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Comment

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Systematic decision frameworks for the socially responsible use of precision medicine

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Deep learning techniques and whole-genome sequencing promise to increase well-being but also risk perpetuating psychological essentialism, potentially justifying inequality. In this Comment, we offer two much-needed systematic frameworks for clinicians and researchers to avoid essentialist inferences and unfair treatment: (1) a data-driven method for detecting causal fairness in precision health and (2) an ethical framework for determining when it is morally permissible to use racial classifications in population health research.

Systematic decision frameworks for the socially responsible use of precision medicine

The simultaneous rise of powerful deep learning techniques and whole-genome sequencing in humans promises to usher in a new era of precision health. Clinicians and researchers are rapidly acquiring the ability to predict, based on genomic data, both the prevalence of disease and the efficacy of treatment. While these capabilities have the potential to drastically increase human well-being, they also come with potentially troubling consequences. These include reinforcing harmful stereotypes surrounding race and other social categories, exacerbating existing health inequalities, and obscuring data in ways that prevent efficient and effective medical interventions.

Perhaps most concerning is the possibility that the use of genomic data in healthcare settings could perpetuate psychological essentialism, or the common bias to treat membership in a socially salient category (e.g., race) as an inherent characteristic of an individual that is causally responsible for the individual's other features and attributes[1–3]. Essentialist beliefs have been historically used to justify deeply unfair treatment and allocation of resources, as well as blatant abuses of human rights from slavery to medical experimentation[4]. This early-emerging and persistent psychological bias continues to shape how people understand human health and behavior today[5,6], especially in the context of genetic attributions for various human conditions[7]. Crucially, psychological essentialism among researchers and practitioners can contribute to unfair medical treatment[8,9] and lead to undervaluing social and environmental determinants of health–key contributors to health disparities[10–13]. Furthermore, recent research on human genetic diversity has been increasingly used to support white nationalist ideology and justify acts of racial violence[14–16].

Given these psychological biases that shape human decision making, and the danger that genomics research may be misinterpreted and misused by the public, how can medical researchers and clinicians capitalize on the advancements of precision health in a socially responsible way? Despite recent increased attention to these issues[17], the literature currently lacks systematic frameworks and recommendations for how to address these challenges. For example, a 2023 report by the National Academies of Sciences, Engineering, and Medicine (henceforth, The NASEM Report) highlighted the importance of addressing the potentially racist consequences of precision health but acknowledged that "[u]ltimate decisions about the use of population descriptors may vary depending on the specific context"[17]. The dearth of concrete strategies is particularly troublesome because, although psychological essentialism exists in the minds of individuals, these individual-level social processes have become deeply embedded in surrounding political, economic, and social structures, which serve to further perpetuate and reinforce these biases in individuals[18,19]. For this reason, implementing concrete strategies at the structural level that help to circumvent individual psychological biases will be crucial for efforts to rectify inequality.

Although it might seem simplest to avoid using racial classifications in precision health all together, we suggest that wholesale elimination of race is also not the answer because this could impede reparative healthcare legislation targeting racial groups historically marginalized in healthcare systems. Many classifications that might serve as alternatives to race (e.g., ancestry, ethnicity, demes) are also vulnerable to the social and moral harms associated with race, and these alternatives often use categories that resemble racial categories, which can also foster essentialist views[20–22]. So, while alternative classifications may be better suited for certain research programs, calling race by another name is not enough. Rather, systematic strategies are needed to prevent either race or these alternatives from perpetuating social harms.

In this Comment, we draw on techniques from psychology, philosophy, statistics, and computer science to make concrete recommendations for how clinicians and researchers can conduct and translate ongoing research and provide patients with precision healthcare while guarding against essentialist inferences and unfair treatment. Specifically, we offer two concrete tools for the socially responsible use of precision health: (1) a data-driven method for the detection of causal fairness and unfairness in precision health contexts, and (2) an ethical framework for determining when (if ever) it is morally permissible to use a racial classification in population health research. We hope that these tools can offer useful strategies to circumvent essentialist biases among individual practitioners and researchers, which we suggest will be particularly effective when they are incorporated into the existing social systems and structures in which these biases are ingrained.

