Review Article

Understanding and Effectively Addressing Breast Cancer in African American Women: Unpacking the Social Context

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Black women have a higher incidence of breast cancer before the age of 40 years, more severe disease at all ages, and an elevated mortality risk in comparison with white women. There is limited understanding of the contribution of social factors to these patterns. Elucidating the role of the social determinants of health in breast cancer disparities requires greater attention to how risk factors for breast cancer unfold over the lifecourse and to the complex ways in which socioeconomic status and racism shape exposure to psychosocial, physical, chemical, and other individual and community-level assaults that increase the risk of breast cancer. Research that takes seriously the social context in which black women live is also needed to maximize the opportunities to prevent breast cancer in this underserved group. ***Cancer*** **2016;122:2138-49**. © *2016 American Cancer Society.*

KEYWORDS: African American women, breast cancer, race, socioeconomic status (SES), stress.

INTRODUCTION

Despite historically having lower incidence rates of breast cancer than white women, African American (or black) women have had markedly higher death rates from breast cancer in comparison with any other racial or ethnic group in the United States.[1] However, these poor outcomes remain largely unexplained. Although much recent research has focused on genetic factors associated with an increased risk of disease, we argue that social contextual factors associated with breast cancer risk have been understudied, particularly as they relate to black women. Understanding and effectively addressing black-white disparities in breast cancer will require a rigorous understanding of black women's lived experiences of racism, segregation, psychosocial stress, and the cumulative stress of living amidst a disproportionate burden of social and environmental assaults and how these experiences undermine health and contribute to breast cancer risk and mortality.

This article reviews the epidemiological literature on breast cancer among black women and addresses the contribution of social factors to racial inequities across the continuum of breast cancer. We begin with a description of the patterns of racial disparities in breast cancer. We then address 3 essential areas in which social-contextual factors have been undervalued in efforts to understand breast cancer disparities. The first area that we address is the importance of the lifecourse perspective; we examine data on how a low socioeconomic status (SES), psychosocial stress, and other adverse exposures starting in early childhood and continuing through adulthood accumulate to increase breast cancer risk. The second area encompasses the multiple risks and resources in the social environment linked to race, and we empirically examine how these are likely related to one another and combine with biological factors to contribute to racial disparities in breast cancer. Finally, we argue for a renewed look at the science of breast cancer prevention and how breast cancer prevention efforts might be improved so that opportunities to reduce breast cancer risks can be maximized.

BLACK-WHITE DISPARITIES IN BREAST CANCER

Historically, black women in the United States have had a lower overall age-adjusted breast cancer incidence rate than their non-Hispanic white counterparts[2] but a higher incidence of breast cancer than their white peers under the age of 40

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TABLE 1. Age-Adjusted and Age-Specific Death Rates for Breast Cancer for Black and Non-Hispanic White Women, 1950–2010

	1950	1960	1970	1980	1990	2000	2010
All ages, age-adjusted							
White	32.4	32.0	32.5	32.1	33.2	26.3	21.5
Black	25.3	27.9	28.9	31.7	38.1	34.5	30.3
Black-white difference	–7.1	–4.1	–3.6	–0.4	4.9	8.2	8.8
Black/white ratio	0.8	0.9	0.9	1.0	1.1	1.3	1.4
35–44 y							
White	20.8	19.7	20.2	17.3	17.1	11.3	8.8
Black	21.0	24.8	24.4	24.1	25.8	20.9	18.3
Black-white difference	0.2	5.1	4.2	6.8	8.7	9.6	9.5
Black/white ratio	1.0	1.3	1.2	1.4	1.5	1.8	2.1
45–54 y							
White	47.1	51.2	53.0	48.1	44.3	31.2	23.9
Black	46.5	54.4	52.0	52.7	60.5	51.5	40.9
Black-white difference	–0.6	3.2	–1.0	4.6	16.2	20.3	17.0
Black/white ratio	1.0	1.1	1.0	1.1	1.4	1.7	1.7
55–64 y							
White	70.9	71.8	79.3	81.3	78.5	57.9	45.9
Black	64.3	63.2	64.7	79.9	93.1	80.9	70.5
Black-white difference	–6.6	–8.6	–14.6	–1.4	14.6	23.0	24.6
Black/white ratio	0.9	0.9	0.8	1.0	1.2	1.4	1.5
65–74 y							
White	96.3	91.6	95.9	103.7	113.3	89.3	73.2
Black	67.0	72.3	77.3	84.3	112.2	98.6	97.4
Black-white difference	–29.3	–19.3	–18.6	–19.4	–1.1	9.3	24.2
Black/white ratio	0.7	0.8	0.8	0.8	1.0	1.1	1.3
75–84 y							
White	143.6	132.8	129.6	128.4	148.2	130.2	110.2
Black	81.0	87.5	101.8	114.1	140.5	139.6	123.2
Black-white difference	–62.6	–45.3	–27.8	–14.3	–7.7	9.4	13.0
Black/white ratio	0.6	0.7	0.8	0.9	0.9	1.1	1.1
≥85 y							
White	204.2	199.7	161.9	171.7	198.0	205.5	186.8
Black	N/A	92.1	112.1	149.9	201.5	238.7	214.6
Black-white difference	—	–107.6	–49.8	–21.8	3.5	33.2	27.8
Black/white ratio	—	0.5	0.7	0.9	1.0	1.2	1.1

Abbreviation: N/A, not available.
The data for this table have been taken from the National Center for Health Statistics. Health, United States, 2013. Hyattsville, MD: US Department of Health and Human Services; 2014.

years.[3] However, as incidence rates have remained stable for whites but continued to increase for blacks in recent years, black-white breast cancer incidence rates converged in 2012.[1] At the same time, black women had a breast cancer mortality rate that was 42% higher than that of whites in 2012; this mortality rate has been consistently higher than that of any other racial or ethnic group.[1]

The pattern of breast cancer mortality among black women is complex (Table 1). In 2010, the age-adjusted breast cancer death rate for black women was 40% higher than that for their white peers.[2] Although black women had lower age-adjusted mortality than their white peers in 1950, their death rates have been on a small but consistent upward trajectory through 1980, with a marked increase in 1990 and a declining trend since then. White women, on the other hand, had fairly stable death rates through 1990, with declining mortality rates in recent decades. This widening absolute and relative disparity since 1990 reflects larger declines in mortality for white women versus black women in recent decades.

