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Race, socioeconomic status, and health: Complexities, ongoing challenges, and research opportunities

David R. Williams,[1,2] Selina A. Mohammed,[3] Jacinta Leavell,[4] and Chiquita Collins[5]

[1]Department of Society, Human Development and Health, Harvard School of Public Health, Boston, MA. [2]Department of African and African American Studies and of Sociology, Harvard University, Cambridge, MA. [3]Nursing Program, University of Washington Bothell, Bothell, WA. [4]Department of Dental Public Health, Meharry Medical College, School of Dentistry, Nashville, TN. [5]Altarum Institute, San Antonio, Texas

Address for correspondence: David R. Williams, Department of Society, Human Development and Health, 677 Huntington Avenue, 6th Floor, Harvard School of Public Health, Boston, MA 02115, USA. dwilliam@hsph.harvard.edu

This paper provides an overview of racial variations in health and shows that differences in socioeconomic status (SES) across racial groups are a major contributor to racial disparities in health. However, race reflects multiple dimensions of social inequality and individual and household indicators of SES capture relevant but limited aspects of this phenomenon. Research is needed that will comprehensively characterize the critical pathogenic features of social environments and identify how they combine with each other to affect health over the life course. Migration history and status are also important predictors of health and research is needed that will enhance understanding of the complex ways in which race, SES, and immigrant status combine to affect health. Fully capturing the role of race in health also requires rigorous examination of the conditions under which medical care and genetic factors can contribute to racial and SES differences in health. The paper identifies research priorities in all of these areas.

Keywords: socioeconomic status and health; race and health; genetics; social factors and health; migration and health

Introduction

This paper provides an overview of recent research on racial disparities in health and the complex ways in which race, ethnicity, and socioeconomic status (SES) combine to affect patterns of the distribution of disease. It begins by considering evidence of the magnitude of racial disparities in health and the striking pattern of earlier onset of illness and more severe disease for minorities compared to Whites. It uses recent national data to illustrate the persistence of racial differences in SES and gives attention to the accumulating evidence that indicates that racial disparities in health persist at every level of SES. Understanding and effectively addressing racial disparities in health requires an appreciation of the contributing factors that importantly affect the racial patterning of the distribution of disease. First, we need to comprehensively delineate the multidimensional social concomitants of race and understand how they relate to each other and combine to affect health. This will require the consideration of SES in all of its complexity, as well as, how other social risk factors and resources combine to affect health. Second, we need to understand the ways in which migration history, status, and context affect health and combine with SES to produce particular patterns of disease distribution. Third, we need to take a renewed look at two historical and often misunderstood potential explanations for social variations in health: medical care and genetics. There has been polarizing discourse regarding the potential contribution of genetics and medical care to racial differences in disease. Both of these explanations are critically evaluated and the paper calls for a new generation of research that will move beyond either/or explanations. We argue that research that will advance our scientific understanding should seek to comprehensively quantify risks and resources in the social environment linked to race and SES and

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examine how they cumulate over the life course and combine with innate and acquired biological factors to affect health.

Racial categorization in the United States and elsewhere has historically reflected oppression, exploitation and social inequality.[1] In health research, these categories were often viewed as meaningful indicators of genetic distinctiveness.[2] This paper views "race" as capturing ethnicity—common geographic origins, ancestry, family patterns, language, cultural norms and traditions, *and* the social history of particular groups. The U.S. Government's Office of Management and Budget (OMB)[1] recognizes five racial categories (White, Black, American Indian or Alaskan Native, Asian, and Native Hawaiian and other Pacific Islander) and one ethnic category (Hispanic). In this paper, in the interest of economy and parsimony of presentation, we use "race" to refer to both the OMB racial and ethnic categories. Several considerations influenced this decision. Both categories capture ethnic or cultural variation, the distinction between the concepts are arbitrary and blurred, with many individuals considering the terms synonymous and with most Hispanics preferring that Hispanic be treated as a "racial" category.[3–5] In addition, race, as an influential social category in the United States, has historically captured not cultural practices and beliefs but societally imposed stigmatization and marginalization that have been consequential for all aspects of life. We use the term ethnicity to refer to subgroups of the global OMB categories. In addition, in recognition of individual dignity, we use the most preferred terms[5] for the OMB categories interchangeably (e.g., Black and African American, Hispanic and Latino, American Indian, and Native American).

Racial disparities in health

Racial disparities in health have been long noted in the United States. In describing these differences, we will be attentive to the role of gender and present gender differences whenever the data are available. It has recently been argued that although black women lag behind other social groups on some societal indicators, they are nonetheless rapidly becoming a "model minority" on a broad range of indicators.[6] For example, black women have a higher rate of college enrollment than black males but also than whites and Hispanics. In addition, they also have lower suicide rates than black males and whites and low rates of crime, cigarette smoking, alcohol use, and the use of illegal drugs.

The routine reports of the National Center for Health Statistics (NCHS) provide life expectancy data only for blacks and whites. In 2004, the life expectancy at birth for blacks was 73.1 years compared to 78.3 years for whites.[7] If blacks could improve their life expectancy at the rate at which overall life expectancy increased in the United States between 1980 and 2000 (an average of 0.2 years annually), it would take them 26 years to close the current 5.2-year gap in life expectancy! Table 1 presents life expectancy at birth and at specific ages for blacks and whites of both sexes.[7] These data illustrate the complex ways in which multiple statuses combine to affect health risks. First, life expectancy differences by race, for both men and women, are large during early and mid adulthood and decline with increasing age. Second, at every age except the eldest, the racial differences in life expectancy are larger for men than for women. Third, factors linked to both race and sex likely contribute to life expectancy such

Table 1. Life expectance at birth and at age 25, 45, 65, 85 by Race 2004

	Male			Female			Gender Differences	
Age	White	Black	Difference	White	Black	Difference	Whites	Blacks
0	75.7	69.5	6.2	80.8	76.3	4.5	5.1	6.8
25	52.0	46.7	5.3	56.6	52.8	3.8	4.6	6.1
45	33.4	29.1	4.3	37.4	34.3	3.1	4.0	5.2
65	17.2	15.2	2.0	20.0	18.6	1.4	2.8	3.4
85	6.0	6.3	−0.3	7.1	7.5	−0.4	1.1	1.2

Arias, 2007[7], NVSS (NCHS).

Table 2. Age specific death rates for 2006 for whites and minority/white ratios

	Males					Females				
Age	White rate	Black/ White ratio	AmInd/ White ratio	API/ White ratio	Hispanic/ White ratio	White rate	Black/ White ratio	AmInd/ White ratio	API/ White ratio	Hispanic/ White ratio
1–4	2.7	1.8	2.2	0.7	1.1	2.3	1.7	2.2	0.9	1.0
15–14	1.6	1.6	1.1	0.7	1.0	1.2	1.5	1.4	0.9	1.0
15–24	10.8	1.6	1.5	0.6	1.1	4.3	1.2	1.5	0.6	0.8
25–34	14.1	1.9	1.4	0.4	0.8	6.2	1.8	1.5	0.5	0.7
35–44	23.3	1.7	1.5	0.4	0.8	13.6	1.9	1.5	0.4	0.6
45–54	51.5	1.8	1.1	0.5	0.8	30.0	1.9	1.1	0.5	0.7
55–64	106.4	1.8	1.0	0.5	0.8	66.8	1.6	1.0	0.5	0.7
65–74	249.0	1.5	0.9	0.5	0.8	167.7	1.4	1.0	0.5	0.7
75–84	627.8	1.2	0.7	0.6	0.7	446.1	1.1	0.8	0.6	0.7
85+	1484.1	0.9	0.5	0.6	0.6	1315.1	1.0	0.5	0.6	0.7

Note: National Center for Health Statistics, 2009.[8,9]
Deaths per 10,000 population.
Both the Black and White category excludes Hispanics.
AmInd = American Indian; API = Asian and Pacific Islander.

that, at every age, black women have higher levels of life expectancy than white men. Fourth, gender differences in life expectancy are consistently larger for blacks than for whites. Finally, the gap in life expectancy between black men and women is consistently larger, at every age, than the racial gap in life expectancy.

National mortality data provide another window on racial disparities in health. Table 2 presents the overall mortality rates for whites by age in the United States in 2005 and the minority/white mortality ratios.[8,9] The data reveal that African Americans and American Indians have a consistent pattern of elevated mortality risk compared to whites. This pattern is evident in early childhood, and persists until advanced age. In contrast, Latinos have rates that are roughly equivalent at the youngest ages but are lower than those of whites at older ages. Asians, a diverse group with an even higher proportion of immigrants than Hispanics, have mortality rates that are markedly lower than those of whites, throughout the life course. The disparities in mortality are generally similar for men and women although there is a consistent trend for the health advantage of Hispanics over whites to be slightly larger for women than for men.

National data on mortality are more accurate for blacks and whites than for Hispanics, Asians and Native Americans. A major problem affecting the quality of mortality data is related to the undercount in the number of deaths because of the misclassification of nontrivial proportions of Hispanics, Asians, and especially American Indians as white on death certificates.[10] This error leads to an undercount in the numerator for death rates for these groups that underestimates their actual death rates. Challenges of population coverage and/or inadequate sample size for small racial populations and ethnic subgroups within the larger racial categories continue to obscure population patterns of health risks. For example, aggregation of the Native Hawaiian and other Pacific Islander (NHOPI) group with the Asian category obscures the reality that the NHOPIs are one of the highest risk populations in the United States in terms of CVD, diabetes, and obesity risk.[11] Similarly, Arab Americans are masked under the white population of the United States and although they have relatively high levels of SES, they have elevated risk of some diseases such as diabetes and certain cancers.[12]

First and worse

One of the characteristics of the elevated rates of disease for minorities compared to whites is the earlier onset of illness, greater severity of disease and poorer survival. For example, a 20-year follow-up of

the CARDIA study's cohort of young adults found that incident heart failure before the age of 50 was 20 times more common in blacks than whites with the average age of onset being 39 years old.[13] National data from the NHANES study also shows that hypertension occurs earlier in blacks than in whites.[14] During the 1999–2002 period of this study, 63% of black adults with hypertension were younger than 60 years of age compared to 45% of their white counterparts. Moreover, for both racial groups the proportions with hypertension under age 60 in 1999–2002 was higher than during the earlier 1988–1994 data collection period (59 and 41%, respectively).

An unexpected patterning of neonatal mortality rates by mother's race and age can be viewed as evidence of premature aging. National data on first births for white and Mexican American women reveal that, as expected, infant mortality rates are lower for mothers who give birth in their twenties compared to those in their teens.[15] The opposite pattern is evident for black and Puerto Rican women, where the lowest rates of neonatal mortality are evident for births during the 15–19 age group, with rates increasing as childbearing is delayed to ages 20–29 or later. Geronimus[15] "weathering hypothesis" argues that for groups residing in unhealthy contexts, chronological age reflects higher levels of exposure to adverse conditions in social and physical environments and greater wear and tear on physiological systems.

