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“White Box” Epidemiology and the Social Neuroscience of Health Behaviors: The Environmental Affordances Model

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Abstract

Crucial advances have been made in our knowledge of the social determinants of health and health behaviors. Existing research on health disparities, however, generally fails to address a known paradox in the literature: While blacks have higher risk of medical morbidity relative to non-Hispanic whites, blacks have lower rates of common stress-related forms of psychopathology such as major depression and anxiety disorders. In this article we propose a new theoretical approach, the Environmental Affordances Model, as an integrative framework for the origins of both physical and mental health disparities. We highlight early empirical support and a growing body of experimental animal and human research on self-regulatory health behaviors and stress coping that is consistent with the proposed framework. We conclude that transdisciplinary approaches, such as the Environmental Affordances Model, are needed to understand the origins of group-based disparities to implement effective solutions to racial and ethnic group inequalities in physical and mental health.

Keywords

stress-buffering effects; race/ethnicity; mental health; allostatic load

Crucial advances have been made over the past five decades in our knowledge of the social determinants of health. While it is established that the major causes of preventable medical morbidity and mortality in mid and late life are related to behavioral factors such as smoking, diet, alcohol and drug use, and physical inactivity, these behaviors, in and of

themselves, do not fully explain social disparities in health (Lantz et al. 2001). The source of this missing variance is thought to be attributable to the contextual physical and social environment (e.g., neighborhood characteristics) (LaVeist et al. 2011) and the psychological stress of social disadvantage (Lantz et al. 2005). Existing models of the origin of social disparities in health, however, generally fail to acknowledge or address a known paradox in the health disparities literature: While pronounced racial/ethnic disparities in physical health (e.g., cardiovascular disease, cancer, type 2 diabetes) favor non-Hispanic whites over blacks (and most other minority groups), blacks have lower rates of common forms of psychopathology (e.g., major depression and anxiety disorders) than whites (Breslau et al. 2006). This phenomenon is illustrated by Figure 1, derived from the National Comorbidity Survey–Replication, a nationally representative population-based study of U.S. adults.

As a complement to existing models of health disparities, in this article we put forth the Environmental Affordances (EA) Model, which focuses on the intersection of stress, health behaviors, mental health, and physical health. We identify a set of interrelated processes and pathways that link social structures and context, stress, and health behaviors to both physical and mental health. We also discuss the limitations of existing models of health disparities, synthesize the theoretical evidence and limited but intriguing empirical support heretofore for this proposed new framework, and outline the implications of this model for efforts to address health promotion among socially disadvantaged groups.

The overall focus and impetus of the EA Model is to offer a perspective with which to reconcile the paradoxical empirical evidence that indicates lower rates of stress-related psychopathology such as depressive and anxiety disorders among blacks relative to non-Hispanic whites, despite large disparities among these racial groups in medical morbidity and mortality in mid and late life (Mensah et al. 2005). Common explanations for the observation in Figure 1 generally invoke either (1) measurement error (i.e., existing measures do not adequately capture the psychopathology of blacks as well as they do of whites), (2) positive coping (i.e., blacks are more likely to use religion or other sources of social support which protect against psychopathology), or (3) sampling bias (i.e., a disproportionate number of blacks are in institutions, and thus the community population is biased to be healthy).

However, there is limited support for these explanations. First, surveys that utilize validated, structured diagnostic interviews (e.g., the National Comorbidity Survey, the National Survey of American Life, the Epidemiologic Catchment Area [ECA] Study, the National Epidemiologic Survey on Alcohol and Related Conditions) consistently indicate that blacks have lower risk of most common psychiatric disorders, particularly mood and anxiety disorders, relative to non-Hispanic whites (Breslau et al. 2006; Schwartz and Meyer 2010). Further, it has been empirically demonstrated that these metrics assess psychopathology just as well in blacks as they do in whites (e.g., Williams et al. 2007) and that the modest measurement inconsistencies that do exist are simply insufficient to explain the black-white gap in psychopathology (Breslau et al. 2008). Second, although positive coping strategies such as religious participation and prayer are more common among blacks, the evidence that these strategies, in and of themselves, are sufficient to protect against the development of psychopathology is weak (Chatters et al. 2008). Religiosity and social support are also associated with better physical health outcomes, including lower risk of mortality (Glass et al. 1999), and thus this explanation would predict that blacks would have both better mental and physical health status relative to whites (contrary to the empirical data in Figure 1). Finally, although a disproportionate number of blacks are in jail or prison, the majority of these individuals are young men, a group that has a low base rate of depressive and anxiety disorders generally (Williams and Earl 2007). Also, surveys show that even within these institutions blacks do not have higher rates of psychopathology than whites (Youman et al.

2010). In sum, we believe that these explanations of the consistent black-white differences exemplified by Figure 1 are insufficient and that this empirical observation requires new theoretical perspectives.

Understanding the social, psychological, and biological mechanisms that produce this “epidemiological paradox” is the central motivating theme of the EA Model. We position this model as a process-based, transdisciplinary framework focused on explicitly interrogating the interactions among societal and individual-level factors. We believe that this model facilitates the development of testable hypotheses as to how these interactions might operate. These predictions are informed by our understanding of the social and physical environment and the psychology and biology of stress and health behaviors. We conclude that process-based approaches to the etiology of health disparities, such as the EA Model, can inform effective ameliorative strategies to reduce racial group disparities in health.

