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Understanding Associations between Race, Socioeconomic Status and Health: Patterns and Prospects

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Racial and ethnic stigmatized peoples experience higher rates of illness, impairment and death than the average of their societies in the U.S. and globally (Williams 2012). Across multiple health outcomes, these disparities are also seen in the earlier onset of illness, more severe disease and poorer quality of care for racial/ethnic minorities compared to their majority peers. Socioeconomic status (SES), whether measured by income, education or occupational status, is among the most robust determinants of variations in health outcomes in virtually every society throughout the world (WHO Health Commission 2008). Understanding the complex ways in which race, ethnicity and SES uniquely and in combination, influence health outcomes is thus a critical task in addressing disparities across the socioeconomic spectrum and among racial/ethnic groups (Williams, Mohammed et al. 2010). This commentary considers empirical evidence regarding these complexities and argues for greater attention to understanding risks and resources in the social environment that are linked to race, ethnicity and SES, and to how they combine and accumulate together with innate and acquired biological factors across the life-course, to influence cardiovascular disease and other health outcomes.

Historically, the categorization of social groups into races has reflected oppression, exploitation and social inequality (American Sociological Association 2003). The identification of specific racial categories over time has been primarily driven by historical circumstances and social and political factors. For example, since the first federal census in the U.S. in 1790, racial categories have changed with every national census (Nobles 2000). For brevity in this paper we will use “race” to refer to both the racial and ethnic categories that the U.S. Government’s Office of Management and Budget requires all Federal agencies to use. In doing so we consider “race” as primarily a social category that encompasses what

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is commonly referred to as ethnicity - common geographic origins, ancestry, family patterns, cultural norms and traditions, *and* the social history of specific groups (Williams, Mohammed et al. 2010).

Biological and genetic explanations of race, and of differences in health between racial groups, dominated in the 19th and early 20th century (American Sociological Association 2003), and still persist among many today (Williams, Mohammed et al. 2010). However human genetic variation does not naturally map into the subgroups that correspond to our racial categories, and most genetic variation is found among individuals and not among population groups. However, although racial categories are not good indicators for biological variation within the human population, considerable research documents the salience of race as a social category, and that existing racial categorization has considerable social consequences (American Sociological Association 2003).

Making sense of "racial" health disparities requires careful consideration of multiple issues captured by racial categories. "Race" reflects simultaneously unmeasured confounding for biological factors linked to ancestral history and geographic origins, and environmental exposures (Williams, Mohammed et al. 2010). These environmental factors include the current psychological, social, physical and chemical environment, as well as, exposures over the life-course and across generations, and biological adaptation to these environmental exposures, including gene expression (Williams, Mohammed et al. 2010). For example, some researchers have historically sought to explain the large black-white differences in hypertension in the U.S. in terms of genetics. In contrast, an internationally comparative study of hypertension among West Africans in Africa and persons of West African descent in other contexts, found a stepwise increase in hypertension as one moved from rural to urban Africa, to the Caribbean and then to the U.S., with persons of African descent in the U.S. having hypertension levels that were twice as high as blacks in Africa (Cooper, Rotimi et al. 1997). Instructively, whites in the U.S., have higher rates of hypertension than blacks in Africa, and other comparative data reveal that the prevalence of hypertension among African Americans in the US was higher than those in some predominantly white populations in Europe such as Sweden and Italy but lower than those in other European countries such as Germany and Finland (Cooper, Wolf-Maier et al. 2005). These data highlight the potential of social, cultural and environmental factors to predict variation in hypertension risk.

SES is a complex and multi-dimensional concept comprising a range of factors encompassing economic resources, power and/or prestige that can influence health at different times in the life course, at different levels (e.g. individual, household, neighborhood), and via different pathways (Braveman, Cubbin et al. 2005). Table 1 shows that all indicators of SES are strongly patterned by race. Asians, a group heavily made up of immigrants, have a high SES profile. Levels of college graduation are almost twice as high for whites compared to blacks and Hispanics. Other data reveal that Pacific Islanders (15%) and American Indian/Alaskan Natives (13%) also have lower levels of college graduation than whites (US Census Bureau 2010). Data on median household income tell a similar story. Asians have the highest median household income in the U.S. but Hispanics earn 70 cents and blacks earn 59 cents for every dollar of income that whites receive. However,

income differences markedly understate racial differences in economic status. Net worth, a measure of wealth, captures the economic assets and reserves that a household has. Wealth facilitates the ability of a household to both plan for the future and cushion shortfalls in income. Racial differences in wealth are striking. For every dollar of wealth that whites have, Asian households have 83 cents but blacks have 6 cents and Hispanics have 7 cents (US Census Bureau 2014).

