

Krieger N. The science and epidemiology of racism & health in the United States: an ecosocial perspective. Invited presentation: the 10th Annual William T. Small Keynote Lecture for the 29th Annual Minority Health Conference, University of North Carolina School of Public Health, Chapel Hill, NC, February 29, 2008.

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Website: www.minority.unc.edu/sph/minconf/

ABSTRACT

Racism harms health. It also creates the very categories of “race.” Racial/ethnic health inequities are a biological expression of racism; their origins lie in injustice, not biology. This is not an ideological argument: it is a scientific statement that rests on rigorous tests of scientific hypotheses about how racial/ethnic inequities in health status and health care arise from unfair and unjust societal conditions. As delineated by ecosocial theory, at issue are the myriad ways racial inequality becomes biologically embodied, over the lifecourse and across generations, thereby creating racial/ethnic health inequities. Relevant pathways include adverse exposure to: economic and social deprivation; toxic substances, pathogens, and hazardous conditions; social trauma; targeted marketing of harmful commodities; and inadequate and degrading medical care. In this presentation I will discuss conceptual and methodological issues involved in analyzing how racial and economic injustice produce health inequities, coupled with empirical examples drawn from research I have done, as a social epidemiologist, on these issues.

Presentation: 45 minutes

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Social epidemiology and ecosocial theory

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"EXPERIENCES OF DISCRIMINATION" (EOD) MEASURE (FREE ACCESS) AT: http://www.hsph.harvard.edu/faculty/NancyKrieger.html
WEBSITE FOR "UNNATURAL CAUSES": http://www.unnaturalcauses.org/

SLIDE 1: INTRODUCTION

Greetings. It is an honor to be here and to give the 10th Annual William T. Small Keynote Lecture for the University of North Carolina School of Public Health's 29th Annual Minority Health Conference. For many years now, this Conference has been playing a critical role in keeping visible, nationally, a critical perspective on health inequities. We need, after all, a critical mass to make a difference – and it is clear, from the history of this conference, its origins in the collective work and organizing done by students and Dr. William T. Small, and the themes addressed over these many years – that a central objective of this conference has been precisely this: to build a critical mass – critical not only in numbers, but also in the sharpness of our thinking. And so I thank the conference organizers, and Dr. Small, for bringing us together today, so that we can listen, learn, engage, get energized, and make the critical connections – in thinking and with each other – so sorely needed to advance the work for social justice and public health.

In this spirit, today I will be speaking on: **The science and epidemiology of racism & health in the United States: an ecosocial perspective.** Noting that science, racism, and health can be linked in more than just one way.

SLIDE 2: WHY SCIENCE? WHY RACISM?

As illustrated rather recently, for example, by Dr. James Watson, who this past October did quite the job in being Exhibit A for scientific racism, claiming that blacks are inherently less intelligent than whites.

The good news is that Watson was widely excoriated by leading scientific institutions and scientists for what he said. The criticisms impaled him for speaking scientific garbage and for attempting to use his scientific laurels as a substitute for scientific evidence. In no short order, Watson was suspended from the Cold Spring Harbor labs, where he served as Chancellor, and he resigned in ignominy a few days later. As Henry Kelley, the president of the Federation of American Scientists said, it was one “sad and revolting way to end a remarkable career.”

The bad news is that the views Watson expressed are all too common, deeply ingrained, and legitimized by centuries of scientific racism. Hence, as part of the work required to debunk this heavy legacy, we need to counter with the science of investigating the health consequences of racism.

SLIDE 3: BEAN COUNTING, GENE COUNTING, & HEALTH INEQUITIES: COMPETING CLAIMS

What are some of the testable claims of this alternative science? The first claim is that the longstanding equation that “race” = “genetics” is wrong. This allegedly scientific view, articulated so baldly by Watson, holds that “races” are “natural” populations, meaning “genetically distinct.” In this approach, biology and ancestry becomes racialized and reduced to a question of genes-R-us (that is, gene counting), and these genetic differences are presumed to explain why racial/ethnic groups can be differentiated and counted (that is, bean counting) and why it is they have different health status. Strong evidence shows, however, that not only can a given phenotypic trait, e.g., skin color, be compatible with many different genotypes (for other traits), but also that genetic variation within groups demarcated by conventional “racial” categories is far greater than between them. The alternative is to posit that race/ethnicity, like social class, is a historically contingent social category with biological consequences.

