ADVERSE BIRTH OUTCOMES IN U.S.- AND FOREIGN-BORN BLACK WOMEN: A CONCEPTUAL AND EMPIRICAL ANALYSIS

by

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This dissertation is dedicated to my Mother, who provided uncompromising support throughout this enduring process; to Dave, whose admiration for my work remained a constant source of inspiration; and to Tosha and Tabar, for whom everything was made worthwhile.

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LIST OF ABBREVIATIONS

CANY	Caribbean-born mothers residing in New York City
CAVI	Caribbean-born mothers residing in the Virgin Islands
CI	Confidence interval
LBW	Low birth weight (< 2500 g, or ≈ 5.5 lbs)
MLBW	Moderately low birthweight (1500-2499 g)
NYC	New York City
NYCDH	New York City Department of Health (and Mental Hygiene)
OR	Odds ratio
PTB	Preterm birth (< 37 completed weeks of gestation)
SGA	Small for gestational age
VI	Virgin Islands (U.S. territory)
VINY	Virgin Islands-born mothers residing in New York City
VIVI	Virgin Islands-born mothers residing in the Virgin Islands
VLBW	Very low birth weight (< 1500 grams, or ≈ 3.3 lbs)

ABSTRACT

Adverse Birth Outcomes In U.S.- And Foreign-Born Black Women:

A Conceptual and Empirical Analysis

by

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Background: This dissertation explored racism and birth outcomes in U.S.- and foreign-born Black women and examined the degree to which commonly-measured risk factors could explain why Black Caribbean immigrants have lower rates of preterm birth than African Americans. It included: a review of how race- and nativity-based disparities have been conceptualized in perinatal health research; an examination of preterm birth predictors among Caribbean- and U.S.-born Black women; and an assessment of preterm birth risk by maternal age and immigrants' duration of U.S. residence. *Methods*: Systematic literature review coupled with logistic regression analyses utilizing birth records from New York City (2000-2010) and the U.S. Virgin Islands (2000-2004). *Results*: The review produced no generalizable evidence for suggested causes of racial or Black ethnic disparities in birth outcomes. However, there is modest support that racism is associated with adverse birth outcomes, and the perinatal health advantage for Black immigrants is ascribed to selective migration and culturally-linked factors, although the evidence is sparse. In this study, Caribbean-born immigrants in New York City sustained lower odds of preterm birth relative to U.S.-born Blacks (*OR* = 0.85, 95% CI: 0.76,

0.94) and Caribbean-born residents in the Virgin Islands (*OR* = 0.54, 95% CI: 0.34, 0.89) despite adjustment for demographic, behavioral, and medical risk factors. Age and education were most influential in explaining the preterm birth advantage for Black Caribbean immigrants, and there was modest support for selective migration. However, the risks of preterm birth with advancing maternal age were similar between Caribbean-born immigrants (*OR* = 1.13, 95% CI: 1.10, 1.15) and U.S.-born mothers (*OR* = 1.15, 95% CI: 1.13, 1.17) in New York City. Further, the odds of preterm birth among Caribbean immigrants increased 7% for every 5 years of U.S. residence (*OR* = 1.07, 95% CI: 1.04, 1.11). *Conclusions*: The "healthy migrant" effect for Black Caribbean immigrants is conditional on national origin and length of time in the U.S. The worsening of immigrant mothers' preterm birth risks with increased duration of U.S. residence warrants additional research into contextual factors, including racism, to yield greater insight into perinatal health disparities among native and foreign-born Black women.

CHAPTER 1

Introduction

For several decades, the U.S. Black-White disparity in infant mortality has averaged two-fold or greater—largely related to the disproportionate number of very low birthweight (< 1500 g) and very preterm births (< 32 weeks) among non-Hispanic Black women (Alexander, Wingate, Bader, & Kogan, 2008; Carmichael & Iyasu, 1998; Centers for Disease Control and Prevention, 2002; Iyasu, Becerra, Rowley, & Hogue, 1992; MacDorman & Mathews, 2011; Singh & Yu, 1995). The persistence of Black-White disparities in preterm birth is also well substantiated (Schaaf, Liem, Mol, Abu-Hanna, & Ravelli, 2013; Schempf, Branum, Lukacs, & Schoendorf, 2007). Notwithstanding, there is little scientific consensus regarding the causal factors that account for these disparities (Behrman & Butler, 2007). The bulk of investigative research into adverse birth outcomes has emphasized demographic, socioeconomic, behavioral, and biomedical risk factors as key explanations for racial disparities. However, it has generally been observed that the Black-White gap persists despite adjustments for a range of risk factors, including ***maternal age*** (Geronimus, 1996; Holzman et al., 2009); ***parity*** (Swamy, Edwards, Gelfand, James, & Miranda, 2012); ***marital status*** (Bennett, Braveman, Egerter, & Kiely, 1994; Reichman, Hamilton, Hummer, & Padilla, 2008); ***income*** (Blumenshine, Egerter, Barclay, Cubbin, & Braveman, 2010; Collins & David, 1990); ***education*** (Din-Dzietham & Hertz-Picciotto, 1998; McGrady, Sung, Rowley, & Hogue, 1992; Schoendorf, Hogue, Kleinman, & Rowley, 1992); ***behavioral health risks*** (Finch, Frank, & Hummer, 2000; Goldenberg et al.,

1996); ***prenatal care*** (Coley & Aronson, 2013; Healy et al., 2006); and ***healthcare technologies*** (Levine et al., 2010).

The failure of traditional risk factors to substantively account for racial disparities in preterm birth, low birthweight, and infant mortality has prompted the examination of ***psychosocial*** (Dole et al., 2004; Dominguez, Schetter, Mancuso, Rini, & Hobel, 2005; Istvan, 1986; Rowley, 2001; Wadhwa, Entringer, Buss, & Lu, 2011); ***neighborhood*** (Buka, Brennan, Rich-Edwards, Raudenbush, & Earls, 2003; Grady, 2006; Morenoff, 2003; O'Campo et al., 2008; Osypuk & Acevedo-Garcia, 2008; Polednak, 1991; Roberts, 1997; Schempf, Kaufman, Messer, & Mendola, 2011); and ***structural*** (Baker & Hellerstedt, 2006; LaVeist, 1993; Pickett, Collins, Masi, & Wilkinson, 2005) contexts that could explain the persistent racial gradient in adverse birth outcomes. ***Racism*** is one such factor receiving emergent attention (Collins, David, Handler, Wall, & Andes, 2004; Collins et al., 2000; Dominguez, 2008, 2011; Misra, Strobino, & Trabert, 2010; Mustillo et al., 2004; L. Rosenberg, Palmer, Wise, Horton, & Corwin, 2002), and empirical studies have shown that racism is positively associated with adverse birth outcomes (Collins et al., 2004; Dole et al., 2004; Mustillo et al., 2004; Rankin, David, & Collins, 2011; L. Rosenberg et al., 2002).

Notwithstanding the contributions that both individually- and contextually-focused studies have made to our understanding of maternal and infant health disparities *between* Blacks and Whites, they do not enhance our understanding of heterogeneity *within* racial groups. In contrast to the plethora of research on Black-White differences, relatively few studies have examined adverse birth outcomes *within* populations of Black women by nativity (see Acevedo-Garcia, Soobader, & Berkman, 2005; Cabral, Fried, Levenson, Amaro, & Zuckerman, 1990; Chavkin, Busner, & McLaughlin, 1987; David & Collins, 1997; Fang, Madhavan, & Alderman, 1999; Friedman et al., 1993; Howard, Marshall, Kaufman, & Savitz, 2006; Hummer et al., 1999;

Kleinman, Fingerhut, & Prager, 1991; Pallotto, Collins, & David, 2000; K. D. Rosenberg, Desai, & Kan, 2002; Singh & Yu, 1996; Stein et al., 2009; Valanis & Rush, 1979). Although studies generally support that Black immigrant women have more favorable birth outcomes than native African American women, it is notable that the infant health advantage has been shown to lessen among immigrants with increased duration of residence (Urquia, Frank, Moineddin, & Glazier, 2010) and among the children of Black immigrants in the U.S. (Collins, Wu, & David, 2002). This Black nativity paradox has rarely been investigated. There is an even greater dearth of studies that explore how racism influences pregnancy and birth outcomes among Blacks in the U.S. by national origin (see Dominguez, Strong, Krieger, Gillman, & Rich-Edwards, 2009). This dissertation seeks to address these gaps.

Purpose

The primary purpose of this dissertation is to explore the enigma of *race x nativity* disparities in birth outcomes by articulating (conceptually) how racism influences pregnancy and birth outcomes among U.S.-born *and* Caribbean-born Blacks and explicating (empirically) the relative contributions of demographic and pregnancy risk factors between immigrant and non-immigrant Black populations to more fully understand the Black immigrant 'advantage' in birth outcomes. The current study explores a fundamental question: *What factors best 'explain' the disparate rates in adverse birth outcomes among U.S.-born and foreign-born Black women*? From a conceptual and empirical stance, this study will articulate and uncover predictors that can potentially explain the variance in maternal and infant health among non-migrant Caribbean Blacks, migrant Caribbean Blacks, and African Americans.

A secondary purpose of this dissertation is to develop a conceptual model of racism as a determinant of differential maternal and infant health risks among diverse Black women in the U.S. I define racism as system of beliefs and structures that denigrate and disadvantage members

of racial groups who are categorized and regarded as inferior. This definition highlights the ideological and structural aspects of racism which contribute to its persistence. Although most perinatal health literature has conceptualized and examined racism as perceived discrimination (i.e., unfair treatment due to one's race), this dissertation responds to the call for studies that elucidate mechanisms by which structural racism impacts health (Ford & Airhihenbuwa, 2010). My model will be used to theoretically explain differential outcomes between U.S.-born Black women and foreign-born Black women and guide the development of empirical studies related to this research focus, two of which are included in this dissertation.

An underlying purpose of this dissertation is to offer a counterargument against implicit and explicit arguments for genetic causes as explanations for racial disparities in perinatal health. A detailed coverage of genetics is beyond the scope of this dissertation. However, the seemingly intractable perinatal health disparities observed between Blacks and Whites has led some researchers to propose a genetic basis for the disparities (Bodnar & Simhan, 2010; Chaudhari et al., 2008; Conley & Bennett, 2000; Dolan, 2010; Esplin, 2006; Jaffe et al., 2013; Kistka et al., 2007; Tsai et al., 2009; Wang et al., 2006). Although genetics is one of many factors that may legitimately contribute to preterm birth, attributing genetics as a primary explanation for *racial* disparities presumes innate differences between human groups based on a biogenetic concept of "race" that is generally not accepted as valid (Cooper, 2003; Foster & Sharp, 2004; Goodman, 2000; Gravlee, 2009; Kaufman, Geronimus, & James, 2007; Reverby, 2010).

My critique of genetic explanations is sustained even in the face of arguments that proffer epigenetic justifications, which, nonetheless, serve to reify the role of genetics to explain racial disparities. For example, it has been observed that birth outcomes among descendants of Black immigrants worsen in as little as one generation (Collins et al., 2002). A genetically-focused argument would contend that Caribbean Blacks and African Americans have genetic variants

that could explain the higher rates of adverse birth outcomes for *both* of these groups relative to Whites. An extension of this argument would reinforce that, through epigenetic mechanisms, these genes become harmfully "expressed" under adverse conditions in the U.S. to worsen the health of Black migrants. I counter that any valid *genetic* factor ascribed to Black "race" would be unlikely to invoke population-wide changes in such a short time-span—even under harsh socio-environmental influences—and this problematizes attributions to genetic factors as a source for perinatal health disparities by race or nativity. My diminution of *genetic* explanations for disparities in birth outcomes does not dismiss the role of biological factors (e.g., allostatic load) as mediators of preterm birth risks—with the presumption that such factors are not deemed immutable or heritable by "race." Accordingly, I include structural contexts and biopsychosocial mechanisms in my conceptualization of Black health disparities.

In summary, this dissertation: (a) provides a conceptual analysis and model of how racism patterns structural and individual risk factors which can influence birth outcomes among native *and* immigrant Black women in the U.S.; and (b) ascertains empirically how preterm birth varies among Black women by nativity (i.e., African Americans, Black Caribbean immigrants in the mainland U.S., and Black Caribbean residents in the U.S. Virgin Islands). The conceptual analysis develops as an integrative literature review of how perinatal health researchers have examined what are believed to be the leading 'causes' for inter-racial and inter-ethnic disparities in birth outcomes among Black women—including how racism has been conceptualized or examined as a source for these disparities. The review is followed by the presentation of a conceptual model that outlines racism-related contexts (e.g., structural racism and socially-patterned risk factors) together with hypothesized moderating or mediating mechanisms (e.g., racism-related stress, racial/ethnic identity, and stress-coping responses) that contribute to racial and ethnic disparities in adverse birth outcomes. The subsequent empirical analyses will

examine: (i) the relative contributions of demographic, medical/behavioral, and infant risk factors to preterm birth disparities among Black mothers by nativity and migrant status; (ii) risks of preterm birth with advancing maternal age—to test the "weathering" phenomenon among foreign-born Black women within and outside of the U.S. mainland; and (iii) risks of preterm birth with duration of U.S. residence—to qualify the extent of the infant health advantage for Black Caribbean immigrants.

This dissertation aims to augment perinatal and immigrant health research with a focus on understudied immigrant populations. This is the first known study to provide a comprehensive review of theoretical frameworks and empirical studies intended explicitly to enrich our understanding of birth outcomes among foreign-born Black women in the U.S. This study also offers a unique examination of migrant health effects in non-Hispanic Black populations by analyzing births to Black women residing in the U.S. Virgin Islands (V.I.) compared to births to their migrant counterparts living in the mainland U.S. This is also the first study to examine "weathering" (Geronimus, 1992) among Black women by nativity and migrant status in relation to preterm birth.

Theoretical Framework

I posit that racism is a fundamental cause of perinatal health disparities impacting Black women and infants in the U.S. The pathways and mechanisms through which racism results in disproportionate rates of adverse birth outcomes integrates structural, psychosocial, and biomedical factors. Specifically, my conceptual model synthesizes three theoretical frameworks: (a) structural racism; (b) lifecourse theory, inclusive of "weathering" and "allostatic load"; and (c) racism-related stress and coping. The first framework borrows from the theory of "fundamental causes" whereby disease causation is attributed to underlying social inequalities (Link & Phelan, 1995). An analogous perspective was adopted by Williams (1997) who

conceptualized racism as a fundamental cause of health in racially-marginalized populations. Racism manifests in many forms. Structural or *institutionalized* racism systematically restricts access to material resources and access to power at a societal and community-wide level (e.g., involuntary racial residential segregation). *Personally-mediated* racism occurs in interpersonal contexts in the form of prejudice and discriminatory behaviors toward persons because of their race (e.g., employment discrimination, unequal healthcare). And *internalized* racism results when persons accept disparaging stereotypes about their racial inferiority (e.g., negative racial self-concept) (Jones, 2000). Racism, particularly its structural components, is viewed as the genesis for racial disparities in adverse birth outcomes.

The second theoretical framework is broadly conceived as the "lifecourse framework." The lifecourse framework posits that maternal and infant health outcomes are consequences of lifelong, cumulative impacts of racial inequality on health. Lu and Halfon (2003) articulated this framework to help explain racial and ethnic disparities in birth outcomes. This framework is closely reminiscent of weathering as described by Geronimus (1992, 1996). Weathering speaks to the cumulative, structural disadvantages that precipitously worsen Black women's health. The lifecourse framework also parallels the concept of allostatic load (McEwen, 1998). Allostatic load is a physiologic consequence of cumulative, chronic stress which results in accelerated wear-and-tear on body systems. Allostatic load impairs adaptive neuroendocrine and inflammatory mechanisms and thus contributes proximate risks for adverse birth outcomes. I use allostatic load as an operational construct for the physiological effects of cumulative, chronic stress that negatively alter the fetal environment and increase risks of adverse birth outcomes.

The third theoretical framework is a modified stress and coping framework that integrates coping styles such as "John Henryism" (James, 1994) and the "Sojourner syndrome" (Mullings, 2005) that have particular relevance to Black populations dealing with racism. My

conceptualization of stress and coping calls attention to stress experienced by Black women as a consequence of both structural- and personally-mediated racism. "Racism-related stress" is conceptualized as distinct from global or pregnancy-specific stress. Although it is admittedly difficult to empirically disentangle the impact of co-existing stressors in Black women's lives, it is presumed that the marginalized position of Black women in the social hierarchy generates *multiplicative* effects for identified (and unidentified) stressors. Examples include the dual impacts of socioeconomic *and* racial disadvantage, or socioeconomic incongruity due to race *and* gender. I outline proposed mechanisms through which both U.S.-born and immigrant Black women experience and cope with structural- and personally-mediated racism that engender racism-related stress and precipitate adverse maternal-infant health.

Dissertation Outline

This dissertation unfolds in six sections, including the present Introduction (Chapter 1), a systematic literature review (Chapter 2), presentation of a conceptual model (Chapter 3), two empirical papers (Chapter 4 and Chapter 5), and a Conclusion (Chapter 6). Chapter 1 describes the background, purpose, and outline for the dissertation. Chapter 2 provides a review and critique of frameworks and propositions that have shaped the scientific discourse in relation to racial and Black nativity disparities in birth outcomes; Chapter 3 introduces a conceptual model that posits racism as a fundamental cause of maternal and infant health disparities by race and nativity. Chapter 4 explores the roles of nativity and migration by analyzing maternal and infant health outcomes between African Americans, Black Caribbean immigrants in the U.S., and Black Caribbean residents in the Virgin Islands. Chapter 5 examines preterm birth by advancing maternal age among non-Hispanic U.S.-born, Caribbean-born, and V.I-born Black women in order to explore the concept of "weathering" in these heterogeneous Black populations. Finally, Chapter 6 summarizes overall study results and implications.

Detailed overview. In Chapter 2, I conduct a systematic literature review of medical and social science inquiry as it relates to racial and Black ethnic disparities in birth outcomes. I review and discuss common explanations for racial/ethnic disparities *vis-à-vis* alternative conceptualizations that highlight racism as a primary cause of observed racial and Black nativity differences in birth outcomes. I provide a distinct review of empirical studies that examine **racism** and **nativity status** in relation to birth outcomes and discuss limitations and opportunities for additional research in these areas.

Chapter 3 presents a conceptual model that emphasizes racism-related factors that are hypothesized as central to race- and nativity-based disparities in maternal and infant health.

Chapter 4 investigates Black ethnic heterogeneity in maternal and infant characteristics with an aim to critically examine a host of predictive and confounding factors for preterm birth *within* and *between* populations of African Americans and immigrant Blacks from the English-speaking Caribbean. In addition to medically-related factors, I carefully analyze socio-demographic covariates that may account for similarities and differences in infant health among these women—particularly the extent to which these commonly-measured risk factors contribute to the perinatal health advantage for foreign-born Blacks.

Chapter 5 examines maternal age in relation to infant health by comparing populations of U.S.-born mothers with Black Caribbean mothers in the U.S. and in the Virgin Islands. Birth data for the U.S. and New York City reveal that foreign-born Blacks, compared to African Americans, have significantly higher proportions of births to women aged 35 and older (Chavkin et al., 1987; Elo, Vang, & Culhane, 2014; Hamilton, Martin, Ventura, Sutton, & Menacker, 2005). These women are considered to be of 'advanced maternal age' with significantly higher risks for adverse birth outcomes. My analysis ascertains patterns of preterm birth by maternal age among African American and Caribbean migrant and non-migrant populations and applies a

test of the weathering hypothesis (Geronimus, 1992, 1996) in these populations. It also examines Black Caribbean immigrants' duration of U.S. residence in association with preterm birth risk.

Chapter 6 summarizes the significance and implications of the findings from this dissertation which proffers racism as a fundamental cause of adverse maternal health and birth outcomes in Black populations in the U.S., regardless of nativity status.

Summary

This dissertation, with its emphasis on adverse birth outcomes by national origin among U.S.-born and foreign-born Blacks, is intended to bridge two currently disconnected streams of research. The bulk of studies on racial disparities in adverse birth outcomes emphasize Black-White differences—with an emerging focus on racial discrimination as a contributing factor. However, Blacks/African Americans are routinely studied as an ethnically homogenous group, and little is known about what factors modify the putative impact of racial discrimination on birth outcomes. A second, but sparser line of research examines national-origin differences in birth outcomes; however, the focus is almost exclusively on Mexican American women and the 'Hispanic health paradox.' A closer examination of the understudied *race x national origin* interaction within populations of Black women may help researchers to better understand the similarities and differences in socio-demographic and pregnancy-related factors between native and foreign-born Blacks that confer protection or risk.

The preponderance of extant research on biomedical, psychosocial, and behavioral risk factors has done little to advance our understanding of fundamental causes of Black/White and African American/Black Caribbean differentials in low birthweight, preterm birth, and neonatal mortality. Racism may be a fundamental cause of the observed race-related disparities—particularly when there is persistent, unexplained variance by "race" after controlling for known risk factors for preterm birth. My research adds to a small but growing number of studies that

address racism as a stressor associated with adverse birth outcomes in Black women. However, my investigation is unique in that it proposes racism as a framework for understanding both inter-racial (Black/White) *and* inter-ethnic (U.S.-born Black/foreign-born Black) disparities in birth outcomes.

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CHAPTER 2

The Scientific Construction of Disparities in Birth Outcomes by Race and Nativity: A Conceptual and Systematic Review

Although racial differences in adverse birth outcomes are descriptively well-documented, there is little scientific consensus as to the underlying *causes* for persistent *disparities* by race (Ashton, Lawrence, Adams, & Fleischman, 2009; Gennaro, 2005). In a comprehensive report on causes and prevention of preterm birth, the Institute of Medicine (Behrman & Butler, 2007) acknowledged our limited understanding of the etiology of disparities between Blacks and Whites, as well as within Black populations by nativity. In an attempt to synthesize the state of the science in these areas, this chapter provides an integrated review of conceptualizations and empirical findings among published articles on these topics, including: (a) reviews and commentaries discussing primary causes for perinatal health disparities by race; and (b) empirical papers specifically examining racism and Black nativity in relation to birth outcomes. The leading conceptualizations and explanations proposed by researchers will be presented, including arguments for socioeconomic, demographic, psychosocial, behavioral, biogenetic, and structural causes, including racism, that have been advanced to explain *why* racial and Black nativity disparities exist.

The notion of "cause" in this paper embraces a liberal understanding of causal inference, acknowledging that most perinatal health studies are observational studies designed to elicit measures of association wherein causal direction often cannot be established (Glass, Goodman, Hernan, & Samet, 2013). Moreover, this paper adopts a 'fundamental causes' perspective which

situates disease causation in the social environment (Kaufman & Cooper, 1999; Krieger, 1994; Link & Phelan, 1995, 2002). Accordingly, it broadens the causal lens from the physiologic processes of parturition to the examination of social context, particularly as related to racism as a stressor. Although an expanding literature has begun to observe biologic and genetic differences by socially-identified "race" to lend insight into the disparate pattern of adverse birth outcomes (Bodnar & Simhan, 2010; Burris & Collins, 2010; Chaudhari et al., 2008; Conley & Bennett, 2000; Dolan, 2010; Esplin, 2006; Kistka et al., 2007; Tsai et al., 2009; Wang et al., 2006), differential biologic and genetic endpoints are not in themselves "causal." Accordingly, it is the antecedent determinants of such differences that arise as the focus of the ensuing investigation. More specifically, this review explores the collective scientific understanding (and the philosophies that compel such understandings) of the fundamental determinants of both racial and national origin differences in adverse birth outcomes.

Integrated with the descriptions of how racial and Black ethnic disparities have been conceptually and empirically examined, this review will highlight the degree to which racism-related frameworks are acknowledged in current perinatal health research. I define racism as a system of beliefs and structures that denigrate and disadvantage members of racial groups who are categorized and regarded as inferior (see Neighbors, Griffith, & Carty, 2009). This conceptualization highlights the normative and systemic nature of racism in racially stratified societies (Bonilla-Silva, 1997). While it is more common for perinatal health studies to conceptualize or examine racism as perceived interpersonal discrimination, this review will highlight how the field has acknowledged or investigated structural racism as a determinant of perinatal health disparities.

Importantly, this review aligns racism-related conceptualizations and findings with the study of Black immigrant populations in the U.S. I juxtapose racism- and nativity-focused

research to offer insight into what contributes to the temporal perinatal health 'advantage' for foreign-born Blacks compared to U.S.-born Blacks. To date, studies of births to immigrant Blacks in the U.S. have compared maternal and infant health outcomes to those of U.S.-born Whites or African Americans, and have speculated on observed differences based on theories and studies of Hispanic immigrants. In contrast, by critically considering how racism may influence those differentials, this chapter offers a theoretical orientation for Black nativity differentials that is more specific to the experiences of Black immigrants. To my knowledge, this will be the first paper to provide an integrative review of theories and empirical studies uniquely focused on birth outcomes among foreign-born Black women in the United States. It will complement a previous study that examined perceptions of racism in U.S.-born and foreign-born pregnant Black women (Dominguez, Strong, Krieger, Gillman, & Rich-Edwards, 2009).

In summary, the following review will systematically examine both leading and alternative explanations for disparate birth outcomes by race *and* nativity. Throughout the review, I will highlight the degree to which researchers conceptualize or examine racism as a cause of perinatal health disparities impacting U.S.-born and foreign-born Black women. My emphasis on racism is not intended to discount the multifaceted nature of causation for adverse birth outcomes or dismiss other determinants of maternal-infant health. Instead, this chapter will offer a more extensive and critical analysis of an acknowledged antecedent cause (i.e., racism) which has received only cursory attention in extant perinatal health studies. It is intended to spur more rigorous examination of racism-associated constructs in perinatal health research and encourage more context-focused interventions to optimize African American and Black immigrant health.

Method

I conducted a review of key conceptualizations and findings among studies that explicitly addressed causation for Black racial and ethnic disparities in adverse birth outcomes. The PubMed database was queried to yield articles specific to pregnancy or birth outcomes (i.e., premature birth, low birth weight, small for gestational age, *or* infant mortality) *AND* racial disparities—with an emphasis on African American and Black immigrant populations. I used Medical Subject Headings (MeSH) syntax to identify causality as a major topic of the article in order to avoid articles that were primarily descriptive or surveillance-based. A separate search was conducted for articles dealing specifically with racism *AND* pregnancy or birth outcomes *AND* African Americans or Blacks. The search criteria were subsequently modified to retrieve articles specific to Black Caribbean women *AND* birth outcomes. Lastly, to ensure coverage of articles focused on perinatal health disparities related to specific etiologies, searches were conducted in reference to psychological, psychosocial, socioeconomic, behavioral, neighborhood, environmental, clinical/healthcare, biological, *or* genetic factors *AND* pregnancy or birth outcomes *AND* racial disparities. Only English-language studies of human populations based in the U.S. were included. There was no limit by year of publication; however, PubMed indexes publications dated back to 1809, and the current search ended on September 30, 2014. Unpublished theses or dissertations are not indexed in PubMed and hence they were not included in this search. Using these search strategies, 772 unduplicated articles were retrieved. These articles were assessed by a single investigator who determined which articles would undergo preliminary review and final selection for the present paper.

The preliminary selection of articles was limited to titles and abstracts that suggested a significant focus on Black or African American women with an emphasis on reviews or empirical studies directed to understanding racial disparities in birth outcomes. I excluded

examinations focused on Hispanic women because the present review aimed to capture findings that were unique to the socialization and health experiences of non-Hispanic Blacks in the U.S. I also excluded articles highlighting acute obstetrical care or other clinical topics that were not focused on explaining population-level disparities. Articles focused on racial disparities in *reproductive* health outcomes or care (e.g., fertility disorders, assistive reproductive technologies) were also excluded. Descriptive epidemiological studies for the mere purpose of describing or monitoring racial disparities in maternal risk factors and adverse birth outcomes without exploring specific causes, per se, were consulted for background information, but they were not made part of the systematic review. With the exception of studies that examined the effects of racism on birth outcomes, where one racial study group was allowed, empirical studies examining perinatal disparities were selected only if they reported measures of association or effect between two or more distinct racial/ethnic populations. Studies were also excluded if they did not measure the end outcomes of preterm birth, low birthweight, small-for-gestational-age, or infant mortality. These exclusions often applied to studies measuring psychosocial stress or biogenetic markers in pregnant women—where factors may have been identified differentially by race but they were not measured in association with birth outcomes. Despite their limited focus on racial disparities, comprehensive perinatal health reports were nonetheless included in this review because these reports reflect the contributions of leading experts in the field; they are often endorsed by official governmental or scientific bodies; and they are intended to drive priority research, policies, and interventions to improve the public's health—hence they are highly influential in reflecting and shaping the knowledge base for perinatal health researchers. Reference lists were cross-checked, and articles deemed relevant that were not retrieved in the original PubMed search were added to the review list.

Based on the above selection and exclusion criteria and a thorough reading of qualified publications, the final list of articles that explicitly discussed explanations or causal factors for racial disparities in birth outcomes totaled 214. These articles were categorized as review papers (*n* = 33); conceptual papers or commentaries (*n* = 37); and empirical investigations (*n* = 144). For space considerations, the empirical papers reviewed in this paper were restricted to the studies of racial discrimination/racism and birth outcomes (*n* = 13) and the studies of births to U.S.-born and foreign-born Black mothers residing in the U.S. (*n* = 25). Therefore, only 108 of the 214 selected articles were summarized for the present paper. Papers were subdivided according to the main focus of the review, commentary, or empirical study (Table 2). The selected papers are outlined in Appendix A: Table A.1 summarizes the reviews and conceptual papers; Table A.2 summarizes the empirical papers examining racism and birth outcomes; and Table A.3 summarizes the empirical papers examining Black women's nativity status and birth outcomes. The reviewed papers are delineated by author, year of publication, birth outcome studied, causal explanations proposed by the authors, and the extent to which racism-focused perspectives were addressed.

Table 2. .Medical/Public Health Articles Highlighting Causation of Racial Disparities in Birth Outcomes Pertaining to U.S.- or Foreign-born Blacks

Reviews *n* **= 33**	**Commentaries** *n* **= 37**	**Empirical Studies** *n* **= 144**
Main Subject Areas	***Primary Explanations Given***	***Main Subject Areas***
Multifactorial/General (*n* = 8)	Social context (*n* = 14)	Discrimination/Racism (*n* = 13)[a]
Biological (*n* = 6)	Biological (*n* =3)	Nativity Status (*n* = 25)[a]
Social context (*n* = 5)	Racism (*n* =4)	Neighborhood/Area-based (*n* = 26)
Racism (*n* = 3)	Stress (*n* =4)	Clinical/healthcare (*n* = 21)
Stress (*n* =2)	Environmental (*n* = 3)	Socioeconomic (*n* = 12)
Healthcare (*n* = 4)	Genetic (*n* = 1)	Biological (*n* = 11)
Environmental (*n* = 2)	Multifactorial or Integrative (*n* = 6)	Sociodemographic (*n* = 5)
Genetic (*n* = 2)	Healthcare (*n* = 2)	Genetic (*n* = 8)
Socioeconomic (*n* = 1)		Stress (*n* = 8)
		Multifactorial (*n* = 6)
		Behavioral (*n* = 4)
		Environmental (*n* = 3)
		Social Factors (*n* = 2)

[a]For space considerations, only these sets of empirical papers were discussed at length in this chapter.

In the ensuing review, I first introduce some papers with novel or alternative conceptualizations that have emerged to speculate about the underlying causes for persistent racial or Black ethnic disparities in birth outcomes. Afterward, I describe some of the more common scientific explanations that have been provided for racial or Black ethnic disparities in perinatal health—organized under the following themes: (a) general/multifactorial/integrative; (b) social context and lifecourse; (c) racism-related stress; (d) neighborhood or structural context; (e) clinical/healthcare; and (f) biogenetic/environmental. Within these dominant frames was a frequent emphasis on (g) demographic; (h) socioeconomic; and (i) behavioral factors which were more often discussed and examined as confounders of perinatal health risk rather than as primary explanatory factors, per se. I end with a review of empirical studies that have specifically examined (j) racism and (k) Black nativity status in relation to birth outcomes.

Alternative Conceptualizations

In 1992, the Centers for Disease Control and Prevention (CDC) developed an initiative to investigate psychosocial and biological causes for persistent racial disparities in preterm birth as they uniquely impact African American women (Hogan & Ferre, 2001). At the time, their approach had no modern-day parallel in perinatal health research. The scientific convention at that time was to investigate causes for preterm birth disparities that were situated largely in the socioeconomic and biomedical domains, with a growing focus on genetic causes. The CDC investigators posed preterm birth as a sociobiological problem and examined psychosocial stressors (e.g., racism) as determinants of health, independent of health risk behaviors. Their aim was to focus research on environmental and social contexts that were believed to pose unique and higher risks for African American women. In other words, they sought to reframe the dominant research position on disparities in preterm birth so as not to construe risks in African American women as an "absence of whiteness," (Rowley, 2001) but to instead examine risks and

assets *within* heterogeneous African American communities in order to better understand variability and ascertain why some members within 'at-risk' groups have healthier outcomes.

Continuing this critical tradition, a handful of papers have proposed novel conceptualizations for understanding racial disparities in adverse birth outcomes. The papers described next were selected because of their creative synthesis of relevant conceptualizations, their specificity to the African American experience, and their resonance within the current scientific public health community. In order of publication, David and Collins (1991) explicitly articulated racism, rather than race, as the genesis for the persistently observed racial disparities in low birthweight. Their paper opposed genetic arguments commonly espoused within medical and public health literatures where race was viewed as a proxy for socioeconomic status or genetic endowment. A related literature introduced similar critiques which have gained traction within the last decade (Goodman, 2000; Gravlee, 2009; Kaufman, Geronimus, & James, 2007; Krieger, 2005; Smedley & Smedley, 2005).

Geronimus (1992, 2001) developed the "weathering" hypothesis which states that African American women experience accelerated health deterioration (i.e., "aging") related to "cumulative socioeconomic disadvantage" (1992, p. 207). This accelerated aging translates into differential age-related reproductive health risks and outcomes for Black women relative to non-Hispanic White women. Age-specific risks rise more sharply for Black women than White women with advancing reproductive age. The weathering hypothesis highlights the importance of stratified analyses which would not presume equivalent risks by age or socioeconomic status between differently-advantaged racial groups.

James (1993) offered a unique cultural perspective that highlighted indigenous cultural strengths within communities that could be employed to buffer against racism. His framework is particularly noteworthy in that he attends to the protective role of indigenous cultural strengths—

a critical construct for the study of Black immigrants in the U.S. (Arthur & Katkin, 2006). Overall, he contributes an alternative perspective to an established literature on racial health disparities that reifies negative or pathological risk orientations almost exclusively.

Beginning with a seminal 1992 research initiative, a cadre of CDC investigators shifted the conversation from race as biology to the social context of race, and they emphasized racism as a chronic, systemic, psychosocial stressor. This CDC initiative was also unique in that it diverted from the common convention of cross-racial comparisons and focused on intra-group study to better understand the variations in both perceptions and health outcomes *within* African American communities. Collectively, the investigators articulated a biopsychosocial framework (Hogue, 2002; Rowley, 2001; Rowley et al., 1993) which conceptualized how differential social stressors experienced by African Americans influence biology in disproportionate ways to produce disparities, and they issued a charge to health providers to address social contexts (Hogan & Ferre, 2001). Wise (1993, 2003), echoed similar sentiments and advocated for the integration of social and biological disciplines to address disparities in infant mortality, with a particular focus on developing effective policy and interventions.

Lu and Halfon (2003) synthesized the lifecourse literature for the perinatal health community and reinforced that the genesis of risk does not begin during the pregnancy period but is manifested over the entire lifecourse, including a women's own fetal environment. Accordingly, this perspective would corroborate that the racial disparities observed for pregnancy and birth outcomes are not due to peculiarities of 'race,' but are more aligned with discrimination, segregation, healthcare inequality, and gene-environment interactions that develop over the lifecourse.

Hogue and Bremner (2005) introduced an integrative framework (see also Hogue, Hoffman, & Hatch, 2001) mirroring the epidemiologic agent-host-environment triad that

specified a range of mediating and moderating pathways of disease susceptibility as well as protection or immunity to the noxious effects of racism as a chronic psychosocial stressor. They coined the notion of 'stress age' which was similar to concept of weathering.

Heeding to an expanded structural context, David and Collins (2007) called for a more enlightened understanding of racial disparities as problem not only for impacted races, but as an indicator of health in the broader population. They commented that the heightened focus on race deflected attention from the salience of *class* inequalities which should be a concern for a large portion of Americans and an issue that can foster alliances and power across class lines to redress health disparities. Therefore, the causal link to infant mortality and other health disparities was attributed not only to race inequalities, but to class disparities as well.

Dominguez (2008, 2011) and Alio et al. (2010) are noted for their explication of the multidimensional nature of racism with its structural as well as individual and interpersonal components. Racism is fundamental (Dominguez, 2008) and embedded (Alio et al., 2010) in all aspects of Black women's lives, which transfers to differential stressors and health risks with consequential racially-disparate perinatal health outcomes.

Finally, commentaries that focus on biological (Morello-Frosch & Shenassa) or epigenetic (Burris & Collins, 2010) aspects of perinatal health disparities have adopted frameworks that conceptualize unequal social systems over the lifecourse which can initiate biogenetic changes that influence disparate perinatal health outcomes.

The contributions that the above papers have made are encouraging, but the degree to which the ideas have carried over into the broader corpus of perinatal health studies is debatable. Integrated with the following review of causal factors studied by perinatal health researchers, I discuss the extent to which the literature has embraced these alternative conceptualizations, with

a particular focus on how racism has been acknowledged as a determinant of perinatal health disparities.

Leading Explanations for Racial Disparities

General/Multifactorial/Integrative. The eight-volume *Report of the Secretary's Taskforce on Black and Minority Health* (1986) was the first official report to summarize epidemiological data and scientific findings on racial/ethnic disparities for all major U.S. minority populations. In a companion review of infant mortality and low birthweight literature developed for the report (Samuels, 1986), socioeconomic status and healthcare access were identified most prominently as areas contributing to the disparities. Although discrimination was not addressed among the report's key findings or recommendations, discrimination was discussed in the review with prescient attention to the role of discrimination, "compounded by a loss of cultural identity and traditional support systems" (Samuels, 1986, pp. 49-50), as a contributing factor for poor maternal-infant health among minority populations adopting the lifestyles of the dominant culture.

Under the auspices of the World Health Organization (WHO), Kramer (1987) prepared a systematic review of low birthweight comprised of studies from 1970 to 1984. Racial/ethnic origin was one of the many "causal factors" under investigation, and it was discussed in the context of exploring possible genetic contributions to low birthweight. Among the 67 studies reviewed for 'race-based' explanations, most characterized as lacking in methodological rigor, Kramer concluded that racial differences in mean birthweight was itself insufficient to establish an independent genetic contribution of "race" or ethnicity. Kramer also noted the importance of controlling for SES, cultural, and environmental factors that vary by race/ethnicity.

Another oft-cited comprehensive review is by Berkowitz and Papiernik (1993), who summarized the epidemiology of preterm birth, which by that time had eclipsed low birthweight

as the focal pathology for prevention of neonatal morbidity and mortality. Similar to the WHO review publication (M. S. Kramer, 1987), Black race was noted as an "established risk factor" for preterm birth. However, Berkowitz and Papiernik (1993) showed preference for differences in "psychological stress stemming from social deprivation" (p. 420) and unmeasured economic, psychosocial, environmental, and medical factors as contributing to Black-White disparities. The review expressed minimum support for the theory of earlier maturation of Black fetuses as evidence of a genetic component to racial disparities.

A comprehensive review on preterm birth compiled by the Institute of Medicine (Behrman & Butler, 2007) acknowledged the complex and interactive nature of many different factors that would contribute to preterm birth—ranging from genetics to environmental exposures—as well as population differences in the burden of preterm birth. Continuing the 'risk' orientation for identification of contributing factors, race-ethnicity was acknowledged as one of the more prominent risk factors, along with maternal age (< 16 and > 35), marital status, and SES. The report acknowledged that no individual-level factors have consistently been associated with preterm birth, and made it clear that the causes for the disproportionate rates of preterm birth by race/ethnicity are largely unknown. The report highlighted socioeconomic conditions, maternal behaviors, stress, infections, and genetics as the most promising areas for understanding racial disparities in preterm birth.

Reviews that characterized "multifactorial" causation (Headley, 2004; MacDorman, 2011; Patrick & Bryan, 2005) tended to acknowledge myriad and complex factors that contributed to preterm birth as well as racial disparities (Anachebe, 2006; Hauck, Tanabe, & Moon, 2011). However, a few studies moved beyond explicating multiple factors to articulating integrative causal frameworks. For example, Menon and colleagues (2011) conceived biological processes as being shaped by multiple factors (e.g., lifetime stress, nutritional deficiencies,

genetic variation, and behavioral, physical, and psychosocial factors) that interacted with each other to confer infection risk for preterm birth.

Social Context/Lifecourse. A number of reviews and commentaries were characterized by attentiveness to the social context. A lifecourse framework was articulated as early as 1989 when Emanuel and colleagues hypothesized that a mother's childhood environment was just as important as her current pregnancy for influencing maternal-fetal health risks (Emanuel, Hale, & Berg, 1989). In addition, Yankauer (1990) offered commentary that emphasized the role of social disadvantage to understand racial disparities in infant mortality. The study of perinatal health disparities has also benefitted from the articulation of a biopsychosocial framework that emphasizes the social context of race through which psychosocial stressors promote physiological mechanisms that cause undue harm to African American pregnancies (Rowley et al., 1993).

In 1992, CDC investigators began to highlight social context and the structural burdens endured by women due to systemic racism (Hogan & Ferre, 2001; Rowley, 2001). Relatedly, Hogue and colleagues (2001) developed a schematic, borrowing from the epidemiologic-triad of agent-host-environment, to model racism as a chronic stressor both interpersonally and in the systemic environment. This model was updated with the notion of "stress age" (Hogue & Bremner, 2005), a concept similar to "weathering" (Geronimus, 1992).

Similar integrative approaches were conceptualized to link social and biological paradigms. Wise (2003), concerned that social and biological disciplines had become too disparate in their approaches, aimed to reconcile this "disciplinary antagonism" with shared goals focused on intervention and parsimonious approaches that could benefit from interdisciplinary input. He reinforced that strategies must be uniquely directed to address racial *disparities* in infant mortality as distinct from tackling infant mortality more generally.

Racism-related Stress. Among the 68 identified reviews and commentaries that specifically addressed *causation* for racial disparities in birth outcomes, three highlighted a racism-stress framework (Dominguez, 2011; Giscombe & Lobel, 2005; Hogue & Bremner, 2005). Hogue and Bremner (2005) provided the most explicit description and integration of how racism contributes to stress, utilizing a host-agent-environment model incorporating a range of psychosocial and biological risk and protective factors that confer relative susceptibility or immunity for individuals impacted by racism as an acute or chronic stressor. The chronic stress of interpersonal and internalized racism was modeled as the putative "agent" which impacts health through a host of possible psychological, socio-cultural, economic, environmental, and gene-environment moderators and mediators. The novel conceptualization with this model was its emphasis on mediating and moderating pathways, which provided a more refined understanding of how racism-related stress impacts perinatal health. It offered ways for understanding the health pathology as well as ameliorative mechanisms with exposure to chronic racism. However, this conceptualization did not emphasize structural components of racism.

Giscombe and Lobel (2005) provided a more substantial overview of racism-related stress as a factor for adverse birth outcome in Black women. Consistent with findings from prior studies, they highlighted that racism is a chronic and unique type of stressor for Black women. Therefore, to not identify racism would serve to underestimate the impact of stress for Black women. They explained that African American women may not necessarily have more frequent stress, but that they are more susceptible to the adverse consequences of stress. This assertion is supported by studies which indicate greater cardioreactivity in African American women to simulated stressors (Hatch et al., 2006; Lepore et al., 2006; McNeilly et al., 1995) as well as greater allostatic load levels (Chyu & Upchurch, 2011), inflammatory markers (Christian, Glaser, Porter, & Iams, 2013; Picklesimer et al., 2008), and hypertension prevalence (Hicken,

Lee, Morenoff, House, & Williams, 2014) in relation to stress. However, studies have also demonstrated no direct association between racism-related stress and adverse physiologic responses (Albert et al., 2008; Krieger et al., 2013) or negative birth outcomes (Harville, Gunderson, Matthews, Lewis, & Carnethon, 2010; Wallace et al., 2013) in Black women.

Dominguez (2011) also reviewed racism and birth outcomes through the lens of a stress and health paradigm. Racism was viewed as an antecedent for stress-induced physiology which precipitates adverse birth outcomes. She emphasized the multidimensional nature of racism (institutional, interpersonal, and internalized) and gave significant weight to institutionalized racism as a source for disparities.

The chronicity and virulence of lifelong racial disadvantage was postulated to facilitate premature aging and consequently impair Black women's reproductive health. This perspective was similarly theorized as allostatic load (McEwen, 1998), 'weathering' (Geronimus, 1992, 2001), and 'stress age' (Hogue & Bremner, 2005). Weathering symbolizes the process of accelerated aging (in the face of chronic social and economic disadvantage) that translates into differential age-specific reproductive health risks and outcomes for Black women relative to non-Hispanic White women. Stress age is influenced by women's responses to the stressful "agent" of racism manifested at the individual and environmental levels—mediated and moderated by levels of host susceptibility (risk factors) as well as immunity (protective factors). Heightened and sustained susceptibility promotes stress age. Measures of chronic stress, especially *self-reported* measures of racism, have had the most significant association with birth outcomes in Blacks (Giscombe & Lobel, 2005). Nonetheless, studies have found no association between racism-related stress *physiology* and birth outcomes in studies designed to examine this link (Harville et al., 2010; Wallace et al., 2013).

Neighborhoods/Structural Context. Studies have found support for the impact of racial residential segregation on birth outcomes. Segregation was found to be an independent predictor of Black infant mortality in several major cities (Polednak, 1991, 1996). After controlling for neighborhood poverty and individual-level risk factors, Grady (2006) found that greater levels of segregation predicted lower birthweights in New York City. Hypersegregation has been associated with higher rates of preterm birth in Black women and larger Black-White disparities relative to less racially-segregated areas (Osypuk & Acevedo-Garcia, 2008). Residential isolation segregation predicted very preterm births in Black women and explained 28% of the geographic variation in Black-White disparities across U.S. metropolitan areas (M. R. Kramer, Cooper, Drews-Botsch, Waller, & Hogue, 2010).

Notwithstanding, segregation and other indicators of neighborhood disadvantage do not always influence birth outcomes in the predicted manner. Roberts (1997) had an unexpected finding that African American mothers who lived in neighborhoods with high Black racial segregation were less likely to have low birthweight births than their counterparts in less segregated neighborhoods—controlling for individual factors such as socioeconomic status. Bell and colleagues (2006) found that the clustering, or contiguousness, of African American neighborhoods was associated with more favorable birth outcomes than when the dissimilarity index (a measure of social isolation) was employed. African American women living in wealthier, non-minority neighborhoods were found to have *increased* risks of LBW and PTB compared to their counterparts living in census tracts with predominantly Black populations (Pickett, Collins, Masi, & Wilkinson, 2005). Moreover, Papacek et al. (2002) found *lower* postneonatal mortality in neighborhoods considered at risk in terms of unemployment, homicide, median income, and lead poisoning.

The inconsistency of neighborhood effects on birth outcomes is illuminated when considering how foreign-born Black populations in the U.S. are impacted. Immigrant Blacks in New York City were found to have lower rates of LBW in high poverty neighborhoods and surprisingly higher rates of LBW in neighborhoods with low levels of poverty (Grady & McLafferty, 2007). In another study (Fang, Madhavan, & Alderman, 1999), foreign-born Blacks in low income communities had lower rates of LBW (relative to Whites) than their foreign-born counterparts in middle- and high-income census tracts in New York City.

Clinical/Healthcare. Following the seminal report by Kessner (1973) on risk factors for infant mortality, prenatal care received significant attention as a modifiable risk factor towards reducing racial disparities in birth outcomes. However, more recent evidence indicates that prenatal care does little to *prevent* spontaneous preterm birth (although healthcare professionals may be more readily able to predict its inevitable occurrence), and it does not consistently contribute to reducing racial disparities (Lu, Tache, Alexander, Kotelchuck, & Halfon, 2003; Rowley, 1995). Some empirical studies selected for this review indicated no reduction in Black-White disparities in birth outcomes with adjustment for prenatal care (Alexander & Cornely, 1987; Coley & Aronson, 2013; Cox, Zhang, Zotti, & Graham, 2011; Rowley, 1995). Conversely, Sparks (2009) demonstrated that controlling for prenatal care dramatically reduced Black-White disparities in preterm birth. One reason for the relative ineffectiveness of prenatal care to reduce disparities in African American women is that African American women tend have high rates of pre-existing chronic health conditions that can negatively impact pregnancy (Arbour, Corwin, Salsberry, & Atkins, 2012; Kiely et al., 2011; Sabol et al., 2014). In two empirical studies selected for the present review (Orr, Blackmore-Prince, James, Griffin, & Raghunathan, 2000; Sparks, 2009), controlling for pre-pregnancy history and medical complications reduced the Black-White disparity in preterm birth.

Differential access to healthcare can be one manifestation of structural racism that influences birth outcomes. Structural inequality can foster segregated healthcare environments that lack the resources to assure quality medical facilities and optimal healthcare for women and children (Griffith, Childs, Eng, & Jeffries, 2007; Haas et al., 2004; Mayberry, Mili, & Ofili, 2000). High-risk obstetric and neonatal offices are often located outside the communities of the women most in need of such services and who lack adequate transportation or economic resources to travel long distances for healthcare. Fossett and colleagues (1990) described how low reimbursement rates, restricted access to provider staff, and residence in concentrated areas of poverty negatively impacted access to quality perinatal services among women receiving Medicaid.

Some advances in perinatal healthcare have had some benefit in improving overall maternal-infant health and survival, but unfortunately, have not reduced racial disparities between Blacks and Whites. For example surfactant and antenatal steroid therapy designed to improve respiratory function in preterm infants has actually resulted in *widening* racial disparities because White infants received greater benefit from these therapies than Black infants (Rowley & Hogan, 2012). Incidentally, VLBW Black infants were more likely to survive than VLBW White infants until these groundbreaking therapies were introduced to the disproportionate advantage of White infants. Moreover, neither progesterone therapy to prevent preterm birth nor the "Black to Sleep" campaign to reduce sleep-related deaths have systematically reduced disparities among non-Hispanic Black infants (Rowley & Hogan, 2012).

Biogenetic/Environmental. Genetic explanations perhaps provide the most provocative explanation for racial disparities in adverse birth outcomes. The fairly robust activity in this area has prompted studies specific to genetic causes for preterm birth (Anum, Springel, Shriver, & Strauss, 2009; Esplin, 2006). However, a genetic contribution to racial disparities has not been

shown (Behrman & Butler, 2007). In addition, perinatal infections, which pose a risk for preterm birth in general, remain somewhat inconclusive as a reason for racial *disparities* (Fiscella, 2004; Menon et al., 2011). Despite the substantial interest in bacterial vaginosis (BV), a common vaginal infection that is found more disproportionately in African American women and which can lead to pregnancy complications if the infection ascends to the upper genital tract, BV has not conclusively predicted racial disparities in birth outcomes (Macones et al., 2004).

There is little support for the possibility that single allelic variants could explain the complexity of preterm birth. However, genetic explanations for racial disparities in birth outcomes have been conceptualized within a lifecourse framework wherein a mother's epigenetic profile is understood as being developed over her lifetime and transgenerationally (Burris & Collins, 2010). Also, genetic explanations have been used to refine our understanding of inflammatory mechanisms during pregnancy. Of note is the research on pro-inflammatory cytokine genes, particularly interleukin-6 (Christian et al., 2013). There also has been support for the role of TNF alpha and spontaneous PTB in association with BV (Macones et al., 2004). The SERPINH1-656T allele has been associated with preterm births and is presumably very prevalent in persons of African and African American ancestry (Anum et al., 2009). Current research documents ancestry-linked genes that elevate obstetric risks in African American women (Anum et al., 2009; Wang et al., 2006), although such studies are rare and cannot be deemed conclusive for genetics as a causal factor for racial disparities. Remarkably, some investigations have declared genetic explanations for birth outcomes even in the absence of supportive genetic data or findings (Conley & Bennett, 2000; Kistka et al., 2007).

Several biogenetic researchers have highlighted epigenetic explanations wherein environmental influences are understood to cause differential gene expressions (due to on and off "switching" of genes) in ways that can be pathologic. This acknowledgement reinforces the

social context and diverts from centering causation solely at the biological level. For example, Fiscella and colleagues (2004), in their review of mediating mechanisms of intrauterine infection and microvascular dysfunction that can help to explain infant and maternal mortality outcomes, attributed the social context of Black women's lives, inclusive of psychosocial stress and intergenerational factors, as the true antecedent risk for these mechanisms. In a similar vein, environmental hazards interacting with individual and place-based stressors have been noted to contribute to adverse birth outcomes (Burris, Collins, & Wright, 2011; Miranda, Maxson, & Edwards, 2009; Morello-Frosch & Shenassa, 2006).

Demographic. The standard practice of examining "race" as the most-cited 'risk factor' for racial disparities in adverse birth outcomes elevates demographic factors as a leading associative cause. Although the field appears to have benefitted from critical discussions of race and its use in biomedical and epidemiologic research (M. W. Foster, 2009; Jones, 2001; Kaufman & Cooper, 2001; Krieger, 2005), research still appears where one can infer a perspective of 'genetic racialism' in the both the investigation and commentary (Kistka et al., 2007). Although it is implied that the observation of more favorable birth outcomes in immigrant Black women contradicts the notion that genetics is a likely explanation for racial disparities (cf. Hauck et al., 2011), such an assertion implicitly buys into the understanding of "race"-specific genes which would contribute to birth outcomes. The understated claim is that populations with African ancestry share similar race-associated genes, and on *this* basis, one would *expect* them to have similar birth outcomes. From an alternative viewpoint, Kramer et al. (2011) affirmed that the more favorable outcomes among immigrant women were related to differential social and health-related exposures over the lifecourse—not 'race' in terms of presumed genetic differences.

Other demographic variables, such as age, were shown to have differential effects by race. This literature review did not conceptualize age as a "causal" factor, and hence studies focused on age-related differences in perinatal health risk (i.e., "weathering") were not included (a separate treatment of this topic is reserved for Chapter 5). Nonetheless, studies have shown different age-related slopes for maternal and infant health risks wherein adverse birth outcomes appear at earlier ages and with greater magnitude for non-Hispanic Black women relative to White women (Buescher & Mittal, 2006; Geronimus, 1992; Holzman et al., 2009; Rauh, Andrews, & Garfinkel, 2001) and for Black women in high-poverty neighborhoods (Collins, Simon, Jackson, & Drolet, 2006; Geronimus, 1996; Love, David, Rankin, & Collins, 2010). The literature on weathering conceptualizes these age-related disparities as a consequence of cumulative social-structural disadvantage in African American populations and living environments resulting in accelerated aging and associated declines in reproductive health.

Another focus of demographic discussion is the role of nativity in perinatal health disparities. Borrowing from theories developed to explain the paradoxical health advantage of Mexican immigrants, *selective migration* (Fang et al., 1999; Valanis & Rush, 1979); *cultural factors* (Friedman et al., 1993; Fuentes-Afflick, Hessol, & Perez-Stable, 1998); and *dietary behaviors* (Cabral, Fried, Levenson, Amaro, & Zuckerman, 1990; K. D. Rosenberg, Desai, & Kan, 2002) have been advanced as explanations for perinatal health disparities between immigrant Blacks and African Americans. In addition, exposure to U.S. *racism* also has been proposed as a reason for why foreign-born Blacks have better perinatal health outcomes than African Americans (Collins, Wu, & David, 2002; Dominguez et al., 2009; Pallotto, Collins, & David, 2000; Stein et al., 2009). Notwithstanding, the theoretical justifications for immigrant health differentials are not as fully articulated by researchers studying Black immigrants. A more detailed review of empirical studies of nativity status and birth outcomes in U.S. Black

populations is provided in the section "Nativity and Birth Outcomes" that appears later in this chapter.

Socioeconomic. Socioeconomic status (SES), particularly income and education, are routinely examined in perinatal health studies (Blumenshine, Egerter, Barclay, Cubbin, & Braveman, 2010). Because SES is viewed as a confounder of perinatal health risk, rather than a causal factor, per se, few studies examine socioeconomic factors as a main source for the racial disparities in perinatal health. The PubMed search strategy produced only two studies where socioeconomic factors were the main focus for examining racial disparities (Colen, Geronimus, Bound, & James, 2006; M. S. Kramer et al., 2001), but other pertinent studies regarding SES and birth outcomes in Black women were identified manually. A mediating link between SES and adverse birth outcomes could be behavioral factors (Schempf, Strobino, & O'Campo, 2009) or the accumulation of chronic stressors (Giurgescu et al., 2012; M. S. Kramer et al., 2001). Complex causal pathways are being investigated to illuminate these and other linkages between SES and preterm birth (M. S. Kramer et al., 2001).

Some general observations hold true among studies. The usual *within-group* pattern of disparity is that women with high SES tend to have healthier birth outcomes than women with low SES. However, unlike the strong SES gradient observed for non-Hispanic Whites, this gradient has been found to be weak or non-existent for Blacks (Nepomnyaschy, 2009). Patterns between SES and birth outcomes also vary when examining *between-group* differences, depending on the chosen measure of socioeconomic position or the birth outcome studied (Blumenshine et al., 2010; Braveman, Cubbin, Marchi, Egerter, & Chavez, 2001; Savitz, Dole, & Herring, 2006). Nonetheless, studies generally find that socioeconomic status does not fully account for Black-White disparities in birth outcomes (Blumenshine et al., 2010). In fact, adjustment for SES often shows *no* impact on reducing the relative effects for adverse birth

outcomes between Blacks and Whites (Collins & Butler, 1997; Din-Dzietham & Hertz-Picciotto, 1997; McGrady, Sung, Rowley, & Hogue, 1992). A recent study of over 10,000 births in California from 2003-2010 (Braveman et al., 2014) did not find any racial disparities in preterm birth between socioeconomically disadvantaged Black and White women in adjusted models; disparities were only present among Blacks and Whites at high socioeconomic levels.

It has also been observed that traditional SES measures (i.e., income, education, employment) do not have equivalent effects across racial groups (David & Collins, 1991; Kaufman, Cooper, & McGee, 1997; Kessel, Kleinman, Koontz, Hogue, & Berendes, 1988; Kleinman & Kessel, 1987). For example, researchers assert that African Americans (Shapiro, 2004) and Black immigrants (Dodoo & Takyi, 2002; Model, 1995) do not reap the same economic or health benefits from higher levels of income or education. These contentions have been supported in perinatal health studies. For example, Schoendorf and colleagues (1992) found that infants born in families where *both* mother and father were college-educated had higher rates of low birthweight, which contributed to higher rates of infant mortality for Blacks relative to Whites at the same parental educational level. A survey of college graduates in Atlanta revealed higher odds of low birthweight and preterm birth among first-born infants to Black compared with White women, adjusting for medical complications and accounting for non-response bias (McGrady et al., 1992). Of note, studies also find that Black-White disparities *widen* at higher SES levels (Collins & David, 1990; Din-Dzietham & Hertz-Picciotto, 1998) or they exist *only* among high SES women (Braveman et al., 2014).

Findings of racial disparities at high income levels have also been confirmed with longitudinal studies that assess the impact of social mobility on birth outcomes. A study (H. W. Foster, Wu, Bracken, Semenya, & Thomas, 2000) that compared descendants of high-SES Black and White women who graduated from Meharry Medical College and Yale School of Public

Health, respectively, found that the Black-White relative risk of adverse birth outcomes for third generation descendants of these women was 1.78 (95% CI: 1.03, 3.09) for LBW and 3.16 (95% CI: 1.89, 5.27) for PTB. (The study did not clarify whether the descendants were also high-SES. Also, these descendants could have been children of the daughters *or sons* of the index graduates.) Colen and colleagues (2006) examined income mobility and low birthweight among females in the Longitudinal Survey of Youth (1979-2002) surveyed from childhood to adulthood. They found that each natural log increase in income level for White women was associated with a 48% decrease in LBW (*OR* = 0.48, 95% CI: 0.30, 0.75); the corresponding decrease for Black women suggested a beneficial trend, although the findings did not attain statistical significance (*OR* = 0.75, 95% CI: 0.51, 1.09).

Attributing socioeconomic status to differential birth outcomes is particularly problematic in perinatal health research because of the reliance on mother's education obtained from birth certificate data. As described, the benefits of education vary by race, with Black women attaining sub-optimal benefit from their educational achievement when translated to infant health outcomes. Moreover, education level is not a reliable proxy for income level (Braveman et al., 2001). To address the absence of income data in birth records, researchers have begun to capture contextual socioeconomic factors that can be related to maternal and infant health. For example, studies have examined neighborhood income (Ward, Mori, Patrick, Madsen, & Cisler, 2010) and other neighborhood socioeconomic characteristics (Collins & David, 1990; Messer, Kaufman, Dole, Savitz, & Laraia, 2006; Pearl, Braveman, & Abrams, 2001) in relation to birth outcomes. Overall findings demonstrate that neighborhood socioeconomic disadvantage is associated with adverse birth outcomes, and this effect is more consistent for U.S. Black residents than White. However, there are mixed findings for Black immigrants such that living in a low-income

community does not always predict worse birth outcomes (Fang et al., 1999; Grady & McLafferty, 2007), and this is counter to the expected inverse relationship (Bloch, 2011).

