Does race or ethnicity play a role in the origin, pathophysiology, and outcomes of preeclampsia? An expert review of the literature

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Introduction

Racial and ethnic disparities in outcomes are pervasive in obstetrics.\(^1\)–\(^4\) Black women have the highest rates of severe maternal mortality compared to other racial and ethnic groups.\(^5\) In the most recent maternal mortality report, the Centers for Disease Control and Prevention confirms that Black women continue to have a maternal mortality rate 2.5 to 3 times higher than White women (14.7 vs 37.1 deaths per 100,000 live births).\(^6\) As we are increasingly more informed on how race is a proxy for life experience\(^7\) and structural and societal racism\(^8\)–\(^11\), the burden of preeclampsia, a substantial contributor to perinatal morbidity and mortality, is not born equally across the population.

The burden of preeclampsia, a substantial contributor to perinatal morbidity and mortality, is not born equally across the population. Although the prevalence of preeclampsia has been reported to be 3% to 5%, racial and ethnic minority groups such as non-Hispanic Black women and American Indian or Alaskan Native women are widely reported to be disproportionately affected by preeclampsia. However, studies that add clarity to the causes of the racial and ethnic differences in preeclampsia are limited. Race is a social construct, is often self-assigned, is variable across settings, and fails to account for subgroups. Studies of the genetic structure of human populations continue to find more variations within racial groups than among them. Efforts to examine the role of race and ethnicity in biomedical research should consider these limitations and not use it as a biological construct. Furthermore, the use of race in decision making in clinical settings may worsen the disparity in health outcomes. Most of the existing data on disparities examine the differences between White and non-Hispanic Black women. Fewer studies have enough sample size to evaluate the outcomes in the Asian, American Indian or Alaskan Native, or mixed-race women. Racial differences are noted in the occurrence, presentation, and short-term and long-term outcomes of preeclampsia. Well-established clinical risk factors for preeclampsia such as obesity, diabetes, and chronic hypertension disproportionately affect non-Hispanic Black, American Indian or Alaskan Native, and Hispanic populations. However, with comparable clinical risk factors for preeclampsia among women of different race or ethnic groups, addressing modifiable risk factors has not been found to have the same protective effect for all women.

Abnormalities of placental formation and development, immunologic factors, vascular changes, and inflammation have all been identified as contributing to the pathophysiology of preeclampsia. Few studies have examined race and the pathophysiology of preeclampsia. Despite attempts, a genetic basis for the disease has not been identified. A number of genetic variants, including apolipoprotein L1, have been identified as possible risk modifiers. Few studies have examined race and prevention of preeclampsia. Although low-dose aspirin for the prevention of preeclampsia is recommended by the US Preventive Service Task Force, a population-based study found racial and ethnic differences in preeclampsia recurrence after the implementation of low-dose aspirin supplementation. After implementation, recurrent preeclampsia reduced among Hispanic women (76.4% vs 49.6%; \(P\ < .001\)), but there was no difference in the recurrent preeclampsia in non-Hispanic Black women (13.7 vs 18.1; \(P\ = .252\)). Future research incorporating the National Institute on Minority Health and Health Disparities multilevel framework, specifically examining the role of racism on the burden of the disease, may help in the quest for effective strategies to reduce the disproportionate burden of preeclampsia on a minority population. In this model, a multilevel framework provides a more comprehensive approach and takes into account the influence of behavioral factors, environmental factors, and healthcare systems, not just on the individual.

Key words: diabetes, DNA variant, ethnicity, fetal death, genetic susceptibility, health disparities, health equity, HELLP syndrome, hypertension, internalized racism, long-term implication, low birthweight, morbidity, mortality, personally mediated discrimination, postpartum preeclampsia, preeclampsia, preterm birth, prevention, race, structural racism

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that directly affects women of different races and ethnicities, we must also examine maternal health outcomes from a life course perspective. The purpose of this expert review is to examine the existing literature on the influence of race and ethnicity in the origin, pathophysiology, and outcomes of preeclampsia.

**Race and Ethnicity: Categories, Definitions, and Limitations**
This review will use the Office of Management and Budget’s categorizations for race and ethnicity (Figure 1). Although there are considerable limitations to this method, this classification system has been primarily used in the medical literature, because it has been historically used in the US census and population-level data. Race is defined with at least the following 5 minimum standards: American Indian or Alaskan Native, Asian, Black or African American, Native Hawaiian or Other Pacific Islander, and White. Ethnicity is defined dichotomously as Hispanic or Latino or non-Hispanic.