A second claim, from this alternative stance, is that what matters for generating and explaining health inequities is gene expression, not gene frequency. Consider, for example, a study I recently conducted involving monozygotic women twins who lived together until at least age 14, and hence tightly matched on genotype and also early life conditions (including in utero!). Tellingly, among twins who had divergent class trajectories, the working class twin fared worse than her professional counterpart on many physiological as well as self-report health measures. These findings highlight that context matters – and the implications for comparing the health status of groups with vastly different childhood as well as adult life circumstances should be glaringly evident.

A third claim, turning racist notions of racial categories on their head, is that there can be meaningful social categories of race/ethnicity that can be used thoughtfully to document, monitor and analyze the impact of racial injustice on people’s lives, including their health. What this statement recognizes is a profound change in the use of racial/ethnic data: from an initial purpose of discriminating adversely to instead providing evidence of discrimination that must be countered. When categories of race/ethnicity are no longer linked to inequity, we will see it in the data. Until then, we cannot afford to be blind to the realities and impact of racism.

And here I remind you that the current reason why we have data on age and race/ethnicity in the 100% population count in the US census is the Voting Rights Act (only just recently renewed) combined with the 1990 Census Redistricting Act. Whereby the collection of racial/ethnic data for the voting age population is mandated to create fair voting districts and prevent racial gerrymandering that would dilute the vote of racial/ethnic minorities. While there still are problems, at least there are data to document the problems and contest illegitimate boundaries in court.

In other words: we cannot escape history – or pretend that it hasn’t happened. The reasons for using “race” data extend far beyond health – and we must understand these reasons to do our health analyses.

SLIDE 4: PROPOSITION 54

Making clear these are not simply academic points is the example of California’s recently defeated Proposition 54, which sought to prohibit the state government from collecting or using any racial/ethnic data, including census data, allegedly on the grounds that because “race” isn’t “real,” biologically, data on “race” should not be collected. Funded by conservative groups, the actual intent of Prop 54 was to end statistical documentation of discrimination, following a time-honored, or perhaps I should say time-disgraced, practice of getting rid of problems by removing them from view, otherwise known as “no data, no problem.” To counter, opponents argued that the social realities of “race” and ongoing racism require

collecting the data to monitor racial/ethnic inequalities in health and other outcomes – and, indeed, it was the public health arguments which proved decisive in helping defeat the measure.

SLIDE 5: DEFINING RACIAL DISCRIMINATION AND RACE: SOME KEY CONSTRUCTS

So let me now turn to the question of analyzing the impact of racism on health. One useful place to start is to clarify a few concepts, as shown on this slide.

Briefly stated, **racism** refers to institutional and individual practices that create and reinforce oppressive systems of *race relations* and their contingent *racial definitions*, whereby dominant groups define themselves and others through the possession of arbitrary physical characteristics, such as skin color, and adversely restrict, by judgment and action, the lives of those against whom they discriminate, and benefit from so doing. Health consequences can thus be conceptualized as **biologic expressions of race relations**, referring to how harmful physical, biological, and social exposures, plus people's responses to these exposures, are ultimately embodied and manifested in racial/ethnic disparities in somatic and mental health. On this slide I list broad categories of these inequitable exposures, involving: economic and social deprivation; toxic substances, pathogens, and hazardous conditions; social trauma; targeted marketing of harmful commodities; and inadequate and degrading medical care.

The recognition that **race/ethnicity** is a *social, not* biological construct, in turn helps clarify that the arbitrary phenotypic characteristics invoked as so-called “markers” of “race” are more accurately understood as **racialized expressions of biology**. The fact that we know what “race” we are – and that racial/ethnic disparities in health exist -- says more about our society than it does our biology.

SLIDE 6: ECOSOCIAL THEORY: CONTEXT MATTERS

Or, to put this another way, using the visual imagery of ecosocial theory, a theory of disease distribution concerned with health inequities that I have been developing since 1994, what we need to understand are

the myriad ways racial inequality becomes biologically embodied, over the lifecourse and across generations, thereby creating racial/ethnic health disparities. In making this statement, I draw on the 4 core constructs of ecosocial theory, which are:

- (1) **“embodiment,”** referring to how we literally embody, biologically, our lived experience, thereby creating population patterns of health & disease;
- (2) **“pathways of embodiment,”** referring to how there are often multiple pathways to a given outcome, via diverse physical, chemical, biological, & social exposures, and involving gene expression, not just gene frequency;
- (3) **“cumulative interplay of both biological and social exposure, susceptibility, and resistance across the lifecourse,”** since all of these matter; and
- (4) **“accountability and agency,”** both for social inequalities in health & for ways they are – or are not – monitored, analyzed & addressed.