It is rare for studies to examine employment in relation to perinatal health disparities by race. Within the occupational health literature, physically demanding work has been associated with increased odds of preterm birth (Bell, Zimmerman, & Diehr, 2008; Mozurkewich, Luke, Avni, & Wolf, 2000). Black women who work tend to have lower rates of LBW than their non-working counterparts (Meyer, Warren, & Reisine, 2010; Poerksen & Petitti, 1991), and the perinatal health benefits of employment increase for women in professional vs. low-skilled occupations (Meyer et al., 2010). Also, "job strain" (low control, high demand work) was found to have a stronger association to preterm delivery for Black women than White women (Brett, Strogatz, & Savitz, 1997), and less occupational stress/fatigue was associated with lower rates of PTB in Black women (Hickey et al., 1995).

Behavioral. Behavioral explanations are generally insufficient and often insignificant to explain racial disparities in birth outcomes (Berg, Wilcox, & d'Almada, 2001; Goldenberg et al., 1996). For example, African American women are generally known to smoke less than White women both before and during pregnancy (Ebrahim, Floyd, Merritt, Decoufle, & Holtzman, 2000; Phares et al., 2004; Tong, Jones, Dietz, D'Angelo, & Bombard, 2009), but this does not appear to reduce the disparities in low birthweight or preterm birth. Among the small proportion of Black and White women who use illicit drugs during pregnancy, no systematic racial differences in rates of usage have been found (Chasnoff, Landress, & Barrett, 1990; Kunins, Bellin, Chazotte, Du, & Arnsten, 2007; Serdula, Williamson, Kendrick, Anda, & Byers, 1991). It should be noted that health risk behaviors are often confounded by factors such as SES (Finch, Frank, & Hummer, 2000).

Synopsis and Discussion of Leading Explanations for Racial Disparities in Birth Outcomes. Despite varying suggestions and targeted studies of potential leading causes for racial disparities in birth outcomes, there is no distinct reason(s) that can be accepted definitively based on current scientific evidence. The source for racial disparities has largely been attributed to socioeconomic factors (Berkowitz & Papiernik, 1993; Samuels, 1986) and psychosocial stress (Behrman & Butler, 2007; Berkowitz & Papiernik, 1993) with emerging interest in maternal behaviors, genetics, and infections (Behrman & Butler, 2007). However, none of these causes have been substantiated scientifically as a primary determinant of racial *disparities*, and, hence, the reasons for Black-White disparities must be characterized as unknown.

Traditional sociodemographic risk factors do not substantively explain racial disparities, which may be due in part to qualitatively different effects and therefore a high potential for measurement bias using standard income and education measures across racial/ethnic populations. It is notable that adjustment for SES often shows little to no impact on reducing the relative effects for adverse birth outcomes between Blacks and Whites (Blumenshine et al., 2010; Collins & Butler, 1997; Din-Dzietham & Hertz-Picciotto, 1997; McGrady et al., 1992), and racial disparities in birth outcomes are more likely to be observed among high-SES Black and White women than their low-income income counterparts (Braveman et al., 2014; Schoendorf et al., 1992). The reason for this inequity may stem from lower levels of wealth accumulation and diminishing economic returns for Black women relative to other races at equivalent levels of education or income. Racism due to residential segregation and employment discrimination or the effects of racism-related vigilance in racially-isolated professional settings can take an economic and health toll on higher-educated Black women (Lekan, 2009; Mullings, 2005).

Behavioral and healthcare measures also do not explain the racial gap in adverse birth outcomes. Black women smoke less than White women (Ebrahim et al., 2000; Phares et al., 2004; Tong et al., 2009) and have no differential rates of alcohol and substance use during pregnancy (Chasnoff et al., 1990; Serdula et al., 1991). Behavioral risk factors did little to explain racial disparities in LBW and PTB (Berg et al., 2001; Goldenberg et al., 1996). Moreover, most evidence indicates little to no effect of prenatal care on reducing racial disparities (Alexander & Cornely, 1987; Coley & Aronson, 2013; Cox et al., 2011; Lu et al., 2003; Rowley, 1995), although some benefit has been shown in select studies (Orr, Reiter, Blazer, & James, 2007; Sparks, 2009).

In published research and commentary, 'social disadvantage' is more likely to be mentioned than 'racism' as a leading contextual cause. Social disadvantage is usually operationalized as racial segregation or census tract income levels that are examined in neighborhood studies. Although neighborhood socioeconomic disadvantage has been associated with adverse birth outcomes, the effects are inconsistent. For example, racial segregation or neighborhood poverty does not always predict higher LBW or PTB (Bell et al., 2006; Grady, 2006; Pickett et al., 2005; Roberts, 1997) in Black women. In current studies, Black immigrant mothers in high poverty or highly segregated neighborhoods (in New York City) have consistently fared better than their counterparts in more affluent or less racially segregated areas (Fang et al., 1999; Grady & McLafferty, 2007).

The differential impact of social-environmental stressors/stress on maternal-fetal biology is a hypothesized pathway linking social disadvantage to biogenetic aberrations which precipitate disparities in adverse birth outcomes (Burris et al., 2011; Fiscella, 2004; Miranda et al., 2009; Morello-Frosch & Shenassa, 2006). Although racism-related stress has been conceptualized as a primary cause for perinatal health disparities by race (Dominguez, 2011; Giscombe & Lobel,

2005; Hogue & Bremner, 2005), and there is evidence of differential markers of stress (i.e., inflammation, allostatic load) in African American women (Christian et al., 2013; Chyu & Upchurch, 2011; Picklesimer et al., 2008), targeted studies have found no direct association between racism-stress *physiology* and adverse birth outcomes (Harville et al., 2010; Wallace et al., 2013). Moreover, a genetic cause for racial disparities in birth outcomes has not been demonstrated (Behrman & Butler, 2007; M. S. Kramer, 1987).

The relative lack of knowledge about what drives racial disparities in birth outcomes may be attributed to: the generally undeveloped causal understanding of some birth outcomes such as preterm birth (Behrman & Butler, 2007) – much less racial disparities in these outcomes; the limited scope of socio-demographic and behavioral health data within standard birth records (the primary source for population health information on birth outcomes); and the impracticality of systematic measurement and research of contextual or structural variables (Savitz et al., 2006). Nonetheless, among the wide assortment of interacting social, environmental, behavioral, psychosocial, and biological factors presumed to contribute to racial disparities (MacDorman, 2011), racism relates to all domains, and hence it is worthy of more in-depth examination as a central source for racial disparities in birth outcomes. Racial discrimination and structural disadvantages by race are key sources of stress and disproportionate hardship for Black women (Dominguez, 2011; Giscombe & Lobel, 2005; Hogue & Bremner, 2005; Nuru-Jeter et al., 2009; Rowley et al., 1993). The relevance of racism in the lives of Black women has propelled a detailed examination of how racism has been empirically examined in relation to birth outcomes.

Racism and Birth Outcomes

The bulk of studies examining racism as a predictor of adverse birth outcomes have appeared in the past decade—with mixed findings. A systematic review including 27 studies of self-reported racism and birth outcomes indicated 15 that showed a positive association and 12

which showed no significant association (Paradies, 2006). This review did not specify which studies were associated with the positive or null findings, but it is likely that the list included studies outside the U.S. and/or included study participants not exclusive to non-Hispanic Blacks. (The present review identified only 13 studies that met the criteria for examining interpersonal racism or race-based discrimination associated with a birth outcome in U.S.-based non-Hispanic Black populations. Studies addressing racial segregation (n = 9) were addressed separately under neighborhood studies.) Giurgescu et al. (2011) submitted the latest systematic review of 10 U.S.-based studies on discrimination and birth outcomes. In general, perceived discrimination was shown to be positively associated with preterm birth and low birthweight, but not with gestational age. It is notable that no strong associative pattern has emerged as many studies report no statistical significance (alpha level = .05) or only marginal significance in predicting adverse birth outcomes.

The first documented empirical study of racism and birth outcomes was by Murrell (1996) who examined perceived racism in a prenatal clinic population (n = 165). Racism was measured using the Perceptions of Racism scale (Green, 1995), a 20-item scale of which 18 items assessed attitudes and options about hypothetical racial situations, and two items assessed direct experiences with racism. In this study, racism showed no association with low birthweight or gestational age, but racism was positively correlated with stress, measured as daily hassles. Shino and colleagues (1997) addressed racial discrimination within a study to determine pregnancy risk factors based in the social environment. Racial discrimination, as reported during pregnancy, was incorporated as part of a summative index including stressful life events, threats to physical safety, residence in public housing, social isolation, single parenthood, and unresolved housing. The authors reported that perceived discrimination was not associated with mean birthweight although the data were not separately shown.

Collins et al. (2000) used a hospital-based case-control design to compare discrimination, coping styles, and social support among *low-income* African American mothers delivering very low birthweight (VLBW) and non-VLBW infants. This study of 25 cases and 60 controls showed a three-fold effect (*OR* = 3.3, 95% CI: 0.9, 11.3) of perceived discrimination on VLBW net of social, medical, and behavioral risk factors. The lack of statistical significance could be related to the sample size as well as the fact that reports of discrimination were restricted to the pregnancy period. A follow-up study (Collins, David, Handler, Wall, & Andes, 2004) with a bigger sample of 104 cases and 208 controls showed a dose-response relationship of discrimination over the life course to VLBW in African American women. Compared to no reports of discrimination, discrimination in 1 or more domains increased the odds of VLBW by 1.7 (95% CI: 1.0, 9.2) and discrimination in 3 or more domains increased the odds of VLBW by 2.6 (95% CI: 1.2, 5.3). Interestingly, the women most at risk were older and college-educated.

Mustillo and colleagues (2004) used an existing survey on cardiovascular health to examine the association between reported race and gender discrimination to LBW and PTB in both Black and White women (*n* = 352). Discrimination helped to explain the Black-White odds ratio for LBW (*OR* = 2.11, 95% CI: 0.75, 5.93) and PTB (*OR* = 2.5, 95% CI: 1.33, 4.85). A research team utilizing the national Black Women's Health Survey found that racial discrimination on the job (*OR* = 1.3, 95% CI: 1.1, 1.6), but not in other areas, predicted preterm delivery among college-educated women (L. Rosenberg, Palmer, Wise, Horton, & Corwin, 2002). In a large, prospective cohort study (Dole et al., 2003), high levels of perceived discrimination predicted preterm delivery in Black women (*RR* = 1.4, 95% CI: 1.0, 2.0) independent of other forms of stress and adjusted for other risk covariates. Similar results (*RR* = 1.8, 95% CI: 1.1, 2.9) were reported in a follow-up study (Dole et al., 2004).

The most recent studies demonstrate a pattern of mixed findings. Dominguez et al. (2008) found that both lifetime racism and racism perceived vicariously in childhood predicted mean birthweight. However, a study by Dailey (2009), using the Everyday Discrimination scale (Forman, Williams, & Jackson, 1997; Williams, Yu, Jackson, & Anderson, 1997), showed no correlation between racial discrimination and infant birthweight, although age and disability discrimination were inversely associated. Misra et al. (2010), using the Racism and Life Experiences (RaLES) scale (Harrell, 1997b) found non-significant results for both acute and lifetime experiences of racism in association with PTB among 832 African American women in Baltimore. Carty and colleagues (2011) analyzed a community survey subsample of 510 women (330 Black and 180 White) who retrospectively reported their birth outcomes for a 10-year period. The adjusted Black-White odds ratio for emotional responses to racism (anger, frustration, etc.) in association with LBW was 1.17 (95% CI: 0.93, 1.48), with mother's race and highest education as covariates—thus indicating a marginal relationship. Finally, Rankin and Collins (2011) used a hospital-based case-control study of African American mothers with preterm births (i.e., cases, $n = 160$) and non-preterm births (i.e., controls, $n = 117$) to examine exposure to perceived interpersonal racial discrimination in public settings (lifetime and past year)—with a particular interest in the modifying effect of coping behaviors. Discrimination was measured with a modified version of the Perceived Racism Scale (McNeilly et al., 1996); tabulated as *frequencies* across the different experiences; and categorized as high or low/medium frequency of interpersonal discrimination. The relative odds of preterm birth for high vs. low/medium frequency was 1.5 (95% CI: 0.9, 2.8) for lifetime discrimination and 2.5 (95% CI: 1.2, 5.2) for past year discrimination. Selected active coping strategies attenuated the relationships.

Synopsis and Discussion of Racism and Birth Outcomes. Although the above-mentioned empirical studies of racial discrimination/racism and birth outcomes are few in number (n = 13), and findings are modest, they do demonstrate empirical support for the influence of perceived racism on adverse birth outcomes. Among the five studies (Collins et al., 2004; Dole et al., 2004; Mustillo et al., 2004; Rankin et al., 2011; L. Rosenberg et al., 2002) that reported statistically significant adjusted odds for LBW or PTB, midpoint estimates ranged from 1.3 to 2.6. Selected active coping strategies (i.e., 'working harder') resulted in attenuated odds ratios in one study (Rankin et al., 2011), but there was no modifying effect of passive or active responses to racism experiences in all other studies that examined this association (Carty et al., 2011; Collins et al., 2000; Dole et al., 2004; Misra et al., 2010; Mustillo et al., 2004).

Notwithstanding the supportive findings, this line of research is challenged by methodological limitations. First, discrimination and racism are measured inconsistently across studies—even when standard scales are used. The majority of studies have utilized the *Experiences of Discrimination* measure (Krieger, 1990; Krieger & Sidney, 1996) which asks about experiences of unfair treatment due to one's race in 5 domains: at school; obtaining medical care; with service at restaurants or stores; and seeking housing. Other studies have utilized the *Everyday Discrimination Scale* (Williams et al., 1997) which asks about day-to-day experiences of unfair treatment such as: being treated with less courtesy and respect; receiving poorer service in restaurants or stores; people acting as if they are afraid of the respondent; or they are better than the respondent; or they think the respondent is unintelligent; …or dishonest; or the respondent has been insulted; called names; or threatened/harassed. In addition, the 3-item *Major Discrimination Scale* (Williams et al., 1997) asks about experiences of unfair treatment related to employment hiring, employment termination, and encounters with the police. The intended usage for the Everyday Discrimination and Major Discrimination scales is for

respondents to attribute the source of the discrimination (e.g., race, gender, disability, age, etc.) only *after* reporting domains of unfair treatment; but researchers often choose to identify race at the onset as the reason for discrimination (see Carty et al., 2011). These scales are also intended as an additive index, but Rosenberg, Palmer, et al. (2002), for example, analyzed each item separately, using an adapted version of the *Everyday Discrimination* scale (Ren, Amick, & Williams, 1999). Murrell (1996) utilized the *Perceptions of Racism* scale (Green, 1995) where only 2 of 20 items assessed direct experiences with racism. Collins et al.'s (2004) use of the Perceived Racism Scale (McNeilly et al., 1996) enabled them to measure (and yield significant findings for) employment related racism which is not part of the Experiences of Discrimination Scale (Krieger, 1990; Krieger & Sidney, 1996) which they also utilized. The Perceived Racism Scale is also noted for its assessment of emotional and behavioral *responses* to racism, as well as experiences. Also, the *Racism and Life Experiences Scale* (RaLES) (Harrell, 1997a, 1997b) was used to assess coping responses/reactions to racism (Carty et al., 2011; Misra et al., 2010).

Second, self-reported experiences of discrimination (measured as a dichotomous "yes/no" variable or a categorized frequency variable) are not an optimal measure for the harmful effects of racism, which include contextual aspects that may not be consciously perceived or reported by participants. A few researchers (Dominguez et al., 2008; Jackson, Hogue, & Phillips, 2005; Nuru-Jeter et al., 2009; Rich-Edwards et al., 2001) have attempted to capture more nuanced perspectives of racism and elicit a lifecourse assessment of the impact of racism. Research into perinatal health disparities can also benefit from increased examination of structural racism. Examples include economic policies; race- and gender-biased employment; family, penal, and immigration policies; residential segregation; healthcare and healthcare financing systems; and other structural aspects of racial inequality that may impact birth outcomes.

More studies are needed, with more varied samples, to compare Black women of varying socioeconomic and demographic characteristics. Current findings are inconsistent regarding racism's impact on perinatal health among high-SES Black women (Collins et al., 2004) in comparison to low SES women (L. Rosenberg et al., 2002). However, reporting of racism tends to occur more frequently among higher-SES Blacks (Murrell, 1996), which is corroborated in psychological and sociological research. Moreover, how racism impacts birth outcomes in U.S. Black immigrants has not been empirically reported to date, although studies have been initiated (see Dominguez et al., 2009).

Another notable observation with the aforementioned studies is the variation in racism frameworks used. Several researchers explicitly embrace racism as a form of stress (Carty et al., 2011; Collins et al., 2004; Dole et al., 2003; Dominguez et al., 2008; Misra et al., 2010; Murrell, 1996; Rich-Edwards et al., 2001; L. Rosenberg et al., 2002). Others additionally highlight the institutionalization or structural forms of racism (Collins et al., 2004; Collins et al., 2000) or how racism is manifested over the lifecourse (Dominguez et al., 2008; Nuru-Jeter et al., 2009). The adoption of a racism-stress framework to explain racial disparities is not surprising considering that racism is a unique stressor for Blacks in the U.S. (Dominguez, 2011; Giscombe & Lobel, 2005), and physiological stress is a plausible mediating mechanism for how psychosocial stressors are related to adverse birth outcomes (M. R. Kramer et al., 2011; Rich-Edwards & Grizzard, 2005). However, the field has re-evaluated prior generalizations with respect to psychosocial stress and birth outcomes. Although earlier studies provided strong support that psychosocial stress is related to adverse pregnancy and birth outcomes (Dole et al., 2003; Istvan, 1986; Lobel, 1994; Paarlberg, Vingerhoets, Passchier, Dekker, & Van Geijn, 1995; Wadhwa, Sandman, Porto, Dunkel-Schetter, & Garite, 1993), a recent meta-analysis qualified the relationship as negligible (Littleton, Bye, Buck, & Amacker, 2010), and leading researchers of

stress and pregnancy have become more speculative about the relationships (Wadhwa, Entringer, Buss, & Lu, 2011).

Moreover, studies that examine racism-related stress exclusively are limited for not simultaneously measuring other psychosocial or behavioral stress responses that could lend insight into proposed pathways between perceived racism and impaired mental and pathophysiologic health states. However, many of the standard psychosocial stress measures—especially stressful life events—have been found inadequate for use with Black women (McLean, Hatfield-Timajchy, Wingo, & Floyd, 1993). Stressful life events in four domains (i.e., emotional, financial, partner-related, and traumatic) were all significantly higher in Black women (than White women), but none were found to predict preterm birth, nor did they contribute to Black-White disparities in preterm birth (Lu & Chen, 2004). In the studies selected for this review, stress was measured as daily hassles (Murrell, 1996); negative life events (Dole et al., 2004); pregnancy related stress (Dominguez et al., 2008); and general stress (Dominguez et al., 2008; Misra et al., 2010). Measures that specifically address racism and other chronic stressors are found to be more relevant for Black women's experiences (Dominguez, Schetter, Mancuso, Rini, & Hobel, 2005; Giscombe & Lobel, 2005; Hogue & Bremner, 2005; O'Campo & Schempf, 2005) and provide more robust and predictive associations with birth outcomes in Black women (Orr et al., 1996).

The articulation of causal links between racism and birth outcomes will be continually challenged in the absence of more sophisticated and valid measures of racism and stress coupled with studies designed to disentangle the effects of racism and stress at various points and with various intensities over the lifecourse. An increase in the number of prospective and longitudinal studies will help to elucidate the causal pathways linking racism to birth outcomes. Also, more population-based studies are needed to enhance generalizability.

Nativity Status and Birth Outcomes

There are a fair amount of empirical studies that have examined Black national origin with respect to birth outcomes. The present review identified 25 analytic studies examining differences in adverse birth outcomes between U.S.-born and foreign-born Black populations. The earliest studies were introduced in the 1970s and 1980s (Chavkin, Busner, & McLaughlin, 1987; Kessner et al., 1973; Valanis & Rush, 1979) followed by state and national studies during the past two decades that compared native and foreign-born Blacks generally (Acevedo-Garcia, Soobader, & Berkman, 2005; Cabral et al., 1990; Cervantes, Keith, & Wyshak, 1999; Collins, Soskolne, Rankin, & Bennett, 2013; Collins et al., 2002; Fuentes-Afflick et al., 1998; Grady & McLafferty, 2007; Howard, Marshall, Kaufman, & Savitz, 2006; Kleinman, Fingerhut, & Prager, 1991; Mason, Kaufman, Emch, Hogan, & Savitz, 2010; K. D. Rosenberg et al., 2002; Singh & Yu, 1996) or stratified by Caribbean or African national origin (Fang et al., 1999; Friedman et al., 1993; Howard et al., 2006; Liu & Laraque, 2006; Pallotto et al., 2000; Stein et al., 2009).

Studies met the full criteria for the present review if they specified Black race among the foreign-born or national ancestry populations studied and examined birth outcomes. However, this review also acknowledged studies of this subject matter that provided empirical data on pregnancy-related risks for Black immigrants in the U.S. but reported no associations with infant outcomes (Dominguez et al., 2009; Elo & Culhane, 2010); or designated foreign-born populations by maternal 'ancestry' (which is not synonymous with maternal nativity) (Howard et al., 2006); or indicated maternal birthplace but not race (Forna, Jamieson, Sanders, & Lindsay, 2003), hence race could only be presumed based on the racial prevalence of the immigrant's home country.

In the following sections, I summarize the reviewed studies according to the conceptual domains or causal factors stated by the authors as most likely to contribute to disparities in birth

outcomes between U.S.-born Blacks and foreign-born Blacks in the U.S. Almost all reviewed studies corroborated the general finding of more favorable birth outcomes in foreign-born Black women compared to U.S.-born African American women. [Exceptionally, Fuentes-Afflick et al. (1998) found no significant difference in VLBW (OR = 1.27, 95% CI: 0.83, 1.95) and only marginal difference in MLBW (OR = 0.83, 95% CI: 0.70, 1.00) between FB- vs. U.S.-born Blacks, after adjusting for maternal and infant risk factors.] Notwithstanding this common conclusion, studies of Black nativity are distinguished by varying conceptualizations and explanations for differences in maternal and infant health between foreign- and U.S.-born Black women.

National Origin as a Determinant of Black Maternal-Infant Health. Early descriptive examinations of immigrant and non-immigrant health highlighted maternal nativity status alone as a risk factor, with findings of lower LBW and infant mortality rates for foreign-born Blacks compared to U.S.-born Blacks (Chavkin et al., 1987; Kessner et al., 1973). County of origin was later explored as a modifier of the relationship between nativity status and birth outcomes. For example, Forna et al. (2003) stimulated attention to national origin differences in their analysis of maternal characteristics and pregnancy and birth outcomes at an Atlanta hospital. Caribbean-born and African-born women had varied estimates across a number of pregnancy and birth outcomes. However, the investigators did not specify maternal race or Hispanic ethnicity, and each national origin group was compared to *all* U.S.-born women in the sample as the reference group. For preterm birth, the relative risk was 0.69 (95% CI: 0.58, 0.83) for Caribbean-born women and a similar RR of 0.6 (95% CI: 0.55, 0.71) for African-born women. Although one can presume that women from the African and Caribbean regions constituted a majority of the 87% Blacks in the foreign-born sample, one cannot make a clear determination about how race may have factored into nativity-status differences.

In another study, with an all-Black sample, Howard and colleagues (2006) found that "ancestry" was more predictive of LBW and PTB for foreign-born Blacks than nativity status alone. Ancestry can refer to one's parentage, therefore ancestry and maternal nativity are not synonymous. Despite the findings for the overall foreign-born/U.S.-born sample, there were no significant differences in LBW or PTB when the "West Indian/Brazilian" group was compared to all U.S.-born Blacks (i.e., nativity) or compared to Blacks in the U.S. who could be second or later generation women with West Indian or Brazilian parentage (i.e., ancestry). Aggregating second or third generation descendants with first-generation immigrant mothers may mispecify true 'nativity effects.' However, classification by ancestry may be a more realistic barometer of the ethnic/cultural component of immigrant health than assuming that a second generation immigrant, although born in the U.S., has the same psychosocial, behavioral, and physical health profile as a native-born African American who is shaped by the historical and cultural context of generations with lifelong experiences in the U.S.

Other studies that have specified immigrant national origin have demonstrated that the health advantage for Caribbean immigrants relative to U.S.-born Blacks is less robust and closer to U.S. estimates than the differences observed between African immigrants and U.S.-born Blacks (Elo, Vang, & Culhane, 2014; Fang et al., 1999; Friedman et al., 1993; Liu & Laraque, 2006; Pallotto et al., 2000; Stein et al., 2009). Such variation would not have been uncovered without stratification of immigrant Blacks beyond foreign-born/U.S.-born status.

Ethnicity/Culture/Selective Migration of Black Immigrant Mothers. Many perinatal health studies presume ethnic and cultural differences between Black immigrants and their U.S. counterparts without a clear description or conceptual framework for what ethnicity or culture is supposed to represent or what aspects of these constructs would logically contribute to differential birth outcomes. For example, Cabral et al. (1990) conceptualized foreign-born status

as approximating 'cultural characteristics' although these characteristics were not defined. Friedman et al. (1993) cited acculturation as possibly contributing to ethnicity (defined by 'ancestry') differences, where ethnicity was regarded as a social category and a reflection of different experiences beyond race. Similarly, Howard et al. (2006) conceptualized ancestry and nativity as proxies for social and environmental contexts—as an alternative to genetically-based perceptions of ethnicity. Most studies, however, conflate nativity status with ethnicity and culture.

Researchers describing nativity differences are also not as critical with respect to articulating reasons for the Black foreign-born advantage. Perinatal health researchers often invoke selective migration/healthy migrant theories (Landale, Gorman, & Oropesa, 2006; Palloni & Morenoff, 2001; Wingate & Alexander, 2006) or cultural explanations (Callister & Birkhead, 2002; Sherraden & Barrera, 1996; Zambrana, Scrimshaw, Collins, & Dunkel-Schetter, 1997) to justify the health advantage of Black immigrants relative to African Americans—although these conceptualizations were developed and tested almost exclusively with Mexican immigrants.

The immigrant selectivity or healthy migrant theories contend that immigrant women have better maternal-infant health outcomes than their U.S.-born counterparts due to their unique socioeconomic, psychological, or cultural resources that favor optimal health. Therefore, successful migrants are not a random selection of their population (Landale et al., 2006; Palloni & Morenoff, 2001; Wingate & Alexander, 2006), but a highly-selected group whose *a priori* health advantages (including physical hardiness, motivation, resilience, and economic and material resources) fostered their migration in the first place—and this social selection trumps normal correlates of health such as socioeconomic status. The socioeconomic profiles for Black immigrants do tend to be more advantageous, particularly for immigrants from African countries. Therefore, immigrant selectivity is a viable proposition. A recent study explored the healthy

migrant theory exclusively in a U.S.-based Black maternal population (Wingate, Swaminathan, & Alexander, 2009)—finding that infant birth outcomes and sociodemographic profiles were most favorable among the foreign-born women, but also, U.S.-born Black women who migrated internally (i.e., moved within or across U.S. states or regions) during their pregnancies had better sociodemographic characteristics and birth outcomes than mothers who did not migrate. These findings corroborate that there may be an aspect of social selection that facilitates migration and is also associated with more optimal health.

Culture tends to be discussed in relation to its role in promoting protective health behaviors such as healthful 'ethnic' diets and tendencies to not smoke. Of the 25 studies selected for their emphasis on nativity status and birth outcomes in Black populations, three studies espoused an immigrant selectivity thesis (Fang et al., 1999; Urquia, Frank, Moineddin, & Glazier, 2010; Valanis & Rush, 1979), and seven highlighted cultural explanations (Cabral et al., 1990; Friedman et al., 1993; Fuentes-Afflick et al., 1998; Grady & McLafferty, 2007; Mason et al., 2010; K. D. Rosenberg et al., 2002; Rumbaut & Weeks, 1996).

Regarding immigrant selectivity arguments, Valanis and Rush (1979) postulated that childhood social status influences health over the lifecourse. In their comparison of foreign-, NYC-, and U.S. Southern-born Black women prospectively enrolled at a prenatal clinic, the foreign-born Blacks had higher infant birthweights. However, higher childhood social status (measured as parents' education) contributed a birthweight advantage for foreign-born women only. Similarly, Fang et al. (1999) adopted an immigrant selectivity thesis within a lifecourse framework by speculating that early life advantages offset poor circumstances later in life for Black immigrants. They found that in low income communities in New York City, the odds of LBW for immigrants from the Caribbean and Africa compared to U.S. Whites were 0.88 (95% CI: 0.79-0.97) and 0.86 (95% CI: 0.61-0.96), respectively. Therefore, Black immigrants

experienced an infant health advantage even in comparison to the native White population (no comparisons were made to U.S. Blacks).

Liu and Laraque (2006) did not find a healthy immigrant effect for migrant women from several Caribbean, African, and Central American countries. Migrant women from these countries had infant mortality rates (IMR) that were higher than the average IMR for all foreign-born women in NYC. Although race was not specified, race may have been a factor for the inter-regional disparities among immigrant women. It is of note that selective migration is less likely to occur among Caribbean nationals than Africans due to the closer regional proximity and more frequent familial migration and sponsorship for Caribbean migrants (Elo & Culhane, 2010; Kent, 2007; Pallotto et al., 2000). Therefore, national origin (i.e., African or Caribbean) is an important consideration for disaggregation when examining the foreign-born Black population in the U.S.

Cultural reasons for the Black foreign-born advantage are attributed to adaptation and acculturation to the U.S. society and loss of indigenous traditions such as reliance on extended social support networks that can be health protective (Friedman et al., 1993; Fuentes-Afflick et al., 1998; Grady & McLafferty, 2007; Mason et al., 2010). But these factors have not been directly tested among Black immigrants within a perinatal health framework. Also, 'cultural' reasons are often conflated with behavioral explanations, as described next.

Behavioral Health Risks and Black Immigrant Birth Outcomes. In the general public health literature, positive health behaviors are said to correlate strongly with socioeconomic position. However, in perinatal studies of Black immigrants, positive health behaviors are more often attributed to ethnic or cultural factors—particularly with respect to favorable practices regarding diet, smoking, and substance use. Caribbean and African immigrant women, for example, are significantly less likely to smoke (Elo et al., 2014) due to cultural norms that deter this practice among women. Accordingly, diet, smoking, and substance use are described as

mediators of the maternal-infant health advantage for foreign-born Black women in the U.S. relative to African Americans (for whom these health risk behaviors are more prevalent) (Elo & Culhane, 2010).

In the present review, three studies reinforced the role of health behaviors for the Black immigrant health advantage. In a prospective study of maternal risk and lifestyle factors among low-income Black women receiving prenatal care at a Boston hospital (Cabral et al., 1990), foreign-born Blacks (n = 201, 72% Caribbean) had more favorable perinatal health risks for marital status, education, pre-pregnancy nutritional status, prenatal care visits, and use of cigarettes, alcohol, and illicit drugs compared to African Americans (n = 616), and the risk of LBW births was lower for foreign-born Blacks, adjusting for these factors. K.D. Rosenberg et al. (2002) speculated that the greater reported intake of vegetables and fruits among Caribbean-born mothers confers a birth outcomes advantage compared to U.S.-born African American mothers who consume more sweets, fats, and proteins. However, detailed monitoring of micronutrient intake might have been a more valid measure of possible nutritional advantages than generalized reports of cultural food norms (see Batis, Hernandez-Barrera, Barquera, Rivera, & Popkin, 2011; Greenberg, Schneider, Northridge, & Ganz, 1998). Finally, a California study (Rumbaut & Weeks, 1996) to explore why immigrants had superior perinatal health outcomes provided little insight into Black immigrants except to conclude that foreign-born (FB) Black women had better diets than U.S.-born Blacks. It is noteworthy that this conclusion was based on only six FB-women in this study who reported less intake of fats and sweets than African Americans. But closer observation also reveals that these foreign-born women had less intake of fruits and vegetables, milk products, and protein, so it would be premature to generalize about the healthier diets of foreign-born Black women.

Health behaviors were the focus in a Philadelphia study of pregnant Black women by nativity (African-, Caribbean-, or U.S.-born) (Elo & Culhane, 2010). Foreign-born women had lower alcohol and substance use, better self-rated health, and less reported stress than native women, despite reporting *more* material hardship and *less* social support than African Americans. Notably, African immigrant women exhibited the most favorable health profiles relative to both Caribbean and African American women. The researchers speculated that Africans were more highly selected and had less lifetime exposure to racial discrimination and marginalization, which translated into a social and health advantage relative to Caribbean and U.S.-born women.

Socioeconomic Status and Black Immigrant Birth Outcomes. Valanis and Rush (1979) conceptualized socioeconomic and environmental influences, operating through behavioral and biological mediators, as a partial explanation for the enigmatic finding of superior birthweights among foreign-born women. In their prospective, clinic-based sample of Black women (n = 766) enrolled in prenatal care, higher childhood social status (measured as parents' occupational class) was associated with a lower proportion of LBW among foreign-born women compared to U.S.-born women.

Notwithstanding this finding, the more normative results across studies are that socioeconomic advantages do not guarantee improved health for foreign-born Blacks. Singh and Yu (1996) found that crude rates of LBW, PTB, and IM were not statistically different between U.S.- and foreign-born Blacks after adjusting for socioeconomic status (SES). Howard and colleagues (2006) likewise found that Black nativity differences in LBW and PTB were not explained by SES. Furthermore, socioeconomic status did not account for the health advantage for foreign-born and native Black pregnant women in relation to substance use and psychological health (Elo & Culhane, 2010).

Counterintuitively, Acevedo-Garcia et al. (2005) found that the protective effect for adverse birth outcomes was stronger for foreign-born Black women with *less* than a high school education compared to higher-educated counterparts. Pallotto and colleagues (2000) discovered an "enigma" in that the *lowest* risk (by age, college education, and marriage to college-educated fathers) Caribbean-born and U.S.-born Black mothers had similar rates of VLBW. But compared to Whites, these Black mothers' VLBW risks *worsened* with improved SES. Education, as reported on birth certificates, consistently did not explain the foreign-born perinatal health advantage (Erhardt & Chase, 1973; Kessner et al., 1973; Valanis & Rush, 1979). Although census data report more favorable socioeconomic profiles for Black immigrants than for African Americans (Kent, 2007), it appears that improved SES yields diminishing returns for foreign-born Blacks in terms of birth outcomes. The reason for this inequity may stem from diminishing economic returns for foreign-born Blacks relative to other ethnic groups at equivalent levels of education or income (Colen et al., 2006; Corra & Kimuna, 2009; Dodoo & Takyi, 2002; Model, 1991, 1995).

Neighborhood Context and Black Immigrant Birth Outcomes. There has been growing attention to neighborhood explanations for Black nativity differences, with investigations of racial residential segregation (Grady & McLafferty, 2007) and ethnic density (Baker & Hellerstedt, 2006; Mason et al., 2010). Segregation was associated with LBW for U.S.-born and Caribbean-born Blacks, but the relationship disappeared for all foreign-born Blacks combined after adjustment for individual maternal factors and national origin (Grady & McLafferty, 2007). There was also an unexpected relationship of neighborhood poverty to LBW among foreign-born Blacks in that foreign-born Blacks had lower rates of LBW in high poverty neighborhoods, higher rates of LBW in low poverty neighborhoods, and an interactive effect of poverty and segregation in medium poverty neighborhoods. This trend was unlike African

Americans where there was an expected, positive gradient in the relationship between neighborhood poverty, segregation, and LBW (Grady & McLafferty, 2007). Fang and colleagues (1999) sought to tease out structural reasons for Black nativity differences by examining LBW by community income strata in New York City. Their study revealed that U.S.-born Blacks had higher LBW than Whites regardless of community income, but foreign-born Blacks in low income communities had even *lower* odds of LBW rates (compared to Whites) than their foreign-born counterparts in middle- and high-income communities.

Ethnic density has been touted as beneficial for promoting immigrant health via facilitating social support and cultural sustenance (inclusive of language, foods, and other culturally-affirming traditions. Presumably such supports would boost psychosocial health, reduce stress, and deter many health risk behaviors such as smoking and alcohol use. However, while ethnic density has contributed to the health advantage for Hispanic and Asian mothers, it does not significantly confer protection against adverse birth outcomes for Black immigrants (Mason et al., 2010).

Lifecourse Perspectives to Understand Black Nativity Health Disparities. The notion that Black immigrants benefit from more favorable lifecourse circumstances than African Americans is noted with varying specificity in nativity and birth outcomes studies. Singh and Yu (1996) sought to clarify the role of nativity independent of SES, and they reported statistically significant adjusted odds ratios of 1.31, 1.61, and 1.33 for PTB, LBW, and IM, respectively, for births to U.S.-born Black mothers in comparison to births to foreign-born Black mothers. Similar to Valanis and Rush (1979), Singh and Yu (1996) speculated on lifecourse SES disadvantage as contributing to the perinatal health disadvantage for U.S.-born Blacks in relation to foreign-born Blacks—although they also added lifecourse discrimination to their conceptualization of disadvantaged status. In a similar vein, Fang et al. (1999) adopted the premise that early life

advantages offset poor circumstances later in life for Black immigrants—an explanation for their counterintuitive findings of *lower* LBW rates for foreign-born Blacks in poor communities than those residing in middle- and higher-income communities, Although Fang et al.'s argument would subscribe to the notion of critical periods (Lu & Halfon, 2003) of reproductive health in a young immigrant's life that can confer protection through adulthood as a U.S. migrant, the researchers did not articulate specific sources of that protection. Liu and Laraque's (2006) examination of infant mortality among U.S.- and foreign born women in NYC emphasized weathering and lifelong exposure to socioeconomic and racial discrimination to explain the relatively *poor* health among U.S. Blacks—compounded by a complex interaction of social, biological, and environmental factors that can elevate risks for adverse birth outcomes—but did not specifically indicate what contributes to *better* health among the foreign-born.

The lifecourse framework emerges as the conceptual lens for Collins, Wu, and David (2002) who studied intergenerational effects among female descendants of U.S.-born and African- and Caribbean-born Black women in Illinois. In contrast to the *increase* in mean birthweight across three generations for U.S.-born Whites and Blacks, third generation descendants of African and Caribbean immigrant women had *lower* mean birthweights and a 40% greater proportion of moderately LBW than their mothers (Collins et al., 2002). This study suggests that the "healthy migrant effect" may not apply as equally to Black immigrants (see also Liu & Laraque, 2006).

Racism and the Perinatal Health of Black Immigrants. Explicit attributions to racism emerge in perinatal health studies of Black immigrants. Lifelong or extended U.S. residence (Collins et al., 2013; Collins et al., 2002; Dominguez et al., 2009; Elo & Culhane, 2010; Elo et al., 2014; Pallotto et al., 2000; Singh & Yu, 1996); institutional racism (Pallotto et al., 2000; Stein et al., 2009); weathering (Liu & Laraque, 2006); and race-related stress (K. D. Rosenberg

et al., 2002) are mentioned as contributing to pregnancy risks and birth outcomes in foreign–born Blacks. While relative exposure to racism is acknowledged as a contributing factor to differential health effects by nativity among African Americans and Black immigrants, the study by Dominguez et al. (2009) is the only one to date to empirically examine measures of racism in foreign-born Blacks in a perinatal heath context.

Dominguez et al. (2009) conceptualized differential exposure to racism over the lifecourse as a crucial factor that would distinguish perinatal health outcomes between U.S.-born (n = 185), Caribbean-born (n = 75), and African-born (n = 30) Black women in the U.S., and they examined differing perceptions of racism between these groups as part of a long-term, prospective birth cohort of pregnant women in Boston. Multiple dimensions of self-reported racism experiences were examined, including personally-experienced racism and group-directed racism over the lifecourse (from childhood (<18) to adulthood), including during the index pregnancy at the time of the study. As expected, U.S.-born Blacks higher reported levels of personal and group racism than foreign-born Blacks, especially for racism experienced in childhood. The percent of U.S.-born, Caribbean-born, and African-born who ever experienced *personal* racism was 79%, 64%, and 30%, respectively. The corresponding percent who ever experienced *group* racism was 90%, 83%, and 50%, and *childhood* racism was 59%, 27%, and 17%. In adjusted models comparing U.S.-born to Caribbean-born women, the relative odds for personal lifetime racism was 2.1 (95% CI: 1.2, 3.8); for personal childhood racism 3.8 (95% CI: 2.1, 7.1); and for group racism 1.9 (95% CI: 0.9, 4.2). The corresponding odds ratios for U.S.-born vs. African-born women were 5.6 (95% CI: 2.5, 12.6) for lifetime racism; 7.0 (95% CI: 2.6, 19.3) for childhood racism; and 9.3 (95% CI: 3.9, 22.0) for group racism. It is notable that the magnitude of experienced racism for Caribbean-born immigrants was closer to U.S.-born women than to African immigrants. Caribbean immigrants did not statistically significantly differ from

U.S.-born women in racism experienced during adulthood, the current pregnancy, or in group racism ever experienced.

Synopsis and Discussion of Nativity and Birth Outcomes. Investigators seeking to explain disparities in birth outcomes among U.S.- and foreign-born Blacks have focused primarily on health behaviors (Cabral et al., 1990; Elo & Culhane, 2010; K. D. Rosenberg et al., 2002); as well as socioeconomic (Howard et al., 2006; Pallotto et al., 2000; Singh & Yu, 1996); ethnic/cultural (Forna et al., 2003); inter-generational (Collins et al., 2002) and neighborhood differences (Baker & Hellerstedt, 2006; Grady & McLafferty, 2007; Mason et al., 2010). Despite the evidence from these studies, one cannot generalize as to the reasons for nativity differences in perinatal health and birth outcomes. Not only is the field still relatively nascent for this purpose, but the review raises a cautionary flag about research methodologies. All foreign-born Blacks are usually collapsed for analysis, although there is evidence that Caribbean-born and African-born Blacks have different infant risk profiles—with African-born Blacks having rates more similar to U.S.-born Whites (David & Collins, 1997; Elo et al., 2014; Urquia, Glazier, et al., 2010). The current trend in studies to disaggregate foreign-born mothers by national origin or ancestry is encouraging. However, many studies use the U.S.-born White population as the reference group (Fang et al., 1999; Friedman et al., 1993; Pallotto et al., 2000; Singh & Yu, 1996; Stein et al., 2009). Inter-racial analyses have amplified differences but deterred our understanding of protective factors specific to health-disadvantaged populations.

Unmeasured factors between socially-disparate racial groups are likely to be substantial which makes interpretation of findings less meaningful than deciphering disparities among women *within* racial groups. Therefore, I consider it more advantageous to examine Black women exclusively than to compare Black and White women and "control" for presumed independent risk factors such as race or socioeconomic status. The latter approach mistakenly

assumes equivalence of covariates across different racial groups. Intra-group analyses can uncover unique patterns of risk and variability within populations (Dominguez et al., 2005; Giscombe & Lobel, 2005). In addition, unlike cross-racial examinations which highlight differences in *negative risk factors* between Blacks and Whites, intra-racial analyses have the advantages of eliciting *protective* factors within and across heterogeneous groups of Blacks that prevent adverse birth outcomes in the face of substantial population-based risks (James, 1993). Examining protective characteristics that go along with foreign status may uncover important clues to the Black foreign-born health advantage.

Perinatal health researchers routinely treat race and ethnicity as distinguishing demographic factors, but rarely consider the social-psychological meanings or consequences of "race" or "ethnicity" for Black immigrants. Aside from commenting on the intrinsic reality or benefit of 'ethnicity' or 'culture' among foreign-born Blacks, more nuanced ethnic conceptualizations are warranted (Branscombe, Schmitt, & Harvey, 1999). For example, the reality of racial stratification in the U.S. makes it helpful to consider immigrant personal and social responses to racism as central to the ethnic identification or adaptation component for Black immigrants (Benson, 2006; Hall & Carter, 2006; Hine-St. Hilaire, 2006). Black immigrants in the U.S. are unique from other immigrants in that their color relegates them to being classified in the lowest stratified racial group. Therefore, the 'ethnic' label assigned to *Black* immigrants is not merely a reflection of culture—but rather, its meaning is closely aligned with relative social position, and it is an indicator for racism. This relative ethno-racial assignment (Ford & Harawa, 2010; Pearson, 2008) can influence health as much or more than attributional or culturally-focused ethnicity. The orientations and cultural strategies employed by immigrants in response to racial/ethnic assignment have health implications in their own right, yet these areas have been virtually ignored in nativity-focused perinatal health research. In a

race-conscious society, any attribution to 'cultural' differences among immigrant and native Blacks would be better informed by explicit examination of beliefs, attitudes, and responses to race and racism. Understanding the ability to cope with racism may be the key to unlocking the enigma of Black nativity differences and the erosion of immigrant health over time.

More in-depth analysis of findings with expanded use of context data may help to discern the underlying reasons for Black nativity differences in birth outcomes, especially in light of the reputed sociopolitical, socioeconomic, and cultural differences within and between U.S.-born and foreign-born Black women. However, most researchers who have looked at birth outcomes by nativity are limited by birth certificate data that provide little insight into more varied and contextual reasons for healthy or unhealthy birth outcomes between immigrant and non-immigrant Black women. The declining health of Black immigrants over time and the wide variation in socioeconomic profiles challenges unqualified theories of migrant selectivity.

A provocative theory has emerged that has conceptualized the Black health advantage in relation to the racial context of the countries of origin. Blacks are predicted to have relatively worse health if they arise from or reside in countries with majority White populations (e.g., European countries or the United States) (Read & Emerson, 2005; Read, Emerson, & Tarlov, 2005). Accordingly, Black African and Caribbean immigrants, who are the racial majority in their home countries, have been theorized to experience more salutary supports, including a more positive racial identity, in contrast to the deleterious psychosocial contexts for Black immigrants and African Americans in the mainland United States.

Read's theory was supported using U.S. National Health Interview Survey data where Black immigrants from Europe were found to have worse self-rated health, hypertension, and activity limitations than Black immigrants from Africa or the West Indies (Read et al., 2005). However, a more recent study (Hamilton & Hummer, 2011), with a much larger, nationally

representative sample of immigrant Blacks, showed no difference in the initial health advantage between African immigrants from majority Black regions and Black immigrants from less concentrated Black areas such as the Caribbean/South America or Europe—and this contrasts with Read's theory. In partial support, however, Hamilton and Hummer (2011) observed that African immigrants sustained their health advantage for over 20 years, whereas Caribbean Black immigrants experienced worsening health with relatively shorter durations in the U.S.

Unlike residential racial segregation of Blacks by race, the neighborhood concentration of Black immigrants by nativity/ethnicity is conceptualized as a *protective* feature—consistent with research on other immigrant groups—and regarded as "ethnic density." Nonetheless, studies have not shown a protective effect of ethnic density on birth outcomes for Black immigrants, even with adjustment for neighborhood poverty and country of origin (Grady & McLafferty, 2007; Mason et al., 2010). In other words, the spatial area density of Black immigrant groups does not produce statistically significant variation in LBW or PTB for Black immigrants. In contrast, Asian and Hispanic immigrants routinely have more positive health outcomes with higher area concentrations of their ethnic group. This difference deserves further exploration. Extended family and community support within ethnic enclaves is an oft-cited reason for immigrant health advantages. But population densities alone cannot uncover the group or neighborhood factors that may actually contribute to presumed immigrant or 'cultural' advantages. Examining community contexts such as political and civic organizations, social support networks, or local economies (i.e., communal cash exchange networks in Caribbean communities) are alternative avenues to highlight ethnic community supports and possible health advantages. Moreover, studies of Black immigrant birth outcomes are hampered by the inability to assess immigrants' length of time in the U.S. – an important marker for acculturation, racism/racial context exposures, and health status.

Examining racism in immigrant Blacks raises some important considerations when exploring perinatal health disparities. Relative lack of lifelong exposure to racial minority status and racism (and hence less exposure to chronic racism-related stress) is a plausible health-protective explanation for foreign-born migrant women which may transfer into healthier birth outcomes (Pallotto et al., 2000; Singh & Yu, 1996). Notwithstanding, it would be incorrect to assume that foreign-born Blacks from the prevalent sending regions of Africa, and particularly the Caribbean, are not exposed to racism prior to U.S. entry. As part of a prospective, longitudinal study of pregnant Black women in Boston (Dominguez et al., 2009), personal racism was experienced by 64% of Caribbean-born women and 40% of African-born women over their lifetime, including 27% and 17%, respectively, who experienced racism during childhood, when some women may not yet have migrated to the U.S. (age at migration was unconfirmed in the study). In addition, internalized racism has been associated with diabetes risk among Blacks in the Virgin Islands (Tull & Chambers, 2001) and Dominica (Butler, Tull, Chambers, & Taylor, 2002). These studies confirm that Blacks who immigrate to the U.S. are not necessarily absent of or immune to racism experiences.

Furthermore, not unlike racism among African Americans, variances in the reporting and impact of racism among Black immigrants are to be expected relative to external and internal factors. *Externally*, institutional racism and racial stratification often dictate an immigrant's social hierarchy in the U.S. Anthropological and sociological research corroborate an externally-imposed Black racial designation for phenotypically-Black migrants to the U.S. regardless of ethnicity, language, socioeconomic status, racial classification in the home countries, or personal preference (Kasinitz, 1992; Waters, 1994). Black immigrants in the U.S. are not likely to escape the 'master' status of Black in daily life or in racially-segregated neighborhoods where Black immigrants often live alongside African Americans (Crowder, 1999). *Internally*, the

psychosocial and health effects of racism would depend on how Blacks (including both U.S.- and foreign-born) internalize a subordinated status and/or resist the racist impositions of society that seek to disparage their individuality or humanity (Benson, 2006; Hine-St. Hilaire, 2006).

Coping mechanisms to resist racial subjugation can impact African American health *positively*: as in positive racial identity (Sellers & Shelton, 2003), ethno-racial identity (Benson, 2006; Painter et al., 2006; Pearson, 2008) and alternative cultural frameworks (James, 1993); or *negatively* as in high effort coping (James, 1994). However, the mechanisms and outcomes of racism resistance become more complex for Black immigrants (Benson, 2006). Black immigrants (i.e., Africans and West Indians) who migrate to the U.S. after the formative childhood period (e.g., after age 10-12) could be said to possess highly resilient buffers to U.S. racism by virtue of implicit socialization experiences as a non-minority in their native countries (Read & Emerson, 2005). On the other hand, inexperience with U.S. racism could be said to put Black immigrants in a uniquely vulnerable position where they are confronted with a starkly unfamiliar context to which they have not yet developed resistant responses (Pearson, 2008). Rather than accepting unthinkingly that Black immigrants have a privileged social or cultural position in the U.S. that protects their general or maternal-infant health, whether Black immigrants are more or less protected from the effects of racism becomes an empirical question that is yet to be resolved in perinatal health literature.

Discussion

Summary. The purpose of a comprehensive scientific review is to summarize the key findings and issues in a particular discipline and offer predictions or guidance for future development of the field. In this regard, the state of knowledge concerning racial (Black/White) and nativity-based (Black foreign-born/Black U.S.-born) disparities in birth outcomes is telling. Most 'alternative' selections among the papers selected for this review tend to highlight social

context as a counter to the prevailing proximal (e.g., biological) focus of many investigations. The lifecourse perspective resounds prominently as a framework within which to conceptualize and examine racial disparities in birth outcomes—emphasizing that sources of racially-disparate perinatal health risks arise from the disproportionate accumulation of social disadvantage and progressive health deterioration over the lifecourse (e.g., due to stress, chronic disease, unhealthy behaviors spawned by inadequate economic and health resources, environmental health risks spawned by systemic structural disadvantages, and biogenetic changes) that threaten maternal health and the maternal-fetal and neonatal environments at critical developmental periods. Causal factors are commonly viewed as operating through multiple complex pathways; however, stress and inflammatory biological processes are the factors acknowledged most frequently. Studies of psychosocial stress as a cause of racial disparities have embraced racism-focused conceptualizations, which can be aptly described as a 'racism-stress' framework. Although epigenetic studies characterize the stressful nature of disadvantaged environments that can influence gene expression, explicit attributions to racism as a source for this stress are rare. The most direct demonstration of interest in racism-related phenomena is with the studies examining either perceived discrimination and birth outcomes or racial segregation and birth outcomes.

Our growing attentiveness to unmasking the enigma of preterm birth in the U.S. (and an expanding urgency to understand what contributes to the persistent racial disparities) has charted a path of inquiry from the social-environmental to the medical, behavioral, psychosocial, and biological realms, with a re-emergence of theories that integrate social and environmental factors and a more enlightened understanding of biochemical and biogenetic pathology not only during pregnancy but also over the lifecourse. Notwithstanding our expanding assortment of risk categories, our knowledge of the primary *causes* for racial disparities in preterm birth is still elusive. Much of this knowledge stagnation could be due to reliance on standard birth certificate

data and epidemiologic approaches which focus on proximal risk factors (social epidemiology is the exception). This review confirms that most investigations into racial disparities in birth outcomes have reified the deficit model or risk factor approach. Despite the burgeoning examination of psychosocial and neighborhood contexts, our context measures remain imperfect and understudied, and there have been few truly integrative conceptualizations and examinations of causality for preterm birth and racial disparities. Hence, there is still limited understanding of the foundational reasons behind persistent racial disparities, and the latest reviews of adverse birth outcomes continue to explore and speculate on the nature of racial disparities (Culhane & Goldenberg, 2011; Hauck et al., 2011; MacDorman, 2011).

Racism-focused Perspectives and Implications. The extent to which racism is discussed as a contributing factor for racial and/or Black inter-ethnic disparities is noteworthy. In perinatal health literature, racism is almost exclusively discussed, theorized, operationalized, and empirically investigated as how Black women perceive and/or experience racial discrimination. Measures of unfair treatment (Krieger & Sidney, 1996; Krieger, Smith, Naishadham, Hartman, & Barbeau, 2005) and 'everyday discrimination' (due to race) (Williams et al., 1997) provide the most frequently used measures for research with individual women in perinatal and public health studies. However, racism is a multidimensional concept (Dominguez, 2011) that includes intrapersonal, interpersonal, and structural dimensions. With the perceived racism construct, an individual must report unfair treatment due to race in order for the exposure to be recorded as a racism-related stressor (e.g., discrimination). But not reporting a stressor as racism-related does not absolve a situation as racist, nor does it lessen the hypothesized association between structural racism and stress. It is also operationally difficult to disentangle the impacts of perceived/latent, reported/unreported, and structural/personally-mediated racism on racism-specific stress responses.

Few studies attempt to conceptualize or explore how women understand, perceive, or experience "racism," in broad strokes, not restricted to specific interpersonal forms. Such a conceptualization, however, requires a historical perspective and a genuine structural analysis of racism and how it is manifested in society (Bonilla-Silva, 1997; Ford & Airhihenbuwa, 2010b). A structural analysis of racism not only identifies "institutional" racism in the manner of residential segregation or healthcare inequality but explores how the *ideology* of racism has been incorporated in laws and policies, reified in social structures, and has pervaded the production of scientific knowledge itself which influences how we address and understand the problem of racial disparities in adverse birth outcomes and develop solutions. A structural analysis reaches beyond how individual women perceive racism and considers how the system impacts African American men, communities, and culture which shapes notable "risk" factors such as fertility timing, unmarried status, and social and economic conditions that determine individual and population mental and physical health.

Notwithstanding the research on racial residential segregation and socioeconomic status over the lifecourse (Colen et al., 2006), and intergenerationally (Collins, David, Rankin, & Desireddi, 2009; H. W. Foster et al., 2000), a number of untapped operational concepts for structural racism remain. These include differential exposures to, and differential quality of, educational *systems*; healthcare and healthcare financing *systems*; other community contexts (e.g., environmental racism); acquisition of *wealth*; structural economic policies; industry and employment policies and practices; state-sanctioned disruption of families and communities (e.g., welfare, penal, and immigration policies); and other conditions of racial inequality that are yet to be examined within and across "races" and communities—both contemporaneously and in varying degrees throughout the lifecourse. These structured conditions may help to explain

perinatal health disparities in lieu of individual perceptions or reporting of racism, and they offer alternative constructs to broaden the 'social context' for perinatal health research.

I surmise that our collective failure to more fully articulate and examine *why* racism is a factor in the persistent perinatal health disparities and *how* racism operates as a determinative antecedent risk for Black populations in the U.S. has compromised our understanding of how racism impacts women's and infants' health. In addition to acknowledging racism's fundamental role in perinatal health disparities (David & Collins, 1991), conceptual models are critically important to highlight specific constructs that can guide research activity regarding associations and mechanisms. For example, Hogue and Bremner (2005) conceptualized the interaction of racism and stress within a stress model that mirrored the epidemiologic triad of agent-host-environment. Alio et al. (2010) modeled racism within an ecological framework, whereby racism was conceptualized to influence many different levels, from the individual to the societal. And Dominguez (2011) highlighted the multidimensional nature of racism which featured three domains (institutional, interpersonal, and internalized) to be considered for perinatal health research. More studies are needed that model or test specific pathways and mechanisms through which racism can cause racial disparities.

Public health and perinatal health researchers are less likely to view contextual factors, including racism, within the purview of professional intervention. Nevertheless, addressing fundamental causes, or the 'cause-of-the cause' (Rose, 1985), has been articulated as necessary for reducing health inequalities (Link & Phelan, 1995), including perinatal disparities, specifically (David & Collins, 1991; Geronimus, 1987). Notwithstanding, the 2007 IOM report on preterm birth (Behrman & Butler, 2007) identified socioeconomic status, maternal behaviors, stress, infections, and racial differences in genetic susceptibility as likely explanations for racial disparities in preterm birth. The report included a review of racism and birth outcomes which

concluded that racism may be a potent lifetime stressor for African American women that may explain racial disparities. However, while the report carefully emphasized that "preterm birth is a complex cluster of problems with a set of overlapping factors of influence" (Behrman & Butler, 2007, p. 2), and it acknowledged both "upstream" and "downstream" factors within a multifaceted web of causation, the report chose to illustrate only the downstream factors which were all in the biological realm, highlighting numerous chemical processes that impacted the placenta and that stimulated preterm labor (see Figure 6-2, p. 178). In contrast, none of the proposed upstream factors were specified and no schematic of these factors was included. The primacy of individual factors still shapes current discussions of perinatal health disparities.

Racial Disparities and Genetically-focused Research…A Slippery Slope. With the advent of genomics and genetic studies, *subcellular* investigations (i.e., genetic polymorphisms) have been introduced to investigate Black-White disparities in low birthweight (LBW) and preterm birth (PTB). These studies have garnered the attention of numerous critical and precautionary public health commentaries (David & Collins, 2007; Dressler, Oths, & Gravlee, 2005; M. W. Foster, 2009; Gravlee, 2009; Krieger, 2005; Reverby, 2010). Science confirms that no genes exclusively or consistently map onto being of 'Black' or 'White' race. However, our ideological predispositions serve to disrupt (or fuel) our observational and empirical logic such that we knowingly or unknowingly "see" or perceive Black and White "races" in biogenetic terms. The predominance of biomedical and public health research that describe differences by "race," in the absence of a clear contextual framework as to how contextual structures shape one's "race," is suggestive of a biogenetic understanding of race even in the absence of explicit adoption of this viewpoint (Krieger, 1987, 2005).

Whether explicit or implicit, genetic perspectives have been an undercurrent for rationalizing persistently adverse birth outcomes by race, and these perspectives are gaining

legitimacy with the growth of biogenetic research publications in this field. It is no benign development that genetically-based theory has encroached upon the discipline of perinatal health which prioritizes individual-level explanations for adverse birth outcomes. The empirical advance of genetic studies is taking place amidst a relative absence of rigorous, socially-based theories and research in public health that could explain racial disparities in birth outcomes (Krieger, 2008; Muntaner, 1999). Some would view epigenetics (i.e., the study of how genes are differentially expressed, although there is no change in the underlying genotype or gene sequence) as the ideal integration of social and biological theories. Indeed, epigenetics could be the "new frontier" to understanding persistent racial disparities (Burris & Collins, 2010). But until there are more refined articulations and measurement of social-structural processes, including racism, added to the body of perinatal health research and until dualistic sociological and biological perspectives are rectified with truly integrative theories, the *genetic* component of epigenetic explanations may eclipse the *epi* (environmental) component, and, moreover, environment may be relegated to the physical environment at the expense of continued examinations of how the broader social structure impinges on pathological gene expressions.

Limitations and Contributions. This integrative conceptual and systematic literature review was limited to publications referenced in the PubMed database. Because of the focus on racism and on immigrant populations, searches of CINAHL, Sociological Abstracts, PsychLit, or other databases could have produced articles from the nursing, psychological, sociological, anthropological, and international health fields relevant to the issues of racial and ethnic health disparities discussed in this paper. Although this author embraces interdisciplinary perspectives, the database search was restricted to PubMed to maximize review efficiency and to target findings for a public health audience.

Also, this review study did not utilize additional reviewers to replicate the search strategies or corroborate the selection of articles, and hence the selection process could not be validated with inter-rater reliability. In addition, there was no standardized evaluation of article quality using uniform rating systems or guides (Armstrong & Waters, 2007; Paradies et al., 2013). These limitations were not felt to detract significantly from the focus of the review which was to illuminate the major theories and conceptual approaches that guide researchers' examination of perinatal health disparities by race. The review's major aims are not suited for the conventional content or format of a meta-analytic review, nor was it felt that the qualitative assessment of researchers' perspectives would benefit from a formal content analysis. For future publication opportunities, the author plans to engage collaborating reviewers to justify search strategies and article selection.

Overall, the current state of the science in perinatal health research demonstrates a need for more critical theory to facilitate our knowledge of what causes persistent adverse birth outcomes by race (Ford & Airhihenbuwa, 2010a; Krieger, 1994; Muntaner, 1999). I contend that few truly integrative models exist to explain racial disparities in birth outcomes, in large part because current conceptualizations lack a sophisticated understanding of race and racism (Ford & Airhihenbuwa, 2010a). Accordingly, I propose more critical development of racism-focused theories to deconstruct the enigma of *race* and *race x nativity* disparities in perinatal health. While it has been customary to examine perinatal health disparities by comparing Black and White women, a more in-depth understanding of disparities calls for more studies *within* Black populations. Such investigations can provide more nuanced examinations of risk and protective factors utilized by Black women against a shared backdrop of racism. The plethora of deficit/risk orientations in perinatal health research subsumes discussions of health-protective individual and community factors, including empowering cultural and community traditions among African

Americans and Black Caribbean and African immigrants. Examining protective mechanisms that may counter the damaging effects of racism may yield insight into the enigmas of Black/White *and* U.S.-born Black/foreign-born Black differentials, and in so doing, bridge these two largely disconnected streams of research.

