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Article

Stress, self-regulation, and context: Evidence from the health and retirement survey



Briana Mezuk^{a,b,*}, Scott Ratliff^a, Jeannie B. Concha^c, Cleopatra M. Abdou^d, Jane Rafferty^b, Hedwig Lee^e, James S. Jackson^{b,f}

^a Department of Epidemiology, University of Michigan School of Public Health, Ann Arbor, MI, USA

^b Institute for Social Research, University of Michigan, Ann Arbor, MI, USA

^c College of Health Sciences, UTEP, El Paso, TX, USA

^d Leonard Davis School of Gerontology, Department of Psychology, University of Southern California, Los Angeles, CA, USA

^e Department of Sociology, University of Washington, Seattle, WA, USA

^f Department of Psychology, University of Michigan, Ann Arbor, MI, USA

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ABSTRACT

Health-related behaviors, such as smoking, alcohol use, exercise, and diet, are major determinants of physical health and health disparities. However, a growing body of experimental research in humans and animals also suggests these behaviors can impact the ways our bodies respond to stress, such that they modulate (that is, serve as a means to self-regulate or cope with) the deleterious impact of stressful experiences on mental health. A handful of epidemiologic studies have investigated the intersection between stress and health behaviors on health disparities (both mental and physical), with mixed results. In this study we use a novel instrument designed to explicitly measure the self-regulatory motivations and perceived effectiveness of eight health-related self-regulatory behaviors (smoking, alcohol, drug use, overeating, prayer, exercise, social support, talking with a counselor) in a subset of the Health and Retirement Study (N = 1354, Mean age = 67, 54% female). We find that these behaviors are commonly endorsed as self-regulatory stress-coping strategies, with prayer, social support, exercise, and overeating used most frequently. The likelihood of using particular behaviors as self-regulatory strategies varied significantly by sex, with only limited variation by race/ethnicity, education, or wealth. We also find that greater stress exposure is associated with higher likelihood of using these behaviors to self-regulate feelings of emotional distress, particularly health-harming behaviors like smoking, alcohol, and overeating. These findings provide an important link between sociological and psychological theoretical models on stress and empirical epidemiological research on social determinants of health and health disparities.

Introduction

The only way to keep your health is to eat what you don't want, drink what you don't like, and do what you'd rather not.

Mark Twain

A compelling body of epidemiologic research indicates that exposure to stressful events contributes to poor health and health disparities over the life course (James, 2009; Miller, Chen & Cole, 2009). “Stress” refers to any threat or challenge to homeostasis (McEwen, 2013), and includes a broad range of exposures such as prenatal insults (Hilmert et al., 2008), early life adversity (Miller, Chen, & Parker, 2011), work (e.g., job strain), finances (e.g., poverty, food insecurity), interpersonal events (e.g., divorce, social isolation), trauma (e.g., emotional, physical,

or sexual abuse), and experiences of discrimination (Abdou, Fingerhut, Jackson, & Wheaton, 2016; Turner, Wheaton, & Lloyd, 1995). While the neurobiological stress response (e.g., hypothalamic-pituitary-adrenal (HPA)-axis, sympathetic nervous system) is well-suited for addressing acute stressors, it is hypothesized that repeated, chronic activation of the body's stress response (commonly operationalized as “allostatic load,” “weathering,” and related constructs) contributes to the development of cardiovascular and metabolic conditions in mid- and late-life (Geronimus, 1992; McEwen & Seeman, 1999; Miller et al., 2011). This process of “wear and tear” is often cited as an explanation of the large racial/ethnic and socioeconomic disparities in physical health seen in the US population (Geronimus, 1992).

However, this explanation of stress as a direct cause of social disparities in physical health does not account for the fact that socially-

* Corresponding author at: Department of Epidemiology, University of Michigan School of Public Health, 1415 Washington Heights, Suite 2649B, Ann Arbor, MI 48109, USA.
E-mail address: bmezuk@umich.edu (B. Mezuk).

disadvantaged groups, particularly African Americans and Hispanics in the US, despite having higher morbidity and mortality, have better mental health relative to non-Hispanic Whites (Jackson, Knight, & Rafferty, 2010; Mezuk et al. 2010; Mezuk et al. 2013). For example, African Americans are less likely to have major depression, anxiety disorders, or substance abuse/dependence relative to non-Hispanic Whites, a finding that has been replicated across numerous nationally-representative samples and measures of psychopathology (Mezuk et al. 2013). Since stress is an established cause of these mental health outcomes, the apparently paradoxical finding that these socially-disadvantaged groups (which are presumably exposed to more stress than socially-advantaged non-Hispanic whites) do not have worse mental health, despite having worse physical health, warrants a reconsideration of the potential pathways linking stress, health behaviors, and health status. Informed by this evidence, we developed the Environmental Affordances Model of Health Disparities (EA Model; Mezuk et al. 2013) a transdisciplinary framework which guides our empirical research on how stress, behavior, and context intersect to influence mental and physical health.

