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Article

The contribution of three dimensions of allostatic load to racial/ethnic disparities in poor/fair self-rated health

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ABSTRACT

Objective: This study evaluates whether different dimensions of physiological dysregulation, modeled individually rather than additively mediate racial/ethnic disparities in self-reported health.
Methods: Using data from the National Health and Nutrition Examination Survey (2005–2010) and the Karlson, Hold, and Breen (KHB) mediation model, this paper explores what operationalization of biomarker data most strongly mediate racial/ethnic disparities in poor/fair self-rated health (SRH) among adults in the United States, net of demographic, socioeconomic, behavioral, and medication controls.
Results: Non-Hispanic blacks and Hispanics had significantly higher odds of reporting poor/fair self-rated health in comparison to non-Hispanic whites. Operationalizations of allostatic load that disaggregate three major dimensions of physiological dysregulation mediate racial/ethnic disparities strongly between non-Hispanic blacks and non-Hispanic whites, but not between Hispanics and non-Hispanic whites. Disaggregating these dimensions explains racial/ethnic disparities in poor/fair SRH better than the continuous score. Analyses on sex-specific disparities indicate differences in how individual dimensions of allostatic load contribute to racial/ethnic disparities in poor/fair SRH differently. All individual dimensions are strong determinants of poor/fair SRH for males. In contrast, for females, the only dimension that is significantly associated with poor/fair SRH is inflammation. For the analytic sample, additive biomarker scores fit the data as well or better than other approaches, suggesting that this approach is most appropriate for explaining individual differences. However, in sex-specific analyses, the interactive approach models fit the data best for men and women.
Conclusions: Future researchers seeking to explain racial/ethnic disparities in full or sex-stratified samples should consider disaggregating allostatic load by dimension.

Introduction

Biomarker data are widely used in population health research, especially in the study of the concept of allostatic load. Most previous research on this topic employs a continuous score approach which indicates how many of a participant's biomarkers exceed a given threshold that is either clinically or empirically determined. This continuous score is intended to capture multi-system physiological dysregulation by combining numerous biomarkers from different biological systems. However, previous research largely employs canonical methods for AL score construction without investigating what operationalization of the underlying biomarkers best serves their research purposes. The continuous score strategy has yielded many research insights, but we argue that a reexamination of this operationalization is timely and appropriate.

This paper reexamines the continuous score strategy by comparing it to alternative operationalizations of the same biomarkers by comparing their explanatory power for individual differences and racial/ethnic disparities in self-rated health (SRH) for a nationally representative sample of adults in the United States. We compare the continuous score measure to dichotomous variables indicating clinically significant biomarker values in the cardiovascular, metabolic, and inflammatory systems, and model the effects of dysregulation in each system both additively and interactively. To our knowledge there is no research investigating whether different specifications of allostatic load mediate racial/ethnic differences in SRH. To achieve this goal, identifying the appropriate operationalization of AL is critical to understanding individual differences and racial/ethnic disparities in this key health measure.

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Background

Allostatic load: concept and measurement

Although the biomarkers used in allostatic load (AL) scores are biological measurements, individual and group differences therein are heavily linked to variations in the social environment. The social determinants of health framework argues that economic, early life, social context, environmental conditions, as well as individual characteristics and behaviors affect health outcomes such as morbidity, health status, functional limitations, healthcare expenditures and mortality (Macgregor, 1961). Exposure to unequal socioeconomic and environmental conditions, paired with individual characteristics, has been associated with homeostatic imbalance (McEwen, 1998), which triggers processes within the body that aim to correct this imbalance (McEwen, 1998; McEwen & Wingfield, 2003). The process of allostasis leads to the adaptation of the organism to these unequal conditions; the lasting effect of adaptation accumulates in the body through "wear and tear" (McEwen & Wingfield, 2003). Researchers have begun to reveal links between the concept of this "wear and tear", or allostatic load (AL), and a wide variety of health outcomes. AL scores are constructed from a variety of biomarkers to summarize the resulting burden of continuing internal processes which aim to attain or maintain stability within the body under stressful conditions (McEwen & Seeman, 1999).

Many AL studies and conceptual models consider three major dimensions of physiological dysregulation, using cardiovascular (CM), metabolic (MM, including anthropometric measures), and inflammatory (IM) biomarkers (McEwen, 1998; Doung, Bingham, Aldana, Chung & Summer, 2017; Morrison, Shenassa, Mendola, Wu & Schoendorf, 2013; Juster, McEwen & Lupien, 2010). Every approach begins with a set of biomarkers, which are then converted to a more informative value either by dichotomizing the underlying value compared to a clinically- or empirically-significant threshold, or by converting the values to a standardized distribution. These converted values are then summed together into a continuous AL score, typically without distinguishing between the biological systems involved, which may limit their explanatory power if each dimension does not contribute equally to health outcomes for individuals or these associations vary by race/ethnicity.

A recent review of the methods employed to construct allostatic load scores (Doung et al., 2017) indicates that researchers vary substantially in which biomarkers are used to construct these indexes of biological dysregulation. Although the number of biomarkers used for each score varies by study, with number of biomarkers considered ranging from 7 to 14, all of them include markers from the aforementioned dimensions. Despite the pervasive use of multiple biological dimensions in the construction of these continuous scores, relatively little research explores how similarly each system influences individual differences and racial/ethnic disparities in these health outcomes.

