

Racial Classifications, Biomarkers, and the Challenges of Health Disparities Research in the African Diaspora

by

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Abstract

Current scholarly research, both sociologically and biologically based, continues to be inundated with notions of race operating as a biological construct and as a proxy for poor health outcomes. Medical research and practice have fostered an environment where diagnostics, treatment, and the creation and dissemination of drug regimens often are influenced by a patient's skin color and ethnicity. The emergence of biological markers in social science-based surveys has fueled recent health disparities research that is shaping the meaning, interpretation, and policy of the health of people of color. Using hypertension as an example, this paper focuses on ways in which biological markers are discussed within the realm of health in the African diaspora. Additionally, the paper discusses how the quantification of disease etiology devoid of social and historical contexts can be troubling to both the social science and medical fields. Finally, the paper identifies the ways in which black scholars can shape the conversation of health inequity in future research.

The notion of “racial diseases”—that people of different races suffer from peculiar diseases and experience common diseases differently—is centuries old. It is tied to the original use of biology in inventing the political category of race. — Dorothy Roberts, Fatal Invention

Introduction

Despite more than two centuries of scholars from diverse disciplines deconstructing and debunking the validity of the existence of human biological races, the use of biological data to investigate purported racial differences in disease etiology and public health persists in genomic, medical and social science research. Biological race, it has been said repeatedly, is not real. Society, however, has made race real through laws, public policies, and social practices designed historically for the primary purpose of circumscribing and controlling the lives of people of color (Freeman, 2003; Haney-López, 2006; Roberts, 2011; Smedley & Smedley, 2005). Biomedical research has played a central role in this process by validating and perpetuating the racial calculus and taxonomies born in the era of racial slavery. In recent decades, genomics, which initially held the promise of ending the pattern and practices of racializing medical science, has resurrected the idea that biological race serves as a useful variable in biomedical research (Roberts, 2011). The social history and purported scientific uses of the concept of race thus contribute to its survival and endurance. Black Studies professor George Lipsitz uses the phrase *the possessive investment in whiteness* to account for the persistence and consequences of the use of race as a cultural artifact and social construct to provide distinct advantages for whites in housing, education, employment and income (Lipsitz, 2006). Few, however, apply this analysis and explanation to biomedical research and genomic science. Yet the common belief that science is objective and thus immune from societal issues and pressures ignores that scientists and science are products of the cultural ethos of the present and the accumulated legacies of the discipline’s past. What arguably is least acknowledged in accounting for the persistence of race in biomedical research is the role of U.S. government policies in mandating and supporting the use of racial classifications.

In the discussion that follows, we describe the federal government’s role in defining race and its implications for medical research. We then examine the use of biological markers in biomedical and social science research, and their misuse in hypertension studies to posit ill-defined concepts of biological race to account for health disparities to the detriment of other explanations. A discussion on the *Slavery Hypertension Hypothesis* follows as an exemplar of the complex problems identified in the prior section. In conclusion, we offer a few thoughts about the role of black scholars in shaping future research on health inequities.

Racial Classifications and Government Policy

Race made its first appearance as a federal classification system in the 1790 census. In the ensuing two centuries, official racial designations increased from three (white, slave, other) to fifteen on the 2010 census form (Bhatnagar, 2007; Hickman, 1996; Racebox.org, 2014). Throughout this period the term and category “white” did not change. Blacks, on the other hand, have been variously identified as slave, free, colored, mulatto, Negro, and African American. The fact that the 2010 Census form provides a combined ten sub-categories for the self-identification of Asians (5.1% of U.S. pop.) and Pacific Islanders (0.2% of U.S. pop.), and none for whites who comprise 78% of the population, is indicative of the pervasive social consciousness that accepts the category as homogenous and the terms “white” and “Caucasian” as self-evident (U.S. Census Bureau, 2014). The classifications Chinese and Indian (Native American) first appeared on the census in 1870. Japanese was added in 1890, and Mexican, Korean, Filipino, and Hindu appeared in 1930. In the past fifty years the decennial forms incorporated new categories (Pacific Islanders) and attempted to refine the collection of data pertaining to persons of Asian geographic origins or Hispanic ethnicity with sub-group labels such as Hmong, Laotian, Thai, etc. for the former, and Cuban, Puerto Rican, Colombian, etc., for the latter (Borak, Fiellin, & Chemerynski, 2004; Hirschman, Alba, & Farley, 2000; Racebox.org, 2014).

