Centers for Disease Control and Prevention, 2004), cognitive learning disabilities (McCormick et al, 1998; Hack et al, 1995; Kiely et al, 1998), and psychiatric disorders, including being inattentive, having difficulty socializing, and low self esteem (Elgen et al, 2002). There is also evidence that substandard nutrition during critical periods of early development may lead to hypertension, heart disease, diabetes, and some forms of cancer later in life (Segelken, 1996). These conditions are probably caused by poorly developed organs or organ systems, which may be attributed to premature birth or IUGR.

Although low birthweight increases the risk of many development disorders, it should be emphasized that most low birthweight infants experience no health or development problems. The smallest infants - especially those with very low birthweight - are much more likely to have health and development problems than are infants born with higher weight. In addition, the risks of short- or long-term complications are higher for infants who do not receive good health care during and after birth. Thus, for some infants, low birthweight has important long-term health consequences; however, these consequences can partly be alleviated by appropriate, high quality health care.

Finally, the increases in health care costs associated with low birthweight are substantial. In 1988, the additional costs of health care, education, and child care for the 3.5 to 4 million children ages 0 to 15 born low birthweight cost were estimated at between $5.5 and $6 billion dollars more than if those children were born normal birth weight. Low

birthweight accounts for 10 percent of all health care costs for children (Lewit et al, 1995).

2.C. RISK FACTORS FOR LOW BIRTHWEIGHT

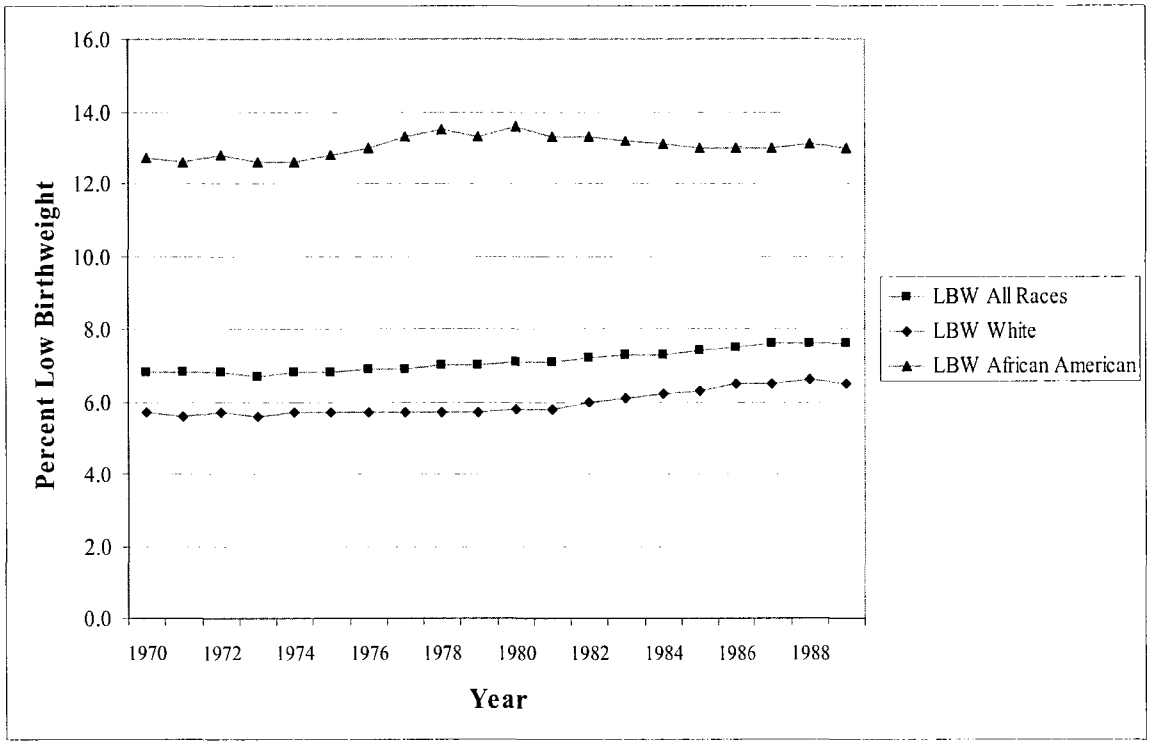
The causes of low birthweight include both individual-level risk factors (e.g., demographic, behavioral, and physiological characteristics of the mother and infant) and contextual factors that reflect the place environment in which the mother lives and interacts during pregnancy. A description of known and suspected individual-level risk factors for low birthweight in the African American population are presented and include: maternal race, origin of birth, age, weight gain and nutrition, parity, birth order, and birth intervals, multiple pregnancies, smoking, alcohol use, illicit drug and other substance use, intergenerational transmission of low birthweight, and individual socioeconomic factors, specifically, marital status, education, occupation, and personal income. In addition, stress and pre-pregnancy and pregnancy-related medical conditions are reviewed.

Individual-Level Risk Factors

Race

Racial disparities in low birthweight have been observed for many decades. Figure 1 is a graph showing the racial disparity in low birthweight in the United States between 1980 and 2000. African American women consistently had higher rates than white women during these years.

Figure 1: Low Birthweight by Race in the Untied States, 1980 to 2000



Source: National Institute of Health, 2000

Table 1 compares the rate of low birthweight in New York City and the United States in 2000. In the United States the rate of low birthweight in African American women is almost double that of white women. In New York City the gap is slightly narrower, but still the rate for African American women is 3.7 percentage points higher than the rate for white women. The smaller gap in New York City may reflect the fact that both the black and white population in the city are very diverse including significant numbers of immigrants and Hispanics. As noted earlier, immigrant blacks tend to have lower rates of low birthweight than native-born, thus the presence of a large immigrant black population would decrease the low birthweight percentage for the city's African American population.

Table 1: Rates of Low Birthweight for African American and White Women in New York City and the United States, 2000

	New York City				United States			
Birth Outcome	**African American**		**White**		**African American**		**White**	
	No.	(%)	No.	(%)	No.	(%)	No.	(%)
Low birthweight	4268	10.9	5047	7.2	80778	12.9	208818	6.5
Not low birthweight	35019	89.1	65396	92.8	544121	87.1	2981487	93.5
Sub Total*	39287		70443		624899		3190305	
Not stated			2		699		3700	
Grand Total	39287	100.0	70445	100.0	622598	100.0	3194005	100.0

*Percentages are calculated on subtotals before missing values added

Sources: New York City Department of Health and Mental Hygiene, Summary of Vital Statistics, 2000 and Centers for Disease Control and Prevention, National Vital Statistics Reports, 2000.

Studies conducted to identify biological causes of racial disparities in low birthweight have been equivocal (National Institute of Medicine, 1985). For example, anthropometrical studies have found persistent racial disparities despite similar stature and weight (National Institute of Medicine, 1985). Some genetic studies suggest a biological component (Van Den Oord and Rowe, 2000, 2001) but this mechanism is highly controversial (Frank, 2001). Van Den Oord and Row (2000) used a quantitative genetics approach to examine variation in birth weight that included measurements of heredity, shared environments, and nonshared environments. Shared environments were those that operated to make siblings alike. Nonshared environments were environmental effects that were unique to a particular child. The authors found that racial differences could be explained by the shared environment of the fetuses, in this case referring to characteristics of the fetal uterine environment. They suggested that possible genetic effects such as those affecting the mother's physical or physiological characteristics could be important because they contribute to the shared environment. In a follow-up study (Van Den Oord, 2001) the authors contributed differences in the fetal uterine environment to maternal genetics and a specific allele of genes that were more prevalent

in African American women than white women. In response to this research Frank (2001) argued that racial groups do not represent discrete genetic entities and this type of research threatens future research on racial/ethnic disparities in health. This argument is supported by the American Association of Physical Anthropologists (AAPA, 1996) who report great genetic diversity within all human populations. Pure races, in the sense of genetically homogeneous populations, do not exist in the human species today, nor is there any evidence that they have existed ever in the past. Kessel et al (1988) also reports that we "do not confuse the biological concept of race with the cumulative consequences of racism." Race as a social construct may cause low birthweight if a woman is exposed to stressor of racial discrimination. Physiological responses to racism include increased blood pressure (Krieger et al, 1990; Dressler, 1990), slow recovery in increased blood pressure (Fang and Myers, 2001), increased heart rate (Daniels et al, 2001; Morris-Prather et al, 1996), decrease in blood flow (Jones et al, 1996), greater cerebral reactivity (Armstead et al, 1989), mood sensitivity (Kingie et al, 1998) and increased anxiety and worry (Harrell et al, 1980). Other physiological responses to stress in general are provided in the section proceeding medical conditions.

Origin of Birth

In health statistics the term 'African American' encompasses West Indian and Caribbean Island immigrants, African-born immigrants, and U.S.-born women. Research on ethnic and racial differences in low birthweight consistently shows that immigrant women have lower rates of low birthweight than U.S.-born women (Cervantes et al, 1999; Gould et al, 2003). These findings are consistent with earlier studies describing this phenomenon

known as the *epidemiological paradox* (Markides and Coreil, 1986; Williams et al, 1986; Rumbaut, 1997; Shiono, 1997). Cervants et al (1999) studied the birth outcomes of Mexican, Puerto Rican, black, and white women in Chicago. These authors found that immigrant Mexican and white women had the lowest rates of low birthweight and preterm births, whereas native and immigrant blacks and Puerto Ricans had the highest rates. Blacks and Mexicans had the largest nativity differentials in the rates of low birthweight and preterm births. For immigrant Mexican women low rates of low birthweight occurred despite having lower socioeconomic status, lower educational levels, and inherent difficulties encountered as immigrants.

Protective features that consistently appear in the literature to explain the *epidemiological paradox* include improved diet, a lower prevalence of smoking and alcohol use, greater family cohesiveness and community support, and original cultural practices. In addition, selection bias and the emigration of primarily healthy women may account for some of these differences. Cervantes et al (1999) point out that with immigration the strong components of cultural perspectives that protect health are retained, while at the same time, specific positive values offered by the American culture are acquired.

Age

Age has a direct biological effect on birth weight only in later reproductive years. Pregnancy in a young woman by itself is not a risk factor because human reproductive functions are intact by puberty. Pregnancy at young ages may cause low birthweight if a woman's nutritional intake is poor or if she is immature and not able to care for herself.

Young mothers generally unmarried and/or often report late for prenatal care, thus requiring additional support (National Institute of Medicine, 1985).

Weight Gain and Nutrition

If the mother's health and nutritional intake are well balanced, the fetus will grow at a natural pace. Women gain on average 25 to 30 pounds over 37 weeks of gestation (Hytten and Leitch, 1964). In general, a 2.2-pound increase in maternal weight results in a 20-gram increase in mean birth weight (Niswander and Jackson, 1974). The optimal weight gain for underweight women is 30 pounds (Naeye, 1979). The optimal weight gain for overweight women is 15 to 20 pounds (Kliegman et al, 1985). Obese women are less likely than non-obese women to have low birthweight infants; however, preterm infants of obese women are smaller than preterm infants born to normal weight mothers. Preterm infants of obese women are also at higher risk of neonatal and infant death (Garbaciak et al, 1985).

Parity, Birth Order, and Birth Intervals

Women who are pregnant for the first time (parity 0) are more likely to have low birthweight infants than women of second and/or subsequent births (parity 1 or greater) (Eisner et al, 1979). This is because women weigh less at their first birth and have fewer nutritional reserves. If a woman's first born is low birthweight she is more likely to deliver another low birthweight infant in subsequent pregnancies. Ekwo and Moawad (1998) found that African American women who previously delivered a premature infant are three times as likely of having another premature delivery. The birth order effect also

varies with mother's age. Women who are pregnant for the first time and 35 years and older are twice as likely as women 18 to 34 years to have a low birthweight infant - this risk is higher for African American women than white women (Naeye and Tafari, 1983). Birth intervals less than one year are also associated with low birthweight (Valero, 2004).

Multiple Pregnancies

The intrauterine growth of twins is similar to single-born infants until the 30th week of gestation, after which time, twins grow slower. Twins or infants born of multiple births are generally smaller than singleton births (Naeye and Tafari, 1983).

Smoking

Smoking is the most important individual risk factor for low birthweight (Sprauve et al, 1999). Smoking one cigarette reduces blood flow to the placenta for up to 15 minutes (Lehlovirta and Forss, 1978). Smoking also restricts blood flow in the uterine arteries causing placenta previa, premature rupture of membranes (Savitz et al, 2001) and/or the development of a single umbilical artery, instead of the normal two (Collaborative Perinatal Project, 1972). Pregnant women who smoke 20 plus cigarettes per day are more likely to have infants born with IUGR (Sprauve et al, 1999). White women are more likely to smoke at younger ages and African American women are more likely to smoke at older ages (Geronimus, 1993; Alexander and Slay, 2002). Poor mothers are more likely to smoke than mothers of higher socioeconomic status (Paneth, 1995). Women who smoke while pregnant have less of an appetite, and lower pregnancy weight gain than nonsmokers (Naeye and Tafari, 1983).

Alcohol Abuse

Alcohol exposure can cause damage to the central nervous system in a developing fetus, leading to permanent brain damage. Alcohol-related nerve damage can also lead to reduced motor skills and touch sensation in infants during the first month of life. Women who drink excessive amounts of alcohol during pregnancy are at higher risk of having a baby with fetal alcohol syndrome. Fetal alcohol syndrome is usually diagnosed when the child enters school and is found to have a learning disability or attention deficit disorder (National Organization of Fetal Alcohol Syndrome, 2004).

Illicit Drug and Other Substance Abuse

Women who use illicit drugs are almost always at higher risk of premature birth or IUGR (American Council for Drug Education, 2004). Illicit drug use during pregnancy is associated with a two-fold increase in the risk of premature rupture of fetal membranes (Berkowitz et al, 1998). Some drugs can be harmful when used any time during pregnancy while others are particularly damaging at specific times during fetal development. Illicit drugs taken during the end of pregnancy may lead to a difficult birthing process and/or withdrawal symptoms in the newborn.

Socioeconomic Characteristics

Marriage may have a positive effect on birth weight through support of the husband. Financial support allows the woman the opportunity to choose how much and how long she will work during her pregnancy; she may be provided with health insurance; and/or she may receive general amenities (e.g. nutrition, better housing) that improve her well-

being. The husband may also provide support by caring for other children or providing general emotional support, both of which can reduce stressors during pregnancy (Hoffman and Hatch, 1996). Education has a positive effect on birth weight through its influence on lifestyle behaviors (e.g. diet), problem-solving capacity, and values (Rowley et al, 1993). Occupation may also have a positive effect on birth weight if the woman has job security, which reflects access to medical care, and the ability to obtain good housing. Personal income can have a positive effect on birth weight by providing access to medical care, good housing, a good diet, and more personal and social amenities in general. Occupation can have a negative impact on birth weight if a woman is standing on her feet for long periods of time (National Institute of Health, 1985).

Intergenerational Transmission

There is also evidence of intergenerational transmission of birth weight – women who were born low birthweight are more likely to deliver low birthweight infants (Collins and David, 1993). Conley and Bennett (2000) found that using grandparent fixed effects models (controlling for socioeconomic status) both father and mother's birth weight status had an effect on offspring outcomes. However, the degree of inheritance was weaker for African Americans than for other races. These findings implied a genetic association that does not work through the uterine environment but rather through the fetus itself. Collins et al (2003) also investigated the relationship between maternal birth weight and infant birth weight but selected only those mothers who received adequate prenatal care. These authors found the rate of low birthweight for white mothers who received adequate prenatal care was 4.0 percent for infants of former low birthweight

mothers compared to 2.1 percent for infants of former non-low birthweight mothers. In comparison, for African American mothers who received adequate prenatal care the low birthweight rate was 15.0 percent for infants of former low birthweight mothers compared to 7.2 percent for infants of former non-low birthweight mothers. The maternal-infant birth weight associations were consistent across all maternal age, education, marital status, and prenatal care categories. These authors concluded that maternal birth weight is a risk factor for infant low birthweight independent of risk status during the current pregnancy. Accounting for adequate prenatal care these authors were able to show that a greater percentage of low birthweight infants born to African American women (compared to white women) were attributable to maternal low birthweight.

Intergenerational studies along the lines of *weathering* suggest a *lifecourse approach* to explain racial disparities in birth outcomes (Lu and Halfon, 2003; Smith, 2004). This approach encompasses an early programming model (e.g., exposure in early life can influence future reproductive potential) and a cumulative pathway model (e.g., a decline in reproductive health results from cumulative wear and tear of the body's allostatic systems). Lu and Halfon (2003) suggest the use of longitudinal data to integrate these mechanisms and explain how different health trajectories develop. These authors comment that the effect of race on birth outcome is likely mediated in part through *weathering* of racism and racial discrimination over the life-course.

Stress

Through interviews of African American women in Detroit, Schulz et al (2001) found that stressors relating to police presence, perception of safety, and unfair treatment were significantly associated with depression; whereas financial and family stress were significantly associated with self-reported general health status. For birth outcomes, premature birth is the most common condition related to stress. Consistent associations have been reported between premature birth and perceived stress (Kramer et al, 2001), perceived negative life events or a greater number of life events (Hedegarrd et al, 1996), and racial discrimination (Dole et al, 2003). There is also a large body of research reporting a strong association between stress and cardiovascular disease, including hypertension, which is also associated with premature birth (Meis et al, 1998). Chronic stress may weaken the immune system increasing the risk of infection (Elenkov and Chrousos, 1999). Infection of the reproductive tract during pregnancy can lead to premature rupture of membranes and premature birth (Gibbs et al, 1992).

Medical Conditions

Medical conditions are considered an individual-level risk factor for low birthweight but often it is the environment that perpetuates the onset of disease. Investigating the medical conditions that African American and white women experience while pregnant can serve as a useful indicator of reproductive health and may lead to improved understanding of the *weathering hypothesis* and the causes of racial disparities in low birthweight. A brief description of the most common medical conditions reported on the birth certificate is outlined in Table 2. Medical conditions are categorized according to

those that are present prior to pregnancy (pre-pregnancy) and those that are diagnosed during pregnancy (pregnancy-related).

Table 2: Medical Conditions and Potential Impact on Birth Outcome	
2A. Pre-Pregnancy Medical Conditions	
Anemia	Reduced oxygenation in the mother's blood may lead to fetal hypoxia and premature birth or reduced intrauterine growth. Anemia may be caused by blood loss, diet deficiency, industrial poisons, diseases of the bone marrow, or any of several other conditions.
Cardiac disease	Poor cardiac output in the mother may lead to fetal hypoxia and premature birth or reduced intrauterine growth. Cardiac disease may be caused by a variety of conditions such as congenital malformations, arteriosclerosis, and a variety of other conditions.
Lung disease	Poor oxygenation in the mother's lungs may lead to fetal hypoxia and premature birth or reduced intrauterine growth. Lung diseases may be caused by asthma, emphysema, chronic obstructive lung disease, and a variety of other conditions.
Diabetes, chronic	Infants may be abnormally large if an excess of glucose is transferred to the fetus or abnormally small if an excess of insulin is transferred to the fetus or if the mother excessively reduces her caloric intake limiting glucose transfer. These infants are prone to IUGR. Diabetic mothers are also prone to infections.
Hypertension, chronic	Diagnosed prior to 20th week of pregnancy. Chronic hypertension in the mother may increase resistance in the uterine artery or reduce uteroplacental blood flow causing premature birth or reduced intrauterine growth. Hypertension may be hereditary, or caused by stress, smoking, obesity, renal artery stenosis, and other known and unknown conditions.
Renal disease	Reduces uteroplacental blood flow and protein transfer to the infant causing premature birth or reduced fetal growth.
Hepatitis	May impair fetal development.
Thyroid disease	Thyroid disease may be a result of poor diet but more likely is diagnosed without a clear picture of its etiology. Untreated, it may cause irregular growth of the fetus.

Table 2.A. (continued)	
Genital herpes and other sexually transmitted diseases	Untreated genital herpes or other sexually transmitted diseases may cause infection in the cervix resulting in premature rupture of the fetal membranes or infection in the fetus resulting in premature intrauterine growth disorder.
Rubella	Rubella (German measles) during pregnancy can cause miscarriage, congenital defects, or developmental problems. Most women have immunity either because they were immunized or had German measles as a child.
Tuberculosis	If diagnosed early in pregnancy and mother is started on anti-tuberculin medications then infant will not be in danger. Infection in the infant is rare unless the mother has a late-stage diagnosis.
Viral disease	Viral disease may cause infection in the cervix resulting in premature rupture of the fetal membranes or infection in the fetus resulting in premature intrauterine growth disorder. Viral disease may also lower the mother's immune system leaving her more vulnerable to other infections.
Obstetrical history	Previous preterm births, small for gestational age infants, or miscarriages increase the risk of low birthweight.
2.B. Pregnancy-Related Medical Conditions	
Diabetes, pregnancy related	Diagnosed during pregnancy. Infant may be abnormally large if an excess of glucose is transferred to the fetus. Infants may be abnormally small if an excess of insulin or an excess of glucose is transferred to the fetus. Fluctuations in glucose concentrations increase the risk of intrauterine growth retardation.
Hydramnios/olighydramnios	Excess or reduced amount of amniotic fluid can impair fetal development.
Hypertension, pregnancy-related (also called gestational hypertension)	Diagnosed after the 20th week of pregnancy. Hypertension may increase resistance in the uterine artery or reduce uteroplacental blood flow resulting in premature birth or reduced fetal growth.
Preeclampsia	Severe gestational hypertension with proteinuria and edema can cause premature birth.
Eclampsia	Characterized by preeclampsia and includes convulsions and/or coma causing premature birth.

Table 2.B. (continued)	
Incompetent cervix	Incompetent cervix may be due to infection, prior cervical instrumentation (e.g., following miscarriage or abortion) or idiopathic. If the cervix dilates before the end of gestation the infant will be born prematurely.
Uterine bleeding and placental disorders	Causes of bleeding in first trimester are unknown but associated with increased low birthweight. Bleeding in last half of pregnancy is usually associated with placenta previa or abrupto placenta.

Contextual Factors

Context refers to neighborhoods, areas, or group properties. Common area characteristics include characteristics of local populations such as median household income, per capita income, percentage unemployment, percentage poverty, and characteristics of the local environment such as the number of prenatal care facilities. The mechanisms, by which, place impacts the reproductive success of African American women is complex. Most of the literature on place as a risk factor for low birthweight focuses on poverty neighborhoods. African American women living in poverty are may lack prenatal care, live in deteriorating housing, and/or be at risk of exposure to environmental pollutants. For example, prenatal clinics may not be available, or if they are available, they may not be accessible because of travel time, lack of financial resources, or language barriers. Prenatal clinics might not be utilized because of culture, lack of knowledge, or poor quality of care. Over the last decade there has been an effort to enroll poor women in Medicaid so they can begin prenatal care early and continued to be monitored throughout their pregnancy. It is recommended that all women visit a prenatal clinic in their first trimester of pregnancy to optimize the intrauterine environment for the fetus (Nathanielsz, 1995).

In addition, African American women living in poverty may be at higher risk of exposure to environmental toxins. We know that older and poorer neighborhoods are more likely to contain deteriorating housing that may contain layers of lead paint. Lead is a known toxin that when ingested (such as through indoor dust) is stored in the bones and leached into the circulatory system during pregnancy. There is no placental barrier for lead and its greatest impact of exposure is during the third trimester, when the fetus is in its most rapid growth phase. Chronic exposure to lead can lead to low birthweight (Agency for Toxic Substances Disease Registry, 1999). Other environmental toxins that may cause low birthweight are volatile organic compounds in the water, particulate matter in the air, and pesticides. It is well known that local industries are often located in poor neighborhoods. These neighborhoods are referred to as environmental justice communities. Poor neighborhoods may also lack general amenities such as grocery stores and other retail outlets essential for meeting basic needs. Woman living in these areas may have to travel long distances or times to acquire essential goods and services. If a woman is unable to pay for the costs of travel, or cannot travel because of children at home, she will be deprived, leading to poor nutrition and/or additional stressors in her life. Over time these additional stressor may deteriorate her health status. These environmental stressors may lead to health deterioration and subsequent medical conditions. In pregnant women, these additional stressors and medical conditions may contribute to low birthweight births.

This next section will focus on residential segregation as a contextual risk factor for low birthweight. It begins with a history of residential segregation in the United States and

provides evidence of why this social phenomenon persists today. There is also a literature review of research specifically pertaining to residential segregation and low birthweight.

2.D. RESIDENTIAL SEGREGATION

There are five dimensions of residential segregation used to describe the geographic distribution of racial groups across space. These dimensions include (a) evenness or differential distribution, (b) exposure or lack of potential contact versus possible interaction, (c) concentration or the relative amount of physical space occupied, (d) centralization or spatial location near the city center, and (e) the degree of spatial clustering. Massey and Denton (1993) explain that a high level of segregation on any one of these dimensions is problematic because it isolates African Americans from amenities, opportunities, and resources that affect social and economic well- being. When three or more of these dimensions are experienced simultaneously racial groups are said to be hyper-segregated characterized by extreme social isolation. New York City is one of many metropolitan areas in the United States considered hyper-segregated (Massey and Denton, 1989).

Historically, residential segregation was measured and reported at the metropolitan scale using local enumeration units. Recently geographic methods have become available to measure residential segregation at the local scale. Indices produced for each local unit measure the degree of racial similarity (using spatial autocorrelation methods produced by Anselin, 1995), and spatial isolation, and/or interaction (using methods produced by

Wong, 2002). While indices alone do not describe the context of residential segregation in local units, they do provide a conceptually sound aggregate measure of social space from which to further evaluate where and under what conditions African American women reside.

Historical Context of Residential Segregation

Residential segregation by race is the outcome of historical, social, economic and political processes. In U.S. cities, these processes can be traced back to emancipation and freeing of African American slaves that occurred in the 19th century. For northern cities like New York, the pace and scope of racial segregation changed markedly in the 20th century. Integral were World Wars I and II and subsequent changes in the American economy. The establishment, growth, and persistence, of residential segregation in New York City are described from the turn of the 20th century.

The first settlement of New York's freed slaves was Five Points (located on the site of the present City Hall and its surrounding blocks) in lower Manhattan (Osofsky, 1965). Migrating African Americans who entered New York City initially lived in Five Points and later settled in Greenwich Village and along the east and west sides of Manhattan near the waterfronts. They lived in poor working class neighborhoods and were surrounded by Irish, German, and Italian immigrant enclaves. The migrant African Americans and European immigrants initially settled in transient communities called ghettos.

A major migration of African Americans from the south to the north occurred between 1910 and 1930 and was attributed to growing racism following the period of Reconstruction and the aftermath of the Civil War, emancipation and the reuniting of families separated by slavery, and the demand for unskilled labor prior to World War I. Those who migrated were primarily first descendants of former slaves. Allen and Turner (1996) report that the 'white elite and established African Americans in the North perceived the African American southerners as culturally backwards, ignorant, and dangerous and reacted to the Great Migration with fear and racism.' European migrants with lighter skin were able to move out of the ghettos into more amenable permanent locations. In contrast, African American migrants looking for homes outside of the ghetto experienced racial steering (term for real estate agents who show housing to African Americans only in African American neighborhoods), blockbusting (a term for enhancing fear among whites living in a transitional neighborhood), and red-lining (financial institutions refusing to lend money to non-white and transitional neighborhoods). These forms of prejudice and discrimination confined the vast majority of African Americans in New York City to racially- segregated neighborhood ghettos.

As the central business district in Manhattan became overcrowded wealthy whites moved out of lower Manhattan into large brownstones with open landscapes in a northern suburb called Harlem. Harlem became an exclusive neighborhood of the rich and remained overwhelmingly white until the expansion of the subway and population growth resulted in the overgrowth of housing construction, and the depreciation of property values. These conditions opened up the opportunity for African Americans to move to Harlem

despite the high rents charged by white landlords. At the same time a large number of African American immigrants from the British West Indies and other Caribbean Islands arrived in Harlem. Although their common experience of migration set them a part from U.S.-born African Americans, there was sense that Harlem was thriving.

With the Great Depression in the 1930s the number of African American migrants from the south and foreign-born immigrants dropped off sharply. African Americans were concentrated in Harlem in Northern Manhattan and South Bronx, and Bedford-Stuyvesant in Brooklyn, and suffered from high rates of unemployment and poverty. They lived in overcrowded housing that contributed to high rates of morbidity and mortality. Poverty and racial segregation became closely tied during this period.

After World War II there was a period of economic growth. Historical analyses suggest that this was a time when residential segregation could have been disbanded but instead it expanded. The increase in segregation resulted from a variety of factors including resistance by whites to integration, suburbanization and the decentralization of central business districts, and the implementation of urban redevelopment and renewal programs, including the construction of public housing. Public housing was initially built to house returning war veterans (Hirsch, 1983). Later it was built for displaced persons during the massive demolition and clearing of slums (Silver and Moeser, 1995). Public housing was generally constructed on the periphery of established ghettos and working class neighborhoods. Those persons displaced were relocated to nearby communities, which over time increased population density causing communities to deteriorate. The core of

the ghetto expanded. Racial residential segregation after World War II is distinct from initial African American ghettos because of its increased size, density, and degree of racial isolation (Kaplan and Holloway, 1998).

During this time, there was a second wave of African American migration from the south, and the numbers that arrived, surpassed that of the first wave prior to the Great Depression. There was a demand for housing by these new immigrants but whites continued to resist residential integration. Real estate agents continued to practice racial steering, blockbusting, and redlining despite victories in the Supreme Court on restricted covenants (Kaplan and Holloway, 1998). Employment opportunities moved to the suburbs as central business districts decentralized. The black ghettos expanded and became poorer while white suburbs grew. Massey and Denton (1993) report that by the 1960s, the psychological alienation produced by decades of segregation led to riots and ultimately the assassination of Martin Luther King.

Contemporary Context of Residential Segregation

Economic restructuring began in the 1970s with a shift from manufacturing-based economy to a decentralized and global economy, heavily reliant on finance, services, and technology. The loss of manufacturing jobs was particularly severe in and around the segregated, inner city neighborhoods of northern industrialized cities like New York. In his book, The Truly Disadvantaged, Wilson (1987) describes the growing ties between poverty and residential segregation, as deindustrialization, employment deconcentration, and occupational bifurcation lead to a growth in ghetto poverty. Growth of jobs and

opportunities for education and training primarily occurred in the suburbs, causing a rise in income inequality and a momentum toward class segregation. Massey and Denton (1993) reported that although racial and class segregation acted independently to concentrate poverty, their simultaneous occurrence caused neighborhoods of poor African Americans to deteriorate substantially more than neighborhoods of poor whites. Furthermore, African Americans of higher socioeconomic status faced stronger barriers to residential mobility than whites of similar social standing. As a result African Americans of different social classes were forced to live in proximity to one another. Those of higher class had fewer resources and amenities available to them as whites of similar background (Massey, 1990).

Over the last three decades, immigration also impacted racially segregated urban neighborhoods. In 1965 immigration reform opened the door to West Indian and Caribbean Island immigration. Those who arrived came from diverse segments of society including urban elite seeking to protect their wealth, middle class households searching for broader opportunities, and a large number of poor people looking for a standard of living above subsistence (Kazinitz, 1992). By the 1980s there were distinct West Indian neighborhoods in New York City with the core community in Harlem shifting to Bedford-Stuyvesant, Crown Heights, East Flatbush and the Flatbush sections of Brooklyn, southeastern Queens, and the northeast Bronx (Kazinitz, 1992. The St. Albans neighborhood in southern Queens was one of New York's first middle-class African American enclaves. Middle-class Caribbean homebuyers also moved into the semi-suburban communities of southeastern Queens. Today East Flatbush in Brooklyn is

New York City's most populated West Indian neighborhood.

Most African immigrants came to New York City after 1980. They were motivated to migrate following economic restructuring and the loss of jobs and potential threat in civil wars. Many African immigrants are professionals with advanced education. Fordham Road in the Bronx is an African immigrant enclave. Although African American immigrants co-exist with native-born African American, there are tensions over economic disparities. While many native-born African Americans live in poverty, the average annual income of an African immigrant family is $45,000 – among the highest of any immigrant group (Bronx Beat, 2004).

The historical development of racial residential segregation and contemporary geographic patterns of African Americans settlement in New York City set the stage for this research.

Residential Segregation and Low Birthweight

New York City's highly segregated residential neighborhoods reflect the social forces that constrain African American's residential choices both historically and today. High rates of residential segregation have been linked to high rates of infant mortality for African American infants (Yankauer, 1950; LaViest, 1989; Polednak, 1991; Polednak, 1996) and rising African American and white disparity in risk (Polednak, 1996). Polednak (1991) found residential segregation to be an important predictor of African American and white infant mortality independent of African American-white differences

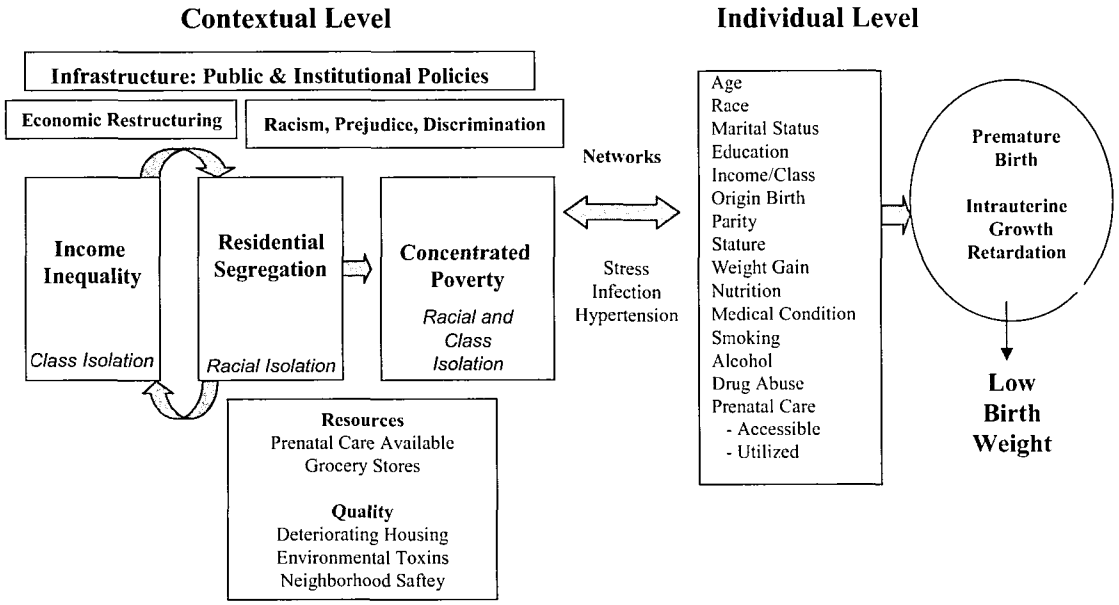
in median family income and poverty prevalence. Polednak (1996) also found a strong relationship between residential segregation and low birthweight across metropolitan areas in the United States, even after controlling for differences in poverty. The author speculates that persistently high rates of low birthweight among African American women living in hyper-segregated metropolitan areas may be related to the concentration of extreme poverty (especially in central core cities of certain large metropolitan statistical areas), poorer neighborhood quality (e.g., inadequate high-density housing, crime, noise, and psychosocial stresses), and a higher prevalence of risk factors.

2.E. CONCEPTUAL MODEL AND STUDY HYPOTHESES

This dissertation will utilize two-stage hierarchical (multilevel) models to identify individual- and contextual-risk factors for low birthweight. Multilevel analyses will be designed such that mothers (individual women) are nested within census tracts (neighborhoods) in which they reside. The effect of residential segregation will be examined by controlling for individual-level confounders (because women may be sorted into census tracts according to maternal characteristics, and these characteristics may be related to low birthweight). Individual level risk factors for low birthweight and residential segregation may also interact: the effect of individual-level variables may differ whether or not a woman lives in residential segregation, and the effects of residential segregation may differ by individual-level variables. In addition, multilevel analyses will allow for the examination of within- and between- neighborhood variability in low birthweight and the extent to which between-census tract variability is "explained" by individual level risk factors and residential segregation.