EXISTING MODELS OF THE ORIGINS OF SOCIAL DISPARITIES IN HEALTH

There have been numerous streams of research situated at the intersection of biology, epidemiology, sociology, and psychology, aimed at integrating social and psychological determinants of health. The EA Model draws on three approaches in particular: the fundamental cause model, the stress process framework, and the socio-biological perspective. It is difficult to draw stark distinctions among these frameworks; careful examination makes clear that there are many examples of work in this area that cross boundaries. Highlighting differences in these approaches, however, can help identify the broad range of factors used to understand health disparities. The fundamental cause model emphasizes the effect of resources on individuals' ability to take action to avoid risks or address health status directly. The stress process framework focuses on the interrelationship among the social environment, stress, and social resources that either mediate and/or moderate the association between stress and health. Finally, the socio-biological perspective aims to identify the specific pathways that may link social conditions to health outcomes; that is, how does the social environment get under the skin? Here we briefly describe how these three frameworks inform the EA Model.

The fundamental cause framework directs attention to the overarching role played by socioeconomic position (SEP) in explaining health inequities (Link and Phelan 1995; Phelan, Link, and Tehranifar 2010). The resources and social connections encompassed by SEP (e.g., education, income, occupation, and residence) both facilitate the avoidance of health risks and provide support if risks are encountered. As a result, SEP is postulated to influence both exposure to and consequences from health risk factors (Link and Phelan 1995). This framework lays the foundation for the EA Model by articulating the role that social position and context play in influencing individual health and behavior.

The stress process model focuses on explaining the link between an individual's position in a set of social institutions and systems of stratification and likely exposure to stress and, in turn, the role played by various social and psychological resources in ameliorating stress or mediating between stress and health (House 2002; McLeod 2012; Pearlin and Schooler 1978; Thoits 2011). The range of resources is broad (e.g., social relations and networks, social support, mastery, and religiosity); the specific nature of the associations between stress and psychosocial resources (e.g., direct, mediating, or moderating) are thought to vary by both individual (e.g., age, race/ethnicity, and gender) and contextual characteristics (e.g., poverty) (House 2002). There are both behavioral and psychological mechanisms underlying how social factors and stress interact. For example, social networks can encourage health-promoting (or -harming) behaviors as a means to cope with stress (Umberson, Crosnoe, and

Reczek 2010). Also, social relations can promote psychological resources such as self-efficacy that have both direct and indirect influences on health (Thoits 2011). This framework highlights the dynamic role of stress and coping in the origin of health and health disparities.

Finally, although previous research has acknowledged the biological correlates and implications of stress, recent streams of work more fully incorporate the measurement and conceptualization of the biological links between social context and health. For example, prolonged emotional distress may have direct consequences for physical health through processes such as allostasis and allostatic load (McEwen and Seeman 1999; Miller, Chen, and Cole 2009). As evidence of the growing importance of using biology to understand population health, biological measurements are now included in many household surveys, and as a corollary many clinical studies have adopted a range of psychosocial assessments (Finch and Vaupel 2001; Harris, Gruenewald, and Seeman 2007). In addition, there has been formal and informal collaboration among social epidemiologists, sociologists, and biologists resulting in ways of conceptualizing the markers of biological dysfunction, which may account for the link between social adversity and health (McEwen and Seeman 1999; Miller et al. 2009).

To date these parallel frameworks have been extremely fertile in broadening our understanding of social disparities in health. We suggest, however, that these perspectives do not fully explain important aspects of racial disparities in mental and physical health. Under fundamental cause, stress process, and socio-biological frameworks, groups with extensive exposures to social and material stressors should have both poor physical health and poor mental health (Schwartz and Meyer 2010). This prediction is not empirically supported, as discussed above and illustrated by Figure 1. We put forth the EA Model as a transdisciplinary approach that explicitly interrogates how individual behavior interacts with environmental resources and context to address this issue of racial disparities in mental and physical health.

THE ROLE OF SOCIAL PSYCHOLOGY IN UNDERSTANDING HEALTH BEHAVIORS

Environmental affordances and constraints (e.g., opportunities and inflexible barriers) promote particular psychological and behavioral responses to stress. Status-based characteristics such as SEP affect health-relevant decision-making processes through their confluence with the physical and social environment (Link and Phelan 1995). As a result, social psychology predicts both within- and between-group differences in psychological and behavioral responses to stress (e.g., Chong et al. 2009; McLeod 2012).

Despite this understanding of the importance of environmental influences on stress-coping strategies, in many fields universal approaches to promoting health behavior change are still applied (Schwarzer 2008). The availability of resources (i.e., material, social, personal, and cultural) largely determine which stress exposures tax individuals and thereby require a coping response (Pearlin and Schooler 1978). Empirical studies of stress coping often focus on positive strategies, such as emotional, material, or social support, and investigations that address negative coping strategies tend to do so in a manner that emphasizes individual psychological processes (Pearlin and Schooler 1978).

