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Correlates and consequences of gendered racial stress among pregnant Black women

By

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Doctor of Philosophy

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Correlates and consequences of gendered racial stress among pregnant Black women

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A.B., Princeton University, 2010

MPH, Emory University, 2016

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Abstract

Correlates and consequences of gendered racial stress among pregnant Black women

By Lasha S. Clarke

Racial disparities in gestational age persist after control for sociobehavioral factors. Gestational ages before term are associated with greater maternal and infant morbidity. A confluence of evidence supports that racism, acting as a stressor for Black women, underlies this disparity. Given their intersecting identities as both Black and women, Black women may experience a distinct, hybrid form of stress called gendered racial stress. Considering a biopsychosocial framework of cumulative risk over the lifecourse, gendered racial stress may be a mechanism through which pregnancy outcomes are affected. Gaps in the growing literature on Black maternal gendered racial stress include: limited use of available intersectional measures; gendered racism's earlier life correlates; its association with embodied biomarkers of stress; and its effect on gestational age. This dissertation uses data from a prospective cohort study of pregnant Black women seeking prenatal care in Atlanta, GA to address these gaps.

In **Aim 1**, we assessed the association of adverse childhood experiences (ACEs) with adult gendered racial stress. Increased ACEs were associated with increased gendered racial stress scores, even after control for confounders. This association appeared stronger among women who reported accepting unfair treatment and keeping it to themselves, versus those who reported saying and/or doing something about it.

In **Aim 2**, we estimated the marginal effect of gendered racial stress on spontaneous preterm (sPTB) and early term birth (sETB). We found null associations of total gendered racial stress and most subscale (i.e., burden, coping, personal history, and work) scores with gestational age. Higher scores on the instrument's racism subscale—which assesses experience/perception of racial stereotypes, White privilege, and the impact of racism on Black children's lives—were associated with increased probability of sPTB, but not sETB, as compared to term birth.

In **Aim 3**, we examined the association of gendered racial stress with C-reactive protein (CRP), an inflammatory biomarker. Median CRP was above previously published thresholds for sub-clinical inflammation. There was a null relationship between exposure to gendered racial stress and elevated CRP, with no moderation by education or body mass index.

Gendered racial stress may exist on a trajectory extending from a woman's childhood to the wellbeing of her offspring. Though limited by sample size, these studies are an important step toward characterizing gendered racial stress pathways among pregnant Black women. Future population-based investigation may further clarify the etiology and embodiment of gendered racial stress over the lifecourse.

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Table of Contents

List of Figures

List of Tables

Chapter 1: Public Health Significance and Literature Review

Public Health Significance

Black women in the US are burdened with a disproportionate rate of adverse birth outcomes, which are inadequately explained by maternal socioeconomic status or prenatal health behaviors. The infant mortality rate for Black Americans is more than twice that for White Americans (Murphy, Xu, Kochanek, & Arias, 2018), and much of this racial disparity can be explained by low birthweight and preterm delivery (Rosenthal & Lobel, 2011). Moreover, the effects of racial disparities on adverse birth outcomes are often far-reaching and intergenerational, extending from the mother's own infancy through her offspring's long-term development. Those born preterm, for instance, are more likely to experience impaired neurodevelopment in infancy and childhood (Jarjour, 2015), and to have greater adult risk for psychiatric illness (Nosarti et al., 2012). Additionally, where a Black woman lives may compound her risk for poor maternal and infant health outcomes (Bell et al., 2006). In the US South, where the legacy of slavery persists (Owens et al., 2019), the pattern of racial disparities is particularly apparent. Georgia, in particular, has one of the highest Black maternal death rates in the US (47 per 100,000 live births).[1] The state's preterm birth rate—11.7 per 100,000 live births—earned it a failing grade of 'F' in a 2020 March of Dimes report, and is driven so high due in part to racial disparities[2]. In Georgia, Black women are 46% more likely to give birth preterm than White counterparts[2]. This disproportionate risk for Black women and infants amplifies the national call to better safeguard their health and lives. The proposed work will contribute to a more nuanced understanding of Black women's lived experience—particularly in Georgia—during pregnancy and throughout their lives, and offer insight into ways to reduce racial disparities in adverse birth outcomes.

[1] https://dph.georgia.gov/maternal-mortality
[2] https://www.marchofdimes.org/peristats/tools/reportcard.aspx?frmodrc=1&reg=06

Literature Review

1.1. Racism and sexism act separately as stressors, and contribute independently to poor mental and reproductive health among Black women. Mounting theoretical and empirical evidence suggests that racism, acting as a stressor for Black women, may help explain racial disparities in birth outcomes (Clark, Anderson, Clark, & Williams, 1999; Tyan Parker Dominguez, Dunkel-Schetter, Glynn, Hobel, & Sandman, 2008; Giscombé & Lobel, 2005; Cynthia Prather et al., 2018; C. Prather, Fuller, Marshall, & Jeffries, 2016; Lisa Rosenthal & Marci Lobel, 2011; Williams, Neighbors, & Jackson, 2003). Interpersonal racism, defined as discriminatory practices enacted on a person due to their racial/ethnic group (C. P. Jones, 2000) has been independently associated with greater psychological distress, including symptoms of depression and anxiety among Black versus White women, perhaps through chronic activation of neuroimmune stress pathways (Goosby, Cheadle, & Mitchell, 2018; H. L. Jones, Cross Jr, & DeFour, 2007; L. Rosenthal & M. Lobel, 2011). In turn, depression—known to be more prevalent among women (Kessler, 2003; Klein & Corwin, 2002) and among Black people (Miranda, Siddique, Belin, & Kohn-Wood, 2005)— may contribute to racial disparities in adverse birth outcomes (Giscombé & Lobel, 2005; M. R. Kramer, Hogue, Dunlop, & Menon, 2011; M. R. Kramer & Hogue, 2009; Ncube, Enquobahrie, & Gavin, 2017; Schulz et al., 2006). Gender-focused studies have also found sexism to be an independent stressor for Black women, for whom it is associated with greater psychological distress as compared to White women (Dawn M. Szymanski & Stewart, 2010). Thus, stress due to racism and sexism experienced by Black women before and during pregnancy are important in understanding birth outcome disparities. Interdisciplinary research further demonstrates that the experience of Black women is unique not just due to race *or* gender, but due to the convergence of their multiple lifelong identities as women *and* Black (Essed, 1991), and in pregnancy, as mothers (Nuru-Jeter et al., 2009; Lisa Rosenthal & Marci Lobel, 2011). Scholars of intersectionality theory posit that instruments and studies, like many cited above, designed to measure racism and sexism as independent effects may not fully or accurately

capture the depth and breadth of Black women's lived experiences (T. T. Lewis & Van Dyke, 2018). Instead, those scholars suggest that empirical study of gendered racism should utilize measures that explore experiences of racism and sexism together, without attempt to disentangle them (Bauer, 2014; Bowleg, 2008; Bowleg & Bauer, 2016; Cho, Crenshaw, & McCall, 2013; J. A. Lewis, Williams, Peppers, & Gadson, 2017; Thomas, Witherspoon, & Speight, 2008). *Although researchers have examined the independent effects that racism and sexism have on Black women's health, this approach limits our understanding of how the two stressors intersect* (Thomas et al., 2008).

1.2. However, relatively few empirical studies employ intersectional frameworks or instruments that consider racism and sexism's joint effect—called gendered racism—in an effort to unpack and contextualize racial disparities in poor health outcomes. This concept of intersectionality has its roots in Black feminist theory (P. H. Collins, 2002), which, for decades, has highlighted the unique experiences that Black women face due to both their sex and race (J. A. Lewis et al., 2017). Kimberlé Williams Crenshaw coined the term intersectionality in her work as a Black feminist and legal scholar after observing the unique position of Black women in antidiscrimination law, which often excluded Black women at the intersection of racial and gender discrimination (Crenshaw, 1989). So, considering that Black women could perceive incidents of discrimination as both racist *and* sexist simultaneously, Crenshaw held that Black women's experiences of their gender and race could be fully understood by looking at their complex intersections, and not by looking at dimensions of identity separately (Crenshaw, 1989, 1990; Thomas et al., 2008). At their intersection, sexism and racism combine to form a distinct, hybrid form of oppression called gendered racism, which is characterized by racist constructions of gender roles (Essed, 1991; J. A. Lewis et al., 2017). Gendered racial theory supports Crenshaw's observations that Black women face discrimination due to their racial minority status *and* because they are women, and that they may perceive discrimination based on the intersection of those identities, and not based on one identity or the other (Essed, 1991; Thomas et al., 2008). *Use of intersectional measurement tools, as planned in the proposed work, may*

best facilitate understanding of Black women's disproportionate health risks (J. A. Lewis et al., 2017; Thomas, Hacker, & Hoxha, 2011; Thomas et al., 2008).

1.3. Gendered racial stress may be a major contributor to Black women's adverse psychosocial and pregnancy outcomes. While race- and gender-related stress contribute separately to high global stress and psychiatric symptoms in Black women throughout their lifecourse (Klonoff & Landrine, 1995; Klonoff, Landrine, & Ullman, 1999; Cheryl L Woods-Giscombé & Lobel, 2008), gendered racism may present extra exposure to stress for Black women[3] (Essed, 1991; F. M. Jackson, Phillips, Hogue, & Curry-Owens, 2001). Exposure to gendered racism creates an environment in which Black women feel they must maintain a persistent vigilance in anticipation of racist events against themselves and their children (F. M. Jackson, Hogue, & Phillips, 2005). Researchers have found this "race-related vigilance" (Powell, Jesdale, & Lemon, 2016) to be associated with feelings of helplessness in Black populations (Davis & Stevenson, 2006). According to the cognitive activation of stress (CATS), stress arises when there is a discrepancy between individuals' expectations and their realities (Ursin & Eriksen, 2004). Individuals can respond to chronic stress with coping (the expectation that she will be able to control a situation and achieve a positive outcome), hopelessness (the expectation that any action taken will result in a negative outcome), and helplessness (the expectation that no control over the outcome is possible) (Ursin & Eriksen, 2004). Gendered racism may reinforce the sense of uncontrollable outcomes; each instance of perceived sexist and racist action may sustain Black women's views that gendered racism is to be expected given their social identities (Broman, Mavaddat, & Hsu, 2000). Consistent with CATS and the learned helplessness model, gendered racial stress may increase susceptibility to depression among Black women

[3] While the concept of gendered racism can apply to multiple groups' experiences of oppression due to the intersection of race/ethnicity and gender, we focus here on gendered racism as experienced by Black women in the U.S. to further understanding of their uniquely disproportionate risk for adverse psychosocial and reproductive health outcomes.

(Abramson, Garber, & Seligman, 1980; Abramson, Seligman, & Teasdale, 1978; Broman et al., 2000; Fernando, 1984).

Poor psychological health and depression during pregnancy are known to adversely affect birth outcomes (Tyan Parker Dominguez et al., 2008; T. P. Dominguez, Schetter, Mancuso, Rini, & Hobel, 2005; C. Dunkel Schetter & Tanner, 2012; Grote et al., 2010; Pagel, Smilkstein, Regen, & Montano, 1990), including gestational age at delivery (Butler & Behrman, 2007). Generally, 37 weeks' gestation has been used to dichotomize births as preterm (<37 weeks) or full term (≥37 weeks). However, research suggests there may be a gradient of risk even beyond 37 weeks, such that early term ($37^{0/7}$-$38^{6/7}$ weeks) infants fare worse developmentally than do full term (≥39 weeks) infants (Boyle et al., 2012; Engle & Kominiarek, 2008; Sengupta et al., 2013). Furthermore, taking the lifecourse under consideration, emerging data consistently suggest that late preterm infants born between $34^{0/7}$ and $36^{6/7}$ weeks' gestation experience greater morbidity than children born at term (Loftin et al., 2010). Births can be additionally can be classified into two groups: (1) spontaneous, or idiopathic, birth in the absence or presence of premature rupture of membranes, and (2) medically indicated, or iatrogenic, births (Menon, 2008). The majority of all preterm births occur spontaneously (Wang et al., 2018), though heterogeneity in birth phenotyping suggests multiple, complex etiologic pathways differentiate gestational age and type of delivery (Wadhwa, Entringer, Buss, & Lu, 2011). Indeed, the contribution of maternal stress may vary between birth phenotypes, necessitating that studies of birth outcomes include their accurate and comprehensive categorization. Maternal stress, in particular, has been hypothesized to have the greatest main effect on spontaneous births near term (Wadhwa et al., 2011), and prior studies have identified Black race and low socioeconomic status as "risk factors" for spontaneous preterm delivery (Menon, 2008). *Thus, studies of the association of gendered racial stress and birth outcomes need nuanced investigation of gestational age beyond a simple preterm or not preterm categorization.*

1.4. Limited empirical research studies extend past demonstration that gendered racism exists to investigate its consequences on pregnant Black women's health. In one of few studies on

gendered racism's pregnancy-related health effects, Rosenthal et al. observed three main findings: (1) Black and/or Latina (vs White) women reported greater stereotype-related gendered racism, (2) stereotype-related gendered racism was associated with greater pregnancy-specific stress, and (3) stereotype-related gendered racism mediated the relationship between race/ethnicity and pregnancy-specific stress (Rosenthal & Lobel, 2018). Stereotype threat, similar to hypervigilance or chronic worry, involves being concerned about the possibility of personally confirming negative group-based stereotypes (Rosenthal & Lobel, 2018). To measure stereotype-related gendered racism, the researchers developed a 15-item instrument based on historically-rooted stereotypes specific to Black and Latina women's sexuality and motherhood. Nine items asked about frequency of general experiences (e.g. 'How often do you feel that people make negative assumptions about how many sexual partners you have, based on being a woman of your racial/ethnic background?'), and 6 asked about pregnancy-related experiences (e.g. 'During your most recent [current] pregnancy, how worried were [are] you that people were [are] making assumptions about whether the father of the child would [will] play a role in raising the child, based on being a woman of your racial/ethnic background?'). However, this study did not examine the role of stereotype-related gendered racism in actual birth outcomes, just in pregnancy-related stress. In a qualitative study of pregnant Black women, Mehra et al. also observed that gendered racism in the form of stereotypes was a source of stress for Black women, who may develop relatively effective (active) or ineffective (passive) coping tools (Mehra et al., 2020). Gendered racism related to stereotype threat likely represents just one form of gendered racism. While stereotypes related to sexuality and motherhood are likely of particular salience to pregnant Black women in healthcare settings (Abdou & Fingerhut, 2014), a host of other stressors (e.g., financial pressure) plausibly contribute to gendered racial stress, and merit investigation.

Within-race investigation of the consequences of gendered racism for Black women has focused mostly on psychosocial outcomes among non-pregnant populations. Findings expectedly suggest that experiences of gendered racism are positively associated with poor mental health, including global psychological distress (J. A. Lewis et al., 2017; Thomas et al., 2008), depression (F. M. Jackson,

Rowley, & Curry Owens, 2012), and suicidal ideation (Perry, Pullen, & Oser, 2012), and that those relationships may be mediated and moderated by protective psychosocial resources. Thomas et al. found a significant positive correlation between gendered racism and global distress (Pearson's correlation coefficient=0.36) where gendered racism accounted for 13% of the variance in global distress (Thomas et al., 2008). Passive or avoidant coping, which itself is associated with increased distress, was found to mediate this relationship, further suggesting that some Black women may cope ineffectively when faced with gendered racial stress. Indeed, previous research has supported that Black women may be more likely to use passive versus active coping strategies, which may stem from perceived helplessness or lack of control over being discriminated against (Thomas et al., 2008; Utsey, Ponterotto, Reynolds, & Cancelli, 2000).

For pregnant Black women, the mental health consequences of gendered racism include hypervigilance to protect not only themselves but also their children from racial discrimination. Research suggests that during pregnancy Black women may anticipate their children later experiencing the racial inequalities already at work in their own lives (F. M. Jackson, James, & Owens, 2017; P. B. Jackson & Mustillo, 2001; Nuru-Jeter et al., 2009). Among Black pregnant women receiving prenatal WIC services, Jackson et al. (2017) found that Black women who agreed that "Black youth are at higher risk for having negative police experiences" was associated with an almost 12-fold odds of depressive symptoms ($p = .001$) when compared with Black women who disagreed with the statement. Results from a within-race study currently under review further suggest that pregnant Black women with higher Jackson, Hogue, Phillips Contextualized Stress Measure scores are more likely to report depressive symptomology ($r=0.49$, $p<.001$). *To date, the mechanisms through which gendered racial stress may causally effect outcomes in pregnancy are not well-established.*

1.5. For Black women, a social environment that interrupts the expectations they have for their lives and for their children's futures creates chronic stressors that may contribute to their

disproportionate vulnerability to depression and poor birth outcomes (Curry Owens & Jackson, 2015; Giscombé & Lobel, 2005; Hogue & Bremner, 2005; M. R. Kramer et al., 2011; M. R. Kramer & Hogue, 2009; Ncube et al., 2017). Ecosocial theory, which joins biological and social analyses of population health to explain social inequity (Krieger, 2001), supports that depression may be an embodied (Krieger, 2005) response to chronic stress due to accumulated experiences with gendered racism (Gravlee, 2009; F. M. Jackson et al., 2012). One physiological pathway through which gendered racial stress may get under Black women's skin is through premature aging, or weathering, of the stress response, which increases allostatic load (A. T. Geronimus, 1992; McEwen, 2000, 2012). The theory of allostatic load describes acute stress as an adaptive physiological response that helps an individual overcome or avoid stressors, in order to maintain balance or allostasis. When faced with a perceived stressor, the hypothalamic-pituitary-adrenal (HPA) axis activates the adrenal glands to release hormones called glucocorticoids, like cortisol. Short-term stress and healthful coping promote the eventual shut-off of HPA activation, and the body's return to balance (Cha & Masho, 2014). By contrast, chronic or cumulative stress is associated with long-term HPA activation and dysregulation of the adaptive system (S. Cohen et al., 2012; Frazier, Hogue, Bonney, Yount, & Pearce, 2018; A. T. Geronimus, 1992; Juster, McEwen, & Lupien, 2010). This dysregulation can lead to alteration of immune function and negative health outcomes, including depression (Iob, Kirschbaum, & Steptoe, 2019). The weathering hypothesis encompasses the known relationship between stressors and allostatic responses, and places allostatic load theory in the context of Black women's lived experience. It explains that the negative effects of racial inequity throughout one's life can result in metabolic, emotional, and/or behavioral patterns which increase the body's susceptibility to poor health (Frazier et al., 2018; Love, David, Rankin, & Collins Jr, 2010). The weathering hypothesis additionally suggests that, for socially-disadvantaged women, younger age is associated with lower accumulated health burden. Emerging evidence that older, rather than younger, age may function as a buffer to psychological outcomes related to racism exposure for non-pregnant Black persons adds complexity to the weathering hypothesis (Greer & Spalding, 2017). In the presence or absence of

weathering over the lifecourse, older adults' greater life experience may confer greater psychosocial resilience to experiences with discrimination (Benson, 2014). Resilience itself—or, the underlying contextual factors necessitating and fostering resilience—may have detrimental physical effects (per, e.g., the John Henryism hypothesis and the Superwoman Schema, described later.) Regardless of the specific effects of age, greater lifecourse stress and worse bodily wear and tear are both risk factors for depression (Leonard, 2000; McEwen, 2003). Weathering then implies that chronic exposure to gendered racial stress likely begins well before pregnancy, and that the embodiment of those pre-pregnancy experiences increases later-in-life risk among expectant Black mothers. The unique stressors Black mothers confront may place them in jeopardy for dysregulation across inter-related physiologic, psychosocial, and behavioral pathways (Curry Owens & Jackson, 2015; Holzman et al., 2009).

The associations between HPA axis hormones, inflammation and pregnancy complications has become a cornerstone for understanding the psychoneuroimmunology of pregnancy (L.M. Christian, 2012; Coussons-Read, 2012; Parker & Douglas, 2010; Ruiz, Fullerton, & Dudley, 2003). Pregnancy involves considerable physiological adaptation to support the growing fetus and mother. During normal gestation, the maternal HPA axis becomes progressively activated, and some literature suggests that depression, in turn, increases with or at least varies across gestation (Bennett, Einarson, Taddio, Koren, & Einarson, 2004; J. Evans, Heron, Francomb, Oke, & Golding, 2001), particularly in the first and third trimesters (Martini et al., 2013). Additionally, HPA axis hormones participate in a network with other hormones and inflammatory mediators to ultimately determine the timing of parturition. A disruption in this network is a proposed mechanism linking chronic stress to inflammation and preterm birth (PTB) (Cassidy-Bushrow, Peters, Johnson, & Templin, 2012; L. M. Christian, 2015; Hogue & Bremner, 2005; M. R. Kramer et al., 2011; Latendresse, 2009; Schetter, 2009; Wadhwa et al., 2001). Biomarker research on the effects of chronic stress highlights two important findings: (1) increased glucocorticoid levels are associated with minority status and low income (L. M. Christian, Glaser, Porter, & Iams, 2013; E. J. Corwin et al., 2013), and (2) without

well-functioning glucocorticoid feedback, a pregnant woman's ability to regulate inflammation is limited, which may contribute to adverse birth outcomes (E. J. Corwin et al., 2013). Notably, Corwin et al. found there were no difference in birth outcomes between minority and low income versus non-minority and high income women in this sample. Given the younger relative age of minority and low-income women, this finding may support the weathering hypothesis' claim that younger age is associated with lower accumulated burden, and thus better pregnancy outcomes. Additionally, given the good health of their sample, Corwin et al. note that indicators of dysregulated inflammation likely reflect weathering over the lifecourse, and are not specific to disease or pregnancy. While their multi-race sample does not allow for nuanced understanding of psychoneuroimmune functioning among just Black women, it suggests an interplay of race and other sociodemographic factors (e.g., income, age) may be at work. *Given that Black women experience a greater risk of chronic stress associated with racial discrimination, as compared to White counterparts, embodied experiences with gendered racism may underlie racial disparities in preterm birth. Variability among Black women is best revealed in within-race study.*

1.6. Variation in key sociodemographic factors and their earlier life correlates may contribute to within-race differences in gendered racial stress among adult Black women. According to the life course approach (Gee, Walsemann, & Brondolo, 2012), the specific timing of social exposures, and whether exposures reoccur over time, can influence the etiology and development of adult health conditions. Thus, contextualizing complex social determinants in an appropriate framework may enrich understanding of the potential pathways through which social exposures act.

