

Ethnicity- and socio-economic status-related stresses in context: an integrative review and conceptual model

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Abstract There continues to be debate about how best to conceptualize and measure the role of exposure to ethnicity-related and socio-economic status-related stressors (e.g. racism, discrimination, class prejudice) in accounting for ethnic health disparities over the lifecourse and across generations. In this review, we provide a brief summary of the evidence of health disparities among ethnic groups, and the major evidence on the role of exposure to ethnicity- and SES-related stressors on health. We then offer a reciprocal and recursive lifespan meta-model that considers the interaction of ethnicity and SES history as impacting exposure to psychosocial adversities, including ethnicity-related stresses, and mediating biopsychosocial mechanisms that interact to result in hypothesized cumulative biopsychosocial vulnerabilities. Ultimately, group differences in the burden of cumulative vulnerabilities are hypothesized as contributing to differential health status over time. Suggestions are offered for future research on the unique role that ethnicity- and SES-related processes are likely to play as contributors to persistent ethnic health disparities.

Keywords Ethnicity · SES · Racism · Psychosocial adversities · Reserve capacity · Cumulative vulnerabilities

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Current epidemiologic evidence indicates a persistent disparity in health status, morbidity and mortality among many racial/ethnic minority groups compared to Caucasians (NCCDPHP 2004). Regardless of the health and social status criteria used, many African Americans, Hispanics, Native Americans, and South Pacific and South East Asians carry a disproportionate burden of morbidity and mortality and are overrepresented among those suffering from the greatest social disadvantage (Keppel et al. 2002). These groups also differ in the onset and trajectory of disorders and in treatment response, and these health differences appear to be heavily influenced by a confluence of psychosocial, behavioral and physiological factors, including differential exposure to and reactivity to stress (Myers et al. 2003), and limited access to quality health care (Smedley et al. 2003). Of these groups, African Americans appear to carry the greatest burden of morbidity and mortality from many different illnesses, including heart disease, hypertension, type 2 diabetes, certain types of cancer, adverse birth outcomes and HIV/AIDS (MMWR 2005; NCCDPHP 2004; CDC 2005a, b, 2006, 2007). Unfortunately, current health trends are anticipated to continue unabated, the overall health status of the United States will decline, along with the attendant increases in health care costs, and declines in the quality of life for all Americans. Therefore, one of the major public health challenges we face today is to identify the complex set of biopsychosocial factors that contribute to or maintain these persistent health disparities and to design innovative interventions to close the health gaps (Williams 1997; Myers et al. 2003).

In this paper we provide a selective review of the literature on stress exposure and other psychosocial and biobehavioral risk factors, including exposure to racism

and discrimination (Clark et al. 1999), as contributors to disease risk (See Myers et al. 2003 for a review). We also include a discussion of psychosocial reserve capacity as a risk mediator (Gallo and Matthews 2003) in helping to account for these ethnic health disparities. This review is informed by a multidimensional biopsychosocial perspective, and we argue that at the core of the ethnic health disparities is differential exposure to psychosocial adversities moderated by inadequate access to and control over essential material, psychological, social, and health care resources over time. We further contend that this disadvantageous stress-resource reserved capacity imbalance is created by social status-defining attributes of race/ethnicity and social class that define the social hierarchy and life opportunities. This disadvantaged health relationship is further maintained and enhanced through debilitating social environments (i.e. poverty and poor health care), and mediated through biological, behavioral and psychological pathways. The interplay of these factors is hypothesized to result in cumulative biopsychosocial vulnerability over the life span, which accounts, at least in part, for the cross-generational persistence of the health disparities that is documented in the epidemiologic literature.

This manuscript is organized into seven sections. First, we briefly review the evidence of ethnic health disparities, with specific emphasis on cardiovascular disease, type 2 diabetes, HIV/AIDS, and birth outcomes, as well as unequal access to quality health care. Second, we review current models of psychosocial adversities, paying particular attention to research on exposure to both generic life stresses that all persons face, as well as to the additional burden of social status-related stresses (i.e. socio-economic disadvantage, racism and discrimination) faced by persons of color and disadvantaged backgrounds. Third, we discuss the role of biological vulnerabilities and stress-response processes as mediating mechanisms through which exposure to psychosocial adversities confer risk for disease and dysfunction. Fourth, we review evidence of the role that processing of negative cognitions and emotions, and the clustering of health injurious behaviors play in mediating health risk. Fifth, we discuss how cultural, interpersonal and intrapersonal resources that constitute psychosocial reserve capacity are hypothesized as mediating vulnerability to psychosocial adversities. Sixth, we argue that the complex interplay of the burden of adversities, dysregulated biological stress responses, ineffective processing of negative thoughts and emotions, the clustering of health injurious behaviors, and lower psychosocial reserve capacity shapes a disadvantaged health trajectory through cumulative vulnerabilities over time to account for persistent ethnic health disparities. Finally, we discuss the

implications of this review and suggest directions for future research.

