Social Science & Medicine 72 (2011) 650–659

Contents lists available at ScienceDirect

Social Science & Medicine

journal homepage: www.elsevier.com/locate/socscimed

Stress, coping, and depression: Testing a new hypothesis in a prospectively studied general population sample of U.S.-born Whites and Blacks

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ARTICLE INFO

Article history:
Available online 17 December 2010

Keywords:
Race
Depression
Mental health
Alcohol
Obesity
Socioeconomic position
African Americans
USA
Stress

ABSTRACT

The scarcity of empirically supported explanations for the Black/White prevalence difference in depression in the U.S. is a conspicuous gap in the literature. Recent evidence suggests that the paradoxical observation of decreased risk of depression but elevated rates of physical illness among Blacks in the U.S. compared with Whites may be accounted for by the use of coping behaviors (e.g., alcohol and nicotine consumption, overeating) among Blacks exposed to high stress levels. Such coping behaviors may mitigate deleterious effects of stressful exposures on mental health while increasing the risk of physical ailments. The racial patterning in mental and physical health outcomes could therefore be explained by this mechanism if a) these behaviors were more prevalent among Blacks than Whites and/or b) the effect of these behavioral responses to stress was differential by race. The present study challenges this hypothesis using longitudinal, nationally-representative data with comprehensive DSM-IV diagnoses. Data are drawn from 34,653 individuals sampled in Waves 1 (2001–2002) and 2 (2004–2005) as part of the US National Epidemiologic Survey on Alcohol and Related Conditions. Results showed that a) Blacks were less likely to engage in alcohol or nicotine consumption at low, moderate, and high levels of stress compared to Whites, and b) there was a significant three-way interaction between race, stress, and coping behavior for BMI only ($F = 2.11$, df $= 12$, $p = 0.03$), but, contrary to the hypothesis, elevated BMI was protective against depression in Blacks at low, not high, levels of stress. Further, engagement in unhealthy behaviors, especially at pathological levels, did not protect against depression in Blacks or in Whites. In sum, the impact of stress and coping processes on depression does not appear to operate differently in Blacks versus Whites. Further research testing innovative hypotheses that would explain the difference in Black/White depression prevalence is warranted.

Introduction

Epidemiologic studies have consistently documented that Blacks living in the United States have higher rates of physical illness such as hypertension and diabetes, and higher rates of mortality, compared with non-Hispanic Whites controlling for indicators of socioeconomic position (SEP) (Heckler, 1985; McCord & Freeman, 1990; Williams & Jackson, 2005). Conversely, major psychiatric epidemiologic household surveys have reported that Blacks have equal or lower rates of most psychiatric disorders, including major depression (Breslau et al., 2006; Hasin et al., 2005; Kessler et al., 1994; Williams et al., 2007). These divergent patterns for mental and physical health outcomes have been termed a 'paradox' (Williams, 2001). Blacks in the U.S. face historic and contemporary institutionalized discrimination which exposes them to disadvantaged SEP, worse living conditions, and greater stress and adversity due to marginalized social status (Kessler et al., 1999; Kreiger, 2000; Williams & Williams-Morris, 2000), all of which seemingly place Blacks at greater risk for depression compared with Whites (Dohrenwend, 2000). Indeed, among Blacks in the U.S., perception of discrimination and adversity due to race is associated with greater psychological distress and depressive symptoms (Kessler et al., 1999; Williams & Williams-Morris, 2000). However, absolute rates of depression remain lower among Blacks compared with Whites.

Many pathways have been posited to explain the elevated rates of physical health problems among Blacks in the U.S. compared with Whites. One well-studied mechanism is stress associated with disadvantaged social status. The physiologic responses to stress via allostatic load have been hypothesized to influence health by a process of 'wear and tear' whereby the body can no longer effectively regulate itself (McEwen, 2000, 2004). "Weathering" (Geronimus, 1994, 1996), which describes a process of accelerated aging as an effect of the cumulative experience of stress and adversity, has been

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doi:10.1016/j.socscimed.2010.12.005

hypothesized to explain why Blacks have lower birth weights as well as higher mortality at younger ages than Whites after controlling for SEP. Further, interpersonal discrimination appraised by the individual as negative can result in fear, anger, and denial, thereby inducing injurious physiologic responses in cardiovascular, endocrine, neurologic and immune systems (Krieger, 1990; Kreiger, 2000; Krieger & Sidney, 1996). Adverse neighborhood conditions, to which Blacks have greater exposure than Whites, can influence health through inadequate access to social and health services, exposures to health hazards, and reduction in social cohesion and connectedness (Massey, 1985, 2004). Greater stress, worse bodily wear and tear, reduced access to medical services, and greater exposure to deleterious neighborhood conditions are all risk factors for depression (Leonard, 2000; McEwen, 2003; Stansfeld, 2005), and yet Blacks consistently generate estimates of depression below those of Whites; this poses a perplexing, unresolved issue for social and psychiatric epidemiology.

