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Short Report

A multifaceted analysis of social stressors and chronic inflammation

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ABSTRACT

Chronic stress has been linked to negative health outcomes, including increased inflammation, which can be measured by high-sensitivity C-reactive protein (CRP). Prior research has focused almost exclusively on relationships between individual social and demographic stressors and CRP. The objective of this study is to assess the role of multiple potential stressors simultaneously to determine which key stressors are related to risk of high CRP, given that sustained stress and resulting inflammation may have long-term health implications. We hypothesized that negative social and environmental factors would be associated with high CRP. Data from two waves of Midlife in the United States were used to predict high CRP with variable selection procedures and logistic regression. Results indicated females, those with greater BMI, those with improvements in family strain, and those with higher A1c had a greater risk of high CRP. There was limited evidence that negative social factors were associated with CRP to the extent seen in prior literature. A key advantage of the study was testing multiple potential determinants of chronic stress and inflammation simultaneously, advancing the existing literature. Results demonstrate the potential usefulness of a multifaceted approach to evaluating the risk of chronic inflammation and high CRP.

1. Introduction

In 2016 and 2017, Americans reported higher stress than 2007 (American Psychological Association, 2017a, 2017b) with symptoms such as anxiety, anger, and fatigue (American Psychological Association, 2017a). Stress profoundly affects humans due to longer periods in psychological stress, compared to other animals who experience short bursts of stress followed by prolonged homeostasis periods (Sapolsky, 2004). Because stress is increasing in Americans, it is imperative to investigate health and quality of life outcomes to understand the impact of prolonged stress.

A multitude of factors have been identified as contributing to prolonged stress, including social factors, such as poverty (distinct from low socioeconomic status (SES)), violence exposure, and caregiving (Oliveira et al., 2016). Social stressors turn into prolonged stress when they initiate a stress response in the body (Oliveira et al., 2016). Chronic inflammation results from a prolonged increase in stress hormones (Barr, 2014) and is linked to negative health outcomes (connected to chronic stress (Kubzansky, Seeman, & Glymour, 2014; Riancho & Brennan-Olsen, 2017) and usually associated with advanced age), such as cardiovascular disease (CVD) (Kubzansky et al., 2014). Elevated levels of CRP (≥10 mg/L) can indicate the development of CVD (Alley et al., 2006), which may lead to poorer health and quality of life. Fig. 1 illustrates the interplay of social/environmental stressors for inflammation developed from the models of Barr (2014), Kubzansky et al. (2014), and Riancho and Brennan-Olsen (2017). CRP is a convenient biomarker for assessing potential diagnoses of CVD, among other medical conditions, as it can be measured through point-of-care in clinical contexts and through minimal processing of saliva and blood samples in research contexts. Thus, understanding the exact nature of its relationship to environmental and biological factors is important.

Demographic indicators, such as race/ethnicity (Nikulina and Widom, 2014), immigration status (Alley et al., 2006), and gender (Loucks et al., 2010; Sbarra, 2009) have been demonstrated to predict elevated CRP, albeit with a highly complex pattern. Relatedly, neighborhood SES was implicated for elevated CRP (Uchino et al., 2016), along with neighborhood disorder (Holmes & Marcelli, 2012), and neighborhood quality (deprivation and problems) (Nazmi et al., 2010), although at least in one study obesity and dietary fat intake explained much of the link (Von Känel et al., 2012). The literature shows inconsistencies in the association between SES and CRP, which is in part due to the many ways in which SES was conceptualized and measured, but poverty was associated with very high CRP levels (> 10 mg/L) when compared to those above the poverty level, and when chronic health conditions and obesity were present (Alley et al., 2006). Given such past findings, we too focus on higher levels of CRP (> 3 mg/L and <

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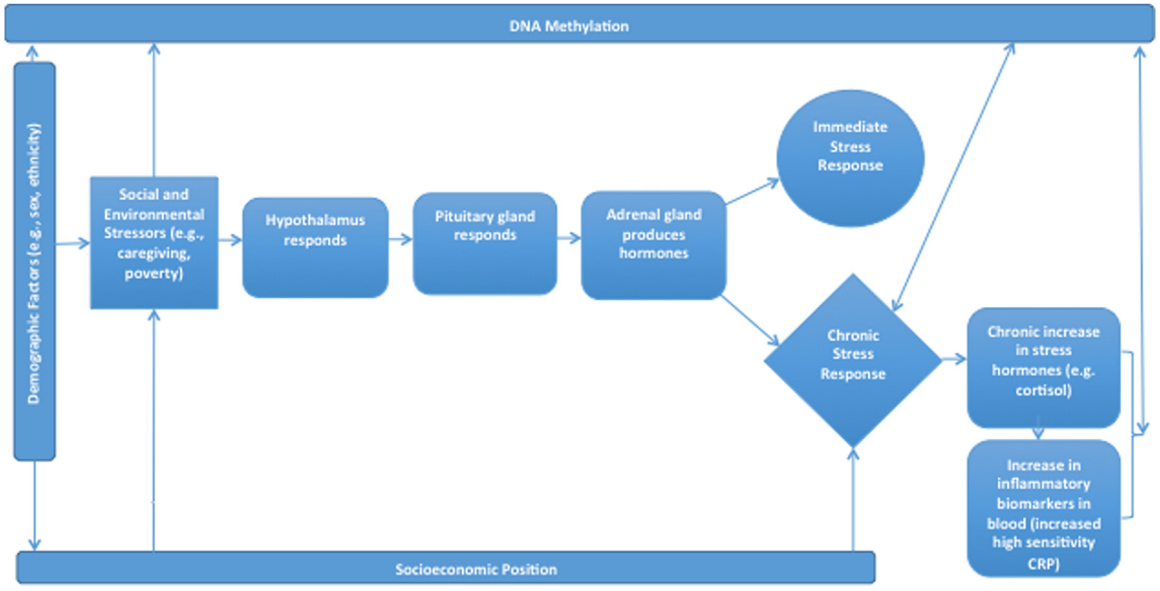