The major limitation of the use of race and ethnicity in medical research is that race is a socially derived label that can either be self-reported or assigned. The assignment of one’s race or ethnicity is not rooted in scientific evidence to justify biological or genetic differences. Human population studies continue to find more genetic variations within racial groups than among them. For these reasons, the validity of race as an indicator of distinct, genetically different population groups has been questioned. Many researchers have shifted paradigms, defining race as a social construct based on phenotypic and genetic expressions rather than as a biological construct. In addition, many individuals self-identify with more than one racial or ethnic group, which can be difficult to ascertain in research when choices may not be adequately comprehensive. Finally, although ethnicity in medical research has traditionally been a dichotomy of Hispanic and non-Hispanic, ethnicity has also been described as attributable to one’s culture, language, country of origin, or traditions, which can leave out populations of interest when these categories are defined by investigators for a scientific inquiry.

Using the existing categorizations of race and ethnicity in medical research and literature is limited by the assignment of race (self-identified vs researcher- or healthcare worker—assigned) and failure to differentiate subgroups (eg, Asian race includes Indian, Samoan, Japanese, and Chinese individuals). Across the United States, studies vary in the amount of racial and ethnic diversity. Most studies that address the disparities focus on Black and White populations. Data on Asian, American Indian or Alaskan Native, and mixed-race individuals are often limited because of the small numbers of poorly designed studies that collapse them into the “other” racial groups.

**Race, Racism, and Preeclampsia**
There are 3 domains or types of racism—structural or institutionalized racism, personally mediated or individual-level discrimination, and internalized racism—that have all been recognized as mediators of health outcomes. Structural racism is defined as “the totality of ways in which societies foster racial discrimination through mutually reinforcing systems of housing, education, employment, earnings, benefits, credit, media, healthcare, and criminal justice.” Increasing the measurements of structural racism, defined most frequently in research as racial inequity (ratio of Black to White population estimates) in educational attainment, median household income, employment, imprisonment, and juvenile custody, was associated with a 5% increase in Black infant mortality (relative risk, 1.05; 95% confidence interval [CI], 1.01–1.10). Black women from an urban population were found to have increased rates of adverse pregnancy outcomes with increasing rates of neighborhood segregation.

Personally mediated racism has also been linked to adverse pregnancy outcomes with 36.9% of Black women (95% CI, 32.9–40.9) and 5.5% of White women (95% CI, 4.5–6.5) reporting chronic worry about racial discrimination. Because it relates to hypertensive diseases, it has been posited that ethnic differences in elevated blood pressures in response to the chronic stress from the experience of racial discrimination contribute to poorer cardiovascular health for Black women and men. The studies to date that have examined the association between preeclampsia and race have mostly grouped preeclampsia with other conditions under the category of hypertensive disorders of pregnancy, thereby limiting our data on preeclampsia itself. To elucidate an answer, researchers need to consider the National Institute on Minority Health and Health Disparities Research Framework (Figure 2).

Traditionally, preeclampsia research focused on the biological influence on an individual level. A multilevel approach would be more comprehensive and take into account the influence of behavioral factors, environmental factors, and healthcare systems, not just the factors...
related to the individual. Including a focus on the interpersonal, community, or a societal influence would be more comprehensive because it would account for cumulative or interactive effects of multiple determinants.29

Finally, the behaviors of healthcare professionals should be examined with a critical lens and examine how medical systems perpetuate racism.30 This can take the form of implicit biases about groups that may lead to differential treatment. In a group of medical students, 73% endorsed at least 1 false belief related to the biological differences in pain between people of Black race and people of White race and more often reported lower pain ratings for Black (vs White) targets.31 Racial inequities in the administration of epidural analgesia32 and then experience, assessment, and treatment of postpartum pain have also been identified.33