These racial differences in SES are an important contributor to racial disparities in health. However, racial disparities in health typically persist, although reduced, across all levels of SES (Braveman, Cubbin et al. 2010; Williams, Mohammed et al. 2010). Table 2 illustrates this complexity by presenting racial and educational variations in heart disease mortality for men and women (Jemal, Ward et al. 2008). Among men, death rates from heart disease are almost twice as high for blacks compared to whites. These racial disparities are smaller, absolutely and relatively, than the education gaps in heart disease mortality, within each race. At the same time, at every level of education a racial gap persists, with the absolute gap similar at the lowest and highest education level and the relative gap wider for college graduates than for those who did not complete high school. Death rates from heart disease are lower for both black and white women than for their male peers but the patterns of disparities by race and education among women are generally similar to those found for men. Heart disease mortality is almost three times higher for black than white women, with the death rates for the lowest education category compared to college graduates being twice as high among black women but almost six times as high among their white counterparts. The persistence of a racial difference in heart disease mortality at every level of education is also evident among women, with the racial gap, absolutely and relatively, being larger at the higher levels of education than at the lowest level. Interestingly, for both men and women, the largest absolute gap in mortality is evident among high school graduates. Further evidence of the added effect of race after SES is accounted for is documented by a 23–25 year follow-up study of medical graduates that found Black physicians, compared to their White peers, had a higher risk of CVD (RR=1.65), earlier onset of disease, higher incidence rates of diabetes and hypertension (twice as high), higher incidence of coronary artery disease (1.4 times) and higher case fatality (52% v 9%) (Thomas, Thomas et al. 1997).

Clearly, race and SES, although related, reflect distinct processes of stratification with each likely to be a proxy for specific exposures that can affect health outcomes (Williams, Mohammed et al. 2010). Research indicates that there are at least four reasons why race still matters for health after SES is considered. Firstly, in addition to being influenced by current SES, health is also affected by exposure to adversity throughout the life-course. Early life adversity, such as poverty, abuse, and traumatic stress, vary by race and SES, and has been shown to influence multiple indicators of physical and mental health later in life, including cardiovascular, metabolic and immune function (Shonkoff, Boyce et al. 2009). An enhanced understanding of the ways in which early life SES and other forms of adversity contribute to adult health disparities, including the psychosocial and physiological pathways, as well as contextual-level influences such as neighborhood factors, is needed. In particular, we need to identify the extent to which childhood poverty among racial minorities is concurrent with a greater concentration of other negative risk factors than among Whites and how multiple

types of stressors combine at various points across the life course and accumulate to influence later health disparities (Williams, Mohammed et al. 2010).

Secondly, race matters to health disparities due to the non-equivalence of SES indicators across racial groups. Compared to Whites, Blacks and Hispanics receive less income at the same education levels, have markedly less wealth at equivalent income levels, and have less purchasing power due higher costs of goods and services in the residential environments where they are disproportionately located (Williams, Mohammed et al. 2010). One national study illustrated how the minority poor are poorer than the White poor. It found that African Americans were more likely than Whites to report six economic hardships, such as being unable to pay full rent and having one's utilities shut off, even after adjustment for multiple demographic, SES, and health status factors that presumably should have accounted for them (Bauman 1998).

Thirdly, arguably the most critical distinctive social exposure experienced by racial minorities is the added burden of racism. Discrimination across both institutional and interpersonal levels remains pervasive in contemporary societies (Pager and Shepherd 2008). Residential segregation by race, an example of an institutional racism, has created racial differences in education and employment opportunities which produces racial differences in SES. In addition, segregation is a major determinant of racial differences in neighborhood quality and living conditions including access to medical care (Williams and Collins 2001). A study of the 171 largest U.S. cities concluded that there is not even one city where whites live under equivalent conditions to blacks and that the worst urban context for whites is better than the average context of blacks (Sampson and Wilson 1995). Although segregation is increasing for Hispanics, the segregation of African Americans remains distinctive. For example, an analysis of national data revealed that middle class blacks are more segregated than poor Hispanics and Asians (Massey 2004). Self-reported experiences of discrimination have been associated with a broad range of disease outcomes, preclinical indicators of disease (e.g. inflammation, visceral fat) and health risk behaviors (Lewis, Cogburn et al. In Press). As with other forms of early life adversity, discrimination is particularly detrimental to children, with associated poor health outcomes reported in childhood and adolescence increasingly documented internationally (Priest, Paradies et al. 2013). Institutional and cultural racism can also harm health through stigma, stereotypes and prejudice all of which can contribute to stunted socioeconomic mobility and reduced access to a broad range of societal resources and opportunities required for health (Williams and Mohammed 2013a).

A fourth reason why there are racial differences in health after SES is controlled is that racial minorities have elevated risk of exposure to a broad range of psychosocial stressors. Institutional discrimination and socioeconomic disadvantages lead to the overrepresentation of minorities in toxic residential and occupational environments that lead to elevated risk of exposure to major hardships, conflicts and disruptions such as crime, violence, material deprivation, loss of loved ones, recurrent financial strain, relationship conflicts, unemployment and underemployment. One recent study documented that African Americans and U.S. born Latinos experienced not only higher levels of multiple psychosocial stressors but also greater clustering of stressors than whites (Sternthal, Slopen et al. 2011). Importantly, in this study psychosocial stressors, including discrimination

helped to account for the residual association between race and health after controls for education and income.

