Under the Skin: Using Theories From Biology and the Social Sciences to Explore the Mechanisms Behind the Black—White Health Gap

Equity and social wellbeing considerations make Black-White health disparities an area of important concern. Although previous research suggests that discrimination- and poverty-related stressors play a role in African American health outcomes, the mechanisms are unclear. Allostatic load is a concept that can be employed to demonstrate how environmental stressors, including psychosocial ones, may lead to a cumulative physiological

We discuss both the usefulness of this framework for understanding how discrimination can lead to worse health among African Americans, and the challenges for conceptualizing biological risk with existing data and methods. We also contrast allostatic load with theories of historical trauma such as posttraumatic slavery syndrome. Finally, we offer our suggestions for future interdisciplinary research on health disparities. (Am J Public Health. 2010;100:S36-S40. doi:10. 2105/AJPH.2009.171140)

toll on the body.

Tiffany L. Green, PhD, William A. Darity Jr, PhD

THE BLACK-WHITE HEALTH

gap is a long-standing problem of great concern to researchers and policymakers. Evidence from the social sciences and public health has suggested that discriminationand poverty-related stressors can affect health outcomes among African Americans and other socially dispossessed groups.^{1,2} However, the discrimination-based literature has not elucidated the precise mechanisms by which these stressors lead to worse health outcomes. To overcome these shortcomings, a field of research has emerged that integrates perspectives from the social and biological sciences.

Allostatic load is a concept that can be used to demonstrate how environmental stressors, including psychosocial stressors, can lead to a cumulative physiological toll on the body.³ We discuss the usefulness of this framework for understanding how discrimination can lead to worse health in African Americans, and we discuss the challenges for conceptualizing biological risk with existing data and methods.

We also contrast the allostatic load framework with theories of historical trauma. These theories, such as posttraumatic slavery syndrome, purport to explain worse health and life outcomes among African Americans through the lens of cultural shortcomings caused by past injustices. Finally, we offer our suggestions for future research endeavors that incorporate perspectives from both the biological and social sciences.

PSYCHOSOCIAL STRESSORS AND HEALTH

Much of the current science focuses on how contemporary psychosocial stressors from poverty and discrimination can predispose individuals to suffer poorer health. Research on nonhuman primate models has provided intriguing evidence of a strong relationship between social hierarchy and health outcomes. For example, 1 study of female cynomolgus monkeys found that low social status was associated with ovarian dysfunction and exacerbated coronary atherosclerosis or heart disease. Socially dominant males did in fact develop heart disease, but they did so only under socially stressful conditions.⁴ Although these relationships are complex, and certainly do not hold over all primates (or even "cultures" within the same species), there seems to be consistent evidence that when subordinate ranking is associated with harassment and a lack of social support, poor health tends to result.5

We find further evidence of the link between socioeconomic status (human rank) and health in humans. The famous Whitehall Studies demonstrated a clear socioeconomic status gradient in the British civil service for deaths from coronary heart disease.⁶ Poor socioeconomic conditions in childhood contributed substantially toward explaining health disparities in nuns who shared otherwise similar environments for many years.⁷ Racial discrimination and unfair treatment also have been linked to cardiovascular reactivity in African Americans and have been labeled chronic stressors that may affect cardiovascular health negatively.⁸ Discrimination compounded by poverty also has been shown to be associated with worse health outcomes. Chronic discrimination from multiple sources also has been indicated as a risk factor for early coronary calcification⁹ and for higher preterm birth rates among Black women.¹⁰

Still, there are serious challenges in quantifying the role of discrimination-related stress in the social sciences, including recall bias (particularly over longer periods of time), underestimation of the degree of unfair treatment, and construct validity.^{11,12} In short, there is a fundamental difference between an individual's perception of discrimination and the potential physiological impact of unfair treatment.¹³ Although there is ample research on the former, there is little on the latter.

