

LETTER TO THE EDITOR

General correspondence

Race-conscious medicine: a response to critique

In a recent Letter to the Editor, Choy encouraged ‘caution’ when ‘abolishing adjustment for race in estimating equations and reference values’, referencing our recent perspective in *The Lancet*.^{1,2} Choy provides examples of clinical findings that are purportedly ‘influenced by race’, including white blood cell (WBC) count, thyroid-stimulating hormone (TSH) and glomerular filtration rate (GFR). Choy also raises the issue of racial diversity in clinical trials, arguing that exclusion of racialised groups could undermine study validity.¹

Here, we reiterate the thrust of our original perspective: that race-conscious medicine ‘emphasises racism, rather than race, as a key determinant of illness and health’.² We thereby identify the flaws in Choy’s concern, contest the cogency of the evidence presented, and comment on the issue of racial diversity in clinical trials.

First, by suggesting that reference values and test interpretations are ‘influenced by race’, Choy implies that race captures a meaningful component of human genetic diversity. As we discussed,² race is a political category devised to enshrine social hierarchies based on scientifically racist notions of human variation. Human phenotypic – and genetic – variation occurs gradually across geographic space, with few discontinuities. Accordingly, humans cannot be divided into distinct biological ‘types’ based on race.³

Second, the evidence advocating for retention of race as a predictor of biological variation is specious. Choy cites significant differences in WBC counts observed between ‘Black’ and ‘non-Black’ participants.^{1,4} Several factors can explain this variation in WBC count, including age, sex, genetics, infection status, comorbidities and exposures, including medications and environmental pollutants;^{5–8} Coates *et al.* only accounted for the former two. Due to structural racism, including environmental racism and racial segregation, distributions of environmental pollutants occur inequitably, disproportionately

harming marginalised communities.⁹ Likewise, the non-systematic review by Surks and Boucai proposes race-based variation in TSH without accounting for multiple confounders related to exposure to racism.¹⁰ Choy also presumes that the 16% higher GFR observed in Black patients reflects a true race-based difference, without acknowledging the evidence to the contrary, the logical flaws for including a race coefficient, and the existence of valid alternatives, such as eGFR computed from cystatin C (eGFR_{cys}).¹¹ In addition, the National Kidney Foundation and American Society of Nephrology task force concluded that race does not belong in eGFR calculations.¹²

Last, Choy raises concerns that ‘excluding racial minority individuals from Phase 1 clinical trials could raise questions on the validity of a trial’s findings’.¹ We agree with this assertion – and note we never argued *against* racial diversity in clinical trials – with an important caveat. Race is a flawed means of capturing human genetic diversity; however, current racial categories may imperfectly encompass geographic variation in alleles that contribute to biological phenotypes. A more fitting alternative would ensure studies capture variation across principal components of genome-wide assessments of ancestry.

As with any policy reform, race-conscious medicine should be adopted with careful consideration paid to potential unintended consequences. That said, we remain confident that our approach – emphasising racism, rather than race – will advance health equity.

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