Age standardization, a useful strategy that provides an equal basis for comparing populations that differ in age structure, can obscure an accurate picture of the nature and extent of racial differences in health because an age-adjusted rate is not an accurate measure of actual risk.[2,4] Age-adjusted rates are typically calculated with the age structure of a standard population. However, the choice of a standard is arbitrary, and the age structure of a standard population can introduce biases that favor one population over another.[5] Age standardization can thus lead to an underestimation of racial disparities in mortality risks, including the breast cancer mortality risk.[4,5]

The age-specific mortality rates in Table 1 provide a picture of the actual racial gap in breast cancer mortality for specific age groups. In 2010, the black/white ratio shows that in contrast to the 40% higher overall age-

adjusted mortality rate, black women had a death rate for breast cancer that was more than twice as high as the rate for whites at the ages of 35 to 44 years, was 70% higher at the ages of 45 to 54 years, and was 50% higher at the ages of 55 to 64 years. The absolute black-white difference was also markedly larger at all age groups than the age-adjusted overall rate of 8.8 deaths per 100,000 population. With the exception of the age groups of 35 to 44 and 75 to 84 years, the difference was twice as large. In terms of trends over time, the age-specific rates show that for the 2 youngest age groups, a racial disparity in breast cancer mortality was evident as early as 1960.

Racial differences in the severity, course, and treatment of breast cancer contribute to these racial differences in mortality. Compared with their white peers, black women are more likely to be diagnosed at a later stage, are less likely to receive stage-appropriate treatment, and are more likely to have lower stage-for-stage survival rates.[3,6] Black women also have a higher risk of poor-prognosis types of cancer. Compared with white women, black women (especially premenopausal black women) are more likely to be diagnosed with estrogen receptor (ER)–negative tumors and ER-negative subtypes, including the triple-negative subtype.[3] They are also more likely to be negative for progesterone receptor (PR) and human epidermal growth factor receptor 2 tumors. Basal-like tumors are another subtype of breast tumors that are also high-grade cancers with poor survival. The joint ER/PR status of tumors is a stronger predictor of mortality than either status considered alone, and kinetic measures of breast cancer are also better predictors of treatment response and outcome than the more traditional static measures.[7,8] Importantly, black women are more likely than their white counterparts to have subtypes of breast cancer tumors that are more aggressive and more resistant to treatment and that do not have clear molecular targets for treatment. These differences in the prevalence of breast cancer subtypes are directly related to differential outcomes.[3,9] For example, the poor-prognosis tumors more common among premenopausal black women have higher proliferation rates, poorer differentiation, greater lymph node involvement, and larger sizes.

The reasons underlying the higher prevalence of poor-prognosis cancers among black women are not fully understood. Reproductive factors that protect against ER/PR-positive breast cancers in black women, including multiparity, a younger age at menarche, and an early age at first pregnancy, also increase the risk of ER-negative and PR-negative breast cancer.[3] Premenopausal aggressive subtypes of breast cancer are also common in black women in West Africa (although overall breast cancer rates are low), Great Britain, and the Caribbean.[3,9] Compared with non-Hispanic white women, Hispanic black women have patterns of late-stage diagnosis and breast cancer mortality that are similar in magnitude to those of non-Hispanic black women but unlike the patterns of Hispanic white women.[10] These patterns suggest that genetic risks linked to African ancestry may play some role. An alternative hypothesis is that populations of African ancestry in different geographic contexts may face common exposures linked to social and economic adversity due to skin color or discrimination that may increase breast cancer risks through poor access to health care, exposure to social and environmental assaults, or increased psychosocial stress. Many biological features of breast cancer are historically contingent and context-dependent in this way. Variations in breast cancer incidence by age and the association of ER-negative tumors with race and SES have varied over time, and even the ER status of a tumor is not a fixed characteristic.[11]

LIFECOURSE APPROACH: THE LINK BETWEEN CUMULATIVE ADVERSITY AND DISEASE

Advancing our understanding of environmental contributions to breast cancer risk and to potential levers for intervention requires greater attention to capturing pathogenic exposures over the lifecourse. Very limited evidence suggests that early-life stressors may increase the risk of breast cancer as well as other chronic illnesses, and some have argued that a perspective that emphasizes early-life exposures can integrate the diverse risk factors for breast cancer that have emerged in the epidemiologic literature.[12] The early onset and severity of breast cancer and poorer survival among black women must be understood within the context of an emerging body of scientific evidence that has documented dramatically earlier onset of disease across multiple health conditions (including cardiovascular and kidney disease) for blacks versus whites. This highlights the central role that racial differences in exposures over the lifecourse in family, neighborhood, and occupational environments may play in racial disparities in health.[13] The terms *accelerated aging*, *premature age*, and *biological weathering* are used in the literature to describe this phenomenon of an earlier onset and poorer prognosis for illness across multiple chronic diseases.

Geronimus et al[14] term this greater physiological wear and tear of US blacks and the more rapid biological aging that they experience in comparison with whites *weathering*. This accelerated aging, in turn, leads to the

early incidence of multiple chronic illnesses. Such weathering among African Americans and other disadvantaged communities is driven by the cumulative impact of repeated exposures to psychological, social, physical, and chemical stressors in their residential, occupational, and other environments and by coping with these stressors. Thus, for groups living in adverse living conditions, chronological age captures the cumulative impact of exposure to these risks.

The concept of allostatic load (AL) has been used to capture the biological dysregulation across multiple physiological systems that results from the cumulative burden of repeated stressors. In a national study by Geronimus et al,[15] AL increased with age for both blacks and whites, but the mean AL score for whites aged 55 to 64 years old was identical to that of blacks who were 10 years younger. Data from 2 national studies also reveal that elevated AL scores capture exposure to adversity over the lifecourse. One study found that economic adversity during childhood and at 2 points in adulthood individually and cumulatively predicted elevated AL scores in later life.[16] The other documented that neighborhood SES was inversely related to AL and that blacks who lived in low-SES neighborhood environments had a 200% increased odds of having high AL in comparison with those in a high-SES neighborhood.[17]

A review of studies using national data also revealed that elevated AL is associated with poor health.[17] Individuals with high AL had a life expectancy that was 6 years shorter than that of individuals with low AL scores, and both blacks and whites under the age of 65 years with high AL scores had mortality rates more than twice the rates of those with low AL scores. Another study found that elevated AL scores were associated with a history of breast cancer for black women but were unrelated for white women.[18] It was unclear whether the AL scores reflected a greater biological burden of breast cancer or captured a risk factor for the incidence of the disease. Telomere length is another indicator of biological aging that captures, at least in part, adversity and stress experienced by the individual. A study of middle-aged women found that at the same chronological age, black women had shorter telomeres than white women; this difference was equivalent to accelerated biological aging of approximately 7.5 years.[15]

Finally, there is a burgeoning body of epigenetic research investigating the impact of early-life stressors on disease via dysregulation of the stress pathway (ie, the hypothalamic-pituitary-adrenal axis). DNA methylation at various points along nuclear receptor 3C1 (*NR3C1*), the gene that codes for the glucocorticoid receptor on the hypothalamic-pituitary-adrenal axis, has been associated with an early childhood history of social adversity (eg, childhood loss of a parent,[19] childhood abuse,[19-21] psychological trauma and stressors,[22] or anxiety[23]), even if the stressful event occurred decades before the DNA was analyzed. These data have established that blood DNA methylation throughout *NR3C1* represents a unique record of past adverse psychosocial experience. Increased methylation of *NR3C1,* in turn, has been associated with increased risk for many illnesses. In a recent case-control study of *NR3C1* methylation and breast cancer, the authors found 15% of breast cancer tumors to be methylated, whereas no samples of normal breast tissue were methylated.[24] These emerging data offer a preliminary suggestion that psychosocial stress, operating via the stress pathway, may contribute to breast cancer risk.