A striking example of a racial difference in the early onset of disease is evident for breast cancer. It is well documented that white women have an overall incidence of breast cancer that is higher than that of blacks. However, the opposite pattern exists under the age of 40, with African American women having a higher incidence of breast cancer than their white counterparts.[16] Thus, despite having a lower overall incidence rate of breast cancer compared to their white peers, black women have a higher risk of early onset, severe types of breast cancer and a reduced risk of late-onset types.[16] The association of some risk factors with breast cancer varies by age: parity is associated with an increased risk of breast cancer incidence in younger women but a reduced risk in older women.[16] Similarly, while obesity is associated with reduced risk in younger women, it is associated with increased risk in older women. Importantly, the crossover in breast cancer incidence by race persists even after adjustment for risk factors.[16] In addition to being more likely to get breast cancer when young, black women are more likely than whites to be diagnosed at an advanced stage, have aggressive forms of the disease that are resistant to treatment, and to have poorer outcomes.[17]

Major depression is another disease for which blacks have a lower rate of illness but a prognosis that is considerably worse than whites. A recent national study found that although blacks have lower current and lifetime rates of major depression than whites, the cases of depression among blacks were more likely to be persistent, severe, disabling, and untreated.[18] National data also reveal that whites are more likely than blacks (but not Hispanics) to develop alcohol dependence.[19] However, once dependence existed, both blacks and Latinos were more likely than whites to persist in alcohol dependence.

Other evidence indicates that the increased risk of disease for minorities is evident very early in life. A study of 23-year-old young men found that compared to whites of similar BMI, body fat, fitness, renal function, blood lipids and glucose levels, black men had comparable brachial blood pressure (BP), but greater central blood pressure, greater augmentation of central BP from wave reflections, and greater macrovascular and microvascular dysfunction reflected in increased central artery stiffness and reduced peripheral endothelial function.[20] A recent report from a longitudinal birth cohort study of 8,550 children in the United States provided further evidence that racial differences in risk factors are established early.[21] By age 4, 13% of Asians and 16% of whites were obese, compared to 21% of blacks, 22% of Hispanics and 31% of American Indians. Neither of these studies included adjustment for SES.

Further evidence of the early health deterioration of African Americans compared to whites comes from analyses of a 10-item measure of allostatic load that was attempting to capture the physiologic burden on multiple biological systems in the NHANES data, due to the wear and tear of exposure to stressors and other environmental adversity.[22] This study found that blacks had higher scores than whites at all ages, with the racial differences being most marked between 35 and 64 years. Moreover, the racial differences persisted after adjustment for

poverty with nonpoor blacks having higher scores than poor whites. In addition, black women consistently had higher allostatic load scores than black men.[22] These differences could be due to the double jeopardy of racial and gender discrimination that women face, and/or to stressors linked to the central role that black women often play as economic providers to their families.

Research also reveals that some risk factors have a more adverse impact on blacks than on whites even when their overall levels are lower than or similar to those of whites. This is evident for both tobacco and alcohol. For example, the risks of lung cancer do not mirror variations in smoking behavior with a given level of smoking associated with an elevated risk of lung cancer for African Americans and Native Hawaiians compared to whites, Japanese Americans and Latinos.[23] In a similar vein, alcohol-related mortality is more than twice as high for black males than for their white counterparts and almost twice as high for females.[24] A general population sample in New York State also found evidence of greater susceptibility to liver damage in blacks compared to whites.[25] Compared to whites, blacks had higher levels of common biomarkers of liver damage at every level of alcohol consumption with the differences being largest at the highest level of alcohol use. This pattern persisted after adjustment for age, sex, education, BMI, and pack years of smoking. We are currently unaware of the extent to which these patterns reflect group variation in the specific types of substance used (compared to whites, blacks are more likely than whites to use menthol cigarettes and to use hard liquor), differential exposure to unmeasured physical and chemical agents in occupational and residential environments, or interactions between health practices and stressors that markedly exacerbate health risks.

Relatedly, several studies find that moderate alcohol consumption is not associated with better health outcomes among African Americans, as it is for whites. In the NHANES Epidemiologic Follow-up Study (NHEFS) there was no beneficial effect of moderate alcohol consumption on all-cause mortality for black men or women.[26] Moreover, although moderate alcohol consumption tends to be cardioprotective in middle-aged adults, in the Atherosclerosis Risk in Communities (ARIC) study, opposite to the pattern for whites and black females, it was positively related to incident coronary heart disease[27] and incident hypertension for black men.[28] Similarly, the CARDIA study found a positive association between alcohol consumption and the development of coronary calcification with the absence of a beneficial effect of moderate alcohol consumption being clearest among black males.[29] The factors underlying these patterns are not well understood, but it is possible that SES is a contributor. There is considerable evidence that at least some of the reported protective effects of moderate drinking are likely due to residual confounding of moderate alcohol consumption with high SES and good health practices,[30–32] and limited evidence that when controls are introduced for multiple measures of SES, the inverse association between moderate consumption and mortality is no longer evident.[33]

Research is also needed to identify the mechanisms and processes that give rise to those situations where whites are more adversely affected by risk factors than racial minorities. In one national study, for example, persistent poverty was unrelated to stunting and wasting for blacks, but positively related for whites and Hispanics.[34] Similarly, black newborns are twice as likely as white ones to be low birth weight, but low birth weight is more strongly linked to neonatal mortality for whites than for blacks.[35] Although the prevalence of obesity is higher for black than for white women, obesity is more strongly related to mortality for white than for black women.[36,37] Future research could profitably explore the extent to which these patterns could reflect weaker, habituation effects for blacks due to earlier exposure and elevated levels of exposure, and/or the conditions under which the presence of cultural, SES, psychosocial, religious and other resources can weaken the health effects of certain risk factors. Research should also examine the extent to which observed racial variations in the effects of risk factors are similar to SES differences. Earlier research on stress and health found that although both blacks and low SES persons were more exposed to stress than whites and high SES individuals, comparable stressors had a more adverse effect on the mental health of both socially disadvantaged groups.[38] More generally, these patterns of findings highlight the importance of attending not only to variations in exposure to risk factors but also to variations in vulnerability as reflected in both differential preparedness for coping with adversity and differential ability to recover.[39]

Table 3. Infant mortality rate,[a] 1940–2006

Year	Males				Females			
	White (W)	Black (B)	Difference (B-W)	Ratio (B/W)	White (W)	Black (B)	Difference (B-W)	Ratio (B/W)
1940	48.3	81.1	32.8	1.7	37.8	64.4	26.8	1.7
1950	30.2	48.3	18.1	1.6	23.1	39.4	16.3	1.7
1960	26.0	49.1	23.1	1.9	19.6	39.4	19.8	2.0
1970	20.0	36.2	16.2	1.8	15.4	29.0	13.6	1.9
1980	12.1	24.6	12.5	2.0	9.5	20.2	10.7	2.1
1990	8.5	19.6	11.1	2.3	6.6	16.3	9.7	2.5
2000	6.2	15.5	9.3	2.5	5.1	12.6	7.5	2.5
2006	6.1	14.4	8.3	2.4	5.0	12.2	7.2	2.4

Note: National Vital Statistics Reports.[41]
[a]Rate of deaths per 1000 live births in specified group.

Trends in racial disparities over time

Another noteworthy characteristic of racial disparities is their persistence over time. Table 3 presents infant mortality rates for blacks and whites, males and females, from 1940 to 2006.[40] For both racial groups, there were marked declines in infant mortality over time, with the absolute difference in infant mortality in 2006 being only about one quarter of what it was in 1940. At the same time the relative difference in infant death rates for both males and females, increased from 1.7 in 1940 to 2.4 in 2006. For both racial groups, infant mortality has also been consistently higher for males and females. Trend data for heart disease and cancer—the two leading causes of death in the United States—indicate that blacks and whites had comparable death rates for these conditions in 1950, but that African Americans currently have higher mortality rates for both of these diseases than whites.[41] Long term trend data is readily available only for blacks and whites. However, trend data exists for the 60% of the American Indian population served by the Indian Health Service (IHS). The IHS began in 1955, and widening disparities in health are evident for Native Americans compared to whites for multiple causes of death such as diabetes and liver cirrhosis.[42]

Unpacking the social context

All indicators of SES are strongly patterned by race, with SES being a key determinant of racial disparities in health. We review recent national data on the relationship between race and SES, as well as, the ways in which race and SES combine to affect health status. We show that indicators of individual-level SES are not equivalent across race. We also show that because residential segregation by race has led racial groups in the United States to live in distinct neighborhood environments, social conditions at the neighborhood and community level make an important contribution to racial differences in health. We need a better understanding of the ways in which pathogenic factors linked to place and other aspects of racism can adversely affect health. Fully understanding the determinants of racial differences in health requires research that explores, across the life course, all aspects of the social context that can have health consequences.

Race and socioeconomic status

Racial categories in the United States embody both historical and contemporary social inequality and any attempt to understand racial disparities in health needs to consider the extent to which race is associated with SES. National data for the United States reveal strikingly high levels of racial inequality in SES, and relatively little change over time. This is in contrast to the perception that racial inequalities have narrowed markedly over time and may even be nonexistent. Some limited evidence indicates that the election of President Barack Obama may represent a setback for addressing racial injustice

Table 4. Percent of persons and children below poverty, 1980–2006

Year	White	Black	Asian and Pacific Islander	Hispanic
All persons below poverty				
1980	10.2	32.5	NA	25.7
1990	10.7	31.9	12.2	28.1
2000	9.5	22.5	9.9	21.5
2006	10.3	24.3	10.3	20.6
Children below poverty[a]				
1980	13.4	42.1	NA	33.0
1990	15.1	44.2	17.0	37.7
2000	12.4	30.9	12.5	27.6
2006	13.6	33.0	12.0	26.6

Note: U.S. Census Bureau, 2008[44] (Tables 689,690).
[a]Children in families under 18 years old.
NA = not available.

in the United States because it has reduced (both among persons who voted and did not vote for him) the perception of the existence of racial injustice and inequality and reduced support for policies designed to address inequality.[43]

Table 4 shows the levels of overall poverty and child poverty by race from 1980 to 2006.[44] Both blacks and Hispanics have levels of overall poverty that are two to three times higher than those of whites. Asians have poverty levels that are generally comparable to those of the white population. Variation in poverty rates are also evident over time with the percent of persons living in poverty being lower in 2000 than in the two prior decades for all racial groups. And for all groups, except Hispanics, the poverty rate in 2006 is higher than it was in 2000. However, at all points in time, poverty varies markedly by race. Table 4 also shows that child poverty rates, for all racial groups, are higher than overall poverty rates and child poverty is also strongly patterned by race. Some limitations of the available data must be noted. The overall data for Asians mask considerable heterogeneity within the Asian population with some Asian groups (Hmong, Laotian and Cambodian) having levels of income that are comparable to those of the African American and American Indian populations.[10] American Indians and Pacific Islanders also have higher levels of poverty compared to whites.

