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Received: 1 July 2022 | Revised: 10 July 2022 | Accepted: 11 July 2022

DOI: 10.1002/hec.4577

PERSPECTIVE

Health Economics WILEY

Healing the past, reimagining the present, investing in the future: What should be the role of race as a proxy covariate in health economics informed health care policy?

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Abstract

In this perspective, the assertion that race-free risk assessment would harm patients of all races is critiqued from the viewpoint that race is not just another covariate in our arsenal. Although race may be associated with outcome, it is nevertheless a proxy for a myriad of other potential explanatory variables that could be genetic/biological but in many circumstances are more likely to be sociological/socioeconomic. It is argued that the pursuit of health maximization through the use of socially constructed variables like race must be done sensitively, recognizing that racial covariates in the medical arena can be subject to structural, institutional or personal biases. Even when such biases are thought to be minimized, the appearance of such bias may be sufficient to justify the removal of its use, particularly where employing a racial covariate could further increase existing disparities. While racial covariates may have descriptive value in helping to understand such disparities, it is beholden on the scientific community to explore alternatives to racial covariates that may provide the same or perhaps even better prognostic value in our analyses.

KEYWORDS

health economic evaluation, race, racial health disparities, risk assessment algorithms

1 | DIFFUSING TENSIONS

In the wake of the global protests sparked by the murder of George Floyd in May 2020, the clinical community has begun a process of re-evaluating the use of race as an explanatory variable in clinical algorithms used to guide clinical decision making for individual patients. Two of the most influential commentaries have been from Vyas and colleagues (2020) writing in the *New England Journal of Medicine* and Cerdeña and colleagues (2020) writing in *The Lancet*. Vyas and colleagues identified a number of prominent clinical algorithms that include race and argued that it was time that the use of race was reconsidered since:

> …relationships between race and health reflect enmeshed social and biologic pathways. Epidemiologists continue to debate how to responsibly make causal inferences based on race. Given this complexity, it is insufficient to translate a data signal into a race adjustment without determining what race might represent in the particular context.

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Cerdeña and colleagues go further still, arguing that:

> Race-adjusted tools should be abandoned or replaced with more precise analytics than currently used.

In this issue of *Health Economics*, Manski (2022) argues that race is a legitimate covariate to be employed in clinical and economic decisions, concluding:

> Writers calling for race-free risk assessment have not studied how it would affect the quality of clinical decisions. Considering the matter from the patient-centered perspective of medical economics yields a disturbing conclusion: Race-free risk assessment would harm patients of all races.

In this perspective, each of Manski's points are examined in the face of the legitimate concerns over the use of race as a covariate for clinical and (by implication) health economic decision making. It is argued that, while it is difficult to refute the central contention that optimal decision making requires the use of all covariates that are associated with outcome, the assumption that racial covariates, and their application within the medical arena, are sufficiently free from bias (structural, institutional or personal) misses the point of the underlying argument: that race is not the same as every other covariate in our arsenal. It is a covariate that is acting as a proxy for a wide range of other explanatory variables that could be genetic/biological but in many circumstances are more likely to be sociological/socioeconomic.

In general, Manski's assertion, in his section on Optimal Clinical Decisions, that "…*maximum achievable well-being is larger if* P_{xz} *is used to assess risk than if* P_x *is used, provided that* P_{xz} *varies with z sufficiently to make optimal care vary with z"* where z signifies the covariate for race and x signifies all other covariates, is uncontentious. Further, previous work illustrates just such a framework for calculating probabilistic assessments of treatment benefit in the presence of covariates. The framework was illustrated with an example of an assertive community treatment (ACT) for homeless persons in Baltimore that employed race as an explanatory covariate—showing not only the prognostic association of the race variable with outcome, but also its predictive interaction with treatment (Hoch et al., 2002; Willan et al., 2004). Unfortunately, although the program appeared to be cost-effective overall, the use of race as a prognostic and predictive covariate suggested that the effectiveness (and therefore cost-effectiveness) was much better for Caucasians than for African Americans.

