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Addressing Structural Racism in Psychiatry With Steps to Improve Psychophysiologic Research

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IMPORTANCE The American Medical Association has acknowledged the public health threat posed by racism in medicine. While clinicians in psychiatry have echoed the sentiment, the research community has largely been silent. Current understanding of the biological domains that underlie psychiatric disorders was historically established by studying White populations, often leaving widely used treatments ineffective for Asian, Black, Hispanic, Indigenous, and other racial and ethnic minority individuals. This article addresses how undersampling of racial and ethnic minority individuals has led to overgeneralized physiological findings, the implications for development of psychiatric treatments, and steps to improve service to racially diverse communities.

OBSERVATIONS Three primary observations regarding differences associated with race and ethnicity have been addressed in the existing psychiatric research: misdiagnosis, medication nonadherence, and treatment efficacy and expression of adverse effects. While cultural factors have been discussed as potential factors associated with these differences, a lack of understanding of physiologic systems may be foundational to each of these issues. Recent evidence points to race differences in psychophysiological measures, likely attributed to factors including the lived experience of racism as opposed to inherent biological differences. This mounting evidence supports a reassessment of existing work to examine potential divergent patterns within racial and ethnic groups. The following strategies may improve understanding of the influence of racism on physiology, allowing clinicians to better address psychiatric symptoms and improve existing treatment approaches. Thus, psychiatric researchers need to (1) understand the historic and current terminology for *race* and *ethnicity* and use appropriate terms and categories as defined by sociologists, population health experts, and databases while respecting individuals' right to self-identify, (2) refine research questions, and (3) reexamine research data to determine whether patterns observed in largely White populations can extend to other groups. To appropriately implement these steps, researchers must accept the discomfort that accompanies growth, invite scientists from diverse backgrounds to participate, and use resources to increase diversity in recruitment of study participants. This will require a commitment from funding agencies to provide adequate support to recruit and investigate large, diverse samples.

CONCLUSIONS AND RELEVANCE To create more suitable medical treatments and improve the quality of care received by those with psychiatric conditions, further discussion is needed surrounding the physiologic toll that racism has had on multiple generations of racial and ethnic minority groups and how that may alter responsivity to biobehavioral interventions. To better inform psychiatric research, the resources provided must be expanded, basic physiologic studies should be replicated with more diverse samples and adequate analyses, and psychiatry scientists must reconsider approaches to clinical research.

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A normal black child, having grown up in a normal family, will become abnormal at the slightest contact with the [white] world.
Frantz Fanon, 1952[1]

Modern understanding of psychiatric disorders is grounded in both behavior and physiology. Constructs of the US National Institute of Mental Health Research Domain Criteria span biopsychosocial dimensions, including genetics, molecular and cellular systems, gross anatomy, behavior, and self-report collections. Despite the growing reliance on psychophysiologic measures, from neural systems assessed by functional magnetic resonance imaging and electroencephalography to peripheral domains like heartrate variability and hypothalamic-pituitary-adrenal (HPA) axis function, basic understanding of these dimensions is rooted in research with predominately White participants.[2] Current comprehension of physiologic symptom clusters is thus limited to a group with social and economic advantages, and the ability to identify these clusters in historically marginalized racial and ethnic minority populations is limited.

The exclusion of race in the study of biological systems in mental health has led to the development of treatment approaches for all that were designed only for some. For example, the treatment response to naltrexone for alcohol use disorder (AUD) has been shown to differ in Black and Hispanic individuals relative to White individuals.[3,4] This has far-reaching health implications for the more than 130 million individuals in the US who do not identify as non-Hispanic White.[5] In effect, the structure of medical research in the US has resulted in a system that has excluded or disenfranchised a large portion of the population whose human biology and development has not been considered in the creation, assessment, and prescription of pharmacological treatment. The pursuit of pharmacological therapies for chronic diseases has revealed the need for a greater understanding of intricacies associated with the health of racial and ethnic minority groups.[6,7]