Many models of stress coping are also largely decontextualized from the physical environment. In reality, many individuals, particularly members of racial minority groups who reside in segregated and poor neighborhoods, are faced with an imbalanced ratio of positive to negative resources. These contexts cannot provide the social or material support

needed to address chronic stressors, such as financial hardship and broader uncontrollability and unpredictability in daily life. Pearlin and Schooler (1978) and many subsequent studies have demonstrated that effective coping strategies are unequally distributed in society and that social circumstances shape not only the capacity to cope but also the specific content of coping responses (Pearlin and Schooler 1978). For example, problem-focused and other forms of approach coping are more useful in high-control contexts (Glanz and Schwartz 2008). However, racial minorities and socioeconomically disadvantaged populations disproportionately live and work in environments characterized by uncertainty and disorganization (Wilson 2009).

We present the EA Model of the relationship between health behaviors and social context and physical and mental health as an example of “white box” epidemiology: an instance in which the mechanisms linking these factors are well articulated from experimental and laboratory data yet the implications and applications of these mechanisms to population health are in the nascent stages. As described below, a key advance of this framework is that it is heavily informed by the neurobiological correlates and consequences of stress and self-regulatory behaviors within the context of sociological theory. This stands in contrast to typical “black box” epidemiology in which population-based observational studies have demonstrated associations between exposures and outcomes but the etiologic mechanisms of these associations are unknown or unspecified (Susser 2004). While existing theoretical models of social disparities in health may acknowledge the role of biology, these models do not directly utilize the biological basis of stress and behavior to make predictions about how individual-level and societal-level factors interact as we do in the EA Model. Finally, we argue that theories of health disparities that ignore the underlying biology of stress and behavior cannot effectively inform the development of programs that will reduce health disparities for socially disadvantaged groups.

THE ROLE OF RACE AND ETHNICITY

Ultimately, the role of race and ethnicity in stress, self-regulatory behavior, and coping must be considered because empirical studies indicate that racial disparities persist even after controlling for SEP (Crimmins, Hayward, and Seeman 2004; Williams et al. 2010). The EA Model embodies Williams and Earl's (2007) perspective that “race is an imprecise variable that captures differential exposure to the resources and rewards in society” (Williams and Earl 2007:759). Compatible with this perspective, we argue that the motivation for and availability to engage in poor health behaviors as stress-coping or self-regulation strategies is influenced by social structures and contexts (e.g., poverty, segregation, access to goods) and that these social structures are in turn differentially distributed across racial groups. We do not posit that the patterns of health behaviors, mental health, and physical health differ by race (i.e., for both blacks and whites, major depression is associated with medical morbidity; persons with depression, regardless of race, are more likely to engage in poor health behaviors than those without).

This perspective on the role of race in health disparities contrasts with the propositions put forth by some investigators who have postulated that genetic, epigenetic, or neurobiological differences between racial groups produce the observed, average differences between whites and blacks. This argument suggests that phenotypic characteristics associated with race, such as facial features, skin tone, and hair texture, reflect much deeper differences than the nature of racial categorization in and of itself or even the different environmental exposures and experiences correlated with this categorization. Although many others have argued against the fallacy in this way of thinking, this explanation, whether explicit or implicit, has persisted in the literature (Tukufu 2000; Tukufu and Bonilla-Silva 2008).

We believe that race, rather than a category of thingness, is actually a category of experiences that reflects the manner in which racial categories evoke particular reactions within social and physical environments. Similar to racial discrimination, it is the accumulation of differential exposures and experiences associated with these categorizations that produces racial disparities in health over the life course. Thus, we argue that race is not best thought of as an immutable category; instead, racial membership is a “destination” that individuals through their experiences arrive at over time. From this perspective, racial minority status can be viewed as a source of both acute and chronic stress. Internalized racism, threats of discrimination and of confirming negative group-based stereotypes, and lower perceived and actual social status are forms of stress commonly associated with ethnic minority status.

We argue that the empirical observation of Figure 1 is produced by the interaction of social and environmental context with race rather than race itself. However, we acknowledge that it is rarely the case that whites find themselves in contexts identical to those in which blacks find themselves, even when living in poverty (LaVeist et al. 2011). Ethnographic work has shown that blacks and whites living in adjacent urban neighborhoods experience profoundly different day-to-day interpersonal and environmental stressors, which highlights the role that race plays in characterizing and shaping stressful experiences (Wilson 2009). However, recent research indicates that whites and blacks living in the same integrated, low-income neighborhood have either nonexistent or much smaller differences in prevalence of hypertension, diabetes, obesity, and other health behaviors compared to national estimates of racial differences (LaVeist et al. 2011). Notably, in these integrated, low-income settings, the prevalence of these health conditions among whites is much more similar to the average for blacks (i.e., whites in these settings have higher prevalence of hypertension, obesity, and diabetes than national averages, as expected based on fundamental cause theory) (LaVeist et al. 2011). Other studies have found that after accounting for multiple indicators of neighborhood socioeconomic status, black-white differences in health are substantially attenuated (Do, Frank, and Finch 2012; Scribner et al. 2009). Together these findings indicate that socioeconomic inequality paired with racial segregation represent mechanisms that contribute to the persistence of health inequalities (Williams and Collins 2001).

