JIM Letter to the Editor

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There is no 'African American Physiology': the fallacy of racial essentialism

We respectfully submit this letter in response to the May 2020 review article 'Obesity in African-Americans: the role of physiology' by Gower and Fowler [1]. We write to call attention to the authors' problematic assumptions about race, which were particularly concerning to see in an influential publication like the *Journal of Internal Medicine*.

Gower and Fowler propose a physiologic basis for obesity in 'obesity-prone' Black women who consume high-glycaemic diets. This premise rests on inappropriate notions of racial essentialism, which reinforce false beliefs in 'innate' genetic abnormalities within Black bodies. Further, it posits that unspecified genomic racial differences justify disparities in a phenotype as multifactorial and complex as *obesity*, without referencing extensive existing literature on the social environment. This inference is unacceptable. It produces illegitimate science that reifies racial determinism and obfuscates the contribution of structural racism to racial health inequalities. We elucidate by discussing verbiage of the original article and conclude by offering recommendations.

First, the authors assume that Black women possess a genetically inherent metabolic phenotype. This presumption is founded in racial essentialism, the theory that an intrinsic genomic essence defines all members of a racial category. In reality, racial groups cannot be categorized as biologic 'types'. 'Race' is a poor proxy for genetic differences, and 'phenotypic' features commonly referenced in discussions of race – such as skin colour or hair texture – fail to correspond to discrete categories or underlying physiology. Indeed, genetic variation within African and African diasporic populations is greater than other human groups because of the antiquity of human evolution in Africa. Human genetic variation is clinically distributed and cannot be distinguished along racial (e.g. Black) or continental (e.g. African) lines [2]. It is imperative to understand that socially constructed racial identities are poor representatives of geographic ancestry and do not warrant genetic inferences. The authors' model of racial–genetic determinism operates contrarily to existing scientific consensus.

Secondly, though they mobilize arguments about genetic racial differences, Gower and Fowler provide no evidence of genetic differences between groups racialized as 'Black' or 'White'. Although narrowly and geographically circumscribed population clusters may possess high genetic similarity, this is not true for all members of a racial group, particularly amongst Black Americans who have considerable admixture and genetic introgression. For instance, there is significant technical difference between 'Yoruba' – an ancestral ethnic group on the West African coast – and 'Black' – a fluid racial descriptor that includes globally dispersed diasporic populations. These are operationally distinct terms and cannot be used as research variables interchangeably [2, 3]. Hypotheses regarding genetic causation require thoughtful, technical analysis of genetic data, of which Gower and Fowler do not review.

Thirdly, the authors' proposed model of insulin sensitivity to high-glyacemic diets as a determinant of obesity in Black women fails to meet scientific rigour. This model, elsewhere termed the carbohydrate–insulin model, predicts that diets relatively low in carbohydrates induce insulin secretion, increase fat oxidation, decrease hunger and increase body fat loss. However, inpatient feeding studies have failed to support these predictions, specifically for body fat loss [4]. When accounting for race, Gower and Fowler build towards a five-way interaction model for obesity that includes (1) female sex, (2) Black race, (3) high insulin secretion, (4) high insulin sensitivity and (5) high-glycaemic index diet. Even excluding problematic racial essentialism, this model of obesity risk originates from a series of hypothesis-generating studies and is not derived from robust, prospective research [4]. Causal inferences cannot be drawn from this model, not only given its complete exclusion of environmental contributors – which impact food security – but also since it depends on underpowered, post hoc analyses of small

intervention trials not designed to assess the interactions tested.

Fourthly, the authors engage in racially inflammatory rhetoric. For instance, they use the term 'Caucasian' to refer to White populations, which is based on archaic colonial-era racial categories, and expressly prohibited by the Council of Science Editors [3]. In addition, the authors cite literature attributing racial differences in insulin sensitivity to atypical fat distribution, an assertion redolent of scientific racism. Similar observations about Black people's skulls, facial features and lung capacity have been marshalled to assert inherent inferiority. The example of fat distribution is perhaps most tragically exemplified by Sara Baartman, a Khoisan woman who – because her breasts and buttocks were large relative to White women – was captured and exhibited throughout Europe as a model of the primitivity and fundamental difference of Black women.

Fifthly, in their discussion of obesity disparities, Gower and Fowler never mention racism, a known contributor to health inequity that operates on institutional and interpersonal levels [5]. For instance, they state that, 'AA [African Americans] lose less weight than EA [European Americans] in clinical weight loss trials' and that African Americans 'engage in less physical activity'. However, they disregard robust evidence that racist policies in Reconstruction-era and contemporary real estate practices reinforce neighbourhood segregation, strand communities of colour in unsafe neighbourhoods with less green space and recreational facilities, and constitute major barriers to regular physical activity and attainment of ideal body weight [6]. Because of continued unequitable distribution of community resources, Black people are also more likely to live in regions that possess higher density of fast-food restaurants without dependable access to fresh groceries. Significant racial inequities in income and wealth also constrain healthy eating. These and other issues of structural inequity – in health care and education access, targeted advertising, beauty standards, mass incarceration and socio-economic status – are obvious and relevant considerations that the authors neglect in their appraisal of obesity disparities.