In other words, as emphasized by this theory, we must pay heed to context, to lifecourse, to historical generation, and to levels of analysis, as well as to the interrelationships between diverse forms of social inequality, including racism, class, gender, among others – as well as to people’s relationship to the rest of the ecosystem.

SLIDE 7: DIRECT AND INDIRECT APPROACHES TO STUDYING DISCRIMINATION AND HEALTH

How, then, to study the impact of racism on health? I would argue we need two kinds of studies: **direct** and **indirect**, each of which can employ quantitative or qualitative methods. First, by **direct**, I mean health studies explicitly obtaining information on people’s self-reported experiences of – and observing people’s physiological and psychological responses to – real-life or experimental situations involving racial discrimination, as both an acute and also, perhaps especially, chronic exposure. The caveat, as well recognized in the enormous body of literature on “stress” and health, is that such research must reckon with not only exposures but perceptions of these exposures, as well as cognitive issues pertaining to

memory and disclosure. Second, by **indirect**, I mean studies that investigate racial/ethnic disparities in distributions of deleterious exposures and/or health outcomes and explicitly infer that racism underlies these disparities – even as they do not per se document discriminatory acts or people's interpretation of these acts. Taken singly, each approach has its flaws, yet both are necessary, as each addresses questions the other cannot.

Specifically, only the direct approach can be used to study the health consequences of social trauma; there is no substitute. By contrast, for most of the other pathways, the indirect approach is required precisely because they involve distributions of exposures that go beyond an individual's perception. For example, knowledge of discrimination in wages, or in occupational hazards, or in medical care, can be obtained only if one has information on what others experience.

SLIDE 8: RACISM & ECONOMIC RESOURCES

Let me start, then, with two examples of the indirect approach, regarding links between racial/ethnic inequality, socioeconomic deprivation, and health.

This slide, drawing on data from my *Public Health Disparities Geocoding Project*, shows the impact of adjusting for census tract poverty on black/white and Hispanic/white age-adjusted health disparities for an assortment of the outcomes, using data from my home state of Massachusetts, centered around the US 1990 census. The first point to note is that the magnitude of racial/ethnic disparities in health varied by race/ethnicity, by outcome, and by gender. African Americans, for example, generally were at excess risk compared to white Americans across the board, with relative risks ranging from around 2 (e.g., premature mortality) to upwards of 20 (gonorrhea and tuberculosis). By contrast, for Hispanics, the picture was more variable, with their often being at higher risk than whites for the childhood and infectious disease outcomes, but at lower risk for premature mortality (possibly a reflection of immigrants returning to their

home countries to die). And in both groups, the racial/ethnic disparity for deaths due to HIV/AIDS was far greater for women compared to men.

Nevertheless, the second major point is that even with this heterogeneity of risk, what you can see is that adjusting for census tract poverty in virtually every case, as shown by the cells highlighted in yellow, reduced the observed racial/ethnic disparities. In some cases, the reduction was quite dramatic, as shown by the outcomes with red font, e.g., nearly a halving of the excess risk, if not more, for childhood lead poisoning, gonorrhea, tuberculosis, and for mortality due to HIV/AIDS and to homicide and legal intervention. If these are the results we obtained with just this one admittedly crude measure, only imagine what we might have found had we been able to use more comprehensive measures of socioeconomic position, across domains and across the lifecourse!

And the third major point is that our results are complex, which is likewise the message of the extant literature on the impact of adjusting for or stratifying by socioeconomic position when analyzing racial/ethnic disparities in health. In other words, there is no one-size-fits-all scenario. That said, it is safe to say that more often than not, racial/ethnic socioeconomic inequities do play a major role in racial/ethnic health inequities, both in the onset of the event and once disease is diagnosed.

SLIDE 9: US POVERTY RATES (2006)

And here I remind you that in the US differences in poverty rates by race/ethnicity loom large. Consider these data from the recently released US Current Population Survey. In 2006, 12.3% of the US population, including 20.7% of children under age 5 – that is, 1 in every 5 children – live under the notoriously stingy US poverty line. This is a very high level of poverty, and especially childhood poverty, compared to other industrialized nations. Only then consider the racial/ethnic inequities in poverty, which translate to fully 4 in 10 black children, 3 in 10 Hispanic children, versus less than 1 in 10 white children are growing up impoverished. At a time when ever more research shows the profoundly devastating

effects of childhood poverty on not only children but also on their later health status as adults, it is obvious that research on health inequities must reckon with huge socioeconomic disparities between US racial/ethnic groups.

