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Long-term physical health consequences of perceived inequality: Results from a twin comparison design

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ABSTRACT

Rationale: Previous research has identified long-term exposure to stress as a risk factor for negative mental and physical health outcomes. This pattern of findings suggests that environmental stimuli that evoke feelings of stress or strain may also result in physiological responses, which may accumulate over the life course and ultimately increase the overall risk of various physical health conditions. This physiological "wear and tear" resulting from sustained levels of stress or strain has been previously operationalized as allostatic load (AL), a comprehensive indicator of stress exposure.
Objective: The current study examines the association between one potential environmental stressor—perceived inequality—and AL with a research design aimed at addressing both observed and unobserved sources of confounding; it also employs a more comprehensive AL measure (comprised of 24 biomarkers tapping seven physiological systems) than previous studies.
Method: The biomarker twin sample from the Midlife Development in the United States (MIDUS) study was used to estimate a series of twin comparison models, which include controls for latent sources of influence that cluster within families. The sibling comparison models also included additional controls for lifestyle choices, overall physical health, and demographics which may confound the examined associations.
Results: The results revealed significant associations between greater perceptions of inequality and greater overall levels of AL. The association persisted even after including controls for both observed and unobserved influences that may confound the examined associations but was limited to more recent measures of perceived inequality. Associations involving earlier measures of perceived inequality, along with a lifetime measure, failed to reach conventional levels of significance.
Conclusion: Perceived inequality appears to be a robust predictor of AL and potentially contributes to subsequent physical health problems, particularly for more proximate forms of perceived inequality.

1. Introduction

Physical health problems present a concerning economic challenge. During 2015, total health care spending in the United States reached $3.2 trillion, with the majority of the overall financial burden shouldered by the U.S. government (37%; Centers for Medicare and Medicaid Services, 2016). President Obama (2016) highlighted the immense cost of U.S. healthcare in a recent Special Communication in the *Journal of the American Medical Association,* which noted that 16% of the U.S. economy was spent on health care in 2008 (p. 526). Along with the financial burden of health care costs on the U.S. government, individuals are also tasked with substantial out-of-pocket costs totaling $338.1 billion in 2015, representing a 2.6% increase from the previous year. Physical health problems can also result in a number of indirect costs including a loss of productivity and absenteeism which may further compound the estimates reported above (Schultz et al., 2009).

Based on these observations, a substantial literature has focused on identifying factors that ultimately contribute to variation in health problems, with a particular emphasis on health disparities resulting from differences in socioeconomic status (SES; Dowd et al., 2009; Goldman, 2001; Sanders-Phillips et al., 2009). Taken together, the results of this expansive literature have revealed that physical health problems including obesity (Gordon-Larsen et al.,

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2006), cardiovascular disease (Diez-Roux et al., 2000), and diabetes (Kumari et al., 2004) are more prevalent among individuals that occupy lower social positions. This pattern of results is so robust it has come to be referred to as a "social gradient in health" in the existing literature (Goldman, 2001, p. 119).

Drawing from Geronimus's (1991) "weathering hypothesis," previous studies have found evidence suggesting that individuals who experience social or economic adversity and political marginalization (such as racial minorities and African-Americans, more specifically) also experience more health problems. While the weathering hypothesis was originally formulated to explain racial differences in chronic morbidity (Geronimus, 1991; Geronimus et al., 2006), the underlying explanations may also provide a better understanding of the mechanisms driving the association between SES and physical health. The weathering hypothesis has enjoyed support in the existing literature, with previous studies reporting evidence of greater overall levels of stress among racial minorities (Brody et al., 2014; Geronimus et al., 2006, 2010; Green and Darity, 2010) and that such increases in stress are significantly associated with a wide range of physical health problems (Geronimus et al., 2010; Jackson et al., 2010; Turner and Avison, 2003). Previous studies have also revealed that greater overall perceptions of inequality and discrimination ultimately result in increased levels of stress (Brunner, 1997; Delhey and Dragolov, 2014). While racially motivated sources of discrimination are the most commonly examined in the existing literature (for example, see Brody et al., 2014; Geronimus et al., 2006), previous studies have also observed discriminatory treatment based on other characteristics including gender (Puhl et al., 2008), sexual orientation (Meyer, 2003), age (Oliver, 2009), and religious belief (Ghumman et al., 2013). Taken together, these findings indicate that inequality and discriminatory treatment, regardless of the impetus for such experiences, may have negative consequences and increase overall stress levels.