The conceptual model for this dissertation is provided in Figure 2. It shows the relationship between residential segregation and individual-level risk factors for low birthweight. Residential segregation interacts with income inequality to create concentrated areas of poverty as a result of macro-structural factors: institutional and policy, economic restructuring, racism, prejudice, and discrimination. If there is a lack of prenatal care facilities, and/or grocery stores, or if women live in deteriorating housing are exposed to environmental toxins, and/or live in unsafe neighborhoods, residential segregation may have a direct effect on low birthweight. Residential segregation may have an indirect effect on low birthweight through mediators such as social networks (informal or formal) and/or health deterioration exhibited by medical conditions (e.g., stress, infection, and/or hypertension). This dissertation will focus on the mediators - medical conditions - and interpret them as biological/physiological responses to the context of residential segregation. In pregnant women these medical conditions will diminish the quality of the intrauterine environment, leading to premature birth or intrauterine growth retardation, otherwise referred to as low birthweight.

Figure 2: Conceptual Model



Study Hypotheses

After reviewing the literature on racial disparities in low birthweight there are specific research gaps that this dissertation will address. First, we do not know how local segregation impacts low birthweight. All of the literature reporting the adverse effect of residential segregation on birth outcomes is reported at the metropolitan scale. This research will provide insight into whether or not African American women living in neighborhoods that are highly segregated by race have poorer birth outcomes than similar women who do not live in segregated neighborhoods. Second, we do not know if the *epidemiological paradox* exists in locally segregated areas. Does the association between segregation and low birthweight differ for immigrant and U.S.-born black women? Third, we know from previous research that maternal age has a positive effect on low

birthweight and that risk is greater for African American women. However, we do not know how much of the disparity in risk is due to health deterioration and the presence of medical conditions prior to and during pregnancy. Furthermore, we do not know the degree to which medical conditions mediate the relationship between residential segregation and low birthweight.

Therefore,

1. It is hypothesized that the probability of low birthweight will be greater for African American women than white women, U.S.-born women than foreign-born women, and U.S.-born African American women than foreign-born African American women.
2. It is hypothesized that African American women living in racially segregated areas will have a greater probability of low birthweight than African American women not living in such areas.
3. It is hypothesized that the probability of medical conditions will increase with increasing age and that risk will be greater for African American women.
4. It is also hypothesized that residential segregation will contribute to low birthweight through the mediating effects of medical conditions.

The data and methodologies that will be used to meet the objectives outlined in Chapter 1 and test the hypotheses presented above are provided in Chapter 3.

CHAPTER 3:

MEASURING THE EFFECT OF RESIDENTIAL SEGREGATION ON LOW BIRTHWEIGHT

This chapter reviews the sources of data, types of data, and the methodologies used in the analyses for this dissertation.

3.A. DATA

Study Area

The study area included the five counties (and their boroughs) of New York City, specifically The Bronx (Bronx), Kings (Brooklyn), New York (Manhattan), Queens (Queens) and Richmond (Staten Island). New York City was selected as the study area because of its high levels of local racial segregation. New York City is one of the most highly segregated cities in the United States (Massey and Denton, 1989). It also has a diverse population with a large black population that includes both native-born African Americans and foreign-born immigrants. New York City's black immigrant population includes large Caribbean-born and African-born groups. Both the size and diversity of the black population made New York City a good site for studying the impacts of residential segregation on low birthweight for immigrant and U.S.-born women.

Study Populations

The populations that were investigated in this study were all black and white women who gave birth in New York City during the year 2000. Black women were the main population of interest, and white women were included for comparison purposes. For

example, Hispanic women can be of either black or white race, and the same is true for women of other ethnic backgrounds. Because racial disparities were the main focus of this research, race was a primary variable that defined the study population. Ethnicity and country of origin were analyzed as sources of difference among women within racial groups.

Individual-level attributes of the mothers and their infants were obtained from the birth certificate using registered vital statistics birth data obtained from the Office of Vital Statistics and Epidemiology, New York City Department of Health and Mental Hygiene. The data were for the year 2000. Only records on live singleton births were utilized in this research. Births that were twins, triplets, etc. were removed from the study because of their usual small size. Other variables included in the data set were maternal characteristics such as age, marital status, education, insurance coverage, and behaviors such as smoking; health data – types of medical conditions such as anemia, diabetes, infections, and hypertension for the mother; and data about the infant such as gender, birth weight, gestational age, and birth defects. Each record also contained geographic information on the mother such as her residential address at time of birth, and the ZIP code and county of residence. Demographic data used to calculate segregation indices and data on poverty were obtained from the SF 1 and SF 3 files, U.S. Bureau of the Census, 2000.

In total there were 122,288 birth records for investigation. Those records excluded from the analysis were women who lived outside of New York City but gave birth in New

York City (n=201), women with an incomplete residential address (n= 3,592), women with 'other' or 'unknown' race (n= 598), women with American Indian or Asian race (n=14,447), and infants with a missing birth weight (n= 6), or missing gestational age (n= 6,562). In total 25,406 (20.7%) records were removed from the birth file because of missing data or race-specific data other than African American and white, leaving 96,882 birth records for analysis. Of these, 36,638 births were to African American women and 60,244 births were to white women.

Geocoding

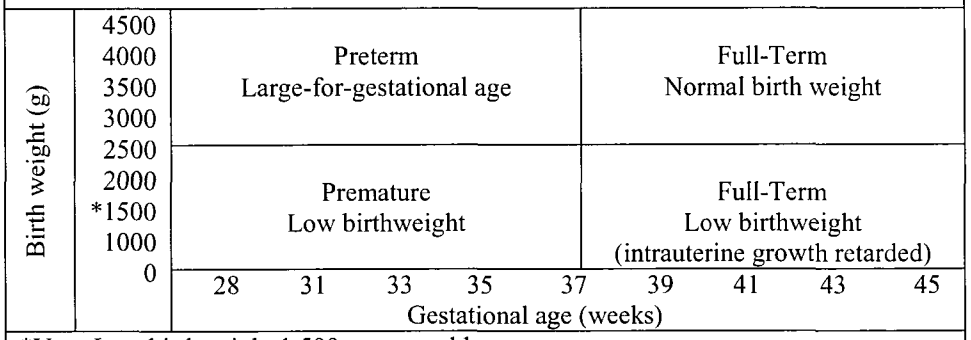
Birth records were matched to New York City's LION street file using standard address matching geocoding procedures in ArcView 3.2. The 'batch' match rate was approximately 85 percent. The remaining records were interactively matched. Each birth record was assigned a census tract identifier by overlaying the birth records onto the New York City census tract boundary file and using a 'spatial join' geoprocessing procedure. This census tract identifier was used to designate the contextual variables in the multilevel analyses.

Individual-Level Variables

All of the individual-level variables used in this research came from the birth data set. The dependent variable used in the analytical analyses was low birthweight (LBW, dichotomous 1=yes and 0=no). Within the low birthweight group, premature births were those infants born less than 2,500 grams and less than 37 weeks gestation and IUGR were

those infants born less than 2,500 grams and greater than or equal to 37 weeks gestation. Figure 3 shows the classification scheme for birth weight.

Figure 3: Classification Scheme for Birth Weight



*Very Low birthweight 1,500 grams and less
Source: Kiely *et al*, 1994

Maternal and infant characteristics (also referred to as individual-level or Level-1 variables) that were included in this analysis are:

Maternal race (MRACE), dichotomous variable
0 = White 1 = Black

Maternal origin of birth (ORIGIN), dichotomous variable
0 = U.S. born 1 = Foreign-born

MRACE*ORIGIN interaction term, dichotomous variable
0 = Other 1 = Black U.S.-born
0 = Other 1 = Black Foreign-born

Maternal age (MAGE), dichotomous variable

0 = Other	1 = Age < 19 years	(MAGE19)
0 = Other	1 = Age 20 to < 25 years	(MAGE24)
0 = Other	1 = Age 25 to < 30 years	(MAGE29)
0 = Other	1 = Age 30 to < 34 years	(MAGE34)
0 = Other	1 = Age 35 to < 40 years	(MAGE39)
0 = Other	1 = Age > 40 years	(MAGE45)

When maternal age was used as a control it was input as a continuous variable, and centered on the mean, (age 27 years) (MAGE27).

Pre-pregnancy and pregnancy- related medical conditions were used in this analysis to analyze the incidence of reproductive health risks in pregnant women. Data on these conditions were available in the birth data set.

Pre-pregnancy medical conditions were set up as dichotomous variables:

0 = No	1 = Anemia	(MRISK395)
0 = No	1 = Cardiac disease	(MRISK396)
0 = No	1 = Lung disease	(MRISK397)
0 = No	1 = Diabetes	(MRISK399)
0 = No	1 = Genital herpes	(MRISK400)
0 = No	1 = Other STDs[a]	(MRISK401)
0 = No	1 = Hepatitis	(MRISK404)
0 = No	1 = Hypertension, chronic	(MRISK405)
0 = No	1 = Renal disease	(MRISK412)
0 = No	1 = Thyroid condition	(MRISK423)
0 = No	1 = Rubella	(MRISK426)
0 = No	1 = Tuberculosis	(MRISK427)
0 = No	1 = Viral disease	(MRISK428)
0 = No	1 = Previous preterm or SGA[b]	(MRISK411)
0 = No	1 = Previous miscarriage	(MRISK421)

(a) Sexually transmitted diseases; (b) Small for gestational age

Pregnancy related medical conditions were defined in a similar way:

0 = No	1 = Diabetes	(MRISK398)
0 = No	1 = Hydramnios/Olighydramnios	(MRISK402)
0 = No	1 = Hypertension	(MRISK406)
0 = No	1 = Preeclampsia	(MRISK407)
0 = No	1 = Eclampsia	(MRISK408)
0 = No	1 = Incompetent cervix	(MRISK409)
0 = No	1 = Uterine bleeding [c]	(MRISK414)
0 = No	1 = Uterine bleeding [d]	(MRISK415)
0 = No	1 = Uterine bleeding [e]	(MRISK416)

(c) first trimester, (d) second trimester, (e) third trimester of pregnancy

Other maternal socio-economic characteristics and risk behaviors included:

Marital status (MARITAL), dichotomous variable
0 = Not Married 1 = Married

College (COLLEGE), dichotomous variable
0 = No College 1 = College

Health insurance during pregnancy (MEDICAID), categorical variable
0 = HMO or other insurance 1 = Medicaid or self pay

Smoking (SMOKE), dichotomous variable
0 = No 1 = Yes

Alcohol (ALCOHOL) number of drinks per day, continuous variable

Substance abuse (SUBUSE), dichotomous variable
0 = No 1 = Yes

Contextual-Level Variables

Residential segregation was the primary contextual variable used in this analysis. Poverty was used as a comparison contextual-level variable. These contextual variables (also referred to as Level-2 variables) represented the place environments in which the mothers resided. Data for these contextual variables were obtained from the 2000 Population Census (SF 1) for all census tracts (n=2095) in New York City.

Residential Segregation -

In this analysis residential segregation (SEG) was used as a dichotomous variable on maps to distinguish segregated areas from non-segregated areas. In hierarchical modeling residential segregation was used as a continuous variable. Residential segregation was calculated using a local spatial segregation index developed by Wong (2002). Professor Wong of George Mason University also provided the script written in Avenue programming language for ArcView 3.2 to calculate the segregation index, and the script was used in this research. The local spatial segregation index is based upon the potential for interaction.

Dr. Wong provided an illustration of this index in his paper "Modeling Local Segregation: a Spatial Interaction Approach" (2002). He reported: 'assuming that the study area has only two population groups A and B, then a*i* and b*i* are the population counts of the two groups in areal unit i, respectively. Using the simple binary condition to identify neighboring areal units, c*ij* is the element of the binary adjacency matrix with '1' indicating that the corresponding i and j units are neighbors, and '0' otherwise. Then the quantity of interaction between A in areal unit i and group B within the neighborhood of i can be modeled as

$$a_i \sum_j^n c_{ij} b_j$$

if the entire study area has *n* subunits. That is all people who belong to group B within the neighboring area are counted. The product of the population count of group A in i and the sum of the population in group B in the neighboring areas indicates the potential level of interaction between the two groups in reference to group A in areal unit *i*. But areal unit i also consists of people in group B and they can also interact with group A in *i*. Therefore, the above formulation is specified more precisely by the following two additional conditions:

1. *i* can be equal to *j*, and

2. $c_{ii} = 1$ instead of 0 as is found in most formulations of the adjacency matrix.

Based upon these two premises in measuring potential interactions for population in area unit *i*, the following local segregation measure is formulated.'

In this dissertation the index measures the lack of potential interaction or the degree of isolation between African Americans (*b*) in census tract *i* and all other racial groups (*a*):

$$S_{i*ba} = 1 - \frac{b_i \sum_j c_{ij} a_j}{bi \sum_j a_j}, j \text{ can be equal to } i$$

where the denominator is the possible interaction between *bi* and all other blacks and racial groups over the entire New York City without any spatial separation. This measure is standardized such that the value ranges from 0 (perfect potential interaction or no segregation) to 1 (no potential interaction or perfect segregation).

It was recognized that segregation indices for census tracts along the borders of New York City may be sensitive to a boundary effect given that an adjacency matrix was used in the computation. Census tracts located along the city boundary that have few adjacent populated tracts may have similar indices than those tracts in the interior of the city. Because in New York City most segregated areas are located in the interior of the city, and even tracts along the boundary generally have at least 3 neighboring census tracts, it was decided not to implement measures to correct for the boundary effect. However, in interpreting the results, I will note specific cases where the boundary effect may have been relevant.

Poverty -

The percentage of families with incomes below poverty was used to characterize census tracts according to poverty level. This measure was based on the ratio of family income to an appropriate poverty threshold. The poverty threshold was based on family size and residential location (urban/rural). A ratio of one or less indicated that a family's income was less than the poverty threshold, and therefore, they lived in poverty. In this analysis poverty (POV) was used as a dichotomous variable on maps to distinguish between

poverty versus non-poverty areas. In hierarchical modeling poverty was used as a continuous variable representing the percentage of families in a census tract with a ratio of income to poverty of 1.0 or less.

Potential Sources of Bias in the Birth Data

The birth certificate data were considered quite complete in that all registered births to women living in New York City were included. Births that were not reported were missing from the data set, but the number of missing births was thought to be very small. More important were errors in the measurement of specific variables in the data set. The mother's race was recorded by a health care provider based on self-reported race or observed race, and there may have been errors or confusion in the reporting process. In addition, several variables such as marital status, education, and occupation and risk behaviors had a large number of missing values. There were also many 'No' responses to smoking, alcohol use, and substance abuse, which probably represented severe underreporting and would need to be considered in the interpretation of the results. Furthermore, data on prenatal care utilization was limited. Data on birth order were not available and because low birthweight may have been more prevalent in a woman's first or second births, birth order may confound the results.

3.B. METHODOLOGY

This research will include a descriptive and analytical data analysis. The methodologies that were implemented to conduct these analyses are provided in the following discussion.

Descriptive Analyses

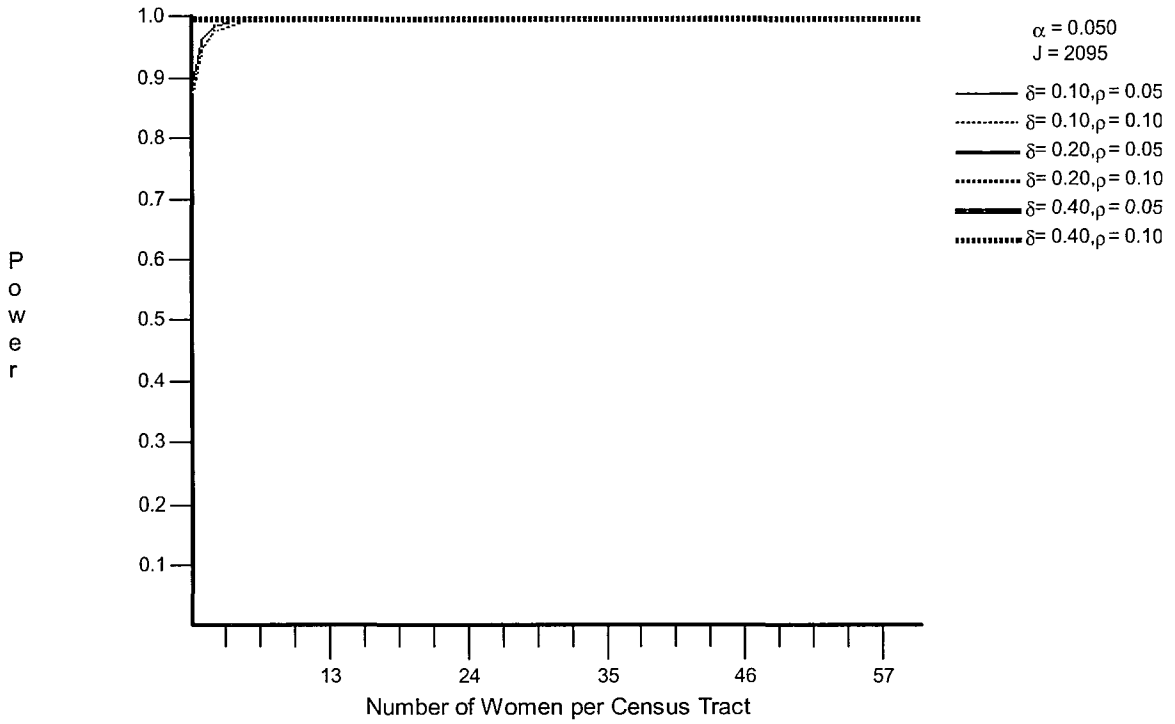
The individual- and contextual-level variables were checked for outliers. The distribution of continuous variables was checked by creating histograms, and if needed the variable(s) was centered. Scatterplots were created for birth weight by gestational age and residential segregation by percent poverty. Maps were also produced to analyze geographic variations in low birthweight, residential segregation, and poverty. Tables were also produced of characteristics of the mother and infant, including the average birth weight by race and place of origin. Place variations in birth outcomes, specifically low birthweight, premature birth, and IUGR, are also presented.

Sample Size

The number of births per census tract was calculated to examine potential sample size problems in small census tracts. Samples size and power calculations were conducted using the Optimal Size software available on the Scientific Software Incorporated website (SSI, 2005). The optimal sample size (number of women per census tract) was calculated for the 2095 census tracts, a power of 0.80, an effect size of 0.10, 0.20, and 0.40 and an alpha 0.05. Figure 4 shows that by using 2095 census tracts, the within census tract sample size can be relatively small for an optimal design. Detecting an effect

size of 0.10 requires approximately, 7 or 8 births, but for effect sizes of 0.2 and 0.4 a minimum of 2 births is sufficient. Less than 1 percent (n=20) of census tracts had 0 or 1 births. These findings concluded that small numbers of births within census tracts was not a problem in this study. Thus, all 2095 of New York City's census tracts were kept in the analysis.

Figure 4: Optimal Design Power Calculations



Most census tracts ranged between 50 and 200 births. Census tracts were used over other administrative geographic units to minimize heterogeneity within larger units and to avoid not having enough births in smaller units. Previous studies using census data in multilevel studies have found that there may be substantial heterogeneity in census tracts, causing the within tract variability to be greater than the between tract variability (Buka

et al, 2003; O'Campo, 2003). In this study, it may be difficult to measure between tract variability in low birthweight if women within census tracts are highly heterogeneous. Likewise, it may be difficult to measure within tract variations in low birthweight if women are highly homogeneous.

Analytical Analyses

Logistic regression models were first run to identify individual-level risk factors for low birthweight. The dependent variable was LBW and the independent variables were MRACE, ORIGIN, MAGE27, MARITAL, COLLEGE, MEDICAID, SMOKE, ALCOHOL, and SUBUSE. Those independent variables that were identified as significantly associated with LBW were input into hierarchical models. The exploratory logistic regression analyses were run prior to hierarchical modeling because they were less computationally intensive.

Binary logistic models were run in SPSS 11.5 using a backwards- conditional procedure. Estimates were based on maximum likelihood and the chi-square test statistic. Those variables with a p-value <0.05 were output. Odds ratio estimates were reported with 95 percent Wald confidence limits.

Two-stage HGLM (Bernoulli models) were run in HLM 5.0 and HLM 6.0. HGLM models were run to determine the contextual effect of residential segregation and poverty on low birthweight and to measure the cross-level effect of residential segregation * individual-level risk factors on low birthweight. Three kinds of parameters were

estimated in the hierarchical model: empirical Bayes (EB) estimates of randomly varying Level-1 coefficients; generalized least squares estimates for the Level-2 coefficients; and maximum likelihood estimates for the variance and covariance components. The Level-1 model in HGLM consists of 3 parts: a sampling model, a Bernoulli distribution logit-link, and a structural model (Raudenbush and Byrk, 2002).

Level-1 Sampling Model:

The Level-1 sampling model for a two-level HLM is written as:

$$\gamma_{ij} \mid \varphi \sim B(m_{ij}, \varphi_{ij})$$

where γ_{ij} is the number of "successes" in m_{ij} trials and φ_{ij} is the probability of success on each trial. In this research, low birthweight is designated as the "success" outcome. According to the binomial distribution, the expected value and variance of γ_{ij} may alternatively be written as

$$\mathrm{E}\left(\gamma_{ij} \mid \varphi_{ij}\right) = m_{ij}\varphi_{ij} \qquad \mathrm{Var}\left(\gamma_{ij} \mid \varphi_{ij}\right) = m_{ij}\varphi_{ij}\left(1-\varphi_{ij}\right)$$

When $m_{ij} = 1, \gamma_{ij}$ is a binary variable taking on a value of either zero or unity. This is known as a Bernoulli distribution.

Level-1 Bernoulli Distribution Logit-Link

The Level-1 predicted value, μ_{ij}, is transformed to η_{ij}, using a link function, to ensure that predictions are constrained to lie within a given interval. The equation for a Bernoulli sampling model and a logit link function in the unconditional model is:

$$\eta_{ij} = \log\left(\frac{\sigma_{ij}}{1-\sigma_{ij}}\right)$$

where η_{ij} is the log-odds of success. If the probability of success, φ_{ij}, is 0.5, the odds of success $\varphi_{ij}/(1-\varphi_{ij}) = 0.5/0.5 = 1.0$ and the log-odds or "logit" is log(1) = 0. When the probability of success is less than 0.5, the odds are less than 1.0 and the logit is negative. When the probability is greater than 0.5, the odds are greater than 1.0 and the logit is positive. While φ_{ij} is constrained to be in the interval (0,1), η_{ij} can take on any real value.

Level-1 Structural Model

The predicted log-odds can be converted to an odds or odds ratio (OR) by taking the exponent exp (η_{ij}). The predicted log-odds can also be converted to a predicted probability by computing

$$\varphi_{ij} = 1/(1+\exp(-\eta_{ij}))$$

The combination of a sampling model, logit link, and structural model, results in a linear model. The following HGLM models will be implemented in this analysis.

Variance Components Model

The Variance Components Model provides information on how much low birthweight rates vary between census tracts.

The Level-1 model is

$$\eta_{ij} = \beta_{0j}$$

where η_{ij}, is the intercept or mean level of low birthweight in each census tract, β_{0j}.

The Level-2 model is

$$\beta_{0j} = \gamma_{00} + u_{0j} \qquad u_{0j} \sim N(0, \tau_{00})$$

where, the mean level of low birthweight in each census tract, β_{0j} , is represented as a function of the grand mean, or level of low birthweight across all census tracts, γ_{00}, plus a random error, u_{0j}. The random error is assumed to be normally distributed with a mean of zero and variance, τ_{00}. τ_{00} is the variance between census tracts, in census tract-average log-odds of low birthweight.

Random Coefficients Model

The Random Coefficients Model adds Level-1 (individual-level) predictors to the previous model. The level-1 predictors were entered into the model as "un-centered."

The Level-1 structural model is

$$\eta_{ij} = \beta_{0j} + \beta_{1j}(\text{MRACE})_{ij} + \beta_{2j}(\text{ORIGIN})_{ij} + \beta_{3j}(\text{MARITAL})_{ij} + \beta_{4j}(\text{COLLEGE})_{ij}$$
$$+ \beta_{5j}(\text{INSURE})_{ij} + \beta_{6j}(\text{MAGE27})_{ij} + \beta_{7j}(\text{SMOKE})_{ij} + \beta_{8j}(\text{SUBUSE})_{ij}$$

where, MAGE27 was centered on the mean and all other Level-1 predictors were in dummy metric form.

The intercepts or mean level of low birthweight in each census tract and the slopes or mean level of each individual-level predictor for each census tract were output as Level-2 variables β_{0j} and β_{1j} through β_{8j}. In the Level-2 structural model the intercept and Level-1 coefficients were fixed.

$$\beta_{0j} = \gamma_{00} + \mu_{0j}$$

$$\beta_{1j} = \gamma_{10} + \mu_{1j}$$

$$\beta_{2j} = \gamma_{20} + \mu_{2j}$$

$$\beta_{3j} = \gamma_{30} + \mu_{3j}$$

$$\beta_{4j} = \gamma_{40} + \mu_{4j}$$

$$\beta_{5j} = \gamma_{50} + \mu_{5j}$$

$$\beta_{6j} = \gamma_{60} + \mu_{6j}$$

$$\beta_{7j} = \gamma_{70} + \mu_{7j}$$

$$\beta_{8j} = \gamma_{80} + \mu_{8j}$$

where γ_{00} is the grand mean, γ_{10} through γ_{80} are the grand slopes, and μ_{0j} and μ_{1j} through μ_{8j} are random effects represented as a variance-covariance matrix:

$$\text{Var} = \begin{bmatrix} & \begin{bmatrix} \tau_{00} & \tau_{01} \end{bmatrix} \\ u_1 & \begin{bmatrix} \tau_{10} & \tau_{11} \end{bmatrix} \\ u_2 & \begin{bmatrix} \tau_{20} & \tau_{21} \end{bmatrix} \\ u_3 & \begin{bmatrix} \tau_{30} & \tau_{31} \end{bmatrix} \\ u_4 & \begin{bmatrix} \tau_{40} & \tau_{41} \end{bmatrix} \\ u_5 & \begin{bmatrix} \tau_{50} & \tau_{51} \end{bmatrix} \\ u_6 & \begin{bmatrix} \tau_{60} & \tau_{61} \end{bmatrix} \\ u_7 & \begin{bmatrix} \tau_{70} & \tau_{71} \end{bmatrix} \\ u_8 & \begin{bmatrix} \tau_{80} & \tau_{81} \end{bmatrix} \end{bmatrix}$$

where

Var (μ_{0j}) = τ_{00} = unconditional variance in the Level-1 intercepts
Var (μ_{1j}) = τ_{11} = unconditional variance in the Level-1 slopes
Var (μ_{1j}) = τ_{21} = unconditional variance in the Level-1 slopes
Var (μ_{1j}) = τ_{31} = unconditional variance in the Level-1 slopes
Var (μ_{1j}) = τ_{41} = unconditional variance in the Level-1 slopes
Var (μ_{1j}) = τ_{51} = unconditional variance in the Level-1 slopes
Var (μ_{1j}) = τ_{61} = unconditional variance in the Level-1 slopes
Var (μ_{1j}) = τ_{71} = unconditional variance in the Level-1 slopes
Var (μ_{1j}) = τ_{81} = unconditional variance in the Level-1 slopes
Cov (μ_{0j}, μ_{1j}) = τ_{01} = unconditional covariance between Level-1 intercepts and slopes
Cov (μ_{0j}, μ_{1j}) = τ_{02} = unconditional covariance between Level-1 intercepts and slopes

Cov (μ_{0j}, μ_{1j}) = τ_{03}= unconditional covariance between Level-1 intercepts and slopes
Cov (μ_{0j}, μ_{1j}) = τ_{04} = unconditional covariance between Level-1 intercepts and slopes
Cov (μ_{0j}, μ_{1j}) = τ_{05} = unconditional covariance between Level-1 intercepts and slopes
Cov (μ_{0j}, μ_{1j}) = τ_{06} = unconditional covariance between Level-1 intercepts and slopes
Cov (μ_{0j}, μ_{1j}) = τ_{07} = unconditional covariance between Level-1 intercepts and slopes
Cov (μ_{0j}, μ_{1j}) = τ_{08} = unconditional covariance between Level-1 intercepts and slopes

Level-2 Predictor for Intercepts-as-Outcomes Model

In this model the Level-1 equation is the same as in previous model but SEGLOG or POVLOG is added as a Level-2 predictor of census tract intercepts, after controlling for individual-level characteristics. The other Level-1 coefficients remain fixed.

$$\beta_{0j} = \gamma_{00} + \gamma_{01}\,(\text{SEGLOG})_j + \mu_{0j}$$

$$\beta_{1j} = \gamma_{10} + \mu_{1j}$$

$$\beta_{2j} = \gamma_{20} + \mu_{2j}$$

$$\beta_{3j} = \gamma_{30} + \mu_{3j}$$

$$\beta_{4j} = \gamma_{40} + \mu_{4j}$$

$$\beta_{5j} = \gamma_{50} + \mu_{5j}$$

$$\beta_{6j} = \gamma_{60} + \mu_{6j}$$

$$\beta_{7j} = \gamma_{70} + \mu_{7j}$$

$$\beta_{8j} = \gamma_{80} + \mu_{8j}$$

where, γ_{00} is the grand mean, γ_{10} through γ_{80} is the effect of SEGLOG on β_{1j} through β_{8j}, and μ_{0j} and μ_{1j} through μ_{8j} is a random error assuming to be ~ independently N(0, τ_{00}). Combining the Level-1 and Level-2 structural models yields a combined model:

$$\eta_{ij} = \gamma_{00} + \gamma_{01}\,(\text{SEGLOG})_j + \gamma_{10}\,(\text{MRACE})_j + \gamma_{20}\,(\text{ORIGIN})_j + \gamma_{30}\,(\text{MARITAL})_j +$$
$$\gamma_{40}\,(\text{COLLEGE})_j + \gamma_{50}\,(\text{INSURE})_j + \gamma_{60}\,(\text{SMOKE})_j + \gamma_{70}\,(\text{SUBUSE})_j +$$
$$\gamma_{80}\,(\text{MAGE27})_j + \mu_{0j}$$

where, η_{ij} the probability of low birthweight, is viewed as a function of γ_{00} the grand mean, γ_{01} is the effect of SEGLOG on η_{ij}, γ_{01} through γ_{08} is the effect of individual-level risk factors on η_{ij}, and a random error μ_{0j} assuming to be ~ independently $N(0, \tau_{00})$.

Level-2 Predictor for Intercepts- and Slopes-as-Outcomes Model

In this model the Level-1 equation is the same as in previous two models but SEGLOG is added as a Level-2 predictor of census tract intercepts and slopes.

$$\beta_{0j} = \gamma_{00} + \gamma_{01}\,(\text{SEGLOG})_j + \mu_{0j}$$

$$\beta_{1j} = \gamma_{10} + \gamma_{01}\,(\text{SEGLOG})_j + \mu_{1j}$$

$$\beta_{2j} = \gamma_{20} + \gamma_{02}\,(\text{SEGLOG})_j + \mu_{2j}$$

$$\beta_{3j} = \gamma_{30} + \gamma_{03}\,(\text{SEGLOG})_j + \mu_{3j}$$

$$\beta_{4j} = \gamma_{40} + \gamma_{04}\,(\text{SEGLOG})_j + \mu_{4j}$$

$$\beta_{5j} = \gamma_{50} + \gamma_{05}\,(\text{SEGLOG})_j + \mu_{5j}$$

$$\beta_{6j} = \gamma_{60} + \gamma_{06}\,(\text{SEGLOG})_j + \mu_{6j}$$

$$\beta_{7j} = \gamma_{70} + \gamma_{07}\,(\text{SEGLOG})_j + \mu_{7j}$$

$$\beta_{8j} = \gamma_{80} + \gamma_{08}\,(\text{SEGLOG})_j + \mu_{8j}$$

Combining the Level-1 and Level-2 structural models yields a combined model:

$$\eta_{ij} = \gamma_{00} + \gamma_{01}\,(\text{SEGLOG})_j + \gamma_{10}\,(\text{MRACE})_j + \gamma_{11}\,(\text{SEGLOG})_j(\text{MRACE})_j +$$
$$\gamma_{20}\,(\text{ORIGIN})_j + \gamma_{12}\,(\text{SEGLOG})_j(\text{ORIGIN})_j + \gamma_{30}\,(\text{MARITAL})_j +$$
$$\gamma_{13}\,(\text{SEGLOG})_j(\text{MARITAL})_j + \gamma_{40}\,(\text{COLLEGE})_j + \gamma_{14}\,(\text{SEGLOG})_j\,(\text{COLLEGE})_j +$$
$$\gamma_{50}\,(\text{INSURE})_j + \gamma_{15}\,(\text{SEGLOG})_j(\text{INSURE})_j + \gamma_{60}\,(\text{SMOKE})_j +$$
$$\gamma_{16}\,(\text{SEGLOG})_j(\text{SMOKE})_j + \gamma_{70}\,(\text{SUBUSE})_j + \gamma_{17}\,(\text{SEGLOG})_j\,(\text{SUBUSE})_j +$$
$$\gamma_{80}\,(\text{MAGE27})_j + \gamma_{18}\,(\text{SEGLOG})_j(\text{MAGE27})_j + \mu_{0j}$$

where, γ_{00} is the grand mean, γ_{01} is the effect of SEGLOG on η_{ij}, γ_{10} through γ_{80} is the effect of individual-level risk factors on η_{ij}, and (SEGLOG)(e.g., individual-level variables) is the effect of the interaction term on $\eta_{ij,}$ with a random effect μ_{0j} and μ_{1j} through μ_{8j} assuming to be ~ independently N(0, $\tau_{00,}\tau_{11}$). The interaction term measures the degree to the association between individual-level risk factors and low birthweight vary according to the level of segregation in the tract.

Unit-Specific Models

There are two models that can be measured in HGLM: unit-specific models and population-average models. The difference between the two models is how the random effects are interpreted. In unit-specific models the coefficients are interpreted as the expected difference in the log-odds of low birthweight associated with a unit-increase in the predictor, holding constant the other predictors *and* holding constant the value of the random effect μ_{0j}. In population-average models the coefficients are interpreted as the expected difference in the log-odds of low birthweight associated with a unit-increase in the predictor, holding constant the other predictors *but* averaging over the distribution of Level-2 random effects. These population-average coefficients are "shrunk" toward zero. This study will interpret the unit-specific model and will be referred to as a census tract-specific model. The census tract-specific model describes the levels of low birthweight for each census tract and how the effect of individual-level risk factors on low birthweight varies by the degree of residential segregation.

Residuals and Model Checking

The output of the HGLM program includes (a) Mahalanobis distance measures useful in assessing the multivariate normality assumption of the Level-2 residuals, and (b) ordinary least squares (OLS) and empirical Bayes (EB) estimates of Level-2 residuals (discrepancies between Level-1 coefficients and fitted values). These measures and residuals were used to assess the adequacy of the HGLM models.

Background:

In general, the accuracy of ordinary least squares (OLS) regression estimates for any census tract depends on the sample size (n_j) and the range in the Level-1 predictor. If n_j is small, the mean estimate β_{0j}, may be imprecise. If there is a restricted range on a Level-1 predictor, the slope estimate, β_{1j}, may also be imprecise. EB estimates of each census tract's regression line take into account imprecision in the OLS estimates by using a "shrinkage" factor.