A noticeable omission in the past twenty years is any attempt to provide sub-groupings to classify recent immigrants from Africa who may differ significantly from African Americans in ethnicity and from an epidemiological standpoint. From 2000 to 2010, the African-born population doubled from 881,300 to 1.6 million (Immigration Policy Center, 2012). Twice as many Africans arrived in the U.S. during the last decade than during three centuries of the transatlantic slave trade. Nigeria, Ethiopia, Egypt, Ghana, and Kenya comprise the top five countries of origin for African immigrants. It should be noted Egyptians in the U.S. are classified as white despite the fact some self-identify as black. It also is noteworthy that with the exception of Nigeria and Ghana, the other countries on the list were not sources of the estimated 46 different ethnic groups that came from West and Central-South Africa to the U.S. during the slave trade (Heywood & Thornton, 2011). Yet like the designation “white,” which, according to the logic of the system, should include a multitude of ethnic sub-groups originating in Europe and the Mediterranean, “black” remains undifferentiated when it comes to identifying recent African immigrants or black immigrants from the Caribbean and Latin America. Thus when race is used as a referent and variable in biomedical research to distinguish and describe populations that are the subjects of comparative research in hypertension, diabetes, cancer or other diseases, the lack of rigor in both classification and nomenclature has major implications for the analysis and reporting of data and findings.

The centrality and significance of federal racial classifications in biomedical research cannot be overemphasized. Current U.S. government policies require the use of federal classifications to structure and report the results of all government-funded research. The passage of civil rights laws in the 1960s provided the rationale and justification for the use of standardized data sets in order to monitor and enforce legal compliance with anti-discrimination regulations. In 1977 the Office of Management and Budget (OMB) issued Directive No. 15, which mandated the adoption of the standards “to provide for the collection and use of compatible, nonduplicated, exchangeable racial and ethnic data by Federal agencies” (U.S. Census Bureau, 2005). In the late 1980s, the National Institutes of Health (NIH) argued for the use of Directive No. 15 guidelines to identify and document problems of discrimination in clinical research and healthcare. In the 1990s the Centers for Disease Control (CDC) drafted a statement on the “Use of Race and Ethnicity in Public Health Surveillance” that included a disclaimer about the validity of biological race but argued nevertheless for improved standards in collecting racial data (CDC, 1993). The Food and Drug Administration (FDA) followed suit with requirements for new drug applications to present effectiveness and safety data on racial sub-groups (FDA, 1998). And in 1998 the U.S. Surgeon General’s Office issued similar guidelines to use OMB Directive No. 15 classifications to administer its Healthy People 2010 program objectives (US Department of Health, 2000). These agency guidelines have been updated at various times since 2000. The controlling authority for the regulations resides within the OMB, which includes in its 2003 update the following disclaimer: “The categories represent a social-political construct designed for collecting data on the race and ethnicity of broad population groups in this country, *and are not anthropologically or scientifically based*” (emphasis added) (OMB, 2003). Notwithstanding these caveats and countless similar statements by scientists and scholars over the decades, theories of biological race have remained standard and pervasive features of biomedical and genomics research.

Although the stated intent of the government’s classification and collection of racial data in biomedical research is to document and reduce health disparities in the nation, the use of race as an independent variable to structure government-funded research belies its history as a social construction that has changed and evolved over the centuries. While the widely accepted conception of whiteness treats it as a fixed and immutable racial category, even the most cursory reading of the history of U.S. immigration laws reveals it to be a fluid concept affected by the vagaries and vicissitudes of popular culture and American nativism (Jacobson, 1998; Tehranian, 2000). Most problematic is the notion of blackness—which began its history with the official designation of “slave” in the first U.S. Census. As a result of the popular theory of genetic dominance embodied in the notion of hypodescent (the “one drop rule” that classifies anyone with a known black ancestor as “black”), the standard definition of blackness commonly accepted since the late 17th century defies any logic but that deemed essential to American apartheid and antiblack racism (Hickman, 1996; Khanna, 2010). Moreover, until the civil rights era and the government’s adoption of the self-designation *African American*, all other federal classifications on the decennial census forms (black, mulatto, colored, Negro) originated in the era of slavery.

