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The interactive effect of change in perceived stress and trait anxiety on vagal recovery from cognitive challenge

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ABSTRACT

The present study tested the hypothesis that the change in state negative affect (measured as perceived stress) after cognitive challenge moderates the relationship of trait anxiety and anger to vagal recovery from that challenge.

Cardiac vagal control (assessed using heart rate variability) and respiratory rate were measured in a sample of 905 participants from the Midlife in the United States Study. Cognitive challenges consisted of computerized mental arithmetic and Stroop color–word matching tasks. Multiple regression analyses controlling for the effects of the demographic, lifestyle, and medical factors influencing cardiac vagal control showed a significant moderating effect of change in perceived stress on the relationship of trait anxiety to vagal recovery from cognitive challenges (Beta = .253, p = .013). After adjustment for respiratory rate, this effect became marginally significant (Beta = .177, p = .037). In contrast, for the relationship of trait anger to vagal recovery, this effect was not significant either before (Beta = .141, p = .257) or after (Beta = .186, p = .072) adjusting for respiratory rate. Secondary analyses revealed that among the individuals with higher levels of trait anxiety, greater reductions in perceived stress were associated with greater increases in cardiac vagal control after the challenge. In contrast, among the individuals with lower levels of trait anxiety, changes in perceived stress had no impact on vagal recovery. Therefore, change in perceived stress moderates the relationship of trait anxiety, but not trait anger, to vagal recovery from cognitive challenge.

1. Introduction

Trait anxiety and trait anger are established risk factors for incident hypertension and coronary heart disease, and for all-cause and cardiovascular mortality (Chida and Steptoe, 2009; Denollet and Pedersen, 2009; Kubzansky et al., 2006; Rutledge and Hogan, 2002). One pathway linking these factors to cardiovascular health outcomes may involve the cardiovascular response to psychological stress. Specifically, both exaggerated (Krantz and Manuck, 1984; Matthews et al., 2004; Treiber et al., 2003) and blunted (Carroll and Phillips, 2010; Phillips et al., 2009) cardiovascular reactivity to psychological stress, and delayed cardiovascular recovery from this stress (Heponiemi et al., 2007; Steptoe and Marmot, 2006; Stewart et al., 2006) predict adverse health outcomes. Evidence suggests that the predictive capacity of cardiovascular recovery from psychological stress may be stronger than that of cardiovascular reactivity (Gerin and Pickering, 1995; Stewart et al., 2006). HR recovery from psychological stress is vagally mediated (Mezzacappa et al., 2001), and cardiac vagal control is an established predictor of cardiovascular morbidity and mortality (Airaksinen, 1999; Kleiger et al., 1987; La Rovere et al., 1998; Tsuji et al., 1996). Thus, vagal recovery from psychological stress has important prognostic implications.

Abbreviations: BP, blood pressure; SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; HRV, heart rate variability; rMSSD, square root of the mean squared differences of successive RR intervals; CVD, cardiovascular disease; CHD, coronary heart disease.

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Previous studies investigating the association between trait anxiety, trait anger, and cardiovascular response (e.g., reactivity and recovery) to psychological stress have produced inconsistent results. While some investigators have reported that individuals with higher levels of trait anxiety have blunted cardiovascular (e.g., HR, systolic/diastolic blood pressure [SBP/DBP]) reactivity and delayed recovery (Girdler et al., 1997; Gonzalez-Bono et al., 2002; Gramer and Sprintschnik, 2008; Vitaliano et al., 1995), others have reported no association between trait anxiety and cardiovascular response to psychological stress (Jorgensen and Zachariae, 2006; Knepp and Friedman, 2008; Ottaviani et al., 2009; Schwerdtfeger, 2004). Similarly, some studies have linked higher levels of trait anger to exaggerated HR, BP (Burns et al., 2004; Ratnasingam and Bishop, 2007), and vagal (Ottaviani et al., 2009) reactivity, and delayed DBP recovery (Vitaliano et al., 1995), while others have linked higher levels of trait anger to blunted SBP reactivity (Laude et al., 1997) and found no association between trait anger and overall cardiovascular recovery (Lache et al., 2007). One possible reason for the inconsistency in these previously reported findings may be heterogeneity across studies. Specifically, previous studies differ in several critical dimensions, including the measures used to assess the trait characteristic, the samples studied, and the types of laboratory stressors utilized (de Rooij et al., 2010; Girdler et al., 1997; Gonzalez-Bono et al., 2002; Gramer and Sprintschnik, 2008; Jorgensen and Zachariae, 2006; Knepp and Friedman, 2008; Lache et al., 2007; Laude et al., 1997; Ottaviani et al., 2009; Ratnasingam and Bishop, 2007; Schwerdtfeger, 2004; Vitaliano et al., 1995). The differences in the study samples represent a particularly important issue as some studies used small samples (Girdler et al., 1997; Gonzalez-Bono et al., 2002; Gramer and Sprintschnik, 2008; Jorgensen and Zachariae, 2006; Laude et al., 1997; Schwerdtfeger, 2004) that were limited to either male (Girdler et al., 1997) or female (Gonzalez-Bono et al., 2002; Gramer and Sprintschnik, 2008; Ratnasingam and Bishop, 2007) participants, while reports based on large samples tended to have limited age range (de Rooij et al., 2010; Ratnasingam and Bishop, 2007; Vitaliano et al., 1995).