β_{0j} can be predicted from the LBW mean in the Level-1 model:

$$\eta_{ij} = \beta_{0j} + \beta_{1j}(\text{MRACE})_{ij} + \beta_{2j}(\text{ORIGIN})_{ij} + \beta_{3j}(\text{MARITAL})_{ij} + \beta_{4j}(\text{COLLEGE})_{ij} + \beta_{5j}(\text{INSURE})_{ij} + \beta_{6j}(\text{MAGE27})_{ij} + \beta_{7j}(\text{SMOKE})_{ij} + \beta_{8j}(\text{SUBUSE})_{ij}$$

or β_{0j} can be predicted from the Level-2 model given SEGLOG:

$$\beta_{0j} = \gamma_{00} + \gamma_{01}(\text{SEGLOG})_j + \mu_{0j}$$

When these two estimators are combined the composite estimate is referred to as an optimal or "weighted" combination or an EB estimate β^*_{0j}. Referring back to a linear

regression model, the EB estimate is equal to the sample mean plus a shrinkage factor times the OLS regression estimate minus the sample mean.

$$\beta^{*}_{0j} = \bar{\gamma} + REL_1(OLS_i - \bar{\gamma})$$

The shrinkage factor is

$$REL_1 = \frac{\tau}{\tau + \frac{\sigma^2}{n_1}}$$

Where τ = the Level-2 variance, σ^2 is the Level-1 variance, and n_1 is the number of women in each census tract.

When the sample mean is a highly reliable estimate, β^{*}_{0j}, puts substantial weight on the sample mean, η_{ij}. If the sample mean is unreliable, the estimated grand mean, γ_{00}, will put more weight on composing, β^{*}_{0j}.

Corresponding to OLS and EB estimates are OLS and EB residuals.

The OLS residuals for the intercepts and slopes are:

$$\mu_{0j} = \beta_{0j} - [\, \gamma_{00} + \gamma_{01} (SEGLOG)_j \,]$$

$$\mu_{0j} = \beta_{1j} - [\, \gamma_{10} + \gamma_{11} (SEGLOG)_j \,]$$

OLS residuals are estimates of the deviation of β_{0j} or β_{1j} from the grand-mean regression line of γ_{00} and γ_{01} or γ_{10} and γ_{11}.

The EB residuals are:

$$\mu^{*}_{0j} = \beta^{*}_{0j} - [\, \gamma_{00} + \gamma_{01} (SEGLOG)_j \,]$$

$\mu^*_{0j} = \beta^*_{1j} - [\ \gamma_{10} + \gamma_{11}\ (SEGLOG)_j\ ]$

EB residuals are estimates of the deviation of β^*_{0j} or β^*_{1j} from a predicted value and in this example is based on SEGLOG.

From these two models μ^*_{0j} is a value of μ_{0j}, "shrunk" toward 0:

$$\mu^*_{0j} = REL\mu_{0j}$$

Thus, if the reliability (REL) is unity, no shrinkage occurs. In contrast if REL is zero, shrinkage toward the predicted value is complete.

This analysis checked the OLS and EB residuals to identify uncommon characteristics of census tracts (e.g., outliers) or unexplained variance in the model.

Examining the Weathering Hypothesis

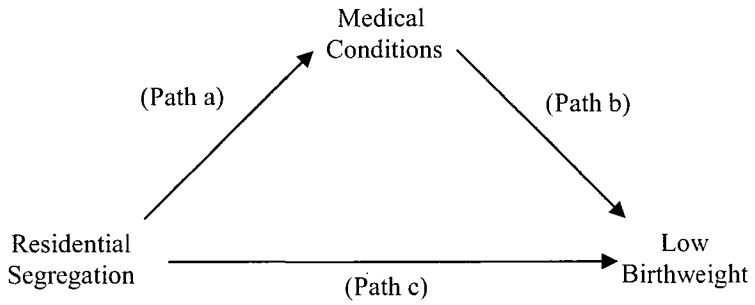
Plots of the log-odds of medical conditions by age were constructed for African American versus white women. These plots provided information on racial disparities in medical conditions over the course of the reproductive years to evaluate if African American women experienced early health deterioration, evidence of the weathering hypothesis. Only those medical conditions that were significantly associated with low birthweight in the logistic regression analysis were plotted.

Testing for Mediating Effects

Testing for mediating effects will answer the question of why an association exists between residential segregation and low birthweight. In this dissertation it is hypothesized that residential segregation will be associated with low birthweight through

mechanisms involving health deterioration, as evidenced by an increase in the number of medical conditions women.

This dissertation will adopt Barron and Kenny's (1986) model to test for mediating effects. It involves a three-variable system with two causal paths feeding into the outcome variable: the direct impact of the independent variable (Path c) and the impact of the mediator (Path b). There is also a third path from the independent variable to the mediator (Path a). In this dissertation Path c represents the impact of residential segregation on low birthweight; Path b represents the impact of medical conditions on low birthweight; and Path a suggests that the stress of living in residential segregation increases the risk of medical conditions. These in turn increase the risk of low birthweight.



To test for mediation, the three following regression equations were estimated: first, the effect of residential segregation on medical conditions was estimated; second, the effect of medical conditions on low birthweight was estimated; and third, the effect of both residential segregation and medical conditions on low birthweight was measured. These models were estimated while controlling for individual level risk factors, specifically, origin of birth, marital status, college education, and Medicaid status. To establish

mediation, the following conditions must hold: first, residential segregation must have an effect on medical conditions in the first equation; second, medical conditions must have an effect on low birthweight in the second equation; and third, medical conditions must have an effect on low birthweight in the third equation. If these conditions all hold in the predicted direction, then the effect of residential segregation on low birthweight in the third equation (Path c') must be less than in the second equation (Path c). The reason that residential segregation might decrease in Path c' is because of the indirect effect of medical conditions explains it away.

To know whether the decrease in residential segregation was significant the following significance test was conducted. The product of Paths a and b was calculated (because c – c' measure the indirect effect of residential segregation on low birthweight) and divided by the error term described by Sobel (1982) and Frazier et al (2004):

$$\sqrt{b^2 s_a^2 + a^2 s_b^2 + s_a^2 s_b^2}$$

The use of regression analysis to estimate a mediational model requires two assumptions: (1) that there be no measurement error in the mediator, and (2) that the dependent variable does not cause the mediator. It was recognized that because a variety of medical conditions were being tested, that certain medical conditions may have a stronger mediating effect than others, and some may not have an effect at all. It is the purpose of this research to identify those medical conditions that do have a significant mediated effect on the residential segregation and low birthweight relationship.

The results from these analyses are presented in Chapters 4 and 5. In Chapter 4 the descriptive analyses are reported. In Chapter 5 the analytical analyses are outlined and interpreted.

CHAPTER 4:

CHARACTERISTICS OF MOTHERS AND INFANTS AND THE ENVIRONMENTS IN-WHICH THEY LIVE

Chapter 4 reports on the descriptive findings of this analysis and offers an interpretation of the results.

4.A. DESCRIPTIVE OVERVIEW

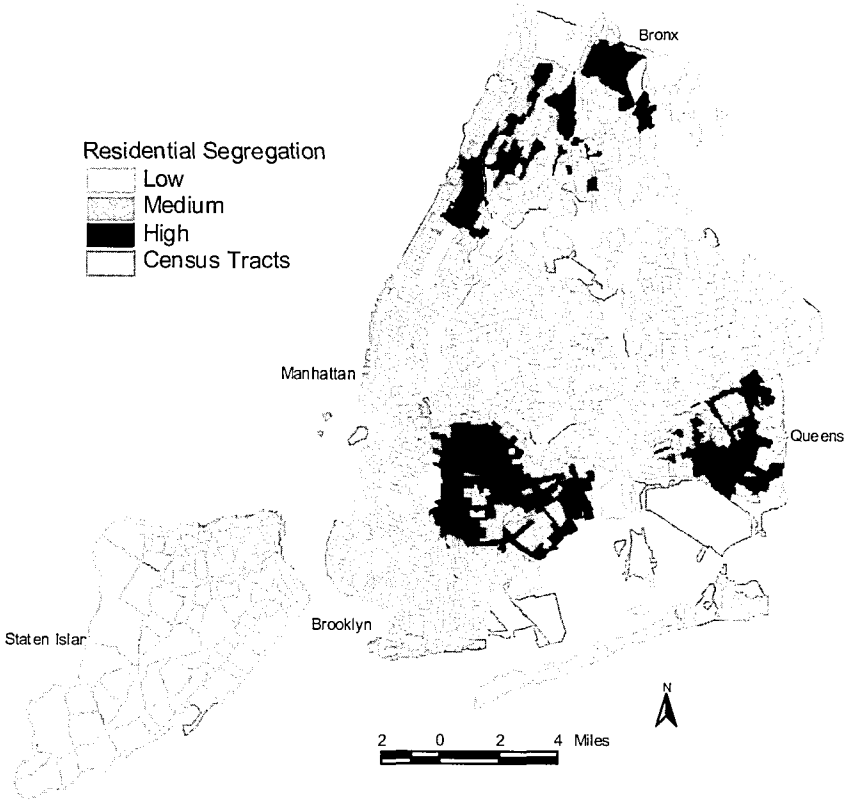
The descriptive portion of this chapter is divided into four parts. The first part describes the geographic distribution of residential segregation and poverty in New York City. The second part examines the demographic characteristics of mothers, focusing on race, ethnic background and socio-economic characteristics. In the third section, variations in birth weight and birth weight by gestational age are analyzed by race and origin of birth. The final section explores geographic place variations in reproductive outcomes.

Residential Segregation

In this study residential segregation refers to predominately black neighborhoods. In 2000, there were 229 (10.32%) highly segregated census tracts in New York City. High segregation was determined as those census tracts with a segregation index greater than or equal to 0.6. This cut-off is commonly used in the literature to measure highly segregated areas at the metropolitan level. The highly segregated tracts were primarily concentrated in Northern Manhattan in Central and East Harlem and Washington Heights, and in the area of West Bronx in Concourse, Melrose and

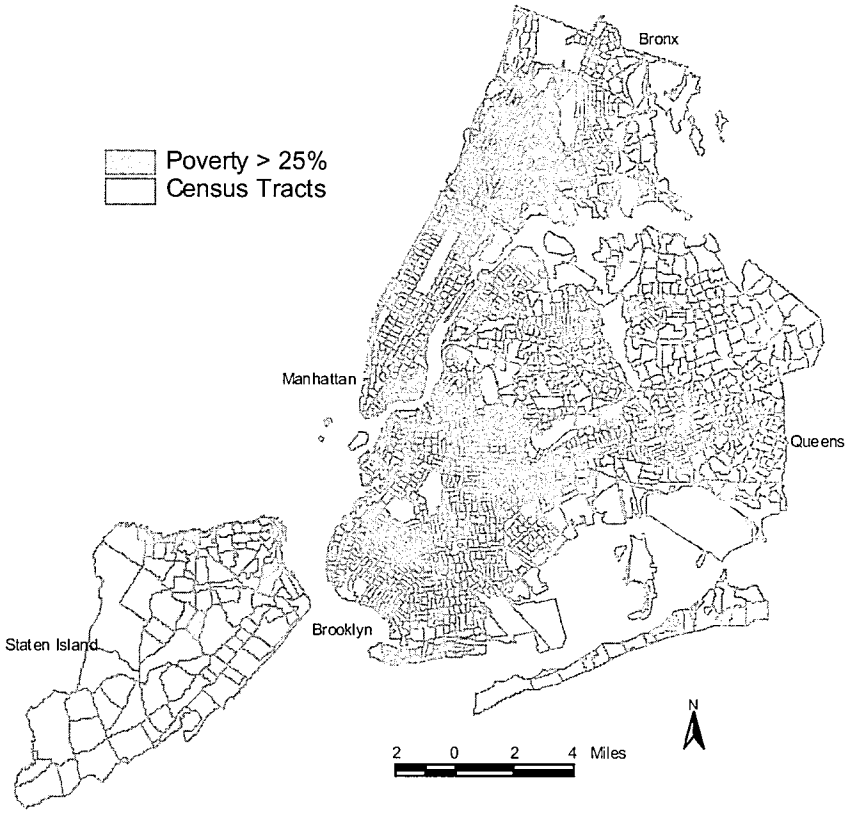
Mott Haven. There were also a large concentration of segregated census tracts in central Brooklyn in Prospect Heights, Crown Heights, Bedford-Stuyvesant, Stuyvesant Heights, Weeksville, Wingate, and Brownsville. Directly east of this area in central Brooklyn were a large number of segregated census tracts in southeast Queens in the areas of Belrose, Queens Village, Bellaire, Saint Albans, Cambria Heights, Springfield Gardens, and Laurelton. In Staten Island there were only a few segregated census tracts in the northeast (Figure 5).

Figure 5: Residential Segregation by Census Tracts, New York City, 2000



The spatial distribution of poverty was somewhat different from that of black residential segregation. Many more census tracts had high poverty than high black segregation. There were 704 (31.75%) census tracts with at least 25 percent of families with a ratio of income to poverty 1.0 or less. These census tracts were generally located within and surrounding areas of residential segregation. One exception was in Brooklyn where a substantial number of high poverty census tracts were located southwest of the segregated area in Sunset Park, Borough Park, Prospect Park South, Kensington, and Parkville. Demographic data show that these communities were comprised primarily of poor whites. In contrast, the southwest area of Queens was highly segregated but had very low poverty rates. There were also areas of high poverty in the lower east- side of Manhattan and northern Manhattan, areas that had large concentrations of Hispanic population. In Staten Island there were a few high poverty areas along the north coast separated from segregated areas. The highest poverty areas were located throughout south and central Bronx (Figure 6).

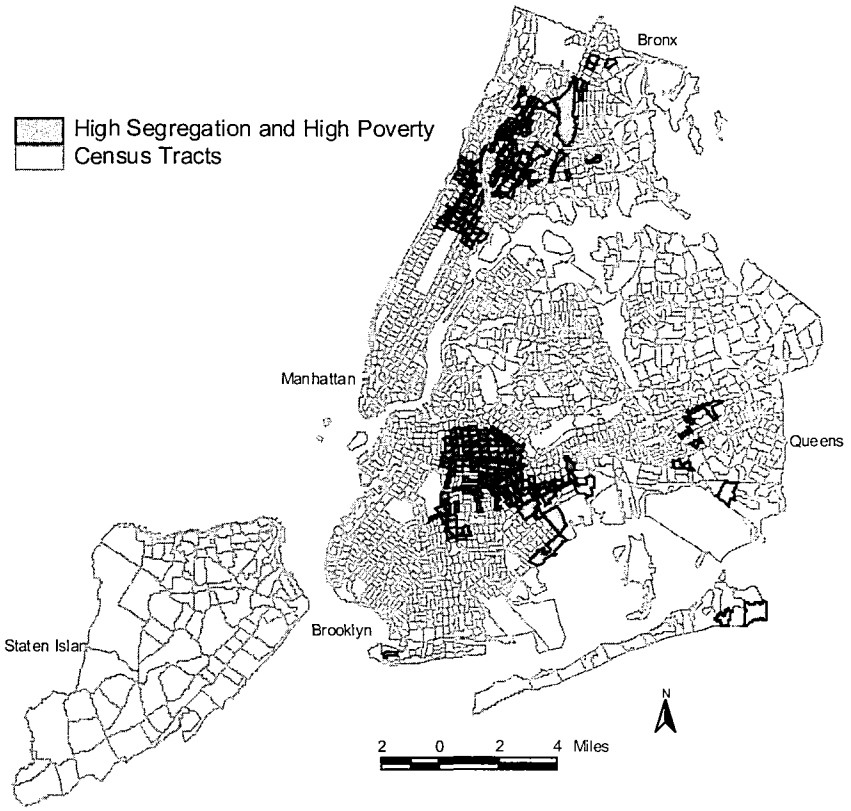
Figure 6: Poverty by Census Tracts, New York City, 2000



* Poverty Indicates Percentage of Families with a Ratio of Income to Poverty 1.0 or less

When the maps of high residential segregation and poverty were combined, specific areas of concentrated poverty and segregated black population emerged (Figure 7). These areas were primarily located in northern Manhattan (Central and East Harlem and Washington Heights), West Bronx (Concourse, Melrose and Mott Haven community), and Central Brooklyn (Prospect Heights, Crown Heights, Bedford-Stuyvesant, Stuyvesant Heights, Weeksville, Wingate, and Brownsville). There were very few census tracts in Queens that were both racially segregated and poor.

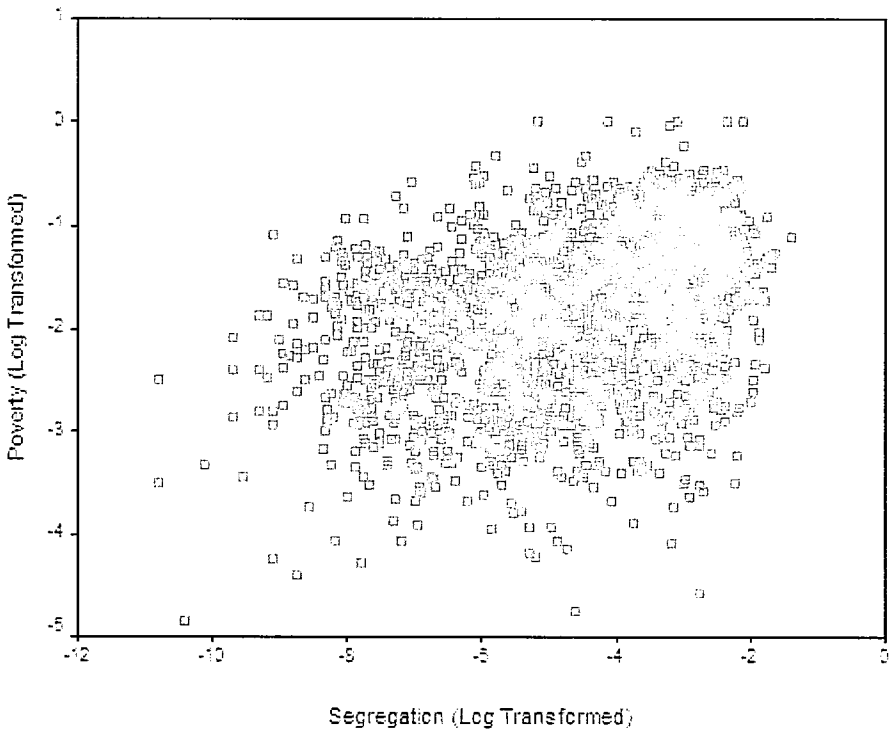
Figure 7: Residential Segregation and Poverty, New York City, 2000



Correlation Between Segregation and Poverty

There was a positive correlation between residential segregation and poverty (Pearson Correlation = 0.307, statistically significant at the 0.01 level). A plot of residential segregation by poverty log transformed is provided in Figure 8.

Figure 8: Scatterplot of Segregation and Poverty, New York City, 2000



With increasing residential segregation there was also an increase in poverty, but the association was not perfect. Some high poverty census tracts were located in low segregated areas, but a substantial number of high poverty census tracts were also located in areas with high residential segregation. When segregation indices and poverty percentages were plotted as histograms both datasets were skewed to the right (Figures 9 and 10); therefore, this data was log transformed (base e) for analysis (Figures 11 and 12).

Figure 9: Histogram of Residential Segregation

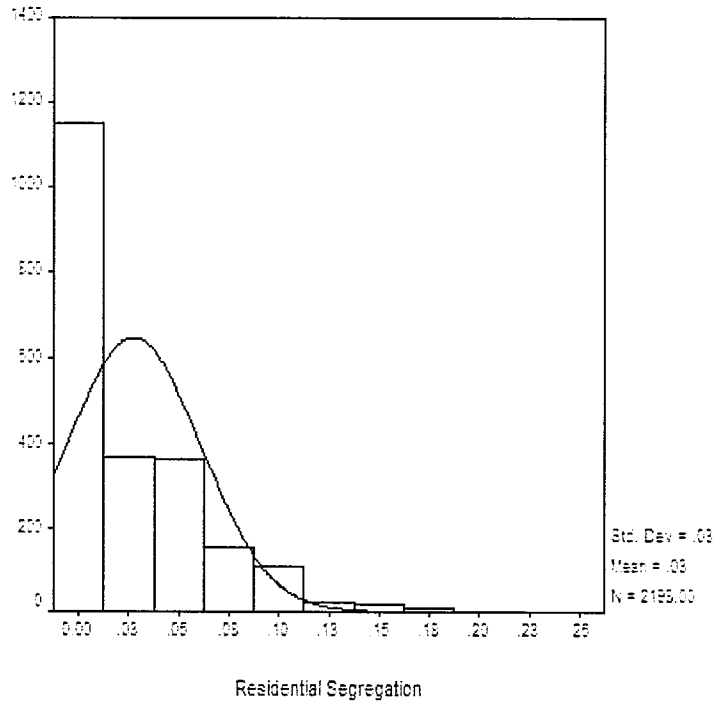


Figure 10: Histogram of Poverty

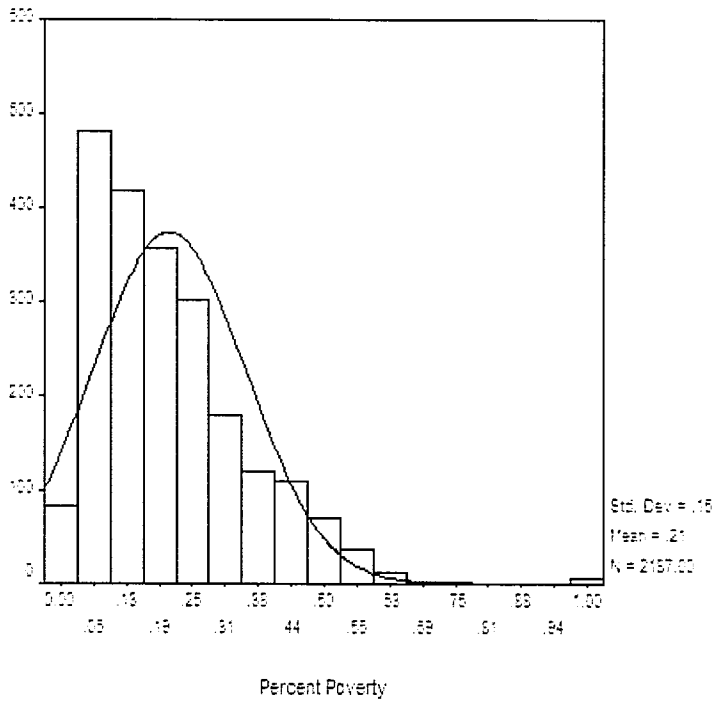


Figure 11: Histogram of Residential Segregation Log Transformed

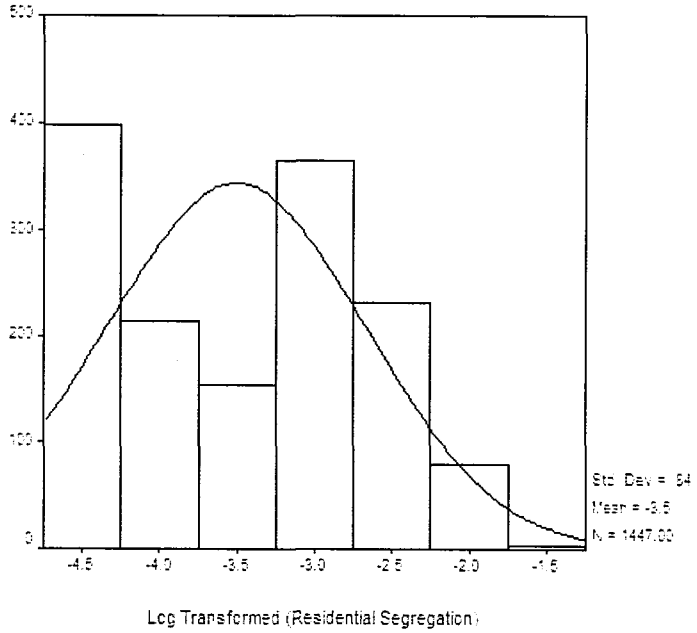
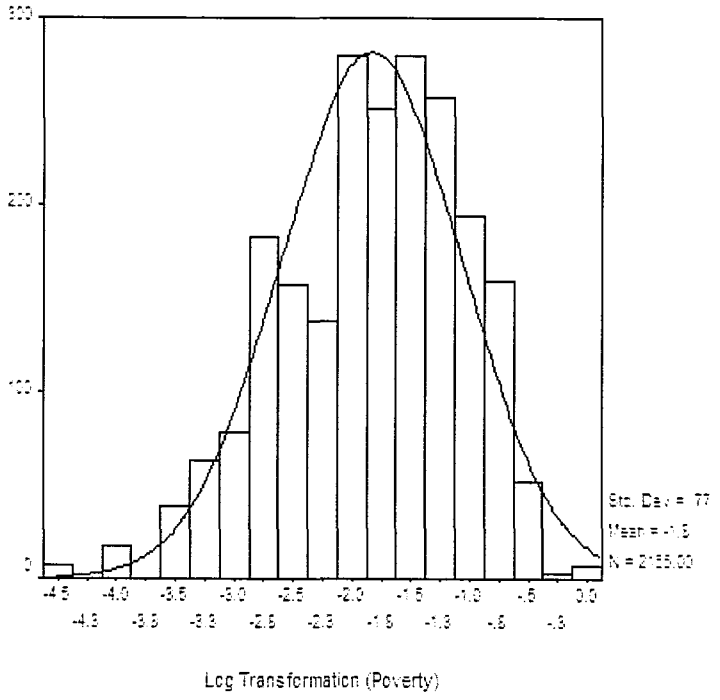


Figure 12: Histogram of Poverty Log Transformed



Demographic Characteristics of Mothers

This study of racial disparities in low birthweight requires that women be categorized by race and origin of birth. There are however, many other important characteristics that describe the women in this study. Women who gave birth in New York City in 2000 were diverse, not only in terms of race and ethnicity, but also in their social, economic, and demographic characteristics. Tables 3 and 4 describe the characteristics of mothers, first for African American and white mothers and then for African American mothers only, disaggregated into U.S.-born and foreign-born groups.

Table 3: Descriptive Characteristics of African American and White Women, New York City, 2000

	African American		White Women	
Characteristics	**No.**	**(%)**	**No.**	**(%)**
Foreign-Born	36498	51.0	60110	50.0
Married	36638	30.0	60244	59.0
College	36287	36.0	59429	43.0
Smoking	36605	5.0	60213	3.0
Alcohol	36568	1.0	60195	0.0
Substance use	36638	2.0	60244	0.1
Medicaid	36257	69.0	60064	52.0
Age (mean)	35775	27.2	60244	28.1

African American women's mean age at pregnancy was 27.2 years, one year less than white women. Slightly more than half of African American women were foreign-born. Twice as many white women were married as African American women, with 70 percent of African American women unmarried at the time they gave birth. White women were a little more likely than African American women to attend college. Although both groups were likely to participate in unhealthy behaviors, a greater percentage of African American women reported that they smoked or used substances

during their pregnancy. About two-thirds of African American women were enrolled in Medicaid, compared to half of white women.

Table 4: Descriptive Characteristics of U.S.-Born and Foreign-Born African American Women, New York City, 2000

	U.S.-Born African American		Foreign-Born African-American	
Characteristics	**No.**	**(%)**	**No.**	**(%)**
Married	17741	20.0	18757	40.0
College	17618	36.0	18574	35.0
Smoking	17716	9.0	18750	1.0
Alcohol	17684	2.0	19854	0.0
Substance use	17741	3.0	18754	0.0
Medicaid	17598	64.4	18528	72.0
Age (mean)	17741	25.7	18757	28.6

The comparison of U.S.-born and foreign-born African-American, women reveals important differences. U.S.-born African American women gave birth at a younger age (25.7 years) than foreign-born African American women (28.6 years). They were also substantially less likely to be married (20 vs. 40 percent) but were equally as likely to have attended college. U.S-born African American women were more likely to participate in unhealthy behaviors than foreign-born African American women (smoking, 9.0 vs. 1.0) and (substance use, 3.0 vs. 0.0). Foreign-born women were more likely to be enrolled in Medicaid. Medicaid provides health care coverage for low-income persons, so the differences in Medicaid percentage suggest that foreign-born African-American women were more likely to be low-income than U.S.-born women.

Table 5 reports on the percentage of medical conditions that African American and white women experienced while pregnant. African American women were more likely to have all medical conditions except renal disease, thyroid disease, tuberculosis, hydramnios/oligohydramnios and uterine bleeding in all three trimesters.

Table 5: Percentage of Medical Conditions in Women by Race, New York City, 2000

Pre-Pregnancy Medical Conditions	African American Women		White Women		
	N	Percent	N	Percent	Difference
Anemia	36638	2.342	60244	1.315	1.027
Cardiac disease	36638	0.415	60244	0.161	0.254
Lung disease	36638	2.178	60244	1.635	0.543
Diabetes, chronic	36638	0.456	60244	0.287	0.169
Hypertension, chronic	36638	1.471	60244	0.629	0.842
Renal disease	36638	0.202	60244	0.252	-0.050
Hepatitis	36638	0.546	60244	0.312	0.234
Thyroid condition	36638	0.366	60244	0.759	-0.393
Genital herpes	36638	0.737	60244	0.609	0.128
Other STDs	36638	2.205	60244	0.666	1.539
Rubella	36638	2.123	60244	0.231	1.892
Tuberculosis	36638	0.038	60244	0.139	-0.101
Viral disease	36638	0.218	60244	0.100	0.118
Previous SGA	36638	0.674	60244	0.483	0.191
Previous miscarriage	36638	0.191	60244	0.189	0.002
Pregnancy-Related Medical Conditions					
Diabetes, pregnancy related	36638	3.857	60244	3.184	0.673
Hydramnios/Oligohydramnios	36638	2.798	60244	3.046	-0.248
Hypertension, pregnancy related	36638	1.946	60244	1.295	0.651
Preeclampsia	36638	2.901	60244	2.171	0.730
Eclampsia	36638	0.079	60244	0.060	0.019
Incompetent cervix	36638	0.540	60244	0.269	0.271
Uterine bleeding 1st trimester	36638	0.267	60244	0.483	-0.216
Uterine bleeding 2nd trimester	36638	0.120	60244	0.163	-0.043
Uterine bleeding 3rd trimester	36638	0.150	60244	0.173	-0.023

The most common pre-pregnancy medical conditions in African American women were anemia, STDs, lung disease, rubella, and chronic hypertension. Anemia and lung disease were the two most frequently reported medical conditions in white

women but their overall rate was much lower than in African American women. Pregnancy-related diabetes was the most common pregnancy-related medical condition in both African American and white women but the rate was higher in African American women. African American women also had higher rates of pregnancy-related hypertension, preeclampsia, eclampsia, and incompetent cervix.

This descriptive analysis shows that African American women had higher rates of almost all pre-pregnancy and pregnancy-related medical conditions compared to white women and suggests the presence of *weathering* in the African-American population. Statistical analyses were conducted to validate this hypothesis and are presented later in this dissertation.

Characteristics of Infants

Descriptive statistics on infant characteristics for African American and white women are presented in Table 6.

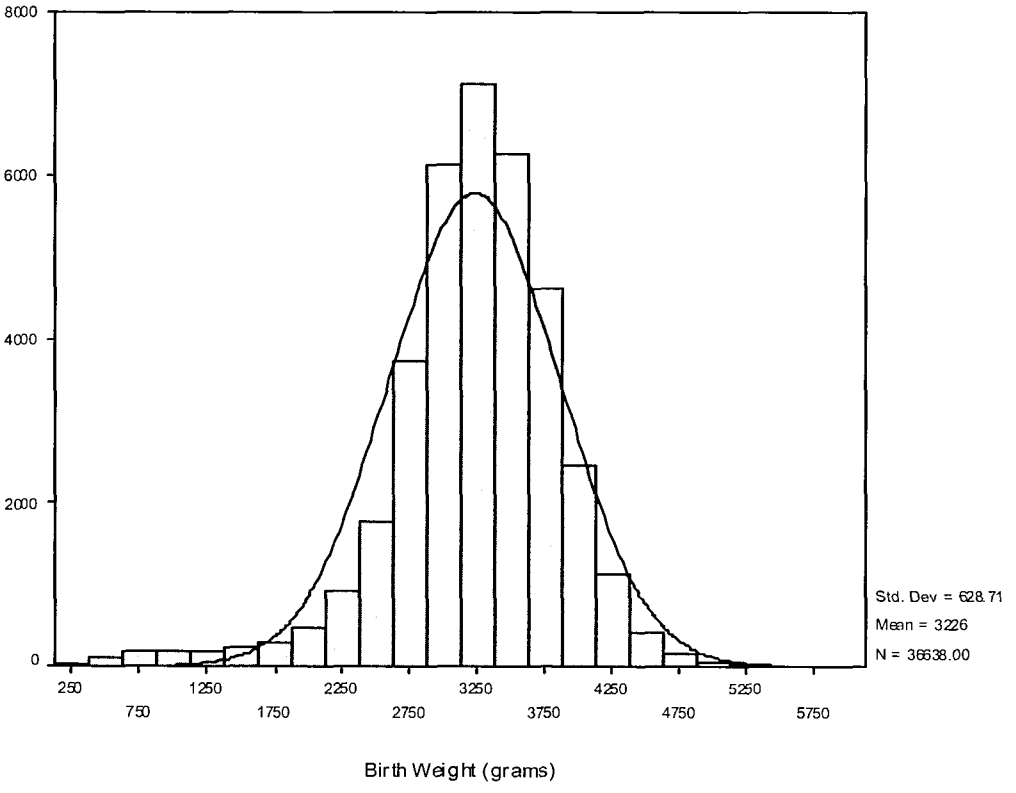
Table 6: Descriptive Statistics for Infants of African American and White Women, New York City, 2000

	African American Women		**White Women**	
Birth Outcome	**No.**		**No.**	
Birth weight (grams)	36638	3225.7	60244	3352.6
Gestation (weeks)	35145	38.4	60244	40.1
	No.	**(%)**	**No.**	**(%)**
Low birthweight	36638	9.0	50244	5.0
Premature	36638	6.0	60244	3.0
IUGR	36638	2.7	60244	1.7
Male sex	36638	49.0	60244	49.0

The mean birth weight for African American infants was 3225 grams (range, 142 to 5925 grams). The mean birth weight for white infants was 3352 grams (range, 198 to 5870 grams). Thus, African American infants were on average 127 grams lighter than white infants. They also experienced higher rates of low birthweight (9.0 vs. 5.0), premature birth (6.0 vs. 3.0), and IUGR (2.7 vs. 1.7). The sex ratio as indicated by the percentage of male births was comparable between the two groups of women.