Efforts to address current social inequality and health inequities experienced by people of color clearly are needed. Yet the failure to recognize how the continued utilization of race affects such research initiatives compromises and subverts them from within. Fundamental questions need to be raised in designing research studies and evaluating research methods and findings that purport to look for and identify racial differences in disease etiology and public health. In each instance consideration should be given to two core concerns: what is the basis used for defining race; and what is the scientific meaning, utility, validity and value of the research? Sociologist Catherine Lee examined these questions in a study that analyzed 204 biomedical research articles funded by NIH and published between 1990 and 1999. Her findings, which identify the same problems in the social science literature, are worth quoting at-length as follows:

Authors tended to see race or ethnicity as important and significant in their research. However, despite seeing the importance of race or ethnicity in their research, authors rarely defined or operationalized the concepts adequately. Moreover, when presenting findings of racial or ethnic difference, authors generally did not provide explanations of the difference. I argue that this under-theorized and unspecified use of race or ethnicity and the biological conclusions drawn about health and difference have the potential to reify “race” and to limit our thinking about what these biomedical differences suggest about health disparities and inequalities in general (Lee, 2009, pp. 1183-1184).

With this background in mind, in the following section we examine how biological markers are used in social science and biomedical research to assess disease occurrence, diagnosis, and responses to treatment, and how the ambiguities of racial classifications complicate and compromise the analysis and assessment of disease etiology and health outcomes in diverse populations.

Assessing Biological Data from a Socio-Cultural Perspective

Biological markers, commonly known as biomarkers, have become a vital tool for assessing health in both medical- and social-based research. The meaning of a biomarker has evolved over time, with no agreed upon standard definition. Due to the ambiguity of the definition, the National Institutes of Health (NIH) assembled a working group in 2001, which devised the following definition: “a biomarker is a characteristic that is objectively measured and evaluated as an indicator of normal biological processes, pathogenic processes, or pharmacological responses to a therapeutic intervention” (Colburn, DeGruttola, & DeMets, 2001). Since then, numerous definitions have surfaced, with more recent measures encompassing genetic information, which is indicative of increasing attempts to establish new, comprehensive markers of the disease process (Crimmins, Vasunilashorn, Kim, & Alley, 2008). One such detailed definition is offered by The National Library of Medicine, which issues the following guideline:

Measurable and quantifiable biological parameters (e.g., specific enzyme concentration, specific hormone concentration, specific gene phenotype distribution in a population, presence of biological substances) which serve as indices for health—and physiology-related assessments such as disease risk, psychiatric disorders, environmental exposure and its effects, disease diagnosis, metabolic processes, substance abuse, pregnancy, cell line development, epidemiologic studies, etc. (2014).

In social science research, biomarkers are commonly referred to as any physiological or functional measurements that, through tests via clinical trials and larger populations, exhibit susceptibility to disease or disease process. Social scientists often use biomarkers, particularly at the population level, to monitor and predict health outcomes (Crimmins et al., 2008) and to examine how biological processes link to typical social factors to potentially create the health disparities researchers often see among different groups of people (Gruenewald, 2013). The validation of disease has been particularly important for social scientists, which partly explains the recent explosion of data combining social indicators of health with biomarker data.

One such example of national datasets including biomarker information involves the Health and Retirement Study (HRS), a U.S.-based longitudinal study of Americans aged 50 and older. Since 1992, the HRS has provided researchers with an array of information on health, financial, and other socio-demographic factors (Brown et al., 2014; Juster & Suzman, 1995). Given the recent success of incorporating biomarkers into the HRS, comparable sister surveys have emerged to assess similar measures. To date, surveys designed specifically for comparison to the HRS span the globe, with similar surveys available in Ghana, South Africa, and Mexico, among other locations (Kowal et al., 2012).

Prior to the inclusion of biomarker data in readily accessible surveys, social scientists concerned with examining how social factors led to possible health disparities were relegated to self-rated measures of general health status and specific health conditions. Survey participants were essentially asked how they would rate their health, with responses ranging roughly from excellent to poor, with variations in possible responses across countries, cultures, and age groups (Addai & Adjei, 2013; Idler, 1993; McGee, Liao, Cao, & Cooper, 1999; Salomon, Nordhagen, Oza, & Murray, 2009). Similarly, prior to the inclusion of biomarker data, researchers who were interested in knowing whether a participant had a specific health condition, such as diabetes, used self-reported responses of that condition. For instance, a typical question would state “Within the last year, has a doctor or health professional ever diagnosed you with hypertension or high blood pressure?” Much debate exists as to whether these self-assessments of health are accurate. Although the research findings are mixed, methodological issues and cultural and socio-economic forces undeniably influence responses to these self-assessment measures.