McRae and Onukwugha have argued the case still further, in a recent opinion piece (2021), proposing that such methods can and should be used to explore issues of intersectionality between race and gender in evaluating the social value of medicines. They outline a five-step approach to conduct socially conscious cost effectiveness analyses that involves the use of a regression framework to understand issues associated with race and intersectionality. Nevertheless, it is a big step from using such methods to understand the social disparities by race (or other factors such as gender) on disease and treatment outcomes, to arguing that welfare maximization requires us to act on those results. This is especially true in those case where to do so (as was the case for the ACT example) would clearly increase social disparities further.

2 | REIMAGINING FOUR ASSERTIONS

Each of Manski's objections to four assertions he describes from the proponents of face-free risk prediction tools are re-examined in light of the position that race is not just another covariate that is required for welfare maximization, but an imperfect proxy variable for a myriad of other effects.

2.1 | Is race a social or a biological construct?

Proponents of race-free risk assessment have argued that evidence that race is a social construct rather than a biological construct justifies its removal. Manski counters:

> …that inclusion [of race] among the covariates used to predict illness does not require that one take any position on the extent to which the classification expresses biological or social characteristics of patients. From the patient-centered perspective, inclusion of race is well-motivated whenever it adds predictive power to the accuracy of risk assessment achievable using other available data. Predictive power with available data has long been the criterion used to choose covariates in medical risk assessment.

Strict application of Manski's position means that, in the case of the ACT intervention for homeless persons in Baltimore, we should offer the intervention only to white homeless persons. In this example, it is not just race that is a social construct but the intervention itself. A much more appropriate response would be to use the descriptive results indicating the racial disparities in the intervention's effectiveness and cost-effectiveness to reassess the design of the intervention such that it becomes effective for all racial groups. This could mean that the intervention itself would be different for different racial groups, which would be consistent with Manski's vision of patient-centered medicine.

Of course the ACT program is an example of a social intervention rather than a clinical intervention, but there are reasons to be wary still of the social construct of racial differences even when dealing with a medical intervention. Social constructs and their association with disease are much more likely to be context dependent and spatially associated with place and time than biological associations. The biological associations of the Framingham risk equations, despite having been based on the small and largely white population of Framingham, Massachusetts in the 1980s, have proved to be generalizable (with calibration) across a diverse set of populations (D'Agostino et al., 2001). It is by no means clear that social associations will prove to be as generalizable—indeed intuitively these are much more likely to be context dependent.

2.2 | Causal linkages with disease

Manski argues, counter to the assertion by Vyas and colleagues that there should be evidence of causality between race and disease, that no such causal effect is required and that only statistical association is needed. There is no further elaboration of his position, which is unfortunate because it is a position that is difficult to accept. The importance of direct causation is in the suggested mechanism by which a potential intervention can act, whereas association does not necessarily imply that treatment will change the outcome. As a general comment, clinical risk assessment only makes sense if there are effective treatments to mitigate the estimated risk such that clinical risk-assessment can guide treatment choice. In this regard, it is important to understand whether treatment can modify the risk that is represented by a racial proxy covariate, as for other clinical/biological risk factors. For example, racial covariates are associated with socioeconomic deprivation. If socioeconomic deprivation is the cause of disparity then better understanding of the mechanism could suggest that social deprivation is the cause to be treated.

2.3 | Potential to perpetuate or amplify inequalities

The argument that including race has the potential to perpetuate or amplify inequalities is suggested by Manski to be "*unfounded if clinicians behave as purely patient-centered planners.*" He goes on to argue that the welfare maximizing approach embodies a notion of 'fairness' or 'justice', but in doing so glosses over the interpretation of inequity as inequality of outcome. As argued above, strict application of his position would mean that the ACT intervention would only be given to white homeless persons which would clearly perpetuate already apparent racial disparities in the rates of homeless persons, despite that fact that the intervention only seems effective (and cost-effective) for that group.

