

The Relationship between Racial Identity, Income, Stress and C-Reactive Protein among Parous Women: Implications for Preterm Birth Disparity Research

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The persistent racial disparity in preterm birth (PTB) remains one of the most obvious yet poorly understood health disparities in the United States, and current evidence suggests that maternal stress, infection and inflammation may play an important role in the etiology of PTB. In this context, we assessed the complex relationships among racial identity; socioeconomic status (SES); psychosocial factors; and serum C-reactive protein (CRP), an inflammatory biomarker, among parous women in King County, WA. African-American women consistently reported a higher number of stressful life events than white American women (4.6 vs. 2.9, $p < 0.001$), as well as slightly higher levels of perceived stress and lower social support (24.7 vs. 22.2, $p = 0.011$, and 3.4 vs. 3.6, $p = 0.06$, respectively). In the multivariate analysis, African-American race, low-income status and their interaction were all independently associated with CRP; when further adjusted for proximal psychosocial, behavioral and infectious factors, race and income associations were significantly reduced. Stressful life events score was the single best proximal predictor of CRP levels ($\beta = 0.07$ per event, $p < 0.001$), while perceived stress and social support were not significantly related to CRP. These results support the hypothesis that differences in CRP by racial identity and income may be mediated by differences in proximal risk factors, including stressful life events and health behaviors such as smoking. Objective life event stressors may be important to consider in future studies investigating a potential inflammatory etiology for preterm birth.

Keywords: health disparities ■ C-reactive protein ■ inflammation ■ obstetrics/gynecology

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INTRODUCTION

Racial disparity in preterm birth (PTB) remains one of the most salient health disparities in the United States today. Despite significantly improved neonatal survival, PTB is still the leading cause of infant mortality.¹ Extensive research, including successful identification of several important risk factors for PTB, has unfortunately not succeeded in reducing the disparity in rates of prematurity between infants born to African-American and white American mothers, which is still largely unexplained.²

Current evidence suggests that both maternal stress³⁻⁶ and genital tract infections⁶⁻¹¹ are involved in the etiology of PTB, and differences in the distribution of these underlying risk factors by race may be at least partly responsible for the observed disparity.² Specifically, chronic stress and infection may cause local or systemic inflammation or an upregulated inflammatory response, which then may put a woman at higher risk of PTB.^{12,13}

C-reactive protein (CRP) is an acute-phase reactant that is a marker of chronic inflammation and remains stable in serum,¹⁴ and even minor elevations in CRP have been shown to be predictive of many adverse health outcomes, including cardiovascular disease (CVD).^{15,16} Several recent studies have also observed an association between elevated CRP during pregnancy, which may in part reflect subclinical genital tract infections and complications, including pre-eclampsia¹⁷⁻¹⁹ and PTB.²⁰⁻²³ Indeed, women who have a history of delivering a premature infant are known to be at higher risk of CVD later in life,²⁴⁻²⁶ suggesting that both pathologies may be related to a common inflammatory pathway.

Numerous studies have observed racial and socioeconomic gradients in CRP levels, with those of nonwhite race and lower socioeconomic status (SES) consistently having higher serum CRP levels.²⁷ This may be in part due to behavioral factors such as smoking and body mass index (BMI), lifestyle factors which are known to be associated with inflammatory markers and strongly racially and socioeconomically patterned. To the extent that elevated CRP is a marker of risk for PTB, underlying differences in CRP by race and SES could partially

explain the racial disparity in PTB.

Recent attention has also focused on how psychosocial factors may be related to inflammation, either directly via immune or endocrine pathways, or through intermediate behavioral factors. One large study found that constructs, including cynical distrust, chronic stress and depression, were positively associated with several biomarkers of inflammation, including CRP.²⁸

Nearly all prior studies have explored CRP in the context of CVD, recruiting predominantly middle-aged and elderly subjects. Only one small study has investigated the effects of psychosocial stress on CRP among pregnant women (n=52), finding that elevated stress and low social support were modestly associated with CRP levels across pregnancy.¹² In the context of prevention of adverse birth outcomes, more comprehensively investigating both the distal and proximal determinants of CRP among women of childbearing age is an important research priority.

The objective of this study was to explore the effects of perceived stress, life events stress, social support, racial identity and SES on serum CRP levels among parous women of childbearing age. Specifically, we aimed to assess how effects of distal factors such as race and SES might be mediated by more proximal psychosocial factors.

METHODS

Data for this study came from the “A Better Chance” (ABC) Project, a population-based cohort study of parous women in King County, WA, which aimed to investigate mechanisms for racial disparity in preterm birth rates. The study was granted human subjects approval by the Washington State Institutional Review Board (WSIRB), and all subjects provided written informed consent.

