

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.

Michael S. Kelly, MD
Aisha K. James, MD, MS
Department of Pediatrics

Massachusetts General Hospital for Children and
Department of Internal Medicine
Massachusetts General Hospital
Boston, Massachusetts

<https://doi.org/10.1016/j.jpeds.2020.11.021>

The authors declare no conflicts of interest.

References

1. Padilla LA, Collins JL, Idigo AJ, Lau Y, Portman MA, Shrestha S. Kawasaki disease and clinical outcome disparities among black children. *J Pediatr* 2020.
2. Fontenot K, Semega J, Kollar M. Income and poverty in the United States: 2017. Suitland. Suitland-Silver Hill (MD): US Census Bureau; 2018.
3. Ogden CL, Carroll MD, Flegal KM. High body mass index for age among US children and adolescents, 2003-2006. *JAMA* 2008;299:2401-5.
4. Rogers R, Eagle TF, Sheetz A, Woodward A, Leibowitz R, Song M, et al. The Relationship between childhood obesity, low socioeconomic status, and race/ethnicity: lessons from Massachusetts. *Child Obes* 2015;11:691-5.
5. Fix AD, Peña CA, Strickland GT. Racial differences in reported Lyme disease incidence. *Am J Epidemiol* 2000;152:756-9.
6. Nolen L. How medical education is missing the bull's-eye. *N Engl J Med* 2020;382:2489-91.
7. Buster KJ, Stevens EI, Elms CA. Dermatologic health disparities. *Dermatol Clin* 2012;30:53-9.
8. McCrindle BW, Rowley AH, Newburger JW, Burns JC, Bolger AF, Gewitz M, et al. Diagnosis, treatment, and long-term management of Kawasaki disease: A scientific statement for health professionals from the American Heart Association. *Circulation* 2017;135:e927-99.
9. Maglo KN, Mersha TB, Martin LJ. Population genomics and the statistical values of race: an interdisciplinary perspective on the biological clas-

sification of human populations and implications for clinical genetic epidemiological research. *Front Genet* 2016;7:22.

10. Rosenberg NA, Pritchard JK, Weber JL, Cann HM, Kidd KK, Zhivotovsky LA, et al. Genetic structure of human populations. *Science* 2002;298:2381-5.
11. Vyas DA, Eisenstein LG, Jones DS. Hidden in plain sight—reconsidering the use of race correction in clinical algorithms. *N Engl J Med* 2020;383:874-82.

Reply



To the Editor:

Kelly and James¹ 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.

Luz A. Padilla, MD
Department of Epidemiology
School of Public Health
University of Alabama at Birmingham
Birmingham, Alabama

Michael A. Portman, MD
Division of Pediatric Cardiology
Department of Pediatrics
University of Washington and
Seattle Children's Research Institute
Seattle, Washington

Sadeep Shrestha, PhD
Department of Epidemiology
School of Public Health
University of Alabama at Birmingham
Birmingham, Alabama

<https://doi.org/10.1016/j.jpeds.2020.11.022>

Supported by the National Institutes of Health (NHLBI-R01HL146130 [to S.S. and M.P.]) and the Quetelet Endowed Professorship Research Fund (to S.S.). The authors declare no conflicts of interests.

References

1. Kelly M, James A. Racial disparities in Kawasaki disease are the effect not the cause. *J Pediatr* 2020.
2. Padilla LA, Collins JL, Idigo AJ, Lau Y, Portman MA, Shrestha S. Kawasaki disease and clinical outcome disparities among black children. *J Pediatr* 2020 Sep 24:S0022-3476(20)31244-0.
3. Elakabawi K, Lin J, Jiao F, Guo N, Yuan Z. Kawasaki disease: global burden and genetic background. *Cardiol Res* 2020;11:9-14.
4. Vyas DA, Eisenstein LG, Jones DS. Hidden in plain sight - reconsidering the use of race correction in clinical algorithms. *N Engl J Med* 2020;383:874-82.
5. Portman MA, Shrestha S. One size does not fit all: genetic prediction of Kawasaki disease treatment response in diverse populations. *Circ Cardiovasc Genet* 2017;10:e001917.
6. 1000 Genomes Project Consortium Auton A, Brooks LD, Durbin RM, Garrison EP, Kang HM, et al. A global reference for human genetic variation. *Nature* 2015;526:68-74.
7. Pan Y, Chen W, Xu Y, Yi X, Han Y, Yang Q, et al. Genetic polymorphisms and clopidogrel efficacy for acute ischemic stroke or transient ischemic attack: a systematic review and meta-analysis. *Circulation* 2017;135:21-33.
8. Shrestha S, Wiener HW, Olson AK, Edberg JC, Bowles NE, Patel H, et al. Functional FCGR2B gene variants influence intravenous immunoglobulin response in patients with Kawasaki disease. *J Allergy Clin Immunol* 2011;128:677-80.
9. Shrestha S, Wiener H, Shendre A, Kaslow RA, Wu J, Olson A, et al. Role of activating FcγR gene polymorphisms in Kawasaki disease susceptibility and intravenous immunoglobulin response. *Circ Cardiovasc Genet* 2012;5:309-16.