Now, the inclusion of biomarker data allows social scientists to craft a more detailed view of health by potentially obtaining validated (diagnosed) occurrences of a disease. As a result, researchers are able to discuss how various social characteristics, such as socioeconomic status (Wolfe, Evans, & Seeman, 2012) and discrimination (Borrell, Kiefe, Williams, Diez-Roux, & Gordon-Larsen, 2006; Green & Darity Jr, 2010), get “under the skin” to influence the disease process throughout the life course (Ferraro & Shippee, 2009; McEwen, 2012; Montez & Hayward, 2011).

How has the inclusion of biomarkers in sociologically driven research influenced our understanding of and the narrative surrounding race and health? At most, the ambiguity of racial classifications in increasingly diverse societies illustrates the complexity of deciphering group membership in which to examine potential population-level differences in health outcomes. In the ensuing section, we use hypertension; one of the most commonly diagnosed conditions among people of African descent, to demonstrate how related biomarkers are employed in disparities research, noting potential pitfalls of analyzing data without a socio-cultural lens.

The Persistent Burden of Hypertension among People of African Descent

Hypertension, or high blood pressure, is defined clinically by two measurements, systolic blood pressure (SBP) and diastolic blood pressure (DBP). The first number, SBP, measures the blood vessel or arterial pressure as heart muscles contract, whereas DBP records the pressure of those arteries when the heart is at rest, or between heart beats (CDC, 2014). Measured in millimeters of mercury (mmHg), a normal blood pressure reading is less than 120/80 mmHg. For people classified as hypertensive, readings are equal to or above 140/90 mmHg. A growing concern for clinicians is the increasing percentage of people considered to be pre-hypertensive, or having blood pressure readings of 120-139 mmHg for systolic blood pressure and between 80 and 89 mmHg for diastolic blood pressure (CDC, 2014).

Hypertension is one of the world’s most persistent health conditions. Its impact is far reaching, as the condition is one of the primary, yet modifiable, risk factors for stroke and cardiovascular disease in the United States (Go et al., 2013). Globally, hypertension is estimated to account for 45% of deaths attributed to heart disease and approximately half of all deaths due to stroke (WHO, 2011). Residents of low- and middle-income countries make up a disproportionately large percentage of hypertensive patients, as countries and communities grapple with treating this growing chronic health condition while simultaneously dealing with the persistent burden of communicable diseases such as malaria, tuberculosis, and HIV/AIDS. In some instances, the prevalence of hypertension in low-income countries is currently similar to, and sometimes exceeding, the occurrence of hypertension in higher-income nations (Addo, Smeeth, & Leon, 2007; Khor, 2001; Vorster, 2002).

Hypertension has proven to be particularly troubling for people of the African Diaspora. After adjusting for age, non-Hispanic blacks have the highest percentage of hypertension in the United States (44%) and, by some estimates, one of the highest percentages in the world (Flack et al., 2010). According to a recent World Health Organization (WHO) report on hypertension, various nations within the African continent are exhibiting some of the highest percentages of people living with hypertension, which some researchers link to the lower economic statuses of these countries (WHO, 2011, 2013) as well as the vast socioeconomic inequality that exists within and between African nations (Sliwa, Stewart, & Gersh, 2011).

What drives this overwhelming hypertension burden in the diaspora? The interplay of genetic, environmental, and social mechanisms makes explaining disparities in hypertension complex. From a socio-demographic standpoint, socio-economic status (Colhoun, Hemingway, & Poulter, 1998; Colin Bell, Adair, & Popkin, 2004), diet/nutrition consumption (Appel, 2000), and obesity (Aneja, El-Atat, McFarlane, & Sowers, 2004) have all been attributed to the disproportionately higher percentages of hypertension in high-income countries such as the U.S. These social factors are less clear among populations of African descent in other, low-income countries. For example, obesity and over-nutrition rates have been increasing in parts of Africa, particularly within larger metropolitan areas. This westernization of urban environments in countries such as Ghana (Bosu, 2010), Tanzania (Edwards et al., 2000), and Cameroon (Mbanya, Minkoulou, Salah, & Balkau, 1998; Sobngwi et al., 2004) illustrates the potential for increasing rates of hypertension and subsequent burden over time.

