as BP tracks from childhood well into the third and fourth decades of life.[12] We are currently investigating how perinatal programming factors (eg, FGR), the RAS, and nutrition (eg, early-life growth and salt intake) interact with the development of hypertension over the life course in our Prenatal Events-Postnatal Consequences birth cohort.[13]

Unfortunately, little is known about how to target preventive and therapeutic nutritional strategies centered on salt intake in this high-risk pediatric population. For now, we must follow current guidelines for the general pediatric population.[14] Intrarenal RAS or its role in programmed hypertension in humans remains to be defined. High-quality clinical trials and observational studies will better define these strategies and delineate these mechanisms across the life course.

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Racial disparities in Kawasaki disease are the effect not the cause

To the Editor:

The report by Padilla et al should be applauded for investigating racial disparity in children with Kawasaki disease, yet the methodology and conclusions must be further examined.[1] When racial or ethnic disparities are identified, it is imperative that we scrutinize what race or ethnicity is serving a proxy for in the observed association, avoiding the pitfall of using race as a proxy for genetics.

The authors cite that socioeconomic status has been associated with worse outcomes in Kawasaki disease and appropriately question whether the known over-representation of black children in this group could explain the disparities.[2] Obesity research highlights how socioeconomic status and race both impact health making it important to factor both in analyses, which was not performed in this study.[3,4]

Rash was identified less frequently in black children in the study.[1] Research shows erythema migrans is identified less often in black individuals, leading to a higher incidence of late stage Lyme disease presentations.[5,6] Racial bias in medical resources and education means that dermatologic conditions are less likely to be identified on black skin, further exacerbating health disparities.[7] It is important to acknowledge the possible impacts of this phenomenon in Kawasaki disease.

Although genetics have been shown to play a role in some individuals' susceptibility to Kawasaki disease, the cause of Kawasaki disease remains unknown and genetics do not explain all cases.[8] Further, the scientific literature continues to show that race cannot be used as a proxy for genetics.[9,10] Thus, the authors' recommendation to include race in treatment algorithms for Kawasaki disease warrants caution. The use of race in diagnostic or therapeutic decision making can exacerbate disparities by providing different care based on race.[11] Racial and ethnic disparities are not intrinsic to the individual's biology, but rather the systemic inequity and racism that plagues our society.

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Reply

To the Editor:

Kelly and James[1] express concerns regarding our study that identified racial disparities in the clinical presentation and outcomes for patients with Kawasaki disease who received similar treatment and were admitted to the same hospital.[1,2] Kawasaki disease shows differences in susceptibility and risk according to populations worldwide.[3] Ignoring potential biological factors responsible for population variation may impede research progress for treatments and propagate disparity.

The letter cites Vyas et al, who opined that race-adjusted algorithms guide decisions that may direct more attention or resources to White patients than to members of racial and ethnic minorities.[4] In contrast, our article highlighted poorer clinical outcomes among Black children with Kawasaki disease, suggesting that they require heightened attention and may be candidates for more aggressive therapy or a different approach.

Race cannot be used as proxy for genetics; however, genetic differences can result in varying health outcomes.[5] The 1000 Genome Project discovered differences in genetic variations among populations with distinct ancestry.[6] Each race or population exhibits "private" genetic variants that could potentially impact biological response and drug mechanisms. For instance, East Asians show the highest frequency for polymorphisms that decrease clopidogrel metabolism and efficacy.[7] Similarly, we previously reported that genetic variants associated with Kawasaki disease intravenous immunoglobulin treatment response differed in frequency by race.[8,9] Our study was retrospective and genetic analyses for ancestry were not available for the studied cohorts. However, we are determined to further identify genetic markers impacting Kawasaki disease, as well as their population-based frequency variation to develop a personalized treatment approach and help decrease disparities faced by some populations.

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