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Lasha Shenel Clarke Date

The association between biomarkers and psychosocial measures of stress and discrimination among pregnant African-American women from the metro Atlanta area

By

Lasha Shenel Clarke

Master of Public Health

Global Epidemiology

Carol Hogue, PhD, MPH

Committee Chair

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Lasha Clarke

B.A., Princeton University, 2010

Thesis Committee Chair: Carol Hogue, PhD, MPH

An abstract of

A thesis submitted to the Faculty of the

Rollins School of Public Health of Emory University

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Abstract

The association between biomarkers and psychosocial measures of stress and discrimination among pregnant African-American women from the metro Atlanta area

By Lasha Shenel Clarke

Objective: African-American (AA) women are at twice the risk of preterm birth (PTB) as compared to white women, and also face unique, intersectional stressors related to their gender and race. Research on the distinct stressors AA women face reveals that chronic stress, often measured during pregnancy, is a risk factor of growing interest in the etiology of PTB. This study aims to explore the associations between biomarkers and psychosocial measures of lifetime stress in a socioeconomically diverse cohort of pregnant, AA women.

Study Design and Setting: This study is a cross-sectional analysis of 144 women enrolled in the ongoing Microbiome Preterm Birth study, a prospective study of pregnant AA women receiving prenatal care at Emory University Midtown Hospital or Grady Hospital in Atlanta, GA. All included women had complete psychosocial exposure (the Jackson Hogue Phillips Contextualized Stress Measure [JHP] & the Experiences of Discrimination Scale [EOD]) and biomarker outcome ($DexIC_{50}$, Dex Top, & Dex Bottom) data. Bivariate and multivariable linear regression analyses were performed.

Results: There was no evidence of a statistically significant relationship between the JHP or EOD and the biomarkers of chronic stress. Further, there was no evidence of effect modification of by depression.

Conclusions: More work is needed to understand the degree to which psychosocial measures of chronic stress are empirically associated with chronic stress biomarkers in pregnant AA women.

Keywords: birth outcomes; discrimination; race; stress

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Abbreviations

AA: African-American

ACTH: adrenocorticotropic hormone

BV: bacterial vaginosis

CRH: corticotrophin-releasing hormone

DST: dexamethasone suppression test

EDS: Edinburgh Depression Scale

EOD: Experiences of Discrimination Scale

GR: glucocorticoid resistance

HPA axis: hypothalamic-pituitary-adrenal axis

JHP: Jackson Hogue Phillips Contextualized Stress Measure

LBW: low birthweight

PNS: prenatal care

PSS: Perceived Stress Scale

PTB: preterm birth

RALES: Racism and Life Experience Scales

SLEI: Stressful Life Events Index

SGA: small for gestational age

VLBW: very low birthweight

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1. Introduction

1.1 Background & Rationale

Although medical advances over recent decades have reduced the incidence of maternal and infant mortality in the United States, racial disparities in preterm birth (PTB) remain blatant and unresolved. African-American (AA) women have disproportionately higher PTB rates when compared to their white counterparts. One explanation for this phenomenon is that AA women's health is compromised because of the relatively higher amounts of stress they experience. Stress—a process in which "environmental demands tax or exceed the adaptive capacity of an organism, resulting in psychological or biological changes that may place persons at risk for disease" [1]—has been linked to a wide range of negative health outcomes in AA women, including cardiovascular disease, mental illness, and of course, poor birth outcomes [2-5]. These findings suggest that AA communities are at increased risk of morbidity and mortality across generations and all throughout the life course [4].

It is believed that the multiple identities and roles that AA women play, as compared to their white counterparts, present more opportunities for exposure to stress [6]. They are not only impacted by the complex roles imposed on them as women (i.e. nurturer, breadwinner, sexual partner), but also have the potential to experience race-based discrimination and racism. Therefore, the intersection of gender and racial stress, or gendered racism, is thought to be one of the primary culprits in the disproportionate exposure to stress among AA women [6].

Research on the unique gender- and race-related stressors AA women face reveals that chronic and acute stress, often measured during pregnancy, are risk factors of

growing interest in the etiology of PTB. Still, preconceptional stress remains a poorly characterized risk factor for understanding and reducing racial disparities in PTB. The lingering racial disparity in PTB calls for further investigation into the impacts of stress on pregnancy and birth in AA populations. Further, the singularity of the stressors faced by AA women calls for improved understanding of how the various stress measures used—both biological and psychosocial—can be interpreted independently and jointly.

Though the body of literature grows, few studies have investigated the links between psychosocial measures of discrimination and biomarkers of chronic stress in pregnant AA women directly. Research that advances the understanding of biological and social processes together promises possibilities for improved intervention to reduce the preterm birth (PTB) rate. Given the complexity inherent in race and racism, it is unlikely that either psychosocial or biomarker data tell the full story independently.

1.2 Objectives

The present cross-sectional analysis explores the associations between biomarkers and psychosocial measures of stress in a socioeconomically diverse cohort of pregnant, AA women. The goals are to:

1. Determine the prevalence of perceived, lifetime racial discrimination, as measured by two psychosocial scales, the Jackson Hogue Phillips (JHP) Contextualized Stress Measure and the Experiences of Discrimination scale (EOD), among the study population;
2. Determine the prevalence of glucocorticoid resistance, an biological indicator of chronic stress measured via the $DexIC_{50}$, among the study population;

3. Assess whether there is an association between the $DexIC_{50}$ and the JHP, or between the $DexIC_{50}$ and the EOD; and
4. Assess any modification of those associations by depressive symptomology.

1.3 Assumptions

This study assumes that participants completing the JHP and EOD scales have reported accurately, and without bias. Furthermore, this study assumes that participants were sufficiently literate to correctly interpret and respond to each measure's items. Finally, this study assumes that blood samples, from which the stress biomarker data were obtained, were collected and assayed accurately.

2. Literature Review

2.1 Background

Within the United States, PTB, defined as birth before 37 completed weeks' gestation, occurs at disproportionately high rates among AA women. Of the known risk markers for PTB, among the strongest is self-reported AA race. The PTB rate is approximately 18% among AA women, and 10.5% to 11.5% among non-Hispanic white, Asian, and Hispanic women [7]. There also exists a strong link between PTB and low birthweight (LBW, <2500g), and infants born small for gestational age (SGA, weight under the 10^{th} percentile for the gestational age) [7]. While PTB has been shown to be more common among women of lower socioeconomic status (SES) [7], racial disparities persist even after adjustment for indicators of SES, like income and level of education [7]. This suggests that there are risk factors yet to be discovered, and that there likely exists potential to advance the methodology guiding exposure and outcome measurement in social epidemiologic research [8].

In spite of the long-standing historical and contemporary existence of racism in American society and culture, the body of literature explicitly examining its effects on AAs has only begun to grow steadily in the recent decades. Numerous authors have noted that racism and discrimination are significant stressors for many AAs, and that racism may play a role in the higher rates of morbidity and mortality that affect AA populations from birth [9, 10]. Specifically, a growing body of research suggests that chronic exposure to racism over the maternal life course may help to explain racial disparities in PTB [11-14].

Racism, as defined by Camara Jones, is a system of oppression that structures opportunity and assigns value based on one's perceived race [15]. It can be institutionalized, personally mediated, and/or internalized [15, 16]. The stress resulting from experiences of perceived racism, across any combination of those three domains, could trigger negative birth outcomes for AA women through a variety of mediators. Namely, racism as a psychosocial stressor could increase the risk of PTB through biological pathways that dysregulate the neuroendocrine immune response to increase and maintain inflammation over the short- and long-term [7]. A growing body of literature supports the hypothesis that racism is not only a perceived stressor, dependent on one's subjective societal experience, but also an embodied experience with objective, biological underpinnings and consequences [17]. That is, the establishment and proliferation of race-related stress may be a biological and social process.

To provide further evidence for a framework that integrates social and biological processes in conceptualizing racism's impact on stress and health, we need a clear understanding of how psychosocial measures of perceived race-related stress over the

lifetime are associated with established biomarkers of chronic stress. Commonly in epidemiologic research, perceived stress is measured via self-report. Survey methods take into consideration that different individuals and groups can experience the same stressful event at the same frequency, but appraise the event variably. While the subjectivity inherent in self-report methods can complicate analysis and interpretation, their data enrich investigations into complex social factors [8, 18, 19].

Many studies with a biomedical or biosocial focus also collect biospecimens, like blood samples, to measure known biomarkers of chronic stress [20-22]. These include glucocorticoids, hormones released by the body's hypothalamic-pituitary-adrenal (HPA) axis. Though studies have suggested that stress and other downstream effects of racism have individual roles in the etiology of PTB [3, 23, 24], it is not yet clear whether, and the degree to which, psychosocial measures of chronic stress are empirically associated with chronic stress biomarkers in pregnant AA women.

This literature review will begin by examining the negative impact stress has on health, especially as related to poor birth outcomes experienced by AA women. It will then discuss the evidence establishing racism as a stressor for AAs, focusing in particular on exploring the association between exposure to lifetime experiences of racism and PTB. Next, this review will discuss selected psychosocial measures of racism and discrimination, detailing their development and application to a study of pregnant AA women. Then, this review will introduce evidence for glucocorticoid resistance (GR) as a biomarker for chronic stress. Finally, this review will underscore the need for a clearer understanding of the association between psychosocial measures of chronic, race-related stress and biomarkers of chronic stress.