A causal fairness model for precision health

The core principle behind our proposed model for precision health is *causal fairness*[23,24]. That is, a process of diagnosis or recommendation is fair with

respect to some protected characteristic to the extent that a person does not receive a different diagnosis or recommendation *because* they possess a particular protected characteristic (i.e., their race or gender). To illustrate, suppose a genetic feature is correlated with both kidney failure and Black race. Here, we make no assumptions about what that genetic feature may be, because we want our framework to be applicable in cases where black-box deep learning methods are used to detect genomic predictors of disease in the absence of richly-formulated biological assumptions[25]. Suppose further that, under these conditions, a clinician using genomic data to predict risk of kidney failure might assign higher levels of risk to Black patients. Under our causal fairness model, such a practice can only be considered fair if the clinician would make the same prediction for an otherwise identical patient who has the same genetic feature but is not Black. In this respect, causal fairness is a species of *counterfactual fairness* (see[26]). Under causal fairness, a person is treated fairly if their treatment in the actual world matches their treatment in a counterfactual scenario in which features that ought to be causally irrelevant to their diagnosis are held fixed.

Although this kind of counterfactual knowledge can never be entirely secured, especially for individual cases, one can examine the extent to which a given data set is consistent with different causal structures. For some causal structures, there is a causal pathway from a genomic feature through the possession of a protected characteristic to the predicted outcome (e.g., the genetic feature causes patients to be perceived as Black and causes them to be at greater risk of kidney failure, while at the same time, being of Black race is itself a probabilistic cause of kidney failure due to systemic racism; Fig. 1a). In such cases, using the genomic feature to generate differential predictions may be unfair with respect to that protected characteristic (e.g., race), since if a person were perceived as having a different race, then the prediction of their risk of kidney failure would be different. Moreover, such predictive practices run the risk of reinforcing the pernicious essentialist biases discussed above. On the other hand, data may be more consistent with a causal structure in which possession of a genomic feature is a common cause of both a person's protected characteristics and the predicted outcome (e.g., people who are Black are more likely to also have the genetic feature), but there is no direct causal relationship between the protected characteristic and the outcome in question (e.g., the feature predicts kidney failure regardless of race; Fig. 1b). In these cases, we have much better evidence for the fairness of using the genetic feature to predict outcomes.

At this stage, it is important to address what we mean by 'causation' for the purposes of this paper. Indeed, one might question how a biological category like a genetic feature could be a cause of a social phenomenon like how someone is racialized. Following Gerstenberg and Tenenbaum, we view causal theories as "probabilistic, generative programs," according to which effects can be computed from their causes via noisy algorithms[27]. One can view the way someone is racialized as computed (via a stochastic, socio-physical algorithm) from a myriad of factors, some of which may include genetic features. By the same token, an increased probability of realizing certain health outcomes may or may not be influenced, via a similarly stochastic, socio-physical algorithm, by someone's race. The exact structure of the probabilistic programs that generate the data we observe in the world leaves statistical traces in that data. For example, though two variables may be correlated even though one is not caused by the other, these correlations will disappear once we condition on the causes of both variables[28]. This principle, known as the "causal Markov condition," enables us to induce causal structure from purely associational data in at least some cases[29]. As an example, the use of windshield wipers and the use of umbrellas on a given street may be correlated over time, but this correlation disappears once we restrict our attention to only those cases in which it is raining.

As a second point of clarification, it is crucial to note that in some cases, we may not be especially concerned with avoiding the use of genetic predictors of disease whose predictive accuracy is largely due to a socio-causal pathway from the presence of the genetic feature to the protected characteristic and then to the presence of the disease. In other words, we may identify a particular genetic trait as responsible for a particular pattern of racialization, which in turn may be responsible for a person's having a higher likelihood of developing a disease. Strong evidence for such a causal pathway could be used to redress health inequalities in a salutary way. What we aim to caution against, however, is the use of such a genetic predictor *without* explicit attention to and communication of the social pathways through which it operates. Such usage risks the essentializing inferences highlighted above, while also suggesting biological solutions to what are often demonstrably social problems.