SLIDE 10: THE FALL AND RISE OF INEQUITIES IN US PREMATURE MORTALITY: 1960-2002.

Consider as well the case of trends in US health inequities. Here I note that in the current literature, articles can be found arguing that racial/ethnic and socioeconomic health disparities are in effect inevitable, because the more educated and wealthier will always be most able to take advantage of the latest health knowledge and medical innovations. But is this necessarily the case? Or might the magnitude of these health inequities be historically contingent, and reflect in part societal priorities – and hence be amenable to social change?

To address this question, we decided to examine, empirically, long-term trends in socioeconomic inequities in US premature mortality and infant death rates, overall and by race/ethnicity, noting that research on this topic has been hampered by the absence of socioeconomic data in US death and birth certificates until 1989 and 1968, respectively – and also because the public access version of the US Compressed Mortality Files only goes back to 1968. Arguably, however, from a policy and public health perspective, the period directly preceding 1968 is crucial. Critical changes included the 1964 US Civil Rights Act, the 1965 establishment of Medicare and Medicaid and consequent desegregation of US medical facilities, the accompanying expansion of community health centers and maternal and child health programs, and the many other federal policies comprising the “War on Poverty.”

We accordingly sought out data to analyze rates of premature mortality and infant death among US counties, ranked by income level, for the period 1960-2002: for both the total population and also stratified by what W.E.B. Du Bois in 1904 famously termed the US “colorline,” which divides the

racially dominant US white population and US populations of color. We decided this dichotomy – between white and of color – was the best way to handle the limitations of the available racial/ethnic mortality data that extended back to 1960, which used only the categories of “white,” “black,” and “other.” We chose age 65 as the cut-point for premature mortality since this age determines eligibility for Social Security and Medicare and also because an average life expectancy of at least 65 years was consistently attained by US black men only in 1995 – compared to 1944 for the total US and white population and 1973 for the black population overall.

SLIDE 11: METHODS

We extracted the 1968-2002 mortality data from the public-use US Compressed Mortality Files, then also obtained US county 1960-1967 mortality data, which additionally required manually locating and identifying the correct county code for each of the 3073 counties. Denominators consisted of US Census decennial counts and intercensal estimates.

As for the socioeconomic data, because of the loss of the computerized 1960 census “100% detail” file (and it really is astounding that the 1960 census data could have been lost!), our economic measure was county median family income, chosen because we were able to locate this for the 1960 as well as the 1970-2000 census. We assigned counties to quintiles of median family income weighted by county population size and then calculated, for each calendar year, each quintile’s aggregated age-standardized premature mortality rate (deaths before age 65, using the year 2000 standard million) and infant death rate (deaths among persons under age 1).

We tested our hypotheses about trends in the socioeconomic inequities in premature mortality and infant death in several ways, using measures of relative risk, absolute difference, changes in slope, and excess fraction of premature deaths. Our *a priori* hypothesis was that inflection points would occur in 1965 and 1980, given major federal policy changes during the Kennedy/Johnson and Reagan administrations.

SLIDE 12: US PREMATURE MORTALITY (<65) BY COUNTY INCOME QUINTILE, 1960-2002

What did we find? This figure basically tells the story. Between 1960 and 2002, in the US overall, even as premature mortality and infant death rates declined in all county income quintiles, the gap between the lower and highest income quintiles persisted and was relatively greatest for premature mortality in 2000 and barely changed for infant deaths. The greatest progress in reducing these income gaps occurred between 1965 and 1980; thereafter, the health inequities widened.

Overall, between 1960 and 2000, 18% of premature deaths would have been averted had the populations in the bottom four quintiles experienced the same yearly age-specific premature mortality rates as the highest quintile. This excess fraction translates to an estimated 4.9 million lives cut short.

SLIDE 13: AND NOW STRATIFIED BY RACE/ETHNICITY ...

The overall picture obscures stark racial/ethnic disparities within and across income quintiles, as shown on this slide. The steep decline in rates in the 1965-1980 period was especially notable in the populations of color, and especially in the two lowest income quintiles. The combined impact of class and color is further underscored by our results showing that between 1960 and 2002, had everyone experienced the same yearly age-specific mortality rates as whites in the highest income county quintile, then 14% of white premature deaths yet fully 30% of the premature deaths among populations of color would have been averted.

Here I note we also used joinpoint regression techniques to identify, empirically, the location of inflection points of, first, significant changes in the annual slope of the premature mortality rate for each income quintile, and second, in the annual slope of the incidence rate ratio and difference for each quintile. And what we found was that, especially for the US populations of color, our *a priori* hypotheses were supported by these post-hoc analyses.