Table 5 considers the extent to which years of formal education—another indicator of SES—is patterned by race. It takes an even longer view than the poverty data by presenting educational attainment from 1960 to 2006.[44] The data reveal remarkable increases in education over time with the percentage of blacks completing high school increasing fourfold from 20% in 1960 to 82% in 2006. Similarly, the percentage of whites completing high school doubled from 43% in 1960 to 86% in 2006. In 2006, the disparity between whites and blacks in high school graduation rates had narrowed dramatically from earlier years. The Asian and Pacific Islander population had a rate of high school graduation that was slightly higher than the white population, but only 60% of Hispanic adults had graduated from high school—a rate that was markedly lower than that of all other racial groups. There is also striking ethnic variation in high school completion within the Latino population with Cubans (80%) and Puerto Ricans (74%) having markedly higher high school graduation rates than Mexicans (54%).

College graduation rates also show steady increases over time for all groups, but with marked variation in 2006. The college graduation rate of Asian and Pacific Islanders (52%) is markedly higher than that of whites (29%) who have a rate that is higher than African Americans (19%) and Latinos (13%). The Hispanic category masks considerable heterogeneity with Cubans having a college graduation rate that is three times higher than Mexicans. Other data reveal that the narrowing of the racial gap in education over time has not led to a commensurate narrowing of the racial gap in income.[45] This gap has been especially acute for black males. For example, in contrast to the rising earnings of black men relative to white men between the early 1960s and the mid-1970s, the relative pay of African American men declined over the next two decades. The relative pay of college-educated black men compared with white men fell by 10% between 1979 and 1997.[45]

Race, SES, and health

Researchers have long assumed that these racial differences in SES make a substantial contribution to racial disparities in health and there is considerable data to assess the role of SES in racial

Table 5. Educational attainment by race/ethnicity 1960–2006s

Year	White	Black	Asian and Pacific	Hispanic			
				All	Mexican	Puerto Rican	Cuban
High school graduate or more							
1960	43.2	20.1	NA	NA	NA	NA	NA
1970	54.5	31.4	NA	32.1	24.2	23.4	43.9
1980	68.8	51.2	NA	44.0	37.6	40.1	55.3
1990	79.1	66.2	80.4	50.8	44.1	55.5	63.5
2000	84.9	78.5	85.7	57.0	51.0	64.3	73.0
2006	86.2	82.3	87.8	60.3	53.9	73.5	79.8
College graduate or more							
1960	8.1	3.1	NA	NA	NA	NA	NA
1970	11.3	4.4	NA	4.5	2.5	2.2	11.1
1980	17.1	8.4	NA	7.6	4.9	5.6	16.2
1990	22.0	11.3	39.9	9.2	5.4	9.7	20.2
2000	26.1	16.5	43.9	10.6	6.9	13.0	23.0
2006	29.1	18.5	52.1	12.7	9.0	16.4	27.2

Note: U.S. Census Bureau, 2008[44] (Table 221).
NA = not available.

health disparities. Research has found that some of the observed racial disparities in health reflect the effect of differences across population groups in socioeconomic circumstances, but that even after taking SES into account racial disparities in health often remain. For example, a study using national data from the Health Interview Survey linked to the National Death Index found that there were large black–white mortality hazard ratios at the youngest age group (ages 18 through 25) and that these declined but remained substantial up through the over 75 age group.[46] Importantly, even after adjustment for SES (income and education), the black–white mortality ratios remained larger than one up through the oldest age categories. A similar pattern is evident across a broad range of outcomes for multiple racial groups. For example, research on posttraumatic stress disorder (PTSD) indicates that blacks, Hispanics, Asians, American Indians, and Native Hawaiians have higher rates of PTSD than whites that are not accounted for by SES and their history of psychiatric disorders.[47]

Further evidence of the elevated disease risk for African Americans after SES is considered comes from national data on chronic disease risk factors for blacks, whites and Hispanics aged 40 and over.[48] This study assessed indicators of blood pressure risk (systolic, diastolic, and pulse rate), inflammation risk (C-reactive protein, fibrinogen, albumin) and metabolic risk (total cholesterol, HDL cholesterol, BMI and glycated hemoglobin). A summary indicator of total risk counted how many of these 10 risk factors were outside of the normal range. This study found that even after adjustment for income, education, gender and age, blacks had higher scores on blood pressure, inflammation, and total risk. Importantly, blacks maintained a higher risk profile even after adjusting for health behaviors (smoking, poor diet, physical activity, and access to care).

There are other examples of racial differences in disease even among high SES groups. A comparison of a cohort of white physicians from Johns Hopkins University with a cohort of black physicians from Meharry Medical College revealed that there were large racial differences in health even in this group of highly educated professional males working in similar social contexts.[49] This study found a higher rate of cardiovascular disease and an earlier onset of disease in the black than the white physicians. Incident diabetes and hypertension, for example, were twice as high in the black compared to the white physicians. Similarly, a study of men in multiple health professions found a higher

incidence of prostate cancer in African Americans than in whites.[50]

National data on life expectancy at age 25 illustrates the contribution of both race and SES. These data reveal that there is a black–white difference in life expectancy at age 25 of 4.4 years for men and 4.3 years for women.[51] There are also large life expectancy differences by income within each racial population. High income white males outlive their low income counterparts by 7.9 years compared to an income gap of 8.6 years among African American males. The income gap is 3.3 years among white women, and 5.0 among black women. Thus, the gap in life expectancy by income for white men and black men and women is larger than the black–white gap. Moreover, at every level of income, white males and females live 2–4 years longer than their African American peers. Similarly, differences in cancer mortality by education within each race are much larger than the overall racial differences. For example, the mortality rate for white men with 8 years of education or less was almost nine times greater than that of their peers with 17 or more years of education.[52] Complex patterns emerged when race, gender, and SES were considered simultaneously. Among men, residual effects of race in which blacks had elevated mortality risk were evident at five of the six education levels.[52] Among women, blacks had higher mortality rates than whites at the three highest education levels while the pattern was reversed at the lower levels of education.

Other complex patterns are also evident. One study found a pattern consistent with a "diminishing returns hypothesis" in which as SES levels increase, blacks do not have the same improvements in health as their white counterparts, with the racial disparity being largest at the highest levels of SES.[53] Other research has found a distinctive pattern of association between SES and health for black men in which poor health and/or risk factors are positively related to SES. In the CARDIA study, for example, education was associated with an increasing risk of a poor lipid profile (high triglycerides, LDL cholesterol, total cholesterol, and lower HDL cholesterol) for black men while the opposite pattern existed for black women and whites.[54] Other studies have found SES to be positively associated with suicide, hypertension and stress among Black men.[55] Cigarette smoking provides another example of these complex interactions. Black men and women have levels of cigarette smoking that are comparable to those of whites, but when race and SES are simultaneously considered, at every level of income and education, cigarette smoking is lower for blacks than for whites, with the differences being especially marked at low SES levels.[56]

A clear priority for research is to understand the complex ways in which SES combines with race and sex to affect patterns of health. We earlier noted the gaps by race and SES in national data on life expectancy at age 25.[51] The patterns become more complex when we simultaneously consider sex. At age 25, white women outlive their male counterparts by 6.6 years and African American women outlive their male peers by 6.7 years. That is, the gender differences in life expectancy are larger than the racial ones. Moreover, the effects of the occupancy of multiple statuses are additive at their intersections. For example, the most advantaged group in terms of health, high income white women have a life expectancy at 25 of 58 years while the most disadvantaged group, low income black men, have a life expectancy at 25 of 42 years. This 16-year difference in life expectancy between these two categories is four times the overall black–white difference and twice the income and gender differences. These examples highlight the need for careful and systematic efforts that pay attention to the indicator of health status, the measure of SES and the racial and gender group under consideration so that we can begin to identify the conditions under which particular patterns in the social patterning of disease are more or less likely to occur. Research is needed to identify how biological factors linked to sex and social factors linked to gender combine with risks and resources linked to SES and race to affect patterns of health risks.

Nonequivalence of SES across racial groups

One contributor to the persistence of racial differences in health after SES is controlled is that traditional measures of SES are not equivalent across race. For example, compared to whites, college-educated blacks are more likely to experience unemployment, employed blacks are more likely to be exposed to occupational hazards and carcinogens even after adjusting for job experience and education, and have less purchasing power because the costs of a broad range of goods and services are higher in Black communities.[45,57,58] A large federal

Table 6. Mean earnings by highest education completed, 2006

Education	Males			Females		
	White	Black	Hispanic	White	Black	Hispanic
0–11 years High school graduate	24,579	21,294	23,060	15,483	14,277	15,072
High school graduate	38,833	30,122	32,148	23,334	22,643	20,608
Some college	39,924	34,033	34,414	25,595	25,832	23,628
Associate degree	49,061	36,534	41,445	32,889	35,328	31,675
Bachelor's degree	71,735	52,569	51,336	43,142	44,326	38,825
Master's degree	89,837	62,396	81,885	53,062	50,916	51,344
Professional degree	133,988	A	97,035	85,112	80,038	B

Note: US Census Bureau, 2008[44] (Table 224).
A = Base figure too small for reliable calculation. Mean for Black males and females is $101,374.
B = Base figure too small for reliable calculation. Mean for Hispanic males and females is $82,627.

survey illustrates how the minority poor are poorer than the white poor. It found that even after adjustment for a broad range of demographic, SES, and health status factors blacks were more likely than whites to report six economic hardships (unable to meet essential expenses, pay full rent or mortgage, pay full utility bill, had utilities or telephone shut off, or was evicted from one's apartment).[59]

Table 6 shows differences in mean earnings, by race and sex, in 2006 by the highest level of education completed.[44] Among men, there are large differences in earnings at every level of education with the gap, especially for black men, widening as education increases. For example, black men with a master's degree earn about $27,000 less than their white counterparts with the same level of education. Among women, a different pattern is evident. For women with a bachelor's degree or less education, racial differences in earnings are nonexistent or small with some of them favoring African American women. In contrast, at the two highest education categories, both black and Hispanic women earn less than their white counterparts. Other data reveal that the small racial differences in individual earnings by education for women mask large differences in household income at every level of education for women.[60] Moreover, racial differences in income understate the magnitude of racial disparities in economic status. There are large racial differences in wealth with African Americans having 9 cents and Hispanics 12 cents for every dollar of wealth that whites have.[61] These racial differences in wealth persist at every level of income. Among persons with income in the bottom 20% of all US households, for every dollar of wealth that whites have, blacks have one penny and Latinos have two. Among the highest earning fifth of U.S. households, blacks have 31 cents and Hispanics have 35 cents for every dollar of white wealth.