ALLOSTATIC LOAD

The concept of allostatic load attempts to bridge the gaps between the physiological, biological, and social sciences. Both allostatic load and related concepts such as inflammation and metabolic syndrome facilitate the exploration of mechanisms whereby different environmental challenges and stressors, broadly defined, may get "under the skin." Allostatic load is a way to capture the cumulative wear and tear

COMMENTARIES

on the body that results from repeated exposures to stressful experiences, whether physical or psychosocial.¹³

For example, fluctuations in blood pressure aid us in sleeping, waking, and other physical activities in the short term. However, repeated surges of blood pressure (e.g., potentially from racial discrimination) can lead to physical damage to blood vessels and, ultimately, atherosclerosis.14 The original allostatic load index was developed with data from the MacArthur Study of Successful Aging¹⁵ and consisted of 10 biological parameters that are markers of physiological activity across the various bodily systems (cardiovascular system, metabolic system, hypothalamic-pituitaryadrenal axis, and sympathetic nervous system). It contained the following 10 physiological markers: systolic blood pressure, diastolic blood pressure, waist-tohip ratio, ratio of total to highdensity lipoprotein cholesterol, high-density lipoprotein cholesterol, glycosylated hemoglobin, cortisol, norepinephrine, epinephrine, and dehydroepiandrosterone sulfate. (See Crimmins and Seeman³ for a more extensive explanation of the development of the original allostatic load index.) The index was a summary measure, consisting of a count of the number of biological risk factors for which each individual scored in the upper quartile of the sample distribution. This index has been shown be strongly correlated with mortality risk as well as a decline in physical and cognitive functioning among the elderly.3

Although allostatic load is often used in aging research, it can be used to compare differences between groups on the basis of social hierarchies involving class and

race. One study of children aged as young as 6 years found that children with low socioeconomic status presented significantly higher levels of cortisol compared with their higher socioeconomic status counterparts.16 Blacks had higher allostatic load scores (signaling early health deterioration) compared with Whites, even after control for impoverished conditions.¹⁷ This race effect appears to have a gendered component as well. Chyu and Upchurch reported results from their study showing that the Black women in their sample had higher allostatic load scores compared with White women.¹⁸ Black women also gained the least benefit from education in lowering allostatic load scores.18

Researchers also have found links between race, neighborhoods, and cumulative biological risk profiles. Merkin et al., using a national sample of US adults, found that neighborhood socioeconomic status bears an inverse relationship with allostatic load. These results were strongest and most consistently significant in the African American subsample.¹⁹ Although these differential findings by race are intriguing, it is important to note that other researchers have found that any experience of unfair treatment is associated with worse outcomes. For example, in a recent study that used an all-White subset of Midlife in the United States (MIDUS) data, respondents demonstrated that greater lifetime exposure to major discrimination and chronic exposure to daily discrimination predicted higher levels of e-selectin (a marker of inflammation) in men but not in women.²⁰

There are many challenges related to the conceptualization of allostatic load in research. First, there are issues of measurement. Many empirical tests, particularly those subsequent to the original MacArthur study,¹⁵ have been limited by available survey data typically not designed to answer questions about the role of allostatic load in disease outcomes. Furthermore, many of these studies are based on cross-sectional data, making it difficult to reach any conclusions about the role of allostatic load in dynamic health processes.^{17,19} Also, it is unclear whether the measures used, such as high blood pressure, succeed in capturing the underlying biological processes, or are outcomes associated with physiological breakdown.²¹ Finally, although the simple count allostatic load index has been shown to predict health outcomes,³ aggregation across multiple measures of system dysregulation (e.g., cardiovascular and metabolic) still may cause researchers to overlook whether 1 or 2 systems are the most important drivers of disease outcomes and potentially important interactive effects.

POSTTRAUMATIC SLAVERY SYNDROME

In contrast to contemporary demographic and biological research, an emerging strand of literature calls for increased attention to be paid to the effects of historical group trauma on health outcomes in traditionally marginalized groups.²² In the case of African Americans, the most wellknown variant is called posttraumatic slavery syndrome²³ or posttraumatic slavery disorder hypothesis.²⁴ The proponents of posttraumatic slavery syndrome and its variants posit that the experience of the Middle Passage and American slavery produced a collective trauma that has been transmitted across generations.

