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A Critical Review on the Use of Race in Understanding Racial Disparities in Preeclampsia

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Abstract

Background: Preeclampsia is a significant cause of maternal morbidity and mortality, affecting up to 8% of pregnancies globally. Although the precise etiology is still under study, the literature suggests that vascular changes reduce placental perfusion and affect the remodeling of spiral arteries to create the hallmark feature of preeclampsia: elevated blood pressure. Screening for preeclampsia is currently recommended for all pregnant women, particularly if risk factors exist. A noted risk factor codified in guidelines is “African-American race.”

Content: We summarize the racial disparities in preeclampsia incidence, morbidity, and mortality. We consider the limitations of using race to understand disparities by also examining multiethnic, immigration, and international studies. We then critically evaluate laboratory analytes

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associated with racial disparities of preeclampsia and explore other mechanisms of action, such as socioeconomic status, stress, and access to care.

Summary: Black and African-American women are consistently at higher risk of preeclampsia incidence, morbidity, and mortality than their white counterparts. Asian women are consistently at lower risk of preeclampsia, whereas the association for Hispanic women remains unclear. When these broad racial categories are subdivided by geographic or cultural origin, preeclampsia disparities within racial groups are identified. The limited literature suggests that sub-Saharan African immigrants tend to have a higher risk of preeclampsia than US-born white populations but a lower risk than US-born Black women. Existing studies seeking to identify racial differences in analytes have limited research designs and tend to operationalize race as a proxy for biologically inherent (i.e., genetic) differences between races despite a plethora of other possible explanatory mechanisms.

A PRIMER ON PREECLAMPSIA

Clinical Features of Preeclampsia

Preeclampsia complicates 2%–8% of pregnancies globally and is the second leading cause of morbidity and mortality in pregnancy (1–3). Although the precise etiology is unknown, current research suggests that preeclampsia results from reduced placental perfusion and impaired remodeling of the spiral arteries (2). These vascular changes may underlie the hallmark clinical feature: new-onset elevation in blood pressure (BP; ≥140/90 mmHg on 2 separate readings) (2). Additional features include nervous system manifestations, liver subcapsular hematoma, and sudden-onset pulmonary edema (1, 2). Eclampsia, characterized by convulsions, is the most severe manifestation of preeclampsia. Fetal impacts include fetal growth restriction, oligohydramnios, placental abruption, and nonreassuring fetal status, all of which may increase the risk of preterm delivery (2).

Lab Values and Screening Modalities

Preeclampsia is screened by taking BP at each prenatal care visit to allow for early identification and diagnosis (3). The laboratory values associated with severe preeclampsia include thrombocytopenia (platelet count <100 000 × 10^9/L), twice the upper limit of normal concentration of liver enzymes, serum creatinine ≥1.1 mg/dL or a doubling of serum creatinine concentration in the absence of other renal disease, and evidence of proteinuria (≥300 mg in 24-hour urine protein collection, protein-to-creatinine ratio [PCR] ≥0.3 mg/dL, or urine dipstick protein reading of 2+) (1).

Risk Factors for Preeclampsia

All pregnant women should be screened for preeclampsia. Prior health conditions such as a patient history of eclampsia or preeclampsia (particularly early onset preeclampsia), maternal vasculopathies including type 1 or 2 diabetes mellitus, chronic hypertension, and renal or autoimmune disease are associated with an increased risk of preeclampsia (1–3). Other conditions include multifetal gestation, nulliparity, obesity, low socioeconomic status (SES), advanced maternal age, and African-American (AA) race (3). In this mini-

review, we explore racial and SES disparities in the prevalence, morbidity, and mortality of preeclampsia.