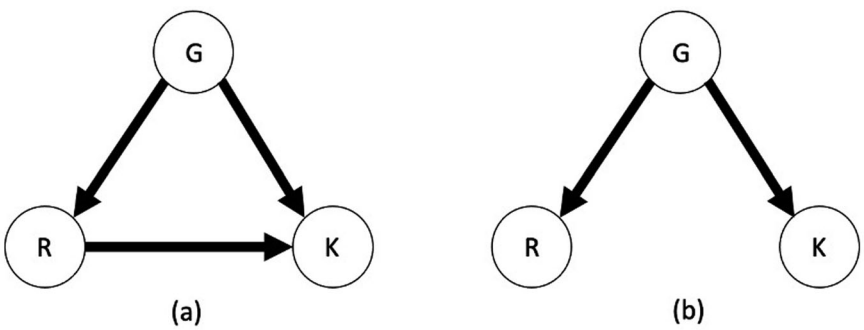

Fig. 1 | Possible causal structures that could produce data consistent with a correlation between race and kidney failure. a A causal structure in which both genetics and perceived race is a cause of kidney failure. **b** A causal structure in which only genetics is a cause of kidney failure, but kidney failure remains correlated with perceived race. The causal structure shown in (**a**) is one in which a genetic trait (**G**) causes a patient both to be more likely to be perceived as being Black (represented by the race variable **R**) and to be more susceptible to kidney failure (represented by the variable **K**). In addition, according to this causal structure, being perceived as being Black is a direct cause of kidney failure. If we observe data generated by this structure and use solely the observed correlation between the genetic trait and kidney failure to predict instances of kidney failure, then we are likely to assign Black people a higher risk of kidney failure *in virtue of their race*, violating causal fairness constraints. By contrast, if we observe data generated by the causal structure in (**b**), then any correlation between Black race and kidney failure will be *solely* due to the fact that possession of the genetic trait is a common cause of both being perceived as being of Black race and kidney failure. In this scenario, if we observe the genetic feature and use it to predict a greater risk of kidney failure, we are not treating any person differently in virtue of their race.

Earlier accounts of causal fairness, such as those given by Nabi and Shpitser (2018) and Chiappa and Isaac (2019), focus solely on those features of a data-generating causal structure that can differentiate between fair and unfair predictions[23,24]. In the real world, however, our data are often compatible with multiple data-generating causal structures and at most provide *better* evidence for one over another. To statistically test whether our data are more likely to have been generated by a fairness-preserving causal structure, and to therefore make a tractable inference about the fairness of using a given genetic trait as a predictor, we can measure the correlation between the protected characteristic (e.g., race) and the outcome (e.g., kidney failure) both in the case where the genetic feature is present and in the case where it is absent. Several mathematical measures of correlation could prove useful in this context, including the chi-square effect size or mutual information[23]. Lower values of these measures indicate a greater degree of observed conditional independence and a greater likelihood that the data is generated by a structure like Fig. 1b. Higher values provide weaker evidence for conditional independence and a greater likelihood that the data is generated by a structure like Fig. 1a[24].

Once we obtain these measures of conditional independence, they can be traded off against measures of other factors–including predictive accuracy, clinical expediency, and risk aversion–to generate an all-things-considered measure of the clinical and societal value of using a genetic feature as a predictor of health outcomes.

A normative framework for morally permissible uses of race in precision health

In addition to the data-driven model of causal fairness, we propose a normative framework to adjudicate morally permissible uses of race in precision health—that is, uses of race in precision health that conform to the *moral* standards (i.e., ethical considerations) necessary for the practice of good medicine[30]. Here, we understand morally permissible to mean morally optional, in that to perform the action (in this case, using race in precision health) is neither morally required nor morally prohibited. We have chosen to focus on morally permissible uses of race in precision health because we are not (yet) convinced that using a racial classification in precision health is ever morally required. However, we are convinced that wholesale elimination of race in precision health is *not* required, so there may be contexts in which it would not be morally prohibited[30].

The moral standards most relevant to the current discourse include the virtues of justice, benevolence, and trustworthiness, where *justice* is understood as respect for another's moral status and the consequent rights that accompany such a moral status; *benevolence* as goodwill toward others, or the proper care and concern of another; and *trustworthiness* as the state of being technically and morally competent in whatever is being entrusted. Here technical competence is a variety of know-how about the technical aspects of precision health, and moral competence is how individuals or institutions are disposed toward the aforementioned virtues[30]. Note, these virtues cohere with The NASEM Report's guiding principles for "scientifically valid and trustworthy research"[17]. Additionally, like The NASEM Report's guiding principles, we believe these virtues are "mutually reinforcing"[17]. Justice, benevolence, and trustworthiness are necessary conditions to promote the aims of precision health in a way that provides total health and well-being and does not compromise the integrity of its recipients[31]. Justice preserves the dignity and respect of those receiving care, and consequently, promotes more holistic well-being. Benevolence ensures proper disposal to the moral considerations relevant to the person(s)/community of interest. Trustworthiness among health care professionals and their institutions is particularly important, given that the asymmetric dynamic between health providers and their patients exposes patients to a certain level of vulnerability.