The present analysis is cross sectional in design, utilizing only data collected at enrollment. Women with a history of prior early preterm (20–34 weeks) or prior term (≥ 37 weeks) birth were recruited through one of two mechanisms. Eligible women were identified via the Washington State Birth Certificate Database from 2002–2006 and were contacted by a Public-Health Seattle & King County liaison to assess interest. Study personnel were only provided contact information for those women who consented. In addition, women were recruited through advertisements at local medical centers, day care centers, women’s shelters, and other public settings. Eligibility criteria included being U.S.-born, being a King County resident at the time of delivery and having had no hypertensive complications during the index pregnancy.

Participants completed a structured in-home interview at enrollment that ascertained basic demographic information, and medical and reproductive history. Racial identity was ascertained by subject self-identification and was categorized as white American or African-American for the purposes of this analysis. Annual

household income was categorized based on a priori cut-points defined by the King County Housing and Urban Development (HUD) statistics for 2005;²⁹ women with household incomes of <50% of the King County median were classified as low income. We chose to reference household income to a local standard in order to evaluate relative as opposed to absolute poverty, especially given the relatively high cost of living in this region. Additionally, income thresholds were specifically based on household size in order to better approximate absolute resources available; thus, women living in households with relatively high incomes may still be classified as low income if the total income must be spread out over many family members. As an example, a household of four with an annual income of <\$38,950 would be considered low income according to the King County HUD criteria.

Psychosocial stress was measured using two different scales designed to measure the distinct constructs of perceived stress and stressful life events. The Cohen scale is a psychometrically validated 14-item instrument that measures an individual’s perception of stress over the prior month and includes questions such as “How often have you felt you were unable to control the important things in your life?” and “How often have you been upset because of something that happened unexpectedly?”³⁰ Responses were coded on a five-point Likert frequency scale (“never” to “very often”), and a summary score was calculated.

In contrast, the Stressful Life Events scale, which was developed as part of the CDC’s Pregnancy Risk Assessment and Monitoring System (PRAMS), is a 13-item survey which asks about the occurrence of specific types of events during the year prior to delivery. Example items include: “You or your partner lost your job,” “Someone close to you had a bad problem with drinking or drugs” and “You had lots of bills you couldn’t pay.”³¹ These items are designed to represent discrete, specific stressful events or situations. While the scale ascertains events that happened during pregnancy and thus may not be perfectly contemporaneous with the year prior to study enrollment (most women were enrolled ≥ 6 months after delivery), only one question is pregnancy specific (“Your partner told you they did not want you to be pregnant”).

Answers to the PRAMS Social Support questionnaire, a four-item scale, were also collected. Sample instrument statements included: “You had someone to drive you to the clinic or doctor’s office if you needed a ride” and “You had someone to loan you \$50”; and women were asked to indicate whether they had such support during their last pregnancy.

Whole blood samples were collected from subjects at a subsequent study visit shortly after the initial interview. Samples were centrifuged, and serum was then stored at -20° C until ready for high-sensitivity C-reactive protein (hsCRP) analysis. Assays were conducted

by the University of Washington Medical Center Clinical Immunology Laboratory using a latex-enhanced nephelometry commercial kit (Dade-Behring, Deerfield, IL). One-milliliter serum was mixed with latex particles coated with mouse monoclonal anti-CRP antibodies, and the concentration of antigen-antibody complexes was measured based on light scatter using a Behring nephelometer. This method has been shown to be effective and comparable to other methods, including ELISA.³² The observed analytical coefficient of variation (CV) for this assay over the study period ranged from 3.1–7.4%, with higher variation typically observed only for the ultralow control values. For the purposes of quantitative analysis, samples which tested below the detection limit (<0.2

mg/L) of the hsCRP assay were assigned a value equal to half the lower limit of detection.