In conclusion, the foregoing conceptual and systematic review suggests that racism can be a viable framework to understand *both* inter-racial (Black/White) *and* inter-ethnic (U.S.-born Black/foreign-born Black) disparities in birth outcomes that highlights Black women's *responses* to racism as potentially *protective* factors that can moderate the negative effects of racism and reduce perinatal risks. Differential experiences and coping responses to racism-related stress may contribute significantly to the nativity differential in Black maternal and infant health in the U.S. and emerge as a critical pathway for understanding the enigma of racial disparities in birth outcomes.

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CHAPTER 3

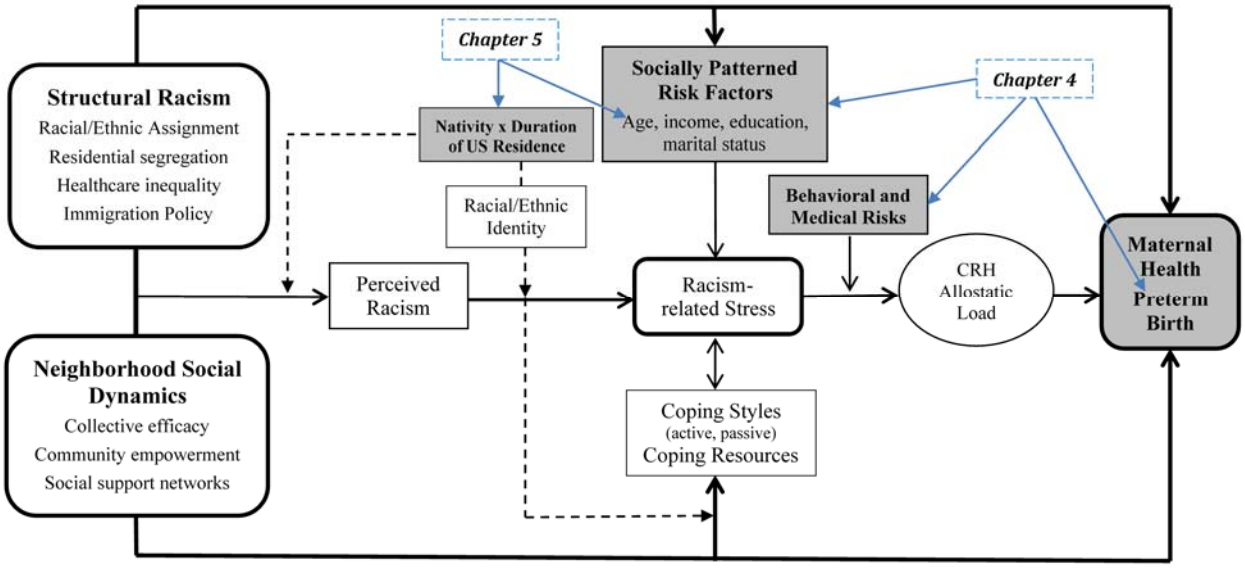
A Conceptual Model of Racism, Nativity, and Birth Outcomes in Black Women

Building on previous conceptualizations and findings, this chapter delineates a model that situates racism as a fundamental cause of adverse birth outcomes for U.S.-based Blacks, both native- and foreign-born. Figure 3 illustrates how structural forces (i.e., structural racism, neighborhood social dynamics), psychosocial orientations (i.e., racial identity, coping styles and resources), and physiologic processes (i.e., stress, corticotrophin releasing hormone (CRH), allostatic load) through which Black women evaluate and cope with racism may explain some of the variance in racial and ethnic disparities in adverse birth outcomes. The model outlines pathways for structural, interpersonal, and internalized racism, depicts psychosocial and biological racism-associated stress responses, and highlights protective individual and community-based moderators of racism-associated stress. Solid lines indicate associations for which evidence exists in the literature. Dashed lines indicate new hypothesized relationships. Shaded boxes will be empirically examined in this dissertation.

My model depicts how racism may be a fundamental cause of observed race-related disparities in maternal health and preterm birth. Intermediary factors in the psychosocial realm are conceptualized to moderate or mediate the effects of racism on birth outcomes. Importantly, the model postulates that the relative contribution of racism and racism-related stress to adverse birth outcomes may differ by national origin. Therefore, differential experiences and coping responses to racism-related stress may contribute significantly to the nativity differential in Black maternal and infant health in the U.S. This chapter includes a more detailed explanation of

structural and psychosocial domains that were not covered as part of the review in Chapter 2 because the associated constructs are rarely discussed in perinatal health research as explanatory factors for racial/ethnic disparities, hence related research would not appear among the articles that were selected based on the review criteria.

Figure 3. Model of Racism-Related Factors and Pathways to Adverse Birth Outcomes



Structural racism and *neighborhood social dynamics* are assumed part of the background context that influences maternal-child health; these factors are unmeasured and untested in the present dissertation study. Also, perceptions (*perceived racism*), moderators (*racial/ethnic identity*), effect modifiers (*coping styles*), and biological indicators (*corticotropin-releasing hormone (CRH), allostatic load*) of racism-related stress could not be empirically tested in the present study with the focus populations. *Sociodemographic risk factors* are conceptualized to be influenced by racism and hence they are characterized as socially patterned characteristics rather than static control measures. *Behavioral* (i.e., smoking, prenatal care) and *medical risks* (chronic health or medical conditions before or during pregnancy) could also be influenced by racism-

related stress or other factors, which, in turn, can contribute to differential risks of preterm birth in Black Caribbean-born immigrants and U.S.-born women.

In Chapter 4, I quantified the degree to which these demographic and medically-related factors predicted the outcome of preterm birth between foreign-born and U.S. born Black women and to what extent these traditional risk factors could explain the preterm birth advantage for foreign-born women. In Chapter 5, I conceptualized differential increased risks of preterm birth with *maternal age* as a consequence of social disadvantage, including racism, using the concept of "weathering" (socially patterned aging and reproductive health decline). Exposure to non-salutary social contexts is central to the concept of weathering. Hence, in Chapter 5, I also examined *duration of U.S. residence* for immigrants in association with risks of preterm birth, although I could not test it as a moderator of social-psychological processes as depicted in the model.

The research evidence for most of the model constructs was summarized in Chapter 2, with respect to how perinatal health researchers conceptualize and examine purported causes of racial and ethnic disparities in adverse birth outcomes. I reiterate some of this information in the present chapter, with an emphasis on the shaded boxes that were empirically examined in Chapters 4 and 5 of this dissertation. The following summary of model constructs reports evidence from the literature that supports the established pathways. Where no research exists, I speculate about the hypothesized pathways that lead from the structural components of the model through the processes of perceived racism, racism-related stress and coping, and stress-related physiology which all culminate to shape differential risks of maternal-child health.

Structural Racism. Structural racism refers to macrosocial or institutional factors that result in systemic racial group inequities (Bonilla-Silva, 1997; Gee & Ford, 2011; Jones, 2000). The mechanisms of structural racism are interactive and self-reinforcing, and they can be

sustained without individual actions or intent (Powell, 2007). I include racial/ethnic assignment, residential segregation, healthcare inequality, and immigration policy as indicators of structural racism that are particularly relevant to racial/ethnic and nativity-based disparities impacting Black populations.

Racial/ethnic assignment. Racial/ethnic assignment is itself a form of racism which involves "the practice of utilizing sociopolitical processes and mechanisms to attribute undesirable characteristics to groups with different origins" (Pearson, 2008, p. 37). The racialized context of U.S. society has important psychological and health consequences for African Americans and Black immigrants (Omi & Winant, 2014). Specifically, the marginalized position of Blacks in the U.S. fosters low social status and low self-regard that contributes to racism-related stress. Foreign-born Blacks are particularly influenced by this racial stratification and marginalization which introduces unique complexities for immigrants forced to contend with involuntary and negative racial attributions which are oppositional to their normative cultural or ethnic orientations (Benson, 2006; Ford & Harawa, 2010; Pearson, 2008). My model suggests that this process of racialization drives the perception of racism which contributes to racism-related stress.

Residential segregation. Residential segregation refers to the composition and spatial distribution of racial groups within metropolitan statistical areas and census tracts (Massey & Denton, 1988). Racially-segregated neighborhoods may have limited access to quality healthcare facilities for women and infants (Fossett, Perloff, Peterson, & Kletke, 1990; Haas et al., 2004; Mayberry, Mili, & Ofili, 2000) or have poor housing stock with safety risks such as lead exposure, fire hazards, and unsafe infant sleeping conditions that increase harm to infants (Hauck, Tanabe, & Moon, 2011). Segregated, low-income neighborhoods are often characterized as 'food deserts' with lack of groceries that stock iron-, calcium-, and folate-rich foods for

healthy pregnancies (Lane et al., 2008). These neighborhood conditions can influence birth outcomes through material hardships which in turn produce mental, behavioral, and physical health consequences that affect pregnancy and birth.

There is evidence for the impact of racial residential segregation on birth outcomes. Segregation was found to be an independent predictor of Black infant mortality in several major cities (Polednak, 1991, 1996). After controlling for neighborhood poverty and individual-level risk factors, Grady (2006) found that greater levels of segregation predicted lower birthweights in New York City. Also, hypersegregation has been associated with higher rates of preterm birth in Black women and larger Black-White disparities relative to less racially-segregated areas (Osypuk & Acevedo-Garcia, 2008).

However, segregation does not always negatively impact birth outcomes. Roberts (1997) found that African American mothers who lived in neighborhoods with high Black racial segregation were less likely to have low birthweight births than their counterparts in less segregated neighborhoods—controlling for individual factors such as socioeconomic status. Papacek et al. (2002) observed *lower* postneonatal mortality in neighborhoods considered at risk in terms of unemployment, homicide, median income, and lead poisoning. And African American women living in non-minority neighborhoods had *increased* risks of LBW and PTB compared to their counterparts in living in predominantly Black census tracts (Pickett, Collins, Masi, & Wilkinson, 2005).

The residential segregation of immigrants is often construed as "ethnic density," and researchers have studied this phenomenon as a potential explanation for the more favorable birth outcomes for Black immigrants relative to African Americans. Using a spatial measure of ethnic density, non-Hispanic Blacks had the highest risk of preterm birth in high vs low density areas among seven ethnic groups (Mason et al., 2011). However, in models adjusted for national origin

and individual factors, segregation was not associated with LBW among Black immigrants (Grady & McLafferty, 2007). In another study, African immigrants had elevated preterm birth risk at high levels of ethnic density, but no ethnic density effect was observed for Black Caribbean women (Mason, Kaufman, Emch, Hogan, & Savitz, 2010). The neighborhood environment had no effect on birthweight among recent immigrants to Canada (Urquia et al., 2009).

A reason for the inconsistency of segregation effects on birth outcomes could be that unmeasured factors such as community cohesion or social support networks can outweigh some of the negative factors of economic disadvantage found in segregated Black communities. Accordingly, my model includes the concept of "neighborhood social dynamics" to represent positive contextual features that may contribute to lower maternal-infant health risks.

Healthcare inequality. Differential access to healthcare can be one manifestation of structural racism. Structural inequality can foster segregated healthcare environments that lack the resources to assure quality medical facilities and optimal healthcare for women and infants (Griffith, Childs, Eng, & Jeffries, 2007; Haas et al., 2004; Mayberry et al., 2000). Fossett and colleagues (1990) describe how low reimbursement rates and restricted access to provider staff by area of residence can negatively impact Medicaid clients receiving perinatal services. High-risk obstetric and neonatal offices are often located far from the communities of women most in need of such services and without adequate transportation or economic resources to navigate long distances for routine healthcare. Moreover, service environments that are not representative of the clients and neighborhoods served can heighten professional biases, communication barriers, mistrust, and stressful encounters for women.

Exploration of healthcare access differences in relation to *race x nativity* disparities in birth outcomes among Black women is an underexplored research area. It is unknown the extent

to which healthcare service factors can independently explain perinatal disparities by race or nativity. One California study found that the proximity of community healthcare and social services had no effect on SGA births—even after adjusting for maternal prenatal care utilization and additional maternal and community factors (Heck, Schoendorf, & Chavez, 2002). An analysis of spatial healthcare access for immigrant groups in NYC indicated a higher density of prenatal clinics for Caribbean immigrants than for other immigrant groups (McLafferty & Grady, 2005), suggesting few barriers with proximity to health care services. Still, more studies are warranted, and an exploration of perceived access to and quality of prenatal healthcare services among U.S.-born and immigrant Black women will be considered for future research.

Immigration policy. Immigration policy represents a form of structural racism via racialized policies that structure the number and descriptions (e.g., preferred sending countries, occupations, social classes, genders, neighborhood residence, phenotype-language) of Black immigrants *vis à vis* other immigrant groups (Gee & Ford, 2011; Viruell-Fuentes, Miranda, & Abdulrahim, 2012). While the 1965 immigration reform opened the doors to millions of immigrants from Africa and the Caribbean (and other regions), the Immigration Act of 1990 has in some ways become more restrictive and discriminatory by prioritizing visas to attract highly skilled professional and technical workers and banning open visas for migrants from Jamaica, Haiti, and the Dominican Republic (all with majority Black migrants) among other areas (Kent, 2007; Kim, 2007). The impact of immigration and healthcare policies on health inequities experienced by native and immigrant Black women will be explored in more detail.

Neighborhood Social Dynamics. Neighborhood social context may predict birth outcomes both directly and mediated by its impact on individuals' stress and coping styles and resources through subsequent race-related paths. While ethnic density has emerged as a popular construct to examine the hypothesized health-protective effects of immigrant enclaves (Becares,

Nazroo, & Stafford, 2009; Mason et al., 2010; Pickett, Shaw, Atkin, Kiernan, & Wilkinson, 2009; Shaw, Pickett, & Wilkinson, 2010; Stafford, Becares, & Nazroo, 2009), my model considers added measures such as social support networks (e.g., density of civic associations) and community empowerment (e.g., community action around infant mortality) that may reflect salutary neighborhood contexts. Collective efficacy, community empowerment, and community support networks represent meaningful and significant social processes in Black communities, but they are rarely considered in predicting adverse birth outcomes, and little is known about how they vary among Black ethnic populations by nativity.

Collective efficacy. "Collective efficacy" is defined as informal social control and social cohesion/trust in neighborhoods (Sampson, Morenoff, & Gannon-Rowley, 2002). Social control is the notion that neighbors will intervene (e.g., calling police, community mobilization) to address problems such as crime and deviance or littering, etc. in one's neighborhood. Social cohesion and trust comes close to having significant social ties within neighborhoods—a sense of connectedness. Collective efficacy may enhance sense of well-being and increase coping resources in neighborhoods. One study found that 'neighborhood social ties' was associated with less maladaptive stress responses (Seeman & McEwen, 1996). The stress mechanisms are significant for hypothesized pathway between structural factors, stress/coping, and birth outcomes. To my knowledge, collective efficacy has not yet been studied relative to birth outcomes.

Community empowerment. The relevance of community empowerment to my research lies in the possibility that empowered communities are more likely to challenge and address neighborhood problems, and they promote adaptive coping styles to individual and community stressors. Community empowerment is "…a social action process that promotes participation of people, organizations, and communities toward the goals of increased individual and community

control, political efficacy, improved quality of life, and social justice…." (Parker et al., 2001). Community empowerment is more of a process than an outcome, and therefore it is difficult to measure (Zimmerman, 2000). However, community empowerment can take many forms. The Harlem Birthright Project mobilized community coalitions to conduct community-based participatory research to improve the community context for maternal-child health. (Mullings et al., 2001). O'Campo and colleagues (1997) measured community empowerment as the number of active community based groups in a neighborhood. Their study did not find an association with this measure of community empowerment and LBW. However, replicability of this concept is desired for future studies, and alternative measures of empowerment such as political activity may be considered. The level of political disempowerment in African American communities has been negatively associated with Black infant mortality rates (LaVeist, 1993).

Social support networks. A previous review and critique of neighborhood studies called for more attention to the collective features of neighborhoods, including networks of community support (Macintyre, Ellaway, & Cummins, 2002). Social networks appear particularly relevant to Black perceptions of community disorganization (Cutrona, Russell, Hessling, Brown, & Murry, 2000). Buka and colleagues (2003) examined racial differences in LBW according to a neighborhood-level measure of social support that combined measures of social cohesion, trust, and reciprocated exchanges. After controlling for individual, behavioral, and socioeconomic risk factors, neighborhood social support was associated with low birthweight births among White mothers, but had no significant relationship to low birthweight among Black mothers.

Strong social networks has been highlighted as a factor that may contribute to the immigrant health advantage (Sherraden & Barrera, 1996). However, most commentaries and research in this area are related to Mexican American families and communities, and no perinatal

health research to my knowledge has examined this factor with non-Hispanic Black immigrants. More studies along these lines are needed.

Nativity and Duration of U.S. Residence. A major limitation with perinatal health studies of immigrants using standard health databases is the inability to ascertain immigrants' length of time in the U.S. Years of residence in the U.S. can be an important moderator of social and health-related exposures and risks (Cho, Frisbie, Hummer, & Rogers, 2004). In my model, it is a critical construct for racism exposure and ethnic identity formation of Black immigrants.

Independently designed population health studies have benefitted from collection of immigrant length of residence data in perinatal health research. Landale observed a 4% annual increase in infant mortality risks for Puerto Rican migrants to the U.S. mainland. Working with Canadian populations, Urquia and colleagues observed a 14% increase in preterm birth for immigrants with each five years of Canadian residence (Urquia, Frank, Moineddin, & Glazier, 2010). There was no reported variation by race or national origin with duration of residence. However, Caribbean immigrants had the highest overall odds of preterm birth relative to immigrants from industrialized countries. In a later study, non-European immigrants in Canada were more likely than European immigrants to experience worsening of maternal health and preterm birth over time (Urquia, O'Campo, & Heaman, 2012).

Nativity itself is an important modifier, in that African and Black Caribbean immigrants have heterogeneous economic (Corra & Kimuna, 2009) and health outcomes (Hamilton & Hummer, 2011) in the U.S., with West Indian Caribbean immigrants faring worse overall.

Psychosocial Factors. Examining psychosocial factors in health outcomes is familiar terrain in public health. By explicitly examining responses to racism-related stress, my proposed research may elicit revelations about coping and resilience against racism in Black women of childbearing age. Integrating a psychosocial perspective acknowledges space for individual and

community agency to counter the effects of racism. In a seminal psychosocial critique, James (1993) implored researchers to shift their investigative gaze to the intrinsic *cultural* environments that can be health-promoting for minority communities. My theoretical framework embraces this cultural paradigm and examines indigenous socio-cultural strengths and resources in Black communities.

Racial Identity. I conceptualize racial identity as a factor that buttresses coping responses in Black women and potentially reduces susceptibility to the stress engendered by structural and interpersonal racism. Racial identity influences the appraisal of racism-related stressors and the coping responses employed to deal with those stressors. It is postulated that the frequency and types of exposure to racial discrimination will not significantly differ by nativity, but cross-cultural differences in racial identity and coping styles may generate responses to racism that are either protective or pernicious. In this manner, racial identity may partially explain inter-ethnic differences in birth outcomes. The hypothesized relationships are in accordance with current social psychological theories in racial identity (Helms, 1990).

Racial identity is defined as "the significance and qualitative meaning that individuals attribute to their membership within the Black racial group within their self-concepts" (R. M. Sellers, Smith, Shelton, Rowley, & Chavous, p. 23). The Multidimensional Inventory of Black Identity (MIBI) (R.M. Sellers, Rowley, Chavous, Shelton, & Smith, 1997) operationalizes three dimensions of racial identity. Centrality refers to the degree to which identification with being Black is central to ones conceptualization of self. Ideology describes one's philosophical orientations about how they feel African Americans should live and interact with society—as nationalist, oppressed, assimilationist, or humanist. Regard refers to the extent to which a person feels positively or negatively about one's race: private regard captures personal judgments and public regard reflects how individuals think the broader society sees their race. Salience is an

added dimension that represents how important "race" is in specific contexts. Conditional on salience, the stable dimensions are more likely to correlate with racial identity-related behaviors or self-esteem. Salience and centrality capture the significance of race to a person, whereas ideology and regard capture the meaning of race to an individual. Racial identity has been associated with lower levels of psychological distress (R. M. Sellers, Caldwell, Schmeelk-Cone, & Zimmerman, 2003; R. M. Sellers & Shelton, 2003). Consistent among these studies is that individuals with high race centrality had more effective coping styles. Racial identity may offer new insights on racism-related stress, coping, and reproductive health outcomes in Black women.

My model implies that African Americans and Caribbean Blacks have qualitatively different meanings and perceptions of racial identity, which in turn, is hypothesized to moderate the link between perceived racism and stress. Racial identity is also believed to influence coping styles. In accordance with Sellers' model (R.M. Sellers et al., 1997), I hypothesize that Caribbean Blacks may ascribe more favorable public and private evaluations of their race. Furthermore, perceived racial oppression is hypothesized to be less central to racial identity formation in Black Caribbean immigrants than in African Americans. These distinctions of racial identity are predicted to generate more favorable health profiles for Caribbean Blacks in response to racism-related stressors. However, it is acknowledged that that racial/ethnic identity among Caribbean Blacks may be more nuanced than can be adequately captured by Sellers' model (Benson, 2006; Butterfield, 2004; Foner, 1998; Joseph, Watson, Wang, Case, & Hunter, 2013; Portes & Zhou, 1993; Waters, 1994). Examining racial identity should simultaneously consider the influence of immigrant and generation status, social class, gender roles or identities, and neighborhood characteristics. No known studies have examined racial identity in relation to

racism-related stress or racism-related coping responses in pregnant and childbearing women, particularly with the foreign-born Black population.

Perceived racism. Structural racism is often not specifically acknowledged when individuals are asked about racism experiences. For this reason, perinatal health studies have operationalized racism as 'perceived racism' (McNeilly et al., 1996), 'unfair treatment' (Krieger, 1990), or 'everyday discrimination' (Williams, Yu, Jackson, & Anderson, 1997). Unfair treatment and everyday discrimination are measured as the frequency of interpersonal acts of unfair treatment (due to one's race) in public settings.

A fair number of studies provide empirical support for the influence of perceived racism on adverse birth outcomes. Among 13 published studies that have investigated perceived racism and birth outcomes, five have reported statistically significant adjusted odds for LBW or PTB, with midpoint estimates ranging from 1.3 to 2.6 (Collins, David, Handler, Wall, & Andes, 2004; Dole et al., 2004; Mustillo et al., 2004; Rankin, David, & Collins, 2011; Rosenberg, Palmer, Wise, Horton, & Corwin, 2002). Active coping strategies have been shown to attenuate the odds of an adverse birth outcome (Rankin et al., 2011). However, no known study has empirically examined mediating factors between perceived racism and adverse birth outcomes.

Racism-related stress. Racism creates a chronic and unique type of stress for Black women that can impact birth outcomes through a host of psychological, behavioral, and physiological mechanisms (Dominguez, 2011; Giscombe & Lobel, 2005; Hogue & Bremner, 2005). Racism is an antecedent for stress-induced physiology which precipitates adverse birth outcomes. Racial discrimination has also been associated with unhealthy coping behaviors such as smoking (Landrine & Klonoff, 1996), and is positively associated with blood pressure in Black immigrants (Ryan, Gee, & Laflamme, 2006). These factors pose risks for unhealthy

pregnancies. Measures of chronic stress, especially self-reported measures of racism, have had the most significant association with birth outcomes in Blacks (Giscombe & Lobel, 2005).

Coping. The working model for this research borrows from a transactional stress-coping framework (Lazarus & Folkman, 1984) which holds that the impact of a stressor is mediated by a person's evaluation of the threat posed by that stressor and perceived control over the stressor. The model highlights the primacy of active vs. passive coping styles and includes sociocultural attributes such as racial identity and neighborhood social dynamics that can influence coping styles. Active or expressive coping involves talking with others or doing something about racism experiences; passive coping includes keeping quiet/suppressing emotions or accepting discriminatory experiences. Across several studies, active coping styles appear more effective than passive coping styles to buffer racism-related stress, and active coping is associated with better mental and physical health outcomes. Active coping against stress has also been associated with a lower likelihood of preterm birth (Dole et al., 2004). In contrast, internalization and denial/avoidance can be maladaptive (Harrell, 2000).

John Henryism is a distinct style of active, high-effort coping in African Americans – exhibited by a dogged determination to fight against chronic stressors (James, 1994). African Americans with high John Henryism and low-incomes have demonstrated worse health outcomes than African Americans with high John Henryism and higher income levels (James, 1994). John Henryism has been associated with lower cortisol levels in young adults (Schmeelk-Cone, Zimmerman, & Abelson, 2003) and decreased blood pressure reactivity in women reporting low levels of acute racism (Clark & Adams, 2004).

Socially Patterned Risk Factors. My empirical studies (Chapter 4 and Chapter 5) examine socio-demographic factors in detail. Demographic factors such as age(ing), income, education, and marital status are conceptualized through the lens of racism, whereby the widely

disparate outcomes by race are a product of structured social conditions and systemic racism. I argue, with supportive evidence, that the meaning or measurement ascribed to these factors operates differently by race or nativity. Hence, these constructs should not be readily construed as static risk factors that can be statistically 'controlled' with presumed equivalent effects in inter-racial or inter-ethnic health studies (Blumenshine, Egerter, Barclay, Cubbin, & Braveman, 2010; Geronimus, 1992; Kaufman, Cooper, & McGee, 1997).

Age. Most research studies on infant mortality treat maternal age as a developmental factor to be controlled for. My model treats age as a socially patterned construct for Black women. Black women have dramatically increased risks for adverse birth outcomes as they age (Hogue & Bremner, 2005). Disparities in age-related risks are demonstrated as early as the 20s and 30s for Black women—in contrast to White women for whom these ages represent more optimal risk profiles for childbearing. Geronimus (1992) calls this phenomenon weathering which is theorized to be the physical consequence of cumulative disadvantage due to structural inequalities that take a toll on Black women's reproductive health.

Income, Education, (and Employment). Socioeconomic status (SES) does not appear to confer equal benefits for improving birth-related risks for Black and White women. For example, college-educated Black women have significantly higher rates of low birthweight and infant mortality than White women at similar socioeconomic levels (McGrady, Sung, Rowley, & Hogue, 1992; Schoendorf, Hogue, Kleinman, & Rowley, 1992). Social patterning seems evident because Black college graduates—known to have disparate birth outcomes compared to Whites at similar levels of income and education, are also more likely to be single, have less annual incomes with respect to occupation, have parents who are less educated (suggesting generational disadvantages), and reside in segregated communities of relative disadvantage (Giscombe & Lobel, 2005). Also, traditional socioeconomic (SES) measures (i.e., income, education,

employment) do not capture differences such as wealth inequalities, and therefore they should not be presumed as equivalent across groups (Williams, 2002).

Employment was not empirically examined in this dissertation, but it should be considered as an important component of the unmeasured background context that may significantly influence socioeconomic context, stress, and potential health risks that may differ between native and immigrant Black women. Employment can be a source of stress to women and shape conditions that are harmful to pregnancies; therefore, female and immigrant work environments may be an important consideration for the examination of race and nativity-status disparities. Understanding unique occupational health risks and stressors may help elucidate why professional Black women have adverse birth outcomes that are not dramatically different from unemployed or working class Black women.

Future planned research will consider the influence of "gendered employment" on reproductive health and how racial and ethnic disparities in working conditions are socially-patterned and representative of the "triple oppression" of gender, race, and class. A disproportionate number of Black immigrant women work as domestics or a nursing- and home-health aids that can cause significant strain and pregnancy-related stress (Rollins, 1985). Gendered employment can also shape reproductive risks for African American women who are subject to stringent criteria of welfare reform employment programs that may impair health and family caretaking roles. An interesting empirical question concerns how labor patterns and employment conditions affect the maternal-infant health differential for Black immigrants and native African American women. Although if one accepts that Black female immigrant workers are subject to restricted economic opportunities and distinctly oppressive work environments, then the impact of such conditions on health would not be in the expected direction given the observed immigrant health advantage. Research is needed to disentangle such inconsistencies.

Marital Status. Seventy-two percent of non-Hispanic Black women are unmarried at the time of delivery compared to 29% of non-Hispanic Whites (Martin, Hamilton, Osterman, Curtin, & Mathews, 2015). Unmarried status is a frequently-acknowledged sociodemographic 'risk' factor for adverse birth outcomes. However, marital status is not consistently associated with infant mortality after adjusting for other factors, and being married does not appear to reduce the risk of adverse birth outcomes among college-educated Black women as it does among Whites (Bennett, Braveman, Egerter, & Kiely, 1994). Caribbean Black women have higher marital rates than African American women, but no known study has identified this characteristic as a key factor for their infant health advantage.

Few investigations describe structural and culturally-relevant implications of marital status for Black women or consider instrumental and relational support in lieu of marriage. The sex ratio in Black communities and the unemployment and underemployment of Black males are structural features that affect marital rates (Raley, 1996). Black male imprisonment and unemployment can take a toll on Black women who are less likely to be married or partnered in economically stable relationships, and this can negatively influence pregnancy and childrearing options and supports. For Black cohabitors, it has been found that socioeconomic disadvantage during childhood reduces the odds of marriage (Manning & Smock, 1995). The relative advantages (or disadvantages) of marriage are conferred differently across racial groups due to social structural forces, suggesting that marital status is not to be uncritically viewed as a sociodemographic risk or protective factor among all women.

Behavioral and Medical Risks. In adjusted models, health risk behaviors such as nutrition, smoking, or substance use, have not been shown to be a major determinant of racial disparities in adverse birth outcomes (Berg, Wilcox, & d'Almada, 2001; Ebrahim, Floyd, Merritt, Decoufle, & Holtzman, 2000; Finch, Frank, & Hummer, 2000; Goldenberg et al., 1996; Phares et

al., 2004; Tong, Jones, Dietz, D'Angelo, & Bombard, 2009). However, they are included in the model to examine variations in health risk behaviors. The presence of chronic diseases can also impact allostatic load measures. With respect to Caribbean immigrant populations, there are noted disparities in Type 2 diabetes that have been observed across several studies, but not discussed as a consequence of influencing the relative disparities between Caribbean, African, and other immigrant groups. Hence, I plan to explore this medical factor in more detail in later studies.

Physiological Factors. Researchers have proposed that physiological changes caused by *chronic* and *cumulative stress* are the mechanisms for racism-related effects and health outcomes (Harrell, 2000; Mays, Cochran, & Barnes, 2007). Stress accelerates the release of cortisol which can activate endocrine systems that induce labor, and stress also reduces antibodies and promotes elevated risk of infections (Rich-Edwards & Grizzard, 2005; Wadhwa, Culhane, Rauh, & Barve, 2001 & Barve, 2001). These biochemical alterations are associated with preterm delivery and low birthweight (Hobel, Dunkel-Schetter, Roesch, Castro, & Arora, 1999 Castro, & Arora, 1999; Wadhwa, Sandman, Porto, Dunkel-Schetter, & Garite, 1993 Dunkel-Schetter, & Garite, 1993). I have conceptualized allostatic load and corticotropin-releasing hormone (CRH) as physiological mediators linking racism-related stress with adverse birth outcomes.

Allostatic Load and CRH. Allostasis is "wear and tear" on the autonomic, nervous, neuroendocrine, and immune systems that affects the body's ability to respond in a healthy manner to stressors (McEwen, 1998). A marker of chronic stress is allostatic load, a maladaptive biological response to frequent and cumulative stress. Most health studies have examined allostatic load in relation to chronic diseases. Only a handful of studies have measured this phenomenon as predictor of adverse birth outcomes. Wallace and colleagues found no relationship of allostatic load to preterm birth or low birth weight and no difference in the effect

of allostatic load on birth outcomes between Black and White women (M. Wallace et al., 2013; M. E. Wallace & Harville, 2013). Lower levels of allostatic load have been observed in Black women (Chen et al., 2010). Corticotropin-releasing hormone (CRH) has been associated with preterm labor ad birth (Hobel et al., 1999; Latendresse & Ruiz, 2011; Wadhwa et al., 2004), but to date there is no evidence to suggest racial disparities in this biological marker. One study found significantly lower CRH levels in African American women.

More studies are needed to understand these phenomena in association with racism-related stress in Black women and as potential sources for racial disparities in birth outcomes.

Discussion

The proposed model intends to spur research in the understudied area of racism and adverse birth outcomes and seeks to improve our understanding of cultural heterogeneity in Black populations with respect to experiences of discrimination, stress-coping responses, and birth outcomes among Black immigrants. However, some challenges with its use are to be noted. The model emphasizes racism-associated constructs and measures that have been intended primarily for studying social-psychological processes and mental health outcomes. Extrapolating the theories and measures to predicting birth outcomes (and presuming certain biological causal mechanisms) takes a leap to which the current state of knowledge is far behind. Also, participant reports of racism/discrimination may be overestimated in studies emphasizing racism or they may be underestimated due to social desirability responses that downplay the personal or public impact of racism. There is also likely underreporting of behavioral risks such as smoking or substance use during pregnancy.

The proposed research studies to be generated based on this model are intended for examining Black women only, and hence, there may be inadequate variance in perceived discrimination and race-related stress to detect significant associations. This limitation is

magnified with the proposed tests for interactions among the various psychosocial variables by national origin. Furthermore, the model does not specify associated constructs such as hostility or self-esteem that could affect coping responses. It also excludes interpersonal social support appraisals and resources.

Notwithstanding the focus on quantitative analyses in the present dissertation, mixed-method approaches would be ideal for use with this model. Qualitative studies have been used to elicit in-depth information about identity formation, processes of acculturation, psychosocial distress, and pregnancy experiences of Caribbean women and communities (Edge & Rogers, 2005; Waters, 1999). Ethnographic approaches have also been used with African American women and communities around the issue of racism-related stress and childbearing. The Harlem Birthright Project is one such study that revealed the nature of social stressors associated with structural forces such as unemployment and female-headed households (Mullings et al., 2001). Women were mobilized and made aware of structural forces that impacted their lives during pregnancy and across the lifecourse. This study also provided insight into individual and community processes to reduce stress and confront the problem of infant mortality. A qualitative study to explore perceptions of racism, stress, and health-specific as well as cultural risk and protective factors among reproductive-aged Black immigrant women is proposed for future study.

This conceptual model encourages a novel line of inquiry into racism, stress, and birth outcomes with careful consideration of ethnic heterogeneity in Black communities. To date, no published studies have examined structural and interpersonal exposures to racism-related stressors in Black U.S.-born/foreign-born differences in relation to birth outcomes. In 1992, the CDC began an initiative to examine the 'social context' of pregnancy for the prevention of adverse perinatal outcomes among Black women, with an emphasis on racially-mediated stress.

Perhaps coincidentally, Sherman James proposed in 1993 a paradigm that reinforced community psychosocial strengths as the key to buffering the effects of disparaging social conditions (James, 1993). Understanding the relative maintenance or erosion of such strengths in native and immigrant Black communities may foster community approaches toward reclaiming indigenous health-promoting psychosocial and cultural orientations to manage stress while at the same time challenging the social conditions that reify toxic and systematic stressors. The anticipated future research based on this model predicts that the relative contribution of racism and discrimination to birth outcomes and the covariates that modify these relationships may differ by nativity. Differential experiences and coping responses to structural racism and race-related stress may contribute significantly to the nativity differential in birth outcomes among Black women.

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CHAPTER 4

Predictors of Preterm Birth by National Origin: An Examination of the Immigrant Health Paradox in Caribbean Black Women

Comparisons of births in the U.S. by mother's nativity have held the interest of researchers who have explored 'paradoxical' observations of healthier maternal and infant health for Mexican-American immigrants, despite their lower income and education, when compared with their U.S.-born Hispanic counterparts and non-Hispanic White women (de la Rosa, 2002; Franzini, Ribble, & Keddie, 2001; R. Hummer, Powers, Pullum, Gossman, & Frisbie, 2007; Palloni & Morenoff, 2001; Vega, Rodriguez, & Gruskin, 2009). However, births to Black immigrant women are less prominently studied. The limited coverage exists despite the fact that births to foreign-born women constitute 14% of all non-Hispanic Black births in the U.S. (Martin, Hamilton, Osterman, Curtin, & Mathews, 2015), and adverse outcomes such as low birthweight and preterm birth are lower among foreign born Blacks as well (Elo, Vang, & Culhane, 2014). The health of immigrant birth cohorts from infancy to adulthood can impact population health for Blacks in the U.S.—especially in areas with large immigrant populations.

Despite the reporting of immigrant health risks and advantages among studies, perinatal health researchers are less forthcoming with respect to articulating the underlying *reasons* for the Black foreign-born advantage. Despite the more favorable measured risk factors such as higher educational attainment, lower smoking rates, and fewer fetal/neonatal health risks for foreign-born Blacks compared to U.S.-born Blacks, it is unknown the extent to which these characteristics contribute to the healthy birth advantage for Black immigrants (Elo & Culhane,

2010; Rosenberg, Desai, & Kan, 2002). Moreover, little is known about what erodes the survival advantage of infants born to immigrant women with longer periods of U.S. residence (Collins, Wu, & David, 2002; Urquia, Frank, Moineddin, & Glazier, 2010). Therefore, the field of immigrant health studies warrants more in-depth investigation of the role of nativity status in perinatal health disparities.

A fair number of studies have examined Black nativity status with respect to birth outcomes. U.S.-based studies on this topic were introduced in the 1970s and 1980s (Chavkin, Busner, & McLaughlin, 1987; Kessner, Singer, Kalk, & Schlesinger, 1973; Valanis & Rush, 1979) followed by studies during the past two decades that compared native and foreign-born Blacks (Acevedo-Garcia, Soobader, & Berkman, 2005; Cabral, Fried, Levenson, Amaro, & Zuckerman, 1990; Collins et al., 2002; Friedman et al., 1993; Fuentes-Afflick, Hessol, & Perez-Stable, 1998; Grady & McLafferty, 2007; Howard, Marshall, Kaufman, & Savitz, 2006; Kleinman, Fingerhut, & Prager, 1991; Mason, Kaufman, Emch, Hogan, & Savitz, 2010; Pallotto, Collins, & David, 2000; Rosenberg et al., 2002; Singh & Yu, 1996; Stein et al., 2009). Less common are studies with results stratified by Caribbean national origin (Elo et al., 2014; Friedman et al., 1993; Howard et al., 2006; Liu & Laraque, 2006; Pallotto et al., 2000; Stein et al., 2009). These studies generally confirm that immigrant Black women have more favorable birth outcomes than U.S.-born African American women (Acevedo-Garcia et al., 2005; Cabral et al., 1990; Elo et al., 2014; Howard et al., 2006; R. A. Hummer et al., 1999; Kleinman et al., 1991; Rosenberg et al., 2002; Singh & Yu, 1996). However, there are disparate health risks by birthplace among Black immigrants. A recent study of preterm birth among U.S.-born and foreign-born Blacks, limited to 27 states and D.C., revealed significant risk variability by country of origin among foreign-born Blacks (Elo et al., 2014). Except for prenatal care where the results were more favorable for Caribbean-born women, Caribbean-born women had worse pregnancy

risk characteristics than women from sub-Saharan Africa, and overall, the adjusted odds of preterm birth were 32% lower for African-born women relative to Caribbean-born women (*OR* = 0.68, CI: 0.66, 0.71) (Elo et al., 2014).

Nativity status may be a crucial link toward understanding *intra*-racial differences in birth outcomes. In a national sample (Kleinman et al., 1991), nativity status had a stronger independent effect than marital status, education, and parity in predicting birth outcomes in Black U.S.- and foreign-born mothers. Foreign-born Black mothers had 23% and 36% lower adjusted odds for infant mortality and low birth weight, respectively, relative to their U.S.-born counterparts. By comparison, the reduced odds for foreign-born Whites compared to U.S.-born Whites were 12% and 11% for IM and LBW, respectively. The larger proportionate differences by nativity within Black populations are striking.

A meta-analysis of migrant status in association with preterm birth and low birthweight revealed a similar pattern: within race, the protective effect of nativity among racial groups was strongest for Blacks in contrast to the moderate protection that nativity status conferred for Hispanics and the negligible impact of nativity among Asians and Whites (Urquia, Frank, & Glazier, 2010). In a 2008 national sample, foreign-born Blacks had 27% lower odds of preterm birth (*OR* = 0.73, CI: 0.73, 0.73) compared to U.S.-born Blacks, despite adjustment for sociodemographic, behavioral, and medical risk factors. These data suggest nativity-related contexts for perinatal health that are more variant for Black women than for other racial/ethnic groups, and they underscore the need for more detailed examination of concurrent risk and protective factors.

Research commentary on why Black immigrant women have healthier births than African American women remains largely speculative, although explanations favor “healthy migrant” (Fang, Madhavan, & Alderman, 1999; Valanis & Rush, 1979) or “cultural” (Cabral et

al., 1990; Friedman et al., 1993; Fuentes-Afflick et al., 1998; Rosenberg et al., 2002) hypotheses. Healthy migrant theories contend that immigrant women have better maternal-infant health outcomes than U.S.-born African American women due to their unique socioeconomic, psychological, or cultural resources that favor optimal health (Landale, Gorman, & Oropesa, 2006; Palloni & Morenoff, 2001; Wingate, Swaminathan, & Alexander, 2009) Namely, migrants in the U.S. are not a random selection of the populations of their countries of origin, but a highly-selected group with characteristics such as better physical and mental health, personal motivation, resilience, social support, and economic resources that foster their ability to migrate (Elo & Culhane, 2010; Fang et al., 1999; Jackson & Antonucci, 2005; Valanis & Rush, 1979; Williams et al., 2007). This social selection confers health advantages.

Culturally-based theories attribute factors such as reliance on family ties and social support networks (Murphy & Mahalingam, 2004; Scribner, 1996; Zambrana & Scrimshaw, 1997) and culturally-linked practices such as ethnic food diets, low smoking, and low substance use that translate into fewer health-risk behaviors. For example, African and Caribbean immigrants are more likely to breastfeed (Singh, Kogan, & Dee, 2007) and less likely to smoke, drink alcohol, or use illicit drugs than African American women (Bennett et al., 2008; Elo & Culhane, 2010; Persaud, 2007). Researchers have also credited the protective role of enhanced nutrition from ethnic diets (Cabral et al., 1990; Rosenberg et al., 2002). Moreover, the relative advantage of immigrant social support networks in ethnically dense neighborhoods compared to U.S. native Black support networks may provide an enhanced buffer against social or material hardships, thus explaining disparities in birth outcomes within Black populations (Mason et al., 2010).

Notwithstanding the logic of selective migration and culture-based arguments, the double burden of race and migrant status introduces complexities for Black migrants that are not

sufficiently acknowledged in theories of immigrant health that are largely directed to the study of Mexican American immigrants (Arthur & Katkin, 2006; Becares, Nazroo, Jackson, & Heuvelman, 2012). For example, the generalizability of healthy migrant theories diminishes when considering the national origin and racial diversity of immigrants. For example, a study found no significant differences in infant mortality risk between recent Puerto Rican migrants to the U.S. mainland and non-migrants who remained in Puerto Rico, hence no healthy migrant effect was demonstrated (Landale et al., 2006). This is unlike the greater consistency of perinatal health advantages found across studies for Mexican migrants. The pattern of declining maternal-infant among U.S. immigrants also appears to have a gradient effect by race and national origin. Landale et al. (2006) observed that infant mortality rates increased significantly for Puerto Rican migrants with increased duration of mainland U.S. residence (Landale et al., 2006). In international studies, the perinatal health advantage appears to be sustained most likely for White European immigrants and much less likely for Black Caribbean immigrants (Urquia, Frank, Moineddin, et al., 2010; Urquia, Glazier, et al., 2010; Urquia, O'Campo, & Heaman, 2012).

The heterogeneity in perinatal health risk found by race among immigrants calls for attention to the role of racial context and social-psychological influences. Puerto Ricans have a larger proportion of phenotypic Blacks in their population, and race/racism have been proposed as factors influencing maternal-infant health among Puerto Ricans (Bermudez-Millan et al., 2011; Landale & Oropesa, 2005). Racism has also been articulated as an important factor in the social and health adaptation of Black immigrants in the U.S. (Dominguez, Strong, Krieger, Gillman, & Rich-Edwards, 2009; Grady & McLafferty, 2007; Pallotto et al., 2000; Spence & Eberstein, 2009).

One alternative theory that has not yet been explored in perinatal health research relates to health consequences associated with the racial contexts of sending and receiving countries for

Black immigrants. Blacks are predicted to have relatively worse health when migrating from or residing in countries with majority White populations in contrast to having better health when migrating from areas with large concentration of minorities (Read & Emerson, 2005; Read, Emerson, & Tarlov, 2005). Accordingly, Black African and Black Caribbean immigrants, who are the racial majority in their home countries, have been theorized to experience more salutary supports, including a more positive racial identity, in contrast to the deleterious psychosocial contexts for Black immigrants and African Americans in the U.S. Social-psychological processes underlie these relationships that can protect or harm health. Read's theory was supported with U.S. National Health Interview Survey data where Black immigrants from Europe were found to have worse self-rated health, hypertension, and activity limitations than Black immigrants from Africa or the West Indies (Read et al., 2005). However, the theory was not upheld in a later study (Hamilton & Hummer, 2011), where it was found that health was not significantly different between African immigrants and immigrants from areas with less majority Black populations such as the Caribbean or Europe. Ethnic variation was observed, however, with respect to length of mainland U.S. residence—the health advantage declined more rapidly over time for Caribbean immigrants than for African immigrants.

When perinatal health researchers allude to the role of immigrant selectivity in the superior health of foreign-born U.S. residents (Fang et al., 1999), they often cannot validate their propositions due to lack of comparable maternal-infant health data from immigrants' home countries with which to compare findings. In addition, when racism is proposed as a determinant of disparate health profiles between U.S. immigrants and U.S.-born Blacks, the absence of racism measures in standard birth data as well as the lack of information on immigrants' duration of U.S. residence precludes examination of racism and birth outcomes for Black immigrants and African Americans when utilizing birth records. The present study addresses these limitations

by: (a) utilizing comparable birth data derived from U.S. natality files to permit a more direct exploration of the immigrant selectivity thesis; and (b) adopting theories of health advantage/disadvantage due to geographic racial context (Read et al., 2005) to explore the validity of this thesis, and by extension, the impact of proxy racism exposures. These premises will be tested using U.S. birth records on a population-based sample of Black immigrant mothers living on the U.S. mainland (New York City) compared to Black non-migrant mothers residing in the U.S. Virgin Islands.

Comparative analyses of births on the U.S. mainland and U.S. territories by maternal nativity and migrant status, focused on non-Hispanic Black mothers, are previously unreported in the public health literature. The closest approximation to the present research was a study by Landale et al. (2006) who analyzed infant mortality among Puerto Rican mothers in Puerto Rico and Puerto Rican migrants to the U.S. mainland—finding no healthy migrant effect for Puerto Rican women. The present study will unveil patterns of maternal and infant health for ethnically-heterogeneous Black women, featuring U.S. Virgin Islands' women in their native and receiving environments. Although the Virgin Islands is a territory of the U.S. and Virgin Islands' natives are U.S. citizens, Virgin Islands' birthplace stands as a proxy for "foreign-born" status relative to the U.S. This assertion holds true because Virgin Islanders identify with and are more heavily characterized by the historical, geographic, demographic, and cultural distinctiveness of the Caribbean region and its people than they are by U.S. nationality (Goulbourne & Solomos, 2004; Roopnarine, 2008). More importantly, the structural and social-psychological contexts of racial stratification are less severe for Blacks in the Virgin Islands and throughout the Caribbean than for Blacks on the U.S. mainland (Benson, 2006; Foner, 1998). The populations selected for this study offer a unique opportunity to explore the role of nativity status to maternal and infant

health in cross-geographic contexts that are presumed to differ by racism exposures (Read & Emerson, 2005).

For expediency of reporting, the mention of "Caribbean" women throughout this paper refers to women born in the Caribbean, *except* for the Virgin Islands—thus reserving "Virgin Islands"-born women as a distinct category. Mention of "migrant" women will refer to the V.I.-born and Caribbean-born women living in New York City who gave birth in New York City. These populations are abbreviated by their nativity and residence as VINY and CANY, respectively. Mention of "native" or non-migrant women will refer to V.I.-born and Caribbean-born residents of the Virgin Islands who gave birth in the Virgin Islands, and they are abbreviated as VIVI and CAVI.

Purpose

The aim of this chapter is to determine the relative contributions of risk factors for preterm birth in Black populations by nativity and migrant status. The risk factors to be tested are construed as socially-patterned and are so indicated in my conceptual model (described in Chapter 3). Although some risk factors for preterm birth are well known, including maternal age, low SES, smoking, infant congenital anomalies, and medical factors such as chronic and pregnancy-induced hypertension and diabetes, this paper will determine the degree to which these commonly measured factors differ between Virgin Islands-born, Other Caribbean-born, and U.S.-born Black women. More importantly, this paper will quantify the extent to which the observed differences can "explain" the maternal and infant health advantage for non-Hispanic Black Virgin Islands and other Caribbean immigrants relative to African Americans. After accounting for observed risk and protective factors contributing to preterm birth, the unobserved background factors believed to contribute to persistent disparities will be addressed. These

unobserved factors, in part, are believed to be among the structural racism and neighborhood social dynamics components of my conceptual model.

Two research questions will be explored. First (Research Question 1), to what extent do traditional pregnancy risk factors explain differences in preterm birth between Virgin Islands-born immigrants, other Caribbean-born immigrants, and African Americans? The populations for Question 1 are all based in New York City. For this question, I will examine the extent to which demographic factors (i.e., nativity, education, marital status), behavioral and medically-related factors (i.e., prenatal care, smoking, weight gain, maternal medical risks), and infant factors (congenital anomalies) explain differences in preterm birth between Virgin Islands-born and other Caribbean-born immigrants in comparison to U.S.-born Blacks. I hypothesize that demographic factors will be more likely than behavioral, medical, and infant factors to explain the health advantage in preterm birth for Virgin Islands immigrants and Caribbean Black immigrants in the U.S. relative to native-born African American women.

Second (Research Question 2), is there a healthy migrant effect, in relation to preterm birth, demonstrated for Virgin Islands-born and other Caribbean-born immigrant mothers in New York City compared to their non-migrant counterparts who reside in the U.S. Virgin Islands? U.S.-born women are not included in this migrant-focused analysis. This study offers a more direct test of healthy migrant theories by comparing non-migrant and migrant populations in their sending and receiving countries rather than presuming a healthy migrant effect based on how immigrants compare to African Americans in the U.S. Question 2 conceptualizes geographic context (i.e., U.S. mainland or U.S. territories) as a proxy for relative exposure to racism. I hypothesize that preterm birth rates and maternal and infant risk characteristics will be lower for V.I.-born mothers in the Virgin Islands (VIVI) and Caribbean-born women in the Virgin Islands (CAVI) than for their migrant counterparts in New York City (VINY and CANY).

I also hypothesize that demographic factors will have a larger impact on preterm birth for V.I.-born and Caribbean-born residents of New York than for their native counterparts living in the Virgin Islands.

Method

Birth data from 2000-2009 were obtained from the New York City Department of Health and Mental Hygiene, and depending on the research question, examined separately (Question 1) or combined with 2000-2004 birth data for the U.S. Virgin Islands (Question 2). Detailed analyses were performed to ascertain the predictive potential of demographic, behavioral, and medical risks on premature birth for foreign-born Black mothers compared to U.S.-born Black mothers. Preterm birth (< 37 completed weeks of gestation) was selected as the outcome of interest because it is a more valid predictor of infant morbidity and mortality than infant birth weight (Klebanoff & Keim, 2011; Shapiro-Mendoza & Lackritz, 2012).

All populations selected for this study are classified as non-Hispanic Blacks based on documentation of the mother's race as "Black" and the mother's ethnicity as "non-Hispanic" in the birth record. By convention, mother's race is based on written self-report of demographic information requested on medical admissions forms. Information included on the standard U.S. birth certificate, which is utilized in both New York City and the U.S. Virgin Islands, is extracted from the medical record by designated staff at local hospitals or public health departments. The extent to which race/ethnicity/nativity documentation was based on proxy report or imputed in the birth records is unknown; nor is it known the extent to which the documented race or ethnicity concur with the primary racial or ethnic self-identification of the respondent.

Data Sources

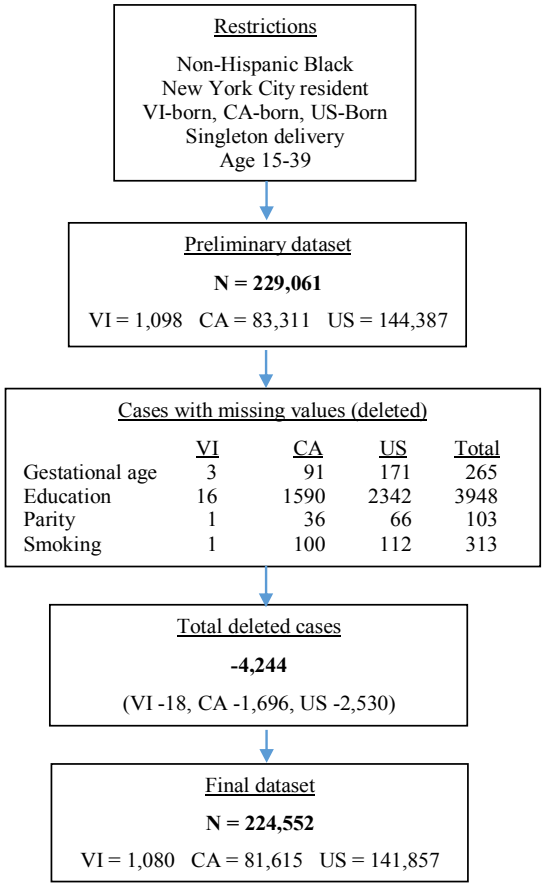
New York data. The New York City birth files were obtained from the New York City Department of Health and Mental Hygiene (NYCDH). Permission to utilize these data for

research was obtained in 2012 from the Institutional Review Boards of both NYCDH and the University of Michigan. Annual birth files were received electronically through an encrypted connection from NYCDH servers. These data were then converted to SAS files and merged across the 2000-2009 birth cohorts.

Mothers aged 15-39 who were coded as non-Hispanic Black (single-race designation), with birthplace in the Virgin Islands, another "non-Hispanic" Caribbean island, or the 50 U.S. states and the District of Columbia (D.C) were extracted for the analysis. This selection included Blacks born in Belize, Guyana, and Haiti but not Blacks from Puerto Rico, the Dominican Republic, Africa, Europe, and other places that were not in the "non-Hispanic" Caribbean or the U.S. Records were further restricted to singleton deliveries and mothers whose permanent residence was in New York City.

The number of New York City birth records meeting the initial selection criteria was 229,061. Cases were further excluded if there was missing information for gestational age, (n = 265, 0.12%), education (n = 3,948 1.73%), parity (n = 103, 0.05%), or smoking status (n = 313, 0.14%). Due to the large number of cases with missing prenatal care information (n = 12,861, 5.62%), an "unknown" prenatal care category was created, and these cases were retained in the dataset. Because some cases had multiple missing values, the aggregate number of missing cases is less than the cumulative count of missing cases for each variable. Therefore, the final working dataset consisted of 224,552 cases (1,080 V.I.-born mothers, 81,615 Other Caribbean-born (CA) mothers, and 141,857 U.S.-born mothers). The proportions of missing and unknown values were distributed similarly among all three groups. Figure 4.1 outlines a sample selection flowchart of the New York City study populations.

Figure 4.1 Flowchart of Missing and Selected Cases from New York City Birth Files (2000-2009)



Ten years of data were collected with the intention to obtain an adequate number of V.I.-born mothers in New York City for the analysis. Despite this strategy, the V.I.-born immigrants in the New York dataset totaled only 1,080, representing less than 0.5% of all births. Chi-squared analyses were done to see if the V.I. and the CA groups were similar enough in the study characteristics to justify combining V.I.-born and Other Caribbean-born women in New York as one group (see Table 4.1). Due to group differences in key study variables in addition to the study's unique interest in distinguishing risk patterns for V.I-born mothers in New York City and

in the Virgin Islands (Research Question 2), the V.I. and CA groups were not collapsed in the current analysis.

A power analysis was conducted on the New York City data utilizing NQuery (Fleiss, Ttun, & Ury, 1980). The analysis was based on disproportionate samples sizes, a difference in the outcome of at least 3% between groups, and a desired power of 80%. With these criteria, 12% power was assessed for V.I./U.S. comparisons and 99% power was obtained for CA/U.S. comparisons.

Virgin Islands data. The U.S. Virgin Islands birth files were downloaded from Vital Statistics Online (National Center for Health Statistics, 2014). As of 2005, mother's birthplace and nativity-status are not included in the public use data files. Therefore, the latest years of data with nativity information present (2000-2004) were utilized. Births to non-Hispanic Black mothers whose usual place of residence was the Virgin Islands were extracted. U.S.-born Blacks in the Virgin Islands (n = 312) were removed due to their small numbers and the lack of a conceptual framework within the context of this study that would hypothesize about how U.S.-born Black mothers would fare outside of a U.S. context. Data were further restricted to singleton births and women aged 15-39. Because some cases had missing values for several variables, the aggregate number of missing cases is less than the cumulative count of missing cases for each variable. In total, the preliminary selection was 4,579 cases (V.I.-born = 3,047; Other/CA-born = 1,532). In the U.S. Territorial birth files, it was not possible to identify women from Caribbean islands other than the Virgin Islands or Puerto Rico. However, based on U.S. census data, it is known that over 90% of foreign-born Blacks in the Virgin Islands are born in another West Indian island (U.S. Census Bureau, 2010). Therefore, foreign-born Blacks in the Virgin Islands were categorized as "Caribbean-born" for the purposes of this study.

V.I.-born women (VIVI). Of the 3,047 V.I.-born resident mothers, cases were removed if there was data missing for gestational age (n = 8, 0.26%), education (n = 12, 0.39%), parity (n = 23, 0.76%), or smoking (n = 27, 0.89%). Cases with "unknown" medical risk factors (n = 107, 3.52%) were also deleted because comparative analyses of maternal medical risks are a central consideration in this paper. Cases with missing maternal weight gain during pregnancy (n = 478, 15.73%), abnormal infant conditions (n = 162, 5.33%), and infant congenital anomalies (n = 145, 4.77%) were retained in the dataset, but neither weight gain nor infant risks were modeled in regression analyses between V.I.-born Virgin Islands residents and V.I.-born NYC residents. Because some cases had missing values for several variables, the aggregate number of missing cases is less than the cumulative count of missing cases for each variable. Therefore, the final number of cases for V.I.-born mothers residing in the Virgin Islands was 2,883.

CA-born women (CAVI). Of the 1,532 Other/Caribbean-born mothers residing in the Virgin Islands, five cases were deleted with missing values for gestational age (n = 5, 0.33%), twenty for missing education (n = 20, 1.31%), seven for missing parity (n = 7, 0.46%), nine for missing data on smoking (n = 9, 0.59%), and 36 for maternal medical risks (n = 36, 2.36%). Cases with missing maternal weight gain (n = 257, 16.83%), abnormal infant conditions (n = 64, 4.19%), or congenital anomalies (n = 64, 4.19%) were retained in the dataset, but neither weight gain nor infant risks were modeled for comparative analyses by migrant status between CA-born V.I. residents and CA-born NYC residents. The final number of maternal-infant records for Other/Caribbean-born mothers residing in the Virgin Islands was 1,459. Figure 4.2 outlines the sample selection process for the V.I. populations.

Chi-squared analyses were done to see if the VIVI and the CAVI groups were similar enough in the study characteristics to justify combining V.I.-born and Other Caribbean-born women in the Virgin Islands as one group for the analyses. The VIVI and CAVI groups differed

significantly in age $\chi^2(1, N = 4332) = 356.3$, $p < .001$; education $\chi^2(3, N = 4332) = 34.9$, $p < .001$; marital status $\chi^2(1, N = 4332) = 187.1$, $p < .001$; and diabetes $\chi^2(1, N = 4332) = 5.72$, $p = .02$, with marginal differences in prenatal care initiation $\chi^2(3, N = 4332) = 6.96$, $p = .07$; birthweight $\chi^2(1, N = 4332) = 0.10$, $p = .07$; and parity $\chi^2(1, N = 4332) = 21.9$, $p = .07$. There were no significant differences in smoking $\chi^2(1, N = 4332) = 0.53$, $p = .47$; weight gain $\chi^2(1, N = 3676) = 0.10$, $p = .95$; hypertension $\chi^2(1, N = 4332) = 0.32$, $p = .57$; maternal medical risks $\chi^2(1, N = 4332) = 0.81$, $p = .36$; abnormal infant conditions $\chi^2(1, N = 4201) = 0.04$, $p = .83$; congenital anomalies $\chi^2(1, N = 4209) = 1.21$, $p = .27$; or gestational age $\chi^2(1, N = 4332) = 0.04$, $p = .84$. For consistency with the New York analyses, I disaggregated the Virgin Islands' sample into V.I-born and Caribbean-born mothers.