OVERVIEW OF THE EA MODEL

The EA Model explicitly assesses the interrelationships among social context, stress, health behaviors, and mental and physical health. It is informed by psychology, sociology, and biology, and we propose that this framework promotes innovative research that moves beyond simply documenting social disparities in health and begins positing specific hypotheses about how these factors work in concert. Figure 2 illustrates our framework, and Table 1 includes important supporting information.

The EA Model rests on three premises: (1) the contextual environment serves as both a source of constraints (or stress) and a source of affordances—here, affordances are defined as opportunities to alleviate stress (Gibson 1977); (2) coping behaviors are influenced by cultural and social norms and contextual factors (Taylor and Stanton 2007); and (3) all organisms, including humans, engage in efforts to mitigate the immediate experience of distress when faced with stressful experiences (Jansen et al. 1995; Umberson, Liu, and Reczek 2008). These strategies are influenced by a number of individual characteristics, including race/ethnicity, gender, and age. Collectively, the intersection between individual characteristics and contextual factors prime individuals to engage in particular behaviors that alleviate immediate symptoms of psychological and physiological stress (Jackson and Knight 2006). How these processes play out over the life course is of central importance in the EA Model: particularly the dynamic and cumulative effects of stress and the availability,

efficacy, and timing and duration of coping strategies, as others before us have suggested (Taylor and Stanton 2007; Umberson et al. 2008).

As illustrated by Table 1 and Figure 2, we believe that social structures and context increase exposure to chronic stress for socially disadvantaged groups (Path 1). In addition to being a source of stress, environmental context is linked to the available range of health-related coping behaviors (Path 2). We propose that health-related self-regulatory coping strategies (e.g., tobacco use, excessive alcohol intake, illicit substance use, and diets high in fat and sugar), which are effective in preserving the mental health of individuals in the short term (Paths 5 and 7), produce the observed physical health disparities in middle age and late life (Paths 5 and 6) (Jackson and Knight 2006; Jackson, Knight, and Rafferty 2010). The impact of these coping behaviors on physical health is compounded by the effects of social, economic, and environmental inequalities (Path 4). A key innovation of the EA Model is the proposition that these self-regulatory health behaviors likely block the neurological cascade linking the experience of emotional distress to stress-related psychopathology (e.g., major depressive disorder, anxiety disorders) (Path 3) by acting on the hypothalamic-pituitary-adrenocortical (HPA) axis and related neuroendocrine systems. These behaviors may have protective effects on the development of psychopathology, especially in the short term, among socially disadvantaged groups (Path 7); that is, self-regulatory health behaviors interrupt the path between stress and development of psychopathology. However, the direct effects of exposure to chronic stress (Path 4), combined with the direct effects of the poor health behaviors themselves (Path 6), create large disparities in morbidity and mortality over the life course.

EVIDENCE FOR THE EA MODEL

Predictions of the EA Model have been tested using three independent longitudinal studies: the Americans' Changing Lives, the Baltimore ECA, and the National Epidemiologic Survey on Alcohol and Related Conditions cohorts (Jackson et al. 2010; Keyes, Barnes, and Bates 2011; Mezuk et al. 2010). Boardman and Alexander (2011) also examined this hypothesis using the National Longitudinal Study of Adolescent Health, and their results were broadly consistent with the EA Model (i.e., the interaction between stress and poor health behaviors was in the predicted direction for blacks and whites but was not statistically significant for either) (Boardman and Alexander 2011:1663). However, this report used the Center for Epidemiologic Studies–Depression Scale rather than a diagnostic instrument as an index of depressive symptomology, and as we revisit in the discussion of distress versus psychopathology below, it therefore does not provide as compelling a test of the model predictions as do the other reports.

These three studies (Jackson et al. 2010; Keyes et al. 2011; Mezuk et al. 2010) longitudinally examined the probability of onset of depressive disorder at follow-up predicted by stressful life events, self-regulatory health behaviors (i.e., smoking, excessive alcohol use, poor diet and/or obesity status to reflect poor nutrition and/or inactivity), and the interaction between stress and health behaviors. The goal of these studies was to investigate the interaction between stress and self-regulatory health behaviors on risk of depression (Path 7 of Figure 2): A negative interaction would indicate that the risk of depression associated with stress was mitigated among those who engaged in these self-regulatory health behaviors, while a positive interaction would indicate that the risk of depression associated with stress was exacerbated among those who engaged in these behaviors.

In both the Americans' Changing Lives and the ECA analyses, Jackson et al. (2010) and Mezuk and colleagues (2010) found that poor health behaviors moderated the relationship

between stress and risk for depressive disorder. However, as predicted by the EA Model, the direction of the effect was drastically different for blacks and whites. Among whites, although not statistically significant, the results suggested a positive interaction, such that poor health behaviors exacerbated the risk of major depression associated with stress (see Figure 3, Panel A). In contrast, among blacks the interaction was negative; that is, poor health behaviors reduced the risk of depression associated with stress (see Figure 3, Panel B). These findings are consistent with a core EA Model hypothesis that self-regulatory health-related behaviors protect against risk of depression among blacks due to the overrepresentation of blacks in environments that are both high stress and resource constrained.

