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Solving the Racial Health Disparities Problem

Ian Shane Peebles[1] and Quayshawn Spencer[2]

[1]School of Historical, Philosophical, and Religious Studies, Arizona State University - Tempe Campus: Arizona State University, Tempe, USA and [2]Department of Philosophy, University of Pennsylvania, USA
Corresponding author: Ian Shane Peebles; Email: ipeebles@asu.edu

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Abstract

Contemporary work in bioethics presupposes a monistic view of race and a structuralist or polysemous view of racism. We aim to show that these received views are mistaken if the aim is to solve the racial health disparities problem. We argue that the theories that best enable us to solve the racial health disparities problem are a pluralist race theory and a virtue-theoretic account of racism because these theories are the best in terms of providing relevant accuracy and scope, causal explanatory power, nonsuperfluous ontological complexity, and practical utility for the task at hand.

1. Introduction

Consider the following empirical findings about human health:

(1) "Thus, in two large cohorts, Black patients had nearly three times the frequency of occult hypoxemia that was not detected by pulse oximetry as White patients" (Sjoding et al. 2020, 2478).
(2) "Our study estimated point prevalence of diabetes for AI/AN [American Indian/Alaska Native] adults aged 18 years and older as 14.6% in 2017, which is higher than for the general US adult population and other racial/ethnic groups" (Bullock et al. 2020, 3).
(3) "We tested whether genetic ancestry itself was associated with treatment outcome, after stratifying for treatment protocols, and found that the cumulative incidence of relapse was significantly associated with higher NA [Native American] ancestry, treated as a continuous variable" (Yang et al. 2011, 2).

Now suppose that a *racial health disparity (RHD)* at a specific time and for a current or past "race talk" in a specific linguistic community is a statistically and medically significant difference in health between one or more existing human races and another existing human race or races (Spencer 2019, 78). Given this definition, we can characterize (1)–(3) as RHDs according to a race talk engaged in among American English speakers. As we see it, *the RHD problem* that medical scholars, medical scientists,

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and health-care professionals have been wrestling with is *solved* at a time *t* when, and only when, all RHDs that ought to be eliminated at *t* are eliminated in a morally permissible way at *t*. There are good reasons to formulate the RHD problem in this way.

For one, we understand an RHD to be a health disparity between current or past races according to a linguistic community's history of race talk because otherwise, a linguistic community could say that it has eradicated an RHD by simply not calling the relevant groups "races" anymore. Second, we understand the RHD problem as focused on the RHDs that ought to be eliminated because *some* RHDs may be immoral to eliminate—for instance, if doing so would undermine an intraracial ethnic group's reasonable religious or other cultural practices. Third, we understand solving the RHD problem as involving doing so in a morally permissible way because it may be more immoral to eliminate an RHD in a particular fashion (e.g., through unwarranted paternalistic means) than to let it remain an RHD. Finally, when we say that a theory *best enables* us to solve the RHD problem, we mean that theory is best in terms of relevant accuracy and scope, causal explanatory power, nonsuperfluous ontological complexity, and practical utility. For example, all else equal, a theory of race that has built-in racial membership conditions—which are conditions for accurately assigning all people to each and every race they're a member of given the relevant theory of race—is better for addressing the RHD problem than one that doesn't because it can be *used* to develop reliable sampling strategies for races in medical research and to promote effective enforcement of relevant civil rights laws, such as the 1993 National Institutes of Health (NIH) Revitalization Act. Additionally, all else equal, a theory of racism that more accurately models the relevant causal structure is a better theory of racism for solving the RHD problem. Just like it's extremely valuable to understand the underlying causes of an infectious disease to develop a safe and effective treatment or prevention for the disease, it's extremely valuable to understand the underlying causes of an RHD's existence in order to eliminate that RHD in a morally permissible way.