As the next several sections show, using a lifecourse approach to understanding how SES, psychosocial stress, or adversity and other prenatal and early-life exposures come together to increase the risk of breast cancer later in life is especially important for trying to understand the development of current disparities in breast cancer incidence and mortality. That is, because minority and underserved populations in the United States are more likely to suffer from greater poverty and various psychosocial stressors early in life, particular attention needs to be paid to how these early-life exposures contribute to disease risk later in life, and only a lifecourse perspective can help to elucidate this.

SES and Breast Cancer

SES is a social factor that is a strong predictor of variation in health for a broad range of outcomes. However, the association between SES and breast cancer is complex. Breast cancer incidence rates for all racial/ethnic groups tend to be positively associated with SES.[25] At the same time, low SES is associated with an increased risk of aggressive premenopausal breast cancers as well as a diagnosis at a late stage and poorer survival.[3,26] However, future research needs to better incorporate SES into studies of breast cancer because there are large racial differences in SES and SES is a likely contributor to the elevated risk of aggressive cancers among young black women. For example, in 2013, black households in the United States earned 59 cents for every dollar of income earned by white households; this is identical to the racial gap in income in 1978.[27] Most large-scale studies of SES and breast cancer in the United States have used area-based measures of SES that imperfectly capture variation at the household level.

However, individual or household levels of income, education, or occupational status understate racial differences in SES because they do not capture the striking racial differences in economic assets and wealth. For example, in 2011, for every dollar of wealth that white households had, Latino households had 7 cents, and black households had only 6 cents.[27]

The ways in which lower SES shapes the trajectory of breast cancer risk factors over the lifecourse are complex and multifactorial. The influence of early childhood exposures on breast cancer risk later in life is particularly complex. Inadequate attention has been paid to the various ways in which SES may contribute to breast cancer risk over the lifecourse, but especially in the early childhood period, via the many pathways that are associated with specific risk factors. The association of low SES with early menarche, which is also associated with an increased risk of breast cancer,[28] is one example. Over the last 50 years, the average age of menarche has declined in the United States for both black and white women, but there has consistently been an almost 2-fold greater risk of early menarche for black females versus white females.[29] Several environmental factors patterned by SES have been linked to an increased risk of early menarche. These include prenatal smoke exposure,[30] obesity and higher rates of change in childhood body mass index,[31] excessive weight gain in the first 9 months of life,[32] low fruit and vegetable intake,[33] chronic stress in the family,[34] and characteristics of the neighborhood environment such as an absence of recreational outlets.[35] Research also reveals that childhood SES may be directly associated with age at menarche, with one study finding that for blacks, whites, and Hispanics, low SES (a composite measure of parental income, education, and occupation) at the age of 7 years and reductions in SES between birth and the age of 7 years were associated with an earlier age at menarche.[36] Future research needs to better assess how early childhood SES combines with SES in later life to predict risks of breast cancer.

Stress and Breast Cancer: Timing Is (Almost) Everything

A second social factor that could potentially contribute to breast cancer risk and that needs to be considered from a lifecourse perspective is psychosocial stress. Prior research on stress in relation to both breast cancer incidence and relapse provides little consistent evidence showing that exposure to stressors is a risk factor,[37,38] but existing studies are limited by an overreliance on measures that assess exposure to only a few recent stressors in adulthood.[13]

Scientific evidence suggests that childhood exposures, however, may shape adult health risks through processes of biological embedding or developmental programming in which early-life experiences can trigger long-term changes in biological processes in stable and predictable ways that lead to elevated health risks over the lifecourse.[39] In addition, severe and/or enduring forms of early-life physical and psychosocial exposures can shape adult risks through their effects on adult health behaviors (smoking, alcohol use, and nutrition).[40] These complex processes may produce epigenetic changes that span both an individual's lifetime and generations.[41]

Some limited evidence suggests that severe stressors in early life are specifically associated with an increased risk of cancer in adults. A study of Canadian adults found that physical abuse as a child by someone close to the respondent was associated with higher odds of cancer.[42] A national study in the United States also found that among men and women, parental emotional and physical abuse were associated with increased cancer risk.[43] Another study found that stressors that occurred at least 20 years before breast cancer hospitalization (maternal death in childhood and chronic depression with severe episodes) predicted an increased risk of breast cancer.[44] Importantly, recent life events and depression and anxiety disorders were not associated with breast cancer risk; this suggests that only certain severe distal conditions were pathogenic.

Limited evidence also suggests that prenatal stress may be associated with an increased risk of cancer. A study of children born in Denmark and Sweden found that children born to women who lost a child or spouse (but not other relatives) during the year before pregnancy or during pregnancy had a 30% increased risk of any childhood cancer.[45] The hazard ratios were largest for non-Hodgkin disease (3.40), hepatic cancer (5.51), and testicular cancer (8.52). Future research needs to better elucidate the conditions under which a broader range of maternal psychosocial stressors and other exposures may be associated with the subsequent risk of breast cancer.

Behavioral pathways are one way that such psychosocial stressors may affect cancer risks. Obesity is a major risk factor for breast cancer, although the risk may be greater in postmenopausal breast cancer; this suggests that increased caloric intake and reduced physical activity leading to obesity are likely to be important for breast cancer. Research reveals that severe childhood adversity (emotional and physical abuse from parents and penetrative childhood sexual abuse) has been associated with being obese or overweight as an adult.[46,47] Research also reveals

that excessive pregnancy weight gain is associated with an increased risk of breast cancer that is independent of the mother's weight at the time of diagnosis.[48]

Finally, some very limited research does suggest that early-life stressors may be directly associated with the progression of breast cancer specifically. A small study of women who had undergone surgery for breast cancer found that early-life abuse and neglect were associated with higher levels of perceived stress, poorer quality of life, and elevated interleukin 6 levels.[49] Similarly, early childhood abuse, neglect, and residence in a chaotic home environment were associated with elevated markers of inflammation among women who had completed primary treatment for breast cancer.[50]