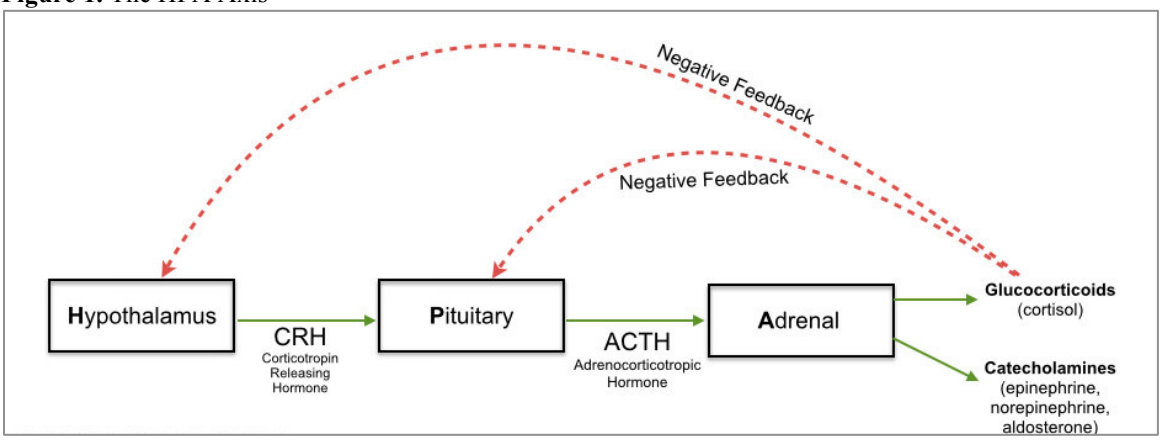
2.2 Stress & Preterm Birth

According to Cohen et al., stress is a process in which "environmental demands tax or exceed the adaptive capacity of an organism, resulting in psychological or biological changes that may place persons at risk for disease" [1]. Stress is generally divided into two time-based categories: acute and chronic. Acute stress is short-term, and has been shown to have some benefits when an individual is faced with a demand or threat [25]. Chronic stress, however, is enduring, and exerts detrimental impacts on the body over time. Geronimus' [26] well-cited weathering hypothesis posits that chronic stressors experienced over much of one's life can result in cumulative wear and tear on the body's physiologic stress response systems. This concept of cumulative wear and tear on the body's systems owing to repeated adaptation to stressors is also known as allostatic load [27].

One such mechanism by which the body responds to and is acted on by a stressor is via the neuroendrocrine system's hypothalamic-pituitary-adrenal (HPA) axis (see Figure 1 below). In the presence of a stressor, the hypothalamus releases corticotrophin-releasing hormone (CRH). CRH then stimulates the secretion of adrenocorticotropic hormone (ACTH) from the pituitary gland; and ACTH serves as an agonist for the production and secretion of glucocorticoids from the adrenal glands. Glucocorticoids, of which cortisol is arguably the best known, have been of particular importance in the recent literature. These hormones play a pivotal role in the HPA axis' feedback mechanism in that they can inhibit the activation and function of cells involved in immune response [25]. Short-term stress promotes the eventual shut-off of HPA activation, and the body's return to homeostasis [25]. Chronic stress, on the other hand, is

associated with hypercortisolemia (the overproduction of cortisol), and the subsequent reduced function of the glucocorticoid receptors. Together, these processes keep the HPA axis in an activated state. The development of this glucocorticoid receptor resistance (GR) in immune cells, which are linked to neuroendrocrine system function, prevents response to negative hormonal feedback, and may lead to uncontrolled inflammation [28]. Evidence of GR, which will be covered in greater detail later, is part of the mounting support linking chronic stress to poor health [20].

Figure 1. The HPA Axis



The question of whether stress impacts preterm birth continues to be of scientific interest. Taken together, findings accumulated over the last few decades, though equivocal, suggest the affirmative. Still, the task of identifying distinct causal mechanisms remains. Notably, a focus on fetal origins of adult disease in recent decades, has produced evidence linking PTB to increased mortality and morbidity later in life [29], thereby strengthening the rationale for further investigation into PTB's etiology, and for longitudinal research looking at the entire life course, and across generations.

Identification of numerous determinants of PTB has pointed to race as a strong marker. The infant mortality ratio for AA infants, which is driven by disparities in PTB, is over twice that of white infants [30]. One hypothesized intermediary between race and

PTB is maternal psychosocial stress [31]. Chronic exposure to psychosocial stressors is said to prematurely age AA women, thereby shifting age-related risk of PTB to that population's peak reproductive years [27, 31].

Evidence for suboptimal HPA axis functioning in pregnant AA women comes from studies that have found elevated maternal serum CRH among those mothers who delivered prior to 37 weeks' gestation [32, 33]. Given that in women who delivered preterm compared with women who delivered full term infants, both absolute and trajectory CRH levels increased as early as the second trimester, CRH may act as an internal clock that can turn on parturition [31, 34]. While CRH is negatively associated with length of gestation across racial groups [35], this association is stronger in black women as compared to white women [36]. In a study linking psychosocial measures of stress with biomarkers, maternal perceptions of stress mid second trimester explained a significant proportion of the difference in CRH in early third trimester [37].

Further, Thayer and Kuzawa [38] recently found that, after controlling for ethnicity and other relevant covariates, women with lower SES had significantly higher evening cortisol, though morning cortisol levels were similar across SES groups. This result is consistent with literature that shows higher evening cortisol levels are associated with chronic stress in pregnancy [39], PTB [40], and restricted fetal growth [41], and suggests that maternal stress physiology is sensitive to SES. While morning cortisol was not significantly related to SES in the relatively small study sample (n = 55), there was a trend toward higher morning cortisol among women with lower SES. Future studies with greater statistical power are required to further elucidate the association between AM cortisol measures and stress. One hypothesis for this phenomenon could be that, for AA

women who often report experiencing racism at the workplace, psychosocial stress builds over the course of the day due to job strain [Collins et al., 2004]. Thus, interracial disparities in cortisol levels may not become detectable until evening measurements are taken.

The evidence makes it increasingly clear that the causes of preterm birth and its racial bases are complex. Given that literature across fields supports a potential causal link between perceived psychosocial stress and the racial disparity in preterm birth, it is worth investigating racism as a psychosocial stressor experienced uniquely by AA women.

2.3 Racism as a Stressor

Investigating racism as a stressor for AAs is warranted for many reasons. First, if exposure to racism is perceived as stressful, it may have negative biopsychosocial consequences. Second, differential exposure (based on sociodemographic factors, for instance) to racism and variability in perception and coping styles may help highlight and account for within-group variability in AA health outcomes. And finally, a strong case for the development of more tailored intervention and prevention strategies can be made if exposure to racism is shown to profoundly impact AA health on a population level [10, 42].

Of note, the research on racism and discrimination as potential stressors is comprised of studies looking at both inter- and intra-group differences. That is, there are studies focusing entirely on AA population (and on AA women more specifically), and there are those with more racial diversity, which focus on differences between racial

groups. Although research exploring intergroup racism abounds in the literature, there are relatively fewer studies assessing the impact of intragroup experiences [24].

The impact of race-related stress on a socioeconomically diverse group of AA women is necessary to promote understanding of intra-racial differences in perceptions and embodiments of racism. The weathering hypothesis was indeed attributed to poverty and discrimination as experienced *uniquely* by black women [26, 30]. Additionally, more recent evidence supports the notion that racial disparities in PTB result from the unique history, context, and experience of AA women, not only during pregnancy, but all throughout their lives [6, 43].

2.3.1 Associations between race- and racism-related stress and birth outcomes in mixed-race study cohorts

In a retrospective cohort study of 152 black and 200 white women originally enrolled in the CARDIA study, the authors found that black women had substantially higher rates of preterm LBW deliveries, and reported more racial discrimination than did their white counterparts [44]. Women reporting high levels of racial discrimination were significantly more likely to deliver LBW infants than women reporting no racial discrimination [44]. A prospective study, which enrolled 29 high-risk (low-income, minority) women, and 66 low-risk (high-income, white) women, found that chronic stress related to minority status and low-income was associated with elevated cortisol without the health-promoting, compensatory decrease in pro-inflammatory cytokine concentration [20]. This evidence supports the existence of GR, or the absence of the negative feedback relationship between cortisol and pro-inflammatory cytokines, in the high-risk group.

Another mixed-race study investigating the contribution of chronic social stressors to racial differences in the rate of bacterial vaginosis (BV) among pregnant women found that AA women had significantly higher rates of BV compared with white women (64% vs 35%) [45]. Further, exposure to chronic stressors at the individual level differed by race such that a significantly greater proportion of AA women reported threats to personal safety as compared with their white counterparts (32% vs 13%). This finding is of note when considering the association of stress with PTB, because an altered vaginal microbial community has been associated with preterm birth [46]. Even more specifically, BV itself has been cited as a strong risk factor for PTB, spontaneous abortion, and other adverse pregnancy outcomes [47]. If AA women are more at risk for BV, due to embodied reactions to chronic, race-related stressors, and if BV is associated with PTB, then BV may act as a mediator along the pathway from chronic stress to adverse maternal and infant outcomes for the AA community.

While numerous studies support a role for stress on adverse birth outcomes, some negative findings suggest no exposure-disease link [48, 49]. A longitudinal mixed-race study of 151 AA and 228 white women found no association between allostatic load and PTB (RR [95% CI]: 1.5 [0.8, 2.9]) or SGA (RR [95% CI]: 1.3 [0.7, 2.5]) [50]. Further, those researchers saw no evidence of any racial disparity in the rates of PTB or SGA among women for whom there was complete biomarker data. However, due to the relatively young age of the study sample (mean age at first birth = 21; allostatic load measurements were taken prior to the first pregnancy), which implies a shorter duration of exposure to stress and less accumulated physiologic wear and tear, the researchers

acknowledge that further investigation is necessary before the association between chronic stress and racial disparities in birth outcomes is discounted [50].

2.3.2 Associations between race- and racism-related stress and birth outcomes in all-African-American study cohorts

Because AA women are regularly exposed to unique societal risk factors closely related to their race, restricting sampling to that group has merit and allows research to shed light on intra-racial disparities that might go undiscovered in inter-group studies [14].

Strong evidence for an association comes from a 2:1 matched case-control study assessing a relationship between maternal exposure to interpersonal racial discrimination and birth to very low birthweight (VLBW, <1500g) infants [51], many of whom were also born preterm, in a racially homogenous cohort. The study of 312 AA women, 104 of whom had delivered VLBW preterm infants, assessed both lifetime and pregnancy exposure to racism across five domains and found that AA mothers who delivered VLBW preterm infants were more likely to report having experienced interpersonal racism during their lifetime than AA mothers who delivered non LBW term infants [51]. Of note, the magnitude of the association between racial discrimination and VLBW was strongest in the "finding a job" and "at work" domains and among college-educated study participants [51]. This finding links to the aforementioned hypothesis that psychosocial stress may increase as time spent at work increases. Of note, no significant association between VLBW and incidents of perceived racial discrimination during pregnancy was found [51]. This suggests the importance of assessing chronic, life course experiences, instead of only measuring stress that occurs during the discrete pregnancy period.