Critical, or sensitive, period models suggest that some development periods are more sensitive to exposure than others, such that exposure in those periods has lasting, often lifelong, effects. In some instances, it is possible that harmful (or beneficial) exposures later in life can modify the health effects initiated by an exposure during an earlier critical period (Mishra, Cooper, & Kuh, 2010). Alternatively, gradual accumulation of risk models do not assume increased sensitivity to exposure in

any one stage, but account for how exposures at each life stage add to one another successively to increase risk over time. The effects of earlier exposures may manifest as observable health outcomes only later in life, after sufficient accumulation (Berkman, Kawachi, & Glymour, 2014). While some cumulative risk models consider the effects of uncorrelated social exposures, a more realistic hypothesis may be that social exposures correlate in patterned ways to create a chain of risk (Mishra et al., 2010). Chain of risk models are a type of cumulative risk model in which a harmful (or beneficial) exposure in one life stage leads to another harmful (or beneficial) exposure later, and so on (Berkman et al., 2014; World Health Organization, 2000). Each exposure in a chain of risk may not only increase the risk of subsequent exposure, but may also have an independent effect on later health outcomes (Mishra et al., 2010).

Pathways associating gendered racism with poor birth outcomes can be conceptualized using Clark et al.'s biopsychosocial model of racism: racism is a perceived stressor that, depending on coping responses, results in negative psychological and physiological stress responses, and subsequently poor health outcomes (Clark et al., 1999). In that model, sociodemographic factors may moderate causal pathways. Considering a biopsychosocial model with chains of risk, adult gendered racial stress, like stress due to racial discrimination more broadly, may be a mechanism through which maternal psychosocial and pregnancy outcomes are affected. Adversity in earlier life stages could create or maintain conditions for other social exposures (including sociodemographic factors, like education and relationship status) later in life (Nurius, Green, Logan-Greene, Longhi, & Song, 2016; Walsh, McCartney, Smith, & Armour, 2019), which in turn, could trigger the experience and embodiment of adult gendered racial stress.

Before we detail the key social & early life exposures for investigation, it is important to note that, in its engagement with theory (Clare R Evans, 2019), this work upholds the fundamental thesis of intersectionality: "that intersectionality does not situate the problems associated with particular identities within individuals or the identities themselves, but within the structural power hierarchies,

social processes, and social determinants that shape the social experiences of individuals with those intersectional identities" (Clare R Evans, Williams, Onnela, & Subramanian, 2018). The chain of risk model proposed sits squarely within ecosocial and eco-epidemiological frameworks, which center the causal influence of societal and structural factors on what we observe on the individual level (Clare R Evans et al., 2018; Krieger, 2001; Merlo, Mulinari, Wemrell, Subramanian, & Hedblad, 2017). As such, we reject the use or interpretation of social labels to homogenize or simplify a group we argue is complexly heterogeneous. Our provisional use of categorical labels (e.g., highly educated, low SES) connotes their purpose as proxies for unmeasured social processes that produce health outcomes for Black women (Bauer, 2014; Clare R Evans et al., 2018).

Returning to our rationale for the study of early adversity and social exposures: Early life adversity is embedded in multiple pathways to health. Adverse childhood experiences (ACEs), including significant dysfunction in the childhood home, are associated with lower socioeconomic and psychosocial resources in adulthood (Nurius et al., 2016), and with a wide range of psychosocial and reproductive outcomes, including preterm birth (Christiaens, Hegadoren, & Olson, 2015; Joshua P Mersky & ChienTi Plummer Lee, 2019; Von Cheong, Sinnott, Dahly, & Kearney, 2017). Education (a proxy for socioeconomic status) shapes employment and income, which help determine economic hardship. Education also influences a sense of personal control (in contrast to a sense of helplessness), social support, and access to skills and information that can mitigate the stresses of life (Ross & Wu, 1995). Similarly, relationship status may connote access to financial and social resources that can provide health advantages. Independently, both education and relationship status (married or not married, for instance) have been shown to influence health (Ross & Wu, 1995; Umberson & Montez, 2010), though control for those sociodemographic factors does not tend to mitigate racial disparities in birth outcomes. *While evidence supports that Black women generally experience higher rates of racial- and gender-based stress than White women, it remains unclear how childhood adversity and related sociodemographic differences in education and relationship status may influence within-race*

differences in gendered racial stress and pregnancy outcomes.

1.7. **A noteworthy caveat to research on gendered racism is that a lower gendered racial stress may not necessarily be positive.** Instead, it may indicate that someone has developed high-effort coping mechanisms to deal with experiences with and perceptions of gendered racism. Based on the John Henryism Hypothesis, high effort coping, defined as "sustained cognitive and emotional engagement," may come at a mental health cost for Black women who put forth greater and more sustained effort to cope with difficult psychosocial stressors, like gendered racial stress (James, 1994). Some studies show that John Henryism protects African Americans of higher (but not those of lower) socioeconomic status (SES) from the negative health effects of race-based stressors (Bonham, Sellers, & Neighbors, 2004; James, 1994). Other studies indicate the opposite: that Black individuals of higher SES take on a detrimental psychosocial stress burden in the process of achieving upward mobility, such that they fare worse than lower SES counterparts (Light et al., 1995; McKetney & Ragland, 1996). It is plausible then that low reported gendered racial stress coupled with high SES may actually indicate that a Black woman has had to perform higher-effort coping over her lifecourse, and not that she has truly experienced relatively fewer instances of or has been less affected by gendered racial stress (Assari, 2017; Darrell L Hudson, Neighbors, Geronimus, & Jackson, 2012). Indeed, a number of studies have found that Black Americans with greater socioeconomic resources report greater exposure to discrimination than those with lower levels of education and income (T. T. Lewis, Cogburn, & Williams, 2015), which may necessitate higher-effort coping to maintain one's occupation, income, or social standing. The implications of this association on mental health, however, are not clear. In a study on the relationship between discrimination on depression in Black men and women, Hudson et al. found that while racial discrimination and higher John Henryism scores were independently associated with increased odds of depression , the relationship between discrimination and depression was not moderated by the effects of John Henryism (Darrell L. Hudson, Neighbors, Geronimus, & Jackson, 2016). There was also no significant association between greater levels of SES (i.e., education and income) and increased John Henryism scores. By contrast,

in a sample of Black women, Bronder et al. found those with more John Henryism were less likely to have depressive symptomatology and that higher John Henryism and income, but not education, were significant predictors of lower depressive symptomology (Bronder, Speight, Witherspoon, & Thomas, 2014).

One pertinent concern with the John Henryism Hypothesis is that it is not exclusive to Black women. Related studies in the literature and described above generally include participants across race/ethnicity and gender. Conversely, while similar to John Henryism, the Superwoman Schema (SWS) is nominally, theoretically, and culturally exclusive to Black women. It describes Black women's contextually-derived obligations to exhibit strength, suppress emotions and vulnerability, succeed independently and against any odds, and help others (Cheryl L. Woods-Giscombé, 2010). SWS adds a theoretical rationale for the paradoxically harmful and helpful coping mechanisms developed by Black women in response to social conditions associated with living in a racist and sexist society (Lekan, 2009). Like John Henryism, SWS connotes a high effort coping style, which is associated with greater psychological and physiological deterioration over time. A qualitative study found gendered racial stress to be a driving force behind participants' internalization of the SWS, leading to silence (e.g., low support seeking) and neglect of their own self-care, including health care (H. J. Jones, Norwood, & Bankston, 2018). A recent empirical study supports a significant association between internalization of the SWS, depression, anxiety and loneliness (Liao, Wei, & Yin, 2020). *Considering the paradoxical complexities underlying John Henryism and the SWS, the unique ways in which reported gendered racial stress may vary by SES (due to associated coping demands and adaptive responses)—and can later impede or advance adverse mental health and pregnancy outcomes—deserves further study in Black women.*

1.8. While a positive association between education and health is well established in the population at large (Berkman et al., 2014), studies of Black maternal health paradoxically demonstrate that

pregnancy outcomes among Black women with the highest level of education are worse than pregnancy outcomes among non-Black women at the lowest level of education. The broader racial disparity in PTB and LBW persists even among women with higher social status. In a 1992 study of Black and White college graduates, McGrady et al. (1992) found that the relative risk of preterm delivery for Black women compared with White women was 1.67, and 2.48 for low birthweight. This finding has been replicated over the decades, suggesting less beneficial effects of upward mobility for Black but not White women (Colen, Geronimus, Bound, & James, 2006; David & Collins Jr, 1997; Rowley, 2001; Schoendorf, Hogue, Kleinman, & Rowley, 1992). The disparity in preterm birth may be brought on by variation in women's cumulative chain of risk, of which educational attainment (whether high or low) is a part.

One way education may influence the likelihood of preterm birth is through its potential associations with the experience and embodiment of discrimination. Black women with higher education report perceptions of racism more often than those of lower educations, perhaps due to detrimental conditions created and sustained by their social environment over the lifecourse (Vines et al., 2006). For instance, some well-educated Black women report experiencing physical and psychological demands associated with being a minority in the workplace (F. M. Jackson et al., 2001). Prior to joining the workforce, Black women pursuing higher education report a more hostile racial climate compared to White women, reporting more racial tension and mistrust (Leath & Chavous, 2018; Jioni A Lewis, Mendenhall, Harwood, & Browne Huntt, 2016). In a study of an older population (ages 52-99), reports of discrimination increased in a monotonic pattern among higher educated Black individuals, but not among White counterparts; and discrimination intensity also rose with educational attainment (Halanych et al., 2011). Notably perceived discrimination was less likely with increasing age, suggesting a buffering effect of age among the oldest women. This finding suggests that older age (even if still accompanied by actual experiences with discrimination) may improve coping resources, including psychosocial maturity (Benson, 2014). As aforementioned, these results suggest a resilience-related psychosocial mechanism, perhaps acting in partnership with

the biosocial weathering process.

Some Black women may worry more about discrimination (irrespective of frequency of actual experience). In a study where 37% of Black women and 6% of White women reported chronic worry about discrimination, Braveman et al. found that reports of chronic worry were highest among Black women at higher education and income levels (Braveman et al., 2017). Among White women, there was a negative association between education and worry. Additionally, chronic worry about racial discrimination was significantly associated with PTB only among Black women (PR 2.00, 95% CI: 1.33–3.01, suggesting the greater risk of of worry among Black women confers greater reproductive health risk (Braveman et al., 2017).

Some study findings do not confirm a positive association between education and discrimination among Black women, but suggest that increased levels of perceived discrimination persist among Black (vs White) women across education levels (Ro & Choi, 2009; Watson, Scarinci, Klesges, Slawson, & Beech, 2002). Ro et al. found that while both racial and gender discrimination were significantly associated with Black but not White race, only gender discrimination was significantly associated with educational attainment (Ro & Choi, 2009). Instead financial difficulty was associated with racial discrimination. It is plausible that the increased exposure to gendered racial stress associated with both high and low educational attainment weathers Black women's stress response system, increasing vulnerability to pregnancy complications. Biologic adaptive responses to this lifecourse stress may be joined by interrelated behavioral responses, including manners of coping with anticipated and actual instances of discrimination (Berkman et al., 2014; Ro & Choi, 2009). *Considering that Black women's educational attainment may foster variation in experience of and response to gendered racial stress, exploring within-race differences may further explain the racial disparities in PTB observed in mixed-race study.*

1.9. Relationship status may serve as an indicator for a variety of social factors that could influence stress measured in the perinatal period, including psychosocial support (e.g. paternal or

partner support), and access to financial and healthcare resources (Chomitz, Cheung, & Lieberman, 1995; El-Sayed, Tracy, & Galea, 2012). Additionally, racial differences in marital patterns (i.e., that Black women marry less frequently, and when they do, experience more marital strain and dissolution than White women) suggest that marital status may contribute to racial differences in health (Keith & Brown, 2018; Thomas Tobin, Robinson, & Stanifer, 2019). Findings that unmarried or non-cohabitating mothers have higher risk of preterm delivery than married or cohabitating mothers implicate relationship status as a determinant of PTB, potentially operating through a stress mechanism (Glynn, Schetter, Hobel, & Sandman, 2008; Merklinger-Gruchala & Kapiszewska, 2019; Rich-Edwards et al., 2006; Schetter & Tanner, 2012; Shah & Births, 2010). Furthermore, these studies reveal a scientific shift over the decades toward investigating implications of relationship types beyond married or not married, to mirror contemporary coupling patterns (Smock, 2000). Some of this literature suggests that married pregnant women report greater levels of social and emotional support than cohabiting pregnant women, but that both groups report greater support than single pregnant women (Smock, 2000). Thus, it is plausible that the presence or absence of social resources associated with relationship status alters the experience and embodiment of chronic stress, including gendered racial stress. In a study of age-diverse Black and White women, marital status was not a significant predictor of racial differences in allostatic load (a measure of chronic stress), although Black women's allostatic load scores were 45% higher than White women's (Thomas Tobin et al., 2019). Notably, this racial gap in allostatic load existed only among current and formally married women; never married Black and White women had similar allostatic load scores. This pattern paradoxically suggests that marriage (like higher education) may confer greater health benefit for White but not Black women (Keith & Brown, 2018). However, given wide age range (from 18-69 years) in that study, and the paucity of similar analyses, additional work is needed to clarify within-race risk pathways for Black women of reproductive age.

Relatively few studies consider the contextual role of race (and therefore racial stress) in the association between marital status and birth outcomes. Among extant studies, results are mixed. Bird

et al. found that non-Hispanic Black women had similar risks of delivering a low birthweight infant, whether they were married, cohabitating, or other (Bird, Chandra, Bennett, & Harvey, 2000). In another study of infant low birthweight among Black and White mothers, unmarried Black adult mothers had the highest risk of delivering a low birthweight infant (RR=2.49) as compared to married White mothers, followed by married Black adults (1.93), unmarried Black teenagers (1.90), and married Black teenagers (1.67) (Sung et al., 1993). This suggests an interactive effect of race with marital status, as well as modification by maternal age to create heterogeneity within race. A study of preterm birth found that Black women who were unmarried or had fewer than 12 years education were more likely to deliver preterm as compared to women who were white/other, married, or had 12 or more years of education, respectively (Margerison-Zilko, Strutz, Li, & Holzman, 2017). Considering a woman's vulnerability to stress and stress-related birth outcomes can be enhanced, even paradoxically, by her relationship status, it follows that relationship status may be associated with Black women's experiences and response to gendered racial stress. *To date, no studies explicitly explore this pathway between relationship status and contextualized stress among Black women. Such investigation could further clarify how social exposures shape Black women's chains of risk toward adverse birth outcomes.*

1.10. C-reactive protein (CRP), a marker of inflammation linked to discrimination, adult depression, preterm birth, and a host of other health conditions, may be heightened among pregnant Black women exposed to gendered racial stress (Danese, Pariante, Caspi, Taylor, & Poulton, 2007; Khera et al., 2005; Ridker, 2003; Sharpley, Bitsika, McMillan, Jesulola, & Agnew, 2019; Szalai, Agrawal, Greenhough, & Volanakis, 1997; Visser, Bouter, McQuillan, Wener, & Harris, 1999). CRP is an inflammatory cytokine produced in response to stress, and subclinical elevations of greater than 3.0 mg/L have been associated with cardiovascular disease, obesity, and race and ethnicity in non-pregnant populations. In a large (n=2,749) population-based study of non-pregnant Black and White participants, Black participants had significantly higher CRP as compared

to White participants (median: 3.0mg/L vs. 2.3 mg/L; $p < 0.001$) (Khera et al., 2005). Furthermore, the highest percentage of those with CRP >3mg/L was found among Black women (vs White women, Black men, and White men) (Khera et al., 2005). In an even larger population-based longitudinal study of over 26,000 participants, 49% of Black participants (compared to 35% of White participants) had CRP >3mg/L (Christina R Evans et al., 2019). Embodiment of experiences with racial discrimination may explain this association, as research suggests that discrimination (for example, based on race and/or socioeconomic status) is also associated with higher levels of CRP (Beatty Moody, Brown, Matthews, & Bromberger, 2014; Cunningham et al., 2012; T. T. Lewis & Van Dyke, 2018; Van Dyke et al., 2017). These results support that there are racial differences in CRP among non-pregnant Black individuals, whose CRP profile may continue to differ from that of White counterparts during the physiologically stressful pregnancy period (Picklesimer et al., 2008). Black women have also been shown to have higher CRP levels during pregnancy, compared to White women (Picklesimer et al., 2008). Picklesimer et al. found that median CRP was 2.6mg/L for pregnant White women and 7.7mg/L for pregnant Black women, well above the 3.0 mg/L threshold. For Black versus White women, the odds of having CRP above 15.7 mg/L (the 75th percentile in that study) were 2.1 (95%CI: 1.3-3.3). Similar odds were reported for low income and unmarried women across race. In a socioeconomically diverse sample of pregnant Black women, similar to that available for the proposed dissertation, Clarke et al. found median first trimester CRP to be 5.3 mg/L (IQR: 2.0-13.6) (Clarke et al., 2020).

Levels of maternal CRP throughout pregnancy have also been positively associated with or even predictive of preterm birth in a number of studies (Catov, Bodnar, Ness, Barron, & Roberts, 2007; Han, Ha, Park, Kim, & Lee, 2011; Pitiphat et al., 2005; Shahshahan & Rasouli, 2014; Vogel et al., 2005), some of which suggest a dose–response relationship between CRP levels and preterm delivery risk (Catov et al., 2007; Pitiphat et al., 2005). These findings suggest CRP may be implicated in the racial disparity in preterm birth among Black women. Still, other research suggests an unclear or weak link between CRP and preterm delivery (Wei, Fraser, & Luo, 2010), especially among women

with higher BMIs (Bullen et al., 2013). In BMI-stratified models, Bullen et al. observed a positive association between CRP and spontaneous PTB among women with pre-pregnancy body mass index (BMI) >25, but not among women with lower BMIs. In our study sample, the mean first-trimester BMI is 28, allowing us the unique opportunity to help clarify the role of CRP in birth outcomes among Black women who may be overweight or obese. *More broadly, the proposed dissertation includes exploration of the role of Black maternal inflammation in relation to experiences with race- and gender- discrimination, which is not fully understood.*

Chapter 2: Overview of Specific Aims and Data Source

Overview

This project proposes an innovative approach to examining life course correlates and consequences of gendered racial stress, and their implications on Black women's mental and perinatal health. It builds on intersectional theory and the recent development of an instrument for quantifying gendered racial stress, and moves the field forward by testing contextual and individual factors that may affect a within-race group of pregnant Black women's exposure and response to gendered racism, by:

- Using an intersectional measure created to assess racism and sexism. Ongoing scientific discourse suggests that limitations may occur when using single identity factor models to understand multifactorial identity issues (Thomas et al., 2011). Still, much of the existing literature leaves out the role of intersections of racism and sexism, or gendered racism (J. A. Lewis et al., 2017). In the proposed study, we will apply and build on intersectionality theory by using a gendered racism scale designed specifically to consider discrimination due to race *and* gender (Curry Owens & Jackson, 2015; F. M. Jackson et al., 2005; F. M. Jackson et al., 2017; F. M. Jackson et al., 2001), without attempt to disentangle them.
- Examining the effect of gendered racism on Black women's birth outcomes. While empirical and theoretical research on the influence of racism on Black women's reproductive health has grown in recent decades, limited empirical research studies extend past demonstration of the existence of *gendered* racism in their lives to investigate its consequences in pregnancy. As Black women and their infants are known to be at disproportionate risk during and after pregnancy, it is of paramount importance that our understanding of processes involved be as complete as possible.
- Focusing on sociodemographic factors contributing to within-race variation in susceptibility to experiences with gendered racism and its consequences. As the paradoxical link between high SES and health for Black but not White individuals suggests, a given social exposure may correspond with a wide diversity of lived experiences, themselves with distinct health consequences (Berkman et al., 2014). Further, though most Black women may experience

gendered racism, homogeneity among its causes and consequences is unlikely, as different mechanisms of achieving the same exposure may also have varying health consequences. Using a study sample made up of sociodemographically-diverse pregnant Black women, we will:

- Analyze whether depression acts as a mediator of the relationship between maternal gendered racial stress and infant gestational age. Mediators reflect the mechanisms by which an exposure may influence an outcome. No studies of gendered racism have specifically tested mediators of its relationship with birth outcomes, though existing studies demonstrate a positive association of gendered racial stress with depression, particularly among college-educated Black women (Curry Owens & Jackson, 2015; F. M. Jackson et al., 2012). Emerging theory suggests depression is one pathway through which experiences of discrimination may result in greater risk of adverse birth outcomes (Christine Dunkel Schetter, 2011; Earnshaw et al., 2013). Increasing evidence demonstrates that experiences of racial discrimination, to which Black women are especially vulnerable, are linked to depression and depressive symptoms (Goosby et al., 2018). Depressive symptoms themselves are associated with greater risk of preterm birth (C. Dunkel Schetter & Tanner, 2012). Thus, this work will help clarify whether depression may be an important mechanism by which exposure to gendered racial stress influences gestational age.
- Consider age as a moderator of the relationship between maternal gendered racial stress and infant gestational age. Moderators may alter the strength of the relationship between racism and health for particular groups (Clark et al., 1999; Y. Paradies et al., 2015). To date, empirical data on the influence of moderators between racism and health outcomes is mixed (Y. Paradies et al., 2015), suggesting a need for additional study. Empirical data on the influence of psychosocial moderators of the relationship between gendered racism and birth outcomes among Black women does not yet exist in the literature. Age, in particular, is implicated in the weathering of Black women's stress response system (Gee et al., 2012; Greer & Spalding, 2017). However, it is most often used as a control variable, precluding understanding of the ways in which age may explain the relationship between racism and health. Additionally, recent evidence raises questions about

whether youth or older age is protective for Black women's health (Earnshaw et al., 2013; Greer & Spalding, 2017; Halanych et al., 2011). Given the range of ages used across studies included here, spanning 14 to 99, exploration of appropriate cutpoints at which age may be more or less protective for Black women is needed. Finally, age is also associated with sociodemographic factors like education and marital status, which themselves influence experiences of discrimination. Thus, this work may fill in gaps on whether the effects of gendered racial stress on birth outcomes vary over the lifecourse.

- Investigate the relationship between gendered racism and a known biomarker of stress. Biosocial processes linking discrimination to health are under study among a vast array of ethnic/racial groups, including Black Americans. However, limited research considers how gendered racial stress experienced by Black women is associated with C-reactive protein, a biomarker whose importance in physiological and psychological wellbeing is established.

Overall, the proposed research will add much-needed context and detail to the growing bodies of literature on the biopsychosocial causes and effects of racial stress. Through use of a multifactorial framework that centers the context created by intersecting social identities, this work holds promise to inform efforts to reduce the disproportionate burden of adverse mental and reproductive health on Black women and infants.