Sixthly, the authors similarly ignore the direct, physiological effects of interpersonal racism. Research establishes that chronic stress exposure – from undue discrimination and scrutiny – can induce long-term activation of the hypothalamic–pituitary–adrenal (HPA) axis, raising plasma glucose levels and mobilizing insulin release [7]. Racism may also promote epigenetic modification of metabolic pathways. The two previous examples of institutional and interpersonal racism – racial segregation and stress-triggered HPA activity – represent toxic health effects of racial injustice. One engineers inequitable external environments that constrain ability to pursue energy balance, the other galvanizes metabolic dysregulation. Both concretely establish that race cannot be conceptualized (or researched) as a simple, genetic variable in isolation of sociopolitical context.

These six problems are evident in the research question itself – 'is there a meaningful physiologic difference in Black and White patients?' The initial presumption of racial essentialism – which proclaims *Yes*, there *is* meaningful genetic racial difference – is so strong that Gowers and Fowler interpret *any* phenotypic difference as evidence of *genotypic* difference. Indeed, they do not even define race, their major operative variable, and make genetic inferences, absent any genetic data. And because the authors do not allow any other explanation – beyond intrinsic racial difference – the only conclusion they can reach is the premise they began with: physiologic differences rooted in unspecified and unmeasured genetic differences between races exist and account for racial obesity disparities. The circular logic reveals a prior commitment to genetic explanation that even their own evidence cannot penetrate.

The issues in Gower and Fowler's use of race reflect a larger concern wherein medical education, research and practice consistently – harmfully – mobilize inappropriate notions of race to patient detriment. The longstanding presence of racial essentialism in medicine amplifies the need to integrate critical race theory and structural competency into physician training. These frameworks prepare trainees to understand how social factors affect patient health outcomes – and shape the questions researchers and clinicians ask – so that physicians do not rely on assumptions of genetic predisposition to rationalize devastating racial health inequities. Better education and professional diversity are required so that doctors and scientists can grasp the complexity of racial formation and institutional injustice, make valid inferences, provide high-quality care and advance health equity.

Researchers face challenges when writing about race, since racial data are inherently imprecise and limited [3]. This is especially true if researchers do not – or cannot – critically engage or even *define* race. Because using race correctly is difficult – and because misuse engenders harm – comprehensive guidelines exist [3]. They recommend, for instance, that racial labels should not be used as proxies for genetic variation, and that researchers must make every effort to consider issues of social class, racism and environmental exposure. These cogent standards are readily available and should always be met by investigators.

Scientific scholarship is meant to facilitate the creation of a healthier and more just society. We recognize this shared mission with Drs. Gower and Fowler and respectfully ask that moving forward, they – and other readers – consider the harms of racial–genetic determinism and scientifically engage with structural determinants of health. We encourage other investigators to make a concerted effort to investigate a robust body of literature that critically appraises the ethical use of race in science. Similarly, we hope this letter reinvigorates conversation amongst scientific editors at large to re-examine protocols to ensure peer-reviewed works meet contemporary standards for discussion of race. Lastly, we ask that the journal leadership commit to publishing more rigorous research that explores the multifactorial pathways between inequity and adverse health, in order to explicitly acknowledge that racism has profound implications for all dimensions of physical, mental and social health.

Conflicts of Interest

All authors have no conflicts of interest to report.

Author Contribution

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References

1 Gower BA, Fowler LA. Obesity in African-Americans: the role of physiology. *J Intern Med* 2020; **288:** 295–304.
2 American Association of Physical Anthropologists, AAPA Statement on Race and Racism, unanimously accepted March 2019. Available at https://physanth.org/about/position-statements/aapa-statement-race-and-racism-2019/
3 Kaplan JB, Bennett T. Use of race and ethnicity in biomedical publication. *JAMA* 2003; **289:** 2709–16.
4 Hall K. A review of the carbohydrate–insulin model of obesity. *Eur J Clin Nutr* 2017; **71:** 323–6.
5 Gravlee C. How race becomes biology: Embodiment of social inequality. *Am J Phys Anthropol* 2009; **139:** 47–57.
6 Bailey ZD, Krieger N, Agénor M, Graves J, Linos N, Bassett MT. Structural racism and health inequities in the USA: evidence and interventions. Series: America: Equity and Equality in Health. *Lancet* 2017; **389:** 453–1463.
7 Cedillo YE, Lomax FO, Fernandez JR, Moellering DR. Physiological significance of discrimination on stress markers, obesity, and LDL oxidation among a European American and African American Cohort of Females. *Int J Behav Med* 2020; **27:** 213–24.

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