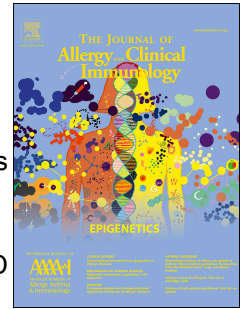


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Time's Up to Adopt a Biopsychosocial Model to Address Racial and Ethnic Disparities in Asthma

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In this issue of the Journal, Fitzpatrick et al(1) present results from an analysis of data from the National Heart, Lung, and Blood Institute's Severe Asthma Research Program (SARP) that aims to understand the contributions of socioeconomic and environmental factors to racial disparities in asthma outcomes. They followed a population of 631 children and adults with asthma, among whom approximately 1/3 were Black, and found that Black participants had a >2-fold greater odds of having an emergency department visit for asthma than White

participants, which is consistent with national data. The investigators then used propensity scores to match the White and Black participant populations on community and family socioeconomic variables and environmental exposure variables, and found that accounting for these factors eliminated the racial disparity in asthma-related ED visits. Other outcomes, including outpatient physician's visit for asthma and hospitalization did not differ by self-reported race. This study highlights an important issue related to racial disparities in asthma, which is that the greater burden of asthma morbidity among the US Black population may be explained, in large part, by socioeconomic and environmental inequities between Black and White populations rather than race-based biology or, more specifically, genetics. This finding has implications for the framing of research, public health, population health, and health care delivery strategies aimed at reducing (and ultimately, eliminating) this disparity.

Our health care and research agendas have a history of conflating race and biology, and emphasizing a predominantly biologic model of disease disparities. Race, a social construct, is important insofar that it is a conveniently measurable proxy for other important factors that influence health outcomes such as health literacy, environment, stress, and community factors. These social determinants of health, which are associated with, but downstream from, race, are most influential in health outcomes (Figure). Not surprisingly, grants for the study of health care disparities have been disproportionately awarded to the study of the influence of individual biological factors rather than community and societal factors(2). This overemphasis on race biology to explain health disparities has perpetuated the idea that disparate health outcomes are more a function of "individual failures" or genetic "defects" than of inherent systemic inequities.

Systems that perpetuate this predominant framing miss a critical opportunity to examine and intervene upon the social and environmental factors that shape the biology. Environmental exposures that are disproportionate among low-income and minority populations, such as pest allergens and pollution exposure(3, 4), and psychosocial factors associated with poverty, such as stress, are well understood to shape the immune phenotype(5) and contribute to symptoms and exacerbations(6). Without sufficient attention and resources paid to these socioeconomic and environmental factors, the findings from Fitzpatrick et al and work from others(7) suggest that we will not be able to shrink the disparity. In fact, we have made little progress in this regard(8) despite decades of focus on this issue and wide availability of highly effective medications.

Although this study highlights an important concept and helps to illuminate a path forward in the area of asthma disparities, there are some methodologic considerations that arise from the conceptual framing of the various factors involved and their hypothesized causal relationships. In particular, there is some ambiguity over what factors confound the relationship between race and ED visits and what factors may lie in the causal pathway between race and ED visits. Making a clear distinction between these two classes of factors is critical from a study design perspective but also implies different analytical approaches. The present analysis uses propensity scores to adjust for all factors as confounders, which implies that the socioeconomic and environmental factors confound any causal relationship between self-reported Black race and asthma ED visit risk. Briefly, propensity score matching is a statistical technique that can be used to make two populations that are being compared similar to each other with respect to known characteristics. For example, one population may have a higher proportion of men than

the comparison population, and propensity scores can be applied to weight the contributions of men and women in each population so that their contributions to each population are similar. However, it might be more plausible to hypothesize that such socioeconomic and environmental factors are in fact a result of self-reported Black race, and that it is these factors that actually lead to the increased risk of ED visit for asthma (Figure). In this conceptualization, the relationship between Black race and asthma-related ED visit is mediated by socioeconomic and environmental factors. While propensity score modeling can play a role in doing a mediation analysis, other approaches, such as principal stratification or counterfactual mediation models, which are specifically designed to allow for estimating the causal effects of intermediate factors, are more appropriate in such an analysis(9).

The logical conclusion of the current work, given the framing of the environmental and socioeconomic factors as confounders, is that there is no difference in ED visits by race once you adjust for other factors, and hence no racial disparity. Therefore, there is no problem to solve! A result of reframing the socioeconomic/environmental factors as mediators is that the observed racial disparity does exist, but operates through a variety of socioeconomic and environmental pathways. The question then is on what factor should we intervene in order to mitigate the disparity, or to break the causal pathway between race and ED visits. One genuine potential confounder is genetics, which could affect one's race as well as one's propensity for asthma ED visits if genetic markers of African ancestry also cause an increased risk of asthma exacerbations, or are linked to polymorphisms that cause asthma exacerbations (Figure). Therefore, genetic factors, if available, would need to be adjusted for, perhaps through propensity scores. Careful conceptual framing of the underlying causal pathways is therefore

essential to designing and analyzing studies focused on asthma among minority populations and asthma racial disparities so that the results are informative and impactful.

Looking to the future, how does this study inform the approach to research in this area so that it leads to advances in minority health and racial and ethnic disparities in asthma? A foundational step is to expand the framing of the issue of asthma disparities from one that is mostly biologic to one that explicitly considers environmental, psychosocial, economic and other factors(10), by applying a biopsychosocial rather than biologic model of disease. Adopting this model would help protect against an explicit – and implicit - focus on the biology of “race,” which distracts from the likely more important social determinants of health. To that end, research programs should have experts on disparities, race, ethnicity, socioeconomic, behavioral health, and environmental health involved in the planning, design, execution and interpretation of studies. Partnership with communities to build these interventions will also be key, as affected communities bring expertise that the scientific community does not, given their lived experiences. Consideration of factors that mediate the relationship between race and asthma outcomes should inform study design and data collection. As datasets are being developed for secondary analyses, linking data across sectors outside of health such as education, housing, and the built environment will be an important strategy for ensuring that environmental, psychosocial and economic factors can be examined. In addition, interpretation of study results should incorporate the roles that socioeconomic factors and environmental exposures may be playing; for example, these factors may explain observed associations between biology and asthma disparities, or biology and asthma outcomes among minority populations. Adopting a biopsychosocial model for asthma has implications for all stages of

research and across the translational spectrum and is no simple task, but given the findings from the Fitzpatrick study and others, and our failure to reduce racial disparities in asthma by emphasizing a biologic model, it is worth the effort.

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Figure: Conceptual representation of the causal pathway between race and ED visits using a biopsychosocial model. Black populations are at greater risk of poverty because of structural racism, which in turn is a cause of a host of social determinants of health such as environmental exposure, stress, etc., which in turn lead to an increased risk of ED visits for asthma.