Specific Aims

Aim 1. To investigate the associations of early life experiences (via Adverse Childhood Experiences, ACEs) with Black women's reported gendered racial stress in pregnancy.

Exploratory Aim 1a. To investigate whether the association of ACEs and gendered racial stress is modified by response to unfair treatment.

Hypothesis: Greater exposure to early life stressors (i.e., increased exposure to ACEs) is associated with higher gendered racial stress scores among pregnant Black adults after control for childhood socioeconomic status. Interaction with response to unfair treatment may demonstrate a stronger association among women more likely to respond to unfair treatment passively, versus actively.

Aim 2. To estimate the effect of maternal gendered racial stress on spontaneous early term and spontaneous preterm birth, as compared to full term birth.

Exploratory Aim 2a. To estimate mediation of the relationships in Aim 2 by prenatal depressive symptomology.

Exploratory Aim 2b. To estimate effect measure modification of the relationships in Aim 2 by maternal age.

Hypotheses: Pregnant Black women with higher gendered racial stress scores will be more likely to give birth at earlier gestational ages than pregnant Black women with lower gendered racial stress scores. Depression significantly mediates this relationship, given an established relationship of gendered racial stress burden and depression. Consistent with the weathering hypothesis, older pregnant women with higher gendered racism scores will be most likely to give birth early.

Aim 3. To evaluate the association between gendered racial stress and C-reactive protein, and determine whether this association is significantly different among women with higher (≥25 mg/k^2) versus lower body mass index.

Hypothesis: C-reactive protein and gendered racial stress are positively associated, particularly among women with high body mass index.

Major components of each aim are represented in the conceptual model shown in Figure 2.1.

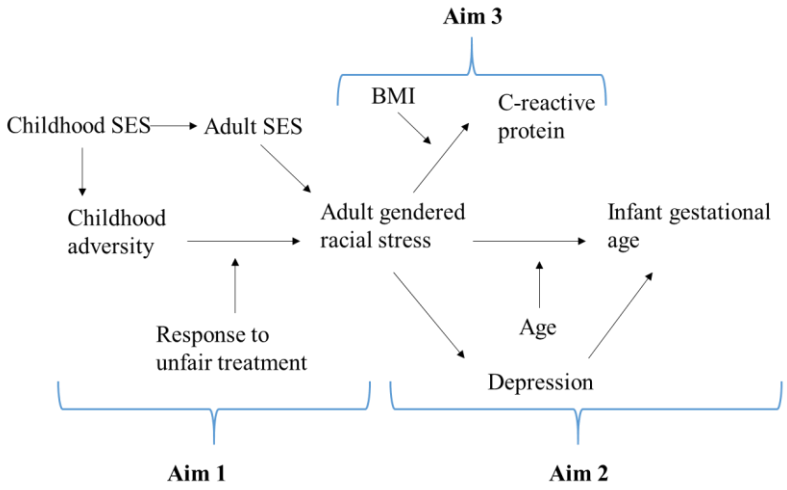


Figure 2.1. Conceptual model for the examination correlates and consequences of gendered racial stress among pregnant Black women.

Study Population

The proposed research uses data from the prospective Emory University Microbiome in Pregnancy Cohort Study (n=485), which aims to provide a biopsychosocial understanding of within-race risk for adverse birth outcomes among a socioeconomically diverse sample of Black women (Elizabeth J Corwin et al., 2017). To be eligible, women were between 18-40 years of age, self-identified as African-American (i.e., US-born and of African-American or Black race), carrying a singleton pregnancy between 8-14 weeks' gestation at the time of enrollment, and had no chronic medical conditions. Participants were receiving prenatal care at either Grady Memorial Hospital or Emory University Hospital Midtown—two

Atlanta-areas facilities (public and private, respectively) that see approximately 10% of Georgia's singleton live births to Black women. In the first study visit at 8-14 weeks' gestation, sociodemographic, psychosocial, and other health information was collected via self-report (Table 2.1); we hypothesize these factors are implicated in a chain of risk culminating in birth outcomes. This visit also included collection of biospecimens, including blood for biomarker analysis. Additional clinical information, including birth modality and birth outcome, was gathered later via maternal medical record abstraction.

Table 2.1. Sociodemographic and psychosocial self-report data collected at first study visit, Emory University Microbiome in Pregnancy Cohort Study

Measure	Information collected
Sociodemographic Survey	Age, education, relationship status, insurance status
Health Survey	Diagnoses, medications, substance use in last month
Measure of Gendered Racial Stress	
Jackson Hogue Phillips Contextualized Stress Measure	Experiences of gendered racism
Additional Psychosocial Measures	
Adverse Childhood Experiences	Dysfunction in childhood home
Edinburgh Depression Scale	Symptoms of depression in last 7 days
Perceived Stress Scale	Stress-related feelings and thoughts in last month

Key Variables

Gendered racial stress: To operationalize gendered racial stress, we will use continuous scores on the 39-item Jackson Hogue Philips Contextualized Stress Measure[4] (JHP-RC©; Table 2.2), a measure of gendered racism designed for Black women's experience (Jackson et al., 2005; Jackson, Hogue, & Phillips, 2014). The JHP-RC© captures racism and sexism together, allowing avoidance of a common limitation in studies that have used separate measures of racism and sexism to understand the constructs' interactive effects (Thomas et al., 2008).

The JHP-RC© uses a Likert scale to assess levels of chronic racial and gendered stress among Black women across five contextual domains: Burden, Coping, Personal History, Racism, and Work. The

[4] JHP-RC© used with permission of the authors.

"burden" domain includes 10 items that capture: the imposed and embraced nurturing and caretaker role associated with gender identity, as well as distress resulting from an imbalance in material and personal resources needed to meet demand (e.g., "I am obligated to provide for the financial well-being of family members who do not live with me"). The 15 "coping" items represent instrumental and expressive support from family and friends, spirituality/religiosity, and racial and gender identification as sources of social support (e.g., "If I have a problem I can get help from people from my religious institution [church, mosque, temple, etc.]"). The five items that make up the "personal history" contextual domain include experiences with mental and physical abuse as individual stressors (e.g., "I have experienced physical abuse in my relationship[s] with men"). The "racism" contextual domain is comprised of five items that capture racist encounters and anticipations associated with nurturing/caretaker roles, racial affiliation, and stereotypes (e.g., "White women have a lot more opportunity than I do"). The "work" contextual domain includes five items that reflect the experiences and perceptions of gender and racial oppression in the work environment, and capture interracial and inter-gender stressors encountered in the workplace (e.g., "Because I am a woman my employer is not usually open to suggestions from me").

Table 2.2. 39-item Jackson Hogue Philips Contextualized Stress Measure (JHP-RC©)[a]

I will read a statement. Please tell me if you agree or disagree which each statement using the following response: strongly agree, agree, unsure, disagree, or strongly disagree.	
Contextual domain: Burden	
1	I am taking care of everyone else, but no one is taking care of me.
2	Everyone expects me to be strong for them.
3	I have a lot of financial pressures.
4	I have the major responsibility for the financial support of my household.
5	By now I should be doing better financially.
6	I feel that I am alone.
7	I have far too much to do.
8	I am obligated to provide for the financial support of my household.
9	I get no time for myself.

10	I am worried that I am going to fail.
Contextual domain: Coping	
11	As an African American woman, I can withstand great pressure.
12	My participation in a religious institution gives me the motivation to perform at my job.
13	I have friends who sense when I have a problem and will help.
14	The African American community has really taken care of me.
15	My religion or spirituality helps me to love myself.
16	I have one or more friends I feel close to.
17	My African American heritage gives me the motivation to perform at my job.
18	I gain strength and/or comfort from a spiritual source through prayer, meditation or reflection.
19	Women from my family and community provide a guide for the way I function in my home and community.
20	If I have problems I can get help from people at my religious institution.
21	My family members offer me emotional support.
22	I feel rewarded when I give back to the community.
23	When other African Americans are successful, I feel it pulls me up, too.
24	Women from my family and community motivate me to perform well at my job.
Contextual domain: Racism	
25	Individuals assume that I am incapable of performing a job because I am African American.
26	Racism is a problem in my life.
27	I have to work harder than white women to earn equal recognition.
28	The African American youth in my community are more likely than other youth to have a negative experience with law enforcement.
29	White women have a lot more opportunities than I do.
Contextual domain: Personal history	
30	I come from a family with a history of alcohol abuse.
31	I come from a family with a history of physical abuse.
32	I have experienced physical abuse in my relationship with men.

33	I come from a family with a history of drug abuse.
34	I have experienced mental abuse in my relationships with men/husband/partner(s).
Contextual domain: Work	
35	I can't trust African American men in the workplace to be supportive of me.
36	I can't trust African American women in the workplace to be supportive of me.
37	I can't trust whites in the workplace to be supportive of me.
38	Because I am a woman, my employer is not usually open to suggestions from me.
39	I am not taken seriously in the workplace.

[a]We compute the total gendered racial stress score from the sum of the positive responses indicating the presence of stressors and stress states, and the absence of stress mediators (F. M. Jackson et al., 2012). Step-wise, we: (1) assign numerical values 1-5 to item responses (with 1 representing "Strongly Disagree" and 5 representing "Strongly Agree," (2) reverse code items 1-11 and 25-29, so that a participant response of 5 is recoded as 1, 4 is recoded as 2, and so on, and (3) sum items within each domain and overall.

Gestational age: Gestational age in weeks was ascertained from maternal medical record abstraction. We will exclude any births before 20 weeks' gestation (n=42). Births beyond 20 weeks' gestation (N=443) will be analyzed both continuously and categorically, as we believe this approach will allow us to investigate whether gendered racial stress might shift the population's gestational age curve left or increase susceptibility only in particular subgroups. We will use 37 weeks' gestation to dichotomize preterm (<37 weeks) or full term births (≥37 weeks). Additionally, to investigate a gradient of risk beyond the 37-week demarcation, we will further categorize births to include an early term birth category, so that groupings represent the following: preterm (>20 weeks-<37 weeks), early term (37 weeks-<39 weeks), and full term (≥39 weeks). Each birth outcome group is further partitioned as shown in Table 2.3.

Table 2.3. Birth outcome categories

All preterm births (n=66)
Spontaneous preterm births (n=49)
Induced preterm births
All early term births (n=122)
Spontaneous early term births (n=93)
Induced early term births
All full term births (n=255)

Healthy[a] full term births (n=190)

[a]Healthy full term births exclude full term births to mothers with pre- and perinatal cardiometabolic complications, including gestational diabetes, preeclampsia or gestational hypertension, and premature rupture of membranes.

Social exposures:

Education. Education was measured with four levels: (1) less than high school, (2) completed high school (or equivalency), (3) some college, and (4) college degree or more. In some analyses, given observed patterning of gendered racial stress, we dichotomize educational attainment as college graduate, or some college or less.

Relationship Status. Relationship status was measured with two broad response categories: (1) Married or cohabiting, and (2) not married or cohabiting.

Age. Self-reported age ranges continuously from 18 to 40 years. To distinguish socially meaningful age categories while still maintaining a roughly equal distribution of the sample across age categories, we partition age into tertiles in some interaction analyses.

Adverse Childhood Experiences (ACEs): The ACEs questionnaire ascertains experiences related to dysfunction in the childhood home (i.e. in the first 18 years of life) by asking about 10 experiences (Felitti et al., 1998). ACEs is used extensively among women of reproductive age across race/ethnicity and socioeconomic status groups. Elevated exposure to ACEs has demonstrated a graded, or dose-response, relationship to myriad health conditions.

Depression: The Edinburgh Depression Scale (EDS), a 10-item self-report questionnaire ascertaining symptoms of depression in the last 7 days, has high sensitivity and specificity for the detection of prenatal major depressive disorder (Grobman et al., 2016; Kozinszky & Dudas, 2015). The scale has been used extensively in studies of pregnant women, and psychometric properties of the EDS for pregnant Black populations have been previously reported (Tandon, Cluxton-Keller, Leis, Le, & Perry, 2012). In this study, a score greater than or equal to 10 will indicate the presence of depressive symptomology [31, 32].

C-reactive protein: After blood collection at the first study visit, C-reactive protein (CRP) was measured using a solid-phase sandwich enzyme-linked immunosorbent assay (ELISA) kit (R&D Systems, Minneapolis, MN) with sensitivity to 0.022 ng/mL. The range of this assay is 0.8-5.0 ng/mL, with inter- and intra-assay variability of approximately 4% and 6%, respectively.

Covariates: We believe selected covariates precede exposures of interest, and indicate risk for outcomes of interest. All individual-level covariates were collected in the Emory University Microbiome in Pregnancy Cohort Study, and will be selected for inclusion where appropriate in fully adjusted models corresponding with each aim. Sociodemographic covariates may include: age, education, relationship status, and insurance status. Psychosocial health indicators may include: adverse childhood events, childhood adversity, prenatal depression, prenatal perceived stress, and stressful life events. Reproductive health indicators may include: body mass index (BMI), previous preterm birth, and parity.

Chapter 3: The association of early life adversity with gendered racial stress among pregnant Black women

Introduction

Adverse childhood experiences (ACEs) that occur prior to the age of 18 have been associated with myriad adverse biological and psychosocial health outcomes later in life, and, thus, are rising rapidly on policy agendas (Campbell, Walker, & Egede, 2016; Felitti et al., 1998; Hamby, Elm, Howell, & Merrick, 2021; Holman et al., 2016; Von Cheong et al., 2017). Exposure to ACEs is also a risk factor for adverse birth outcomes, including earlier gestational age, low birth weight, although the mechanisms by which maternal ACEs may influence birth outcomes have not been fully explored in the literature (Joshua P. Mersky & ChienTi Plummer Lee, 2019; Smith, Gotman, & Yonkers, 2016). Numerous studies have confirmed a graded, or dose-dependent, relationship of ACEs with adult health, suggesting the impact of ACEs over one's life is not only strong, but cumulative (Felitti et al., 1998; Merrick et al., 2017). Disparities in ACEs illustrate their social patterning: ACEs are more prevalent among Black versus non-Black children in the U.S. (V. Sacks, Murphey, & Moore, 2014), are linked to indicators of lower socioeconomic status (Walsh et al., 2019), and are remarkably common among pregnant women experiencing perinatal mood and anxiety disorders (H. G. Kim, Kuendig, Prasad, & Sexter, 2020). This patterning reflects chains of risk in which a primary harmful exposure leads to later harmful exposures, thereby perpetuating the impact of earlier adversity, and adding social, behavioral, and psychosocial pathways to biological mechanisms of stress reactivity (Berkman et al., 2014; Nurius et al., 2016; Umberson, Williams, Thomas, Liu, & Thomeer, 2014).

Researchers who adopt the lifecourse perspective as an explanation for racial disparities in birth outcomes emphasize the importance of what individuals experience at different times of their lives and how those experiences combine to impact health outcomes (Braveman & Barclay, 2009; Lu & Halfon, 2003). One physiological pathway through which ACEs may be embodied over time is through premature aging, or weathering, of the stress response, which increases allostatic load (A. T. Geronimus, 1992; McEwen, 2000, 2012). The theory of allostatic load describes acute stress as an adaptive physiological response that helps an individual overcome or avoid stressors, in order to maintain balance or allostasis.

When faced with a perceived stressor, the hypothalamic-pituitary-adrenal (HPA) axis activates the adrenal glands to release hormones called glucocorticoids, like cortisol. Short-term stress and healthful coping promote the eventual shut-off of HPA activation, and the body's return to balance (Cha & Masho, 2014). By contrast, the chronic or cumulative stress is associated with long-term HPA activation and dysregulation of the adaptive system (S. Cohen et al., 2012; Juster et al., 2010). This dysregulation can lead to toxic stress, and long-term alteration of immune function, which can hasten mental and physical illness (Iob et al., 2019; Wesarg, Van Den Akker, Oei, Hoeve, & Wiers, 2020) . The weathering hypothesis encompasses the known relationship between toxic stress and a maladaptive physiological response, and places allostatic load theory in the context of Black women's lived experience. It explains that the negative effects of racial inequity—rooted in historical, social, economic, and medical disadvantage—throughout Black women's lives can result in metabolic, emotional, and/or behavioral patterns which increase the body's susceptibility to poor health, even at younger ages (Frazier et al., 2018; A. T. Geronimus, 1992; Love et al., 2010). Thus, a possible explanation for racial variation in patterns of birth outcomes is the long-term biopsychosocial cost of childhood stress and adversity.

Preliminary evidence suggests that negative effects of ACEs not only endure over the exposed person's lifetime, but can be transmitted intergenerationally (Buss et al., 2017). Toxic stress experienced by women prior to pregnancy can negatively affect fetal development, which can contribute poor health for the infant before, at, and well after birth (Madigan, Wade, Plamondon, Maguire, & Jenkins, 2017; Scorza et al., 2020). For instance, infants born to women who experienced at least four childhood adversities were two to five times as likely to have poor physical and emotional health outcomes by 18 months of age when compared with . . . (Madigan et al., 2017). ACEs may thus link the maternal chain of risk to her child's, to place particular infants at a relative disadvantage right from the start. As Black women and their infants are known to be at disproportionate health risks during and after pregnancy, it is imperative to understand the role of ACEs in proliferating types of stress specific to American Black women's sociohistorical context.

Researchers have found that racism is a stressor for pregnant Black women (Clark et al., 1999; Tyan Parker Dominguez et al., 2008; Stancil, Hertz-Picciotto, Schramm, & Watt-Morse, 2000), and that racial stress is linked with adverse psychosocial and birth outcomes (T. T. Lewis et al., 2015; Mays, Cochran, & Barnes, 2007; Murrell, 1996; Williams & Mohammed, 2009). However, for Black women, additional exposure to stress over the lifecourse may not only be due to race, but both race and gender (Essed, 1991; J. A. Lewis et al., 2017; Thomas et al., 2008). This type of stress—contextualized in Black women's intersecting social identities—is known as gendered racial stress, and is brought on by gendered racism, a hybrid form of oppression (Essed, 1991; F. M. Jackson et al., 2001; T. T. Lewis & Van Dyke, 2018). Gendered racism suggests that, by virtue of being both Black and women, Black women uniquely experience psychosocial stressors that go beyond reports of perceived instances of racism (F. M. Jackson et al., 2012), and which may operate independently of other known sources of stress (L. Rosenthal & M. Lobel, 2011). Research supports that Black women appraise experiences of gendered racism as stressful due to the simultaneous experience of racism and sexism, and not just due to one or the other (Thomas et al., 2008). Further, experiences with gendered racism related to Black women's sexuality and gendered role as mothers are associated with greater pregnancy-specific stress (Rosenthal & Lobel, 2018).

While ACEs are implicated in stress pathways affecting maternal and infant health, it is unknown whether ACEs confer risk for gendered racial stress among pregnant Black women, who may then go on to experience adverse pregnancy outcomes. The present study examines the association between maternal ACEs and the intersectional construct of gendered racial stress, and explores moderating pathways, among socioeconomically diverse Black women. We hypothesize that our findings will support a graded relationship of ACEs with gendered racial stress, which may be modified by adaptive coping responses. While our investigation focuses on the individual level, we emphatically acknowledge that the enduring historical underpinnings of racism negatively influence health outcomes of Black women in America (Michael R Kramer et al., 2017; Cynthia Prather et al., 2018).

Methods

Participants

This cross-sectional study uses first trimester prenatal data from the participants enrolled in the prospective Biobehavioral Determinants of the Microbiome and Preterm Birth in Black Women study (n=485), which aims to provide a biopsychosocial understanding of within-race risk for adverse birth outcomes among a socioeconomically diverse sample of Black women (Elizabeth J Corwin et al., 2017). At enrollment, participants were receiving prenatal care at either Grady Memorial Hospital or Emory University Hospital Midtown—two Atlanta-area facilities (public and private, respectively) that see approximately 10% of Georgia's singleton live births to Black women. Eligible women were between 18-40 years of age, identified as African-American (i.e., US-born and of African-American or Black race), were carrying a singleton pregnancy between 8-14 weeks' gestation, and had no reported chronic medical conditions or conditions requiring long-term prescription management. During the baseline study visit, sociodemographic, psychosocial, and other health information was collected via self-report. The Emory University Institutional Review Board granted ethical approval for all data collection, and all participants provided written informed consent. Eighty women were excluded from these analyses due to missing data on gendered racial stress (n=36) or due to ineligible birth outcomes (n=44; e.g. gestational age less than 20 weeks), leaving an analytic sample of 405. While not explicitly examined here, birth outcomes are relevant to the broader context in which our specific research question is situated (see directed acyclic graph in Figure 2.1)

Measures

Gendered racial stress

Our dependent variable was self-reported gendered racial stress as measured in early pregnancy. To operationalize gendered racial stress, we use continuous scores from the 39-item Jackson Hogue Phillips Contextualized Stress Measure (JHP), which we call the JHP-Reduced Common (JHP-RC©; see Table

2.2). This reduced scale includes items from the original 68-item JHP (F. M. Jackson et al., 2005; F. M. Jackson, Hogue, & Phillips, 2014) that were found to be most commonly experienced by Black women who worked at one of two public hospitals and were enrolled in a federally funded worksite fitness study (Freeman, 2009). The JHP-RC© assesses specific exposure to chronic racial and gendered stress among Black women, and consists of four subscales measuring stressors (burden, personal history, racism, and work) and one measuring stress mediators (coping), with 5-level Likert scoring from greatest agreement to greatest disagreement. The burden subscale includes 10 items that measure gendered role strain, and distress due to inadequate resources to meet demands. It includes 3 of the 4 items from the original JHP© Scale referred to as "stress states" (F. M. Jackson et al., 2005; F. M. Jackson et al., 2014). The personal history subscale includes 5 items that assess experiences with emotional or physical abuse, and family or partner substance abuse. The 5 racism items capture racial stereotypes, White privilege, and the perceived impact of racism on Black children's lives. The 5-item work subscale assesses racism and sexism in the workplace. The 14 coping items measure coping resources (including support from and belonging to one's community) and capacity for coping based on race and gender. To calculate total and subscale JHP-RC© scores, we coded items 1-5 and reverse coded items, as necessary, so higher values for each item indicated greater gendered racial stress. In our sample, total JHP-RC© score ranges from 47 to 159. Ranges for the subscales are: burden=10-50, coping=16-66, personal history=3-25, racism=4-25, and work=2-25. Cronbach's alpha coefficient for the full JHP-RC© was .81 in the present study. The subscales had the following alpha coefficients: burden=.77, coping=.83, personal history=.78, racism=.81, and work=.77. These values indicate acceptable to high internal reliability and consistency. Given the theoretical overlap and relatively strong correlation (r=.49) between the personal history subscale and items in the Adverse Childhood Experiences Questionnaire (described below), we created a modified 34-item JHP-RC (named the Modified JHP-RC henceforth) for this study that excludes the 5 personal history items for clearer interpretation of associations between gendered racial stress and adversity in childhood.