Notably, though, this study is susceptible to selective recall, wherein even if events were initially interpreted as neutral, they may be remembered to reinforce an individual's expectation. Thus, if a study participant expects to be treated discriminatorily, they may remember experiences in such a way that confirms that expectation.

Another study investigated prenatal care (PNC) initiation as a behavioral pathway from race-related stress to PTB among AA women. Receiving early and adequate PNC has been shown to reduce the risk of maternal & infant morbidity and mortality [52]. The retrospective/prospective cohort study of 872 AA women used a denial of racism index based on the well validated Racism and Life Experiences Scales (RALES) [53]. Results from this index, which assessed both personal and group experiences of racism, showed that women who denied group experiences of racism were most likely to enter PNC late (during their 3rd trimester) or not at all [53]. Late entry in PNC has been shown to be associated with AA race and with negative birth outcomes [53]. Even further, racism has been hypothesized as a barrier to PNC entry for pregnant AA women [52].

A study using RALES to assess lifetime exposure to racism found no main effect of RALES score on risk of PTB in its sample of 832 AA women [16]. This negative finding might suggest that unmeasured factors may moderate the effects of racism and may point to possible areas in which to develop interventions. Even in light of a negative main effect results, that same study did report that higher levels of self-reported lifetime racism influenced PTB risk in a complex manner with significant interaction with prenatal depressive symptomology (CES-D > 16) [16, 41].

Another study that also analyzed effect modification by depression looked at exposure to racial micro-aggressions as markers of perceived interpersonal racism in a

sample of 1,232 AA women, and found that, among those with severe depressive symptoms (CES-D ≥ 23), perceived racism was not associated with PTB [49]. However, when the CES-D cut-off was decreased by 1-point (CES-D ≥ 22) to include those the authors categorized as possessing mild to moderate symptoms, perceived racism was significantly associated with PTB [47]. These results speak to the complexity involved in measuring racism and its impacts, and suggest that adverse effects of racism may be missed if especially vulnerable subgroups (e.g. those with mental illness) are not identified and context is not thoroughly considered [16, 49].

2.3.3 Limitations in the reviewed literature on the association between racism as a stressor and PTB

A fundamental issue each of the reviewed prospective studies is recall bias, which is inherent in reliance on self-report [16, 20, 49, 51, 53]. However, given that racism, one's perception of its manifestations, and one's coping mechanisms are subjective, it is difficult to recommend elimination of self-report of either race or perceived racial discrimination. That many studies that used more objective measures of stress do find a positive association between minority status and chronic inflammation offers support for similar evidence produced by use of subjective measures [51].

Another point of discrepancy across the studies of racial stress in pregnant women is the time period over which and at which perceived racism was measured. The study that found no association between VLBW and perceived discrimination *during* pregnancy highlights the importance of considering timing when measuring experiences with racism, as the effect may be obscured if research focuses exclusively on a single time

period (e.g. pregnancy) rather than also looking at one's cumulative experience over the lifecourse [51].

In addition to timing, the type of exposure also varied across studies. One study in particular [53] assessed both personally mediated and group experiences with racism. Their results support the notion that researchers' conceptualization of racism-related stress and discrimination can influence data collection and analysis.

Additionally, selection bias, stemming from inclusion/exclusion criteria and participation, calls the results of the reviewed studies into question. The age distribution in many studies of pregnant women tends to skew young, which is logical given women's peak reproductive years. Those samples are also generally comprised of healthy, non-smokers [16, 20, 51, 53]. In an investigation of lifetime experiences of racism, a relatively young sample may limit detection of that exposure, as the women enrolled may not have had a sufficiently long period over which to accrue the lifetime experiences hypothesized to have detrimental impacts on birth outcomes [27].

Other limitations of the reviewed studies include: inability to infer causality due to issues with temporality in exposure and outcome measurement, sparse data precluding further stratified analyses, and residual confounding. More research is needed to determine whether inconsistencies in the reviewed studies, and in the greater body of literature on race and birth outcomes, reflects differences in unmeasured contextual variables [51].

The literature reviewed reveals evidence for an association between racism and poor birth outcomes, both of which disproportionately impact AA families. Varying study methodology and results do not necessarily weaken that relationship, but

underscore the complexity inherent in measuring and analyzing a multi-dimensional, pervasive, and historical construct like racism. Indeed, depending on individual factors, any events could be deemed stressful and as involving racism [24], hence the heavy reliance on psychosocial measures of racism and discrimination in social epidemiology literature.

2.4 Psychosocial Measures of Racism & Discrimination

Assessment of perceived racism focuses mainly on use of self-report measures, a technique that highlights the importance of subjectivity in the pathogenesis of stress [8]. One critique of self-report instruments for assessing psychosocial stress is the extensive variability among the measures used [54], as shown in the previously reviewed studies. A search of the PTB and LBW literature on psychosocial stress scales reveals a broad range of measures, both validated and non-validated, and suggests that inconsistent associations may be, at least in part, a methodological issue [54]. This review and the subsequent analysis will focus on two measures that have been found to be reliable and valid for use among pregnant AAs – the Jackson, Hogue, Phillips (JHP) Contextualized Stress Measure and Krieger's Experiences of Discrimination (EOD) scale.

2.4.1 The Jackson, Hogue, Phillips (JHP) Contextualized Stress Measure

Psychosocial scales assessing stress among AA populations need to take AA women's unique intersectional context into explicit account. For this reason, the JHP was developed as a race and gender specific stress measure for AA women [6].

To address the limitations in measuring multiple intersecting stressors, or what the authors deem "gendered racism" among AA women, a study was conducted with the purpose of developing a stress measure targeted to that population [55]. Content analysis

of qualitative data from a socioeconomically diverse group of college-educated AA women informed the development of a 71-item questionnaire which consists of six subscales [55]. Four of the subscales include sources of stress: race/racism, burden, work, and personal history. Another subscale measures support and coping as stress mediators; and the last measures stress states, or distress. The race/racism sub-scale is comprised of items that capture racist encounters and anticipations associated with nurturing/caretaker roles, racial affiliation, and stereotypes. The burden subscale is made up of two parts: the first has items representing the imposed and embraced nurturing and caretaker role associated with gender identity; the second part has statements capturing distress as the result of the absence of material and personal resources in the presence of high demand. The work subscale is comprised of items that reflect the experiences and perceptions of gender and racial oppression in the work environment. The work subscale items also capture intra-racial and intra-gender stressors encountered in the workplace. Items that make up the personal history subscale include experiences of mental and physical abuse as individual stressors. The support/coping subscale items represent instrumental and expressive support from family and friends, spirituality/religiosity, and racial and gender identification as sources of social support. The coping items, specifically, replicate active individual engagement in activities intended to counteract stress. Finally, the stress states subscale captures affective responses to stressors.

In its initial application, the internal consistency reliabilities for the subscales were sufficiently high. Validity testing included investigating associations between the stress subscales and existing measures and found the JHP measure is compatible to the well-established Perceived Stress Scale (PSS) [55, 56]. Later testing of the JHP supports

its overall efficacy in identifying gender- and race-related stressors in AA women, including in pregnant AA women specifically (unpublished data). A principal components analysis was conducted, and a 39-item scale was developed (unpublished data); that 39-item scale is used in the present analysis.

In one recent study exploring associations between socioeconomic position and mobility, depression, and contextualized stress, 101 well-educated, pregnant AA women were administered the JHP [56]. While contextualized stress was found to be predictive of depression, the relationship was not modified by the AA woman's socioeconomic position over her life course [56]. Still, the study offers enlightening evidence that racial and gender stressors persist regardless of whether there is financial gain or poverty throughout childhood and adulthood. Additionally, that work supports further applicability of the JHP in a population of pregnant, AA women, and promotes the need for identification of risk factors of PTB that exist intra-racially.

2.4.2 Experiences of Discrimination (EOD) Scale

The EOD is another psychosocial stress measure that was developed as evidence mounted showing that racial minorities who perceive and report discrimination experience higher mortality and morbidity than their white counterparts. It is among of the most commonly used instruments of exposure to racism [57].

The EOD is a 9-item measure assessing the occurrence and frequency of discrimination due to race/ethnicity. Participants indicate whether they have experienced discrimination over their lifetime (Yes/No) in the following nine settings: at school, getting hired or getting a job, at work, getting housing, getting medical care, getting service in a store or restaurant, getting credit, bank loans or a mortgage, on the street or in

a public setting, or from the police or in the courts. The scale's frequency measure assesses the frequency (once, 2-3 times, or 4+ times) of experiences of discrimination at any of the endorsed settings. This scale has high test-retest reliability and predictive validity for health outcomes in AA adults [58, 59]. Moreover, validation studies indicate that scores are not related to social desirability, which is another known bias in self-report data [58, 60, 61].

While it was not developed expressly for use among pregnant, AA women, the EOD has been used in studies investigating the effects of race and pregnancy on stress-induced inflammatory responses [61]. While prior investigations have lacked adequate statistical power to compare inflammatory responses on the basis of perceived racial discrimination, that work points to within-group variability in cellular immune function among those reporting greater racial discrimination [7, 60]. These findings call for further investigation into the predictive validity of the EOD among a cohort of pregnant, AA women.

2.5 Biomarkers of Chronic Stress

The probability that a stress-related adverse health outcome will occur is a function of at least two factors: the amount of actual or perceived stress exposure over time, and an individual's biological propensity to react to that stress [62]. Objective measures of race-related stress have focused on collection and analysis of inflammatory biomarkers. Inflammation is a key biological pathway by which stress may impact birth outcomes. With mental or physical stress, a complex, bidirectional neuroendocrine response is initiated, leading not only to activation of the HPA axis, and secretion of

cortisol, but also to increased release of pro-inflammatory signaling molecules called cytokines [21, 22].