Unlike The NASEM Report, we emphasize virtue, that is "excellences" of character. Where principle-based frameworks (like The NASEM Report) often focus primarily on action guidance, virtue-based frameworks (like ours) prioritize the character of the relevant persons and institutions, while also providing action guidance. Thus, in what follows, while we provide requirements for adjudicating morally permissible uses of race in precision health, we will be less concerned with providing a mechanical decision procedure and more concerned with highlighting the proper conditions for cultivating the sort of dispositions that promote good medicine when working with diverse populations. We are concerned both with what medical practitioners and stakeholders *do* and with how they are disposed toward their practice/investment (i.e., how they *are*). This is, in part, because rules can be misapplied and abused if those beholden to them do not know or are not inclined to value the virtues necessary for good medical practice. Still, despite the differing approaches, we take our convergence on moral standards with The NASEM Report to add further support for those standards; it demonstrates a sort of robustness of evidence.

Finally, note that these standards distinguish themselves from legal norms in that legal norms are standards of evaluation that are primarily concerned with the scope, nature, and legitimacy of political structures, whereas justice, benevolence, and trustworthiness—while relevant to legal norms—are also concerned more generally with human and societal flourishing and well-being. Given these considerations, we will understand morally permissible actions to be actions that, whether performed or not, are conducive to the promotion of justice, benevolence, and trustworthiness, and thus, human and societal flourishing and well-being.

Morally permissible uses of race should be able to prevent or mitigate the social harms associated with race-based medicine, including (amongst other things) unjust treatment due to essentialism[8], while also preserving the benefits of using race in medicine, including the ability to track racism's impact on health outcomes and medical care[13,32] and the etiology of medically relevant genetic differences between human subpopulations for both monogenic and polygenic traits and diseases[33]. Given the above, we propose that researchers and clinicians use the race-in-medicine (RIM) normative framework to determine morally permissible uses of racial classifications in precision health[30]. According to RIM, it is morally permissible to use race in medicine *if and only if:* (a) when applicable, social determinants of health are sufficiently engaged prior to or in tandem with the use of race (*social determinants requirement*), (b) the medical end(s) sought cohere with the aim of medicine and are best acquired using race (*harm minimization requirement*), and (c) the use of race does not violate the relevant just legal norms constraining medical practice more generally (*legal norms requirement*). These requirements are necessary to fulfill, because without fulfilling them, practitioners of precision health risk violating the virtues of justice, benevolence, and trustworthiness[30]. What's more, these requirements are sufficient, because taken together, they properly address the relevant objections and concerns raised when considering the use of race in medicine[30].

Note, RIM is agnostic toward race theories in that it does not privilege a particular theory of race and is applicable whatever one's theory of race may be[30]. Additionally, RIM is comprehensive in that it is meant to cover all branches of medicine—medical research, diagnosing, treatment, and education—and consequently, all these branches as they relate to precision health. Finally, RIM is comprehensive in its application at the individual (patient-physician) level, at the institutional level (e.g., medical schools and hospitals), and at the governmental level.

Given the above, how might precision health practitioners and stakeholders apply RIM's requirements? Note, fulfillment of each condition will vary depending on context and competency. So, in what follows, we offer a case study of glomerular kidney disease, providing additional examples when useful. Glomerular kidney disease is associated with APOL1 genotype, which is common in populations of sub-Saharan African ancestry[33], and is characterized by racial and ethnic disparities in incidence, prevalence, treatment, and outcome, with Black American adults and Hispanic adults disproportionately negatively impacted[34–37]. Despite glomerular kidney disease's association with APOL1 genotypes, risk for the disease is modulated by social determinants including, but not limited to, socioeconomic status[36–42], neighborhoods and housing[43,44], diet (including access to certain foods)[45,46], insurance[47], and exposure to air pollutants[48,49]. Given these known socioenvironmental factors, according to the social determinants requirement, research programs seeking to investigate glomerular kidney disease along racial lines would need to prioritize engaging these exogenous factors. For research teams, this may mean partnering with others or creating a subgroup of researchers who focus on the correlations between these exogenous factors and glomerular kidney disease before dividing genomes into groups that reflect some sort of racial classification. This shift may, amongst other things, require hiring a technically diverse group of researchers, including those trained to engage social determinants of health. Increased demographic diversity generates novel solutions[50,51] so increasing the demographic diversity of precision health's research workforce could help illuminate novel pathways previously overlooked. At the institutional level, this priority shift might be implemented through incentives like targeted grants, which could dramatically alter the research landscape. For example, the National Human Genome Institute currently earmarks just 5% of its congressional budget for studying ethical and social implications of genomics[52], despite billions of dollars spent each year to treat illnesses linked to genetic as well as social and environmental factors[53].