Now that we've made these clarifications, it should be obvious that to solve the RHD problem, one needs suitable theories of *race* and *racism*. In this article, we argue that a *pluralist race theory (PRT)* and a *virtue-theoretic account of racism (VAR)* best enable us to solve the RHD problem. We defend our thesis with a conjunction argument. In section 2, we argue that a PRT best enables us to solve the RHD problem if there's no monist race theory that's better than some PRT in terms of relevant accuracy and scope, causal explanatory power, practical utility, and nonsuperfluous ontological complexity. And (to date) there is no monist race theory that's better than some PRT in terms of relevant accuracy and scope, causal explanatory power, practical utility, and nonsuperfluous ontological complexity. In section 3, we argue that the best candidate accounts of racism for solving the RHD problem are a VAR, a structuralist theory of racism, or a polysemous account of racism. And of these candidates, VAR best enables us to solve the RHD problem because it overcomes the limitations of its competitors without compromising the relevant epistemic standards. We close by considering how application of a PRT and a VAR might influence investigation in RHDs.

2. In defense of a pluralist race theory

We begin by clarifying what a PRT is. A PRT is a theory of race that states that race has more than one essence or more than one existence status. This contrasts with a *monist*

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theory of race, that is, a theory of race that posits a single essence and existence status for race. There are plenty of monist theories of race that scholars have adopted when addressing the RHD problem. For example, in Haslanger's (2012, 260–62) response to Outlaw's handling of the RHD problem, she defines *race* as a nonbiological division of people into what she calls *racialized groups*. To date, no author has defended a pluralist approach to addressing the RHD problem, although Hardimon has come close.[1]

Suppose, now, there's no monist race theory that's better than some PRT in terms of relevant accuracy and scope, causal explanatory power, practical applicability, and nonsuperfluous ontological complexity. In that case, because a race theory can either be pluralist or monist, and because a PRT's being better than any monist race theory in each aforementioned respect makes that PRT *best enabled* to help us solve the RHD problem, by definition, it follows that a PRT best enables us to solve the RHD problem. This premise wasn't difficult to defend because it was constructed to be analytically true. The more difficult lift will be to show that there's no monist race theory that's better than some PRT in terms of the relevant desiderata. There are different strategies one can take to defend this premise, but the strategy that we'll employ is to identify two RHDs that ought to be eliminated, then show that one of these RHDs would not be identifiable as an RHD without a PRT. In fact, we'll use two of the RHDs introduced at the beginning of this article to do this: (2) and (3).

For review, (2) states that American Indian or Alaska Natives (AIANs) have a higher prevalence of diabetes than non-AIANs and to a medically significant degree. As for (3), it states that a child's probability of relapse in two years after receiving the standard chemotherapy for acute lymphoblastic leukemia (ALL) continuously rises with that child's degree of Native American (NA) genomic ancestry, and it does so to a medically significant degree. In fact, the probability of ALL relapse rose twofold for every 100% increase in NA genomic ancestry (Yang et al. 2011, table S1). What we intend to show is that there is no monist theory of race that defines the race terms 'AIAN' and 'NA' in such a way as to pick out the correct referents for these terms, given how they're used in (2) and (3). There is, however, a way to do so with a PRT.

To start, note that Bullock et al. (2020, 1–2) sample health information from the AIAN population using data from the Indian Health Service (IHS), and they consider this sample to be "large" and "representative." They also note that the main eligibility condition for receiving IHS health services is "membership in a federally recognized tribe." In our review of the literature, the best monist race theory for defining 'AIAN' in this kind of situation is Ásta's (2018) conferralist theory of race. According to this theory, the essence of a race is a social status conferred onto its members by the relevant linguistic authority. Moreover, in *Categories We Live By*, Ásta considers a hypothetical scenario that leads her to define 'AIAN' as people with membership in a federally recognized tribe (Ásta 2018, 102). In addition to being relevantly accurate, Ásta's race theory is also practically useful because, unlike a lot of social constructionist race theories, her theory has built-in racial membership conditions for who is AIAN. They are whomever the federally recognized tribes count as tribal members. Interestingly, for some tribes, tribal membership doesn't require any

[1] We think that Hardimon has come close but hasn't quite done so because Hardimon (2017, 113, 137) doesn't think that socialrace is a type of race, and he thinks that populationist race is a type of minimalist race.