The health equity framework integrates the individual-level biomedical and behavioral causes of a maternal disease, with the population-level differences in the sociocontextual environment, which might be the drivers of disparities in disease rates among groups (Figure 3). In the framework, social determinants of health contribute to the circumstances around health, economic opportunity, and chronic stressors. Differences in exposure and opportunities then contribute to disparate outcomes. As we investigate ways to reduce the disparities in outcomes, a health equity framework will help address the drivers more comprehensively. This can take the form of years of oppression that causes excess stress on the body or weathering that can affect one’s health.34,35 It could also be the implicit or explicit biases acted on by healthcare providers that result in inequitable treatment.31,33,36

The Role of Race and Ethnicity on the Prevalence of Preeclampsia

The prevalence of preeclampsia is reported to be 2% to 8%.37 An increased rate of preeclampsia among non-Hispanic Black women compared with non-Hispanic White women has been found in a number of studies.38,39 and Black race has been cited as a risk factor for preeclampsia.40—42 In a study using
data from the National Inpatient Sample (NIS), the largest publicly available all-payer hospital inpatient care database in the United States, 4.7% of the 177,000 deliveries included were complicated by preeclampsia.43 Black women experienced preeclampsia or eclampsia in 69.8 of every 1000 deliveries, compared with 43.3 per 1000 deliveries in White women, 46.8 per 1000 deliveries in Hispanic women, 28.8 per 1000 deliveries in Asian or Pacific Islander, and 46.6 per 1000 for all women, overall.43 Preeclampsia is understudied among the American Indian and Alaskan Native populations, but the prevalence is estimated to be 7% to 11%.44 Higher rates of preeclampsia have also been found in the American Indian and Alaskan Native populations compared with non-Hispanic White women (odds ratio [OR], 1.17; 95% CI, 1.06–1.29).44 The sum of the data appears to indicate an increased prevalence of preeclampsia among Black and American Indian or Alaskan Native women. Racial differences have also been reported in the timing of preeclampsia diagnosis (early vs late gestation). In a cohort of 9149 women who were prospectively evaluated for preeclampsia risk factors, compared with their White counterparts, women who self-identified as a member of the Black race were more likely to develop early preeclampsia (<34 weeks’ gestation) (OR, 3.64; 95% CI, 1.84–7.21) and late preeclampsia (≥34 weeks gestation) (OR, 2.97; 95% CI, 1.98–4.46).45 Women who self-identified as Indian or Pakistani (OR, 2.66; 95% CI, 1.29–5.48) or mixed race (OR, 3.31; 95% CI, 1.55–7.06) were more likely than their White counterparts to develop late preeclampsia.45

The prevalence of new-onset postpartum hypertensive disorders has reported rates ranging from 0.3% to 27.5%, with Black race and Latino ethnicity described as risk factors.46 A recent retrospective cohort study indicated that women with new-onset postpartum preeclampsia (n=121) were more likely to be of non-Hispanic Black race (31.4% vs 18.0%; P<.01).47 Postpartum preeclampsia contributes to hospital readmission rates, severe maternal morbidity, and mortality.48 Given that more than one-third of maternal deaths occur in the postpartum period, a focus on postpartum preeclampsia is imperative to decrease the US maternal mortality rates.49

One explanation for the increased prevalence of preeclampsia among Black women has been postulated to be related to the increased incidence of chronic hypertension in that population, resulting in misclassification of chronic hypertension as solely pregnancy-related hypertension, or to the increased risk of developing preeclampsia or eclampsia or pregnancy-aggravated hypertension among women with chronic hypertension.50 An observational study of 101 women appears to challenge that theory.51 Among women without chronic hypertension, Black
women were more likely to be diagnosed with preeclampsia (78% vs 53%; \( P = 0.04 \)) and more likely to have had systolic blood pressures of \( > 160 \) mm Hg (43% vs 17%; \( P = 0.01 \)). In addition, this study found that Black multiparous patients with chronic hypertension were more likely than White multiparous patients to receive a diagnosis of preeclampsia (80% vs 27%; \( P = 0.01 \)).