The report by Keyes et al. (2011) did not support the EA Model; that is, there was no evidence of an interaction between poor health behaviors and risk of depression among blacks. While we acknowledge that these findings are important to consider as we refine the EA Model, one relevant difference between this analysis and the others is that stress was modeled as a categorical rather than a continuous variable. In particular, we feel it is worth noting (as did the authors themselves) that in this sample there was not a linear relation between stress (treated as a continuous variable) and risk of depression for blacks (Keyes et al. 2011:653). We also note that Keyes and colleagues conducted several sensitivity analyses to examine whether their specific three-level categorization of stress influenced the results and report that it did not. Additional research testing the hypotheses of the EA Model is needed, as we outline below, but we feel that these early findings suggest that frameworks such as this one are useful for examining the epidemiologic paradox illustrated by Figure 1.

SELF-REGULATORY BEHAVIORS IN THE CONTEXT OF SOCIAL DISADVANTAGE

Several social science literatures predict that socially disadvantaged groups will be more likely to select alternatives, including health-related ones, which favor short-term over long-term benefits (Frederick, Loewenstein, and O'Donoghue 2002). This tendency may be adaptive in high-stress, low-control environments that require constant vigilance and readiness to respond, as in the case of impoverished or unsafe environments of the type in which racial minorities in general, and blacks in particular, tend to live (Williams and Collins 2001). These factors make it challenging to initiate and sustain future-oriented behaviors, such as dietary changes or tobacco and alcohol cessation (Epstein et al. 2010; Hill, Jenkins, and Farmer 2008). Consistent with this idea, there is strong experimental evidence that socially disadvantaged individuals engage in more risk taking and less delayed gratification, forms of self-regulation that might be referred to as impulsivity or future discounting (Ellis et al. 2011; Hill et al. 2008). There is also emerging evidence that propensity to self-regulate becomes biologically embedded over the course of development (Miller et al. 2009), again indicating that the origin of disparities in health, including health behaviors, emerges early and intensifies over the lifespan.

Link and Phelan's (1995) notion that social structures and contexts put individuals "at risk of risks" applies here. Socially disadvantaged groups experience considerable difficulties in obtaining reasonably priced healthy food and safely engaging in outdoor recreational activities that promote health and prevent disease (Lovasi et al. 2009). These limitations are often compounded by time constraints due to inflexible work schedules and juggling demands of work and family life. These factors may also limit the ability to exercise and prepare fresh, healthy meals, habits that help to preserve long-term health (Wethington and Johnson-Askew 2009). On the other hand, these same groups have ready access to tobacco and alcohol retailers (LaVeist and Wallace 2000), fast food restaurants, and corner stores (Lovasi et al. 2009). As these examples illustrate, the positive coping resources available to

disadvantaged groups are greatly truncated relative to more advantaged groups, while riskier strategies for material (e.g., smoking, drinking, eating) self-regulation strategies are more highly accessible.

SELF-REGULATORY BEHAVIORS AND THE EMERGENCE OF STRESS-RELATED PSYCHOPATHOLOGY

We feel the distinction between psychological distress and psychopathology is essential to understanding how stress, coping, and mental health relate over the life course. While we agree that both distress and depression are dimensional constructs, it is not necessarily the case that they lay on the same continuum (i.e., that depression is simply the severe end of a dimension of distress). This is a critical distinction because while blacks have lower prevalence of psychopathology than whites, numerous population-based studies of nonspecific psychological distress (indexed by metrics such as the Center for Epidemiologic Studies–Depression Scale, Kessler-6, or General Health Questionnaire) indicate higher levels of distress among blacks relative to whites (e.g., Bratter and Eschbach 2005; Eaton and Kessler 1981; Schnittker 2012; Williams et al. 1997). These differences are often, but not always, attenuated after accounting for SEP (e.g., Bratter and Eschbach 2005; Williams et al. 1997).

The EA Model offers an explanation for the observation that blacks report higher levels of distress despite having lower levels of clinically relevant psychopathology. Encompassing the self-medication hypothesis of depression and substance misuse, we argue that (1) individuals are cognitively aware of feeling psychologically distressed long before a clinical syndrome such as major depression onsets, and that (2) when distressed, individuals engage in coping strategies (including those that involve health behaviors) to mitigate the psychological and biological experience of distress. While it is true that substance dependence disorders are comorbid with major depression among both blacks and whites, it is less clear how nonpathological use of these psychoactive substances (e.g., alcohol, tobacco, illicit drugs, and palatable foods) is related to depressive disorders.

For example, over the short term, alcohol is associated with lower risk of depression (Aneshensel 1983); similarly, alcohol abstainers have higher prevalence of depression than moderate alcohol users, and this finding is not explained by individuals' quitting drinking due to health consequences (Skogen et al. 2009). Similarly, existing studies of the relation between eating behavior and depression are mixed. Major depression is generally associated with appetite and weight loss, not gain (Sullivan, Kessler, and Kendler 1998), and only severe obesity is consistently associated with depression (Onyike et al. 2003). In contrast, psychological distress has generally been associated with appetite gain (particularly a preference for palatable, high-calorie foods) in animal and human studies (Dallman 2010; Pecoraro et al. 2004).