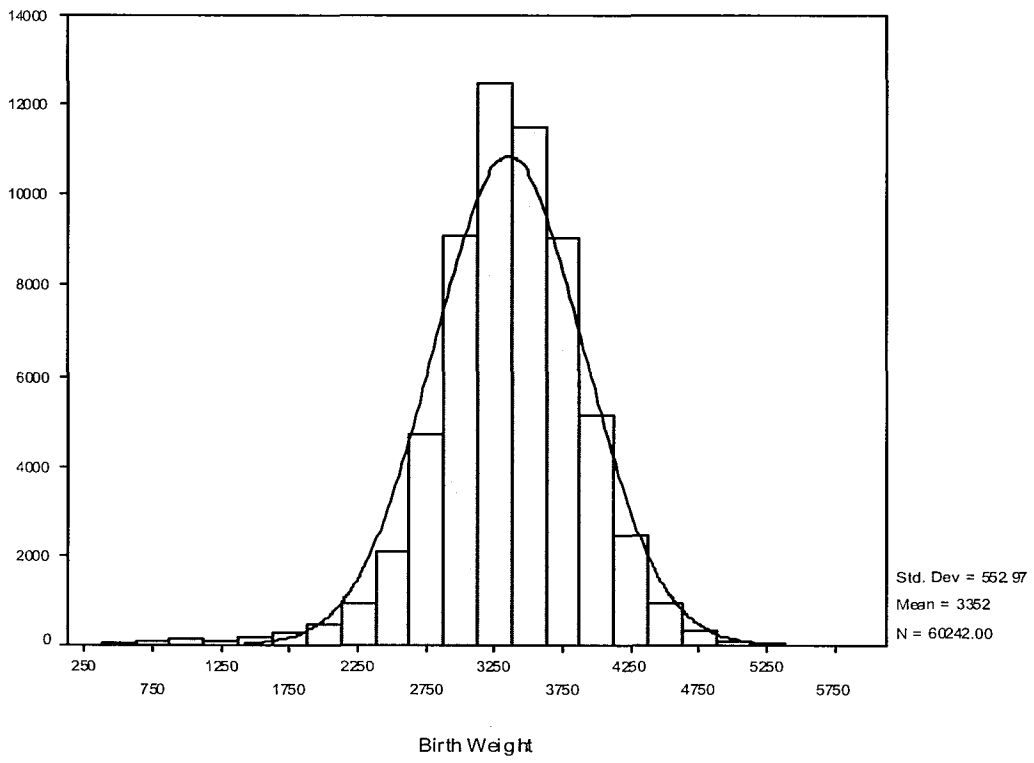
The histograms of birth weight show that infants of African American and white women were normally distributed around the mean. Infants of African American women had a slightly lower peak than infants of white women, because there were fewer births. There was also a wider tail at the lower end of the birth weight scale, indicating that African American women had a greater number of low birthweight births (Figures 13 and 14).

Figure 13: Histogram of Birth Weight for African American Infants



When infant birth weight was plotted by gestational age, there were three white infants with a birth weight greater than or equal to 6000 grams or 13.3 pounds. These records were considered outliers and removed from the dataset. In addition, there were a substantial number of records with very high gestational ages for both races. Outliers above 45 weeks (equal to approximately 315 days or 49 days past normal gestation) were also removed from the dataset for this descriptive analysis. Scatterplots showing African American and white infant birth weight by gestational age, after removing outliers, are presented in Figures 15 and 16. The line on the horizontal axis marks 37 weeks of gestation and the line on the vertical axis marks 2,500 grams. Low birthweight infants fall below the horizontal axis. Premature infants fall below the horizontal axis and to the left of the vertical axis. IUGR infants

Figure 14: Histogram of Birth Weight for White Infants



fall below the horizontal axis and to the right of the vertical axis. In general, there appears to be a greater number of low birthweight births, premature births, and IUGR infants among African American women, a finding supported by the histogram of African American infants. These descriptive statistics highlight the racial disparity in low birthweight in New York City.

Figure 15: Scatterplot of Birth Weight by Gestational Age for African American Infants

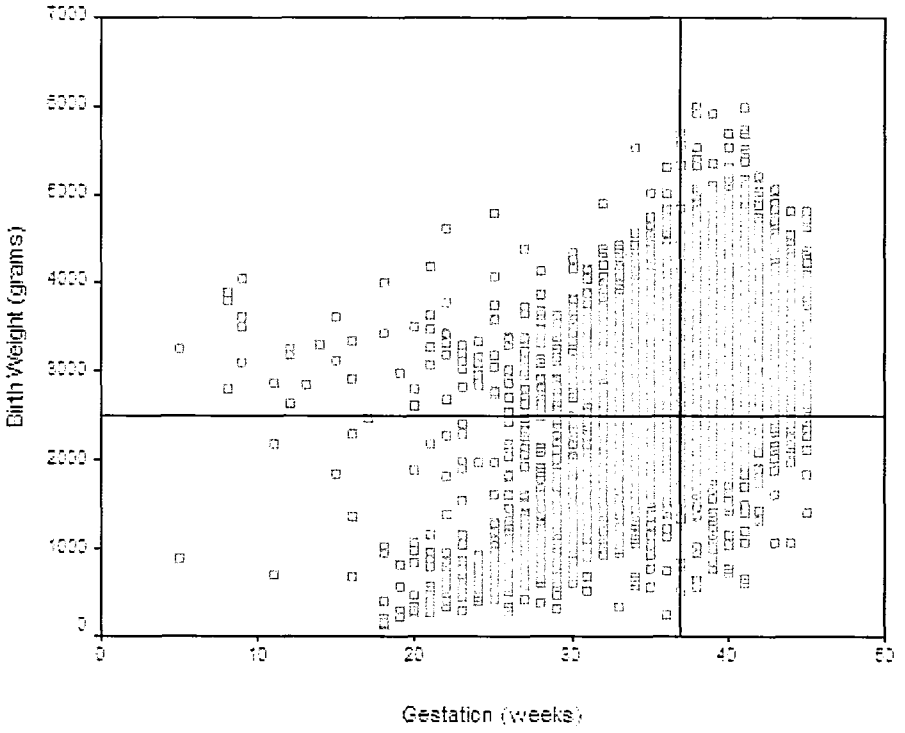


Figure 16: Scatterplot of Birth Weight by Gestational Age for White Infants

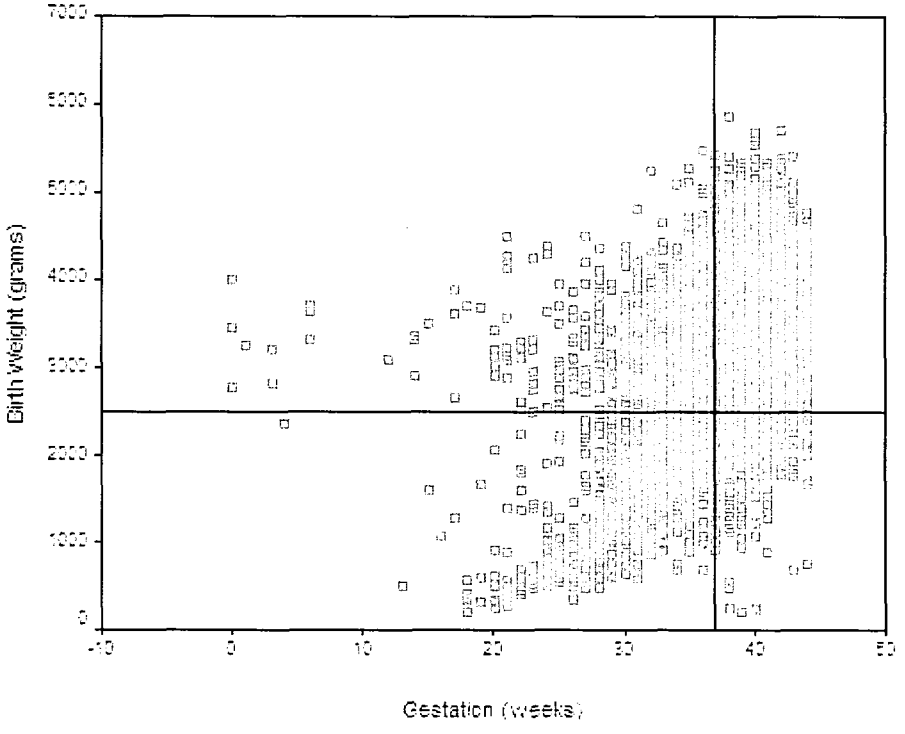


Table 7 highlights information on infants and birth outcome for U.S.-born and foreign-born African American women. U.S.-born African American infants on average had a mean birth weight of 3180 grams (range, 142 to 5760 grams). The average birth weight for foreign-born African American infants was 3270 grams (range, 165 to 5925 grams). Thus, U.S.-born African American infants were on average 90 grams lighter than foreign-born African American infants. U.S.-born African American mothers were more likely than foreign-born mothers to have low birthweight infants (11.0 vs. 8.0), premature infants (6.0 vs. 5.0), and IUGR infants (3.3 vs. 2.1). The sex ratio was similar for U.S.-born and immigrant African American women.

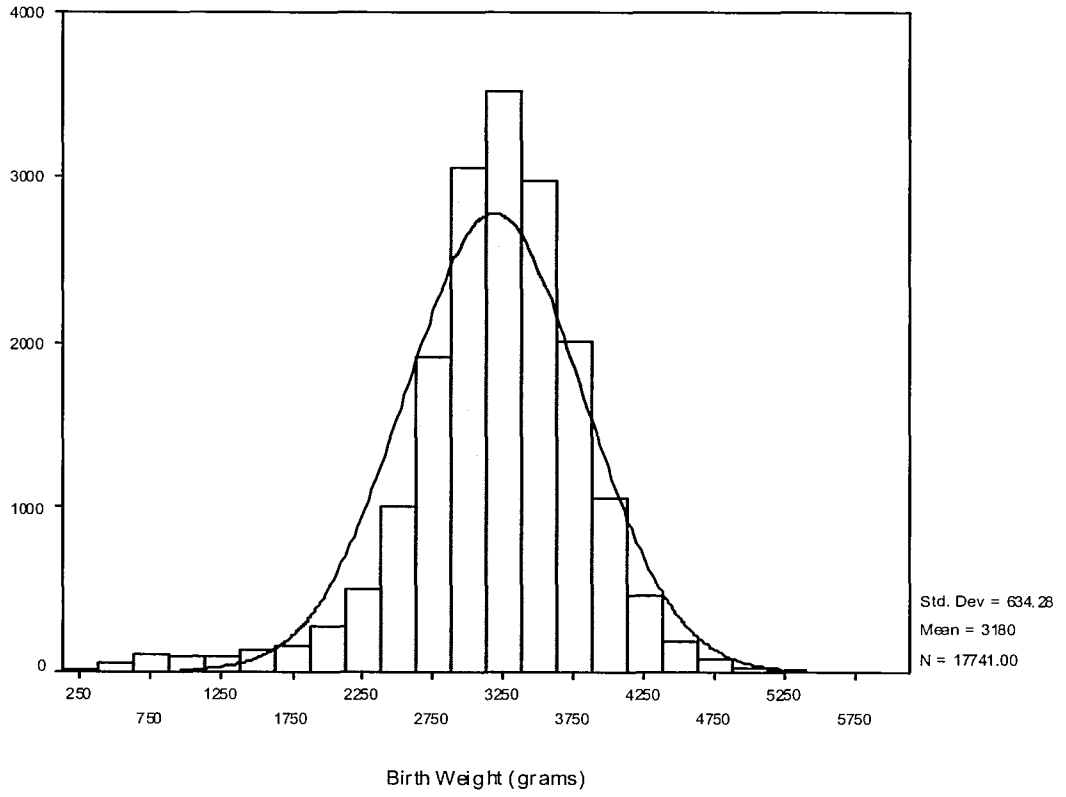
Table 7: Descriptive Statistics for Infants of U.S.-Born and Foreign-Born African American Women, New York City, 2000

	U.S.-Born African American		**Foreign-Born African American**	
Birth Outcome	**No.**		**No.**	
Birth weight (grams)	17741	3180.0	18757	3270.9
Gestation (weeks)	17741	40.2	18757	40.0
	No.	**(%)**	**No.**	**(%)**
Low birthweight	17741	11.0	18757	8.0
Premature	17741	6.0	18757	5.0
IUGR	17741	3.3	18757	2.1
Male sex	17741	49.0	18757	49.0

The histograms of birth weight also differed (Figures 17 and 18). The histogram for infants of U.S.-born mothers had a lower peak as indicated by fewer births. Both groups had a negatively skewed distribution but the tail for U.S.-born African American women was greater than that for foreign-born women. These findings showed that U.S.-born African American women had a higher proportion of low

birthweight births.

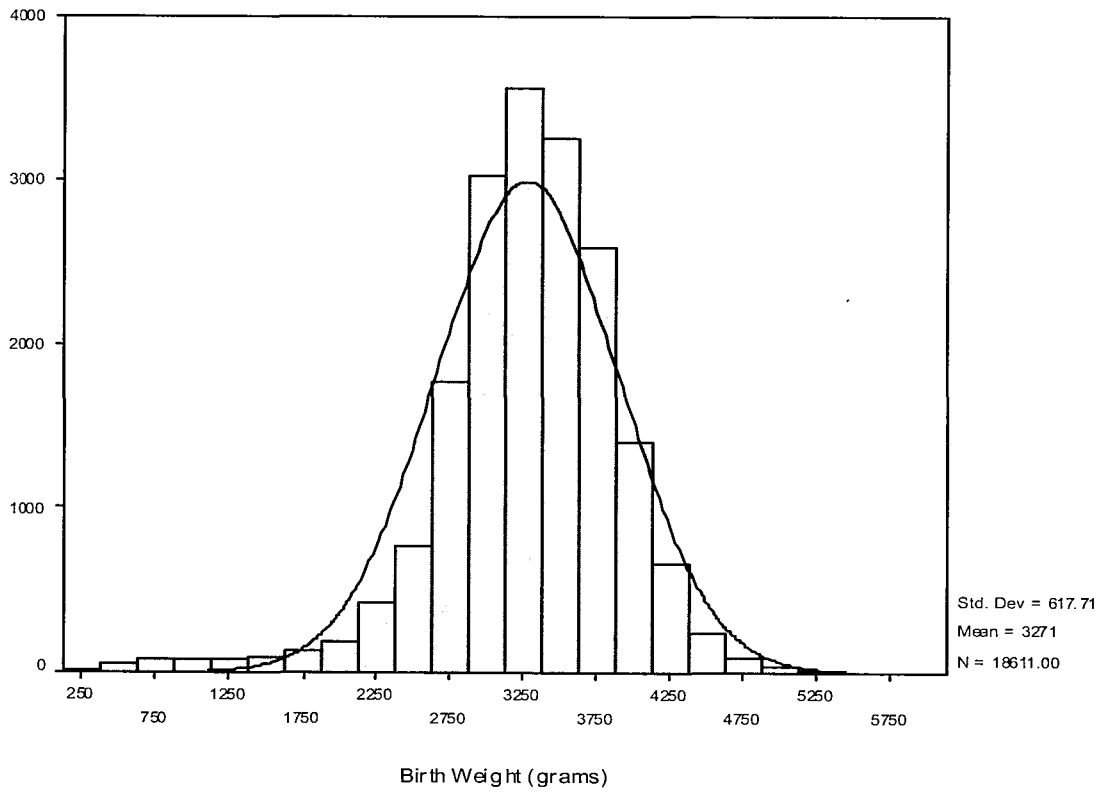
Figure 17: Histogram of Birth Weight for U.S.-Born African American Infants



The scatterplots of birth weight by gestational age for U.S.-born and foreign-born African American women (Figures 19 and 20) showed a greater number of low birthweight births, premature births, and IUGR infants. It appears that the difference in the number of low birthweight births in these two groups of women was due to IUGR and to a lesser extent premature birth.

These descriptive statistics on infant characteristics and birth outcomes demonstrated substantial racial disparity in low birthweight in New York City, with African American women at higher risk than white women. When African American women were stratified by origin of birth, U.S.-born women were at greater risk of low

Figure 18: Histogram of Birth Weight for Foreign-Born African American Infants



birthweight than foreign-born women. Although foreign-born African American women appear to be the poorest group of women as evidenced by Medicaid status, U.S.-born African American women appear to have the worst profile of birth outcomes among all population groups.

Figure 19: Scatterplot of Birth Weight by Gestational Age for U.S.-born African American Women

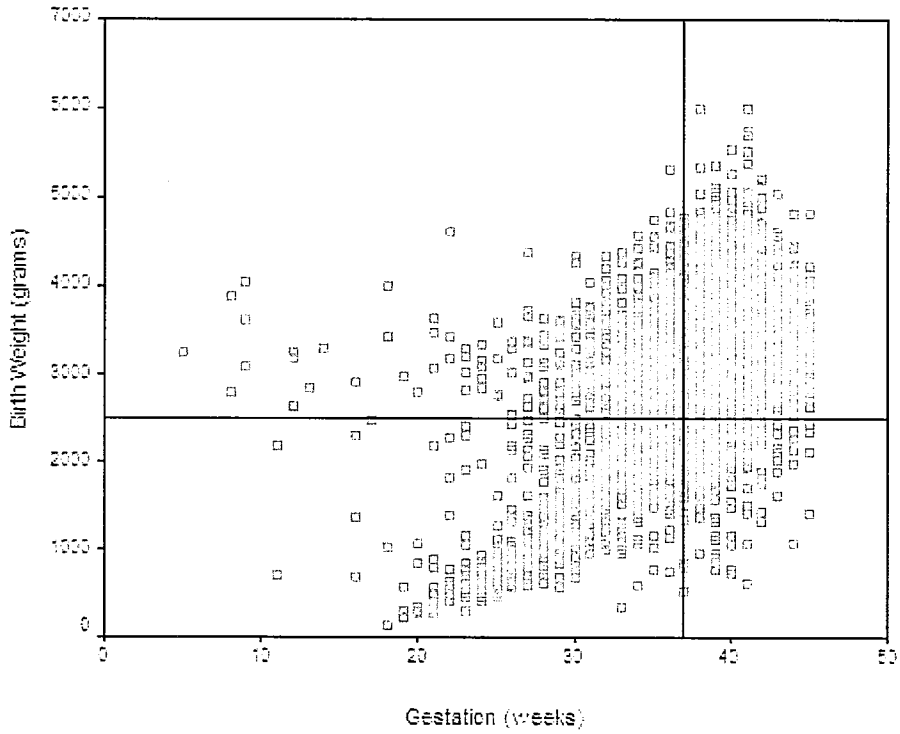
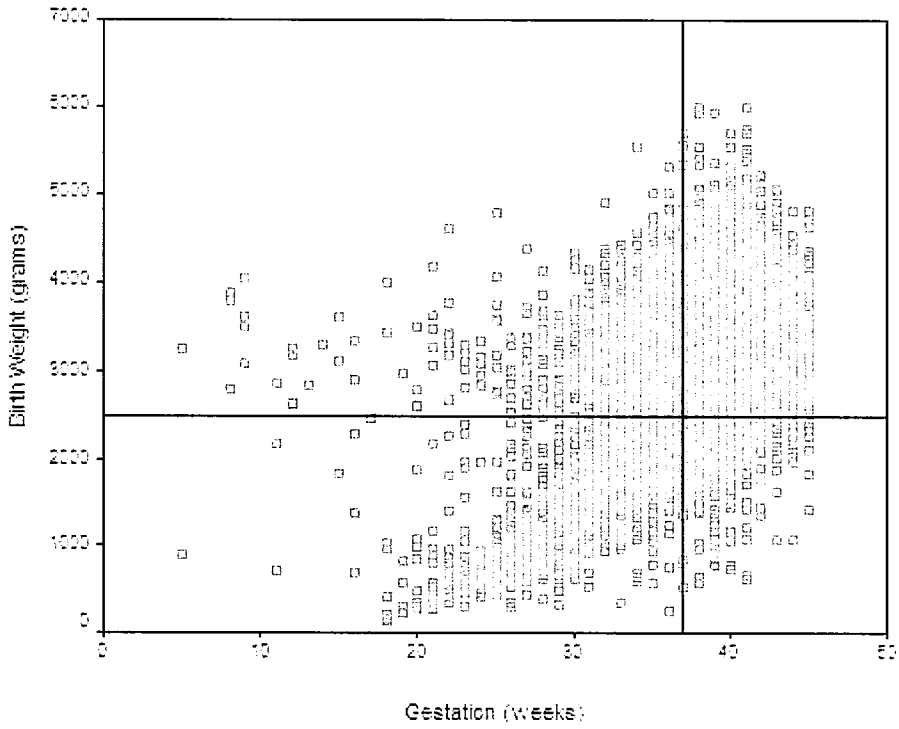


Figure 20: Scatterplot of Birth Weight by Gestational Age for Foreign-Born African American Women



Geographic Variations in Low Birthweight

A majority of African American infants were born in Brooklyn and the Bronx followed by Queens, Manhattan, and Staten Island. A majority of white infants were also born in Brooklyn, followed by Queens, Manhattan, the Bronx, and Staten Island. These geographic patterns of births by race follow demographic racial patterns in the general population. When African American women were studied by origin of birth there were more U.S.-born African American births in Brooklyn than foreign-born African American births, although Brooklyn was where a majority of black births occurred for both groups. U.S.-born African American infants were also likely to be born in the Bronx, followed by Queens, Manhattan and Staten Island. Foreign-born African American infants were more likely to be born in Manhattan and Staten Island than in the Bronx or Queens (Table 8).

Table 8: Borough of Residence for Women by Race, New York City, 2000

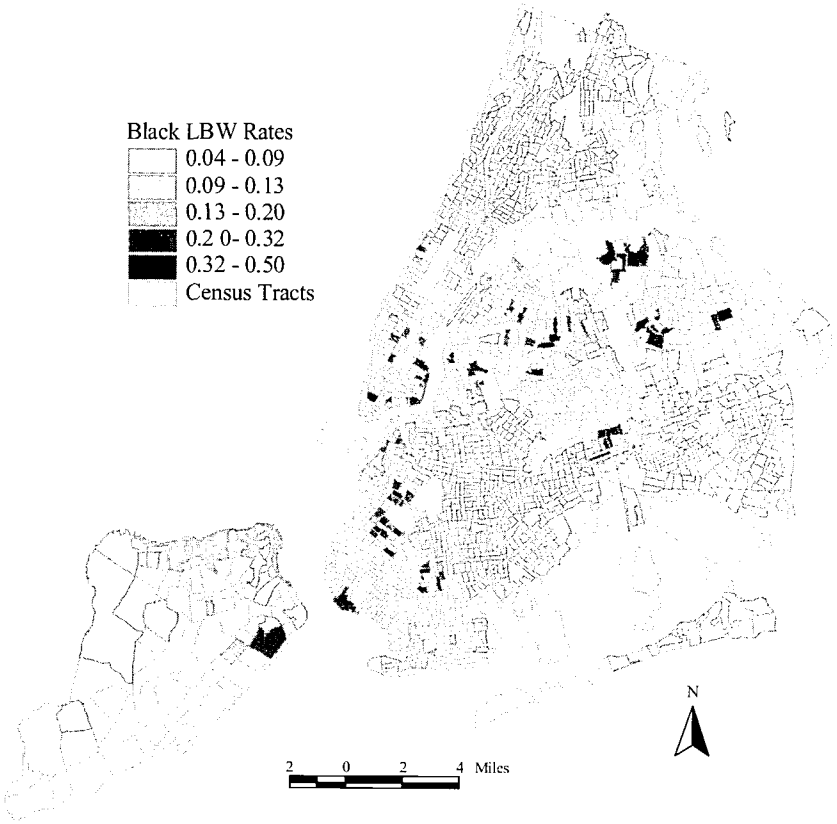
	Black Women		White Women		U.S.-Born Black Women		Foreign-Born Black Women	
Borough	**No.**	**Percent**	**No.**	**Percent**	**No.**	**Percent**	**No.**	**Percent**
Bronx	9740	(26.5)	10429	(17.3)	4566	(25.4)	5056	(16.4)
Brooklyn	14938	(40.7)	19390	(32.1)	7283	(40.6)	10513	(34.1)
Manhattan	4671	(12.7)	11714	(19.4)	2530	(14.1)	6865	(22.2)
Queens	6491	(17.7)	14260	(23.6)	3062	(17.1)	4871	(15.8)
Staten Island	798	(2.1)	4451	(7.3)	496	(2.7)	3504	(11.3)

Race-specific spatially weighted smoothed rate maps were created using GEODA version 9.5.i (2004). The spatial weight used was a nearest neighbor (4 neighbors). The rate was calculated by the number of low birthweight births divided by the total number of births per census tract. Figure 21 shows the spatially weighted rates of low birthweight for African American women. Those census tracts with high rates were

generally located within areas of residential segregation. In Northern Manhattan and Southern Bronx higher rates of low birthweight were centrally located. In Brooklyn there were higher rates within and to the north of the segregated area. There were also two groups of census tracts with higher rates of low birthweight in the Queens area. Census tracts with higher rates were also located along the northern coast and eastern portion of Staten Island – in between lower rate census tracts.

Figure 22 shows the spatially weighted rates of low birthweight for white women. Census tracts with high rates of low birth weight were distributed throughout the city with little concentration in segregated areas, as expected.

Figure 21: Spatially Weighted Rates of Low Birthweight in African American Women, New York City, 2000



Smoothed spatial rates calculated using nearest neighborhood weighting scheme in GEODA 9.5.i.

Figure 22: Spatially Weighted Rates of Low Birthweight in White Women, New York City, 2000

Smoothed spatial rates calculated using nearest neighborhood weighting scheme in GEODA 9.5.i.

Table 9 shows the place variations in the number and percentage of low birthweight births, premature births, and IUGR infants for African American and white mothers by residential segregation and poverty. The racial disparity between African American and white women was slightly greater than that for the population as a whole (9.2 vs. 5.3). African American women living in residential segregation were more likely than similar women not living in residential segregation to have low

birthweight births (9.6 vs. 8.7). Residential segregation also contributed to higher rates of premature birth (5.9 vs. 5.2). The rate of UGR for African American women living in segregated and non-segregated neighborhoods was similar.

African American women living in poverty were also more likely to have a low birthweight birth compared to similar women not living in poverty (9.3 vs. 8.7). There was also an increase in premature births (5.6 vs. 5.3) and IUGR births (2.8 vs. 2.5) for African American women living in poverty versus a non-poverty neighborhood.

The percentage of low birthweight births for African American women living in segregated neighborhoods (9.6 percent) was greater than African American women living in poverty neighborhoods (9.3 percent). Women living in residential segregation were also more likely to have a premature birth as women living in poverty (5.9 vs. 5.6). There was little difference in IUGR births for African American women living in residential segregation versus poverty.

Although white women living in poverty were also at elevated risk of low birthweight (6.1 vs. 4.8), premature birth (3.4 vs. 2.6), and IUGR (2.0 vs. 1.6) compared to similar women who did not live in poverty, their overall risk was substantially lower than that for African American women.

Table 9: Place Variations in Birth Outcomes* by Race and Origin of Birth, New York City, 2000				
Race and Context	Low Birth Weight N	Low Birth Weight (%)	Premature (%)	IUGR (%)
African Americans	3362	9.2	5.6	2.7
US-Born	1870	10.5	6.2	3.2
Foreign-Born	1465	7.8	4.8	2.1
African Americans not in Segregation	1802	8.7	5.2	2.6
African Americans in Segregation	1522	9.6	5.9	2.7
US-Born	843	10.9	6.5	3.4
Foreign-Born	672	8.3	5.3	2.2
African Americans not in Poverty	1358	8.7	5.3	2.5
African Americans in Poverty	1963	9.3	5.6	2.8
US-Born	1231	11.0	6.5	3.4
Foreign-Born	712	7.3	4.4	2.1
Whites	3214	5.3	3.0	1.8
US-Born	1662	5.6	3.1	1.8
Foreign-Born	1532	5.1	2.8	1.8
Whites not in Poverty	1724	4.8	2.6	1.6
Whites Poverty	1447	6.1	3.4	2.0
US-Born	741	6.6	3.9	2.0
Foreign-Born	717	5.6	3.1	1.9
* Calculations might not add up because of missing variables				

The percentage of low birthweight births for U.S.-born African American women was substantially higher than that for foreign-born African American women (10.9 vs. 8.3). The difference in the percentage of premature births (6.5 vs. 5.3) and IUGR births (3.4 vs. 2.2) was also remarkable. U.S.-born women living in segregated neighborhoods were more likely to have a low birthweight birth than foreign-born African American women also living in segregated neighborhoods (10.9 vs. 8.3). This was also true for premature births (6.5 vs. 5.3) and IUGR (3.4 vs. 2.2). This disparity was also observed for U.S.-born and foreign-born African American women

living in poverty (11.0 vs. 7.3). These findings suggest that the protective factors related to being foreign-born persist in segregated and poverty environments, evidence of an *epidemiological paradox.*

These descriptive findings demonstrated substantial racial disparity in birth outcomes in New York City. Further research on the *epidemiological paradox* is also warranted given the large disparity in risk between U.S.-born and foreign-born African American women. While disparities persisted in different place environments, suggesting that individual characteristics of the mother play a very important role in low birthweight, there were still increases in risk associated with segregation and poverty. The relationship between individual- and contextual-level risk factors for low birthweight is investigated further and the results of these analyses are reported in Chapter 5 of this dissertation.

pregnancy-related medical conditions instead of *pre-pregnancy* medical conditions. Two additional models were run to measure the probability of low birthweight for U.S.-born and foreign-born African American women: (3) controlling for the individual-level variables MRACE*ORIGIN, MARITAL, COLLEGE, MEDICAID, SMOKE, ALCOHOL, SUBUSE and *pre-pregnancy* medical conditions; and (4) controlling for the same individual-level predictors but adding *pregnancy-related* medical conditions instead of *pre-pregnancy* medical conditions.

The results from the first model are presented in Table 10. They show that the odds of low birthweight for a white, U.S.-born woman, who was unmarried, without a college education, and medically insured, was 0.067, after controlling for pre-pregnancy medical conditions. The risk of low birthweight for African American women was significantly greater than the risk of low birthweight for white women, OR = 1.548 (1.467, 1.634) demonstrating racial disparity in low birthweight in New York City. In other words, black women were 50 percent more likely than white women to have a low birthweight birth, after controlling for individual-level risk factors. The odds of low birthweight for foreign-born women was less than the odds of low birthweight for U.S.-born women, OR = 0.818 (0.774, 0.865), suggesting the presence of an *epidemiological paradox.* Women were also at higher risk of low birthweight with increasing age (each unit increase in age increases the odds of low birthweight by 1.011), if they were unmarried, OR = 0.724 (0.679, 0.771), did not attend college, OR = 0.840 (0.790, 0.893), on Medicaid, OR = 1.142 (1.069, 1.221), if they smoked, OR = 1.531 (1.374, 1.706), if they drank alcohol (each drink increased the odds of low birthweight by 1.075), or if they used illicit

substances, OR = 2.559 (2.169, 3.019). These findings confirm the importance of traditional behavioral and socio-demographic risk factors in affecting low birthweight risk.

The pre-pregnancy medical conditions that significantly predicted low birthweight, after controlling for individual-level risk factors, included lung disease, OR = 1.250 (1.064, 1.467), chronic diabetes, OR = 1.941 (1.426, 2.642), chronic hypertension, OR = 3.443 (2.919, 4.060), renal disease, OR = 1.989 (1.328, 2.978), thyroid disease, OR = 1.721 (1.305, 2.271), viral disease, OR = 1.791 (1.100, 2.916), a previous preterm or SGA infant, OR = 4.821 (3.940, 5.899), or a previous miscarriage, OR = 2.112 (1.372, 3.252) (Table 10). Having a preterm or SGA infant in a previous pregnancy was the strongest pre-pregnancy medical risk factor for low birthweight. Chronic hypertension was also a strong predictor of low birthweight. Although white women had a higher rate of thyroid disease the odds associated with low birthweight was greater for African American women than white women. Those pre-pregnancy medical conditions that had no statistically significant association with low birthweight were anemia, cardiac disease, hepatitis, genital herpes, sexually transmitted diseases (STDs), rubella, and tuberculosis. Since these medical conditions were not statistically associated with low birthweight, they were removed from further analyses.

Table 10: Individual-Level Risk Factors (Including Pre-Pregnancy Medical Conditions) for Low Birthweight, New York City, 2000		
Risk Factors	**OR**	**Confidence Interval**
Intercept	0.067	
MRACE	1.548	(1.467, 1.643)
ORIGIN	0.818	(0.774, 0.865)
MARITAL	0.724	(0.679, 0.771)
COLLEGE	0.840	(0.790, 0.893)
MEDICAID	1.142	(1.069, 1.221)
SMOKING	1.531	(1.374, 1.706)
ALCOHOL	1.075	(1.017, 1.136)
SUBSTANCE	2.559	(2.169, 3.019)
MAGE27	1.011	(1.006, 1.015)
LUNG	1.250	(1.064, 1.467)
DIABETES	1.941	(1.426, 2.642)
HYPERTENSION	3.443	(2.919, 4.060)
RENAL	1.989	(1.328, 2.978)
THYROID	1.721	(1.305, 2.271)
VIRAL	1.791	(1.100, 2.916)
PREVIOUS PRETERM/SGA	4.821	(3.940, 5.899)
PREVIOUS MISCARRIAGE	2.112	(1.372, 3.252)
N	96,882	

The results from the second model are presented in Table 11. These findings were consistent with those for model 1, with the various individual-level risk factors affecting low birthweight in the expected directions. The pregnancy-related medical conditions that significantly predicted low birthweight, after controlling for individual-level risk factors, included hydramnois or olighydramnios, OR = 1.986 (1.761, 2.240), pregnancy-related hypertension, OR = 1.928 (1.657, 2.243), preeclampsia, OR = 5.875 (5.328, 6.478), eclampsia, OR = 8.168 (4.775, 13.974), incompetent cervix, OR = 5.916 (4.639, 7.545), and uterine bleeding in the second, OR = 2.295 (1.438, 3.662) or third, OR = 5.905 (4.058, 8.592) trimesters. These are well-known pregnancy-related medical conditions that can lead to premature delivery and thus low birthweight. Pregnancy-related diabetes and uterine bleeding in the first trimester were not significant risk factors

for low birthweight and thus were removed from future analyses. Eclampsia was the strongest pregnancy-related medical risk for low birthweight. Incompetent cervix and bleeding in the third trimester also put women at substantial risk of low birthweight. Eclampsia, incompetent cervix, and bleeding in the third trimester are all leading causes of premature birth.

Table 11: Individual-Level Risk Factors (Includes Pregnancy-Related Medical Conditions) for Low Birthweight, New York City, 2000

Risk Factors	OR	Confidence Interval
Intercept	0.061	
MRACE	1.530	(1.457, 1.625)
ORIGIN	0.790	(0.747, 0.835)
MARITAL	0.783	(0.692, 0.787)
COLLEGE	0.827	(0.777, 0.880)
MEDICAID	1.152	(1.031, 1.172)
SMOKING	1.606	(1.441, 1.791)
ALCOHOL	1.069	(1.009, 1.132)
SUBUSE	2.745	(2.327, 3.239)
MAGE27	1.014	(1.010, 1.019)
HYDRAMNIOS/OLIGHYD_	1.986	(1.761, 2.240)
HYPERTENSION	1.928	(1.657, 2.243)
PREECLAMPISA	5.875	(5.238, 6.478)
ECLAMPSIA	8.168	(4.775, 13.974)
INCOMPETENT CERVIX	5.916	(4.639, 7.545)
UTERINE 2ND TRIMESTER	2.295	(1.438, 3.662)
UTERINE 3RD TRIMESTER	5.905	(4.058, 8.592)
N	96,882	

A key question in this dissertation concerns differences in low birthweight risk for African American women by origin of birth. To assess these differences an interaction term MRACE*ORIGIN was added to each of the two regression models. The results from the third model controlling for individual-level risk factors, including pre-pregnancy medical conditions are presented in Table 12. They showed that the risk of low birthweight for U.S.-born African American women was 72 percent greater than the odds

of low birthweight for white women. Along the same lines, the odds of low birthweight for foreign-born African American women, (OR = 1.393 (1.302, 1.490)) was 39 percent greater than the odds of low birthweight for white women. Although both U.S.-born and foreign-born African American women were at increased risk of low birthweight after controlling for pre-pregnancy medical conditions and other individual-level risk factors, that risk was substantially greater for U.S.-born African American women. Black immigrant women have significantly better reproductive outcomes than black U.S.-born women, even after a range of risk factors are controlled.