2.5.1 Glucocorticoid Receptor Resistance

Although protective against infection, acute inflammation can increase the risk of PTB [63]. Chronic inflammation can also increase the risk of PTB by inhibiting the body's natural feedback circuit, which under normal conditions, works to shut off the pro-inflammatory response [64]. Multiple recent studies propose a model wherein chronic stress results in glucocorticoid receptor resistance (GR). GR is defined as a decrease in the sensitivity of immune cells to glucocorticoid hormones that normally terminate the inflammatory response. Evidence for GR in response to chronic stress has been found in those reporting high levels of loneliness, and in spouses and parents of cancer patients [65, 66]. A recent study found that after controlling for known covariates, those with recent exposure to a long-term threatening stressful life event demonstrated GR, and those with GR were at higher risk of developing a cold when administered rhinovirus [65]. Congruously, women with lifetime histories of discrimination may be more likely to demonstrate GR, and then to be at higher risk for negative birth outcomes.

Corwin et al. [20] recently published evidence of GR in minority and low-income pregnant women resulting in both dysregulated inflammation and hypercortisolemia, two abnormal neuroendocrine profiles that may explain how chronic stress related to social disadvantage over the lifecourse influences PTB. Further investigation into how imbalances in the stress-related biological responses conspire to influence poor birth outcomes in high-risk populations, like pregnant AA women, is required [20, 23]

2.6 Summary

To date, few studies have assessed whether racially based psychosocial stress is empirically associated with known biomarkers of chronic stress. Recently, Harville et al. [67] found non-significant correlations between cortisol, CRH, and psychosocial measures among a cohort of mixed-race pregnant women. Notably in that study, the role of race was not explicitly examined; it was merely controlled for. The findings underscore that the relationship between measurements of reported stress and biomarkers is not straightforward, and that further investigation bridging analysis of multiple measurements is required.

The most direct way to reduce the influence of race-related stress on health is to reduce the amount of that type of stress one encounters. Though attainable, this reality appears distant given that racism has persisted for centuries in the United States, and that individuals often cannot control their appraisals and embodiments of stressful, racially charged situations. Furthermore, it is unlikely that any single measure will be able to fully capture all instances, perceptions, and biological sequelae of discriminatory experiences [18]. Thus, it is not only necessary to continue to hone existing methodology, but also to understand how multiple measures of stress, data for which are often collected in tandem, work together to describe complex and interwoven biosocial processes, like stress, racism, and birth.

The present analysis will explore the associations of the JHP & EOD, as cumulative psychosocial measures of racism and discrimination, with the $DexIC_{50}$, Dex Top, and Dex Bottom, biomarkers of glucocorticoid resistance and chronic stress, in a socioeconomically diverse cohort of pregnant, AA women. The author hypothesizes that

the JHP & EOD are positively associated with $DexIC_{50}$, Dex Top, and Dex Bottom. That is, higher contextualized stress and higher experiences of discrimination are anticipated to be associated with glucocorticoid resistance in this population of pregnant AA women. Regarding the *a priori* decided covariates, the author hypothesizes that a higher $DexIC_{50}$, Dex Top, and Dex Bottom would be associated with older age, lower household income, public insurance status, a less committed relationship status, and higher EDS, PSS, and SLEI scores.

3. Methods

3.1 Study design & Sample

The Microbiome and Preterm Birth (MPTB) Study (5R01NR014800-03, PI: Elizabeth Corwin), an ongoing population-based investigation, is prospectively enrolling 960 nulliparous, pregnant AA women receiving prenatal care (PNC) at Emory University Midtown Hospital or Grady Hospital in Atlanta, Georgia. Those medical facilities, private and public respectively, see approximately 10% of Georgia's singleton live births to AAs. However, the characteristics of AA women delivering at either hospital are different, allowing for recruitment of a socioeconomically diverse cohort. Given that socioeconomic status is a determinant of the stress and health behaviors under investigation, the diversity across these hospitals will provide sufficient variation in the biobehavioral factors of interest to probe their impact on the microbiome and PTB. Furthermore, the diversity across the two hospitals will allow for a robust within-race investigation of the risk of PTB among AA women.

Women are invited to participate in the study at their first prenatal visit (6-14 weeks' gestation) and, upon eligibility confirmation and consent, are followed through

delivery. Over the course of the follow-up period, data are collected at three time points: twice via direct contact during PNC appointments (at 6-14 and 26-30 weeks' gestation), and once via medical record review post-delivery. Gestational age is to be determined by standard criteria based on last menstrual period and/or first trimester ultrasound. Data collection is being conducted by experienced research coordinators, trained in all aspects of the protocol, and will include biological sample, and clinical and questionnaire data. Women who deliver preterm, that is, prior to 37 completed weeks' gestation, will be designated as cases. An equal number of controls will be randomly selected from those with a term delivery to carry out nested case-control analysis in accordance with MPTB study aims.

The present cross-sectional study, aimed at investigating the association between biomarkers and psychosocial measures of cumulative, discrimination-related stress among pregnant African-American women, is utilizing data collected from the first 184 women enrolled in the MPTB study who have completed their initial study visit at 6-14 weeks' gestation. This visit includes collection of vaginal, oral, and rectal swabs, blood and hair samples, and completion of sociodemographic, health, nutrition and stressor exposure questionnaires. Using the blood drawn from the first visit, members of the research team carried out a white blood cell dexamethasone (Dex) suppression test (DST) and assessment of the cytokine production profile to measure glucocorticoid resistance, a biological indicator of the stress-immune axis. The Dex suppression test measures how much endogenous levels of cortisol change with the administration of dexamethasone, an exogenous steroid. More specifically, the assay is based on the suppression of lipopolysaccharide (LPS)-induced tumor necrosis alpha (TNF-a) production by

dexamethasone in whole blood samples. TNF-a is among the strongest known pro-inflammatory cytokines and has been shown to impair glucocorticoid receptor function, making it a target of glucocorticoid receptor resistance research [68]. The amount of TNF-a produced by LPS-stimulation in the presence or absence of Dex was measured, as was the degree of Dex inhibition of TNF-a production. In the present analysis, the $DexIC_{50}$, or the point at which Dex inhibits 50% of the administered LPS-stimulation, is the index of cell sensitivity to glucocorticoids, and the main outcome measure of GR. A relatively higher $DexIC_{50}$ indicates the potential presence of glucocorticoid receptor resistance and embodied chronic stress, as a higher $DexIC_{50}$ indicates resistance to suppression of dexamethasone, and a sustained increase of plasma glucocorticoid concentrations [Bremner, P. & Pearce, B. (2014), *Neurotransmitter, Neurohormonal, and Neuropeptidal Function in Stress and PTSD*, chapter submitted for publication].

Dex Top and Dex Bottom are two related measures being considered to paint a more complete picture of glucocorticoid resistance. The Dex Top represents the amount of inflammatory cytokine made in response to LPS without any Dex, and therefore indicates one's inherent or current inflammatory state. A higher Dex Top might indicate a greater likelihood of GR, and may be associated with a high $DexIC_{50}$. The Dex Bottom indicates where one's inflammatory cytokines "level off." Theoretically, Dex Bottom values should approach or reach zero. Dex Bottom values that plateau at much higher values may also indicate a greater likelihood of GR.

Women were selected for this analysis if they answered the JHP and EOD scales in their entireties, as those well-validated and population-appropriate measures of psychosocial stress probe lifetime experiences with and perceptions of racism and

discrimination specifically. Additionally, eligible women also completed the Perceived Stress Scale (PSS), a 14-item questionnaire that measures experiences of stress over the last month, the Edinburgh Depression Scale (EDS), which ascertains symptoms of depression in the last 7 days, as depression and general stress were considered as potential covariates or effect modifiers, and the Stressful Life Events Index (SLEI) which measures whether major life events associated with long-term threat have occurred at any time over the life course. The study population on which analyses were performed included 144 women, with complete psychosocial exposure (JHP & EOD) and biomarker ($DexIC_{50}$, Dex Top, & Dex Bottom) outcome data.

3.2 Scale Scoring

EOD

Consistent with prior literature [4, 58], EOD responses to unfair treatment were combined to classify participants into one of three categories: (1) Engaged: talk to others and try to do something about it (talk, act), (2) Moderate: talk to others and accept it as a fact of life (talk, accept), or keep it to myself and do something (quiet, act), and (3) Passive: keep it to myself and accept it (quiet, accept). The EOD situation count score was obtained by tallying the number of situations in which a participant reported experiencing racial discrimination [4, 58]. The weighted frequency score measured total occurrences, assigning the value of 0 to ‘‘never,’’ 1 to ‘‘once,’’ 2.5 to ‘‘2–3 times,’’ and 5 to ‘‘4 or more times,’’ and then summed across items [4, 58].

JHP

The individual items were scored from 1 to 5 with 1 indicating strongly disagree and 5 indicating strongly agree. The total stress score was computed from the sum of the

positive responses indicating the presences of stressors and stress states (distress), and the absence of stress mediators [56]. The total scores ranging from 65 to 151 were maintained in a single JHP Total Score continuous variable, and were also divided into tertiles representing low, moderate, and high contextualized stress.

EDS

Depressive symptomology was a score of >13 on the EDS. The EDS, a 10-item self-report questionnaire with possible scores ranging from 0 – 30, has high sensitivity and specificity for the detection of major depression in pregnancy and has been widely used with pregnant women. Psychometric properties of the EDS for pregnant, African-American populations have been previously reported [69].

PSS

Perceived stress was measured using the PSS-14, which includes 14 items designed to address one's sense of control over life's daily demands [70]. Because there are not yet any well-established and culturally appropriate cut-offs for the PSS, women in the top quartile of stress were compared with those in lower quartiles. A cut-off score of 33 to define high perceived stress was chosen based on the average of the 75th percentile scores across the educational attainment groups [70].

SLEI

Exposure to stressful life events over the entire life course was measured using the 13-item SLEI scale. Given that there are not yet any well-established and culturally appropriate cut-offs to signify high exposure to stressful life events, the same method as was used to define a cutoff for the PSS was used for the SLEI. A score of 5.5 or greater defined exposure to a high number of stressful life events.

3.3 Statistical Analysis

All statistical analysis was performed using SAS 9.3 (SAS Institute, Inc. Cary, NC). Covariates were selected a priori based on the existing stress and PTB literature, and descriptive statistical analysis was conducted on all variables of interest. Distribution of continuous variables was first examined with histograms, and confirmed statistically using the Kolmogorov-Smirnov goodness-of-fit test for normal distribution. Distribution of categorical variables was examined with boxplots. Linear relationships between outcome and independent variables were first assessed visually using scatterplots. Normality and homoscedasticity of residuals were examined with partial plots, and independence of outcome variables was confirmed by examining parent study design and its random sampling methodology. Taken together, these steps assessed whether assumptions were met for simple and multivariable linear regression. Where necessary to meet statistical testing assumptions, outcome and exposure variables were log-transformed to correct for skewness and non-normality.