Finally, we note that effective stress coping does not necessarily equate to an absence of psychological distress but may instead be associated with the delay or prevention of progressively severe forms of psychopathology, such as major depression. As emphasized above, a life course approach is necessary to understand how stress, health behaviors, and mental and physical health relate over time.

THE BIOLOGY OF SELF-REGULATORY BEHAVIORS

As described above, the reason we offer the EA Model as an example of white box epidemiology is that it is directly informed by experimental research in humans and animals on the biological mechanisms underlying self-regulatory behaviors in the context of stress.

While a comprehensive examination of these mechanisms is beyond the scope of this article (see Koob 2008 for a recent review), two broad neuroendocrine systems are particularly relevant to the EA Model: (1) the stress response system, including the HPA axis, and (2) the reward/reinforcement system, including dopamine/endorphin pathways. The HPA axis is a complex hormone-signaling system through which the body responds to stress and is a component of the flight or fight response. The HPA axis produces cortisol (and other glucocorticoids), which regulates and influences a wide variety of biological responses to stress, including blood pressure and immune function (Miller et al. 2009). The reward/reinforcement system involves the mesocorticolimbic dopaminergic system in the brain; this system has innervations to the amygdala and hippocampus (structures key to memory formation) and opioid neurons (e.g., enkephalins, endorphins, and dynorphins) (Koob 2008). Endogenous opioids are associated with pain relief and feelings of euphoria and reward. Experimental studies have established that the stress response and reward systems interact and adapt to each other over time (Armario 2010; Koob 2008).

We also note two aspects of self-regulatory health behaviors that are particularly relevant to the EA Model: (1) the biological and psychological consequences of health behaviors differ for acute, short-term use as compared to chronic and/or pathological use (Koob 2004), and (2) over time, the physical health problems that develop from persistent engagement in poor health behaviors overwhelms any protective effect they have on development of stress-related psychopathology. That is, these behaviors increase risk of conditions such as cardiovascular disease, cancer, and type 2 diabetes, which are all associated with the development and recurrence of depression in later adulthood (Benton, Staab, and Evans 2007). This emphasizes the need to understand the relationship between stress, health behaviors, and mental and physical health as one that is dynamic and changes over the life course.

Although the exact mechanisms are not completely understood, evidence from both human and animal research suggests that ingestion of nicotine, alcohol, and palatable foods simultaneously activate the body's stress response system and elicit rewarding and reinforcing effects when consumed in response to stress (see Table 1, Path 7) (Koob 2008; Miller et al. 2011). For example, ingestion of nicotine, alcohol, and other drugs elicits the release of dopamine and β -endorphin, which activates the reward/reinforcement system, and that these reinforcement circuits are modulated by the HPA axis (Uhart and Wand 2009). Glucocorticoids can increase preference for palatable foods (Warne 2009), and the consumption of these foods increases dopamine release, similar to illicit drugs (Gearhardt et al. 2011; Volkow and Wise 2005). This is thought to be driven by the endogenous opioid system, which heightens the rewarding and reinforcing effects of these foods (Volkow and Wise 2005). The EA Model posits that these health behaviors may mitigate the negative effects of chronic stress on risk of psychopathology through their actions on the biological correlates of the stress and reward/reinforcement systems.

IMPLICATIONS, UNRESOLVED ISSUES AND SUGGESTIONS FOR FUTURE RESEARCH

Overall, the EA Model suggests that mental and physical health must be considered jointly within the context of the social and physical environment when investigating the causes of disparities across racial groups. The model argues that the observed paradox of mental and physical health disparities observed in the United States is due to the interaction of individual behaviors with environmental contexts over the lifespan and that effectively matching whites and blacks on similar contexts would result in similar behaviors. It also calls into question implicit assumptions about the relationships between social disadvantage, health behaviors, and mental health. It recognizes that individuals may face competing

demands between preserving immediate mental health status and long-term physical health and that this is experienced as a zero-sum game for groups under chronic stress. Below we briefly discuss the implications of the EA Model for both social theory and public health efforts to reduce social disparities.

The central hypothesis of the EA Model is that observed racial differences in mental and physical health, and differences in the moderating role of self-regulatory coping behaviors on stress, would not exist if whites and blacks lived, worked, and grew up in comparable environments. In other words, we would observe the same moderating effect of poor health behaviors on the relationship between stress and the risk of psychopathology shown in Panel B of Figure 3 among whites if whites experienced the same contextual environments, over the life course and in the same way, as typical blacks do. That is, what appear to be racially based differences may in fact reflect explicable and demonstrable differences in social and material environments and the ensuing psychological and social processes that are a product of these environments. It is difficult to test this hypothesis because blacks and whites often have qualitatively different experiences within quantitatively similar socioeconomic contexts. Nevertheless, as described above there is a growing body of research that is consistent with this postulate.