This emerging body of literature suggesting that early-life stressors leave a biological imprint that lasts decades into adulthood and subsequently increases the risk of cancer and other chronic illnesses emphasizes the importance of racial differences in exposures to early-life stressors and thus the social context of disease. It is well documented, for example, that black children experience higher rates of abuse and neglect than whites.[51] Child abuse and neglect in the United States have in turn been tied to poverty, and black children suffer from a rate of poverty 3 times higher than that for white children.[52] Furthermore, black children are twice as likely as white children to witness domestic violence and 20 times more likely to witness a murder.[53] The critical role of psychosocial stress in early childhood at the individual and community levels in shaping the trajectory of breast cancer risk for black women cannot be ignored if we are to have a full appreciation of the social context and its impact on health over the lifecourse. Our current knowledge is fairly limited, however, about which specific markers of childhood adversity are most pathogenic, the length of exposure necessary to trigger adverse health effects, and the pathways (psychological, behavioral, and physiological) that link early childhood exposures to breast cancer risk.[40]

Other Prenatal and Early-Life Exposures

Nutrition and other related factors in early life and in adulthood also affect breast cancer risk. In contrast to cardiovascular disease (CVD), a lower birthweight tends to be associated with a lower risk of breast cancer.[54,55] The risk of breast cancer increases in a graded manner with increasing birthweight, and the cancer risk linked to birthweight is often strongest for premenopausal breast cancer. A higher birthweight likely reflects an abundance of prenatal nutrition and the relative amounts of specific nutrients in the maternal diet. Other recent evidence suggests that although red meat consumption in midlife is not a consistent predictor of breast cancer risk, red meat in early adulthood and especially in adolescence is associated with breast cancer overall and premenopausal breast cancer in particular.[56] Research also documents the importance of a lifecourse perspective in understanding the effects of alcohol on breast cancer: the more alcohol a woman drinks between puberty and her first full-term pregnancy, the greater her risk of developing breast cancer.[57] A priority for future research should be a more systematic examination of how social contexts shape access to healthy foods and how the prenatal and early nutritional environment may be associated with breast cancer risk through epigenetic changes that increase health risks over the life course.[54,55]

COMPREHENSIVELY ASSESSING ENVIRONMENTAL EXPOSURES

Research seeking to shed light on racial differences in breast cancer should begin with a clear recognition that self-identified racial categories capture simultaneous confounding for unmeasured social, biological, and environmental factors.[13,58] Contemporary racial categories vary over a broad range of social, behavioral, nutritional, psychological, residential, occupational, and other variables. However, most of the specific environmental factors have not been identified. Research is needed to comprehensively characterize the multiple exposures in the social, psychological, physical, chemical, and built environment that can contribute to breast cancer risk and to assess potential interactions between the social environment and both inherited and acquired biological factors.

Research on stress indicates that failure to measure stressors comprehensively can dramatically understate the effects of stress on health.[59] It has also been noted that the measures of stress in prior research on breast cancer fail to capture the full range of acute and chronic stressful experiences.[37] Compared with whites, blacks experience higher levels of stressors in multiple domains of life, greater clustering of stressors, and probably greater duration and intensity of stressors.[60] Large racial differences in income and wealth suggest that greater attention should be paid to capturing all of the stressors linked to social and material deprivation and the extent to which their greater clustering could lead to more adverse effects on minorities versus whites. Compared with whites, blacks and Hispanics receive less income at the same education levels, have markedly less wealth at equivalent income levels, have less purchasing power because of higher costs of goods and services in the residential environments where they are disproportionately located, and live in more disadvantaged

neighborhoods at the same income level.[13] Middle-class black women make larger contributions to the financial and social well-being of poorer relatives than their white counterparts; they are also more likely to live in neighborhoods with higher poverty, more female-headed households, and fewer college graduates.[61] It is important for future research to capture the duration and intensity of poverty and other economic and social stressors.

The comprehensive assessment of stressors in future research also needs to include measures of discrimination, a distinctive social exposure experienced by racial minorities. A recent review documented that self-reported measures of discrimination were adversely related to multiple disease conditions, early indicators of clinical disease potentially relevant for breast cancer (eg, inflammation, visceral fat, obesity, AL, oxidative stress, shorter telomeres, and cortisol dysregulation), and health behaviors (eg, poor sleep, cigarette smoking, and substance use).[62] In the Black Women's Health Study, the largest cohort of black women in the United States, racial discrimination was recently associated with an increased incidence of breast cancer.[63] This association was stronger among women aged 50 years or younger and among those who reported discrimination in multiple contexts. Discrimination was also associated with an increased incidence of obesity.[64] Discrimination should be assessed over the lifecourse because a recent study documented that diurnal cortisol rhythms at the age of 32 years were predicted by experiences of discrimination in the prior 20 years.[65]

Residential segregation by race has created pathogenic neighborhood conditions, with African Americans in the United States living in markedly health-damaging environments.[13] Accordingly, the neighborhoods where black women live have more adverse environmental conditions, including lower income levels, education levels, and home ownership rates and higher rates of poverty, crime, residential instability, overcrowding, and unemployment, in comparison with those of whites.[66] Neighborhood conditions are also associated with access to a broad range of exposures that are related to health, including medical care, the quality and availability of nutritious food, safe places to exercise, access to and quality of public services, and environmental pollutants.[66] Future research needs to elucidate how all of these exposures may combine to determine breast cancer risk.

Residential and occupational segregation is also distinctive in triggering exposure to toxic substances in the physical, built, and chemical environment. More research attention should also be given to the potential contribution of chemical exposures to breast cancer risk and to racial disparities in breast cancer. Laboratory studies reveal that hundreds of common chemicals activate biological pathways and cause mammary tumors, that hormone disrupters interact with ER and promote tumor proliferation, and that developmental toxicants alter mammary gland development and cancer susceptibility in rodents.[67] These chemicals are widespread in air and water pollution, consumer products, house, dust, and human tissues. These environmental factors could be directly related to breast cancer risks and could also interact with psychosocial factors to influence risk. Because of the disproportionate poverty of African Americans and the accompanying poor-quality housing and neighborhood conditions, the role of the home and residential environment in breast cancer risk remains a strikingly understudied area of research.

There has also been a lack of study of positive influences within the social context of African Americans that may contribute to resiliency and health. Supportive influences, such as psychosocial support and emotional and religious coping, for example, may attenuate the negative consequences of psychosocial stress by modifying the reaction to stress.[68,69] Religious coping/religiosity has been associated inversely with ambulatory blood pressure, colon cancer risk, and overall mortality.[70-72] Religious women with breast cancer were found to have a decreased risk of death from breast cancer in comparison with nonreligious women.[73]

Genomic research in the area of breast cancer disparities is also needed that would give increased attention to the comprehensive, detailed, and rigorous characterization of the risk factors/resources in the psychological, social, chemical, and physical environment that may interact with genetic factors to predict health risks. That is, we need an integrated science to give systematic attention to understanding the contribution of epigenetics and somatic mutations to disease risk.