A PRIMER ON RACE

Race is often listed as an associated risk factor for preeclampsia. However, the social sciences have shown that race is not a robust biological category but rather a social, cultural, and political concept with deep historical roots. The whimsicality of racial categories is immediately apparent in the lack of a uniform classification system. Racial categories are commonly nonuniformly based on skin color (e.g., white or Black), geographical origin (e.g., Asian), citizenship (e.g., African American), and language (e.g., Hispanic). The concept of race was socially engineered to justify inequality and rationalize the disparate treatment of conquered and enslaved peoples (4). Scientific developments in this century have demonstrated there is more genetic variation within than between racial groups (5), further illustrating the imprecision of racial categories. Nevertheless, race is often treated as a genetic risk factor, devoid of social context (6). Failing to socially contextualize data perpetuates the misunderstanding of race as a legitimate genetic proxy to explain biological differences rather than a proxy for cultural experiences, which further fuels racial bias in science and medicine. Today's literature relies heavily on racial categories (as opposed to genetic ancestry), necessitating the summary of data along these same racial categories in this mini-review. Although we summarize trends by racial categories, we also make a point of demonstrating how the imprecision of these racial categories may obscure data.

RACIAL DIFFERENCES IN PREECLAMPSIA AND PREGNANCY-RELATED MORTALITY

Incidence, Morbidity, and Mortality in Black and AA Women

Preeclampsia incidence in the United States is rising: between 1987 and 2004, there was a 25% increase in the incidence of cases (1). In the United States, preeclampsia is more prevalent among Black or AA women than white women (1, 3), and some studies have found that this Black–white gap is widening (7). The disparity in preeclampsia risk between AA women and women of other races is a robust finding (8–13). The increased incidence in AA is thought to be secondary to a variety of factors including AA women being disproportionately affected by risk factors of preeclampsia (e.g., hypertension, diabetes mellitus, obesity), SES, and unequal access to adequate prenatal care (1, 3). Preeclampsia leads to a 1.7-fold increased risk of cardiovascular-related mortality (8). Tucker et al. (14) found that even in a population in which the prevalence of preeclampsia in Black women was only slightly higher than in white women (Black-to-white ratio, 1.2), the maternal fatality rate was significantly higher in Black women (Black-to-white ratio, 2.7). The racial disparity in maternal fatality has also been documented more recently by Shahul et al. (12) in 2015 [AA women at increased risk of inpatient maternal mortality: odds ratio (OR), 2.85; 95% CI, 1.38–5.53] and by Bibbins-Domingo et al. (3) in 2017 (case fatality by AA vs white race: 73.5 vs 27.4 per 100 000 cases, respectively).

Incidence in Other Races

Some data exist on the incidence of preeclampsia in Hispanic and Asian populations in the United States. Large-scale multiethnic studies illustrate the variability within these broad racial categories, almost urging clinicians to reconsider the utility of race as a risk factor for preeclampsia (Table 1).

Within the Hispanic ethnic group, varying risks of preeclampsia have been reported; some studies show an increased risk (7, 15), whereas others show comparable or decreased risk (8, 10) compared with white women. This comparable or decreased risk of disease in Hispanics compared with non-Hispanic white women is known as the “Hispanic paradox.” However, this paradox is not universally observed and may differ by geographical context. Tanaka et al. (7) and Gong et al. (15) did not observe the Hispanic paradox in New York City. Tanaka et al. noted increased preeclampsia risk in Hispanic women compared with non-Hispanic white women, a disparity that increased between 1999 and 2002 (7). Gong et al. separated Hispanics by geographical location: Hispanic Caribbean, Central American, and South American. All 3 groups of women experienced greater risk of preeclampsia than non-Hispanic white women, with the greatest disparity experienced by Mexican women [adjusted OR (aOR), 2.9; 95% CI, 2.7–3.1], adjusting for maternal age, maternal education, parity, maternal weight, smoking, and year of delivery (15).

Several studies have shown that Asian women have a lower risk of preeclampsia than white women (8, 11, 15). However, when Asian women are subdivided by geographical origin, there is huge variability in preeclampsia relative risk (RR). RR was 0.30 in Iranian (South Central Asia), 0.70 in Chinese, and 2.0 in Filipino women compared with non-Hispanic white women (15). Nakagawa et al. (17) evaluated preeclampsia risk in women residing in Hawaii and discovered that women who were Native Hawaiian (4.6%; RR, 1.62), Pacific Islander (4.5%; RR, 1.57), and Filipino (4.6%; RR, 1.63) had higher incidence than their counterparts who were majority white (2.9%; RR, 1.00) or Chinese (2.0%; RR, 0.70). The perception that Asian women are at lower risk of preeclampsia can be detrimental to subgroups of Asians if such perceptions translate into lower screening rates and fewer outreach campaigns.