Area-based differences in SES

Pronounced racial differences in SES at the neighborhood and community level are likely an important contributor to the residual effects of race after adjustment for individual and household level indicators of SES. These differences in neighborhood quality and community conditions are driven by residential segregation by race—a neglected but enduring legacy of institutional racism in the United States[62] Considerable evidence suggests that because of segregation, the residential conditions under which African Americans, American Indians and an increasing proportion of Latinos live are distinctive from those of the rest of the population. A recent study documented striking differences in opportunities for growth and development of children in the 100 largest metropolitan areas in the United States where children reside. It found that 76% of African American children and 69% of Latino children live under worse conditions than the worst off white children.[63] Another study found that in one third of the largest metro areas, there is no overlap in neighborhood poverty between blacks and whites and that neighborhood poverty distributions of whites overlapped those of blacks and Latinos only 27% of the time.[64] Similarly, a

national study of African Americans and Caribbean blacks that attempted to compare the black population to whites who live in similar residential contexts found that only 14% of whites in the United States reside in Census tracts or block groups where 10% or more of the population is black.[18] A study of the 171 largest cities in the United States reported that there was not even one city where blacks lived under similar ecological conditions to those of whites in terms of concentrated poverty and female headed households.[65] It concluded that the worst urban context in which whites reside was better than the average context of black communities.

Segregation probably has a larger impact on the health of African Americans than other groups because blacks currently live under a level of segregation that is higher than that of any other immigrant group in United States history.[62] In addition, the association between segregation and SES varies by minority racial group. For Latinos and Asians, segregation is inversely related to household income but segregation is high at all levels of SES for blacks.[66] In the 2000 Census, the highest SES blacks (incomes greater than $50,000) were more segregated than the poorest Latinos and Asians (incomes less than $15,000).[66] A study of 6 community areas in Chicago illustrates how increasing household income does not translate into markedly improved neighborhood conditions or health for blacks.[67] It found that in two heavily African American areas that varied markedly in income (one having a median income of $38,000 and the other of $18,000), residents of the better off black neighborhood did better than those of the poorer one on only 4 out of 13 health measures. Because of the relatively high levels of disadvantage of African American neighborhoods regardless of SES, studies of neighborhood conditions and health have often used race-specific measures of neighborhood factors and have found that neighborhood variables were more strongly related to the health of whites than of blacks.[68]

There are multiple pathways through which segregation can adversely affect health.[62,69] First, segregation limits socioeconomic mobility by limiting access to quality elementary and high school education, preparation for higher education and employment opportunities. Second, the conditions created by concentrated poverty and segregation make it more difficult for residents to adhere to good health practices. The higher cost, poorer quality, and lower availability of healthy foods in economically disadvantaged neighborhoods can lead to poor nutrition. The heavy targeting of disadvantaged minority communities with advertising for tobacco and alcohol can encourage the use of these products. The lack of recreation facilities and concerns about personal safety can discourage leisure time physical exercise. Third, the concentration of poverty can lead to exposure to elevated levels of economic hardship and other chronic and acute stressors at the individual, household and neighborhood level. Fourth, the weakened community and neighborhood infrastructure in segregated areas can also adversely affect interpersonal relationships and trust among neighbors. Fifth, the institutional neglect and disinvestment in poor, segregated communities contributes to increased exposure to environmental toxins, poor quality housing and criminal victimization. Finally, segregation adversely affects both access to care and the quality of care.

Research has linked residential segregation to an elevated risk of illness and death and shown that it contributes to the racial disparities in health.[62,70] A recent study of US metro areas found that residential area mattered for birth outcomes for blacks. In contrast to prior work using a single dimension of segregation, this study operationalized hypersegregation as areas scoring high on four or five of the distinct dimensions of segregation.[71] It noted that although only 9% of metro areas representing 28% of U.S. births were hypersegregated, some 40% of black women of childbearing age lived in hypersegregated areas. In addition, black infants in hypersegregated areas were more likely to be preterm than those in less segregated areas and black–white differences in preterm birth were larger in hypersegregated areas than in less segregated ones. It also found that the association of increasing age with poorer birth outcome for African Americans was greater in hypersegregated areas than in other areas.[71] Another study found that the elevated prevalence of CVD risk factors for blacks and Hispanic premenopausal women compared to white women was markedly reduced when adjusted for geographic location and education.[72] Similarly, a Baltimore, MD study found that disparities in health between blacks and whites are attenuated when they live in similar SES and socio-environmental conditions, compared to nationally observed patterns.[73]

Needed research on segregation

Future research must seek to identify the optimal size of a geographic unit for characterizing health effects. Some evidence suggests that the size of the geographic unit matters for capturing and potentially intervening on residential factors. A study in Michigan found that large racial differences in prostate and breast cancer survival in large geographic areas (15 federal house legislative districts) were markedly reduced and sometimes eliminated in smaller geographic units (110 state house districts and 212 neighborhood areas).[74] Interestingly, there were three neighborhood areas, with high and persistent poverty for whites, where blacks had better survival than whites.

Research is also needed that gives more systematic attention to understanding the conditions under which segregation can have positive effects for particular health outcomes and social groups. There is nothing inherently negative about living in close proximity to members of one's own race. The problem of segregation is not racial composition per se but the concentration of poverty and social adversity that co-occurs with segregation given America's history of racial inequality and injustice. At the same time, there are conditions under which greater geographic concentration of one's group can have beneficial effects on health. One study found that mortality rates are lower in high density Mexican American and Cuban American neighborhoods than in low density areas[75] and a recent national study from the UK found that reported levels of discrimination among racial minorities was lower in areas of high racial density, and greater geographic concentration of one's group was associated with lower levels of mental health symptoms but not physical health.[76] Understanding the relative contribution of concentrated poverty and racial density to health is an important research priority.

Another important priority of future research is to examine the extent to which normal adaptive and regulatory systems are affected by the harsh residential environments of blacks and other minorities. It is plausible that biological adaptation to distinctive residential environments can lead disadvantaged racial and SES groups to have some biological profiles that are different from others and to distinctive patterns of interactions between biological and psychosocial factors. A study of 249 adults in Pittsburgh found that area-based measures of SES predicted brain serotonergic responsivity independent of individual SES.[77] Individuals residing in census tracts with indicators of social deprivation such as elevated rates of poverty, unemployment, and receipt of public assistance showed diminished central nervous system (CNS) serotonergic responsivity. Indices of CNS serotonergic activity are believed to play a role in trait variation in impulsiveness and aggressiveness and are related to risks of mental and behavioral disorders such as depression, substance abuse, violence and antisocial personality. Much is yet to be learned about the ways in which specific features of residential areas leads to altered biological processes that have adverse consequences on health and the extent to which such exposures are reflected in the elevated health risks of minority populations.

Other aspects of racism

Residential segregation is an institutional mechanism by which racism affects health. There are multiple other ways by which racism can adversely affect health.[78,79] More research is needed to identify the ways in which segregation and other aspects of institutional racism combine with other mechanisms of racism, such as experiences of discrimination and internalized racism to affect health.

Perceived racial discrimination is one aspect of racism that is increasingly receiving empirical attention as a class of stressors that could have consequences for health and for understanding racial disparities in health. A recent review identified 115 studies on this topic in the PubMed database between 2006 and 2008.[80] It revealed that recent studies have found an association between discrimination and a broad range of health status indicators ranging from violence, poor sexual functioning and less stage 4 sleep, to abdominal fat, hemoglobin A1c, coronary artery calcification, the incidence of uterine myomas (fibroids) and breast cancer. Studies have also found that discrimination is adversely related to health care seeking and adherence behaviors, and increased risk of multiple substances, such tobacco, alcohol and illicit drug use. Another striking pattern in the recent research is the broad range of contexts that have been considered. In addition to including all the major racial groups in the United States, recent studies have included samples from New Zealand, Australia, South Africa and immigrant groups in most European countries.

It is also noteworthy that very few studies explicitly examine the role of discrimination in accounting for racial disparities in health. Some early studies provided evidence that discrimination made an incremental contribution to SES in explaining disparities.[81] A few recent studies find that perceived discrimination accounts for some of the racial disparities in health. This is evident for Maori-European disparities on four indicators of self-reported health in a national study of New Zealand,[82] Aboriginal-non Aboriginal variations in self-reported physical and mental health in Australia,[83] and in United States studies for black–white differences in health care trust,[84] sleep quality and physical fatigue[85] and Hispanic-white differences in PTSD symptoms.[86]

Internalized racism or self-stereotyping is another mechanism by which negative stereotypes about race in the larger society can adversely affect health. Cultural racism has led to pervasive negative racial stereotypes of racial groups regarded as inferior.[79] One response of stigmatized racial populations to the societal beliefs about their biological and/or cultural inferiority is to accept as true the dominant society's ideology about them. This acceptance of negative cultural images by stigmatized groups appears to create expectations, anxieties and reactions that can adversely affect social and psychological functioning.[87,88] U.S. research indicates that when a stigma of inferiority was activated under experimental conditions, performance on an examination was adversely affected.[87] African Americans who were told in advance that blacks perform more poorly on exams than whites, women who were told that they perform more poorly than men, and white men who were told that they usually do worse than Asians, all had lower scores on an examination than control groups who were not confronted with a stigma of inferiority.[87,88] Some evidence indicates that in addition to adversely affecting academic performance, the activation of the stigma of inferiority also leads to increases in blood pressure.[89] A recent review also indicated that studies have found a positive association between internalized racism and alcohol consumption and psychological distress among African Americans.[80] In addition, internalized racism was positively associated with the risk of being overweight or abdominal obesity among black women in the Caribbean and with cardiovascular outcomes among black women in Africa.[80]

Future research needs to explore the extent to which elevated health risks are located at multiple intersections of stigmatization and discrimination. Understanding how experiences of racial discrimination relate to internalized racism and combine to affect health is also important. Self-reported experiences of discrimination must also be situated within the context of the total stress burden of respondents' lives with the recognition that racism (interpersonal and institutional) is only one source of stress. That is, understanding the potential contribution of stressful life experiences to racial disparities in health requires the assessment of perceived discrimination *and* a systematic effort to assess all of the other social, psychological and environmental (physical and chemical) stressors that respondents face. Research is also needed to identify the coping and adaptive resources that respondents use to respond to racism. One recent study using longitudinal data from the National Study of Black Americans, found that religious involvement was a health enhancing resource in the face of racial discrimination.[90] Frequency of attendance at religious services and the degree of guidance provided by religion in daily life buffered the negative effects of reports of discrimination on health. Future research needs to identify all of the resistance and coping resources used by societally stigmatized populations in responding to stressors.