Thus, as predicted, significant inflection points demarcated the period between the mid-1960s and the early 1980s, the era of the greatest declines in US premature mortality rates, especially among US populations of color residing in the poorest counties. Subsequently, as marked by the significant inflections points detected between 1982 and 1984, the average annual percent change in premature mortality rates dropped to less than half that of the preceding period for all socioeconomic-racial/ethnic strata – except for the white population living in the two highest county income quintiles, whose rate of decline stayed the same or increased. The same pattern was evident for changes in the magnitude of the incidence rate ratio and difference, and also for the analogous analyses of the infant death rates.

SLIDE 14: INTERPRETATION

In other words, what our results show is that within the United States, the burden of premature mortality and infant deaths, while declining, is strongly patterned by income and the color line. Is this surprising? At one level, no; the finding that risk of premature mortality increases with economic deprivation and racial inequality obviously is not new; research documenting these social facts easily extends back to the late 18th century. That said, what our results newly underscore is that contemporary US inequities are not immutable: they shrunk considerably between 1965 and 1980 and increased or stagnated thereafter. The early trends give grounds for hope; the latter augur poorly for the *Healthy People 2010* objective of eliminating US socioeconomic and racial/ethnic health disparities.

The finding of a declining -- then increasing -- gap is unlikely to be an artifact of inaccurate numerator or denominator data. Since 1960, 99% of all US deaths and births have been registered. The US census undercount (disproportionately affecting lower income populations and populations of color) has declined considerably (for example, for blacks, from 6.6% in 1960 to 2.8% in 2000), a trend that would increasingly reduce, not inflate, estimates of social disparities in mortality. Results are also unlikely to be affected by racial/ethnic misclassification, given the broad groupings employed. Rather, demographic trends should have lowered risk of premature mortality among US populations of color, given the increase

in foreign-born US populations of color and their associated “healthy immigrant” effect and the corresponding proportional decline in the US African American population (from 92% of US populations of color in 1960 to 72% in 2000).

What, then, might explain the observed trends? First, the rising US per capita gross domestic product (GDP) likely contributed to the overall decline in premature mortality rates (GDP grew, for example, by 32% for 1961-1970, 23% for 1970-1980, 25% for 1980-1990, and 22% for 1990-2000). But this rising GDP cannot explain the observed pattern of a diminishment and then increase in the socioeconomic gradient. Nor can the observed trends be explained simply by relative positioning in a social hierarchy, which doesn’t account for either the overall falling rates of premature mortality or the shrinking and then widening of the gap. It is also unlikely that purely individual-level behavioral factors can explain the faster and then slower decline in premature mortality among persons in the lower income quintiles, unless an argument can be made that health promotion efforts in this group were more successful in the earlier rather than later time period, which is dubious.

As an alternative explanation, consider two likely major societal determinants of health: economic priorities and civil rights. Likely contributing to the 1965-1980 improvements are the positive impact of the “War on Poverty” and the civil rights legislation that expanded economic opportunity and resources, and also availability of health services, for both the poor and populations of color, especially African Americans. Conversely, the subsequent slow-down likely reflects the adverse impact of post-1980 neo-liberal and neo-conservative policies to “roll-back” the welfare state. Here I mean: cutting federal responsibility and funds for public health and anti-poverty programs, opposing affirmative action, blocking rises in the minimum wage, and selectively decreasing taxes on the wealthy, coupled with rising medical uninsurance and persistent racial/ethnic disparities in quality of care.

To summarize, our results refute the view that widening health disparities necessarily accompany improvements in population health. Death is inevitable. Premature mortality is not. With one potentially hopeful hypothesis prompted by our findings being that if addressing social injustice and its embodied health consequences becomes a priority, we can make progress.

Moreover, as our data illustrate, the point is not “race” vs “class”: both matter, and we need to understand not only how class inequalities harm health within each and every racial/ethnic group, but also how racial inequality harms health, including within and across class strata.

SLIDE 15: EOD VALIDATION STUDY

Hence the need also for research on the direct impact of racial discrimination. What I will next do is briefly describe a validation study of a measure of self-reported experiences of racial discrimination that we published in 2005, based on an instrument I first developed in 1990 and have used with slight modification since, after which I will discuss current research on measuring racial discrimination and its health consequences.