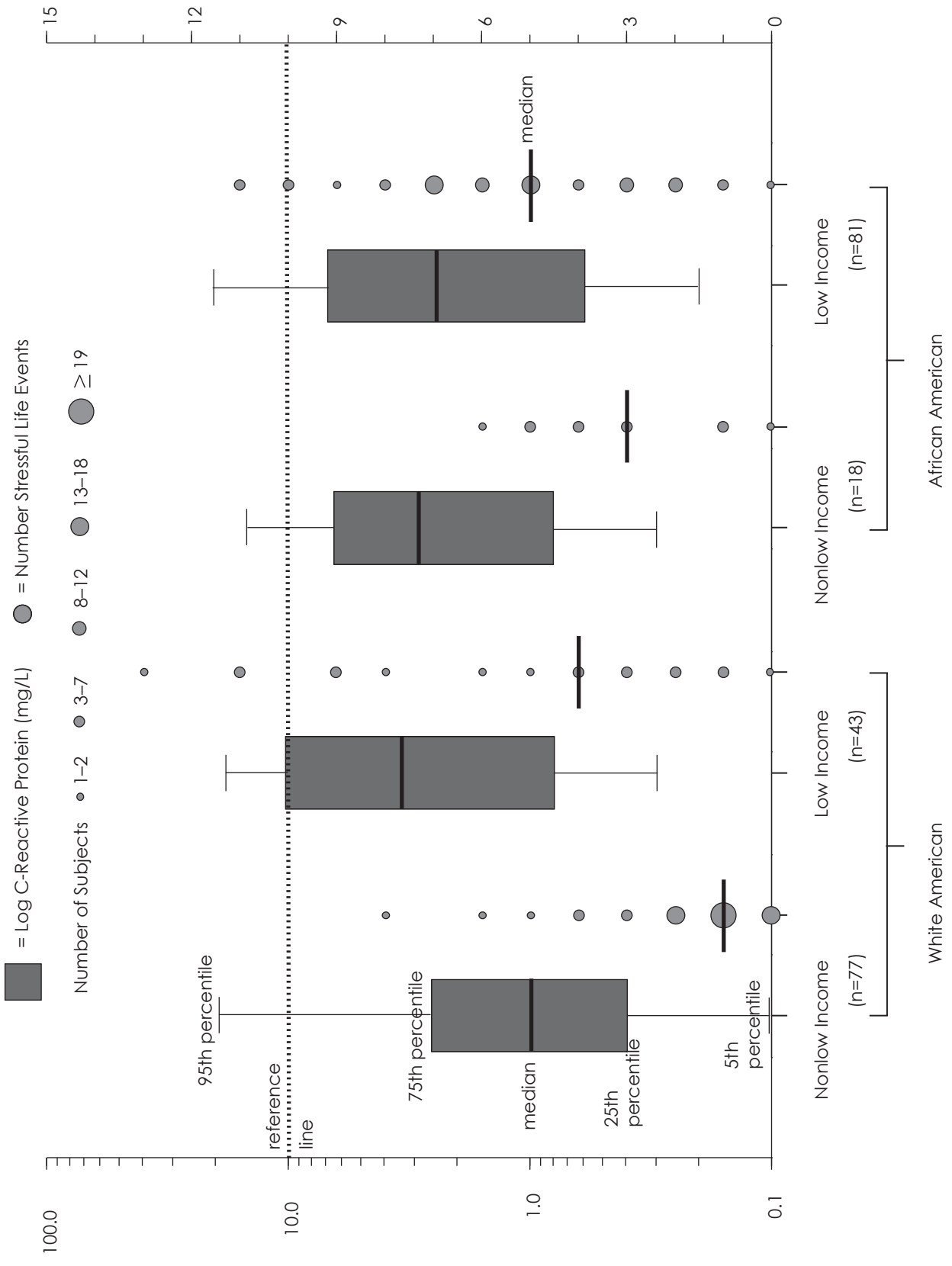
Vaginal swabs were collected at enrollment (or soon thereafter if a subject was menstruating), and BV status was ascertained by Gram stain based on Nugent criteria: a score of 0–3 was considered normal flora, 4–6 was intermediate and 7–10 corresponded to BV.³³ Subjects were also tested for common sexually transmitted infections (STIs), including *Trichomonas vaginalis* by culture (In-Pouch TV),³⁴ and *Chlamydia trachomatis* and *Neisseria gonorrhoeae* by nucleic acid amplification (Amplacor PCR).³⁵ Treatment was offered as appropriate.

