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Nonlinear associations between chronic stress and cardiovascular reactivity and recovery☆

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ABSTRACT

A mixed literature on the influence of chronic and acute stress on cardiovascular reactivity (CVR) and recovery suggests a need for improved modeling of these associations. We examined these associations using both linear and nonlinear (quadratic) models. Data were collected on 129 healthy adults [59% female, ages 18–29 years]. Participants completed the Perceived Stress Scale (PSS) after engaging in a mental arithmetic and a stress recall task. Heart rate (HR), systolic and diastolic blood pressure (SBP, DBP) were measured during rest, task, and recovery periods. Hierarchical ordinary least squares regression was used to examine the association of chronic stress to CVR and recovery with initial cardiovascular values and body mass index entered first as covariates. Hierarchical linear modeling (HLM) was also used for recovery. For reactivity, a quadratic relationship between PSS scores and DBP was observed in females such that those scoring at moderate levels of stress displayed lesser reactivity than those scoring either low or high. For recovery, a quadratic model was supported for SBP among females, with moderate levels of stress associated with greater recovery relative to either low or high levels. For females the quadratic model was also supported for SBP and DBP when examined using HLM. Quadratic modeling may better represent current theories of how chronic stress influences CVR and recovery. Our findings further suggest that these associations may be differentially evident by gender, perhaps due to gender differences in reported stress levels or gender-related task relevance.

1. Introduction

The cumulative effects of stress are apparent on many physiological systems and are thought to play a significant role in disease (McEwen and Stellar, 1993). With regard to cardiovascular disease, an extensive literature has identified individual differences in cardiovascular reactivity (CVR) during acute stress as one potential marker of risk that may contribute to atherogenesis through physical and neuroendocrine damage (Krantz and Manuck, 1984; Manuck, 1994). Poor cardiovascular recovery from acute stress is another increasingly appreciated marker of risk for hypertension and cardiovascular disease more generally (Brosschot and Thayer, 1998; Carroll et al., 2001; Linden et al., 1997; Steptoe and Marmot, 2005). Stress occurring over a longer duration may also impact cardiovascular risk insofar as it affects the degree of physiological response and recovery to acute stress, and alters basal levels of cardiovascular function (Dienstbier, 1989; Evans and Kim, 2007; Rozanski et al., 1999; Suarez et al., 1997).

Despite progress in this field, the literature is rather mixed. Among the 19 studies available a decade ago (Gump and Matthews, 1999), about half reported potentiating effects of "background stressors" (i.e., chronic stressors) on CVR, with the remaining studies reporting null findings, or even attenuated reactivity to acute stress. More recent findings overall show a dampening effect of chronic stress on CVR. For example, dampened CVR to acute laboratory stress was reported among adolescents with a greater lifetime exposure to violence (Murali and Chen, 2005). Blunted reactivity has also been associated with exposure to poverty among adolescents (Evans and Kim, 2007), and the occurrence of disruptive life events among middle-aged and older adults (Carroll et al., 2005). In contrast, adults reporting a greater degree of past racial discrimination have demonstrated greater reactivity to an anger recall task (Richman et al., 2007).

Recovery from acute stress has received comparatively less attention. A handful of laboratory studies report that chronic stress is associated with slower recovery from acute laboratory stress (Lepore et al., 1997). This is consistent with findings outside of the laboratory that chronic stress is associated with prolonged cardiovascular activation (Pieper and Brosschot, 2005). However, despite the dynamic nature of cardiovascular recovery from acute stress, a majority of investigators analyze recovery data using change scores or repeated-measures analysis of variance (ANOVA). Currently, more advanced statistical models, such as hierarchical linear modeling (HLM) are available that may better capture individual change

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during recovery (Llabre et al., 2004), and thus may offer greater insight to how recovery from an acute challenge is impacted by chronic stress.

Researchers have also invariably adopted linear conceptualizations of how chronic stress relates to both CVR and recovery, such that greater stress produces either enhanced or blunted CVR, and greater stress produces either quicker/better or slower/poorer recovery. Despite some theoretical and empirical support for each view, these alternatives might not be mutually exclusive. Rather, each may describe different points on a single curve representing the relation between chronic stress severity and cardiovascular function. Thus, cardiovascular reactivity may initially increase as the level of chronic stress increases to a moderate degree, and then be more attenuated at the highest levels.

A concurrent examination of stress physiology models supports this nonlinear conceptualization. Dienstbier (1989) in his "physiological toughness" model, reviewed studies indicating that animals with moderate amounts of stress relative to animals with low stress displayed a physiological toughening of catecholamine responses to, and recovery from, acute stress. That is, moderately stressed animals demonstrated a greater reactivity response to acute stressors followed by a more rapid recovery relative to low stressed animals. He suggests that moderate levels of life stress in humans may similarly condition or "toughen" individuals to show a heightened but adaptive physiological reaction to acute challenges followed by relatively quick recovery. In contrast, under conditions of severe ongoing stress, Selye's work on adaptation would suggest that individuals become physiologically depleted and fail to react sufficiently to environmental demands (i.e., exhaustion) (Selye, 1973). Likewise, McEwen's conception of allostatic load suggests that individuals under significant long-term stress may demonstrate inadequate physiological responses to stress, and/or exhibit prolonged physiological responses (i.e., poor recovery) (McEwen, 1998). More specifically, Porges (1995) suggested that severe stress is associated with blunted sympathetic reactivity. This is consistent with the idea that patients with generalized anxiety disorder, who arguably experience chronic and severe stress levels, characteristically exhibit lesser sympathetic reactivity to and slower recovery from acute stress compared to nonanxious individuals (Hoehn-Saric and McLeod, 1988).