SLIDE 16: EXPERIENCES OF DISCRIMINATION

Here, first, are the questions included in what I now call the “Experiences of Discrimination” (or EOD) measure, with the validation study nested within a larger on-going study, *United for Health*, focused on the health impact of social and occupational hazards experienced by a cohort of unionized yet still low-income workers in the Greater Boston Area. For each of the situations listed on this slide, participants were asked: “Have you ever experienced discrimination, been prevented from doing something, or made to feel inferior in any of the following situations because of your race, ethnicity, or color?” Those answering “yes” were asked a follow-up question about frequency. Overall 38% and 24% of the black and Latino participants reported experiencing discrimination in 3 or more of the 9 situations, compared to only 12% of the white participants, with similar differences in magnitude evident for the frequency score.

Analyses indicated all of the EOD items for frequency of discrimination were positively correlated and had a high Cronbach's alpha. Confirmatory factor analysis, taking into account correlated variances, produced an acceptable fit to the data for a single underlying factor. Differential item functioning analysis, which included data on race/ethnicity, birthplace, gender, age, education, and social desirability, indicated that no group-specific bias was present in the item responses, and also showed that EOD levels were not influenced by social desirability, age, or educational attainment. Additional analyses showed high test-retest reliability for the EOD (i.e., a correlation of 0.69 or higher). By contrast, test-retest reliability was disturbingly low (yielding correlations of at most 0.4) for several single-item measures we included asking about racial discrimination. These latter findings underscore why it is inadequate, if not invalid, to use single-item questions to assess experiences or awareness of racial discrimination, even though such single-item questions have been used in quite a number of studies.

SLIDE 17: SOCIAL HAZARDS: IN COMBINATION

Next, I present the social patterning of the self-reported experiences of racial discrimination within the full cohort of 1202 workers, in relation to not only race/ethnicity and gender but also the two additional social hazards on which we collected data: sexual harassment and workplace abuse. Among the black workers, the 2 most common combinations, together reported by over half the women and men, were, first, racial discrimination combined with workplace abuse (shown in yellow), followed by all 3 types combined (shown in red), with the latter most common among the black women. Among the Latino workers, a different set of combinations were the two most common, again reported by over half the women and men: first, racial discrimination plus workplace abuse (shown in yellow), followed by workplace abuse alone (shown in purple). Lastly, among the white workers, the most common category by far was workplace abuse alone (shown in purple), reported by slightly over 40% of the women & men. Additional analyses showed that, in the case of sexual harassment, one additional social category was relevant: that of sexuality, with the LGBT workers reporting twice as much sexual harassment as their heterosexual counterparts.

With one key point being that racial discrimination matters – and does not occur in isolation. This too is the point of the ecosocial construct of embodiment and its recognition that each and every day our bodies daily integrate our experiences. Hence, in order to understand the distributions of social hazards – and health more generally -- we have to remember that we are not one day a woman or a man, another day white or a person of color, another day working class or a professional, and still another day US-born or foreign-born: we are all of these at once, with the implication being that our research must reckon with diverse yet combined and embodied aspects of social position.

SLIDE 18: RACIAL DISCRIMINATION & RISK OF PRETERM DELIVERY

Next, demonstrating that it does make a difference, empirically, to include data on self-reported experiences of discrimination in epidemiologic research, are results of our study of racial discrimination and risk of preterm delivery, a major determinant of low birthweight and infant mortality. In this investigation, led by my colleague Dr. Sarah Mustillo, we used data from the US CARDIA study, a multi-city population-based longitudinal investigation concerned with black/white differences in risk of cardiovascular disease.

SLIDE 19: RACISM & PRETERM DELIVERY

The study population consisted of 367 women who gave birth between the Year 7 and 10 CARDIA exams. From the Year 7 exam, we obtained prospective data on self-reported experiences of racial discrimination, socioeconomic position, and other relevant baseline characteristics; from the Year 10 exam, we obtained the data on the birth outcomes. And what we found was:

- First: overall, the black women were 2.5 times more likely to have a preterm delivery than white women.
- Second: in analyses that took into account other major risk factors for preterm delivery, such as income, education, smoking, alcohol, and depression, controlling for these risk factors somewhat reduced the black/white difference, but black women were still at about 2 times the risk of having a preterm delivery.

-- Third: when we additionally included the data on racial discrimination, we showed there was no longer any racial/ethnic difference: the black and white women were at equal risk. Moreover, women who reported experiencing racial discrimination in 1 or 2 versus no situations were at twice the risk, and women who reported racial discrimination in 3 or more situations were at three times the risk, of having a preterm delivery compared to women who reported no racial discrimination.