ENHANCING THE SCIENCE OF PREVENTION

Research is also needed to strengthen the science base that would identify optimal strategies to increase the awareness of behavioral risk factors for breast cancer and that could be used to effectively intervene with the social factors that often initiate and sustain these risk behaviors. Some of the key behavioral areas for primary prevention that are applicable to breast cancer are physical activity, alcohol consumption, breastfeeding, early-life conditions, dietary factors, and overweight and obesity.[74] Social and behavioral research can also contribute to maximizing the

contribution of medical care to reduce breast cancer risks through secondary prevention (early diagnosis and screening) and to reducing racial disparities in treatment.

Need for Primary Prevention

A meta-analysis of prospective studies of the association between physical activity and breast cancer documented an inverse association between both occupational and nonoccupational physical activity and breast cancer risk.[75] Increased physical activity in early life and adulthood could contribute to reducing obesity in early and adult life, and physical activity may be an especially potent prevention strategy for African American women because the protective effect was more marked among premenopausal women and for ER-negative and PR-negative breast cancer tumors.

Alcohol is a human carcinogen,[76] and a dose-response relation exists between alcohol consumption and breast cancer as well as certain other cancers.[76] One drink of alcohol per day, regardless of type, is associated with approximately a 10% increase in breast cancer; 3 drinks per day are associated with approximately a 40% increase.[77] The extent to which alcohol use contributes to racial disparities in breast cancer is not clearly understood. Black women tend to consume less alcohol than their white peers, but alcohol has more negative effects on blacks than whites, with the cardiovascular benefits of moderate alcohol consumption evident in research for whites being nonexistent for blacks.[13] Evidence of confounding in research on moderate alcohol use and CVD and the methodological limitations of this literature raise questions about the extent of the moderate alcohol benefit for CVD.[78-81] Public health experts emphasize that reducing alcohol use is a vital and neglected cancer prevention strategy and that greater attention should be given to effectively communicating the role of alcohol as a risk factor for breast cancer.[76]

Breastfeeding, including the duration and the number of children breastfed, is associated with a reduced risk of aggressive premenopausal breast cancer.[3,82] White women have higher levels of breastfeeding and longer breastfeeding duration than black women.[83] One population-based study estimated that increasing breastfeeding and reducing abdominal obesity could eliminate 68% of basal-like breast cancers in young black women and more than half of breast cancers in the general population.[82] Research reveals that experiences during hospitalization for childbirth are important for initiating breastfeeding, and hospitals in areas with a higher percentage of black residents are less likely to offer their patients recommended practices supportive of breast feeding.[83] The use of community doulas (trained professionals who provide nonmedical support to mothers) can lead to marked increases in breastfeeding.[84]

For all of these different risk factors, considerable evidence highlights the need to start prevention early. Data from a prebirth cohort found that by the age of 7 years, black and Hispanic children were twice as likely as whites to be overweight and obese.[85] Infancy and childhood risk factors contributing to this pattern included early feeding behaviors (nonoptimal breast feeding and early solid foods), accelerated weight gain, and obesity-related risk factors (television in the child's bedroom, inadequate sleep, sugar-sweetened drinks, and fast food). Other research reveals that maternal economic disadvantage (reflected in low education levels, a minority racial status, and being unmarried) during the prenatal period is adversely related to health at birth and has long-term negative associations with adult health and SES.[86] Interventions, including some that begin before birth, can have positive effects on maternal and child health. These include reductions in negative health behaviors (eg, smoking during pregnancy), enhanced environments (eg, policies that lead to lower pollution and reduced violence exposure), increased access to medical care and family planning, nutritional supplements, early childhood enrichment programs (especially programs that start before the age of 3 years), and postnatal programs that provide additional income.[86]

Data also emphasize the need to appreciate the fact that prevention does not take place in a vacuum. Cancer prevention efforts are received by communities that are differentially burdened by stress, and this stress can affect individuals' capacity to respond and change health behaviors. For example, a recent study found that chronic stress alters energy homeostasis by activating peripheral mechanisms in fat tissue that augment the negative effects of sugar and fat on visceral tissue accumulation.[87] Women who ate an unhealthy diet and scored high on chronic stress had larger increases in waist circumference than those who ate the same diet but had less stress.[87] Other research indicates that addressing stress can facilitate behavioral change.[88]

Prevention science must also take a more nuanced approach in tackling health disparities. Often, such efforts focus solely on specific racial/ethnic communities without taking into account the important dimension of socioeconomic position or the heterogeneity within racial and ethnic groups. Such approaches often assume that improving overall health within a minority group is the only valid

goal and ignore the fact that higher status groups within a particular community typically have greater knowledge about and access to interventions and higher levels of utilization and often receive greater benefit from the intervention. Reducing child and adolescent obesity is a recent example. Although national data show a plateauing of the increases in adolescent obesity, for whites, blacks, and Hispanics, adolescent obesity is increasing for children of parents with a high school education or less but declining for children of parents with a college degree or more education.[89] Accordingly, we need intervention strategies that improve the health and health-enhancing behaviors of black women more rapidly than those of the rest of the population. Research is needed to identify the conditions under which interventions for behavioral risk factors have the greatest effect on socially marginalized and vulnerable populations, who tend to have the highest levels and greatest clustering of risk factors. We also need a better understanding of how we can best remove the social, economic, and psychological barriers that need to be addressed to ensure that historically disadvantaged populations experience the maximum benefits from interventions.

Health Care System Interventions

Interventions within the health care system can also play a critical role in the secondary prevention of breast cancer. The failure of black women to receive timely diagnoses and optimal treatment for breast cancer, including the aggressive subtypes, is likely to be a major contributor to their elevated mortality risk.[90] Disparities in the quality and intensity of care exist along the continuum of breast cancer; interventions for patients, providers, and the health care system to reduce disparities have been identified.[90,91] Evidence suggesting that equal treatment is associated with equal outcomes highlights the need for improvements in health care quality for black women with breast cancer.[6] Research reveals that many health care providers are unaware that racial disparities in health care treatment exist, and some still question the existence of disparities.[92] Research is needed to identify the optimal strategies for raising awareness regarding provider bias and the ways such bias influences clinical decision making and for generating the motivation and commitment among health care providers to tackle such bias.