Adverse Childhood Experiences (ACEs)

ACEs data were collected via retrospective self-report, as previously described (Elizabeth J Corwin et al., 2017). The ACE questionnaire ascertains experiences related to dysfunction in the childhood home (i.e. in the first 18 years of life) by asking the respondents experience of each of the following 10 experiences:

1. Living with someone who was a problem drinker or alcoholic
2. Living with someone who used street drugs
3. Parents were separated or divorced
4. Living with stepfather or stepmother
5. Living in a foster home
6. Running away from home for more than one day
7. Someone in household was depressed or mentally ill
8. Someone in household attempted to commit suicide
9. Someone in household went to prison
10. Someone in household committed a serious crime

We scored each experience dichotomously: 0 for 'No,' 1 for 'Yes.' The total ACEs score was calculated from the sum of scores across the 10 items, allowing a continuous range from 0 to 10.

Covariates

Educational attainment. Educational attainment refers to the highest level of education completed by the respondent. Participants self-reported whether they had completed less than high school, high school, some college, or were a college graduate or more (coded categorically as 0, 1, 2, or 3, respectively). In regression models, we included education as a potential confounder and used dummy coding, with "less than high school" as the referent group.

Age. Participants self-reported age in years. In multivariable regression models, we included continuous age (centered at the sample mean) as a potential confounder.

Response to unfair treatment. We used two items from the Experiences of Discrimination (EOD) Scale (Krieger, Smith, Naishadham, Hartman, & Barbeau, 2005) to operationalize response to unfair treatment. Each participant was asked to consider how she and others like her are treated, and how she typically responds. Items—which were previously shown to have acceptable test-retest reliability (Krieger et al., 2005)—and their response options read:

1. If you feel you have been treated unfairly, do you usually:
    a. accept it as a fact of life?
    b. try to do something about it?
2. If you have been treated unfairly, do you usually:
    a. talk to other people about it?
    b. keep it to yourself?

To explore potential interaction by response to unfair treatment, we dichotomized responses to the above items into two categories: passive responders and active responders. Passive responders reported that they both accepted unfair treatment as a fact of life, and kept it to themselves. Active responders were those who reported that they tried to do something about it, and/or talked to other people about it.

Our categorization of responses to these items is novel and exploratory. We note that extant studies typically classify respondents into four categories to describe response to unfair treatment (Ertel et al., 2012; Krieger & Sidney, 1996; Krieger et al., 2005). While our dichotomization is inconsistent with conventional practice to date, our method allows for basic ascertainment of potential interaction, which, if found, could be further explored in studies powered *a priori* for detecting interaction.

Statistical Analysis

We performed preliminary testing to check for the conditions required for our statistical analyses (e.g., normally distributed dependent variable and absence of multicollinearity); results indicated that analytic

assumptions were met. Using univariate analyses, we tabulated descriptive statistics for all variables of interest among the analytic dataset. We assessed relationships between continuous the JHP-RC© and ACEs using Pearson correlation coefficients and simple linear regression. We used linear regression to confirm bivariate associations of potential confounder with our exposure and outcomes variables, and used mean plots to visualize the relationship of educational attainment with the JHP-RC©. We then used multivariable linear regression, including age and educational attainment as covariates, to evaluate the adjusted association of ACEs on JHP-RC© scores. All regression analyses were repeated separately for each of the five JHP-RC© subscales (burden, coping, personal history, racism, and work) and for the Modified JHP-RC© (which excludes personal history subscale items). Finally, to explore potential interaction by response to unfair treatment, we added the dichotomous response to unfair treatment variable and an interaction term (response to unfair treatment*ACEs) to the multivariable models. We used a likelihood ratio test (LRT)—which compares the -2 Log L of the model including the interaction term to that of the no-interaction model—to determine whether there was evidence of statistically significant interaction. To visualize potential interaction effects, we created scatterplots demonstrating a regression line for the relationship between ACEs and gendered racial stress at each level of response to unfair treatment (i.e. active and passive). We considered $p<.05$ statistically significant, and used SAS 9.4 for all analyses.

Results

We present characteristics of our analytic sample of 405 Black women in early pregnancy (8-14 weeks' gestation) in **Table 3.1**. Mean (SD) age was 24.9 (4.7) years. Across education categories, the largest proportion of participants (38.3%) completed high school only. College graduates represented the smallest proportion (16.6%). 82% of the sample reported at least one ACE, and mean JHP-RC© score was 95.1 (20.6). The majority (76.5%) of participants responded actively (versus passively) to unfair treatment. Sensitivity analyses revealed no significant characteristic differences between women included and excluded from our analytic dataset (data not shown).

Of the JHP-RC© subscales, ACEs were most strongly correlated with personal history (**Table 3.2**; r=.49, p<.0001), followed by burden (r=.33, p<.0001). We found weak but statistically significant linear relationships with all other subscales, except racism (p=.12). College graduates reported the lowest coping scores and the highest racism scores, as compared to the other educational attainment groups (**Figure 3.1**). Mean burden, personal history, and work scores were not significantly different across educational attainment groups (data not shown).

Table 3.3 summarizes results from the no-interaction linear regression models including ACEs, educational attainment, and age. Together, these parameters were statistically significantly associated for the JHP-RC and all subscales. Concurrent with bivariate analyses, ACEs were statistically significantly associated with JHP-RC and all subscales except racism (b=.15, 95% CI: -.08-.37), after control for educational attainment and age. A 1-unit increase in ACEs (i.e., report of an additional ACE) was associated with an increase in the JHP-RC and all subscale scores. The strength of the effect of ACEs was greatest for burden (b=1.4, 95% CI: .98-1.8), followed by personal history (b=1.3, 95% CI: 1.1-1.6). High educational attainment was associated with a significant decrease in coping scores (b=-4.4, 95% CI: -7.8, -.99) and an increase in racism scores (b=2.5, 95% CI: .82-4.1), as compared to the lower educational attainment groups. There were minimal changes in R^2 (which represents the proportion of variance in the dependent variable explained by the independent variable/s) between simple and multivariable linear regression models (**Table 3.4**). Comparing R^2 for the ACEs-only models to the multivariable models, ACEs drove the majority of the variance explained by the set of independent variables for the JHP-RC© and all subscales (ΔR^2 =0-.01), except racism (ΔR^2 =.05), where variance appeared to be explained most by education.

After including an interaction term between ACEs and response to unfair treatment in hierarchically well formulated regression models (**Table 3.5**), likelihood ratio tests suggest significant statistical interaction for the full and modified JHP-RC©, and the racism and work subscales (LRT p-values<.05). Scatterplots (**Figure 3.2**) further illustrate a steeper regression line among passive versus

active responders, suggesting a stronger positive association between ACEs and gendered racial stress may exist for passive responders.

Discussion

Our results suggest that childhood adversity carries over into adulthood to affect the patterning of gendered racial stress in pregnancy. In this cohort of pregnant Black women, in which 82% were exposed to at least one ACE and 21% were exposed to 4 or more ACEs, increased adverse childhood experiences were associated with increased gendered racial stress scores, particularly among those who respond passively versus actively to unfair treatment. Adjustment for educational attainment and age did not attenuate the magnitude of associations of ACEs with gendered racial stress and its measured components. While extant literature confirms the cumulative, detrimental impact of early adversity on toxic stress over the lifecourse (Felitti et al., 1998; Hamby et al., 2021) this study is the first to our knowledge to demonstrate the relationship between ACEs and Black women's intersectional gendered racial stress in adulthood.

Empirical studies of gendered racial stress among Black women have focused predominantly on associated risk factors and outcomes in adulthood. Those studies have found positive associations of gendered racism with psychosocial stress (Carr, Szymanski, Taha, West, & Kaslow, 2014; Curry Owens & Jackson, 2015; F. M. Jackson et al., 2017; F. M. Jackson et al., 2012; Jioni A Lewis & Neville, 2015; J. A. Lewis et al., 2017; Mehra et al., 2020; Perry et al., 2012; Dawn M Szymanski & Lewis, 2016; Thomas et al., 2008). Additional studies have found positive association of gendered racism and adverse birth outcomes (Giscombé & Lobel, 2005; Rosenthal & Lobel, 2018). JHP scores, specifically, have been shown to be associated with adult depression, anxiety, anger, perceived stress, relationship status, household income, and parity (F. M. Jackson et al., 2017; F. M. Jackson et al., 2001; F. M. Jackson et al., 2012). However, the lifecourse approach urges attention to the role of early life experiences in patterning variation in toxic stress and related outcomes, which may then accumulate to create relative advantage or disadvantage in adult wellbeing (Ben-Shlomo & Kuh, 2002; Lu & Halfon, 2003). Results from the

present study are consistent with the premise that ACEs function as part of this patterning (Hamby et al., 2021), and add that the long reach of ACEs extends to Black women's contextualized gendered racial stress in pregnancy. Given that ACEs (Christiaens et al., 2015; Joshua P Mersky & ChienTi Plummer Lee, 2019) and racial stress (Giscombé & Lobel, 2005; Lu & Halfon, 2003; L. Rosenthal & M. Lobel, 2011) are associated with greater likelihood of preterm birth, and perinatal depression, gendered racial stress may exist on a causal pathway linking exposure to childhood adversity to Black women's birth outcomes. This mechanism warrants further testing with research that examines gendered racial stress not only as an outcome, but also as a mediating variable.

Differences in associations of ACEs across the JHP-RC© subscales may shed light on nuanced pathways linking pregnant Black women's childhood experiences to adult stress. Of the subscales, ACEs were most strongly associated with personal history (in unadjusted analyses) and burden (in adjusted analyses). The relationship with personal history can be explained partially by the conceptual overlap between the ACEs and personal history items, which both assess early life traumatic experiences. The relationship with burden is particularly notable. Indeed, pregnant women may have greater family burdens than women who are not mothers or women whose childrearing days have passed (Umberson, Pudrovska, & Reczek, 2010). In addition to assessing perceived lack of material and personal resources, the burden subscale captures the "imposed and embraced nurturing and caretaker roles associated with gender identity" (Jackson et al., 2005). As such, items associated with burden may approach measurement of some components of the Superwoman Schema (SWS), which describes Black women's sociocontextually-derived obligations to: (a) exhibit strength, (b) suppress emotions, (c) resist vulnerability (d) succeed independently and against any odds, and (e) prioritize care for others over self-care (Cheryl L. Woods-Giscombé, 2010). Specifically, burden items like "I am taking care of everyone else but no one is taking care of me" may reflect Black women's obligation to nurture others at their own expense (i.e., (e) above), which was found to be most strongly correlated with depression (Woods-Giscombe et al., 2019). Other items like "Everyone expects me to be strong for them" may assess an

obligation to appear strong and hide emotions, which Woods-Giscombé (2019) found to be moderately correlated with perceived stress. Given aforementioned associations of ACEs and gendered racial stress with these poor psychosocial outcomes, early adversity may encourage Black women's adoption of the Superwoman role later in life, which could heighten accumulation of gendered racial stress. Notably, a history of mistreatment or abuse is among the contextual factors identified as a contributor to the Superwoman role (Woods-Giscombe et al., 2019), which may further explain the relatively strong correlation between ACEs and personal history items. Still, additional study is needed to locate the role of SWS in the context of early life experiences and adult gendered racial stress.

Consistent with some previous research (Ertel et al., 2012; Krieger et al., 2005; Polanco-Roman, Danies, & Anglin, 2016), the majority of study participants responded to unfair treatment with active strategies (i.e., talking to others and/or trying to do something about it) versus passive strategies (i.e., accepting it and keeping it to themselves). Other studies have found that Black women are more likely to use avoidance rather than problem-solving or seeking social support (Thomas et al., 2008; Utsey et al., 2000), and that avoidance is associated with heightened distress conferred by experiences with gendered racism (Thomas et al., 2008). We demonstrate that a stronger association between childhood adversity and gendered racial stress may exist among passive responders, as hypothesized. This significant interaction was apparent only for the racism and work subscales, as well as the full and modified JHP-RC©. Researchers have identified factors that may contribute to the employment of active versus passive coping strategies in response to unfair treatment. For example, strong racial/ethnic identity—the degree to which an individual identifies with a racial or ethnic group—may be one mechanism that allays the harmful effects of racial discrimination (Brondolo, Ver Halen, Pencille, Beatty, & Contrada, 2009). Additional research shows that ACEs are associated with avoidant coping (e.g. acceptance) versus problem-focused coping (e.g. soliciting support), which in turn, is associated with poorer health in adulthood (Sheffler, Piazza, Quinn, Sachs-Ericsson, & Stanley, 2019). Together, these results suggest that accumulation of early life risk factors (e.g., ACEs) can alter the adult coping response, which can alter

Black women's reports of gendered racial stress in pregnancy. Given that our study was not optimally powered for interaction analysis, we offer limited explanation for our findings. However, if confirmed in future studies, our results point to avenues for the mitigation of the effects of childhood adversity on perinatal gendered racial stress through opportunities that bolster racial/ethnic identity, or otherwise promote active responses to unfair treatment.

Education served as a confounder in our multivariable models, and demonstrated notable bivariate relationships with components of gendered racial stress. Specifically, we found that high educational attainment (i.e., achieving or surpassing college graduation) was associated with increased coping resources in the context of greater exposure to racism, but not with greater burden. Due to institutionalized racism (C. P. Jones, 2000), women with greater education are more likely to be educated and later work in settings characterized by a predominantly White population (Leath & Chavous, 2018; Jioni A Lewis et al., 2016), and in which they are more likely to experience discrimination because of their race and sex (P. B. Jackson & Mustillo, 2001; Nuru-Jeter et al., 2009). High levels of gendered racial discrimination have been shown to place Black women at greater reproductive and psychosocial risk (J. W. Collins, Jr., David, Handler, Wall, & Andes, 2004; Mustillo et al., 2004). While it is surprising that educational attainment was not associated with the work subscale ($p>.05$; data not shown), our findings corroborate that highly educated women report greater racist encounters or anticipations, White privilege, and racial stereotypes (Jackson 2012). That burden scores did not differ significantly across educational attainment groups suggests that Black women's sociocultural obligations (Cheryl L. Woods-Giscombé, 2010; Woods-Giscombe et al., 2019) may apply broadly, regardless of socioeconomic status. Indeed, although a positive association between education and health is well established in the population at large (Berkman et al., 2014), studies of Black maternal health paradoxically demonstrate that college-educated Black women experience adverse birth outcomes more frequently than non-Black women of lower educational attainment (Curry Owens & Jackson, 2015).

Finally, lower coping subscale scores among women with higher education may point to a combination of multiple factors, including: (1) place- and community-based protective effects of

proximity to the city of Atlanta, with its majority Black population (51.0%)[5], and/or (2) an increased prevalence of high effort coping among the most educated. Described by the John Henryism Hypothesis, high-effort coping is "sustained cognitive and emotional engagement," which may come at a health cost (James, 1994). Studies testing the hypothesis have yielded mixed results. Some confirm that Black individuals of higher SES take on a detrimental psychosocial stress burden in the process of achieving or maintaining upward mobility (Curry Owens & Jackson, 2015; Light et al., 1995; McKetney & Ragland, 1996). Indeed, the effects of weathering may be greatest among Black individuals who consistently engage in high-effort coping (A. T. Geronimus, Hicken, Keene, & Bound, 2006). Other studies suggest the opposite: that John Henryism protects African Americans of higher SES from the negative health effects of race-based stressors (Bonham et al., 2004). Further study is needed to define the social contexts in which a lower gendered racial stress score (for the coping subscale, in this case) indicates a positive or negative health effect. The present study's finding that the magnitude of the effect of ACEs on gendered racial stress was not attenuated by control for education adds that higher SES may not offer protection to pregnant Black women against the lifecourse effects of childhood adversity.

Limitations

Our study has several limitations. First, the cross-sectional nature of data collection precludes causal interpretation. While the ACEs Questionnaire specifically asked participants to consider experiences in the first 18 years of life, future ACE research would benefit from measuring adversity prospectively. Second, ACEs data were gathered via retrospective self-report, which could introduce recall bias. Underestimates of childhood exposure to adversity would result in downwardly biased estimates of the relationship between ACEs and adult gendered racial stress. Third, we used a non-exhaustive list of covariates in our analyses, given limited data on factors temporally associated with childhood exposures. Thus, it is possible that we did not consider variables (e.g. direct versus proxy measures of maternal early socioeconomic status, and family resilience (Goldstein, Topitzes, Miller-Cribbs, & Brown, 2020)) that

[5] Data accessed here: https://www.census.gov/quickfacts/atlantacitygeorgia

explain some of the main and/or interaction effects. Finally, while the ACEs Questionnaire we employed included many constructs common to similar measures (e.g., household mental illness/suicide, household alcohol or substance abuse, incarceration of household member), it excluded others like bullying, and sexual abuse (Bethell et al., 2017), which may be uniquely related to gendered racial stress. Future ACE research among Black women may benefit from not only using a broader range of ACEs (Finkelhor, Shattuck, Turner, & Hamby, 2013), but from also using culturally-informed measures that include childhood exposure to racism-related experiences (Bernard et al., 2021). Still, quantifying the long-term stress burden of child adversity in this perinatal population is an important step in broadening our understanding of Black pregnant women's lived experiences.

Conclusions

This study demonstrates that associations between ACEs and perinatal gendered racial stress in adulthood remain after adjustment for demographic and socioeconomic factors. This enhances the call for inequities in Black maternal health to be targeted intersectionally, at multiple levels (inclusive of medical, political, social, and institutional factors), and at multiple points over time (Crenshaw, 1990; Cynthia Prather et al., 2018).

Table 3.1. Characteristics of Atlanta-area Black women in the study sample (N=405)

	Mean (SD)	**n (%)**
Sociodemographic factors		
Age	24.9 (4.7)	
Education		
Less than high school		68 (16.8)
High school		155 (38.3)
Some college		115 (28.4)
College graduate or more		67 (16.6)
Psychosocial factors		
JHP-RC©[a]	95.1 (20.6)	
Burden	28.2 (8.3)	
Coping	32.8 (9.1)	
Personal History	10.4 (5.4)	
Racism	13.3 (4.5)	
Work	10.4 (4.1)	
ACEs[a, b]	2.2 (1.9)	
0		73 (18.0)
1		93 (23.0)
2-3		144 (35.6)
4+		87 (21.4)
Response to unfair treatment[b]		
Active		310 (76.5)
Passive		66 (16.3)

[a]JHP-RC©: 39-item Jackson Hogue Phillips Reduced Common Contextualized Stress Measure; ACEs: 10-item Adverse Childhood Experiences Questionnaire

[b]Variable (n, % missing): ACEs (8, 2.0%); response to unfair treatment (29, 7.2%) There were no missing data for variables without this superscript.

Table 3.2. Bivariate association (Pearson's correlation coefficient, r) and simple linear regression of the JHP-RC©[a] and subscale scores on total ACEs[a] score (n=397)

	r	**R^2**	**Estimate (SE)[b]**	**p-value**
Full JHP-RC©	.42	.17	4.5 (.50)	**<.0001**
Modified JHP-RC[c]	.34	.12	3.1 (.43)	**<.0001**
Burden	.33	.11	1.4 (.21)	**<.0001**
Coping	.24	.06	1.1 (.23)	**<.0001**
Personal history	.49	.24	1.4 (.12)	**<.0001**
Racism	.08	.01	.18 (.12)	.12
Work	.17	.03	.36 (.11)	**.0006**

Bolded p-values are significant at alpha=.05 level

[a]JHP-RC©: 39-item Jackson Hogue Phillips Reduced Common Contextualized Stress Measure; ACEs: 10-item Adverse Childhood Experiences Questionnaire

[b]Parameter estimate (SE) for the total ACEs score

[c]Modified JHP-RC excludes items from the personal history subscale given conceptual overlap and relatively strong correlation between personal history items (See Table 2.1) and ACEs items

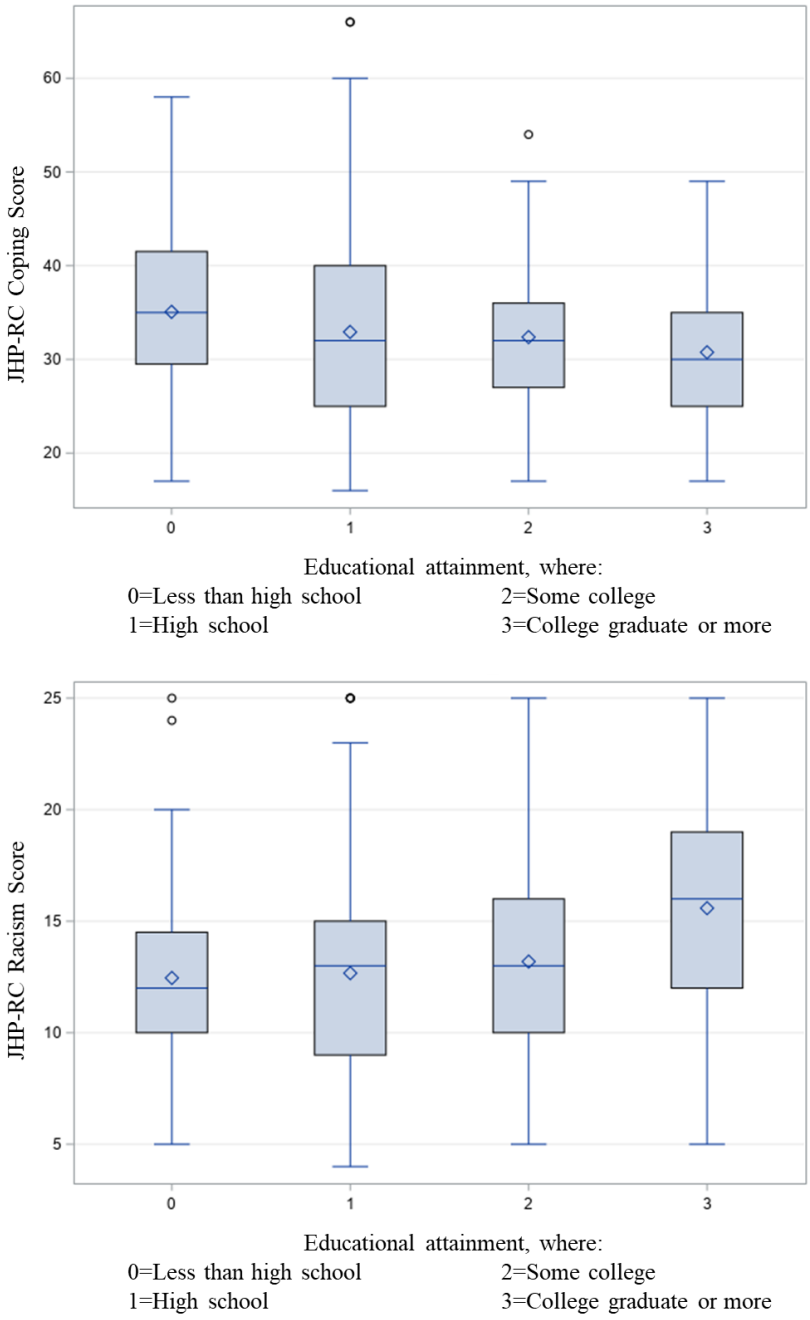


Figure 3.1. Mean JHP-RC©[a] coping (top) and racism (bottom)subscale scores across the educational attainment groups (n=405). College graduates (where 'Educational attainment'=3) reported the lowest mean coping scores (top) and the highest mean racism scores (bottom) relative to other educational attainment groups.

