When Health Disparities Hit Home: Redlining Practices, Air Pollution, and Asthma

Asthma disproportionately burdens minority and low-income populations, and factors such as poor housing quality, the indoor environment, and ambient pollutants have consistently been linked to worse asthma outcomes (1). Recently, there has been increasing awareness of the role of institutional racial bias in creating and perpetuating respiratory health disparities (2). In this issue of the *Journal*, Schuyler and Wenzel (pp. 824–837) have focused their lens on the impact of one undeniable incarnation of structural racism: historical redlining (3). Redlining was a real estate practice of ranking non-White neighborhoods as credit unworthy and was designated as part of the federal housing policy from the 1930s to 1960s through the Home Owner's Loan Corporation (HOLC) and Federal Housing Authority. The consequences of redlining are complex and multifaceted. Redlining has not only limited the ability of people of color to build wealth through home ownership but has also been linked to overall substandard, poor-quality urban housing. Redlining designations have also contributed to other environmental disparities and specifically have been demonstrated to be associated with a variety of established environmental risk factors for asthma, including greenspace, tree canopy, and urban-heat exposure (4–7), as well as intraurban pollutant gradients for nitrogen dioxide (NO_2) and particulate matter (8). Thus, understanding the impact of redlining on asthma morbidity deserves particular consideration; however, to date, limited studies have investigated these relationships. A single ecological study conducted by Nardone and colleagues (9) explored HOLC risk grades based on location of population-weighted centroids on security maps and asthma exacerbation data obtained using census tract–level rates of emergency department visits across eight California cities. Historically redlined census tracts (i.e., risk grade D) were found to have a 39% higher rate of emergency department visits due to asthma than that of the lowest-risk census tracts in adjusted models, suggesting that this discriminatory practice might have contributed to current racial and ethnic asthma health disparities.

In the current study in this issue of the *Journal*, Schuyler and Wenzel (pp. 824–837) analyzed an existing, geocoded asthma registry, and examined exposures to multiple airborne air pollutants (carbon monoxide [CO], fine particulate matter [$PM_{2.5}$], sulfur dioxide [SO_2], and volatile organic compounds [VOCs]), as well as asthma health outcomes, across neighborhoods in Pittsburgh/Allegheny, Pennsylvania demarcated by a 1930s HOLC map-based assignment (grades A–D, in decreasing desirability for mortgage lending) (3). Their work represents an advancement from prior broader population-based studies as it synthesized individual-level geographic and environmental data with highly resolved measures of asthma severity, morbidity, and quality of care within the context of structurally imposed racial segregation. As expected, the proportion of Black families in each neighborhood was inversely correlated with superior HOLC grades, representing redlining against financing opportunities for Black applicants, with persistent race-based segregation extending to 2010. Notably, the authors found that HOLC D neighborhoods had the highest amounts of air pollutants (CO, $PM_{2.5}$, SO_2, and VOCs), with decreased $PM_{2.5}$ exposures occurring with increasing distance away from D neighborhoods. In addition, rates of uncontrolled and/or severe asthma were progressively higher when transitioning from A to D neighborhoods, with similar relationships observed for measures of asthma-related morbidity. It is interesting to note that despite increasing asthma prevalence and morbidity in D neighborhoods, there was no increase in asthma specialist care, and these neighborhoods had an even lower use of asthma biologic therapy, suggesting increased likelihood of suboptimal asthma management. Overall, these findings provide stark evidence for the downstream consequences of a geographically entrenched, race-based institutional practice on asthma health for Black populations manifesting decades after its inception. The results, although not surprising in themselves, provide substantive and direct evidence of respiratory health disparities derived from government-sanctioned racist legal practices, with clear implications for advocacy and opportunities for reform.

This work highlights the importance of considering upstream systemic and historical policy actions in health disparities research and further motivates the consideration of racism, over race, as a risk factor for health inequities and a target for intervention. Although race is recognized as one of the strongest predictors of environmental health disparities, recent federal efforts to address environmental injustice are expected to take a race-neutral approach to avoid being stymied in legal challenges (10). On the contrary, the investigation of racist systems, practices, and policies provides compelling evidence for systematic changes to undo these unjust structures. Indeed, systemic biases may be so embedded into society that the entire regional economy—not just local conditions—may contribute to a higher pollution burden in communities of color (11); thus, equitable solutions for pollution control will often require consideration of both local and regional sources. It is imperative that the research community continues to grow the scientific evidence base for social determinants of health disparities such as institutional racial bias to inform strategies for action. The study by Schuyler and Wenzel, therefore, is an important contribution to our understanding of the

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pervasive consequences of institutional racism and the wide-ranging systems and structures that have led to asthma disparities in the United States. Future work will need to build upon these findings and, in particular, develop novel measures of structural racism influencing environmental asthma risk among vulnerable populations. ■

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Use of Computed Tomography Lung Densitometry as an Outcome Measure for Emphysema Progression

The Case of Losartan

The current treatments available for chronic obstructive pulmonary disease (COPD) have shown to improve lung function, relieve symptoms, and reduce the risk of exacerbations, but there is an urgent need for therapies that can change the natural history of the disease (1). In the current issue of the *Journal*, Wise and colleagues (pp. 838–845) provide the results of a randomized, placebo-controlled trial aimed at demonstrating the efficacy of the angiotensin receptor blocker losartan in reducing emphysema progression (2). Credit must be given to the authors for conducting this trial without support from industry and overcoming the hurdles of the coronavirus disease (COVID-19) pandemic, but above all, for investigating a possible new mechanism to treat lung emphysema and prevent disease progression.

Investigating a new treatment is always associated with a high risk of negative results. In this trial, losartan administered to patients with COPD and emphysema did not demonstrate any radiological reduction in the rate of emphysema progression; in fact, subgroup analysis showed that former smokers treated with losartan had significantly more emphysema progression than current smokers on placebo, which is difficult to understand (2). On the other hand, some possible positive effects of losartan were a reduction in the risk of hospitalizations and improvement in the score of the PROMIS-20a

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