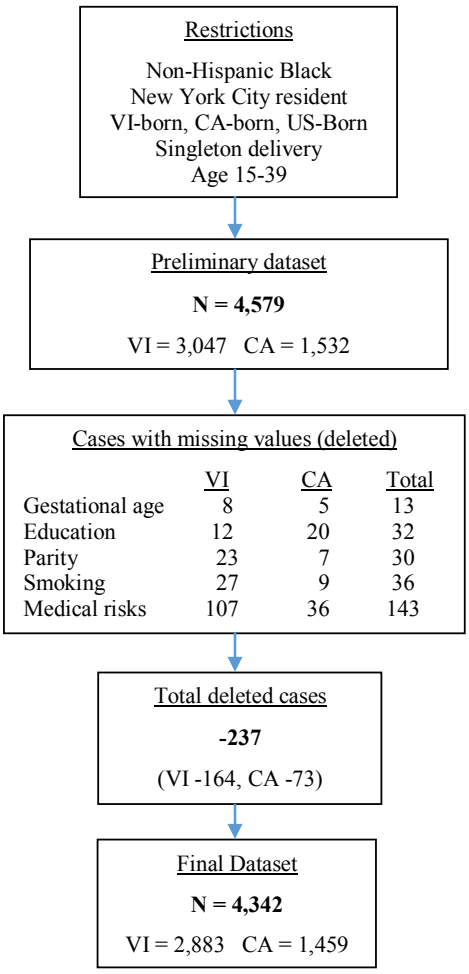
A power analysis was conducted on combined New York and Virgin Islands data utilizing NQuery (Fleiss et al., 1980). The analysis was based on disproportionate samples sizes, a difference in the outcome of at least 3% between groups, and a desired power of 80%. In order to permit migrant comparison, the Virgin Islands and New York data were combined. Based on these criteria, 86% power was obtained for comparisons between V.I.-born mothers in the Virgin Islands and V.I-born mothers in New York, and 96% power was obtained for comparisons between Caribbean-born women in the V.I. and New York.

Measures

Outcome variable. *Preterm birth* (PTB) was defined as a live-born, singleton infant delivered at less than 37 completed weeks of gestation. Preterm birth was dichotomized as <37 completed weeks of gestation or ≥37 completed weeks of gestation. I did not distinguish between spontaneous and induced preterm births. The clinical estimate of gestation (based on the birth attendant report and aided by baseline ultrasound measurements where possible) was used. In the 2000-2007 New York City birth records, the clinical estimate of gestational age was reported

only for fetuses ≥20 weeks. From 2008-2009, the reported range for fetal gestational age was ≥17 weeks. The correlation of the gestational age based on last menstrual period (LMP) and the gestational age based on the clinical estimate was high—both in the presence of reporting and in the consistency between the LMP estimate and the clinical estimate. Therefore, reliance on the clinical estimate did not substantially reduce the number of cases available for analysis.

Figure 4.2 Flowchart of Missing and Selected Cases from Virgin Islands Birth Files (2000-2004)



Explanatory variables. ***Nativity*** was designated for mothers as Virgin Islands-born (V.I.), Caribbean-born (CA), or U.S.-born (U.S.). Models comparing V.I.-born, CA-born, and

U.S.-born mothers in New York City used the U.S.-born mothers as the reference group. U.S.-born mothers were designated as the reference group because of the study's primary interest in specifying detailed estimates for the foreign-born populations who are more understudied than the U.S.-born population. Also, preterm birth and covariate estimates for African Americans are well-established in the literature. The size and stability of the U.S.-born Black population makes it suitable for use as a reference. Models comparing V.I.-born and CA-born migrant mothers in New York City with their native counterparts in the Virgin Islands used the New York-based mothers as the reference group. The NYC-based migrants were used as the reference in order to highlight estimates for the non-migrant group for whom no information is currently available in the perinatal health literature. ***Maternal age*** was categorized as 15-19, 20-24, 25-29, 30-34, and 34-39, with age group 20-24 being the reference category. The age categories represent developmental stages for motherhood (teen 15-19, young adult 20-29, middle adult 30-34; 34-39 older/advanced maternal age mother) and they afford refined reporting of preterm birth risks with advancing maternal age. Mothers aged 20-24 are the reference because the largest proportion of births occurred during these ages and they had the lowest risk of preterm birth in the overall study population. Mother's highest years of ***education*** was categorized as 0-11 years (less than high school), 12 years (high school graduate), 13-15 years (some college), and ≥16 years (college graduate). The reference category is high school graduates. ***Marital status*** was dichotomized as married or unmarried based on an imputation by NYCDH using a non-disclosed algorithm (NYC does not report marital status on the birth certificate). The reference category is married. ***Smoking*** was dichotomized as tobacco use during pregnancy or no tobacco use during pregnancy (reference). ***Prenatal care*** was categorized as 1st trimester initiation, 2nd trimester initiation, 3rd trimester initiation, and no prenatal care/unknown prenatal care with the reference

category being first trimester initiation. ***Parity*** was dichotomized as primiparous or multiparous, with multiparous being the reference category.

Any report of a ***maternal medical risk*** or complication was categorized as '1 or more medical risk factors' or 'no reported medical risk factors' (reference is none). Maternal medical risks in this analysis included any the following: chronic or gestational diabetes, chronic or gestational hypertension, cardiac structural or functional defects, other serious chronic illness, anemia, asthma or other chronic lung disease, rh sensitization, poly- or oligo-hydramnios, hemoglobinopathies, abruptio placenta, eclampsia, poor previous pregnancy outcome (including previous preterm birth), prelabor referral for high-risk care, sexually transmitted infection, hepatitis B or C, rubella, or infertility treatment.

The selected maternal medical conditions were justified as risk factors for preterm birth for the following reasons. Chronic and pregnancy hypertension are significant risks for pre-eclampsia and eclampsia which are highly correlated with preterm birth. Chronic diabetes as well as cardiac, hematologic, and chronic respiratory conditions can cause vascular and other pathologies which can restrict blood flow and oxygen to the fetus causing fetal hypoxia and a physiologic cascade leading to preterm labor and preterm birth. Polyhydramnios and oligohydramnios refer to excess or inadequate amniotic fluid, respectively; these conditions can lead to uterine overdistention or biochemical changes precipitating spontaneous or induced preterm birth. Mothers who are referred for high risk care or who have chronic medical conditions may have a higher likelihood of medically-indicated preterm birth. Sexually transmitted infections, whether diagnosed or subclinical, pose risks for intrauterine infection which is correlated with preterm birth. Infertility is associated with preterm birth, including preterm singletons, due to unexplained factors associated with subfecundity (lower ability to reproduce). The primary medical risk factors for preterm birth are previous preterm birth,

hypertension, diabetes, preterm premature rupture of membranes, low prepregnancy BMI, excessive or inadequate maternal weight gain during pregnancy, uterine cervical anomalies, and short interpregnancy interval (Behrman & Butler, 2007). The more complete and precise documentation of maternal medical conditions and infant risk conditions in the New York City dataset could have contributed to the higher prevalence of medically-related risk factors for mothers in New York than for mothers in the Virgin Islands.

Congenital anomalies were dichotomized as 'any reported congenital anomaly' and 'no reported congenital anomaly,' with no reports as the reference. Congenital anomalies included any of the following: anecephaly, spina bifida, congenital heart disease, diaphragmatic hernia, omphalocele, gastroschisis, limb reduction defect, chromosomal disorders, and hypospadias. Congenital malformations are associated with preterm birth (Honein et al., 2009; Rasmussen, Moore, Paulozzi, & Rhodenhiser, 2001). ***Abnormal newborn conditions*** were categorized dichotomously as 'no reported abnormal infant condition' or 'any reported abnormal infant condition,' including assisted ventilation, admission to the neonatal intensive care unit, seizures, surfactant therapy, suspected neonatal sepsis, and significant birth injury. No reports are the reference category. Abnormal conditions of the newborn are reported in this paper for descriptive purposes, but they are not modeled as predictors of preterm birth.

Analyses

The research questions for this paper utilized two distinct study populations. Analyses by nativity (Question 1) referred to New York residents only. Analyses by migrant status (Question 2) combined data for immigrant groups in New York and non-migrant women in the Virgin Islands. Each question employed similar analytic techniques to estimate preterm birth odds and evaluate the degree to which various risk factors contribute to the outcome in the respective groups.

Descriptive analyses. For descriptive analyses, cross-tabulations were calculated for the outcome and covariate variables and these tables were estimated with the Wald chi-square test to evaluate proportionate frequency differences by nativity or migrant group.

Stratified model estimates. Logistic regression models, stratified by nativity (Question 1) or migrant group (Question 2), were estimated to evaluate the contribution of demographic, behavioral/medical, and infant-specific risk factors to preterm birth within each nativity or migrant population. The models were estimated in a forward stepwise manner, beginning with a base model that included only maternal age and parity, and adding the remaining covariates in conceptually-linked groups to evaluate the change in estimates contributed by each set of predictors. The covariate groups were added hierarchically beginning with the demographic variables (age, education, marital status), followed by the behavioral and medical variables (prenatal care, smoking, weight gain, maternal medical conditions), and lastly the infant-specific variable (congenital anomalies). Covariates were not removed once added to the model.

Combined model estimates. Logistic regression models were also estimated with nativity group populations combined (Question 1) or migrant group populations combined (Question 2) which permitted the evaluation of interaction effects by nativity or migrant status within each set of covariates. In the combined nativity-group models (Question 1), V.I.- and Caribbean-born mothers in New York City were compared to U.S.-born mothers in New York City as the reference group. In the migrant-group models (Question 2), V.I.-born non-migrant women in the Virgin Islands were compared to the reference group of V.I-born migrants in New York City; and CA-born women residing in the Virgin Islands were compared to the reference group of CA-born migrants in New York City. I began with a model estimated only for nativity (Question 1) or a model estimated only for migrant status (Question 2), followed by the subsequent addition

of demographic, behavioral/medical, and infant-specific risk factors to the respective models as described previously.

Two-way interaction terms were added last, combining nativity status with each demographic, behavioral/medical, or infant-specific covariate (Question 1) or migrant status with each demographic, behavioral/medical, or infant-specific covariate (Question 2). The interaction terms were added all at the same time. This study was interested in how index groups, by nativity or migrant status, uniquely differed from the reference population across a range of traditionally-measured maternal and infant risk characteristics. In New York City, that involved comparing V.I.-born mothers and CA-born mothers to U.S.-born mothers. Evaluating the healthy migrant effect involved comparing V.I-born and CA-born mothers in the Virgin Islands with the reference populations of V.I.-born and CA-born mothers who migrated and give birth in New York City.

Interaction effects were estimated as the ratio of the estimates for the index nativity group compared to the reference nativity group. The midpoint estimate for the interaction odds ratio can be calculated as

$$\boldsymbol{exp}\ (\boldsymbol{\beta_1} + \boldsymbol{\beta_3}),$$

where β_1 the log odds estimate for the index nativity term and β_3 is the log odds estimate for the interaction term involving nativity and the other covariate of interest. The betas were estimated from a fully adjusted logistic regression model that included all main and interactive parameters.

The interaction log odds ratio can also be represented as:

$$\frac{\boldsymbol{OR}_{\textbf{index nativity group}}}{\boldsymbol{OR}_{\textbf{reference nativity group}}} = \frac{\boldsymbol{exp}\ (\boldsymbol{\beta_0} + \boldsymbol{\beta_1} + \boldsymbol{\beta_2} + \boldsymbol{\beta_3})}{\boldsymbol{exp}\ (\boldsymbol{\beta_0} + \boldsymbol{\beta_2})} = \boldsymbol{exp}\ (\boldsymbol{\beta_1} + \boldsymbol{\beta_3}) = \boldsymbol{e}^{\boldsymbol{\beta 1}} \times \boldsymbol{e}^{\boldsymbol{\beta 3}}$$

The betas can be approximated from the main effects and interactive odds ratios reported in the results tables, with some variation in final estimates due to rounding error. For example,

$$\frac{OR_{CA\ aged\ 35-39}}{OR_{US\ aged\ 35-39}} = \exp\left(\beta_{1CA\ nativity} + \beta_{3CA\ nativity*aged\ 35-39}\right) = e^{-.1628} \times e^{-.1098}$$

$$= .8498 \times .8960 = .76 \approx [.85 \times .90 = .77]$$

To calculate the confidence intervals for the interaction effect, I utilized the *covb* option to produce a variance-covariance matrix for fully-adjusted logistic regression models that included interaction parameters. The variance-covariance matrix displays the variances for the main effects involved in an interaction, the interaction terms, and importantly, the joint variance (i.e., covariance) shared between the main effects and the interaction parameters of interest. The total variance must be calculated in order to correctly estimate the significance of interaction effects. The formula for the total variance is:

Variance β_1 + Variance β_3 + 2(Covariance β_1 + β_3)

The square root of the total variance produces the standard error (SE), from which the confidence interval can be calculated using the formula (log odds $\beta_3 \pm 1.96$ * SE). Odds ratios can be produced by exponentiating the log odds.

Likelihood ratio tests. After estimating the stratified or combined-group logit models, I performed likelihood ratio (LR) tests to further assess the contribution of demographic, behavioral/medical, and infant-specific covariates to the outcome of preterm birth. The LR test was used as an accompaniment to the observance of odds ratios which indicated magnitude of effects for the respective predictors. The LR test compares the log likelihoods of a full model with one or more predictors and a nested reduced model with a restricted set of predictors and tests whether the difference between the two models is statistically significant (Hosmer, Lemeshow, & Sturdivant, 2013). The LR test statistic follows a chi-square distribution with

degrees of freedom equal to the difference in the degrees of freedom between the full and reduced models. If the difference is statistically significant, then the full model is said to fit the data significantly better than the reduced model. A significant LR test provides evidence of the degree to which a predictor contributes to the model.

For stratified, single-group models, I started with a saturated model, inclusive of all predictors under study, then I removed one predictor at a time, comparing the fit of the fully-saturated model versus the fit of the model minus the predictor that was removed. I computed the difference in -2 Log Likelihood (-2LogL) between the saturated and reduced models. Higher values for -2LogL are associated with smaller *p*-values and indicate better model fits, thus validating that the tested predictor(s) do make a difference in the outcome of preterm birth.

For the combined-group models, I employed LR tests with a focus on testing the significance of interactions between nativity (i.e., VI-born or CA-born) and demographic variables, medically-related variables, and infant-specific variables. For this procedure, I estimated a combined-group logit model for preterm birth, inclusive of all main effect predictors plus interaction terms with the non-reference nativity or migrant population combined with each predictor. I then ran a reduced model absent of the demographic, medical, or infant interactions for the index population of interest. Next, I evaluated the significance of the LR test statistic (at alpha level .05). A significant result indicated that there was a unique multiplicative effect of the acknowledged risk factor in the index population.

Counterfactual analyses. Finally, to aid in quantifying the contribution of unobserved background factors to differences in the predicted occurrence of preterm birth between populations, I conducted counterfactual analyses. For Question 1, restricted to the mothers in New York City, I estimated the probability of preterm birth among U.S.-born mothers as if they had the population characteristics of the V.I.-born or Caribbean-born mothers, respectively. To

do this procedure, I estimated a logit model for preterm birth in the U.S.-born mothers with all risk factor variables under study and saved an output dataset based on this model with the individual predicted probabilities set to 'missing.' Next, the population characteristics of the V.I.-born and CA-born mothers were applied to the model estimated for U.S.-born women. This step entails a "switching" of coefficient values in a manner that roughly mimics the Blinder-Oaxaca decomposition technique (Fairlie, 2005). The counterfactual probabilities were then averaged for the U.S.-born women based on the V.I.- or CA-born populations, respectively. For Question 2, I repeated similar procedures to estimate the probability of preterm birth among V.I.-born and Caribbean-born migrant mothers in New York City as if they had the population risk characteristics of their native counterparts who remained in the Virgin Islands.

SAS procedures. All analyses were performed with SAS 9.4 statistical software (SAS Institute, Inc., Cary, North Carolina). Logistic regression models were run using the *proc logistic* function in SAS using reference coding and the *clparm* and *exbp* options, respectively, which compute log odds and log odds confidence intervals which are subsequently exponentiated to produce an odds ratio for each non-reference level of the predictors. The significance of each parameter was evaluated at the 95% confidence level, indicated by the Wald X^2 test of significance for the log odds for each term.

In SAS, there is no automated likelihood ratio test for non-linear models involving interactions with categorical predictors. Therefore, I manually computed the differences in likelihood ratios and degrees of freedom and utilized these values to calculate a *p*-value utilizing a Chi-squared function in SAS. For the counterfactual analyses, I employed a background computational procedure in SAS that allowed for the listing of predicted probabilities based on a logit model estimated for a reference population; I applied these predicted probabilities to the cases in one or more index populations (thus switching coefficient values for the covariates

between the reference and index populations); and then averaged these 'counterfactual' probabilities with *proc means*. These steps were done as a proxy method to mimic the Blinder-Oaxaca decomposition technique (Fairlie, 2005).

Results

Research Question 1: Variations by Nativity Status (VI-born and Caribbean-born immigrants in New York City compared to U.S-born mothers in New York City)

Maternal and Infant Characteristics. Table 4.1 displays maternal and infant characteristics by nativity for the New York City births. Among demographic factors, African Americans had the highest percentage (16.4%) of teen births, and Caribbean women had the highest percentage of births to mothers 35-39 (19%) which was almost double the birth rate for African Americans in this older age group. Educational attainment was highest for V.I.-born women of whom 18% had a four-year college degree. Marital rates were low in all groups, but particularly among U.S-born women where only 19% were married compared to marital rates of 26% and 39% for V.I.-born and Caribbean-born women, respectively.

For behavioral factors, early initiation of prenatal care was the norm for all groups, where roughly 60% of women in each group began prenatal care in the first trimester. Caribbean women were the least likely to have no prenatal care. Reported smoking during pregnancy was highest among African Americans (5%), which far surpassed the smoking rates for V.I. women (1.2%) and Caribbean women (0.57%).

About 30% of women in all groups had at least one reported medical risk factor for pregnancy, including previous preterm birth, infections, chronic diseases, and reproductive health conditions. Among the medical risk factors, the proportion of hypertensive disorders, including pre-existing hypertension and pregnancy-related hypertension, was roughly equivalent in all New York-based groups, ranging from 4.1% to 4.6%. Diabetes in the New York-resident

mothers was highest among Caribbean immigrants (5.5%) and V.I. immigrants (4.26%) with a lower proportion of diabetes (3.49%) among U.S.-born women. The groups were roughly equivalent for low weight gain during pregnancy, and African Americans were most likely to have excessive weight gain.

Low birthweight and preterm birth rates were statistically different, but not widely disparate, between the groups. Low birthweight percentages were about the same between Virgin Islands women (11.4%) and African Americans (11.5%) compared to 9.3% of low birthweight births for Caribbean women. Preterm births were lowest for Caribbean immigrant women (10.7%) and V.I.-born immigrant women (11.6%) and slightly higher for African American women (12.1%).

In summary, the differences in observed characteristics between V.I.-, CA- and U.S-born women in New York City the groups were largest for age, education, marital status, and smoking during pregnancy. The three nativity groups were fairly equivalent for prenatal care initiation, weight gain during pregnancy, and maternal medical risk factors. Due to the large sample sizes for the Caribbean and U.S.-born women, even small group differences would be evaluated as statistically significant. Indeed, chi-squared analyses comparing all three groups produced *p* values of <.0001 on average which confirm significant differences by nativity group for each risk factor. The only exception was for infant congenital anomalies for which no significant differences were found.

Stratified models. As an extension of the examination of differences in risk factors between the nativity groups, I conducted stratified analyses (by nativity group) to elicit information on the magnitude and degree to which each risk factor independently predicted preterm birth for each population. Stratified models permitted assessment of the degree to which the range of predictors contributed to the outcome of preterm birth solely *within* each respective

population, and not in relation to the combination of risk factors in another population. Lastly, stratified models afforded within-group examination of covariates involved in interaction effects that were discovered in combined models.

In Table 4.2, results stratified by nativity are shown for Virgin Islands-, Caribbean-, and U.S.-born mothers in New York City. In preliminary models (Appendix Table B.1), there was hierarchical adjustment for covariate risk factors in each population, starting with parity and age, and continuing with the addition of education, marital status, prenatal care, smoking, maternal weight gain during pregnancy, maternal medical risks, and infant congenital anomalies. With rare exceptions, coefficient values for the progressively-adjusted risk factors did not change in excess of 10% from one model to the next, and the confidence limits overlapped with adjustments for additional risk factors (Appendix Table B.1). Because few differences emerged for risk factor estimates in the restricted models compared to the fully-adjusted models, I will focus only on the results from the fully-adjusted models, as displayed in Table 4.2.

Virgin Islands-born mothers, NY. Table 4.2 indicates that only age, prenatal care, and maternal medical risks significantly predicted preterm birth for Virgin Islands immigrants in New York. Not surprisingly, mothers aged 35-39 had over twice the odds of preterm birth relative to mothers 20-24 (*OR* = 2.53, 95% CI: 1.30, 4.91). Women with no prenatal care/unknown prenatal care were 2.7 times more likely to have a preterm birth (*OR* = 2.72, 95% CI: 1.46, 5.05) compared to women who initiated care in the first trimester. Women with maternal medical risks were over twice as likely to have a preterm birth compared to women with no documented medical risks (*OR* = 2.11, 95% CI: 1.42, 3.14).

Caribbean-born mothers, NY. As noted in Table 4.2, the odds of preterm birth for Caribbean immigrants delivering their first child were 21% higher (*OR* = 1.21, 95% CI: 1.15, 1.27) compared to women with previous children. For demographic factors, there was an age-

graded effect such that preterm birth odds increased progressively with advancing age. Caribbean immigrant mothers 35-39 demonstrated a 51% increased odds of preterm birth (*OR* = 1.51, 95% CI: 1.40, 1.63) relative to women aged 20-24 in their nativity group. College education was only slightly protective relative to attaining only a high school education (*OR* = 0.92, 95% CI: 0.86, 0.99), and being unmarried increased the preterm birth odds by 12% (*OR* = 1.12, 95% CI: 1.07, 1.18) in consideration of other factors.

With respect to medically-related risk factors, the impact of prenatal care for Caribbean immigrants was somewhat counterintuitive; women who initiated care in the second and third trimesters had 9% and 34% *reduced* odds of preterm birth compared to women who began care in the first trimester. Mirroring expected patterns, women with no prenatal care/unknown prenatal care had increased preterm birth odds (*OR* = 1.72, 95% CI: 1.58, 1.86). Elevated weight gain during pregnancy significantly *reduced* the odds of preterm birth (*OR* = 0.77, 95% CI: 0.73, 0.82) for Caribbean immigrants relative to women who had a 'normal' weight gain of 16-40 lbs, and Caribbean women with low weight gain had expected elevated odds (*OR* = 1.78, 95% CI: 1.68, 1.89). Maternal medical risks (*OR* = 2.14, 95% CI: 2.05, 2.24) and infant risks (*OR* = 1.58, 95% CI: 1.36, 1.84) also increased the odds of preterm birth in this population. The effect of smoking on preterm birth (*OR* = 1.45, 95% CI: 1.13, 1.88) was reduced by 10% (*OR* = 1.35, 95% CI: 1.04, 1.75) after adjusting for other medically-related risks (Appendix Table B.1, CA-born).

U.S.-born mothers, NYC. Table 4.2 also shows the risk-factor adjusted preterm birth odds for African American mothers in New York City. Not unlike Caribbean immigrants, there was a graded effect of age, and women aged 35-39 had the highest odds of preterm birth (*OR* = 1.69, 95% CI: 1.59, 1.79) compared to women aged 20-24. College education had a definite protective effect for African Americans, such that the odds of preterm birth were reduced 21% (*OR* = 0.79, 95% CI: 0.74, 0.84) for college graduates compared to high school graduates.

Unmarried African Americans had an 18% increased odds of preterm birth (*OR* = 1.18, 95% CI: 1.12, 1.23).

Among medically-related risk factors, women who initiated care in the second and third trimesters had an 8% and 24% reduced odds of preterm birth, and women with no/unknown prenatal care had an 80% increased odds compared to women who initiated care in the first trimester. For African American women, maternal medical risks doubled the odds of preterm birth (*OR* = 2.00, 95% CI: 1.94, 2.07) compared to women with no medical risks, while infant risks increased the odds by 41% (*OR* = 1.41, 95% CI: 1.26, 1.59). Smoking and maternal medical risks reduced the protective effect of college education for African American women (Table A.4). While smoking was associated with a 37% increase in preterm birth odds in this group (*OR* = 1.37, 95% CI: 1.28, 1.46), the independent effect of smoking reduced to 21% (*OR* = 1.21, 95% CI: 1.13, 1.29), in consideration of maternal medical risks (Table 4.2).

Combined models. The primary focus for Research Question 1 was to determine the extent to which commonly-measured demographic, behavioral/medical, or infant risk factors can explain the putative immigrant health advantage for V.I.- and Caribbean-born Blacks. I conjectured that demographic factors would be most likely to explain the heath advantage in preterm birth for V.I. and Caribbean women in comparison to African Americans. To test this hypothesis, I examined the odds of preterm birth for each predictor in combined nativity-group models (with African Americans as the reference group) and evaluated the degree to which demographic, medically-related, or infant-specific risk factors modified the reduced odds of preterm birth estimated for nativity status in unadjusted and adjusted models. Thereafter, I examined interactive parameters to determine the degree to which demographic, behavioral/medical, or infant risks had unique associations with preterm birth in V.I.-born and Caribbean-born women relative to U.S.-born women.

As a baseline assessment, I modeled the odds of preterm birth for Virgin Islands- and Caribbean-born populations in New York City relative to African Americans in New York City, adjusted only for parity (Appendix Table B.3). Preterm birth odds were 5% lower for V.I.-born women (*OR* = 0.95, 95% CI 0.79, 1.15) though not statistically significant, and 12% lower for Caribbean-born women (*OR* = 0.88, 95% CI: 0.85, 0.90) compared to U.S.-born women. With hierarchical adjustment, there were no changes greater than 10% in the nativity effect except for the addition of age, which reduced the odds of preterm birth an additional 7% in Caribbean-born women, although the protective effect of age was confounded by medical risks (Table B.3). When all risk factors were considered together, the preterm birth advantage remained about the same as the unadjusted estimates in each immigrant group relative to the U.S.-born mothers ($OR_{VI\text{-}born}$ = 0.98, 95% CI: 0.81, 1.19; $OR_{CA\text{-}born}$ = 0.89, 95% CI: 0.87, 0.92) (Table 4.3, 2nd column).

Interaction parameters were then modeled to determine how risk factors differed uniquely among the index nativity populations compared to the reference U.S. born population (Table 4.3, 3rd column). For Caribbean-born women, there were statistically significant interaction effects demonstrating reduced odds of preterm birth for mothers aged 35-39; mothers with less than 12 years of education; and mothers who initiated prenatal care in the third trimester. The interaction effects were estimated comparing the Caribbean-born population to the U.S.-born populations at the same covariate levels. For example,

$$\frac{OR_{CA\ aged\ 35-39}}{OR_{US\ aged\ 35-39}} = \exp\left(\beta_{1CA\ nativity} + \beta_{3CA\ nativity*aged\ 35-39}\right)$$

$$= e^{-.1628} \times e^{-.1098} = .8498 \times .8960 = .76$$

$$\frac{OR_{CA<12\ years\ edu}}{OR_{US<12\ years\ edu}} = \exp(\beta_{1CA\ nativity} + \beta_{3CA\ nativity*<12\ yr\ edu})$$

$$= e^{-.1628} \times e^{-.1377} = .8498 \times .8714 = .74$$

$$\frac{OR_{CA\ 3rd\ trimester\ PNC}}{OR_{US\ 3rd\ trimester\ PNC}} = \exp(\beta_{1CA\ nativity} + \beta_{3CA*3rd\ trimester\ PNC})$$

$$= e^{-.1628} \times e^{-.133} = .8498 \times .8755 = .74$$

Therefore, for Caribbean immigrants aged 35-39, the odds of preterm birth were 24% lower than the estimated age-related effect that would be predicted for U.S.-born women in New York (*OR* = 0.76, 95% CI: 0.69, 0.84). Whereas college education reduced the odds of preterm birth in the overall sample**,** higher educational attainment did not confer any added advantage in reducing the risk of preterm birth for Caribbean immigrants in New York. In fact, Caribbean-born women with less than 12 years of education had substantially lower odds of preterm birth (*OR* = 0.74, 95% CI: 0.66, 0.83) compared to the lowest-educated African American women, based on the interaction effects. Finally, Caribbean-born women who initiated prenatal care in the 3rd trimester had 26% lower odds of preterm birth (*OR* = 0.74, 95% CI: 0.64, 0.86) compared to African Americans with late prenatal care.

To extend my analysis of interaction effects (and uncover clues to the preterm birth advantage for Black Caribbean women), I conducted likelihood ratio tests to gauge if there was a unique influence of demographic, behavioral/medical, or infant-specific risk factors on estimating the preterm birth advantage for V.I.-born or CA-born mothers relative to U.S.-born mothers. Tables 4.3 and B.4 report *p*-values for the joint significance of the coefficients on the interaction terms for nativity status and demographic factors (i.e., nativity, education, marital status); behavioral and medically-related factors (i.e., prenatal care, smoking, weight gain, maternal medical risks); and infant factors (i.e., congenital anomalies), respectively. The results

indicate that, for Caribbean-born women, demographic factors estimated jointly had the strongest influence on the preterm birth advantage ($p < .001$), driven largely by population differences in age and education. Medically-related risk factors were second in influence ($p < .03$), and infant congenital anomalies did not contribute significantly to the preterm birth advantage for Caribbean women. Smoking had no independent effect on preterm birth for Caribbean-born women in consideration of the other risk factors ($p < .49$).

For V.I.-born women, demographic factors also contributed most to nativity-group distinctions, albeit with marginal significance ($p = .12$). Smoking emerged as the only risk factor ($p = .05$) contributing to the preterm birth advantage for V.I.-born mothers compared to U.S.-born mothers. This finding is driven by the fact there was no reporting smoking among V.I.-born women. However, overall differences in medical or infant factors did not contribute significantly to V.I.-born/U.S-born differences.

Counterfactual analyses. As a final approach to exploring what contributes to the preterm birth advantage for V.I.-born and Caribbean-born immigrants *vis à vis* African American women, I estimated predicted probabilities for preterm birth for African American mothers in New York City conditional on the demographic and maternal health conditions of the V.I.- and Other Caribbean-immigrant populations in New York City. The goal was to estimate the probability of preterm birth in the U.S.-born mothers as if they had the population characteristics of the V.I.-born and Caribbean-born immigrants in New York City. This procedure produces a counterfactual analysis of preterm birth among African Americans relative to Caribbean-immigrant groups—as if everything were equivalent among the groups for all measured factors. Any estimated differences observed between the U.S.- and the immigrant women could then be attributed to unmeasured background factors that were different between the groups.

As shown in Table 4.4, based on the adjusted main effects model in Table 4.3, the predicted probabilities of preterm birth for V.I.-born, Caribbean-born, and U.S.-born non-Hispanic Blacks in New York City were 11.57%, 10.73%, and 12.05% respectively. When the measured characteristics of the V.I. and Caribbean groups were applied to African Americans in New York City, the probabilities of preterm birth among U.S.-born women were reduced to 11.79% and 11.87%, respectively. These figures suggest that 2.21% of the difference in preterm birth rates between U.S.-born and V.I-born women could be explained by *measured* maternal and infant characteristics in the model (comparing 12.05% and 11.79%), and 1.52% of the difference in rates between U.S.-born and Caribbean-born women could be explained by measured characteristics (comparing 12.05% and 11.87%). Correspondingly, *unmeasured* background characteristics accounted for about 2% of difference in preterm birth rates between U.S.-born women and V.I.-born women (comparing 11.79% and 11.57%) and 10% of the difference in rates between U.S.-born women and CA-born women (comparing 11.87% and 10.73%).

Summary of findings: Research Question 1. The foregoing analyses aimed to elucidate factors that contributed most to the preterm birth advantage for Caribbean-born, non-Hispanic Black immigrant mothers in New York City relative to U.S.-born, non-Hispanic Black resident mothers in New York City. In combined-group models, the unadjusted odds ratio for preterm birth for Caribbean women was 0.88 (95% CI: 0.85, 0.90). This ratio remained fairly stable even after adjusting for parity, age, education, marital status, prenatal care, smoking, maternal weight gain during pregnancy, maternal medical risks, infant congenital anomalies, and interactive effects (*OR* 0.85, 95% CI: 0.76, 0.94). Relative to African Americans, there was a unique protective effect for Caribbean immigrant mothers aged 35-39, and some protection was also garnered for Caribbean immigrant mothers with a less than high school education and those who

initiated prenatal care in the third trimester. Although smoking rates were substantially low among Caribbean women, smoking status did not independently confer any added protection against preterm birth relative to African American mothers. Moreover, the health advantage for Caribbean women was not related to maternal medical risk factors combined or to infant congenital anomalies.

Due to the similarity in preterm birth rates between V.I.-born immigrants and African Americans (11.57% vs. 12.05%, respectively), coupled with the disproportionately small population of V.I.-born immigrants in New York, reliable estimates for preterm birth odds could not be produced for most maternal and infant characteristics in V.I-born women relative to African American women. Being a smoker was the only factor to significantly contribute to the slight preterm birth advantage observed for V.I.-born women relative to African Americans. Although smoking was virtually non-existent among V.I.-born mothers (n = 13 smokers), all smokers had a preterm birth and none of the non-smokers delivered preterm.

Marital status demonstrated some conflicting patterns that suggest effect modification for immigrant women. In fully-adjusted combined-group models, unmarried women had 18% overall increased odds of preterm birth relative to married women (Table 4.3). This marital status estimate corresponds in direction and magnitude to the estimates for CA-born and U.S.-born women in stratified analyses (Table 4.2). In contrast, for V.I-born mothers, the bivariate effect (OR = 0.67, 95% CI = 0.45, 1.01, Table B.1); adjusted main effect (OR = 0.80, 95% CI: 0.51, 1.25, Table B.1); and interactive effect (OR = 0.68, p = .09, data not shown) indicate reduced odds of preterm birth for unmarried V.I.-born mothers, although results were not statistically significant.

Among traditional risk factors, combined demographic factors appeared most likely to explain the preterm birth advantage for Caribbean immigrants. Likelihood ratio analyses

confirmed that age and education were the driving factors for the preterm birth advantage for Caribbean-born women. Assessed by the robust interactive effect for age, clues to the preterm birth advantage for Caribbean women in New York appear to be related in part to a unique age-related effect. Specifically, Caribbean-born women 35-39 have lower odds of preterm birth than would be expected in consideration of the relatively high proportion of births to Caribbean-born women in this age range and considering the fact that maternal age >35 is a known risk factor for preterm birth. As suggested by the interaction estimates, some counterintuitive "protective" effects are also noted with respect to education and prenatal care, whereby Caribbean women with *less* than 12 years of education and Caribbean women with *late* prenatal care have lower predicted odds of preterm birth. The lower proportion of composite medical risks among Caribbean women do not contribute significantly to their preterm birth advantage (*OR* = 0.91, 95% CI = 0.81, 1.01, data not shown).

The maintenance of an average 15% reduced odds of preterm birth for Caribbean women across a range of known risks suggests unexplored factors that may yield greater insight into nativity-related perinatal health disparities. Counterfactual analyses confirm that 10% of the difference in preterm birth rates between non-Hispanic Black Caribbean immigrants and African Americans in New York City can, indeed, be attributed to unmeasured background factors unaccounted for in the present analysis.

Research Question 2: Variations by Migrant Status (VI-born and Caribbean-born mothers in the Virgin Islands compared to their migrant counterparts in New York City)

Maternal and Infant Characteristics. Table 4.5 shows maternal and infant characteristics among mothers born in the Virgin Islands (V.I.-born) and those born elsewhere in the Caribbean (CA-born) yet reside in the U.S. Virgin Islands. The V.I -born and CA-born mothers in the Virgin Islands were compared to their migrant counterparts in New York City in

order to explore variations by migrant status and investigate the potential roles of immigrant selectivity and geographic racial context on differential risks of preterm birth.

V.I.-born mothers, V.I. As depicted in Table 4.5, V.I.-born mothers in the Virgin Islands (non-migrants) had a substantially higher proportion of teen births (19.7%) compared with their New York counterparts (9.2%). The proportions switched at older ages such that the percent of births to V.I.-born non-migrant and migrant mothers aged 35 and older was 6.5% and 15.4%, respectively. About 25% of V.I.-born non-migrant women did not complete a high school education compared to 18% among V.I. migrants to New York. The marital rate for V.I. residents (19.4%) was significantly lower than the rate for their migrant counterparts in New York City (26.4%).

Overall, V.I.-born women in New York and in the Virgin Islands tended to receive early prenatal care. Rates of smoking were only 0.42% for V.I-born non-migrants and 1.4% for V.I-born migrants to New York. Medical risks could not be fully ascertained for the population of V.I. resident mothers due to missing medical history data. Among the cases with reported medical risk conditions, the rate of diabetes was lower for V.I.-born non-migrants (2.3%) than migrants to New York (4.3%), although the proportion of maternal medical conditions combined was not significantly different between V.I.-born residents in the V.I. and NY. The proportion of low birthweight was significantly but not vastly different between V.I.-born women in the Virgin Islands (10.2%) and New York (11.4%).

Caribbean-born mothers, V.I. As indicated in Table 4.5, the proportion of births to teens was higher for CA-born mothers living in the Virgin Islands (8.4%) compared to CA-born migrants living in New York (5.8%), and the proportion of births to mothers 35 and older was also higher in the Virgin Islands (20.2%) than in New York (19.0%). CA-born mothers in the V.I. were least educated compared to CA-born mothers in New York. A larger proportion of CA-

born mothers in New York gained excess weight during pregnancy (22%) than their counterparts in the V.I. (15.6%). There were no significant differences in rates of diabetes, total medical conditions, or low birthweight between CA-born intra-regional migrants in the Virgin Islands and CA-born extra-regional migrants to New York.

Preterm birth rates by migrant status. It was hypothesized that preterm birth and associated risk characteristics would be lower for V.I.-born and CA-born mothers in the Virgin Islands compared to their counterparts on the U.S. mainland and that demographic factors will have a larger impact on preterm birth for V.I.-born and Caribbean-born residents of New York than for their native counterparts living in the Virgin Islands. Viewing comparisons by migrant status (Table 4.5), the rate of preterm birth was actually substantially *higher* for V.I.-born mothers in the Virgin Islands (14.9%) than their counterparts in York City (11.6%). Preterm birth rates were also disproportionately high for other CA-born mothers in the Virgin Islands (15.1%) relative to their counterparts in New York City (10.7%). The disparities were largest for the Caribbean-born mothers.

For non-migrant mothers in the Virgin Islands, variations in demographic characteristics did not result in statistically significant differences in preterm birth odds *within* groups (VIVI or CAVI) (Table 4.6). The covariates with the greatest magnitude of effect on predicting preterm birth *within* groups were prenatal care and maternal medical risks. Specifically, VIVI mothers with no/unknown prenatal care were 2.5 times more likely to deliver a preterm infant than women initiating care in the first trimester. Within groups, VIVI and CAVI mothers with documented medical risks had 64% and 127% higher odds of preterm births than mothers with no medical risks (Table 4.6).

With adjustment for the range of risk factors, V.I.-born residents in the Virgin Islands had a 43% increased odds of preterm birth relative to their migrant counterparts in New York City

(*OR* = 1.43, 95% CI: 1.14, 1.79) (Table 4.7), and Caribbean-born residents in the V.I. had 55% increased odds for preterm birth (*OR* = 1.55, 95% CI: 1.34, 1.79) than Caribbean-born migrants in New York City (Table 4.8). Details for each non-migrant/migrant group pair are described next, with attention to the relative contributions of demographic and other covariates on preterm birth differences by migrant status.

V.I.-born, by migrant status. As indicated in Table 4.7, the risk factors under study contributed to about 7% of the disparity by migrant status for V.I.-born mothers, as indicated the change in the unadjusted (i.e., parity only) and adjusted main effects estimates for V.I. vs. NY residence. With consideration of the range of risk factors, V.I.-born non-migrants had a 43% increased odds of preterm birth relative to their migrant counterparts in New York (*OR* = 1.43, 95% CI: 1.14, 1.79) (Table 4.7). There were no statistically significant interactive effects for V.I.-born mothers in the Virgin Islands relative to the migrant counterparts in New York City. Mm, Overall, demographic factors appeared to contribute most to the population disparities, indicated by the significance value (p = .01) for LR tests of the influence exerted by demographic factors relative to medically-related factors for V.I.-born non-migrants compared to migrants in New York (Table 4.7). Age appeared to be the main driver of differential preterm birth risks for V.I.-born women by migrant status, and educational differences were somewhat influential as well (Appendix Table B.10).

CA-born, by migrant status. Table 4.8 displays adjusted models for Caribbean-born mothers in the V.I. and NY with NY residents (extra-regional migrants) as the reference group. Among CA-born mothers, the factors that contributed to the infant health advantage by migrant status were more ambiguous than the findings for V.I.-born mothers. Preterm birth estimates with hierarchical adjustment of individual risk factors showed insignificant changes until there was control for maternal medical risk factors (Appendix Table B.9). CA-born women in the V.I.

with less than 12 years of education had higher than expected odds of preterm birth (*OR* = 2.69, 95% CI: 1.59, 4.55). Age and education were effect modifiers that contributed significantly the final adjusted elevated risk of preterm birth for Caribbean-born non-migrants in comparison to their migrant counterparts (*OR* = 1.84, 95% CI: 1.15, 2.94) (Table 4.8). As hypothesized, demographic factors had more influence than medically-related risks combined in predicting preterm birth differences between Caribbean-born non-migrants and migrants ($p = .03$) (Table 3.8). Age was a leading factor for the differential (Appendix Table B.10).

Counterfactual analyses. Predicted probabilities for preterm birth were estimated for the reference migrant (NY-based) populations as if they had the measured population characteristics of the V.I.-born and Caribbean-born women in the environment of the Virgin Islands. This procedure estimates the extent to which unmeasured factors, presumably correlated with migrant status, were different between the groups. Based on the fully-adjusted models for each nativity group estimated in Tables 3.7 and 3.8, the predicted probabilities of preterm birth for V.I.-born and Caribbean-born migrant women in New York City were 11.57% and 10.73%, respectively. When the measured characteristics of the V.I.-born and CA-born groups living in the Virgin Islands were statistically applied to their migrant counterparts living in New York City, the probabilities of preterm birth among the NY-based migrants were reduced to 9.31% and 10.38%, respectively. Therefore, *measured* factors in the model accounted for almost 20% of the difference in preterm birth rates between V.I-born migrants and natives (comparing 11.57% and 9.31%), and 3% of the difference between CA-born extra-regional migrants to New York and CA-born intra-regional migrants to the Virgin Islands (comparing 10.73% and 10.38%). *Unmeasured* background characteristics were responsible for a substantial 37% of the difference in preterm birth rates between V.I-born migrants and natives (comparing 14.85% and 9.31%) and

31% of the difference in rates between CA-born extra-regional and intra-regional migrants (comparing 15.08% and 10.38%).

Summary of findings: Research Question 2. When V.I.-born and Other CA-born mothers were compared in cross-geographic contexts, preterm birth was substantially higher for mothers living in the Virgin Islands than those living in New York City. Overall, maternal age distributions and educational attainment were more favorable for New York-based migrants whereas smoking rates and prenatal care were better in the Virgin Islands. CA-born women in the Virgin Islands with less than 12 years of education had higher than expected odds of preterm birth compared to their migrant counterparts in New York.

The reasons for the immigrant health advantage for migrant mothers from the Virgin Islands and elsewhere in the Caribbean are not completely clear. For V.I-born women, combined measured factors explained 7% of the difference in preterm birth, comparing unadjusted to adjusted odds ratios (1.33 to 1.43). Virgin Islands mothers are much younger in the Caribbean than in the U.S. When counterfactual approaches were used to equalize the age distributions between V.I.-based and U.S.-based populations (based on the population in the Virgin Islands), about 20% of the difference in preterm birth rates could be explained by measured characteristics and 37% were attributed to unmeasured factors yet unaccounted for in the present paper. For Caribbean-born women, there was a 5% change from unadjusted to adjusted odds ratios for preterm birth (1.48 to 1.55). Although Other CA-born women had more widely disparate preterm birth rates in the V.I. and in the U.S., when the distribution of demographic and other risk factors were equalized between the groups, only 3% of the difference was accounted for by measured factors whereas 31% of the difference was attributable to unmeasured background characteristics.

Overall, migrant non-Hispanic Black V.I-born and other Caribbean-born women in New York are older than their non-migrant counterparts living in the Virgin Islands. However, the suggested interaction of migration and age was not statistically significant when considering both marginal and covarying effects among risk factors ($OR_{\text{CAVI 30-34}} / OR_{\text{CANY 30-34}} = 0.92$, 95% CI: 0.59, 1.43). Also, educational attainment of NY-based migrants did not significantly explain the immigrant health advantage. Over 30% of difference in preterm birth rates between migrant and non-migrant women was attributed to background characteristics not measured in this study.

Discussion

The present study demonstrates a preterm birth advantage for non-Hispanic Black Caribbean immigrants in New York City relative to U.S-born Black women in New York City. This finding corroborates the majority of U.S.-based studies that find healthier low birth weight and preterm birth among foreign-born Blacks relative to African Americans. However, no previous study has employed a direct test of the "healthy migrant effect" in birth outcomes between Black immigrants in the U.S. and counterparts in their countries of origin. Using comparable birth records data from the Virgin Islands as a sample of Caribbean-region births, this study confirmed that non-Hispanic Black women who were born in or reside permanently in the U.S. Virgin Islands have substantially higher demographic and medical risk characteristics coupled with higher preterm birth rates than their migrant counterparts in New York City. This finding validates the "healthy migrant" effect for non-Hispanic Caribbean-born Black mothers in New York City. Notwithstanding, there was no significant nativity effect for the subset of Caribbean women from the U.S. Virgin Islands. Notably, V.I.-born women who migrated to NY, despite having higher educational levels, fared worse than migrants from elsewhere in the Caribbean. In fact, their preterm birth rates approached the high levels for African Americans.

Therefore, viewed by national origin, these findings challenge the generalized assertion of a maternal-infant health advantage for all immigrants from the Caribbean.

I surmised that demographic factors would explain more of the foreign-born/U.S. born differences in birth outcomes than behavioral and especially physical health differences. The idea behind this proposition was that demographic factors were socially patterned and more reflective of racism-related contexts than behavioral or physical health outcomes. Also, health behaviors and medical risks were expected not to be vastly disparate *within* populations of Black women who were all assumed to all have elevated levels of risk due to adverse racial contexts in the U.S. In partial support of my hypothesis, there was somewhat greater variation and contribution of demographic factors than medical factors to the observed perinatal heath advantage for Caribbean-born women relative to African Americans in New York City. However, the overall effect of demographic factors was negligible to low-moderate at best. Notably, there was no protective influence of medical factors for foreign-born women; and, therefore, it would be incorrect to assume that Caribbean-born women have superior physical health status in relation to pregnancy. Unlike the comparisons for Caribbean women and African Americans in New York City, demographic factors were inconsequential in explaining differences by migration status between Caribbean immigrants in New York City and Caribbean residents in the Virgin Islands.

The findings in this study lend support to the healthy migrant hypothesis, but not in the expected manner. Theoretical explanations for the immigrant health advantage preference superior socioeconomic status, lower behavioral risks, and disproportionately favorable health status for migrants which would render them systematically different from the populations in their home countries—and these superior characteristics would presumably contribute to a health advantage in their countries of origin, not to mention in the U.S. (Wingate et al., 2009).

Psychosocial orientations and the maintenance or loss of cultural traditions is also offered as reasons for differential health among U.S.-based immigrants (Esses, Deaux, Lalonde, & Brown, 2010; Schwartz, Unger, Zamboanga, & Szapocznik, 2010; Zambrana, Dunkel-Schetter, Collins, & Scrimshaw, 1999). In this study, the descriptive analysis of demographic, behavioral, and medical risk factors indicated a higher percentage of teen births, lower educational attainment, and lower marital rates among V.I.-born and Other Caribbean-born women in the Virgin Islands (relative to their non-migrant counterparts) which suggested added risks for non-migrant women. However, prenatal care and smoking behaviors were superior and LBW rates and abnormal newborn conditions were lower among women in the Virgin Islands. With the exception of hypertension, documented medical risk factors were not higher among these non-migrant women. Moreover, inferential statistics pointed primarily to age for both V.I.-born and Caribbean-born women, and to a lesser extent education for Caribbean-born women, as the main source of the immigrants' health advantage relative to African Americans. Medically-related factors did not drive the differentials.

As a clue to why migrant women in the U.S. had lower rates of preterm birth than their non-migrant counterparts in the Caribbean, educational attainment, age, and education played marginally influential roles, but not factors such as prenatal care or medical risks. Based on counterfactual analyses, there was still up to 10% unexplained risk between V.I.- and Caribbean-born non-Hispanic Blacks and U.S.-born non-Hispanic Blacks attributable to unmeasured, background factors that varied by nativity. Between Caribbean migrants and non-migrants, the unaccounted variance in risk was lower, but still approached 10%. I postulated that a portion of this difference is encapsulated in some component of ethnic status that has not yet been sufficiently ascertained—and pointed to exposures and responses to racism or racial context as possible contributors.

In this study, higher educational attainment did not confer the expected health advantages for V.I-born and Caribbean-born women as it did for African Americans. However, this study shares limitations that are common with the use of standard birth records. Although education is used almost universally in perinatal health research to represent SES level, education as reported in birth records is a poor approximation for socioeconomic position, and its effect varies considerably by race and ethnicity (Blumenshine, Egerter, Barclay, Cubbin, & Braveman, 2010; Braveman et al., 2014). Using education as an SES measure is particularly problematic for Black and immigrant women who are the focus of this study. Education does not have the expected gradient effect in Black women that would confer perinatal health advantages at higher levels (Nepomnyaschy, 2009). Black women with higher education are found to have no substantive protection from adverse birth outcomes or they can actually fare worse than lower-educated Black women (Collins & David, 1990; Din-Dzietham & Hertz-Picciotto, 1998). The use of education as an SES measure in this study may also reflect measurement bias because education levels are qualitatively different in the Caribbean and the U.S. In many West Indian Islands, comprehensive high school content can be completed by the equivalent of the 10th grade in the U.S. Therefore, completing less than 12 years of education would not be considered an educational deficit for Caribbean-born women.

Other potential explanatory variables that were excluded from the present analysis are neighborhood factors. Linking neighborhood economic data with maternal address information is a technique commonly employed in perinatal health studies. For the present study, I was not able to incorporate data on neighborhood income or other census-related factors. Although zip code information was available with the NYC data, FIPS codes (i.e., standard geographic identifiers) were not added to the records until 2008. Moreover, the census tract codes provided with the records were in an incorrect format such that I was unable to validly match residence information

for the birth mothers. Ecologic linkages also were not possible with the Virgin Islands' data due to suppression of geographic identifiers due to the small populations and need to maintain confidentiality. Another contextual feature not assessed in this study was ethnic density. Ethnic density could theoretically contribute to the salutatory effect for Black immigrants due to enhanced social networks, especially in an ethnically-rich area such as New York City. However, unlike the supportive findings usually reported for Hispanic immigrants, previous studies examining adverse birth outcomes have not observed a protective effect of ethnic density for Black immigrants (Mason et al., 2010).

In this descriptive study, I examined an outcome for which there is little variance within the race-exclusive population of Black women that I have selected to study. However, the preterm birth rates between U.S.-based native and immigrant women in this paper mirror a recent national study of Black nativity differences in preterm birth rates which reported a 1.4% difference between non-Hispanic U.S.-born Blacks and Caribbean-born Blacks (PTB rates 12.4% and 11%, respectively) (Elo et al., 2014). The same study highlighted the variance among foreign-born Blacks in that the PTB rate for African immigrants was 7.3%. The stark variation between immigrant Blacks should compel researchers to minimally separate results for African and Caribbean immigrants, as there is enough evidence to suggest a generalized trend of worse outcomes for Caribbean immigrants in the U.S. The present study affirms the heterogeneity of perinatal risks and outcomes by national origin among Blacks, and it provides previously unreported information by nativity and migrant status for birth mothers on the U.S. mainland and in the Virgin Islands.

The notably higher proportion of diabetes and macrosomic babies among foreign-born Blacks warrants further investigation. Despite the inherent medical risks from these conditions, infant health outcomes remain more favorable for foreign-born Blacks. Other data sources such as

the National Health Interview Survey can be investigated to verify if there are comparably higher rates of diabetes and/or overweight and obesity in the Black immigrant population relative to their native African American counterparts. Observing higher rates of Type 2 diabetes or overweight/obesity in immigrant Blacks would question the oft-cited claim of better nutrition among FB Blacks.

It is acknowledged that the observed differences in this study, while statistically significant, are of small magnitude in most cases—on the order of a few percentage points in the relative odds of the outcome by nativity. The observation of nativity-related effects is even more challenging to justify as substantive in light of the limited range of explanatory factors afforded by the birth data. With the exception of age, the nativity effect was stable across most measured predictors. Collectively, the examined predictors explained about 10% of the variance in odds in preterm birth between African American and Caribbean women in New York. And the overall effects of most predictors were eclipsed in consideration of infant risks. The influence of co-morbid neonatal health risks on the odds of preterm birth is entirely predictable, and I could have modified the study to focus exclusively on maternal components of risk.

Also, my approaches may not have been optimal for specifying what exactly *explains* the foreign-born advantage. My methods could have been aided by more statistical techniques that may have been more applicable, including alternatives to logistic regression such as structural equation modeling (SEM). A desired improvement in technique would have been to employ decomposition methods such as the Blinder–Oaxaca procedure to separate effects based on the distribution of individual characteristics by nativity in the populations and true nativity-related effects. Logistic regression estimates effects with an assumption that the factors operate the same in all groups. Although interaction analyses afford the ability to estimate unique effects, it is challenging to fully comprehend or explain interactive effects in a logistic regression framework

among the numerous covariates and even more daunting covariate categories analyzed in this study. The Blinder–Oaxaca technique has the advantage of simultaneously estimating different models for each group and fully decomposing covariate effects and reporting results as probabilities. While the Stata program has a package that can automate the Blinder–Oaxaca procedure, the SAS program cannot duplicate this method, and hence I could not reasonably attempt this method for this paper. Moreover, these decomposition techniques are used rarely, if at all, in perinatal health studies and in the field of public health in general. Future opportunities exist to employ SEM or formal decomposition methods to this line of research and potentially offer unique insights regarding racial and nativity-status disparities in birth outcomes.

In this study, no *statistically* significant differences were found between Virgin Islands women in New York and African American women, or between Virgin Islands women and the other Caribbean-born women in New York. The sample of Virgin Islands women in New York was inadequately powered to detect any effects at the desired level of confidence. It may have been statistically advantageous to balance sample sizes via random selection and reduction of cases from the large New York-based African American and Caribbean immigrant samples to more closely match each other and the much smaller V.I.-born samples.

Furthermore, because the nature of this study was to quantify the relative influence of maternal and infant risk characteristics between groups using multiple logistic regression, it was essential that identical sets of covariates were estimated across all groups. Accordingly, if any *one* covariate value of interest was missing from a case, the entire record was deleted from the model estimations. While the New-York based records are superior in their level of completeness, the Virgin Islands records were problematic, particularly in the documentation of maternal and infant medical risk factors where the level of missing values was substantial. About five percent of cases ($n = 237$) were deleted due to missing values. The level of case deletions

required was undesirable in a population sample that was already small. Sensitivity analyses revealed that preterm birth was slightly higher among the cases with missing covariate values (15.17%) compared to the working dataset with missing values removed (14.85%). The working dataset was also differentiated with slightly higher proportions of college graduates, earlier prenatal care initiation, and a slightly lower proportion of maternal medical risks. Multiple imputation methods could be employed to substitute medical risks based on case matching so as to avoid case deletions for future studies. An advantage to techniques like SEM or the Blinder-Oaxaca decomposition is that they may be more liberal in accommodating cases with missing values.

The range of factors provided by birth data is insufficient for empirical testing of context factors. However, this study afforded an opportunity to extend empirically-based hypotheses to speculate about differential birth outcomes by nativity based on unmeasured contextual factors related to exposure and adaptation to a racialized U.S. social context. Accordingly, Caribbean migrants and V.I. migrants in New York were expected to have more favorable health outcomes then their U.S.-born counterparts. Although supportive findings along these lines are not surprising due to well-established evidence about the health advantage of U.S. immigrants relative to U.S. natives, what is surprising is that this advantage was sustained even relative their native counterparts still residing in the Caribbean, utilizing the Virgin Islands as a test case. Immigrant selectivity appeared evident for non-Hispanic Black women who migrated from the Caribbean. Regardless of this unexpected finding, the third and primary extension of my hypothesis was that the primary determinants of the relative advantage for Caribbean migrants compared to African Americans, as well as V.I.-based residents compared to their counterparts in NYC would *not* be related to medically-based factors. This proposition was upheld, and demographic factors, particularly age and to a lesser extent education drove the nativity and

migrant status differences. It was also expected that place of residence would still prove substantial amidst other explanatory factors. However, my data were not specified to test the effect of residence. Because the groups were coded according to their place of residence (i.e., New-York or the Virgin Islands), the residence variable became fully linear with nativity status and it could not be modeled. Other placed-based features would have needed to be specified in order to separate geographic factors from nativity effects.

Women in the Virgin Islands who were born in another Caribbean island are technically intra-regional migrants (i.e., migrants within the Caribbean). Their migrant status as well as the reasons prompting their migration could make them demographically or culturally different from native-born Virgin Islanders in ways that can differentiate health status. Comparisons between Caribbean-born women (non-V.I. born) in the Virgin Islands and Caribbean immigrants (non V.I. born) in New York preclude true migrant/non-migrant contrasts. Consistent with healthy migrant theories, it is possible that migrant status confers psychological or physical hardiness that result in health advantages and, therefore, V.I-born women in NY as well as CA-born women in both the V.I. and NY would be subject to immigrant selectivity effects. Alternatively, the similarity in socioeconomic status and pan-Caribbean identity shared by non-Hispanic Black Virgin Islands residents, regardless of Caribbean birthplace, may render native-born Virgin Islanders and Caribbean-born women in the Virgin Islands similar enough to characterize as an aggregate group for monitoring population health. In this study, CA-born women in the Virgin Islands did have a better pregnancy risk profile than native-born Virgin Islanders. Moreover, the maternal and infant health characteristics were not as disparate between CA-born women in the V.I. and NY as they were between V.I.-born women in the V.I. and NY. These observations suggest that immigrant selectivity plays a role for these populations.

A critical moderator not assessed in this study is duration of residence for immigrants, which can qualify the meaning and effect of nativity-status. One can be born in the U.S., yet be a second generation immigrant, and this status can produce qualitatively different social contexts for this index mother vs. a U.S.-born mother with American ancestry. In the Virgin Islands context, if a woman was born elsewhere in the Caribbean, yet came to the Virgin Islands as an infant or child, her acculturation experiences and environmental exposures would not be that different from the experiences of native-born Virgin Islanders. Moreover, a large proportion of native-born Virgin Islanders have Caribbean ancestry—that is, their parents were born in another Caribbean island. Therefore, many mothers who state their nativity as "V.I.-born" are second generation Caribbean immigrants. Unlike child immigrants, if the "foreign-born" woman came to the Virgin Islands in her late teens or as an adult, her socialization and acculturation experiences would more readily mirror her status as "immigrant," and it is more likely that her differential health outcomes could be attributable to a healthy migrant effect. Also important to the present analysis would be data on inter-regional travel between the U.S. and the Caribbean, and residence or pregnancy/birth in both places which would influence social/racial context exposures. Without data on parental ancestry, date(s) of migration, or "push" or "pull" factors for migration, the reasons for differences in maternal-infant health by nativity in the U.S. and the V.I. are largely speculative.

It is of note that the assessment of non-Hispanic Caribbean-born Black women in the Virgin Islands is subject to measurement error because I was not able to extract Black women from the Dominican Republic in that sample. Nonetheless, this subsample is estimated to be small based on census data reporting Dominicans, all races and both sexes, as 12% of the foreign-born population in the Virgin Islands (U.S. Census Bureau, 2010). Lastly, it was assumed that due to ethnicity, culture, and sociopolitical history, Hispanic Blacks would have

distinct sociodemographic influences which would make their background and health contexts incomparable with other Black Caribbean women. Evidence of this is indicated by studies which demonstrate better perinatal outcomes for Black Hispanics than Blacks from the English-speaking Caribbean. The present study also did not include African-born Blacks because of the special interest in migrant comparisons and the inability to analyze natality information from African countries.

The data in this study provide information that is not readily available for researchers interested in the maternal-infant health of Black immigrants. Additional analyses of maternal and infant heath among foreign-born Blacks would be to compare past and current trends in natality data for the foreign-born population, both nationally and disaggregated by U.S. region. New York City provides valuable regional information for that locale, but regional data can also be extracted from migrant enclaves in cities like Miami, Chicago, Atlanta and others. Census data can be explored in more detail to document demographic information on the foreign-born Black population in these areas. Although Caribbean populations would be most numerous, opportunities exist to delineate information for African-born populations as well.

Public health investigations are rife with inter-racial comparisons to evaluate the state of health in populations. In the case of African Americans or Blacks, the barometer is almost exclusively how these populations compare to Whites. The result of these normative associations is a perpetual focus on inter-racial disparities which detracts from health patterns and improvements within populations that may be noteworthy in their own right. Although investigators may claim that relative health comparisons are the most valuable for scientific interest, expediency (due to data availability), and fostering public health improvement as a whole, the basis of such arguments are grounded in philosophical—not scientific—orientations with somewhat arbitrary justifications. It may be beneficial in the interest of the communities

being studied to highlight in-group risk associations, especially with regard to ameliorative factors that may contribute to absolute health improvements. Minimally, the comparative lens should extend to racially- and ethnically-similar populations and communities such that the background effects are more fixed and the estimated effects would be a more valid representation of a population's baseline health risks and advantages.