Lessons can be learned from a concerted and comprehensive colorectal cancer initiative in the state of Delaware.[93] This screening and treatment program covered the costs of cancer care for uninsured residents and involved a nurse navigator system and special outreach efforts for African Americans. Within 8 years, the program eliminated disparities in screening and equalized incidence rates. The mortality gap was almost eliminated with a mortality decline of 42% for blacks and 13% for whites. Furthermore, a metropolitan task force established in 2007 in Chicago to address barriers in access to quality mammography screening and recommended treatment for breast cancer shows promise and illustrates how multiple sectors of a community (74 area organizations) can come together to improve breast cancer care.[94]

CONCLUSIONS

Black women are disadvantaged in multiple dimensions of access to economic and social resources in society. We need to better understand the ways in which these risk factors, at multiple levels of exposure, combine over the life-course with social and psychological resources or exposures to predict the development and course of breast cancer. Effectively reducing breast cancer disparities for black women will thus require disentangling risk factors driven by racism from those driven by SES at these multiple levels (eg, individual, neighborhood, and health system). As discussed previously, some social exposures associated with being black in America, such as racism or SES, will be associated directly with breast cancer risk. In other cases, black race or SES will mediate breast cancer risk through another variable (eg, childhood adversity leading to adult obesity[46,47]). When one is investigating the social context of black women and their risk for breast cancer, however, it must be remembered that black women are not a monolithic category. Rather, they are a varied and diverse group that encompasses many cultural communities and socioeconomic strata. Being black in America is, therefore, not a risk category for developing breast cancer a priori but rather makes up an intersectionality, as Crenshaw[95] writes, of various cultural, social, economic, and biological factors that together give shape to the risk for breast cancer. Only when we can disaggregate the influences of the social factors affecting breast cancer risk related to race, SES, and other relevant dimensions of identity will we be able to develop truly effective interventions.

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AUTHOR CONTRIBUTIONS

David R. Williams: Conceptualization, methodology, investigation, original draft, writing–review and editing, project administration, and funding acquisition. **Selina A. Mohammed:** Conceptualization, methodology, investigation, data curation, writing–original draft, and writing–review and editing. **Alexandra E. Shields:** Conceptualization, methodology, investigation, data curation, writing–original draft, writing–review and editing, visualization, supervision, and project administration.

REFERENCES

1. DeSantis CE, Fedewa SA, Goding Sauer A, Kramer JL, Smith RA, Jemal A. Breast cancer statistics, 2015: convergence of incidence rates between black and white women. *CA Cancer J Clin.* 2016;66:31–42.
2. National Center for Health Statistics. Health, United States, 2013. Hyattsville, MD: US Department of Health and Human Services; 2014.
3. Dunn BK, Agurs-Collins T, Browne D, Lubet R, Johnson KA. Health disparities in breast cancer: biology meets socioeconomic status. *Breast Cancer Res Treat.* 2010;121:281–292.
4. Williams DR. The health of U.S. racial and ethnic populations. *J Gerontol B Psychol Sci Soc Sci.* 2005;60B:53–62.
5. Fu M, Todem D, Fu WJ, Ma S. A millennium bug still bites public health—an illustration using cancer mortality. http://arxiv.org/abs/1401.7686. Accessed April 8, 2015.
6. Brawley OW. Health disparities in breast cancer. *Obstet Gynecol Clin North Am.* 2013;40:513–523.
7. Dunnwald L, Rossing M, Li C. Hormone receptor status, tumor characteristics, and prognosis: a prospective cohort of breast cancer patients. *Breast Cancer Res.* 2007;9:R6.
8. Dunnwald LK, Doot RK, Specht JM, et al. PET tumor metabolism in locally advanced breast cancer patients undergoing neoadjuvant chemotherapy: value of static versus kinetic measures of fluorodeoxyglucose uptake. *Clin Cancer Res.* 2011;17:2400–2409.
9. Newman LA. Breast cancer disparities: high-risk breast cancer and African ancestry. *Surg Oncol Clin N Am.* 2014;23:579–592.
10. Banegas M, Li C. Breast cancer characteristics and outcomes among Hispanic black and Hispanic white women. *Breast Cancer Res Treat.* 2012;134:1297–1304.
11. Krieger N. History, biology, and health inequities: emergent embodied phenotypes and the illustrative case of the breast cancer estrogen receptor. *Am J Public Health.* 2012;103:22–27.
12. Trichopoulos D, Adami HO, Ekbom A, Hsieh CC, Lagiou P. Early life events and conditions and breast cancer risk: from epidemiology to etiology. *Int J Cancer.* 2008;122:481–485.
13. Williams DR, Mohammed SA, Leavell J, Collins C. Race, socioeconomic status, and health: complexities, ongoing challenges, and research opportunities. *Ann N Y Acad Sci.* 2010;1186:69–101.