[a]JHP-RC©: 39-item Jackson Hogue Phillips Reduced Common Contextualized Stress Measure

Table 3.3. Multivariable regression of JHP-RC©© on ACEs[a] (n=397)

	Model parameters	Model p-value	Standardized estimate	Parameter estimate (SE)	Parameter estimate 95% CI	Parameter p-value
Full JHP-RC©	Total ACEs	**<.0001**	.40	4.3 (.49)	3.3, 5.3	**<.0001**
	<High school (ref.)		--	--	--	--
	High school		-.07	-2.9 (2.8)	-8.5, 2.6	.29
	Some college		-.07	-3.1 (3.0)	-9.0, 2.7	.29
	College grad+		-.10	-5.4 (3.6)	-12.5, 1.8	.14
	Age		.13	.56 (.23)	.12, 1.0	**.01**
Modified JHP-RC[b]	Total ACEs	**<.0001**	.33	2.9 (.43)	2.1, 3.8	**<.0001**
	<High school (ref.)		--	--	--	--
	High school		-.06	-2.2 (2.4)	-7.0, 2.6	.37
	Some college		-.07	-2.9 (2.6)	-8.0, 2.2	.27
	College grad+		-.10	-4.5 (3.2)	-10.7, 1.7	.15
	Age		.12	.46 (.20)	.07, .84	**.02**
Burden	Total ACEs	**<.0001**	.32	1.4 (.21)	.98, 1.8	**<.0001**
	<High school (ref.)		--	--	--	--
	High school		.01	.13 (1.2)	-2.2, 2.5	.91
	Some college		.03	.53 (1.3)	-1.9, 3.0	.68
	College grad+		-.05	-1.2 (1.5)	-4.2, 1.8	.43
	Age		.11	.19 (.09)	.00, .38	**.046**
Coping	Total ACEs	**<.0001**	.23	1.1 (.24)	.65, 1.6	**<.0001**
	<High school (ref.)		--	--	--	--
	High school		-.07	-1.4 (1.3)	-4.0, 1.3	.31
	Some college		-.12	-2.5 (1.4)	-5.3, 0.3	.08
	College grad+		-.18	-4.4 (1.7)	-7.8, -.99	**.01**
	Age		.04	.08 (.11)	-.13, .29	.46
Personal History	Total ACEs	**<.0001**	.47	1.3 (.13)	1.1, 1.6	**<.0001**
	<High school (ref.)		--	--	--	--
	High school		-.07	-.76 (.71)	-2.1, .64	.29

	Some college		-.02	-.27 (.75)	-1.8, 1.2	.72
	College grad+		-.06	-.86 (.92)	-2.7, .94	.34
	Age		.09	.10 (.06)	-.01, .22	.06
Racism	Total ACEs	**<.0001**	.06	.15 (.12)	-.01, .38	.19
	<High school (ref.)		--	--	--	--
	High school		.02	.16 (.65)	-1.1, 1.4	.81
	Some college		.04	.43 (.69)	-.93, 1.8	.53
	College grad+		.21	2.5 (.84)	.82, 4.1	**.004**
	Age		.10	.10 (.05)	-.00, .20	.06
Work	Total ACEs	**.002**	.15	.33 (.11)	.12, .54	**.002**
	<High school (ref.)		--	--	--	--
	High school		-.13	-1.1 (.60)	-2.3, .07	.06
	Some college		-.15	-1.4 (.64)	-2.6, -.08	**.04**
	College grad+		-.13	-1.4 (.78)	-2.9, .14	.08
	Age		.10	.09 (.05)	-.01, .19	.06

Bolded p-values are significant at alpha=.05 level

[a]JHP-RC©: 39-item Jackson Hogue Phillips Reduced Common Contextualized Stress Measure; ACEs: 10-item Adverse Childhood Experiences Questionnaire

[b]Modified JHP-RC© excludes items from the personal history subscale given conceptual overlap and relatively strong correlation between personal history items (See Table 2.1) and ACEs items

Table 3.4. Comparison of R^2 across unadjusted and adjusted regression models

	ACEs only[a]	**ACEs + education + age**
Full JHP-RC©[a]	.17	.18
Modified JHP-RC[b]	.12	.13
Burden	.11	.11
Coping	.06	.06
Personal history	.24	.24
Racism	.01	.06
Work	.03	.03

[a]JHP-RC©: 39-item Jackson Hogue Phillips Reduced Common Contextualized Stress Measure; ACEs: 10-item Adverse Childhood Experiences Questionnaire

[b]Modified JHP-RC© excludes items from the personal history subscale given conceptual overlap and relatively strong correlation between personal history items (See Table 2.1) and ACEs items

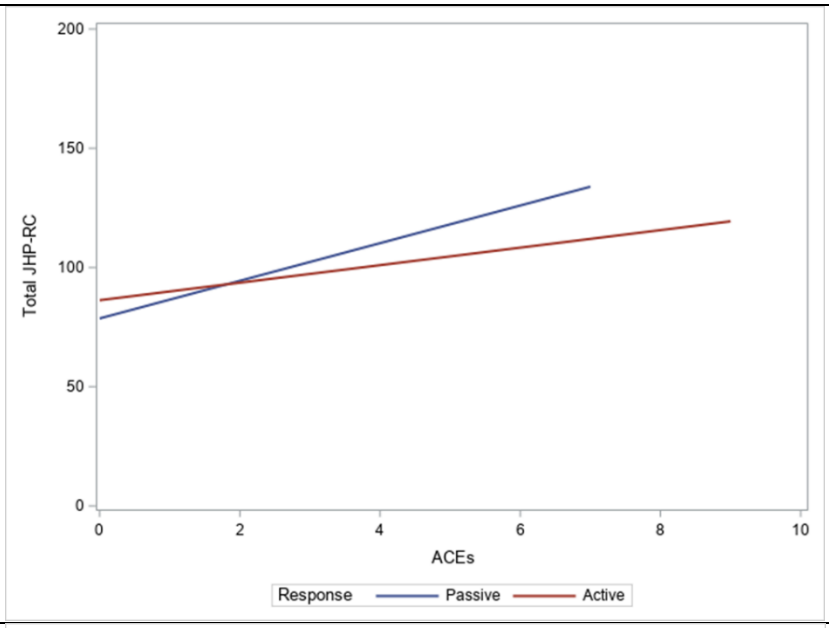
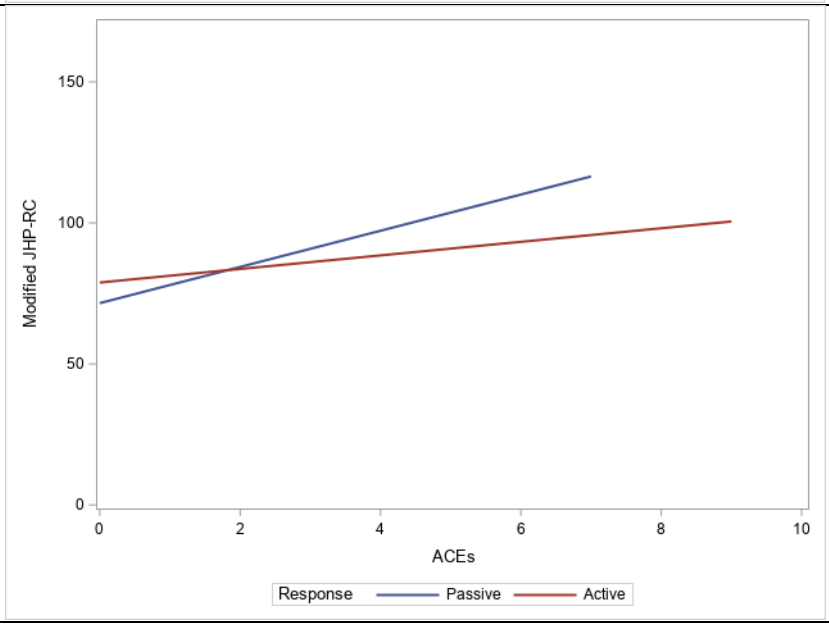
Table 3.5. Variance explained by the interaction model (R^2) & likelihood ratio test (LRT) statistical significance (n=371)

	R^2	**LRT p-value**
Full JHP-RC©[a]	.24	**.009**
Modified JHP-RC[b]	.21	**.002**
Burden	.17	.14
Coping	.07	.38
Personal history	.23	.59
Racism	.17	**.005**
Work	.07	**.02**

Bolded p-values are significant at alpha=.05 level

[a]JHP-RC©: 39-item Jackson Hogue Phillips Reduced Common Contextualized Stress Measure; ACEs: 10-item Adverse Childhood Experiences Questionnaire

[b]Modified JHP-RC© excludes items from the personal history subscale given conceptual overlap and relatively strong correlation between personal history items (See Table 2.1) and ACEs items

Total JHP-RC©	
Modified JHP-RC	

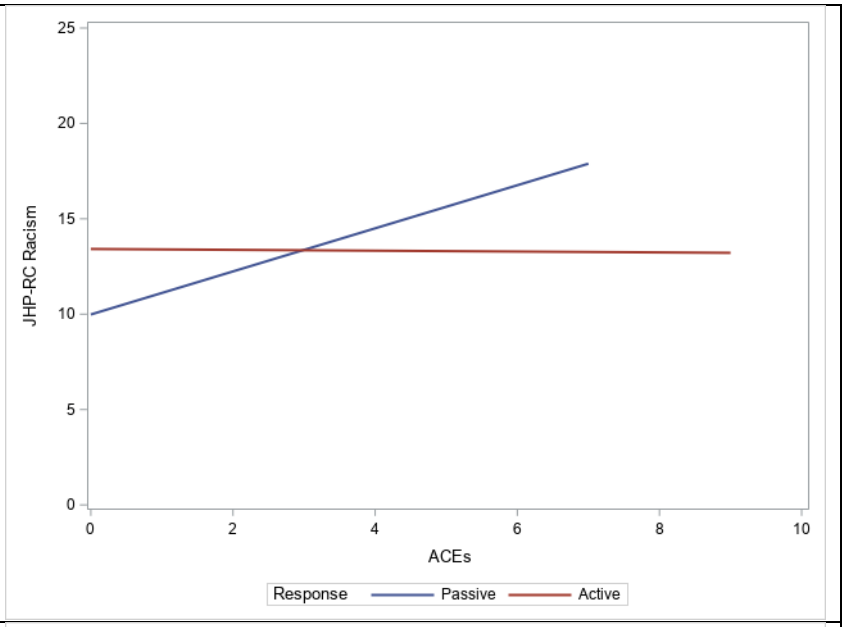
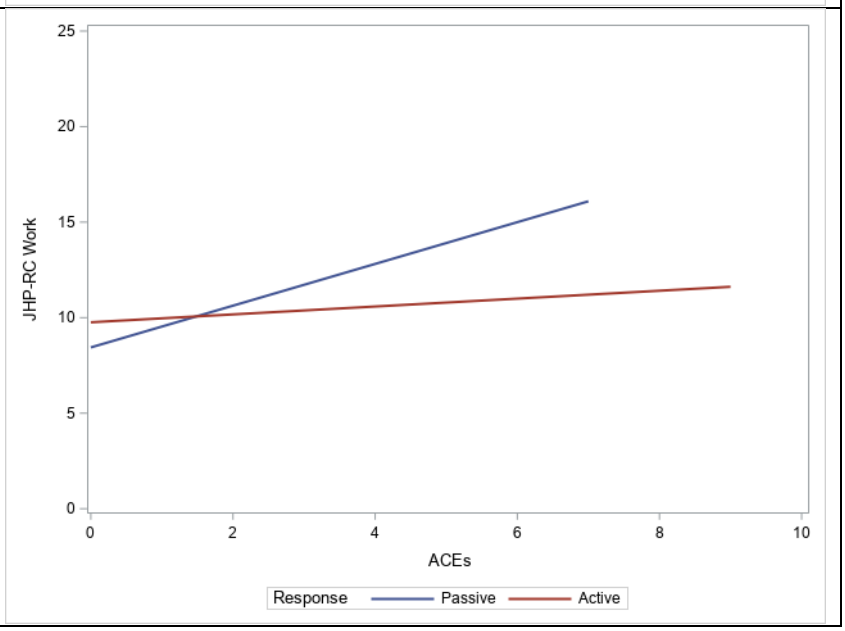
Racism Subscale	
Work Subscale	

Figure 3.2. Visualizing effect measure modification of ACEs-gendered racial stress relationship by response to unfair treatment among JHP-RC© variables with significant likelihood ratio tests (i.e. total JHP-RC, modified JHP-RC [excluding personal history items], and the racism and work subscales). Across panels, a steeper regression line exists among participants who respond passively (shown in blue), versus actively (shown in red), to unfair treatment.

Chapter 4: The association of maternal gendered racial stress with gestational age at birth among pregnant Black women

Introduction

Black women in the US are burdened with a disproportionate rate of adverse birth outcomes, which are inadequately explained by maternal socioeconomic status or prenatal health behaviors. The infant mortality rate for Black Americans is more than twice that for White Americans (Murphy et al., 2018), and much of this racial disparity can be explained by low birthweight and preterm delivery (L. Rosenthal & M. Lobel, 2011). Generally, 37 weeks' gestation has been used to dichotomize births as preterm (<37 weeks) or full term (≥37 weeks). However, research suggests that births before term are not heterogeneous: there may be a gradient of risk even beyond 37 weeks, such that early term ($37^{0/7}$-$38^{6/7}$ weeks) infants fare worse developmentally than do full term (≥39 weeks) infants (Boyle et al., 2012; Engle & Kominiarek, 2008; Sengupta et al., 2013). Furthermore, taking the lifecourse under consideration, emerging data consistently suggest that late preterm infants born between $34^{0/7}$ and $36^{6/7}$ weeks' gestation experience greater morbidity than children born at term (Loftin et al., 2010). Births can be additionally can be classified into two groups: (1) spontaneous, or idiopathic, birth in the absence or presence of premature rupture of membranes), and (2) medically indicated, or iatrogenic, births (Menon, 2008). The majority of all preterm births occur spontaneously (Wang et al., 2018), though heterogeneity in birth phenotyping suggests multiple, complex etiologic pathways differentiate gestational age and type of delivery (Wadhwa, Entringer, Buss, & Lu, 2011). Indeed, the contribution of maternal stress may vary between birth phenotypes, necessitating that studies of birth outcomes include their accurate and comprehensive categorization. Maternal stress, in particular, has been hypothesized to have the greatest main effect on spontaneous births near term (Wadhwa et al., 2011), and prior studies have identified Black race and low socioeconomic status as "risk factors" for spontaneous preterm delivery (Menon, 2008). Thus, studies of the association of gendered racial stress and birth outcomes need nuanced investigation of gestational age beyond a simple preterm or not preterm categorization. This literature calls for more nuanced investigation of factors associated with delivery at early gestational age for Black women with studies that consider various birth phenotypes (Wadhwa et al., 2011).

Mounting theoretical and empirical evidence suggests that racism, acting as a stressor for Black women, may help explain racial disparities in birth outcomes (Clark et al., 1999; Tyan Parker Dominguez et al., 2008; Giscombé & Lobel, 2005; Cynthia Prather et al., 2018; C. Prather et al., 2016; Lisa Rosenthal & Marci Lobel, 2011; Williams et al., 2003). Interpersonal racism, defined as discriminatory practices enacted on a person due to their racial/ethnic group (C. P. Jones, 2000) has been independently associated with greater psychological distress, including symptoms of depression and anxiety among Black versus White women, perhaps through chronic activation or weathering of neuroimmune stress pathways (A. T. Geronimus et al., 2006; Goosby et al., 2018; H. L. Jones et al., 2007; L. Rosenthal & M. Lobel, 2011). In turn, depression—known to be more prevalent among women (Kessler, 2003; Klein & Corwin, 2002) and among Black people (Miranda et al., 2005) may contribute to racial disparities in adverse birth outcomes (Giscombé & Lobel, 2005; M. R. Kramer et al., 2011; M. R. Kramer & Hogue, 2009; Ncube et al., 2017; Schulz et al., 2006). Gender-focused studies have also found sexism to be an independent stressor for Black women, for whom its associated with greater psychological distress as compared to White women (Dawn M. Szymanski & Stewart, 2010). Thus, stress due to racism and sexism experienced by Black women before and during pregnancy are important in understanding birth outcome disparities. However, interdisciplinary research further demonstrates that the experience of Black women is unique not just due to race *or* gender, but due to the convergence of their multiple lifelong identities as women *and* Black (Essed, 1991), and in pregnancy, as mothers (Nuru-Jeter et al., 2009; Lisa Rosenthal & Marci Lobel, 2011). Thus, scholars of intersectionality theory posit that empirical study of gendered racism should utilize measures that explore experiences of racism and sexism together, without attempt to disentangle them (Bauer, 2014; Bowleg, 2008; Bowleg & Bauer, 2016; Cho et al., 2013; J. A. Lewis et al., 2017; Thomas et al., 2008). To date, relatively few empirical studies employ intersectional frameworks or instruments that consider racism and sexism's joint effect in an effort to unpack and contextualize racial disparities in poor health outcomes.

At their intersection, sexism and racism combine to form a distinct, hybrid form of oppression called gendered racism, which is characterized by racist constructions of gender roles (Essed, 1991; J. A. Lewis et al., 2017). Gendered racism may present extra exposure to stress for Black women (Essed, 1991; F. M. Jackson et al., 2001), and creates an environment in which Black women feel they must maintain a persistent vigilance in anticipation of racist events against themselves and their children (F. M. Jackson et al., 2005). In one of few studies on gendered racism's pregnancy-related health effects, Rosenthal et al. (2018) observed three main findings: (1) Black and/or Latina (vs White) women reported greater stereotype-related gendered racism, (2) stereotype-related gendered racism was associated with greater pregnancy-specific stress, and (3) stereotype-related gendered racism mediated the relationship between race/ethnicity and pregnancy-specific stress. Stereotype threat, similar to hypervigilance or chronic worry, involves being concerned about the possibility of personally confirming negative group-based stereotypes (Rosenthal & Lobel, 2018). However, this study did not examine the role of stereotype-related gendered racism in actual birth outcomes, just in pregnancy-related stress. In a qualitative study of pregnant Black women, Mehra et al. also observed that gendered racism in the form of stereotypes was a source of stress for Black women, who may develop relatively effective (active) or ineffective (passive) coping tools (Mehra et al., 2020). Gendered racism related to stereotype threat likely represents just one component of gendered racism. While stereotypes related to sexuality and motherhood are likely of particular salience to pregnant Black women in healthcare settings (Abdou & Fingerhut, 2014), a host of other stressors (e.g., obligation to care for others; workplace discrimination) plausibly contribute to gendered racial stress, and merit investigation.

Additionally, sociodemographic factors may influence the relationship of gendered racial stress with birth outcomes. According to the weathering hypothesis, younger age is associated with lower accumulated health burden for socially-disadvantaged women (A. T. Geronimus, 1992). By regarding maternal age as both a biological and psychosocial characteristic, the weathering hypothesis provides a useful framework to explore how the life course progression of Black women's stress exposure in

women's health culminates into their birth outcomes (S. Kim, Im, Liu, & Ulrich, 2020). To date, however, limited studies prospectively examine the role gendered racial stress in pathways leading to adverse birth outcomes.

Thus, in the present study, we investigate the associations of Black women's intersectional gendered racial stress in early pregnancy with spontaneous preterm birth and spontaneous early term birth. We further assess whether maternal age may moderate these associations, and whether these associations are mediated by symptoms of depression.

Methods

Participants

The prospective Biobehavioral Determinants of the Microbiome and Preterm Birth in Black Women Study (n=485) aimed to provide a biopsychosocial understanding of within-race risk for adverse birth outcomes among a socioeconomically diverse sample of Black women (Elizabeth J Corwin et al., 2017). At enrollment, participants were receiving prenatal care at either Grady Memorial Hospital or Emory University Hospital Midtown—two Atlanta-area facilities (public and private, respectively) that see approximately 10% of Georgia's singleton live births to Black women. Eligible women were between 18-40 years of age, self-identified as African-American (i.e., US-born and of African-American or Black race), were carrying a singleton pregnancy between 8-14 weeks' gestation, and had no reported chronic medical conditions or conditions requiring long-term prescription management. During the first study visit at 8-14 weeks' gestation, sociodemographic, psychosocial, and other health information was collected via self-report. Post-delivery, additional clinical information, including gestational age at birth, type of delivery, previous preterm birth, and pre-pregnancy BMI, were gathered via maternal medical record abstraction. We excluded women from these analyses due to missing data on gendered racial stress (n=36), ineligible birth outcomes (i.e., gestational age less than 20 weeks [n=44], medical induction of labor [n=36: 17 preterm, 19 early term], or due to perinatal cardiometabolic conditions among women who gave birth full term [n=65]). Thus, the present cohort study is comprised of 304 women. The Emory

University Institutional Review Board granted ethical approval for all data collection, and all participants provided written informed consent.

Measures

Birth outcomes

Our outcomes of interest include spontaneous early term birth and spontaneous preterm birth. At the first study visit, participants underwent early pregnancy dating, via last menstrual period and/or early ultrasound assessment. Gestational age at birth was determined from abstraction of the maternal delivery record, based upon the date of delivery in relation to early pregnancy dating. Spontaneous (versus induced) births were deemed most relevant to evaluating the direct contribution of gendered racial stress to gestational age at delivery. Spontaneous preterm births were those occurring after 20 weeks' gestation but before 37 weeks' gestation. Spontaneous early term births were those occurring between gestational week $37^{0/7}$ and gestational week $38^{6/7}$. Our comparison group, full term births delivered at or after 39 weeks' gestation, excludes full term births to mothers with pre- and perinatal cardiometabolic complications, including gestational diabetes, preeclampsia or gestational hypertension, and premature rupture of membranes (n=65). As such, we label births in this category healthy full term births henceforth.