Collective dysfunction consequent upon the race-related traumatic events also is thought to explain the propensity of African Americans to engage in self-defeating behaviors that contribute to adverse health outcomes.

However, there are major difficulties with applying this approach to African American health deficits. The approach does not clarify how daily indignities and discrimination²⁵ affect psychological well-being above and beyond the memory of slavery. It is difficult to sort between the effects of an immediate, unexpected trauma (e.g., an assault) and experiences of slavery and discrimination that have lasted for many generations.²⁶ Moreover, how does one determine, at least in a quantitative sense, the most relevant tragic historical event or events; was it the Middle Passage, slavery itself, or the White terror campaign in the post-Reconstruction era conducted by groups such as the Ku Klux Klan and the Red Shirts, or contemporary daily traumas associated with the deaths like that of unarmed Guinean immigrant Amadou Diallo?27

Second, groups that have been subjected to collective trauma are not all relatively low-performing with respect to health or other indicators. European Jews after the Holocaust and Japanese Americans after mass incarceration during World War II indicate that a shared history of groupbased trauma does not inevitably lead to poor group outcomes on social and economic achievement indicators. Indeed, one can readily explain poor outcomes for a number of racial/ethnic groups by pointing to contemporary discriminatory barriers and inequalities without appealing to

a syndrome driven by a past injustice.

Inevitably, postttraumatic slavery syndrome and other historical trauma theories, however wellintentioned, make problematic assumptions about marginalized groups.²⁸ Alleged dysfunctional traits might include oppositional attitudes toward education (e.g., Fordham and Ogbu's "acting White" hypothesis²⁹) and higher rates of consumerism.²³ However often these traits are refuted in systematic research, ^{30,31} these ideas have become common wisdom echoed by influential public figures.³² A more useful approach would be to examine the actual biological pathways that explain why less-educated individuals have worse health and how grossly uneven intergenerational transfers of wealth may be far more salient in affecting a group's profile of wellbeing than the alleged transmission of psychological trauma across several generations.31,33

Finally, a major flaw of historical theories is their lack of predictive power and the near impossibility of deriving independent parameter estimates of the pure "trauma" effect. To understand the causal effect of slavery on health, the researcher must at least establish a quasi-experimental framework that demonstrates differences in health and behavior pre- and posttrauma.34 For African Americans, there is no clear period that marks the termination of trauma when we consider what we know about the continued influence of racism on socioeconomic outcomes in the United States. Thus, any remembrance of historical trauma necessarily will be correlated with current experiences of discrimination and economic hardship-both factors that are associated with inferior health outcomes.

FUTURE RESEARCH

We briefly offer some suggestions for future interdisciplinary research in population health that integrates biological and social determinants. First, we urge a continued emphasis on the collection of comprehensive longitudinal data. This will enable researchers to study individuals and social groups over the life course, as well as pinpoint critical periods where health disparities begin to emerge or worsen. Most notably, increasing evidence suggests that adult diseases can be traced back to developmental and biological disruptions during the prenatal and early childhood periods.35

In addition, it is vital to collect data on biological markers over time to be able to understand the connections between racism, other forms of stress, and health. Although cross-sectional studies can be useful starting points, they can be misleading in the context of interpreting biomarker data. For example, hormones such as cortisol have natural cycles that fluctuate over the course of the day and vary from weekdays to weekends.13,36 Thus, in order for studies to yield meaningful findings, researchers must collect both baseline (resting) measures and measures over time to evaluate whether any significant differences between groups are present. Using these improved data, investigators will be able to develop and test more precise measures of allostatic load at the population level. One promising example of a long-term, longitudinal study that incorporates these elements is the upcoming National Children's Study.37

Last, we emphasize the potential for allostatic load research to continue to move the field of population health away from race as a fundamental determinant of health. Geneticists repeatedly have shown that there is not enough genetic variation among human groups to constitute biologically valid subspecies or "races." 38,39 It is also spurious to assume that phenotypical traits correspond to "race" differences in propensity for diseases.⁴⁰ In spite of this, many researchers in biomedicine and the social sciences, clinicians, and others continue to treat race as an immutable scientific category. For example, Graves and Rose discussed the position of physicians Alastair Wood and Sally Satel,⁴¹ both of whom advocated in print for racial profiling in medical research and practice. This mode of thought also is promulgated in the popular media. Distressingly, whereas the "success" of racespecific drug therapy on cardiovascular disease rates among Blacks was widely reported,⁴² the lack of consistency across trials as well as the type I error that drove the original result³⁹ was not reported.