Immigration Preeclampsia Data

In New York City, both North African women (2.2%; RR, 1.10) and sub-Saharan African women (3.5%; RR, 1.75) have lower risk of preeclampsia than African-American women (4.6%; RR, 2.30) but higher risk than non-Hispanic white women (2.0%; RR, 1.0). Although foreign-born North African women had lower odds of preeclampsia than US-born North Africans (aOR, 0.6; 95% CI, 0.3–1.3), the finding was not significant (15). These limited data tentatively suggest that the US environment increases preeclampsia risk in those of African ancestry. Intriguingly, the statistically significant differences by nativity identified the same relationship: foreign-born mothers were at higher risk of preeclampsia than native-born mothers, specifically from the Philippines, the Dominican Republic, and Panama (15). The only notable differences by nativity among Asian women were observed in those from Southeast Asia and the Pacific Islands, specifically the Philippines (15).

A study of preeclampsia risk by nativity conducted across 6 industrialized nations revealed 2 trends. The first finding is that the receiving country appears to play a role in preeclampsia risk: immigrants to countries such as Australia, Denmark, and Sweden tended to have lower odds of preeclampsia than native-born individuals, whereas the opposite was true for Spain. The second finding is that immigrants from sub-Saharan Africa (OR, 1.72; 95% CI, 1.63–1.80) and Latin America/Caribbean (OR, 1.63; 95% CI, 1.57–1.69) tended to be at higher risk of preeclampsia than native-born women (19). In these studies, immigrants were compared with the "majority nonimmigrant populations" (likely white) of each respective receiving country. These results suggest that Black–white disparities also exist outside the United States.

Global Racial Disparities in Preeclampsia

Global studies on racial and ethnic disparities in preeclampsia emphasize the whimsical nature of racial categories, reflecting the different composition of minority populations in each country. In New Zealand, Chinese women (aOR, 0.56; 95% CI, 0.41–0.76) have a significantly lower risk of preeclampsia than European women, whereas the native Māori women (aOR, 1.51; 95% CI, 1.16–1.96) have a significantly higher risk compared with European women. Being of Indian descent (aOR, 1.20; 95% CI, 0.92–1.56) was not significantly related to preeclampsia risk. Chinese and Māori disparities persisted even after adjusting for risk factors such as obesity (18). In the Netherlands, Turkish women (OR, 0.32; 95% CI, 0.18–0.58) and Moroccan women (OR, 0.28; 95% CI, 0.14–0.58) had lower incidence of preeclampsia, and Cape Verdean women (OR, 2.22; 95% CI, 1.22–4.07) had higher incidence in comparison to their Dutch counterparts (20). In contrast to the use of "Asian" and "Black/African American" categories commonly used in the United States, New Zealand distinguished between Chinese and Indian, and the Netherlands distinguished among individual North African countries.

LABORATORY ANALYTES OF PREECLAMPSIA

Protein-to-Creatinine Ratio

Proteinuria has historically been a hallmark diagnostic feature of preeclampsia. The spot PCR is used to evaluate protein excretion. Bhatti et al. (21) concluded that a PCR cutoff of 30 mg/mmol would misclassify 41.4% of Black women as nonproteinuric in comparison to 22.9% of non-Black women. This would suggest that the cutoff excluded 41.4% of Black women from being diagnosed with preeclampsia, preventing them from accessing appropriate care. Proteinuria is no longer required for preeclampsia diagnosis. In 2013 and 2014, respectively, the International Society for the Study of Hypertension in Pregnancy and the American College of Obstetricians and Gynecologists revised their definitions of preeclampsia. A diagnosis of preeclampsia can now be made in the absence of proteinuria because studies of maternal and fetal risk in nonproteinuric preeclampsia cases suggested a different phenotype of preeclampsia instead of a distinctly different disease. This new definition has already increased cases of preeclampsia and superimposed preeclampsia by up to 10% (2). The increased cases would likely lead to an increase in treatment by induction of labor. An increase in induction of labor increases the risk of preterm deliveries and subsequent neonatal complications (2). This change in guidelines has expanded inclusivity,

which should enable more Black women to access a diagnosis and treatment. It remains unclear how the revised guidelines will differentially affect racial groups.