Comprehensively addressing the social context

A priority for future research is to comprehensively and systematically quantify all of the risks in the social environment that differ markedly by race. For example, we need an enhanced understanding of the contribution of specific conditions in work environments to racial differences in health outcomes. Meyer *et al.*[91] show that low maternal job control and substantive complexity at work are associated with low birth weight and prematurity in the state of Connecticut. Moreover, while there is an overall beneficial effect of maternal employment on birth outcomes, maternal employment in some industries (such as textile, food service, personal appearance, material dispatching or distributing and retail sales work) was associated with increased risk of low birth weight.[92] Given the racial segregation in occupations, the extent to which occupational risks are clustered by race should be explored.

Research on labor market segmentation finds that many racial minority groups and immigrants are concentrated in job sectors with low status and low pay.[93] Environmental hazards, injury risk and occupational stress are also patterned by industry.[94] Some evidence suggests that compared to whites, minorities have higher levels of exposure to occupational hazards, greater risk of occupational injury and job-related death.[95] Inadequate attention has been given in research on racial health disparities on assessing the contribution of occupational stress and disease to the disproportionate burden of illness.

Exposure to community violence is another aspect of the social context that is not sufficiently integrated into studies of health. A recent study of women in an urban community in the northeastern United States illustrates the importance of this risk factor.[96] It found that exposure to community violence was high with 67% of the sample reporting at least one event. These exposures were consequential, with the risk of depressive and anxiety symptoms being twice as high for women who witnessed neighborhood violence. Other data reveal that neighborhood violence is one of the major chronic stressors that predicts the risk of childhood asthma and that can also interact with physical and chemical exposures to increase asthma risk.[97]

The United States has the highest incarceration rate in the world and a relatively high proportion of minority males spend time in prison during young adulthood. A history of incarceration is not typically included as a measure of stress in health studies, but recent studies suggest that this may be an important risk factor. In the National Longitudinal Survey of Youth, any exposure to incarceration as a teenager or young adult was associated with an increased risk of severe functional limitations.[98] Intriguingly, any contact with prison was more important than the amount of contact, even after adjustment for a number of controls. The CARDIA study which has followed a cohort of young adults aged 18 to 30 at baseline, also found that any time in jail during the first 3 years of the study was associated with increased risk of incident hypertension 3 years later and higher end organ damage related to hypertension.[99] Incarceration is likely to be what has been called a "disorderly transitional" stressful event in which this exposure creates role changes that are nonnormative, undesired, involuntary and sometimes irreversible.[100] Such events are critical in creating social disadvantage and placing an individual on a trajectory of low education, low job prospects, and low income and that often lead to the proliferation of other stressors. These examples emphasize the importance of fully characterizing the multiple aspects of the social context that may have health consequences.

In sum, research on the nonequivalence of SES indicators across race, the substantial differences in residential context by race, and on the multiple other ways in which race is associated with differential exposure to health risks, emphasizes that adjusting for individual and household-level measures of SES fail to fully characterize the social environment and to capture the vast number of unmeasured characteristics that differ substantially between racial minorities and whites.

Migration and health

In the 2000 U.S. Census, 67% of Asians and 40% of Latinos were foreign born.[101] Processes linked to migration make an important contribution to the observed mortality rates for these groups in Table 2. National data reveal that white, Black, Asian and Latino immigrants have lower rates of adult and infant mortality than their native born peers.[102–104] However, with increasing length of stay and generational status, the health of immigrants declines. The pattern of immigrant health where time in the United States is associated with declining health is somewhat of a paradox, since one would expect that increasing length of stay would be associated with higher SES (as wages increased and working conditions improved) and thus better health. Hispanic immigrants, especially those of Mexican background, have low levels of SES and relatively low levels of access to health insurance in the United States. However, their levels of health are equivalent and sometimes superior to that of the white population. This pattern has been called the Hispanic paradox.[105] Some research documents that poorer health is evident among second generation Latinos compared to immigrants, even though they have higher levels of SES than their first generation peers.[106]

A closer examination of the data on migration status and health reveals that the association varies by the health outcome and population subgroup

under consideration. For example, maternal mortality is higher for immigrants than for the native born across multiple populations.[107] Similarly, women of all Hispanic groups have a higher risk of low birth weight and prematurity than whites.[108] Unlike the pattern for other Latinos, recent Puerto Rican immigrants to the United States mainland have higher levels of chronic disease than island Puerto Ricans and, in contrast to the overall pattern for Asians, Chinese, Japanese and Filipino immigrants, have higher overall death rates than their native-born peers.[103] In the California Health Interview Survey (CHIS), virtually all immigrants reported better health than the native born for measures of physical health status, such as chronic physical conditions.[109] In contrast, for psychological distress, many immigrant groups (most Latino groups, Pacific Islanders and Koreans) reported worse health than the native born, while other immigrants groups (black, Puerto Rican and Filipino) had better health and still others (Vietnamese, Japanese and Chinese) did not differ from their native born counterparts. Adjusting for socioeconomic status made these pattern more pronounced.

Migration and SES

Migration status combines in complex ways with SES upon arrival in the United States and the trends of socioeconomic mobility over time to affect the trajectories of immigrant health. Immigrant populations differ in SES upon arrival in the United States. Asian immigrants have markedly higher levels of education than other immigrant groups with some Asian immigrant groups being more than twice as likely as whites in the United States to graduate from college.[10] While there is diversity among Hispanic immigrants in terms of years of education level, the largest subgroup of Hispanics, immigrants from Mexico, have low levels of education at the time of migration to the United States and face major challenges with socioeconomic mobility in the second generation. The SES of immigrants compared to the native born population of their group also varies across population groups. Analyses of CHIS data found that immigrant whites, Latinos, Asians and Pacific Islanders had higher rates of poverty than their native-born counterparts.[109] The opposite pattern was evident for blacks, and these overall patterns mask divergence for some ethnic subgroups. For Puerto Ricans, South Asians, Koreans and Vietnamese, the native born have higher poverty than immigrants. These data highlight the importance of paying attention to the particularities of each immigrant group and the ethnic diversity within the broad racial categories.

It is likely that variations in SES contribute to some of the complexities that are evident in the relationship between migration and health. First, for immigrant populations largely made up of low SES individuals, traditional indicators of SES tend to be unrelated to health in the first generation. For example, using national data from a sample of preretirement age Latino adults, Angel *et al.*[110] found that the association between SES and health varied by immigrant status. Income and education were unrelated to both self-reported physical and mental health among the foreign-born but predicted health, as expected, among the native-born. Financial assets, on the other hand, showed a stronger inverse relationship with self-reported ill health among the foreign-born than the native-born. Second, the SES of immigrants upon arrival to the United States affects an immigrant group's trajectory of health. Thus, given the low SES profile of Hispanic immigrants and their ongoing difficulties with educational and occupational opportunities, the health of Latinos is likely to decline more rapidly than that of Asians and to be worse than the United States average in the future. Consistent with this view, the gap in mortality between immigrants and the native born is smaller for Asians than for whites, blacks, and Hispanics.[103] Similarly, recent national data reveal that declines in subsequent generations in mental health were less marked for Asians than for blacks and Hispanics. For black Caribbean immigrants, the lifetime rate of psychiatric disorders increased from 19%, to 35%, to 55% for the first, second and third generation immigrants, respectively.[111] Similarly, among Hispanics, the increased prevalence of psychiatric disorders went from 24% to 30% to 43% across the three generations.[112] For Asian immigrants, there is an increase in the prevalence of lifetime psychiatric disorders from 15% to 24% from the first to the second generation, but there was no substantial increase for the third generation (26%).[113] The lifetime rate for psychiatric disorder is 31% for African Americans and 37% for whites.[114] Thus, black, Latino and Asian first generation immigrants all have lower disorder rates than the general population of blacks and whites, but by

the third generation the disorder rates of Latino and black immigrants are higher.

Third, it has been recently shown that SES is a critical determinant of variation across immigrant groups in the degree to which immigrants are selected on health. Analyses of the New Immigrant Survey found that positive health selection occurs among new legal permanent residents of the United States but varies by region of origin, with immigrants from all regions experiencing higher levels of positive health selection than immigrants from Mexico.[115] Differences in the SES of immigrant streams was the key determinant of variations in health selection by region.

Research priorities on migration and health

The available evidence on the health of immigrant populations highlights priorities in future research. Understanding the context of specific immigrant populations is important. A recent cross-national study highlights the promise of attending to the context of migration by comparing race-related risk factors and the health of Caribbean immigrants in the United Kingdom to that of Black Caribbean immigrants in the United States[116] The study found that the Caribbean English reported higher levels of work discrimination than African Americans and Caribbean blacks in the United States who reported similar levels of discrimination. Health patterns were complex. The Caribbean blacks in the United States tend to have better health than African Americans but worse health than the Caribbean English. Similarly, the pattern of worsening health beyond the age of 35 was stronger for the Caribbean English than for either African Americans or Caribbean blacks in the United States These differences in health between Caribbean immigrants in two contexts could be due to differences in the composition of the two groups of immigrants. Compared to black Caribbean immigrants in the United States, the English Caribbean group was more likely to be second generation, to have migrated under the age of 12 and before the 1970s.[116]

Relatedly, more attention should be given to identifying the role that reception factors in the new society can play in immigrants' health. A recent analysis of CHIS data for 2001 found that, in contrast to earlier research using both national and California samples, Mexican immigrants in California reported higher levels of psychological distress than their native born peers.[109] Given that the data were collected in 2001, a time of considerable anti-immigrant rhetoric and legislative proposals in California, the researchers speculate that the threat of discrimination against immigrants raised by anti-immigrant sentiment and impending policies could have adversely affected immigrants' levels of emotional distress.[109] These findings suggest that entry into a hostile climate could adversely affect the well being of immigrant groups.

Another priority for future research is to characterize all of the risk factors and resources in immigrant populations and identify how they relate to each other and combine to affect health.[117] Neglected risk factors include stressors and strains associated with migration and adaptation, inadequate health care in the country of origin and factors linked to larger social structures and context, such as institutional racism and interpersonal discrimination.[117] For example, because of challenges to socioeconomic mobility for Mexican immigrants, increasing length of stay in the United States could reflect greater exposure to blocked opportunity and thwarted aspirations, which in turn could lead to growing levels of alienation and poorer health.[118] A study of 1001 adult migrant Mexican workers in Fresno, CA assessed the association between stressors linked to acculturation and health.[118] The three aspects of acculturation stress assessed were stressors linked to discrimination, legal status and problems speaking English. The study found that acculturation stressors were inversely related to self-reported measures of physical and mental health and partially accounted for the declines in these health indicators with years in the United States In addition, acculturation stressors had a more severe negative effect on migrants who were more acculturated than those who were less acculturated.