Engaging social determinants of health requires technical competence to identify confounding variables in medical research, as well as moral competence to intervene in environments where health burdens are largely modulated by unjust structures (e.g., housing discrimination and access to healthcare)[54]. One might worry that our recommendation to 'engage' social determinants of health is insufficiently prescriptive to offer genuine guidance to practitioners. However, like The NASEM Report, we acknowledge the context-sensitivity of clinical research and practice [17, p. 99]. We aim here to reflect that sensitivity by offering examples that illustrate how addressing individual psychological biases like essentialism may require modifying the surrounding social structures in which these biases are embedded, rather than providing a mechanical decision procedure that is ill-equipped to capture all the relevant medical and social contexts as well as "evolving best practices over time" [17, p. 99]. At the same time, the causal fairness framework described above serves as a more explicitly prescriptive framework for engaging social determinants of health that compliments RIM and can be embedded into RIM's social determinants requirement (along with other frameworks for engaging social determinants of health).

We move now to the harm minimization requirement. Although the APOL1 genotype is common in populations of sub-Saharan African ancestry[33], not every person of sub-Saharan African ancestry possesses the APOL1 genotype[55]. What's more, not every person with the APOL1 genotype will develop kidney disease[56]. This suggests that a racial classification is ill-suited to accomplish the ends sought in studying the (epi)genetic and environmental structures implicated in glomerular kidney disease. Instead, a more fine-grained classification system that distinguishes between at-risk populations is better suited for investigation. This coheres with and encompasses The NASEM Report's principles of equity and justice, validity and reproducibility, and transparency and replicability, as it takes seriously the ethical and social implications of model choice in precision health (equity and justice), demands that the level of classification reflect the population(s) observed (validity and reproducibility), and requires researchers to consider why and whether the classification scheme being used is best suited given the aims of the research program. It is also here, at the harm minimization requirement, that we believe our causal fairness model for precision health plays an important role. Again, under our causal fairness model, if the use of a racial classification for (say) diagnosis or recommendation does not lead to the same prediction for an otherwise identical patient who has the same genetic feature as a racial group but is not categorized in that race, then, according to the harm minimization requirement, an alternative classification scheme better suited to causal fairness should be used.

It should be noted, however, that in the case of medical research regarding glomerular kidney disease, racial self-reports may still be of value. This is especially true when considering the relationship between race and social determinants of health, given that the distribution of these determinants is modulated, in part, by racism[13,31]. Additionally, where genomic information is unavailable and it is too inefficient to test everyone, the use of self-reported racial membership may aid in evaluating how risk for glomerular kidney disease is distributed within and between populations. Finally, although the harm minimization requirement appears to preclude the use of racial classifications in precision health overall (as there appears to always be a better alternative), wholesale elimination of race is also not the answer because alternative classifications are subject to the same essentialist biases as race, as described above.

Finally, *the legal norms requirement* will require endorsement and adherence to current legal norms *that demonstrate respect for the moral status of research participants and the consequent rights that accompany such a moral status*. It is not enough that the practice be legal, since history is replete with examples of laws that violated the rights of individuals based on their racial membership. The legal norms informing the use of race in precision health must, then, be just[30]. For example, the NIH Revitalization Act of 1993 was meant to establish guidelines for the inclusion of minority populations in clinical research funded by the US federal government. However, with the advent of genomic research and population genetics, blind adherence to this act risked further entrenching the social imagination in essentialist assumptions. Thus, to prevent *unjust* use of the NIH Revitalization Act of 1993, application of these legal norms must be modified to address such risks.

Several legal norms constrain research on glomerular kidney disease. For example, the right to privacy torts can be understood as just legal norms, given they were created to protect the dignity of persons by preserving their ability to exercise autonomy over the most fundamental and intimate parts of their lives[57]. Any use of race in glomerular kidney disease research that unwarrantedly jeopardizes the anonymity of research participants should be emended or else eschewed. Additionally, in keeping with equal protection under federal law, glomerular kidney disease researchers reliant on genomic tests should make sure their use of participants' results cohere with the Genetic Information Nondiscrimination Act of 2008, which is meant to protect individuals against group-based, genetic discrimination in health coverage and employment.