The article by Bhatti et al. (21), although well intentioned, is an example of the perpetuation of racial ideas in science and medicine. The authors note the high variability in performance of PCR to predict significant proteinuria in pregnancy and hypothesize that the inclusion of ethnicity data would improve predictive performance. They justify the inclusion of race by citing studies suggesting that Black populations excrete more urinary creatinine than similarly sized white populations—studies that are eerily similar to older studies that aimed to "scientifically prove" that Black bodies were biologically different from white bodies. Moreover, the inclusion of women of mixed ethnicity with white women based on similar proteinuria levels affirms the arbitrariness of their Black versus non-Black comparison and inappropriately conflates proteinuria levels with race. Their discussion implies that racial differences are due to inherent biology (suggestive of genetic determinism) rather than biology resulting from different sociocultural experience (e.g., racist oppression, structural violence of Jim Crow laws and slavery).

Placental Growth Factor

Circulating angiogenic factors have been considered as clinical markers for the onset of preeclampsia. Low placental growth factor (PlGF) is suggestive of early onset preeclampsia; as such, evaluation of PlGF can be utilized as an early screening tool for preeclampsia before the onset of clinical features. However, Yang et al. (22) demonstrated that the association of low PlGF levels with the development of preeclampsia may be weaker in Black women (aOR, 0.219; 95% CI, 0.12–0.39) compared with white women (aOR, 0.048; 95% CI, 0.03–0.09), and Hispanic women (aOR, 0.028; 95% CI, 0.01–0.06). The aORs in white and Hispanic women were >4.4 times the aOR in Black women. In addition, soluble vascular endothelial growth factor receptor-1 levels were associated with early onset preeclampsia in white and Hispanic women but not in Black women (22). The authors suggest that each racial-ethnic group (i.e., heterogenous white, Black, Hispanic) may have its own pathophysiological mechanisms underlying early onset preeclampsia, without any sociocultural contextualization.

Nkx2–5

A novel study evaluating the association of placental Nkx2–5 (cardiovascular development transcription factor) with preeclampsia demonstrated that Nkx2–5 is expressed in a racially disparate manner, with a greater prevalence in white women (23). In correlation with soluble fms-like tyrosine kinase 1, the increase of Nkx2–5 was strongly correlated with preeclampsia in white women but not in African-American women. The authors argue that this result demonstrates possible differential contributions or gene effects responsible for preeclampsia that are not governed by Nkx2–5 regulation. The classification of race is poorly defined, and racial differences cannot be concluded from the small sample size (n = 33). Nkx2–5 laboratory assays are not readily available and thus have not been introduced into routine patient care.

Small-Nucleotide Variants

It is important to carefully consider how race is operationalized when investigating genetic etiologies of preeclampsia. Studies of human genetic variation have shown that phenotypic racial classifications (i.e., skin color) are inadequate and that individual ancestry is a more important consideration for biomedical studies (5). The limitations of cultural categories of race are revealed in the literature on rs7579169 and preeclampsia risk. The minor allele T of single-nucleotide polymorphism rs7579169 near the inhibin, beta B gene was first identified in an Australian cohort of "confirmed-Caucasian ancestry" to increase the risk of preeclampsia (OR, 1.57; 95% CI, 1.32–1.87) (24). It is unclear how the word "Caucasian" is being used in this context and exactly how this classification was extracted from the medical records (suggestive of self-report rather than genetic ancestry). The term "Caucasian" technically refers to peoples from the Caucasus region (modern-day Armenia, Azerbaijan, Georgia, Russia) but is commonly and erroneously used as a term synonymous to "white Europeans" (25). The relationship between rs7579169 and preeclampsia failed to replicate in a Norwegian cohort and a Finnish cohort (24) but was replicated in a Polish cohort (26). The race of participants was not specified in any of these 3 cohorts, so the veracity of assuming a homogeneously white population is questionable. In the Han Chinese, there is also no consensus on how rs7579169 affects preeclampsia risk, with one study finding that the CT genotype (OR, 1.76; 95% CI, 1.07–2.87) and the TT genotype (OR, 5.03; 95% CI, 1.99–12.73) increase preeclampsia risk (27) but another study finding that the C allele (OR, 1.44; 95% CI, 1.05–1.98) increases preeclampsia risk (28). Genetic studies must carefully consider how race is used and avoid conflating racial differences with genetic differences.