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"blood" connection to Native Americans. For example, all descendants of the Black people who were enslaved in the Cherokee Nation before 1866 are eligible for Cherokee citizenship as part of a reparations package that the tribe has granted.

However, despite how accurate and practically useful Ásta's race theory is for identifying the races referenced in some RHDs, its scope isn't wide enough to perform just as well for some other RHDs. For example, (3) is not an RHD that's about any conferralist races. The researchers who identified (3), Yang et al. (2011), had a hunch that the standard chemotherapy for childhood ALL wasn't working as effectively for NA children compared with all other children, where they understood *NA* to be an ancestry group. So, they sampled humans in a way to obtain a large enough group of NAs to have enough statistical power to detect a significant difference between NA and non-NA children when it came to relapse rate. In particular, the researchers sampled not only AIAN tribal members but also Mestizo Hispanics who, on average, had a significant amount of NA ancestry. Next, Yang et al. (2011, 9, fig. 2) sorted study participants into a "Native American" group (colored blue in fig. 2) using only *structure*-informed genomic ancestry components. If you had an NA chromosomal component, you were in the group. If not, you weren't. Using this classification scheme, the researchers observed a twofold increase in relapse rate among NA subjects for every 100% increase in NA genomic ancestry. So, it's plausible to say that the researchers identified Native Americans as people with "Native American genetic ancestries" (Yang et al. 2011, 5).

Given our reading of the relevant literature, Spencer's (2019) 1997 US Office of Management and Budget (OMB) race theory best defines 'NA' in this RHD. For clarity, Spencer's theory states that the racial division picked out by the OMB in 1997 is identical to a fivefold subdivision of our species into biological populations that Spencer (2019, 99) refers to as "the human continental populations." Spencer's race theory implies that Native Americans (so understood) are just people with "Native American genetic ancestries" (Yang et al. 2011, 5). Spencer's race theory strikes us as not only the most accurate race theory to use here but also a practical one to use as well because it has a built-in way to determine racial membership. Namely, one can use reliable genomic ancestry tests (e.g., 23andMe, AncestryDNA, etc.) to measure an individual's degree of genomic ancestry from a continental population, which will double as that individual's degree of racial membership. Note that competing biological race theories are either not as accurate as Spencer's for capturing the essence of NA in the relevant RHD, such as Glasgow's (2009) (H1*)-race theory, or they're not as practically useful due to lacking theory-driven racial membership conditions, such as Hardimon's (2017) minimalist race theory.

However, just like how Ásta's conferralist race theory cannot identify as a race all and only people with NA genomic ancestry, Spencer's OMB race theory is not able to identify as a race all and only people with federally recognized tribal membership. So, even the best monist race theories for these two particular RHDs do not have a wide enough scope to allow us to identify all RHDs that ought to be eliminated. The solution to this puzzle is simple. We should conjoin these and all other monist race theories that best handle certain RHDs to create a PRT that works *best* in identifying as RHDs all RHDs that should be eliminated. For instance, the best race theory for solving the RHD problem is the PRT that consists of, at the least, Ásta's conferralist race theory and Spencer's 1997 OMB race theory. This is the *minimum* ontological complexity that's

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going to yield the level of accuracy, wideness of scope, practical utility, and in fact, causal explanatory power needed to solve the RHD problem—assuming that the races themselves are relata in the causal relations whose structure is important to understand in order to eliminate RHDs in morally permissible ways.

While we trust that the reader won't disagree that (2) and (3) are RHDs that should be eliminated, there are other concerns that the reader may have at this point. One concern may be that our argument that a PRT should be used to solve the RHD problem suffers from a linguistic error. Namely, while it's okay to talk about Native Americans as an ancestry group, it's not correct to talk about the NA ancestry group as a race. For example, Yudell et al. (2016, 565) have said explicitly that races and ancestry groups are separate things. According to these authors, ancestry groups are "process-based," based on processes like "genetic heritage," whereas race is "pattern-based," based on "common hereditary traits (such as skin color)" (Yudell et al. 2016, 564–65). So, a Yudell et al.–like concern would boil down to the claim that health disparities like (3) aren't *racial* health disparities at all.