Though hypertension remains a debilitating disease within black communities around the world, some scholars contend that the relationship between race and hypertension is much more complex and that blacks do not possess the highest rates of hypertension globally as many agency-level data indicate. In a cross-national study of Europeans and people of African descent, Cooper and colleagues found that Spain (46.8%), Finland (48.6%), and Germany (55.3%) all had a higher percentage of adults aged 35 to 64 living with hypertension than Nigeria (13.5%), Jamaica (28.6%), and U.S. Blacks (44%). Additionally, Italy, Sweden, England, Spain, Finland, and Germany all reported higher total mean blood pressures readings (both systolic and diastolic) than the three African-origin populations (R. S. Cooper et al., 2005). Variations in hypertension prevalence between groups of African-origin populations is evident in other studies, with blacks in Africa and the Caribbean generally exhibiting lower rates of hypertension than U.S. Blacks (R. Cooper et al., 1997; R. S. Cooper, Rotimi, & Ward, 1999). This variation in hypertension prevalence among African-origin populations suggests that any race/ethnic disparities evident between these populations and those of European origin, as is the case with U.S blacks and whites, are not solely based on genetics, as some have argued in the past (Grim & Robinson, 1996). The ensuing section briefly discusses the ongoing (and unsolved) debate of whether African Americans possess a genetic predisposition to higher blood pressure today as a result of the genetic selection during the Middle Passage.

The Slavery Hypertension Hypothesis

As early as the 1930's, data existed underscoring the significantly higher mean blood pressure of African Americans (Adams, 1932; Kaufman & Hall, 2003b). As additional surveys and subsequent data became readily available to examine the race-hypertension link, no single factor could explain the higher prevalence among African Americans. An array of scholars in both the social and biological sciences started to look to history and genetics for possible answers to these findings. The link between salt sensitivity and high blood pressure was targeted as a potential reason, with early speculations of the Middle Passage as a historical marker to which salt retention became a unique aspect of African Americans, as evidence existed as early as the 1960's that blood pressure was quite low for several African populations (Curtin, 1992; Henry & Cassel, 1969).

Although the connection to slavery, the Middle Passage, and salt retention was posited earlier (Blackburn & Prineas, 1983), Thomas Wilson and Clarence Grim are often credited with the conceptualization of the slavery hypothesis (1991). In general, the hypothesis asserts that African Americans are predisposed to salt retention as a result of selection characteristics during the Middle Passage and succeeding enslavement. Those who were able to survive the diseases and deplorable physical conditions of the journey and the grueling demands of slavery were those slaves who were genetically predisposed to retain salt. As a result, descendants of these slaves retain a high amount of salt. The ability to retain salt, according to this hypothesis, was a protective mechanism for slaves, yet remains a hindrance for African Americans today, as salt retention is associated with hypertension and cardiovascular disease (Pollock, 2012; Wilson & Grim, 1991). In his critique of the hypothesis, historian Philip Curtin claims that, through various examinations, the hypothesis has been divided into three separate hypotheses in which people have debated. He goes on to state the following:

The first hypothesis, which concerns the ancestral experience in Africa, emphasizes the possible genetic consequences of a low-salt diet over many centuries. In the second, which concerns genetic changes caused by trauma of the ocean passage from Africa to America during the slave trade, the case is made that individuals who were losing salt from sweat, diarrhetic stools, and vomit were more likely to survive if they already had an ability to conserve salt. This ability, which had survival value in the slave trade, would be passed on to their children and would later cause hypertension and death in the African-American community. For the third hypothesis, which concerns the genetic consequences of life under slavery, the argument is less precise, but it holds that bad conditions caused high death rates and hence genetic change among the survivors (1992, pp. 1681-1682).

In examining the first hypothesis, discrepancies exist as to whether slaves that arrived in the Americas were in fact from salt-deficient regions of Africa. Seaside areas were accessible to the salt derived from saltwater. However, according to reports, the availability of salt varied in West Africa, and areas that potentially lacked salt were able to obtain it via detailed trading networks (Curtin, 1992; Wilson, 1986), although some contend that Central Africa was and remains a “salt-poor” region (Jackson, 2005). Moreover, the fact that salt was a cheap commodity also leads some historians to suggest that there were possibly adequate supplies of it (Curtin, 1992), although accounts of this are not available.

