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## Racial differences in biomarkers should point towards structural, not genetic, determinants

We read with interest the article by Commodore-Mensah and colleagues (July 2023) [1] describing observed racial and ethnic differences in N-terminal pro-brain-type natriuretic peptide (NT-proBNP) levels in adults. The study documents important biomarker differences in different population groups which could help better understand cardiovascular inequities. However, we are concerned that the paper references studies that “speculated that there may be a genetic basis for the racial differences in NT-proBNP” [1] without giving context, moves between socially defined racial groups and ancestry without clear explanation, and does not explicitly name racism as a driver of racial inequities. We are not questioning the authors’ intentions, as we all have a shared goal of ending health inequity, but we want to ensure that the manuscript does not inadvertently bolster beliefs in inherent, categorical differences between people of different racial groups. Racial health inequities including observed differences in biomarkers are evidence of the clinical and biological impacts of structural racism [2], therefore specifically naming racism is needed to focus future studies and solutions on rectifying the structural causes.

In the paper, the authors state that “race/ethnicity is not a biological construct but a social one;” that “the potential for residual confounding cannot be ruled out,” and that they “did not have information on genetic ancestry.” However, they later state that differences in NT-proBNP levels appear in young, healthy adults and “may hint at underlying genetic determinants that differ by ancestry” [1]. They do not present evidence in their study population to support the jump from self-identified race to ancestry. Even if they had data on ancestry in the study population, there would remain questions about what is captured by ancestry designations, as they too are poor proxies for genetics and correlate with exposure to unmeasured social and environmental confounders [3,4].

As is mentioned in the limitations section, residual confounding could explain the observed association between NT-proBNP levels and racial and ethnic categories [1]. Specifically, when adjusting for socioeconomic factors the model uses ‘income’ not ‘wealth,’ which can be less specific. In addition, the model uses binary variables to adjust for ‘employment,’ ‘health insurance status,’ ‘hypertension,’ and ‘diabetes’ [1]. Furthermore, there is no adjustment for neighborhood level factors or experiences of chronic stress, racism, or other discrimination. These are common limitations for datasets and models and are not disqualifying. However, finding that NT-proBNP levels vary by racial and ethnic category after adjustment for traditional cardiovascular risk factors and some socioeconomic factors, should lead us to look towards unmeasured structural confounders before suggesting that the

remaining variation could be genetic [2,5]. Documenting health inequities is vital, but knowing the massive impact of structural racism on health we must look at how racism produces differences [2] so that we can focus on solutions to end the inequalities.

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### CRediT authorship contribution statement

**Francois Rollin:** Conceptualization, Writing – original draft, Writing – review & editing. **Amy Miller:** Writing – review & editing. **Alex Galloway:** Writing – review & editing.

### Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Francois Rollin\*, Amy Miller, Alex Galloway  
 Department of Medicine, Faculty Office Building, Emory University School of Medicine, #496, 69 Jesse Hill Jr Drive, Atlanta, GA, United States

\* Corresponding author.  
 E-mail address: [frrollin@emory.edu](mailto:frrollin@emory.edu) (F. Rollin).

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