Implications for Research and Policy

Future research needs to examine how the levels, timing and accumulation of experiences of racial bias over the life-course, particularly pre-conception, in pregnancy, early childhood and pre-adolescence combine with a broad range of other toxic stressors to influence the onset and course of illness. Extant research has failed to comprehensively examine how the distinctive residential environments of blacks affect normal physiological processes, including adaptive and regulatory systems, and how racial minorities' biological adaption to their residential environments produces distinct biological profiles and patterns of interactions between biological and psychosocial factors (Williams, Mohammed et al. 2010). There is an urgent need to develop a more integrated science that will assess how multiple dimensions of the social and physical environment combine additively and/or interactively together with innate and acquired biological factors (including epigenetic effects) and accumulate over the life-course to influence disease risk.

The patterns described in this article also have implications for how race data is collected, and reported. Whenever racial data is collected or reported greater attention should be given to why race is being used, the limitations of race data, and the interpretation of findings. Data on racial differences in health should routinely stratify them by SES within racial groups to reduce mis-specifying complex health risks and the perpetuation of harmful social stereotypes (Williams 1999). Renewed research attention needs to be given to identify markers that are better than race to identify potential contribution of genetic factors. It is also important to remember that there is diversity of experiences and exposures with each of the major racial groups in the U.S. as well as between groups. While there are processes of stigmatization and exclusion that affect multiple minority groups, the SES and health of each group is shaped by its own distinctive history and social, political and cultural context. Accordingly, as we look across various racial and ethnic populations, we should expect to find both similarities and differences with the patterns observed for African Americans.

The persistence of striking inequalities in health by race and SES also highlight the urgent need for a renewed research focus of identifying the interventions at multiple societal levels that will be effective in reducing and ultimately eliminating racial inequities in health. Importantly, although there is much that we need to learn about maximizing the impact of interventions to reduce the observed gaps, there is substantial evidence now that provides clear direction and promising strategies to tackle health inequalities (Williams and Mohammed 2013b; Cohen and Sherman 2014; Osypuk, Joshi et al. 2014). Thus our greatest need is to build the needed political support to utilize the best available science to reduce the massive loss of lives that social inequalities in health reflect. The available evidence also suggests the need for generic interventions targeted at reducing SES inequalities and targeted interventions that address the added effects of race. Both of these classes of interventions should focus on improving the health of disadvantaged groups more rapidly than the rest of the population so that progress can be made in eliminating inequalities. Many interventions that promise the greatest benefit in improving population health are

likely to widen disparities because of the greater health benefits received by the most advantaged (Mechanic 2002).

In conclusion, there are large and pervasive racial disparities in health that reflect larger social inequalities within societies, of which SES is only one part. Race, still matters for health when SES is considered, as indeed does gender. In particular, racism is an organized system of social stratification that combines with, and even transforms SES to influence health. Action is thus critically required to illuminate and address, how the health of socially disadvantaged groups is determined by exposures to risks and resources linked to living and working conditions, and how intervening in these contexts can lead to improvements in health.

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Table 1

Socioeconomic Status by Race and Ethnicity

Indicator	Whites	Blacks	Hispanics	Asian	Ratios		
					B/W	H/W	A/W
Education (% College Grad+), 2010[1]	31	17.8	13	50.2	0.57	0.42	1.62
Median Household Income, 2013[2]	58,270	34,598	40,963	67,065	0.59	0.70	1.15
Median Wealth, 2011[3]	110,500	6,314	7,683	89,339	0.06	0.07	0.81
Non-Home Wealth, 2011[3]	33,408	2,124	4,010	29,339	0.06	0.12	0.88
Home Ownership, 2014 (%)[4]	72.3	42.1	44.5	--	0.58	0.62	--

Whites = Non-Hispanic Whites;

[1] U. S. Census (2010)

[2] (DeNavas-Walt and Proctor 2014)

[3] (Vornovitsky, Gottschalck et al. 2014)

[4] (Callis and Kresin 2015)

Table 2

Heart disease death rates, age-standardized, for Blacks and Whites, aged 25–64, 2001

Education	Blacks	Whites	B/W Ratio	B-W Difference
		Men		
All	194.9	100.7	1.94	94.2
<12 years	262.9	214.9	1.22	48
12 years	258.2	145.2	1.78	113
13–15 years	120	73.1	1.64	46.9
16+ years	99.2	51.1	1.94	48.1
Low-High Difference	164	164		
Low/High Ratio	2.65	4.21		
		Women		
All	106.1	37.9	2.8	68.2
<12 years	132.9	97.8	1.36	35.1
12 years	142.1	50.1	2.84	92
13–15 years	73	25.1	2.91	47.9
16+ years	62.8	16.9	3.72	45.9
Low-High Difference	70	81		
Low/High Ratio	2.12	5.79		

Rates per 100,000; Jemal et al 2008