A large majority of scholars tend to focus on the second hypothesis, which contends that, those slaves that were able to adapt to these adverse conditions of the Middle Passage were those who were able to retain salt. Anthropologist Fatimah Jackson (1991) has focused on the potential genetic variability of the transatlantic slave trade in previous work. While positing that the evidence of the hypothesis is weak and more detailed tests and analyses of interdisciplinary databases should be conducted, Jackson asserts that the following selective and diverse pressures endured by slaves after arrival and throughout the more than four hundred years since the transatlantic slave trade has led to a much more heterogeneous group of African Americans. Considering the socio-cultural diversity, nutritional deprivation, and psychological stress occurring among slaves *before* encountering the transatlantic journey, Jackson claims that the Middle Passage led to a variety of acute and chronic stressors that scholars have yet to understand fully (2005).

Proponents of the *Slavery Hypertension Hypothesis* ignore the neglected history of white servitude in North America as a possible data source for study and analysis. If the Middle Passage and slavery is indeed an historical marker that suggests a possible link to black rates of hypertension, then, according to this theory, similar studies of hypertension rates in whites should be conducted on the descendants of 17th and 18th century English and German indentured servants to determine the possible long-term effects of the Northern Passage and servitude on that population. The term “indentured servitude” identifies a form of “bound labor” that “encompassed Indians, apprentices, domestic poor, debtors, convicts, foreign convicts, and those foreigners entering the country as servants” (Bilder, 1996, p. 752). It also describes the first form of unfree labor in the British colonies of North America (Bilder, 1996; Jordan & Walsh, 2008). England and Germany supplied the vast majority of immigrant workers to this system of bound labor that peaked around 1772 (prior to the American Revolution), declined significantly sometime after 1820 (more than a decade after the cessation of the transatlantic slave trade from Africa), and ended in the 1830s (Grubb, 1994; Jordan & Walsh, 2008). Estimates range from half to three quarters of the colonists that came to British North America were indentured, with the Chesapeake tobacco-growing region receiving the largest percentages (Bilder, 1996). More than 300,000 persons arrived in bondage from Europe during the period between 1620 and 1775, as compared to approximately 390,000 enslaved Africans imported directly from Africa of which 72% arrived before 1776 (Eltis, 2008; Jordan & Walsh, 2008).

Maligned and stereotyped as rogues, whores, and vagabonds by their colonial masters, demographic profiles of the indentured prior to their departure to North America reveal a large segment of the population came from the lower classes in Europe and thus suffered from malnutrition, a host of chronic and communicable diseases, and the psychological and physical traumas associated with poverty and oppression (Jordan & Walsh, 2008; Souden, 1978). They were transported across the Atlantic in abysmal conditions, faced severe hardships of starvation and disease during the voyages, and experienced mortality rates ranging from ten to fifteen percent before arrival (the same as African mortality on the Middle Passage) (Grubb, 1987). Those who survived were sold at auction individually or in groups as legal chattel for specified periods of time to pay off debts or transportation costs. In some instances they were stripped naked for inspection prior to sale (Roediger, 2007). With the exception of the contracted lengths of their servitude, which set dates for their freedom that varied according to skill, gender and age, they were essentially slaves to masters who could whip them, sell them, and forbid them to marry (Kolchin, 2003).

In the 17th century these unfree whites initially outnumbered enslaved Africans five-to-one. Over the next century, they shared housing, socialized, occasionally married, and labored side-by-side in the fields with blacks whose numbers increased dramatically each decade (Breen & Innes, 2004; Morgan, 1998; Sobel, 1987). Consequently, they experienced many of the same physical and psychological stressors that have been linked to purported hereditary factors of hypertension in African Americans. Where are the epidemiological studies of the descendants of these white “slaves”? What role if any did indentured servitude play in disease etiology within that population? The point of this digression goes to our main contention pertaining to the problems of using race to explain disease etiology in blacks to the disadvantage of other explanations. Rather than providing scientific evidence, the blackening of disease in U.S. medical research, like the pathologizing of blackness in U.S. culture, historically has served the singular purpose of perpetuating the fiction of racial differences and biological races to maintain the socio-political status quo of white supremacy/black inferiority. Even biomedical researchers with the best intentions unwittingly become trapped in this “epistemology of ignorance,” as Charles Mills describes the cognitive dysfunction that forms and informs the social construction of race (Mills, 1997, p. 18).