Gendered racial stress

To operationalize maternal exposure to gendered racial stress, we use continuous scores from the 39-item Jackson Hogue Phillips Contextualized Stress Measure (JHP), which we call the JHP-Reduced Common (JHP-RC©; see Table 2.2). This reduced scale includes items from the original 68-item JHP (F. M. Jackson et al., 2005; F. M. Jackson et al., 2014) that were found to be most commonly experienced by Black women who worked at one of two public hospitals and were enrolled in a federally funded worksite fitness study (Freeman, 2009). The JHP-RC© assesses specific exposure to chronic racial and gendered stress among Black women, and consists of four subscales measuring stressors (burden, personal history, racism, and work) and one measuring stress mediators (coping), with 5-level Likert scoring from greatest agreement to greatest disagreement. The burden subscale includes 10 items that measure gendered role

strain, and distress due to inadequate resources to meet demands. It includes 3 of the 4 items from the original JHP Scale referred to as "stress states" (F. M. Jackson et al., 2005; F. M. Jackson et al., 2014). The personal history subscale includes 5 items that assess experiences with emotional or physical abuse, and family or partner substance abuse. The 5 racism items capture racial stereotypes, White privilege, and the perceived impact of racism on children's lives. The 5-item work subscale assesses racism and sexism in the workplace. The 14 coping items measure coping resources (including support from and belonging to one's community) and capacity for coping based on race and gender. To calculate total and subscale JHP-RC© scores, we coded items 1-5 and reverse coded items, as necessary, so higher values for each item indicated greater gendered racial stress. In our sample, total JHP-RC© score ranges from 47 to 159. Ranges for the subscales are: burden=10-50, coping=16-66, personal history=3-25, racism=4-25, and work=2-25. Cronbach's alpha coefficient for the full JHP-RC© was .86 in the present study. The subscales had the following alpha coefficients: burden=.84, coping=.82, personal history=.87, racism=.75, and work=.82. These values indicate acceptable to high internal reliability consistency.

Covariates

The following demographic and psychosocial factors—deemed risk factors for early gestational age (i.e., <39 weeks)—were self-reported at the first study visit:

- Via sociodemographic survey,
    - Age (years)
    - Educational attainment: participants reported whether they had completed less than high school, high school, some college, or college graduate or more (coded 0, 1, 2, or 3, respectively). For adjusted regression analyses, we used dummy coding, with "less than high school" as the referent group.
    - Insurance status: participants reported whether they were privately or publicly insured (i.e. receiving low-income or Right from the Start Medicaid). We coded these responses as either 0 (public insurance) or 1 (private insurance).

  - Relationship status: participants reported whether or not they were married or cohabiting. We coded these responses as either 0 (not married or cohabiting) or 1 (married or cohabiting).

- Via health survey,
  - Tobacco use in the last month
  - Alcohol use in the last month
- Via measures of psychosocial stress,
  - Perceived stress: Cohen's 14-item Perceived Stress Scale (PSS) assesses "the degree to which individuals appraise situations in their lives as stressful" over the last month (Sheldon Cohen, Kamarck, & Mermelstein, 1983). We summed items, coded 0 to 4, where higher values indicate greater perceived stress. In our sample, scores ranged from 0 to 45. The PSS is among the most widely used instruments of perceived stress, and demonstrates acceptable internal consistency in this sample (Cronbach's alpha=.72).
  - Childhood adversity: The Adverse Child Experiences Questionnaire (ACE) ascertains experiences related to dysfunction in childhood home (i.e. in the first 18 years of life) by asking about 10 experiences (Felitti et al., 1998). We scored each item dichotomously: 0 for 'No,' 1 for 'Yes.' The total ACEs score was calculated from the sum of scores across the 10 items, allowing a continuous range from 0 to 10. ACE is used extensively among women of reproductive age of various race/ethnicity and income status. Cronbach's alpha in this sample was .67.
  - Depressive symptomology: The Edinburgh Depression Scale (EDS) is a 10-item self-report questionnaire ascertaining symptoms of depression in the last 7 days (Cox, Holden, & Sagovsky, 1987). While originally developed for use in the postnatal period, the scale has been used and validated in studies of pregnant women and shows high sensitivity and specificity for the detection of prenatal major depressive disorder (Bergink et al., 2011; Grobman et al., 2016; Kozinszky & Dudas, 2015). The EDS maintains high internal

consistency reliability in studies of Black pregnant populations (Tandon et al., 2012; Yonkers et al., 2001). We summed items, coded 0 to 3, where higher scores indicate greater depressive symptomology. In our sample, scores range from 0 to 25. Using generally recommended thresholds, an EDS score ≥10 indicates the presence of depressive symptomology (Cox et al., 1987; Kendler, Karkowski, & Prescott, 1998; Kessler, 2003). In this sample, Cronbach's alpha for the EDS was .85.

After delivery, information detailing the following clinical covariates—also deemed risk factors for early gestational age—was gathered via medical record abstraction:

- Pre-pregnancy body mass index (BMI, kg/m^2): categorized conventionally as <18.5, 18.5-<25. 25-<30, or ≥30.
- Parity: dichotomized as 0 for primiparity or 1 for multiparity (at least one previous live birth).
- Previous preterm birth, which was assessed only among multiparous women, and dichotomized as 0 for no history of preterm birth or 1 if the participants had given birth preterm (spontaneous or induced) at least once before.

Statistical Analysis

All analyses were conducted at the $p<.05$ level for statistical significance, with SAS version 9.4. Using univariate analyses, we assessed model assumptions and descriptive statistics for all variables of interest. For each predictor and covariate, we compared the means and distributions among spontaneous preterm births (n=46) versus healthy full term births (n=171), and separately, among spontaneous early term births (n=87) versus healthy full term births. We used Student's t-tests to assess differences between quantitative variables. To test differences in frequencies across levels of categorical variables, we used Pearson's Chi-square tests.

Binary outcomes, as in the present study, are often analyzed with logistic regression models, which yield odds ratios comparing groups. We report average marginal effects here given consensus in

the literature that the adjusted odds ratio derived from logistic regression does not approximate the risk ratio when the outcome of interest is common (>10%), and thus is more difficult to interpret and compare. One report more specifically defines the criteria for using the odds ratio: "in a cohort study, if the incidence of the outcome is more than 10% and the odds ratio is more than 2.5 or less than .5, correction of the odds ratio may be desirable to more appropriately interpret the magnitude of association" (Zhang & Kai, 1998). Given that the prevalence of both spontaneous preterm and spontaneous early term birth were common in our sample, comparison of risk, not odds, was deemed most appropriate for our analyses. To accommodate our binary outcome measures, we used multiple logistic regression analysis in combination with the predictive margins method (Korn & Graubard, 1999) to compute the average marginal effects (AME) and 95% confidence intervals (CI) of the association of gendered racial stress with spontaneous PTB or spontaneous ETB.[6] As described by Korn and Graubard (1999), the average marginal prediction allows for comparisons of predicted outcomes, or risks, between subsets of people in a population defined by exposure. For a continuous predictor, like the JHP-RC©, the average marginal effect assesses instantaneous rate of change, and thus approximates the effect of a 1-unit change in the predictor on the percentage point change in probability of the outcome (Cameron & Trivedi, 2010).

Conventionally, if the exposure-outcome association changes by at least 10% when a confounder is included, that confounder is maintained in adjusted models (Maldonado & Greenland, 1993). Of the risk factors we examined, none altered marginal effects by ≥10%. Given our *a priori* selection of covariates as factors theoretically and/or empirically associated with racial stress (Beatty Moody et al., 2014; Broman et al., 2000; L. M. Christian et al., 2013; Clark et al., 1999; T. P. Dominguez et al., 2005) and gestational age (Blackmore, Savitz, Edwards, Harlow, & Jr., 1995; Frazier et al., 2018; Lilliecreutz, Larén, Sydsjö, & Josefsson, 2016), we acknowledge their importance in the causal pathways under study and maintained all risk factors in adjusted models. As such, we present only adjusted measures of association. To further verify that the psychosocial covariates were appropriate to include

[6] Margins macro accessed here: https://support.sas.com/kb/63/038.html

altogether in adjusted models, we checked their correlation coefficients (r). Correlations between perceived stress, stressful life events, and childhood adversity ranged from .2 to .4, suggesting that the corresponding measures assess distinct stress-related constructs. Similarly, those measures were not highly correlated with components of gendered racial stress (r= <.1-.5).

To investigate moderation by age in fully adjusted models, we included an interaction term between age (categorized into data-driven tertiles) and gendered racial stress, along with individual variables for the social exposures and gendered racial stress. We computed average marginal effects of the exposure-outcome association within each age tertile, and assessed significant differences of the average marginal effects across tertiles of age. To visually verify potential interaction, we created an effect plot.

Finally, we used the CAUSALMED procedure[7] to examine whether the presence of perinatal depressive symptoms mediated the gendered racial stress-birth outcome association. The procedure computes total effects and decomposed effects (e.g. indirect effects) that can be interpreted as marginal effects, consistent with prior mediation analysis literature (Valeri & VanderWeele, 2013).

Results

We summarize the demographic, behavioral, clinical, and psychosocial characteristics of women in the sample, stratified by birth outcome, in **Table 4.1**. Public insurance (93.5% vs. 76.6%; p<.05) as well as previous preterm birth (41.4% vs. 5.7%; p<.05) were more common among the spontaneous preterm birth (sPTB) group as compared to the healthy full term group. Previous preterm birth was also more common among the spontaneous early term birth (sETB) group relative to the health full term group (27.6% vs. 5.7%; p<.05). However, primiparity (33.3% vs. 48.5%; p<.05) and stressful life events (average of 3.3 vs. 4.2; p<.05) were significantly less frequent among women with sETB versus healthy full term births. Depressive symptomology, measured by an EDS score ≥10, was common (27.5%-39.1%) across the three

[7] Additional information found here: https://support.sas.com/documentation/onlinedoc/stat/143/causalmed.pdf; https://www.sas.com/content/dam/SAS/support/en/sas-global-forum-proceedings/2018/1991-2018.pdf

birth outcome strata, though the frequency of depressive symptoms among sPTB or sETB women was not significantly different than that among the healthy full term group (p-values>.05).

Table 4.2 presents mean gendered racial scores, and average marginal effects (AMEs) for components of gendered stress and sPTB vs. healthy full term birth. The estimated margin, .013 (95% CI: .002, .024), indicates that increasing JHP-RC racism subscale significantly increases the probability of sPTB as compared to healthy full term delivery (p=0.02). These results suggest a 1-unit increase in score on the JHP-RC racism subscale was associated with a 1.3 percentage point increase in the probability of sPTB as compared to healthy full term birth. Average marginal effects of other components of gendered racial stress on sPTB were smaller in magnitude (-.002 to .007) and non-significant. Methodological literature supports two ideas: (1) that average marginal effects are easiest to interpret when categorical or continuous variables are set at least 2 specific values of interest (Bieler, Brown, Williams, & Brogan, 2010; Graubard & Korn, 1999), and (2) that the effect of changing a continuous predictor from one level to another may not be constant across the scale on which the predictor is measured (Cameron & Trivedi, 2010). To explore the variation in AMEs across the JHP-RC© racism scale, we set racism scores at four levels across the range of the subscale (**Table 4.3**). On the low end, increasing JHP-RC© racism score by 1-unit from a score of 5, is associated with a .9 percentage point increase in probability of sPTB (p=.0002). On the high end, increasing JHP-RC© racism score from a score of 20 to 21 is associated with a 1.8 percentage points increase in probability of sPTB (p=.054). **Table 4.4** presents mean gendered racial scores, and non-significant average marginal effects for subscales of gendered stress and sETB vs. healthy full term birth.

To explore moderation by age, we included age tertiles in an interaction term with JHP-RC© racism. The UNIVARIATE procedure yielded cutpoints at 22 and 28 years. There was a significant average marginal effect of racism on sPTB within the oldest age tertile (percentage point increase in probability of sPTB: 1.5; 95% CI: .3, 3; p=.02), but not within the youngest tertile (percentage point increase in probability of sPTB: 1.1; 95% CI: -1, 3; p=.02), or the middle tertile (percentage point

increase in probability of sPTB: .6; 95% CI: -1, 2; p=.02). There were also no significant differences across the tertile-specific average marginal effects (p-values≥.35). The effect plot in **Figure 4.1** illustrates that, at lower racism scores, risk of sPTB is highest among the youngest tertile, followed by the middle tertile, then the oldest tertile. However, at higher racism scores, the risk of sPTB among the oldest tertile surpasses that of the middle tertile.

Testing mediation of the JHP-RC© racism and sPTB association by depression in no-interaction models yielded a null indirect effect (percentage mediated: 3.3, 95% CI: -16, 22.5, p=.74; **Table 4.6**).

Multiple logistic regression models without use of the predictive margins approach yielded odds ratio less than 2.5 and greater than .5, so we report crude and adjusted odds ratios for the associations tested here in this chapter's appendix. The qualitative description of main findings remains largely the same, irrespective of the measure of association.

Discussion

Previous studies suggest that Black women's racial stress contributes to the racial disparity in adverse birth outcomes, including earlier gestational age (L. Rosenthal & M. Lobel, 2011; Rosenthal & Lobel, 2018). However, few studies have used intersectional measures of gendered racial stress to investigate its association with gestational age. We predominantly found non-significant marginal effects of the gendered racial stress-gestational age association. However, results suggest that experiences with and/or perception of racial stereotypes, White privilege, and the impact of racism on children's lives (as measured by JHP-RC© racism items) are associated with increased probability of spontaneous preterm birth, but not spontaneous early term birth, as compared to full term birth.

Lack of statistical significance elsewhere masks that the average marginal effects are largely similar in absolute value. Moreover, that no significant effect was found in all associations except racism-sPTB could point to insufficient sample size across birth outcome groups. Given that lack of statistical significance does not make our study findings meaningless or uninformative *per se*, we offer the

following exploratory interpretations of the associations of subscales of gendered racial stress with early onset gestational age, relative to healthy full term delivery. In general, higher burden, personal history, racism and work scores were associated with an increase in probability of sPTB, while higher coping scores were associated with a decrease in probability of sPTB. Greater burden, coping, and personal history appeared to confer greater risk for sETB, while higher racism and work scores decreased likelihood of sETB. That gendered racial stress burden and personal history of adversity in earlier life are associated with earlier gestational age is unsurprising, and corroborates extant literature on the effects of racial and psychosocial stress pathways in pregnancy (L. Rosenthal & M. Lobel, 2011; Rosenthal & Lobel, 2018). In our sample, racism scores are patterned by educational attainment: college graduates report significantly higher scores than all other groups ($p<.0001$). Our findings may support that the social and financial status offered by greater educational attainment still leave Black women and infants at risk for the worst birth outcomes (G. A. McGrady, J. F. Sung, D. L. Rowley, & C. J. Hogue, 1992). Control for education did not attenuate the average marginal effect.

The relationship of coping to sPTB and sETB in the negative and positive directions, respectively, may echo mixed results in the John Henryism literature (Bonham et al., 2004; Curry Owens & Jackson, 2015; A. T. Geronimus et al., 2006; James, 1994). Coping scores were also patterned by educational attainment, and were lowest among the most educated women in our sample. Educated women may be performing more high-effort coping (James, 1994), which may have protective or harmful health effects. We refrain from drawing conclusions based on our mostly null findings, and recommend future studies to confirm the effect sizes we observed.

Returning to our main significant finding, i.e., that that gendered racial stress may be associated with sPTB but not with sETB, this supports a gradient of risk across gestational age. A confluence of study results suggests that fetal maturation is not linear across gestation (Raju, 2013). Indeed, morbidity among early-term infants is considerably less than that among late-preterm infants (Sengupta et al., 2013). Previous findings that each additional week between 34 and 38 weeks substantially diminished the

relative risk of infant morbidity (Gouyon et al., 2010; Shapiro-Mendoza et al., 2008) suggest that if gendered racial stress is associated with delivery prior to 36 weeks, it may put Black mothers and babies at additional risk than delivery even a week later. In 2018, infant mortality at 34-36 weeks gestation (known as late preterm) was 8.21 per 1,000 live births, but 2.05 per 1,000 live births at 37-41 weeks gestation (Ely & Driscoll, 2020). In the same year, mortality rates were highest among Black infants (10.75 per 1,000 live births) than among infants in other race/ethnicity groups. This disparity may be more apparent in Georgia, one of sixteen states with significantly higher infant mortality than the US rate (Ely & Driscoll, 2020). Of the 46 preterm births in our analytic sample, nearly three-quarters (n=34) were late preterm, compared with 87 early term births. These results match national statistics wherein late preterm infants account for approximately 70% of preterm births in the United States (Stewart & Barfield, 2019), and where early-term infants outnumber late-preterm infants by about 3 to 1 in the United States (Sengupta et al., 2013). While sample size limits generalization of our study results to the US population of Black women, the combination of our results and national statistics suggests a role for gendered racial stress in increasing hazard for Black women and babies at earlier gestational ages. Additionally, given the aforementioned association of racism scores with education, our results add that gendered racial stress may help explain the persistence of racial disparities in early onset gestational age after adjustment for sociodemographic factors (Goldenberg et al., 1996). Among White women, only low maternal education increases the risk of preterm delivery (Michael S Kramer, McLean, Eason, & Usher, 1992; G. A. McGrady et al., 1992). Among highly educated Black women who may face greater exposure to interpersonal racism, the risk of preterm delivery is not fully attenuated by their social status (Johnson, Green, Vladutiu, & Manuck, 2020; G. A. McGrady et al., 1992). Sociodemographic determinants of pregnancy risk may act indirectly through stress mechanisms that enable gendered racism to get under Black women's skin (M. R. Kramer et al., 2011; M. R. Kramer & Hogue, 2009; Michael S. Kramer et al., 2009), though further investigation is needed to better define the pathways.

Results from our moderation and mediation analyses go in the direction of testing particular stress-related pathways to early onset gestational age, but could not confirm the mechanisms at work in our study sample. A significant average marginal effect of JHP-RC© racism on sPTB in just the oldest age tertile offers support for our hypothesis that older pregnant women with higher gendered racism scores would be most likely to give birth early. Examination of the effect plot of age further reveals that JHP-RC© racism scores may pattern the maternal age-related increase, or weathering, associated with sPTB. Higher gendered racial stress tended to amplify the maternal age-related increase in probability of sPTB among the youngest and oldest groups, but not as much among the middle age group. At lower gendered racial stress scores, we observed the ordinal or linear relationship of maternal age to sPTB risk found in other studies of Black women, where the youngest women experience the greatest risk (A. T. Geronimus, 1992; Arline T Geronimus, 1996; Rich-Edwards, Buka, Brennan, & Earls, 2003). From the weathering perspective, maternal age for Black women could be reconceptualized as the duration of their exposure to stressful conditions over the lifecourse (A. T. Geronimus, 1992; S. Kim et al., 2020). Thus, the increasing maternal age effect among women 28 and above in the face of greater chronic gendered racial stress may indicate the cumulative impact of stress on the body over time. While this notion supports the weathering hypothesis, it is contradictory to emerging evidence that older, rather than younger, age may function as a buffer to health outcomes related to racism (Greer & Spalding, 2017).

Irrespective of the specific effects of age, greater lifecourse stress and worse bodily wear and tear are both risk factors for depression (Leonard, 2000; McEwen, 2003). Null results for mediation of the gendered racial stress-sPTB relationship by depression are surprising given previous study findings that depression is associated with gendered racial stress (unpublished manuscript)—particularly its burden subscale—and with racial stress more broadly (Giscombé & Lobel, 2005; M. R. Kramer et al., 2011; Ncube et al., 2017). Previous studies have also found a mediating role for depression in the long-term effects of late preterm birth (Rogers, Lenze, & Luby, 2013). We believe additional adequately-powered studies are needed to verify these and other moderating and mediating pathways linking Black women's contextualized stress to their birth outcomes.

Limitations

The results of this prospective cohort study should be considered in light of its limitations. First, small sample size across birth outcome groups limits study power and may obscure the true magnitude of main and interactive effects of interest. Future research using a large population-based sample may confirm whether associations are stronger than observed, or due to chance. The prevalence of preterm birth among Black women in our sample (15.1% spontaneous PTB) likely exceeds the national race-specific preterm birth rate of 14.4% in 2019,[8] which includes spontaneous and induced cases. In addition to violating the rare outcome assumption necessary for multiple logistic regression, this further limits generalizability of study findings to the overall population. Furthermore, the relatively young age of women in our sample (mean, SD: 24.9, 4.7), does not lend itself to robust testing of the weathering hypothesis. We recommend including an even distribution of participants across the childbearing age range in later studies of maternal age patterning of gendered racial stress-birth outcome associations. Second, we focused on spontaneous labor and excluded births that were medically induced. Though we did consider multiple gestational age categories (i.e. preterm and early term), early onset gestational age is not only a heterogeneous entity in terms of the extent to which the birth occurs before 39 weeks' gestation, but also in terms of whether the birth is medically indicated (i.e., induced), spontaneously initiated, or precipitated by premature rupture of membranes (Menon, 2008; Wadhwa et al., 2011). Given that distinct and/or interrelated etiologic mechanisms may underlie the type of birth (Menon, 2008), we acknowledge that the contribution of maternal gendered racial stress to adverse pregnancy outcomes cannot be fully understood by study of spontaneous births alone. Future studies should take these points under consideration to more precisely identify the role of gendered racial stress in: (1) additional hazard related to each successive week of gestation, (2) competing risks affecting spontaneous and induced delivery (Joseph & Kramer, 2018), and (3) maternal lifecourse physiological health complications.

[8] Data accessed here: https://www.cdc.gov/reproductivehealth/maternalinfanthealth/pretermbirth.htm

Conclusions

Spontaneous preterm births—especially those in the late preterm period—represent the majority of all preterm deliveries in the United States, and early term births represent about 30% of all national live births (Martin, Hamilton, Osterman, & Driscoll, 2019). For decades, Black women have been at twofold or greater risk of spontaneous preterm birth compared with White women (Demissie et al., 2001). This persistent racial disparity amplifies the public health importance of studying spontaneous preterm and early term birth outcomes in cohorts of Black women, wherein within-race variation in susceptibility to and consequences of risk factors can be best understood. Our investigation of gendered racial stress as a risk factor along the pathway toward early delivery is an initial step toward elucidating additional structural and biopsychosocial determinants of adverse birth outcomes among Black women.