Our response to this concern is that the critic is simply incorrect about their claim that the NA ancestry group isn't a race and that races must be distinguished through patterns instead of processes. For one, the researchers who discovered (3) use the terms 'Native American' and 'American Indian/Alaska Native' interchangeably (Lim et al. 2014, 2). Second, these researchers also say that they are comfortable with definitions of 'race' that are "completely genetics-based (genetic ancestry)" (Lim et al. 2014, 2). Finally, the racial language that the researchers who discovered (3) are borrowing is ultimately from the OMB's 1997 revisions, which do not require races to be pattern-based. For instance, the 1997 revisions say that the Pacific Islander race consists of people "with origins in any of the original peoples of Hawaii, Guam, Samoa, or other Pacific Islands" (Spencer 2019, 79). There's no skin color, facial features, or hair type mentioned in that. So, the critics are assuming that racial discourse is tidier than it actually is. Having defended a PRT as the best theory of *race* for addressing the RHD problem, we now turn our attention to defending a VAR as the best theory of *racism* for addressing the RHD problem.

3. In defense of a virtue-theoretic account of racism

The most predominant theories of racism in contemporary bioethics are structuralist accounts of racism, with one example defining structural racism as "involv[ing] interconnected institutions ... refer[ring] to the totality of ways in which societies foster racial discrimination, through mutually reinforcing inequitable systems ... that in turn reinforce discriminatory beliefs, values, and distribution of resources" (Bailey et al. 2017, 1454).[2] While structural racism is a social determinant of health (Krieger 1999; Bailey et al. 2017; Williams et al. 2019), structuralist accounts of racism present several limitations. First, such accounts fail to adequately address that much of health care and medical research is interpersonal and that the structures being critiqued have individuals implicated in their persistence and functioning. Relatedly, when considering the proposed solutions for intervening into these racist structures,

[2] Note that the emphasis on institutions and systems coheres with the philosophical literature on structural racism, where structures are taken to be the primary unit of analysis in regard to racism (see Shelby 2014).

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it becomes readily apparent that to garner the critical mass of people necessary, or at least the people with the capital and power to amend or abolish the relevant structures, individual persons are a necessary target of reform in the mitigation and elimination of racism (Peebles 2024, 2519).

Additionally, structuralist accounts of racism are vulnerable to both Type I (false-positive) and Type II (false-negative) errors. Consider, for example, structuralist accounts that treat racial disparate impact as sufficient for identifying racism without knowing the cause of disparate impact (e.g., Williams and Mohammed 2013; Paradies et al. 2015; Jones 2018; Williams et al. 2019; Thomas et al. 2021; National Academies of Sciences, Engineering, and Medicine 2023). Disparate impact alone is insufficient for adjudicating instances of racism *because appealing to disparate impact alone fails to explain in virtue of what makes the outcome racist* (Matthew, 2022, 9–11; Peebles 2024, 2518). For example, racial disparities in inheriting a monogenic disease do not straightforwardly reflect racism, given that the inheritance of such diseases is not caused by social and environmental factors modulated by racism.[3] In contrast, racial disparities in multifactorial traits may or may not be influenced by racism, depending on the factors implicated in the trait(s) under consideration. This is important because the causal mechanisms implicated in a particular context will dictate the relevant interventions, and racism may not be the relevant causal mechanism in all RHDs. Thus, for disparate impact to be identified as racist, other explanatory features are required outside of disparate impact itself. Without said explanatory features, our analysis risks Type I errors.

