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Stress, Inflammation and Preterm Birth in African American Women

Carmen Giurgescu, PhD, RN, WHNP [a,*], Christopher G. Engeland, PhD [b,c,d], Shannon N. Zenk, PhD, RN [b], Karen Kavanaugh, PhD, RN, FAAN [a,e]

[a] College of Nursing, Wayne State University, Detroit, MI
[b] College of Nursing, University of Illinois at Chicago, Chicago, IL
[c] Department of Biobehavioral Health, The Pennsylvania State University, University Park, PA
[d] College of Nursing, The Pennsylvania State University, University Park, PA
[e] Children's Hospital of Michigan, Wayne State University, Detroit, MI

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ABSTRACT

In 2011, 11.7% of babies in the U.S. were born prematurely. African American women have higher rates of preterm birth compared with non-Hispanic white women. Chronic stressors experienced by African American women, such as living in disadvantaged neighborhoods and experiencing racial discrimination, have been related to higher rates of preterm birth. One potential pathway by which neighborhood disadvantage and racial discrimination can affect preterm birth is by increasing cumulative stress burden for these women. Psychological stress has been linked to preterm birth. The effects of chronic stress on preterm birth may occur through alterations of immune functions, thereby predisposing women to infection/inflammation. This review focuses on stress and inflammation as potential mechanisms for disparities in preterm birth in African American women. Prior research provides some evidence that stress-related immune interactions may contribute to preterm birth. Nurses need to be aware that African American women may experience chronic stressors in addition to the acute stress of having a premature infant.

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Preterm birth (<37 completed weeks gestation)[1] is a public health challenge that costs our society $26 billion annually.[2] In 2011, almost half a million babies were born prematurely (11.7%) in the U.S.[1] Preterm birth has been related to higher rates of infant mortality, neurodevelopmental delays, and chronic illness.[3] Even late preterm infants (34–36 weeks gestation) have increased risk for breathing and feeding complications.[4]

Preterm birth is influenced by multiple factors including intrauterine infections/inflammation, hemorrhage, uterine over-distension, cervical disease, stress, and endocrine disorders.[5,6] Maternal socio-demographic characteristics, such as young maternal age,[7,8] lower levels of education,[9,10] and low income[8] have been associated with preterm birth. However, preterm birth rates remain high even when controlling for socio-demographic characteristics. In addition, smoking,[9,11–14] short inter-pregnancies interval,[15,16] genitourinary infections,[14,17] previous history of preterm birth,[18] and chronic hypertension each increase the risk of preterm birth. However, the majority of preterm births (65–70%) are due to spontaneous preterm labor or preterm premature rupture of membranes.[19] Furthermore, medical interventions to treat preterm labor (e.g., tocolytics) have not decreased the rates of preterm birth.[6,20–22] Thus, focusing only on women's obstetrical and medical history, and targeting treatments for such, will not decrease the incidence of preterm birth.

There are persistent disparities in preterm birth which are not easily explained by the abovementioned socio-demographic characteristics and other risk factors.[23,24] African American women have higher rates of preterm birth compared with non-Hispanic white women (16.8% and 10.5%, respectively, in 2011)[1] after controlling for socio-demographic characteristics. It has been proposed that genetic predisposition may increase the risk for preterm birth.[25] However, disputing this possibility, research has found that compared with foreign-born Black women, U.S.-born African American women have higher risk for preterm birth.[26–29]

The causes of preterm birth disparities are not well understood. Chronic stressors experienced disproportionately by African American women, such as living in neighborhoods with high levels of violent crime and experiences of racial discrimination, have been related to higher rates of preterm birth.[9,11,12,30–39] One potential pathway by which negative aspects of the neighborhood environment and racial discrimination can affect preterm birth is by increasing cumulative stress burden for these women. Psychological stress has also been related to preterm birth. This includes perceived stress,[7,13,40–42] psychological distress,[43] anxiety,[11,12,41,44] and depressive symptoms.[45–47] Supporting this notion, African American women are more likely to report higher levels of perceived stress,[48] anxiety,[49] and depressive symptoms[34,50,51] compared with non-Hispanic white women. Despite a large body of literature linking psychological stress

* Address correspondence to Carmen Giurgescu, PhD, RN, WHNP, Assistant Professor, College of Nursing, Wayne State University, Cohn Bldg., Room 335, 5557 Cass Ave, Detroit, MI 48202.
E-mail address: carmen.giurgescu@wayne.edu (C. Giurgescu).

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to preterm birth, research on the biological pathways to explain this association has been limited. A likely mechanism linking psychological stress with preterm birth is inflammation. This review will focus on psychological stress and inflammation as potential mechanisms for preterm birth.