Tables

Table 4.1 Maternal and Infant Characteristics, by Maternal Nativity, non-Hispanic Blacks, Singleton Births, New York City, 2000-2009

	V.I.-born, NY (*n* = 1,080)	Caribbean-born, NY (*n* = 81,615)	*p* value[a]	U.S.-born, NY (*n* = 141,857)	*p* value[b]
	%	%		%	
Maternal Characteristics					
Age, years					
15-19	9.2	5.8	**<.0001**	16.4	**<.0001**
20-24	23.3	21.0		31.0	
25-29	23.7	27.8		25.0	
30-34	25.7	26.4		17.6	
35-39	15.4	19.0		10.0	
Education, years					
<12	17.9	15.7	**<.0001**	27.9	**<.0001**
12	32.2	40.6		32.1	
13-15	31.6	28.8		28.5	
16+	18.3	14.8		11.5	
Married	26.4	39.2	**<.0001**	19.2	**<.0001**
Prenatal care initiation					
1st trimester	62.8	59.4	**.013**	60.7	**<.0001**
2nd trimester	22.9	24.4		25.3	
3rd trimester	7.7	10.3		6.5	
No prenatal care	1.0	0.6		1.7	
Unknown prenatal care	5.7	5.4		5.7	
Parity					
Primiparous, 1	41.6	44.4	.068	46.3	**<.0001**
Multiparous, 2 or more	58.4	55.7		53.7	
Smoked during pregnancy	1.2	0.6	**.007**	5.0	**<.0001**
Weight gain during pregnancy					
< 16 lb	14.5	13.1	.401	14.0	**<.0001**
≥ 41 lb	21.7	22.1		24.1	
Diabetes (chronic or gest.)	4.3	5.5	.075	3.5	**<.0001**
Hypertensive disorders	4.3	4.1	.790	4.6	**<.0001**
Maternal medical risks	28.6	30.5	.172	32.6	**<.0001**
Infant Characteristics					
Abnormal newborn conditions	6.1	6.6	.564	8.5	**<.0001**
Congenital anomalies	1.4	1.5	.718	1.4	.187
Low birthweight (< 2500 g)	11.4	9.3	**.020**	11.5	**<.0001**
Preterm birth (< 37 weeks)	11.6	10.7	.374	12.1	**<.0001**

[a] *p* value refers to the χ^2 test for differences between VI- and CA-born.
[b] *p* value refers to the χ^2 test for differences between VI-, CA-, and US-born.

Table 4.2 Predictors of Preterm Birth, Stratified by Maternal Nativity, non-Hispanic Blacks, New York City, 2000-2009

	V.I.-born, NY		CA-born, NY		U.S.-born, NY	
	Within-Group Estimates	Influence of Risk Factors[a]	Within-Group Estimates	Influence of Risk Factors[a]	Within-Group Estimates	Influence of Risk Factors[a]
	OR 95% CI	*p* value	*OR* 95% CI	*p* value	*OR* 95% CI	*p* value
Primiparous, 1	1.11 (0.72, 1.73)		**1.21** (1.15, 1.27)		1.01 (0.97, 1.05)	
Multiparous, 2 or more (ref.)	1.0		1.0		1.0	
Aged 15-19 years	0.75 (0.28, 2.02)	Demographic	1.09 (0.98, 1.22)	Demographic	1.01 (0.95, 1.06)	Demographic
Aged 25-29 years	1.61 (0.88, 2.95)	**.034**	**1.12** (1.04, 1.20)	**<.001**	**1.15** (1.09, 1.20)	**<.001**
Aged 30-34 years	1.58 (0.84, 2.97)		**1.30** (1.21, 1.40)		**1.35** (1.28, 1.42)	
Aged 35-39 years	**2.53** (1.30, 4.91)		**1.51** (1.40, 1.63)		**1.69** (1.59, 1.79)	
Aged 20-24 years (ref.)	1.0		1.0		1.0	
< 12 yrs education	1.68 (0.93, 3.05)		0.95 (0.89, 1.02)		**1.09** (1.05, 1.14)	
13-15 yrs education	1.58 (0.96, 2.59)		**0.93** (0.88, 0.98)		**0.93** (0.89, 0.97)	
16+ yrs education	1.01 (0.54, 1.90)		**0.92** (0.86, 0.99)		**0.79** (0.74, 0.84)	
12 yrs education (ref.)	1.0		1.0		1.0	
Unmarried	0.80 (0.51, 1.25)		**1.12** (1.07, 1.18)		**1.18** (1.12, 1.23)	
Married (ref.)	1.0		1.0		1.0	
2nd trimester prenatal care	0.93 (0.57, 1.51)	Medically-	**0.91** (0.86, 0.96)	Medically-	**0.92** (0.88, 0.95)	Medically-
3rd trimester prenatal care	0.47 (0.18, 1.26)	related	**0.66** (0.61, 0.72)	related	**0.76** (0.71, 0.82)	related
None/unknown prenatal care	**2.72** (1.46, 5.05)	**<.001**	**1.72** (1.58, 1.86)	**<.001**	**1.80** (1.71, 1.90)	**<.001**
1st trimester prenatal care (ref.)	1.0		1.0		1.0	
Smoked during pregnancy	0.00 (0, ∞)		**1.35** (1.04, 1.76)		**1.21** (1.13, 1.29)	
Did not smoke (ref.)	1.0		1.0		1.0	
Weight gain <16 lb	1.11 (0.65, 1.88)		**1.78** (1.68, 1.89)		**1.67** (1.61, 1.75)	
Weight gain ≤41 lb	0.66 (0.38, 1.13)		**0.77** (0.73, 0.82)		**0.74** (0.71, 0.77)	
Weight gain 16-40 lb (ref.)	1.0		1.0		1.0	
Maternal medical risks	**2.11** (1.42, 3.14)		**2.14** (2.05, 2.24)		**2.00** (1.94, 2.07)	
No maternal medical risks (ref.)	1.0		1.0		1.0	
Infant congenital anomalies	2.71 (0.79, 9.22)	Infant specific	**1.58** (1.36, 1.84)	Infant specific	**1.41** (1.26, 1.59)	Infant specific
No congenital anomalies (ref.)	1.0	.100	1.0	**<.001**	1.0	**<.001**

Note: Significant terms are bolded.

[a]The *p* -value indicates the contribution of risk factors to the model based on likelihood ratio tests comparing models with and without the designated risk factors.

Table 4.3 Predictors of Preterm Birth, by Mother's Nativity, Inclusive of Interaction Effects, V.I.-born and Caribbean-born Compared to U.S.-born, non-Hispanic Blacks, New York City, 2000-2009

VI, CA, US combined (NYC)	Unadjusted	Adjusted Model (main effects)	Adjusted Model (with interactions)	Nativity Interactions[a]	
				V.I.-born	CA-born
	OR 95% CI	*OR* 95% CI	*OR* 95% CI	*p* -value	*p* -value
Virgin Islands-born	0.96 (0.79, 1.15)	0.98 (0.81, 1.19)	0.79 (0.37, 1.71)		
Caribbean-born	**0.88** (0.85, 0.90)	**0.89** (0.87, 0.92)	**0.85** (0.76, 0.94)		
US-born (ref.)	1.0	1.0	1.00		
Primiparous, 1	**0.91** (0.88, 0.93)	**1.08** (1.04, 1.11)	**1.01** (0.97, 1.05)		
Multiparous, 2 or more (ref.)	1.0	1.0	1.00		
Aged 15-19 years	**1.08** (1.03, 1.13)	1.01 (0.96, 1.06)	1.01 (0.95, 1.06)	Demographic	Demographic
Aged 25-29 years	**1.08** (1.04, 1.12)	**1.15** (1.10, 1.19)	**1.15** (1.09, 1.20)	.124	**<.001**
Aged 30-34 years	**1.23** (1.19, 1.28)	**1.33** (1.28, 1.39)	**1.35** (1.28, 1.42)		
Aged 35-39 years	**1.53** (1.47, 1.60)	**1.60** (1.53, 1.68)	**1.69** (1.59, 1.79)		
Aged 20-24 years (ref.)	1.0	1.0	1.00		
< 12 yrs education	**1.09** (1.05, 1.12)	**1.06** (1.02, 1.10)	**1.09** (1.05, 1.14)		
13-15 yrs education	**0.92** (0.89, 0.95)	**0.93** (0.90, 0.97)	**0.93** (0.89, 0.97)		
16+ yrs education	**0.85** (0.81, 0.89)	**0.85** (0.81, 0.89)	**0.79** (0.74, 0.84)		
12 yrs education (ref.)	1.0	1.0	1.00		
Unmarried	**1.15** (1.11, 1.18)	**1.16** (1.12, 1.20)	**1.18** (1.12, 1.23)		
Married (ref.)	1.0	1.0	1.00		
2nd trimester prenatal care	**0.94** (0.91, 0.97)	**0.91** (0.89, 0.94)	**0.91** (0.88, 0.95)	Medical/	Medical/
3rd trimester prenatal care	**0.77** (0.73, 0.81)	**0.72** (0.68, 0.76)	**0.76** (0.71, 0.82)	Behavioral	Behavioral
None/unknown prenatal care	**1.87** (1.79, 1.96)	**1.79** (1.71, 1.87)	**1.80** (1.71, 1.90)	.246	**.026**
1st trimester prenatal care (ref.)	1.0	1.0	1.00		
Smoked during pregnancy	**1.63** (1.54, 1.72)	**1.25** (1.17, 1.33)	**1.21** (1.13, 1.29)		
Did not smoke (ref.)	1.0	1.0	1.00		
Weight gain <16 lb	**1.82** (1.76, 1.88)	**1.71** (1.65, 1.77)	**1.67** (1.60, 1.74)		
Weight gain ≤41 lb	**0.78** (0.75, 0.80)	**0.75** (0.72, 0.77)	**0.74** (0.71, 0.77)		
Weight gain 16-40 lb (ref.)	1.0	1.0	1.00		
Maternal medical risks	**2.12** (2.06, 2.17)	**2.05** (2.00, 2.11)	**2.00** (1.94, 2.07)		
No maternal medical risks (ref.)	1.0	1.0	1.00		
Infant congenital anomalies	**1.61** (1.47, 1.77)	**1.48** (1.35, 1.62)	**1.41** (1.25, 1.59)	Infant	Infant
No congenital anomalies (ref.)	1.0	1.0	1.00	.315	.229
Significant marginal interactions:					
CA-born * aged 35-39			**0.90**[b] (0.81, 0.99)		
CA-born * < 12 years education			**0.87**[b] (0.80, 0.94)		
CA-born * 3rd trimester prenatal care			**0.88**[b] (0.78, 0.98)		

Note: Significant terms are bolded.

[a]Interaction effects are indicated by *p* -values for the significance of V.I. or CA nativity interacting with combined risk factor categories.

[b]The OR_{CA}/OR_{US} estimates based on these interaction parameters are as follows: Aged 35-39: OR_{CA}/OR_{US} = **0.76** (0.69, 0.84); <12 yrs education: OR_{CA}/OR_{US} = **0.74** (0.66, 0.83); 3rd trimester prenatal care initiation: OR_{CA}/OR_{US} = **0.74** (0.64, 0.86)

Table 4.4 Actual and Counterfactual Probabilities of Preterm Birth, by Maternal Nativity, non-Hispanic Blacks, New York City, 2000-2009

	V.I.-born, NY	Caribbean-born, NY	U.S.-born, NY
	A	B	C
Actual predicted probability	11.57%	10.73%	12.05%
Counterfactual predicted probability[a] for U.S. born based on V.I. population characteristics	—	—	11.79%
Counterfactual predicted probability[a] for U.S.-born based on CA population characteristics	—	—	11.87%

Note: Unmeasured factors are indicated by group percentage change differences between columns A and C, or B and C.
Measured factors are indicated by group percentage change differences in column C.
[a]Conditional on parity, age, education, marital status, prenatal care, smoking, weight gain, maternal medical risks, and congenital anomalies.

Table 4.5 Maternal and Infant Characteristics by Nativity and Migrant Status, V.I.-born and Caribbean-born Mothers, non-Hispanic Blacks, Virgin Islands Births (2000-2004) and New York City Births (2000-2009), Singleton Deliveries

	Virgin Islands-born			Caribbean-born		
	Non-Migrants[a] (VIVI) *n* = 2,883	**Migrants[b] (VINY)** *n* = 1,080	*p* value[c]	**Non-Migrants[a] (CAVI)** *n* = 1,459	**Migrants[b] (CANY)** *n* = 81,615	*p* value[c]
	%	%		%	%	
Maternal Characteristics						
Age, years						
15-19	19.7	9.2	**<.0001**	8.4	5.8	**<.0001**
20-24	32.9	23.3		19.7	21.0	
25-29	24.8	23.7		25.0	27.8	
30-34	16.2	25.7		26.7	26.4	
35-39	6.5	15.4		20.2	19.0	
Education, years						
<12	24.8	17.9	**<.0001**	29.3	15.7	**<.0001**
12	41.2	32.2		45.2	40.6	
13-15	20.4	31.6		14.5	28.8	
16+	13.6	18.3		11.0	14.8	
Married	19.4	26.4	**<.0001**	38.6	39.2	**<.0001**
Prenatal care initiation						
1st trimester	64.5	62.8	**<.0001**	62.0	59.4	**<.0001**
2nd trimester	26.0	22.9		28.7	24.4	
3rd trimester	5.9	7.7		6.7	10.3	
No prenatal care	3.2	1.0		2.6	0.6	
Unknown prenatal care	0.3	5.7		0.0	5.4	
Parity						
Primiparous, 1	38.2	41.6	**.052**	31.0	44.4	**<.0001**
Multiparous, 2 or more	61.8	58.4		69.0	55.7	
Smoked during pregnancy	0.4	1.2	**.005**	0.3	0.6	.132
Weight gain during pregnancy						
< 16 lb	17.9	14.5	**<.0001**	17.5	13.1	**<.0001**
≥ 41 lb	15.7	21.7		15.6	22.1	
Diabetes (chronic or gest.)	3.0	4.3	**.046**	4.4	5.5	**.064**
Hypertensive disorders	7.0	4.3	**.001**	6.6	4.1	**<.0001**
Medical risk factors	26.9	28.6	.277	28.2	30.5	**.052**
Infant Characteristics						
Abnormal newborn conditions	2.2	6.1	**<.0001**	2.3	6.6	**<.0001**
Congenital anomalies	1.0	1.4	.256	1.3	1.5	.574
Low birthweight (<2500 g)	10.2	11.4	.276	8.5	9.3	.294
Preterm birth (<37 weeks)	14.9	11.6	**.008**	15.1	10.7	**<.0001**

[a]Non-migrants are VI-born or Caribbean-born women who are natives/residents of the Virgin Islands and whose index birth was in the Virgin Islands.
[b]Migrants are VI-born or Caribbean-born women who are migrants in New York City and whose index birth was in New York City.
[c]*p* values refer to the χ^2 test for differences within each nativity group by migrant status.

Table 4.6 Predictors of Preterm Birth, Stratified by Maternal Nativity, non-Hispanic Blacks, U.S. Virgin Islands, 2000-2004

	V.I.-born, V.I. (VIVI)		CA-born, V.I. (CAVI)	
	Within-Group Estimates	Contribution of Predictors[a]	Within-Group Estimates	Contribution of Predictors[a]
	OR 95% CI	*p* value	*OR* 95% CI	*p* value
Primiparous, 1	1.21 (0.94, 1.55)		1.10 (0.77, 1.57)	
Multiparous, 2 or more (ref.)	1.0		1.0	
Aged 15-19 years	1.14 (0.82, 1.57)	Demographic	1.19 (0.67, 2.10)	Demographic
Aged 25-29 years	1.27 (0.95, 1.70)	.377	0.77 (0.48, 1.22)	.116
Aged 30-34 years	1.01 (0.70, 1.44)		0.64 (0.40, 1.04)	
Aged 35-39 years	0.97 (0.59, 1.58)		1.04 (0.64, 1.71)	
Aged 20-24 years (ref.)	1.0		1.0	
< 12 yrs education	1.13 (0.87, 1.49)		1.41 (0.99, 2.00)	
13-15 yrs education	0.79 (0.59, 1.07)		1.41 (0.90, 2.19)	
16+ yrs education	0.95 (0.66, 1.35)		1.03 (0.59, 1.78)	
12 yrs education (ref.)	1.0		1.0	
Unmarried	0.97 (0.72, 1.31)		0.94 (0.67, 1.31)	
Married (ref.)	1.0		1.0	
2nd trimester prenatal care	1.09 (0.85, 1.40)	Medically-related	1.00 (0.71, 1.41)	Medically-related
3rd trimester prenatal care	1.02 (0.65, 1.60)	**<.001**	1.19 (0.67, 2.10)	**<.001**
None/unknown prenatal care	**2.46** (1.54, 3.91)		1.33 (0.59, 3.00)	
1st trimester prenatal care (ref.)	1.0		1.0	
Smoked during pregnancy	1.23 (0.26, 5.73)		1.82 (0.18, 18.28)	
Did not smoke (ref.)	1.0		1.0	
Maternal medical risks	**1.64** (1.32, 2.05)		**2.27** (1.68, 3.08)	
No maternal medical risks (ref.)	1.0		1.0	

Note: Significant terms are bolded.

[a]The *p* -value indicates the within-group contribution of risk factors to preterm birth in each population based on the likelihood ratio tes comparing models with and without the designated risk factors.

Table 4.7 Predictors of Preterm Birth for V.I.-born Mothers, by Migrant Status, V.I.-born Residents in the Virgin Islands (2000-2004) and V.I.-born Immigrants in New York City (2000-2009)

V.I.-born, by Migrant Status	Unadjusted	Adjusted Model (main effects)	Adjusted Model (with interactions)	Interaction Effects VIVI[a]
	OR 95% CI	*OR* 95% CI	*OR* 95% CI	*p* value
V.I.-born, V.I. Resident (VIVI)	**1.33** (1.08, 1.65)	**1.43** (1.14, 1.79)	2.09 (0.90, 4.83)	
V.I.-born, NY Resident (VINY) (ref.)	1.0	1.0	1.0	
Primiparous, 1	1.07 (0.89, 1.29)	1.18 (0.95, 1.46)	1.13 (0.73, 1.75)	
Multiparous, 2 or more (ref.)		1.0	1.0	
Aged 15-19 years	1.30 (1.00, 1.70)	1.14 (0.84, 1.53)	0.70 (0.26, 1.89)	Demographic
Aged 25-29 years	1.20 (0.94, 1.53)	**1.32** (1.02, 1.71)	1.60 (0.87, 2.92)	**.010**
Aged 30-34 years	0.98 (0.74, 1.29)	1.12 (0.83, 1.52)	1.60 (0.86, 3.01)	
Aged 35-39 years	1.25 (0.89, 1.75)	1.43 (0.99, 2.06)	**2.56** (1.32, 4.97)	
Aged 20-24 years (ref.)	1.0	1.0	1.0	
< 12 yrs education	1.23 (0.98, 1.54)	1.22 (0.95, 1.56)	1.69 (0.93, 3.06)	
13-15 yrs education	0.94 (0.74, 1.20)	0.97 (0.76, 1.25)	1.58 (0.96, 2.59)	
16+ yrs education	0.95 (0.72, 1.26)	0.92 (0.68, 1.25)	1.00 (0.53, 1.87)	
12 yrs education (ref.)	1.0	1.0	1.0	
Unmarried	1.00 (0.80, 1.24)	0.89 (0.70, 1.14)	0.75 (0.49, 1.17)	
Married (ref.)	1.0	1.0	1.0	
2nd trimester prenatal care	1.08 (0.87, 1.33)	1.09 (0.88, 1.35)	0.94 (0.58, 1.52)	Behavioral/Medical
3rd trimester prenatal care	0.92 (0.62, 1.37)	0.90 (0.60, 1.35)	0.52 (0.20, 1.35)	.572
None/unknown prenatal care	**2.54** (1.78, 3.61)	**2.56** (1.77, 3.69)	**2.68** (1.45, 4.98)	
1st trimester prenatal care (ref.)	1.0	1.0	1.0	
Smoked during pregnancy	0.54 (0.13, 2.27)	0.57 (0.13, 2.47)	0.00 (0, ∞)	
Did not smoke (ref.)	1.0	1.0	1.0	
Maternal medical risks	**1.76** (1.46, 2.13)	**1.72** (1.43, 2.09)	**2.09** (1.41, 3.10)	
No maternal medical risks (ref.)	1.0	1.0	1.0	

Note: Significant terms are bolded.

[a]Interaction efffects are indicated by *p* -values for the interactions of V.I. residence with demographic or medical risk factors in a fully-adjusted model. There were no statistically significant migrant group interactions with individual predictors.

Table 4.8 Predictors of Preterm Birth for Caribbean-born Mothers, by Migrant Status, CA-born Residents in the Virgin Islands (2000-2004) and CA-born Immigrants in New York City (2000-2009)

CA-born, by Migrant Status	Unadjusted	Adjusted Model (main effects)	Adjusted Model (with interactions)	Interaction Effects CAVI[a]
	OR 95% CI	*OR* 95% CI	*OR* 95% CI	*p* value
CA-born, V.I. Resident (CAVI)	**1.48** (1.28, 1.71)	**1.55** (1.34, 1.79)	**1.84** (1.15, 2.94)	
CA-born, NY Resident (CANY) (ref.)	1.0	1.0	1.0	
Primiparous, 1	**1.06** (1.01, 1.11)	**1.19** (1.13, 1.25)	**1.19** (1.13, 1.25)	
Multiparous, 2 or more (ref.)		1.0	1.0	
Aged 15-19 years	**1.14** (1.03, 1.27)	1.10 (0.98, 1.22)	1.09 (0.97, 1.22)	Demographic
Aged 25-29 years	1.06 (0.99, 1.14)	**1.12** (1.05, 1.20)	**1.13** (1.05, 1.21)	**.034**
Aged 30-34 years	**1.22** (1.14, 1.30)	**1.31** (1.22, 1.40)	**1.32** (1.23, 1.42)	
Aged 35-39 years	**1.48** (1.38, 1.59)	**1.55** (1.44, 1.67)	**1.56** (1.45, 1.69)	
Aged 20-24 years (ref.)	1.0	1.0	1.0	
< 12 yrs education	0.97 (0.91, 1.04)	0.97 (0.91, 1.04)	0.96 (0.90, 1.03)	
13-15 yrs education	0.92 (0.87, 0.97)	**0.93** (0.88, 0.98)	**0.92** (0.87, 0.97)	
16+ yrs education	0.94 (0.88, 1.01)	**0.90** (0.84, 0.97)	**0.90** (0.84, 0.96)	
12 yrs education (ref.)	1.0	1.0	1.0	
Unmarried	**1.07** (1.02, 1.12)	**1.11** (1.06, 1.17)	**1.12** (1.06, 1.17)	
Married (ref.)	1.0	1.0	1.0	
2nd trimester prenatal care	0.92 (0.87, 0.97)	**0.92** (0.87, 0.97)	**0.91** (0.87, 0.97)	Medically-related
3rd trimester prenatal care	0.71 (0.65, 0.77)	**0.68** (0.62, 0.74)	**0.67** (0.61, 0.73)	.726
None/unknown prenatal care	**1.68** (1.55, 1.81)	**1.73** (1.59, 1.87)	**1.73** (1.59, 1.87)	
1st trimester prenatal care (ref.)	1.0	1.0	1.0	
Smoked during pregnancy	**1.49** (1.16, 1.92)	**1.32** (1.03, 1.71)	**1.32** (1.02, 1.71)	
Did not smoke (ref.)	1.0	1.0	1.0	
Maternal medical risks	**2.18** (2.09, 2.28)	**2.16** (2.07, 2.26)	**2.16** (2.07, 2.26)	
No maternal medical risks (ref.)	1.0	1.0	1.0	
Significant marginal interactions:				
CAVI * <12 years education			**1.47**[b] (1.03, 2.09)	

Note: Significant terms are bolded.

[a]Interaction effects are indicated by *p* -values for the significance of V.I. or CA nativity interacting with combined risk factors.

[b]Marginal interactive effect only. The OR_{CAVI}/OR_{CANY} estimate for <12 years education is **2.69** (1.59, 4.55).

Table 4.9 Actual and Counterfactual Probabilities of Preterm Birth, by Migrant Status, non-Hispanic Blacks, U.S. Virgin Islands (2000-2004) and New York City (2000-2009)

	V.I.-born		Caribbean-born	
	Non-migrant (VI resident: VIVI)	Migrant (NY resident: VINY)	Non-migrant (VI resident: CAVI)	Migrant (NY resident: CANY)
	A	B	A′	B′
Actual predicted probability[1]	14.85%	11.57%	15.08%	10.73%
Counterfactual predicted probability[a] based on population characteristics of non-migrant (V.I.-resident) group	—	9.31%	—	10.38%

Note: Unmeasured factors are indicated by group percentage change differences between columns A and B (second row), or A′ and B′ (second row). Measured factors are indicated by group percentage change differences in column B or B′.
[a]Conditional on parity, age, education, marital status, prenatal care, smoking, and maternal medical risks.

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CHAPTER 5

Maternal Aging and Preterm Birth Risks In U.S.-born and Caribbean-born Black Women

This chapter will (1) summarize the conceptual approaches and empirical findings on the concept of weathering (as related to adverse birth outcomes between non-Hispanic Blacks and Whites); (2) highlight gaps and opportunities with respect to examining weathering *within* Black populations by nativity status; and (3) present an original study exploring patterns of weathering in immigrant and native-born Black women in New York City and the U.S. Virgin Islands. The overall aim of this chapter is to examine risks of preterm birth with advancing age among non-Hispanic Black mothers in consideration of both national origin and migrant status. Maternal aging is conceptualized as a socially patterned risk factor as outlined in my conceptual model (see Chapter 3). Exposure to the U.S. social context, with an emphasis on racial stratification/racism, may be particularly harmful for Black immigrants who may be less socialized to this reality. Accordingly, this chapter explores immigrants' duration in the U.S. in association with preterm birth risk, which is depicted in my conceptual model.

Background

Geronimus (1992) coined the term "weathering" to describe the precipitous decline of African American women's reproductive health with advancing maternal age. The weathering hypothesis argues that adverse birth outcomes in African American women are the result of the impaired health of *women* that ultimately compromises fetal health (Geronimus & Bound, 1990). Further, weathering holds that health onslaughts are precipitated by contextual circumstances

such as socioeconomic disadvantage and race-based stressors, coupled with psychological and physical responses, that increase morbidity and mortality of African American women and infants (Geronimus, 1992, 2001). The central thesis of weathering highlights the observance of a reverse J-shaped curve whereby African American females in their teens have a lower risk of neonatal mortality and low birthweight than African American women in their 20s, and the risks rise steeply with maternal age. In contrast, a J-shaped or U-shaped curve is more representative of the risk pattern for White women, who experience the highest risks of adverse birth outcomes among teens and the lowest risks for women in their 20s, with elevated risks that do not recur until the 30s.

Perinatal health researchers have sought to replicate findings of weathering. Overall, the weight of the evidence among 14 published studies is supportive of the hypothesized patterns of weathering in African American populations, including no evidence of worsening risks for Black teens relative to young adult Black mothers (Ekwo & Moawad, 2000; Swamy, Edwards, Gelfand, James, & Miranda, 2012); increasing within-group age slopes with advancing age (Geronimus, 1996; Holzman et al., 2009); and larger Black-White odds ratios with advancing age (Buescher & Mittal, 2006; Rich-Edwards, Buka, Brennan, & Earls, 2003). However, non-supportive findings have been demonstrated as well (Ananth, Misra, Demissie, & Smulian, 2001).

There is significant variability across studies which makes it difficult to systematically compare empirical investigations of weathering. Existing studies have observed diverse geographic and racial populations, including significant variability among the teenage years observed (from as young as 11 to the typical 15-19 age range); the age reference groups used (from <20 to 20-24 or 25-29); and the upper maternal age cut-offs (ranging from 34, to 35 and above, and 40 and above). The inclusion of actual or potentially confounding factors is also quite

varied—ranging from crude analyses with no testing of confounding variables (Buescher & Mittal, 2006; Geronimus, 1992) to systematic testing of a range of main and interactive effects (Rauh, Andrews, & Garfinkel, 2001; Rich-Edwards et al., 2003). Importantly, there are no standard definitions or analytic approaches used to determine "weathering." Approaches range from stratified comparisons of the increases in the age-related slopes *within* one population compared to another (usually Blacks and Whites) (Geronimus, 1986, 1992; Holzman et al., 2009; Khoshnood, Wall, & Lee, 2005; Love, David, Rankin, & Collins, 2010; Schempf, Branum, Lukacs, & Schoendorf, 2007)—to relative comparisons of the change in the age-specific odds ratios or rate ratios *between* Black and Whites, using Whites as the reference group (Buescher & Mittal, 2006; Rich-Edwards et al., 2003) (Osypuk & Acevedo-Garcia, 2008; Rauh et al., 2001).

Most weathering studies have examined Black populations as an ethnically homogeneous group. Studies have intentionally excluded foreign-born populations from their samples so as not to introduce nativity effects which may lessen the magnitude of estimates for Black or Whites (Osypuk & Acevedo-Garcia, 2008; Rauh et al., 2001). To date, only one published study has analyzed weathering in foreign-born Blacks (Deal, Bennett, Rankin, & Collins, 2014). In this study, consistent with original theories of weathering, weathering was operationalized as a pattern of lowest risks of low birthweight for teens with progressively increasing risks across the reproductive age span. This is the pattern that has been observed for African Americans, which is in contrast to the J-shaped or U-shaped curve for non-Hispanic Whites where teens have elevated risks. In a 2003-2004 U.S. national sample of non-Hispanic foreign-born Blacks, Deal et al. (2014) found J- and U-shaped patterns of age-related risk (restricted by marital status, parity, and prenatal care) that were similar to what is observed among non-Hispanic Whites, and hence concluded that there is no weathering pattern for foreign-born Blacks. This study had no comparison group, and the outcome was low birthweight.

With the exception of the latest study that was exclusive to foreign-born Blacks (Deal et al., 2014), all prior weathering studies have included general populations of Black women. One of the earliest studies of weathering as an explanatory framework for Black-White differences in birth outcomes examined neonatal mortality in three southern states (Geronimus, 1986). For crude estimates, neonatal mortality was highest for Blacks younger than 15, began an upward climb in the mid-20s, and declined starting at age 27. Adjusting for race and prenatal care confounded the association of age with neonatal mortality. For example, adjusting for race revealed a higher risk for first-time older than younger Black mothers, relative to primiparous Whites aged 24-26. Data consistent with the weathering framework were also observed for African American women in a study of LBW and VLBW controlled for maternal medical risks, prenatal care, and smoking (Geronimus, 1996). However, when neighborhood SES was added as a control, weathering persisted only for Blacks in lower income communities; higher-income Blacks did *not* demonstrate weathering in comparison to Whites. Holzman and colleagues (2009) explored weathering utilizing an extensive population pool across several states. Maternal age, neighborhood disadvantage, and smoking (where smoking was conceptualized as high-risk coping) were examined simultaneously in relation to PTB. In this study, a weathering pattern was found for Black smokers and non-smokers, and a more pronounced effect was demonstrated for Black mothers living in deprived (sic) neighborhoods (Holzman et al., 2009).

Weathering investigators have linked census data to explore the contribution of neighborhood factors to birth outcomes. Median neighborhood income was found to influence weathering in relation to LBW for low income Blacks, but not for Blacks in higher income neighborhoods (Geronimus, 1996). In another study, extreme racial segregation worsened the age effect for Black women (Osypuk & Acevedo-Garcia, 2008); this study intentionally excluded foreign-born Blacks from the analysis. Collins et al. (2006) broadened the concept of

neighborhood disadvantage to include rates of unemployment, homicide, and lead poisoning in addition to median household income. Similar to Geronimus' (1996) findings, MLBW increased with maternal age, especially in the most impoverished neighborhoods. However, poverty modified the effect of maternal age on LBW only for low-parous mothers. Like most weathering studies, the effect was not strong for the outcome of VLBW which includes a significant component of preterm births.

Multilevel modeling affords greater methodological precision in parsing out individual and group effects—thus helping to rectify the problem of statistical non-independence when group measures are used to predict individual birth outcomes. Rich-Edwards and colleagues' (2003) multi-level analysis analyzed the slopes of the age gradient on LBW for Blacks and Whites while simultaneously addressing individual maternal risk factors and census tract poverty. The age-slope divergence between Blacks and Whites disappeared when the interaction of age with neighborhood poverty and other sociodemographic and medical risk factors were controlled. Therefore, in the face of socioeconomic disadvantage, the age-related weathering effect worsened for *both* Blacks and Whites, although Blacks fared worse than Whites for individual-level risk factors and outcomes. In a multi-level analysis utilizing New York City birth records, Rauh et al. (2001) discovered that community poverty as a main effect influenced MLBW for Black, but not White, women. However, community poverty did not modify the effect of individual income on MLBW nor did community poverty modify the age effect found at the individual level. With adjustments, there was *no* weathering effect (i.e., Black/White disparity with advancing age) for *non-poor* women so indicated by their non-receipt of Medicaid. In general, studies demonstrate that both individual and neighborhood income influence weathering, with poor women in poor neighborhoods being at highest risk.

The inclusion of confounders or effect modifiers varies significantly in weathering studies of birth outcomes. Maternal medical characteristics explained much of the age effect in Geronimus' weathering study (1996), and there was no statistically significant age effect when all relevant risk factors were accounted for. The study by Holzman et al. (2009) demonstrated that smoking contributed to weathering for both Black and White women, and the age-gradient was sharper as neighborhood deprivation increased. In another study, adjustment for interactions of age with maternal education, marital status, prenatal care, cigarette smoking, and neighborhood poverty eliminated the age-dominant effect on birth outcomes (see Rich-Edwards et al., 2003). Adequate prenatal care did not ameliorate the effect of age on infant birthweight in highly impoverished neighborhoods (Collins et al., 2006). Schempf and colleagues' (2007) analysis of preterm birth highlighted parity in association with maternal age and race/ethnicity. Weathering was demonstrated for all older, primiparous women. But for moderate PTB (32-36 weeks) and very PTB (28-31 weeks), only older, multiparous, Black and Hispanic women demonstrated weathering. The results by Schempf et al. (2007) lend insight into the largely absent findings for weathering in relation to preterm birth; disaggregated categories of parity and preterm birth elicited divergent weathering findings for the study populations. Unlike Schempf et al. (2007) who highlighted multiparous mothers, many weathering studies are limited to primiparous mothers, referring to their first births only.

Despite the acknowledged importance of cumulative, lifecourse disadvantage to the weathering phenomenon, prospective cohort studies are rare. Love et al. (2010) utilized an inter-generational dataset in Chicago to examine weathering for Black and White women from birth through their childbearing years with respect to areas of residence. Black women who were born in poor areas and also lived in poor neighborhoods as adults exhibited weathering for LBW and SGA, but *not* preterm birth. Black and White women living in upper income areas at birth and

during adulthood did not demonstrate weathering and actually had declining LBW and SGA with advancing age, with no observed effect on preterm birth. Once again, weathering was most restricted to poor women in poor neighborhoods.

Weathering patterns have not held true in all studies. Ananth et al. (2001) found similar age patterns of risk for preterm birth in both Black and White women across birth periods and cohorts with the lowest risk found in the 25-29 age group for both Blacks and Whites. Thus, they concluded that the weathering construct (in relation to the importance of social antecedents) was an unlikely explanation for race- and age-related disparities in PTB, and they reinforced that maternal aging operated similarly in Blacks and Whites. As previously described, weathering patterns observed in unadjusted analyses have disappeared or remained partial with adjustments for income (Geronimus, 1996), for parity (Schempf et al., 2007), for age x race interactions (Rich-Edwards et al., 2003), or dependent on the birth outcome observed (Love et al., 2010; Rauh et al., 2001).

In weathering studies, the findings are robust in relation to MLBW (<2500 g), but less consistent for VLBW (<1500 g) and PTB (<37 weeks gestation). Ascertaining the weathering phenomenon for VLBW and PTB is important because these outcomes confer risks for adverse birth outcomes more consistently than MLBW, and they are acknowledged to lie on the causal pathway to infant mortality (Gage, Fang, O'Neill, & Stratton, 2009; Gage, Fang, O'Neill, & DiRienzo, 2010; Wilcox & Russell, 1983). Among only five studies using preterm birth as an outcome to examine weathering in Black women, three found evidence of weathering (Holzman et al., 2009; Osypuk & Acevedo-Garcia, 2008; Schempf et al., 2007), and two had non-significant findings (Ananth et al., 2001; Love et al., 2010). Preterm birth did not significantly increase with age or among Blacks in any socioeconomic group, including low income Black women (Love et al., 2010). Of four studies that looked at weathering for VLBW, three found

supportive findings, including one descriptive study comparing unadjusted rates by age (Buescher & Mittal, 2006) and two studies with conditional findings that varied by population (Geronimus, 1996; Rauh et al., 2001). Another study (Collins et al., 2006) found no neighborhood impact on the age effect for VLBW in contrast with positive findings for MLBW. The authors deduced that contextually-laden phenomena such as weathering have a greater influence on intrauterine growth retardation (IUGR), which represents a significant component of MLBW, than it does on VLBW or PTB. Love et al. (2010) attributed null findings for PTB risk to a greater likelihood of measurement error for gestational age. Notwithstanding these speculations, the foundational reasons for why weathering is less likely to influence PTB and VLBW have not been articulated.

Conceptually, weathering speaks to divergent health and birth outcomes of African American and White women in the U.S., particularly with respect to the hypothesized accelerated aging that takes place in African American populations with accompanying declines in maternal and fetal health. Although weathering has been employed as an explanatory framework to study birth outcomes and maternal age in Hispanic (Collins, Rankin, & Hedstrom, 2012; Wildsmith, 2002) and Asian (Wang & Lee, 2012) populations, African Americans and Whites remain the primary index populations for the weathering framework. Indeed, African Americans and Whites are viable populations to examine in weathering research due to the longstanding evidence of race-based inequities that disproportionately disadvantage Black populations in the U.S. Furthermore, due to smaller populations and fewer events of adverse birth outcomes, there is often inadequate power to conduct analyses of weathering in populations other than non-Hispanic Blacks and Whites, including subpopulations of these groups.

For the above noted reasons, the literature on weathering is replete with studies restricted to disaggregated, U.S.-based Blacks and Whites. Consequently, little published information

exists on weathering patterns among ethnic subpopulations such as non-Hispanic Caribbean Blacks. Although cross-sectional studies demonstrate that foreign-born Blacks in the U.S. have more favorable birth outcomes than U.S.-born Blacks (Acevedo-Garcia, Soobader, & Berkman, 2005; Cabral, Fried, Levenson, Amaro, & Zuckerman, 1990; Elo, Vang, & Culhane, 2014; Howard, Marshall, Kaufman, & Savitz, 2006; Hummer et al., 1999; Kleinman, Fingerhut, & Prager, 1991; Rosenberg, Desai, & Kan, 2002; Singh & Yu, 1996) the foreign-born advantage, specifically for Caribbean-born Blacks, tends to lessen over time (Collins, Wu, & David, 2002; Valanis & Rush, 1979), and the rates of adverse birth outcomes are routinely higher for foreign-born Blacks than foreign-born Whites at *all* times. An interesting observation across studies is that Caribbean immigrant women in the U.S. have a higher proportion of births in their 30s and 40s compared to African Americans. Analyses of births to women in the Caribbean reveal a similar pattern of fairly high percentages of births at older maternal ages. For example, the birth rate for Caribbean-born women aged 35-39 in the Virgin Islands (20.2%) is even higher than the birth rate for Caribbean-born women aged 35-39 living in the U.S. (18.9%).

The degree to which the weathering phenomenon (i.e., age-related trajectories of risk) can help to explain the differential outcomes between foreign-born and U.S.-born Blacks is an empirical question that will be explored in the present paper. In particular, the general findings of Caribbean Blacks as a more socioeconomically advantaged population (*vis à vis* African Americans), with lower maternal health risks, evokes a special curiosity regarding if and how weathering manifests in this population. Moreover, the higher proportion of births to older migrant women, especially in consideration of the comparably lower rates of adverse birth outcomes in this population (relative to African Americans), focuses the weathering lens on the older age group (in contrast to teens), and evokes speculation about the social patterning of this phenomenon. This and other age-related findings (both crude and adjusted for factors that

confound and interact with age) are the focus of this paper. Only one previous study, very recently published, has examined weathering in foreign-born Blacks (Deal et al., 2014). However, this study did not differentiate the foreign-born population by national origin. Moreover, it did not have a comparison group to assess how the observed patterns would differ from U.S.-born Blacks (or other racial/ethnic groups) in the same birth cohort, and the outcome was restricted to low birthweight. The present study provides a more detailed analysis of weathering within Black populations by nativity and by migrant status in relation to the outcome of preterm birth.

Purpose

This chapter will examine maternal age in relation to hypothesized racism-related effects on maternal health and preterm birth for non-Hispanic Black Caribbean immigrants—in and outside of the U.S. racial context. The present study is couched within a framework that conceptualizes racism as a source of weathering in foreign-born Blacks as well as U.S.-born Blacks. My operationalization of weathering does not predict any specific linear or curvilinear relationships between teen mothers and their older-age counterparts. Rather, drawing upon the concept of cumulative health disadvantage across the reproductive age spectrum, I will focus attention on differences in the age-related slopes for preterm birth between V.I.-born/Caribbean-born mothers and African American mothers in New York City, as well as between women in the V.I. and their migrant counterparts residing in New York City. In addition, I will explore the concept of cumulative disadvantage by examining preterm birth in relation to length of residence in the U.S. for foreign-born Black mothers. Length of U.S. residence is operationalized as an exposure to racism.

In this chapter, weathering is operationalized threefold as: (a) increasing risks of preterm birth with advancing age from ages 15-39 *within* discrete populations of Black mothers stratified

by nativity and migrant status; (b) a *widening* of the gap in age-related risks of preterm birth with advancing age indicated by the increasing magnitude of the odds ratio *between* populations of Black mothers; and (c) an increase in the *rate* at which hypothesized maternal aging occurs as indicated by a statistically significant *interaction* in a positive direction between age and nativity or migrant status. For between-group estimations, the populations being compared are (i) Black V.I.-born and Other Caribbean-born migrants in New York relative to Black U.S.-born women in New York as the reference; and (ii) Black V.I.-born and Other Caribbean-born migrants in New York compared to their non-migrant counterparts living in the Virgin Islands as the reference. The estimates for Black Caribbean migrants relative to U.S.-born Blacks are expected to be lower (i.e., less than 1). Therefore, I will attempt to confirm that this index population is *less* "weathered" in accordance with my operational definitions.

The following research questions will be investigated. First (Research Question 1), to what extent is weathering, in relation to preterm birth, present for V.I.-born and Caribbean-born, non-Hispanic Black mothers in New York in comparison to U.S.-born non-Hispanic Black mothers in New York? I hypothesize that V.I.-born and CA-born Black women residing in New York City will demonstrate weathering trends (i.e., age-specific preterm birth risks) that are similar to African Americans, although the *magnitude* of weathering for Black immigrants will be less severe (i.e., flatter age-related slopes) relative to U.S.-born non-Hispanic Black women. Second, (Research Question 2), how does weathering operate for V.I.-born and Caribbean-born mothers migrants to New York compared to their native counterparts residing in the Virgin Islands? I hypothesize that V.I.- and Caribbean-born Black women residing in New York will demonstrate more severe "weathering" (i.e., steeper age-related slopes) than V.I.- and Caribbean-born women living in the Virgin Islands. Third (Research Question 3), how does length of U.S. residence influence preterm birth in non-Hispanic Black Caribbean immigrants? I hypothesize

that there will be a graded, linear increase in the odds of preterm birth by length of U.S. residence for Caribbean immigrants.

Method

Data Sources. This study utilized natality files from New York City (2000-2009) and the U.S. Virgin Islands (2000-2004). A detailed description of these sources is provided in Chapter 4. The present study also utilizes 2008-2010 birth data from New York City to explore length of residence in the U.S. as a predictor of preterm birth risk; length of residence information did not become available until 2008. The 2008-2010 NYC data are identical to 2000-2009 data utilized in Chapter 4 except for the length of residence variable and number of cases used. The analysis of duration of residence utilized 22,542 cases. V.I.-born immigrants (n = 204) and Caribbean-born immigrants (n = 22,338) were aggregated as one group for this sub-analysis.

Measures. *Preterm birth* (PTB) is defined as a live-born, singleton infant delivered at < 37 completed weeks of gestation. Preterm birth was dichotomized as < 37 completed weeks of gestation or ≥ 37 weeks of gestation. *Maternal age* was categorized as 15-19, 20-24, 25-29, 30-34, and 34-39, with age group 20-24 being the reference category. For predicted probability models, age was utilized as a continuous variable restricted to ages 15-39 and plotted in five-year increments. Continuous age was also modeled as age^2 to account for non-linearity. *Length of U.S. residence* was based on self-report of mothers who were not born in the United States. This question was asked of all immigrant mothers delivering in New York City beginning in 2008. Length of U.S. residence was modeled as mean years for this study, including calculation of intervening months 1-11. A detailed description of the other variables selected for this study can be found in Chapter 4.

Analyses. Frequencies and cross-tabulations were estimated with the Wald chi-square statistic to evaluate differences in expected frequency proportions by nativity for the outcome

and predictor variables. The descriptive assessment was followed by logistic regression models estimating preterm birth with nativity and age exclusively as well as adjusted for other pregnancy risk factors. Parity was included as a control in all models. In addition to main effects-only models, I tested interaction terms for nativity and categorical age as well as nativity and continuous age, in separate models, with and without adjustment for additional risk factors.

Preliminary analyses that were modeled with age^2, age^3, and age^4 indicated marginal significance for age^2 only, so the higher-order age terms were not included in the models described in this study. Age was centered at age 15 and modeled in five-year increments. The age-related effect for each nativity group was determined by adding the betas for the age term together with the age^2 term and significant nativity*age interactions. The formula can be represented as follows,

$$\log\frac{p}{1-p} = \beta_0 + \beta_{1\,Nativity} + \beta_{2Age} + \beta_{3Age^2} + \beta_{4Nativity*Age}$$

Due to the significance of age-squared, the change in slope for a five-year period within each population is indicated as follows, where ***x*** represents a given age.

$$\beta_{2Age} + (\beta_{3Age^2} \times 2x) + \beta_{3Age^2} + \beta_{4Nativity*Age}$$

These effects were charted to illustrate the probability of preterm birth with advancing age for mothers aged 15 to 39.

The first set of weathering analyses in this paper include V.I.-born, CA-born, and U.S.-born mothers in New York City. These are followed by analyses by migrant status which compared V.I.-born migrants in NY with V.I.-born natives in the Virgin Islands and also compared CA-born migrants in NY with their CA-born counterparts in the Virgin Islands. All models were estimated using the *proc logistic* function in SAS 9.4 (SAS Institute, Inc., Cary, North Carolina).

Results

Population Characteristics

Table 5.1 shows descriptive results for the three nativity groups. Overall, pregnancy risk characteristics were most favorable for Caribbean-born mothers and least favorable for U.S.-born mothers, with V.I.-born mothers falling in-between. Regarding age distributions, most births to African American and immigrant Black women occurred at the expected population frequencies during women's reproductive age cycles (Figure 5.1). Birth proportions were most equivalent during ages 25-29, where roughly a quarter of all births in each group occurred at these ages. However, there was significant inter-ethnic variation outside of this range. African Americans had highest birth rates at 15-19 and 20-24 years, with declining birth rates at older ages compared to Black immigrants, particularly after 30. After age 30, immigrants had significantly higher birth rates. African American teen birthrates were double the rates for the V.I.-born and Caribbean-born teens. For African Americans, 16.4% of births were to women 15-19 compared to 9.2% of births to V.I.-born teens and 5.8% of births to Caribbean-born teens (Table 5.1). The most dramatic and influential difference in the age distributions for births occurred at the older maternal ages. For Caribbean-born women and V.I.-born women, 19.0% and 15.4% of births, respectively, occurred to women aged 35-39. In comparison, the birth rate percentage for African American women aged 35-39 was 10%. Figure 5.1 illustrates the age distributions of births for Virgin Islands' mothers, Caribbean-born mothers, and African American mothers in New York City.

Preterm Birth by Age

Table 5.2 indicates the number and percent of preterm births, stratified by age, in each nativity group. For V.I.-born women, crude preterm birth rates were lowest for teenage mothers. For Caribbean-born and U.S.-born mothers, preterm birth rates were slightly elevated for teenage

mothers, declined slightly at ages 20-29, and rose progressively thereafter. Figure 5.2 illustrates the patterns of preterm birth by age. A J-shaped pattern is depicted for both Caribbean-born and U.S.-born mothers, with teens (15-19) having slightly elevated rates than women in their early 20s. The V.I.-born mothers are an exception with lower preterm birth rates for teens. Not unexpectedly, preterm birth rates are higher for African Americans than Caribbean-born women at all ages, with the exception of a slightly higher rate for V.I.-born women at ages 25-29.

Some important group distinctions are represented by age patterns. First, the difference in preterm birth rates was narrowest for Caribbean-born and U.S.-born women at 15-19 and 20-24 years old, and the disparities widened progressively thereafter, but most noticeably after age 30. The most dramatic difference in age slopes is with the V.I.-born population. Due to the small population and fewer cases of preterm birth in the V.I. population, the charted rates depict significant variability. However, there is a linear trend with a rapidly increasing slope after age 24 except for an unexplained dip during ages 30-34.

The review of crude preterm birth rates in this section has determined that preterm birth rates are highest for African Americans and V.I-born immigrants and lowest for Caribbean-born immigrants in New York. The worsening of preterm birth risk was particularly steep after age 30 for the V.I.-born and U.S.-born women, whereas the curve of increasing risk with advancing age was flatter for CA-born immigrants. For the V.I. populations, the lower preterm birth risk for teens with a fairly progressive increase in risk thereafter is consistent with the generally-observed weathering phenomenon in U.S. Black women. In this New York-based sample, it is of note that African Americans did *not* exhibit a normative weathering pattern (i.e., lowest risks among teens), which contrasts with findings in other studies that examine national samples of Black women or samples in other U.S. states or regions.

Research Question 1: Weathering by Nativity Status

It was hypothesized that V.I.- and Caribbean-born, non-Hispanic Black mothers would demonstrate a weathering pattern for preterm birth, although to a lesser degree than African American women. Table 5.3 compares age estimates, stratified by nativity, which reveals the age-related patterns for V.I.-born, CA-born, and U.S.-born women. The results are best examined horizontally to distinguish similarities or differences in age-specific estimates across nativity groups. When viewing age effects adjusted only for parity (Model A, top row), V.I.-born women 35-39 had over 2.5 times the odds of preterm birth for mothers aged 35-39 compared to mothers aged 20-24 (*OR* = 2.51, 95% CI: 1.35, 4.67). This compares to a 62% elevated odds for older CA-born and U.S.-born mothers. Model A best approximates the crude, age-related trends depicted in Figure 5.2 and confirms that, even with a parity adjustment, the pattern of 'weathering' is similar for Caribbean- and U.S.-born Black women. Model B (middle row) in Table 5.3 controls for demographic factors, and this model depicts more variation in age-related risks and higher relative odds for CA-born women overall. Older mothers in all groups had the highest risks of preterm birth, but the relative odds between CA-born women aged 35-39 vs. 20-24 (*OR* = 1.71, 95% CI: 1.58, 1.84) were 11% lower than the corresponding odds for African American older mothers (*OR* = 1.90, 95% CI: 1.79, 2.01). V.I.-born mothers had the highest predicted odds overall, especially for older women (*OR* = 2.58, 95% CI: 1.34, 4.96).

A more thorough evaluation of weathering takes into account confounders of preterm birth risk, in addition to age. This was attempted in Model C (Table 5.3, bottom row) with the addition of medically-related risk factors. When medically-related factors were considered, the age-specific estimates did change, but variably across groups. The patterns demonstrated a marked change in age-specific estimates for CA-born women starting at age 35-39 from Models A to C, presumably mediated by demographic factors. For U.S.-born mothers, the modification

in weathering patterns began during the 30-34 age range. In contrast, there was no sizeable shift in age-specific estimates for V.I.-born women between restricted and fully-adjusted models, and V.I.-born women sustained the highest overall odds of preterm birth, especially at the older ages, among the three nativity groups. Interestingly, with adjustments for parity, demographic factors, and medical risk factors (Model C), teen mothers 15-19 did not significantly differ from mothers 20-24 in preterm birth odds within any nativity group.

Due to observed effect modification by age and education in this study (detailed data not shown) and to mimic an approach in several weathering studies to restrict the sample to first births and/or mothers aged 20 and older, I investigated weathering in a sample restricted to *first births* among mothers who had *completed at least 12 years of education*. Table 5.4 adjusts for demographic and medical risk factors on this restricted sample of primiparous mothers with at least a high school education. The age-related patterns of risks in this restricted sample are similar to the patterns observed on the full sample; however, the age-specific odds are of greater magnitude in the restricted sample. This indicates that, within groups, preterm birth risks with advancing age are worse among primiparous mothers with at least a high school education.

Tables 5.5 to 5.10 afford a closer look at the age effect for V.I.-born and Caribbean-born immigrants. Interactive effects demonstrate unique age-slopes for each population, which provide a more refined indicator of differential patterns of aging between groups. Significant interactive effects in a positive direction, at younger ages, are suggestive of 'accelerated aging'—hence weathering. V.I.-born and Caribbean-born women were compared to African Americans to evaluate if there were any slope differences in age-related risks. The interactive age effect was examined with adjustments for parity, education, and other demographic and medical risk factors.

Unrestricted Data. In Table 5.5, there was a unique categorical age slope for Caribbean-born women aged 35-39. The interaction effect was estimated comparing Caribbean-born mothers 35-39 to U.S.-born mothers at the same age. For example,

$$\frac{OR_{CA\ aged\ 35-39}}{OR_{US\ aged\ 35-39}} = \exp\left(\beta_{1CA\ nativity} + \beta_{3CA\ nativity*aged\ 35-39}\right)$$

$$= e^{-.1511} \times e^{-.1239} = .8598 \times .8834 = .76$$

The marginal odds ratios reported in Table 5.5 will approximate the log odds (data not shown) used to calculate the interactive effect, with the possibility of slight rounding error.

The results in Table 5.5 indicate that Caribbean-born mothers had 24% *lower* odds of preterm birth compared to U.S.-born women at that age (*OR* = 0.76, 95% CI: 0.71, 0.81). This protective effect was sustained with adjustment for demographic, behavioral, and medical risk factors (*OR* = 0.76, 95% CI: 0.69, 0.84). For V.I.-born mothers, there was no statistically significant interactive effect with age, although the trend suggests higher than expected odds starting at age 25. These findings were based on the full sample of mothers, unrestricted by parity or education. Table 5.5 shows results by categorical age ranges with the sample restricted to primiparous mothers with at least a high school education.

Table 5.7 models age as a continuous term. Due to the significance of age^2, the age-related effects are difficult to interpret from the estimates as shown. Therefore, Table 5.8 provides the results of the calculated effects within each nativity group to provide a gross assessment of differential risks by age. Reported in odds ratios that represent the mid-point within each 5-year age range, the magnitude of risks are highest for V.I.-born women. Although U.S.-born women have the highest overall age-related risks, the slope effects between CA-born women and U.S.-born women are not as disparate as compared to the age-slopes for V.I-born women.

Figure 5.3 illustrates the probability of preterm birth with advancing age for the three nativity groups. The figure depicts the sustained reduced odds of preterm birth with advancing age for CA-born women relative to U.S.-born mothers. Importantly, the unique age effect for CA-born women is evident after age 35, where there is a slight flattening of the curve for CA-born older women relative to the simultaneous steeper slope for U.S.-born women. However, as indicated by the marginal interactive effect for CA-born women reported in Table 5.7 (*OR* = 0.97, 95% CI: 0.97, 0.99) coupled with the similarity in age-slopes, there is no evidence of a differential pattern of "weathering" between CA-born mothers and U.S.-born mothers.

Primiparous Mothers Who Graduated From High School. Many weathering studies restrict their samples to first births in order to avoid confounding related to pregnancy history or prior adverse birth outcome. Women delivering for the first time have a higher risk for preterm birth than women with existing children. A previous preterm birth is also the leading risk factor for subsequent preterm births. Moreover, restriction to women 20 and over is a common practice to avoid effect modification due to sociodemographic factors, particularly education. Women less than 20 could potentially still be in high school, and they are also less likely to have been married at that time or have no or low income, thus making them at particularly high risk. Tables 5.6 and 5.9 restrict the study populations to first-time mothers who had completed high school (12 or more years of education). With this specification, there were no significant differences in the advancing age slope for either V.I.-born or Other Caribbean-born women relative to U.S. women. Due to the small subsample size, the estimates for the V.I.-born population are not reliable. However, a gross weathering pattern is observed for Virgin Islands women, whereby the lowest risk of preterm birth occurred among teens, and there was a trend of worsening risk with advancing age. The negligible difference in preterm birth risk with advancing maternal age between CA-born and US-born mothers can be verified by comparing the age slopes in Table

5.10. Figure 5.4 plots the predicted probabilities of preterm birth across the 15-39 age range for the restricted group of mothers.

Summary. The findings for Research Question 1 indicated that age-graded patterns of preterm birth were fairly similar between Caribbean-born and U.S.-born mothers in New York. In the full sample, an effect modification was demonstrated for Caribbean-born mothers aged 35-39 signifying a lower probability of preterm birth than would be expected at later ages relative to U.S.-born Black women. However, this interactive effect was no longer significant in samples restricted to primiparous women with at least a high school education.

Consequently, there are mixed findings regarding weathering comparing V.I.-born and Caribbean-born immigrants to African Americans. Based on stratified models estimated separately by nativity group, U.S.-born mothers demonstrated earlier-onset risks of preterm birth that began during the 30-34 age range. This contrasted with risks that began during 35-39 for Caribbean-born women and suggested a slight advantage for Caribbean-born mothers who appeared to have a later age-related increase in preterm birth. Indeed, in categorical age models adjusted for demographic and medical risk factors, there was a 0.24 times *lower* odds of preterm birth for Caribbean-born women 35-39 than would be expected at that age (*OR* = 0.76, 95% CI: 0.71, 0.81), adjusted for demographic factors, smoking, prenatal care, and other medically-related risks. This unique age effect for CA-born immigrant women translated to an average 24% reduced odds of preterm birth relative to African Americans aged 35-39. However, when the population sample was restricted to first births among women with at least a high school education, the unique age effect for older Caribbean-born women was no longer statistically significant.

The contrasting findings for Caribbean-born women between the unrestricted and restricted samples suggested effect modification by parity or education. Indeed, in the

unrestricted sample, a significant interaction effect was confirmed for Caribbean women with a less than high school education, who had 26% lower odds of preterm birth compared to U.S. born women with low education (*OR* 0.74, 95% CI: 0.66, 0.83, data not shown). Although V.I.-born immigrants had higher probabilities of preterm birth with advancing age, age interactions were not statistically significant for this group in fully adjusted models, as the V.I. population was small and inadequately powered for most analyses.

In addition to the conflicting results with respect to age interactions, the contribution of medical risk factors to the preterm birth advantage for Caribbean-born mothers was also unclear. When viewing age-related preterm birth estimates in main effects models comparing V.I.-born and CA-born mothers with U.S.-born mothers as a reference group, the addition of medical risk factors did not change the 12% reduced odds in preterm birth between Caribbean-born women and U.S.-born women, nor did they modify the age-specific estimates. Furthermore, there were no significant interactions for Caribbean-born nativity * medical risks (*OR* 0.91, 95% CI: 0.82, 1.02, data not shown) nor for nativity*age*medical risks (*OR* 0.99, 95% CI: 0.86, 1.14, data not shown). This confirms that medical factors do *not* explain the preterm birth advantage for Caribbean-born women.

In closing, weathering effects were highly conditional, but a basic conclusion is the observance of no differential weathering pattern between Caribbean-born immigrants and African Americans. When both first and subsequent births were estimated together, with control for parity and education, there was a sustained reduced odds of preterm birth with advancing age for CA-born women relative to U.S.-born. Moreover, there was a slight widening of the gap at older ages due to a flattening of slope for CA-born women at older ages. This pattern indicated less "weathering" for CA-born women relative to U.S.-born women across the maternal age spectrum. Notwithstanding, the disparity was reduced considerably when the sample was

restricted to primiparous mothers with at least a high school education. In this population subsample, the gap between CA-born and U.S.-born women narrowed considerably, and there were no statistically significant differences in preterm birth between these groups with advancing maternal age.

Research Question 2: Weathering by Migrant Status

It was hypothesized that V.I.- and Caribbean-born Black mothers in New York City would demonstrate more weathering than V.I.- and Caribbean-born Black mothers in the Virgin Islands. Although not testable in this study, noxious features of the U.S. social environment were conceptualized to contribute to worsening general and reproductive health for U.S. migrants. An unexpected preliminary finding was the *higher* rates of preterm birth for V.I.-born women and Caribbean-born women living in the Virgin Islands than their counterparts living on the U.S. mainland. The preterm birth rate was 11.6% for V.I.-born migrants to New York City (2000-2009) (Table 5.1) and 14.9% for V.I.-born natives remaining in the Virgin Islands (2000-2004).

Table 5.11 reports relative odds of preterm birth by migrant status and by age, using non-migrant mothers as the reference group. The computed interaction effects, in consideration of the shared variance between nativity and age, are reported in the table footnote; these calculations are based on Model 3, the fully adjusted model. The calculations used the odds ratios to approximate the log odds betas, therefore there is rounding error relative to the more precise calculations using the log odds betas. Based on the final calculations, there was evidence of a differential age effect for V.I.-born migrants relative to V.I-born women living in the Virgin Islands (*OR* = 0.39, 95% CI: 0.26, 0.66), but the comparisons between CA-born women in NYC and in the V.I. were not significantly different (*OR* = 0.62, 95% CI: 0.38, 1.01). When both VI-born and CA-born women were combined, there was evidence of an overall reduced age slope for the NY-based migrants (*OR* = 0.57, 95% CI: 0.44, 0.74). These findings suggest that the

NYC residents, especially the V.I.-born women, were less "weathered" across the reproductive age spectrum than women living in the Virgin Islands.