Table 12: Individual-Level Risk Factors for African American Women by Origin of Birth (Including Pre-Pregnancy Medical Conditions) for Low Birthweight, New York City, 2000

Risk Factors	OR	Confidence Interval
Intercept	0.067	
BLKUS	1.722	(1.613, 1.838)
BLKFB	1.393	(1.302, 1.490)
MARITAL	0.724	(0.679, 0.771)
COLLEGE	0.840	(0.790, 0.893)
MEDICAID	1.142	(1.069, 1.221)
SMOKING	1.531	(1.374, 1.706)
ALCOHOL	1.075	(1.017, 1.136)
SUBSTANCE	2.559	(2.169, 3.019)
MAGE27	1.011	(1.006, 1.015)
LUNG	1.250	(1.064, 1.467)
DIABETES	1.941	(1.426, 2.642)
HYPERTENSION	3.443	(2.919, 4.060)
RENAL	1.989	(1.328, 2.978)
THYROID	1.721	(1.305, 2.271)
VIRAL	1.791	(1.100, 2.916)
PREVIOUS PRETERM/SGA	4.821	(3.940, 5.899)
PREVIOUS MISCARRIAGE	2.112	(1.372, 3.252)
N	96,882	

The results from the fourth model which, controls for pregnancy related medical conditions are presented in Table 13. As in the previous model, the risk of low

birthweight for U.S.-born African American women was 73 percent greater than the odds of low birthweight for white women. Along the same lines, the odds of low birthweight for foreign-born African American women, (OR = 1.370 (1.280, 1.467)) was 37 percent higher than the odds of low birthweight for white women. Although both U.S.-born and foreign-born African American women were at increased risk of low birthweight after controlling for pregnancy-related medical conditions and other individual-level risk factors, that risk was again substantially greater for U.S.-born African American women. The increased risk of low birthweight for U.S.-born versus foreign-born African American women in both models supported the *epidemiological paradox* in New York City, which identifies better health outcomes among immigrants compared to their U.S.-born counterparts.

Table 13: Individual-Level Risk Factors for African American Women by Origin of Birth (Includes Pregnancy-Related Medical Conditions) for Low Birthweight, New York City, 2000

Risk Factors	OR	Confidence Interval
Intercept	0.056	
BLKUS	1.735	(1.625, 1.853)
BLKFB	1.370	(1.280, 1.467)
MSTATUS	0.739	(0.694, 0.788)
COLLEGE	0.832	(0.782, 0.885)
MEDICAID	1.098	(1.029, 1.172)
SMOKING	1.656	(1.486, 1.846)
ALCOHOL	1.067	(1.008, 1.130)
SUBUSE	2.801	(2.374, 3.305)
MAGE27	1.013	(1.009, 1.017)
HYDRAMNIOS/OLIGHYD_	1.975	(1.751, 2.227)
HYPERTENSION	1.940	(1.667, 2.256)
PREECLAMPSIA	5.814	(5.273, 6.409)
ELCAMPSIA	8.169	(4.780, 13.959)
INCOMPETENT CERVIX	5.979	(4.696, 7.613)
UTERINE 2ND TRIMESTER	2.325	(1.458, 3.708)
UTERINE 3RD TRIMESTER	5.935	(4.081, 8.632)
N	96,882	

CONTEXTUAL-LEVEL RISK FACTORS

Hierarchical models were constructed to measure individual, contextual, and cross-level effects on low birthweight. Four models were implemented: (1) variance components model, (2) random coefficients model, (3) Level-2 predictor for intercepts-as-outcomes model, and (4) Level-2 predictor for intercepts and slopes-as-outcomes model.

Variance Components Model

The variance-components model will measure the intercept or low birthweight mean for New York City, and the unexplained variance in census tract low birthweight intercepts or means. The expected log-odds of low birthweight in New York City was γ_{00} –2.652, which translated to an odds ratio (OR) exp (-2.652) = 0.070 (0.055, 0.090), or about 1 in 14 births (Table 14).

The variation in low birthweight intercepts at the individual-level within census tracts was ($\sigma^2 = 0.340$). The variation in low birthweight intercepts at the census tract level was ($\tau_{00} = 0.116$). This variation at the census tract level was statistically significant (p-value 0.000) demonstrating significant variability in the rates of low birthweight across census tracts in New York City (Table 14).

Table 14: Variance Components Model for Low Birthweight, New York City, 2000

Fixed Effect	β	*se*	t-ratio	df	p-value
Average census tract mean, γ_{00}	-2.652	0.016	-165.532	2081	0.00
	Standard	Variance			
Random Effect	Deviation	Component	df	X^2	p-value
Census tract mean, μ_{0j}	0.340	0.116	2081	2643.836	0.000

Random-Coefficient Model

A random-coefficient model was implemented to estimate the low birthweight intercept and average slope for each individual-level predictor of LBW (e.g., MRACE, ORIGIN, MAGE27, MARITAL, COLLEGE, MEDICAID, SMOKE, and SUBUSE). This model also provided information on the variability in the intercepts and slopes. The results from this analysis is presented in Table 15.

The expected log-odds of low birthweight was γ_{00} -2.638 translating into an OR = 0.071 (0.048, 0.105), or about 1 in 14 births, after controlling for individual-level risk factors. This result was similar to the previous model. The odds of low birthweight for African American women was exp (0.435) = OR 1.544 (1.106, 2.157) times the odds of low birthweight for white women, demonstrating racial disparity in low birthweight. The odds of low birthweight for foreign-born women was exp (-0.226) = OR 0.802 (0.571, 1.113) the odds of low birthweight for U.S.-born women, suggestive of the *epidemiological paradox.* This model also showed that women were at higher risk of low birthweight if they were unmarried, exp (-0.321) = OR 0.725 (0.508, 1.035), without a college education, exp (-0.192) = OR 0.825 (0.581, 1.171), on Medicaid exp (0.140) = OR 1.150 (0.801, 1.651), or if they smoked exp (0.470) = OR 1.599 (1.006, 2.544) or used illicit substances, exp (0.967) = OR 2.263 (1.485, 4.657). For each year increase in age the odds of low birthweight increased by 1.016. In general, the coefficients in this model were similar to the coefficients in the logistic regression analyses.

The variation in low birthweight intercepts at the individual-level within census tracts was (σ^2 = 0.338), which was similar to the previous model. The similarity between models in the within census tract variability was expected because the composition of women did not change. The variation in low birthweight intercepts at the census tract level (τ_{00} = 0.114), remained statistically significant (p-value 0.027). Thus, controlling for individual-level risk factors did not substantially change the within or between census tract variability in low birthweight intercepts (Table 15).

In addition to significant variability in low birthweight across census tracts, there was also significant variability in MRACE (τ_{11} = 0.089) across census tracts (p-value 0.014). Thus, the racial composition of women within census tracts is assumed to be largely homogeneous, which is expected given the high levels of racial segregation in New York City. Homogeneity within census tracts probably contributes to between census tract racial differences (Table 15).

Table 15: Random Coefficients Model for Low Birthweight, New York City, 2000						
	Fixed Effect	**β***	***se***	**t-ratio**	**df**	**p-value**
Intercept, β_{0j}	Intercept, γ_{00}	-2.638	0.040	-65.835	2095	0.000
MRACE, slope, β_{1j}	Intercept, γ_{10}	0.435	0.029	14.849	2095	0.000
ORIGIN, slope, β_{2j}	Intercept, γ_{20}	-0.226	0.029	-7.761	2096	0.000
MARITAL, slope, β_{3j}	Intercept, γ_{30}	-0.321	0.033	-9.487	2096	0.000
COLLEGE, slope β_{4j}	Intercept, γ_{40}	-0.192	0.032	-5.987	2096	0.000
MEDICAID, slope, β_{5j}	Intercept, γ_{50}	0.140	0.034	4.061	2096	0.000
SMOKE, slope, β_{6j}	Intercept, γ_{60}	0.470	0.056	8.351	2096	0.000
SUBUSE slope, β_{7j}	Intercept, γ_{70}	0.967	0.085	11.319	2096	0.000
MAGE27, slope β_{8j}	Intercept, γ_{80}	0.016	0.002	7.212	2096	0.000
* Unstandardized coefficient						
	Standard	**Variance**				
Random Effects	**Deviation**	**Component**	**df**	X^2	**p-value**	
Intercept, μ_{0j}	0.338	0.114	444	503.222	0.027	
MRACE, μ_{1j}	0.299	0.089	444	512.238	0.014	
ORIGIN, μ_{2j}	0.210	0.044	444	448.657	0.429	
MARITAL, μ_{3j}	0.321	0.103	444	392.566	>0.500	
COLLEGE, μ_{4j}	0.195	0.038	444	475.967	0.142	
MEDICAID, μ_{5j}	0.174	0.030	444	391.926	>0.500	
SMOKE, μ_{6j}	0.373	0.139	444	446.047	0.464	
SUBUSE, μ_{7j}	0.449	0.202	444	447.206	0.449	
MAGE27, μ_{8j}	0.011	0.000	444	439.431	>0.500	

Residential Segregation –

The effects of residential segregation on low birthweight were examined by adding the log of segregation (SEGLOG) as a contextual or Level-2 variable, while keeping individual-level variables in the model. The results from the Level-2 Predictor for Intercepts-as-Outcomes Model are provided in Table 16. The results from the Level-2 Predictor for Intercept- and Slopes-as-Outcomes Model is presented in Table 17.

Level-2 Predictor for Intercepts-as-Outcomes Model

A level-2 predictor for intercept-as-outcomes model was implemented to measure the contextual effect of residential segregation (SEGLOG) on low birthweight. The results

from this model provided estimates on the low birthweight intercept if residential segregation = 0 (SEGLOG = 0), the change in intercept with SEGLOG, and the unexplained variance in census tract low birthweight means.

This modeled showed that the impact of residential segregation on low birthweight was statistically significant. For women living in areas with no segregation (SEGLOG=0) the expected log-odds of low birthweight was –2.637, corresponding to an OR = 0.071 (0.048, 0.105), or about 1 in 14 births. This result was similar to both previous models. However, the coefficient for SEGLOG was positive (0.069) and statistically significant (p-value = 0.00) demonstrating that higher levels of residential segregation were associated with higher rates of low birthweight. Translating SEGLOG back to SEG the log-odds was 1.071, corresponding to an OR = 2.915. Thus, a unit increase in SEG increased the odds of low birthweight by 2.915. Therefore, if comparing two women who were similar in other ways but lived in areas that differed by one unit of SEG, we could expect the odds of low birthweight of the higher-SEG woman to be 2.915 times the odds of low birthweight of the lower-SEG woman. The meaning of "one unit" of SEG can be interpreted in terms of the standard deviation. In the descriptive figures (Chapter 4), the standard deviation for SEG was 0.03. We can therefore say that a one standard deviation difference in SEG is associated with a difference in the log-odds of low birthweight of 0.03 * (1.071) = 0.032 or a relative odds of exp (0.032) = 1.032.

The effects of individual variables were similar to those observed in previous models. Being African American was associated with a higher odds of low birthweight, exp

(0.336) = 1.399 (0.985, 1.986). Being foreign-born was associated with a lower odds of low birthweight, exp (-0.232) = 0.792 (0.567, 1.107). The magnitude of the odds ratios associated with the individual-level predictors after controlling for residential segregation were slightly higher than in the previous models, except for women on Medicaid where the odds ratios were relatively similar. For example, the protective effects of marriage (OR = 0.749 vs. 0.725) and college education (OR = 0.827 vs. 0.825) slightly increased. In addition, women who used illicit substances were at substantially greater risk of low birthweight after controlling for residential segregation (OR = 2.640 vs. 2.263).

The variation in low birthweight intercepts remained relatively the same within census tracts (σ^2 = 0.324) as expected since the composition of women did not change. However, the variation in low birthweight intercepts across census tracts (τ_{00} = 0.105) was not significant (p-value = 0.106) indicating that residential segregation sufficiently explained census tract variability in low birthweight means. In addition, the variability in MRACE across census tracts was also explained by residential segregation (Table 16).

Table 16: Level-2 Predictor for Intercepts-as-Outcomes Model (Residential Segregation), New York City, 2000

	Fixed Effects	**β***	***se***	**t-ratio**	**df**	**p-value**
Intercept, β_{0j}	Intercept, γ_{00}	-2.637	0.039	-65.952	2080	0.000
SEGLOG	Intercept, γ_{01}	0.069	0.011	6.308	2080	0.000
MRACE, slope, β_{1j}	Intercept, γ_{10}	0.336	0.032	10.335	2081	0.000
ORIGIN, slope, β_{2j}	Intercept, γ_{20}	-0.232	0.029	-7.956	2081	0.000
MARITAL, slope, β_{3j}	Intercept, γ_{30}	-0.288	0.034	-8.421	2081	0.000
COLLEGE, slope β_{4j}	Intercept, γ_{40}	-0.189	0.032	-5.910	2081	0.000
MEDICAID, slope, β_{5j}	Intercept, γ_{50}	0.127	0.034	3.685	2081	0.000
SMOKE, slope, β_{6j}	Intercept, γ_{60}	0.473	0.056	8.376	2081	0.000
SUBUSE slope, β_{7j}	Intercept, γ_{70}	0.971	0.086	11.266	2081	0.000
MAGE27, slope β_{8j}	Intercept, γ_{80}	0.016	0.002	7.282	2081	0.000
* Unstandardized coefficient						

Random Effects	**Standard Deviation**	**Variance Component**	**df**	X^2	**p-value**	
Intercept, μ_{0j}	0.324	0.105	432	469.029	0.106	
MRACE, μ_{1j}	0.251	0.063	432	473.572	0.087	
ORIGIN, μ_{2j}	0.198	0.039	432	437.105	0.436	
MARITAL, μ_{3j}	0.310	0.096	432	378.065	>0.500	
COLLEGE, μ_{4j}	0.193	0.037	432	453.364	0.241	
MEDICAID, μ_{5j}	0.168	0.028	432	372.223	>0.500	
SMOKE, μ_{6j}	0.372	0.138	432	432.084	>0.500	
SUBUSE, μ_{7j}	0.442	0.195	432	434.456	0.471	
MAGE27, μ_{8j}	0.011	0.000	432	422.940	>0.500	

When coefficients from the Level-2 Predictor for Intercept-as-Outcomes Model were compared with the coefficients from the Random Coefficients Model, there was a substantial decrease in the odds ratio for MRACE (1.399 vs. 1.544). Measuring residential segregation (a social construct) in the context of women's race (also a social construct) may therefore be a form of over-control. To further evaluate these findings MRACE was removed from the model and the change in effect of SEGLOG on low birthweight has re-evaluated.

In the new analysis (Table 17) the expected log-odds of low birthweight was -2.498, corresponding to an odds ratio = 0.082 (0.056, 0.119), or 1 in 12 births. The result showed slightly greater risk of low birthweight than in the previous model that included MRACE. The expected log-odds of SEGLOG = 0.112, associated with an expected log-odds of SEG = 1.118. A one-unit increase in SEG without controlling for MRACE increased the log-odds of low birthweight by 1.118 corresponding to an OR = 3.058. Therefore, if we compared two women who were similar in other ways but differed by one unit in SEG we could expect the odds of low birthweight of the higher-SEG woman to be 3.058 times the odds of low birthweight of the lower-SEG woman. This risk differential was slightly greater than that observed in the model controlling for MRACE (OR = 2.919). When the risk associated with residential segregation was measured in terms of a one standard deviation difference in SEG associated with a difference in the log-odds of low birthweight the result was 0.03 * (1.118) = 0.033 or a relative odds of exp (0.033) = 1.033 the result was relatively the same (OR = 1.031).

These findings demonstrated that the overall impact that SEGLOG had on low birthweight increased slightly. However, it was decided that MRACE should be left in the model. Since this research focuses on racial disparities in low birthweight and the literature demonstrates strong racial disparities, it makes sense to keep race as an individual-level variable in the model. Omitting race would cause the effects of residential segregation to be overestimated because they partially include the effects of race on low birthweight.

Table 17: Level-2 Predictor for Intercepts-as-Outcomes Model (Residential Segregation), New York City, 2000*
***MRACE excluded from model**

	Fixed Effects	**β***	***se***	**t-ratio**	**df**	**p Value**
Intercept, β_{0j}	Intercept, γ_{00}	-2.498	0.037	-66.247	2080	0.000
SEGLOG	Intercept, γ_{01}	0.112	0.010	12.153	2080	0.000
ORIGIN, slope, β_{1j}	Intercept, γ_{10}	-0.245	0.029	-8.368	2081	0.000
MARITAL, slope, β_{2j}	Intercept, γ_{20}	-0.328	0.034	-9.607	2081	0.000
COLLEGE, slope β_{3j}	Intercept, γ_{30}	-0.181	0.032	-5.608	2081	0.000
MEDICAID, slope, β_{4j}	Intercept, γ_{40}	0.130	0.034	3.765	2081	0.000
SMOKE, slope, β_{5j}	Intercept, γ_{50}	0.487	0.056	8.668	2081	0.000
SUBUSE slope, β_{6j}	Intercept, γ_{60}	0.975	0.085	11.351	2081	0.000
MAGE27, slope β_{7j}	Intercept, γ_{70}	0.017	0.002	7.767	2081	0.000
* Unstandardized coefficient						

Random Effects	**Standard Deviation**	**Variance Component**	**df**	X^2	**p-value**	
Intercept, μ_{0j}	0.313	0.098	449	451.296	0.461	
ORIGIN, μ_{1j}	0.221	0.049	450	441.580	>0.500	
MARITAL, μ_{2j}	0.331	0.109	450	386.023	>0.500	
COLLEGE, μ_{3j}	0.203	0.041	450	468.760	0.261	
MEDICIAD, μ_{4j}	0.174	0.030	450	395.918	>0.500	
SMOKE, μ_{5j}	0.369	0.136	450	454.992	0.426	
SUBUSE, μ_{6j}	0.451	0.203	450	463.793	0.316	
MAGE27, μ_{7j}	0.012	0.000	450	428.120	>0.500	

Residential Segregation -

A level-2 predictor for intercept- and slopes-as-outcomes model was implemented to measure individual- and contextual-level interactions at the census tract level, and to identify and map census tracts with large unexplained residuals. The results from this analysis are provided in Table 18. When this model is compared to the "previous" model, the comparison is referring to the Level-2 Predictor for Intercepts-as-Outcomes Model that included the variable MRACE.

Level-2 Predictor for Intercepts- and Slopes-as-Outcomes Model

In this model SEGLOG was used to predict the low birthweight intercepts and slopes for each individual-level predictor of low birthweight. The results showed that SEGLOG did not significantly change the direction or magnitude of the individual-level predictors and their low birthweight relationships. In other words none of the individual-level risk factors for low birthweight were exacerbated by residential segregation.

Finally, the within census tract variability in low birthweight intercepts remained relatively constant ($\sigma^2 = 0.302$) as expected because the composition of women did not change. In addition, residential segregation significantly explained the variation in low birthweight intercepts across census tracts ($\tau_{00} = 0.091$, p-value = 0.136), a finding similar to the previous analysis.

Table 18: Level-2 Predictor for Intercepts- and Slopes-as-Outcomes Model (Residential Segregation), New York City, 2000

	Fixed Effects	**β***	***se***	**t-ratio**	**df**	**p-value**
Intercept, β_{0j}	Intercept, γ_{00}	-2.607	0.043	-59.355	2080	0.000
SEGLOG	Intercept, γ_{01}	0.060	0.026	2.291	2080	0.022
MRACE, slope, β_{1j}	Intercept, γ_{10}	0.352	0.041	8.460	2080	0.000
SEGLOG	Intercept, γ_{11}	-0.016	0.025	-0.672	2080	0.502
ORIGIN, slope, β_{2j}	Intercept, γ_{20}	-0.232	0.032	-7.078	2080	0.000
SEGLOG	Intercept, γ_{21}	-0.015	0.019	-0.793	2080	0.428
MARITAL, slope, β_{3j}	Intercept, γ_{30}	-0.307	0.037	-8.233	2080	0.000
SEGLOG	Intercept, γ_{31}	0.038	0.022	1.714	2080	0.086
COLLEGE, slope β_{4j}	Intercept, γ_{40}	-0.195	0.035	-5.572	2080	0.000
SEGLOG	Intercept, γ_{41}	0.011	0.020	0.538	2080	0.590
MEDICAID, slope, β_{5j}	Intercept, γ_{50}	0.123	0.038	3.194	2080	0.002
SEGLOG	Intercept, γ_{51}	-0.004	0.022	-0.187	2080	0.852
SMOKE, slope, β_{6j}	Intercept, γ_{60}	0.512	0.064	7.959	2080	0.000
SEGLOG	Intercept, γ_{61}	-0.004	0.037	-1.194	2080	0.233
SUBUSE slope, β_{7j}	Intercept, γ_{70}	0.837	0.129	6.453	2080	0.000
SEGLOG	Intercept, γ_{71}	0.110	0.077	1.411	2080	0.158
MAGE27, slope β_{8j}	Intercept, γ_{80}	0.014	0.002	5.664	2080	0.000
SEGLOG	Intercept, γ_{81}	0.002	0.001	1.644	2080	0.100
* Unstandardized coefficient						

Table 18 (continued)						
	Standard	**Variance**				
Random Effects	**Deviation**	**Component**	**df**	X^2	**p-value**	
Intercept, μ_{0j}	0.302	0.091	432	464.944	0.136	
MRACE, μ_{1j}	0.236	0.055	432	469.944	0.101	
ORIGIN, μ_{2j}	0.197	0.038	432	437.463	0.4118	
MARITAL, μ_{3j}	0.283	0.080	432	373.633	>0.500	
COLLEGE, μ_{4j}	0.189	0.035	432	451.293	0.251	
MEDICAID, μ_{5j}	0.152	0.023	432	369.950	>0.500	
SMOKE, μ_{6j}	0.361	0.130	432	435.668	0.442	
SUBUSE, μ_{7j}	0.435	0.189	432	434.588	0.456	
MAGE27, μ_{8j}	0.011	0.000	432	420.924	>0.500	

Comparison of Hierarchical Models

Table 19 lists the estimates of low birthweight and coefficients for the different models for comparison purposes. The odds of low birthweight in non-segregated areas remained fairly constant across models (in order of increasing complexity OR ranged from exp (-2.652) = 0.070 to exp (-2.607) = 0.073)). Residential segregation had a statistically significant and positive effect on low birthweight the last two models. This confirmed the hypothesis that women who live in highly segregated areas have a higher risk of low birthweight than similar women who do not live in segregated areas. Residential segregation significantly explained the variability in mean low birthweight rates and the variability in the effect of race on low birthweight between census tracts. Black segregation is an important factor underlying the large racial disparities in low birthweight in New York City.

The individual-level risk factors for low birthweight identified in the logistic regression analysis remained significant in the hierarchical models, after controlling for residential segregation. In general, women were at increased risk of low birthweight if they were

older, not married, without a college education, on Medicaid, or if they smoked, or used illicit substances. These factors have been cited in the literature, and these findings provide additional supporting evidence. Individual-level risk factors for low birthweight however were not exacerbated by residential segregation, suggesting that other factors explain how residential segregation impacts low birthweight. Later in this chapter the medical conditions will be analyzed for their mediating effect on the residential segregation and low birthweight relationship.

Table 19: Comparison of Four Hierarchical Models Analyzing the Effects of Individual-Contextual- (Residential Segregation) and Cross-Level Risk Factors for Low Birthweight, New York City, 2000

Parameter	Variance Components	Random Coefficients Model	Level-2^ Predictor Intercepts	Level-2^ Predictor Intercepts/ Slopes
Within census tract variability in intercepts, σ^2	0.340*	0.338*	0.324	0.302
Between census tract variability in intercepts, τ_{00}	0.116*	0.114*	0.105	0.091
Intercept LBW, Υ_{00}	-2.652*	-2.638*	-2.637*	-2.607*
Change in intercept with SEG#, Υ_{01}			-1.567*	-1.546*
Slope MRACE, Υ_{10}		0.435*	0.336*	0.352*
Slope ORIGIN, Υ_{20}		-0.226*	-0.232*	-0.232*
Slope MARITAL, Υ_{30}		-0.321*	-0.288*	-0.307*
Slope COLLEGE, Υ_{40}		-0.192*	-0.189*	-0.195*
Slope MEDICAID, Υ_{50}		0.140*	0.127*	0.123*
Slope SMOKE, Υ_{60}		0.470*	0.473*	0.512*
Slope SUBUSE, Υ_{70}		0.967*	0.971*	0.837*
Slope MAGE27, Υ_{80}		0.016*	0.016*	0.014*
Change in MRACE slope with SEGLOG, Υ_{11}				0.336
Change in ORIGIN slope with SEGLOG, Υ_{21}				-0.247
Change in MARITAL slope with SEGLOG, Υ_{31}				-0.269
Change in COLLEGE slope with SEGLOG, Υ_{41}				-0.184
Change in MEDICAID slope with SEGLOG, Υ_{51}				0.119
Change in SMOKE slope with SEGLOG, Υ_{61}				0.468
Change in SUBUSE slope with SEGLOG, Υ_{71}				0.947
Change in MAGE27 slope with SEGLOG, Υ_{81}				0.016

*Significant < 0.05
Based on SEGLOG transformed back to SEG
^ Includes models controlling for MRACE

The results from the last two models controlling for residential segregation, where compared to similar models, controlling for poverty. The first two models were not rerun because they did not include a contextual variable. The findings from the first two models will be used however in the interpretation of the results. This comparison is made

to see if the effect of segregation on low birthweight is greater than or less than the effect of poverty on low birthweight.

Poverty -

Level-2 Predictor for Intercepts-as-Outcomes Model

The results from this model are presented in Table 20 and show that the expected log-odds of low birthweight if POVLOG = 0 was –2.640, corresponding to an OR = 0.071 (0.048, 0.105), or about 1 in 14 births. The expected log-odds of POVLOG was 0.045. Translating POVLOG back to POV the log-odds was 1.046, corresponding to an OR = 2.846. This OR was slightly lower than the odds associated with residential segregation (OR = 2.915). In the descriptive analysis the standard deviation of POV was 0.15. Therefore, a one- standard deviation difference in POV was associated with a difference in the log-odds of low birthweight of 0.15 * (1.046) = 0.156 units apart in the log-odds of low birthweight, a relative odds = 1.168. This result was slightly greater than a one-standard deviation difference observed with SEG (OR = 1.031).

Controlling for POVLOG, African American women were exp (0.423) = OR 1.526 (1.093, 2.131) times more likely than white women to have a low birthweight birth. This OR was slightly greater than that observed for African American women living in residential segregation OR = 1.399 (0.985, 1.986). This finding shows that the effect of poverty is not explained by race as observed for race and residential segregation in the previous analysis. Foreign-born women were exp (-0.266) = OR 0.766 (0.548, 1.070) less likely than U.S.-born women to have a low birthweight birth. This finding was

relatively comparable to that observed in the model controlling for residential segregation, OR = 0.792 (0.567, 1.107).

The magnitude of effect of individual-level predictors varied slightly from the models controlling for residential segregation. For example, the protective effect of being married in the poverty model (OR = 0.731) was less than in the segregation model (OR = 0.749). Still the magnitudes and signs of the individual-level predictor variables were similar in both models.

Two important differences between the poverty and residential segregation models were (a) poverty did not explain the variability in low birthweight intercepts across census tracts, but residential segregation did; and (b) poverty did not explain variability in the effect of race on low birthweight, but residential segregation did. Controlling for poverty, there was still significant unexplained variability in the low birthweight intercepts (τ_{00} = 0.108, p-value = 0.042) and in the effect of race on low birthweight (τ_{11} = 0.082, p-value = 0.017). The within census tract variation for both models was relatively similar and therefore could not account for these differences.

When coefficients from the Level-2 for Intercepts-as-Outcomes Model, controlling for poverty, were compared to coefficients in the Random Coefficients Model, there was not a substantial decrease in the odds ratio for MRACE (1.526 vs. 1.544) as observed in the same model controlling for residential segregation. This finding suggests that residential

segregation has more of an impact on racial disparities in low birthweight, than poverty alone.

Table 20: Level-2 Predictor for Intercepts-as-Outcomes Model (Poverty), New York City, 2000

	Fixed Effects	**ß***	***se***	**t-ratio**	**df**	**p-value**
Intercept, β_{0j}	Intercept, γ_{00}	-2.640	0.040	-65.700	2080	0.000
POVLOG	Intercept, γ_{01}	0.045	0.022	2.057	2080	0.000
MRACE, slope, β_{1j}	Intercept, γ_{10}	0.423	0.029	14.347	2081	0.000
ORIGIN, slope, β_{2j}	Intercept, γ_{20}	-0.226	0.029	-7.748	2081	0.000
MARITAL, slope, β_{3j}	Intercept, γ_{30}	-0.313	0.034	-9.161	2081	0.000
COLLEGE, slope β_{4j}	Intercept, γ_{40}	-0.181	0.032	-5.611	2081	0.000
MEDICAID, slope, β_{5j}	Intercept, γ_{50}	0.130	0.034	3.733	2081	0.000
SMOKE, slope, β_{6j}	Intercept, γ_{60}	0.467	0.056	8.262	2081	0.000
SUBUSE slope, β_{7j}	Intercept, γ_{70}	0.977	0.086	11.334	2081	0.000
MAGE27, slope β_{8j}	Intercept, γ_{80}	0.016	0.002	7.344	2081	0.000
* Unstandardized coefficient						

Random Effect	**Standard Deviation**	**Variance Component**	**df**	X^2	**p-value**	
Intercept, μ_{0j}	0.329	0.108	432	483.994	0.042	
MRACE, μ_{1j}	0.286	0.082	433	497.706	0.017	
ORIGIN, μ_{2j}	0.206	0.042	433	440.642	0.389	
MARITAL, μ_{3j}	0.323	0.104	433	383.285	>0.500	
COLLEGE, μ_{4j}	0.188	0.035	433	456.090	0.214	
MEDICAID, μ_{5j}	0.164	0.027	433	379.037	>0.500	
SMOKE, μ_{6j}	0.376	0.141	433	435.175	0.462	
SUBUSE, μ_{7j}	0.432	0.186	433	437.251	0.434	
MAGE27, μ_{8j}	0.010	0.000	433	424.629	>0.500	

Poverty -

The next model examined the cross-level effects of poverty and individual-level risk factors for low birthweight. These results are presented in Table 21.

Level-2 Predictor for Intercepts and Slopes Model

In this model, the expected log-odds of low birthweight if POVLOG = 0 was –2.657, corresponding to an OR = 0.070 (0.046, 0.105), or about 1 in 14 births. The log-odds of

POVLOG was 0.156. Translating POVLOG back to POV the log-odds was 1.168, corresponding to an OR = 3.215. This OR was slightly higher than that observed for residential segregation (OR = 2.866), controlling for the same individual-level risk factors. In addition, a one- standard deviation difference in POV was associated with a difference in the log-odds of low birthweight of 0.150 * (1.168) = 0.175, or a relative odds = 1.191. This difference was slightly greater than a one standard deviation difference associated with SEG (OR = 1.031).

With regard to the slopes, POVLOG, like SEGLOG did not significantly change the direction or magnitude of effect for individual-level predictors of low birthweight. In other words, as with segregation, the poverty of census tracts did not influence the effect of individual-level risk factors on low birthweight. This means that residential segregation and census tract poverty impact low birthweight through other mechanisms.

In this model, the variability in low birthweight intercepts, and the effect of race on low birthweight, across census tracts was explained by poverty. There was however, some variability in ORIGIN between census tracts (τ_{00} = 0.042, p-value 0.056), suggesting that populations of women within census tracts were fairly homogeneous in terms of origin of birth. This is expected because immigrant populations are spatially clustered in distinct neighborhoods within the city. A high degree of homogeneity by origin of birth within census tracts may have led to greater variability between census tracts. In this same model controlling for residential segregation, there was not significant variability in ORIGIN between census tracts.

Table 21: Level-2 Predictor for Intercepts- and Slopes-as-Outcomes Model (Poverty), New York City, 2000						
	Fixed Effects	**ß***	***se***	**t-ratio**	**df**	**p-value**
Intercept, β_{0j}	Intercept, γ_{00}	-2.657	0.044	-59.482	2080	0.000
POVLOG	Intercept, γ_{01}	0.156	0.059	2.614	2080	0.022
MRACE, slope, β_{1j}	Intercept, γ_{10}	0.478	0.033	14.259	2081	0.000
POVLOG	Intercept, γ_{11}	-0.189	0.045	-4.162	2081	0.502
ORIGIN, slope, β_{2j}	Intercept, γ_{20}	-0.123	0.032	-6.564	2081	0.000
POVLOG	Intercept, γ_{21}	-0.097	0.043	-2.251	2081	0.428
MARITAL, slope, β_{3j}	Intercept, γ_{30}	-0.298	0.037	-7.866	2081	0.000
POVLOG	Intercept, γ_{31}	-0.007	0.052	-0.147	2081	0.086
COLLEGE, slope β_{4j}	Intercept, γ_{40}	-0.183	0.034	-5.287	2081	0.000
POVLOG	Intercept, γ_{41}	0.024	0.047	0.516	2081	0.590
MEDICAID, slope, β_{5j}	Intercept, γ_{50}	0.125	0.038	3.250	2081	0.002
POVLOG	Intercept, γ_{51}	-0.004	0.052	-0.086	2081	0.852
SMOKE, slope, β_{6j}	Intercept, γ_{60}	0.438	0.069	6.310	2081	0.000
POVLOG	Intercept, γ_{61}	0.075	0.085	0.893	2081	0.233
SUBUSE slope, β_{7j}	Intercept, γ_{70}	0.930	0.124	7.504	2081	0.000
POVLOG	Intercept, γ_{71}	0.075	0.149	0.503	2081	0.158
MAGE27, slope β_{8j}	Intercept, γ_{80}	0.017	0.002	6.683	2081	0.000
POVLOG	Intercept, γ_{81}	0.000	0.003	0.027	2081	0.100
* Unstandardized coefficients						
	Standard	**Variance**				
Random Effects	**Deviation**	**Component**	**df**	X^2	**p-value**	
Intercept, μ_{0j}	0.311	0.096	432	476.677	0.068	
MRACE, μ_{1j}	0.244	0.060	432	479.669	>0.500	
ORIGIN, μ_{2j}	0.205	0.042	432	439.473	0.056	
MARITAL, μ_{3j}	0.320	0.102	432	383.156	>0.500	
COLLEGE, μ_{4j}	0.186	0.034	432	454.632	0.218	
MEDICAID, μ_{5j}	0.152	0.023	432	376.629	>0.500	
SMOKE, μ_{6j}	0.386	0.149	432	435.211	0.448	
SUBUSE, μ_{7j}	0.450	0.202	432	436.299	0.433	
MAGE27, μ_{8j}	0.011	0.000	432	476.677	>0.500	

Comparison of Models

Table 22 lists the estimates of low birthweight and coefficients for the different models for comparison purposes. As mentioned earlier, the first two models do not control for poverty. The odds of low birthweight in non-poverty areas remained fairly constant

across models (in order of increasing model complexity the odds of low birthweight ranged from 0.070 to 0.071), a result similar to that in non-segregated environments.

The individual-level risk factors for low birthweight identified in the logistic regression analysis remained significant in the hierarchical models, after controlling for poverty. However, individual-level risk factors for low birthweight were not exacerbated in women who lived in poverty. This finding was similar to that observed for residential segregation. Similar to residential segregation there may be other factors playing into the poverty and low birthweight relationship.