Moreover, there is extensive historical, sociological, and anthropological research that demonstrates clearly how the meaning of race in the United States has shifted over time. Before 1930, when all Blacks were collapsed into 1 category on the Census, mixed-race Blacks were counted as a separate category ostensibly for "scientific purposes." Census data also reveal the attempt to categorize Whites as well, where immigrant groups such as Italians, Hebrews, and Greeks were initially considered separate (and lesser) races from the White, Anglo-Saxon founders of the United States. Again, post-1930, these different "White" races became consolidated into 1 group. However,

there were still exclusionary barriers in employment and housing imposed against some White ethnic groups such as Jews.⁴³

In short, we must, as Cooper and Kaufman stressed, soundly reject race as a legitimate measure of intrinsic risk in etiological research.⁴⁴ As Michael Omi noted,

> the idea of 'race' and its persistence as a social category is only given meaning in a social order structured by forms of inequality—economic, political, and cultural—that are organized, to a significant degree, along racial lines.^{45(p254)}

Employing interdisciplinary perspectives from the biological and social sciences will allow researchers to continue to deconstruct the black box of race. We can gain insight into how the disproportionate life stressors that African Americans tend to experience⁴⁶⁻⁴⁸ physiologically translate into worse health over time. We also can use this work to understand why some individuals have better health outcomes than others in the same marginalized group, even when faced with the same stressors. Also, allostatic load research may help to explain some of the relatively superior health outcomes initially experienced by Afro-Caribbean immigrants compared with nativeborn Blacks,49 and why their health outcomes deteriorate over time.⁵⁰

A major task in population health research is to examine the ways in which underlying social hierarchies produce an unjust distribution of health and other life outcomes. This entails the rejection of spurious theories involving the assumption of inherent racial or cultural shortcomings in favor of insights based upon meaningful and systematic research. Work involving allostatic load may inspire researchers to understand



how economic and emotional deprivation may help prompt physiological breakdown. At the same time, researchers in the biological sciences must marry their laboratory findings with work from social scientists that examines the role that institutions play in creating conditions that lead to poor health. Thus, the analysis of the problem of health disparities is fundamentally an interdisciplinary endeavor that will require creative ways of thinking about how social conditions get "under the skin."

About the Authors

Tiffany L. Green is with the Department of Population Health Sciences, School of Medicine and Public Health, University of Wisconsin–Madison. William A. Darity Jr is with the Sanford School of Public Policy at Duke University, Durham, NC.

Correspondence should be sent to Tiffany L. Green, PhD, 610 Walnut St, 707 WARF, Madison, WI 53726-2397 (e-mail: tlgreen2@wisc.edu). Reprints can be ordered at http://www.ajph.org by clicking the "Reprints/Eprints" link.

This commentary was accepted September 28, 2009.

Contributors

T.L. Green developed the article framework and served as the principal writer. W.A. Darity Jr also developed the article framework, performed extensive edits, and contributed to the main text.

Acknowledgments

The authors thank the Robert Wood Johnson Health and Society Scholars program for its financial support during the writing of this article. The project is also supported by the National Institute of Child Health and Human Development (award T32HD049302).

The authors also thank the anonymous reviewers for their helpful editorial comments.

Note. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institute of Child Health and Human Development or the National Institutes of Health.

References

1. Borrell LN, Kiefe CI, Williams DR, Diez-Roux AV, Gordon-Larsen P. Self-reported health, perceived racial discrimination, and skin color in African Americans in the CARDIA study. *Soc Sci Med.* 2006;63(6):1415–1427.