Ethnic health disparities

Current evidence indicates that the health of the United States has improved significantly in the last decade (Health United States 2005). However, this improvement has not been uniform, with many Caucasian Americans and Asian Americans enjoying a significant overall health advantage over members of other racial/ethnic groups. Despite limitations in the amount and quality of health data across ethnic groups (e.g. comparatively less data on Native American, Hispanic American, Asian American and Caucasian American subgroups because of the tendency to ignore important within-group differences), current epidemiologic evidence indicates significant and persistent ethnic group differences on virtually all major health status indicators in adults (NCCDPHP 2004; Williams and Jackson 2005), as well as in children (Chen et al. 2006). In this section, we provide a brief overview of ethnic health disparities in coronary heart disease (CHD), type 2 diabetes, HIV, birth outcomes, and healthcare access. Because of their greater burden of morbidity, we will use African Americans as an exemplar.

Heart disease

Although there has been improvement in the rate of Cardiovascular Disease (CVD) in the U.S. in the last decade, CHD continues to be the major cause of death in all ethnic groups (American Heart Association 1998, 2007). However, there are substantial ethnic differences in relative risk for CHD. For example, African Americans have higher rates of CHD and stroke than Caucasian Americans, Chinese and Japanese Americans exhibit higher rates of stroke but not CHD, Mexican Americans have higher prevalence of both stroke and CHD, and Native Americans have high rates of CHD (Forouhi and Sattar 2006). In addition, African Americans have a greater risk for complications from CHD and unstable angina, and higher CHD death rate, as well as more severe strokes and greater stroke mortality than Caucasian Americans (Health United States 2005; Keppel et al. 2002; Saunders and Ofili 2008). These disparities in CVD rates have been related directly to differences in the clustering of CVD risk factors, such as smoking, obesity, and limited physical activity (Hayes et al. 2005), as well as to differences on other risk factors, including access to effective CVD treatments (Smedley et al. 2003), and levels of visceral adiposity, insulin resistance, and novel risk markers such as C-reactive pro-

tein (CRP), adiponectin and plasma homocysteine (Forouhi and Sattar 2006).

Diabetes

More Americans than ever are suffering from non-insulin dependent or type 2 diabetes mellitus (NIDDM), which is the 6th leading cause of death in the U.S. (CDC 2005b). This disease is associated with a variety of disabling conditions, including enhanced risk for end-stage renal disease, blindness, lower extremity amputations, gum disease, CHD, stroke, complications of pregnancy, functional limitations, and increased mortality. There are substantial ethnic differences in the prevalence of this disease and its sequelae, with age-adjusted rates higher for Native Americans (18%), African Americans (15%) and Hispanic Americans (14%) than for Caucasian Americans (8%) (CDC 2005a, b).

HIV/AIDS

In the United States, the HIV/AIDS epidemic disproportionately affects African Americans. Of all newly diagnosed cases of HIV in 2005, 49% were African American compared with 31% who were Caucasian, 18% Hispanic, 1% Asian/Pacific Islander and <1% Native American (CDC 2007). Among women, 78% of those who are HIV-infected are African American (Hader et al. 2001), as are 65% of perinatally-infected infants (CDC 2006).

Reproductive outcomes

There is also ample evidence of persistent disparities in birth outcomes between African Americans and all other ethnic groups (Alexander et al. 1999; Geronimus 1992; Keppel et al. 2002). This discrepancy is evident on most maternal or child outcomes examined. African American women are more likely to die from ectopic pregnancies, and are three times more likely than Caucasian Americans to die in hospitals following hysterectomies. Compared to Caucasian Americans, African Americans have rates of low birthweight (LBW = 5 lb, 8 oz) and preterm delivery (PTD \leq 37 weeks gestation) that are almost twice as high, experience three times the rate of very low birthweight (VLBW \leq 3 lb, 4 oz) babies and four times the rate of very early delivery (i.e. <28 weeks gestation) (Keppel et al. 2002). Also, African American infants are three times as likely to die of causes attributable to perinatal events and are twice as likely to die in the first month of life as Caucasian infants (See Lu and Halfon 2003 for a review and discussion).