Table 22: Comparison of Hierarchical Models Analyzing the Effects of Individual-Contextual- (Poverty)- and Cross-Level Risk Factors on Low Birthweight, New York City, 2000

Parameter	Variance Components	Random Coefficients Model	Level-2^ Predictor Intercepts	Level-2^ Predictor Intercepts/ Slopes
Within census tract variability in intercepts, σ^2	0.340	0.338	0.392	0.311
Between census tract variability in intercepts, τ_{00}	0.116*	0.114*	0.108	0.096
Intercept LBW, Υ_{00}	-2.652*	-2.638*	-2.640*	-2.657*
Change in intercept with POVLOG#, Υ_{01}			-1.594*	-1.489*
Slope MRACE, Υ_{10}			0.423*	0.478*
Slope ORIGIN, Υ_{20}			-0.226*	-0.123*
Slope MARITAL, Υ_{30}			-0.313*	-0.298*
Slope COLLEGE, Υ_{40}			-0.181*	-0.183*
Slope MEDICAID, Υ_{50}			0.130*	0.125*
Slope SMOKE, Υ			0.467*	0.438*
Slope SUBUSE, Υ_{70}			0.977*	0.930*
Slope MAGE27, Υ_{80}			0.016*	0.017*
Change in MRACE slope with POVLOG, Υ_{11}				-0.189
Change in ORIGIN slope with POVLOG, Υ_{21}				-0.097
Change in MARITAL slope with POVLOG, Υ_{31}				-0.007
Change in COLLEGE slope with POVLOG, Υ_{41}				0.024
Change in MEDICIAD slope with POVLOG, Υ_{51}				-0.004
Change in SMOKE slope with POVLOG, Υ_{61}				0.075
Change in SUBUSE slope with POVLOG, Υ_{71}				0.075
Change in MAGE27 slope with POVLOG, Υ_{81}				0.000

* Significant < 0.05

POVLOG transformed back to POV

^ Includes models controlling for MRACE

Model Checking

In this analysis the Level-2 ordinary least squares (OLS) and empirical Bayes (EB) residuals will be examined to assess the fit of the model. EB residuals are the deviation of an empirical Bayes estimate from its predicted value based on the Level-2 model, in this example SEGLOG. EB residuals from the Level-2 Predictor for Intercepts- and Slopes-as-Outcomes Model using SEGLOG were evaluated.

Figures 23 and 24 are Q-Q plots of Mahalanobis distances or the distance between the residual estimates for each census tract relative to the expected distances based on the model. If the plot resembles a 45 degrees line there is evidence that the random effects at Level-2 are normally distributed. Figure 23 demonstrates outlying census tracts in the upper right corner, where the Mahalanobis distances are larger than expected. After those outliers were removed from the model the Mahalanobis and expected distances were linear (Figure 24).

Figure 23: Mahalanobis Distances versus Expected Distances with Outliers

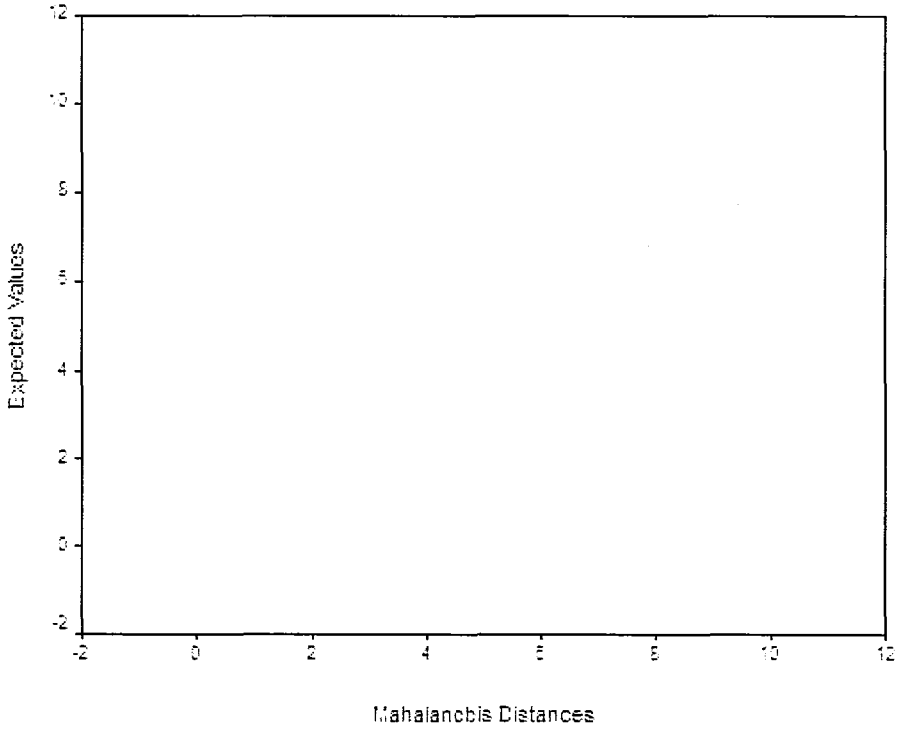
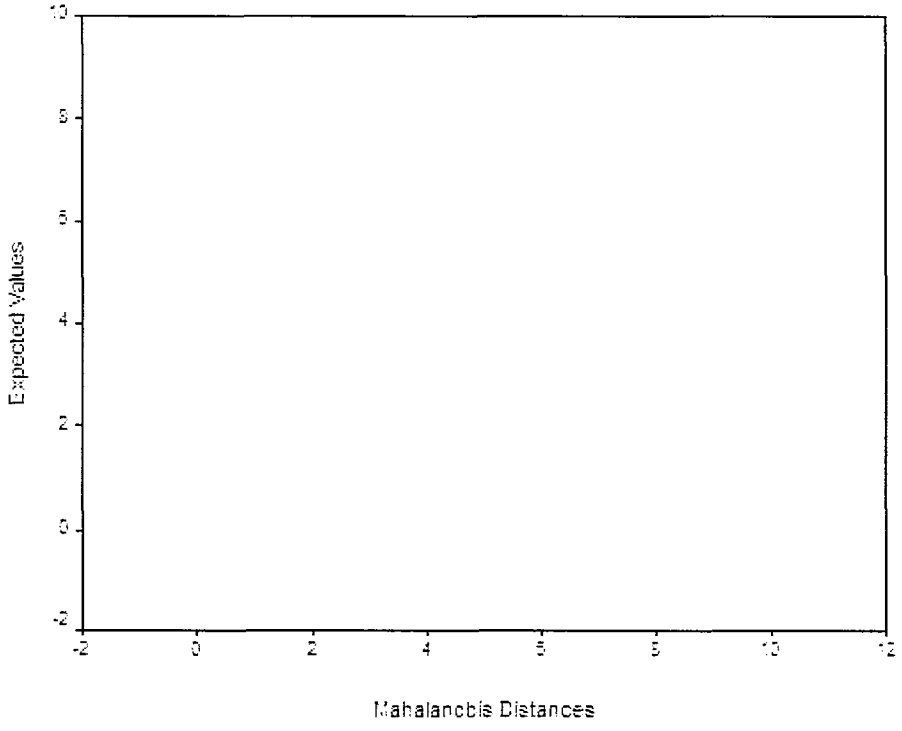


Figure 24: Mahalanobis Distances versus Expected Distances without Outliers



Figures 25 and 26 are histograms of OLS and EB intercept residuals after the outliers identified in Figure 23 were removed. The OLS residuals had a large number of negative values and were slightly skewed to the right. The EB residuals were normally distributed around the mean, as expected.

Figure 25: Histogram of OLS Intercept Residuals without Outliers

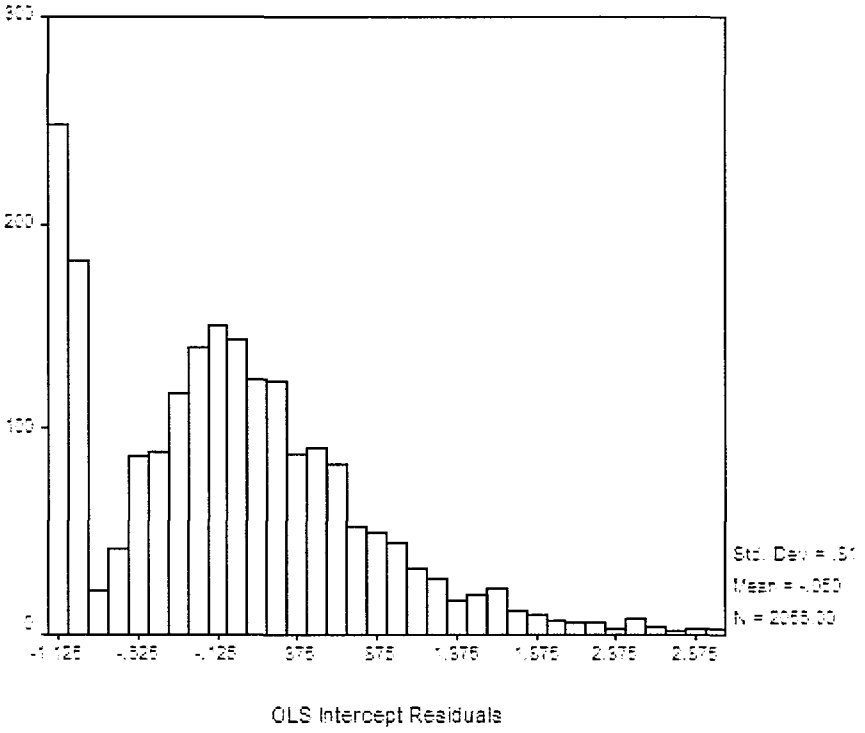
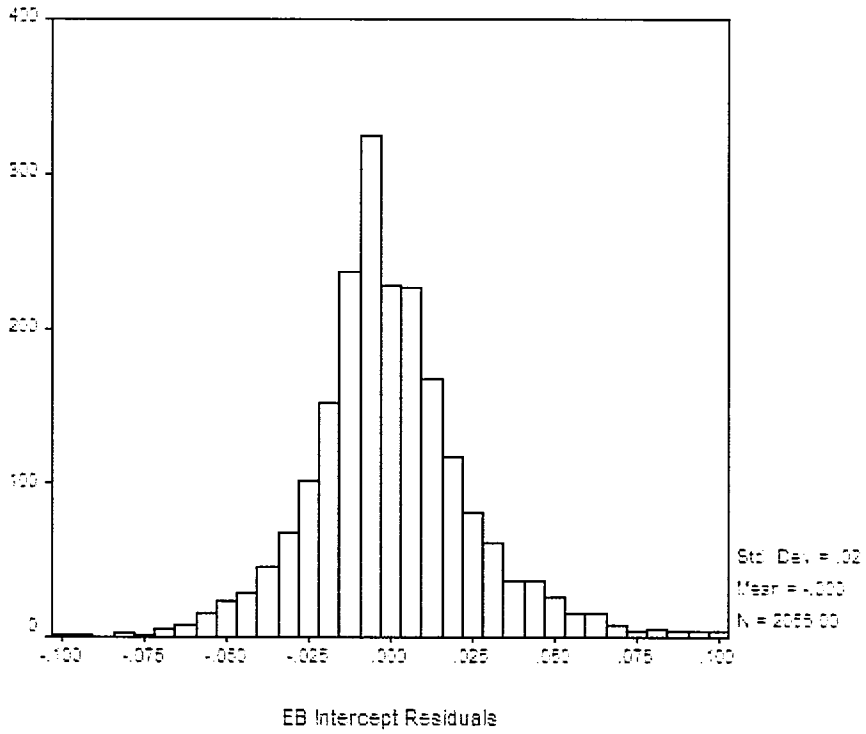


Figure 26: Histogram of EB Intercept Residuals without Outliers



These figures of OLS and EB residuals demonstrated that the Level-2 Predictor for Intercepts- and Slopes-as-Outcomes Model was adequately fit.

Figure 27 is a scatterplot of the EB residuals by proportion of low birthweight by census tracts. In general, census tracts with the proportion of low birthweight births less than 0.1 (or less than 10 percent) the residuals were negative, meaning the predicted values were greater than the actual. The residuals for census tracts with a proportion of low birthweight births greater than 0.1 (or greater than 10 percent) were generally positive, meaning the predicted values were less than the actual.

Figure 27: EB Intercept Residuals by Proportion Low Birthweight in Census Tracts

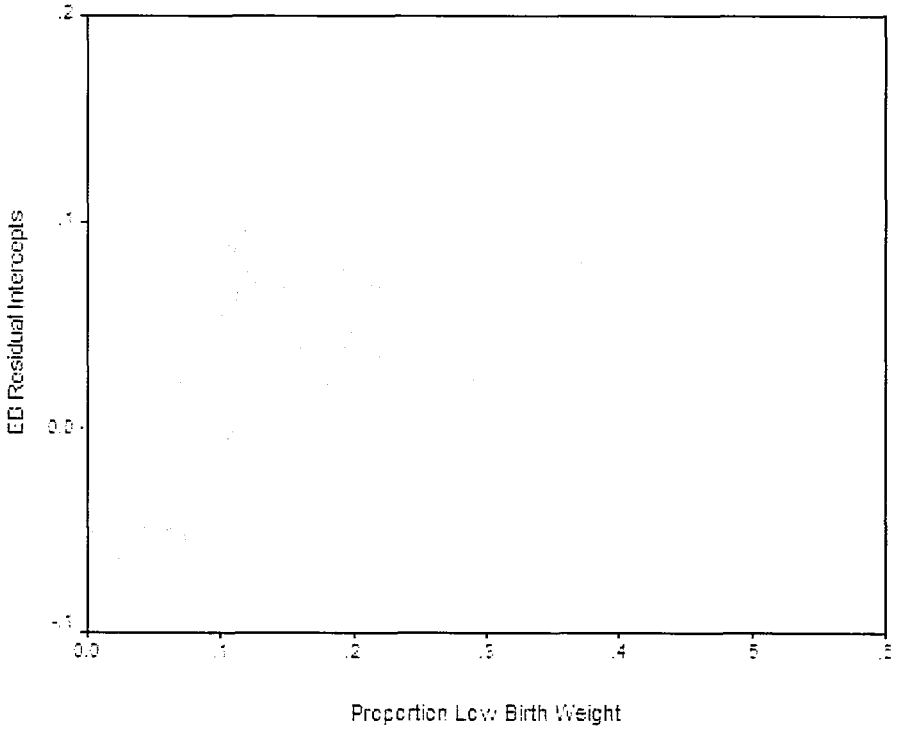


Figure 28 is a scatterplot of EB coefficients by proportion low birthweight births. The EB coefficients are the sum of the fitted values and the EB residuals. When the EB coefficients were converted to probabilities, the mean probability of low birthweight was 0.064 (range, 0.041 to 0.085). Thus, 95 percent of census tracts ranged between 4.1 and 8.5 percent, in terms of the rate of low birthweight. This compares to the range in proportion of low birthweight births (0 to 50 percent). Thus, using the proportion of low birthweight may over-emphasize census tract variability in New York City.

Figure 28: EB Coefficients for Low Birthweight Intercepts by Proportion Low Birthweight in Census Tracts

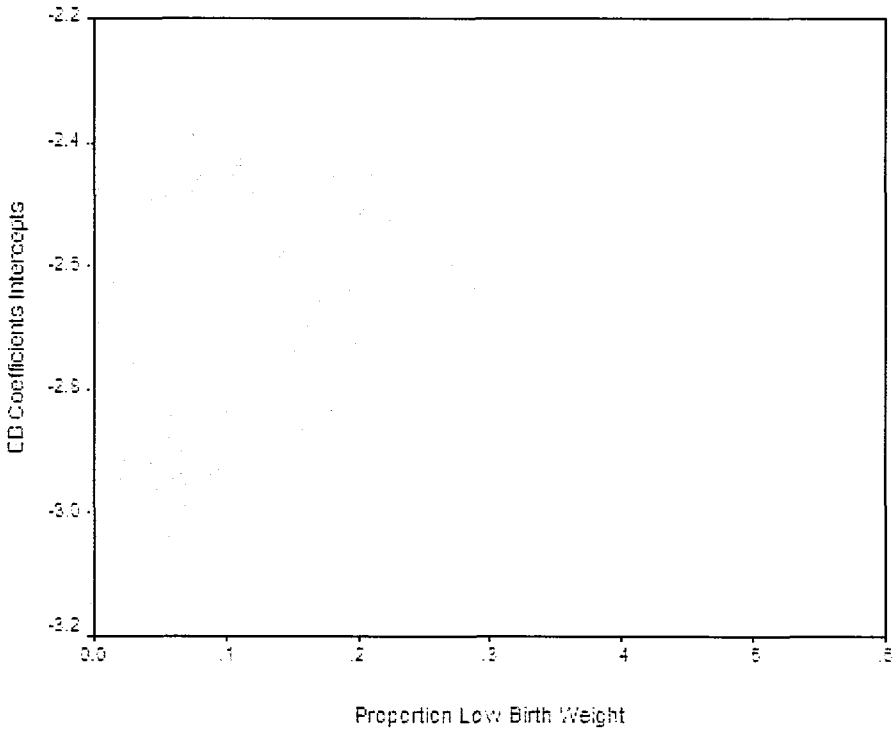


Figure 29 is a scatterplot of the EB residuals by SEGLOG. EB residuals in low-segregated census tracts lie along the mean suggesting little variability in the low birthweight means across census tracts. However, in higher- segregated census tract residuals were more widely dispersed around the mean, suggesting greater variability.

Figure 29: Plot of EB Intercept Residuals by Residential Segregation

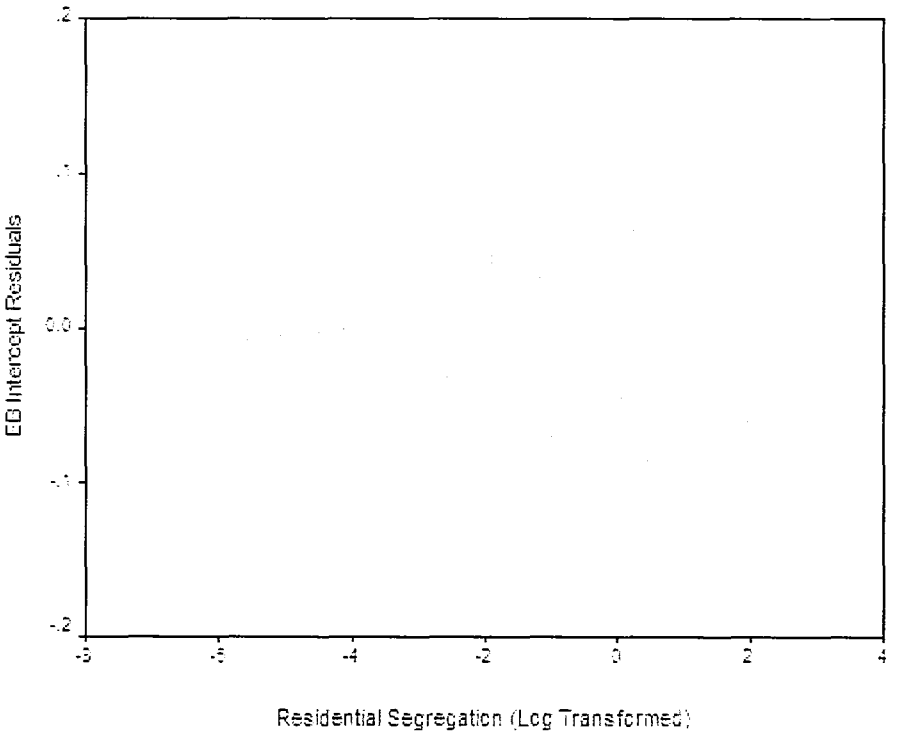


Figure 30: Plot of EB Coefficients by Residential Segregation.

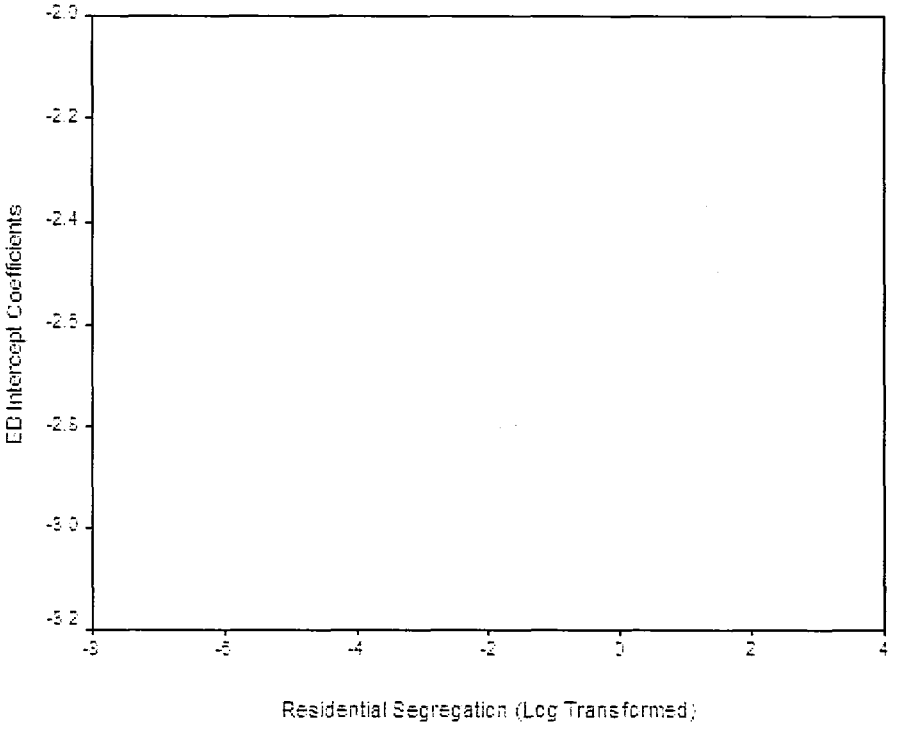
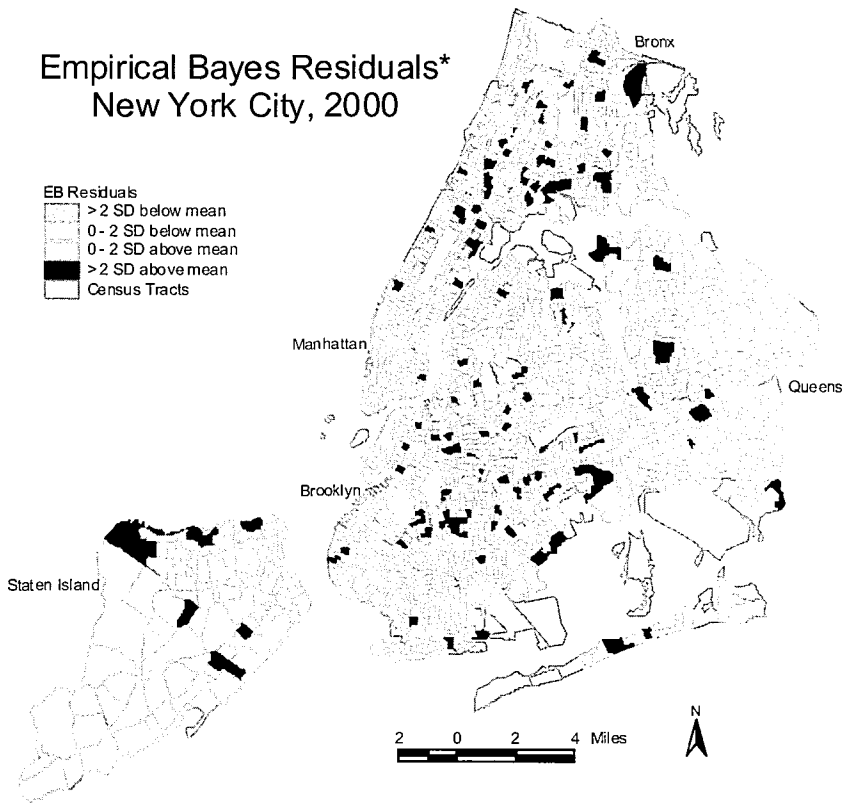


Figure 30 is a scatterplot of EB coefficients by residential segregation. There is a definite linear relationship between these two variables. When the EB coefficients are converted to probabilities, the mean probability of low birthweight is 0.076 (range, 0.041, 0.110). Thus, 95 percent of census tracts range between 4.1 and 11.0 percent, in terms of the rate of low birthweight.

Figure 31 is a map of the EB intercept residuals by census tract in New York City. The residuals refer to areas where low birthweight was not sufficiently explained by the model. The residual are classified by the number of standard deviations, above or below the mean. The dark colors represent those census tracts identified as having intercept residuals greater than or equal to two standard deviations above the mean. The lighter gray census tracts are those with intercept residuals greater than or equal to two standard deviations below the mean.

In total, there were 123 census tracts identified as having EB intercept residuals greater than or equal to two standard deviations above the mean. The mean rate of low birthweight in those census tracts was 14.87 (range, 5.85 to 36.84). Census tracts with high EB residuals were scattered throughout New York City, specifically in the Bronx (n=30), Brooklyn (n=48), Manhattan (n=13), Queens (n=22), and Staten Island (n=10). There is no clear spatial pattern or clustering in the residuals, indicating that the residuals are not spatially autocorrelated and that the model adequately represented spatial variation in low birthweight.

Figure 31: Map of EB Intercept Residuals by Census Tract, New York City, 2000



* Residuals from Level-2 Predictor for Intercepts and Slopes-as-Outcomes Model (Residential Segregation)

EPIDEMIOLOGICAL PARADOX

In general, the results from the analyses provided sufficient evidence that an *epidemiological paradox* exists in New York City. The logistic regression model demonstrated that U.S.-born women were at greater risk of low birthweight than foreign-born women, OR = 1/0.818 = 1.222. When African American women were stratified by origin of birth, U.S.-born African American women were almost twice as likely as white women to have a low birthweight birth. Foreign-born African American women were also more likely than white women to have a low birthweight birth but their risk was less than that of U.S.-born women. These risk differentials may be due to differences in the duration of exposure to residential segregation.

In models that controlled for residential segregation, being foreign-born was associated with a lower odds (OR = 0.792 (0.567, 1.107) of low birthweight than the odds of low birthweight for U.S.-born women. In addition, models that controlled for poverty, being foreign-born was associated with a lower odds (OR = 0.766 (0.548, 1.107)) of low birthweight for U.S.-born women. These findings demonstrated the persistent protection associated with being foreign-born despite living in disadvantaged (residential segregation and/or poverty) areas of New York City.

In models that controlled for residential segregation, there was no variability in origin of birth by census tract. However, in the Level-2 Predictor for Intercepts-as-Outcomes Model that controlled for poverty, there was significant variability in origin of birth by

census tract. This finding suggests that foreign-born and U.S.-born women lived apart from one-another within poverty environments but not racially segregated environments.

One more analysis was conducted to evaluate the *epidemiological paradox* in New York City. This model input the RACE*ORIGIN interaction term into a Level-2 Predictor for Intercepts-as-Outcomes Model to measure the differences in risk of low birthweight for U.S.-born (BLKUS) and foreign-born (BLKFB) African American women. The results from this analysis are presented in Table 23. The coefficients for BLKUS and BLKFB are both positive and statistically significant, indicating that both groups have a higher likelihood of low birthweight after controlling for individual-level risk factors and segregation. However, the coefficient for U.S.-born black women is double that for foreign-born black women which shows a substantially higher risk. This finding supports the *epidemiological paradox* and suggests that the health impacts of residential segregation may differ between U.S.-born and foreign-born black women.

Table 23: Level-2 Predictor for Intercepts-as-Outcomes Model for U.S.-Born and Foreign-Born African American Women, Controlling for Residential Segregation, New York City, 2000

	Fixed Effects	**ß***	***se***	**t-ratio**	**df**	**p-value**
Intercept, β_{0j}	Intercept, γ_{00}	-2.735	0.038	-70.244	2080	0.000
SEGLOG	Intercept, γ_{01}	0.067	0.011	6.151	2080	0.000
BLKUS, slope, β_{1j}	Intercept, γ_{10}	0.456	0.037	12.094	2081	0.000
BLKFB, slope, β_{2j}	Intercept, γ_{20}	0.224	0.039	5.627	2081	0.000
MARITAL, slope, β_{3j}	Intercept, γ_{30}	-0.288	0.034	-8.424	2081	0.000
COLLEGE, slope β_{4j}	Intercept, γ_{40}	-0.181	0.031	-5.671	2081	0.000
MEDICAID, slope, β_{5j}	Intercept, γ_{50}	0.085	0.033	2.554	2081	0.011
SMOKE, slope, β_{6j}	Intercept, γ_{60}	0.504	0.056	8.955	2081	0.000
SUBUSE slope, β_{7j}	Intercept, γ_{70}	0.995	0.086	11.552	2081	0.000
MAGE27, slope β_{8j}	Intercept, γ_{80}	0.015	0.002	6.749	2081	0.000
* Unstandardized coefficient						
	Standard	**Variance**				
Random Effect	**Deviation**	**Component**	**df**	X^2	**p-value**	
Intercept, μ_{0j}	0.296	0.087	408	424.105	0.281	
BLKUS, μ_{1j}	0.275	0.075	409	419.000	0.355	
BLKFB, μ_{2j}	0.353	0.125	409	433.792	0.191	
MARITAL, μ_{3j}	0.312	0.097	409	35.567	>0.500	
COLLEGE, μ_{4j}	0.173	0.030	409	419.186	0.353	
MEDICAID, μ_{5j}	0.144	0.020	409	369.298	>0.500	
SMOKE, μ_{6j}	0.362	0.131	409	401.622	>0.500	
SUBUSE, μ_{7j}	0.425	0.180	409	404.418	>0.500	
MAGE27, μ_{8j}	0.013	0.000	409	404.816	>0.500	

WEATHERING HYPOTHESIS

The *weathering hypothesis* predicts that African American women will experience health deterioration early in the life course due to their exposure to residential segregation and racism. Focusing on reproductive health, this health deterioration should be evidenced by an increase in the number of medical conditions or a more complex array of medical conditions during pregnancy at a relatively young age. These conditions are likely to have a negative impact on birth outcome. This section explores the *weathering hypothesis* by analyzing the risk of pre-pregnancy and pregnancy-related medical

conditions as a function of maternal age for black and white women in New York City. The following plots show racial disparities in medical conditions by age for African American and white women. The medical conditions presented are those that were significantly associated with low birthweight in the logistic regression analyses. In these models the medical conditions were not controlled for by other variables. Thus, they represent overall patterns.

Pre-pregnancy Medical Conditions

The pre-pregnancy medical conditions that will be examined by race and age group include:

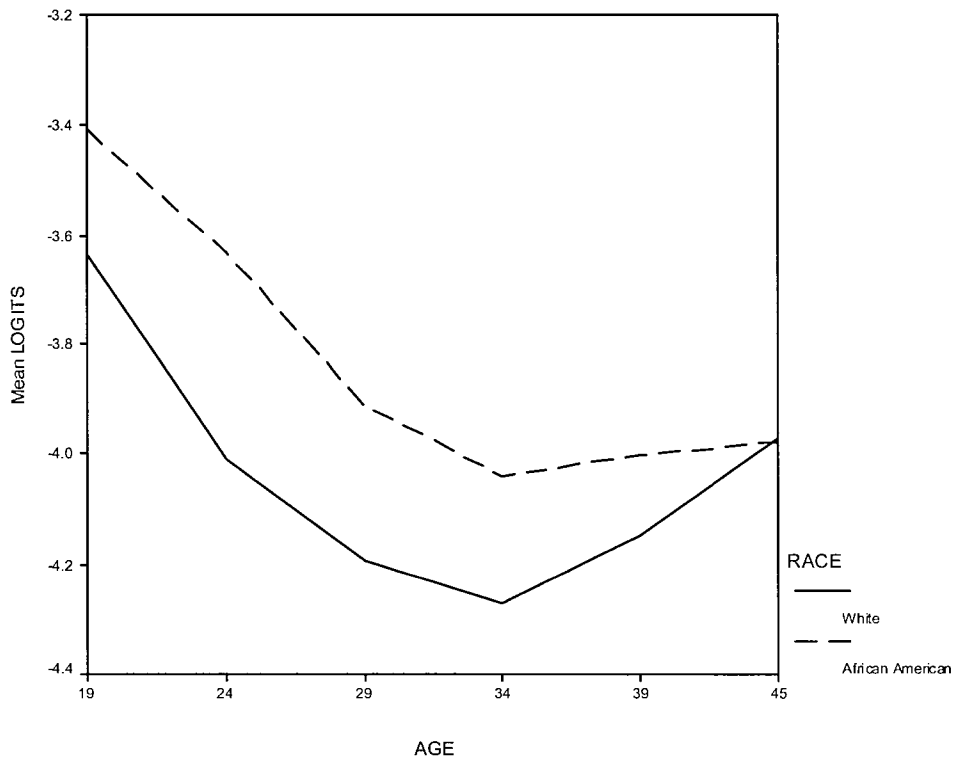
Lung disease
Diabetes, chronic
Hypertension, chronic
Renal disease
Thyroid disease
Viral disease
Previous preterm or small for gestational infant (SGA)
Previous miscarriage

Lung Disease

A plot of the log-odds of lung disease by age group showed an L shaped trend with the highest risk in the youngest age groups and declining risk until the mid-30s. Thereafter, risk levels off for African American women and rose slightly for white women. Racial disparities are present across the reproductive years. At the youngest age group the log-odds of lung disease for African American women was approximately –3.40, corresponding to an OR = 0.030, or about 1 in 33 births. In this same age group the log odds of lung disease for white women was approximately -3.65, corresponding to an OR

= 0.025, or about 1 in 40 births (Figure 32). The racial gap in lung disease risk is relatively constant through age 35, after which the gap diminishes. The large gap is consistent with the *weathering hypothesis*, which predicts increased health risks among young African American mother.

Figure 32: Log-Odds of Lung Disease by Race and Age Group

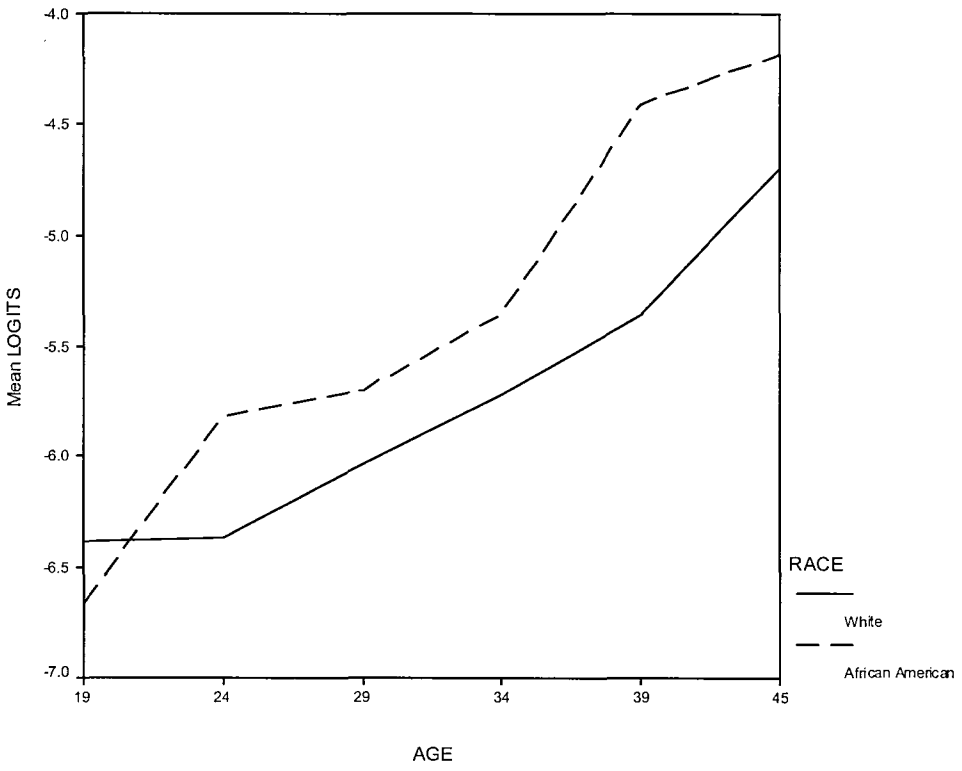


Diabetes, Chronic

A plot of the log-odds of chronic diabetes showed that risk was greater in the younger age group for white women, but thereafter, a rapid increase in risk was observed for African American women, exceeding that for white women over the remaining reproductive years. This rapid increase in the early reproductive years for African American women may be suggestive of the *weathering hypothesis*. At age 39 years the disparity in risk is

most pronounced. The log-odds of chronic diabetes for African American women at that age was approximately –4.50, corresponding to an OR = 0.011, or about 1 in 90 births. This was substantially greater than the log-odds of chronic diabetes in white women which was approximately -5.70, corresponding to an OR = 0.003, or about 1 in 333 births (Figure 33).