14. Geronimus AT, Hicken MT, Pearson JA, Seashols SJ, Brown KL, Cruz TD. Do US black women experience stress-related accelerated biological aging?: a novel theory and first population-based test of black-white differences in telomere length. *Hum Nat.* 2010;21:19–38.
15. Geronimus AT, Hicken M, Keene D, Bound J. "Weathering" and age patterns of allostatic load scores among blacks and whites in the United States. *Am J Public Health.* 2006;96:826–833.
16. Gruenewald TL, Karlamangla AS, Hu P, et al. History of socioeconomic disadvantage and allostatic load in later life. *Soc Sci Med.* 2012;74:75–83.
17. Beckie TM. A systematic review of allostatic load, health, and health disparities. *Biol Res Nurs.* 2012;14:311–346.
18. Parente V, Hale L, Palermo T. Association between breast cancer and allostatic load by race: National Health and Nutrition Examination Survey 1999–2008. *Psychooncology.* 2013;22:621–628.
19. Tyrka AR, Price LH, Marsit C, Walters OC, Carpenter LL. Childhood adversity and epigenetic modulation of the leukocyte glucocorticoid receptor: preliminary findings in healthy adults. *PLoS One.* 2012;7:e30148.
20. Romens SE, McDonald J, Svaren J, Pollak SD. Associations between early life stress and gene methylation in children. *Child Dev.* 2015;86:303–309.
21. Perroud N, Paoloni-Giacobino A, Prada P, et al. Increased methylation of glucocorticoid receptor gene (NR3C1) in adults with a history of childhood maltreatment: a link with the severity and type of trauma. *Transl Psychiatry.* 2011;1:e59.
22. Steiger H, Labonte B, Groleau P, Turecki G, Israel M. Methylation of the glucocorticoid receptor gene promoter in bulimic women: associations with borderline personality disorder, suicidality, and exposure to childhood abuse. *Int J Eat Disord.* 2013;46:246–255.
23. Hompes T, Izzi B, Gellens E, et al. Investigating the influence of maternal cortisol and emotional state during pregnancy on the DNA methylation status of the glucocorticoid receptor gene (nr3c1) promoter region in cord blood. *J Psychiatr Res.* 2013;47:880–891.
24. Nesset KA, Perri AM, Mueller CR. Frequent promoter hypermethylation and expression reduction of the glucocorticoid receptor gene in breast tumors. *Epigenetics.* 2014;9:851–859.
25. Yin D, Morris C, Allen M, Cress R, Bates J, Liu L. Does socioeconomic disparity in cancer incidence vary across racial/ethnic groups? *Cancer Causes Control.* 2010;21:1721–1730.
26. Andaya AA, Enewold L, Horner MJ, Jatoi I, Shriver CD, Zhu K. Socioeconomic disparities and breast cancer hormone receptor status. *Cancer Causes Control.* 2012;23:951–958.
27. Williams DR, Priest N, Anderson NB. Understanding associations between race, socioeconomic status and health: patterns and prospects. *Health Psychol.* In press.
28. Anderson BM, MacLennan MB, Hillyer LM, Ma DWL. Lifelong exposure to n-3 PUFA affects pubertal mammary gland development. *Appl Physiol Nutr Metab.* 2014;39:699–706.
29. Krieger N, Kiang MV, Kosheleva A, Waterman PD, Chen JT, Beckfield J. Age at menarche: 50-year socioeconomic trends among US-born black and white women. *Am J Public Health.* 2014;105:388–397.
30. Behie AM, O'Donnell MH. Prenatal smoking and age at menarche: influence of the prenatal environment on the timing of puberty. *Hum Reprod.* 2015;30:957–962.
31. Lee JM, Appugliese D, Kaciroti N, Corwyn RF, Bradley RH, Lumeng JC. Weight status in young girls and the onset of puberty. *Pediatrics.* 2007;119:e624–e630.
32. Ong KK, Emmett P, Northstone K, et al. Infancy weight gain predicts childhood body fat and age at menarche in girls. *J Clin Endocrinol Metab.* 2009;94:1527–1532.
33. Mervish NA, Gardiner EW, Galvez MP, et al. Dietary flavonol intake is associated with age of puberty in a longitudinal cohort of girls. *Nutr Res.* 2013;33:534–542.
34. Walvoord EC. The timing of puberty: is it changing? Does it matter? *J Adolesc Health.* 2010;47:433–439.
35. Deardorff J, Fyfe M, Ekwaru JP, Kushi LH, Greenspan LC, Yen IH. Does neighborhood environment influence girls' pubertal onset? Findings from a cohort study. *BMC Pediatr.* 2012;12:27.
36. James-Todd T, Tehranifar P, Rich-Edwards J, Titievsky L, Terry MB. The impact of socioeconomic status across early life on age at menarche among a racially diverse population of girls. *Ann Epidemiol.* 2010;20:836–842.
37. Nielsen NR, Gronbaek M. Stress and breast cancer: a systematic update on the current knowledge. *Nat Clin Pract Oncol.* 2006;3:612–620.
38. Heikkila K, Nyberg ST, Theorell T, et al. Work stress and risk of cancer: meta-analysis of 5700 incident cancer events in 116 000 European men and women. *BMJ.* 2013;346:f165.
39. Hertzman C, Boyce T. How experience gets under the skin to create gradients in developmental health. *Annu Rev Public Health.* 2010;31:329–347.
40. Cohen S, Janicki-Deverts D, Chen E, Matthews KA. Childhood socioeconomic status and adult health. *Ann N Y Acad Sci.* 2010;1186:37–55.
41. Kuzawa CW, Sweet E. Epigenetics and the embodiment of race: developmental origins of US racial disparities in cardiovascular health. *Am J Hum Biol.* 2009;21:2–15.
42. Fuller-Thomson E, Brennenstuhl S. Making a link between childhood physical abuse and cancer. *Cancer.* 2009;115:3341–3350.