This African American–Caucasian differential is further complicated by the paradoxical finding that the infant mortality rate is lower among infants born to African American teens, the age-group associated with highest risk for adverse birth outcomes, compared to Caucasian Americans and to older African American women (Geronimus 2001, 2002). Also, while the low birthweight and infant mortality rates are higher in less educated and poorer women of all ethnic groups, the African American–Caucasian American differential in LBW and in infant mortality are smaller among the less educated and larger among the most educated (Shiono et al. 1997). This suggests that African American women derive less reproductive benefit from upward mobility than their Caucasian counterparts.

The birth outcome picture is even more interesting when the data on Hispanic Americans are examined. For example, although Hispanic women share many of the socioeconomic deprivations, burdens of morbidity and lifestyle risk factors as many African American women, and in some cases may be worse off because of the added stresses of immigrant status, Hispanic American women as a whole have birth outcomes that are comparable to those of Caucasian American women. Thus, despite being significantly poorer on average and having a poorer health behavior profile than Caucasian American women, Hispanic American women, especially Mexican Americans, have comparable infant mortality, low birthweight, and very low birthweight rates compared with Caucasians American women (Shiono et al. 1997). A number of sociocultural explanations have been offered to account for this “Hispanic Paradox”, including the role of selective migration and the protective effects of low acculturation, as well as the availability of effective family social networks of support that counteract other risk factors (Franzini et al. 2002; Lara et al. 2005; Gallo et al. 2008). It is important to note, however, that there is considerable debate about how to interpret these paradoxical findings because of the lack of longitudinal studies with adequate representation of the various Hispanic subgroups, as well as diversity with respect to socioeconomic status, nativity, level of acculturation, etc.

Obstetrical outcome data are also limited for the other ethnic groups, but the available evidence suggests that controlling for SES, the birth outcomes for Native American women are only slightly worse than for non-Hispanic Caucasian American women, and the outcomes for Asian/Pacific Islanders are more favorable overall. However, our ability to gain an accurate picture of birth outcomes across ethnic groups is limited by the tendency to report summary data by aggregated ethnic groups which underestimates possible subgroup differences (e.g. differences between

Mexican Americans, Cubans, Puerto Ricans, Dominicans, and women from Latin America).

Health care access

There is compelling evidence that differences in access and utilization of quality health care are an important contributor to the disproportionate ethnic burden of morbidity and mortality (Fiscella et al. 2002; Williams and Collins 2002; Klonoff 2008). In their comprehensive review, Smedley et al. (2003) document the evidence indicating ethnic differences in availability, access and utilization of a range of health services, and how patient level variables (e.g. health beliefs, differences in symptom recognition and tolerance, system mistrust, etc), health care system variables (e.g. costs, availability of treatment procedures in ethnic service hospitals, language barriers, etc), and health care process level variables (e.g. physicians' biases in recommending certain treatments) contribute to racial/ethnic differences in care (also see Klonoff this issue).

How do we account for these health disparities?

Health outcomes, whether they are chronic conditions such as heart disease, diabetes or cancer, discrete events such as acquiring a life threatening infection such as HIV or the birth of a pre-term infant, or whether and when the decision to seek medical care is made, are the byproducts of the complex interaction of many factors over time. As we have suggested in previous work (Myers et al. 2003; Myers and Hwang 2004), although there is general consensus within the scientific community on the major factors that contribute to disease risk, there remains little synergy between the biological, psychosocial, cultural and behavioral explanations that have been offered to account for ethnic disparities in health. Therefore, we propose a modified biopsychosocial model of stress and disease as a useful conceptual tool for integrating the disparate literatures and advancing our understanding of ethnic group differences in health and disease. This model was built on the work of several stress and life-course theorists, including McEwen and colleagues' work on allostatic load (McEwen 1998, 2004; McEwen and Seeman 1999), the work by Elder and colleagues on life course and intergenerational transmission of risk (Elder 1998; Elder and Crosnoe 2002; Wickrama et al. 1999), Singer & Riff's work on life history method for estimating risk for negative health outcomes from the balance or imbalance of life adversities and advantages (Singer and Riff 1999), Geronimus' work on the weathering hypothesis (Geronimus 1992), and Clark et al. (1999) biopsychosocial model of racism as a stressor