Bivariate analysis using simple linear regression was performed between all independent variables and each exposure, and between all independent variables and each outcome measured at the 5% significance level. Associations between continuous variables were analyzed using linear correlation. The relationship between continuous and categorical variables was analyzed using t-tests or ANOVAs. Chi-square tests were performed to examine the association between categorical variables. Logistic regression was performed to examine the association between categorical dependent variables and continuous independent variables. Stratified univariate and bivariate analyses were also

performed by education level as previous work has shown that having gone to college does not mediate poor health outcomes in AA female populations as it often does for white college-educated women [6, 71]. Another rationale for stratified analysis by educational level is that the JHP was originally developed within the context of AA women who had attended college, and it has not yet been validated for AA women with less educational attainment.

Multivariable linear regression was used to determine what portion of the variance in the outcome measures was accounted for by the exposures and covariates, and to determine a valid estimate of the association between the exposure and outcome. Predictive analysis was not performed or reported, as it was deemed beyond the scope of this analysis. A separate model was constructed for each psychosocial scale. As noted, variable selection occurred a priori. To avoid issues with multicollinearity, if a covariate had been coded as both continuous and categorical, only the continuous version of the covariate was included in the modeling procedures. To avoid decreasing the sample size and power any further, no influential or outlying outcome or exposure values were excluded in this analysis. Still, the assumptions for linear regression were deemed not grossly violated, allowing for valid multivariable testing.

Interaction assessment was performed via chunk test, where a reduced model containing no interaction terms was compared to a full model containing all interaction terms. Even when the chunk test for interaction was not significant, backwards elimination, wherein the least significant interaction term is dropped one at a time and the model is re-run, was performed to confirm no interaction. In the presence of no interaction, confounding assessment was then carried out using the standard backwards

elimination approach. To build the most valid model, all potential confounders were retained in each model.

After models were run on the main outcome measures in question, a separate set of linear regression models were run on the same sample (n = 144) using EDS Total Score as the outcome variable.

4. Results

4.1 Univariate Analyses

The dataset used for all analyses included 144 observations with no missing values for the exposure or outcome measures. All study participants were pregnant AA women receiving prenatal care at either Grady Hospital or Emory Midtown Hospital in Atlanta, Georgia. Descriptive statistics are summarized in Table 1. The mean age of the sample was 24.27 (std dev 4.27). Age ranges from 18 to 35, with about 57% of the sample indicating an age between 18 and 24. Sixty-eight (47.22%) women reported that they had attended at least some college, and were therefore categorized as "College Educated." All but 13 women indicated they were single. More specifically, 32 (22.22%) women indicated they were single and not cohabitating with a partner, 41 (28.47%) indicated being in a relationship, but not cohabitating with a partner, and 58 (40.28%) indicated being in a relationship and cohabitating with a partner. A minority of the sample (9.03%) indicated being married and cohabitating with their spouse. No one was married, but not cohabitating with their spouse at the time of survey administration.

Given that the study sample was drawn from two Atlanta-area hospitals, one private and one public, women were asked to indicate the type of insurance they had. Most women (77.78%) reported having public insurance, or Medicaid. An insurance

classification was missing for just one participant. There were 39 missing values for income, possibly due to the sensitive nature of the question,. Of the 105 women who did respond, only 28 (19.44%) reported a household income at least 200% above the federal poverty level.

The mean Edinburgh Depression Scale (EDS) score was 7.01 (std dev 5.37). Roughly 17% of the sample scored at or above 13, indicating depressive symptomology. The mean Perceived Stress Scale (PSS) score was 27.63 (std dev 9.10). Thirty-four (23.61%) women scored at or above 33, indicating a higher level of perceived stress based on a cut off at the 75th percentile. The mean Stress Life Events Index (SLEI) score was 4.01 (std dev 2.49). Thirty women scored at or above 5.5, indicating a higher number of stressful life events based on a cut-off at the 75th percentile.

Descriptive statistics were also stratified by education level. The mean age of college educated women, 25.85 (std dev 4.57) was slightly above that of the overall sample. As might be expected, a greater proportion of college-educated women (33.82%), as compared to the overall sample (19.44%), reported a household income at least 200% above the federal poverty level. Mean EDS, PSS, and SLEI scores among college educated women did not differ substantially from scores reported by the overall sample (data not shown).

Table 2 details how the sample answered the Experiences of Discrimination scale. The mean response to unfair treatment was 1.36 (std dev .74), where 0 indicates passive, 1 indicates moderate, and 2 indicates engaged. Just over half the sample (52.08%) were classified as engaged, 31.94% as moderate, and 15.97% as passive. Among the entire study population, the mean number of situations in which participants reported having

experienced discrimination was 1.94 (std dev 2.03). The maximum number of situations that could have been reported was 9, indicating a relatively unexposed sample. The mean weighted frequency with which discrimination was experienced was 4.14 (std dev 5.35); the maximum frequency was 45. Further support that this sample was relatively unexposed to experiences of discrimination comes from a detailed look at responses to the EOD's situation-based questions (data not shown). Well over half of the overall sample (65.28% - 93.06%, depending on the situation), reported never having experienced discrimination in any of the situations outlined in the scale. This held true when EOD responses were stratified by educational attainment. Psychometric testing of the EOD revealed high reliability, which is consistent with the existing literature [58].

Table 3 details responses to the Jackson Hogue Phillips (JHP) Contextualized Stress measure and its subscales. The mean total JHP Total Score for the entire sample was 97.51 (std dev 18.16). Scores were similar when stratified by educational attainment.

Table 4 details the $DexIC_{50}$, Dex Top, and Dex Bottom biomarker descriptive statistics. There were no significant differences in the biomarker measurements (when looked at independently of each other) across levels of education.

Table 1. Demographic Characteristic of Study Participants (n = 144), Stratified by Education Level

	Entire sample (n = 144)					College Educated (n = 68)					Not College Educated (N = 76)				
Variable	**n**	**%**	**Mean**	**SD**	**n missing**	**n**	**%**	**Mean**	**SD**	**n missing**	**n**	**%**	**Mean**	**SD**	**n missing**
Age (yrs)															
Overall			24.27	4.27				25.85	4.57				22.86	3.44	
18-24	82	56.94				32	47.06				50	65.79			
25-35	62	43.06				36	52.94				26	34.21			
Education															
College educated	68	47.22				68	100								
Not college educated	76	52.78									76	100			
Relationship status															

Single															
Not in a relationship	32	22.22				16	23.53				16	21.05			
In a relationship, not cohabitating	41	28.47				17	25				24	31.58			
In a relationship, cohabitating	58	40.28				23	33.82				35	46.05			
Married															
Living together	13	9.03				12	17.65				1	1.32			
Insurance															
Public	122	77.78			1	41	60.29			1	71	93.42			
Private	31	21.53				26	38.24				5	6.58			
Poverty level (household)															
<100% poverty	51	35.42			39	19	27.94			14	32	42.11			25
100-199%	26	18.06				12	17.65				14	18.42			
200%+	28	19.44				23	33.82				5	6.58			
EDS															
Overall			7.01	5.37	3			6.12	4.87				7.84	5.7	3
<13	116	80.56				60	88.24				56	73.68			
>= 13	25	17.36				8	11.76				17	22.37			
PSS															
Overall			27.63	9.10	2			28.84	7.33				26.51	10.4	2
<33	108	75.00				50	73.53				58	76.32			
>= 33	34	23.61				18	26.47				16	21.05			
SLEI															
Overall			4.01	2.49	1			4.4	2.77				3.65	2.17	1
<5.5	113	78.47				50	73.53				63	82.89			
>= 5.5	30	20.83				18	26.47				12	15.79			

Table 2. Experiences of Discrimination Scale Descriptive Statistics, Stratified by Education Level

	Entire Sample (n = 144)				College Educated (n = 68)				Not College Educated (n = 76)			
	n	**%**	**Mean**	**SD**	**n**	**%**	**Mean**	**SD**	**n**	**%**	**Mean**	**SD**
Response to unfair treatment												
Summary score			1.36	0.74			1.56	0.63			1.18	0.8
Engaged	75	52.08			43	63.24			32	42.11		
Moderate	46	31.94			20	29.41			26	34.21		
Passive	23	15.97			5	7.35			18	23.68		
Situations mentioned												
0	52	36.11			19	27.94			33	43.42		
1 to 2	43	29.86			23	33.83			20	26.31		
3+	49	34.03			26	38.23			23	30.27		
Summary score												

Situation (possible range: 0 - 9)			1.94	2.03			2.22	2.1			1.7	1.95
Frequency (possible range: 0 - 45)			4.14	5.35			4.88	5.33			3.47	5.32
Cronbach's alpha												
Situation	.726											
Frequency	.759											

Table 3. JHP Descriptive Statistics, Stratified by Education Level

	Overall sample (n = 144)		**College Educated (n = 68)**		**Not College Educated (n = 76)**	
Score	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
JHP Total	97.51	18.16	96.26	18.47	98.62	17.94
Subscales						
Burden	28.47	8.08	28.09	8.1	28.8	8.09
Coping/Support	31.63	9.54	30	8.11	33.09	10.49
Racism	12.66	4.24	13	4.45	12.36	4.05
Personal History	10.24	5.22	10.34	5.46	10.14	5.04
Work	14.51	2.48	14.84	2.64	14.22	2.3

Table 4. $DexIC_{50}$, Dex Top, and Dex Bottom Descriptive Statistics, Stratified by Education Level

	Overall sample (n = 144)		**College Educated (n = 68)**		**Not College Educated (n = 76)**	
Score	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
$DexIC_{50}$	9910.52	5164.7	10000.15	5366.94	9830.33	5011.31
Dex Top	763.93	410.67	812.01	426.22	720.92	394.08
Dex Bottom	56.24	62.49	58.73	66.24	54.02	59.29

4.2 Bivariate Analyses

Independent sample student t-tests were performed to assess whether answers to the EOD varied by educational level (data not shown). Those bivariate analyses revealed that the Response to Unfair treatment did vary significantly by education ($p = .002$). A significant Chi-square test of association ($p = .009$) between response to unfair treatment and education showed that college educated women were more likely to be classified as engaged as compared to women who were not college educated (63.24% vs 42.22%).

