

## Things We Do for No Reason™: Routine Inclusion of Race in the History of Present Illness

Jonathan Gau, MD<sup>1</sup>, Christle Nwora, MD<sup>2</sup>, Shaden T Eldakar-Hein, MD, MSc<sup>3</sup>,  
Mita Sanghavi Goel, MD, MPH<sup>4</sup>, Tim Lahey, MD, MMSc<sup>5</sup>, Allen B Repp, MD, MSc<sup>5\*</sup>

<sup>1</sup>Internal Medicine Residency Program, UC Irvine School of Medicine, Orange, California; <sup>2</sup>Pediatrics Urban Health Residency Program, Johns Hopkins, Baltimore, Maryland; <sup>3</sup>Lewis Katz School of Medicine at Temple University/St. Luke's University Health Network, Philadelphia, Pennsylvania; <sup>4</sup>Division of General Internal Medicine & Geriatrics, Feinberg School of Medicine, Northwestern University, Chicago, Illinois; <sup>5</sup>University of Vermont Medical Center and the Larner College of Medicine at the University of Vermont, Burlington, Vermont.

Inspired by the ABIM Foundation's Choosing Wisely® campaign, the "Things We Do for No Reason™" (TWDFNR) series reviews practices that have become common parts of hospital care but may provide little value to our patients. Practices reviewed in the TWDFNR series do not represent clear-cut conclusions or clinical practice standards but are meant as a starting place for research and active discussions among hospitalists and patients. We invite you to be part of that discussion.

### CLINICAL SCENARIO

On teaching rounds, a medical student presents the following case to the attending hospitalist: "Mrs. L is a 54-year-old Black female with chronic kidney disease who was admitted with community-acquired pneumonia. She continues to improve symptomatically on ceftriaxone. Currently, she is afebrile and her vitals are stable. Supplemental oxygen has been weaned to 2 L/min by nasal cannula. Exam reveals improved crackles in the left lower chest without dullness to percussion. Labs are notable for down-trending leukocytosis and a stable serum creatinine of 2.8 mg/dL." The hospitalist considers how including racial descriptors in clinical presentations may influence the care of the patient.

### WHY YOU MIGHT THINK INCLUDING RACE IN THE HISTORY OF PRESENT ILLNESS IS HELPFUL

For decades, medical educators have taught learners to include sociopolitical constructs such as race in the opening sentence of the history of present illness (HPI). This practice presumably stems from the assumption that race accurately reflects biogenetic information about patients and serves as a key attribute in problem representations.<sup>1</sup> Proponents of including race in the HPI suggest doing so aids the clinical assessment of patients' risks for particular diseases and may inform the selection of race-appropriate therapies.<sup>2</sup>

The construct of race does sometimes correlate with the risk of disease or response to therapies. For example, sickle cell disease (SCD) occurs more commonly among patients who identify as Black rather than White. Specifically, ancestry from African nations such as Nigeria or the Democratic Republic of Congo increases the likelihood of having the disease-associated hemoglobin gene variant HbS.<sup>1</sup> Popular genomic ancestry tests often report ancestral groupings that map to racial categories and may reinforce the perception that race has a genetic basis.<sup>3</sup>

### WHY IT IS NOT HELPFUL TO INCLUDE RACE IN THE HPI

Race, a construct of sociopolitical origins, incorrectly conflates skin color with genetic variation. Associations between race and disease have the potential to cause diagnostic and therapeutic errors and inequitable allocation of resources. Increased illness burden in minority populations results primarily from social factors such as environment, access to care, housing instability, food insecurity, and experiences of discrimination, rather than genetic differences. The resulting chronic and recurrent physiologic stress—known as allostatic load—also contributes to the inequitable health outcomes observed in vulnerable populations, including patients who identify as Black.<sup>4</sup>