As mentioned earlier, this debate on the validity of this hypothesis, or in Curtin’s case, hypotheses, is ongoing and quite extensive and outside of the scope of this paper (Armelagos, 2005; Armelagos & Maes, 2006; Kaufman & Hall, 2003a). However, it is important to emphasize a few critiques made by scholars in regards to whether these claims are true or are actually measurable. Scholars questioning the slavery hypothesis and other theories meant to examine intergenerational trauma among minority populations point to the methodological quandary of teasing apart the current and historical impact of generations of discrimination and mental and physical violence (Cross, 1998; Green & Darity Jr, 2010). As scholars Tiffany Green and William Darity suggest,

Moreover, how does one determine, at least in a quantitative sense, the most relevant tragic historical event or events; was it the Middle Passage, slavery itself, or the White terror campaign in the post-Reconstruction era conducted by groups such as the Ku Klux Klan and the Red Shirts, or contemporary daily traumas associated with the deaths like that of unarmed Guinean immigrant Amadou Diallo? (2010, p. S37).

Considering the brevity and continuity of these stressors, it is extremely difficult to identify the effect of a single event on health. Research concerned with present-day health outcomes would benefit greatly from increased focus on methodological frameworks geared toward examining health within a given social context over time as well as variation of health and socio-cultural factors within individuals and within racial categories. Likewise, researchers must also look to different approaches that capture what the social construct of race really means within health disparities research.

Research on the effects of skin color (as opposed to racial classification) on hypertension provides an excellent example of the importance of the conceptualization of measures in disparities scholarship. Previous studies have found a significant relationship between skin color and high blood pressure in African-descent populations both in the U.S. (Armstead, Hébert, Griffin, & Prince, 2013; Harburg, Gleibermann, Roeper, Schork, & Schull, 1978) and abroad (Dressler, Balieiro, & Dos Santos, 1999). In fact, in a sample of adults in southeastern Puerto Rico, skin color proved to be a better predictor of high blood pressure than estimated genetic ancestry (Gravlee, Non, & Mulligan, 2009). Research results vary in regards to both the degree and manner in which skin color and blood pressure are associated among African-descended groups. Several studies suggest that the skin color-blood pressure relationship is influenced by socioeconomic status (SES) (Keil, Tyroler, Sandifer, & Boyle Jr, 1977; Klag, Whelton, Coresh, Grim, & Kuller, 1991), whereas some contend that the relationship remains significant, independent of SES (Harburg et al., 1978).

The impact of SES on the relationship between skin color and blood pressure is complex. In a longitudinal study of young adults in the U.S., Elizabeth Sweet and colleagues found that African Americans with lighter skin experienced an SES gradient where those with higher incomes had lower systolic blood pressure. However, there was no significant impact of income on the skin color-blood pressure link, thus indicating that gains in economic success, a social factor that typically leads to better health, does not improve blood pressure readings for darker African Americans (Sweet, McDade, Kiefe, & Liu, 2007). Gravlee and Dressler's research on skin pigmentation and blood pressure among Puerto Ricans addressed the role of SES, but yielded different results (2005). There was no significant relationship between high blood pressure and self-rated color, which was measured by a nine-point scale spanning from *claro* (light) to *oscuro* (dark).

However, when considering respondents' self-ratings of skin color and actual measurements of skin color, Gravlee and Dressler found that this discrepancy ("color incongruity"), once an interaction with SES was considered, was associated with differential systolic blood pressure results. Specifically, people who self-reported a darker skin color relative to the measured skin color reading were more likely to have a higher systolic blood pressure, *only if* they were of a lower SES background. People reporting similar measures on the color incongruity scale, but of a higher SES background, had lower systolic blood pressure averages (Gravlee & Dressler, 2005). Research exploring Africoid facial features, discrimination, and health is also pushing the boundaries of disparities scholarship. These results and emerging methodology undoubtedly show the importance and complexity of socio-cultural processes, above and beyond racial classification, and the need for their continued inclusion into data focused on understanding health experiences.

Given the greater possibilities of creating a detailed health profile of individuals through more biological data, there are drawbacks regarding the use of biomarkers within the social sciences. As mentioned earlier, there is no clear consensus on a general definition of a biomarker, and in many cases, there is even greater ambiguity as to which biomarker should be used to assess a particular health condition, leading one to question whether a given biomarker actually measures what it claims (Strimbu & Tavel, 2010). This ambiguity is reflected in disparities research on blood pressure. In addition to the usage of diastolic and systolic blood pressure as commonly accessible measures for large datasets, some scholars, particularly those studying the aged, prefer to use the measurement pulse pressure, which takes the difference between the two traditional measures (Crimmins et al., 2008). However, because hypertension is known to be a risk factor for cardiovascular disease (Albert, 2007), some medical and public health scholars contend that individual biomarkers for specific conditions limit the ability to assess multiple biological pathways in which to diagnose and subsequently treat multiple health conditions (Wang et al., 2007). The high correlation of biomarkers with each other can create some complexity for research involving large scale datasets, as these more clinically based biomarkers may not all be included in the data. The biomarkers for hypertension and other health conditions that are included in social surveys are commonly derived from clinical trials that, despite the increasing focus on race-based health disparities research, have a paucity of racially diverse people (Albert, 2007; Sliwa et al., 2011). As social scientists, we must give careful consideration to how we may utilize this increasingly available biological data.