Similar bivariate testing was performed to assess whether response to the JHP varied by educational level (data not shown). While neither the total JHP Total Score nor the scores across JHP tertiles were significantly different based on education level, the Coping subscale did show marginal significant difference ($p = 0.05$). The mean JHP Coping subscale score among college educated women was 30.00 (std dev 10.49), while the mean score for non-college educated women was 33.09 (std dev 8.11). Non-college educated women were more likely to be in the highest JHP tertile group than were college educated women (36.84% vs 27.94%).

Similar bivariate testing was performed to assess whether response to the biomarker outcome measures varied by educational level (data not shown). Neither $DexIC_{50}$ ($p = .84$) nor Dex Top ($p = .18$) nor Dex Bottom ($p = .65$) was significantly different between the two education groups.

Table 5 details bivariate association between the exposures and covariates. Most associations were not significant at the $p \leq .05$ level, indicating a non-significant relationship between the psychosocial scale and the covariate in question. Only significant associations will be discussed herein.

When considered in association with the JHP, the continuous EDS score ($r = .43$, $p < .0001$) showed a moderate, positive linear relationship, as did the continuous SLEI score ($r = .30$, $p = .0002$). Performance of an ANOVA revealed that relationship status was also significantly associated with the JHP score ($F = 3.15$, $p = .03$), indicating that at least one relationship status group's JHP mean was different from the others. A post hoc Tukey comparison test showed that the significant difference was between the single, not cohabitating group and the married group (Difference between the means [95% CI]:

15.44 [.25, 30.63]). All three categorical covariate scales (i.e. EDS, PSS, and SLEI) were significantly associated with the JHP total score. Mean JHP scores were significantly higher among those with higher EDS scores as compared to lower EDS scores (Difference in means [95% CI]: 16.76 [9.35, 24.18]), higher among those with higher PSS scores as compared to lower PSS scores (Difference in means [95% CI]: 8.34 [1.40, 15.29]), and also higher among those with higher SLEI scores as compared to lower SLEI scores (Difference in means [95% CI]: 10.47 [3.28, 17.67]),

When considered in association with the EOD response to unfair treatment, only one continuous covariate showed significant association: PSS score ($p = 0.05$). The maximum likelihood estimate of -0.03 suggests than a 1-unit increase in PPS score decreases EOD Response to Unfair Treatment by .03. Of the categorical covariates, only education was significantly associated with EOD response to unfair treatment ($p = .01$). A Chi-square test of association revealed that engaged study participants, as compared to passive study participants, were more likely to be college educated (57.33% vs 42.67%).

When considered in association with the EOD situation count, the following continuous covariates showed significant association: EDS score ($r = .31$, $p = .0001$), and SLEI score ($r = .36$, $p < .0001$). Based on their correlation coefficients, EDS and SLEI scores had moderate, positive linear relationships with EOD situation count, while PSS score had a weak, positive linear relationship with that exposure. Of the categorical covariates, EDS, PSS, and SLEI were significantly associated with EOD situation count ($p = .0001$, .01, and .002, respectively).

When considered in association with the EOD weighted frequency, the following continuous covariates showed significant association: EDS score ($r = .28$, $p = .001$), PSS

score (r = .20, p = .02), and SLEI score (r = .40, p < .0001). Based on their correlation coefficients, EDS and PSS scores had weak, positive linear relationships with EOD weighted frequency, while SLEI score had a moderate, positive linear relationship with that exposure. Of the categorical covariates, education, EDS, PSS, and SLEI were significantly associated with EOD weighted frequency (p = .03, .002, .001, and .001, respectively).

Table 6 details bivariate associations between the outcome measures, and the exposures and covariates. Neither $DexIC_{50}$ nor Dex Bottom was significantly associated with any of the included independent variables. Dex Top was significantly associated with JHP Total Score (r = .21, p = .01) and insurance status (t = -2.75, p = .01).

Table 5. Bivariate Associations of Covariates with Exposure Measures

	Exposure							
	JHP Total Score		**EOD Response to Unfair Treatment**		**EOD Situation Count**		**EOD Weighted Frequency**	
	Test statistic	**p-value**	**Test statistic**	**p-value**	**Test statistic**	**p-value**	**Test statistic**	**p-value**
Covariate								
Continuous								
Age	0.03[a]	0.71	2.43[d]	0.12	0.06[a]	0.46	0.08[a]	0.33
EDS Score	0.43[a]	<.0001**	3.61[d]	0.06	0.31[a]	0.0001**	0.28[a]	0.001**
PSS Score	0.06[a]	0.48	3.79[d]	0.05**	0.15[a]	0.08	0.20[a]	0.02**
SLEI Score	0.30[a]	0.0002**	0.01[d]	0.92	0.36[a]	<.0001**	0.40[a]	<.0001**
Categorical								
College Educated	0.78[b]	0.44	9.33[e]	0.01**	-1.07[b]	0.09	-2.19[b]	0.03**
Income	1.14[c]	0.32	4.93[e]	0.29	0.87[c]	0.42	0.69[c]	0.51
Insurance	1.11[b]	0.27	2.75[e]	0.25	-0.89[b]	0.38	1.35[b]	0.31
Relationship Status	3.15[c]	0.03**	3.39[e]	0.07	0.23[c]	0.88	0.45[c]	0.72
EDS	-4.47[b]	<.0001**	5.01[e]	0.08	-3.40[b]	0.001**	-3.12[b]	0.002**
PSS	-2.38[b]	0.02**	0.73[e]	0.70	-2.59[b]	0.01**	1.18[b]	0.001**
SLEI	-2.58[b]	0.01**	5.12[e]	0.07	-3.18[b]	0.002**	-3.59[b]	0.001**

[a] denotes a correlation analysis; r is reported as the test statistic.
[b] denotes a T-test; t is reported as the test statistic.

[c] denotes an ANOVA; F is reported as the test statistic.
[d] denotes logistic regression; Wald Chi-square is reported as the test statistic..
[e] denotes a Chi-square test of association; Chi-square is reported as the test statistic.
** denotes significance at the $p \leq .05$ level.

Table 6. Bivariate Associations of Covariates and Exposure Measures with Outcome Measures

	Outcome					
	$DexIC_{50}$		**Dex Top**		**Dex Bottom**	
	Test statistic	**p-value**	**Test statistic**	**p-value**	**Test statistic**	**p-value**
Exposure						
JHP Total Score	-0.11	0.19	-0.21	0.01**	-0.12	0.17
EOD Response to Unfair Treatment	0.09	0.91	0.18	0.84	0.56	0.57
EOD Situation Count	-0.60	0.48	-0.08	0.36	-0.06	0.44
EOD Weighted Frequency	-0.11	0.20	-0.13	0.13	-0.14	0.10
Covariate						
Continuous						
Age	-0.09	0.71	0.10	0.22	-0.10	0.23
EDS Score	-0.06	0.45	-0.15	0.07	-0.09	0.30
PSS Score	-0.95	0.26	-0.09	0.31	-0.05	0.54
SLEI Score	-0.004	0.96	0.003	0.97	0.09	0.29
Categorical Demographics						
College Educated	0.17	0.86	-1.62	0.11	-0.48	0.63
Income	0.26	0.77	2.42	0.09	0.79	0.46
Insurance	-1.61	0.11	-2.75	0.01**	-0.7	0.49
Relationship Status	0.15	0.93	2.25	0.08	0.62	0.60
Categorical Scales						
EDS	0.54	0.59	1.77	0.09	1.43	0.16
PSS	0.31	0.75	2.1	0.04	0.44	0.67
SLEI	0.74	0.46	0	0.99	-0.79	0.43

Note: Correlational analyses were performed to test the bivariate association of the outcome with continuous exposures and covariates. T-tests or ANOVAs were performed to test the bivariate association of the continuous outcomes with categorical exposures and covariates. T-tests were performed against 2-level covariates; the t-value is reported as the test statistic. ANOVAs were performed against covariates and exposures with greater than 2 levels; the F value is reported as the test statistic.
** denotes significance at the $p \leq .05$ level.

4.3 Multivariable Analyses

Multiple linear regression was performed to estimate the effects of the JHP and EOD variables (each scale considered in separate model) with covariates on the three Dex outcome measures (only one outcome per model). In Tables 7 & 8 only the best model for each outcome variable is presented. In all cases, neither significant interaction

nor confounding were discovered (data not shown), making the gold standard model including all covariates the most valid. Thus, the full, no interaction model is reported for each outcome measure.

JHP total score, age, education level, income, insurance, relationship, and the continuous EDS, PSS, and SLEI scores explained just 3.1% of the variance in $DexIC_{50}$ (Table 7). Only the JHP total score was significant at the $p < 0.05$ cutoff. The variable estimates indicate that, in this study sample, $DexIC_{50}$ decreases by .01 pg/ml for each 1-unit (1-U) increase in JHP total score, by .02 for each 1-year increase in age, by .06 from not college educated to college educated, by .05 from a less committed relationship status to a more committed one (e.g. single and not cohabitating, to single and cohabitating), by .002 for each 1-U increase in EDS total score, and by .01 for each 1-U increase in PSS total score. The variable estimates also indicate that, in this study sample, $DexIC_{50}$ increases by .02 from lower to higher income, by .27 from public to private insurance, and by .04 for each 1-U increase in SLEI total score.

The JHP total score and included covariates explained less than 1% of the variance in Dex Top, and none of the variance in Dex Bottom.

The three EOD exposure variables were included together in multivariable models, and along with the aforementioned covariates, explained 4.2% of the variance in $DexIC_{50}$ (Table 8). Only the weighted EOD frequency score was statistically significant at the $p<0.05$ cutoff. The variable estimates indicate that, in this study sample, $DexIC_{50}$ decreases by .06 pg/ml from passive to moderate, or moderate to engaged, by .53 for each 1-U increase in EOD weighted frequency, by .02 for each 1-year increase in age, by .03 from lower to higher income, by .01 from a less committed relationship status to a more

committed one (e.g. from single and not cohabitating, to single and cohabitating), by .02 for each 1-U increase in EDS score, and by .01 for each 1-U increase in PSS total score. The variable estimates also indicate that, in this study sample, $DexIC_{50}$ increases by .53 for each 1-U increase in EOD situation count, by .06 from not college educated to college educated, by .32 from public to private insurance, and by .06 for each 1-U increase in SLEI score.