Figure 33: Log-Odds of Chronic Diabetes by Race and Age Group

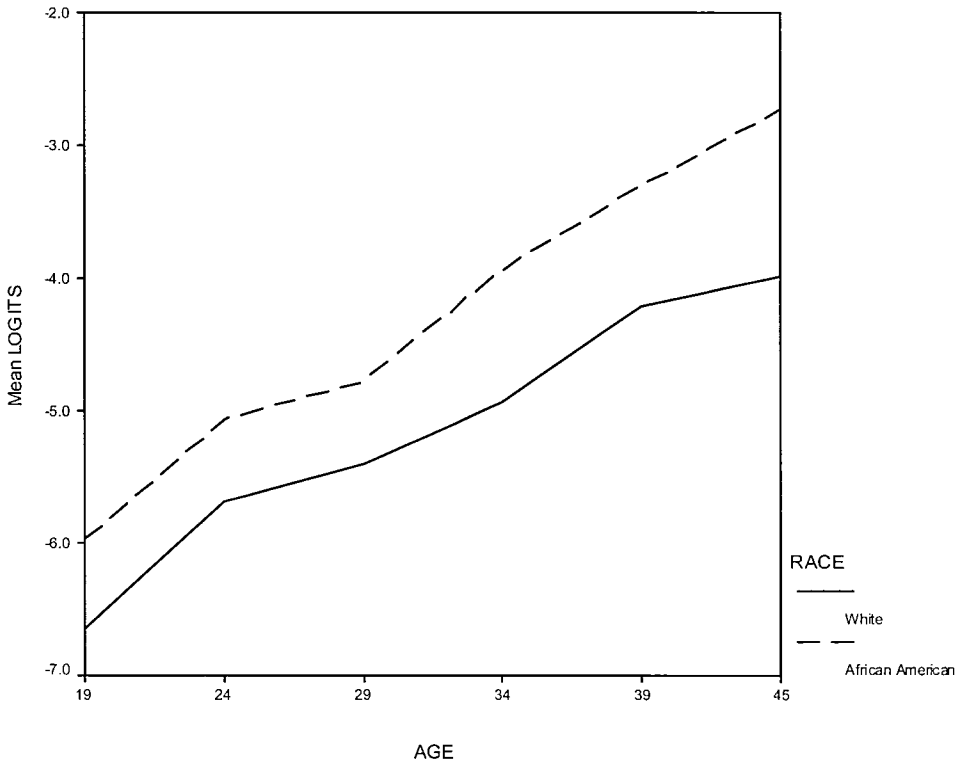


Chronic Hypertension

Chronic hypertension is one of the most important pre-pregnancy risk factor for low birthweight based on earlier analysis. For both racial groups the risk of chronic hypertension in the earliest age group was relatively low but increased over the reproductive years. There is consistent racial disparity in risk, with African-American women having higher risks across the age spectrum, as expected according to the

weathering hypothesis. The pattern was relatively similar to chronic diabetes with the widest disparity in risk around age 39 years. At that time the log-odds of chronic hypertension in African American women was approximately -3.20, corresponding to an OR = 0.04, or about 1 in 25 births. The log-odds for white women was approximately –4.20, corresponding to an OR = 0.014, or about 1 in 70 births (Figure 34).

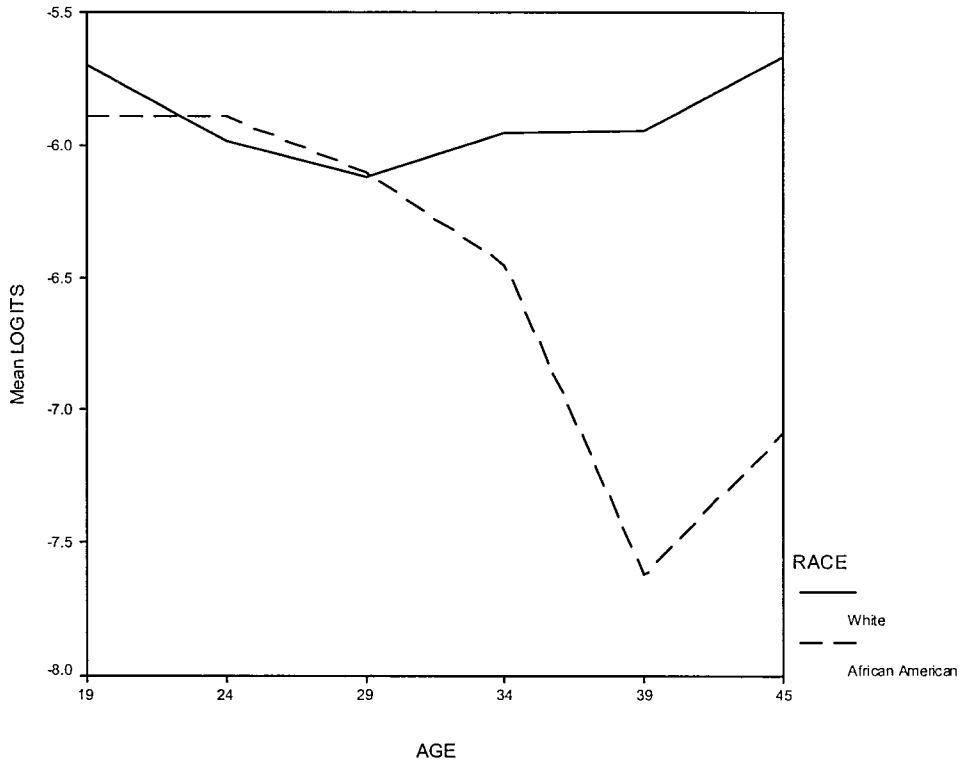
Figure 34: Log-Odds of Chronic Hypertension by Race and Age Group



Renal Disease

Renal disease shows a more complex pattern with no clear racial disparity. The log-odds of renal disease was approximately equal for African American and white women in the early reproductive years. However, by age 29 years risk decreased for African American women but remained virtually the same for White women (Figure 35).

Figure 35: Log-Odds of Renal Disease by Race and Age Group

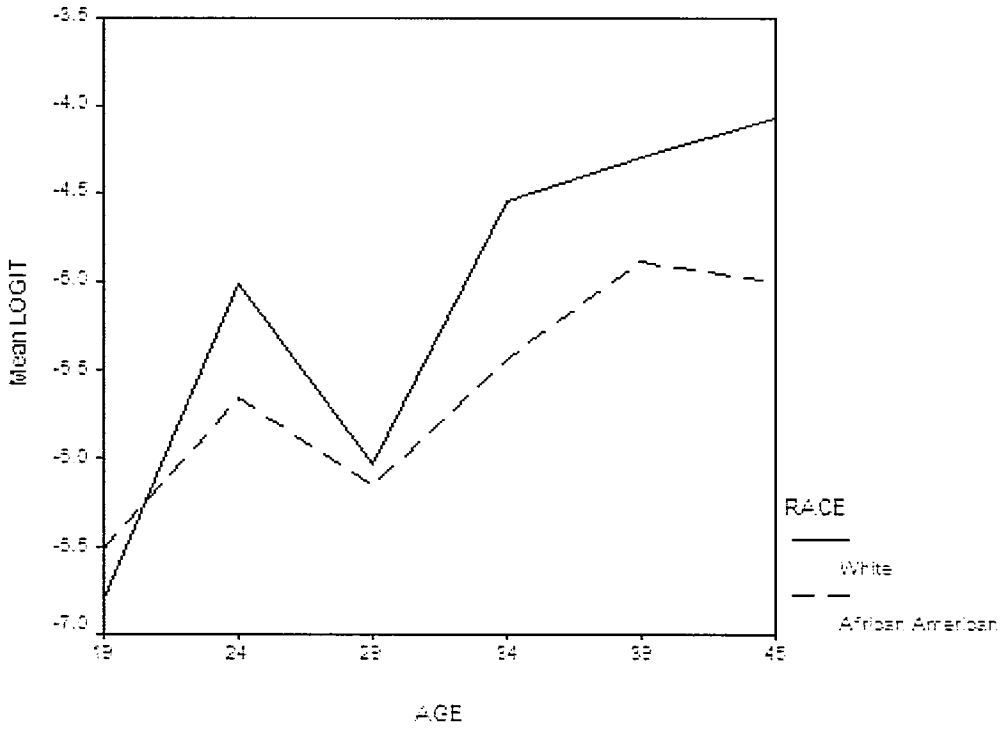


Thyroid Disease

The log-odds of thyroid disease was relatively low for both groups of women at the younger age groups with African American women at slightly greater risk than white

women. In general, risk increased over their reproductive years for both groups but risk was greater for white women (Figure 36).

Figure 36: Log-Odds of Thyroid Disease by Race and Age Group

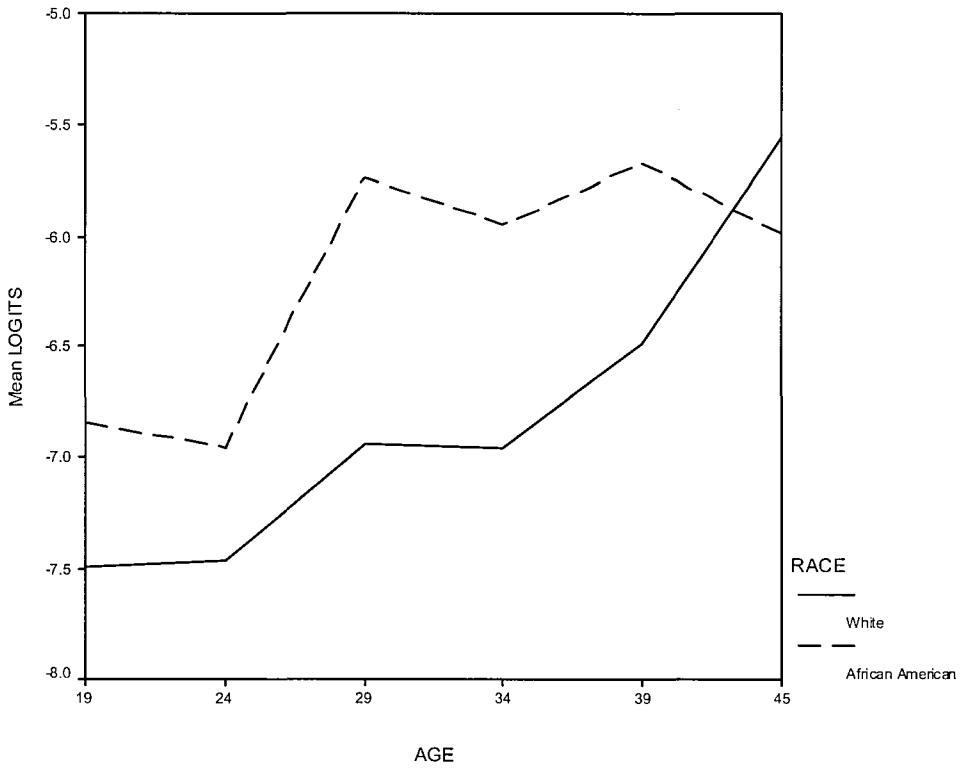


Viral Disease

The risk of viral disease was substantially greater for African American women than white women with the greatest disparity of risk around age 29 years and continuing across the reproductive years. At this age the risk of viral disease for African American women was approximately –5.7, corresponding to an OR = 0.003 or about 1 in 333 births. At the same age the risk of viral disease for white women was approximately –7.0, corresponding to an OR = 0.0009, or about 1 in 1110 births (Figure 37). In the oldest age group the risk of viral disease in white women surpassed that in African

American women, but these results may not be reliable due to the small number of births in this age group.

Figure 37: Log-Odds of Viral Disease by Race and Age Group

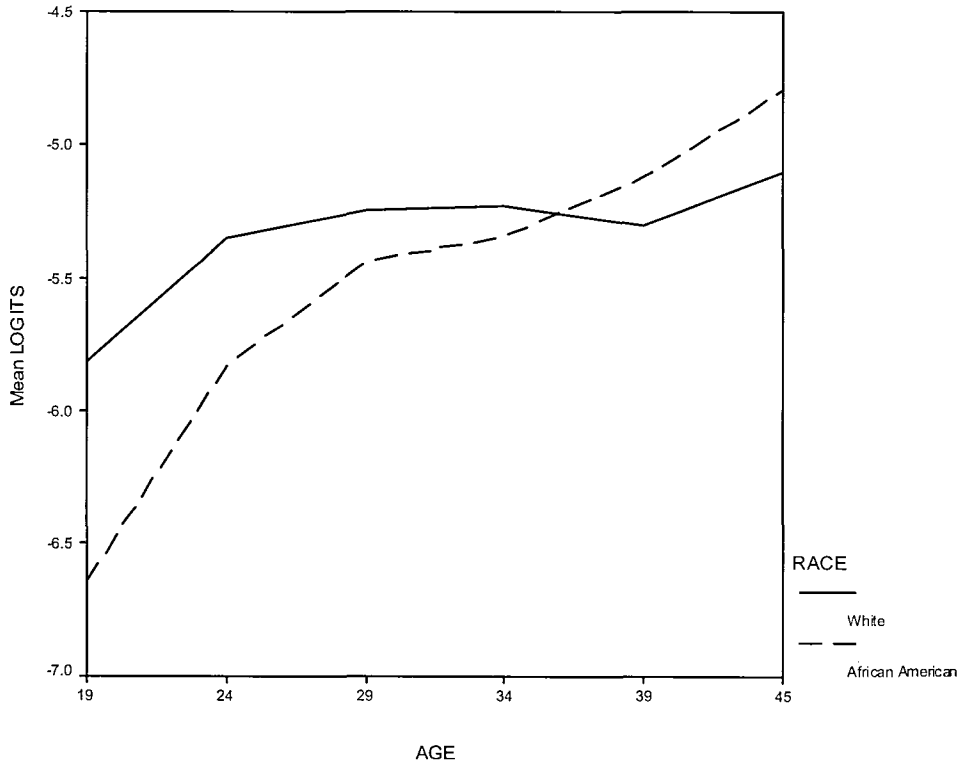


Previous Premature or SGA Infant

Having a premature or SGA infant in a previous pregnancy is the most important risk factor for low birthweight. As expected this risk is greater in the early age groups when women are less likely to have had a previous pregnancy. The risk is higher among White women until the mid-30s, at which time the risk for African American women exceeds that for White women (Figure 38). Although risk is lower for African American women

in the early age groups, the risk increases more rapidly with age than that for White women, possibly suggestive of a *weathering hypothesis.*

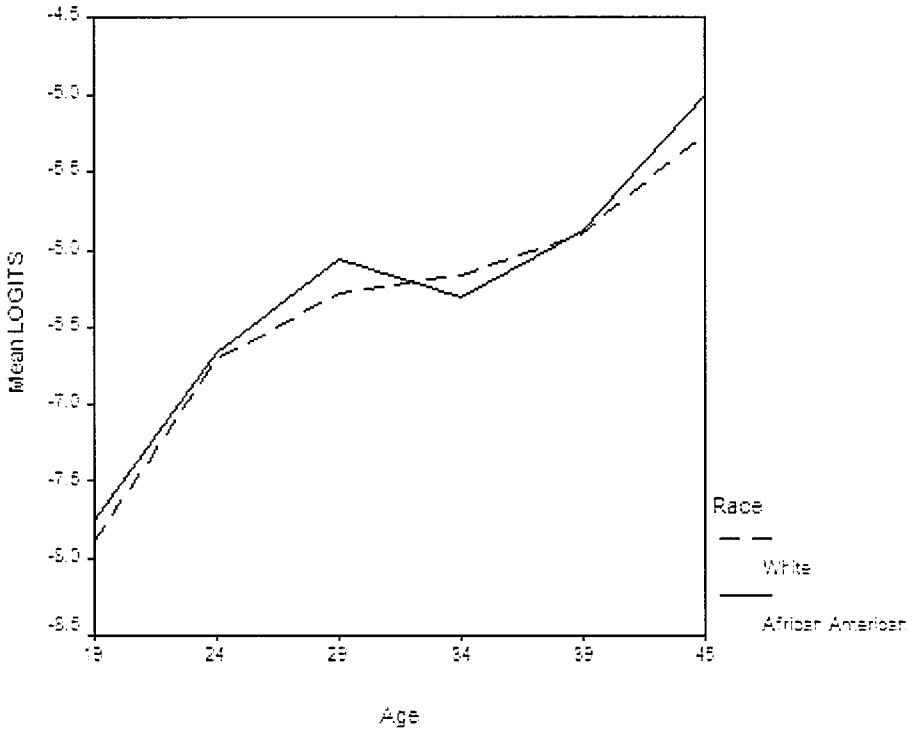
Figure 38: Log-Odds of Previous Preterm or SGA infant by Race and Age Group



Previous Miscarriage

Both groups of women were at low risk of having a previous miscarriage in the early age groups, with the greatest risk in the oldest age groups. This finding may partially be due to the fact that younger women have not had a previous birth but as age increases the likelihood of having a previous miscarriage increases. There is no apparent racial disparity in this medical condition (Figure 39).

Figure 39: Log-odds of Previous Miscarriage by Race and Age Group



PREGNANCY RELATED MEDICAL CONDITIONS

This section focuses on pregnancy-related medical conditions – conditions that arise during pregnancy that increase the risk of adverse birth outcomes for the mother and infant. The conditions that will be examined are those that were significantly associated with low birthweight in the logistic regression analysis.

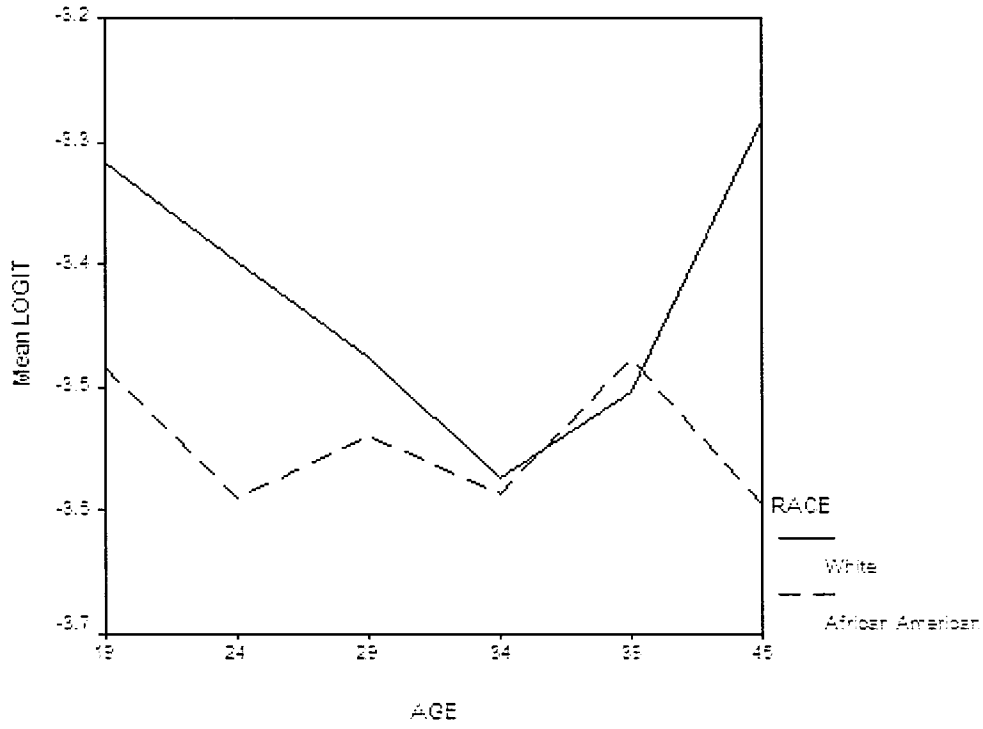
These include:

Hydramnios or olighydramnios
Hypertension, pregnancy related
Preeclampsia
Eclampsia
Incompetent cervix
Uterine bleeding in the second or third trimesters

Hydramnios/Olighydramnios

Hydramnios and olighydramnios are conditions where there is either an excess or reduction in the amount of amniotic fluid needed for the fetus to survive. The risk of hydramnios or olighydramnios is greatest in the early reproductive years and gradually declines until approximately age34 years. After that risk increases sharply for white women and moderately for African American women. After age39 risk decreases for African American women but increases sharply for white women (Figure 40). There is no consistent racial disparity in risk, although White women have higher risks at both younger and older ages.

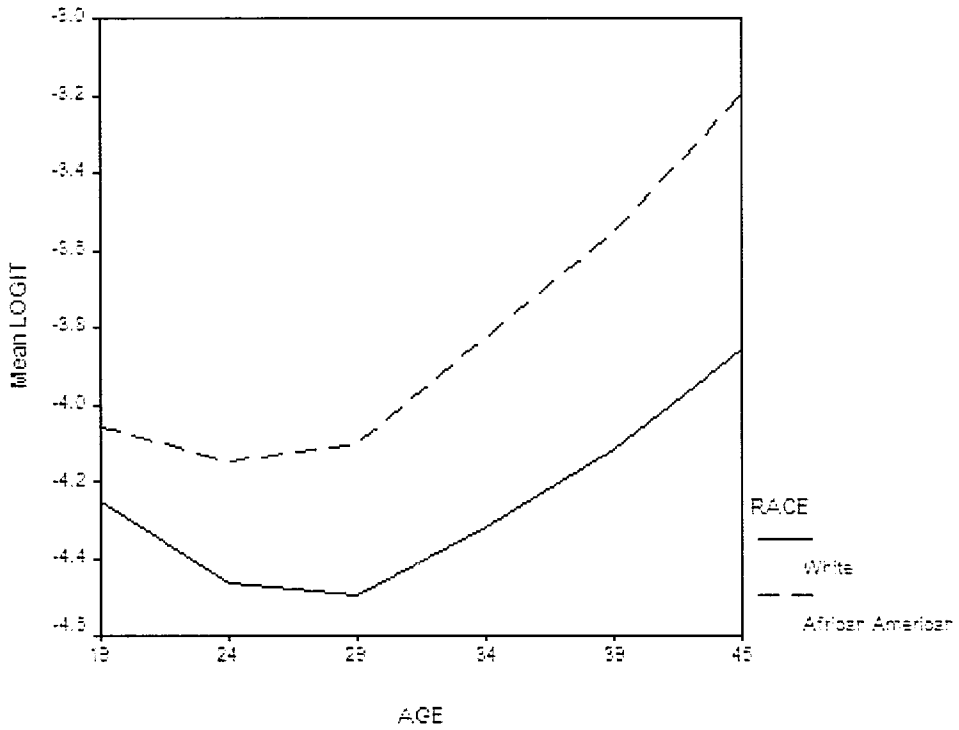
Figure 40: Log-Odds of Hydramnios or Olighydramnios by Race and Age Group



Pregnancy-Related Hypertension

Pregnancy-related hypertension is a sever hypertensive state diagnosed after the 20th week of pregnancy. Hypertension may restrict blood flow to the fetus resulting in premature birth or reduced fetal growth. There is substantial disparity in risk of pregnancy-related hypertension at all age groups, with African American mothers having substantially higher risk. Risks are most similar in the earliest age group at which time risk decreases for white women and remains steady for African American women until age29 years at which time risk increases for both races. At age 29 the risk of pregnancy-related hypertension for African American women was approximately -4.02, corresponding to an OR = 0.017, or about 1 in 55 births. The risk for white women was approximately -4.25, corresponding to an OR = 0.014, or about 1 in 70 births. Thus, young African American women were at greater risk of pregnancy-related hypertension than young white women (Figure 41). These findings supported the weathering hypothesis.

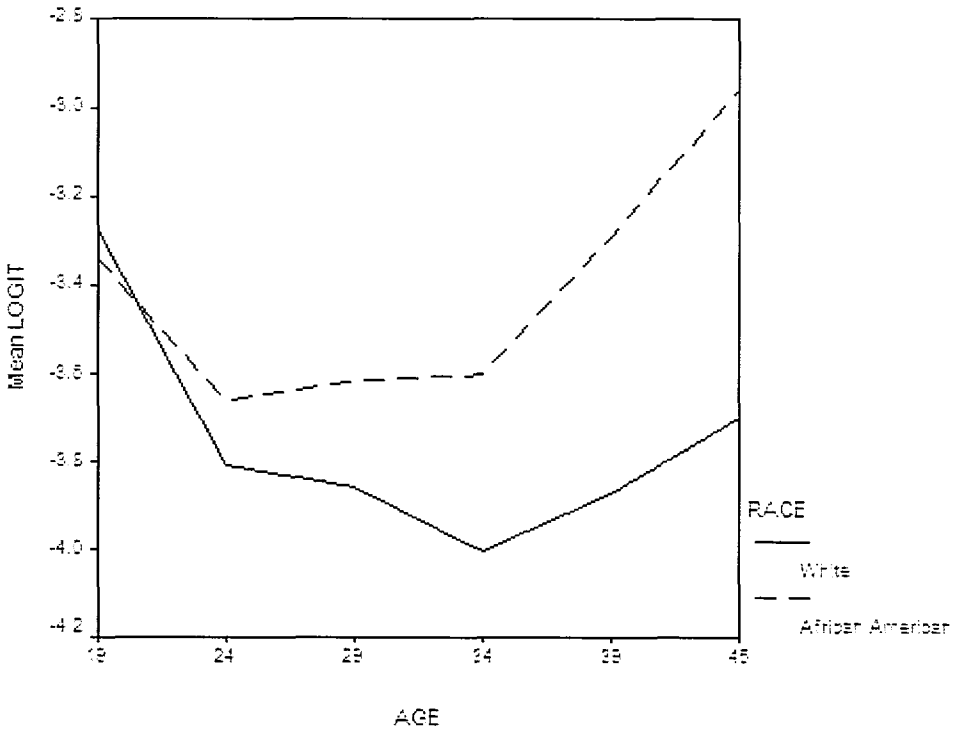
Figure 41: Log-Odds of Pregnancy-Related Hypertension by Race and Age Group



Preeclampsia

Preeclampsia is severe hypertension that occurs during pregnancy, which also includes symptoms of proteinuria (protein in the urine) and edema (swelling). Untreated preeclampsia can cause premature birth. The risk of preeclampsia is higher for women in their early reproductive years but drops off until age 24 years for both races. By age 35 years there is substantial racial disparity. At age 39 years the log-odds of preeclampsia for African American women was –3.2, corresponding to an OR = 0.040, or about 1 in 25 births. The log-odds for white women at the same age was approximately – 3.9, corresponding to an OR = 0.020, or about 1 in 50 women. African American women are twice as likely as white women to have preeclampsia in their late 30s. This finding may represent further evidence of the *weathering hypothesis* (Figure 42).

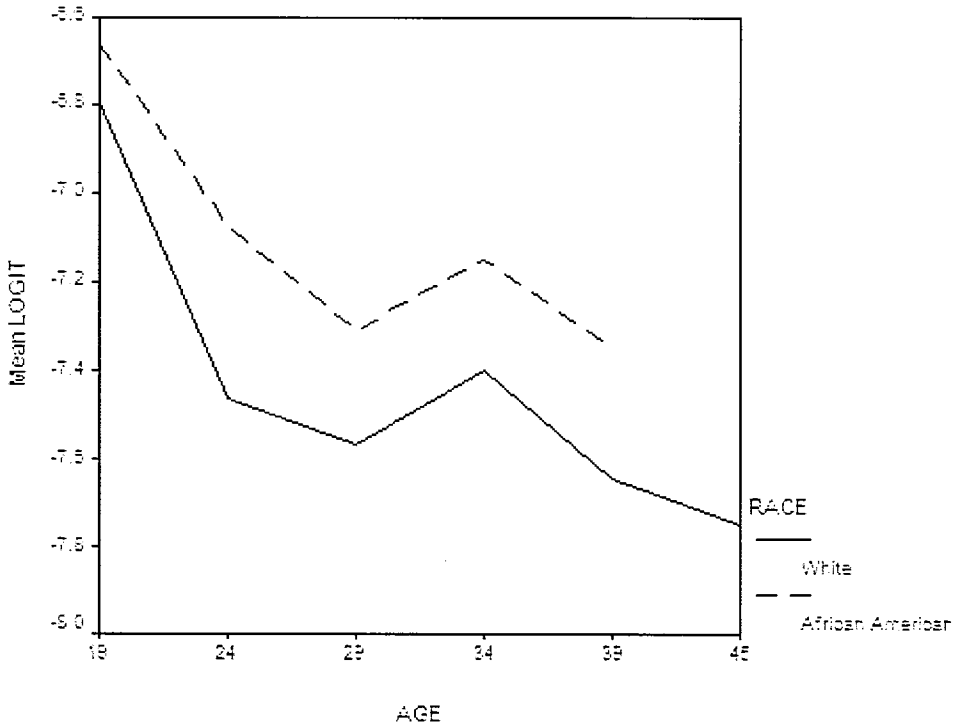
Figure 42: Log-Odds of Preeclampsia by Race and Age Group



Eclampsia

Eclampsia is characterized by preeclampsia but also includes convulsions and/or coma. Women with eclampsia are at very high risk of having a premature birth. The risk of eclampsia is greatest in the early reproductive years for both groups of women. Risk sharply declines until age29 years and then increases slightly. After age34 years the risk again begins to decline over the remainder of the reproductive years (Figure 43).

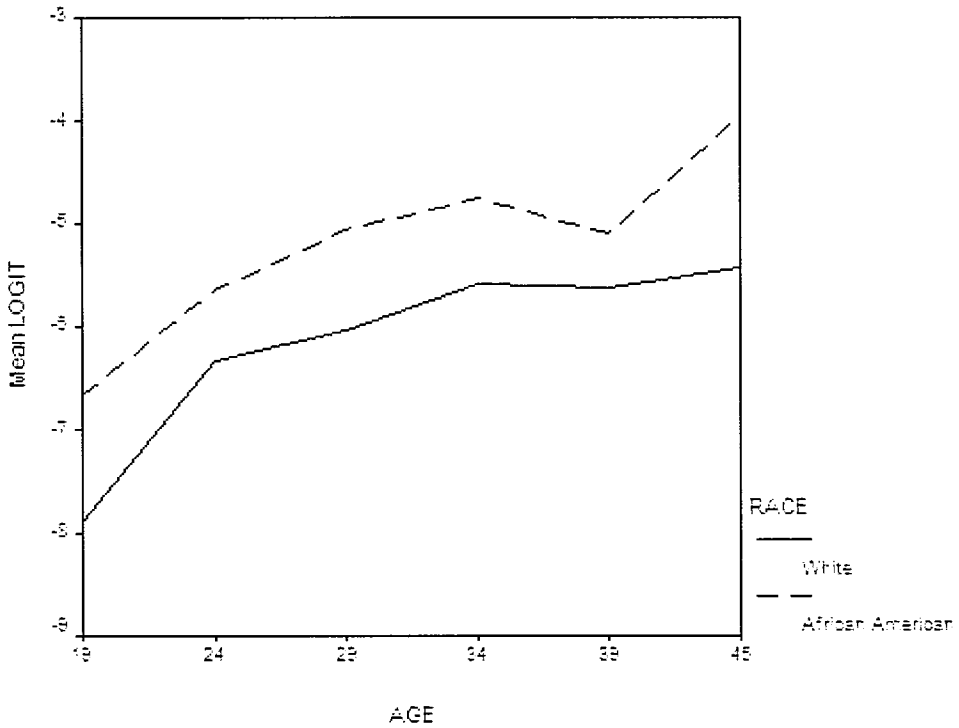
Figure 43: Log-Odds of Eclampsia by Race and Age Group



Incompetent Cervix

An incompetent cervix may be due to infection, prior cervical instrumentation (e.g., following miscarriage or abortion) or idiopathic. If the cervix dilates before the end of gestation the infant will be born prematurely. The risk of developing an incompetent cervix increases gradually over the reproductive years for both African American and white women, with greater risk for African American women (Figure 44).

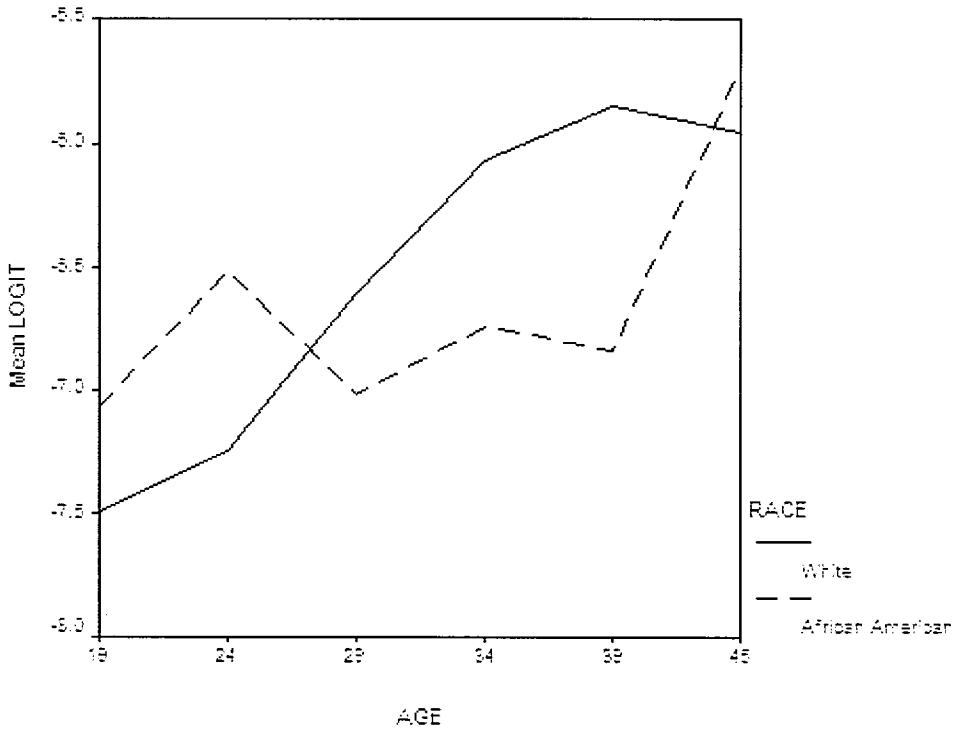
Figure 44: Log-Odds of Incompetent Cervix by Race and Age Group



Uterine Bleeding 2nd Trimester

Uterine bleeding in the second trimester of pregnancy is an important risk factor for prematurity and low birthweight. The risk of this condition is greater for African American women in the early and later reproductive years with the middle years relatively moderate. The risk for white women gradually increases over the reproductive life cycle and is greatest in the older age group, exceeding that for black women (Figure 45). There is no consistent racial disparity in this condition.

