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Naming racism: the first step

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In this issue of *Blood*, Abraham et al demonstrate the striking contribution of neighborhood to survival disparities between non-Hispanic White and non-Hispanic Black patients with acute myeloid leukemia (AML).[1] They show that structural racism defined by using measures of concentrated neighborhood disadvantage and racial segregation mediates almost all of the difference in leukemia survival. By using a rich multi-institutional retrospective cohort of 822 adult patients with AML from 6 centers in the Chicago area, the authors evaluate the role of structural racism in the context of more traditional predictors of leukemia outcomes, including leukemia biology and treatment received.

Numerous studies have documented racial disparities in outcomes of patients with AML[2]; however, racism is rarely named as a potential mechanism. Increasing attention is being paid to racism within our hematology community[3] as well as within our society at large. Racism as a concept is often defined at 3 levels: structural, interpersonal, and internalized.[4] Although the most blatant manifestations of racism are interpersonal, in which there is a clear actor perpetrating the racist behavior, the influence of structural racism is far-reaching, and its pervasive nature can make it difficult to recognize. Structural racism refers to the way in which our society fosters interrelated and mutually reinforcing discriminatory systems, institutions, and laws that confer preferential access to societal goods, services, opportunities, rights, and power by race.[5] As a result of centuries of slavery, segregation, and discrimination, racism is deeply woven into the fabric of American society and culture.

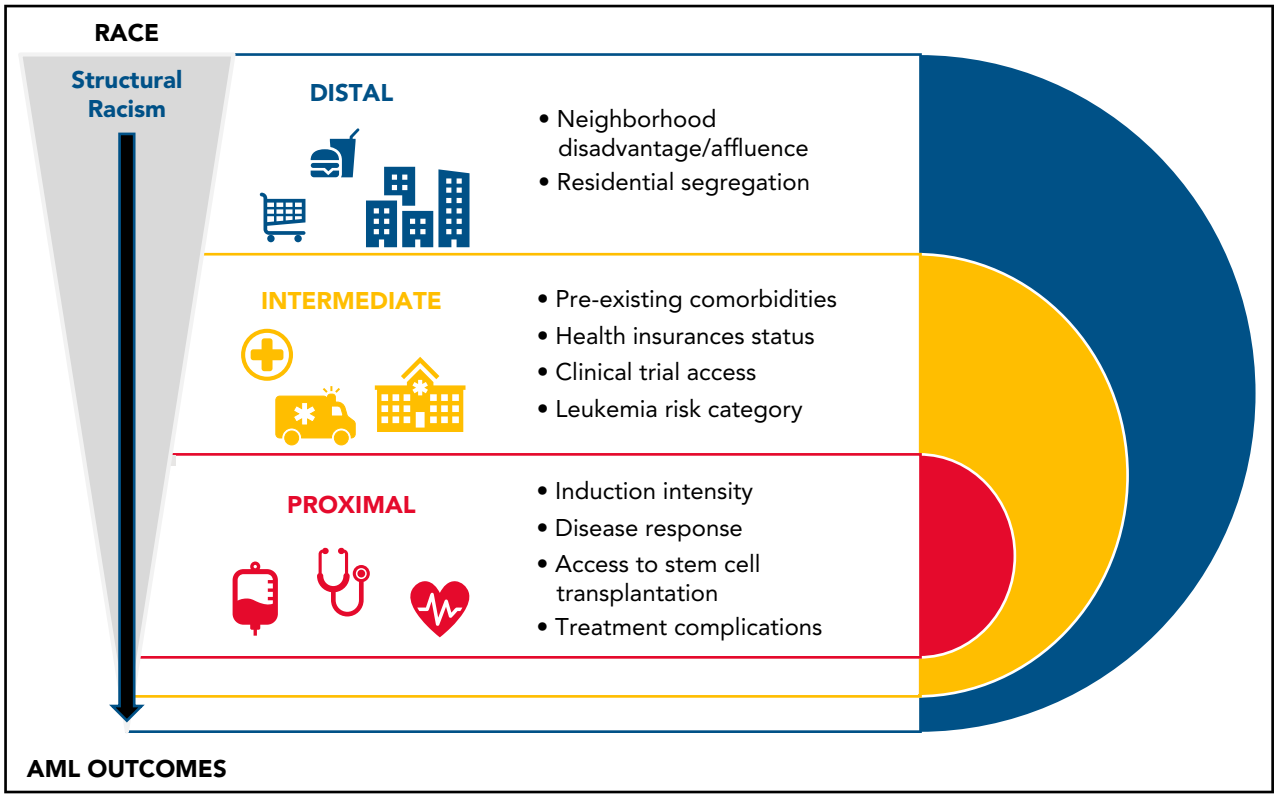
The authors use the Chicago metropolitan area, one of most diverse and at the same time most segregated cities in the United States, as a case study of the effect of structural racism on leukemia survival. Almost half of Chicago's Black residents live in majority-Black neighborhoods, and these neighborhoods bear the legacy of race-based discrimination. Starting in 1916, Black Americans left the oppression of the Jim Crow South in a movement known as The Great Migration. Moving to industrial cities like Chicago held the prospect of better opportunities and higher paying jobs. However, racism was codified in the discriminatory practice of redlining, in which banks denied federally backed loans to Black Americans, limiting their ability to purchase homes. This resulted in starkly racialized residential segregation that is self-perpetuating to this day.[6] Systematic disinvestment in majority-Black communities continues to limit economic opportunity for the residents of these majority-Black neighborhoods. It reduces the likelihood that neighborhood residents will go to college and renders them significantly more likely to be unemployed than people who live in majority-White neighborhoods.