To test this hypothesis, it is necessary to statistically model the counterfactual state of the world, that is, to build a representation in which whites and blacks experience the world in the same way. Methods such as propensity score techniques (which can be used to account for the confounding of race with SEP and therefore allow for a more unbiased estimation of the relationship between stress, health behaviors, and mental health) (Rosenbaum and Rubin 1983) and marginal structural modeling (which go beyond propensity scores methods to account for the presence of time-dependent exposures that may be acting simultaneously as confounders and mediators, such as stress) (Robins, Hernan, and Brumback 2000) are useful approaches for testing this counterfactual prediction. Other viable approaches include latent variable techniques, which have been used for decades to model SEP, to treat race as an unmeasured construct indicated by multiple environmental factors that reflect experiences over the life course (e.g., poverty and stress in childhood). This approach would provide a means to unpack the numerous environmental factors that correlate with race simultaneously and allow for the possibility of matching individuals on their "latent" trait of race rather than on their observed racial group membership (Borsboom, Mellenbergh, and van Heerden, 2003). These analytic approaches (propensity score, marginal structural modeling, and latent variable techniques) reflect our argument that race is a complex set of experiences rather than an immutable categorical characteristic over the life course (Tukufu 2000).

We readily acknowledge that this work is in its infancy and that there are important outstanding questions that need to be addressed. It is not yet clear how positive and negative coping behaviors work together, especially factors such as cultural and psychological resources (e.g., communalism, resilience, flourishing; Campos et al. 2008). For example, does having a smoke break relieve feelings of psychological distress through the biological effects of nicotine alone, or is it also the social interactions that come from talking with coworkers during the break? Are so-called comfort foods a particularly potent form of psychological release when consumed with family and friends? Both population-based and clinical experimental studies are needed to address this intersection between positive and negative coping strategies.

A second area in need of research is to examine the relevance of the EA Model in other materially disadvantaged racial/ethnic populations, including some Latino groups and specifically in relation to the apparent "Hispanic paradox" in health (Palloni and Morenoff 2001). There is also a need to integrate objective measures of stress exposure and response

into population-based studies to more directly interrogate the hypothesis that poor health behaviors are associated with lower risk of psychopathology among blacks under high stress through the biological pathways outlined above.

Finally, we acknowledge the need for an iterative approach to evaluating the EA Model: one that moves back and forth between black box population-based studies (which provide insight into the real-world social and environmental contexts in which people live but which generally cannot directly investigate causal pathways) and white box mechanistic clinical and animal work (which provide insight into precise biological and psychological mechanisms but may not be readily generalizable to the real world). This approach is needed to investigate the robustness of model predictions and identify new questions that the EA Model may (or may not) be able to address.

IMPLICATIONS FOR HEALTH PROMOTION EFFORTS

To date, the translation of theories of the origins of health disparities into practice has resulted in only limited improvements for socially disadvantaged groups. We propose that a key reason for this lack of progress has been the focus on psychological processes, such as learning, motivation, attitude, and persuasion, which emphasize the influence of personal characteristics on behavior. Most models of behavior modification do not consider the effects of the physical and social environment, nor do they incorporate structural factors related to race, SEP, gender, and age in determining individuals' ability to initiate, maintain, and benefit from behavior change (Thoits 2010).

The EA Model has direct implications for efforts to reduce poor health behaviors and prevent development of chronic medical problems among materially disadvantaged groups. Sustained reductions in poor health behaviors will likely not be achieved without addressing the contextual sources of stress and incentivizing healthy self-regulatory behaviors (Winkleby et al. 1999). Health promotion efforts that adopt a three-pronged approach to simultaneously (1) provide evidence-based interventions to prevent initiation or promote cessation of harmful behaviors, (2) mitigate sources of stress (e.g., financial strain, exposure to trauma and violence) or reinforce positive resources (e.g., positive social networks), and (3) act on structural forces (e.g., high concentrations of fast food restaurants or alcohol outlets) that encourage engagement in these behaviors are more likely to succeed than efforts to modify any of these factors in isolation. Policy and infrastructure changes that are not directly related to health may have downstream influences on health behavior; for example, efforts to improve educational attainment among urban youth may translate into improvements in self-regulatory health behaviors in adulthood (Thoits 2010).

The EA Model also has implications for the effective management of chronic diseases, particularly for conditions that require intensive patient self-management and substantial behavioral modifications to effectively control (e.g., asthma, diabetes, and hypertension) (Chodosh et al. 2005). There is increasing recognition of the need to incorporate psychosocial interventions (e.g., problem solving, stress management, social support) into chronic disease management (Katon et al. 2010). For example, in the Collaborative Care Model for diabetes, care managers enhance patient self-care with education, encourage exercise through engaging in enjoyable physical activities, and promote goal-setting and problem-solving skills. This model is successful, at least partially, because it is focused on open communication between patients and clinicians about health goals and provides a social resource (in the form of a care manager) to help patients achieve those goals (Katon et al. 2010).