Historically, race evolved as a sociopolitical framework stemming from colonialism, discrimination, and exploitation.<sup>5</sup> Numerous studies reveal a lack of genetic precision in racial categories. In fact, genetic data compared across major continental groups found greater variation of microsatellite loci and restriction fragment length polymorphisms within racial groups than between them.<sup>6</sup> The evidence indicates that racial categories do not reflect homogenous population groups but rather "arbitrary division[s] of continuous variation" that cannot serve as a surrogate to genetic diversity.<sup>5</sup> Not only are racial categories genetically inaccurate, but data on race within the electronic health record often differ from patients' self-description of race, underscoring the problematic nature of even identifying race.<sup>7</sup> In one study, up to 41% of patients self-reported identification with at least one other racial or ethnic group than the race or ethnicity documented in their electronic health record.<sup>7</sup>

Additionally, conflating race with genetic variation can lead to diagnostic errors. As an example, the incidence of cystic fibrosis (CF) varies widely across populations of European

\*Corresponding Author: Allen B Repp, MD, MSc;  
Email: allen.repp@uvmhealth.org; Telephone: 802-847-7911.

Received: February 16, 2021; Revised: April 25, 2021; Accepted: May 4, 2021

© 2021 Society of Hospital Medicine DOI 10.12788/jhm.3650

ancestry. The primary focus on CF's occurrence in patients of European descent may divert attention from the identification of mutations causing CF in populations of African descent or the decreased survival observed in the United States among CF patients of Hispanic descent.<sup>8,9</sup> Similarly, India represents one of the countries largely affected by SCD, suggesting that a myopic focus on SCD among those identifying as Black can lead to underdiagnosis of SCD among those with Indian ancestry.

Perhaps more insidiously, linking disease to race or other social constructs can result in differential support for affected individuals. SCD offers a striking illustration of this point. Reflecting the legacy of transatlantic slave trading, the majority of people with SCD in the United States are Black and face interpersonal and structural racism within society and health-care that amplify the effects of this devastating illness.<sup>10</sup> Compulsory screening programs for sickle cell trait introduced by many states in the 1970s targeted Black Americans and resulted in stigmatization and the denial of insurance, educational opportunities, and jobs for many identified with sickle cell trait. Federal funding for SCD research remains low, particularly in comparison to the tenfold higher funding for CF, which afflicts fewer, but primarily White, Americans.<sup>10</sup>

The incorporation of race into risk models and guidelines—alongside biologically relevant variables such as age and comorbid conditions—has received increasing attention for its potential to compound racial disparities in health outcomes. The American Heart Association Heart Failure Risk Score, for instance, may lead to the exclusion of some Black patients from necessary care because “Black” race, for no clear physiologic reason, serves as a protective factor against heart failure mortality.<sup>11</sup> Likewise, race adjustments in pulmonary function tests, breast cancer risk models, and estimated glomerular filtration rate calculations, among others, have limited biological basis and the potential to divert care disproportionately from minority populations.<sup>11</sup>

Researchers have even called into question the application of race to pharmacotherapies. A 2001 investigation on geographic patterns of genetic variation in drug response concluded that common racial and ethnic labels were “insufficient and inaccurate representations” of the individual genetic clusters.<sup>12</sup> Further, numerous experts have criticized two landmark studies of vasodilators and angiotensin-converting enzyme inhibitors in Black patients with heart failure for inconsistent results and nonsignificant associations between race and major outcomes, such as the development of heart failure or death.<sup>13</sup>

Race-based labels can also divert attention from true causes of health inequities. The National Academy of Sciences concluded that social determinants of health and structural racism are the root causes of health inequities, rather than genetics.<sup>14</sup> Medical professionals may perpetuate these disparities: Most US physicians demonstrate an unconscious preference—or implicit bias—for White Americans over Black Americans.<sup>15</sup> Beyond obscuring the role of social determinants of health and structural racism in health outcomes,

race-based labels may exacerbate the ways in which physicians' implicit biases contribute to racial and ethnic health disparities, primarily affecting Black Americans.<sup>2</sup> In a recent study, clinicians documented race in the HPI for 33% of Black patients compared with 16% of White patients, and White clinicians were twice as likely to document race as Black physicians.<sup>16</sup> Moreover, training medical students to view race as an independent risk factor of disease without discussing structural inequities can pathologize race and reinforce implicit biases linking race and disease.<sup>15</sup>

Based on the current evidence, we believe routine use of race-based labels in clinical presentations confuses providers at a minimum and potentially produces far more damage by obscuring or perpetuating the role of racism in health inequities.