Health researchers should also pay attention to the ways in which segregation may affect the health of black immigrants. In the 2000 Census, foreign-born blacks and Hispanic blacks experienced even higher levels of residential segregation than U.S.-born blacks,[119] primarily because they tend to reside in metropolitan areas where black–white segregation tends to be very high. The effects that this has on their exposures to health risks and their trajectories of health over time need to be examined. More generally, this raises the issue of paying attention to the extent to which variations in skin tone

within immigrant and other groups may be associated with differential exposures such as discrimination that may have health consequences. Recent research indicates that Hispanics who self-identify as black have poorer health than those who identify as white.[120]

Understanding the complex ways in which migration affects health also requires more systematic attention to the social and psychological resources that immigrants bring with them and empirical verification of their effects on health and the ways in which these resources may change over time. Social and family ties, cultural traditions, identity and religious engagement have all been identified as potential positive resources.[117,118] However, one national study found that adjusting for multiple measures of SES, both formal support (religious attendance) and informal social support were unrelated to physical and mental health for the foreign-born but predictive of health for the native-born.[110] This study also found that social support does not account for the better health of Hispanic immigrants.

There is also increasing recognition that a full understanding of the health effects of migration requires an assessment of the ways in which migration impacts the health of sending communities. A study of infant health in two high migration sending states in Mexico found that infants born to fathers who had migrated to the United States had a lower risk of low birth weight and prematurity compared to infants born to fathers who had never migrated.[121] This study also sheds light on the mechanisms. It found that women with partners in the United States had lower levels of social support and higher levels of stress during pregnancy than women with nonimmigrant partners but the positive effects of the receipt of remittances and the avoidance of adverse behavioral practices led to better infant health outcomes. The effects of migration were complex. Although women with international partners had a healthier behavioral profile in general (lower rate of smoking, higher level of exercise and a lower level of insufficient weight gain), they were nonetheless less likely to plan on exclusively breast feeding. Greater attention to the bi-directional effects of migration processes is clearly warranted in future research.

Research is also needed to understand the extent to which methodological factors are contributing to some of the observed patterns of health for immigrants versus nonimmigrants. For example, in contrast to the patterns of lower overall mortality than whites, Hispanics have higher rates of self rated ill health than whites.[41] Immigrant Latinos also report lower levels of fair and poor health than the native born.[110] Some limited evidence suggests that at least some of the observed difference between whites and Latinos, may be an artifact of the translation of the question into Spanish. In particular, the translation of "fair" (English) to "regular" (Spanish) may give that term a more positive connotation in the Spanish language than is intended in English. Research reveals that adjusting for language of interview reduces the self rated health differences between Hispanics and non-Hispanic whites.[110,122] More generally, there is a need to critically and explicitly examine the role of culture and to conceptually and empirically identify the relevant aspects of culture that may affect the health of immigrant populations.

Genetics

In the 19th and early 20th century, any and all observed racial disparities in health were presumed to reflect biological differences between racial groups.[2] This view has been shown to be problematic. Human genetic variation, including the genetic characteristics that are of interest to health researchers and clinicians, does not naturally aggregate into subgroups that match our racial categories.[3,123] It follows that any population categorization system is arbitrary and our racial categories fail to provide good sensitivity and specificity for the presence of specific genetic variants.[123] Moreover, genetic diseases that vary across population groups are rare and single gene disorders such as cystic fibrosis, Tay-Sachs disease and sickle cell that are cited as examples of the contribution of genetics to racial health disparities do not vary by continent of origin.[123]

There is a resurgence of interest in using self-identified race to capture at least part of the genetic differences between population groups. This has been fueled in part by research that indicates that data from multiple loci on the human genome can provide fairly accurate characterization of individuals into continental ancestral groups that approximate our current racial categories.[124] Data on "continental ancestry" have been used to suggest that there is value in race as a biological category. In fact, Risch *et al.*[124] concluded that "the greatest genetic structure that exists in the human population occurs

at the racial level." However, Serre and Pablo[125] have reanalyzed data from the largest study of human genetic variation to date, and have shown that sampling biases play a key role in conclusions about the degree of continental clustering of populations. They found that when individuals are sampled from around the world in a way that reflects the geographic distribution of humans across continents, the human gene pool does not consist of continental clusters, but reflects gradients of allele frequencies across the world. This absence of major discontinuity in genetic traits is consistent with earlier research showing that most genetic variation is found among individuals and not among population groups. They concluded that "most alleles influencing susceptibility to disease or outcome of medical interventions cannot be expected to show significantly different frequencies between 'races'."[125] Thus, although genetic markers can uniquely identify most individuals, variation in biological characteristics is not inherently structured into meaningful "racial" categories at the population level and identifying ancestry provides very limited direct information regarding whether an individual carries specific genetic traits.[3,126] It has also been noted that there is a growing trend in health disparities research to keep the logic of genetic racial differences intact but to substitute the language of "ancestral background" for the language of "race."[127]

The continuing misuse of genetics

Deeply entrenched ideas die very slowly. A 2005 national survey of U.S. physicians found that 81% of physicians believed that race should be used as a biological basis for determining diseases and 85% indicated that drugs targeted toward specific ethnic and racial groups may have therapeutic advantages.[128] Not surprisingly, the research literature provides many examples of the misuse of genetics in an effort to understand racial disparities in health. We briefly highlight a few examples. A 2000 study in the journal *Demography* claimed to examine the contribution of genetic and environmental factors to racial differences in low birth weight.[129] The authors used self-identified race as markers of both genetic and social factors. They argued that racial categories are genetic entities because "generations of 'reproductive isolation' have led to differences in gene frequency across racial groups." However, their quantitative genetic approach used self-identified race as the only marker of genetics, and the authors indicated that their analysis assumed that "genetic and environmental factors do not correlate or interact." There are detailed critiques of the limitations of this approach.[130,131]

A 2004 paper in the *New England Journal of Medicine* provided a rationale for studying BiDil only in blacks in the A-HeFT trial, by indicating that there were racial differences in the prevalence, risk factors, causation, disease severity, outcomes and response to therapy between black and white patients with heart failure.[132] Accordingly, the authors argued, studying only blacks would avoid the "substantial variation in genetic and environmental factors that influence disease progression and the response to therapy." They hypothesized that blacks were biologically different from whites due to lower levels of nitric oxide in the blood or other unspecified biological characteristics. Importantly, the study made no effort to measure these characteristics but used self-identified race as the marker of genetics.[127] Another example of the misuse of genetics is a 2001 *New England Journal of Medicine* study that claimed to demonstrate a greater response to angiotensin-converting enzyme inhibitor therapy in white compared to black patients with left ventricular dysfunction.[133] In his detailed critique of this study, Kaufman[134] notes that it has been highly cited as evidence for a differential response to therapy due to innate differences in physiology between blacks and whites. However, he shows that the analyses were flawed, violated basic principles of causal inference and provided no analysis of genetic variants. Accordingly, this paper does not provide support for the notion that genetic factors are responsible for racial differences in response to therapy.

The slavery hypothesis[135] is a final example of the misuse of genetics in contemporary health disparities research. Using a model of genetic determinism, it argues that the elevated rates of hypertension for blacks compared to whites in the United States are due to the selection of a genetic trait that occurred during the capture of slaves in Africa and their journey to the New World. African American hypertension is viewed as a response to sodium due to the selective effects of heat stress, salt and water deprivation during the transport of African slaves that created a genetic bottleneck of black people in the United States with distinctive genetic traits. A critical review of this hypothesis shows that it is

inconsistent with the processes of population genetics, historical data on the scarcity of salt in Africa and on the level of mortality during the slave trade as well as on the proportion of mortality attributed to diarrhea.[136]

Detailed methodological critiques have been published for all of the examples included here but although helpful, they appear to be having limited impact on the field because much of the thinking on race and genetics is driven consciously or unconsciously by assumptions or ideology that tend to view racial groups as valid biological categories.[126,127]

Limits and opportunities: genetic research on race and health

The idea that genes alone will make a major contribution to understanding racial disparities in health is not consistent with the available evidence.[137] First, there are differences in genetic susceptibility in human populations but they are unlikely to be strongly patterned by race.[123] The genetic alleles that predict susceptibility to common diseases tend to be present in many populations, although at varying rates. Second, many genes are likely to be involved in most common chronic illnesses and each gene is likely to have many variants.[138] Thus, the chronic diseases that make a large contribution to the burden of disease in contemporary society are seldom driven by any single risk factor, whether genetic or environmental. Third, the overall contribution of genetics to population health is likely to be modest. A review of the more than 300 replicated associations that have been identified for more than 70 diseases from genome-wide association studies concluded that their effect on disease risk is very modest, with an identified variant typically increasing disease risk by 10 to 30%.[139] Thus, genetic effects are small and hard to detect and there are few examples of gene-environment interactions for modest gene effects or small environmental effects.

Does this mean that research on genetics has no place in the study of racial disparities in health? We think not. Instead, we call for more research on the potential contribution of genetics to population health and provide broad guidelines for the needed research that seeks to understand how social exposures combine with biology to affect patterns of disease. Race remains an important social category in contemporary society.[1] It is strongly related to many environmental factors and racial groups are likely to differ on a broad range of environmental risks and exposures. Given the extent to which a broad range of social, behavioral, nutritional, psychological, residential, occupational and other variables vary by race, race is a crude category that likely reflects simultaneous confounding for unmeasured genetic *and* environmental factors.[137] Given that biology is not static but is adaptive to the environmental conditions in which the human organism exists, it is important for research to assess potential interactions between the social environment and both innate and acquired biological factors.