When included in models for Dex Top and Dex Bottom, all EOD independent variables and covariates explained none of the variance in either outcome measure.

Table 7. Multivariable Linear Regression on Dex Outcomes by JHP & all Covariates

Psychosocial Scale used as Exposure	Independent Variables	Adjusted R^{2*}	B*	SE B*	β*	p-value
JHP	*Model for DexIC50*	0.03				
	JHP Total Score		-0.01	0.00	-0.25	0.03
	Age		-0.02	0.02	-0.11	0.32
	College Educated		-0.06	0.14	-0.05	0.69
	Income		0.02	0.09	0.02	0.84
	Insurance		0.27	0.18	0.19	0.13
	Relationship Status		-0.05	0.07	-0.08	0.46
	EDS Total Score		0.002	0.02	-0.02	0.87
	PSS Total Score		-0.01	0.01	-0.18	0.13
	SLEI Total Score		0.04	0.03	0.16	0.19
	Model for Dex Top	0.01				
	JHP Total Score		-0.01	0.00	-0.16	0.16
	Age		0.01	0.01	0.10	0.38
	College Educated		0.06	0.13	0.05	0.68
	Income		0.07	0.08	0.10	0.44
	Insurance		0.17	0.17	0.13	0.31
	Relationship Status		0.05	0.07	0.07	0.50
	EDS Total Score		0.01	0.01	0.07	0.60
	PSS Total Score		0.00	0.01	-0.07	0.58
	SLEI Total Score		0.01	0.03	0.03	0.83

	Model for Dex Bottom	0				
	JHP Total Score		-0.01	0.01	-0.21	0.09
	Age		-0.02	0.03	-0.08	0.48
	College Educated		0.00	0.28	0.00	0.99
	Income		-0.09	0.18	-0.06	0.61
	Insurance		0.48	0.35	0.18	0.17
	Relationship Status		-0.07	0.14	-0.06	0.60
	EDS Total Score		0.00	0.03	0.00	0.98
	PSS Total Score		-0.01	0.02	-0.06	0.63
	SLEI Total Score		0.05	0.06	0.11	0.36

*Adjusted R^2 denotes the adjusted proportion of the variance explained by the model.
B denotes the variable estime.
SE B denotes the standard error of the variable estimate, B.
β denotes the standardized variable estimate.

Table 8. Multivariable Linear Regression on Dex Outcomes by EOD & all Covariates

Psychosocial Scale used as Exposure	**Independent Variables**	**Adjusted R^{2*}**	**B***	**SE B***	**β***	**p-value**
EOD	*Model for DexIC50*	0.04				
	EOD Response to Unfair Treatment		-0.06	0.09	-0.07	0.52
	EOD Situation Count		0.53	0.31	0.62	0.09
	EOD Weighted Frequency		-0.53	0.23	-0.86	0.02
	Age		-0.02	0.01	-0.16	0.15
	College Educated		0.06	0.15	0.05	0.70
	Income		-0.03	0.09	-0.04	0.74
	Insurance		0.32	0.18	0.23	0.07
	Relationship Status		-0.01	0.07	-0.01	0.94
	EDS Total Score		-0.02	0.01	-0.14	0.26
	PSS Total Score		-0.01	0.01	-0.10	0.42
	SLEI Total Score		0.06	0.03	0.25	0.06
	Model for Dex Top	0				
	EOD Response to Unfair Treatment		0.01	0.09	0.01	0.94
	EOD Situation Count		0.19	0.30	0.24	0.53
	EOD Weighted Frequency		-0.09	0.22	-0.16	0.67
	Age		0.01	0.01	0.07	0.52
	College Educated		0.04	0.14	0.04	0.75
	Income		0.06	0.09	0.08	0.52
	Insurance		0.16	0.17	0.12	0.34
	Relationship Status		0.06	0.07	0.09	0.40
	EDS Total Score		0.00	0.01	-0.02	0.88
	PSS Total Score		0.00	0.01	-0.02	0.87

	SLEI Total Score		-0.01	0.03	-0.02	0.86
	Model for Dex Bottom	0				
	EOD Response to Unfair Treatment		-0.32	0.18	-0.20	0.08
	EOD Situation Count		0.62	0.61	0.37	0.32
	EOD Weighted Frequency		-0.69	0.45	-0.58	0.13
	Age		-0.03	0.03	-0.12	0.28
	College Educated		0.22	0.29	0.09	0.46
	Income		-0.15	0.18	-0.11	0.41
	Insurance		0.55	0.35	0.20	0.12
	Relationship Status		0.00	0.14	0.00	1.00
	EDS Total Score		-0.03	0.03	-0.12	0.32
	PSS Total Score		0.01	0.02	0.05	0.69
	SLEI Total Score		0.09	0.06	0.19	0.15

*Adjusted R^2 denotes the adjusted proportion of the variance explained by the model.
B denotes the variable estime.
SE B denotes the standard error of the variable estimate, B.
β denotes the standardized variable estimate.

4.3.1 Depression as Outcome

Table 9 shows modeling results where the JHP was used as the psychosocial exposure. Only age was statistically significant at the $p<0.05$ cutoff. Along with the full set of covariates, this model accounted for 33% of the total variance in EDS Total Score.

Table 10 shows results from modeling where the EOD was used as the psychosocial exposure. PSS and SLEI were significantly associated with EDS. Together, the model accounted for 28% of the variance in EDS Total Score.

Table 9. Multivariable Linear Regression on EDS Total Score by JHP & all Covariates

Psychosocial Scale used as Exposure	**Independent Variables**	**Adjusted R^{2*}**	**B***	**SE B***	**β***	**p-value**
JHP	*Model for $DexIC_{50}$*	0.33				
	JHP Total Score		-2.71	3.40	0.33	0.14
	Age		0.09	0.02	-0.17	0.01
	College Educated		-0.19	0.10	-0.14	0.42
	Income		-1.44	0.93	-0.02	1.07
	Insurance		-0.14	0.61	-0.05	1.76
	Relationship Status		-0.60	1.19	-0.04	0.75
	PSS Total Score		-0.20	0.48	0.29	0.27

	SLEI Total Score		0.16	0.05	0.25	0.86

*Adjusted R^2 denotes the adjusted proportion of the variance explained by the model.
B denotes the variable estime.
SE B denotes the standard error of the variable estimate, B.
β denotes the standardized variable estimate.

Table 10. Multivariable Linear Regression on EDS Total Score by EOD & all Covariates

Psychosocial Scale used as Exposure	**Independent Variables**	**Adjusted R^{2*}**	**B***	**SE B***	**β***	**p-value**
EOD	*Model for DexIC$_{50}$*	0.28				
	EOD Response to Unfair Treatment		-0.98	0.64	-0.15	0.13
	EOD Situation Count		3.09	2.17	0.44	0.16
	EOD Weighted Frequency		-1.77	1.60	-0.35	0.27
	Age		-0.13	0.10	-0.11	0.22
	College Educated		-1.03	1.04	-0.10	0.32
	Income		-0.21	0.65	-0.04	0.74
	Insurance		-0.97	1.24	-0.08	0.44
	Relationship Status		-0.36	0.50	-0.07	0.47
	PSS Total Score		0.15	0.06	0.27	0.01
	SLEI Total Score		0.70	0.21	0.36	0.001

*Adjusted R^2 denotes the adjusted proportion of the variance explained by the model.
B denotes the variable estime.
SE B denotes the standard error of the variable estimate, B.
β denotes the standardized variable estimate.

5. Discussion

By and large, the results from this analysis suggest no significant relationship between the measures of psychosocial exposures analyzed and the biomarker outcomes. Moreover, the direction of the relationships between the independent and dependent variables was often opposite that which was hypothesized.

While limited, there was support for the author's hypotheses. In regression models on DexIC$_{50}$ including JHP and all covariates, the SLEI, education, and relationship status variables were associated as anticipated, though not significantly. That is, a higher DexIC$_{50}$, or greater likelihood of glucocorticoid resistance, was associated with a higher number of stressful life events, a lower educational attainment, and a less

committed relationship status. In regression models on $DexIC_{50}$ including the EOD and all covariates, EOD situation count, SLEI, income, and relationship status were associated as anticipated, though also not significantly. In that case, a higher $DexIC_{50}$ was associated with a greater number of endorsed situations in which discrimination was experienced, a higher number of stressful life events, lower household income, and a less committed relationship status. Still, all independent variables accounted for a negligible amount of the outcome's variance in either model.

In the adjusted models with EDS score as the outcome, the one including JHP as the exposure showed that increasing age was associated with EDS. The model including EOD as the exposure revealed that PSS and SLEI were associated with EDS. These findings suggest that each scale is differentially associated with depression, though further investigation is required before firm conclusions can be drawn.

The lack of significant findings can be attributed, at least in part, to a number of known study limitations. First, the relatively young age of the study population might have precluded ascertainment of cumulative stress, or allostatic load. Per Geronimus' [26] weathering hypothesis stress accumulates over the lifecourse, indicating that older women may be at particular risk. In a 2006 study, Geronimus et al. [27] found little difference in allostatic load scores for participants younger than 35 years old, but did find that allostatic load for AAs was consistently higher than that for whites for adults aged 35 to 64. The entire study sample used for the current analysis included women 35 years old or younger, with the mean age of about 24. Over half the sample consisted of women 24 years old or younger. In keeping with this proposed limitation and its rationale is the result that a majority of the sample was unexposed to experiences of discrimination.

Additional studies should investigate how younger AA women appraise, experience, and internalize racially-based stress differentially from older AA women.

A second limitation is that the current study is nested within a larger ongoing study (the Microbiome Preterm Birth Study), and thus only included those women who were enrolled by the time data were included in the current analysis. Among the women enrolled in MPTB prior to this analysis, only a subset had complete psychosocial exposure and biomarker outcome data. Future analyses using the MPTB cohort will include sensitivity analyses to compare women with complete exposure and biomarker outcome data to those women with missing values.