When the sample was restricted to primiparous mothers who had completed high school (Table 5.12), there was no evidence of differently-patterned age-related slopes between V.I-born or Caribbean-born migrants and their non-migrant counterparts, indicated by the non-significance of the interaction effects for migrant status and age ($OR_{V.I.}$ = 0.88, 95% CI: 0.21, 3.66; OR_{CA} = 1.19, 95% CI: 0.40, 3.52). These findings indicate that differential age-related risks do not explain preterm birth disparities by migrant status among first-time mothers who graduated from high school; this restricted group of Black immigrant women residing in New York were no more 'weathered' than their migrant counterparts living in the Virgin Islands (comparisons are plotted in Figure 5.6). However, these results were not adjusted for duration of residence in the U.S., and this facet will be explored in the next section with a subset of data that includes this information.

Research Question 3: Preterm Birth by Length of U.S. Residence

Weathering theories attribute accelerated reproductive and fetal health declines in Black women to cumulative disadvantage related to exposure to structural and interpersonal racism exposures in the U.S. Accordingly, the third hypothesis for this study speculated that immigrant length of stay in the U.S. (a proxy for potential racism exposure) would be positively associated with preterm birth. For this analysis, both V.I.-born (n = 204) and Other Caribbean-born (n = 22,338) women were collapsed as one group.

Table 5.13 shows maternal and infant characteristics by immigrant status as well as immigrant duration of U.S. residence. On average, immigrant mothers had lived in the U.S. for 11.7 years, with a standard deviation of 8.1 years, and a range of U.S. residence from less than one month to 43 years (continuous data not shown). The bulk of births occurred to women who

had lived in the U.S. for about 5-9 years at the time of the index birth. Incidentally, about 6.8% delivered an infant after being in the U.S. for 0 to 6 months and 3.9% delivered an infant after being in the U.S. for 7 to 12 months. Educational attainment, marital status, first trimester prenatal care, and maternal smoking generally increased with duration of U.S. residence, and so did low birthweight and preterm birth. However, medically-reported maternal medical risks were not significantly different across years. As an aggregate group (i.e., not stratified by duration of residence), the immigrants under study differed significantly from U.S.-born Blacks on all maternal and infant health measures (Table 5.13).

Among the Caribbean immigrant residents who had given birth in New York City from 2000-2009, there was a notable effect of years of residence on a preterm birth outcome. Specifically, for every 5 years a Caribbean immigrant woman lived in the U.S., the odds of preterm birth increased by 10% on average (*OR* = 1.10, 95% CI: 1.07, 1.13). Adjustment for traditional demographic and medical factors explained only 3% of this difference (*OR* = 1.07, 95% CI: 1.04, 1.11) (Table 5.14). Using recent immigrants less than 5 years of U.S. residence as a baseline, the odds of preterm birth began a statistically significant incline during 10-14 years of residence (*OR* = 1.33; 95% CI: 1.16, 1.54) and tapered at 20 or more years of residence (*OR* = 1.30, 95% CI: 1.13, 1.50) with adjustment for traditional maternal-infant risk factors.

Table 5.15 provides a perspective of worsening risk for immigrant Blacks relative to native, U.S.-born Blacks in New York City. Overall, immigrant Blacks had an 11% reduced odds of preterm birth relative to native Blacks, with a more modest 2% reduction for immigrants delivering their first child in the U.S., as demonstrated by the interactive effect for parity (0.82 x 1.20 = 0.98), all other factors remaining equal between the groups. Immigrant women in the U.S. for less than 10 years had a sustained preterm birth advantage compared to non-immigrant Blacks (*OR* = 0.80, 95% CI: 0.72, 0.88). This protection was lost, however, after a decade of

residence—especially for immigrant women delivering their first child after extended U.S. residence. As demonstrated by the interactive effects for primiparous immigrant mothers, immigrants delivering their first child with over 15 years of U.S. residence had about a 20% increased odds of preterm birth compared to U.S. mothers with the same measured demographic and medical risk characteristics ($OR_{15\text{-}20\ years}$ = 1.35 x 0.88 = 1.19; $OR_{\geq 20 years}$ = 1.38 x 0.87 = 1.20). Figure 5.7 illustrates these duration of residence-associated relationships. There is a sharp incline in preterm birth risk for immigrants with increasing residence—and in fact, the predicted preterm birth rate for immigrants meets and exceeds the average rate for native-born Blacks shortly after 10 years of U.S. residence (Figure 5.7).

Discussion

This analysis confirmed that the age-related trends (or the overall shape of the curves) are similar for Caribbean and U.S.-born Blacks. When age-effects are examined by nativity, within race, there was nothing to suggest a significantly different pattern of weathering for Caribbean-born immigrants and African American women. Caribbean immigrants had lower preterm birth rates overall, but the developmental effect of age seems to be similar, particularly in models restricted to primiparous mothers with at least a high school education. The risk of preterm birth did not rise more quickly with age for Caribbean women. Age slope differences, indicating differential rates of weathering, were also not significantly different between Black Caribbean immigrants loving in New York and non-migrant counterparts living in the Virgin Islands. With respect to self-reported years of U.S. residence, recent Black Caribbean immigrants to the U.S. who delivered an infant within five years had a substantial preterm birth advantage (i.e., lower risks of preterm birth) relative to U.S.-born Blacks, and this protection was sustained for up to a decade of U.S. residence. After ten years, however, the advantage was lost, and long-term immigrants had higher predicted rates of preterm birth than U.S.-born mothers in New York.

Based on the results of this study, weathering is a conditional phenomenon in Black Caribbean immigrant populations. Despite lower average risks of preterm birth across the reproductive age-spectrum for Black Caribbean migrants in the mainland U.S., the rate of worsening maternal-infant health with advancing age for these immigrant populations does not appear to statistically significantly differ from the rate observed in U.S.-born maternal-infant populations or among immigrant counterparts living in the Virgin Islands. When the foreign-born Black populations under study were examined by years of residence in the U.S., weathering was observed for immigrant mothers in the U.S. for over 10 years, who had higher predicted rates of preterm birth than the rate for U.S.-born mothers.

Previous weathering studies have observed that the ages associated with the lowest risk of birth outcomes for African Americans—the teens and early 20s—happen to coincide with the ages with the largest proportion of first births (Geronimus, 1992). In the present study, for non-Hispanic Black Caribbean-born immigrant women in New York City, the largest proportion of singleton *first* births occurred to women aged 20-24 (32%) and 25-29 (28%), while 12% of first births were to teens. Also, in this study, for *both* immigrants and U.S.-born Blacks, the risk of adverse birth outcomes mimics the standard J-shaped curve where teens have slightly elevated risks of preterm birth than women in their 20s – not a "reverse J" as highlighted by Geronimus (1992) with respect to crude neonatal mortality rates. However, the difference between Black teens and women 20-24 was slight, and not statistically significant. This observation is the usual trend across weathering studies that have examined teen births with respect to preterm birth and low birth weight outcomes. Among the 19% of all births to older Caribbean immigrant women aged 35-39 years, 9% were first births—in contrast to 10% of births to African American women aged 35-39 among which 4.7% were first births. Something about this added fertility could transfer into lower reproductive health risks for Caribbean immigrants. In within-group analyses,

the elevated preterm birth odds for Caribbean-born immigrants 35-39 compared to mothers 20-24 was 61% among first births to women with a high school education. The corresponding relative odds were 87% for U.S.-born women 35-39. Although preterm birth predictions in this study did not extend past 39, there appeared to be a slight convergence between the CA-born and U.S.-born age slopes starting around age 38—with a steeper slope observed for CA-born women relative to African Americans. The variability in preterm birth for women after age 35 likely contributed to the marginal significance of advanced age for CA-born women in the unrestricted sample of mothers. In a previous study focused on LBW with advancing age, there were similar within-group odds ratios between older vs. younger women for non-Hispanic Blacks, non-Hispanic Whites, Mexicans, and Puerto Ricans, although Blacks were not differentiated by nativity status.

The magnitude of the odds ratios as well as the evidence of accelerated risks with advancing maternal age (the latter indicated by the significance of the interaction terms for nativity and age or migrant status and age) differed depending on adjustments for parity and education. It was expected that adjusting for parity (with two or more births as the reference) would produce similar results as estimates produced by models restricted to first births—especially considering that parity was not statistically significant in adjusted models. However, results differed significantly between models run on the full dataset versus a subsample restricted only to first births *and* mothers who had at least completed high school. Weathering studies are mixed in the choice to present results separately or exclusively for first births (Ekwo & Moawad, 2000; Geronimus, 1986, 1996; Holzman et al., 2009; Khoshnood et al., 2005; Schempf et al., 2007), or to adjust for parity as was done in this study (Love et al., 2010; Osypuk & Acevedo-Garcia, 2008; Rauh et al., 2001; Rich-Edwards et al., 2003; Urquia, Frank, Moineddin, & Glazier, 2010). In the interest of estimating valid effects for education and marital status in

populations with teens who would be less likely to have completed high school or be married, weathering researchers have restricted their samples to mothers over 20. Because this study was conducted on a previously unstudied population with respect to weathering, it was deemed important to examine the entire age spectrum. Nonetheless, sensitivity analyses with this age restriction produced estimates that were not dramatically different from the estimates in the parity- and education-restricted samples in the present study. Furthermore, because the present findings revealed no statistically significant differences between teens 15-19 and younger mothers aged 20-24, it would be prudent for future analyses or publication purposes to restrict the study sample to women aged 20 and over to avoid introducing measurement bias or effect modification by education or marital status which can complicate findings. Stratifying results for primiparous and multiparous mothers is another option.

The present study joins only two other weathering studies (Rauh et al., 2001; Rich-Edwards et al., 2003) that have considered interactive effects for the age slopes. Examining interactions provides a more valid assessment of age-related differences between population groups in combined analyses. In Rich-Edwards' study (2003) the weathered effect for Black vs. White women was only marginal in adjusted main effects models and disappeared with control for the interaction with age and Black race and race and SES, thus demonstrating no differential weathering pattern among poor Blacks and Whites. The present study revealed a modest reduction of the age effect for Caribbean immigrants 30-34 which disappeared among primiparous older mothers who had completed high school, and this prompted an interest in more detailed studies of older immigrant mothers. For example, previous weathering studies have explored how main effects change the interactions—to provide clues about risk or protective factors contributing to age-specific estimates.

The findings in this study of the declining preterm birth risks for immigrants over time in the U.S. are similar to findings in study of Black immigrant women in New York City (Valanis, 1979) and a study of immigrants and non-immigrants in Canada (Urquia et al., 2010). Valanis and Rush (1979) found that years of U.S. residence was inversely related to birthweight among over 700 foreign-born Black mothers prospectively enrolled at a prenatal clinic in Harlem. As in the present study, immigrants to Canada sustained a preterm birth advantage relative to non-immigrants for about 10 years before there was convergence of preterm birth risk with non-immigrant Canadians. Although Urquia and colleagues (2010) did not specify race in their analyses, racial background can be presumed from the reported immigrants' region of birth. For example, a majority of Caribbean immigrants in Toronto, Canada are of African ancestry from the West Indian islands—mirroring the index population in this study. In Canada, the highest odds of preterm birth were to immigrant mothers from the Caribbean region relative to immigrants from industrialized countries (*OR* = 1.72, 95% CI: 1.37, 2.17); this surpassed the estimates for immigrants from sub-Saharan Africa (*OR* = 1.25, 95% CI: 1.00, 1.56) which were statistically insignificant.

One important consideration yet to be resolved is the extent to which the U.S. racial context may influence the reproductive health advantage for foreign-born Blacks. An underlying tenant of this study is that racism is a central feature of the U.S. racial context that could impact the perinatal health of Black immigrants—particularly when considering the worse birth outcomes for non-Hispanic Black immigrants relative to immigrants of other races or ethnicities, and also considering the declining health for Black immigrants over time. For preterm birth, this study revealed an infant health advantage for Black Caribbean-born immigrants to the U.S. that lasted for about a decade. Afterwards, preterm birth rates approached the levels for U.S.-born Blacks. Therefore, the immigrant health advantage is not indefinite. In one of the few studies to

examine intergenerational birth outcomes in Black immigrant women, Collins and colleagues (2002) found that the mean infant birthweight was lower for 3rd generation descendants of African and Caribbean immigrants in Chicago than for 2nd generation descendants. In this study, I was able to ascertain declining reproductive health by duration of U.S. residence among first generation immigrant women controlling for standard demographic, behavioral health, and medical risk factors. However, specific contributing features of the U.S. context could not be ascertained due to unavailability of data.

A critical data limitation in this study is the absence of a valid socioeconomic indicator—a crucial determinant of weathering. Mimicking previous weathering studies, inclusion of neighborhood economic indicators linked to the individual birth records would have been desired, and multilevel modeling in consideration of neighborhood status as a predictor would have been even more ideal. Several weathering studies have examined indicators of neighborhood disadvantage believed to contribute to weathering, particularly neighborhood poverty (Collins et al., 2006; Geronimus, 1996; Love et al., 2010; Rauh et al., 2001; Rich-Edwards et al., 2003) and racial segregation (Osypuk & Acevedo-Garcia, 2008). The dataset upon which this study is based has residential information that can be used for neighborhood context analyses. However, errors in the coding of census tracts prevented valid linkages of neighborhood poverty or other context data which could have been important moderators of the observed weathering patterns.

Another limitation of the current study is the need for a more developed and conceptually consistent framework to justify examining weathering in Caribbean immigrant women, as well as the need for testable data that could link the results with racism-related factors presumed to contribute to weathering. Although I proposed racism as a central feature of the U.S. racial context that could impact the perinatal health of Black immigrants, no racism-specific constructs

were available for empirical testing. Moreover, I may have 'misappropriated' the weathering hypothesis by applying the concept to immigrant Blacks on the U.S. mainland as well as Blacks in the Virgin Islands who may have had little or no exposure to historical and current realities of racism within a U.S. context. My foregoing analysis may also be conceptually inconsistent with my choice to examine Black-only populations, not in relation to Whites. Comparing foreign-born Blacks to foreign-born Whites may have been more consonant with a weathering-focused analysis. However, I sought to examine the heterogeneity of risks *within* Black populations, and therefore, U.S.-born Blacks were the reference group for Black Caribbean-born populations in the U.S. and Black Caribbean residents of the Virgin Islands were the reference population for their migrant counterparts residing in New York. Black-White comparisons are the *sine qua non* in weathering studies and for health disparities research in the U.S. in general. It would be possible for me to reframe the analysis to examine weathering in foreign-born Blacks relative to foreign-born Whites or examine how weathering impacts other foreign-born Black populations in addition to Caribbean-born immigrants. A previous weathering-focused analysis using New York City birth data excluded immigrants and hence provide no data to support or refute my findings on weathering for immigrant Black populations in New York (Rauh et al., 2001). Other perinatal health studies utilizing New York City data to examine birth outcomes by Black nativity have not been framed in the context of weathering (Grady & McLafferty, 2007; Mason, Kaufman, Emch, Hogan, & Savitz, 2010; Stein et al., 2009). The only published study to date of weathering in foreign-born Blacks (Deal et al., 2004) utilized a nationwide sample (2003-2004 birth cohort) that was not disaggregated by Caribbean or African national origin, or by U.S. region, and included no relative comparisons to U.S.-born Blacks. Therefore, there are welcome opportunities to contribute additional research on this topic.

Notably, the present study views maternal age-related effects within the context of race-based cumulative disadvantage and speculates about differential exposures and responses to racism among Black Caribbean immigrants as a possible explanation for contrasting results for this population. Results that demonstrate erosion of the preterm birth advantage for immigrants with 10 or more years of U.S. residence was not explained by demographic or medical factors in this study, and is unlikely to be related to socioeconomic decline since SES would be likely to increase, not decrease, over time for immigrants. Indeed, the declining immigrant health advantage could be related to an erosion of culturally-protective factors, but it could also be reasonably attributed to the negative influence of the U.S. racial context (Pallotto, Collins, & David, 2000; Stein et al., 2009). Concomitant results in this study that suggest moderated preterm birth risk for Caribbean women at older ages could be related to biological (enhanced fertility); personal (racial identity, coping styles, resilience); or structural factors (neighborhood supports, permissive or restrictive immigration policies) that nonetheless buffer the impact of racism.

Overall, the factors that can confound the relationships between race, age, and adverse birth outcomes are still largely empirically unexplored. Weathering studies tend to examine Black-White disparities almost exclusively, and consequently, there is limited understanding of this concept within heterogeneous Black populations. The weathering framework was not devised for foreign-born Blacks, presumably because the health outcomes of immigrant Blacks are generally better than those of African Americans, and the exposure to U.S. racism (the conceptual genesis of weathering) in addition to the psychological and physiological responses, are not deemed to be as consistent or applicable for immigrants as they would be for U.S.-born Blacks. Moreover, the relative unavailability of disaggregated population health data on foreign-born Blacks precludes extant work and an established pattern of group-specific, weathering-

related findings upon which to build. For these reasons, my assertion of "weathering" as an explanation for age-related patterns of preterm birth in foreign-born Blacks is speculative and diverges from the original theorizing and intended study populations for the weathering framework. However, my application of "weathering" to foreign-born Blacks lays a foundation for further theoretical development expected to emerge on the basis of the empirical findings that were presented here.

Tables and Figures

Table 5.1 Maternal and Infant Characteristics by Nativity, non-Hispanic Blacks, Singleton Births, New York City, 2000-2009

	V.I.-born (*n* = 1,080)		**Caribbean-born** (*n* = 81,615)		**U.S.-born** (*n* = 141,857)		***p* value**
	%	*n*	%	*n*	%	*n*	
Maternal age, years							
15-19	9.2	99	5.8	4,721	16.4	23,211	**<.0001**
20-24	23.3	252	21.0	17,173	31.0	43,916	
25-29	23.7	285	27.8	22,685	25.0	35,478	
30-34	25.7	278	26.4	21,543	17.6	25,013	
35-39	15.4	166	19.0	15,493	10.0	14,239	
Parity							**<.0001**
Primiparous, 1	41.6		44.4		46.3		
Multiparous, 2 or more	58.4		55.7		53.7		
Education (years completed)							**<.0001**
<12	17.9		15.7		27.9		
12	32.2		40.6		32.1		
13-15	31.6		28.8		28.5		
16+	18.3		14.8		11.5		
Married	26.4		39.2		19.2		**<.0001**
Prenatal care initiation							
1st trimester	62.8		59.4		60.7		**<.0001**
2nd trimester	22.9		24.4		25.3		
3rd trimester	7.7		10.3		6.5		
No prenatal care	1.0		0.6		1.7		
Unknown prenatal care	5.7		5.4		5.7		
Smoked during pregnancy	1.2		0.6		5.0		**<.0001**
Weight gain during pregnancy	21.7		22.1		24.1		
<16 lb	4.3		5.5		3.5		**<.0001**
≥41 lb	4.3		4.1		4.6		
Maternal medical risk factors	28.6		30.5		32.6		**<.0001**
Preterm birth (< 37 weeks)	11.6		10.7		12.1		**<.0001**

Figure 5.1 Singleton Births by Maternal Age and Nativity, non-Hispanic Blacks, New York City, 2000-2009

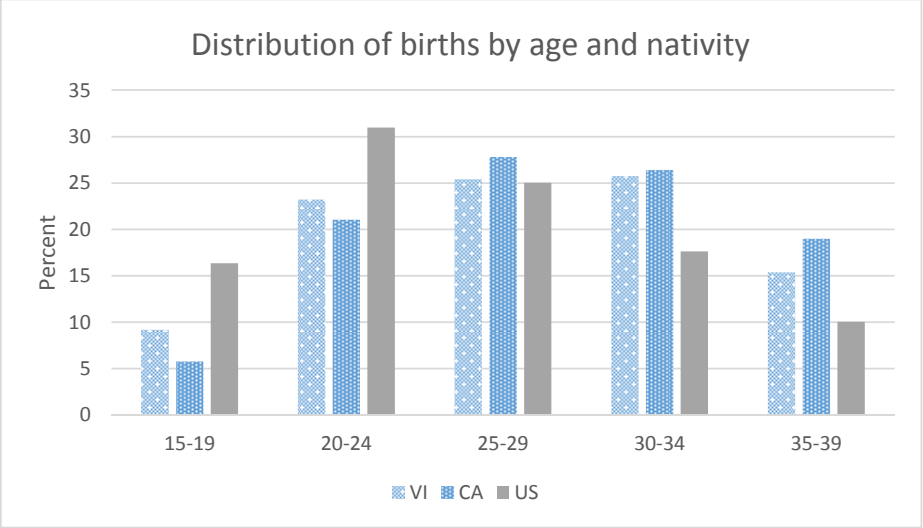


Table 5.2 Number, Percent, and Rate Ratios of Preterm Births by Maternal Age and Nativity, non-Hispanic Blacks, New York City, 2000-2009

	V.I.-born		CA-born		US-born		Rate Ratios	
Age	%	*n*	%	*n*	%	*n*	V.I./US	CA/US
15-19	6.06	6	10.27	485	11.14	2,585	0.54	0.92
20-24	8.33	21	9.26	1,591	10.62	4,664	0.78	0.87
25-29	12.28	35	9.85	2,235	11.66	4,136	1.05	0.84
30-34	11.87	33	11.15	2,403	13.33	3,334	0.89	0.84
35-39	18.07	30	13.19	2,044	16.67	2,374	1.08	0.79

Figure 5.2 Percent Preterm Births by Maternal Age and Nativity, non-Hispanic Blacks, New York City, 2000-2009

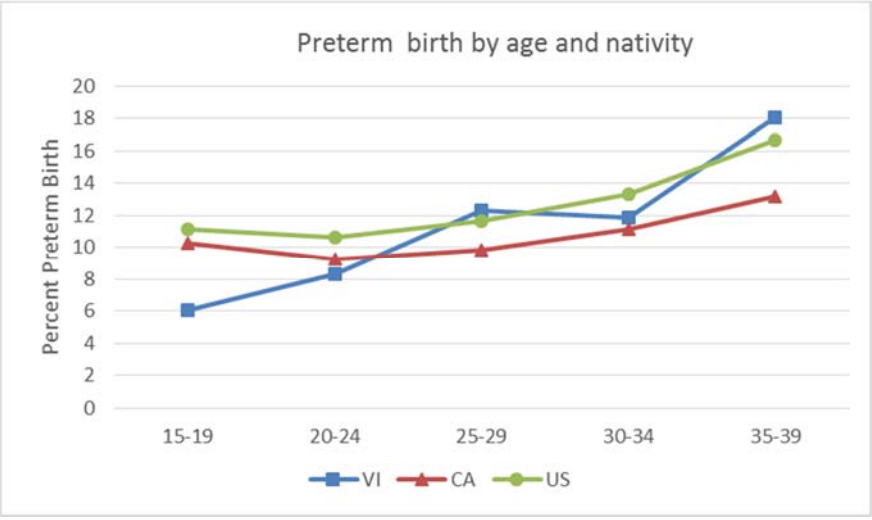


Table 5.3 Odds Ratios for Preterm Birth by Age, Adjusted for Other Risk Factors, Stratified by Mother's Nativity, V.I.-born, Caribbean-born, and U.S.-born, non-Hispanic Blacks, New York City, 2000-2009, Unrestricted Data

	V.I.-born		CA-born		U.S.-born	
	OR	95% CI	*OR*	95% CI	*OR*	95% CI
Model A: *Age, Parity*						
Aged 15-19 years	0.70	(0.27, 1.79)	1.08	(0.97, 1.20)	**1.09**	(1.03, 1.15)
Aged 25-29 years	1.57	(0.88, 2.80)	**1.11**	(1.04, 1.19)	**1.08**	(1.04, 1.13)
Aged 30-34 years	1.52	(0.84, 2.76)	**1.31**	(1.23, 1.41)	**1.25**	(1.19, 1.31)
Aged 35-39 years	**2.51**	(1.35, 4.67)	**1.62**	(1.50, 1.74)	**1.62**	(1.54, 1.72)
Aged 20-24 years (ref.)						
Model B: *Age, Parity, Education, Marital Status*						
Aged 15-19 years	0.65	(0.25, 1.72)	1.04	(0.93, 1.16)	0.97	(0.92, 1.02)
Aged 25-29 years	1.60	(0.88, 2.90)	**1.16**	(1.08, 1.24)	**1.19**	(1.13, 1.24)
Aged 30-34 years	1.53	(0.82, 2.86)	**1.39**	(1.29, 1.49)	**1.44**	(1.37, 1.52)
Aged 35-39 years	**2.58**	(1.34, 4.96)	**1.71**	(1.58, 1.84)	**1.90**	(1.79, 2.01)
Aged 20-24 years (ref.)						
Model C: *Age, Parity, Education, Marital Status, Prenatal Care, Smoking, Medically-related risks*[a]						
Aged 15-19 years	0.72	(0.27, 1.95)	1.09	(0.97, 1.22)	1.01	(0.96, 1.06)
Aged 25-29 years	1.60	(0.87, 2.93)	**1.12**	(1.04, 1.20)	**1.15**	(1.10, 1.20)
Aged 30-34 years	1.56	(0.83, 2.93)	**1.30**	(1.21, 1.40)	**1.35**	(1.28, 1.42)
Aged 35-39 years	**2.51**	(1.29, 4.88)	**1.51**	(1.40, 1.63)	**1.69**	(1.59, 1.79)
Aged 20-24 years (ref.)						
Adjusted 5-year within-group average	**1.31**	(1.09, 1.59)	**1.13**	(1.10, 1.15)	**1.15**	(1.13, 1.17)

Note: Estimates for covariates are not shown. Bolded estimates are significant at $p < .05$ or less.
[a]Medically-related risks include prenatal care initiation, smoking, maternal weight gain during pregnancy, and other medically-related risk factors for preterm birth.

Table 5.4 Odds Ratios for Preterm Birth by Age, Adjusted for Other Risk Factors, Stratified by Mother's Nativity, V.I.-born, Caribbean-born, and U.S.-born, non-Hispanic Blacks, New York City, 2000-2009, First-time Mothers with 12 or More Years of Education

	V.I.-born		CA-born		U.S.-born	
	OR	95% CI	*OR*	95% CI	*OR*	95% CI
Model A: *Age*						
Aged 15-19 years	0.33	(0.04, 2.69)	1.07	(0.91, 1.24)	**1.14**	(1.05, 1.24)
Aged 25-29 years	1.96	(0.79, 4.86)	**1.23**	(1.12, 1.35)	**1.18**	(1.09, 1.28)
Aged 30-34 years	1.82	(0.67, 4.95)	**1.51**	(1.37, 1.67)	**1.36**	(1.24, 1.49)
Aged 35-39 years	**5.05**	(1.76, 14.5)	**1.94**	(1.73, 2.19)	**1.79**	(1.61, 2.00)
Aged 20-24 years (ref.)						
Model B: *Age, Education, Marital Status*						
Aged 15-19 years	0.34	(0.04, 2.85)	1.03	(0.88, 1.20)	1.07	(0.99, 1.17)
Aged 25-29 years	2.19	(0.85, 5.61)	**1.27**	(1.15, 1.39)	**1.29**	(1.19, 1.39)
Aged 30-34 years	1.67	(0.57, 4.93)	**1.57**	(1.41, 1.74)	**1.56**	(1.42, 1.72)
Aged 35-39 years	**5.42**	(1.69, 17.4)	**2.01**	(1.78, 2.27)	**2.10**	(1.87, 2.36)
Aged 20-24 years (ref.)						
Model C: *Age, Education, Marital Status, Prenatal Care, Smoking, Medically-related risks*[a]						
Aged 15-19 years	0.35	(0.04, 3.03)	1.09	(0.93, 1.28)	**1.13**	(1.03, 1.23)
Aged 25-29 years	2.15	(0.81, 5.69)	**1.22**	(1.11, 1.35)	**1.25**	(1.15, 1.36)
Aged 30-34 years	1.77	(0.58, 5.43)	**1.45**	(1.30, 1.61)	**1.45**	(1.32, 1.61)
Aged 35-39 years	**4.00**	(1.16, 13.8)	**1.73**	(1.53, 1.96)	**1.87**	(1.66, 2.11)
Aged 20-24 years (ref.)						
Adjusted 5-year within-group average	**1.64**	(1.13, 2.34)	**1.18**	(1.13, 1.22)	**1.17**	(1.14, 1.21)

Note: Estimates for covariates are not shown. Bolded estimates are significant at $p < .05$ or less.

[a]Medically-related risks include prenatal care initiation, smoking, maternal weight gain during pregnancy, and other medically-related risk factors for preterm birth.

Table 5.5 Categorical Odds Ratios for Preterm Birth by Maternal Nativity and Age, non-Hispanic Blacks, New York City, 2000-2009, Unrestricted Data

	Model 1[a]		Model 2[b]		Model 3[c]	
Nativity and Age Terms	*OR*	95% CI	*OR*	95% CI	*OR*	95% CI
V.I.-born	0.91	(0.76, 1.10)	0.77	(0.49, 1.20)	0.81	(0.38, 1.74)
Caribbean-born	**0.82**	(0.80, 0.85)	**0.86**	(0.81, 0.91)	**0.85**	(0.77, 0.95)
U.S.-born (ref.)	1.0		1.0			
Primiparous, 1	1.00	(0.97, 1.02)	1.00	(0.97, 1.02)	1.01	(0.97, 1.05)
Multiparous, 2 or more (ref.)	1.0		1.0			
Aged 15-19	**1.06**	(1.01, 1.11)	1.06	(1.00, 1.11)	1.01	(0.95, 1.06)
Aged 25-29	**1.10**	(1.06, 1.14)	**1.11**	(1.06, 1.16)	**1.15**	(1.09, 1.20)
Aged 30-34	**1.28**	(1.23, 1.33)	**1.29**	(1.23, 1.36)	**1.35**	(1.28, 1.42)
Aged 35-39	**1.60**	(1.54, 1.67)	**1.68**	(1.59, 1.78)	**1.69**	(1.59, 1.79)
Aged 20-24 (ref.)	1.0		1.0		1.0	
V.I.-born, aged 15-19			0.67	(0.26, 1.72)	0.72	(0.27, 1.94)
V.I.-born, aged 25-29			1.39	(0.78, 2.46)	1.39	(0.76, 2.55)
V.I.-born, aged 30-34			1.14	(0.64, 2.04)	1.16	(0.62, 2.19)
V.I.-born, aged 35-39			1.44	(0.79, 2.62)	1.49	(0.76, 2.89)
CA-born, aged 15-19			1.06	(0.94, 1.20)	1.08	(0.96, 1.23)
CA-born, aged 25-29			0.96	(0.89, 1.05)	0.98	(0.90, 1.06)
CA-born, aged 30-34			0.95	(0.88, 1.03)	0.97	(0.88, 1.06)
CA-born, aged 35-39			**0.88**[d]	(0.81, 0.96)	**0.90**[e]	(0.81, 0.99)
U.S.-born, aged 20-24 (ref.)			1.0		1.0	

Note: Significant terms are bolded. Coefficient and CI cut-offs reflect rounding. All models controlled for parity.
[a]Model 1 is the base parity and age-adjusted model.
[b]Model 2 adds nativity x age interactions.
[c]Model 3 adds controls for marital status, prenatal care, smoking, weight gain, medical risk factors, and all nativity x covariate interactions.
[d]Marginal effect reported. The OR_{CA}/OR_{US} estimate for Caribbean vs. US mothers aged 35-39 is **0.76** (0.71, 0.81).
[e]Marginal effect reported. The OR_{CA}/OR_{US} estimate for Caribbean vs. US mothers aged 35-39 is **0.76** (0.69, 0.84).

Table 5.6 Odds of Preterm Birth by Maternal Nativity and Age, non-Hispanic Blacks, New York City, 2000-2009, First-time Mothers with 12 or More Years of Education

	Model 1[a]		Model 2[b]		Model 3[c]	
Nativity and Age Terms	*OR*	95% CI	*OR*	95% CI	*OR*	95% CI
V.I.-born	0.89	(0.64, 1.25)	0.68	(0.34, 1.33)	0.88	(0.25, 3.08)
Caribbean-born	0.97	(0.92, 1.01)	0.93	(0.86, 1.01)	0.94	(0.81, 1.10)
U.S.-born (ref.)						
Aged 15-19	**1.12**	(1.04, 1.21)	**1.14**	(1.04, 1.24)	**1.13**	(1.03, 1.23)
Aged 25-29	**1.20**	(1.13, 1.27)	**1.18**	(1.09, 1.28)	**1.25**	(1.15, 1.36)
Aged 30-34	**1.43**	(1.33, 1.52)	**1.36**	(1.24, 1.48)	**1.45**	(1.32, 1.61)
Aged 35-39	**1.87**	(1.72, 2.02)	**1.79**	(1.61, 2.00)	**1.87**	(1.66, 2.11)
Aged 20-24 (ref.)						
V.I.-born, aged 15-19			0.29	(0.04, 2.37)	0.31	(0.04, 2.69)
V.I.-born, aged 25-29			1.66	(0.67, 4.13)	1.72	(0.65, 4.57)
V.I.-born, aged 30-34			1.34	(0.49, 3.67)	1.22	(0.39, 3.75)
V.I.-born, aged 35-39			2.82	(0.98, 8.13)	2.14	(0.62, 7.41)
CA-born, aged 15-19			0.94	(0.79, 1.12)	0.97	(0.81, 1.16)
CA-born, aged 25-29			1.04	(0.92, 1.18)	0.98	(0.86, 1.11)
CA-born, aged 30-34			1.12	(0.97, 1.28)	0.99	(0.86, 1.15)
CA-born, aged 35-39			1.08	(0.92, 1.27)	0.93	(0.78, 1.10)
U.S.-born, aged 20-24 (ref.)						

Note: Significant terms are bolded. Coefficient and CI cut-offs reflect rounding.
[a]Model 1 is the base age-adjusted model.
[a]Model 2 adds nativity x age interactions.
[c]Model 3 adds controls for marital status, prenatal care, smoking, weight gain, medical risk factors and all nativity x covariate interactions.

Table 5.7 Odds Ratios for Preterm Birth by Maternal Nativity and Age, non-Hispanic Blacks, New York City, 2000-2009, Unrestricted Data

	Model 1		Model 2		Model 3[a]	
	OR	95% CI	*OR*	95% CI	*OR*	95% CI
V.I.-born	0.91	(0.76, 1.10)	0.63	(0.38, 1.02)	0.68	(0.28, 1.61)
CA-born	**0.82**	(0.80, 0.85)	**0.91**	(0.85, 0.98)	0.90	(0.80, 1.02)
U.S. born (ref)	1.0		1.0		1.0	
Age (5-year increase)	**0.90**	(0.86, 0.94)	**0.89**	(0.85, 0.94)	0.99	(0.94 1.04)
Age^2	**1.05**	(1.04, 1.06)	**1.05**	(1.04, 1.06)	**1.03**	(1.02, 1.04)
V.I.-born x age			1.14	(0.98, 1.34)	1.14	(0.94, 1.38)
CA-born x age			**0.96**	(0.94, 0.99)	**0.97**	(0.94, 0.99)

Note: Significant terms are bolded. Coefficient and CI cut-offs reflect rounding. All models controlled for parity.
[a]Model 3 adjusted for parity, education, marital status, prenatal care, smoking, weight gain, medical risk factors, and all nativity x covariate interactions.

Table 5.8 Slope Estimates for Preterm Risks with Advancing Maternal Age,[a] for V.I-born, Caribbean-born, and U.S.-born Mothers, non-Hispanic Blacks, New York City, 2000-2009, Unrestricted Data

Nativity Group	Age				
	15-19	**20-24**	**25-29**	**30-34**	**35-39**
VI-born	1.16	1.24	1.32	1.40	1.49
CA-born	0.99	1.05	1.12	1.19	1.26
US-born	1.02	1.09	1.16	1.23	1.31

[a]Interpreted as the rate of change within each nativity group across five-year age periods, controlling for all other covariates.

Figure 5.3 Odds of Preterm Births by Maternal Nativity and Age, non-Hispanic Blacks, New York City, 2000-2009

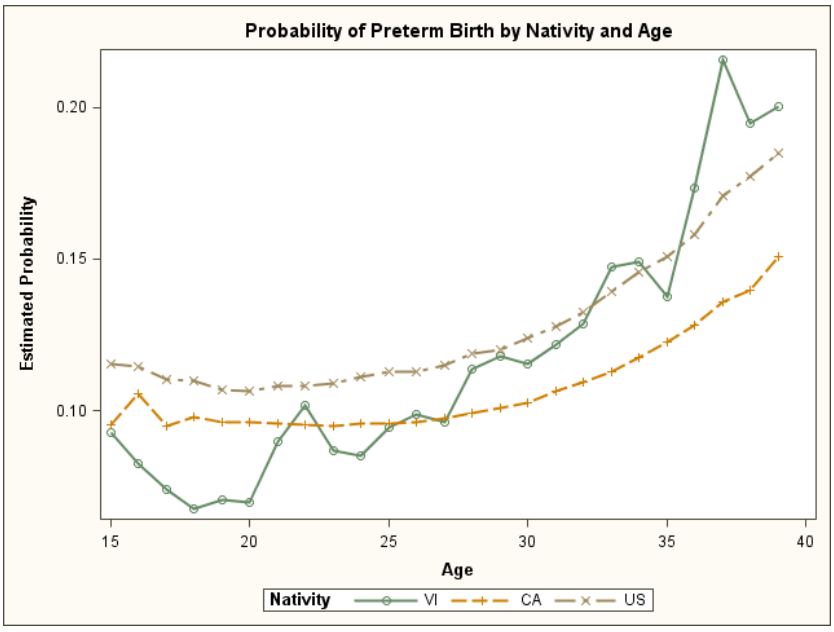


Table 5.9 Odds Ratios for Preterm Birth by Maternal Nativity and Age, non-Hispanic Blacks, New York City, 2000-2009, First-time Mothers with 12 or More Years of Education

	Model 1		Model 2		Model 3[a]	
	OR	95% CI	*OR*	95% CI	*OR*	95% CI
V.I.-born	0.89	(0.64, 1.25)	0.34	(0.14, 0.81)	0.43	(0.10, 1.81)
CA-born	0.97	(0.92, 1.01)	**0.87**	(0.79, 0.97)	0.94	(0.79, 1.12)
	1.0		1.0		1.0	
Age (5-year increase)	**0.91**	(0.83, 0.99)	**0.91**	(0.83, 0.99)	0.99	(0.90, 1.09)
Age^2	**1.05**	(1.04, 1.07)	**1.05**	(1.03 1.07)	**1.04**	(1.02, 1.05)
V.I.-born x age			**1.48**	(1.10, 2.00)	1.38	(0.95, 1.99)
CA-born x age			1.04	(1.00, 1.09)	0.99	(0.94, 1.04)

Note: Significant terms are bolded. Coefficient and CI cut-offs reflect rounding.
[a]Model 3 adjusted for education, marital status, prenatal care, smoking, weight gain, and medical risk factors, plus all nativity x covariate interactions.

Table 5.10 Slope Estimates for Preterm Risks with Advancing Maternal Age,[a] for V.I-born, Caribbean-born, and U.S.-born Mothers, non-Hispanic Blacks, New York City, 2000-2009, First-time Mothers with 12 or More Years of Education

Nativity Group	Age				
	15-19	**20-24**	**25-29**	**30-34**	**35-39**
VI-born	1.41	1.51	1.62	1.73	1.85
CA-born	1.02	1.09	1.16	1.24	1.33
US-born	1.03	1.10	1.18	1.26	1.35

[a]Interpreted as the rate of change within each nativity group across five-year age periods, controlling for all other covariates.

Figure 5.4 Odds of Preterm Births by Maternal Nativity and Age for First-time Mothers with 12 or More Years of Education, non-Hispanic Blacks, New York City, 2000-2009

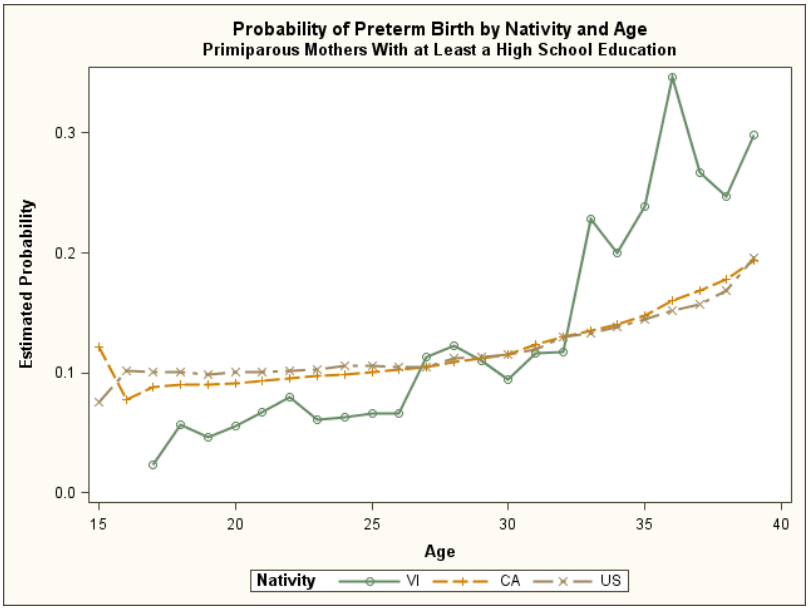


Table 5.11 Preterm Birth by Maternal Nativity and U.S. Migrant Status Interacting with Age, New York City Resident Mothers (2000-2009) Compared to Virgin Islands Resident Mothers (2000-2004), Unrestricted Data

Migrant Status and Age Terms	Model 1		Model 2		Model 3[a]	
	OR	95% CI	*OR*	95% CI	*OR*	95% CI
V.I.-born mothers						
VINY (Migrant)	**0.75**	(0.60, 0.93)	**0.42**	(0.29, 0.62)	**0.42**	(0.19, 0.90)
VIVI (Non-migrant) (ref.)	1.0		1.0		1.0	
Age (5-year increase)	**1.00**	(0.93, 1.08)	**1.94**	(1.38, 2.72)	**1.80**	(1.24, 2.62)
VINY x Age			**0.93**	(0.89, 0.96)	**0.94**[b]	(0.90, 0.98)
CA-born mothers						
CANY (Migrant)	**0.68**	(0.59, 0.78)	**0.55**	(0.43, 0.70)	0.63	(0.39, 1.01)
CAVI (Non-migrant) (ref.)	1.0		1.0		1.0	
Age (5-year increase)	**1.12**	(1.10, 1.14)	**1.27**	(1.12, 1.43)	**1.22**	(1.05, 1.41)
CANY x Age			0.98	(0.95, 1.00)	0.98[b]	(0.96, 1.01)
V.I. and CA-born mothers combined						
NY Residents (Migrants)	**0.65**	(0.60, 0.71)	**0.52**	(0.46, 0.59)	**0.58**	(0.46, 0.74)
VI Residents (Non-migrants) (ref.)	1.0		1.0		1.0	
Age (5-year increase)	**1.11**	(1.09, 1.13)	**1.34**	(1.23, 1.45)	**1.23**	(1.12, 1.35)
NY x Age			**0.97**	(0.95, 0.98)	**0.98**[b]	(0.96, 1.00)

Note: Significant terms are bolded. Coefficient and CI cut-offs reflect rounding. All models controlled for parity.
[a]Model 3 adds controls for education, marital status, prenatal care, smoking, and medical risk factors, plus all nativity x covariate interactions.
[b]Marginal effect reported. The OR_{VINY}/OR_{VIVI} estimate for VI-born migrant vs non-migrant mothers is **0.39** (0.26, 0.66).
The OR_{CANY}/OR_{CAVI} estimate for CA-born migrant vs non-migrant mothers is 0.62 (0.38, 1.01).
The OR_{NY}/OR_{VI} estimate for migrant vs non-migrant mothers (VI-born and CA-born mothers combined) is **0.57** (0.44, 0.74).

Figure 5.5 Probability of Preterm Birth by Migrant Status, Unrestricted Data

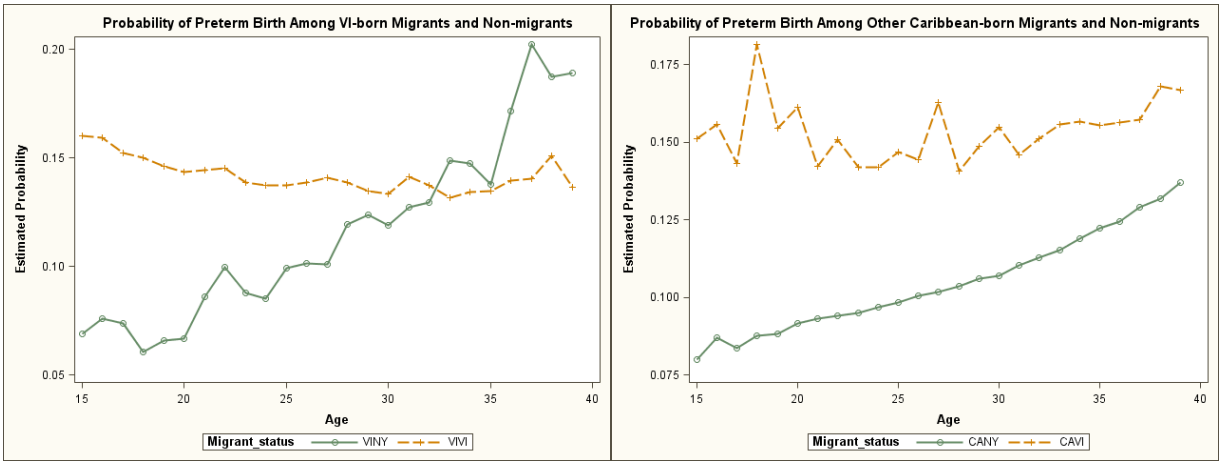


Table 5.12 Preterm Birth by Maternal Nativity and U.S. Migrant Status Interacting with Age, New York City Resident Mothers (2000-2009) Compared to Virgin Islands Resident Mothers (2000-2004), First-time Mothers with 12 or More Years of Education

Migrant Status and Age Terms	Model 1		Model 2		Model 3[a]	
	OR	95% CI	*OR*	95% CI	*OR*	95% CI
V.I.-born mothers						
VINY (Migrant)	**0.58**	(0.39, 0.86)	**0.30**	(0.16, 0.59)	0.95	(0.24, 3.76)
VIVI (Non-migrant) (ref.)	1.0		1.0		1.0	
Age (5-year increase)	**1.23**	(1.04, 1.44)	**2.84**	(1.49, 5.40)	**2.35**	(1.09, 5.06)
VINY x Age			**0.91**	(0.84, 0.98)	0.93	(0.85, 1.02)
CA-born mothers						
CANY (Migrant)	**0.70**	(0.51, 0.96)	0.79	(0.48, 1.30)	1.14	(0.39, 3.28)
CAVI (Non-migrant) (ref.)	1.0		1.0		1.0	
Age (5-year increase)	**1.22**	(1.18, 1.26)	1.11	(0.84, 1.46)	1.00	(0.71, 1.42)
CANY x Age			1.12	(0.97, 1.08)	1.04	(0.97, 1.11)
V.I. and CA-born mothers combined						
NY Residents (Migrants)	**0.64**	(0.54, 0.76)	**0.60**	(0.48, 0.75)	0.95	(0.57, 1.53)
VI Residents (Non-migrants) (ref.)	1.0		1.0		1.0	
Age (5-year increase)	**1.22**	(1.18, 1.26)	**1.31**	(1.11, 1.55)	1.12	(0.91, 1.38)
NY x Age			0.99	(0.96, 1.02)	1.01	(0.97, 1.06)

Note: Significant terms are bolded. Coefficient and CI cut-offs reflect rounding.
[a]Model 3 adds controls for education, marital status, prenatal care, smoking, medical risk factors, and all nativity x covariate interactions.
[b]Marginal effect reported. The OR_{VINY}/OR_{VIVI} estimate for VI-born migrant vs non-migrant mothers is 0.88 (0.21, 3.66).
The OR_{CANY}/OR_{CAVI} estimate for CA-born migrant vs non-migrant mothers is 1.19 (0.40, 3.52).
The OR_{NY}/OR_{VI} estimate for migrant vs non-migrant mothers (VI-born and CA-born mothers combined) is 0.96 (0.58, 1.58).

Figure 5.6 Probability of Preterm Birth by Migrant Status, First-time Mothers with 12 or More Years of Education

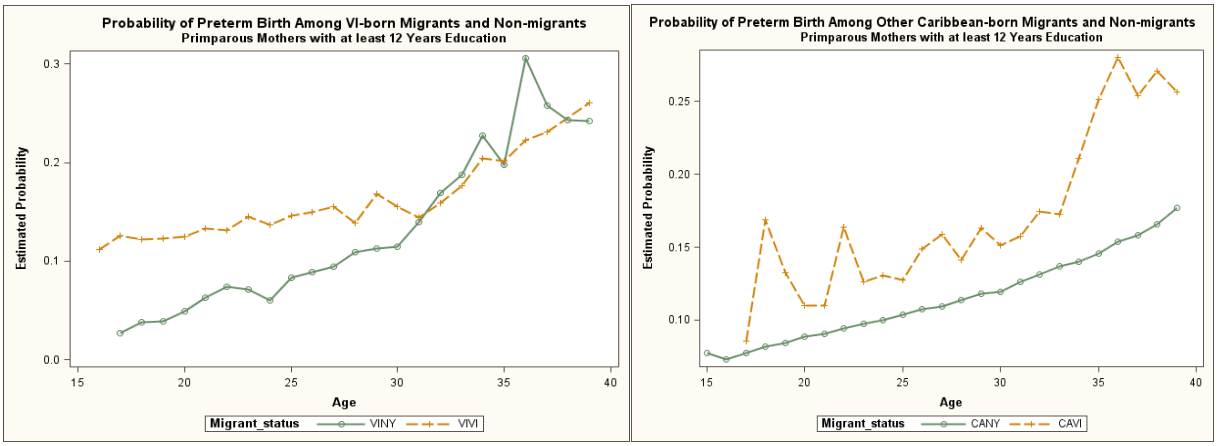


Table 5.13 Maternal and Infant Characteristics of V.I.-born and Other Caribbean-born Immigrants by Migrant Status and Immigrant Duration of U.S. Residence, non-Hispanic Blacks, Singleton Births, New York City, 2008-2010

	Immigrants By Duration of Residence						All Immigrants	Non-immigrants	
	<5 years	5-9 years	10-14 years	15-20 years	≥ 20 years	p-value			p-value
No. of Births	4,890	5,167	4,582	3,403	4,499		22,542	44,782	
Characteristics	%	%	%	%	%		%	%	
Maternal age, years									
15-19	5.7	6.0	3.8	2.5	0.0	<.0001	3.8	13.8	<.0001
20-24	22.4	24.3	19.0	14.7	5.6		17.5	30.0	
25-29	25.1	29.0	31.8	29.4	21.4		27.1	25.6	
30-34	25.0	20.2	27.3	30.4	32.4		26.7	18.2	
35-39	21.9	20.6	18.2	23.0	40.6		24.9	12.4	
Parity									
Primiparous, 1	53.5	48.3	42.9	37.9	32.8	<.0001	43.6	48.4	<.0001
Multiparous, 2+	46.5	51.7	57.1	62.1	67.3		56.4	51.6	
Education									
<12	18.0	19.1	15.2	14.9	8.9	<.0001	15.4	27.3	<.0001
12	39.9	40.8	33.5	25.6	18.3		32.4	26.0	
13-15	30.4	30.9	35.8	39.3	39.0		34.6	31.9	
16+	11.7	9.3	15.6	20.2	33.8		17.6	14.9	
Married	37.4	38.6	41.2	41.9	46.0				
Prenatal care initiation									
1st trimester	35.9	60.3	64.3	67.0	70.0	<.0001	57.6	58.1	<.0001
2nd trimester	24.7	24.6	23.6	22.2	20.7		23.1	25.4	
3rd trimester	32.1	8.1	6.0	4.6	3.8		12.6	7.4	
No/Unknown care	7.3	6.9	6.1	6.3	5.5		6.7	9.1	
Smoked during pregnancy	0.35	0.77	0.74	1.0	1.47	<.0001	0.85	6.21	<.0001
Maternal medical risks	30.9	31.1	31.0	32.4	32.2	.42	31.5	33.6	<.0001
Low birthweight (<2500 g)	8.8	9.1	10.3	10.6	11.7	<.0001	10.2	12.0	<.0001
Preterm birth (< 37 weeks)	9.8	10.6	12.4	12.9	13.2	<.0001	11.7	12.5	.01

Table 5.14 Odds Ratios for Preterm Births Among non-Hispanic Black Caribbean Immigrants by Duration of U.S. Residence, Singleton Births, New York City, 2008-2010

	Model 1		Model 2[a]	
	OR	95% CI	*AOR*	95% CI
5-yr average estimate	**1.10**	(1.07, 1.13)	**1.07**	(1.04, 1.11)
<5 years	1.00		1.00	
5-9 years	1.13	(0.99, 1.29)	1.08	(0.94, 1.24)
10-14 years	**1.34**	(1.18, 1.53)	**1.33**	(1.16, 1.52)
15-20 years	**1.40**	(1.22, 1.61)	**1.33**	(1.15, 1.54)
≥ 20 years	**1.44**	(1.27, 1.64)	**1.30**	(1.13, 1.50)

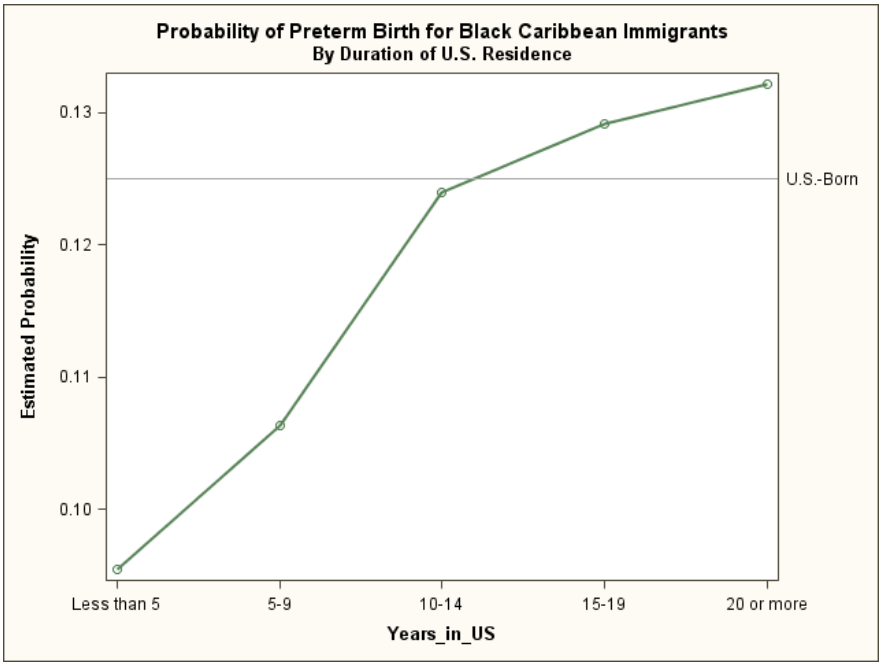
[a]Adjusted for parity, education, marital status, prenatal care, maternal weight gain during pregnancy, smoking, and medical risk factors.

Table 5.15 Odds Ratios for Preterm Births Between Caribbean Immigrants and U.S. Born Blacks Overall, and by Immigrant Duration of U.S. Residence, Singleton Births, non-Hispanics, New York City, 2008-2010

	Model 1		Model 2[a]		Model 3[b]	
	OR	95% CI	*AOR*	95% CI	*AOR*	95% CI
U.S.-born Blacks	1.00		1.00		1.00	
Caribbean Immigrants	**0.93**	(0.88, 0.97)	**0.89**	(0.84, 0.94)	**0.82**	(0.77, 0.88)
Immigrants x Parity					**1.20**	(1.08, 1.33)
Immigrants by duration in U.S.						
<5 years	**0.76**	(0.69, 0.84)	**0.75**	(0.67, 0.83)	**0.69**	(0.59, 0.80)
5-9 years	**0.83**	(0.76, 0.92)	**0.80**	(0.72, 0.88)	**0.74**	(0.65, 0.85)
10-14 years	0.99	(0.91, 1.09)	0.98	(0.89, 1.08)	0.93	(0.82, 1.05)
15-20 years	1.04	(0.94, 1.15)	0.99	(0.89, 1.10)	0.88	(0.77, 1.01)
≥ 20 years	1.07	(0.98, 1.17)	0.97	(0.89, 1.07)	**0.87**	(0.77, 0.98)
15-20 years x first birth					**1.35**	(1.09, 1.68)
>20 years x first birth					**1.38**	(1.14, 1.68)

[a]Adjusted for parity, education, marital status, prenatal care, maternal weight gain during pregnancy, smoking, and medical risk factors.
[b]Adjusted for noted main effects and significant interactions.

Figure 5.7 Probability of Preterm Birth Among Black Caribbean Immigrants By Duration of U.S. Residence



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CHAPTER 6

Conclusion

This dissertation sought to articulate racism as framework through which perinatal health researchers can better understand reasons for persistent racial and ethnic disparities in maternal-infant health that impact Black populations by race (i.e., Black/White disparities) as well as nativity (i.e., U.S.-born Black/foreign-born Black disparities). I defined racism as a system of beliefs and structures that denigrate and disadvantage members of racial groups who are socially categorized and regarded as inferior. Racism was postulated to exert influence through various mechanisms starting at the structural level and impacting social, psychological, and biological factors to harm maternal, fetal, and infant health. Although the systemic nature of racism defies direct epidemiological study, I proposed an integrated model of racism-associated constructs and pathways that are plausibly testable and could lend insight into perinatal health disparities that are currently unexplained by routinely-measured socioeconomic, behavioral, or medical risk factors.

In the absence of data to directly examine racism-related constructs in association with birth outcomes in the study population, I completed detailed analyses to more fully understand the extent through which standard risk factors could explain reduced rates of preterm birth for non-Hispanic Black immigrant populations relative to U.S.-born Blacks. Guided by my conceptual analysis and model, 'risk factors' such as race, nativity, income, education, and medical care were examined as socially-patterned constructs influenced significantly by racism. The persistent, unexplained gradients that were observed within Black populations by nativity

and migrant status echoed the need for greater exploration of background contexts. Moreover, the contrasting patterns of risk by national origin and by duration of U.S. residence raised questions about the "healthy immigrant" label as applied to Black Caribbean immigrants.

In Chapter 2, I conducted a literature review which synthesized the state of knowledge concerning racial disparities in birth outcomes. Despite varying suggestions and targeted studies of potential leading causes for racial disparities in birth outcomes, there are no distinct reason(s) that can be accepted definitively based on current scientific evidence. My aim was to reconcile the research in the currently divergent fields of racism and health, perinatal health, and immigrant health in order to understand the enigmas of race and nativity-based disparities in birth outcomes. The bulk of studies reviewed in this dissertation that addressed causal factors for racial/ethnic perinatal health disparities impacting Black populations in the U.S. have highlighted the social-environmental context, largely driven by a Centers for Disease Control and Prevention (CDC) initiative that propagated studies investigating the social contexts for Black women that could elevate risks for preterm birth, augmented by community studies that have examined racial segregation and other neighborhood socioeconomic contexts in association with adverse birth outcomes. The exposure that the reviewed papers have contributed to the field is encouraging, but the degree to which enlightened perspectives have carried over into other studies is debatable.

In the conceptual literature, causal factors for racial disparities were commonly viewed as stemming from social-environmental exposures mediated by stress-induced biological and genetic mechanisms that threaten maternal and fetal health (Rowley et al., 1993). Among empirical investigations, the most direct attribution to racism as a contributing cause of perinatal health disparities appeared in studies examining either perceived discrimination and birth outcomes or racial segregation and birth outcomes. Studies of psychosocial stress as a cause of

racial disparities have also embraced racism-focused conceptualizations, which can be aptly described as a 'racism-stress' framework (Dominguez, 2011; Giscombe & Lobel, 2005; Hogue & Bremner, 2005). The racism-stress framework has influenced epigenetic studies that characterize the stressful nature of disadvantaged environments that can influence gene expression (Fiscella, 2004; Menon, Dunlop, Kramer, Fortunato, & Hogue, 2011; Morello-Frosch & Shenassa). Specifically, African Americans are alleged to have unique epigenetic susceptibilities that are intensified by cumulative psychosocial and physical health-damaging exposures over the lifecourse (Burris & Collins, 2010).

The review additionally explored literature relative to immigrant Black populations in the U.S. that could lend insight into our understanding of disparities affecting native African Americans. Underlying the general observations of a health advantage for immigrant populations were some telling observations. Mirroring the pattern of Black/White disparities in the U.S.-born population, Black Caribbean immigrants have higher rates of LBW and preterm birth than White immigrants. This represents a clear racial disparity. However, disparities impacting Black immigrants have rarely been explored because the dominant research focus has been on the 'healthy immigrant' advantage for foreign-born Blacks relative to African Americans. Another understudied observation is that the immigrant health advantage deteriorates fairly rapidly over time for Black Caribbean immigrant mothers but remains unchanged or declines more modestly for White immigrant mothers. Moreover, ethnic density has not shown a protective effect for U.S. Black immigrants unlike the advantages shown for Hispanic and Asian immigrants. Further examination of racial disparities that impact Black migrants in the U.S., particularly persons of West Indian and Puerto Rican ancestry, could potentially offer insights into Black/White disparities among the U.S.-born. The peculiar negative impact of Black racial designation in the

U.S. for immigrant *and* native populations suggests a shared influence of racism on perinatal health that calls for deeper examination in the literature.