for African Americans. In the initial model we made explicit that race/ethnicity, social class and environments (i.e. community, family, work, etc.) are interacting contributors to chronic stress burden that includes both generic life stresses and ethnicity-related stresses (i.e. racism, discrimination). This chronic burden of stress, in turn, is hypothesized as contributing to disease through a biological pathway that includes constitutional predispositions or vulnerabilities, the triggering of physiological response mechanisms (i.e. allostasis), and allostatic load (i.e. wear and tear on the system). This allostatic load, in turn, is hypothesized to contribute overtime to cumulative vulnerability, which ultimately results in disease and dysfunction. The model also acknowledged that the hypothesized stress-biological processes-disease pathway is likely moderated by a number of psychosocial and behavioral factors, including psychological characteristics (e.g. personality dispositions), lifestyle factors (e.g. diet, exercise, smoking, substance use), stress appraisal and coping strategies used, and by the availability and use of social resources (See Myers and Wang 2004 for a review). Therefore, it is all of these factors operating synergistically overtime that we hypothesize to result in differential health status and health trajectories for the different racial/ethnic and social class groups.

We expand and update this earlier model and propose a reciprocal and recursive lifespan biopsychosocial model of cumulative vulnerability and minority health. This revised model includes consideration of the psychosocial reserve capacity model of Gallo and Matthews (2003), the health care disparities model of Smedley et al. (2003), the more recent reviews of the impact of racism and health by Brondolo et al. (2003), Paradies (2006), and Mays et al. (2007), and the reviews and conceptual work by Hertzman (2004) and Lu and Halfon (2003) on life-course contributions to ethnic health disparities. In this new meta-model, which is depicted in Fig. 1, we propose that the persistent racial/ethnic health disparities can best be understood as the byproducts of the complex interactions between race/ethnicity and SES factors whose effects over the lifespan are mediated through 6 primary pathways: (1) long-term chronic exposure to psychosocial adversities, (2) psychosocial reserve capacity, (3) cognitive-emotional processing, (4) clustering of health injurious behaviors, (5) biological, and (6) health care pathways. We hypothesize that these paths intersect in recursive and reciprocal ways to create cumulative biopsychosocial vulnerabilities that ultimately shape the disadvantaged health trajectories of racial/ethnic minority groups over the lifespan. It is important to note that this meta-model should be considered as a broad conceptual framework for thinking and a guide for future programmatic research that investigate more discrete and testable models.

LIFESPAN BIOPSYCHOSOCIAL MODEL OF CUMULATIVE VULNERABILITY AND MINORITY HEALTH

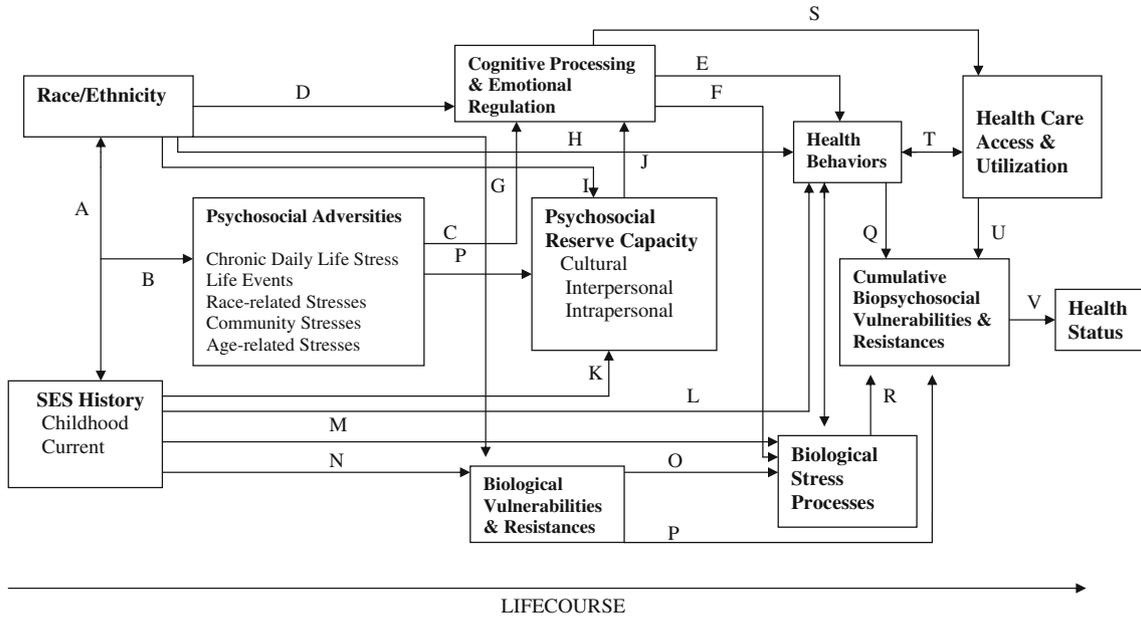


Fig. 1 This is a reciprocal and recursive model of the complex relationships between race/ethnicity and SES on psychosocial adversities, reserve capacity and cumulative vulnerabilities in predicting health status over the lifespan. The model posits that race/ethnicity and SES history interact (path A) over the lifespan to predict health outcomes by shaping exposure to psychosocial adversities (path B) through cognitive-emotional (path D), behavioral (paths E, H & L), and biological pathways (paths F, M, N & O). The