Fig. 1. Conceptual Model of Biological Response to Social Stressors.

10 mg/L) to investigate disparities.

Education, another potential marker of SES, has also been inversely linked to CRP levels (Loucks et al., 2010), consistent with greater trends in health conditions. Later first marriage in men has appeared to be protective in relation to CRP (Sbarra, 2009), but previously married older men displayed higher CRP levels, like married and unmarried women (Sbarra, 2009). Caregiving for a spouse with Alzheimer's has been associated with increased CRP until the spouse's death, after which CRP declined (Von Känel et al., 2012), suggesting inflammation increases during prolonged stress.

Social support displayed an inverse relationship to CRP levels in the literature. Research has demonstrated higher CRP levels are associated with social factors in some studies, including low social support or social isolation, with results typically seen in men and sometimes only in narrow age ranges (Heffner et al., 2011; Ford, Loucks, & Berkman, 2006). However, there is limited evidence that high social support can buffer the relationship between chronic stress and CRP (Runsten et al., 2014). Further, spousal support displayed an inverse relationship with age- and sex-adjusted levels of CRP (Yang, Schorpp, & Harris, 2014).

Individuals who have sustained trauma experience prolonged stress (Williamson et al., 2015; Johnson, Delahanty, & Pinna, 2008), but many other circumstances can also affect levels of inflammation in adulthood through epigenetic mechanisms (McDade et al., 2017) (e.g., stressful life events like parental absence). Childhood adverse events are important to examine as they have been associated with greater levels of inflammation in adults, specifically CRP (Danese et al., 2007; Kiecolt-Glaser et al., 2011), and enhance inflammation from adult stressors, such as caregiving (Kiecolt-Glaser et al., 2011). Exposure to violence in childhood was linked to increased CRP continuing into adulthood (Runsten et al., 2014; Danese et al., 2007), as was exposure to poverty (Nikulina & Widom, 2014). Furthermore, childhood adverse events seem to be associated with less social support (Runsten et al., 2014). Therefore, this paper endeavors to examine factors in childhood, their impact on adulthood, and adulthood-specific events, to investigate how they impact markers of inflammation.

The variety of stressors examined in the CRP literature creates a complex picture of stress's role in elevating CRP. However, the relationship among all of the stressors has not been quantified. Because of the potential for omitted variable bias, it is important to evaluate stressors concurrently (Alley et al., 2006), an approach this paper will take. Drawing from the wide-ranging literature on individual stressors associated with CRP, we developed the following research question: What are the key social stressors affecting Americans that lead to chronic inflammation, as measured by high CRP? This question is of interest because sustained stress and resulting inflammation may compromise long-term health outcomes and quality of life. The main hypothesis is the following: Negative social and environmental factors will be associated with high CRP.

2. Materials and methods

2.1. Materials

Data come from Midlife in the United States: A National Longitudinal Study of Health & Well-Being (MIDUS). We used MIDUS 1, collected in 1995 and 1996, and MIDUS 2, the longitudinal follow-up collected between 2004 and 2006. MIDUS 1 was used to obtain background variables. For MIDUS 2 we used Projects 1 and 4. Project 1 includes a follow-up of MIDUS 1 data, plus additional questions about relevant topics, such as caregiving. Project 4 is the biomarker study, which allowed us to study CRP and potentially related biological factors, such as A1c and cholesterol. There were 1054 individuals who responded to both MIDUS 1 and MIDUS 2, including the biomarker study.

2.2. Sample

We restricted the sample to participants who responded to MIDUS 1, and MIDUS 2 Projects 1 and 4 with full data on the CRP outcome (45 cases dropped); with no exclusionary health conditions (i.e., conditions that may unduly affect the outcome: tuberculosis, burns, autoimmune/Lupus disease) (31 cases dropped); and with full data on our selected predictor variables (201 cases dropped; data missing primarily for BMI at time 1 and menopause status; some additional missing data on biomarkers and predictors such as strain, social support, neighborhood quality, and poverty). Total analytic sample size was 777. Supplementary Table 1 includes descriptive statistics for the analytic sample compared to the full biomarker sample. The full biomarker sample is slightly more female, slightly older on average, and slightly more likely to be caring for an adult at MIDUS 2.

2.3. Variables

The outcome variable was high CRP, measured at MIDUS 2. All