OTHER DISPARITIES IN PREECLAMPSIA

Socioeconomic Disparities

Several studies suggest that the noted disparities between AA women and their counterparts may be due to SES and healthcare access issues, which are often compounded for lower income minority women (11). However, recent studies have demonstrated that increases in financial stability do not confer the same protective effects against preeclampsia in AA women as in other races (7, 16, 29). In studies that looked at the role of income and race on preeclampsia risk, AA women did not benefit from the protective effects (longer gestational length or reduced preeclampsia risk) garnered by higher SES as women of other races (16, 30). In these studies, AA women developed preeclampsia at higher rates than white women, independent of education (OR, 1.56; 95% CI, 1.48–1.64) or insurance status (OR, 1.55; 95% CI, 1.48–1.63) (16). Even among women who are relatively low income, AA women who earn higher incomes within this subset have higher postpartum cardiometabolic risk compared with their poorer AA counterparts and all Hispanic and white women (29). Tanaka et al. (7) also demonstrate that the protective effects of a higher income were seen only in Hispanic women and not AA women. These studies contribute to the growing body of evidence supporting the phenomenon known as "diminishing returns" and "reverse gradient," in which higher SES does not provide the same health outcome benefits in AA as in white patients. Some explanations for this phenomenon may be that AA women with higher SES are exposed to the added stress of combatting racism in a white-dominated cultural space.

SES may affect preeclampsia risk through psychosocial mechanisms. A meta-analysis found that maternal stress is associated with increased preeclampsia risk (OR, 1.49; 95% CI, 1.27–1.74; $P < 0.001$), as was work-associated stress (OR, 1.50; 95% CI, 1.15–1.97) (31). This point is important because economic disparities by race are well documented. Moreover, perceived racial discrimination is associated with hypertensive status (32). Chronic hypertension is a strong risk factor for preeclampsia (OR, 10.2; 95% CI, 7.0–14.9) and, when combined with high maternal lifetime stress, dramatically increases the risk of preeclampsia (OR, 21.3; 95% CI, 10.2–44.3) (33).

Obesity

Obesity is associated with higher rates of preeclampsia, and minority communities experience disproportionate rates of obesity. In a study evaluating preeclampsia rates in Hawaii, Nakagawa et al. (17) reported higher rates of preeclampsia in women who were obese native Hawaiian (OR, 1.80; 95% CI, 1.24–2.60), other Pacific Islander (OR, 1.68; 95% CI, 1.14–2.49), and Filipino (OR, 1.64; 95% CI, 1.04–2.59) compared with white. In the United States, there are notably higher rates of obesity among Black pregnant women (12). An Australian study evaluating the effect of obesity on pregnancy found that obese mothers were more likely to have a low SES, chronic hypertension, and greater odds of preeclampsia (aOR, 3.143; 95% CI, 1.694–5.830) compared with normal-weight mothers (34). Interventions to address obesity, such as eliminating food deserts, increasing access to out-door exercise facilities, and healthcare access to treat long-term health conditions before the onset of pregnancy may help address the disparities seen in obese women with preeclampsia.