model also hypothesizes that race/ethnicity’s and SES history’s effects on health are mediated through Reserve Capacity (paths A, B, P, I, K), which also affects cognitive-emotional processing, and that health behaviors, health care access and utilization and allostatic load lead to the development of cumulative biopsychosocial vulnerabilities (paths P, Q, R, S, Q, T and U), and these, in turn, ultimately predict health status (path V) over the lifecourse

Social status stresses, psychosocial adversities and health

Perhaps the most important factor underlying these persistent racial/ethnic health disparities is the overrepresentation of the most affected groups among the lowest social classes. In fact, research in the U.S. and other industrialized societies indicates a consistent pattern of findings linking SES in a monotonic relationship with diverse health outcomes, including birth outcomes, life expectancy, chronic disease rates, quality of life, and mortality rates (Adler et al. 1994). Part of the SES disparities in health, at least at the lower SES levels, is due to differences in the distribution of material and psychological resources, environmental risks, the clustering of risk behaviors, inadequate health care, etc. (Link and Phelan 1995; Lynch et al. 2000; Marmot et al. 1997; Williams and Collins 2002). However, SES health differentials are evidenced at all steps in the gradient, including among the most affluent (Adler et al. 1994). Gallo and Matthews (2003) argue that these SES-related health disparities are likely mediated through a number of psychosocial, biological and behavioral pathways, including greater vulnerability to negative cognitions and emotions, reductions in individual reserve capacity,

and clustering of health risk behaviors over time (See Gallo and Matthews 2003 for a review of the relevant literature). In a recent paper, Geronimus et al. (2006) reported results of their analysis of allostatic load scores of African American and Caucasian American men and women from the National Health and Nutrition Examination Survey (NHNES). They found that African Americans had a greater probability of a high allostatic load score than Caucasian Americans at all ages, especially among those that were poor and 35–64 years of age. They also noted that poor and non-poor African American women had the highest scores compared to their male counterparts. Furthermore, and contrary to expectations, these ethnic and gender differences were not explained by poverty. These data argue for conceptualizing ethnic group health disparities as by-products of the interaction of ethnicity, SES and gender as social status variables rather than simply as the product of either variable alone (*bidirectional pathway A in the model*). Thus, there is growing recognition in the field that both long-term exposure to socio-economically disadvantaged environments and ethnic-minority status are associated with disproportionate risk for adverse health outcomes. In fact, the greatest burden of medical morbidity and mortality is among those who are dually disadvantaged

by poverty and ethnic minority status, and this is especially true for poorer African Americans who have the worst overall health profile of all ethnic and SES groups in the U.S. (Williams 1997; Williams and Jackson 2005).

Several studies have indicated that those from the lower social classes, especially ethnic minorities, often report a greater number of negative life events, greater and more frequent exposure to “generic life stressors” (i.e. stressors that are a usual part of modern life—financial, occupational, relationships, parental, etc.) (Gallo and Matthews 2003), perceive these events as more stressful and report greater psychological distress from these stressful life experiences (Collins et al. 1998; Chen and Matthews 2001) than their Caucasian American counterparts. As such, therefore, they are likely to be particularly vulnerable to the long term effects of high allostatic load because of their relative social position (Geronimus et al. 2006) (*pathway A, B in the model*).

On the other hand, several other studies have qualified this assertion and noted that low SES is associated with fewer, but more severe, daily hassles and life events (Gryzwacz et al. 2004), that this effect of disadvantage may be specific to race/ethnicity, but not to gender or sexual orientation (Meyer et al. 2008), and that some people may even become less reactive to social disadvantage (Nguyen and Peschard 2003) perhaps because they develop more effective, context-specific coping resources. These studies suggest the need for a more complex analysis of social disadvantage and health that considers possible differences in relative risk for adverse health outcomes as a function of ethnicity, gender, sexual orientation, and we would also suggest considering age, level of acculturation, generational status, etc. as additional moderators.