In the present study we measured perceived stress over the past 30 days and related this to indices of cardiovascular function during a mental arithmetic (MA) and a stress recall task using both linear and nonlinear (quadratic curve) analytic approaches. Based on the above models, we expected that heart rate (HR) and blood pressure reactivity would increase from low to moderate levels of perceived chronic stress. In contrast, models highlighting the physiological depletion associated with high levels of chronic stress suggest that reactivity would be blunted across all cardiovascular domains among the highest stress appraisers. For recovery, we expected that moderate levels of stress would be associated with more rapid and complete recovery compared to the lowest or highest levels of stress. Because of known gender differences in CVR and recovery (Light et al., 1993), we examined the associations between stress and CVR and cardiovascular recovery separately for men and women.

2. Method

2.1. Participants

This study was approved by the University of Michigan-Dearborn Institutional Review Board and conformed to United States federal ethical standards. All participants provided informed consent prior to inclusion in the study. Three hundred eighty seven students were recruited from introductory psychology classes. The posting for this study stated that participants must refrain from alcohol (12 h) and caffeine (6 h) prior to attending. Self-reported adherence to this was assessed along with other exclusionary criteria that included a family history of early cardiovascular disease, psychiatric conditions, and medical conditions or medication use that could affect cardiovascular function. Participants were excluded from data analysis if they were a current smoker, had a body mass index >30, high blood pressure (systolic >140, diastolic >90), or an age greater than 35 years. The final sample consisted of 129 participants (female = 76) aged 18–29 years ($M = 19.34$, $SD = 2.06$), self-identifying as White (74), Arab (27), African American (10), Asian (9), Hispanic (3), American Indian or Alaskan Native (1), Pacific Islander (1), and Other (4).

2.2. Protocol timeline

Data were collected with the following timeline: post-instrumentation rest (4 min); baseline (4 min); MA task (4 min); affect assessment; post-MA rest (2 min), stress recall task (4 min), affect assessment; re-engagement in stress recall task (2 min); recovery (10 min); assessment of chronic stress appraisal; debriefing.

2.3. Tasks

Our first task, verbal mental arithmetic (MA) is widely used in CVR studies and offers comparability with prior investigations. Participants mentally subtracted by seven from a large 3 digit number, responding aloud with their answers. During the task, the experimenter periodically changed the starting number and prompted the participant to work faster, make fewer mistakes, and try harder (Brown et al., 2007; Waldstein et al., 1999).

The second task was a stress recall task, modeled after an anger recall task used in prior research (Brown et al., 2007; Waldstein et al., 2000). Participants were instructed to think about "a very stressful time in your life, but one that you are willing to talk to the researcher about". Participants were given 2 min to recall this scenario and were then asked to try and "re-live the situation" by describing the event to the experimenter. Throughout the task, the experimenter actively prompted participants for emotional expression and to state what they said and did in the situation. Prompts included probing for loss of personal control, feelings of helplessness, and reflection of emotional content. After completing the PANAS following the stress recall task, participants were re-engaged in the task for 2 min in order to eliminate the potential confound of self-reporting affect during the recovery phase (Lai and Linden, 1992; Neumann et al., 2004).

2.4. Measures

2.4.1. Perceived Chronic Stress

The Perceived Stress Scale (PSS) (10-items) assesses the degree to which individuals perceive themselves as having a sense of control over, and ability to cope with, nonspecific life events during the past 30 days (Cohen et al., 1983). The PSS has good internal consistency (coefficient alphas .84–.86) and has demonstrated construct validity (Cohen et al., 1983). Although various domains of stress may differentially relate to CVR (Gump and Matthews, 1999), objective measures of stress are likely to be confounded by differences in stress severity and duration across participants. Because measures of perceived life stress may better reflect a range of current background stressors for the individual, measuring stress this way should allow for a more valid examination of stress level across individuals and domains.

2.4.2. Affect

The Positive and Negative Affect Schedule (PANAS) (Watson et al., 1988) was administered immediately prior to and following the tasks to assess the emotional impact of the challenge. The PANAS consists of 20 adjectives describing positive and negative emotions, on a 5 point response scale. Coefficients alpha have been reported at .85 or greater for both the positive and negative affect subscales (Watson et al., 1988).

2.4.3. Physiological measures

An electrocardiogram (ECG) was collected with a Biopac MP150 system (Biopac Systems Inc., California) and data were digitized at a