Alternatively, the inconsistency may be explained by the inherent lack of evidence regarding whether trait anxiety or trait anger alone are sufficient to generate a physiological response in the laboratory. It is possible that the stressfulness of the task — e.g., the degree to which the stressor elicits an increase in *state* negative affect, and the speed with which this state resolves after the stressor has ended, also may be important. For example, studies have demonstrated that state negative affect induced by laboratory stress is a strong predictor of the consequent cardiovascular reactivity and recovery (Demaree et al., 2004; Feldman et al., 2004; Gerin et al., 2006; Gramer and Sprintschnik, 2008; McClelland et al., 2009). Here too however, the findings are not fully consistent as some studies have found no association between state negative affect, and either cardiovascular reactivity or recovery (Gramer and Saria, 2007; Papousek et al., 2010; Schwerdtfeger, 2004).

In summary, previous studies investigating the association between trait anxiety and anger and cardiovascular reactivity to and recovery from psychological stress produced inconsistent results with some studies reporting significant associations (Burns et al., 2004; de Rooij et al., 2010; Girdler et al., 1997; Gonzalez-Bono et al., 2002; Gramer and Sprintschnik, 2008; Laude et al., 1997; Ottaviani et al., 2009; Ratnasingam and Bishop, 2007; Vitaliano et al., 1995) and some studies reporting none (Jorgensen and Zachariae, 2006; Knepp and Friedman, 2008; Lache et al., 2007; Ottaviani et al., 2009; Schwerdtfeger, 2004). Similarly, the literature that evaluated the links between state negative affect and cardiovascular reactivity and recovery is contradictory (Demaree et al., 2004; Feldman et al., 2004; Gramer and Saria, 2007; Gramer and Sprintschnik, 2008; McClelland et al., 2009; Papousek et al., 2010; Schwerdtfeger, 2004). Methodological heterogeneity among the studies, especially reliance on samples that were limited in their size or demographic representativeness may explain these inconsistencies. Moreover, it may be possible that state negative affect may impact cardiovascular response to stress only against the background of high state negative affect. In other words, state negative affect may moderate (Kraemer et al., 2008) the association between trait negative affect and the cardiovascular stress response.

The goal of the present study was therefore to test the hypothesis that state negative affect moderates the relationship of trait negative affect (trait anxiety and anger) to vagal recovery from challenge. We also investigated whether the hypothesized moderating effect is specific to either trait anxiety or anger, or whether this effect is non-specific and may be generalized to the both types of trait negative affect.

2. Method and materials

2.1. Participants

The data for the current study are from MIDUS II, a 9-year follow-up of the MIDUS I cohort. MIDUS is a national study of midlife development in the United States. MIDUS II included four new studies, one of which, the Biomarker Project conducted from December 2004 to March 2009, included a laboratory-based psychophysiology protocol, from which the current data were drawn. The detailed description of MIDUS study is available elsewhere (Love et al., 2010; Radler and Ryff, 2010).

2.2. Procedures

Participants traveled to one of three regional sites (Georgetown University, UCLA, or University of Wisconsin, Madison) for an overnight stay in a General Clinical Research Center. The measures of trait anxiety and anger (described below) were completed by the participant in the evening of their arrival. The following morning after a light breakfast with no caffeinated beverages, the psychophysiology protocol was administered. The patient reported to the study room. ECG electrodes were placed on the left and right shoulders, and in the left lower quadrant. The participant was seated, and a keypad for responding to the stress tasks was secured in a comfortable position relative to the dominant hand. Respiration was monitored by inductive plethysmography using the Inductotrace Respiration Monitor (Ambulatory Monitoring, Inc., Ardsley, NY). To measure respiration, stretch bands were placed around the participant's chest and abdomen. Analog signals from chest and abdomen bands were digitized at 20 Hz.

The protocol order (see Fig. 1) was: seated baseline (11 min); cognitive challenge 1 (mental arithmetic or Stroop task — 6 min); recovery 1 (6 min); cognitive challenge 2 (mental arithmetic or Stroop task — 6 min); recovery 2 (6 min). Task order was counterbalanced. Participants were instructed to remain silent throughout the procedures.

2.2.1. Cognitive stressors

2.2.1.1. Mental arithmetic task. A computer-administered mental arithmetic task (Turner et al., 1986) was utilized. The participant was presented with addition or subtraction problems on the computer monitor. After the problem appeared, the participants saw the word "equals" followed by an answer to that problem. The participants' task was to determine if the answer was correct or incorrect by pressing "Yes" or "No" on the keypad within 1 s. The level of difficulty was adjusted based on their performance. *The participants were told that their performance was being evaluated in terms of both speed and accuracy.*

2.2.1.2. Stroop color–word matching task. In this computer-administered version of the Stroop task, a color name (blue, green, yellow, or red) was presented on the computer monitor in a color that was either congruent