43. Morton PM, Schafer MH, Ferraro KF. Does childhood misfortune increase cancer risk in adulthood? *J Aging Health.* 2012;24:948–984.
44. Jacobs JR, Bovasso GB. Early and chronic stress and their relation to breast cancer. *Psychol Med.* 2000;30:669–678.
45. Li J, Vestergaard M, Obel C, et al. Antenatal maternal bereavement and childhood cancer in the offspring: a population-based cohort study in 6 million children. *Br J Cancer.* 2012;107:544–548.
46. Greenfield EA, Marks NF. Violence from parents in childhood and obesity in adulthood: using food in response to stress as a mediator of risk. *Soc Sci Med.* 2009;68:791–798.
47. Mamun AA, Lawlor DA, O'Callaghan MJ, Bor W, Williams GM, Najman JM. Does childhood sexual abuse predict young adult's BMI? A birth cohort study. *Obesity.* 2007;15:2103–2110.
48. Kinnunen TI, Luoto R, Gissler M, Hemminki E, Hilakivi-Clarke L. Pregnancy weight gain and breast cancer risk. *BMC Womens Health.* 2004;4:1–10.
49. Witek Janusek L, Tell D, Albuquerque K, Mathews HL. Childhood adversity increases vulnerability for behavioral symptoms and immune dysregulation in women with breast cancer. *Brain Behav Immun.* 2013;30(suppl):S149–S162.
50. Crosswell AD, Bower JE, Ganz PA. Childhood adversity and inflammation in breast cancer survivors. *Psychosom Med.* 2014;76:208–214.
51. Wildeman C, Emanuel N, Leventhal JM, Putnam-Hornstein E, Waldfogel J, Lee H. The prevalence of confirmed maltreatment among US children, 2004 to 2011. *JAMA Pediatr.* 2014;168:706–713.
52. Lanier P, Maguire-Jack K, Walsh T, Drake B, Hubel G. Race and ethnic differences in early childhood maltreatment in the United States. *J Dev Behav Pediatr.* 2014;35:419–426.
53. Finkelhor D, Ormrod R, Turner H, Hamby SL. The victimization of children and youth: a comprehensive, national survey. *Child Maltreat.* 2005;10:5–25.
54. Burdge GC, Lillycrop KA, Jackson AA. Nutrition in early life, and risk of cancer and metabolic disease: alternative endings in an epigenetic tale? *Br J Nutr.* 2009;101:619–630.
55. Xue F, Michels KB. Intrauterine factors and risk of breast cancer: a systematic review and meta-analysis of current evidence. *Lancet Oncol.* 2007;8:1088–1100.
56. Farvid MS, Cho E, Chen WY, Eliassen AH, Willett WC. Dietary protein sources in early adulthood and breast cancer incidence: prospective cohort study. *BMJ.* 2014;348:g3437.
57. Liu Y, Colditz GA, Rosner B, et al. Alcohol intake between menarche and first pregnancy: a prospective study of breast cancer risk. *J Natl Cancer Inst.* 2013;105:1571–1578.
58. Shields AE, Fortun M, Hammonds EM, et al. The use of race variables in genetic studies of complex traits and the goal of reducing health disparities: a transdisciplinary perspective. *Am Psychol.* 2005; 60:77–103.
59. Thoits PA. Personal agency in the stress process. *J Health Soc Behav.* 2006;47:309–323.
60. Sternthal MJ, Slopen N, Williams DR. Racial disparities in health: how much does stress really matter? *Du Bois Rev.* 2011;8:95–113.
61. Thomas CS. A new look at the black middle class: research trends and challenges. *Sociol Focus.* 2015;48:191–207.
62. Lewis TT, Cogburn CD, Williams DR. Self-reported experiences of discrimination and health: scientific advances, ongoing controversies, and emerging issues. *Annu Rev Clin Psychol.* 2015;11:407–440.
63. Taylor TR, Williams CD, Makambi KH, et al. Racial discrimination and breast cancer incidence in US black women: the Black Women's Health Study. *Am J Epidemiol.* 2007 166:46–54.
64. Cozier YC, Yu J, Coogan PF, Bethea TN, Rosenberg L, Palmer JR. Racism, segregation, and risk of obesity in the Black Women's Health Study. *Am J Epidemiol.* 2014;179:875–883.
65. Adam EK, Heissel JA, Zeiders KH, et al. Developmental histories of perceived racial discrimination and diurnal cortisol profiles in adulthood: a 20-year prospective study. *Psychoneuroendocrinology.* 2015; 62:279–291.
66. Miranda ML, Maxson P, Edwards S. Environmental contributions to disparities in pregnancy outcomes. *Epidemiol Rev.* 2009;31:67–83.
67. Brody JG, Kripke ML, Kavanaugh-Lynch MH, Rizzo J, Forman MR. Breast cancer and environmental research. *Science.* 2014;344: 577.
68. Antonovsky A. Health, Stress, and Coping. San Francisco, CA: Jossey-Bass; 1979.
69. Pargament KI, Smith BW, Koenig HG, Perez L. Patterns of positive and negative religious coping with major life stressors. *J Sci Study Relig.* 1998;37:710–724.
70. Chida Y, Steptoe A, Powell LH. Religiosity/spirituality and mortality. A systematic quantitative review. *Psychother Psychosom.* 2009;78: 81–90.
71. Kinney AY, Bloor LE, Dudley WN, et al. Roles of religious involvement and social support in the risk of colon cancer among blacks and whites. *Am J Epidemiol.* 2003;158:1097–1107.
72. Steffen PR, Hinderliter AL, Blumenthal JA, Sherwood A. Religious coping, ethnicity, and ambulatory blood pressure. *Psychosom Med.* 2001;63:523–530.
73. Van Ness PH, Kasl SV, Jones BA. Religion, race, and breast cancer survival. *Int J Psychiatry Med.* 2003;33:357–375.
74. Holman DM, Grossman M, Henley SJ, Peipins LA, Tison L, White MC. Opportunities for cancer prevention during midlife. *Am J Prev Med.* 2014;46:S73–S80.
75. Wu Y, Zhang D, Kang S. Physical activity and risk of breast cancer: a meta-analysis of prospective studies. *Breast Cancer Res Treat.* 2013; 137:869–882.
76. Nelson DE, Jarman DW, Rehm J, et al. Alcohol-attributable cancer deaths and years of potential life lost in the United States. *Am J Public Health.* 2013;103:641–648.
77. Yang S, Lynch JW, Raghunathan TE, Kauhanen J, Salonen JT, Kaplan GA. Socioeconomic and psychosocial exposures across the life course and binge drinking in adulthood: population-based study. *Am J Epidemiol.* 2007;165:184–193.
78. Naimi TS, Brown DW, Brewer RD, et al. Cardiovascular risk factors and confounders among nondrinking and moderate-drinking U.S. adults. *Am J Prev Med.* 2005;28:369–373.
79. Fillmore KM, Kerr WC, Stockwell T, Chikritzhs T, Bostrom A. Moderate alcohol use and reduced mortality risk: systematic error in prospective studies. *Addict Res Theory.* 2006;14:101–132.
80. Holmes MV, Dale CE, Zuccolo L, et al. Association between alcohol and cardiovascular disease: Mendelian randomisation analysis based on individual participant data. *BMJ.* 2014;349: g4164.
81. Knott CS, Coombs N, Stamatakis E, Biddulph JP. All cause mortality and the case for age specific alcohol consumption guidelines: pooled analyses of up to 10 population based cohorts. *BMJ.* 2015; 350:h384.
82. Millikan RC, Newman B, Tse CK, et al. Epidemiology of basal-like breast cancer. *Breast Cancer Res Treat.* 2008;109:123–139.
83. Lind JN, Perrine CG, Li R, Scanlon KS, Grummer-Strawn LM. Racial disparities in access to maternity care practices that support breastfeeding—United States, 2011. *MMWR Morb Mortal Wkly Rep.* 2014;63:725–728.
84. Edwards RC, Thullen MJ, Korfmacher J, Lantos JD, Henson LG, Hans SL. Breastfeeding and complementary food: randomized trial of community doula home visiting. *Pediatrics.* 2013;132(suppl 2): S160–S166.
85. Taveras EM, Gillman MW, Kleinman KP, Rich-Edwards JW, Rifas-Shiman SL. Reducing racial/ethnic disparities in childhood obesity: the role of early life risk factors. *JAMA Pediatr.* 2013;167: 731–738.
86. Aizer A, Currie J. The intergenerational transmission of inequality: maternal disadvantage and health at birth. *Science.* 2014;344:856–861.
87. Aschbacher K, Kornfeld S, Picard M, et al. Chronic stress increases vulnerability to diet-related abdominal fat, oxidative stress, and metabolic risk. *Psychoneuroendocrinology.* 2014;46:14–22.
88. Williams DR, Mohammed SA. Racism and health II: a needed research agenda for effective interventions. *Am Behav Sci.* 2013;57: 1200–1226.
89. Frederick CB, Snellman K, Putnam RD. Increasing socioeconomic disparities in adolescent obesity. *Proc Natl Acad Sci U S A.* 2014; 111:1338–1342.
90. Wheeler SB, Reeder-Hayes KE, Carey LA. Disparities in breast cancer treatment and outcomes: biological, social, and health system

determinants and opportunities for research. *Oncologist.* 2013;18: 986–993.
91. Roberts MC, Wheeler SB, Reeder-Hayes K. Racial/ethnic and socioeconomic disparities in endocrine therapy adherence in breast cancer: a systematic review. *Am J Public Health.* 2015;105(suppl 3): e4–e15.
92. van Ryn M, Burgess DJ, Dovidio JF, et al. The impact of racism on clinician cognition, behavior, and clinical decision making. *Du Bois Rev.* 2011;8:199–218.
93. Grubbs SS, Polite BN, Carney JJ, et al. Eliminating racial disparities in colorectal cancer in the real world: it took a village. *J Clin Oncol.* 2013;31:1928–1930.
94. Ansell D, Grabler P, Whitman S, et al. A community effort to reduce the black/white breast cancer mortality disparity in Chicago. *Cancer Causes Control.* 2009;20:1681–1688.
95. Crenshaw K. Demarginalizing the intersection of race and sex: a black feminist critique of antidiscrimination doctrine, feminist theory and antiracist politics. *Univ Chicago Leg Forum.* 1989;140:139–167.