Historically, race has been used as a proxy for presumed genetic biological differences between racial groups[8] despite evidence that within-race genetic variability far exceeds that of between-group evaluations.[9] These arguments were used to support eugenics and related notions about the intellectual and moral inferiority of Black individuals and other historically marginalized groups in the US.[10] The presence of these ideas in scientific circles contributed to the subsequent inattention to race in physiological research. Over the past several decades, there have been a number of calls to action from scholars to address racism in psychiatry[11,12]; however, the message has been largely ignored by those in the majority and seats of power.[12] Some progress has been made in recent years, with federal policy requiring the consideration of race and ethnicity in sponsored research. The US Public Health Service Act (42 USC §289a-2)[13] became policy in 1993 and mandated that racial and ethnic minority groups be included in all National Institutes of Health–funded studies in a manner that is appropriate to the scientific question under study.[14] In 2020, the American Medical Association published a statement recognizing racism as a public health threat and introduced a point-by-point plan to mitigate its impact on medicine through updated practice guidelines.[15] The statement also called for an increase in the number and type of medical studies examining the health impacts of racism as well as those evaluating interventions designed to reduce the risk and damage caused by racist structures, institutions, and practices. This topic is particularly salient in psychiatry and has been echoed by clinicians and academic leaders alike.[12,16-18] But the general discourse about the development and promotion of antiracist strategies in psychiatric research has been remarkably muted. Psychiatry is intimately linked with the lived experiences of racial and ethnic minority individuals and can contribute to this enterprise. But psychiatry's approach to race and racism requires considerable evolution.

Clarity of terms and concepts is essential for any discussion about race and racism. Race is a social construct that emerged in the US from the effort to justify slavery and subsequent forms of second-class citizenship.[9,19] Historically, the term *race* is related to but was not synonymous with *ethnicity*. The term *ethnicity* has cultural and biologic implications (eg, Ashkenazi vs Sephardic, Afrocarribean vs African American). Racism is a system of structuring opportunity in a manner that unfairly disadvantages some individuals and groups and, consequently, unfairly advantages other individuals and communities.[1,7,10] In discussions of race and ethnicity in psychophysiology, it is important to note that genetic differences are not prerequisites of physiological differences and that observed between-group contrasts can emerge from transgenerational exposures to the societal constructs of ethnicity, race, and racism.[20] Additionally, racism has multiple dimensions, and its effect on physiology can vary by place and population. A 2019 book edited by Ford and colleagues[19] explains how racism has implications for other racial and ethnic minority groups, including Asian, Hispanic and Latinx, Indigenous, and religious minority groups in the US. The following discussion largely focuses on the effects of racism on Black individuals and serves as a foundation to further the conversation.

Existing Work Supports Racism-Related Changes in Physiologic Responsivity

There are currently 3 major cornerstones of race-related research in psychiatry: (1) differences in symptomology and misdiagnosis,[21,22] (2) discrepancies in medication access and adherence,[23,24] and (3) treatment efficacy and expression of adverse effects.[25,26] Misdiagnosis is often reported to be because of differences in cultural norms. That is, non-Anglocentric behaviors are seen as aberrant, sometimes even by Black psychiatrists.[22] Nonadherence to medication is attributed to race-related stigma, lack of access to health care, and broken patient-physician trust,[24,27] although these factors do not fully explain the discrepancies.[24] Racial differences in adverse effects of treatments have not been discussed as thoroughly; while some have argued for differences in rates of metabolism,[28] others point to clinician bias.[29] A greater understanding of how the constructs of race and racism present physiologically in psychiatric disorders may add clarity to each of these issues.

Studies have reported higher levels of medication nonadherence in addition to a greater diagnostic prevalence of psychotic disorders in Black individuals compared with White individuals in the US.[24,30] Antipsychotic medications primarily function through antagonism of dopamine D2 receptors, which results in a number of adverse effects, including tardive dyskinesia (TD). TD is a highly prevalent and debilitating disorder related to D2 hypersensitivity, affecting approximately 20% of individuals who take a dopamine-blocking drug.[31] Multiple studies and meta-analyses over the last 25

years have indicated a greater risk of developing TD among racial and ethnic minority individuals relative to White individuals,[31-34] even when analyses were limited to those prescribed second-generation antipsychotics (eg, haloperidol), which hold a lower risk of developing TD. Another study identified differences in striatal D2 availability associated with ancestry,[35] although there is evidence that this is likely associated with lived adverse experiences.[8,36] Despite the mounting evidence that Black individuals are at a greater risk of developing TD, to our knowledge, no work has discussed the possible contribution of differing D2 availability following lifelong exposure to discrimination. Studies that advance comprehension of racism-related changes in receptor density, with the possibility of targeted treatments, have not yet been reported.