Obesity, \(^5\) sleep-disordered breathing, \(^3\) and diabetes, \(^4\) all risk factors for preeclampsia, are more prevalent among Black women and American Indian or Alaskan Native women. \(^38\) Using Washington state hospital–linked birth discharge data, investigators found an association between the American Indian or Alaskan Native race and preeclampsia after adjusting for sociodemographic and clinical confounders (OR, 1.17; 95% CI, 1.06–1.29). However, after adjustment for body mass index, the odds of preeclampsia (OR, 1.05; 95% CI, 0.95–1.16) was no longer statistically significant. \(^4\) These findings are aligned with other more recent data that indicate obesity as a risk modifier may depend on race or ethnicity. In a birth certificate study, it was found that the effects of maternal race, ethnicity, and obesity were not uniform, varying among racial groups and also by the specific outcome being analyzed. \(^32\) In a study examining the relationship between severe preeclampsia and preexisting medical comorbidities, the effect of comorbidities on preeclampsia risk was least pronounced in Hispanic women and most pronounced in non-Hispanic Black women with similar comorbidities. This effect persisted in the presence of adverse sociodemographic factors in both groups and is consistent with what has been labeled the “Hispanic paradox.” \(^57\) The “Hispanic paradox” is a term used to describe the epidemiologic mystery of why Hispanic individuals in the United States live longer than their White counterparts despite generally lower socioeconomic status and healthcare access. \(^59\) Over time, it has also been used to describe scenarios where other health outcomes are better than expected among Hispanic women. \(^57\) \(^60\)

**The Role of Race and Ethnicity on the Pathophysiology of Preeclampsia**

Abnormalities of placental formation and development, \(^31\)–\(^63\) immunologic factors, \(^64\),\(^65\) vascular changes, \(^66\) and inflammation have all been identified as contributing to the pathophysiology of preeclampsia. \(^67\) There has been a desire to investigate genetic causes of preeclampsia. Observations that led to a suggestion of a genetic component to preeclampsia are as follows: (1) studies that found an increased risk of recurrence of preeclampsia in subsequent pregnancies, \(^68\)–\(^70\) (2) studies that observed an increased risk in first-degree relatives of women with preeclampsia, \(^71\),\(^72\) and (3) familial studies that found that a family history of hypertension and cardiovascular disease is associated with the occurrence of preeclampsia. \(^73\),\(^74\) Preeclampsia manifests as complex phenotypes, resulting from both maternal and fetal genetic features. \(^75\) Candidate genes have been investigated, but much of the focus of racial differences has been on African ancestry.

Diminished or aberrant human leukocyte antigen G expression patterns may contribute to the development of preeclampsia. \(^76\) Recent studies of women identified as having African ancestry or being a member of the Black race observed an increased risk of preeclampsia when the mother carried the 1597C allele. In contrast, the presence of the 1597C allele in the fetus was not associated with preeclampsia risk. \(^76\) Another effort to explain the higher rates of preeclampsia among Black women has focused on the finding that common coding variants in the apolipoprotein L1 gene (APOL1) are potent risk factors for a spectrum of kidney diseases in Black Americans. Investigators hypothesized that APOL1 variants play a role in the excess risk for preeclampsia among Black women. \(^77\) The study included 121 infants born to women with preeclampsia at 1 center, and 886 women with and without preeclampsia at a second center. The investigators concluded that fetal APOL1 high-risk genotype was associated with the development of preeclampsia in both centers with an OR of 1.84 (95% CI, 1.11–2.93) and 1.92 (95% CI, 1.05–3.49). Maternal APOL1 did not indicate the same effect. \(^78\) Researchers theorized that the presence of a high-risk variant of APOL1 protein in the fetus may adversely affect the placental function leading to preeclampsia.

Soluble fms-like tyrosine kinase (sFlt1), a soluble-deactivating receptor for the vascular endothelial growth factor (VEGF) and the placental growth factor, has been implicated in the pathogenesis of preeclampsia. \(^79\) Both VEGF and placental growth factor are potent stimulators of angiogenesis and regulators of endothelial function. \(^79\) In a study of more than 600 women, the association between the genetic variation in 6 angiogenesis pathway genes and preeclampsia was investigated in White and Black women from our large case-control study. \(^80\) Notably, 3 single nucleotide polymorphisms (SNPs) in Black women and a different 3 SNPs in White women were associated with preeclampsia. \(^80\) However, the allelic variants that were associated with preeclampsia in each racial group were not associated with differences in the serum sFlt levels within the cases and control subjects in those racial groups. \(^80\)