These social status effects are further complicated by exposure to racism and discrimination. Much of this work has focused on the impact of exposure to racism and discrimination in African Americans (Clark et al. 1999; Williams et al. 2003; Paradies 2006; Mays et al. 2007), although more recent work has been extended to include other ethnic groups in the U.S. and in other countries (Moradi and Risco 2006; Harris et al. 2006). Studies by Brondolo and colleagues (2005; Broudy et al. 2007) using daily diary methodology report that persons of color have relatively frequent experiences of discrimination, that these experiences are linked to more anger, are associated with greater intensity of their ratings of social interactions as harassing, exclusionary and unfair, and appear to increase expectancy of future discrimination. Their results suggest that racism and discrimination are not simply another stressor because they have the capacity to condition the appraisals of both current and future interpersonal interactions, especially interactions that are ambiguous (i.e. incidents when racism is suspected, but difficult to confirm)

(Brondolo et al. 2005; Bennett et al. 2004). Thus, these stressful experiences not only can add to the person’s overall stress burden, but may also serve to exacerbate the impact of other life stresses (e.g. job loss, legal difficulties, etc.).

Clark et al. (1999) also argue that studies of racism and discrimination-related stresses should not focus exclusively or primarily on more “objective” experiences and ignore or underestimate the importance of exposure to more subtle racism (i.e. micro insults) because these involve some degree of subjectivity. In fact, it is how experiences are appraised subjectively that determines the magnitude of the response. Therefore, it is reasonable to expect greater psychological and physiological reactivity and greater allostatic load in those who report greater exposure to both objectively measurable chronic and episodic stressors as well as subjectively experienced greater exposure to racism-related stresses (Harrell et al. 2003).

It is also likely that ethnic individuals and groups will differ in the degree to which they have developed “racial” filters, cognitive schemas or scripts that mediate how they interpret and respond to experiences that may be “racially meaningful” (*pathway D in the model*). Some groups (e.g. African Americans) who may have more politicized racial identities and thus more sensitive racial filters and lower response thresholds that may predispose them to interpret a wider range of experiences and events as “racially meaningful” and have a more intense reaction to them (Chen and Matthews 2001). This would be especially true for ambiguous events, which would increase their psychological salience and increase their adverse impact on health and well-being (Bennett et al. 2004).

It is also true that there are substantial individual differences in response to stressors, including racism-related stressors, and such factors as constitutional predispositions, cognitive and emotional processing, resources for coping, and psychological and behavioral factors are likely to mediate or moderate their effects on health and well-being (Myers et al. 2003; Gallo and Matthews 2003; Paradies 2006). Unfortunately, although there is considerable debate about racism and its effects, there continues to be inadequate attention investigating factors that might moderate or mediate the effects of exposure to SES- and race/ethnicity-related stresses on health.

Psychosocial reserve capacity, cognitive-emotional processing and health

Gallo and colleagues (Gallo and Matthews 2003; Gallo et al. 2005) provide a comprehensive review of the extant literature and propose a framework for accounting for SES-related health disparities. They argue that health disparities

can be explained, at least indirectly, by the strength of the associations between low SES and such negative cognitive-emotional factors as hostility, anxiety, depression and feelings of hopelessness (*pathways C, G and D in the model*). Thus, persons from disadvantaged backgrounds are exposed to more chronic stressors and greater likelihood of experiencing more hostility, anxiety, depression and hopelessness, all of which in turn, have been linked to adverse health outcomes (Hatch and Dohrenwend 2007) (*pathway J in the model*). These SES-related stressors are exacerbated for persons of color, such that low SES and ethnic minority status may combine to create higher chronic stress burden that accumulates to contribute to disproportionate burden of morbidity and mortality over the lifecourse (Myers et al. 2003; Myers and Hwang 2004). However, at this point it is unclear whether the SES and ethnicity effects combine additively or synergistically, which is an important question for future research.

Gallo and Matthews (2003) also argue, using concepts from resource models of stress and coping (Hobfoll 2001) that the availability of tangible, cultural, interpersonal and intrapersonal resources create a reserve capacity that can moderate or mediate the negative impact of life stresses over time (*pathways I, K and P in the model*). They argue that low SES confers risk for adverse health outcomes by reducing reserve capacity for coping with stress. This is consistent with the evidence of the role of social resources in moderating adverse life circumstances over the lifecourse (Singer and Ryff 1999). We take this argument further and suggest that persons with the dual burdens of a disadvantaged socio-economic background and the additional burden of race/ethnicity-related stressors should be at even greater risk of more limited access to tangible (e.g. income, childcare, transportation), interpersonal (e.g. supportive social relationships), intrapersonal (i.e. optimistic future orientation, personal agency), socio-cultural (e.g. supportive family networks, familismo, biculturalism, strong ethnic identity), and health care (i.e. access to quality care) reserve resources for coping. It is also very likely that the demands from chronic exposure to social adversities require the overutilization of limited resources and few opportunities to replenish those resources. Thus, it is useful to consider many of those with excess social status burdens to be in a state of relatively chronic resource deficit, and as such, at disproportionate risk for adverse health and functional outcomes.