Table 1. Characteristics of the study population, stratified by racial identity

	White American N=120	African American N=99	P
<i>Demographic</i>			
Age	32.6 ± 6.8	29.7 ± 7.5	0.004
Marital Status			
Single/no partner	19 (16)	31 (32)	
Partnered/married	96 (80)	56 (57)	
Separated/divorced	2 (2)	4 (4)	
Other	3 (3)	7 (7)	0.004
Gravidity	2.8 ± 1.9	3.7 ± 2.3	0.002
Occupation			
Homemaker	37 (31)	14 (14)	
Employed	64 (53)	47 (48)	
Student	7 (6)	8 (8)	
Unemployed	12 (10)	30 (30)	<0.001
Household income			
Median \$	70,000	18,050	<0.001
Low income*	43 (36)	81 (82)	<0.001
Household size	3.4 ± 1.0	3.8 ± 1.8	0.058
<i>Behavioral</i>			
Ever douched	49 (41)	78 (79)	<0.001
Current smoker	31 (26)	35 (36)	0.134
Ever used cocaine	39 (33)	19 (19)	0.029
<i>Infections</i>			
Chlamydia	0 (0)	5 (5)	0.018
Gonorrhea	0 (0)	2 (2)	0.203
Trichomoniasis	0 (0)	10 (10)	0.002
Bacterial vaginosis	19 (16)	45 (46)	<0.001
<i>Psychosocial</i>			
Stressful life events	2.9 ± 2.9	4.6 ± 2.7	<0.001
Social support	3.6 ± 0.9	3.4 ± 1.0	0.064
High social support	99 (83)	62 (63)	0.001
Perceived stress	22.2 ± 6.9	24.7 ± 7.3	0.011

Data are presented as n (%) for categorical variables and mean ± SD for continuous variables unless otherwise specified; * Household income of <50% of the 2005 King County median for household size.

Figure 1. Log CRP and number of stressful life events by racial identity and income groups (reference line, CRP=10 mg/L)



Statistical Analysis

Differences in baseline characteristics by racial group were explored using the Chi-square or Fischer’s exact test for categorical variables and Student’s t test or Mann-Whitney U test for continuous variables. CRP values were log transformed to reduce skewness and improve modeling behavior.

Multivariate linear regression was used to assess associations among race, SES, stress and CRP levels. We approached the analysis by conceptualizing potential predictors into two distinct levels based on their hypothesized role in the pathway leading to elevated CRP: distal factors (race and income) and proximal factors (psychosocial stress constructs, behaviors, genital tract infections) thought to be potential mediators. Two separate but closely related multivariate models were constructed to test these hypotheses. Model 1 included African-American race, low-income status, and the interaction between race and income, representing the aggregate effect of race and SES on CRP levels. Model 2 additionally adjusted for a set of proximal variables that were known or plausibly hypothesized to be associated with CRP and therefore were potential mediators of any observed race and SES effects.

All analyses were conducted using SPSS® 10 (SPSS Inc., Chicago, IL) and Stata® 9.2 (StataCorp., College Station, TX).

RESULTS

A total of 219 women had complete data on income, psychosocial stress and CRP. Overall, African-American women were slightly younger and more likely to be single, unemployed, current smokers, and to have a low income; they were also more likely to have a history of douching and to be diagnosed with an STI at enrollment. African-American women reported consistently higher perceived stress scores and a higher number of stressful life events as well as lower social support scores than

white American women. Relevant sociodemographic, behavioral and psychosocial characteristics of the study population, stratified by racial identity, are presented in Table 1.

Figure 1 presents median levels of CRP and median number of stressful life events experienced, by racial identity and income group. White American, nonlow-income women had the lowest median CRP, while white American, low-income women were observed to have the highest median CRP. African-American women had intermediate median CRP levels, which did not differ significantly by income status. The vast majority of women in all racial and income groups had CRP levels of <10 mg/L, the reference value typically indicative of active clinical inflammation.

Low-income African-American women experienced the highest number of stressful life events on average, followed by low-income white American women, non-low-income African-American women and nonlow-income white American women, who reported the fewest number of stressful life events.

In the multivariate analysis (Table 2, model 1), identifying as African American and having a low income were both independently associated with significant increases in CRP levels ($\beta=0.37$, $p=0.027$ and $\beta=0.41$, $p=0.001$, respectively). The interaction between African-American race and low-income status was also significantly predictive of CRP ($\beta=-0.47$, $p=0.022$), indicating that the effect of having a low income differed by racial identity. Specifically, while there was a strong income effect among white American women, no such effect was observed among African-American women.