2. Williams DR. Race, socioeconomic status, and health. The added effects of racism and discrimination. *Ann N Y Acad Sci.* 1999;896:173–188.

3. Crimmins E, Seeman T. Integrating biology into the study of health disparities. *Popul Dev Rev.* 2004;30: 89–107.

4. Kaplan JR, Adams MR, Anthony MS, Morgan TM, Manuck SB, Clarkson TB. Dominant social status and contraceptive hormone treatment inhibit atherogenesis in premenopausal monkeys. *Arterioscler Thromb Vasc Biol.* 1995;15(12):2094– 2100.

 Sapolsky R. Why Zebras Don't Get Ulcers: The Acclaimed Guide to Stress, Stress-Related Diseases, and Coping. 3rd ed. New York, NY: Holt Paperbacks; 2004.

6. Marmot MG, Smith GD, Stansfeld S, et al. Health inequalities among British civil servants: the Whitehall II study. *Lancet.* 1991;337(8754):1387–1393.

 Snowdon DA, Ostwald SK, Kane RL. Education, survival, and independence in elderly Catholic sisters, 1936– 1988. *Am J Epidemiol.* 1989;130(5): 999–1012.

 Guyll M, Matthews KA, Bromberger JT. Discrimination and unfair treatment: relationship to cardiovascular reactivity among African American and European American women. *Health Psychol.* 2001; 20(5):315–325.

9. Lewis TT, Everson-Rose SA, Powell LH, et al. Chronic exposure to everyday discrimination and coronary artery calcification in African-American Women: The SWAN Heart Study. *Psychosom Med.* 2006;68(3):362–368.

 Dole N, Savitz DA, Hertz-Picciotto I, Siega-Riz AM, McMahon MJ, Buekens P. Maternal stress and preterm birth. *Am J Epidemiol.* 2003;157(1):14–24.

11. Williams DR, Mohammed SA. Discrimination and racial disparities in health: evidence and needed research. *J Behav Med.* 2009;32(1):20–47.

12. Coleman MG, Darity WA, Sharpe RV. Are reports of discrimination valid? Considering the moral hazard effect. *Am J Econ Sociol.* 2008;67(2):149–175.

 McEwen BS, Wingfield JC. The concept of allostasis in biology and biomedicine. *Horm Behav.* 2003;43(1):2– 15.

14. McEwen BS, Seeman T. Protective and damaging effects of mediators of stress. Elaborating and testing the concepts of allostasis and allostatic load. Ann N Y Acad Sci. 1999;896:30-47.

15. Seeman TE, Singer BH, Rowe JW, Horwitz RI, McEwen BS. Price of adaptation-allostatic load and its health conseqences. MacArthur Studies on Successful Aging. *Arch Intern Med.* 1997;159(11): 1176.

16. Lupien SJ, King S, Meaney MJ, McEwen BS. Child's stress hormone levels correlate with mother's socioeconomic status and depressive state. *Biol Psychiatry*. 2000;48(10):976–980.

17. Geronimus AT, Hicken M, Keene D, Bound J. "Weathering" and age patterns of allostatic load scores among Blacks and Whites in the United States. *Am J Public Health.* 2006;96(5):826– 833.

 Chyu L, Upchurch D. Racial and ethnic profiles of allostatic load among adult women in the US: findings from the National Health & Nutrition Examination Survey 1999–2004. Abstract presented at: Annual Meeting of the Population Association of America; April 17–19, 2008; New Orleans, LA.

19. Merkin SS, Basurto-Davila R, Karlamangla A, et al. Neighborhoods and cumulative biological risk profiles by race/ethnicity in a national sample of U.S. adults: NHANES III. *Ann Epidemiol.* 2009;19(3):194–201.

20. Friedman EM, Williams DR, Singer BH, Ryff CD. Chronic discrimination predicts higher circulating levels of E-selectin in a national sample: the MIDUS study. *Brain Behav Immun.* 2009;23(5): 684–692.

21. Goldman N, Turra CM, Glei DA, Lin YH, Weinstein M. Physiological dysregulation and changes in health in an older population. *Exp Gerontol.* 2006;41(9): 862–870.