Thus what our results showed -- for the first time -- is that experiences of racial discrimination not only predict risk of preterm delivery but also explain the excess black/white risk that the other conventional risk factors could not. Up until our investigation, however, most studies had included only these kinds of conventional variables, thereby concluding that the remaining unexplained black excess risk of premature delivery must be due to some innate biological difference between black and white women. But we show this interpretation is wrong because our study clearly demonstrates that by including information on experiences of racial discrimination we could explain the observed black/white disparities. This is a very powerful finding – and adds further evidence to the claim that the source of racial/ethnic disparities is the injustice in our society, not innate biology.

SLIDE 20: ARE EXPLICIT MEASURES ENOUGH?

Yet is reliance on what people self-report adequate? There are good grounds for skepticism. One concern, typically found in the writings of those dubious about claims of discrimination, is that self-reports of racial discrimination are likely to be inflated. I note, however, that even if this were indeed the case, the net impact would be to yield conservative estimates of the impact of self-reported experiences of racial discrimination on health, precisely because of problems of misclassification. In other words, including persons who weren't actually highly exposed in the "high exposure" category presumably would dilute comparisons of the effect of "high" vs "low" exposure on whatever the chosen outcome was ...

More worrisome, however, is the possibility that self-reports may in fact under-represent the experiences people have with racial discrimination. This is because of the well-known “person/group discrimination discrepancy” phenomena, whereby people from groups historically discriminated against tend to report more discrimination for their group compared to for themselves personally – a finding that social psychologists suggest may reflect how it is costly, psychologically, for people to consider themselves as being victims. Stated another way, it may be beneficial to distance oneself cognitively from negative attributes, as part of the overall psychological tendency to view and present oneself positively (even when such denial may not be in one’s self interest).

What led me to think about these issues were some interesting findings in my first major investigation of the relationship between self-reported experiences of racial discrimination and blood pressure, and which still stands as the largest study on this topic. What I found was that the relationship was linear (dose-response) among the professional African Americans, but curvilinear among the working class African Americans (who exhibited a J-shaped curve). One potential explanation for these findings is that for African Americans with more power and resources, a “no” may truly mean “no,” whereas among more disenfranchised persons, a “no” may reflect positive illusion, denial or internalized oppression. Here I note that what I observed is not a one-time fluke finding: several other studies on discrimination and health have replicated the finding of a dose-response relationship among professionals and highly educated persons, compared to a J-shaped curve among working class and less educated persons.

SLIDE 21: IAT STUDY

Back in 2000, I accordingly began to consider how we might benefit from using new approaches developed by social psychologists to study phenomena for which self-reported data might not fully capture what people think and feel. Specifically, I became interested in extending use of what is called the Implicit Association Test, or IAT, to study experiences of racial discrimination, building on its prior use to study racial prejudice.

In brief, the IAT is a computer-based reaction-time methodology designed to capture phenomena that lie outside of the reaches of introspective access. The test contrasts the time it takes to make associations between two sets of items, e.g., “flowers” with the word “good,” and “bugs” with the word “bad” – and then compares what happens when participants alternatively are asked to pair “flower” with “bad” and “bugs” with “good.” A difference in average matching speed for opposite pairings determines the IAT score. Participants are typically aware that they are making these connections but unable to control them given the rapid response times and structure of the test. More than 500 studies have employed numerous versions of the IAT and have found the results to be robust.

Right now, I’m currently engaged in research to test whether implicit measures of discrimination could perhaps usefully be combined with explicit measures to provide a more complete picture of the effects of discrimination on health. Today what I can share with you are some very preliminary results, based on a just-completed pilot study of a random sample of members of a community health center, conducted as preparation for a just-funded grant I’ve received on racial discrimination and risk of chronic disease.

SLIDE 22: EXPLICIT (EOD) & IMPLICIT (IAT) RESULTS

Here I show results for the 18 adult black participants included in our pilot study, all of whom were working age adults (between 25 and 64 years old) and US-born (a restriction imposed because research indicates immigrants of color, as categorized by US schemas of race/ethnicity, may have different understandings of – and likelihoods of reporting – experiences of racial discrimination, as compared to their US-born counterparts). Economically, 44% of the black participants were below the poverty line, 28% were at 100-199% above the poverty line, and 38% were at 200% or more above the poverty line.

Two findings stand out. First, the IAT and EOD were not correlated ($p > 0.7$). Second, the black participants explicitly reported higher levels of discrimination against blacks as a group than for themselves personally, whereas on the IAT, there was no discrepancy: they showed equally high

associations for discrimination against blacks as a group and themselves personally. These results suggest the IAT picked up experiences of discrimination the EOD did not – a finding that clearly has provocative implications for conducting and interpreting research on the health impact of discrimination.