In Chapter 3, building on previous conceptualizations and findings, I proposed an integrative model that situates racism as a fundamental cause of adverse birth outcomes for U.S.-based Blacks, both native and foreign-born (Figure 3). The model outlined pathways for structural, interpersonal, and internalized racism; depicted psychosocial and biological racism-associated stress responses; and highlighted protective individual and community-based responses to racism-associated stress. Racism, not race *per se*, was viewed as the genesis for race and nativity disparities in birth outcomes. The current perinatal health disparities literature has an undercurrent that views 'race' from a genetic stance. This orientation seems apparent with discussions that seek to reject genetic explanations for Black-White differences by claiming that foreign-born Blacks should hypothetically have health risk profiles that are similar to African Americans (and different from Whites) and, therefore, the observed nativity differences within Black populations should disprove race (i.e., genetics) as the source for racial disparities. Ironically, such arguments reify genetically-based arguments by presuming that U.S. and immigrant Blacks should have similar genes due to their racial designation. But birth outcome similarities and difference, whether within or across races, may have nothing to do with genes at all, but should serve to highlight the influence of differential social contexts between the groups. For example, while foreign-born Blacks do fare better in perinatal health outcomes than U.S.-Blacks, only Africans are more similar to Whites, while Caribbean Blacks maintain race-based disparities in comparison to both foreign-born and U.S.-born Whites.

Why would African immigrants, Caribbean immigrants, and African Americans be so disparate? Genes are unlikely the answer except to the extent that external contexts over long periods are so influential as to modify gene expressions (i.e., epigenetic modifications). Race is

not inconsequential, however, insofar as structured racial and ethnic social contexts shape the trajectory for racial/ethnic groups by normatively structuring opportunities and constraints over the lifecourse (Geronimus, 2001). The social assignment is just as important based on race for African Americans as it is for race/ethnicity among Black immigrants who must endure subordinate positioning as a racial minority which makes their immigrant 'ethnic' identification relatively subjugated as well (Ford & Harawa, 2010; Pearson, 2008).

Considering the acknowledged importance of race and racism for both U.S.-born and foreign-born Blacks, I included expanded constructs of racism, including structural racism, to encourage examination of how racist structures impact the convergence, or divergence, of the social and health trajectories for immigrant and native Blacks that could lend insight into perinatal health disparities. This study adds to earlier racism-focused models that outlined integrative racism-stress conceptualizations, yet were absent of structural domains (Hogue & Bremner, 2005). Dominguez (2009) made advances with her multidimensional conceptualization of racism and birth outcomes that highlighted the structural domain. My conceptualization advances even further and calls attention to racialized policies that have structured the number and descriptions (e.g., preferred sending countries, occupations, social classes, genders, neighborhood residence, phenotype-language) of Black immigrants *vis à vis* other immigrant groups (Kim, 2007). I also proposed how neighborhood social contexts can predict birth outcomes directly and as mediators of individuals' stress and coping styles and resources. My conceptualization of "neighborhood social dynamics" gave preference to health-promoting contexts such as collective efficacy, community empowerment, and the durability and resourcefulness of social networks which represent meaningful social processes in Black communities.

Racism as a foundational framework for understanding Black health disparities is particularly salient because of the stress that it engenders – a key mediator between structural racism and its perinatal health impacts. Accordingly, I conceptualized how differential experiences and coping responses to structural racism and race-related stress could contribute to the nativity differential in birth outcomes among Black women. This stress can have a basis in structural disadvantages at the societal level, whether consciously perceived or not, that curtail health-promoting resources and opportunities. An underlying framework for the proposed research is that African Americans and Caribbean-born Blacks have qualitatively different meanings and perceptions of racial identity. These distinctions of racial identity are predicted to generate more favorable health profiles for Caribbean Blacks in response to racism-related stressors. Racial identity influences the appraisal of racism-related stressors and the coping responses employed to deal with those stressors. It is anticipated that the frequency and types of exposure to racial discrimination will not significantly differ by nativity, but cross-cultural differences in racial identity and coping styles may generate responses to racism that are either protective or pernicious. In this manner, racial identity may partially explain inter-ethnic differences in birth outcomes. Examining racial or ethnic identity should simultaneously considering the influence of immigrant and generation status, neighborhood characteristics such as socio-demographic concentration, class, and gender roles or identities. More studies are warranted to unearth the interactions of racism, stress, and the immigration experiences of Caribbean Black women that could explain the Black nativity difference in birth outcomes.

The link between racism and birth outcomes is further mediated through physiological mechanisms. In line with current research, my conceptual model highlights both allostatic load and corticotrophin releasing hormone (CRH) as markers for the biological mechanisms that increase risks for birth outcomes. Research suggests that CRH may be a more valid biochemical

marker of maternal-fetal health than allostatic load which has been studied more often in relation to chronic diseases. The extent to which higher chronic disease rates exacerbate perinatal health *disparities* remains an empirical question. Higher rates of both essential and pregnancy-related hypertension and diabetes do occur in African Americans, but there has been no definitive link with these medical conditions and persistent racial disparities in LBW, PTB, or IM. Although chronic disease conditions have a lower prevalence in foreign-born Black mothers, they have not emerged as primary explanatory factors in either racial or nativity-based disparities in birth outcomes.

In Chapter 4, I conducted an empirical study to examine the extent to which nativity, migration, and a range of traditionally measured risk factors could explain differential risks of preterm birth among Virgin Islands-born, Caribbean-born, and U.S.-born Black women both in New York City and in the Virgin Islands. The interaction of nativity, migration status, and geo-racial context was conceptualized to represent varied racism-related exposures that would translate into differential maternal health and birth outcomes. In this study, the unadjusted odds of preterm birth for non-Hispanic Black Caribbean immigrants vs. non-Hispanic U.S.-born Blacks women was 0.88 (95% CI: 0.85, 0.90). This estimate remained fairly stable after adjusting for sociodemographic, behavioral, and medical risks (*OR* 0.85, 95% CI: 0.76, 0.94). However, medically-related factors were not the primary determinants of the preterm birth advantage for Caribbean immigrants. There was no protective influence of medical factors for foreign-born women; and, therefore, it would be incorrect to assume that Caribbean-born women have superior physical health status in relation to pregnancy. Although smoking rates were substantially low among Caribbean women, smoking status did not emerge as a factor that conferred added protection for Caribbean women against preterm birth when evaluated together with demographic factors that appeared more risk protective in the models.

Demographic factors had the strongest influence on the preterm birth advantage for Caribbean women based in the U.S. compared to African Americans, driven largely by population differences in age and education. However, a college education did not confer the expected advantages for Caribbean immigrants relative to African Americans. Caribbean women with less than 12 years of education had lower predicted odds of preterm birth than African Americans. Age was the only predictor that substantively modified the impact of nativity status on preterm birth, particularly among women aged 35-39. Except for age, the range of other independent predictors typically analyzed in birth outcomes studies did not appear to explain the protective effect of nativity for Caribbean-born mothers. Rather, there is a 10% variance in preterm birth that can be attributed to unaccounted background factors between Caribbean immigrants and African Americans. Among these currently unexamined factors may lie one or more determinants of disparate risks with substantive explanatory power that could be considered for more routine analysis when evaluating nativity-based disparities.

Analyses comparing foreign-born Black mothers in New York City and the U.S. Virgin Islands provided some support for the theory of U.S. immigrants being selectively healthier than their non-migrant counterparts. Caribbean-born non-Hispanic Black mothers in the Virgin Islands had more teen births, lower educational attainment, lower marital rates, and higher rates of hypertension and preterm birth than their migrant counterparts in New York. Age and education were effect modifiers that contributed significantly an 84% increased odds for preterm birth for Caribbean-born residents in the Virgin Islands in comparison to their migrant counterparts in New York City (*OR* = 1.84, 95% CI: 1.15, 2.94). A related but publicly understated finding, is the fairly rapid deterioration of this health advantage for Black Caribbean immigrants relative to African immigrants and other immigrants to the U.S.

No previous studies have attempted an analysis of weathering and preterm birth with a focus on foreign-born Blacks. Hence, in Chapter 5, I examined if and how weathering operated for V.I.-born, Caribbean-born, non-Hispanic Black mothers residing in New York in comparison to U.S.-born non-Hispanic Black mothers in New York. Findings indicated that age-graded patterns of preterm birth were fairly similar between Caribbean-born and U.S.-born residents in New York; hence, there was no weathering by Black nativity status. Age-related effects were therefore highly conditional, but the overall conclusion supported no differential weathering pattern between Caribbean-born immigrants and African Americans. Additionally, there was no statistical evidence of differential age-related risks in preterm birth by migrant status; hence, Caribbean-women residing in the Virgin Islands were no more 'weathered' than their migrant counterparts in the U.S. A conditional finding is that women from the Virgin Islands demonstrated significant maternal health and preterm birth disparities that were persistent across the reproductive age spectrum. However, the V.I.-born sample was small and inadequately powered for most analyses. One can conclude that weathering was present for Caribbean-born Black women; however, an effect modification was demonstrated for Caribbean-born migrants aged 35-39 signifying a lower probability of preterm birth than would be expected at later ages for Black women. Although the effect modification by age for Caribbean-born women 35-39 was readily apparent in models highlighting interaction effects across categorical age ranges the effect was more muted in models estimated for continuous age interactions and the magnitude of effect for the age interaction was negligible. This could explain why no sustained weathering pattern was found. There could be a unique aspect to the 'weathering' pattern for Caribbean immigrants that must be explored further. Findings also indicated that differential age-related risks did not explain preterm birth disparities by migrant status.

Lastly, this study found evidence that the immigrant health advantage is attenuated with exposure to the U.S. context, which was conceptualized in this dissertation as a marker for racism exposure. For Black Caribbean women, the odds of preterm birth increased 7% for every 5 years of U.S. residence with adjustment for age, medical risks and other maternal-infant risk factors. Moreover, the predicted risk of preterm birth approached the levels for native African Americans after 10 years of residence. These findings are notable in light of studies that observe relative declining health for African immigrants after 20 years of U.S. residence and no substantive health deterioration for White European immigrants. These results lend support for cumulative health disadvantage for Caribbean immigrant mothers related to harmful background contexts that are have not been empirically reported in current literature.

Limitations. The above findings for the noted immigrant and U.S.-born group comparisons provided unique insight into the variability within Black populations that defies common generalizations and calls for additional exploration of background factors for a more refined understanding of adverse birth outcomes in Black women. However, this dissertation study has some limitations. Previous explanations for nativity differences have focused on socioeconomic and behavioral health differences between immigrant Blacks and African Americans. This study was not able to test socioeconomic data except for mother's educational level. Inclusion of proxy SES indicators such as Medicaid coverage or median neighborhood income was planned, but these analyses could not be conducted due to changes in how health insurance coverage was categorized in the database across the cohort periods and due to mispecified coding of census tracts such that a large proportion of cases could not be matched to a valid residence.

A statistical concern was the relatively small number of V.I.-born immigrants (n = 1,080) that were pooled for this study. A power analysis assessed 12% power to detect differences

between V.I. migrants in New York and African Americans. Accordingly, it may have been best to collapse the V.I. and Caribbean groups in New York which would have simplified the analysis, although the study questions would have to be reframed accordingly. Had I collapsed the V.I. and other Caribbean populations, I could not have compared a migrant population (e.g., Virgin Islands'-born migrants to New York) with their "pure" counterparts in the Caribbean (i.e., Virgin Islands' natives in the Virgin Islands), but instead, I would have had to consider more generalized regional similarities by comparing Caribbean migrant populations in New York with a generalized Caribbean population based in the Virgin Islands. Such a comparison, while preferable for statistical reasons, is problematic largely because of the economic and political standing of the U.S. Virgin Islands relative to other Caribbean islands that would presumably make V.I. migrants to the U.S. mainland quite distinct from other Caribbean migrants to the U.S. For instance, the passage to and from the U.S. for V.I.-born natives is unrestricted by virtue of the U.S. citizenship, and the economic "push" factors for migration among V.I. migrants are probably not as intense as for other Caribbean migrants.

My rationalization for keeping V.I. and other Caribbean populations separate for the analysis was not to suggest that V.I. migrants in New York and their counterparts at home are homogeneous groups except for migrant status. Migrant studies have observed distinct differences between people who voluntarily migrate (not including political refugees) and those who remain at home. With some exceptions, people who migrate are typically more "advantaged," whether it is psychological or socioeconomic. Psychological advantage refers to the enhanced self-efficacy and resilience of those who choose to 'take the plunge' to move away from their family, culture, and native land. While a significant reason for migration is to accrue economic advantages, many migrants move *because* they are more economically advantaged. The "costs" of migration, and the associated material and psychological resources required of

migrants, increase relative to geographic distance between the sending and receiving countries. Relatively speaking, it is easier to migrate from Mexico to the U.S. or from the Caribbean to the U.S., than it is to migrate from Africa to the U.S. The socioeconomic advantages of African migrants to the U.S. have been cited as a reason for the more favorable health profiles for African immigrants compared to Caribbean immigrants. In fact, African migrant birth outcomes more closely approximate the outcomes for U.S. Whites than for Black immigrants or U.S. Blacks (Urquia et al., 2010). African migrants to the U.S. are deemed a truly unique group, not representative of their non-migrant counterparts in Africa or Black migrants from countries outside of Africa. The significant educational and economic advantages alone are an important consideration for health.

My study presumed that there are unique psychosocial contexts that could potentially explain disparities in birth outcomes between foreign-born and U.S.-born Black women. Differential experiences and coping responses to racism-related stress are thought to contribute to the nativity differential in Black maternal and infant health in the U.S. and emerge as a critical pathway for understanding the enigma of racial disparities in birth outcomes. For example, individual traits such as psychological strengths or resilience could be more prevalent among women who choose to voluntarily migrate. These traits may serve to buffer to the ill-effects of racism and could in part explain the immigrant health advantage. However, it is also possible that Caribbean-born mothers who migrate to New York City have a unique health advantage due to factors not related to nativity. Some of this protection could be related to *a priori* supreme health or fertility status of Caribbean migrants—evidenced by the large proportion of mothers aged 35 and older who are Caribbean-born. This finding would also support an immigrant selectivity component to Caribbean-born women in New York City. Subgroup analyses did not bear this out for Virgin Islands women, however.

Recommendations. Overall, this dissertation aims to spur more rigorous examinations of racism-associated constructs and encourage context-focused interventions to optimize African American and Black immigrant health. There is a sizeable gap in the current research which calls for better explication of how racism influences birth outcomes in both African American and Black immigrant populations. Except for neighborhood-based studies that examine racial segregation and indices of socioeconomic disadvantage, only a handful of empirical studies explicitly study racism and birth outcomes. Almost all rely on measures of perceived interpersonal racism, which is a helpful construct, but limited in its explanatory power. My review of the studies in this area revealed wide variability in how basic measures of everyday discrimination are used. A call is needed among the cadre of researchers in this area for more consistency of measurement to improve validity and reliability of measurement and aid in generalizing the findings regarding how discrimination is associated with birth outcomes (Blank, Dabady, & Citro, 2004).

A blatant gap in this area of research is the study of how racism impacts birth outcomes in foreign-born Blacks. Several research commentaries acknowledge racism-related factors as determinants of maternal-infant health for Black immigrants (Collins, Soskolne, Rankin, & Bennett, 2013; Collins, Wu, & David, 2002; Dominguez et al., 2009; Elo & Culhane, 2010; Elo, Vang, & Culhane, 2014; Liu & Laraque, 2006; Pallotto, Collins, & David, 2000; Rosenberg, Desai, & Kan, 2002; Singh & Yu, 1996; Stein et al., 2009). However, only one published study to date has explored racism in a perinatal population of Black immigrant women in the U.S. (Dominguez et al., 2009). Although that study did not measure birth outcomes, it made a critical contribution to the currently sparse literature and articulated views echoed in this dissertation regarding the importance of racism to Black immigrants. As the field of discrimination and perinatal health research develops further, it will bode well to widen its examination of context

measures. Future studies are needed that examine structural and interpersonal exposures to racism-related stressors as mediating factors in Black U.S.-born/foreign-born differences in birth outcomes. A welcome trend in discrimination studies would be the addition of measures to ascertain how Black women *cope* with racist experiences and how this may moderate intermediate outcomes related to stress and risk behaviors (Ertel et al., 2012). In the interim, until more sophisticated data collection and methods are developed for measuring context variables, qualitative studies can be very helpful to unearth the interactions of racism, stress, and the immigration experiences of Caribbean Black women that could explain the Black nativity difference in birth outcomes.

I offer several recommendations for additional research to refine studies that examine racism as it impacts both African American and Black immigrant health. First, the transitory nature of the immigrant health advantage for Black Caribbean immigrants in the U.S. will continue to be enigmatic without more attention to social context. The present study suggested that the healthy immigrant effect for Caribbean migrants is not driven by socioeconomic indicators such as maternal education. More studies are needed to refine constructs regarding assimilation and culture that may contribute to the healthy birth advantage for immigrants. A solution to this knowledge deficit would be to conduct qualitative research to enhance theory-generating to understand more about the cultural component of the healthy immigrant advantage for Black immigrants, and the process of acculturation amidst racial adaptation.

Another recommendation stemming from the findings in this research is the need for interdisciplinary study of the impact of racism on health, racial and ethnic health disparities, and immigrant health to better integrate knowledge regarding the complex interplay of mechanisms leading from racism to maternal-fetal health. Moreover, a critical consideration is the need to focus not only on improving perinatal health generally, but to specifically focus our research and

interventions on the *disparities* that are observed (Wise, 2003). History demonstrates that racial disparities can worsen when racial inequities are not tackled directly. An example was the introduction of surfactant therapy to improve neonatal survival which resulted in African American babies attaining less access and benefit from this therapy than White infants, and in the process, losing the survival advantage that had been observed for Black infants for some time (Rowley & Hogan, 2012). With the current emphasis on preventing preterm births, much enthusiasm has been generated regarding the use of progesterone therapies to prevent preterm labor. But perinatal health practitioners should be cautious lest this and other medically-focused interventions widen Black/White disparities related to social and healthcare access barriers. Similar to the caution that I implored related to genetic causes as an explanation for racial disparities in preterm birth, it is important not to lose site of the social causes that fundamentally contribute to disparities (Link & Phelan, 1995; Phelan, Link, & Tehranifar, 2010) among the range of medically-focused that are proposed.

Implications. The examination of racism as a fundamental cause of perinatal health disparities has important practical and policy considerations. Although addressing fundamental causes is necessary for reducing perinatal disparities (David & Collins, 1991; Geronimus, 2000), public health and perinatal health researchers are less likely to view contextual factors, including racism, within the purview of professional intervention. The multidimensional nature of my conceptualization offers avenues for ameliorative intervention outside of the structural realm. Importantly, my model highlights individual and community responses to racism as potentially *protective* factors that can moderate the negative effects of racism and reduce perinatal risks. If it can be demonstrated that there are modifiable intrapersonal or community-based supports that will buffer the impact of racism, these factors can be encouraged via individual or group interventions (e.g., stress regulation programs) of women during the perinatal period, and also in

association with general health promotion throughout the reproductive ages. Targeted interventions can also be fostered via healthcare or community grant programs to benefit pregnant women and families. The Centers for Disease Control and Prevention has embraced proposals supportive of social determinants of health interventions. Also, the National Institutes of Health has been supportive of interdisciplinary research of socially-mediated factors impacting health. These and other resources forecast favorable support and scholarly development in this field.

Summary. In this dissertation, I proposed racism as a framework for understanding both inter-racial (Black/White) and inter-ethnic (U.S.-born Black/foreign-born Black). My empirical analyses demonstrated that weathering patterns were not significantly different between Black Caribbean immigrants and African Americans. Moreover, the preterm birth advantage for Black immigrants was not sustained with increasing duration of U.S. residence, controlling for concurrent health risks. These findings lend some support to my thesis that both U.S.- and foreign-born Black women may be negatively impacted within a structured context of racial disadvantage. If it can be shown in future studies that racial identity, racism-related stress, and coping styles are also influential toward determining health status in both U.S.-and foreign-born Blacks, these contributions would offer alternative constructs to broaden the purview of social and structural investigations within perinatal health research.

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APPENDICES

Appendix A:

Chapter 2 Reference Tables

Table A.1 Summary of Reviews and Conceptual Papers Addressing Racial Disparities in Birth Outcomes

Reviews/Conceptual Papers

Author, Year (references Chap. 2)	**Birth Outcome**	**Main Explanations or Frameworks**	**Emphasis on Racism** --none; + low; ++ med.; +++ high
Institute of Medicine, 1985	LBW	**General** (Review) Emphasis on risk factors of LBW including Black race, low income and education, and a number of medically-related risks; acknowledged more research needed to understand why Black race is a 'risk factor'; significant emphasis on prenatal care education and contraception as preventive interventions	**--**
Samuels, 1986	IM, LBW	**General** (Review) Literature review of IM and LBW among minority groups in the US; written to accompany the first US government report on Black and minority health disparities Socioeconomic status and access to healthcare received the most policy-related attention. Stress mentioned slightly as research possibility.	+ Discrimination identified in the review but not in the report's key findings or recommendations
Kramer, 1987	LBW	**General** (Review) Review of 67 studies from 1970 to 1984 addressing disparities (includes European literature) 43 factors identified across studies, categorized (by the author) under: genetic & constitutional; demographic & psychosocial; obstetric; nutritional; maternal morbidity; toxic exposure; antenatal care The most important factor contributing to disparities (in developed countries) was cigarette smoking followed by nutrition and pre-pregnancy weight. Most studies compared differences in mean birthweight which was concluded as insufficient to suggest an independent genetic contribution of 'race' or ethnicity. Racial/ethnic origin was of many causal factors investigated – intended to rule out possible genetic differences.	**--**
Emanuel et al., 1989	LBW	**Lifecourse Perspective** A mother's socioeconomic environment during pregnancy *and* childhood influence pregnancy outcome.	**--**
Yankauer, 1990	IM	**Social Context** Provided a historical perspective on understanding the mechanisms of racial disparities in IM.	+ Social disadvantage
David & Collins, 1991	Adverse Birth Outcomes	**Racism** Called for shift in focus from 'race' to 'racism' to better understand disparities Racism central to explaining disparities.	+++ Racism is significantly responsible for the disparities
Geronimus, 1992	IM	**Social Context** Weathering framework; Black women experience accelerated aging and health deterioration during reproductive years; infant mortality disparities are related to health status differences and age-variation of these differences	++ Weathering is a "cumulative pattern of racism." [Referring primarily to structural inequality.]
Berkowitz & Papiernik, 1993	PTB	**General** (Review) General review of PTB Authors highlighted racial differences in economic, psychosocial, environmental, and medical factors (acknowledging these cannot be readily controlled in epidemiologic studies). They also acknowledged "psychological stress stemming from social deprivation," but summarized that the research findings on stress as a factor in PTB disparities were conflicting and inconclusive.	**--** Although "stress stemming from social deprivation" could be inferred as relating to racism, a racism context was not emphasized by the authors

Reviews/Conceptual Papers

Author, Year (references Chap. 2)	**Birth Outcome**	**Main Explanations or Frameworks**	**Emphasis on Racism** --none; + low; ++ med.; +++ high
Blackmore et al., 1993	PTB	**Social Context-Stress** Conceptualized a stress model with interactions of social, environmental, and medical factors unique to women of color Rejected race as a risk 'factor'; race described is a risk 'marker' for stress which can stimulate preterm delivery through various mechanisms	+ Racism mentioned as a chronic stressor
James, 1993	IM, LBW	**Social Context** Conceptualized interaction of cultural, psychological, and economic factors – a 'structurally-rooted biopsychosocial process.' Racism conceptualized as a fundamental cause. Called for more attention to "structural factors" (poor housing, poor nutrition, violence, discrimination due to race/ethnicity/ gender); structural factors deemed responsible for intractable disparities.	++ Reference to structural racism: psychological and economic marginalization by mainstream US culture
Rowley et al., 1993	PTB	**Biopsychosocial** Reducing racial PTB disparities requires understanding the problem in Black women. PTB was addressed as a sociobiological problem. Social behavior and culture, history, political, and economic forces influence disease with the intersection of gender, race, and class.	++ Racism mentioned as a psychosocial stressor
Rowley, 1994	VLBW, PTB	**Social Context-Racism-Stress** Discrimination is an important stressor that should be addressed toward reducing excess deaths in outcomes among African Americans Prevention research must consider social, cultural, political context of African American women Understanding of protective factors needed as well	++
Wise, 1993	IM	**Integrative** IM framed as both a technical and a social concern. Author attempts to reconcile the 'disciplinary antagonism' toward a coherent public understanding and policy direction to address IM. Proposes that an analytic approach should: "…distinguish between the causes of IM and the causes of disparities in IM"; be linked to intervention; embrace the social and the biological without artificial separation; have implications for local action and social policy; be simple and amenable to a range of disciplines.	+ Need to better address social context of race
Hogue & Hargraves, 1995	PTB	**Social Context** (Review) Focus on causes and prevention of PTB within the African American community from the perspective of their cultural traditions and social environments; "Little is known about how African American women translate these barriers from within their own cultural traditions, and social environments" (260).	++ Stressors caused by the long history of structural barriers and discrimination
Paneth, 1995	LBW	**Social Context** Variation in birthweight described as both pathological and non-pathological. Need to 'decode the biological expression of social stratification' (influenced by Wise, 1993).	+ Stated that the roots of inequality for African Americans are social and economic
Fiscella, 1996	PTB	**Biological** (Review) Review of infections related to racial disparities in preterm birth	

Reviews/Conceptual Papers

Author, Year (references Chap. 2)	**Birth Outcome**	**Main Explanations or Frameworks**	**Emphasis on Racism** --none; + low; ++ med.; +++ high
Fuller, 2000	LBW	**Biological** Racial disparities due to lower Vitamin D synthesis associated with heavy pigmentation; alters calcium levels which is a risk for IUGR, premature labor, and hypertension.	**--** Counters racism as a key explanation
Hogan, Njoroge et al., 2001	Perinatal health disparities	**Social Context** Need to shift from the individual factors to the larger social context. More intra-group studies are needed to understand variability *within* populations and why some groups have healthier outcomes.	**--**
Hogan, Richardson et al., 2001	PTB	**Social Context-Stress** Chronic stress is internalized with altered stress reactivity. Recommended public health framework: address social context of PTB and chronic stress; address structural challenges to eliminating disparities; preventive health for all women of reproductive age; improve quality of prenatal care; increased collaboration to address social factors	++ Disparities attributed to experiences of living in the US
Rowley, 2001	PTB	**Biopsychosocial** Integrative examination of biologic factors, psychological factors related to stress, and the social and political impact of being Black in the US	++ Systemic racism: "…structural burdens of systematic racial inequality"
Hogue, 2002	Black Women's Health Disparities	**Biopsychosocial** Presented agent-host-environment schematic to model the impacts of racism. Interpersonal and systemic racism compromises host responses and increases susceptibility to disease.	+++ Racism modeled as an agent, a host factor, and a mediator(s) of disease
Hogue & Vasquez, 2002	IM, VLBW	**Lifecourse** Emphasized the psychosocial stress pathway. Called for a focus on preventive efforts, not just prenatal care, for all women although disparities primarily affect women of color.	++ Racism as a psychosocial stressor
Anachebe, 2003	IM	**General** (Review) Review of studies 1996-2002	
Fuller, 2003	Adverse Birth Outcomes	**Biological** Argues against framing disparities in terms of race/racism; favors "phenotype/environmental mismatch."	**--**
Lu & Halfon, 2003	Birth Outcomes	**Lifecourse** Merged conceptualization of early programming and cumulative pathways. Perinatal health risks should be examined and contextualized throughout the lifecourse, not only during pregnancy; consider both risk and protective factors over the lifecourse.	++ Discrimination over the lifecourse can impact health
Wise, 2003	IM	**Integrative** Need perinatal interventions for birthweight-specific mortality; and prenatal interventions for birthweight distribution as they distinctly contribute to neonatal mortality.	+ Discrimination acknowledged within a stress framework
Fiscella, 2004	IM, Maternal mortality	**Biological** (Review) Review of 70 studies cumulatively examining 32 different genetic variants. Author's approach to examining the biological is to highlight the social context as the antecedent risk. Highlighted the confluence of intrauterine infectious and microvascular pathways.	+ Disparities embedded in social context of Black women's lives

Reviews/Conceptual Papers

Author, Year (references Chap. 2)	**Birth Outcome**	**Main Explanations or Frameworks**	**Emphasis on Racism** --none; + low; ++ med.; +++ high
		'Racial disparities…may be related to environmental and intergenerational factors including psychosocial stress, genes, diet, douching, bottle-feeding, lead exposure, IUGR.'	
Headley, 2004	IM	**Multifactorial** Authors examined factors related to racial disparity; poverty alone was discounted. Acknowledged "entrenched social, economic, or political barriers." Mentioned stress and anxiety only in the traditional sense. Speculated on importance of community support—especially considering immigrant outcomes (only references the work on Hispanic women).	**--** No explicit mention of racism
Fiscella, 2005	PTB	**Environment** Emphasizes environmental factors in shaping differential risk for preterm birth even in the presence of genetic polymorphisms. Author's notion of the environment includes SES factors, discrimination/social marginalization, and the importance of a lifecourse orientation. Clarifies that knowledge of genetic contributions to PTB should not imply that genetics is a source of racial disparities in PTB.	+
Giscombe & Lobel, 2005	Adverse Birth Outcomes	**Racism-Stress** (Review) Conceptualized greater susceptibility to adverse impact of stress in African Americans. The prominent disparities explanations in the literature were mentioned as socioeconomic status and behavioral differences confounded with ethnicity; and differences in stress. Important to examine racism with African American women to prevent underestimation of stress.	+++ Conceptualized racism as a unique and chronic form of stress impacting African American pregnancies
Hogue & Bremner, 2005	PTB	**Stress** Racism conceptualized as an acute or chronic stressor (agent) that contributes to excess health risk. Introduced concept of 'stress age'—premature aging due to cumulative effects of social disadvantage. Stress age is influenced by *host susceptibility* (stress reactivity, self-assessment, personality trait/ gene/environment); *host immunity* (blame reflection, stress reducers, spirituality, social resources, economic resources, resilience); and *environment* (social/cultural/physical level stressor).	++ Racism is a direct as well as indirect stressor that contributes to host susceptibility to stress
Parham, 2005	Reproduc-tive Health Disparities	**General**	
Patrick & Bryan, 2005	Adverse Birth Outcomes	**Multifactorial** Explanations: race/genetics; stress; environment; healthcare delivery systems; interactions of genetic susceptibility, stress-induced risk behaviors, and social support); biobehavioral pathways mediated by stress	++ Racial inequality as psychosocial stressor or due to structural factors (neighborhood, healthcare)
Rich-Edwards & Grizzard, 2005	PTB	**Stress-Neuroendocrine** (Review) Focuses on contribution of psychosocial stressors and neuroendocrine mediators for the racial-ethnic disparities in PTB. Allostatic load used as a framework; chronic stress can prime the reaction.	+ 'Chronic exposure to poverty, racism, and insecure neighborhoods may condition stress responses and physiologic changes that increase risk of preterm delivery'

Reviews/Conceptual Papers

Author, Year (references Chap. 2)	**Birth Outcome**	**Main Explanations or Frameworks**	**Emphasis on Racism** --none; + low; ++ med.; +++ high
Anachebe, 2006	IM and Maternal Mortality	**Multifactorial** A complex interaction of risk factors to be examined No mention of genetics	++ Related models are discussed, particularly Hogue's (2005) stress model
Esplin, 2006	PTB	**Genetic** (Review) PTB is unlikely to be explained by a single gene mutation or risk factor; a complex of factors. "The fact that racial differences exist in the rates of PTB […] supports the hypothesis for a genetic component." Familial clustering (repeat of PTB in individuals and families) and racial disparities support a genetic argument (within multifactorial causation). Differing allelic frequencies in Black women that contribute to increased inflammatory response	--
Morello-Frosch & Shenassa, 2006	Maternal-Child Health Disparities	**Environmental** Incorporates stress and allostatic load theories into a conceptual model Environmental hazards interact with psychosocial and place-based stressors – and buffer social support, civic engagement/political empowerment, and social capital to influence health	--
Institute of Medicine (Behrman & Butler, Eds.) 2007	PTB	**Multifactorial** (Review) Identified SES, maternal behaviors, stress, infections, and genetic susceptibility as likely explanations for racial disparities in preterm birth—with stress and infections considered the most promising explanatory factors. Included a brief review of racism and birth outcomes and concluded that racism may be a potent lifetime stressor for African American women that may explain racial disparities.	+
David & Collins, 2007	IM	**Social Context (Race and Class)** Questions the aims of investigators who attempt to "control for" the complex and historical effects of racism. 'Genetic racialism' and the marginalization of race has suppressed class unity across racial lines—which diminishes collective power. Understanding race as a social construct *and* a social class is needed to understand health outcomes in the majority population.	++ Genetic racialism Racism and social context studies should counter the growth of genetic studies Structural factors of racism are implied with the discussion of class
Dominguez, 2008	Adverse Birth Outcomes	**Racism** Racism and stress stimulate biological pathology leading to adverse birth outcomes Healthcare professionals should be aware of ingrained racial stereotypes. Black nativity differences undermine genetic explanation for racial disparities.	+++ Highlights the multidimensional nature of racism – interpersonal, institutional, internalized
Howell, 2008	IM	**Clinical/Healthcare** Highlighted quality of care perspective as a 'new framework to address disparities.'	--
Anum et al., 2009	PTB	**Genetic** (Review) Emphasizes 'mounting evidence' that genetics account for some of the disparity. Single gene defects are rare and do not account for the disparities (per review). Candidate gene-based association studies focus on pro-inflammatory cytokine genes. TNFA and IL6 are not strong genetic determinants due to inconsistent findings.	--

Reviews/Conceptual Papers

Author, Year (references Chap. 2)	**Birth Outcome**	**Main Explanations or Frameworks**	**Emphasis on Racism** --none; + low; ++ med.; +++ high
		Stronger support for SERPINH1 -656T allele (an ancestry informative marker very prevalent in African Americans and Africans) associated with preterm birth. Epigenetic investigations are emerging.	
Collins & David, 2009	LBW, IM	**Social Context** Look beyond traditional risk factors to the social context of race and lifelong disadvantage for pregnancy outcomes. Disputes genetic causation arguments.	++ Lifelong exposure to interpersonal discrimination
Kramer & Hogue, 2009	PTB	**Social Context** Biosocial: linking the social construct of race with the biological Main intermediary pathways include stress, preconceptional health, and genotype/epigenetics. Key causes: socially patterned maternal stress; inflammatory, vascular, neuroendocrine mechanisms	++ Interpersonal and institutionalized racism as mechanisms
Menon, 2009	PTB	**Genetic** (Review) Identified candidate gene associations from maternal and fetal case-control analyses in 370 White mothers and 319 fetuses and 279 Black mothers and 243 fetuses total. Concluded that the pathways and mechanisms to PTB differed by race, but did not suggest a genetic component to population-level disparities.	--
Miranda et al, 2009	PTB, LBW	**Environmental** (Review) Environmental (air pollution, metals, water quality, secondhand smoke) and social (neighborhood built environment, economic conditions, and racial composition) conditions interacting with personal factors (physical and emotional stress responses) are a source of cumulative stress that contributes to persistent disparities in birth outcomes.	+
Pachter & Coll, 2009	PTB, LBW	**Racism** (Review) Reviews research on racism in association with child health; includes studies of discrimination and birth outcomes.	+++
Alio et al, 2010	Perinatal Mortality	**Racism** Adopts a socioecological, historical, and lifecourse perspective for the role of racism. Birth outcomes are impacted by family and community characteristics, which are influenced by the larger community and society. Institutional racism is the root of racial disparities, including slavery, segregated healthcare facilities, residential segregation. Disparities are related to lifelong accumulation of the impact of racism.	++ Racism is embedded and permeates all aspects of African American women's lives.
Bodnar, 2010	PTB, IUGR	**Biological** (Review) Reduced synthesis of Vitamin D contributes to PTB, fetal growth restriction, and pre-eclampsia.	--
Blumenshine, 2010	Adverse Birth Outcomes	**Socioeconomic Status** (Review) 93 of 100 studies showed a significant association between a socioeconomic measure and birth outcome; range from 1.1 to 1.5 in over half of studies. Education and income are the most common measures used. Poverty and income measures are most prevalent among neighborhood studies.	-- Not much detail on specific racial groups in this review. No particular focus on African Americans

Reviews/Conceptual Papers

Author, Year (references Chap. 2)	**Birth Outcome**	**Main Explanations or Frameworks**	**Emphasis on Racism** --none; + low; ++ med.; +++ high
Burris & Collins, 2010	PTB	**Genetics** Differences in gene *expression* (epigenetics) rather than *genotype* is the most plausible genetic-based explanation for racial differences. Transgenerational effects exemplify epigenetic mechanisms.	--
Bryant et al., 2010	Obstetric outcomes (including PTB)	**Clinical/Healthcare** Used McGinnis et al. (1993) framework [which quantifies contributing factors to disease: behavior (40%), genetics (30%); social circumstances (15%); environmental (5%); medical care (10%)] to describe how these domains contribute to disparities in obstetric outcomes, including PTB. Social factors (unspecified) received an "A" rating indicating good evidence for their role in PTB.	+ Authors note that racism can be a contributing factor to disparities
Dolan, 2010	PTB	**Genetic/Environmental** (Review) A brief review of genetic and environmental contributions to racial disparities in PTB.	--
Lu, et al., 2010	Birth Outcomes	**Lifecourse** Present 12-point plan for reducing the racial gap in 3 categories (healthcare, family and community, socioeconomic) with 4 points each Emphasized importance of addressing early life disadvantages and allostatic load over the lifecourse.	
Misra et al., 2010	Birth Outcomes	**Fatherhood**	
Ranjit et al, 2010	Adverse Birth Outcomes	**Biological** (Review) Bisphenol A—endocrine disrupting compounds in the environment; hazardous to a developing fetus Possible association of Biphenol A with fetal death and decreased fetal growth	+ Sociopolitical discrimination and segregation of African Americans puts them in environments with more hazardous exposures
Burris et al., 2011	PTB	**Environmental** Environmental contributions to PTB include epigenetic variation, lead, air pollution, cigarette smoke. Genetic factors interact with the environment.	
Culhane & Goldenberg, 2011	PTB	**Social Context** (Review) Review emphasized social factors; "[PTB due to] complex mechanisms originating from social inequities." Discussed the social context of neighborhood experiences; maternal nativity; infection-inflammation; preconception healthcare differentials. Claimed that addressing individual risk factors is not an effective approach for reducing racial disparities.	+ Non-native Blacks may have different perceptions about racism or social marginalization due to race
Dominguez, 2011	Adverse Birth Outcomes	**Racism-Stress** Stress and health paradigm Racism is a social determinant of health disparities. Discusses key issues with respect to racism's role in race-based disparities: genetics, SES, multidimensional racism, stress-induced physiology.	+++ Multidimensional nature of racism
Dunlop, 2011	PTB	**Nutrition** (Review) Contribution of nutrient deficiencies to Black-White disparities in PTB	--

Reviews/Conceptual Papers

Author, Year (references Chap. 2)	**Birth Outcome**	**Main Explanations or Frameworks**	**Emphasis on Racism** --none; + low; ++ med.; +++ high
Giurgescu et al., 2011	Adverse Birth Outcomes	**Racism-Stress** (Review) Review of studies of racial discrimination/stress and birth outcomes in African Americans; focus on empirical findings. Reviewed 10 articles: consistent positive relationship to PTB, LBW, VLBW but not gestational age Used McEwen's allostatic load as framing theory. Also emphasized lifecourse perspective	++ Discrimination leads to chronic stress and allostatic load One of few articles to explicitly define racism
Hauck et al., 2011	IM	**Multifactorial** Article set out to address *why* there are persistent IM disparities. Proposed integrative model of factors affecting health and well-being of infants/girls/pregnant women over the lifecourse (e.g., healthcare access, environment, toxins, genetic susceptibility, knowledge, racism/discrimination, maternal deprivation, social support, substance use). Causal pathway for IM disparities: "complex interactions of biological, behavioral, health access, social, and political factors that make some infants more vulnerable."	+ Black women have greater exposure to lifetime stressors
Hogue et al., 2011	PTB	**Biological** (Review) Emphasis on short interpregnancy interval (<12 months) which explained about 4% of the Black-White gap in PTB; intervals < 6 months increase PTB by 40%. Biopsychosocial framework	--
Kramer et al., 2011	PTB	**Stress** (Review) Reviewed studies and mechanisms of preconceptional stress and racial disparities 3 theories of stress-related mechanisms in PTB disparities: 1) early life developmental plasticity & early life programming of neuroendocrine function; 2) chronic stress-related blunting, weathering, or dysfunction of neuroendocrine mechanisms over the life course; 3) stress and risky behaviors Evidence is circumstantial for early life programming and moderate for chronic stress and stress-associated behaviors.	++ Discusses "interpersonal discrimination or racism"
Menon et al., 2011	PTB	**Biological** (Review) Review of racial disparity in genetic and biomarkers for PTB (1990-2010 publications) Infection and inflammatory responses as cause of disparities examined from the perspective of multiple and interactive causation (e.g., genetics, epigenetics, variable exposure to infection, nutritional deficiencies, early adverse experiences, lifetime exposure to chronic stress, behavioral, physical, and psychosocial factors) Argues that: "…infection may <u>not</u> be a cause of the racial disparity but in association with other risk factors such as stress, nutritional deficiency, and differences in genetic variations in PTB, pathways, and their complex interactions may produce differential inflammatory responses that may contribute to racial disparity."	--
Rosenthal & Lobel, 2011	Adverse Birth Outcomes	**Stress** Unique sources of stress for Black women include abuses and power by the medical system, contradictory societal pressures regarding their childrearing, and Black stereotypes of sexuality and motherhood.	++ Interaction of racism with gender bias against African American women (i.e., gendered racism)

Reviews/Conceptual Papers

Author, Year (references Chap. 2)	**Birth Outcome**	**Main Explanations or Frameworks**	**Emphasis on Racism** --none; + low; ++ med.; +++ high
Rowley & Hogan, 2012	IM	**Healthcare** (Review) Inequitable healthcare contributes to increasing IM racial/ethnic disparities.	+
Schaaf, 2013	PTB	**General** (Review) Review of effect of maternal ethnicity on PTB	

Table A.2 Empirical Studies of Racial Discrimination/Racism and Birth Outcomes

Racism and Birth Outcomes

Author, Year (references Chap. 2)	**Birth Outcome**	**Framework**	**Sample**	**Measure/Method**	**Findings**	**Racism Focus** --none; + low; ++ med.; +++ high
Murrell, 1996	LBW, PTB	Theoretical model of stress Racism and self-esteem linked to biological markers	"Low risk" African American women *(N* = 165) in a prenatal clinic	Perceived racism (general) Perceptions of racism scale (Green, 1995): 20 items, 4-point Likert scale 18 items assessed attitudes and opinions about hypothetical racism situations 2 items assessed individual's direct experience with racism	Women who reported the highest levels of racism were older, more educated, married Racism was associated with increased stress Neither racism nor stress were related to LBW or PTB	++
Shiono et al., 1997	Mean infant birthweight	Racial discrimination was one of several "social adversities" evaluated as risk factors for pregnancy outcomes	Black women *(N* = 346) Also included: White (*n* = 215) Chinese Dominican Mexican Puerto Rican	Perceived racism (during pregnancy) Also measured racial discrimination as part of a summative index of stressors	Perceived racism during current pregnancy not associated with mean birthweight (no data shown) No individual findings were presented for racial discrimination which was included as part of a summative stress scale	+
Collins et al., 2000	VLBW	Institutional racism Stress hypothesis	VLBW infants (*n* = 25) Normal birthweight infants (*n* = 60)	Perceived racism (during pregnancy) Used Modified 'Experiences of Discrimination' scale (Krieger 1990, 1996) which measures self-reported exposure to racism in 5 domains (school, medical care, service at restaurants or stores, seeking housing) Experience of racism was measured as "Yes" with at least one reported domain Design: restrospective case-control study	Unadjusted *OR* = 1.9 (0.5 - 6.6) With adjustments for maternal age, previous pregnancy, prenatal care, social support, smoking, alcohol, drugs: AOR = 3.3 (0.9 - 11.3) Odds ratios were highest for women with other risk behaviors	+++

Author, Year (references Chap. 2)	**Birth Outcome**	**Framework**	**Sample**	**Measure/Method**	**Findings**	**Racism Focus** --none; + low; ++ med.; +++ high
Rosenberg et al., 2002	PTB	Black Women's Health Study Stress-related risks	*N* = 4966 Preterm (*n* = 422) Full-term (*n* = 4544)	Women were asked about racism in 1997, after which their pregnancies were followed over 2 years Study used modified version of the Everyday Racism scale (Ren et al., 1999) 9 questions total regarding: Unfair treatment: -on the job -in housing -with police At least once a week: -poorer service in restaurants -poorer service in stores People act as if: -they are afraid of you -you are not intelligent -you are dishonest Participants were also asked: -How often they thought about their race Investigators summed the positive responses; also analyzed each question separately. Design: historical prospective cohort study	There was a modest increase in PTB in association with: Unfair treatment on job: AOR = 1.3 (1.1 –1.6) People act afraid: AOR = 1.4 (1.0 – 1.9) ORs by education level: <12 years education: OR Receive poorer service 3.5 People act afraid of you 3.4 Unfair housing 2.4 People act as if you are not intelligent 2.0 13-15 years education: People act as if you are dishonest 1.7 >16 education: Unfair treatment on the job 1.6	+++
Dole et al., 2003	PTB	Psychosocial factors	Blacks (*n* = 707, 36%) Whites (*n* = 1134, 58%)	Prospective collection of data in 2nd and 3rd trimesters Experiences of Discrimination scale (Krieger 1990, 1996): measures self-reported exposure to racism in 5 domains (school, medical care, service at restaurants or stores, seeking housing)	Relative risk of PTB by level of discrimination, adjusted for SES, medical, and behavioral covariates: No discrimination non-significant Some discrimination non-significant High discrimination RR=1.4 (1.0, 2.0)	+

Racism and Birth Outcomes

Author, Year (references Chap. 2)	**Birth Outcome**	**Framework**	**Sample**	**Measure/Method**	**Findings**	**Racism Focus** --none; + low; ++ med.; +++ high
Collins et al., 2004	VLBW	Interpersonal racism as a chronic stressor with acknowledgement of the cumulative impact of societal-institutional racism	Exclusive Black women sample VLBW infants (n = 104) Normal birthweight infants (n = 208)	Interpersonal racism (lifetime exposure) Experiences of Discrimination scale (Krieger 1990, 1996): measures self-reported exposure to racism in 5 domains (school, medical care, service at restaurants or stores, seeking housing) Also used Perceived Racism Scale (McNeilly et al., 1996) Design: restrospective case-control study	Odds ratios for levels of racism (vs. no racism) in predicting VLBW Racism in 1 or more domains: UOR = 1.9 (1.2 - 3.1) AOR = 1.7 (1.0 - 9.2) Racism in 3 or more domains: UOR = 3.2 (1.5 - 6.6) AOR = 2.6 (1.2 - 5.3) Strongest effects with employment-related racism and among higher-educated women Null findings for racism reported during pregnancy	+++
Dole et al., 2004		Effect of psychosocial factors on PTB will differ by race	Blacks (n = 724)	Experiences of Discrimination scale (Krieger, 1990, 1996): measures self-reported exposure to racism in 5 domains (school, medical care, service at restaurants or stores, seeking housing) Administered at 24-29 weeks gestation Design: prospective study	African American women reporting perceived racial discrimination had higher risks of PTB RR = 1.8 (1.1 - 2.9) African American women also had more negative life events	+
Mustillo et al., 2004	PTB, LBW (based on self-report)	Racism as a psychosocial stressor	CARDIA study Blacks (n = 152) Whites (n = 200)	Self-reported racial discrimination in at least 3 situations Adaptation of Krieger (1990) scale Women asked if they had ever experienced discrimination, been hassled, or felt inferior, due to race: -at school -getting a job -getting housing -getting medical care, -on the street -in public setting -from the police or courts	Black/White ORs for PTB and LBW reduced when racism estimated as a factor (though not significant) Black/White ORs for <u>PTB</u> Unadjusted 2.54 (1.33 - 4.85) Add racial discrim. 1.71 (0.84 - 3.48) Add other factors 1.11 (0.51 - 2.41) Black/White ORs for <u>LBW</u> Unadjusted 4.24 (1.31 - 13.7) Add racial discrim. 2.11 (0.75 - 5.93) Add other factors 2.43 (0.79 - 7.42) The independent effect of racial discrimination was significant for 3+ reported domains vs. 0 (for PTB, LBW)	+++

Author, Year (references Chap. 2)	**Birth Outcome**	**Framework**	**Sample**	**Measure/Method**	**Findings**	**Racism Focus** --none; + low; ++ med.; +++ high
				Responses combined across categories and summed as 0, 1, 2, or 3+ experiences Also asked about response to unfair treatment—whether acceptance or trying to do something	Response to unfair treatment did not differ between Whites and Blacks and was not modeled.	
Dominguez et al., 2008	Mean BW Gestational age	Lifecourse perspective Acknowledged both direct and vicarious experiences of racism	Blacks (n = 51) Whites (n = 73)	Adaptation of Krieger (1990) scale Asked about perceived racism experiences as a child (≤16) or adult (>16) in interpersonal, educational, employment, housing, or other domains Also assessed general and pregnancy-related stress	Perceived racism across the lifetime, and vicariously as a child, predicted mean birthweight and racial disparities in birthweight—controlling for medical and demographic risk factors and adult exposure to racism Vicarious racism in childhood predicted birthweight, even after control for SES Perceived racism accounted for 6% additional variance in birthweight	+++
Dailey, 2009	LBW	Racism as a stressor, in association with potential buffers such as spirituality	Black women (N = 119)	Everyday Discrimination Scale (Forman, Williams, Jackson, 1997). Asked about discrimination in 9 domains; obtained sum of individual reported experiences; then asked about reason for the discrimination, and report of each reason (1 or none) scored independently Prospective study design	No correlation between racial discrimination and LBW Age and physical disability discrimination were significantly related to LBW	++
Nuru-Jeter et al., 2009	Birth Outcomes	Multidimensional nature of racism (interpersonal institutional, internalized)	Black women (N = 40), childbearing age, socio-economically diverse	Qualitative, exploratory study; 6 focus groups Toward development of a multidimensional, gender-sensitive measure of racism so as not to underestimate women's racism experiences	Women reported racism throughout the lifecourse with acknowledged significance of childhood experiences Direct and vicarious experiences reported, especially in relation to their children Active and passive responses to racism reported	+++

Racism and Birth Outcomes

Author, Year (references Chap. 2)	**Birth Outcome**	**Framework**	**Sample**	**Measure/Method**	**Findings**	**Racism Focus** --none; + low; ++ med.; +++ high
Misra et al., 2010	PTB	Racism-stress and lifecourse frameworks	Low-income African American women in Baltimore (N = 832)	Acute and lifetime racism measured with the Racism and Lifetime Experiences Scale (RaLES), (S.P. Harrell, 1997) modified to include the three most variable items. RaLES asks about perceptions and experiences of racism for nine items using a 5-point (0 to 4) Likert scale; also includes scale for responses to racism Combined prospective and retrospective study	Racism, experienced 6 months prior or during pregnancy, had no association with PTB Also, lifetime experiences of racism had no effect on risk of PTB	+++
Carty et al., 2011	LBW (based on retrospective self-report of births over a 10-year period)	Racism (including personal, and structural) as a social determinant of health	Blacks (n = 330) Whites (n =180)	Assessment of 5 emotional reactions (i.e., angry, anxious, depressed, bothered, powerless) to racism-related experiences with Likert scale responses (1=none to 5=extreme) for each reaction. Adapted from the RaLES, Racism-related Experiences Scale (S.P. Harrell, 1997)	LBW marginally related to more extreme emotional reactions to racism, among Blacks and Whites combined Unadjusted OR = 1.24 (0.99, 1.55) Adjusted for race and education OR = 1.17 (0.93, 1.48)	+++

Table A.3 Empirical Studies of Births to Black Foreign-born Women in the U.S.

Nativity and Birth Outcomes (FB=Foreign-born, CA=Caribbean)

Author, Year (references Chap. 2)	**Birth Outcome**	**Framework**	**Sample**	**Measure/Method**	**Findings**	**Racism Focus** --none; + low; ++ med.; +++ high
Valanis, 1979 Valanis & Rush, 1979	Birthweight Gestational age	Presumed that the black nativity advantage could be explained by childhood social status Conceptual-ized social differences translated into biological outcomes Selective migration	New York City (1971-1973) Black women (n = 766) NY-born (55%) US Southern-born (35%) Foreign-born (10%)	Prospective enrollment of women in prenatal care in Harlem All at high risk for LBW as determined by previous LBW, low pre-pregnancy weight or pregnancy weight gain, low 24-hr protein intake Modeled nativity in association with childhood SES (parents' occupation)	FB Blacks had higher mean BW and lower proportion of LBW (3.8 v 15.6 and 18.4%) and preterm birth (11.4 vs. 24.3%) than US Blacks. FB Blacks had better childhood social status and more healthful behaviors such as lower smoking rates Years of residence was inversely related to birthweight among FB Blacks Childhood (and current) social class provided a birthweight advantage for the FB women only	--
Chavkin et al., 1987	LBW IM	No causal association with immigrant status was suggested	New York City (1980-1984) Caribbean mothers (n >100,000) stratified as: Puerto Rican Other Hispanic non-Hispanic	Descriptive study of percentage distributions by mother's race/ethnicity and national origin Did not identify 'non-Hispanic Caribbean' women by race although most were Jamaican and Haitian and assumed Black	Over one-third of births were to non-Hispanic Caribbean women over 30 years old. Higher education US Black 21%, FB Black (nh CA) 27% Caribbean LBW rate 6% compared to 10.8% among US Blacks Caribbean IM rate 14.2 compared to 21.3 among US Blacks	
Cabral et al., 1990	Pregnancy and birth outcomes	FB status approximates cultural characteristics ('cultural' not defined)	Boston Black women, low-income US-born (n = 616) FB (n = 201): (72% from the Caribbean, includes Hispanics)	Unadjusted, bivariate analyses of risk differences between US and FB women.	FB Blacks had better pre-pregnancy, nutrition, prenatal care visits, higher IUGR, lower risk of LBW than US Blacks FB advantage independent of SES, marital status, prenatal care, health behaviors No nativity difference in gestational age	--

Nativity and Birth Outcomes (FB=Foreign-born, CA=Caribbean)

Author, Year (references Chap. 2)	**Birth Outcome**	**Framework**	**Sample**	**Measure/Method**	**Findings**	**Racism Focus** --none; + low; ++ med.; +++ high
Kleinman et al., 1991	IM	No guiding framework Purpose to yield clues for prevention strategies	US (1983-1984) White and Black US and FB mothers	'Maternal risk' quantified as low, moderate, high Differences in IM by maternal risk characteristics larger than differences by nativity Nativity was the only factor that had stronger effects among Blacks than Whites	Black FB had less LBW and VLBW than Black US-born FB Blacks had 22% and 24% lower adjusted neonatal and post-neonatal mortality than US Blacks No differences in neonatal IM among Whites by nativity, but 20% lower post-neonatal IM for FB Whites	--
Friedman et al., 1993	LBW	Acculturation as a possibility for Black nativity differences Ethnicity construed as social category and a reflection of different experiences beyond race Cultural explanatory framework	Massachusetts (1987-1989) non-Hispanic White (n = 206,358) Black (n = 18,571) Blacks included: US-born (n = 11,075) West Indian (n = 1626) Haitian (n = 2579) Cape Verdean (n = 895) Hispanic (n = 575)	Groups delineated by race and self-reported ancestry – not strictly nativity Analyzed with multiple linear regression (mean birthweight) and multiple logistic regression (LBW) Comparison group non-Hispanic Whites	Caribbean-born women had lower LBW rates than US-born women All Black ethnic groups higher LBW than Whites, regardless of SES US-born and non-US-born in the full sample each had non-significant LBW odds in adjusted models—not including race or ancestry—prompting the conclusion: "Clear results regarding the relationship between maternal place of birth and birthweight do not emerge from the MA data." However, ancestry groups (Black) were not analyzed by nativity status	--
Rumbaut & Weeks, 1996	Mean BW and Mean Gestational Age	Assimilation, cultural explanatory framework	San Diego Sample included only 28 U.S. born Blacks and 6 FB Blacks	Mean and percent comparisons: US born vs FB	No significant differences in mean birthweight or mean gestational age between US and FB Blacks Conclusion of 'Better diets among FB Black women than US Blacks': based on fewer servings of fats and sweets However, fruit and vegetable intake by FB Blacks was lower as well as milk products and protein	--

Nativity and Birth Outcomes (FB=Foreign-born, CA=Caribbean)

Author, Year (references Chap. 2)	**Birth Outcome**	**Framework**	**Sample**	**Measure/Method**	**Findings**	**Racism Focus** --none; + low; ++ med.; +++ high
Singh & Yu, 1996	IM, PTB, LBW	Speculated on behaviors, but also life-course SES disadvantage and **discrimination** for US compared to FB Blacks	US (1985-1987) Non-Hispanic Whites, Blacks, plus Asian and Hispanic groups. All FB Blacks lumped together US Territories considered foreign	Estimated fully-adjusted models including nativity with FB as the reference; race with White non-Hispanic as the reference; and other pregnancy risk variables considered together Crude and adjusted ORs were stratified for each US-born group, as well as the total US population, with corresponding FB groups as the reference populations Control for SES to see if nativity still matters	Black US/FB adjusted odds ratios: IM 1.33 (1.21, 1.45) LBW 1.61 (1.55, 1.67) PTB 1.31 (1.27, 1.35) Lower smoking rates for FB than USB	+
David & Collins, 1997	LBW Mean birthweight	Interest in determining if LBW a result of social or genetic factors	Illinois (1980-1995) US-born Blacks African-born Blacks US-born Whites	Descriptive study Examined distribution of factors Used random sample of US-born Blacks and Whites from the cohorts	Mean birthweight US Black 3089g Africans 3333g US White 3446g LBW US Black 13.2%, RR = 3.1 African Black 7.1%, RR = 1.6 US White 4.3% (ref.) LBW among lowest risk women: 20-39, 12 years maternal/paternal education, early prenatal care, gravida 2-3, no previous fetal losses US Black 7.5% Africans 3.6% US White 2.4%	
Fuentes-Afflick, 1998	MLBW VLBW	Importance of cultural factors and social support for the immigrant health advantage	California (1992) Black, White, Asian, and Latina women FB Black *n* = 2,490 US Black *n* = 41,513	Women born in Puerto Rico or the US territories categorized as foreign-born Stratified racial/ethnic groups Nativity comparisons: reference group US born	FB Blacks had more favorable maternal characteristics but there was no significant difference in VLBW or MLBW between FB and US-born Blacks after adjustment for covariates VLBW: FB/USB = AOR 1.27 (0.83, 1.95) MLBW: FB/USB = AOR 0.83 (0.70, 1.00) Both FB and US Blacks significantly higher AORs than US Whites VLBW: FB 2.44 (1.56, 3.83), USB 1.85 (1.63, 2.11) MLBW: FB 1.59 (1.32, 1.91), USB 1.88 (1.79, 1.98)	--

Nativity and Birth Outcomes (FB=Foreign-born, CA=Caribbean)

Author, Year (references Chap. 2)	**Birth Outcome**	**Framework**	**Sample**	**Measure/Method**	**Findings**	**Racism Focus** --none; + low; ++ med.; +++ high
Fang et al., 1999	LBW	Aimed to counter genetic explanations for racial differences by examining nativity within race Early life experiences for FB may buffer poor circumstance in the U.S. Selective migration	New York City (1988-1994) Non-Hispanic native and foreign-born White and Black mothers	Estimated logistic models for race, and for race/nativity groups Also estimated group models for each tertile of community income Used all Whites as the group reference in adjusted models.	In low-income communities, Caribbean and African Blacks had lower LBW rates compared to Whites; therefore, FB Blacks had an even greater advantage in poor communities There were no significant nativity difference (NYC whites as reference) when controlling for individual-level maternal factors—Black CA 0.95 (0.87, 1.03); African 0.86 (0.69, 1.02)—only the neighborhood context had an effect Authors conclude that the equivalency of risk between Caribbean-born and US-born Blacks would eliminate the Black-White race disparity	--
Pallotto et al., 2000	MLBW VLBW	Institutional **racism** negatively impacts health Lifelong minority status experiences contribute to birth outcomes	Illinois (1985-1990) US-born Blacks Caribbean-born Blacks (may include Hispanics in 85-88 cohort) US-born Whites	Selected random samples of US White and US Black births US Whites are reference group for relative risks (RR) Categorized maternal risk (low/high) and estimated RR of LBW for each group according to risk category	Caribbean-born risk profile lower than US-born Blacks but higher than Whites FB had better outcome, regardless of SES or medical risk status MLBW: USB 10%, CA 6%, USW 4% RR CAB/USW = 1.7 (1.4, 2.0) VLBW: USB 2.6%, CA 2.4%, USW 0.7% RR CAB/USW = 3.3 (2.5, 4.4) Low risk mothers MLBW: USB 10%, CA 6%, USW 4% RR CAB/USW = 1.7 (1.4, 2.0) Relative higher rates for CA immigrant women (disaggregated from African immigrants)	+
Collins et al., 2002	Mean birthweight	Lifecourse framework Pregnancy risks are not independent of prior life experiences	US and FB Blacks and Whites in Illinois Africans and Caribbean Blacks were combined as FB	Compared infant birthweight across generations (2nd and 3rd) by maternal race/nativity	African/Caribbean 3rd gen descendants had lower birthweight (mean 57 g) then gen 2 and 40% greater MLBW (9.6% v 6.7%). This contrasts with increased birthweights for US-born White and Black descendants.	+ 'Unidentified aspects of American society are deleterious to the reproductive health of African American women'

Nativity and Birth Outcomes (FB=Foreign-born, CA=Caribbean)