CONCLUSION

The EA Model presented here challenges existing notions of health disparities in important ways. Overall, we believe that the EA Model and empirical work deriving from this framework raise significant questions about how we understand racial classification and how racial group membership functions over the life course. This framework embodies a transdisciplinary approach that aims to examine the relationships among stress, mental health, and self-regulatory health behaviors across multiple levels of analysis. It draws from experimental animal and human studies, epidemiology, and quantitative and qualitative psychology and sociology. Finally, it provides a means for understanding and addressing seemingly paradoxical findings regarding ostensibly social sources of inequalities in health.

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Figure 1.

Lifetime prevalence of common psychiatric and chronic medical conditions by race/ethnicity: National Comorbidity Survey–Replication (2001–2003).

Note: Analysis is limited to adults aged 40 and older, and values are adjusted for sampling weights. MDE = major depressive episode; GAD = generalized anxiety disorder.

Figure 2.

The Environmental Affordances Model.

Note: The Environmental Affordances Model incorporates status-based stressors, coping, and mental and physical health.

Figure 3.

Predicted probability of depression at follow-up by race, number of poor health behaviors (PHB), and life stress at baseline for A) whites and B) blacks in the Baltimore Epidemiologic Catchment Area Study.

Note: From the *American Journal of Epidemiology* (2010), 172(11):1238–49. Reprinted with permission of Oxford University Press.

Table 1

Postulates and Evidence for the Environmental Affordances Model (Paths Indicated in Figure 2).

Path 1	A. Exposure to chronic social stress is patterned by social structures and context, including race.	a. Neighborhood contexts are racially segregated; racial/ethnic minorities (e.g., blacks and Hispanics) are more likely to live in socially disadvantaged neighborhoods (Williams and Collins 2001).
	B. Over the life course, differential exposure to chronic stress creates and perpetuates differences between social groups.	b. Socially disadvantaged neighborhoods have higher incidence of stressors such as crime and exposure to violence, low social capital, high unemployment, and single-headed households (Sternthal, Slopen, and Williams 2011).
		c. Racial/ethnic minorities are more likely to experience both institutional and interpersonal discrimination relative to whites (Williams and Collins 2001).
Path 2	The social context is associated with material coping behaviors.	a. Socially disadvantaged neighborhoods have limited access to healthy foods and recreational physical activity opportunities (Lovasi et al. 2009).
		b. Socially disadvantaged neighborhoods have greater access to tobacco and alcohol outlets, corner stores, and illicit drugs (LaVeist and Wallace 2000).
		c. Persons living in poor neighborhoods are more likely to use tobacco, alcohol, and illicit drugs, have high-fat diets, and engage in risky sexual behaviors (Mulia et al. 2008)
Path 3	Chronic stressors increase risk of stress-related psychopathology.	a. Negative life events predict incidence of major depression, even accounting for genetic risk (Kendler, Karkowski and Prescott 1999).
Path 4	Chronic stressors increase risk of medical morbidity and mortality.	a. Social adversity is associated with high allostatic load (McEwen and Seeman 1999; Miller, Chen, and Cole 2009).
		b. Stressful life events are associated with elevated risk of chronic health conditions in mid and late life (Lantz et al. 2005)
		c. Perceived discrimination is associated with increased mortality (Barnes et al. 2008).
Path 5	Chronic stress increases propensity to engage in poor health behaviors.	a. Laboratory-induced stress increases food intake and reduces feelings of reward from food consumption, which promotes overeating (Epel et al. 2001; Adam and Epel 2007); chronic stress promotes ingestion of palatable foods (Foster et al. 2009; Pecoraro et al. 2004).
		b. Stress is predictive of initiation and persistence of use of smoking (Slopen et al. 2012), alcohol and illicit drugs, and relapse (Brady and Sonne 1999).
		c. Stress is associated with poor health behaviors among both blacks and whites (Boardman and Alexander 2011; Keyes, Barnes, and Bates 2011).
Path 6	A. Self-regulatory health behaviors increase risk of medical morbidity and mortality.	a. Tobacco smoking, alcohol misuse, poor diet, physical inactivity, illicit drug use, and risky sexual behaviors are the leading preventable sources of chronic conditions such as cardiovascular disease, cancer, type 2 diabetes, hypertension, and premature mortality (Mensah et al. 2005).
	B. The effect of poor health behaviors on development of physical health problems is exacerbated by stress.	b. The interaction between stress and poor health behaviors on risk of physical health problems is positive and similar for both blacks and whites (Jackson, Knight, and Rafferty 2010; Mezuk et al. 2010).
Path 7	A. Self-regulatory health behaviors reduce the biological consequences of the stress experience in the brain.	a. Consumption of palatable foods is associated with lower levels of corticotropin-releasing hormone and cortisol (Dallman 2010; Foster et al. 2009; Pecoraro et al. 2004)
	B. These substances also reduce the psychological and behavioral consequences of stress.	b. Consumption of palatable foods after stress increases affiliative behavior (Ulrich-Lai et al. 2010)
		c. Smoking influences the hypothalamic-pituitary-adrenocortical axis (Richards et al. 2011).
		d. Chronic alcohol and cocaine use dampens the biological response to laboratory stressors (Sayette 1999; Waldrop et al. 2010).