Conclusion

The wide-ranging discussion presented above addresses several key issues in the ongoing efforts to account for health disparities that illuminate the unique burden of illness and disease in black communities in the U.S. and throughout the African diaspora. Of critical concern to this analysis is the problematic use of race in social science and biomedical research. As noted at the beginning, the broad scholarly consensus that race is a social construction is well established. In fact, scientific racist ideology and the racism it engendered came under immediate assault from black scholars and others upon its emergence in the 18th century. The early confrontation and contestation of biological race is perhaps best exemplified in this remark written by a former slave in 1789:

Can it be contended, that a difference in colour alone can constitute a difference in species?—if not, in what single circumstance are we different from mankind? what variety is there in our organization? what inferiority of art in the fashioning of our bodies? what imperfections in the fashioning of our minds? (Bay, 2000, p. 16)

The above comment is both noteworthy in terms of its date and source and because the author questions the key conceptual components—skin color, physiology, and intelligence—that have been cited for centuries to define, explicate and defend the existence of human biological races. Comprehensive analyses and critiques of biological race from various disciplines (anthropology, sociology, genetics, Black Studies, etc.) followed this early rebuttal and can be seen in the works of scholars like Charles Darwin, W.E.B. Dubois, Ruth Benedict, Ashley Montagu, and more recently in books and articles by C. Loring Brace, Joseph Graves, and Dorothy Roberts to name but a few. Even the federal government, the historical and current source and authority for racial classifications in the U.S., discounts the anthropological and biological basis of race as noted above. Nevertheless, in 2010 when geneticist Joseph Graves conducted a search of PubMed (the electronic database of the National Library of Medicine) using the term “race,” the search “returned 114,305 articles from the human biomedical literature. More specifically, searches on Caucasian, Mongoloid, and Negroid race returned 52,846, 22667, and 38,792 citations, respectively” (Graves, 2011, pp. 144-145). We conducted a similar search of PubMed on April 10, 2014 that returned 180,551 articles using the term “race” with limiters set to “human.” Our searches on Caucasian, Mongoloid, and Negroid identified 75,356, 40,834, and 65,821 citations, respectively. During the search we also noted the availability of MESH terms (Medical Subject Headings) for searching the database as follows: black race (66,846 citations) and race differences (31,629 citations). Admittedly, an undetermined number of the references we retrieved were cross-indexed. Nevertheless, of all the various categories of citations we identified, only *two* were published before 1961.

While we recognize the fact that the federally mandated use of racial classifications to structure research affects how studies are titled and indexed in the database, the fact remains the vast majority of researchers continue to draw inferences of racial differences from their research data to analyze outcomes and formulate conclusions. The obvious result is the social reinscription and reification of biological race rather than its deracination from biomedical research.

In view of this situation, it is imperative to reconsider what bioethics truly means in the genomic era. While the basic definition of the term—the philosophical implications of right and wrong in healthcare, and what constitutes professionalism in the field of medicine—is both accurate and apposite, its implementation in our healthcare system, given the preponderance of health disparities and lack of affordable care, has always been questionable. Much more discussion in the media, the healthcare system, the classroom, the laboratory, and the halls of government is needed to effect positive changes in the ethical conduct of bioresearch and healthcare going forward. From our perspective as scholars of Pan African Studies celebrating the 40th anniversary of our department, the multidisciplinary nature of our field provides a unique vantage point from which to meet and address these issues. We who labor “in the vineyard”—the phrase Perry A. Hall uses to describe the mission of building Black Studies in academia—have always understood the need to balance social activism with scholarship (Hall, 1999). We also understand and are prepared for the necessity of crossing disciplinary boundaries to tackle the issues of racism and injustice that challenge and confront us as scholars and agents for change on campuses and in our communities. This special issue of the *Journal of Pan African Studies* reflects that commitment and sets the stage for the next forty years of our department’s work *in the vineyard* to produce first-rate Pan African Studies scholars and scholarship for generations to come.

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