For one thing, our results suggest that it potentially is misleading to interpret an explicit response of “no” discrimination as meaning the person is unaware of having ever been a target of discrimination. Second, they suggest that the growing use of the term “perceived discrimination” in the public health literature for studies using self-report measures is problematic, since such measures can, by definition only capture what people are able and willing to report; either the term should be changed to refer to “consciously perceived discrimination” or else it should be dropped and the more accurate phrase “self-reported experiences of discrimination” should be employed. Third, our results encouragingly show that it is feasible to use implicit as well as explicit measures in population-based health research – and likely is desirable to do so. Further underscoring the feasibility of using this enhanced approach, I am happy to note that we soon will be analyzing data from our just-about-completed web-based study that uses both implicit and explicit measures of racial discrimination to investigate their associations with several health behavior and health status outcomes.

SLIDE 23: CURRENT ISSUES IN STUDYING RACISM & HEALTH

So, to start wrapping up, as I hope my talk has made clear, we have a lot of questions to sort out for the science of health inequities as it pertains to the impact of racism on health. This is not surprising, given that work in this field is in many ways still in its infancy – albeit growing quickly, as you can see from the increase in number of studies included in recent review articles: from 15 in the article I wrote in 1999, the first review article on the topic, to 138 in an epidemiologic review published in 2006. A big increase, yes, but still less than a drop in the bucket when considering health research on race/ethnicity.

On this slide I list some of the important topics under discussion for research investigating how racism harms health, many – but not all – of which I have touched on in this presentation. In the case of studies using the direct approach, these questions include:

- (1) how we explicitly ask about people's experiences of discrimination, noting that the two most widely used measures, the EOD that I have described today and the one developed by my friend and colleague Dr. David Williams, use different approaches – whereby the former asks directly about these experiences and the latter asks first about unfair treatment and then whether this is attributed to race or some other reason (and here I note some ongoing psychometric research is testing whether these different approaches yield different results);
- (2) the importance – or not – of getting data on the timing and frequency of exposure;
- (3) resolving issues related to the cognitive biases of self-report data, e.g., by using the IAT; and
- (4) paying more heed to where immigration & generation fits into the picture.

Additionally, for studies using the indirect approach, important challenges pertain to:

- (1) improving our measures of structural or institutional discrimination, in relation to both process and outcome; and
- (2) determining which sorts of data about material and social conditions need to be collected at which level (e.g., individual, household, neighborhood, workplace, region, country, etc.) to capture the pathways by which racial inequality becomes embodied.

For example, new research on racial residential segregation is making very clear that there is nothing, ipso facto, that is bad for health about being in a predominantly “black neighborhood” – what matters instead is whether the segregation adversely concentrates economic and social deprivation. There is, after all, a world of difference between a reasonably affluent African American enclave and a destitute and largely African American neighborhood – and the two should not be conflated.

SLIDE 24: UNNATURAL CAUSES

And why bother to do this research? Consider the upcoming series “Unnatural Causes: Is Inequality Making Us Sick” that will be shown on PBS starting in late March. Powerfully bringing alive the kinds of public health research I have discussed, this series focuses on the impact of racial discrimination and socioeconomic deprivation on health; by way of disclosure, I’ve been involved with it as both a scientific advisor and as one of the scientists interviewed. This past October, I organized its Boston premiere screening as well as a session at last November’s American Public Health Association on how people in public health can use this series to aid efforts to rectify health inequities. In both places, the series had an electrifying effect. For the Boston screening; we had over 500 people attend at a standing-room only event; at APHA, over 550 jammed into the room, not counting those turned away. What so stood out was how this series affirms the experiences of those now suffering the brunt of health inequities, breaking through the individualism so rampant in our society, and making clear that, in the old-fashioned language of the day, the personal is in fact political. People’s health woes are at once individual and societal: we experience them as the unique individuals we are, within a context of societally-structured options and constraints. In my city and many others throughout the US, health departments and organizations are holding public events when it airs, to build on the series’ clear message that racism and economic deprivation harm health and can be challenged and countered. You can see the list of planned events if you go to the website listed on this slide.

SLIDE 25: CONCLUSION

In conclusion, we need to keep doing the rigorous work needed to establish the science and epidemiology of racism and health. Taking seriously the notion of embodying inequality entails engaging with intimate links between bodily truths and the body politic. With the goal being to generate knowledge that, if put into action, can inform current efforts to eliminate health disparities and promote instead social equity in health. Thank you.