Health behaviors and health outcomes

Ethnicity and SES also exert negative effects on health through health behaviors. There is substantial evidence of

greater clustering of health undermining behaviors, such as smoking, poor diet, limited physical activity, obesity, use/abuse of recreational drugs and alcohol, high risk sexual behaviors, and limited health care access and utilization among the poor and persons of color (NCCDPHP 2004; Henderson et al. 2007) (*pathways H and L in the model*). In addition, recent studies have found that perhaps because of their excess burden of other risk factors, these risky health behaviors have greater negative effect on low SES persons of color than on Caucasian Americans. Thus, for example, while there are ethnic differences in smoking compared to Caucasian Americans (i.e. Fewer African Americans, especially women, smoke compared to their Caucasian American counterparts), minorities who smoke have higher smoking-related mortality rates than Caucasian Americans (Haiman et al. 2006). At least for African Americans, the smoking-related mortality differences may be due, in part, to the fact that African Americans who smoke are likely to smoke more cigarettes and to prefer menthol cigarettes than Caucasian American smokers (68.9 vs. 22.4% of smokers) (Giovino et al. 2004) and have higher levels of serum cotinine at all levels of cigarette smoking than Caucasian and Hispanic Americans (Caraballo et al. 1998). The latter findings suggest that there may be a number of factors (e.g. stress reactivity) that may contribute to ethnic group differences in the way cigarette smoke, nicotine and other additives in cigarettes are metabolized.

Biological vulnerabilities, stress response processes and health

There is a substantial body of evidence that exposure to psychosocial adversities impact on health through the dysregulation of a variety of biological mechanisms (*pathways G, M, N and O in the model*), and that there are some documented ethnic group differences in apparent biological vulnerabilities that enhance risk for adverse health outcomes (i.e. hypertension in African Americans, diabetes mellitus in some Native American groups, cancer in some Asian American groups, etc.). Thus, macro-social factors such as the concentration of poverty in minority communities due to residential segregation and chronic social status stresses have been associated with persistently higher cortisol and other glucocorticoids, higher blood pressure reactivity and heart rate variability, corticotropin-releasing hormone levels, as well as inflammatory and immunologic processes (Massey 2004). All of these effects over time are believed to aggregate to contribute to higher allostatic load (McEwen 1998, 2004; Geronimus et al. 2006) and greater vulnerability to disease and other adverse health outcomes such as hypertension and coronary heart

disease (Brondolo et al. 2003; Harrell et al. 2003), adverse birth outcomes (Sandman et al. 1997; Wadhwa et al. 1998; Dunkel-Schetter 1998), and psychiatric disorders (McEwen 2004). There is suggestive evidence of some group differences in the distribution of genetic factors (e.g. sodium conservation trait among African Americans) (See Grim et al. 1995 for a review of this evidence) that confer vulnerability to these adverse environmental conditions that are also likely to contribute to the pattern of persistent health disparities.

Recent evidence is also available linking race-based discrimination and brain function using sophisticated brain imaging methods focusing on the amygdala and the anterior cingulate cortex (See Mays et al. 2007 for a review of this evidence). Collectively, this evidence points to the opportunity to further elucidate complex biopsychosocial pathways through which exposure to chronic social adversities over time contribute to the disproportionate burden of disease and dysfunction in racial/ethnic groups.

Disparities in health care

In their comprehensive Institute of Medicine report on “*Unequal Treatment: Confronting Racial and Ethnic Disparities in Health Care*”, Smedley et al. (2003) review the substantial body of research that document racial/ethnic differences in access, utilization, and quality of health care. These differences in the quality of care received persist even after accounting for differences in access-related factors (e.g. health insurance), clinical needs, treatment preferences, and the appropriateness of interventions, and are an independent contributor to the persistent racial/ethnic health disparities. While there is considerable debate about the reasons for these health care disparities, Smedley et al. (2003) offer a multi-dimensional explanatory model that recognizes the contribution of both individual and health-care system-level variables as contributing to racial/ethnic differences in care. Their analysis includes patient level variables (e.g. health beliefs, differences in symptom recognition and tolerance, system mistrust, etc), health care system variables (e.g. costs, imbalances between level of patient demand and the availability of resources, availability of treatment procedures in hospitals and clinics that serve primarily communities of color, language barriers, etc), and health care process level variables (e.g. physicians’ racial and social class prejudices and biases in recommending certain treatments, ineffective physician-patient communication, etc.). As such, efforts to reduce these disparities in care will require interventions to produce changes at all of these levels.