Though not an exhaustive examination of how just legal norms factor into precision health, these observations demonstrate the inescapability of legal systems when practicing precision health, and thus the need for a normative framework that explicitly considers the legal norms constraining precision health and medical practice more generally[30]. Of course, writing and emending legislation should not fall on the shoulders of precision health

researchers and practitioners, given their limited resources and lack of legal expertize. But the legal norms requirement does signal that precision health should be a multidisciplinary collaboration, where medical researchers, IRB committees, and health institutes regularly partner with legal scholars, lawyers, social scientists, and (legal) philosophers with an eye toward the relationship between historically marginalized communities and the law.

Though not a mechanical decision procedure, RIM reinforces the virtues necessary for ethical precision health by addressing social determinants of health (some of which are modulated by historical injustices and racism), highlighting competing population descriptors, and acknowledging the need for just legal norms to constrain medical practice. An upshot of RIM is its broad scope allows it to be comprehensive and capture all relevant contexts in which precision health takes place.

Conclusion

In this Comment we offered two concrete suggestions for how clinicians and researchers can capitalize on the power of precision health to dramatically improve human well-being, while also safeguarding against the troubling psychological biases that shape human reasoning about genetic information. The causal fairness (CF) model demonstrates that, with sufficient data, researchers can measure whether the inductive use of a particular genomic trait in a clinical context is fair to members of socially salient groups that may be more or less likely to possess that trait. The race-in-medicine (RIM) normative framework proposes that it is morally permissible to use a racial classification in medicine just in case population descriptors do not obscure the role of social factors, best cohere with the aim of the research, and comport with just legal policies that inform medicine. By offering these tools, we hope to enable researchers and clinicians to both conduct more robust research and provide more just medical treatment.