Similarly, one of the body's primary stress response systems, the HPA axis, has been implicated in some mental and physical health disorders and has been thoroughly studied since the ability to isolate neuroendocrine hormones was reported 50 years ago.[37] For example, dysregulation of the HPA axis among recently abstinent (2 to 8 weeks) individuals with AUD is a well-established finding that has been replicated using multiple pharmacologic and behavioral stressors.[28] However, recently, several investigators reported not being able to replicate this observation in new cohorts.[38-40] The key consistency across these nonreplicating studies was the inclusion of racially diverse samples relative to previous reports. Follow-up analyses by 2 of us (J.L.P. and B.A.) found the hypothesized association between AUD and HPA axis function was present only when limited to White participants.[41] We have posited that the association between chronic alcohol exposure and Black men's HPA axis function may have been confounded by potential effects of lifelong exposure to racism.[20,41] Medications used to treat AUD have been shown to have direct or indirect effects on the HPA axis (for example, naltrexone[3,4]). The hypothesized association between racism and HPA axis function among Black men may explain some of the variability in efficacy and adherence to such medications.

Another example builds from the cardiovascular health literature, which has advanced to address racial disparities in health outcomes and health care. For example, the carotid and arterial baroreflex system modulates blood pressure via a bidirectional feedback loop between the heart and the brain and has clinical relevance in multiple psychiatric conditions.[42] Baroreceptors are downstream effectors of medications used to ameliorate symptoms of depression, anxiety, and substance use disorder (tricyclic antidepressants, serotonin-norepinephrine reuptake inhibitors, and benzodiazepines) and are central to the concept of heartrate variability biofeedback, a practice in which the neurocardiac signal is altered through slow, controlled pacing of the breath.[43] Studies have demonstrated that this technique may be associated with acute health benefits both in the general population and among patients in clinical studies.[42,43] This research, however, does not take into account differences in baroreflex sensitivity between normotensive Black and White individuals.[44,45] Pharmacologic and behavioral interventions that affect cardiovascular function may differentially benefit individuals from varying racial groups. Considering the growth of app-based, self-administered treatment approaches,[46] racial minority groups have expressed openness and interest in precision medicine (despite concerns regarding trust, privacy, and marginalization[47,48]), and the discourse surrounding medication adherence and efficacy for racial minority groups, exploring biobehavioral interventions, such as heartrate variability biofeedback, may be a promising route to provide better care for racial minority patients with psychiatric disorders.

The Call

As presented by the American Medical Association statement,[15] antiracist medical practice requires a foundation of antiracist medical science. To achieve this, psychiatry researchers and clinicians cannot continue to ignore the constructs of race and racism in studies of physiological systems and psychiatric conditions. Deliberate and meticulous consideration of racial differences in experience and resistance of the urge to draw inferences without rigorous examination is needed. The cost to address previous shortcomings will be large, and the final product of truly inclusive science will require larger systemic solutions. But smaller, more immediate steps can also be taken to mitigate the problem and pave the way for representative science.

1. **Understand the terms associated with race and ethnicity:** Language should be precise. Research protocols and methods and published reports must accurately reflect the appropriate terminology defined by sociologists, population health experts, and databases while respecting individuals' right to self-identify. Vague terms such as *non-White* should be replaced with more precise descriptors that convey specific racial and ethnic categories, country or origin, culture, and exposure to racism.
2. **Refine questions:** Address whether questions or answers change if race and racism are included variables in the outcome assessment. Do the patterns identified in a specific study extend to other racial and ethnic minority groups? Or are the findings limited to majority White individuals represented in that study? Based on the work conducted in other fields and the existing research directly examining race in psychophysiological indices of psychiatric disorder, unique patterns may emerge within different racial groups. Further, inclusion of measures of lived experiences of adversity and racism may serve as useful covariates in understanding identified patterns. These newly formed questions will reform the approach to understanding psychiatric conditions and, thus, clinical trials and treatment. Medicine will continue to lack precision if patient context, experience, and environment are not included.
3. **Reexamine existing research and data:** The psychiatry research communities are experiencing a replication crisis, possibly compounded by the confounding effect of subgroups. Researchers may be biased by an assumption that subpopulations will show directionally similar patterns of varying magnitude between healthy and clinical populations, as exemplified by the report of the association between AUD and HPA axis function being limited to White men, possibly explaining previously reported null findings.[41] Existing work should be examined for similar confounding associations. Previously null findings and physiologic function that provided foundation for later interventions must be reconsidered within the existing variance across and within racial groups. Analyses should assess if potential associations have been missed owing to divergence of patterns between racial groups. This type of error can be mollified by examining associations within groups, such as racial and ethnic subgroups, that may be responsible for unexpected findings.