This historical context provides the basis for the association between socioeconomic status (SES) and race, which is itself a sociopolitical construct. The persistence of the association between SES and race is the product of contemporary structural injustices that continue to reinforce historical wrongs.[4] As researchers, we should be mindful not to invoke SES as the explanation for racial disparities without acknowledging that the source of that SES disparity is also rooted in structural racism.

These societal factors are complex, are inextricably linked, and interact with one another. As a result, structural racism is difficult to measure and quantify. Abraham et al empirically define structural racism using census tract–level measures of residential segregation (defined to include tertiles of the proportion of non-Hispanic Black and non-Hispanic White residents), concentrated disadvantage (defined to include the proportion of families with incomes that are below the poverty line, proportion of families receiving public assistance, proportion of unemployed adults, and proportion of female-headed households with children), and concentrated affluence (defined to include the proportion of families with incomes greater than $75 000, proportion of college-educated adults, and proportion employed in professional or managerial occupations). This is not the first use of these measures; the Index of Concentration at the Extremes (ICE) was introduced more than 20 years ago to measure social polarization and has since been used to evaluate economic and racial/ethnic segregation as a joint construct that represents structural racism.[7] The act of identifying the root of segregation and naming structural racism is a critical first step in the effort to recognize and address the source of disparities in hematology.

The application of methods for causal inference, in this case mediation, adds to the novelty and rigor of the study. Aiming to quantify the relative contributions of comorbidities, access to health care, leukemia biology, treatment, admission to the intensive care unit, and structural racism (see figure), the authors define distal, intermediate, and proximal mediators that may be part of the causal pathway between race and leukemia death. Applying this level of methodologic rigor is essential for moving the study of disparities toward a deeper understanding of underlying mechanisms.

The naming of racism has opened the door for asking and answering the question of how racism is operating in a given place or population, thus opening myriad avenues for future research. The findings of this study need to be validated in a broader setting and confirmed using additional measures of structural racism. Another important step will involve interrogating how cumulative lifetime exposure to racism may influence a patient's transcriptional and epigenetic pathways and disease manifestations. Universal screening for social determinants of health, of which individual experiences of



Potential mediators along the causal pathway between structural racism (blue) and AML survival include intermediate factors (yellow) that impact access to care and leukemia risk stratification and more proximal factors (red) such as treatment approaches and complications. Structural racism (as defined by Abraham et al) leverages the contrasts between membership in a neighborhood characterized by concentrated affluence (high income, college-educated, employed in professional/managerial occupations) or concentrated disadvantage (low income, requiring public assistance, high unemployment, and female-headed households) and majority-White or majority-Black census tracts. Illustration by Justine M. Kahn.

racial discrimination are one, will allow for the rigorous study of the "exposome" and inform development of interventions to address the impact of social determinants of health on hematology outcomes.

Prioritizing efforts to address race-based inequities in the United States is daunting and requires conscious appreciation of the impact of racism on health, changing cultural norms, and both community-based and institutional-level efforts. Ultimately, because the root causes of inequities are often structural, many of the solutions will likely also need to be structural. As a first step, however, we can shift the discourse toward continuing to name structural racism as a foundational root cause of hematology health disparities, as Abraham and colleagues have.

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