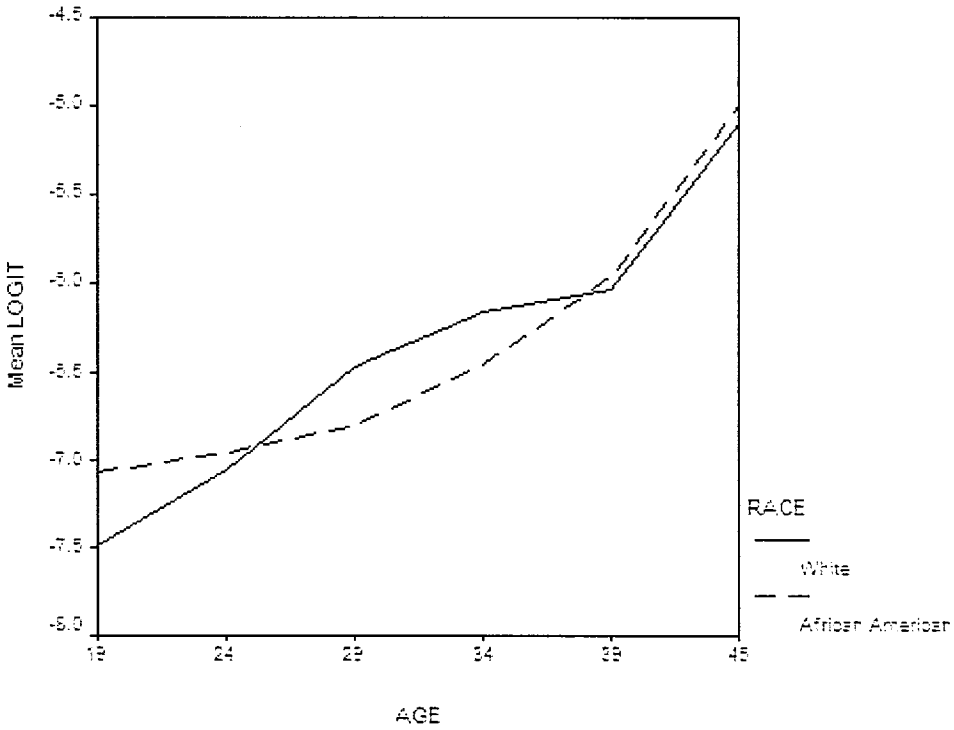
Figure 45: Log-Odds of Uterine Bleeding 2nd Trimester by Race and Age Group



Uterine Bleeding 3rd Trimester

The risk of uterine bleeding in the third trimester of pregnancy is greater for African American women until age 24 years at which time the risk for white women exceeds the risk for African American women. This risk gradually increases over the reproductive years for both groups of women (Figure 46). Overall, the difference in risks between the two racial groups is small.

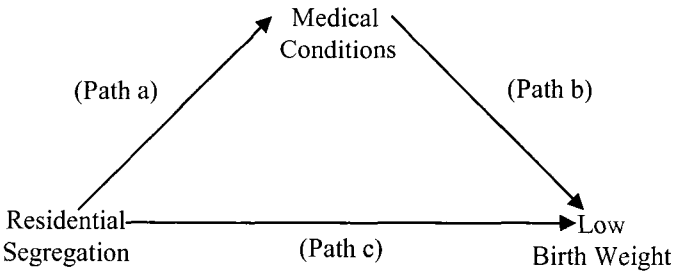
Figure 46: Log-Odds of Uterine Bleeding 3rd Trimester by Race and Age Group



MEDICAL CONDITIONS AS MEDIATORS

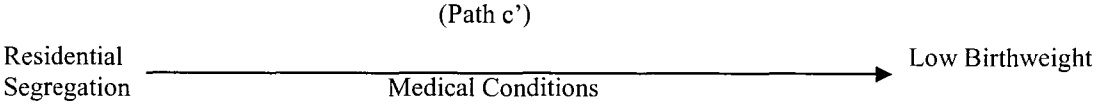
As discussed earlier, one of the aims of this research is to determine if medical conditions mediate the residential segregation and low birthweight relationship. The multilevel model results showed that segregation has a statistically significant association with low birthweight after individual risk factors are controlled. Does this association exist because segregation increases the risk of adverse medical conditions, which in turn heighten the risk of low birthweight for African American women? Or is the pathway primarily direct? Those medical conditions identified as significant predictors in the logistic regression models will be used to explain why residential segregation contributes to increasing rates of low birthweight.

These analyses were conducted on African American women only since they live in residentially segregated areas. The first step in testing for mediation was to show that there is a significant relationship between residential segregation and low birthweight (Path c). The previous section demonstrated that residential segregation was associated with higher rates of low birthweight, while controlling for MRACE, ORIGIN, MARITAL, COLLEGE, MAGE27, SMOKE, ALCOHOL, and SUBUSE. The models that were used in this analysis were similar to previous models, except the variables SMOKE and SUBUSE were removed because they both cause certain medical conditions and low birthweight. Thus, keeping them in the model could eliminate the mediating effects of medical conditions. Path c was used as a reference model from which subsequent models were compared.



The second step in testing for mediation is to show that residential segregation is related to the possible mediators (medical conditions) (Path a). Two-stage multilevel modeling was conducted to identify those medical conditions predicted by residential segregation.

The third step in testing for mediation is to show that the mediator (medical conditions) was related to low birthweight (Path b). Path b was estimated controlling for the effects of residential segregation. The final Path c' measures the change in effect of residential segregation on low birthweight after the mediator (medical conditions) is added to the model. Path c' is estimated using the Path b model.



The significance of the mediating effect was measured as outlined in the Chapter 3. The pre-pregnancy and pregnancy-related medical conditions that were input into this analysis were those significantly associated with low birthweight in the logistic regression analysis and plotted by race and age in the previous section on the *weathering hypothesis*. Path c will be presented first, and as mentioned, will be used as a comparison for all future models.

Path c -

Table 24 presents the results from the Path c analysis. As in previous models, there was a positive association between residential segregation and low birthweight; therefore, (Path c) was validated and further analyses could be conducted to test for mediating effects. In this Path c model, the expected log-odds of low birthweight was –2.137, corresponding to an OR = 0.118 (0.075, 0.183). SEGLOG was positively associated with increasing rates of low birthweight = 0.054. Translating SEGLOG back to SEG the expected log-odds was 1.055, corresponding to an OR = 2.871. Thus, a unit- increase in SEG increased odds of low birthweight by 2.871.

Table 24: Risk of Residential Segregation on Low Birthweight (Path c)

Fixed Effects	**ß***	***se***	**t-ratio**	**df**	**p-value**
Step 1 (Path c)					
Outcome: LBW	-2.137	0.051	-41.915	1711	0.000
Predictor: SEGLOG	0.054	0.019	2.735	1711	0.007
Controls: ORIGIN	-0.389	0.039	-9.884	34554	0.000
MARITAL	-0.254	0.046	-5.449	34554	0.000
COLLEGE	-0.180	0.042	-4.221	34554	0.000
MEDICAID	0.149	0.045	3.291	34554	0.001
MAGE27	0.031	0.003	10.578	34554	0.000
Random Effects: $\sigma^2 = 0.045$ $\tau_{00} = 0.002$ df = 1711 $X^2 = 1685.337$ p-value = >0.5					
*Unstandardized coefficient					

The other individual-level coefficients were in the same direction and of similar magnitude to that observed in the previous models predicting low birthweight, while controlling for residential segregation.

Lung Disease

In the Path a model, residential segregation was not significantly associated with lung disease; therefore, this medical condition was not studied further.

Chronic Diabetes

Residential segregation was also not significantly associated with chronic diabetes in the Path a model; therefore no further analyses were conducted on this medical condition.

Chronic Hypertension

Residential segregation was significantly associated with chronic hypertension in African American women; therefore, the analysis of chronic hypertension as a mediator was continued. The results are presented in Table 25. In the Path a model for chronic hypertension, the expected log-odds was –4.669, corresponding to an OR = 0.009 (0.004, 0.019), or about 1 in 111 pregnant women. The expected log-odds of SEGLOG γ_{01} = 0.122. Translating SEGLOG back to SEG the expected log-odds was 1.129, corresponding to an OR = 3.092. Thus, a unit- increase in SEG increased the odds of chronic hypertension by 3.092. The coefficients for the individual-level predictors showed that marital status and college education were not significantly associated with higher rates of chronic hypertension. Foreign-born African American women were at slightly lower risk of chronic hypertension (OR = 0.644) and women on Medicaid were at slightly higher risk (OR = 1.462).

Table 25: Mediating Effects of Chronic Hypertension on Residential Segregation and Low Birthweight Relationship (Paths a, b, and c')

Fixed Effects	**ß***	***se***	**t-ratio**	**df**	**p-value**
Step 2 (Path a)					
Outcome: Chronic Hypertension	-4.669	0.131	-35.579	1711	0.000
Predictor: SEGLOG	0.122	0.050	2.446	1711	0.015
Controls: ORIGIN	-0.439	0.094	-4.670	35444	0.000
MARITAL	-0.032	0.101	-0.323	35444	0.746
COLLEGE	0.009	0.098	0.094	35444	0.925
MEDICAID	0.380	0.106	3.569	35444	0.001
MAGE27	0.140	0.007	19.388	35444	0.000
Random Effects: $\sigma^2 = 0.181$ $\tau_{00} = 0.032$ df = 1711 $X^2 = 1552.306$ p-value = >0.5					
Step 3 (Paths b and c')	ß*	*se*	t-ratio	df	p-value
Outcome: LBW	-2.162	0.051	-42.271	1711	0.000
Predictor: SEGLOG	0.051	0.019	2.599	1711	0.010
Mediator: Hypertension (Path b)	1.154	0.104	11.069	35443	0.000
Controls: ORIGIN	0.378	0.039	-9.562	354443	0.000
MARITAL	-0.251	0.046	-5.372	35443	0.000
COLLEGE	-0.178	0.042	-4.168	35443	0.000
MEDICAID	0.137	0.045	3.017	35443	0.003
MAGE27	0.027	0.003	9.155	35443	0.000
Random Effects: $\sigma^2 = 0.043$ $\tau_{00} = 0.001$ df = 1711 $X^2 = 1694.913$ p-value = >0.5					
*Unstandardized coefficient					

(*Path b*)

Chronic hypertension was positively associated with higher rates of low birthweight, (log-odds = 1.154), corresponding to an OR = 3.170.

(Path c')

In Path c', the expected log-odds of SEGLOG was 0.051, after controlling for individual-level risk factors This result was slightly lower that that observed in Path c = 0.054, controlling for the same individual-level risk factors. To determine if this difference was significant, the product of the coefficients for Paths a and b were divided by the standard error = (0.140)/0.057 = 2.469. The mediated effect was a z score greater than 1.96 at a

0.05 significance level. The confidence interval around the indirect effect of chronic hypertension was 0.028, 0.251. This confidence interval does not include zero, which is consistent with the conclusion that there is mediation. Thus, chronic hypertension partially explains the pathway by which residential segregation contributes to higher rates of low birthweight.

Renal Disease

The effect of SEGLOG on renal disease (Path a) was not significant (p-value = 0.178); therefore, renal disease could not be a mediator in the residential segregation and low birthweight relationship and was removed from further analyses.

Thyroid

The expected log-odds of SEGLOG on thyroid disease was -0.074, corresponding to an OR = 0.928. Since SEGLOG did not increase the risk of thyroid disease in pregnant women this medical conditions was also removed from further analyses.

Viral Disease

The effect of SEGLOG on viral disease (Path a) was not significant (p-value = 0.073); therefore, viral disease could not be a mediator and was removed from further analyses.

Previous Preterm or SGA Infant

The effect of SEGLOG on previous preterm or SGA infant (Path a) was also not significant (p-value = 0.101); therefore, this medical condition was removed from further analyses.

Previous Miscarriage

The effect of SEGLOG on having a previous miscarriage was not significant in Path a (p-value 0.824); therefore, this medical condition could not be a mediator and was not studied further.

Hydramnios/Olighydramnios

The effect of SEGLOG on hydramnios/olighydramnios (Path a) was not significant (p-value = 0.694); therefore, this medical condition was removed from further analyses.

Pregnancy-Related Hypertension

The effect of residential segregation on pregnancy-related hypertension was significant; therefore, the analysis of possible mediator was continued. The results are presented in Table 26. In Path a, the expected log-odds of having pregnancy related hypertension was –3.93, corresponding to an OR = 0.019 (0.010, 0.037), or about 1 in 52 pregnant women. The expected log-odds of SEGLOG γ_{01} was = 0.90. Translating SEGLOG back to SEG the expected log-odds was 1.094, corresponding to an OR = 2.986. Thus, a unit- increase in SEG increased the odds of pregnancy-related hypertension by 2.986.

(Path b)

Having pregnancy-related hypertension was positively associated with higher rates of low birthweight, $\gamma_{05} = 0.887$, corresponding to an OR = 2.492 (1.314, 4.484).

(Path c')

In Path c', the expected log-odds of low birthweight was the same as in Path b, γ_{00} – 2.162, corresponding to an OR = 0.114 (0.073, 0.179). SEGLOG was positively associated with increasing rates of low birthweight, γ_{10} 0.052. To determine if the decrease in the log-odds of SEGLOG from Path c = 0.054 was significant, the product of the coefficients for Paths a and b were divided by the standard error = 0.079/0.039 = 2.073. The mediated effect was a *z* score greater than 1.96 at a 0.05 significance level. The confidence interval around the indirect effect of pregnancy-related was 0.004 and 0.155. This confidence interval does not include zero, which is consistent with the conclusion that there is mediation.

Table 26: Mediating Effect of Pregnancy-Related Hypertension on Residential Segregation and Low Birthweight Relationship (Paths a, b, and c')					
Step 2 (Path a)	**ß***	***se***	**t-ratio**	**df**	**p-value**
Outcome: Pregnancy- Hypertension	-3.930	0.105	-37.245	1711	0.000
Predictor: SEGLOG	0.090	0.042	2.146	1711	0.032
Controls: ORIGIN	-0.190	0.081	-2.344	35444	0.019
MARITAL	0.030	0.089	0.337	35444	0.736
COLLEGE	0.078	0.085	0.929	35444	0.353
MEDICAID	-0.053	0.089	-0.592	35444	0.554
MAGE27	0.037	0.006	6.015	35444	0.000
Random Effects: $\sigma^2 = 0.236$ $\tau_{00} = 0.055$ df = 1711 $X^2 = 1711.183$ p-value = 0.494					
Step 3 (Paths b and c')	**ß***	***se***	**t-ratio**	**df**	**p-value**
Outcome: LBW	-2.162	0.051	-42.227	1711	0.000
Predictor: SEGLOG	0.052	0.019	2.630	1711	0.009
Mediator: Hypertension (Path b)	0.887	0.098	8.999	35443	0.000
Controls: ORIGIN	0.385	0.039	-9.771	35443	0.000
MARITAL	-0.255	0.046	-5.472	35443	0.000
COLLEGE	-0.182	0.042	-4.255	35443	0.000
MEDICAID	0.151	0.045	3.323	35443	0.001
MAGE27	0.030	0.003	10.265	35443	0.000
Random Effects: $\sigma^2 = 0.039$ $\tau_{00} = 0.001$ df = 1711 $X^2 = 1675.799$ p-value = >0.5					
*Unstandardized coefficient					

These findings showed that both chronic hypertension and pregnancy-related hypertension are pathways by which residential segregation is associated with low birthweight. They are both hypertensive medical conditions that are likely stress-related.

Preeclampsia

The effect of residential segregation was also significant for preeclampsia, a severe hypertensive disorder. The results from these analyses are presented in Table 27. In Path a, the expected log-odds of preeclampsia was –3.652, corresponding to an OR = 0.025 (0.014, 0.046), or about 1 in 40 pregnant women. SEGLOG was positively associated with increasing rates of preeclampsia; therefore, the analysis of preeclampsia as a mediator was continued. The expected log-odds of SEGLOG γ_{01} was = 0.71. Translating

SEGLOG back to SEG the expected log-odds was 1.073, corresponding to an OR = 2.925. Thus, a unit- increase in SEG increased the odds of preeclampsia by 2.925.

(Path b)

Having preeclampsia was positively associated with higher rates of low birthweight γ_{05} = 1.775, corresponding to an OR = 5.90 (3.525, 9.873). Thus women are at substantially higher risk of low birthweight if they develop preeclampsia during pregnancy.

(Path c')

In Path c', the expected log-odds of low birthweight was the same as in Path b, γ_{00} – 2.219, corresponding to an OR = 0.108 (0.069, 0.169). SEGLOG was positively associated with increasing rates of low birthweight γ_{10} 0.050. To determine if this decrease in the log-odds of SEGLOG from 0.054 in Path c to 0.050 in Path c' was significant, the product of the coefficients for Paths a and b were divided by the standard error = (0.126)/0.061 = 2.065. The mediated effect was a *z* score greater than 1.96 at a 0.05 significance level. The confidence interval around the indirect effect was 0.005 and 0.246. This confidence interval does not include zero, which is consistent with the conclusion that there is mediation.

Table 27: Mediating Effect of Preeclampsia on Residential Segregation and Low Birthweight Relationship (Paths a, b, and c')					
Step 2 (Path a)	**ß***	***se***	**t-ratio**	**df**	**p-value**
Outcome: Preeclampsia	-3.652	0.090	-40.360	1711	0.000
Predictor: SEGLOG	0.071	0.034	2.036	1711	0.042
Controls: ORIGIN	-0.031	0.067	-0.471	35444	0.637
MARITAL	-0.272	0.079	-3.437	35444	0.637
COLLEGE	0.083	0.071	1.173	35444	0.241
MEDICAID	0.186	0.078	2.391	35444	0.017
MAGE27	0.019	0.005	3.710	35444	0.000
Random Effects: $\sigma^2 = 0.198$ $\tau_{00} = 0.039$ df = 1711 $X^2 = 1662.504$ p-value = >0.5					
Step 3 (Paths b and c')	**ß***	***se***	**t-ratio**	**df**	**p-value**
Outcome: LBW	-2.219	0.051	-42.853	1711	0.000
Predictor: SEGLOG	0.050	0.020	2.498	1711	0.013
Mediator: Preeclampsia(Path b)	1.775	0.069	25.698	35443	0.000
Controls: ORIGIN	-0.397	0.039	-9.940	35443	0.000
MARITAL	-0.235	0.047	-4.987	35443	0.000
COLLEGE	-0.191	0.043	-4.431	35443	0.000
MEDICAID	0.136	0.045	2.970	35443	0.003
MAGE27	0.030	0.003	10.140	35443	0.000
Random Effects: $\sigma^2 = 0.046$ $\tau_{00} = 0.002$ df = 1711 $X^2 = 1693.254$ p-value = >0.5					
*Unstandardized coefficient					

Very importantly, these findings now show that three hypertensive disorders in African American mediate the relationship between residential segregation and higher rates of low birthweight. All three conditions are substantial risk factors for premature birth and low birthweight.

Eclampsia

The expected log-odds of SEGLOG on eclampsia (Path a) was not significant (p-value = 0.991); therefore, this medical condition was removed from further analyses.

Incompetent Cervix

The expected log-odds of SEGLOG on incompetent cervix (Path a) was not significant (p-value = 0.137); therefore, this medical condition was removed from further analyses.

Uterine Bleeding 2nd and 3rd Trimesters

The expected log-odds of SEGLOG on uterine bleeding in the second trimester (Path a) was not significant (p-value = 0.525); therefore, this medical condition was also removed from further analyses. The expected log-odds of SEGLOG on uterine bleeding in the 3rd trimester (Path a) was also not significant (p-value = 0.710); therefore, this medical condition was removed from further analyses.

Comparison of Mediated Effects Models

The medical conditions that were identified as mediators in the residential segregation and low birthweight relationship were chronic hypertension, pregnancy-related hypertension, and preeclampsia. The other pre-pregnancy and pregnancy-related medical conditions that significantly predicted low birthweight in the logistic regression analyses did not meet the criteria for mediation because they were not statistically associated with residential segregation. Women who live in segregated areas are not at higher risk of these conditions compared to similar women living in non-segregated areas. The factors and paths contributing to these medical conditions need to be explored in future research.

In review, the purpose of identifying mediators was to understand why residential segregation contributed to higher rates of low birthweight. This analysis found that the pathways by which residential segregation impacts low birthweight are through chronic hypertension (mediating effect = 2.469), pregnancy-related hypertension (mediating effect = 2.073), and preeclampsia (mediating effect = 2.065). Of these, chronic hypertension had the strongest mediating effect in the residential segregation and low birthweight relationship, followed by pregnancy-related hypertension, and preeclampsia. These mediators and their underlying processes are explored.

Chronic hypertension was identified as the strongest mediator in the residential segregation and low birthweight relationship. Hypertension is associated with stress, and in African American women that stress may be caused by racism and living in residential segregation. The stressors associated with living in residential segregation were outlined in the background section of this dissertation, and include financial stressors, family stressors, and/or stressors from living in unsafe neighborhoods. Chronic hypertension is a leading cause of premature birth and if untreated, may exacerbate into other pregnancy-related medical conditions, such as pre-eclampsia, eclampsia, and/or uterine bleeding due to abrupto placenta or premature rupture of fetal membranes.

Pregnancy-related hypertension is also a strong mediator in the residential segregation and low birthweight relationship. The plot of logits by age group found that African American women were more likely than white women to have pregnancy-related hypertension over the course of their reproductive years. The risk began at about age 29

increased rapidly over the remaining reproductive years. Pregnancy-related hypertension is diagnosed after the 20th week of pregnancy. The degree to which untreated and/or treated chronic hypertension exacerbates into pregnancy-related hypertension in African American women who live in residential segregation, warrants further research. Pregnancy-related hypertension is also a major cause of premature birth.

Preeclampsia is a sever hypertensive disorder in pregnant women and requires immediate health care attention. The risk of preeclampsia in African American women sharply increases after 35 years of age. African American women with preeclampsia are over 5 times (OR 5.9) as likely as similar women to have low birthweight. Women with preeclampsia are at very high risk of premature birth.

In summary, these three hypertensive medical conditions help to explain why residential segregation is associated with higher rates of low birthweight. Living in segregated areas heightens the risk chronic hypertension, pregnancy-related hypertension, and preeclampsia, which in turn increase the risk of low birthweight. All of these medical conditions have been linked to stress and environmental exposures that are much more prevalent in segregated areas. The indirect pathways through which segregation influences these medical conditions, and their varying effects by age, socio-economic status and country of origin, require further investigation.

This analysis however, did not explain why other medical conditions did not mediate the residential segregation and low birthweight relationship. Specifically, it was interesting

that residential segregation was not associated with infectious diseases as hypothesized earlier in this dissertation. It may be that the pathways by which residential segregation impacts infectious diseases are more complex and multiple mediators are required to have an impact on low birthweight. Future research on these pathways is warranted.

The three medical conditions that were identified in this dissertation as having a mediating effect on the residential segregation and low birthweight relationship require not only further research but also public health intervention. The final chapter of this dissertation will summarize the findings from all of these analyses in place them context of what we know and do not know about residential segregation and low birthweight, and how these findings may contribute to public health policy initiatives.

CHAPTER 6:

SUMMARY AND CONCLUSIONS

The purpose of this research was to investigate the geography of racial disparities in low birthweight in New York City. Race was viewed as a social construct and not a biological construct; therefore, it was hypothesized that racial disparities in low birthweight could be explained by geographic inequalities in income, class, race, ethnicity, and other social dimensions. This cross-sectional multilevel study focused on residential segregation and its effect on the risk of low birthweight among African American infants and mothers.

Previous literature on racial disparities in low birthweight has focused on individual-level risk factors, or characteristics and behaviors of the mother that placed her infant at increased risk. Strategies to reduce the number of low birthweight births have focused on reducing individual-level risk factors through health education related to reproduction and family planning, reduction of risky behaviors, increasing the accessibility of early and regular prenatal care services, and removing barriers to prenatal care. To address racial differentials the National Institute of Medicine recommended that research focus on the cumulative effects of poverty and social neglect, and the interaction of these factors with biological parameters. This change in emphasis from individual- to contextual-level risk factors resulted in wave of research investigating the relationship between poverty and low birthweight. Overall, these studies found a positive and significant relationship between poverty and increasing rates of low birthweight, for both

African American and white women, but as yet they have not sufficiently explained why racial disparities in low birthweight exist, and persist today. This research specifically focused on the environment of residential segregation as a contextual-level risk factor for low birthweight, while controlling for individual-level risk factors. While previous studies have been conducted on residential segregation and low birthweight at the metropolitan scale, this is the first comprehensive investigation at the intraurban scale of residential segregation and its contribution to racial disparities in low birthweight.

In this study residential segregation was considered a hazard to which African American women were exposed. Exposure may have occurred prior to or during pregnancy and risk may have been acute or cumulative. The level of exposure depended on the level of local segregation. The duration of exposure was based on nativity or place of birth of the mother. It was hypothesized that the duration of exposure to residential segregation would be longer for U.S.-born African American women than immigrant black women. It was also hypothesized that cumulative exposure in African American women would lead to early health deterioration, which would reduce the quality of the intrauterine environment, increasing fetal vulnerability to low birthweight. Premature birth and intrauterine growth retardation are two subgroups of low birth weight, each associated with their own set of risk factors and pathways by which they occur. African American women are particularly prone to premature birth.

The first objective of this research was to measure the extent of racial disparities in low birthweight in New York City and to identify populations at increased risk. In all

analyses, African American women were at substantially greater risk of low birthweight than white women, after controlling for individual-level risk factors. In addition, U.S.-born women were at higher risk of low birthweight than foreign-born women. When African American women were stratified by origin of birth, both U.S.-born and foreign-born black women were at elevated risk. However, U.S.-born African American women were at greater risk than immigrant black women reflecting higher risks associated with race and place of birth. These findings demonstrated that individual-level characteristics of the mother were important predictors of low birthweight, but they still did not explain racial disparities and differences by origin of birth.

The *epidemiological paradox* is described in the literature as a phenomenon by which foreign-born women have improved birth outcomes over native-born women, despite their living in similar disadvantaged neighborhoods. This research found that African American women living in racially segregated areas were more likely to have a low birthweight birth than similar women not living in racially segregated areas; and the health effects of residential segregation differed in U.S.-born and foreign-born African American women. In fact, U.S.-born African American women living in segregated neighborhoods were the most likely of population groups to have a low birthweight birth, evidence that prolonged exposure to residential segregation may be detrimental to birth outcome.

Furthermore, this study found that immigrant black women were more likely to have lower income levels than U.S.-born African American women determined by the

percentage of women on Medicaid, although these differentials were small. Yet despite the contextual- and individual-level disadvantages, foreign-born black women had lower rates of low birthweight compared to U.S.-born African American women. Although these analyses could not explain the protective effects associated with being foreign-born, they did demonstrate the presence of a strong *epidemiological paradox* in New York City's racially segregated neighborhoods.

The second objective was to measure the association between residential segregation and low birthweight, controlling for individual-level risk factors. In general, women were at increased risk if they were unmarried, did not attend college, were on Medicaid, if they smoked, consumed alcohol, or if they reported using illicit substances. Age had a slightly positive association with low birthweight, such that with increasing age the likelihood of low birthweight also increased. Previous literature reports that age has a direct biological effect on birth weight only in the later reproductive years. Pregnancy in a young woman is a risk factor only if she is immature or does not have the skills to care for herself during her pregnancy. This study found that on average African American gave birth at a younger age than white women and U.S.-born African American women gave birth at the youngest ages.

Marriage has a positive effect on birth weight through support of the husband. Financial support allows the woman the opportunity to choose how much and how long she will work during her pregnancy, she may be provided with health insurance, and/or she may receive general amenities (e.g. nutrition, better housing) that improve her well being.

The husband may also provide support by caring for other children or providing general emotional support, both of which can reduce stressors during pregnancy. This research found that unmarried women were more likely to have a low birthweight birth than married women. African American women were less likely than white women to be married at the time of their infant's birth. The most compromised group were U.S.-born African American women with only 20 percent married at the time of their infant's birth. When controlling for residential segregation, the protective effects of marriage increased.

Women with higher education are more likely to have higher incomes, which support improved health behaviors such as nutrition and seeking early prenatal care. These women are also less likely to participate in detrimental lifestyle behaviors. In this study, the percentage of women with a college education did not vary substantially between population groups; however, having a college education was consistently protective of low birthweight. These protective effects of college education increased after controlling for residential segregation.

This study also found that women on Medicaid were more likely to have a low birthweight birth than similar women using private insurance. Women on Medicaid are generally of low income, and Medicaid status may be interpreted as individual-level poverty. In this study, the effect of individual-level poverty on low birthweight did not change after controlling for residential segregation. Likewise, residential segregation did not exacerbate the effect of individual-level poverty on low birth weight, a finding consistent with previous literature on the cross-level effect of poverty.

Smoking is the most important individual-level risk factor for low birthweight. White women are more likely to smoke at younger ages than African American women but this trend reverses with increasing age. This study found higher rates of smoking among African American women than white women. The highest rates of smoking were among U.S.-born African American women. Smoking was consistently associated with increases in low birthweight. African American women were also more likely to report substance use than were white women. The highest rate of substance usage was among U.S.-born African American women. Although maternal behaviors detrimental to birth outcomes are probably underreported on the birth records these results on high-risk behaviors are consistent with high-risk population groups for low birthweight.

The individual-level risk factors identified in this study support findings in previous research. In addition, this study used multilevel modeling to measure the cross-level effect of residential segregation and individual-level risk factors on low birthweight. The results showed that individual-level risk factors were not exacerbated by residential segregation. This finding did not support the original hypothesis that stressors related to residential segregation would increase detrimental lifestyle behaviors, such as smoking, or substance use, which in-turn would increase the risk of low birthweight. Instead the results indicated that other mechanisms were contributing to the residential segregation and low birthweight relationship.

Another significant finding from this research was that individual-level risk factors alone could not explain census tract variability in low birthweight means across New York

City. In other words, characteristics of the mother could not explain geographic variation in low birthweight. Poverty could not explain the variation either. Geographic variability in census tract low birth weight means could be explained however by residential segregation. Thus, when the racial context of neighborhoods was considered geographic variation in census tract low birth weight means disappeared. This finding demonstrated that residential segregation is a strong determinant of racial disparities in low birthweight in New York City.

The third objective was to measure and plot the probability of medical conditions by age for African American and white women. It was hypothesized that African American women would have a greater number of medical conditions with age or a more complex array of medical conditions with age because of their exposure to residential segregation. These medical conditions would be indicative of early health deterioration, which is a key element of the *weathering hypothesis.* Only those medical conditions that were significantly associated with low birth weight were used in this analysis. This study found substantial racial disparities in medical conditions over the reproductive years with African American women almost always having a higher prevalence than white women. These findings support the *weathering hypothesis* of early health deterioration in African American women.

The fourth objective of this research was to determine how residential segregation impacted birth outcomes by identifying mediating effects. It was hypothesized that medical conditions mediated this relationship. In other words, residential segregation

would contribute to health deterioration through medical conditions, which in-turn would impact birth outcomes. This study found three medical conditions that were significant mediators in the residential segregation and low birth weigh relationship, specifically chronic hypertension, pregnancy-related hypertension, and preeclampsia. All three of these medical conditions are stress-related hypertensive diseases that appear to be related to exposure to residential segregation. Hypertensive disorders have the potential to reduce blood flow to the fetus, resulting in premature contractures and premature labor. These are important findings that explain a pathway by which residential segregation contributes to low birth weight.

Limitations of the Study

There are a few limitations of this study. At the individual-level there are important data variables missing from the analyses, specifically, data on birth order, nutritional status of the mother, weight gain during pregnancy, the intergenerational transmission of low birth weight (maternal or paternal), and mother's occupation. In addition, low birthweight was not stratified by premature birth and IUGR. Studying the individual- and contextual-effects of specific birth outcomes may result in different risk mechanisms. In addition, we do not know how long a mother resided at her place of residence – in other words, the duration of exposure used in this analysis may be miss-specified. This study estimated the duration of exposure using origin of birth, and although significant differences emerged, a more detailed longitudinal history of women's exposure to residential segregation would strengthen the study.

In regards to the contextual analysis residential segregation and its effect on low birthweight was studied independently and compared with the effect of poverty. It would also be useful to measure the effect of residential segregation on low birthweight, while controlling for poverty. It may be that a substantial portion of the residential segregation effect is explained by poverty, although the fact that segregation has a stronger association than poverty with low birthweight suggests that there is an independent segregation effect.

Contribution to Geographic Research

There are many contributions of this research to the field of geography. First, this research defined residential segregation at the local scale in New York City. Previous literature on residential segregation at the metropolitan scale has referred to New York City as a highly segregated city. This research confirmed those findings by mapping the geographic distribution of segregation indices at the census tract level. Second, this research confirmed the presence of an *epidemiological paradox* among black women in New York City. This finding demonstrates the importance of defining different places of origin when measuring health outcomes in different populations of women. African American women are not homogeneous, and immigrant African American women have considerably different birth outcomes than native-born African American women. Third, this research identified areas of elevated unexplained risk of low birthweight for African American women, using spatially weighted regression models, controlling for individual-level risk factors. These areas were identified along the periphery of residential segregation and suggest the need to further evaluate these neighborhoods for transitional

qualities such as deteriorating socioeconomic status or gentrification. Forth, using multilevel modeling this research was able to explain the within- and between- census tract variability in low birthweight births for African American and white women. We now know that within census tracts women are racially homogeneous because of New York City's racially segregated areas. With this knowledge geographers can study individual-level similarities and differences among women of the same racial group and examine the specific place environments in which they live. This research indicates that the place environment, specifically the environment of racial segregation, is closely tied to the risk of adverse birth outcomes for mothers and infants.

Suggestions for Future Research

Future research on residential segregation and low birth weight should focus on defining areas of concentrated poverty and distinguishing between areas of high segregation and high poverty versus areas of high segregation and low poverty. The effects of these local contexts on the risk of low birth weight require further investigation. In addition, future research should investigate the boundaries of residential segregation for physical barriers. There may be additional mechanisms contributing to highly segregated areas other than transitional neighborhoods that cause them to be separated such as highways, parks, or waterways. These physical barriers may have important effects on women's social and spatial interactions and their ability to access health care services.

The findings from this research also support the need to further investigate the *epidemiological paradox* to identify protective qualities attributed to foreign-born women. It would also be useful to stratify foreign-born women by place of birth to

examine differences in their risk of low birthweight. In addition, future research should focus on the *weathering hypothesis* and distinguishing between early programming and cumulative exposure pathways for low birth weight. A path- analysis may help to explain the multiplicity of contributing factors. There is also a need to specify low birth weight by premature birth and IUGR to measure change in the mediating effects identified in this analysis. For example, chronic hypertension may have a greater mediating effect on the residential segregation and low birthweight relationship for premature birth than IUGR. Finally, the results of these analyses should be tested at different geographic scales to measure the sensitivity of the results.

Recommendations for Public Health Policy Initiatives

Future public health policy should evaluate the availability and accessibility of health care facilities in racially segregated neighborhoods. While an important focus should be on prenatal care there is also a need for chronic disease prevention in the African American population of women. Promoting health and preventing illnesses before pregnancy may enhance the fitness of women during pregnancy. During pregnancy African American women should be closely screened for hypertensive complications and treated promptly. Prenatal and health care should be culturally sensitive to meet the needs of the diverse African American population.

In addition, this research supports strengthening the link between public health policy and social policy. Future public health policy initiatives should include collaborating with social policy makers to assess areas of high-risk for low birthweight in the African American population of women. This study showed that residential segregation is

strongly associated with adverse birth outcomes. Now there is a need to identify specific environmental risks within the context of residential segregation that place women at greater risk of low birthweight. Public health representatives need to be stronger advocates in the social policy arena for community development and reducing neighborhood risks.

Finally, there is a need to hear from African American women what stressors they are experiencing and if improvements could be made in their life, what would they be? There may be a need for individual change but there may also be a greater need for social change, such as providing transportation, low- cost grocery stores, or day-care facilities to residentially segregated areas. Public health policy initiatives should quantify the economic costs associated with chronic diseases and low birthweight in the African American population of women and advocate the burden of these costs on society. By broadening the social definition of health for African American women responsibilities associated with disease prevention are more likely to be delegated.

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