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Embracing Complexity When Examining the Social Construct of Race: Nuanced Strategies are Needed to Reduce Harm and Improve Neurodevelopmental Science

Although advances in neurodevelopmental sciences and clinical care have progressed at lightning speed, our headway at addressing the long history of racism in science and medicine has been notably slower and muddled. Only recently has there been widespread acknowledgement in the scientific community that race is a social construct¹—with greater genetic variability between categorical groups than within—spurring contemporary guidance from NASEM² confirming “race” is not biologically-based and analyses suggesting otherwise are “misleading and harmful.” Others have illuminated the problematic use of race-based diagnostic algorithms and practice guidelines given their contributions to health inequities.³ Race/ethnicity has historically been treated as a confounding or stratification variable in health research, but leaders have argued for revamping this approach^{4,5} to avoid implication that biological differences underlie observed inequities. Given the pervasiveness of racial/ethnic inequities in neurodevelopmental outcomes and policies and social structures that have led to substantial correlations between poverty and race, the complexity of studying socioeconomic effects precludes one-size-fits all approaches—particularly in the current sociopolitical climate.

In this issue, Semanaz et al.⁶ test a recent suggestion to incorporate social determinants of health (SDoH), rather than including race as a confounder or stratification variable, when examining poverty effects on neurodevelopment. In a sample of ~8,000 10-year-olds from the ABCD study, they found significant race*poverty interactions in the prediction of psychiatric symptoms, but not cognitive performance, cortical thickness, or brain volume. Analyses also showed poverty associations with brain morphology diminished after adjusting for race, and that SDoH did not fully account for confounding effects of race/ethnicity. From these analytic results, Semanaz et al. conclude that “effects of poverty differ for children of varying race and ethnicity,” inferring that differences found are due to structural racism that prior attempts to explain racial inequities using SDoH alone have been insufficient. These extensive analyses shed light on the issue of structural racism’s impact on associations between poverty neurodevelopment, but also provoke questions. We agree that examination of SDoH can advance understanding of the interplay of race, poverty, and neurodevelopment, and Semanaz et al contribute to this line of inquiry in several ways. The ABCD cohort is well-powered and sociodemographically diverse relative to other neurodevelopmental epidemiological databases. The directed acyclic graphs (DAGs) are valuable contributions and their articulation of the theorized causal pathways among poverty, race/ethnicity, SDoH, and child neurodevelopment informs future endeavors in this space. Their Discussion section raises important reflections and acknowledges study limitations, highlighting key considerations for future research.

In our review of the paper’s results, we were struck by how, more often than not, the effect of poverty on the range of outcomes did *not* differ by group. While their study finds

circumstances in which group effects differed, statistically significant interaction tests in this sample of ~8000 were found only within some models with reporter-based measures (vs. cognitive tasks or MRI). This pattern of results highlights that the *main effect* of poverty on neurodevelopment remains a key exposure in this area of research and demonstrates the importance of transparent researcher choices and their consequent impact on both the estimate of poverty's effect and conceptual interpretations of found effects.

Further, there are important points to expand upon and additional big picture considerations to highlight for those engaging in this type of work. First, while categorical race/ethnicity is sometimes a useful *proxy variable* for exposure to racism, it is not equivalent to *structural or individual experiences of racism*. Indeed, the concept of categorical race/ethnicity emerged in the Americas as a way to discriminate against Black and Indigenous groups and restrict their access to resources and participation in American society,⁵ and today, structural racism persists in myriad forms of group-based discrimination within societal systems.⁷ However, minoritized racial/ethnic categories also encompass a multitude of *health-promotive, strengths-based factors*, such as connection with family and community, participation in cultural practices, and artistic expression. Studies that utilize categories solely to indicate racism risk emphasizing a deficit-based view of minoritized groups and overlooking the strengths that also accompany group membership. Moreover, coarse approaches to assessing racial and ethnic groups (such as only 3 categories) create unreliable proxies for social, cultural, and ancestral differences and obscures crucial complexities. The tensions in work attempting to thoughtfully incorporate race into models are not easily resolved, though inclusion of validated measures of structural racism⁸ in analytic models is a strong start.