Table 4.1. Characteristics of study participants by birth outcome (N=304)

	Healthy full term[a] (n=171)	sPTB (n=46)	sETB (n=87)
Age, mean (SD)	24.8 (4.7)	24.7 (4.9)	25.6 (4.8)
Education, n (%)			
Less than high school	28 (16.4)	7 (15.2)	12 (13.8)
High school	56 (32.8)	24 (52.2)	37 (42.5)
Some college	54 (31.6)	11 (23.9)	23 (26.4)
College graduate or more	33 (19.3)	4 (8.7)	15 (17.2)
Insurance status, n (%)			
Public (Medicaid)	131 (76.6)	43 (93.5)*	67 (77.0)
Private	40 (23.4)	3 (6.5)*	20 (23.0)
Relationship status, n (%)			
Either married or cohabiting	92 (53.8)	29 (63.0)	41 (47.1)
Neither	79 (46.2)	17 (37.0)	46 (52.9)
Prepregnancy BMI, kg/m^2, n (%)			
<18.5	8 (4.9)	1 (2.2)	0 (0)
18.5-<25	61 (35.7)	20 (43.5)	40 (46.0)
25-<30	41 (24.0)	17 (37.0)	19 (21.8)
≥30	61 (35.7)	8 (17.4)	28 (32.2)
Primiparous, n (%)	83 (48.5)	17 (37.0)	29 (33.3)*
Previous PTB[b], n (%)	5 (5.7)	12 (41.4)*	16 (27.6)*
Tobacco use, n (%)	18 (10.5)	7 (15.2)	11 (12.6)
Alcohol use, n (%)	6 (3.5)	3 (6.5)	6 (6.9)
PSS, mean (SD)	23.2 (8.0)	24.5 (6.0)	23.9 (6.6)
ACEs, mean (SD)	2.4 (1.9)	2.2 (1.9)	2.0 (1.7)
EDS, mean (SD)	7.1 (5.4)	7.8 (5.1)	6.6 (5.0)
≥10, n (%)	47 (27.5)	18 (39.1)	24 (27.6)

Abbreviations: ACEs, Adverse Childhood Experiences [Questionnaire]; BMI, body mass index; EDS, Edinburgh Depression Scale; PTB, preterm birth; PSS, Perceived Stress Scale; SD, standard deviation; sETB, spontaneous early term birth; sPTB, spontaneous preterm birth.

*p<.05 versus healthy full term

[a]Healthy full term births defined as full term births delivered at or after 39 weeks' gestation, excludes full term births to mothers with pre- and perinatal cardiometabolic complications, including gestational diabetes, preeclampsia or gestational hypertension, and premature rupture of membranes

[b]Among multiparous participants (N=175)

Note. Missingness was between 0% and 2% for participant characteristics across birth outcome groups.

Table 4.2. Average marginal effect of gendered racial stress on spontaneous preterm birth

	Mean (SD)		AME[a]	SE	95% CI	p-value
	Healthy full term (n=171)	sPTB (n=46)				
JHP-RC© Total	94.6 (20.6)	94.6 (24.4)	.0005	.001	-.002, .003	.73
Burden	28.3 (8.1)	27.0 (9.7)	.004	.004	-0.01, .004	.33
Coping	32.7 (9.2)	32.0 (8.7)	-.002	.003	-.008, .004	.47
Personal history	10.3 (5.4)	10.4 (5.9)	.007	.006	-.003, .018	.18
Racism	13.0 (4.5)	14.6 (5.3)	**.013**	.006	.002, .024	.02
Work	10.3 (4.1)	10.6 (4.6)	.003	.006	-.009, .015	.59

Abbreviations: AME, average marginal effect; CI, confidence interval; JHP-RC©, Jackson Hogue Phillips Reduced Common Contextualized Stress Measure; SD, standard deviation; SE, standard error; sPTB, spontaneous preterm birth.

Bolded marginal effects are statistically significant at $p<.05$

[a]Logistic models adjusted for all variables in Table 4.1, except EDS score

Table 4.3. Average marginal effect of JHP-RC© racism on sPTB at selected levels of JHP-RC© racism exposure

Racism Score	AME[a]	SE	95% CI	p-value[b]
5	.009	.002	.004, .013	.0002
10	.011	.004	.003, .020	.007
15	.015	.006	.001, .026	.032
20	.018	.009	-.000, .036	.054

Abbreviations: AME, average marginal effect; CI, confidence interval; JHP-RC©, Jackson Hogue Phillips Reduced Common Contextualized Stress Measure; SE, standard error; sPTB, spontaneous preterm birth.

[a]Logistic models adjusted for all variables in Table 4.1, except EDS score.

[b]There were no significant differences in AMEs across racism scores ($p>.05$; data not shown).

Table 4.4. Average marginal effect of gendered racial stress on spontaneous early term birth

	Mean (SD)		AME[a]	SE	95% CI	p-value
	Healthy full term (n=171)	sETB (n=87)				
JHP-RC© Total	94.6 (20.6)	95.0 (19.7)	.0002	.002	-.003, .004	.90
Burden	28.3 (8.1)	27.8 (8.1)	.006	.004	-.015, .002	.15
Coping	32.7 (9.2)	33.4 (9.5)	.004	.003	-.003, .01	.27
Personal history	10.3 (5.4)	10.5 (5.2)	.007	.007	-.006, .02	.30
Racism	13.0 (4.5)	13.0 (4.4)	-.004	.007	-.017, .010	.62
Work	10.3 (4.1)	10.3 (4.0)	-.001	.007	-.016, .014	.88

Abbreviations: AME, average marginal effect; CI, confidence interval; JHP-RC©, Jackson Hogue Phillips Reduced Common Contextualized Stress Measure; SD, standard deviation; SE, standard error; sETB, spontaneous early term birth.

[a]Logistic models adjusted for all variables in Table 4.1, except EDS score

Table 4.5 Average marginal effect of JHP-RC© racism on sPTB within tertiles of age

Age Tertile	AME [a]	SE	95% CI	p-value	p-value for difference with…		
					18-21	22-27	28-40
18-21	0.011	0.01	-0.01, 0.03	.33	--	0.71	0.71
22-27	0.006	0.01	-0.01, 0.02	.55	0.71	--	0.35
28-40	0.015	0.006	0.003, 0.03	.02	0.71	0.35	--

Abbreviations: AME, average marginal effect; CI, confidence interval; JHP-RC©, Jackson Hogue Phillips Reduced Common Contextualized Stress Measure; SE, standard error; sPTB, spontaneous preterm birth.

[a]Logistic models adjusted for all variables in Table 4.1, except EDS score

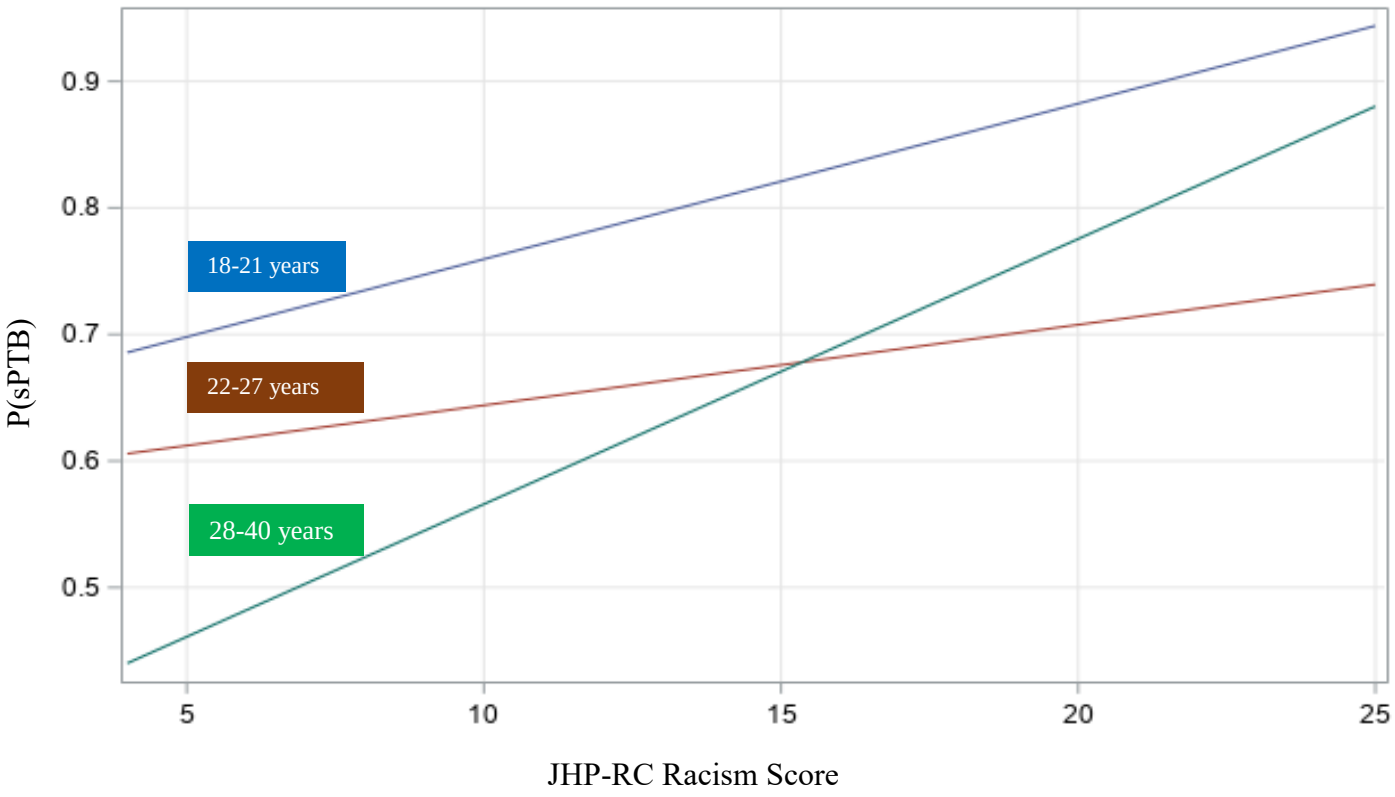


Figure 4.1. Effect plot illustrating effect measure modification by maternal age (tertiles) of the JHP-RC racism & spontaneous preterm birth relationship. The steepest slope exists among the oldest women (shown by the green line).

Table 4.6. Mediation of the JHP-RC© Racism-sPTB association by depressive symptoms

	Estimate	SE	95% CI	p-value
Total Effect	.013	.006	0.002, 0.03	.024
Controlled Direct Effect	.013	.006	0.001, 0.03	.029
Natural Direct Effect	.013	.006	0.001, 0.03	.028
Natural Indirect Effect	.0005	.001	-0.002, 0.003	.74
Percentage Mediated	3.3	9.8	-16.0, 22.5	.74

Abbreviations: CI, confidence interval; JHP-RC©, Jackson Hogue Phillips Reduced Common Contextualized Stress Measure; SE, standard error; sPTB, spontaneous preterm birth.

Appendix

Table A4.1. Associations of gendered racial stress and spontaneous preterm birth

	Mean (SD)		Crude OR	95% CI	Adjusted OR[a]	95% CI
	Healthy full term (n=171)	sPTB (n=46)				
JHP-RC© Total	94.6 (20.6)	94.6 (24.4)	1.00	.99, 1.02	1.00	.98, 1.03
Burden	28.3 (8.1)	27.0 (9.7)	.98	.94, 1.02	.97	.92, 1.03
Coping	32.7 (9.2)	32.0 (8.7)	.99	.96, 1.03	.98	.94, 1.03
Personal history	10.3 (5.4)	10.4 (5.9)	1.00	.95, 1.07	1.06	.97, 1.15
Racism	13.0 (4.5)	14.6 (5.3)	**1.07**	1.001, 1.15	**1.11**	1.02, 1.21
Work	10.3 (4.1)	10.6 (4.6)	1.02	.94, 1.09	1.02	.93, 1.13

Abbreviations: sPTB, spontaneous preterm birth; JHP-RC©, Jackson Hogue Phillips Reduced Common Contextualized Stress Measure; OR, odds ratio; CI, confidence interval

Bolded ORs are statistically significant at p<.05

[a]Adjusted for all variables in Table 4.1, except EDS score

Table A4.2 Associations of gendered racial stress and spontaneous early term birth

	Mean (SD)		Crude OR	95% CI	Adjusted OR[a]	95% CI
	Healthy full term (n=171)	sETB (n=87)				
JHP-RC© Total	94.6 (20.6)	95.0 (19.7)	1.00	.99, 1.01	1.00	.98, 1.02
Burden	28.3 (8.1)	27.8 (8.1)	.99	.96, 1.02	.97	.92, 1.01
Coping	32.7 (9.2)	33.4 (9.5)	1.00	.98, 1.04	1.02	.99, 1.06
Personal history	10.3 (5.4)	10.5 (5.2)	1.01	.96, 1.06	1.04	.97, 1.11
Racism	13.0 (4.5)	13.0 (4.4)	1.00	.94, 1.06	.98	.91, 1.06
Work	10.3 (4.1)	10.3 (4.0)	1.00	.94, 1.07	.99	.92, 1.07

Abbreviations: sETB, early preterm birth; JHP-RC©, Jackson Hogue Phillips Reduced Common Contextualized Stress Measure; OR, odds ratio; CI, confidence interval

[a]Adjusted for all variables in Table 4.1, except EDS score

Table A4.3. Association of JHP-RC© racism subscale and spontaneous preterm birth

	Estimate	SE	p-value	Adjusted OR	95% CI
Intercept	-2.6	1.2	.03	--	--
JHP-RC© Racism	.10	.05	.02	**1.11**	1.02, 1.21
Age	-.01	.05	.80	.99	.89, 1.10
Education[a]	-.36	.50	.47	.69	.26, 1.85
Insurance	-1.0	.75	.17	.36	.08, 1.55
Relationship status	.22	.44	.60	1.26	.53, 2.97
Prepregnancy BMI[a]	-.10	.42	.81	.90	.40, 2.05
Primiparity	-.13	.48	.79	.87	.34, 2.23
Previous PTB	3.2	.81	<.0001	**25.2**	5.1, 125.3
Tobacco Use	.64	.69	.36	1.9	.49, 7.3
Alcohol Use	.23	1.1	.84	1.26	.14, 11.4
PSS	.01	.03	.67	1.01	.95, 1.08
SLEI	-.11	.11	.30	.90	.73, 1.10
ACEs	-.04	.14	.76	.96	.73, 1.26

Abbreviations: BMI, body mass index; PTB, preterm birth; sPTB, spontaneous preterm birth; sETB, spontaneous early term birth; PSS, Perceived Stress Scale; SLEI, Stressful Life Events Index; ACEs, Adverse Childhood Experiences [Questionnaire]; SE, standard error

Bolded adjusted ORs are statistically significant at p<.05

[a]Dichotomized (high school or less; some college or more) to improve model parsimony given no meaningful differences when 4 categories versus 2 were used.

Table A4.4. Mediation of the JHP-RC© Racism-sPTB association by depression

	OR estimate	95% CI	p-value
Total effect	1.11	1.001, 1.21	0.03
Natural indirect effect	1.00	0.986, 1.01	0.82
Natural & controlled direct effect	1.11	1.001, 1.22	0.04

Abbreviations: JHP-RC©, Jackson Hogue Phillips Reduced Common Contextualized Stress Measure; OR, odds ratio; CI, confidence interval; sPTB, spontaneous preterm birth

Chapter 5: The association of gendered racial stress with C-reactive protein among Black women in early pregnancy

Introduction

C-reactive protein (CRP) is an extensively studied marker of inflammation that has been linked to experiences of discrimination, preterm birth, cardiovascular conditions, and a host of health factors over the lifecourse, (Danese et al., 2007; Khera et al., 2005; T. T. Lewis, Aiello, Leurgans, Kelly, & Barnes, 2010; Ridker, 2003; Sharpley et al., 2019; Szalai et al., 1997; Visser et al., 1999). CRP is an inflammatory protein induced in response to stress (Sproston & Ashworth, 2018). Exposure to stressors triggers activation of the hypothalamic-pituitary-adrenal (HPA) axis, which, in turn triggers a cascade of hormonal reactions resulting in increased production of glucocorticoids and greater levels of proinflammatory cytokines (Miller, Maletic, & Raison, 2009), which regulate CRP levels (Sproston & Ashworth, 2018). Although glucocorticoids exert negative feedback on inflammatory processes under acute stress conditions, the inhibitory effects of glucocorticoids on inflammation are reduced in the context of chronic or toxic stress (Miller et al., 2009). Thus, CRP has been a focus in studies of the inflammatory stress response.

The weathering hypothesis states that exposure to chronic stress weathers this physiological response to put Black Americans—who experience greater social adversity—at greater health risk (A. T. Geronimus, 1992). Black women face extra adversity due to their race and gender, known as gendered racism (Essed, 1991). This hybrid form of oppression may place Black women in double jeopardy for inflammatory dysregulation across inter-related physiologic, psychosocial, and behavioral pathways (Curry Owens & Jackson, 2015; Holzman et al., 2009; Thomas et al., 2008). These pathways are especially important in pregnancy, which involves considerable physiological adaptation to support the growing fetus and mother. During normal gestation, the maternal HPA axis becomes progressively activated, and a network of hormones and inflammatory mediators ultimately determine the timing of parturition. A disruption in this network is a proposed mechanism linking chronic stress to inflammation and preterm birth (Cassidy-Bushrow et al., 2012; L. M. Christian, 2015; Hogue & Bremner, 2005; M. R. Kramer et al., 2011; Latendresse, 2009; Schetter, 2009; Wadhwa et al., 2001).

Levels of maternal CRP throughout pregnancy have also been positively associated with or even predictive of preterm birth in a number of studies (Catov et al., 2007; Han et al., 2011; Pitiphat et al., 2005; Shahshahan & Rasouli, 2014; Vogel et al., 2005), some of which suggest a dose–response relationship between CRP levels and preterm delivery risk (Catov et al., 2007; Pitiphat et al., 2005). Additionally, biomarker research in our group highlights that well-functioning glucocorticoid feedback, a pregnant woman's ability to regulate inflammation (including CRP production) is limited, which may contribute to adverse birth outcomes (E. J. Corwin et al., 2013). Conversely, some research suggests an unclear or weak link between CRP and preterm delivery (Wei et al., 2010), especially among women with higher body mass index (BMI) (Bullen et al., 2013). In BMI-stratified models, Bullen et al. observed a positive association between CRP and spontaneous PTB among women with pre-pregnancy BMI >25, but not among women with lower BMIs. Taken together, these findings suggest CRP may be implicated in the racial disparity in preterm birth among Black women. Indeed, dysregulated inflammation and its reproductive health consequences may reflect weathering over the lifecourse, in addition to pregnancy-specific mechanisms.

The connection between experiences with racial discrimination and inflammation has been investigated in several studies, with mixed results (Brody, Yu, Miller, & Chen, 2015; Cunningham et al., 2012; T. T. Lewis et al., 2010). Contradictory findings may reflect the distinct measures of racial discrimination used across study populations (Yin Paradies, 2006; Williams & Mohammed, 2009), which leave the etiology of biosocial processes linking racism and discrimination to Black maternal health unclear. Given the intersectional nature of gendered racism, scholars suggest that researchers should utilize frameworks and measures that explore experiences of racism and sexism together, in an effort to contextualize racial disparities in adverse health outcomes.

There are no studies to our knowledge that explore of the role of Black maternal inflammation in relation to experiences with intersectional race- and gender- discrimination. The present study builds upon the literature on discrimination and CRP (Goosby et al., 2018) and is one of the first to examine the

association of gendered racial stress and inflammation, and the moderating role of BMI, among a socioeconomically diverse group of pregnant Black women.

Methods

Participants

This cross-sectional study uses first trimester prenatal data from the participants enrolled in the prospective Biobehavioral Determinants of the Microbiome and Preterm Birth in Black Women study (n=485), which aimed to provide a biopsychosocial understanding of within-race risk for adverse birth outcomes among a socioeconomically diverse sample of Black women (Elizabeth J Corwin et al., 2017). At enrollment, participants were receiving prenatal care at either Grady Memorial Hospital or Emory University Hospital Midtown—two Atlanta-area facilities (public and private, respectively) that see approximately 10% of Georgia's singleton live births to Black women. Eligible women were between 18-40 years of age, self-identified as African-American (i.e., US-born and of African-American or Black race), were carrying a singleton pregnancy between 8-14 weeks' gestation, and had no reported chronic medical conditions or conditions requiring long-term prescription management. The first-trimester study visit included collection of blood for bioassays, and completion of demographic, psychosocial, and other health questionnaires via self-report. The Emory University Institutional Review Board granted ethical approval for all data collection, and all participants provided written informed consent. Women were excluded from these analyses due to missing data on gendered racial stress (n=36) or C-reactive protein (n=30), or due to ineligible birth outcomes (n=44; e.g. gestational age less than 20 weeks), leaving an analytic sample of 375. While not explicitly examined here, birth outcomes are relevant to the broader context in which our specific research question is situated (see directed acyclic graph in Figure 2.1)

Measures

C-reactive protein

Serum high-sensitivity CRP levels (mg/L) were measured from venous blood samples using a solid-phase sandwich enzyme-linked immunosorbent assay (ELISA) kit (R&D Systems, Minneapolis, MN) with

sensitivity to 0.022 ng/mL. The range of this assay is 0.8-5.0 ng/mL, with inter- and intra-assay variability of approximately 4% and 6%, respectively.

Gendered racial stress

To operationalize gendered racial stress, we use continuous scores from the 39-item Jackson Hogue Phillips Contextualized Stress Measure (JHP), which we call the JHP-Reduced Common (JHP-RC©; see Table 2.2). This reduced scale includes items from the original 68-item JHP (F. M. Jackson et al., 2005; F. M. Jackson et al., 2014) that were found to be most commonly experienced by Black women who worked at one of two public hospitals and were enrolled in a federally funded worksite fitness study (Freeman, 2009). The JHP-RC© assesses specific exposure to chronic racial and gendered stress among Black women, and consists of four subscales measuring stressors (burden, personal history, racism, and work) and one measuring stress mediators (coping), with 5-level Likert scoring from greatest agreement to greatest disagreement. The burden subscale includes 10 items that measure gendered role strain, and distress due to inadequate resources to meet demands. It includes 3 of the 4 items from the original JHP Scale referred to as "stress states" (F. M. Jackson et al., 2005; F. M. Jackson et al., 2014). The personal history subscale includes 5 items that assess experiences with emotional or physical abuse, and family or partner substance abuse. The 5 racism items capture racial stereotypes, White privilege, and the perceived impact of racism on children's lives. The 5-item work subscale assesses racism and sexism in the workplace. The 14 coping items measure coping resources (including support from and belonging to one's community) and capacity for coping based on race and gender. To calculate total and subscale JHP-RC© scores, we coded items 1-5 and reverse coded items, as necessary, so higher values for each item indicated greater gendered racial stress. In our sample, total JHP-RC© score ranges from 47 to 159. Ranges for the subscales are: burden=10-50, coping=16-66, personal history=3-25, racism=4-25, and work=2-25. Cronbach's alpha coefficient for the full JHP-RC© was .80 in the present study. The subscales had the following alpha coefficients: burden=.77, coping=.83, personal history=.77, racism=.81, and work=.77. These values indicate acceptable to high internal reliability consistency.