Older studies focused more on oxidative stress and inflammatory pathways with inconsistent results. \(^81\)–\(^85\) In a systematic review of 35 studies looking at the endothelial nitric oxide synthase polymorphism and preeclampsia risk stratified by ethnicity, a higher risk of preeclampsia was observed in the White or mixed-race populations with the 894 guanine> adenine polymorphism but not in those of Asian or African descent. \(^81\) Another systematic review and meta-analysis based on 21 studies investigated the role of interleukin-10 (IL-10) polymorphisms and preeclampsia risk. IL-10–1082 guanine> adenine polymorphism stratified by ethnicity found a markedly increased risk of developing preeclampsia in Asian and mixed-race populations but not in White populations. \(^82\) Although some racial and ethnic trends in gene expression have
Preeclampsia and its related morbidity are substantial contributors to pregnancy-related deaths in the United States. The presentation and outcomes of preeclampsia have also been disparate among racial and ethnic groups. In a study of 473 women with severe preeclampsia, non-Hispanic Black women were more likely to have severe hypertension at presentation (45% vs 32% and 27%; P<.005) and require antihypertensive medications (45.8% vs 36.8% and 25%; P=.01) than White or Hispanic women, respectively. White women had a more frequent diagnosis of HELLP (hemolysis, elevated liver enzymes, and low platelet count) syndrome than Black women (30 vs 19; P=.03). In another study of similar size, the increased risk was not confirmed.

The infants of Black women also do not fare well. Black infants of women with preeclampsia are more likely than their White counterparts to be very low birthweight and very preterm (OR, 3.77 [95% CI, 2.77–5.13], and OR, 3.66 [95% CI, 2.66–5.03], respectively). Black women with preeclampsia are also more likely to have fetal demise. In a study of 3921 women, despite similar severity of preeclampsia, Hispanic women had lower rates of preeclampsia-related infant mortality, consistent with the “Hispanic paradox.”

Examining the outcomes of the more uncommon complications has been challenged by the use of administrative databases in which the diagnosis of preeclampsia and race and ethnicity may lack specificity. In a study using the NIS, a large publicly available all-payer inpatient care database in the United States, non-Hispanic Black women when compared with non-Hispanic White, Hispanic, and all other women, had a higher rate of preeclampsia-related severe maternal morbidity, including stroke (17.1 vs 6.5, 12.7, and 9.3 per 10,000 deliveries, respectively; P<.01), and pulmonary edema or heart failure (56.2 vs 32.7, 30.2, and 38.4 per 10,000 deliveries, respectively; P<.01). The disparate outcomes persist into the postpartum period. Black women were more likely to have hospital readmission for cardiovascular disease diagnosis (6.8 vs 1.7 vs 1.0 per 1000 deliveries, respectively; P<.001) than White women. Black women diagnosed with preeclampsia with severe features have lower rates of postpartum follow-up than women of other racial or ethnic groups (47.0% vs 70.5%; P=.007).

For women with preeclampsia, the risk of maternal mortality is higher for Black women than all other groups (121.8 per 100,000 deliveries [95% CI, 69.7–212.9] vs 24.1 per 100,000 deliveries [95% CI, 14.6–39.8], respectively; P<.01). Most concerning was the fact that 60% of the maternal deaths are preventable and Black women are more likely to have a preventable death. Targeted efforts to address preeclampsia reduction in Black women would significantly move the needle in the fight against US maternal mortality for all women.

Survivors of preeclampsia face a 2-fold increased risk of cardiovascular disease, and that risk can be seen within 3 to 5 years after delivery. Black women are more likely to have long-term consequences of preeclampsia, including chronic hypertension and cardiovascular disease progression. Obesity, chronic hypertension, sleep-disordered breathing, and the development of diabetes have been implicated. However, there seem to be short-term differences in the manifestations of the cardiovascular changes after the development of preeclampsia.

In a cohort of 29 matched case-control pairs of Black women, there was more abnormal cardiac function as evidenced by worse cardiac systolic function (longitudinal strain), increased chamber stiffness (end-systolic elastance), and worse diastolic function as measured by noninvasive echocardiographic tissue Doppler assessment in preeclampsia cases compared with controls. These findings persisted 4 to 12 weeks after delivery, and worse diastolic function and increased arterial stiffness were noted in the postpartum period. These cardiac changes and propensity to hypertension in the postpartum period are further compounded by low postpartum follow-up rates. However, novel use of text messaging was associated with a higher adherence rate across all racial and ethnic groups. It appeared to close the disparity gap in postpartum blood pressure management. Further studies are needed to examine the trajectory of cardiovascular diseases to identify potential therapeutic targets and models of care that will address the disparities. Left undeterred, there will continue to be substantial disparities in cardiovascular diseases among different ethnic or racial groups.