Regarding Type II errors, structuralist accounts often treat contingent features associated with racism as if they are necessary. For example, while disparate impact can be a harbinger for racism, it is unnecessary for instantiating racism because *intending to generate racial disparate impact, but failing to do so, is sufficient for instantiating racism* (Peebles 2024, 2517–18). Additionally, treating features like certain economic structures (e.g., capitalism; Bright et al. 2022; Jabloner and Walker 2023) or racial superiority (e.g., white supremacy; Feagin and Bennefield 2014) as necessary features of racism will fail to capture the ways in which racism can (and does) persist outside such conditions. To be clear, we acknowledge that these features are commonly implicated in racism, and the relationships are worth investigating. The problem is that they aren't necessary for racism to manifest itself in medical practice and research. In sum, while it is important to track and intervene on the ways in which structural racism affects health (care) and medical research, structuralist accounts of racism are too narrow in scope because of their neglect and undertheorizing of individuals implicated in RHDs, and they risk classification errors that can obscure or confuse (social) scientific research meant to collect, process, and analyze empirical data on the relationship between racism and RHDs.

To better highlight the various levels of racism, some have suggested polysemous accounts of racism (e.g., Williams et al., 2019; Danis et al. 2016; Chadha et al. 2020). Polysemous accounts of racism understand the term 'racism' as having (very) different meanings across different contexts, and these different meanings are (or can

[3] Although social and environmental factors modulated by racism can influence the expression and severity of monogenic diseases, in which case, disparities in expression and severity may point to racism's influence.

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be) independent of each other (Hardimon 2019, 224; 2023, 369–70). In a polysemous account of racism, structural racism is (or can be) distinct from and independent of interpersonal racism, and vice versa. For example, Chadha et al. define structural and institutional racism in terms of systemic disparities, but internalized and interpersonal racism are predicated on attitudes (and behavior), and cultural racism has beliefs as its essential component (Chadha et al. 2020, 10). Thus, while described as interlocking levels of racism, the framework offered suggests that these various levels can be demarcated into three distinct and independent essences.

While polysemous accounts of racism correctly highlight the various expressions of racism, the problem is that these accounts risk unnecessary conceptual conflation between racism and other social ills (e.g., xenophobia and ethnocentrism). This is particularly troublesome given that the nature of a phenomenon often informs what treatment(s) to pursue. Consider, for example, viral versus bacterial infections. Each *kind of* infection is caused by a different *kind of* pathogen, and the pathogen dictates the most effective treatment (for bacterial infections, it is antibiotics, and for viral infections, it is antiviral medications). Similarly, if nonracist social ills are confused for racism, yet have distinct causal pathways, a polysemous account risks obfuscating the proper treatments for each *kind of* ill.

Additionally, polysemous accounts of racism compromise simplicity and coherence by lacking a unifying core (Peebles 2024, 2515). This, in turn, can obscure rather than clarify the etiology of RHDs and their persistence, making the connection between the causes opaque in the wake of several independent features. This, ultimately, undermines the explanatory power of polysemous accounts because the connection between the various forms of racism is often not clearly articulated. In contrast, bioethicists and health-care professionals have acknowledged the need to synthesize the data on racism (at all levels). For example, Williams et al. write, "Tying together interconnected data on health and racism will be critical for health disparities researchers moving forward" (2019, 117). Polysemous accounts of racism undermine this aspiration.

How, then, might we overcome these limitations? Consider, now, a VAR as presented by Peebles (2024), which can be summarized as follows:

> **Virtue-theoretic account of racism (VAR):** (i) Racism is fundamentally race-based ill-will or race-based deficient goodwill, where race-based ill-will is understood as willing individual (or a group of) persons ill based on their assigned or perceived race, and race-based deficient goodwill is understood as the failure to be disposed to the moral considerations relevant to the person(s) of interest because of their assigned or perceived race. (ii) Both fundamental forms of racism can manifest themselves interpersonally, institutionally, and systemically, with racism of one kind being able to transfer to another kind in a potentially bidirectional fashion.