A third limitation is the unique setting in which the MPTB study is occurring. All women in this analysis, and all those enrolled in larger MPTB prospective cohort, are receiving prenatal care at one of the two metropolitan Atlanta hospitals. Historically and presently, Atlanta has been known as a "mecca" for AAs [6]. The city has often offered AA populations a lifestyle less readily achievable and less overtly visible than do other metropolitan American locations. A number of factors contribute to Atlanta's relative "friendliness" to AAs, including the city's reputation for lavish lifestyles among AAs (including the availability of housing commensurate with education and income), the presence of the world's largest consortium of historically black universities and colleges, and an established so-called "civil rights elite" [6]. Given these unique qualities of Atlanta, and the type of AA community the city boasts, it may be that residence in Atlanta, in and of itself, mitigates experiences racism and discrimination. Additional studies, then, might investigate the same research question presented here in other

settings that do not possess Atlanta's distinct context. Together, the age and location distribution of the sample suggests the presence of selection bias.

A fourth limitation could be that there are yet to uncovered differences in perceived vs. experienced discrimination. Perceived discrimination, as is measured by the JHP, may depend upon one's appraisal [19]. That is, a situation or encounter, however objectively discriminatory, may not be recognized or perceived as such by all, thereby introducing variability in how the JHP's items are conceptualized by different respondents [19]. Different groups, depending upon coping styles, denial, and a host of other factors, may also label experiences of discrimination variably. Endorsement of a situation on the EOD, for instance, certainly requires the respondent to have ascribed the label of "discriminatory" to an experience in their past, and to have held on to that memory up until the time of survey administration. Thus, until researchers' conceptualizations of what different measurement of racism and discrimination truly measure are refined, it may prove difficult to understand how multiple scales used in a single study support or refute each other. Similarly, further understanding of psychosocial scales may need to precede inclusion of biomarkers in analyses, as there is not yet an established hypothesis as to how which psychosocial scales and which biomarkers are measuring the same embodied processes [67].

A fifth limitation pertains to the measurement of the Dex outcome measures. Currently, Dex is not a well-established biomarker for chronic stress in the literature on racial disparities in PTB. As such, no cut-off points for GR exist for any population, which introduces difficulty in assigning a "diagnosis" of GR to any individual or group in

the current analysis of all AA women. Thus, further validity testing of Dex, including in samples of pregnant, AA women is necessary.

Further, it may be that the psychosocial exposure scales and the Dex biomarkers measure distinct components of the chronic stress embodiment process. Recent work has found that the pathways leading to allostatic load may differ from those leading to metabolic syndrome, which is characterized specifically by the *biological* alterations that accumulate over time to confer risk of clinically detectable disease [50, 72, 73]. This might explain the lack of an association between exposure and outcome in the present study, as they may represent separate underlying processes. This also suggests that future studies are needed to ascertain the temporal sequence of the development of chronic stress (i.e. to determine which biomedical processes happen when, and in what relation to perceptions and experiences of stress externally) [50].

Finally, because variable selection occurred a priori, it may be that the full set of confounding and effect modifying variables was not included in this analysis. That is, there is likely residual confounding by both unmeasured and measured, but not analyzed, factors.

In spite of this study's results and shortfalls, the evidence is indisputable that AA women, from all socioeconomic backgrounds, experience higher rates of PTB than all other racial and ethnic groups [74]. These negative results do not discount the strong evidence that shows that chronic stress may be a driver of racial disparities in PTB. Instead, this study highlights the need for further refinement of existing stress measures (both psychometric and biological) that assess the impact of racism and/or sexism in AA population.

6. Appendix – All Scales Used

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Edinburgh Depression Scale

Subject ID _____

For each item, please indicate the answer that most closely captures how you have felt in the last 7 days.

1. I have been able to laugh and see the funny side of things
- ☐ As much as I always could
- ☐ Not quite so much now
- ☐ Definitely not so much now
- ☐ Not at all

2. I have looked forward with enjoyment to things
- ☐ As much as I ever did
- ☐ Rather less than I used to
- ☐ Definitely less than I used to
- ☐ Hardly at all

3. I have blamed myself unnecessarily when things went wrong
- ☐ Yes, most of the time
- ☐ Yes, some of the time
- ☐ Not very often

4. I have been anxious or worried for no good reason
- ☐ No, not at all,
- ☐ Hardly ever
- ☐ Yes, sometimes
- ☐ Yes, very often

5. I have felt scared or panicky for no very good reason
- ☐ Yes, quite a lot
- ☐ Yes, sometimes
- ☐ No, not much
- ☐ No, not at all

6. Things have been getting on top of me
- ☐ Yes, most of the time I haven't been able to cope at all
- ☐ Yes, sometimes I haven't been coping as well as usual
- ☐ No, most of the time I have coped quite well
- ☐ No, I have been coping as well as ever

7. I have been so unhappy that I have had difficulty sleeping
- ☐ Yes most of the time
- ☐ Yes, sometimes
- ☐ Not very often
- ☐ No, not at all

8. I have felt sad or miserable
- ☐ Yes, most of the time
- ☐ Yes, quite often
- ☐ Not very often
- ☐ No, not at all

9. I have been so unhappy that I have been crying
- ☐ Yes, most of the time
- ☐ Yes, quite often
- ☐ Only occasionally
- ☐ No, never

10. The thought of harming myself has occurred to me
- ☐ Yes, quite often
- ☐ Sometimes
- ☐ Hardly ever
- ☐ Never

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Krieger Experiences Of Discrimination

Subject ID _____

Now I'm going to ask you questions about how you and others like you are treated, and how you typically respond.

1. If you feel you have been treated unfairly, do you usually
   - ☐ accept it as a fact of life?
   - ☐ try to do something about it?

2. If you have been treated unfairly, do you usually
   - ☐ talk to other people about it?
   - ☐ keep it to yourself?

Have you ever experienced discrimination, been prevented from doing something, or been hassled or made to feel inferior in any of the following situations because of yoru race ethnicity, or color? If yes, how many times?

	never	once	2-3 times	4+ times
3a. At school?	☐	☐	☐	☐
b. Getting hired or getting a job?	☐	☐	☐	☐
c. At work?	☐	☐	☐	☐
d. Getting housing?	☐	☐	☐	☐
e. Getting medical care?	☐	☐	☐	☐
f. Getting service in a store or restaurant?	☐	☐	☐	☐
g. Getting credit, bank loans, or a mortgage?	☐	☐	☐	☐
h. On the street or in public settings?	☐	☐	☐	☐
i. From the police or in the courts?	☐	☐	☐	☐

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Jacksonhogue Phillips Stress Meas For Aa Women

Subject ID _____

I will read a statement and please tell me if you agree or disagree with each statement using these responses: Strongly Agree, Agree, are Unsure, Disagree, or Strongly Disagree

	Strongly Agree	Agree	Unsure	Disagree	Strongly Disagree
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 emotional support to family members who don't live with me.	☐	☐	☐	☐	☐
9. I get no time to myself.	☐	☐	☐	☐	☐
10. I am worried that I am going to fail.	☐	☐	☐	☐	☐
11. As an African American woman, I can withstand great pressure	☐	☐	☐	☐	☐
12. My participation in a religious institution gives me a sense of community	☐	☐	☐	☐	☐
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.	☐	☐	☐	☐	☐

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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.	☐	☐	☐	☐	☐
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 opportunity than I do.	☐	☐	☐	☐	☐
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(s) with men	☐	☐	☐	☐	☐
33. I come from a family with a history of drug abuse	☐	☐	☐	☐	☐
34. I have experienced mental abuse in my relationship(s) with men.	☐	☐	☐	☐	☐
35. I can't trust African American men in the workplace to be supportive of me	☐	☐	☐	☐	☐

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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	☐	☐	☐	☐	☐

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Perceived Stress Scale

Subject ID _____

I will read several questions to you. The questions ask you about your feelings and thoughts during the last month. Then, tell me if which of the following best describes how often you felt or thought a certain way during the last month using these responses: Never, Rarely, Sometimes, Often, Very often or Always IN THE LAST MONTH, HOW OFTEN HAVE YOU:

	Never	Rarely	Sometimes	Often	Very often or Always
1. Been upset because of something that happened unexpectedly?	☐	☐	☐	☐	☐
2. Felt that you were unable to control the important things in your life?	☐	☐	☐	☐	☐
3. Felt nervous and "stressed?"	☐	☐	☐	☐	☐
4. Dealt successfully with irritating life hassles?	☐	☐	☐	☐	☐
5. Felt that you were effectively coping with important changes that were occurring in your life?	☐	☐	☐	☐	☐
6. Felt confident about your ability to handle your personal problems?	☐	☐	☐	☐	☐
7. Felt that things were going your way?	☐	☐	☐	☐	☐

	Never	Rarely	Sometimes	Often	Very often or Always
8. Found that you could not cope with all the things you had to do?	☐	☐	☐	☐	☐
9. Been able to control irritations in your life?	☐	☐	☐	☐	☐
10. Felt that you were on top of things?	☐	☐	☐	☐	☐
11. Been angered because of things that happened that were outside of your control?	☐	☐	☐	☐	☐
12. Found yourself thinking about things you have to accomplish?	☐	☐	☐	☐	☐
13. Been able to control the way you spend your time?	☐	☐	☐	☐	☐
14. Felt difficulties were piling up so high that you could not overcome them?	☐	☐	☐	☐	☐

Confidential

Stressful Life Events Index

Subject ID _____

Please indicate which of these events has occurred to you at any time in the past by responding "yes" or "no" after each item I read.

	No	Yes
1. A close family member was very sick and had to go into the hospital	☐	☐
2. I got separated or divorced from my husband or partner	☐	☐
3. I moved to a new address	☐	☐
4. I was homeless	☐	☐
5. My husband or partner lost his job	☐	☐
6. I lost my job even though I wanted to go on working	☐	☐
7. I argued with my husband or partner more than usual	☐	☐

	No	Yes
8. My husband or partner said he didn't want me to be pregnant	☐	☐
9. I had a lot of bills I couldn't pay	☐	☐
10. I was in a physical fight	☐	☐
11. My husband or partner or I went to jail	☐	☐
12. Someone very close to me had a bad problem with drinking or drugs	☐	☐
13. Someone very close to me died	☐	☐

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