Cumulative vulnerabilities and health over the lifecourse

Finally, we argue that in the final analysis racial/ethnic health disparities are likely the byproduct of the reciprocal, recursive and synergistic interaction among the major factors in the model over the lifecourse. We hypothesize that chronic exposure to social adversities exert their adverse effects, mediated through negative cognitive-emotional processing, clustering of health risk behaviors, reduced psychosocial reserve capacity for coping, excess allostatic load and inadequate health care. In turn, these are hypothesized to lead to cumulative biopsychosocial vulnerabilities over the lifespan, which ultimately result in greater burden of medical morbidity and mortality (*pathways Q, R, S, T, U and V in the model*). We also suggest that the racial/ethnic groups with the worst health profiles (e.g. African Americans, Puerto Ricans, Native Americans, Cambodians, Hmong, Pacific Islanders) are likely to be those with the greatest burden of adversities, highest allostatic loads, weakest reserve capacity and poorest health care histories. These latter hypotheses remain to be tested and will require comprehensive, lifecourse studies that include consideration of issues such as timing of exposure to adversities (e.g. childhood vs. adult exposure), chronicity of exposure (i.e. life events vs. exposure to unremitting stressors), the clustering of adversities and risk factors, and whether these factors exert their effects additively or synergistically. We agree with Hertzman (2004), who notes the need to consider latency of effects (i.e. exposure at one point in the lifecourse and health expression at a later point), as well as Singer and Ryff (1999) and Gallo and Matthews (2003) whose work underscores the importance of considering the balance between the cumulative burden of adversities and the resources for coping or reserve capacity in predicting the negative health impact of these life adversities (See Singer and Ryff 1999, Hertzman 2004, and Gallo and Matthews 2003 and Gallo et al. 2005 for more detailed discussion of these issues).

Implications for future research and interventions

As a metamodel, the proposed lifespan biopsychosocial model of cumulative vulnerability and minority health is designed as a framework that identifies a number of hypothesized pathways leading to health disparities rather than a model that can be tested directly in a single study. Ideally, this framework will stimulate the development and testing of smaller models. For instance, we suggest that race/ethnicity-related stressors not only make independent contributions to greater stress burden (additive effect), but may also exacerbate the impact of other life stressors (synergistic effect). Although there is evidence to support

the former, support for the latter remains to be demonstrated. Similarly, while there is a strong body of evidence demonstrating the impact of exposure to psychosocial adversities on the dysregulation in some biological mechanisms, few studies have investigated the impact of such life adversities on multiple mechanisms simultaneously over time. Further, while there continues to be attention focused on many of these questions among African Americans, much of this work continues to focus on low income samples which preclude our ability to disentangle the independent effects of ethnicity and SES from their interaction. Most studies also give less systematic attention to other racial/ethnic groups and to subgroups within the major ethnic groups, which limits the ability to test whether ethnic groups and subgroups within each ethnic group differ in the degree to which the hypothesized pathways are confirmed or disconfirmed. For example, do ethnic groups and sub-groups within each differ in reserve capacity, and are these differences linked to differences in health status and health trajectories over time?

This model may also be useful in informing the development of interventions targeted at modifying these mediating pathways (See Lu and Halfon 2003 for suggestions of how resource building interventions may be effective in reducing disparities in birth outcomes). At present, there are many health risk reduction and health promotion interventions targeting health behaviors mainly in low SES ethnic groups. However, there are few studies that we are aware of that are specifically designed to enhance the development or replenishment of psychosocial reserve capacity for coping, either singly or as part of a multi-component intervention that also targets other risk factors in these groups. Finally, it is apparent that the ultimate test of models such as this will require longitudinal studies with large samples and adequate ethnic and SES representation, sophisticated methodologies and experienced multi-disciplinary research teams or consortia (e.g. the MacArthur Network). While such an expensive and ambitious enterprise will face many challenges, especially in the current fiscal and political climate, smaller more targeted studies are clearly feasible and can serve as the important first steps in building the foundation for the larger research effort. We hope that the ideas shared here will stimulate more discussion of these issues and guide future studies.

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