22. Stam R. PTSD and stress sensitisation: a tale of brain and body Part 2: animal models. *Neurosci Biobehav Rev.* 2007;31(4):558–584.

23. Leary JD. Post Traumatic Slave Syndrome: America's Legacy of Enduring Injury and Healing. Milwaukie, OR: Uptone Press; 2005.

24. Reid O, Mims S, Higginbottom L. Post Traumatic Slavery Disorder: Definition, Diagnosis, and Treatment. Charlotte, NC: Conquering Books; 2005.

25. Brown DJ. Everyday life for black American adults: stress, emotions, and blood pressure. *West J Nurs Res.* 2004; 26(5):499–514.

26. Cross WE. Black psychological functioning and the legacy of slavery: myths and realities. In: Danieli Y, ed. *International Handbook of Multigenerational Legacies of Trauma*. New York, NY: Plenum Press; 1998.

27. Jane F. The Diallo verdict: the overview: 4 officers in Diallo shooting are acquitted of all charges. *New York Times*. February 26, 2000:1A.

28. Brave Heart MY. The historical trauma response among natives and its relationship with substance abuse: a Lakota illustration. *J Psychoactive Drugs*. 2003;35(1):7–13.

29. Fordham S, Ogbu JU. Black students' school success: coping with the "burden of acting white." *Urban Rev.* 1986;18:176–206.

30. Tyson K, Castellino DR, Darity W. It's not "a black thing": understanding the burden of acting white and other dilemmas of high achievement. *Am Sociol Rev.* 2005;70(4):582–605.

 Gordon Nembhard J, Chiteji N. Wealth Accumulation and Communities of Color in the United States: Current Issues. Ann Arbor, MI: The University of Michigan Press; 2006.

32. Obama B. Democratic National Convention Keynote Address. Presented at Democratic National Convention; Boston, MA; July 27, 2004. Available at: http://www.2004dnc. com/barackobamaspeech. Accessed on October 5, 2009.

 Pollack CE, Chideya S, Cubbin C, Williams B, Dekker M, Braveman P.
Should health studies measure wealth? A systematic review. *Am J Prev Med.* 2007; 33(3):250–264.

34. Shadish WR, Cook TD, Campbell DT. *Experimental and Quasi-Experimental Designs for Generalized Causal Inference*. Boston, MA: Houghton Mifflin; 2002.

35. Shonkoff JP, Boyce WT, McEwen BS. Neuroscience, molecular biology, and the childhood roots of health disparities: building a new framework for health promotion and disease prevention. *JAMA*. 2009;301(21):2252–2259.

36. Kunz-Ebrecht SR, Kirschbaum C, Marmot M, Steptoe A. Differences in cortisol awakening response on work days and weekends in women and men from the Whitehall II cohort. *Psychoneuroendocrinology*. 2004;29(4):516– 528.

37. Landrigan PJ, Trasande L, Thorpe LE, et al. The National Children's Study: a 21-year prospective study of 100,000 American children. *Pediatrics*. 2006; 118(5):2173–2186.

 Long JC, Kittles RA. Human genetic diversity and the nonexistence of biological races. *Hum Biol.* 2003;75(4):449– 471.

39. Cooper RS, Kaufman JS, Ward R. Race and genomics. *N Engl J Med.* 2003;348(12):1166–1170.



40. Graves JL. *The Emperor's New Clothes: Biological Theories of Race at the Millennium*. New Brunswick, NJ: Rutgers University Press; 2001.

41. Graves JL, Rose RM. Against racial medicine. *Patterns Prejudice*. 2006;40(4–5):481–493.

42. Wade N. Race-based medicine continued. *New York Times*. November 14, 2004;sect 4:12.

43. Oppenheimer GM. Paradigm lost: race, ethnicity, and the search for a new population taxonomy. *Am J*

Public Health. 2001;91(7):1049-1055.