First, conclusions about the contribution of genetics should be based on direct tests of genetic traits. Since an individual's race is of very limited use as an indicator of genetic traits, directly measuring the relevant gene is the reliable method for assessing genetic risk.[123,125,140] The underlying biological mechanisms of disease sometimes vary across race[141] and differential treatment effectiveness is sometimes found across racial groups.[142] Residual racial differences in biological processes after SES is adjusted for, are often interpreted as due to innate genetic differences.[123] However, racial differences in observed biological factors do not necessarily reflect racial variation in genotype. Given the marked differences in environmental conditions noted earlier, they could also reflect differences in unmeasured environmental factors, gene expression, and other biological adaptations to the environment, or some combination of these factors. Researchers should also pay attention to variation within race, as well as, differences across racial groups. For example, a meta-analysis of 15 clinical trials found that blacks and white patients differ in their responses to antihypertensive medications with white patients responding better to beta blockers and ACE inhibitors and blacks responding better to diuretics and calcium channel blockers.[143] However, the variation within each race was three to four times the size of the average racial difference with almost 90% of blacks and whites having similar responses to all antihypertensive medications. Thus, in contrast to conventional medical wisdom, a patient's race provides little guidance to the clinician in the choice of antihypertensive medication. The bottom-line is that a genetic explanation of residual race effects conflates self-reported racial status with inferred genetic traits and neglects evidence that indicates that

exposures to social factors over the life course can increase risk for disease.[144]

Second, gene-environmental interactions are central to understanding the role of genetics in disease. Even in the cases of single gene disorders, the severity and timing of genetic expression are affected by environmental triggers, and known genetic risks can be exacerbated or become protective because of specific environmental exposures.[138,145] Intriguing patterns of interaction between genetic factors and psychosocial risk factors have been identified that could shed light on the patterning of disease by race.[146] A recent study of 590 participants from the 2004 Florida Hurricane Study found that two aspects of the macro-social environment (county level crime rate and unemployment rate) both interacted with a specific genetic trait (5-HTTLPR) in predicting PTSD risk.[147] Similarly, analysis of data for adolescents in the Add Health Study found that genetic traits interacted with family processes (e.g., daily family meals), school processes (e.g., repeating a grade) and friendship network variables (e.g., friend delinquency) to predict delinquency and violence among male adolescents.[148]

Olden and White[138] have reviewed the tools and databases that provide new opportunities and a new paradigm for studying how genes and the environment combine to affect health. Racial and SES groups may face differential social, physical, and chemical exposures in residential and occupational environments that could interact with susceptibility genes to affect the risk of disease. Socially disadvantaged populations may also have higher levels of particular genetic variants that could increase their vulnerability to the environmental exposures that they face. Research to date has also given scant attention to the extent to which genetic effects may vary across SES groups or for social categories that reflect the simultaneous consideration of race, SES, and gender. Socioeconomic groups often occupy different occupational and residential contexts. Ellis' review[149] suggests, for example, that genetic and other physiological factors play a larger role in adult SES (education and earning levels) than is generally recognized. These deserve serious empirical examination.

Research on racial disparities in health could also benefit from the methodological and conceptual recommendations that have been made to advance future research on gene-environment interactions in general.[146,150] One of the key recommendations is the need for careful replication and meta-analyses. Given the large number of variants examined the probability of chance findings is high. Over the last decade, genetic association studies have created "one of the largest bodies of irreproducible results yet accumulated."[137] A meta-analysis of 370 studies that examined 36 genetic associations for various diseases found that the first published study typically suggests a stronger genetic effect than is found in subsequent studies.[151] Sampling biases are potential contributors to this pattern. There has been particular interest in interactions between the serotonin transporter gene (5-HTTLPR) and stress in affecting depression but a recent meta-analysis of 14 studies that examined the interaction between this genotype and stressful life events in predicting depression found that although stress is associated with an increased risk of depression, there was no interaction evident between the genetic trait and stress.[152] However, the studies varied in sample designs, measures of both stress and depression and analytic approaches. Relatedly, research on race and genetics (and in other areas of health researcher more generally), should exercise great caution in making generalizations and inferences to larger populations when the analyses are based on data from nonrepresentative samples.

Ramos and Rotimi[153] recently provided examples of the potential of genomics to address racial disparities in health through treatment and prevention. They show how recent comprehensive research identifying a genetic variant that is associated with an increased survival rate against cardiac failure and ischemia,[154] has shed important new light on the confusing data on racial differences in response to beta blockers. The study revealed that only about 40% of African Americans carry this protective variant and the majority of blacks who do not carry it will benefit from the use of beta blockers in the treatment of heart failure. Ramos and Rotimi[153] also describe how research that has identified genomic regions that contain risk variants for prostate cancer holds promise for increasing our understanding of the markedly elevated risk of this disease in African Americans. These susceptibility variants for prostate cancer exists in many populations but the estimate of the population attributable risks (the proportion of cases that would not occur if this risk factor were not present) ranges from 32% in whites to 45% in

Native Hawaiians, 46% in Latinos, 60% in Japanese Americans and 68% in blacks.[155] Importantly, in both of these examples, although the genetic variant of interest is differentially distributed by race, racial group membership is an unreliable predictor of its presence.

Finally, given the distinctive environments of racial minorities in the United States, more systematic attention should be given to identifying potential epigenetic effects. Epigenetics refers to changes in the patterns of gene expression in the absence of changes in the nucleotide sequences of the DNA. While variation in genotype is unlikely to play a major role in accounting for disparities, differences in gene expression linked to occupancy of different environmental contexts could play a critical role. Research on the role of genetics in social disparities in health has mistakenly emphasized gene frequency over gene expression.[126] Although racial group membership and continental origin are poor predictors of genetic variation, they are strongly related to distinctive patterns of social exposures that can produce epigenetic changes in gene expression and tissue and organ function.[144] Recent research has highlighted the potential of the social environment to produce epigenetic changes. For example, by analyzing the brain tissue from adults who had committed suicide one study found genetic changes in those who had been abused as a child.[156] That is, hippocampal gene expression was decreased in victims of child abuse compared to controls with no abuse. Another recent study found changes in prostate gene expression among men with low-risk prostate cancer in response to lifestyle changes in diet, exercise, stress management and social support.[157]

Medical care

Much of the national health policy focus around social disparities in health in the United States has been on initiatives seeking to improve access to and the coverage of medical care, as well as, addressing the quality and intensity of health care. Researchers studying social inequalities in health tend to emphasize, in contrast, that medical care is a relatively small contributor to the overall health status of the population and to social inequities in health. These researchers note that inadequacies in medical care account for only about 10% of the variation in health status,[158] medical care has contributed relatively little to improvements in health in the last 250 years,[159] and most health problems occur long before individuals get to their health care provider or hospital. In contrast, we argue that the contribution of medical care to racial and SES disparities in health deserves renewed examination and research.

First, even if medical care has only a limited role as a determinant of health, it is nonetheless a health enhancing resource that is socially patterned. Table 7 shows national data on health care access and usage.[160] There are large racial differences in insurance coverage. In the pre-retirement years, Hispanics and American Indians are much less likely than whites, blacks and Asians to have any health insurance. The reduced access of many racial minorities to educational and employment opportunities noted earlier leads them to be overrepresented in poor quality jobs that do not provide health insurance. Some persons who do not receive employer-based insurance are eligible for public (means-tested) insurance coverage. Table 7 also shows that blacks, Hispanics and American Indians under the age of 65, are more likely than whites and Asians to have public insurance only. Moreover, for all racial groups, there is a strong positive relationship between having insurance and income and an inverse association between income and public insurance. However, there are substantial racial variations in insurance coverage at every level of income.

A dental care visit in the past year is one indicator of health care usage. Strikingly, Table 7 shows that the use of dental services is relatively low in the United States for persons of all racial and income groups. However, use of dental care is strongly patterned by both race and income with persisting racial gaps in dental care at every level of income. A similar pattern is evident among adults age 50 and over who received any colorectal screening test (colonoscopy, sigmoidoscopy, proctoscopy or a fecal occult blood test) within the prior 2 years. Table 7 also provides one indicator of unmet medical need. Whites were less likely than all other racial groups to indicate that during the past year, they sometimes or never got care as soon as they wanted for an illness, injury, or condition. Large variations were also evident by income. For both blacks and whites, the gaps by income were larger than the overall racial gap. Instructively, Asians had the highest level of unmet medical need and unmet medical need did not

Table 7. Health care access and usage by race and income

Race	All	Poor	Near Poor	Mid Income	High Income
% People under age 65 with any health insurance, 2006					
White	87.5	74.6	75.0	87.6	94.5
Black	82.5	77.7	76.6	84.7	92.6
Hispanic	65.0	55.7	56.1	71.4	85.0
Asian	85.0	74.8	71.8	84.3	93.2
American Indian	62.0	60.7	48.9	63.7	80.5
% People under age 65 with public insurance only, 2006					
White	24.2	68.7	49.9	20.2	8.5
Black	46.8	80.4	55.9	29.8	14.7
Hispanic	59.4	89.1	70.9	41.0	21.0
Asian	27.4	64.8	50.4	25.9	10.0
American Indian	62.9	87.3	81.8	45.2	25.2
% People with dental visit in calendar year, 2005					
White	49.5	32.3	34.9	45.8	60.0
Black	30.4	22.9	25.2	32.2	42.4
Hispanic	27.8	22.9	20.8	29.5	43.1
Asian	41.0	24.2	31.8	39.1	49.0
American Indian	32.6	–	–	–	–
% of adults age 50 and over with a colorectal screening test in past 2 years, 2005					
White	58.5	43.1	51.4	57.3	64.7
Black	48.5	38.8	46.0	49.0	63.6
Hispanic	37.3	30.7	34.5	36.5	46.6
Asian	42.4	–	37.2	33.0	56.6
American Indian	38.1	–	–	–	–
% of adults with unmet medical need in last 12 months, 2005					
White	12.8	22.8	19.0	11.8	8.6
Black	20.9	30.1	23.9	18.8	11.0
Hispanic	17.7	18.9	18.1	16.0	18.1
Asian	27.4	–	–	–	–
American Indian	–	–	–	–	–

Note: Agency for Health care Research and Quality.[160]
Both the black and white categories exclude Hispanics; – = data not available.
Poor = below federal poverty level (FPL); near poor = over FPL but less than twice the FPL; middle income = more than twice FPL but less than 400% FPL: high income = 400% FPL or more.

vary by income for Hispanics. Access differences linked to language and cultural barriers are likely contributors to this pattern.