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References

1. Gelman, S. A. Psychological essentialism in children. *Trends Cogn. Sci.* **8**, 404–409 (2004).
2. Medin, D. L. & Ortony, A. Psychological essentialism. *Similarity Anal. Reason.***179**, 195 (1989).
3. Prentice, D. A. & Miller, D. T. Psychological essentialism of human categories. *Curr. Dir. Psychol. Sci.* **16**, 202–206 (2007).
4. Washington, H. A. *Medical apartheid: The Dark History of Medical Experimentation on Black Americans From Colonial Times to the Present* Reprint edn, Vol. 528 (Doubleday Books, 2006).
5. Keller, J. In genes we trust: the biological component of psychological essentialism and its relationship to mechanisms of motivated social cognition. *J. Personal. Soc. Psychol.* **88**, 686–702 (2005).
6. Williams, M. J. & Eberhardt, J. L. Biological conceptions of race and the motivation to cross racial boundaries. *J. Personal. Soc. Psychol.* **94**, 1033 (2008).
7. Dar-Nimrod, I. & Heine, S. J. Genetic essentialism: on the deceptive determinism of DNA. *Psychol. Bull.* **137**, 800 (2011).
8. Hoffman, K. M., Trawalter, S., Axt, J. R. & Oliver, M. N. Racial bias in pain assessment and treatment recommendations, and false beliefs about biological differences between blacks and whites. *Proc. Natl Acad. Sci. USA* **113**, 4296–4301 (2016).
9. Lett, E., Asabor, E., Beltrán, S., Cannon, A. M. & Arah, O. A. Conceptualizing, contextualizing, and operationalizing race in quantitative health sciences research. *Ann. Fam. Med.* **20**, 157–163 (2022).
10. Braveman, P. & Gottlieb, L. The social determinants of health: it's time to consider the causes of the causes. *Public Health Rep.* **129**, 19–31 (2014).
11. Marmot, M. & Wilkinson, R. (eds). *Social Determinants of Health* 2nd edn (Oxford University Press, 2005).
12. Roberts, D. E. *Fatal Invention: How Science, Politics, and Big Business Re-Create Race in the Twenty-First Century* 50852nd edition, Vol. 400 (New Press, 2011).
13. Krieger, N. Embodying inequality: a review of concepts, measures, and methods for studying health consequences of discrimination. *Int. J. Health Serv.* **29**, 295–352 (1999).
14. Li, J. Z. et al. Worldwide human relationships inferred from genome-wide patterns of variation. *Science* **319**, 1100–1104 (2008).
15. Kozlov, M. 'All of Us' genetics chart stirs unease over controversial depiction of race. *Nature* https://doi.org/10.1038/d41586-024-00568-w (2024).
16. Carlson, J., Henn, B. M., Al-Hindi, D. R. & Ramachandran, S. Counter the weaponization of genetics research by extremists. *Nature* **610**, 444–447 (2022).
17. National Academies of Sciences, Engineering, and Medicine. *Using Population Descriptors in Genetics and Genomics Research: A New Framework for an Evolving Field*, Vol 234 (National Academies Press, 2023).
18. Lei, R. F., Foster-Hanson, E. & Goh, J. X. A sociohistorical model of intersectional social category prototypes. *Nat. Rev. Psychol.* **2**, 297–308 (2023).
19. Skinner-Dorkenoo, A. L., George, M., Wages III, J. E., Sánchez, S. & Perry, S. P. A systemic approach to the psychology of racial bias within individuals and society. *Nat. Rev. Psychol.* 18, 1–15 (2023).
20. Donovan, B. Playing with fire? The impact of the hidden curriculum in school genetics on essentialist conceptions of race. *J. Res. Sci. Teach.* **51**, 462–496 (2014).
21. Donovan, B. Framing the genetics curriculum for social justice: an experimental exploration of how the biology curriculum influences beliefs about racial difference. *Sci. Educ.* **100**, 586–616 (2016).
22. Donovan, B. Learned inequality: racial labels in the biology curriculum can affect the development of racial prejudice. *J. Res. Sci. Teach.* **54**, 379–411 (2017).
23. Nabi, R. & Shpitser, I. Fair inference on outcomes. *Proc. AAAI Conf. Artif. Intell.* **32**, 1931–40 (2018).
24. Chiappa, S. & Isaac, W. S. A causal Bayesian networks viewpoint on fairness. In, *Privacy and Identity Management. Fairness, Accountability, and Transparency in the Age of Big Data.* (eds. Kosta, E., Pierson, J., Slamanig, D., Hübner, S. F., Krenn, S.) 291 (Springer Cham, 2019).
25. Troyanskaya, O. Abstract SY36-01: decoding the biochemical, regulatory, and clinical impact of genomic mutations. *Cancer Res.* **79**, SY36–01 (2019).
26. Kusner, M. J., Loftus, J., Russell, C. & Silva, R. Counterfactual fairness. *Adv. Neural Inf. Process. Syst.* **30**, 1–11 (2017).
27. Gerstenberg, T. & Tenenbaum, J. B. Intuitive Theories. In *Oxford Handbook of Causal Reasoning* (ed. Waldmann, M.) 515–548 (Oxford University Press, 2017).
28. Pearl, J. Causality: Models, reasoning, and inference. *Econ. Ther.* **19**, 675–685 (2000).
29. Spirtes, P., Glymour, C. N. & Scheines, R. *Causation, Prediction, and Search* 1st edn, Vol. 530 (MIT Press, 2000).
30. Peebles, I. S. To race or not to race: A normative debate in the philosophy of race. *Philosophers' Imprint;* https://doi.org/10.3998/phimp.4295 (forthcoming).
31. Roberts, D. E. Understanding the impact of inequality on health, disease, and who participates in research. In *An Examination of Emerging Bioethical Issues in Biomedical Research: Proceedings of a Workshop.* 51-61 (National Academies Press, 2020).
32. Burchard, E. G. et al. The importance of race and ethnic background in biomedical research and clinical practice. *N. Engl. J. Med.* **348**, 1170–75 (2003).
33. Elliott, M. D. et al. Clinical and genetic characteristics of CKD patients with high-risk APOL1 genotypes. *J. Am. Soc. Nephrol.* **34**, 909–919 (2023).
34. Peralta, C. et al. Racial and ethnic differences in kidney function decline among persons without chronic kidney disease. *J. Am. Soc. Nephrol.* **22**, 1327–34 (2011).
35. Derose, S. et al. Racial differences in estimated GFR decline, ESRD, and mortality in an integrated health system. *Am. J. Kidney Dis.* **62**, 236–244 (2013).
36. Krissberg, J. et al. Racial-ethnic differences in health-related quality of life among adults and children with glomerular disease. *Glomerular Dis.* **1**, 105–117 (2021).
37. Krissberg, J. et al. Racial and ethnic disparities in acute care utilization among patients with glomerular disease. *Am. J. Kidney Dis.* **81**, 318–328 (2023).
38. Ward, M. Socioeconomic status and the incidence of ESRD. *Am. J. Kidney Dis.* **51**, 563–572 (2008).
39. Bello, A., Peters, J., Rigby, J., Rahman, A. & El Nahas, M. Socioeconomic status and chronic kidney disease at presentation to a renal service in the United Kingdom. *Clin. J. Am. Soc. Nephrol.* **3**, 1316–23 (2008).
40. Patzer, R. & McClellan, W. Influence of race, ethnicity and socioeconomic status on kidney disease. *Nat. Rev. Nephrol.* **8**, 533–541 (2012).
41. Vart, P., Gansevoort, R., Joosten, M., Bültmann, U. & Reijneveld, S. Socioeconomic disparities in chronic kidney disease: a systematic review and meta-analysis. *Am. J. Prev. Med.* **48**, 580–592 (2015).
42. Zeng, X. et al. Associations between socioeconomic status and chronic kidney disease: a meta-analysis. *J. Epidemiol. Community Health* **72**, 270–279 (2018).
43. Lapedis, C., Mariani, L., Jang, B., Hodgin, J. & Hicken, M. Understanding the link between neighborhoods and kidney disease. *Kidney* **360**, 845–854 (2020).
44. Novick, T. & Baweja, M. Housing: A critical contributor to kidney disease disparities. *J. Am. Soc. Nephrol.* **33**, 1471–73 (2022).
45. Klahr, S., Buerkett, J. & Purkerson, M. Role of Dietary factors in the progression of chronic renal disease. *Kidney Int.* **24**, 579–587 (1983).
46. Hunsicker, L. et al. Predictors of the progression of renal disease in the modification of diet in renal disease study. *Kidney Int.* **51**, 1908–19 (1997).
47. Jurkovitz, C. et al. Association between lack of health insurance and risk of death and ESRD: results from the kidney early evaluation program (KEEP). *Am. J. Kidney Dis.* **61**, S24–32 (2013).