Second, a critical risk of using categorical race/ethnicity as a default “confounder” or “modifier,” regardless of a researcher’s stated definition of what it is capturing, is that presentation of group-based differences in health or physiology (especially neuroscience, as in Semanaz et al.) carries the real risk of misconstrued interpretation of findings that differences detected are innate, instead of socioculturally-determined. This “two-edged sword” of racialized data⁵ has been discussed deeply by other scholars^{4,5,7,9} and was noted by Semanaz et al. Even when group differences are well-contextualized in manuscripts, modern-day racist agents in positions of power co-opt pieces of scientific findings, especially decontextualized tables and figures, to further their agendas.^{5,9,10} Additionally, when comparing outcomes across groups, researchers must consider the likelihood of racial/ethnic bias in the development of outcome measures (e.g., psychiatric scales, brain anatomical templates), given historical exclusion of minoritized groups from research.^{1,9} Thus, *researchers should exercise caution when presenting race-based group differences*.

Third, although ABCD is well-powered, and Semanaz et al. includes 1798 Black and 1236 Hispanic/Latino/a/x youth, the sample doesn't have comparable distributions of the

exposure of interest across the three racial/ethnic groups tested. While only 4.9% of White-non-Hispanics were below the poverty threshold, 38.6% and 24.8% of the Black and Hispanic/Latino subsamples, respectively, met this criterion. Additionally, the socioeconomic composition of those in the non-poverty groups is quite disparate by race/ethnicity (Figure S2). Data sparsity and lack of SDoH overlap across groups may produce unreliable estimates for the modifier effects of interest. Ideal samples would be comprised of better-overlapping distributions of poverty among the racial/ethnic groups tested, and thus well-powered at both ends of income within groups, in order to rigorously test differential effects between groups—though admittedly such samples are not typical in American science, and when achieved are not representative of US demographics.

Semanaz et al. articulate some of the reasons examination of race*poverty interactions, or stratified models, may be of high value in poverty research. Readers eager to test for these interactions, however, should take care and avoid a default-mode approach and ask themselves critical questions raised by Krieger et al⁵: "Who produces and controls the data? To what end? And engaging with what history?" (p. 2). Some among the Semanaz et al. author team are longstanding experts in the study of race, poverty, and health, and their work in this issue highlights how well-defined conceptualization and modeling can lead to improved precision or biased estimates. However, when conducted with an ahistorical lens and without socioecological content experts to guide the design and interpretation, findings from similar research could be used to argue for harmful redistribution of interventions and resources. Accordingly, a crucial approach to prevent these harms is to actively involve people with lived experience and research expertise in structural racism in the research process to ensure accurate approaches for presenting these complexities.^{4,9} Emphasizing subgroup variation in effects on children's brain and behavior, particularly when heterogeneity is the norm among humans, warrants careful reflection on its ultimate goal and whether the advantages outweigh the potential harm. Overall, we are wary of the suggestion that others "should assess interaction between poverty and race/ethnicity and/or stratify" to look for differences, given huge variability in most scientists' readiness to do so responsibly.

We share enthusiasm for refining understanding of the interplay between race/ethnicity, poverty, and neurodevelopment and have long struggled with ascertaining the best methods for doing so. We appreciate the opportunity to reflect on the complexities highlighted by Semanaz et al.'s investigation. While study of subgroups or potential confounding due to group membership is necessary and valuable given specific conditions,^{4,5} findings from poorly designed or articulated subgroup studies have the potential to reify rigid categorizations that perpetuate negative stereotypes, oversimplify complex realities, and contribute to misguided intervention efforts and policies. When approached with intention to address societal inequities and rigor, further research into the complex interplay among social constructs of race/ethnicity, structural and individual racism, socioeconomic status, and variations in environments will advance the health of our population, particularly for those who need it most.

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