44. Cooper RS, Kaufman JS. Race and hypertension: science and nescience. *Hypertension*. 1998;32(5):813–816.

 Omi M. The changing meaning of race. In: Smelser NJ, Wilson WJ, Mitchell F, eds. America Becoming: Racial Trends and Their Consequences. Washington, DC: National Academy Press; 2001:243– 263.

46. Darity WA Jr. Employment discrimination, segregation, and health.

Am J Public Health. 2003;93(2):226–231.

47. Ross SL, Yinger J. The Color of Credit: Mortgage Discrimination, Research Methodology, and Fair-Lending Enforcement. Cambridge, MA: MIT Press; 2002.

48. Weinick RM, Krauss NA. Racial/ ethnic differences in children's access to care. *Am J Public Health.* 2000;90(11): 1771–1774.

49. Acevedo-Garcia D, Soobader MJ, Berkman LF. The differential effect of foreign-born status on low birth weight by race/ethnicity and education. *Pediatrics*. 2005;115(1):e20–e30.

50. Williams DR, Haile R, Gonzalez HM, Neighbors H, Baser R, Jackson JS. The mental health of Black Caribbean immigrants: results from the National Survey of American Life. *Am J Public Health*. 2007;97(1):52–59.

Community-Based Participatory Research Contributions to Intervention Research: The Intersection of Science and Practice to Improve Health Equity

Community-based participatory research (CBPR) has emerged in the last decades as a transformative research paradigm that bridges the gap between science and practice through community engagement and social action to increase health equity.

CBPR expands the potential for the translational sciences to develop, implement, and disseminate effective interventions across diverse communities through strategies to redress power imbalances; facilitate mutual benefit among community and academic partners; and promote reciprocal knowledge translation, incorporating community theories into the research.

We identify the barriers and challenges within the intervention and implementation sciences, discuss how CBPR can address these challenges, provide an illustrative research example, and discuss next steps to advancethetranslational science of CBPR. (*Am J Public Health.* 2010;100:S40–S46. doi:10.2105/AJPH.2009. 184036) Nina Wallerstein, DrPH, and Bonnie Duran, DrPH

ALTHOUGH MUCH EVIDENCE

exists of health and social disparities within populations of color and other marginalized groups, the real challenge lies ahead-to develop, implement, and sustain effective strategies to eliminate disparities in clinical and public health systems and population health status. Community-based participatory research (CBPR) represents a transformative research opportunity to unite the growing interest of health professionals, academics, and communities in giving underserved communities a genuine voice in research, and therefore to increase the likelihood of an intervention's success.¹ In this article, we add to the literature on intervention and implementation sciences by identifying barriers and challenges to building bridges between science and community-based practice and policy. We illustrate ways to address these challenges through an example of successful CBPR work done among American Indians in the Southwest, and

through presenting CBPR as an overall translational strategy for diverse communities to improve health equity.

Several definitions of CBPR circulate widely. In their 1995 study of participatory research in Canada, Green et al. defined CBPR as an "inquiry with the participation of those affected by an issue for the purpose of education and action for effecting change."² In the definition offered by the Agency for Healthcare Research and Quality in 2004, CBPR is an approach that incorporates formalized structures to ensure community participation.3 Focusing on disparities, the Kellogg Foundation Community Health Scholars Program states that CBPR

equitably involves all partners ... with a research topic of importance to the community with the aim of combining knowledge and action for social change to improve community health and eliminate health disparities.^{1(p6)}

These definitions set the stage for CBPR to be able to address core

challenges in intervention research.

CHALLENGES WITHIN TRANSLATIONAL INTERVENTION RESEARCH

The widening socioeconomic and racial/ethnic health disparities documented in the past 20 years,^{4,5} the chasm in the quality of health care delivery, and the extended time it takes for research findings to translate into practice⁶ have created a national urgency to design effective interventions, including an increased emphasis by the National Institutes of Health (NIH) on public health significance and impact. This context for the translational intervention sciences has produced an important new area of investigation that is now emerging as its own disciplineimplementation science⁷⁻⁹-with a new Implementation Science journal, conferences, and calls by the NIH for proposals. According to the NIH, "Implementation [research] is the use of strategies to adopt and