Second, some evidence suggests that medical care that is prevention oriented and seeks to address the social determinants of health along with the delivery of clinical services can play an important role in reducing disparities in health.[161] Moreover, for at least some health status indicators, medical care may have a greater positive effect on socially disadvantaged populations than on their more advantaged counterparts.[159] Research reveals that primary care is associated with better overall health and with smaller SES and racial disparities in health.[162] Specifically, health is better in areas with more primary care physicians and for persons who have primary care physicians as their usual source of care. Blacks in the United States are two to three times more likely

than whites to have diabetes-related lower extremity amputations, a disparity that is not evident in black–white comparisons in London. It has been suggested that the more primary care oriented British health system has eliminated this disparity.[162]

International comparisons also support a critical role for primary care in population health. Costa Rica has a relatively good health profile compared to other countries and some evidence suggests that this country's introduction of integrated primary care teams may be a key contributor to its positive health profile.[162] Similarly, although Cuba is a poorly performing country in economic terms, it has health outcomes that are similar to those of the highest income countries in the world, and markedly better than those of countries with similar economic status. The delivery of primary care may well be the explanation of Cuba's paradoxical status. Cuba has the highest physician-to-population ratio of any country in the world, and Cuba's doctors address medical and nonmedical determinants of health in their delivery of care to the entire population of specific, geographically defined areas.[163]

Research is needed to provide a clearer understanding of the conditions under which particular health care systems and specific aspects of care can combine with social factors to affect social disparities in health. Social disparities in health exist for the onset of illness, as well as, for the severity and progression of disease. It is likely that primary, preventive care, along with social factors outside the health care system can play a key role in reducing the onset of disease. However, the provision of health insurance to socially vulnerable populations that is not accompanied by the reduction or elimination of other system barriers to care, can result in the persistence of high levels of unmet medical needs. System barriers are characteristics of the health care system that can serve as a deterrent to use such as fragmentation of services, distance, waiting time, appointment scheduling, availability of transportation and poor provider communication. Recent U.S. data document that although the proportion of kids uninsured declined as a result of the expansion of children's health insurance between 1998 and 2006, the proportion of kids reporting no usual source of care increased, and not having a usual source of care was a strong determinant of unmet medical need.[164]

Disparities in the course of disease are sometimes larger than the disparities in the incidence of illness.[165] Thus, timely and appropriate therapeutic care can make a major contribution to reducing social disparities in the progression of disease. Considerable evidence suggests that there are large racial differences in the quality and intensity of medical care with blacks and other minorities receiving fewer medical procedures and poorer quality medical care than whites even after SES is considered. An Institute of Medicine report documented a pervasive pattern of racial differences in the quality and intensity of care that persisted even when even when differences in health insurance, SES, stage and severity of disease, co-morbidity, and the type of medical facility were taken into account.[166] It is likely that similar patterns exist by SES, but since racial identifiers are much more likely to be on patient records than SES, our knowledge of SES differences in the quality and intensity of care is more limited.

There are suggestions in the literature that medical care can play an important role in reducing racial differences in the severity and progression of disease. For example, compared to whites, Hispanics and Chinese, African Americans show an accelerated disease process in the development of congestive heart failure (CHF) suggesting that the aggressive control of hypertension and diabetes could prevent incident CHF in blacks.[141] Recent research suggests that although there has been progress in increasing awareness in the black community regarding hypertension and getting blacks into treatment, major challenges persist with regards to the effectiveness of treatment and blood pressure management. National data reveals that compared to whites, blacks are more likely to have hypertension, more likely to be aware of their disease, more likely to be pharmacologically treated, but less likely to achieve control of blood pressure, given treatment.[14] These differences could reflect racial differences in the quality of care and/or differences in exposure to stressors that could affect blood pressure control.

Towards an integrative science of the determinants of disease

We need new integrative, life course and intergenerational scientific models that will seek to understand how the accumulation of social adversities and resources can alter biological processes, including gene expression, to affect health.[144,167] Given that high levels of stress in early childhood can alter

biological structures and processes and lead to increased risk of disease in adulthood, these models must pay particular attention to early childhood exposures.[167] This will include giving adequate attention to capturing the role of socioeconomic adversity over the life course. Carson *et al.*[168] found that a cumulative measure of life course SES that captured childhood, young adulthood (age 30) and older adulthood (ages 45–64) SES was associated with greater subclinical arthrosclerosis for both blacks and whites. A recent national study found that among women who spend their childhoods in or near poverty, upward social mobility was associated with improved birth outcomes for white but not black women.[169] We do not know the extent to which this finding reflects differences in the effects of early childhood adversity across race or whether early childhood adversity among African Americans is associated with a larger concentration of other negative risk factors than the presence of childhood poverty among whites. Other research indicates that childhood SES accounts for part of the racial disparities in adult health. A recent study found that childhood SES as captured by father's occupation, childhood health and height accounted for a part of the increased risk of first stroke for blacks compared to whites.[170] This association was independent of adult SES (income, education and occupation, and wealth) and CVD risk factors. Thus, increasing evidence points to the need to capture exposure to health risks over the life course and to understand the contribution of early life SES to disparities in adult health.

Multiple forms of stress and other risk factors tend to co-occur in individuals, and individuals and households who are at elevated risk are often clustered in geographic space. Multidisciplinary research teams are needed to capture the full range of exposure to acute and chronic chemical, physical, biological and psychosocial stressors and model how they can combine in additive and interactive ways to affect health. Attention should be given to the ways in which adversities and resources cumulate over time and over generations. Recent research suggests that there is value in this approach. For example, research on maternal low birth weight (LBW) shows how conditions early in a woman's life can have a long reach that affects outcomes for the next generation. Collins *et al.*[171] found that maternal LBW is a risk factor for infant LBW even after adjusting for age, education, marital status and prenatal care. This pattern was evident for both blacks and whites. However, given the high prevalence of LBW among blacks, this effect likely has a larger impact on LBW among black infants. This research also suggests that prenatal development and growth may be a critical period that has life-long consequences. Almond and Chay[172] provide another example of intergenerational effects. They compared black women born in 1961–1963 with those born 1967–69. This latter group had benefited from the economic gains and greater access to medical care that were attendant to the Civil Rights movement and other social policies of the 1960s. They found that black women born in 1967–69 had lower risk factors as adults and were less likely to have infants that were LBW and had low Apgar scores.

Exposures to social adversity over the life course and over generations ultimately affect health through biological processes, including genetic ones. Kuzawa and Sweet[144] have recently provided a detailed description of an epigenetic model in which prenatal nutritional deficits and/or psychosocial stress can combine with early childhood exposures to produce epigenetic changes in the patterns of gene expression that can contribute to the elevated risk of CVD in adult African Americans. They have also described several pathways by which the effects of these epigenetic changes can be perpetuated across generations. This model is an example of the needed integrative scientific approaches that will require increased collaboration between geneticists and social scientists, and that hold enormous promise for advancing our understanding of the determinants of group variations in disease.

Racial and SES disparities exist across the continuum of disease and the needed research needs to give greater attention to identifying both the determinants of disparity and the optimal intervention strategies at each specific point of the disease continuum. A study by Wong *et al.*[173] examined the contribution of the risk factors that predicted disparities in cancer incidence, stage at diagnosis and survival after diagnosis to disparities in life expectancy. It found that across all cancers combined, differences in cancer incidence contributed more to racial differences in cancer mortality than the differences in cancer stage or survival after diagnosis. For example, for lung cancer among men, incidence accounted for

almost half of a year difference in life expectancy—that is more than the impact of stage and survival after diagnosis of all cancers combined. This has important implications for the design and targeting of interventions for lung cancer. It indicates, for example, that smoking cessation may be an especially effective strategy to reduce racial disparities in cancer mortality among men. A similar pattern of the primacy of social risks to the onset over the course of disease may not be evident for other health conditions and future research needs to attend to the particular patterns that emerge for specific diseases. Research also needs to identify the factors that can affect the success of particular intervention strategies across social groups. For example, although attempts to quit smoking are comparable across racial groups, whites are markedly more likely to be successful quitters than blacks, Latinos, and Asians.[56] While attempts to quit did not follow an SES gradient, success at quitting did. This pattern was evident for most racial groups. Other research indicates that when smoking cessation programs for blue collar workers are integrated with efforts to reduce job-related health and safety hazards, workers are more likely to quit than if offered only a smoking cessation program.[174]

Research on reducing social disparities in health should also identify the critical targets for intervention and the particular indicators that are likely to maximize potential intervention effects. Schulz *et al.*[175] show that income affects risk factors for CVD (such as waist circumference, BMI and cigarette smoking) through a chain of effects to which psychosocial stress and psychological distress contribute. Even in a sample with a relatively restricted range of income, they found that even modest increases in income could lead to a "cascade of effects" that could trigger improvements in CVD risk at the population level. Future research needs to replicate these cross-sectional findings and identify the key leverage points for intervention and the target indicators that are likely to produce cascading effects.

Conclusions

The research reviewed indicates that social disparities in health are large, pervasive, and persistent over time. These inequalities in health reflect larger inequalities in society. Race, SES, and gender all matter for health separately and in combination. We need to better understand what happens when these social statuses interact. Moreover our review reveals that the patterning of health status and risk factors for males versus females is neither simple nor straightforward. We need research that elucidates how risks and resources linked to multiple social statuses and social roles can create particular patterns of living and working conditions that combine over time to affect health. Our review also reveals that individual level indicators of SES do not fully capture lifetime exposure to social adversity or social advantage for racial groups. Understanding the joint effects of race and SES will require greater attention to comprehensively quantifying risks in the social and physical environment and understanding how they interact with biological mechanisms to affect health. Importantly, fully understanding the biological processes shaping health disparities will require attention to social risk factors. As research on the human genome moves forward, there will be an increasing need for comprehensive, detailed and rigorous characterization of the risk factors and resources in the psychological, social and physical environment that may interact with biological predispositions, and alter gene expression, to affect patterns of health risk.

A focus on biology is not inconsistent with attention to the upstream causes of disease. Kuzawa and Sweet,[144] for example, argue that a comprehensive and integrative epigenetic model of health disparities emphasizes that life course interventions on social and economic factors are the key to effectively addressing social disparities in health. Addressing the developmental and epigenetic processes that shape adult health points to the need for large scale interventions that need to be implemented over the life course that seek to increase access to social and economic resources that can buffer women of childbearing age from social and economic stress and inadequate nutrition. These interventions could include expanding government programs to provide adequate nutrition, ensuring that all workers have paid sick days for their own health and adequate parental leave, and increasing the extent to which both neighborhoods and workplaces are healthy environments.[176]

A recent study highlights the urgency of addressing social inequalities in health. It documents that declines in overall cancer mortality between 1993

and 2001 mainly reflects declines among the highly educated.[177] For example, for lung cancer—the leading cause of cancer deaths in the United States—for both blacks and whites, men and women, each higher education level experienced a progressively steeper decline. The persistence and magnitude of social inequalities in health requires renewed attention to identifying the modifiable environmental factors in the places where individuals and groups live, learn, work, play and worship that can be manipulated to reduce social disparities in health.

Finally, efforts to reduce social inequalities in health in the United States must be combined with efforts to improve the health of all social groups. Throughout this paper, whites and high SES groups have been the standard of comparison for the health of more socially vulnerable groups. Recent reports from the Robert Wood Johnson Foundation Commission to Build a Healthier America show that whites in the United States and the college-educated in almost every state, fall below an achievable national benchmark of both child[178] and adult health.[179] Thus, even the most advantaged residents of the United States have less than optimal health, with even more dramatic shortfalls for many racial minorities and low SES groups. Thus, we need a new national commitment to improve the health of all that would also give specific emphasis to improving the health of the most vulnerable, more rapidly than that of the overall population, so that social inequalities in health can ultimately be eliminated.

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Conflicts of interest

The authors declare no conflicts of interest.

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