## WHAT YOU SHOULD DO INSTEAD

Instead of routinely presenting race in the HPI, we recommend including racial or ethnic information in the social history only when the patient reports it as a meaningful identity or when it informs health disparities stemming from structural or interpersonal racism. Clinicians should include physical characteristics pertaining to race, such as skin tone, in the physical exam only if required to describe exam findings accurately. When presenting race, clinicians should explicitly justify its use and take care to avoid obfuscatory, inaccurate, or stigmatizing mention of associations between race and disease. Clinicians should not use race in clinical algorithms. Medical educators should emphasize the role of social determinants of health and structural racism in health outcomes to inform the use of race in medicine, in hopes that doing so will help students minimize implicit biases and learn to mitigate racial inequities in health-care.<sup>2,16</sup> In short, clinicians and medical educators alike should ensure that clinical care and the medical curriculum avoid presenting race as a proxy for pathology.

There is little evidence to guide proper inclusion of race in clinical interviews. In the absence of clear guidance about how to approach patients about race, we suggest not asking about it unless there is a reasonable probability that doing so will improve clinical care. If a clinician decides to ask about race, it is important to provide a rationale—such as explaining that the information can be used to assure high-quality care for all patients—since many patients are uncomfortable with questions about race.<sup>17</sup> If clinicians report information about race in the social history, we advise using the patient's description of race rather than traditional racial categories.

Clinicians who ask their patients about race should approach every patient in a uniform manner to avoid perpetuating biases. We hope future studies will inform equitable, inclusive, and person-centered approaches to discussing race with patients and promote a shared understanding of how racism contributes to illness.

## RECOMMENDATIONS

- Avoid using racial descriptors in the HPI.
- Include racial and ethnic information in the social history

only when it serves as a meaningful identity or it informs disparities stemming from racism.

- If racial or ethnic information is asked for, explain to patients why and how it will be used.
- Mention physical characteristics such as skin tone, rather than race, in the physical exam if required to describe findings accurately.
- Advocate for the replacement of race or race-adjusted algorithms in patient care.
- Expand the medical curriculum in the social determinants of health and structural racism, and develop systems to avoid the use of stigmatizing, race-based labels.

## CONCLUSION

Race, a sociopolitical construct, does not accurately represent genetic variation. The routine use of race in the HPI can perpetuate racial biases and muddle both diagnoses and treatment. Only mention race in the social history if it is meaningful to the patient's self-identity or explains health disparities arising from racism. All documentation and presentations should avoid the use of stigmatizing, race-based labels.

In the clinical scenario mentioned earlier, the attending hospitalist raises the issue of race-based labels in patient care in a nonjudgmental fashion. To provide illustrative specificity, she notes how the incorporation of race in formulas of glomerular filtration rate can lead to under-referral for renal transplant. The hospitalist then facilitates an open and inclusive discussion with the team regarding the use of race in clinical presentations and its potential impact on health disparities.

*Do you think this is a low-value practice? Is this truly a "Thing We Do for No Reason™"? Let us know what you do in your practice and propose ideas for other "Things We Do for No Reason™" topics. Please join in the conversation online at Twitter (#TWDFNR)/Facebook and don't forget to "Like It" on Facebook or retweet it on Twitter.*

---

Disclosures: Dr Repp serves as a co-investigator on unrelated grants from the National Institutes of Health and the Centers for Disease Control and Prevention. He also serves as a member of the Board of Governors for the American

College of Physicians and has received reimbursement for travel to Board of Governors meetings. All other authors have nothing to disclose.