After adjustment for psychosocial, behavioral and infection-related variables, race and income associations with CRP were reduced in magnitude and no longer statistically significant (Table 2, model 2). The single strongest proximal predictor of CRP was number of stressful life events experienced ($\beta=0.07$, $p<0.001$), although

Table 2. Multivariate associations with log CRP

	Model 1		Model 2	
	β (95% CI)	P	β (95% CI)	P
African American	0.37 (0.04–0.70)	0.027	0.27 (-0.05–0.59)	0.10
Low income	0.41 (0.17–0.65)	0.001	0.13 (-0.15–0.40)	0.36
African American,* low income	-0.47 (-0.88–-0.08)	0.022	-0.26 (-0.66–0.15)	0.22
Stressful life events			0.07 (0.03–0.10)	0.001
Perceived stress			-0.01 (-0.02–0.00)	0.07
Social support			0.05 (-0.04–0.15)	0.27
Cigarette smoking			0.20 (-0.02–0.42)	0.07
Bacterial vaginosis			-0.02 (-0.23–0.19)	0.87
Current STI ¹			-0.30 (-0.64–0.04)	0.09

Model 1: includes race, income and race*income interaction. $R^2=0.07$; Model 2: Model 1 variables plus potential mediating variables. $R^2=0.15$; 1: Sexually transmitted infection: *T. vaginalis*, *C. trachomatis* or *N. gonorrhoeae*.

there was also a marginal association between current smoking and CRP ($\beta=0.20$, $p=0.07$). Other psychosocial variables, including perceived stress and social support, were not independently associated with CRP, nor were any genital tract infections.

DISCUSSION

African-American women consistently reported a higher number of stressful life events, lower social support and higher perceived stress than their white American counterparts. Interestingly, the largest racial differences were observed in stressful life events, with smaller disparities in social support and perceived stress. This finding might suggest that some African-American women are habituated to higher levels of chronic stress, perceiving such stressors as a routine part of life. Racial identity is increasingly understood in biomedical research as a social construct, indicative of shared socioeconomic and cultural features as opposed to intrinsic biologic similarity.^{36,37} It seems plausible that these shared socioeconomic and cultural features also extend to a shared psychosocial environment, one which negatively contributes to chronic inflammation and associated adverse health outcomes among African-American women.

Women living in households with low incomes also reported a higher number of stressful life events than those in nonlow-income households. This may reflect the measurement instrument sensitivity to stressful life events that are financial in nature (“You couldn’t pay all of the bills”), although it is likely that having a low income puts women at higher risk of other types of stressors related to housing, relationships and employment as well.

African-American women also had higher serum CRP levels on average than white American women. We observed evidence of an interaction between race and income; specifically, income did not appear to be protective among African-American women. In fact, nonlow-income African-American women had slightly higher median CRP levels than low-income African-American women, the opposite trend as was observed among white American women.

The strongest proximal predictor of CRP levels was the number of stressful life events experienced; and no significant effects of race, income or their interaction remained after adjustment for psychosocial, infection and behavioral variables. Although it is impossible to infer causal relationships due to the cross-sectional nature of the study design, our results are consistent with the hypothesis that stressful life events and behaviors such as smoking are important mediators of the observed effects of race and income on CRP levels. Research indicates that being a racial minority and having a low income may predispose women to exposure to more chronic stressors or contexts in which smoking is viewed as an acceptable behavior. Environmental expo-

sure to stress and smoking can be viewed as proximal contextual risk factors that in turn may lead to systemic subclinical inflammation.

Interestingly, genital tract infections were not associated with inflammation in this population, although the small number of women with such infections limits our ability to draw strong conclusions. Larger studies will be needed to more comprehensively investigate how infections may be involved in the inflammatory pathway and whether they may partially mediate the observed relationship between stress and inflammation.

Several prior studies have reported an association between maternal stress during pregnancy and increased levels of proinflammatory cytokines and/or CRP,^{12,13} but to our knowledge this is the first to separately assess the impact of stressful life events and perceived stress on inflammation among parous women (albeit in a non-pregnant cohort).

An important limitation of this study was the strong correlation between identifying as African American and having a low income in this population, which limited our ability to discern the independent effects of these factors. We also lacked information on BMI, a known predictor of systemic inflammation. However, like smoking, BMI is a lifestyle factor that is more likely to be a mediator of the effects of race, SES and stress, as opposed to a true confounder. A recent review noted that studies which fully adjust for potentially mediating behavioral and lifestyle factors may actually underestimate the true effects of race and SES.²⁷

It is important to note that the majority of women in this study had CRP levels below the threshold typically indicative of clinical inflammation (10 mg/L), and there is not yet a well-established threshold for CRP with respect to risk of PTB. Future studies will be needed both to establish cutoff values predictive of clinical risk and to investigate whether interventions aimed at reducing chronic stressors during pregnancy can successfully reduce CRP levels and rates of PTB.

Insofar as PTB and other pregnancy complications may be related to an inflammatory pathway, it is critical to continue exploring both the distal and proximal causes of such inflammation among women of childbearing age. Prospective studies will allow direct testing of the extent to which psychosocial, behavioral and infectious factors may mediate persistent racial and socioeconomic disparities in adverse reproductive outcomes.

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