The US Preventive Services Task Force recommended low-dose aspirin for the prevention of recurrent preeclampsia in 2014. A single-center study evaluating the population-based effect of this recommendation on the prevalence of recurrent preeclampsia found differences among racial and ethnic groups. Although the relative proportion of Hispanic women who experienced recurrent preeclampsia after the implementation of low-dose aspirin was lower in the post-aspirin group (76.4% vs 49.6%; P<.0001), there was no difference in recurrent preeclampsia in non-Hispanic Black women (13.7 vs 18.1; P=.252).

Other therapeutic agents used to prevent preeclampsia, which have been investigated and remain controversial, include heparin, calcium supplementation, and folic acid. There are insufficient data on racial or ethnic differences to comment on disparities in safety, efficacy, or outcomes.

Weight loss, particularly through bariatric surgery, has been associated with a reduction in recurrent preeclampsia and adverse pregnancy outcomes.
outcomes in most but not all studies. Data on disparities in outcomes and effectiveness are limited. However, given the well-documented disparities in access to obesity treatment and uptake of bariatric surgery, there would need to be careful consideration of the sample size needed for a meaningful analysis.

Finally, although socioeconomic status (SES) has been implicated as a modifiable risk factor for adverse birth outcomes, this relationship has been called into question. In a cohort of 718,604 Black and White women drawn from a population-based California cohort, high SES in White women was associated with a decreased risk of preeclampsia. However, Black women continued to have a higher risk of developing preeclampsia independent of education (OR, 1.56; 95% CI, 1.48–1.64) or insurance status (OR, 1.55; 95% CI, 1.48–1.63). This phenomenon of Black women not receiving the same protective benefit from the improved sociodemographic risk factors has been labeled Minorities’ Diminished Returns (MDR). MDR can be defined as the observation of smaller health gains from SES indicators such as education attainment among ethnic minorities compared with the majority group. These findings should not discourage providers from supporting all patients in risk-reducing behaviors. However, they should be aware that even in the absence of these sociodemographic risk factors some racial or ethnic groups continue to be at a higher risk of preeclampsia.

Future Research and Solutions
To continue to decrease health disparities, researchers and clinicians should continue to systematically and reliably include race and ethnicity in their studies. Researchers should acknowledge the limitations of race and ethnicity as a scientific variable when critiquing their study design. Kaplan and Bennett outline some of the challenges in race and ethnicity research—including the importance of distinguishing between a risk factor and a risk marker. However, it is essential that it is done so in a way that does not perpetuate racism. Finding an association between a disease and race and ethnicity does not imply causality. It should prompt us to inspect the underlying intersectionality of health disparities, including, but not limited to, the social context of one’s community, socioeconomic factors, and environmental exposures. Finally, translational and clinical research dedicated to health disparities should consider how to ask questions that are not only health equity—focused but also solution-oriented, such as the utilization of the public health critical race methodology. Despite the pervasive reports of disparities, strategies to reduce them remain limited in a disease that disproportionately affects the short-term and long-term health of minority populations. Considerations for research priorities are listed in the Table.

Comment
Racial and ethnic disparities are commonly reported in the preeclampsia literature, and people of Black and American Indian or Alaskan Native races are widely reported to have a higher risk of preeclampsia. However, the contemporary understanding of race as a social construct as opposed to a biological or genetic factor points to race as a risk marker. Differential experiences based on race and ethnicity influence the origins and outcomes of the disease, leading to disparate outcomes. There remain substantial gaps in the literature, most prominently in the strategies to reduce disparities in preeclampsia management and outcomes. Studies of interventions should include a multilevel approach that accounts for the dynamic interplay of multiple levels of influence to be effective. Continued research should be made looking at how one’s experience has generational effects on their and their family’s health outcomes. As stated previously, in discussions about health disparities by racial group, it is imperative to separate the social construct of race from what is driving a lot of the observed differences, which is racism.


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NIMHD, National Institute on Minority Health and Health Disparities.


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