

# How to Reduce Racial Disparities?

## *Upon What to Intervene?*

Tyler J. VanderWeele<sup>a</sup> and Whitney R. Robinson<sup>b</sup>

Our article on the causal interpretation of race in regression analyses<sup>1</sup> was intended to clarify (1) how such analyses could be interpreted causally without conceptualizing hypothetical interventions to somehow alter race itself, and (2) how the causal interpretation of the race coefficient differed depending on whether socioeconomic status (SES) variables were controlled for early, or later, in life. We thank Glymour and Glymour<sup>2</sup> and Kaufman<sup>3</sup> for their thoughtful commentaries.

We agree with most of the points made by Glymour and Glymour<sup>2</sup> but feel that their commentary had as its primary target not our article, but certain comments made by Rubin,<sup>4</sup> which were also reflected in an influential article by Holland.<sup>5</sup> We did not claim that race and sex are not causes. We believe they could be understood as causes, as indicated in the “stronger interpretation” of race in our article. In fact, Glymour and Glymour’s discussion of race as a cause seemed to very much resemble our stronger interpretation. We also did not claim that causation is meaningful only when there is an intervention in mind, and we have in fact argued to the contrary.<sup>6,7</sup> Part of what we tried to accomplish in our article, however, was to provide a causal interpretation of the race coefficient in regression models that would be palatable to someone who was opposed to discussing causal effects for nonmanipulable variables. We believe that it is important to develop methodological approaches that can be used by researchers with differing philosophical positions, and that is what we attempted to do.

We also hoped that the methods proposed in our article<sup>1</sup> would contribute to generating hypotheses about the relative effectiveness of various potential interventions to reduce health disparities. We agree with Glymour and Glymour<sup>2</sup> that changing all aspects of SES is not a possible practical intervention—although, as noted by Kaufman,<sup>3</sup> this may be the question that at least some social epidemiologists are trying to answer (ie, what if we could disable the arrow from race to SES entirely?). This is in fact the question our methods would answer if it were possible to perfectly measure all aspects of SES, and if the other assumptions required for the analysis held perfectly as well—which, as indicated by Kaufman,<sup>2</sup> and by Glymour and Glymour,<sup>3</sup> will never be the case. Interventions that fundamentally alter legal and social structures may better correspond to “disabling the arrow from race to SES” and can sometimes dramatically reduce disparities,<sup>8,9</sup> although even in these cases it will generally be only the arrow from race to subsequent SES that is disabled (not the arrow to earlier SES). Historical legacies of racism and consequent unequal residential and economic opportunity may persist even if interpersonal racism suddenly stopped. This was also indicated in our diagrams and necessitated control for early

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From the <sup>a</sup>Departments of Epidemiology and Biostatistics, Harvard School of Public Health, Boston, MA; and <sup>b</sup>Department of Epidemiology, University of North Carolina at Chapel Hill, Chapel Hill, NC.

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Correspondence: Tyler J. VanderWeele, Departments of Epidemiology and Biostatistics, Harvard School of Public Health, 677 Huntington Avenue, Boston, MA 02115. E-mail: tvanderw@hsph.harvard.edu.

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individual and neighborhood SES so that the effects either were conditional on these values or concerned what would happen if early individual and neighborhood SES were equalized across racial groups.

We pointed out in our article that because it is, in any case, not possible to capture all aspects of SES, the interpretation of the effect estimates will always be with respect to potential interventions on the actual SES variables used in the analysis. We noted that this may, in fact, allow researchers to help assess whether interventions on certain SES variables are more likely than others to reduce racial disparities. This too can be challenging, as many SES variables are themselves likely to be correlated with one another.

Some of the recent discussion in social epidemiology has placed emphasis on finding practical interventions to reduce health disparities rather than continually merely documenting the disparities themselves.<sup>10–12</sup> This has proved to be challenging. Policy efforts in the United Kingdom to reduce disparities have proved relatively ineffective.<sup>13,14</sup> Part of the difficulty may have to do with the distinction between association and causation: the association of a particular SES variable with health does not mean that a change in that variable would ultimately alter health. As we<sup>1</sup> and our commentators<sup>2,3</sup> have indicated, for any of the effect estimates in our article to have the causal interpretation we gave them control for confounding needs to be sufficient for the associations between the SES variable and the outcome to reflect causal effects. Determining what associations are or are not causal is thus difficult. However, some of the challenge in reducing disparities is also likely related to the difficulty of finding the right place, time, and aspect of social and economic conditions upon which to intervene.

As a negative example, if the analyses in our article<sup>1</sup> are to be believed, they would indicate that even if we could intervene to equalize years of education comparing white and black persons, this would remove only 1% of the differences in BMI. Such an intervention would be relatively ineffective at altering BMI inequalities. However, other outcomes, such as income, may be more susceptible to such an intervention on years of education; and other types of potential interventions may be more effective still at changing outcomes. In recent analyses by Fryer,<sup>15</sup> (mirroring earlier analyses by Neal and Johnson)<sup>16</sup> the authors used regression models to control for a standardized test measure of educational achievement among black and white men. There was a 77% reduction in the racial differences in wages, a 75% reduction in racial differences in unemployment, and a 69% reduction in racial differences in incarceration rates --and all of the racial differences in self-reported physical health vanished. If the associations between educational achievement and these outcomes truly reflect the effects of education (rather than something correlated with it), then roughly three-quarters, or more, of the disparities in all of these outcomes could be eliminated if we

were able to equalize such educational achievement across black and white populations.

The analyses by Fryer,<sup>15</sup> unfortunately, did not control for any measures of family SES early in life (even though some data were available for this), and thus it is not clear whether early SES measures might confound the effects of later educational achievement. Control for confounding by other earlier SES factors would be needed to better understand what aspects of SES, if intervened upon, would be most effective at reducing disparities. Thus, the question remains: Is it really educational achievement that has an effect, or something correlated with it?

Even if the associations are causal, to what extent can we intervene to actually change such educational achievement? Recent evidence does seem to indicate that changes in schooling and education structure can substantially alter early educational achievement.<sup>17</sup> It remains to be seen whether such interventions can be used more broadly, and whether those changes in early educational achievement affect subsequent health, income, incarceration, and employment outcomes for the same study participants. If so, such early educational changes could reduce disparities—more effectively perhaps than other potential interventions. And, as noted before, the methods proposed in our article<sup>1</sup> may be helpful in comparing the effectiveness of interventions in reducing disparities. The results of Fryer's regression analyses are, if nothing else, certainly striking. If the results are even roughly indicative of the effects of early schooling achievement, social epidemiology may need to turn to education for cues.

Regression analyses with observational data cannot, of course, definitively determine causality. Still, when such analyses are carried out and interpreted carefully, they can provide clues. They can help generate hypotheses. They can help decide where to experiment first, what to try to intervene upon, and what strategy might be most effective. Of course, when we do intervene, it takes time to determine whether an intervention had the desired effect or whether our analyses had led us astray. The best we can hope for is that the methods and framework laid out in our article<sup>1</sup> might, in some small way, contribute to this task of deciding upon what to try to intervene.

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