## References

1. Burchard EG, Ziv E, Coyle N, et al. The importance of race and ethnic background in biomedical research and clinical practice. *N Engl J Med*. 2003;348(12):1170-1175. <https://doi.org/10.1056/NEJMs025007>
2. Tsai J, Ucik L, Baldwin N, et al. Race matters? Examining and rethinking race portrayal in preclinical medical education. *Acad Med*. 2016;91(7):916-920. <https://doi.org/10.1097/ACM.0000000000001232>
3. Roth WD, Yaylacı S, Jaffe K, et al. Do genetic ancestry tests increase racial essentialism? Findings from a randomized controlled trial. *PLoS One*. 2020;15(1):e0227399. <https://doi.org/10.1371/journal.pone.0227399>
4. Beckie TM. A systematic review of allostatic load, health, and health disparities. *Biol Res Nurs*. 2012;14(4):311-346. <https://doi.org/10.1177/1099800412455688>
5. Fuentes A, Ackermann RR, Athreya S, et al. AAPA Statement on race and racism. *Am J Phys Anthropol*. 2019;169(3):400-402. <https://doi.org/10.1002/ajpa.23882>
6. Barbujani G, Magagnoli A, Minch E, et al. An apportionment of human DNA diversity. *Proc Natl Acad Sci U S A*. 1997;94(9):4516-4519. <https://doi.org/10.1073/pnas.94.9.4516>
7. Klinger EV, Carlini SV, Gonzalez I, et al. Accuracy of race, ethnicity, and language preference in an electronic health record. *J Gen Intern Med*. 2015;30(6):719-723. <https://doi.org/10.1007/s11606-014-3102-8>
8. Stewart C, Pepper MS. Cystic fibrosis in the African diaspora. *Ann Am Thorac Soc*. 2017;14(1):1-7. <https://doi.org/10.1513/AnnalsATS.201606-481FR>
9. Rho J, Ahn C, Gao A, et al. Disparities in mortality of Hispanic patients with cystic fibrosis in the United States. A national and regional cohort study. *Am J Respir Crit Care Med*. 2018;198(8):1055-1063. <https://doi.org/10.1164/rccm.201711-2357OC>
10. Power-Hays A, McGann PT. When actions speak louder than words—racism and sickle cell disease. *N Engl J Med*. 2020;383(20):1902-1903. <https://doi.org/10.1056/NEJMp2022125>
11. Vyas DA, Eisenstein LG, Jones DS. Hidden in plain sight—reconsidering the use of race correction in clinical algorithms. *N Engl J Med*. 2020;383(9):874-882. <https://doi.org/10.1056/NEJMms2004740>
12. Wilson JF, Weale ME, Smith AC, et al. Population genetic structure of variable drug response. *Nat Genet*. 2001;29(3):265-269. <https://doi.org/10.1038/ng761>
13. Cooper RS, Kaufman JS, Ward R. Race and genomics. *N Engl J Med*. 2003;348(12):1166-1170. <https://doi.org/10.1056/NEJMs022863>
14. National Academies of Sciences, Engineering, and Medicine. *Communities in Action: Pathways to Health Equity*. National Academies Press; 2017.
15. Chapman EN, Kaatz A, Carnes M. Physicians and implicit bias: how doctors may unwittingly perpetuate health care disparities. *J Gen Intern Med*. 2013;28(11):1504-1510. <https://doi.org/10.1007/s11606-013-2441-1>
16. Balderston JR, Gertz ZM, Seedat R, et al. Differential documentation of race in the first line of the history of present illness. *JAMA Intern Med*. 2021;181(3):386-388. <https://doi.org/10.1001/jamainternmed.2020.5792>
17. Baker DW, Hasnain-Wynia R, Kandula NR, Thompson JA, Brown ER. Attitudes toward health care providers, collecting information about patients' race, ethnicity, and language. *Med Care*. 2007;45(11):1034-1042. <https://doi.org/10.1097/MLR.0b013e318127148f>