Author, Year (references Chap. 2)	**Birth Outcome**	**Framework**	**Sample**	**Measure/Method**	**Findings**	**Racism Focus** --none; + low; ++ med.; +++ high
Rosenberg et al., 2002	IM	Speculation on maternal nutrition and stress as hypotheses for birth outcome differences among Blacks by nativity Authors speculate that CA had better childhood and adult diets.	New York City (1988-1992) Non-Hispanic US and FB Blacks ---------- (Framework) Also, CA had hopefulness and social supports to mediate stress of poverty and dislocation	Linked mean household income from census tract data Compared IMR across risk factors, stratified by native and foreign born Then adjusted model with backwards removal of non-significant factors; included US/FB as a covariate	Lower IMR among FB Blacks	--
Acevedo-Garcia et al., 2005	LBW	Does not propose reasons for the FB advantage, but acknowledges ideas about culture and immigrant selectivity in reference to Latina findings Makes no attributions to any of these hypotheses in relation to her study	US (1998) Non-Hispanic Black, Asian, Hispanic, and White (ref.) women aged 20+	Estimated LBW ORs in total population: Blacks, Asians, and Hispanics compared to non-Hispanic Whites as the reference group Then adjusted for FB status and tested interactions for race*nativity and FB status*education When interaction term proved significant, stratified models were estimated Stratified models run by race that used the US-born as the reference group. All models controlled for SES and medical risk factors	FB status associated with reduced LBW among Black (25% reduced odds, AOR 0.75, and Hispanic women AOR 0.81) The protective effect was stronger for women with low education (0-11) compared to 16+ education. Black educ effect: FB/US 0.64 Black FB educ gradient 1.05, 1.14, 1.10 Black US educ gradient 1.32, 1.21, 1.02 The educational gradient is less pronounced in FB than in US-born '…diminishing returns of FB status as education increases." (26)	--

Nativity and Birth Outcomes (FB=Foreign-born, CA=Caribbean)						
Author, Year (references Chap. 2)	**Birth Outcome**	**Framework**	**Sample**	**Measure/Method**	**Findings**	**Racism Focus** --none; + low; ++ med.; +++ high
Howard et al., 2006	LBW, PTB	Ancestry and nativity as proxies for social and environmental contexts—not genetics "Ancestry" self-reported and presumed based on parentage. The term not defined in the paper.	New York City (1998-2002) Non-Hispanic Blacks of varying ancestries (US, African, Asian, Cuban, European, Puerto Rican, and Central American) Brazilian and West Indian lumped together	Examined LBW and PTB odds in Black ancestry groups with African Americans as the reference group Adjusted for covariates including nativity status (USB or FB) and an ancestry*nativity interaction	Caribbean and Cuban women had the highest unadjusted LBW rate among the foreign Blacks, relative to African Americans There was no significant nativity effect on LBW or PTB for women of Caribbean ancestry (WI-Brazilian) Ancestry models adjusted for nativity (US/FB) and other covariates: WI-Brazilian/US Black LBW ARR = 0.98 (0.92, 1.05) PTB ARR = 1.05 (0.99, 1.11) Direct comparison of FB/US: WI-Brazilian FB/ WI-Brazilian US LBW ARR = 0.96 (0.83, 1.11) PTB ARR = 0.99 (0.85, 1.13) WI-Brazilians had highest RR of LBW and PTD than all other Black ancestry groups Study suggests that country of origin may be more predictive of birth outcomes than foreign-born status alone	--
Liu & Laraque, 2006	IM	Suggests maternal risk factors as contributing to the FB advantage Acknowledged the lifecourse perspective & interaction of social, environment-tal and biological factors as contributing to disparities	New York City (1995-1998) US-born and foreign-born mothers, by race and Hispanic ethnicity US-born includes the US Territories in this study.	Estimated logit models separately for each cause of infant death, controlling for covariates Model A includes nativity and race as separate variables Model B includes a nativity*race interaction	Healthy migrant effect not shown for many Caribbean immigrants, by national origin, where their IMR was higher than the NYC average for FB mothers Immigrants from the Caribbean, Central America, and Africa had higher IM rates compared to immigrants from Europe, Asia, and South America US (12.7) and FB Blacks (10.0) had highest IMR of all other racial and ethnic groups; and highest ORs relative to US Whites - 2.03 and 1.89, respective	

Nativity and Birth Outcomes (FB=Foreign-born, CA=Caribbean)

Author, Year (references Chap. 2)	**Birth Outcome**	**Framework**	**Sample**	**Measure/Method**	**Findings**	**Racism Focus** --none; + low; ++ med.; +++ high
Grady & McLafferty 2007	LBW	Ethnic density as protective for immigrants (promotes cultural and social capital), but segregation hypothesized to offset ethnic density	New York City (2000) US-born and foreign-born Black women in NYC Black race groups include Hispanics	Multilevel analysis by racial segregation (more/less) Stratified by race/nativity group and neighborhood poverty (high/med/low) Estimates adjusted for individual maternal characteristics	Segregation unconditionally associated with LBW in both US-born and FB Blacks. Segregation also associated with LBW in FB Blacks combined, but after control for national origin and maternal characteristics, the area segregation and poverty effects were no longer significant The area poverty gradients differ by nativity: AA-high poverty, higher LBW AA-low poverty, lower LBW FB-high poverty, lower LBW FB-low poverty, higher LBW Could be a function of neighborhood concentration of immigrants with relatively low risks in segregated neighborhoods (i.e., W. Indian, Dominican) and relatively high risk in less segregated neighborhoods (i.e., Haitian)	+
Dominguez & Strong, 2009	None. Sample of pregnant Black women with focus on nativity differences in perceptions of racism. Part of prospective study to later examine associations with birth outcomes.	Differential exposure to racism over the lifecourse may help to explain differences in US-born and foreign-born Black women's birth outcomes	Black women, Boston, US-born (n = 185) FB (n = 114) 67% of FB sample from the Caribbean 55% of FB sample emigrated to the US before age 18	Women sampled from larger prospective study Self-reported personal racism and group-directed racism examined over lifecourse stages Used validated Experiences of Discrimination scale (Krieger, Smith, Naishadham, Hartman, & Barbeau, 2005) Participants asked about: How they respond to unfair treatment: Talking to others (active) Keeping to self (passive) Accept as part of life (passive) Do something (active)	US-born reported higher odds of personal and group racism than FB—especially for racism experienced in childhood The magnitude of experienced racism for Caribbean-born closer to US-born than African-born FB immigrants <18 did not significantly differ from US-born women in reported personal racism except during childhood Caribbean immigrants did not significantly differ from US-born in racism during adulthood or the current pregnancy or in group racism ever experienced. Percent who experienced racism:	+++

Nativity and Birth Outcomes (FB=Foreign-born, CA=Caribbean)

Author, Year (references Chap. 2)	**Birth Outcome**	**Framework**	**Sample**	**Measure/Method**	**Findings**	**Racism Focus** --none; + low; ++ med.; +++ high
				Experiences of unfair treatment by race (racism), either personally or for their racial group, in 8 domains: at school, getting a job, at work, getting housing, getting medical care, getting service in store or restaurant, on the street public setting, from police or courts Categorized racism experiences as 0, 1-2, or ≥3	Personal Childhood Group US 79% 90% 59% CA 64% 83% 27% AF 30% 50% 17% Personal racism US/CB: Ever 2.1 (1.2, 3.8) Childhood 3.8 (2.1, 7.1) Adulthood 1.5 (0.9, 2.7) ns Current Pregnancy 2.6 (0.0, 7.0) ns Group-directed racism US/CB: Ever 1.9 (3.9, 22.0) Prior to 18 5.9 (3.2, 10.9)	
Stein et al., 2009	PTB, SGA, LBW	Speculated on racism as the contributor for all Black population differences in comparison to White populations.	New York City (1995-2003) FB populations examined by nationality/ ethnicity, not by race, but one can presume a majority Black sample from the NH Caribbean nationalities observed (e.g. Jamaica)	Examination of birth outcomes by ancestry Analyses did not include any covariates	Non-Hispanic Caribbean (i.e., Caribbean Black) women had higher PTB rates than non-Hispanic Whites SGA and LBW rates were similar among all race/nationality/ethnic groups socially recognized as Black	++
Urquia et al., 2010a	PTB, SGA	Convergence hypothesis of immigrants achieving health outcomes over time to approach the levels of the native population—related to changes in	Immigrants and non-immigrants in Ontario Canada Includes analysis of Caribbean immigrants Not specified by race, but majority of Caribbean immigrants in	Population-based study of all immigrants vs all non-immigrants; sub-analysis by region of birth which provided some data on Caribbean immigrants Logistic regression to estimate effects of duration of residence using hierarchical models that account for clustering of births by country of birth	Recent immigrants with less than 10 years residence had lower odds of PTB than the native population (OR 0.92, CI 0.87, 0.97). Immigrants with residence 10-14 years (OR 1.07, CI 1.01, 1.13 and ≥ 15 years (OR 1.20, CI 1.12, 1.28) had higher PTB odds than native population Caribbean immigrants had the highest odds of preterm birth relative to other immigrants 1.72 (1.37, 2.17), including sub-Saharan Africans who were not significantly different from other immigrants 1.25 (1.00, 1.56)	--

Nativity and Birth Outcomes (FB=Foreign-born, CA=Caribbean)						
Author, Year (references Chap. 2)	**Birth Outcome**	**Framework**	**Sample**	**Measure/Method**	**Findings**	**Racism Focus** --none; + low; ++ med.; +++ high
		health behaviors	Canada are from West Indies.		PTB advantage for immigrants lost after 10 years	
Urquia et al., 2010b	PTB, LBW	Heterogeneity of migration effects by race/ethnicity and region of origin and destination	Meta-analysis of 24 studies, international and US	Meta-analysis of international migration	Black immigrants to the US have lower risk than AA African immigrants to the US have risks closer to Whites than Blacks White and Asian immigrants to the US have prematurity risk equal or greater to their native counterparts in the US	--
Mason et al., 2010	PTB	Views FB ethnic density as the reason for Black nativity differences via food preferences, social and cultural support, and lower perceived race-based barriers	Non-Hispanic Blacks, NYC US-born, Caribbean-born, African-born	Compared groups in neighborhoods with 90th and 10th percentile ethnic density Computed risk differences, using intercept and variable coefficients to back calculate from odds to risks (Austin, 2010)	Unlike the pronounced relationship for African Americans, there was little significant effect of ethnic density among Black Caribbeans and Africans (no statistical significance), despite higher risk difference estimates in more disadvantaged(deprived) neighborhoods Little support for heterogeneity across groups by country of origin No evidence of health promoting enclaves for Black immigrants Adjusted OR / Deprived / Non-deprived US 8.1 (3.3, 12.8) / -4.0 (-12.1, 4.2) / 12.5 (6.6, 18.4) CA 1.5 (-3.2, 6.3) / -1.5 (-8.6, 5.5) / 4.4 (-1.6, 10.4) AF 4.8 (2.1, 7.4) / 2.8 (-1.4, 7.0) / 6.1 (1.9, 10.2)	--
Collins et al., 2013	IM	Contextual factors and experiences associated with lifelong residence in the U.S. harm health of minority women	US linked infant birth-death files (2003-2004) Non-Hispanic White, African American, & Mex. American mothers with TERM infants	Multivariable binomial regression models with interaction term for ethnicity and LBW; controlled for age, education, parity, region U.S. of birth Term birth: 37-42 weeks	Higher IM RR for US-born except for infant survival advantage among US born with term, LBW infants 0.7 (.5, .9) Rel. risks US-born/Foreign born Black IMR US = 4.1 IMR FB = 2.2 Unadjusted RR = 1.8 (1.6, 2.1) Adjusted RR = 1.7 (1.5, 2.1) SIDS 2.7 (1.8, 4.2); neonatal IM 1.1 (0.9, 1.1); postneonatal IM 1.7 (1.4, 2.0)	-- Never mentioned, although could be inferred among the 'unmeasured contextual factors' that could be detrimental
Elo, et al., 2014	PTB, SGA	Worsening risks for immigrants with exposure to U.S. racial discrimination	US (2008), non-Hispanic Black women in 27 states where maternal nativity recorded	Comparison of FB and UB-born births via percent comparisons and both unadjusted and adjusted multiple logistic regression Did not include U.S. Territorial births.	FB/US PTB AOR = 0.73 (0.73, 0.73) FB/US SGA AOR = 0.74 (0.74, 0.75) FB / US Mothers 30 and older 54% / 24% Married 60% / 25% Residence US South 54% / 47% Smoked in Pregnancy 11% / 0.5% Diabetes 4.1% / 6.1%	++

Appendix B:

Chapter 4 Reference Tables

Table B.1 Stepwise Adjusted Predictors of Preterm Birth, Stratified by Maternal Nativity, non-Hispanic Blacks, New York City, 2000-2009

Virgin Islands-born, NYC	Unadjusted	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8
	OR 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI
Primiparous, 1	0.83 (0.57, 1.22)	1.08 (0.71, 1.64)	1.14 (0.74, 1.77)	1.17 (0.75, 1.80)	1.13 (0.73, 1.74)	1.13 (0.73, 1.74)	1.12 (0.73, 1.74)	1.14 (0.73, 1.77)	1.11 (0.72, 1.73)
Multiparous, 2 or more (ref)	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
Aged 15-19 years	0.71 (0.28, 1.81)	0.70 (0.27, 1.79)	0.65 (0.25, 1.71)	0.65 (0.25, 1.72)	0.68 (0.26, 1.81)	0.67 (0.25, 1.78)	0.69 (0.26, 1.83)	0.72 (0.27, 1.95)	0.75 (0.28, 2.02)
Aged 25-29 years	1.54 (0.87, 2.72)	1.57 (0.88, 2.80)	1.62 (0.90, 2.93)	1.60 (0.88, 2.90)	1.56 (0.86, 2.85)	1.57 (0.86, 2.86)	1.57 (0.86, 2.86)	1.60 (0.87, 2.93)	1.61 (0.88, 2.95)
Aged 30-34 years	1.48 (0.83, 2.64)	1.52 (0.84, 2.76)	1.62 (0.88, 2.99)	1.53 (0.82, 2.85)	1.52 (0.82, 2.83)	1.56 (0.84, 2.91)	1.53 (0.82, 2.87)	1.56 (0.83, 2.93)	1.58 (0.84, 2.97)
Aged 35-39 years	2.43 (1.34, 4.41)	**2.51** (1.35, 4.67)	**2.76** (1.45, 5.25)	**2.58** (1.34, 4.96)	**2.50** (1.30, 4.81)	**2.53** (1.31, 4.87)	**2.49** (1.29, 4.80)	**2.51** (1.29, 4.88)	**2.53** (1.30, 4.91)
Aged 20-24 years (ref)	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
< 12 yrs education	1.45 (0.83, 2.55)		1.63 (0.92, 2.92)	1.67 (0.93, 2.98)	1.71 (0.95, 3.08)	1.70 (0.94, 3.06)	1.67 (0.92, 3.01)	1.66 (0.92, 3.01)	1.68 (0.93, 3.05)
13-15 yrs education	**1.72** (1.07, 2.77)		1.58 (0.97, 2.56)	1.54 (0.95, 2.51)	1.60 (0.98, 2.62)	1.57 (0.96, 2.57)	1.56 (0.96, 2.56)	1.57 (0.96, 2.58)	1.58 (0.96, 2.59)
16+ yrs education	1.21 (0.68, 2.18)		0.94 (0.51, 1.73)	0.87 (0.47, 1.62)	0.93 (0.50, 1.74)	0.90 (0.48, 1.69)	0.90 (0.48, 1.69)	1.00 (0.53, 1.87)	1.01 (0.54, 1.90)
12 yrs education (ref)	1.0		1.0	1.0	1.0	1.0	1.0	1.0	1.0
Unmarried	0.67 (0.45, 1.01)			0.75 (0.49, 1.15)	0.74 (0.48, 1.14)	0.75 (0.48, 1.16)	0.78 (0.50, 1.20)	0.79 (0.51, 1.23)	0.80 (0.51, 1.25)
Married (ref)	1.0			1.0	1.0	1.0	1.0	1.0	1.0
2nd trimester prenatal care	0.92 (0.57, 1.47)				0.96 (0.59, 1.54)	0.95 (0.59, 1.53)	0.95 (0.59, 1.54)	0.95 (0.58, 1.54)	0.93 (0.57, 1.51)
3rd trimester prenatal care	0.50 (0.20, 1.27)				0.57 (0.22, 1.47)	0.58 (0.22, 1.50)	0.58 (0.22, 1.51)	0.52 (0.20, 1.36)	0.47 (0.18, 1.26)
None/unknown prenatal care	**2.41** (1.33, 4.37)				**2.58** (1.40, 4.75)	**2.60** (1.41, 4.80)	**2.60** (1.41, 4.82)	**2.68** (1.44, 4.98)	**2.72** (1.46, 5.05)
1st trimester prenatal care (ref)	1.0				1.0	1.0	1.0	1.0	1.0
Smoked during pregnancy	0.00 (0, ∞)					0.00 (0, ∞)	0.00 (0, ∞)	0.00 (0, ∞)	0.00 (0, ∞)
Did not smoke (ref)	1.0					1.0	1.0	1.0	1.0
Weight gain <16 lb	1.17 (0.71, 1.95)						1.14 (0.68, 1.93)	1.13 (0.67, 1.92)	1.11 (0.65, 1.88)
Weight gain ≤41 lb	0.64 (0.38, 1.07)						0.69 (0.41, 1.18)	0.65 (0.38, 1.11)	0.66 (0.38, 1.13)
Weight gain 16-40 lb (ref)	1.0						1.0	1.0	1.0
Maternal medical risks	2.01 (1.37, 2.95)							**2.15** (1.44, 3.19)	**2.11** (1.42, 3.14)
No maternal medical risks (ref.)	1.0							1.0	1.0
Infant congenital anomalies	2.84 (0.89, 9.05)								2.71 (0.79, 9.22)
No congenital anomalies (ref.)									1.0

Note: Significant terms are bolded.

(table continues)

Table B.1 (continued)

Caribbbean-born, NYC	Unadjusted	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8
	OR 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI
Primiparous, 1	**1.06** (1.01, 1.11)	**1.19** (1.14, 1.25)	**1.21** (1.15, 1.27)	**1.20** (1.15, 1.27)	**1.21** (1.15, 1.27)	**1.21** (1.15, 1.27)	**1.24** (1.18, 1.30)	**1.21** (1.15, 1.28)	**1.21** (1.15, 1.27)
Multiparous, 2 or more (ref)	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
Aged 15-19 years	**1.12** (1.01, 1.25)	1.08 (0.97, 1.20)	1.06 (0.95, 1.18)	1.04 (0.93, 1.16)	1.06 (0.95, 1.18)	1.06 (0.95, 1.18)	1.06 (0.95, 1.19)	1.09 (0.97, 1.22)	1.09 (0.98, 1.22)
Aged 25-29 years	1.07 (1.00, 1.15)	**1.11** (1.04, 1.19)	**1.13** (1.06, 1.22)	**1.16** (1.08, 1.24)	**1.15** (1.08, 1.24)	**1.15** (1.08, 1.24)	**1.14** (1.07, 1.23)	**1.12** (1.04, 1.20)	**1.12** (1.04, 1.20)
Aged 30-34 years	1.23 (1.15, 1.31)	**1.31** (1.23, 1.41)	**1.35** (1.25, 1.44)	**1.39** (1.29, 1.49)	**1.39** (1.29, 1.49)	**1.39** (1.29, 1.49)	**1.36** (1.27, 1.46)	**1.30** (1.21, 1.40)	**1.30** (1.21, 1.40)
Aged 35-39 years	1.49 (1.39, 1.60)	**1.62** (1.50, 1.74)	**1.65** (1.53, 1.78)	**1.71** (1.58, 1.84)	**1.71** (1.58, 1.84)	**1.71** (1.58, 1.84)	**1.65** (1.53, 1.78)	**1.51** (1.40, 1.63)	**1.51** (1.40, 1.63)
Aged 20-24 years (ref)	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
< 12 yrs education	0.95 (0.89, 1.01)		0.97 (0.91, 1.04)	0.96 (0.90, 1.03)	0.96 (0.90, 1.03)	0.96 (0.90, 1.03)	0.95 (0.89, 1.01)	0.95 (0.89, 1.02)	0.95 (0.89, 1.02)
13-15 yrs education	0.91 (0.87, 0.97)		**0.91** (0.86, 0.96)	**0.91** (0.87, 0.97)	**0.91** (0.87, 0.97)	**0.91** (0.87, 0.97)	**0.92** (0.87, 0.98)	**0.93** (0.88, 0.98)	**0.93** (0.88, 0.98)
16+ yrs education	0.94 (0.88, 1.01)		**0.87** (0.81, 0.93)	**0.89** (0.83, 0.95)	**0.88** (0.82, 0.95)	**0.88** (0.82, 0.95)	**0.90** (0.84, 0.97)	**0.92** (0.86, 0.98)	**0.92** (0.86, 0.99)
12 yrs education (ref)	1.0		1.0	1.0	1.0	1.0	1.0	1.0	1.0
Unmarried	1.07 (1.02, 1.12)			**1.13** (1.08, 1.19)	**1.14** (1.08, 1.19)	**1.14** (1.08, 1.19)	**1.14** (1.09, 1.20)	**1.12** (1.07, 1.18)	**1.12** (1.07, 1.18)
Married (ref)	1.0			1.0	1.0	1.0	1.0	1.0	1.0
2nd trimester prenatal care	0.92 (0.87, 0.97)				**0.93** (0.88, 0.98)	**0.92** (0.88, 0.98)	**0.92** (0.87, 0.97)	**0.91** (0.86, 0.96)	**0.91** (0.86, 0.96)
3rd trimester prenatal care	0.70 (0.64, 0.76)				**0.70** (0.64, 0.76)	**0.70** (0.64, 0.76)	**0.69** (0.64, 0.76)	**0.67** (0.61, 0.73)	**0.66** (0.61, 0.72)
None/unknown prenatal care	1.68 (1.55, 1.82)				**1.69** (1.56, 1.83)	**1.68** (1.55, 1.83)	**1.67** (1.54, 1.81)	**1.72** (1.58, 1.87)	**1.72** (1.58, 1.86)
1st trimester prenatal care (ref)	1.0				1.0	1.0	1.0	1.0	1.0
Smoked during pregnancy	1.49 (1.16, 1.93)					**1.45** (1.13, 1.88)	**1.49** (1.15, 1.92)	**1.35** (1.04, 1.75)	**1.35** (1.04, 1.76)
Did not smoke (ref)	1.0					1.0	1.0	1.0	1.0
Weight gain <16 lb	1.85 (1.75, 1.96)						**1.82** (1.71, 1.92)	**1.78** (1.68, 1.89)	**1.78** (1.68, 1.89)
Weight gain ≤41 lb	0.80 (0.75, 0.84)						**0.78** (0.74, 0.83)	**0.77** (0.73, 0.82)	**0.77** (0.73, 0.82)
Weight gain 16-40 lb (ref)	1.0						1.0	1.0	1.0
Maternal medical risks	2.18 (2.09, 2.28)							**2.15** (2.06, 2.25)	**2.14** (2.05, 2.24)
No maternal medical risks (ref.)	1.0							1.0	1.0
Infant congenital anomalies	1.74 (1.50, 2.02)								**1.58** (1.36, 1.84)
No congenital anomalies (ref.)	1.0								1.0

Note: Significant terms are bolded.

(table continues)

Table B.1 (continued)

US-born, NYC	Unadjusted	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8
	OR 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI
Primiparous, 1	**0.83** (0.81, 0.86)	**0.90** (0.87, 0.93)	0.98 (0.95, 1.02)	0.97 (0.94, 1.01)	0.98 (0.94, 1.02)	0.99 (0.95, 1.03)	1.02 (0.98, 1.06)	1.01 (0.97, 1.05)	1.01 (0.97, 1.05)
Multiparous, 2 or more (ref)	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
Aged 15-19 years	1.06 (1.00, 1.11)	**1.09** (1.03, 1.10)	0.97 (0.92, 1.02)	0.97 (0.92, 1.02)	0.98 (0.92, 1.03)	0.99 (0.94, 1.04)	0.99 (0.94, 1.05)	1.01 (0.95, 1.06)	1.01 (0.95, 1.06)
Aged 25-29 years	**1.11** (1.06, 1.16)	**1.08** (1.04, 1.13)	**1.17** (1.12, 1.23)	**1.19** (1.13, 1.24)	**1.18** (1.13, 1.24)	**1.18** (1.12, 1.23)	**1.16** (1.11, 1.22)	**1.15** (1.10, 1.20)	**1.15** (1.09, 1.20)
Aged 30-34 years	**1.29** (1.23, 1.36)	**1.25** (1.19, 1.30)	**1.40** (1.33, 1.47)	**1.44** (1.37, 1.52)	**1.43** (1.36, 1.50)	**1.41** (1.34, 1.49)	**1.39** (1.32, 1.46)	**1.35** (1.28, 1.42)	**1.35** (1.28, 1.42)
Aged 35-39 years	**1.68** (1.60, 1.78)	**1.62** (1.54, 1.72)	**1.83** (1.73, 1.94)	**1.90** (1.79, 2.01)	**1.87** (1.76, 1.98)	**1.83** (1.73, 1.94)	**1.79** (1.68, 1.89)	**1.69** (1.59, 1.79)	**1.69** (1.59, 1.79)
Aged 20-24 years (ref)	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
< 12 yrs education	**1.10** (1.06, 1.14)		**1.16** (1.11, 1.21)	**1.15** (1.10, 1.20)	**1.14** (1.09, 1.19)	**1.12** (1.07, 1.17)	**1.10** (1.06, 1.15)	**1.09** (1.05, 1.14)	**1.09** (1.05, 1.14)
13-15 yrs education	**0.90** (0.87, 0.94)		**0.87** (0.83, 0.90)	**0.88** (0.85, 0.92)	**0.90** (0.86, 0.94)	**0.91** (0.87, 0.95)	**0.92** (0.88, 0.96)	**0.93** (0.89, 0.97)	**0.93** (0.89, 0.97)
16+ yrs education	**0.79** (0.74, 0.84)		**0.68** (0.64, 0.72)	**0.72** (0.68, 0.77)	**0.73** (0.69, 0.78)	**0.74** (0.70, 0.79)	**0.76** (0.72, 0.81)	**0.79** (0.74, 0.84)	**0.79** (0.74, 0.84)
12 yrs education (ref)	1.0		1.0	1.0	1.0	1.0	1.0	1.0	1.0
Unmarried	**1.17** (1.12, 1.22)			**1.22** (1.16, 1.27)	**1.20** (1.15, 1.26)	**1.19** (1.14, 1.24)	**1.19** (1.14, 1.25)	**1.18** (1.12, 1.23)	**1.18** (1.12, 1.23)
Married (ref)	1.0			1.0	1.0	1.0	1.0	1.0	1.0
2nd trimester prenatal care	0.96 (0.92, 1.00)				**0.94** (0.91, 0.98)	**0.94** (0.90, 0.98)	**0.94** (0.90, 0.98)	**0.92** (0.88, 0.95)	**0.92** (0.88, 0.95)
3rd trimester prenatal care	**0.85** (0.79, 0.91)				**0.82** (0.77, 0.89)	**0.82** (0.76, 0.88)	**0.80** (0.74, 0.86)	**0.76** (0.71, 0.82)	**0.76** (0.71, 0.82)
None/unknown prenatal care	**1.95** (1.85, 2.06)				**1.84** (1.75, 1.94)	**1.82** (1.73, 1.92)	**1.81** (1.72, 1.91)	**1.80** (1.71, 1.90)	**1.80** (1.71, 1.90)
1st trimester prenatal care (ref)	1.0				1.0	1.0	1.0	1.0	1.0
Smoked during pregnancy	**1.64** (1.54, 1.74)					**1.37** (1.28, 1.46)	**1.36** (1.27, 1.45)	**1.21** (1.13, 1.29)	**1.21** (1.13, 1.29)
Did not smoke (ref)	1.0					1.0	1.0	1.0	1.0
Weight gain <16 lb	**1.80** (1.73, 1.88)						**1.71** (1.64, 1.78)	**1.68** (1.61, 1.75)	**1.67** (1.61, 1.75)
Weight gain ≤41 lb	**0.76** (0.73, 0.80)						**0.75** (0.72, 0.78)	**0.74** (0.71, 0.77)	**0.74** (0.71, 0.77)
Weight gain 16-40 lb (ref)	1.0						1.0	1.0	1.0
Maternal medical risks	**2.08** (2.01, 2.15)							**2.01** (1.94, 2.08)	**2.00** (1.94, 2.07)
No maternal medical risks (ref.)	1.0							1.0	1.0
Infant congenital anomalies	**1.54** (1.37, 1.73)								**1.41** (1.26, 1.59)
No congenital anomalies (ref.)	1.0								1.0

Note: Significant terms are bolded.

Table B.2 Log Likelihood Tests of the Significance of Individual Risk Factors on Predicted Odds of Preterm Birth for V.I.-born, Caribbean-born, and U.S.-born Mothers, non-Hispanic Blacks, New York City, 2000-2009

	-2 Log Likelihood	df	Difference in -2 Log L	Chi-square Distribution	p-value
VI-born, NY (Stratified)					
Saturated Model	720.711	17			
Reduced Models					
Minus Age	729.99	13	9.28	χ^2 (4)	**.054**
Minus Education	726.36	14	5.65	χ^2 (3)	.130
Minus Marital Status	721.68	16	0.97	χ^2 (1)	.325
Minus Prenatal Care	733.74	14	13.03	χ^2 (3)	**.005**
Minus Smoking	733.85	16	13.14	χ^2 (1)	**.000**
Minus Weight gain	723.73	15	3.01	χ^2 (2)	.222
Minus Maternal Medical Risks	733.83	16	13.11	χ^2 (1)	**.000**
Minus Congenital Anomalies	722.91	16	2.20	χ^2 (1)	.138
Minus all demographic factors (age, education, marital status)	737.35	9	16.64	χ^2 (8)	**.034**
Minus medically-related factors (prenatal care, smoking, weight gain, medical risks)	750.96	10	30.25	χ^2 (7)	**.000**
Minus infant factors (congenital anomalies)	722.91	16	2.70	χ^2 (1)	.100
CA-born, NY (Stratified)					
Saturated Model	53478.82	17			
Reduced Models					
Minus Age	53611.36	13	132.54	χ^2 (4)	**.000**
Minus Education	53488.22	14	9.40	χ^2 (3)	**.024**
Minus Marital Status	53499.58	16	20.76	χ^2 (1)	**.000**
Minus Prenatal Care	53744.89	14	266.07	χ^2 (3)	**.000**
Minus Smoking	53483.71	16	4.89	χ^2 (1)	**.027**
Minus Weight gain	55637.15	15	2158.34	χ^2 (2)	**.000**
Minus Maternal Medical Risks	54528.11	16	1049.29	χ^2 (1)	**.000**
Minus Congenital Anomalies	53510.31	16	31.49	χ^2 (1)	**.000**
Minus all demographic factors (age, education, marital status)	53626.77	9	147.95	χ^2 (8)	**.000**
Minus medically-related factors (prenatal care, smoking, weight gain, medical risks)	55337.96	10	1859.14	χ^2 (7)	**.000**
Minus infant factors (congenital anomalies)	53510.31	16	31.49	χ^2 (1)	**.000**
US-born, NY (Stratified)					
Saturated Model	100148.05	17			
Reduced Models					
Minus Age	100490.62	13	342.57	χ^2 (4)	**.000**
Minus Education	100246.44	14	98.39	χ^2 (3)	**.000**
Minus Marital Status	100196.32	16	48.27	χ^2 (1)	**.000**
Minus Prenatal Care	100738.31	14	590.26	χ^2 (3)	**.000**
Minus Smoking	100177.72	16	29.67	χ^2 (1)	**.000**
Minus Weight gain	101114.00	15	965.95	χ^2 (2)	**.000**
Minus Maternal Medical Risks	101827.03	16	1678.98	χ^2 (1)	**.000**
Minus Congenital Anomalies	100178.40	16	30.35	χ^2 (1)	**.000**
Minus all demographic factors (age, education, marital status)	100564.15	9	416.1	χ^2 (8)	**.000**
Minus medically-related factors (prenatal care, smoking, weight gain, medical risks)	103490.01	10	3341.96	χ^2 (7)	**.000**
Minus infant factors (congenital anomalies)	100178.40	16	30.35	χ^2 (1)	**.000**

Table B.3 Stepwise Adjusted Predictors of Preterm Birth by Mother's Nativity, V.I.-born and Caribbean-born vs. U.S.-born, non-Hispanic Blacks, New York City, 2000-2009

VI, CA, US combined (NYC)	Unadjusted	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8	Model 9
	OR 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI
Virgin Islands-born	0.96 (0.79, 1.15)	0.95 (0.79, 1.15)	0.91 (0.76, 1.10)	0.93 (0.77, 1.12)	0.93 (0.77, 1.13)	0.94 (0.78, 1.14)	0.96 (0.79, 1.15)	0.95 (0.79, 1.15)	0.98 (0.81, 1.19)	0.98 (0.81, 1.19)
Caribbean-born	**0.88** (0.85, 0.90)	**0.88** (0.85, 0.90)	**0.82** (0.80, 0.85)	**0.82** (0.80, 0.85)	**0.84** (0.82, 0.87)	**0.86** (0.84, 0.88)	**0.88** (0.85, 0.90)	**0.88** (0.85, 0.91)	**0.89** (0.87, 0.92)	**0.89** (0.87, 0.92)
US-born (ref.)	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
Primiparous, 1	**0.91** (0.88, 0.93)	**0.91** (0.88, 0.93)	1.00 (0.97, 1.03)	**1.06** (1.03, 1.09)	**1.05** (1.02, 1.08)	**1.05** (1.02, 1.08)	**1.06** (1.03, 1.09)	**1.09** (1.06, 1.12)	**1.08** (1.04, 1.11)	**1.08** (1.04, 1.11)
Multiparous, 2 or more (ref)	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
Aged 15-19 years	**1.08** (1.03, 1.13)		**1.06** (1.01, 1.11)	0.97 (0.92, 1.02)	0.97 (0.92, 1.01)	0.98 (0.93, 1.03)	0.99 (0.94, 1.04)	1.00 (0.95, 1.04)	1.01 (0.96, 1.06)	1.01 (0.96, 1.06)
Aged 25-29 years	**1.08** (1.04, 1.12)		**1.10** (1.06, 1.14)	**1.16** (1.12, 1.21)	**1.18** (1.14, 1.23)	**1.18** (1.13, 1.22)	**1.17** (1.13, 1.22)	**1.16** (1.12, 1.21)	**1.15** (1.10, 1.19)	**1.15** (1.10, 1.19)
Aged 30-34 years	**1.23** (1.19, 1.28)		**1.28** (1.23, 1.33)	**1.38** (1.32, 1.43)	**1.42** (1.36, 1.48)	**1.41** (1.35, 1.47)	**1.40** (1.34, 1.46)	**1.38** (1.32, 1.43)	**1.33** (1.27, 1.39)	**1.33** (1.28, 1.39)
Aged 35-39 years	**1.53** (1.47, 1.60)		**1.60** (1.54, 1.67)	**1.73** (1.66, 1.81)	**1.80** (1.72, 1.88)	**1.78** (1.70, 1.86)	**1.76** (1.68, 1.84)	**1.71** (1.63, 1.79)	**1.60** (1.53, 1.68)	**1.60** (1.53, 1.68)
Aged 20-24 years (ref)	1.0		1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
< 12 yrs education	**1.09** (1.05, 1.12)			**1.12** (1.08, 1.16)	**1.10** (1.07, 1.14)	**1.10** (1.06, 1.14)	**1.08** (1.04, 1.12)	**1.06** (1.03, 1.10)	**1.06** (1.02, 1.09)	**1.06** (1.02, 1.10)
13-15 yrs education	**0.92** (0.89, 0.95)			**0.89** (0.86, 0.92)	**0.90** (0.87, 0.93)	**0.91** (0.88, 0.94)	**0.91** (0.88, 0.95)	**0.92** (0.89, 0.95)	**0.93** (0.90, 0.97)	**0.93** (0.90, 0.97)
16+ yrs education	**0.85** (0.81, 0.89)			**0.75** (0.72, 0.79)	**0.79** (0.75, 0.82)	**0.79** (0.76, 0.83)	**0.80** (0.76, 0.84)	**0.82** (0.78, 0.86)	**0.84** (0.81, 0.88)	**0.85** (0.81, 0.89)
12 yrs education (ref)	1.0			1.0	1.0	1.0	1.0	1.0	1.0	1.0
Unmarried	**1.15** (1.11, 1.18)				**1.19** (1.15, 1.22)	**1.18** (1.14, 1.22)	**1.17** (1.13, 1.21)	**1.17** (1.13, 1.21)	**1.16** (1.12, 1.20)	**1.16** (1.12, 1.20)
Married (ref)	1.0				1.0	1.0	1.0	1.0	1.0	1.0
2nd trimester prenatal care	**0.94** (0.91, 0.97)					**0.94** (0.91, 0.97)	**0.94** (0.91, 0.97)	**0.93** (0.90, 0.96)	**0.92** (0.89, 0.95)	**0.91** (0.89, 0.94)
3rd trimester prenatal care	**0.77** (0.73, 0.81)					**0.78** (0.73, 0.82)	**0.77** (0.73, 0.81)	**0.76** (0.72, 0.80)	**0.72** (0.68, 0.76)	**0.72** (0.68, 0.76)
None/unknown prenatal care	**1.87** (1.79, 1.96)					**1.82** (1.74, 1.90)	**1.80** (1.72, 1.88)	**1.78** (1.71, 1.86)	**1.79** (1.72, 1.88)	**1.79** (1.71, 1.87)
1st trimester prenatal care (ref)	1.0					1.0	1.0	1.0	1.0	1.0
Smoked during pregnancy	**1.63** (1.54, 1.72)						**1.42** (1.33, 1.51)	**1.41** (1.33, 1.50)	**1.25** (1.17, 1.33)	**1.25** (1.17, 1.33)
Did not smoke (ref)	1.0						1.0	1.0	1.0	1.0
Weight gain <16 lb	**1.82** (1.76, 1.88)							**1.74** (1.68, 1.80)	**1.71** (1.65, 1.77)	**1.71** (1.65, 1.77)
Weight gain ≤41 lb	**0.78** (0.75, 0.80)							**0.76** (0.73, 0.79)	**0.75** (0.72, 0.78)	**0.75** (0.72, 0.77)
Weight gain 16-40 lb (ref)	1.0							1.0	1.0	1.0
Maternal medical risks	**2.12** (2.06, 2.17)								**2.06** (2.01, 2.12)	**2.05** (2.00, 2.11)
No maternal medical risks	1.0								1.0	1.0
Infant congenital anomalies	**1.61** (1.47, 1.77)									**1.48** (1.35, 1.62)
No congenital anomalies (ref.)	1.0									1.0

Note: Significant terms are bolded.

Table B.4 Log Likelihood Tests of the Significance of Interactions of Nativity with Demographic, Medical, and Infant Risks on the Odds of Preterm Birth for V.I.-born and Caribbean-born Mothers vs. U.S.-born Mothers, non-Hispanic Blacks, New York City, 2000-2009

	-2 Log Likelihood	df	Difference in -2 Log L (test statistic)	Chi-square Distribution	p-value
VI-born, NYC					
Saturated Model	154379.57	51			
Reduced Models					
Minus VI * Age	154382.37	47	2.80	χ^2 (4)	.592
Minus VI * Education	154384.55	48	4.98	χ^2 (3)	.173
Minus VI * Marital Status	154382.32	50	2.75	χ^2 (1)	.097
Minus VI * Prenatal Care	154382.48	48	2.91	χ^2 (3)	.406
Minus VI * Smoking	154383.33	50	3.76	χ^2 (1)	**.052**
Minus VI * Weight gain	154382.08	49	2.51	χ^2 (2)	.285
Minus VI * Maternal Medical Risks	154379.64	50	0.07	χ^2 (1)	.791
Minus VI * Congenital Anomalies	154380.58	50	1.01	χ^2 (1)	.315
Minus VI * demographic factors (age, education, marital status)	154392.24	43	12.67	χ^2 (8)	.124
Minus VI * medically-related factors (prenatal care, smoking, weight gain, medical risks)	154388.66	44	9.09	χ^2 (7)	.246
Minus VI * infant factors (congenital anomalies)	154380.58	50	1.01	χ^2 (1)	.315
CA-born, NYC					
Saturated Model	154379.57	51			
Reduced Models					
Minus CA * Age	154409.81	47	30.24	χ^2 (4)	**.000**
Minus CA * Education	154420.57	48	41.00	χ^2 (3)	**.000**
Minus CA * Marital Status	154380.86	50	1.29	χ^2 (1)	.256
Minus CA * Prenatal Care	154386.12	48	6.55	χ^2 (3)	.088
Minus CA * Smoking	154380.04	50	0.47	χ^2 (1)	.493
Minus CA * Weight gain	154382.68	49	3.11	χ^2 (2)	.211
Minus CA * Maternal Medical Risks	154385.63	50	6.06	χ^2 (1)	**.014**
Minus CA * Congenital Anomalies	154381.02	50	1.45	χ^2 (1)	.229
Minus CA * demographic factors (age, education, marital status)	154441.75	43	62.18	χ^2 (8)	**.000**
Minus CA * medically-related factors (prenatal care, smoking, weight gain, medical risks)	154395.48	44	15.91	χ^2 (7)	**.026**
Minus CA * infant factors (congenital anomalies)	154381.02	50	1.45	χ^2 (1)	.229
Reference: US-born, NYC					

Note: All of the -2LogL differences in this table were originally negative values.

Table B.5 Stepwise-Adjusted Predictors of Preterm Birth, V.I.-born, non-Hispanic Blacks, U.S. Virgin Islands, 2000-2004

VI-born, VI Resident (VIVI)	Unadjusted	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
	OR 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI
Primiparous, 1	1.17 (0.95, 1.44)	1.09 (0.86, 1.37)	1.14 (0.89, 1.45)	1.14 (0.89, 1.45)	1.23 (0.96, 1.57)	1.23 (0.96, 1.57)	1.21 (0.94, 1.55)
Multiparous, 2 or more (ref.)	1.0	1.0	1.0	1.0	1.0	1.0	1.0
Aged 15-19 years	1.33 (1.00, 1.77)	1.29 (0.96, 1.74)	1.15 (0.84, 1.59)	1.15 (0.84, 1.58)	1.12 (0.81, 1.54)	1.12 (0.81, 1.54)	1.14 (0.82, 1.57)
Aged 25-29 years	1.17 (0.89, 1.54)	1.19 (0.90, 1.57)	1.24 (0.93, 1.65)	1.24 (0.93, 1.66)	1.29 (0.97, 1.73)	1.29 (0.97, 1.73)	1.27 (0.95, 1.70)
Aged 30-34 years	0.92 (0.66, 1.27)	0.93 (0.67, 1.30)	0.98 (0.69, 1.39)	0.99 (0.69, 1.40)	1.04 (0.73, 1.48)	1.03 (0.72, 1.48)	1.01 (0.70, 1.44)
Aged 35-39 years	0.92 (0.58, 1.47)	0.95 (0.59, 1.52)	0.97 (0.60, 1.57)	0.97 (0.60, 1.58)	1.02 (0.63, 1.66)	1.02 (0.62, 1.65)	0.97 (0.59, 1.58)
Aged 20-24 years (ref.)	1.0	1.0	1.0	1.0	1.0	1.0	1.0
< 12 yrs education	1.18 (0.92, 1.52)		1.15 (0.88, 1.50)	1.15 (0.88, 1.50)	1.14 (0.87, 1.49)	1.14 (0.87, 1.49)	1.13 (0.87, 1.49)
13-15 yrs education	0.78 (0.58, 1.04)		0.78 (0.58, 1.05)	0.78 (0.58, 1.05)	0.80 (0.59, 1.08)	0.80 (0.59, 1.08)	0.79 (0.59, 1.07)
16+ yrs education	0.94 (0.68, 1.30)		0.93 (0.66, 1.31)	0.93 (0.65, 1.33)	0.95 (0.66, 1.35)	0.95 (0.66, 1.36)	0.95 (0.66, 1.35)
12 yrs education (ref.)	1.0		1.0	1.0	1.0	1.0	1.0
Unmarried	1.13 (0.87, 1.48)			1.03 (0.77, 1.38)	0.98 (0.73, 1.32)	0.98 (0.73, 1.32)	0.97 (0.72, 1.31)
Married (ref.)	1.0			1.0	1.0	1.0	1.0
2nd trimester prenatal care	1.12 (0.88, 1.42)				1.10 (0.86, 1.40)	1.10 (0.86, 1.40)	1.09 (0.85, 1.40)
3rd trimester prenatal care	1.13 (0.73, 1.75)				1.10 (0.70, 1.72)	1.10 (0.70, 1.72)	1.02 (0.65, 1.60)
None/unknown prenatal care	**2.77** (1.78, 4.31)				**2.79** (1.76, 4.41)	**2.79** (1.76, 4.41)	**2.46** (1.54, 3.91)
1st trimester prenatal care (ref.)	1.0				1.0	1.0	1.0
Smoked during pregnancy	1.15 (0.25, 5.26)					1.24 (0.27, 5.76)	1.23 (0.26, 5.73)
Did not smoke (ref.)	1.0					1.0	1.0
Maternal medical risks	**1.70** (1.37, 2.11)						**1.64** (1.32, 2.05)
No maternal medical risks (ref.)	1.0						1.0

Note: Significant terms are bolded.

(table continues)

Table B.6 Stepwise-Adjusted Predictors of Preterm Birth, CA-born, non-Hispanic Blacks, U.S. Virgin Islands, 2000-2004

CA-born, VI Resident (CAVI)	Unadjusted	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
	OR 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI
Primiparous, 1	1.21 (0.90, 1.64)	1.12 (0.79, 1.58)	1.09 (0.77, 1.56)	1.09 (0.76, 1.56)	1.12 (0.78, 1.61)	1.13 (0.79, 1.61)	1.10 (0.77, 1.57)
Multiparous, 2 or more (ref.)	1.0	1.0	1.0	1.0	1.0	1.0	1.0
Aged 15-19 years	1.40 (0.82, 2.40)	1.36 (0.79, 2.34)	1.21 (0.69, 2.12)	1.21 (0.69, 2.12)	1.18 (0.67, 2.07)	1.17 (0.67, 2.06)	1.19 (0.67, 2.10)
Aged 25-29 years	0.80 (0.51, 1.23)	0.82 (0.52, 1.28)	0.79 (0.50, 1.25)	0.80 (0.50, 1.26)	0.81 (0.51, 1.28)	0.81 (0.51, 1.27)	0.77 (0.48, 1.22)
Aged 30-34 years	0.70 (0.45, 1.09)	0.73 (0.46, 1.15)	0.69 (0.43, 1.10)	0.70 (0.43, 1.12)	0.71 (0.44, 1.14)	0.71 (0.44, 1.14)	0.64 (0.40, 1.04)
Aged 35-39 years	1.17 (0.76, 1.81)	1.23 (0.78, 1.95)	1.18 (0.74, 1.87)	1.18 (0.73, 1.91)	1.23 (0.76, 1.99)	1.23 (0.76, 1.99)	1.04 (0.64, 1.71)
Aged 20-24 years (ref.)	1.0	1.0	1.0	1.0	1.0	1.0	1.0
< 12 yrs education	**1.58** (1.13, 2.21)		**1.53** (1.09, 2.16)	**1.53** (1.09, 2.16)	**1.50** (1.07, 2.12)	**1.50** (1.06, 2.12)	1.41 (0.99, 2.00)
13-15 yrs education	1.40 (0.92, 2.15)		1.45 (0.94, 2.25)	1.46 (0.94, 2.25)	1.46 (0.94, 2.26)	1.45 (0.94, 2.26)	1.41 (0.90, 2.19)
16+ yrs education	0.98 (0.58, 1.65)		1.04 (0.61, 1.78)	1.04 (0.61, 1.79)	1.06 (0.62, 1.82)	1.05 (0.61, 1.81)	1.03 (0.59, 1.78)
12 yrs education (ref.)	1.0		1.0	1.0	1.0	1.0	1.0
Unmarried	1.09 (0.81, 1.47)			1.02 (0.74, 1.41)	1.00 (0.72, 1.38)	0.99 (0.71, 1.38)	0.94 (0.67, 1.31)
Married (ref.)	1.0			1.0	1.0	1.0	1.0
2nd trimester prenatal care	1.08 (0.78, 1.50)				1.02 (0.73, 1.43)	1.02 (0.73, 1.43)	1.00 (0.71, 1.41)
3rd trimester prenatal care	1.37 (0.80, 2.36)				1.30 (0.74, 2.28)	1.30 (0.74, 2.28)	1.19 (0.67, 2.10)
None/unknown prenatal care	1.87 (0.86, 4.04)				1.77 (0.80, 3.93)	1.78 (0.80, 3.94)	1.33 (0.59, 3.00)
1st trimester prenatal care (ref.)	1.0				1.0	1.0	1.0
Smoked during pregnancy	1.88 (0.20, 18.2)					2.08 (0.21, 20.9)	1.82 (0.18, 18.3)
Did not smoke (ref.)	1.0					1.0	1.0
Maternal medical risks	**2.38** (1.77, 3.19)						**2.27** (1.68, 3.08)
No maternal medical risks (ref.)	1.0						1.0

Note: Significant terms are bolded.

[a]The *p* -value indicates the contribution of predictors to the model based on the likelihood ratio test comparing models with and without the designated predictors.

Table B.7 Log Likelihood Tests of the Significance of Individual Predictors on Odds of Preterm Birth for non-Hispanic Black Mothers, U.S. Virgin Islands, 2000-2004

	-2 Log Likelihood	df	Difference in -2 Log L (test statistic)	Chi-square Distribution	*p* value
VI-born, V.I. Resident (Stratified)					
Saturated Model	2373.32	14			
Reduced Models					
Minus Age	2377.04	10	3.72	χ^2 (4)	.445
Minus Education	2377.47	11	4.15	χ^2 (3)	.246
Minus Marital Status	2373.35	13	0.03	χ^2 (1)	.862
Minus Prenatal Care	2386.51	11	13.19	χ^2 (3)	**.004**
Minus Smoking	2373.38	13	0.06	χ^2 (1)	.806
Minus Maternal Medical Risks	2392.18	13	18.86	χ^2 (1)	**<.001**
Minus all demographic factors (age, education, marital status)	2381.92	6	8.60	χ^2 (8)	.377
Minus medically-related factors (prenatal care, smoking, medical risks)	2409.40	9	36.08	χ^2 (5)	**<.001**
CA-born, V.I. Resident (Stratified)					
Saturated Model	1189.49	14			
Reduced Models					
Minus Age	1197.09	10	7.60	χ^2 (4)	.107
Minus Education	1194.37	11	4.88	χ^2 (3)	.181
Minus Marital Status	1189.63	13	0.14	χ^2 (1)	.708
Minus Prenatal Care	1190.23	11	0.74	χ^2 (3)	.864
Minus Smoking	1189.72	13	0.23	χ^2 (1)	.632
Minus Maternal Medical Risks	1216.70	13	27.21	χ^2 (1)	**<.001**
Minus all demographic factors (age, education, marital status)	1202.38	6	12.89	χ^2 (8)	.116
Minus medically-related factors (prenatal care, smoking, medical risks)	1219.51	9	30.02	χ^2 (5)	**<.001**

Note: All of the -2LogL differences in this table were originally negative values.

Table B.8 Stepwise Adjusted Predictors of Preterm Birth for V.I.-born Mothers, by Migrant Status, Virgin Islands Residents vs. New York City Residents

VI-born by Migrant Status	Unadjusted	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
	OR 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	OR 95% CI	*OR* 95% CI
VI-born, VI Resident (VIVI)	**1.33** (1.08, 1.65)	**1.34** (1.08, 1.65)	**1.35** (1.09, 1.69)	**1.35** (1.09, 1.69)	**1.35** (1.09, 1.69)	**1.44** (1.15, 1.80)	**1.44** (1.15, 1.80)	**1.43** (1.14, 1.79)
VI-born, NY Resident (VINY)	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
Primiparous, 1	1.07 (0.89, 1.29)	1.08 (0.90, 1.30)	1.08 (0.88, 1.32)	1.13 (0.91, 1.40)	1.13 (0.91, 1.40)	1.19 (0.96, 1.47)	1.19 (0.96, 1.47)	1.18 (0.95, 1.46)
Multiparous, 2 or more (ref)		1.0	1.0	1.0	1.0	1.0	1.0	1.0
Aged 15-19 years	1.30 (1.00, 1.70)		1.25 (0.94, 1.65)	1.13 (0.84, 1.51)	1.13 (0.84, 1.52)	1.11 (0.83, 1.50)	1.11 (0.83, 1.49)	1.14 (0.84, 1.53)
Aged 25-29 years	1.20 (0.94, 1.53)		1.24 (0.97, 1.59)	**1.31** (1.01, 1.68)	1.30 (1.00, 1.68)	**1.34** (1.03, 1.73)	**1.34** (1.03, 1.73)	**1.32** (1.02, 1.71)
Aged 30-34 years	0.98 (0.74, 1.29)		1.05 (0.79, 1.39)	1.11 (0.83, 1.50)	1.10 (0.81, 1.48)	1.14 (0.84, 1.54)	1.14 (0.85, 1.55)	1.12 (0.83, 1.52)
Aged 35-39 years	1.25 (0.89, 1.75)		1.38 (0.97, 1.95)	**1.45** (1.01, 2.07)	1.43 (0.99, 2.04)	**1.46** (1.02, 2.10)	**1.47** (1.03, 2.12)	1.43 (0.99, 2.06)
Aged 20-24 years (ref)	1.0		1.0	1.0	1.0	1.0	1.0	1.0
< 12 yrs education	1.23 (0.98, 1.54)			1.23 (0.97, 1.57)	1.23 (0.97, 1.57)	1.23 (0.96, 1.57)	1.23 (0.96, 1.56)	1.22 (0.95, 1.56)
13-15 yrs education	0.94 (0.74, 1.20)			0.96 (0.75, 1.23)	0.96 (0.75, 1.22)	0.98 (0.77, 1.26)	0.98 (0.76, 1.25)	0.97 (0.76, 1.25)
16+ yrs education	0.95 (0.72, 1.26)			0.90 (0.67, 1.22)	0.89 (0.65, 1.20)	0.91 (0.67, 1.24)	0.91 (0.67, 1.24)	0.92 (0.68, 1.25)
12 yrs education (ref)	1.0			1.0	1.0	1.0	1.0	1.0
Unmarried	1.00 (0.80, 1.24)				0.93 (0.73, 1.18)	0.89 (0.70, 1.14)	0.90 (0.70, 1.14)	0.89 (0.70, 1.14)
Married (ref)	1.0				1.0	1.0	1.0	1.0
2nd trimester prenatal care	1.08 (0.87, 1.33)					1.09 (0.88, 1.35)	1.09 (0.88, 1.36)	1.09 (0.88, 1.35)
3rd trimester prenatal care	0.92 (0.62, 1.37)					0.97 (0.65, 1.44)	0.97 (0.65, 1.45)	0.90 (0.60, 1.35)
None/unknown prenatal care	**2.54** (1.78, 3.61)					**2.77** (1.92, 3.98)	**2.77** (1.92, 3.98)	**2.56** (1.77, 3.69)
1st trimester prenatal care (ref)	1.0					1.0	1.0	1.0
Smoked during pregnancy	0.54 (0.13, 2.27)						0.58 (0.13, 2.50)	0.57 (0.13, 2.47)
Did not smoke (ref)	1.0						1.0	1.0
Maternal medical risks	**1.76** (1.46, 2.13)							**1.72** (1.43, 2.09)
No maternal medical risks (ref.)	1.0							1.0

Note: Significant terms are bolded.

(table continues)

Table B.9 Stepwise Adjusted Predictors of Preterm Birth for CA-born Mothers, by Migrant Status, Virgin Islands Residents vs. New York City Residents

CA-born by Migrant Status	Unadjusted	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
	OR 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	*OR* 95% CI	OR 95% CI	*OR* 95% CI
CA-born, VI Resident (CAVI)	**1.48** (1.28, 1.71)	**1.49** (1.29, 1.72)	**1.50** (1.30, 1.74)	**1.49** (1.28, 1.72)	**1.49** (1.29, 1.72)	**1.51** (1.31, 1.75)	**1.52** (1.31, 1.75)	**1.55** (1.34, 1.79)
CA-born, NY Resident (CANY)	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
Primiparous, 1	**1.06** (1.01, 1.11)	**1.06** (1.02, 1.11)	**1.19** (1.14, 1.25)	**1.21** (1.15, 1.27)	**1.20** (1.15, 1.26)	**1.21** (1.15, 1.27)	**1.21** (1.15, 1.27)	**1.19** (1.13, 1.25)
Multiparous, 2 or more (ref)		1.0	1.0	1.0	1.0	1.0	1.0	1.0
Aged 15-19 years	**1.14** (1.03, 1.27)		1.09 (0.98, 1.22)	1.07 (0.96, 1.19)	1.05 (0.95, 1.17)	1.07 (0.96, 1.19)	1.07 (0.96, 1.19)	1.10 (0.98, 1.22)
Aged 25-29 years	1.06 (0.99, 1.14)		**1.11** (1.03, 1.18)	**1.13** (1.05, 1.21)	**1.15** (1.07, 1.23)	**1.15** (1.07, 1.23)	**1.15** (1.07, 1.23)	**1.12** (1.05, 1.20)
Aged 30-34 years	**1.22** (1.14, 1.30)		**1.30** (1.21, 1.39)	**1.33** (1.24, 1.42)	**1.37** (1.28, 1.47)	**1.37** (1.28, 1.47)	**1.37** (1.28, 1.47)	**1.31** (1.22, 1.40)
Aged 35-39 years	**1.48** (1.38, 1.59)		**1.61** (1.50, 1.73)	**1.64** (1.53, 1.77)	**1.70** (1.58, 1.83)	**1.70** (1.57, 1.83)	**1.70** (1.57, 1.83)	**1.55** (1.44, 1.67)
Aged 20-24 years (ref)	1.0		1.0	1.0	1.0	1.0	1.0	1.0
< 12 yrs education	0.97 (0.91, 1.04)			0.99 (0.92, 1.05)	0.98 (0.92, 1.04)	0.98 (0.92, 1.04)	0.98 (0.91, 1.04)	0.97 (0.91, 1.04)
13-15 yrs education	0.92 (0.87, 0.97)			**0.92** (0.87, 0.97)	**0.92** (0.87, 0.97)	**0.92** (0.87, 0.97)	**0.92** (0.87, 0.97)	**0.93** (0.88, 0.98)
16+ yrs education	0.94 (0.88, 1.01)			**0.87** (0.81, 0.93)	**0.89** (0.83, 0.96)	**0.89** (0.83, 0.95)	**0.89** (0.83, 0.95)	**0.90** (0.84, 0.97)
12 yrs education (ref)	1.0			1.0	1.0	1.0	1.0	1.0
Unmarried	**1.07** (1.02, 1.12)				**1.13** (1.08, 1.19)	**1.13** (1.08, 1.19)	**1.13** (1.08, 1.19)	**1.11** (1.06, 1.17)
Married (ref)	1.0				1.0	1.0	1.0	1.0
2nd trimester prenatal care	0.92 (0.87, 0.97)					**0.93** (0.88, 0.98)	**0.93** (0.88, 0.98)	**0.92** (0.87, 0.97)
3rd trimester prenatal care	0.71 (0.65, 0.77)					**0.71** (0.65, 0.77)	**0.71** (0.65, 0.77)	**0.68** (0.62, 0.74)
None/unknown prenatal care	**1.68** (1.55, 1.81)					**1.69** (1.56, 1.83)	**1.69** (1.56, 1.83)	**1.73** (1.59, 1.87)
1st trimester prenatal care (ref)	1.0					1.0	1.0	1.0
Smoked during pregnancy	**1.49** (1.16, 1.92)						**1.45** (1.13, 1.87)	**1.32** (1.03, 1.71)
Did not smoke (ref)	1.0						1.0	1.0
Maternal medical risks	**2.18** (2.09, 2.28)							**2.16** (2.07, 2.26)
No maternal medical risks (ref.)	1.0							1.0

Note: Significant terms are bolded.

Table B.10 Log Likelihood Tests of the Interactive Effect of Migrant Status with Demographic and Medically-related Predictors on the Odds of Preterm Birth, V.I.- and CA-born Residents of the Virgin Islands vs. V.I.- and CA-born Migrants to New York

	-2 Log Likelihood	df	Difference in -2 Log L (test statistic)	Chi-square Distribution	p-value
VIVI (non-migrant) with VINY (migrant) as the reference					
Saturated Model	3099.65	28			
Reduced Models					
Minus VI * Age	3109.64	24	9.99	χ^2 (4)	**.041**
Minus VI * Education	3106.39	25	6.74	χ^2 (3)	.081
Minus VI * Marital Status	3100.56	27	0.91	χ^2 (1)	.340
Minus VI * Prenatal Care	3101.69	25	2.04	χ^2 (3)	.564
Minus VI * Smoking	3102.39	27	2.74	χ^2 (1)	.098
Minus VI * Maternal Medical Risks	3100.72	27	1.07	χ^2 (1)	.301
Minus VI * demographic factors (age, education, marital status)	3119.64	20	19.99	χ^2 (8)	**.010**
Minus VI * medically-related factors (prenatal care, smoking, medical risks)	3105.38	23	5.73	χ^2 (7)	.572
CAVI (non-migrant) with CANY (migrant) as the reference					
Saturated Model	55218.81	28			
Reduced Models					
Minus CA * Age	55229.58	24	10.77	χ^2 (4)	**.029**
Minus CA * Education	55224.74	25	5.93	χ^2 (3)	.115
Minus CA * Marital Status	55219.86	27	1.05	χ^2 (1)	.306
Minus CA * Prenatal Care	55223.09	25	4.28	χ^2 (3)	.233
Minus CA * Smoking	55218.88	27	0.07	χ^2 (1)	.791
Minus CA * Maternal Medical Risks	55218.90	27	0.09	χ^2 (1)	.764
Minus CA * demographic factors (age, education, marital status)	55235.49	20	16.68	χ^2 (8)	**.034**
Minus CA * medically-related factors (prenatal care, smoking, medical risks)	55223.27	23	4.46	χ^2 (7)	.726

Note: All of the -2LogL differences in this table were originally negative values.