4. **Accept discomfort:** There is likely an internal threat to the introspective aspect of this call to action. But the benefits to science and perhaps society outweigh individual apprehension. By using the previous steps, researchers, academicians, and clinicians can accept this as an uncomfortable moment of growth for the greater good of the science. There are students, mentees, and trainees who want to learn about racism in science. Considering the impact of race on and within scientific study may lie beyond personal expertise; however, it presents an opportunity to co-learn with students. The necessity of racial inclusion in practice and research, while likely a new terrain for many, will advance psychiatry and psychiatric research.
5. **Invite scientists from other backgrounds to participate:** Adherence to these recommendations does not require a reinvention of the wheel. The assistance from scientists with varying areas of expertise, cultural backgrounds, and community sensibilities can improve the quality of research considerably. No one should speak for or over groups that they do not represent. By focusing on a broader collaborative network, psychiatric researchers can continue to cultivate working groups that apply to more broad populations and bolster individualized treatment approaches. For some, the focus on race may be unfamiliar and difficult. This cannot be an excuse for continued omission. Researchers can invite other researchers who study race and ethnicity and culture to participate in grant applications and research designs and ensure compensation for such assistance. This approach to science can produce findings that are transdisciplinary and transformative.
6. **Use tools and invite collaborators that increase diversity in study recruitment:** A number of reasons have been presented to explain the lack of studies focusing on psychiatric disorders among Black and other racial and ethnic minority individuals. It has been suggested that Black individuals are less likely to participate in research studies; however, study participation among racial minority groups may be linked to discriminatory experiences with research institutions and personnel.[49] A debt is owed to the individuals who participate in studies (beyond financial compensation), and researchers' actions must reflect this. Researchers should ask how the study might benefit participants and, more importantly, listen to the response. Researchers can identify the resources and information they have to offer the communities that they seek to help. Collaborators may be able to assist in improving inclusive recruitment techniques, but researchers should not ask their Black science colleagues for a solution. Researchers should engage with communities outside their own, not solely for the purpose of benefiting participant recruitment but also because these are the communities that are being served.
7. **Invitation to funding agencies:** The move to inclusive research will be expensive, as it will require larger study samples and involvement of more collaborators. For a swift and effective transition, funding agencies should be prepared to support studies that investigate the psychiatric effects of racism. The National Institutes of Health recently announced their dedication to end structural racism in biomedical research.[50] Through a new initiative, UNITE, the National Institutes of Health made calls for change in structure, funding, and data dissemination to stop the progression of systemic racism in biomedical research. Under this call for new research on health disparities, racial and ethnic minority health, and health equity, the National Institutes of Health is urged to provide sufficient funding that will allow researchers to recruit diverse samples, examine within-group patterns of pathology, and support replication and reinvestigation of previously established findings.

Conclusion

W. E. B. DuBois prophesized in The Philadelphia Negro[51] and The Souls of Black Folk[52] that race was a deeply foreboding issue with far-reaching implications for the Black community and other historically marginalized populations. The remarkable accuracy of this prophesy is manifested in the deep and persistent disparities in health care and health outcomes. Psychiatry cannot move forward with treatment development when understanding of human physiologic systems is historically grounded in studies conducted by White researchers that primarily include White participants. To create more suitable medical treatments and improve the quality of care received by those with psychiatric conditions, further discussion is needed surrounding the physiologic toll that multiple generations of racism have on individuals from racial and ethnic minority groups and how that may alter responsivity to biobehavioral interventions. To better inform psychiatric research, the resources provided must be expanded, basic physiologic studies should be replicated with more diverse samples and adequate analyses, and psychiatric scientists must reconsider approaches to clinical research.

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