The underlying connection between stress and physical health has been inherently tied to the theoretical concept of *allostatic load* (AL), which refers to the cumulative wear and tear on various physiological systems due to exposure to chronic and acute sources of stress (McEwen, 1998; McEwen and Seeman, 1999; Sterling and Eyer, 1988). More specifically, AL is related to the process of *allostasis* (Sterling and Eyer, 1988), which refers to the activation of physiological systems including the cardiovascular, metabolic, immune, and endocrine systems based on demands stemming from encountered environmental experiences (McEwen, 1998; McEwen and Seeman, 1999). In most situations, allostasis is considered adaptive and provides the physiological responses necessary for encountering and overcoming an environmental obstacle. However, the process of allostasis is primed for acute stressors that persist for only a relatively short period of time, as physiological responses to such stressors are expected to be activated intermittently and normalize after the stressor subsides. When such physiological systems are activated more consistently and for more sustained periods of time, overall levels of allostatic load (or system dysregulation) may increase. Previous studies have indicated that greater overall levels of AL are associated with a wide range of deleterious physical and mental health outcomes (Juster et al., 2010; Mattei et al., 2010; Seeman et al., 2001).

Despite the connection between AL and such outcomes, the underlying process that ultimately links these two concepts is complex and involves multiple mediators. McEwen and Seeman (1999) provided an overview of these processes by distinguishing between *primary mediators*, *primary effects*, *secondary outcomes*, and *tertiary outcomes*. *Primary mediators* refer to stress hormones (i.e., cortisol and other glucocorticoids) released during the process of allostasis that have widespread influences on multiple physiological processes (or *primary effects*) and operate through "cellular events" which involve enzymes, receptors, ion channels, and structural proteins (Juster et al., 2010). Over time, downstream physiological systems attempt to compensate for the over- or underproduction of primary mediators to overcome the detrimental impact of primary effects, resulting in functional changes in such systems. These changes, or *secondary outcomes*, can impact cardiovascular, immune, and metabolic (among other) systems, causing such systems to operate at sub-optimal levels and ultimately resulting in dysfunction.

Directly in line with these findings, previous studies have demonstrated a positive and significant association between unequal treatment and physical health problems, such that individuals with greater perceptions of inequality display greater overall levels of inflammation (which has been linked to increased risk for cardiovascular disease and diabeates; Cunningham et al., 2012); increased levels of cortisol (Fuller-Rowell et al., 2012); increased sympathetic nervous system activity (Sawyer et al., 2012); and, greater overall levels of AL (which has been linked to neural atrophy, heart disease, and memory problems; Brody et al., 2014; Geronimus et al., 2006; Green and Darity, 2010; Gruenewald et al., 2012). In addition, the results of a recent meta-analysis synthesizing the results of 36 studies and 303 individual effects found a negative and significant association between perceived discrimination and physical health outcomes ($r = -0.13$), indicating that individuals who experienced more discrimination also exhibited poorer physical health outcomes (Pascoe and Smart Richman, 2009).

Despite the significant number of studies reporting associations between perceived inequality or discrimination and AL, determining whether such associations may be interpreted as causal effects represents a methodological challenge. The process of selection, whereby individual characteristics that increase the likelihood of physical health problems may also be more likely to elicit unfair treatment, remains particularly problematic. Although some previous studies have employed sophisticated multivariate research designs to limit selection effects, such designs are limited in that they only control for *observed* measures of selection. Additional latent or *unobserved* sources of selection remain particularly important, but are more difficult to address methodologically (Goldman, 2001). These sources of influence can encompass virtually any background variables that potentially impact the examined association (such as family experiences or genetic influences) and failing to take such influences into account may result in biased findings. For example, Hamdi et al. (2016) recently examined the association between educational attainment (a common measure of SES) and AL. The results revealed a significant association in preliminary models; but after including additional controls for unobserved sources of selection, including genetic and environmental influences that cluster within families, the association fell from significance.

Directly in line with these findings, the current study aims to examine the potential association between perceived inequality and AL with a twin comparison model, which includes controls for both observed and unobserved sources of selection (Hamdi et al., 2016; Turkheimer and Harden, 2014). In addition to the use of a twin comparison model, the current study also makes use of a comprehensive measure of AL. Few existing studies have employed comprehensive measures of AL when examining the potential ramifications of unequal or discriminatory treatment, with the majority of the existing literature focusing on dysfunction within a single physiological system or a subset of systems (Cunningham et al., 2012; Fuller-Rowell et al., 2012; Sawyer et al., 2012). Of the few studies that have employed a comprehensive measure of AL (Brody et al., 2014; Geronimus et al., 2006), the number of