Literature Review

Conceptual Framework

The theory of allostatic load[52–54] may guide our understanding of health disparities in preterm birth. The theory describes acute stress as an adaptive process and chronic stress as a maladaptive process. Acute stress causes an adaptive cascade of physiological events to occur that helps the individual overcome (or avoid) the stressor. During such times, the body endeavors to maintain a balance between the sympathetic and parasympathetic nervous systems. This balance is called allostasis. Conversely, chronic or cumulative stress increases one's allostatic load (dysregulation of the adaptive system that can lead to disease), thereby altering allostasis, and can subsequently affect health outcomes. It is possible that the persistent exposures to chronic stressors by African American women may lead to allostatic overload. Preterm birth is a potential outcome when allostatic load becomes too great in pregnant women. Researchers have examined both acute and chronic stressors and their relationships with preterm birth. Acute stressors may be catastrophic events such as earthquakes and have been related to preterm birth.[55,56] These acute stressors impact all racial groups. Chronic stressors, for the purpose of this review article, are stressors over a significant portion of a woman's life such as poverty, living in neighborhoods with high levels of violent crimes, and experiences of racial discrimination. African American women are more likely to be exposed to such stressors than non-Hispanic white women.[32,34,35,37,57] The effects of chronic stress on preterm birth may occur through a dysfunction of the hypothalamic-pituitary-adrenal (HPA) axis, and a subsequent alteration of immune functions, thereby predisposing women to infection/inflammation. The HPA axis produces cortisol which under normal conditions down-regulates the body's inflammatory responses. However, during chronic stress, the HPA axis becomes dysregulated and cortisol becomes less effective at suppressing inflammation. We hypothesize that chronic stressors experienced by African American women result in higher levels of psychological stress. In turn, this leads to alterations in the HPA axis, thereby dysregulating inflammation and predisposing African American women to preterm birth[58] (see Fig 1).

Chronic Stressors and Psychological Stress

Like their non-pregnant counterparts, pregnant African American women are more likely to live in neighborhoods with high rates of poverty,[35,37] violent crime,[32,57] and abandoned buildings,[57] to be exposed to racial discrimination,[34] and to report more stressful events[12,34,48] compared with pregnant non-Hispanic white women. These chronic stressors have been related to higher rates of preterm birth.[9,11,12,30–39] For instance, residence in a high poverty census tract was a risk factor for preterm birth for mothers reporting emotional stress (e.g., moving to new address, becoming homeless, and mother/partner going to jail).[59] One potential pathway by which these chronic stressors relate to preterm birth is by increasing psychological stress for these women. However, research on the relationships of chronic stressors and psychological stress in pregnancy has been limited.

It is possible that the life-long stress caused by poverty and experiences of discrimination accumulates and contributes to preterm birth. In a sample of 72 postpartum women within the first three days after birth, psychological distress mediated the effects of neighborhood social disorder and perceived neighborhood crime on preterm birth.[60] Similarly women who reported more racial discrimination had higher levels of perceived stress[61] and psychological distress.[60] Such lifetime chronic stressors increase a woman's cumulative stress burden (allostatic load), and appear to contribute to disparities in preterm birth.[62] Positive coping strategies and social support may ameliorate pregnant women's psychological stress,[63] and also appear to play a role as African American women are more likely to report lower levels of social support compared with non-Hispanic white women.[48] In addition, pregnancy-specific stress predicts cigarette smoking, caffeine consumption, and unhealthy eating.[64] Such unhealthy behaviors (e.g., smoking[9,11–14]) have been related to preterm birth and provide an indirect mechanism by which stress can affect birth outcomes. Women who continued use of tobacco, alcohol, and illicit drugs throughout pregnancy exhibited the highest levels of depression and anxiety compared with women who quit during pregnancy.[65] Therefore, the life-long stress experiences by some African American women may increase their psychological stress and use of unhealthy behaviors.

Psychological Stress and Inflammation

Psychological stress has been related to dysregulation of the HPA axis resulting in increased levels of cortisol[66] and alterations in immune functions leading to higher levels of inflammation.[66,67] In plasma, cortisol is mostly bound to corticosteroid-binding globulin, with a small amount bound to albumin. The remainder is free and, thus, biologically active. Less than 10% of cortisol circulates in the body as a “free” fraction of the hormone.[68] Cortisol is potently anti-inflammatory and, under normal conditions, activation of the HPA axis down-regulates the body's inflammatory responses (see Table 1 for key terms). Cytokines are substances produced by the immune system and other cells that regulate these inflammatory responses. Pro-inflammatory cytokines such as interleukin(IL)-1β, IL-6, and tumor necrosis factor-α (TNF-α) stimulate the HPA axis to secrete cortisol.[69] In turn, cortisol down-regulates pro-inflammatory cytokines[70] and stimulates the production of anti-inflammatory cytokines such as IL-10. In this manner, the immune system can signal to regulate the body's level of inflammation. However, during chronic stress, the HPA axis becomes dysregulated, often resulting in high levels of circulating cortisol. These unremitting high levels cause immune cells to become less sensitive to cortisol's anti-inflammatory effects. As a result, cortisol becomes less effective at suppressing

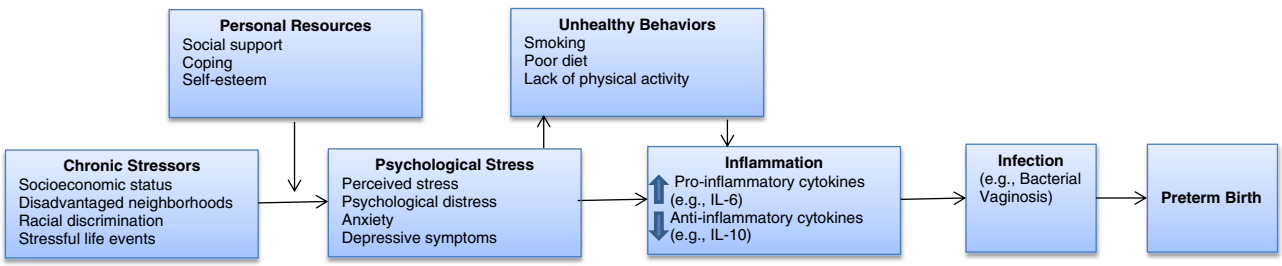

Fig 1. Potential pathways linking chronic stressors, psychological stress, inflammation and preterm birth. (Color version of figure is available online.)

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