Two methodological hypotheses advanced to explain this mental/physical health paradox posit that rates of depression among Blacks are underestimated in major psychiatric epidemiologic studies due to selection bias and measurement error. The selection bias hypothesis reflects the fact that all major psychiatric epidemiologic surveys conducted in the U.S. exclude institutionalized populations. Young Black men in the U.S. are overrepresented in prison and jail populations (Petit & Western, 2004), where depression is more prevalent compared with household populations (Teplin, 1990; Teplin et al., 1996). Thus, the underestimation of depression prevalence in household samples could affect Blacks to a greater extent compared with Whites, though the effect of this bias would primarily be age- and gender-specific. The measurement error hypothesis suggests potential diagnostic bias in the major survey instruments used to capture depression. Given the same symptom presentation, Blacks interviewed by clinicians in unstructured or semi-structured formats are more likely to be diagnosed as having a disorder in the psychotic spectrum and Whites as having a disorder in the mood spectrum (Neighbors et al., 1999, 2003; Strakowski et al., 2003). Additionally, some argue that depression may manifest differently in Blacks compared with Whites, and current diagnostic nosology more appropriately captures depression in Whites compared with Blacks (Baker, 2001; Brown, 2003; Kleinman, 2004; Rogler, 1999). Available data suggest that while these hypotheses may explain some of the Black/White difference in depression, methodological issues cannot account for the all of the difference (Breslau et al., 2008; Williams et al., 2007). Thus, hypotheses exploring alternative mechanisms through which Blacks may have a lower prevalence of depression compared with Whites remain necessary.

In contrast to methodological hypotheses explaining the mental/physical health 'paradox', a recently advanced alternative hypothesis is that the patterning in physical and mental health outcomes in Blacks versus Whites arises from mechanisms for coping with stressors that on average operate differently for Black and White Americans (Jackson & Knight, 2006; Jackson et al., 2009). Jackson and colleagues have argued that Blacks in the U.S. face greater, and unique, stressors compared with Whites, and that strategies deployed to cope emotionally with this increased stress may protect mental health while having deleterious consequences for physical health. Recently, Jackson and colleagues reported that at high levels of stress, Blacks with elevated body mass index (BMI) and/or who smoke cigarettes and/or drink alcohol (collectively termed 'unhealthy behaviors' or 'UHBs' (Jackson et al., 2009)) were less likely than Blacks not engaging in these behaviors to develop depression, whereas the pattern trended in the opposite direction for Whites (Jackson et al., 2009). Further empirical support for this hypothesis was recently reported using data from the Baltimore Epidemiologic Catchment Area Study (Mezuk et al., 2010). Evidence indicates that UHBs can ameliorate immediate anxiety and depressive symptoms in response to stressful experiences by regulating corticotropin-releasing factor in the hypothalamic–pituitary–adrenocortical (HPA) axis (Benowitz, 1988; Dallman et al., 2003; Koob et al., 1998). However, long-term heavy alcohol consumption, smoking, and high BMI can lead to a cascade of physical health consequences. This hypothesis suggests that, in the context of chronic stress, Blacks' engagement in UHBs may serve to buffer the deleterious consequences of stress on depression through the HPA pathway, leading to a lower prevalence of depression but a greater prevalence of physical health problems than would have otherwise occurred. This hypothesis also suggests that the same processes operate differently or with different consequences in Whites. In the interest of brevity, we refer to these potentially differential patterns in the relationships between stress, coping, and depression between Blacks and Whites as "group-specific," meaning that they arise from the unequal distribution of exposures and coping resources engendered by a racialized environment, rather than differences embedded in the individual.

Differences in stress and coping processes between Blacks and Whites could account for the mental/physical health 'paradox' under two scenarios. (1) UHBs are indeed protective against depression, among both Blacks and Whites, but Blacks are much more likely to engage in them compared with Whites at a given level of stress. This is unlikely in light of previous epidemiologic evidence suggesting that a) substance disorders and obesity are comorbid with depression (Hasin et al., 2005; Kessler et al., 1997; Reiger et al., 1990) and b) Blacks are less likely than Whites to engage in alcohol and nicotine consumption (Grant et al., 2004; Hasin et al., 2007). However, patterns of comorbidity and Black/White differences in depression at all levels of stress have not been investigated systematically. (2) UHBs operate differentially by race, whereby they protect against depression to a greater extent among Blacks compared with Whites (either overall or variably by level of stress). This hypothesis is supported by data from the Americans' Changing Lives Survey (Jackson et al., 2009) and the Baltimore Epidemiologic Catchment Area Study (Mezuk et al., 2010), as described above.

We propose to comprehensively investigate each of the above scenarios in a large nationally-representative prospective study of U.S. adults. The present study is intended to both replicate and extend the analyses presented in Jackson et al. (2009) to provide a comprehensive test of the underlying theory. Using the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) we accomplish five main aims. First, we construct as exact a replication as possible of Jackson et al. (2009) in order to provide a baseline for comparison and from which to broaden the analyses. The remaining four aims systematically test the theory underlying the two scenarios outlined above. We examine whether alcohol consumption, nicotine consumption, and body mass index (as a proxy for overeating, consistent with Jackson et al. (2009)) are prospectively protective against depression; we examine whether Blacks engage in more of these behaviors than Whites at low, moderate, and/or high levels of stress; and we test the hypothesis that Blacks exposed to high levels of stress are protected against depression if engaged in UHBs at the time of the stressors and, simultaneously, that Whites are not similarly conferred such protection from these behaviors. Finally, the hypothesis outlined by Jackson et al. (2009) suggests that the stress exposure of Blacks is qualitatively different compared to that of Whites. The NESARC data allows us to examine a measure of perceived racial discrimination in order to test whether Blacks who report high levels of discrimination and engage in UHBs have less depression than Blacks who report high levels of discrimination and do not engage in UHBs.

The data used in the present study have distinct advantages over those in Jackson et al. (2009): namely a larger sample size, DSM-IV diagnoses of major depression at two time points, DSM-IV diagnoses