48. Afsar, B. et al. Air pollution and kidney disease: review of current evidence. *Clin. Kidney J.* **12**, 19–32 (2018).
49. Wu, M.-Y., Lo, W.-C., Chao, C.-T., Wu, M.-S. & Chiang, C.-K. Association between air pollutants and development of chronic kidney disease: a systematic review and meta-analysis. *Sci. Total Environ.* **706**, 1–11 (2020).
50. Barjak, F. & Robinson, S. International collaboration, mobility and team diversity in the life sciences: impact on research performance. *Soc. Geogr.* **3**, 23–36 (2008).
51. Intemann, K. Why diversity matters: understanding and applying the diversity component of the national science foundation's broader impacts criterion. *Soc. Epistemol.* **23**, 249–66 (2009).
52. National Human Genome Research Institute, U.S. *National Human Genome Research Institute: Congressional Justification FY 2023.* https://www.genome.gov/about-nhgri/Budget-Financial-Information (2023).
53. Center for Disease Control and Prevention. *Chronic Kidney Disease Basics*. https://www.cdc.gov/kidney-disease/about/?CDC_AAref_Val=https://www.cdc.gov/kidneydisease/basics.html (2022).
54. Yearby, R. Structural racism and health disparities: Reconfiguring the social determinants of health framework to include the root cause. *J. Law, Med. Ethics* **48**, 518–26 (2020).
55. Daneshpajouhnejad, P., Kopp, J., Winkler, C. & Rosenberg, A. The evolving story of apolipoprotein L1 nephropathy: the end of the beginning. *Nat. Rev. Nephrol.* **18**, 307–320 (2022).
56. Friedman, D. & Pollak, M. Nephropathy: From genetics to clinical applications. *Clin. J. Am. Soc. Nephrol.* **16**, 294–303 (2021).
57. Allen, A. L. & Rotenberg, M. *Privacy Law and Society* 3rd edn. Vol. 1643 (West Academic Publishing, 2016).

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Author contributions

E.F.H. conceptualized, investigated, analyzed, wrote and revised all portions on psychological essentialism, and served as project supervisor and administrator. D.K. conceptualized, investigated, analyzed, wrote and revised the section offering a causal fairness model for precision health, and created the figure included in the final manuscript. I.P. conceptualized, investigated, analyzed, wrote and revised the section offering a normative framework for the morally permissible use of race in precision health, and organized all sources for the final manuscript. All authors contributed to the visualization of the published manuscript. All authors reviewed, commented, and edited every iteration of the manuscript from pre- to post-publication. All authors read and approved the final manuscript.

Competing interests

The authors declare no competing interests.

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