Manski acknowledges that pursuit of welfare-maximization objectives may be more efficient but less equitable. This is clear, but this is a broader problem for health systems and is not limited only to those systems that impose a cost-effectiveness requirement. Manski may well be correct in his assertion that even when society values a reduction of inequality over the pursuit of welfare maximization, removal of racial covariates may not be the best way to do this in the first best case where racial associations are not founded on structural or institutional racial biases and where physicians do indeed behave as perfect agents for their patients. However, this ignores the arguments, made most forcefully by Cerdeña and colleagues (2020), that racial profiling in medicine is subject to structural and institutional bias and that medical education received by clinicians is insufficient to avoid unconscious bias of racial stereotypes. In this second-best world, removal of racial risk-adjustment, especially where that adjustment perpetuates or amplifies existing inequality, may well represent the most practical solution to at least some of the problem. Certainly, this seems to be the view of the National Kidney Foundation (NKF)/American Society of Nephrologists' (ASN) Task Force on reassessing the inclusion of race in diagnosing kidney disease (Delgado et al., 2021). While acknowledging a modest level of misclassification in excluding race as a covariate they argue that the cost of this misclassification is minor, with the burden apportioned equally across racial groups, relative to the benefit of avoiding racial profiling in the diagnostic tool and recognizing the underdiagnosis of kidney disease in non-white Americans for a host of other reasons.

2.4 | Use of race as a covariate may offend

Manski acknowledges that people may fear that the use of race in clinical prediction tools may perpetuate a culture of structural and institutional racism. He describes such fear as understandable, but is concerned that such fears should not block legitimate attempts to make clinical risk-assessment tools as accurate and useful as possible. Such a fear is understandable in light of important evidence that health outcomes are impacted by the level of structural and institutional racism (Lukachko et al., 2014). The current debate is potentially helpful in seeking to understand how the accuracy of risk-assessment tools can be preserved without unnecessary use of proxy variables for race.

3 | INVESTING IN THE FUTURE

In looking to the future, Manski appears to accept that future risk-assessment re-evaluations may remove the use of race as a covariate, much as the NKF-ASN Task Force has recommended in the calculation of eGFR used to diagnose kidney disease. His conclusion? That in choosing to treat race as a different form of covariate, proponents of race-free risk-assessment should be challenged to show how the removal of race generates benefits in terms of reduced inequality that offsets the loss of efficiency.

Economists will likely be sympathetic to this proposed cost-benefit analysis. Nevertheless, there are reasons to be optimistic that such an analysis will be positive for two main reasons. First, the call to arms is not for the unthinking removal of the race covariate. As the opening quote from Cerdeña and colleagues suggests—the race covariate can and should be replaced by more accurate analytics. Once race is recognized as a proxy for other influences at play then it is beholden on the scientific community to strive to be more precise. As social scientists, economists should be even more sensitive to the potential inflammatory power of race as a social construct to do more harm than good (Smedley & Smedley, 2005). Second, Vyas and colleagues lay out an impressive agenda as to how the use of race as a covariate can be reconsidered. This agenda seeks to minimize the use of race as a proxy again focusing on underlying mechanisms while still allowing for its use where a good biological/genetic rationale exists and where it is clear that its use will narrow inequalities rather than perpetuate or exacerbate them. And there is clear evidence that such a strategy can be successful. In *'A troubled Calculus'* Madhusoodanan (2021) sympathetically highlights the re-examination of the clinical guideline to diagnose urinary tract infection (UTI) in children between the ages of 2 and 24 months. The original guideline included race as a covariate that reduced the risk that a child had a UTI among non-whites (Roberts et al., 2011). This guideline was recently retired by the American Association of Pediatrics after a re-examination found that the same level of accuracy could be achieved by replacing the race covariate with a prior history of UTIs and duration of the child's fever (Wright et al., 2022).

In terms of economic analysis, there is a continued role for the race covariate in health economic evaluations. Not in the blind pursuit of welfare maximization, but in the pursuit of better understanding of the insidious inequalities that persist in our society that are patterned by race. Welfare maximization remains a legitimate aim, but as social scientists a responsible approach to a sensitive covariate such as race is required. To that end, the initiatives of our clinical colleagues to better understand the determinants of health that the race covariate is merely a proxy for is to be welcomed and supported. Indeed, the hope is that health economists can also contribute to this important debate.

CONFLICT OF INTEREST

The author declares that there is no conflict of interest that could be perceived as prejudicing the impartiality of the research reported.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

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How to cite this article: Briggs, A. H. (2022). Healing the past, reimagining the present, investing in the future: What should be the role of race as a proxy covariate in health economics informed health care policy? *Health Economics*, 1–5. https://doi.org/10.1002/hec.4577