

Editorials

Fallacies and Dangers of Practicing Race-Based Medicine

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Race does not have a biologic definition, but rather a political and social one that is fluid and independent of science.^{1,2} Nevertheless, the medical community uses race as a scientific variable in several prominent clinical situations, including calculation of atherosclerotic cardiovascular disease (ASCVD) risk and glomerular filtration rate (GFR), decisions on pharmacotherapy for hypertension, and interpretation of pulmonary function testing. These diagnostic and clinical prediction tools do not recognize the social and environmental factors that influence racial differences in health outcomes. Furthermore, they do not highlight the potentially harmful effects of using race as a factor in making medical decisions. Although we may hear about biologic or genetic differences between races, there is more variation *within* races than there is *between* them.³

When using identical blood pressure and lipid data in the ASCVD Pooled Cohort calculator, a 40-year-old White male smoker has a lower cardiovascular risk than a 40-year-old Black male nonsmoker. Without considering lifestyle, drug use, or the significant heterogeneity in the Black population, this decision-making tool lumps all Black people together and determines that being a Black man is more dangerous than smoking.

The guidelines from the Joint National Committee on the management of high blood pressure recommend against using monotherapy with angiotensin-converting enzyme (ACE) inhibitors in the treatment of Black patients with hypertension.⁴ This recommendation was heavily influenced by the findings of the Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT), which showed worse cardiovascular outcomes for Black patients treated with lisinopril.⁵ This is sometimes translated into the idea that ACE inhibitors are ineffective in Black people, despite the fact that these questionable findings have not been shown in Black patients treated with combination therapy.^{6,7}

Even taken at face value, a racial difference in the effectiveness of ACE inhibitor monotherapy is explained by social factors, not genetic causes. More than a decade ago, the American Heart Association published a report from the International Society on Hypertension in Blacks that linked poor blood pressure control in Black patients to nonphysiologic factors, such as high stress levels, less physical activity,

higher rates of obesity, and low-potassium diets.⁸ Racial differences in renin-angiotensin activity resolve when Black patients decrease sodium intake, and there is more variation in renin-angiotensin activity within the Black race than there is when comparing Black and White people.⁸ Treating Black patients as a monolithic group stereotypes them without considering that they are not all obese and that many follow the Dietary Approaches to Stop Hypertension (DASH) diet and exercise regularly.

Despite Black patients having worse outcomes for chronic kidney disease, race-based equations assign a higher GFR (i.e., better kidney function) to Black patients with the same creatinine value as people of other races. This is based on questionable racial differences in muscle mass, bone density, and nutritional status.⁹⁻¹¹ As is the case with hypertension, these racial differences are often presented without considering nonbiologic causes or the significant variation within individual races.

As one expert asserted, the use of race-based GFR equations will “systematically miss a high-risk group of blacks at a time in the disease course when interventions are crucial.”¹² Inflating GFR in Black patients leads to underdiagnosis of chronic kidney disease in this population and makes it less likely that they will be referred to a nephrologist or become eligible for kidney transplantation. Additionally, the erroneous beliefs about lower effectiveness of ACE inhibitors in Black patients mentioned earlier can prevent those with chronic kidney disease from receiving one of the key medications that can stop disease progression.

We should reconsider the use of race in other clinical decision-making tools, such as the calculator for predicting the likelihood of a successful vaginal birth after cesarean delivery (VBAC). Even though Black patients have significantly higher maternal morbidity and mortality, this calculator assigns a lower chance of VBAC success for them compared with White or non-Black Hispanic patients.¹³ Instead of encouraging a mode of delivery that can improve recovery time and lower the risk of infection or postpartum hemorrhage, this calculator potentially discourages maternity care clinicians from offering Black patients a trial of labor after a cesarean delivery. Instead of treating these patients as a homogeneous group, we should be aggressively studying the social and systemic factors that have historically resulted in more cesarean deliveries.

Although the VBAC and ASCVD calculators typically assign higher risk to Black patients, the FRAX tool for estimating osteoporotic fracture risk assigns a 10-year fracture risk to non-White women that is approximately one-half that of White women. Black women are less likely to be screened or treated for osteoporosis, less likely to receive a dual-energy x-ray absorptiometry scan following a hip fracture, and more likely to die or become disabled following a hip fracture.¹⁴ Yet, FRAX systematically assigns a lower fracture risk to the entire Black community without considering its diversity.

We should focus on the social factors that create the group-level differences that we see among races, and we should also discuss how to screen for these social factors and behaviors instead of making assumptions based on skin color. The rigor that is demanded in evidence-based medicine often eludes studies of race, as exemplified by the studies that led to the Black and Asian correction factors for pulmonary function testing. A systematic review of spirometry revealed that only 17.3% of studies actually defined race and/or ethnicity, and 94% of articles failed to examine socioeconomic status as a potential confounder.^{15,16}

Race is a sloppy, inconsistent proxy for genetics and biology, and we should move toward eliminating it from medical decision-making. None of the calculators and predictor tools mentioned provide any scientific rationale about what race to input if a patient has one parent who is Black and one who is not. There are almost no other aspects of medicine that result in such dramatic differences in medical management simply based on a patient's subjective report about their identity.

Race-based approaches to medicine reinforce a system that assumes biologic causes of health inequities, which can lead us to ignore the social determinants of health that are the true drivers for racial disparities in health outcomes.¹⁷ This creates a racist system that withholds organ transplants and preventive services from Black patients with chronic kidney disease and ACE inhibitors from Black patients who might benefit from them, and ignores the social or environmental factors that cause differences in the lung function of some Black and Asian people. As family physicians, we should inform patients of these limitations when we use race-based algorithms, and we should look for social explanations to racial/ethnic disparities. As we continue to improve the ways in which we competently address social determinants, members of the American Academy of Family Physicians should be proud that it has joined the ranks of the American Medical Association and other organizations that have declared that race is a social construct instead of a biologic one.^{18,19}

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