Covariates

Potential confounders were selected *a priori* based on existing literature regarding factors related to racial stress and/or CRP (Black, Johnson, & VanHoose, 2015; Frazier et al., 2018; A. T. Geronimus et al., 2006; Goosby et al., 2018; F. M. Jackson et al., 2012; T. T. Lewis et al., 2010; Nazmi & Victora, 2007; Pitiphat et al., 2005; Van Dijk et al., 2013; Van Dyke et al., 2017). They included:

- Age in years.
- Educational attainment, which refers to the highest level of education completed. Participants self-reported whether they had completed less than high school, high school, some college, or were a college graduate or more. We conceptualized educational attainment as a proxy for socioeconomic status (Van Dyke et al., 2017), and in adjusted regression models, we dichotomized educational attainment as higher educated (college graduate or more) or lower educated (less than high school, high school, or some college).
- Tobacco use in the last month.
- Prepregnancy body mass index (BMI; kg/m^2): to assess interaction and confounding by BMI, we dichotomize our sample as BMI <25 or BMI >=25, consistent with prior literature (Bullen et al., 2013).
- Parity: dichotomized as primiparous or multiparous (i.e. at least one previous live birth).
- Previous preterm birth, which was assessed only among multiparous women, and dichotomized as no history of preterm birth or at least one prior preterm.
- Symptoms of depression: The Edinburgh Depression Scale (EDS) is a 10-item self-report questionnaire ascertaining symptoms of depression in the last 7 days (Cox et al., 1987). While originally developed for use in the postnatal period, the scale has been used and validated in studies of pregnant women and shows high sensitivity and specificity for the detection of prenatal major depressive disorder (Bergink et al., 2011; Grobman et al., 2016; Kozinszky & Dudas, 2015). The EDS maintains high internal consistency reliability in studies of Black pregnant

populations (Tandon et al., 2012; Yonkers et al., 2001). We summed items, coded 0 to 3, where higher scores indicate greater depressive symptomology. In our sample, scores range from 0 to 25. Using generally recommended threshold, an EDS score ≥10 indicated the presence of depressive symptomology (Cox et al., 1987). In this sample, Cronbach's alpha for the EDS was .84.

- Perceived stress: Cohen's 14-item Perceived Stress Scale (PSS) assesses "the degree to which individuals appraise situations in their lives as stressful" over the last month (Sheldon Cohen et al., 1983). We summed items, coded 0 to 4, where higher values indicate greater perceived stress. In our sample, scores ranged from 0 to 45. The PSS is among the most widely used instruments of perceived stress, and demonstrates acceptable internal consistency in this sample (Cronbach's alpha=.72).

Statistical Analysis

We log-transformed CRP because univariate analyses and the distribution of residuals from multivariable regression models revealed that CRP was right skewed. We performed preliminary testing to check for the conditions required for our statistical analyses (e.g., normally distributed dependent variable and absence of multicollinearity); results indicated that analytic assumptions were met. The arithmetic mean of log CRP was back-transformed (i.e., exponentiated) to yield the geometric mean in CRP original units (mg/L) for descriptive statistics. Transformed CRP (henceforth called CRP) was used for all estimates in correlation analyses and regression models. Relationships of CRP with continuous JHP-RC© variables were examined using Pearson correlation coefficients. We used multivariable linear regression to model the relationship of CRP with total JHP-RC© and subscale scores, adjusted for potential confounders. In order to examine interaction effects of education (given social patterning of gendered racial stress observed in *Aim 1*) and BMI, we initially included the interaction terms of interest (i.e. JHP-RC© score*BMI) in adjusted multivariable linear regression models. All models were run separately for total

JHP-RC© score and each subscale. We used SAS version 9.4 for all analyses, and considered $p<.05$ statistically significant.

Results

We summarize the demographic, behavioral, clinical, and psychosocial characteristics of women in the sample in **Table 5.1**. Mean CRP in early pregnancy was 5.2 mg/L (standard error: 2.3). Mean JHP-RC© total score was 94.8 (20.4).

Correlation analyses (**Table 5.2**) revealed small, non-significant relationship of CRP with all measured components of gendered racial stress. We found no statistically significant interaction effects of prepregnancy BMI or educational attainment, and thus, report non-stratified multivariable regression results in **Table 5.3**. Linear models confirmed small, non-significant associations of CRP with total JHP-RC© score, controlling for all potential confounders. Associations of CRP with JHP-RC© subscales were similarly small and non-significant (see detailed results in this chapter's appendix). The proportion of variance in the dependent variable that a model's parameters explain is quantified by R^2. In **Table 5.4**, non-significant R^2 values less than 1% further demonstrate a weak association of CRP with gendered racial stress in this sample, irrespective of control for confounders.

Discussion

Previous studies confirm that CRP is elevated in early pregnancy (G. Sacks, Seyani, Lavery, & Trew, 2004), particularly among Black women (Picklesimer et al., 2008) who may face additional exposure to chronic stress due to their intersecting racial and gender identities (Essed, 1991). In our cohort, we demonstrate that clinically elevated CRP is very prevalent in this population pregnant Black women. However, we could not confirm an association between exposure to gendered racial stress and elevated CRP.

Among the socioeconomically diverse women in our sample, mean first trimester CRP was 5.2 mg/L. Median CRP was similar (5.3 mg/L) with an interquartile range from 2.1 mg/L to 13.9 mg/L. In the literature, CRP values above 3.0 mg/L suggest sub-clinical inflammation. CRP values surpass this

threshold for the majority of our study sample, and are consistent with some studies of CRP in Black women. In a longitudinal analysis aimed at characterizing CRP in a racially diverse group of pregnant women, median CRP for pregnant Black women with gestational ages < 14 weeks (as in our sample) was 7.68 mg/L, and was significantly above median CRP among pregnant White women (2.59 mg/L) (Picklesimer et al., 2008). That study also demonstrated a strong association between elevated serum CRP (i.e. defined as above 15.7 mg/L) and Black race (OR [95%CI]: 2.1 [1.3-3.3]), and socioeconomic status. Additionally, unlike CRP for White women which showed a slight upward trend with increasing gestational age, CRP for Black women remained high and relatively flat throughout pregnancy (Picklesimer et al., 2008).

It is surprising that we did not observe an association between gendered racial stress and CRP overall, or across levels of educational attainment or BMI. However, if Black women's CRP is elevated homogeneously (e.g. irrespective of SES) in early pregnancy as a function of both normal inflammatory response to pregnancy and lifecourse psychosocial stress, this could also explain our null associations with gendered racial stress. No previous studies have explored the association of CRP and gendered racism among a within-race cohort of pregnant Black women, which precludes direct comparison of results. However, limited studies of Black and White women in pregnancy suggest that Black women report greater discrimination and have higher CRP levels than White counterparts (Borders et al., 2015). Population-based studies of non-pregnant Black populations further confirm racial disparities in CRP. In those studies, Black participants had significantly higher CRP as compared with White counterparts, including CRP above 3 mg/L (Christina R Evans et al., 2019; Khera et al., 2005); and SES discrimination was associated with elevated CRP levels only among college educated Black adults versus Black adults with less education or White adults at any education level (Van Dyke et al., 2017). While important, study of racial differences in CRP does not clarify within-race variation in CRP, or in associated risk factors, which may be especially relevant to pregnancy outcomes (Borders et al., 2015; Picklesimer et al., 2008; Pitiphat et al., 2005). Indeed, elevated serum CRP levels may result from toxic stress, or weathering of the stress response, caused by cumulative socioeconomic disadvantage (A. T. Geronimus, 1992). Given that

pregnancy can be conceptualized as a biological stressor—adding to risk that has already accumulated earlier in the life course—future investigation is necessary to understand contributions to within-race risk for elevated CRP.

Limitations

Our study is subject to a few limitations. First, the cross-sectional collection of self-reported gendered racial stress data and the collection of venous blood for CRP assays precludes causal interpretations, and obscures the directionality of the association of interest. Future studies should assess the longitudinal trajectory of CRP throughout gestation to improve understanding of correlates of elevated CRP over and above what might be expected during pregnancy. Second, our small sample size limits power to detect main and interactive effects found elsewhere (Sims, Sims, Glover, Smit, & Odden, 2020; Van Dyke et al., 2017). Finally, CRP represents just one biomarker of stress and inflammation with which racial stress and pregnancy outcomes may be associated (Clarke et al., 2020; Szarka, Rigó, Lázár, Bekő, & Molvarec, 2010). Inclusion of related cytokines and chemokines involved in inflammatory processes may more comprehensively depict the relationship between self-reported gendered racial stress and its biological embodiment.

Conclusion

Greater exposure to chronic stress dysregulates neuroendocrine and inflammatory stress mechanisms, which may play a role in racial disparities in preterm birth. We observed elevated CRP (relative to established thresholds) in our cohort of pregnant Black women, but did not detect an association with gendered racial stress. Future studies with larger sample sizes should further investigate the role of gendered racial stress on CRP, given associations of gendered racial stress with spontaneous preterm birth. With additional data to identify differences in social and environmental stressors among pregnancy Black women, we can gain a better etiologic understanding of the complex mechanisms underlying birth outcomes.

Table 5.1. Characteristics of Atlanta-area Black women in the study sample (N=375)

	Mean (SD)	**n (%)**
Age	25.9 (4.8)	
Education		
Less than high school		63 (16.8)
High school		141 (37.6)
Some college		107 (28.5)
College graduate or more		64 (17.1)
Insurance type		
Medicaid		293 (78.1)
Private		82 (21.9)
Tobacco use in last month		49 (13.1)
Prepregnancy BMI		
<18.5		11 (2.9)
18.5-<25		154 (41.1)
25-<30		84 (22.4)
≥30		126 (33.6)
Primiparous		177 (47.2)
History of preterm birth		40 (20.2)
Depressive symptoms[a]		110 (29.3)
Perceived stress[a]	23.6 (7.4)	
CRP (mg/L), geometric mean	5.2 (2.3)	
JHP-RC©	94.8 (20.4)	
Burden	28.2 (8.3)	
Coping	32.6 (9.1)	
Personal History	10.3 (5.3)	
Racism	13.2 (4.5)	
Work	10.4 (4.0)	

Abbreviations: CRP: C-reactive Protein; JHP-RC©: 39-item Jackson Hogue Phillips Reduced Common Contextualized Stress Measure

[a]Variable (n, % missing): PSS (13, 3.5%); EDS (11, 2.9%) There were no missing data for variables without this superscript.

Table 5.2. Correlations (r) of log CRP with gendered racial stress

	r	**p-value**
JHP-RC© Total	.01	.77
Burden	.01	.84
Coping	.04	.50
Personal History	.02	.64
Racism	-.03	.52
Work	-.02	.69

Abbreviations: CRP: C-reactive Protein; JHP-RC©: 39-item Jackson Hogue Phillips Reduced Common Contextualized Stress Measure

Note. Pearson coefficients are reported.; Spearman coefficient s were not meaningfully or statistically different

Table 5.3. Linear regression of CRP on total gendered racial stress

Model type	**Model parameters**	**Model p-value**	**Parameter estimate (SE)**	**Parameter p-value**
Unadjusted	JHP-RC© Total	**.77**	.00 (.003)	**.77**
Adjusted	JHP-RC© Total	.23	.002 (.004)	.51
	Age		-.008 (.02)	.62
	College education		.002 (.22)	.99
	Private insurance		-.14 (.19)	.43
	Multiparity		-.00 (.15)	.99
	Previous preterm birth		.09 (.23)	.70
	Tobacco use		-.35 (.20)	.08
	BMI		.27 (.13)	.19
	Depressive symptoms		-.00 (.02)	.98
	Perceived stress		.00 (.01)	.96

Abbreviations: BMI: body mass index; CRP: C-reactive Protein; JHP-RC©: 39-item Jackson Hogue Phillips Reduced Common Contextualized Stress Measure
*p<.05

Table 5.4. Comparison of R^2 across unadjusted and adjusted regression models associating CRP with gendered racial stress

Main effect	**Unadjusted model, R^2**	**Adjusted[a] model, R^2**
Full JHP-RC©	.003	.006
Burden	.003	.005
Coping	.001	.007
Personal history	.002	.006
Racism	.002	.005
Work	.002	.005

Abbreviation: CRP: C-reactive Protein; JHP-RC©: 39-item Jackson Hogue Phillips Reduced Common Contextualized Stress Measure

[a]Adjusted for all covariates in Table 5.3

Appendix

Table A5.1. Linear regression of CRP on JHP-RC © Burden Subscale

Model type	Model parameters	Model p-value	Parameter estimate (SE)	Parameter p-value
Unadjusted	JHP-RC© Burden	.84	.00 (.01)	.84
.72Adjusted	JHP-RC© Burden	.71	.00 (.01)	.71
	Age		-.00 (.02)	.73
	College education		.00 (.22)	.98
	Private insurance		-.13 (.19)	.50
	Multiparity		.02 (.15)	.86
	Previous preterm birth		.06 (.23)	.81
	Tobacco use		.39 (.20)	.06
	BMI		.17 (.13)	.20
	Depressive symptoms		-.00 (.01)	.75
	Perceived stress		.00 (.01)	.63

Abbreviations: BMI: body mass index; CRP: C-reactive Protein; JHP-RC©: 39-item Jackson Hogue Phillips Reduced Common Contextualized Stress Measure

Table A5.2. Linear regression of CRP on JHP-RC Coping Subscale

Model type	**Model parameters**	**Model p-value**	**Parameter estimate (SE)**	**Parameter p-value**
Unadjusted	JHP-RC© Coping	.50	.00 (.01)	.50
Adjusted	JHP-RC© Coping	.65	.01 (.01)	.39
	Age		-.00 (.02)	.73
	College education		.01 (.22)	.97
	Private insurance		-.11 (.19)	.57
	Multiparity		.02 (.15)	.88
	Previous preterm birth		.07 (.23)	.75
	Tobacco use		-.39 (.20)	.06
	BMI		.18 (.13)	.18
	Depressive symptoms		-.00 (.02)	.85
	Perceived stress		.00 (.01)	.74

Abbreviations: BMI: body mass index; CRP: C-reactive Protein; JHP-RC©: 39-item Jackson Hogue Phillips Reduced Common Contextualized Stress Measure

Table A5.3. Linear regression of CRP on JHP-RC Personal History Subscale

Model type	**Model parameters**	**Model p-value**	**Parameter estimate (SE)**	**Parameter p-value**
Unadjusted	JHP-RC© Personal History	.64	.01 (.01)	.64
Adjusted	JHP-RC© Personal History	.69	.01 (.01)	.57
	Age		-.00 (.02)	.72
	College education		-.00 (.22)	.98
	Private insurance		-.12 (.19)	.54
	Multiparity		.03 (.15)	.86
	Previous preterm birth		.05 (.23)	.81
	Tobacco use		-.40 (.20)	.06
	BMI		.17 (.13)	.20
	Depressive symptoms		-.00 (.02)	.77
	Perceived stress		.01 (.01)	.62

Abbreviations: BMI: body mass index; CRP: C-reactive Protein; JHP-RC©: 39-item Jackson Hogue Phillips Reduced Common Contextualized Stress Measure

Table A5.4. Linear regression of CRP on JHP-RC Racism Subscale

Model type	**Model parameters**	**Model p-value**	**Parameter estimate (SE)**	**Parameter p-value**
Unadjusted	JHP-RC© Racism	.52	-.01 (.01)	.52
Adjusted	JHP-RC© Racism	.72	-.00 (.02)	.93
	Age		-.00 (.02)	.76
	College education		.01 (.22)	.96
	Private insurance		-.12 (.20)	.52
	Multiparity		.03 (.15)	.83
	Previous preterm birth		.06 (.23)	.79
	Tobacco use		-.39 (.20)	.06
	BMI		.17 (.13)	.20
	Depressive symptoms		-.00 (.02)	.87
	Perceived stress		.01 (.01)	.58

Abbreviations: BMI: body mass index; CRP: C-reactive Protein; JHP-RC©: 39-item Jackson Hogue Phillips Reduced Common Contextualized Stress Measure

Table A5.5. Linear regression of CRP on JHP-RC Work Subscale

Model type	**Model parameters**	**Model p-value**	**Parameter estimate (SE)**	**Parameter p-value**
Unadjusted	JHP-RC© Work	.69	-.01 (.02)	.69
Adjusted	JHP-RC© Work	.72	-.00 (.02)	.96
	Age		-.01 (.02)	.76
	College education		.01 (.22)	.97
	Private insurance		-.13 (.19)	.50
	Multiparity		.03 (.15)	.83
	Previous preterm birth		.06 (.23)	.79
	Tobacco use		-.39 (.20)	.06
	BMI		.17 (.13)	.20
	Depressive symptoms		-.00 (.02)	.86
	Perceived stress		.01 (.01)	.58

Abbreviations: BMI: body mass index; CRP: C-reactive Protein; JHP-RC©: 39-item Jackson Hogue Phillips Reduced Common Contextualized Stress Measure

Chapter 6: Conclusion

Summary of Findings

Decades of study have demonstrated that Black women of all socioeconomic backgrounds experience worse birth outcomes than women of other race/ethnicity groups. A confluence of theoretical and empirical research has further put forth chronic exposure to both racial and gendered stress as a key explanation for the racial disparity. Though the literature is growing, there are limited studies to date exploring correlates and consequences of gendered racial stress among pregnant Black women. Thus, the pathways through which Black women's contextualized stress shapes maternal and infant health remain poorly understood.

In *Aim 1* (Chapter 3), we assessed whether adversity in the childhood home was related to reports of gendered racial stress among adult Black women in early pregnancy. In this study, we confirmed that adverse childhood events (ACEs) are common, and found that increasing ACEs were associated with increasing JHP-RC scores, particularly for the personal history and burden subscales. These associations persisted after control for educational attainment—a proxy for childhood socioeconomic status—and age. Results from models examining interaction by response to unfair treatment suggest an even stronger association among women who accept unfair treatment as a fact of life and keep it to themselves, as compared to women who try to do something or speak to someone about it. This aim also revealed social patterning in gendered racial stress scores. Highly educated women (i.e. college graduates) reported: (1) most experiences and/or perceptions of racism, White privilege, and the impact of racism of children's lives; and (2) lowest (i.e., more positive) scores on the coping subscale. These findings may be related to Black women's socialization to appear strong in a cultural context that devalues them based on race and gender (Thomas et al., 2008; Cheryl L. Woods-Giscombé, 2010).

Aim 2 (Chapter 4) examined whether gendered racial stress was prospectively associated with categorical birth outcomes, and exploratorily tested the weathering hypothesis by examining interaction of the association by maternal age. Comparing JHP-RC© scores among women who experienced

spontaneous preterm (sPTB) and spontaneous early term birth (sETB), to women with no cardiometabolic conditions who gave birth full term, we found a significant marginal effect of the JHP-RC© racism subscale with sPTB, but not with sETB. This effect appeared moderately stronger among women in the oldest age tertile (i.e., between 28 and 40 years), who, at higher (but not lower) JHP-RC© racism scores, experienced similar sPTB risk as the youngest women in our study (i.e. between 18 and 21 years). However, we found no evidence of mediation by depressive symptoms. Given that most sPTB in our sample occurred between 34 and 36 weeks' gestation, this study adds to emerging literature on risks associated with late preterm birth (Loftin et al., 2010; Raju, 2013).

In *Aim 3* (Chapter 5), we investigated the relation of gendered racial stress to C-reactive protein (CRP). This study adds that CRP is above established threshold for systemic-inflammation in Black women's early pregnancy, which may be a product of expected inflammatory processes in pregnancy already underway and/or dysregulated inflammation related to chronic stress. However, we found null associations between gendered racial stress and CRP overall, and across educational attainment and body mass index groups, using unadjusted and adjusted models.

Taken together, findings across aims offer preliminary evidence that patterns of maternal gendered racial stress begin in childhood and may have long-reaching consequences on birth outcomes

Strengths and Limitations

This dissertation includes numerous strengths. First is its novel examination of gendered racial stress as part of a biopsychosocial lifecourse framework, which contextualizes Black women's lived experiences within the "structural power hierarchies, social processes, and social determinants" that shape opportunities for wellbeing over time (Clare R Evans et al., 2018). Second, while the term gendered racism was coined 20 years ago (Essed, 1991), there remain limited intersectional measures of gendered racial stress. We not only used one such measure, but selected the Jackson Hogue Phillips Contextualized Stress Measure, which was developed with, for, and by a diverse group of Black women's voices (F. M.

Jackson et al., 2005). Third, prospective data collection allowed measurement of gendered racial stress to precede birth outcomes of interest. Thus, our study is among the first to empirically and prospectively assess whether gendered racism may contribute to gestational age.

Our studies are also subject to several limitations. Most notably, sample size across all aims was relatively low, and studies were not powered *a priori* for assessment of interaction. Thus, interactive effects are presented as exploratory findings that warrant additional study in the future. Second, collection of key variables in *Aims 1 and 3* occurred cross-sectionally, which limits our understanding of the directionality of associations of interest. Third, the available data precluded comprehensive study of: (1) additional exposures in childhood (particularly pertinent for *Aim 1*), (2) factors conferring resilience in the face of gendered racism, and (3) longitudinal trajectories of gendered racial stress, and other psychosocial and socioeconomic factors, which may alter health risk throughout pregnancy and over the lifecourse (Curry Owens & Jackson, 2015; Love et al., 2010; Osypuk et al., 2016).

Future Research

These studies provide an initial step toward characterizing the causes and outcomes of gendered racial stress among pregnant Black women. Findings from large population-based studies would best confirm the relationships of gendered racial stress, early life stress, and maternal and infant health (Johnson et al., 2020). Additional investigation could bolster these findings to elucidate whether there is a mediating role of gendered racial stress in the association of ACEs and CRP (Iob, Lacey, & Steptoe, 2020), and in the association of ACEs and adverse birth outcomes (H. G. Kim et al., 2020; Merrick et al., 2017; Joshua P Mersky & ChienTi Plummer Lee, 2019; Scorza et al., 2020). To achieve equitable birth outcomes for Black women, future studies must also examine the multilevel components of gendered racial stress. Robust collection and analysis of these data hold promise to inform necessary improvements in interventions on the clinical, community, and structural levels (F. M. Jackson et al., 2012).

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