Let us, then, return to the limitations previously mentioned and see how a VAR overcomes them. To begin, in a VAR, the limitations associated with structuralist accounts of racism are resolved. With race-based ill-will and race-based deficient goodwill being essentially constituted by cognitive and volitional states (Peebles 2024, 2510–14) and cognitive and volitional states being attributes of persons, a VAR

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immediately calls into account individual persons. Additionally, a VAR can accommodate a story for how certain economic structures, disparate impact, or racial superiority complexes might factor into the creation, maintenance, and perpetuation of racism without making these necessary features of racism. This is because all such features can be bolstered by race-based ill-will or race-based deficient goodwill, whereas not all race-based ill-will or race-based deficient goodwill is caused by these features.

Next, by providing a unifying core, a VAR does not risk the conceptual conflation invited through polysemous accounts of racism, and it preserves the sort of simplicity and coherence that allow for a unifying explanation between the various forms of racism. Again, this is important for research into the relationship between racism and RHDs. Under a VAR, several independent studies on racism, at various levels, can coalesce for more robust results. Thus, a VAR overcomes the limitations of its competitors while preserving the relevant accuracy and scope, causal explanatory power, nonsuperfluous ontological complexity, and practical utility that best enable solving the RHD problem.

Some might object to the practicality of a VAR, skeptical of whether it is possible to identify the volitional states or doxastic content influencing various RHDs. But it is unclear why circumstantial evidence wouldn't be sufficient for identifying and tracking the volitional states and doxastic content highlighted by a VAR. In fact, the US judicial system adjudicates many court cases with motives and intent in mind, *and it does so using circumstantial evidence*. While it is beyond the scope of this article to pursue this topic further, let us state that prima facie legal distinctions based on motives and intent are warranted and rightly demarcate the severity of certain violations. Presumably, a similar approach can be made to aid in distinguishing between the motivations and intent implicated in RHDs. Again, this is important because insofar as volitional states and doxastic content are implicated in RHDs, and insofar as modulating these states will be a necessary component to mitigating RHDs, we want a theory of racism that highlights these states.

Finally, some might object to our critical view of polysemous accounts of racism while promoting a PRT. Put simply, why is a PRT best suited for solving the RHD problem, but not a polysemous account of racism? In response, again consider the epistemic standards used to arrive at a PRT as our chosen race theory. These included accuracy and scope, causal explanatory power, nonsuperfluous ontological complexity, and practical utility. As demonstrated in section 2, the problem with monist theories of race is that although they are ontologically parsimonious, they lack the accuracy and scope, causal explanatory power, and practical utility to enable solving the RHD problem. What's more, despite being more ontologically complex than monist theories of race, a PRT (correctly constructed) will not produce *superfluous* ontological complexity. So, as it relates to the phenomenon of race in health (care), a PRT best adheres to the epistemic standards posited. In contrast, fulfillment of these epistemic standards in regard to racism does not require a polysemous account but can be fulfilled using a VAR, as demonstrated by VARs' ability to overcome the mentioned limitations. The result is that application of these epistemic standards provides the interesting result of promoting a pluralist theory of race and a monist theory of racism *in the context of addressing RHDs*.

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4. Conclusion

We have argued that a PRT and a VAR best enable us to solve the racial health disparities problem because both theories possess greater accuracy and scope, causal explanatory power, and practical utility than their competitors, and they avoid superfluous ontological complexity. We close by considering how application of a PRT and a VAR might influence investigation in RHDs. First, using these theories will influence how we understand and whether we participate in race-based medicine (see Peebles 2025). As demonstrated in section 2, not all race-based medicine warrants the charge of racism, and eschewing all race-based medicine may be counterproductive to eliminating RHDs. Second, using these theories will influence how we collect, process, and analyze empirical data on RHDs. Third, in regard to racism, a VAR suggests that altering certain policies and procedures is, while necessary, insufficient for mitigating RHDs. We also need to cultivate (or, perhaps, excise) certain character traits in health-care workers and the relevant stakeholders to promote the sort of goodwill toward all necessary for the practice of good medicine. Ignoring or rebranding RHDs in need of elimination will not make them go away. We hope that the conceptual tools offered in this article will allow the medical community to effectively and properly face them head-on.

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