Racial/ethnic and sex disparities in self-rated health

In the United States (U.S.), racial/ethnic disparities are frequently documented for SRH, as higher proportions of non-Hispanic Blacks (NH Blacks) and Hispanics report poor or fair health when compared to non-Hispanic Whites (NH Whites) (Woo & Zajacova, 2016; Borrell & Dallo, 2008), a difference that remains strong even when models are adjusted for social status, access to healthcare services, and health behaviors (Lo, Howell & Cheng, 2013). Moreover, NH Blacks-NH White differences exist for the majority of health outcomes. NH Blacks have been found to have higher mortality rates (Levine, Foster & Fullilove, 2001), disability rates (Fuller-Thomson, Nuru-Jeter, Minkler & Guralnik, 2009; Hayward, Hummer, Chiu, González-González & Wong, 2014), lower life expectancy (Harper, MacLehose & Kaufman, 2014; Elo, Beltrán-Sánchez & Macinko, 2014), higher rates of engagement in risky health behaviors (Kawachi, Kennedy & Glass, 1999), and lower levels of engagement in exercise or healthy diets (August & Sorkin, 2011). In a study of the reliability of SRH measures, where respondents reported SRH on 2 occasions (about 1 month apart), NH Blacks were more likely than NH Whites to change their SRH answer and report worse health status (Zajacova & Dowd, 2011). Most recent approaches to understanding the NH Black-NH White gap in SRH have incorporated controls for period and cohorts (Beck, Finch, Lin, Hummer & Masters, 2014), wealth (Hajat, Kaufman, Rose, Siddiqi & Thomas, 2011), health conditions (Banerjee, Perry, Tran & Arafat, 2010), and contextual variables (Subramanian, Acevedo-Garcia & Osypuk, 2005; Bjornstrom & Kuhl, 2014) (i.e. residential segregation, percent NH Black within the county, etc.) but none of these have been able to eliminate the NH Black-NH White disparity.

The difference between NH Whites and Hispanics continues to puzzle researchers as the latter group has been found to have lower or similar mortality rates (Markides & Coreil, 1986), infant mortality risk (Hummer, Powers, Pullum, Gossman & Frisbie, 2007), poor/fair self-rated mental health (Santos-Lozada, 2016), self-reported hypertension among Hispanic-Whites (Borrell, 2009), and low birth-weights (Johnelle Sparks, 2009) when compared to NH Whites. This pattern has been termed the epidemiological paradox (Markides & Coreil, 1986) because it is inconsistent with these groups' respective socioeconomic positions in US society. SRH is one of the few outcomes where evidence that contradicts this paradox is present (Dubard & Gizlice, 2008; Viruell-Fuentes, Morenoff, Williams & House, 2011; Kandula, Lauderdale & Baker, 2007), however. The contrast between the usual pattern of Hispanic health advantage and poorer SRH has been referred to as the "Latino health puzzle" (Viruell-Fuentes et al., 2011). Numerous factors have been hypothesized to explain this puzzle, including language of interview (Dubard & Gizlice, 2008; Kandula et al., 2007), rating health based on different factors (Bzostek, Goldman & Pebley, 2007), socioeconomic and cultural influences (Kandula et al., 2007; Markides & Martin, 1979), and contextual effects (Patel, Eschbach, Rudkin, Peek & Markides, 2003), among others (Bzostek et al., 2007).

Furthermore, in the U.S., differences in health status by sex are well documented, as women tend to report higher poor/fair SRH when compared to males despite incorporating controls for demographic and socioeconomic characteristics (Prus, 2011). Because of this, a growing body of literature has started to pursue analyses of SRH and its determinants stratifying by sex. Two reasons for doing so are particularly salient here. First, men have lower odds of reporting poor/fair SRH than females but lower life expectancy (Gorman & Read, 2006; Zajacova, Huzurbazar & Todd, 2017; Oksuzyan et al. 2009; Case & Paxson, 2005). This pattern has been termed the male-female health-survival paradox and has been roughly translated to imply "men die, women suffer". Others have indicated that "females are sicker, but males die sooner" (Arber & Cooper, 1999). In light of gender differences in these later life outcomes, it is likely that allostatic load and its individual dimensions differentially explains individual and racial/ethnic differences in SRH by gender.

Second, poor/fair SRH predicts mortality better for males than for females (Hirve, Juvekar & Sambhudas, 2012; Ross, Masters & Hummer, 2012). Given that the predictive power of SRH varies by sex, it may be possible that SRH is capturing different elements of subjective health and this produces the differences in reporting SRH. Because differences exist in SRH reporting (i.e. male-female health survival paradox) and the difference in predictive power for subsequent mortality varies by sex, a sex-specific analyses is deemed both appropriate and essential to better understand the contribution of the dimensions of allostatic load to SRH. In summary, given significant differences by sex in self-rated health, biomarkers, and potentially the relationship between them, we pursue this sex-specific analysis to examine whether our findings are comparable to those found in the complete analytical sample or not.