Access to Care

Medicaid covers approximately half of pregnancies in the United States (35). Although pregnancy-related hypertension is similar between women with private and public (i.e., Medicaid) insurance, women who obtain public insurance have more severe hypertension (systolic BP ≥160 mmHg; 42.6% vs 33.2%, $P = .001$) (36). These findings were further supported by Webster et al. (37), who showed that AA women were more likely to have higher diastolic BP at their prenatal visit (88 vs 85 mmHg; $P = 0.03$). Before and during pregnancy, AA women are more likely to have chronic hypertension (8, 38, 39). AA and Hispanic women were more likely to receive healthcare through public insurance and to live in lower income neighborhoods (9). Brown et al. (13) reported that despite the comparable rates of poverty and Medicaid insurance status between Hispanic and AA women, AA women still had higher rates of preeclampsia than women of other races. Although these studies demonstrate that the differences in preeclampsia between AA and Hispanic women are not entirely explained by poverty or insurance status, this finding should not be interpreted as support for underlying genetic differences between races.

CONCLUSIONS

The personal stories of prominent AA celebrities such as Serena Williams and Beyoncé Knowles have helped shed light on the persistence of racial disparities in pregnancy outcomes, even among women who have wealth, privilege, and access to world-class

healthcare. Racial disparities in preeclampsia exist globally and may actually be increasing. To end racial disparities, we must understand and accurately identify them.

The first takeaway is to carefully consider how race is defined and used in a study. We have shown that incidence rates of preeclampsia differ not only by race but also within a race. Familiar racial categories are actually quite heterogeneous. The second takeaway is to distinguish between “the biology of race” and the “biology of racism.” The former is derived from a history of colonial eugenics that equates racial differences with genetically determined biology, even though genetic variation does not match racial categories. The latter is a recognition that our societal history of racism and structural violence may become biologically embodied, such that SES, differences in risk factors (e.g., obesity rates, hypertension rates), and differential access to care cannot entirely explain away racial disparities in preeclampsia. This mindset enables us to think more critically about how to apply laboratory medicine to rectify racial disparities. It reminds us to be cautious of calls for race-based cutoffs; even if laboratory tests or screen cutoffs could be personalized by race, how can “race” be operationalized? Or does precision medicine only need be as precise as “Black,” “white,” and “Hispanic” in a world with ever increasing genetic admixture?

This year marks the end of Healthy People 2020, a set of nationwide health-promotion and disease-prevention goals from the US Department of Health and Human Services (40). One of the overarching goals is the achievement of health equity through the improvement of health in all groups and will likely be renewed for Healthy People 2030. To achieve this goal, we must improve our understanding of racial disparities through better and more thoughtful research design and discussions about the intersectionality of race and medicine.

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IMPACT STATEMENT

This article provides a broad overview of racial disparities in preeclampsia incidence, morbidity, and mortality and explores potential mechanisms of action. We focus on the nuances of race and provide critical assessment of how laboratory studies to date have used race to understand the pathophysiology of preeclampsia. This article will be a useful reference for those who seek to study and understand the biology underlying racial disparities.

Table 1.

Relative risk of preeclampsia by race and ethnicity in the United States.

Race	Relative Risk[a]	References
White	1.0 (Reference)	
Black or African American	1.64 (1.24–2.30)	(7, 8, 11–16)
North Africa	1.10	(15)
Sub-Saharan Africa	1.75	(15)
Ivory Coast	2.10	(15)
Guinea	1.35	(15)
Hispanic	1.03 (0.69–1.5)	(7, 8, 11–13)
Hispanic Caribbean	1.95	(15)
Dominican Republic	2.10	(15)
Cuba	1.25	(15)
Mexico	2.50	(15)
South America	1.65	(15)
Ecuador	1.70	(15)
Argentina	1.30	(15)
Central America	1.85	(15)
Asian	0.82 (0.68–0.96)	(8, 11)
East Asia	0.70	(15)
China	0.63 (0.48–0.70)	(15, 17, 18)
Southeast Asia/Pacific Islands	1.65	(15)
Philippines	1.82 (1.63–2.00)	(15, 17)
South Central Asia	1.10	(15)
Iran	0.30	(15)
Multiracial/Other	1.25 (1.15–1.39)	(7, 8, 17)

[a]Relative risk mean (range) are shown only if multiple studies are represented.