Re-conceptualization of coping behaviors under the EA model

While much is known about the direct effects of stress exposure on health, there has been less focus on how the intersection between stress and coping behaviors (i.e., efforts to self-regulate the body's stress response) relates to health and health disparities (Ellis & Del Giudice, 2014; Mezuk et al., 2013). Under the conceptualization of stress as a direct cause of poor physical health, behaviors are treated as confounders (i.e., correlates of stress and causally related to health, but not part of the pathway linking the two (Umberson, Liu & Reczek, 2008)). This conceptualization of health behaviors as confounders may stem from an inappropriately narrow scope of coping typologies. Coping is traditionally defined as the “cognitive and behavioral efforts to manage specific external and/or internal demands that are appraised as taxing or exceeding the resources of the person,” (Lazarus & Folkman, 1984, pp. 141), and is generally divided into *approach* and *avoidance* typologies (Taylor & Stanton, 2007). Approach-oriented coping involves processes that directly address either the source of stress or the resulting homeostatic imbalance (e.g., *cognitive* strategies like planning, strategizing, humor, and acceptance), and implicitly focus on coping as a psychological experience. When *behaviors* are examined as approach-oriented strategies the focus is often on seeking social support (e.g., talking to others or seeking advice). Within this typology, other behaviors (e.g., doing activities as a distraction, disengaging from the situation) are regarded as avoidant coping. The term *avoidant* invokes processes that prevent individuals from effectively addressing the stressful situation and/or do not address the homeostatic imbalance induced by the stressor. Health-related behaviors (i.e., smoking, drinking alcohol, eating, exercise) are chief among these avoidant strategies (Umberson et al., 2008). For both approach and avoidant coping strategies there is little consideration of the biological underpinnings by which these processes translate into improved mental health; however, all mental experiences are derived from the brain, even if we do not fully understand how this derivation occurs.

A growing body of experimental research (both in animal models and humans) suggests that these “avoidant” health behaviors act on a common set of reward and stress-response pathways and have the same (short-term) salutary impact on restoring homeostasis (both psychological and physiological) as approach-oriented coping behaviors (Mezuk et al., 2013). For example, in a placebo-controlled study of current smokers (i.e., smoking as usual vs. nicotine patch vs. placebo patch), cigarette use reduced the cortisol response to a laboratory stressor, indicating a biological underpinning between stress and relapse from smoking cessation (Wardle, Munafo, & de Wit, 2011). In another example, women randomized to consume high-sugar beverages over a 2-week period had a reduced cortisol response to a laboratory stressor

compared to women receiving aspartame-sweetened beverages, suggesting a negative feedback loop between glucose consumption and HPA-axis reactivity (Tryon et al., 2015). There are similar examples of linkages between the HPA-axis and other reinforced behaviors (Koob, 2008) including alcohol use (Stephens & Wand, 2012), eating (Pecoraro, Reyes, Gomez, Bhargava, & Dallman, 2004), drug use (Chaplin et al., 2010), meditation (Rosenkranz et al. 2016), and exercise (Childs & de Wit, 2014). This suggests a need to consider the neuroscience of coping as much as we consider the psychology of coping.

In sum, a growing body of research indicates that the relationships among stress exposure, stress reactivity, and health behaviors are intrinsically linked in two important ways: (1) Stress exposure impacts the likelihood of engaging in health behaviors, and these behaviors, in turn, impact physiological reactivity to subsequent stressors; and (2) These behaviors engage reinforcing (e.g., dopaminergic and opioid) pathways in the brain, which are also connected to the HPA-axis and related stress-response systems. Thus, in the short-term, these health behaviors can serve as effective stress-coping strategies and preserve mental health, just as traditional approach-oriented coping strategies are known to do. However, unlike these traditional coping strategies, over the long-term behaviors such as smoking, excessive alcohol use, and poor diet contribute to disparities in physical health (Lantz et al., 1998). Moreover, sociological studies have shown that the impact of poor health behaviors (e.g., smoking, physical inactivity) on physical health is amplified by stress for individuals with fewer socioeconomic resources (Krueger & Chang, 2008), and that the strength of the relationship between health behaviors and health outcomes varies by race/ethnicity, largely because of racial/ethnic stratification of socioeconomic resources (Krueger, Saint Onge, & Chang, 2011). This illustrates the need for transdisciplinary frameworks like the EA Model that seek to link biology, behavior, and social context.

Limitations of existing research testing the EA model

In this paper we refer to health behaviors as self-regulatory coping behaviors (SRCB) to emphasize that they are coping efforts aimed at addressing the neurobiological stress response and returning the individual to a homeostatic state. As with traditional social psychology theories of coping, the EA Model posits that the specific set of SRCBs prompted in response to stress is influenced by context. By context, we mean the affordances and constraints of the environment, including both physical context (i.e., neighborhood attributes, such as the availability of fast food restaurants) and sociocultural context (i.e., social norms, social integration, social cohesion, and other cultural resources). In this way, structural sources of health disparities (i.e., poverty, residential segregation, social capital) influence health disparities by both acting as a source of stress (i.e., financial strain) and by truncating the opportunities individuals have to cope with stressors (Bird & Rieker, 2008; Link & Phelan, 1995).

However, to date epidemiologic research testing hypotheses of the EA Model has not directly assessed whether health behaviors are actually being used as efforts to self-regulate (i.e., it has been assumed, rather than directly measured, that the behaviors are used to cope with stress) (Boardman & Alexander, 2011; Jackson et al., 2010; Keyes, Barnes & Bates, 2011; Mezuk et al., 2010). This is because these analyses have relied on existing data that assessed these behaviors in traditional ways (i.e., asking respondents if, but not why, they smoke, overeat, exercise, etc.). There is also little information known about the perceived effectiveness of these behaviors at reducing feelings of distress in a general population sample (i.e., do individuals experience a reduction in psychological distress as predicted by the biological experimental data, and how does that perceived effectiveness vary across behaviors?) In addition, there has been little attention to how SRCBs that harm physical health (e.g., smoking) relate to SRCBs that promote health (either mental or physical: e.g., exercise). For instance,

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