Neuroticism and Cardiovascular Response in Women: Evidence of Effects on Blood Pressure Recovery

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ABSTRACT  Neuroticism is a unifying personality trait that underlies a number of psychosocial risk factors for cardiovascular disease. One means by which Neuroticism may influence health risk is through effects on cardiovascular reactivity and recovery. Eighty-six women scoring high or low in Neuroticism took part in a paired interpersonal stressor task with a laboratory confederate. Conditions differed on the basis of the confederate’s interpersonal behavior: hostile, neutral, or friendly. Neuroticism interacted with condition to affect blood pressure recovery such that women high in Neuroticism showed less recovery following hostile interactions and greater recovery following friendly interactions. Main effects of Neuroticism on anger and anxiety reactivity were found. Results indicate that Neuroticism is relevant to cardiovascular health in the context of valenced social interactions. Implications for future study of Neuroticism and interpersonal stressors as risk factors for cardiovascular disease are discussed.

Increasingly, evidence supports an association between stable or chronic psychosocial factors and physical health. For example, depression, anxiety, hostility, and anger proneness are all associated with the development of health problems (Barth, Schumacher, & Herrmann-Lingen, 2004; Chida & Steptoe, 2009; Miller, Smith, Turner, Guijarro, & Hallet, 1996; Suls & Bunde, 2005), whereas optimism, conscientiousness, and the tendency to experience positive

The authors would like to thank Danielle Dorn, Julia Martin, and Alexandra Terrill for their important contributions toward the successful completion of this study.

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Journal of Personality 79:2, April 2011
© 2011 The Authors
Journal of Personality © 2011, Wiley Periodicals, Inc.
DOI: 10.1111/j.1467-6494.2010.00679.x
emotion are associated with health advantages (Kern & Friedman, 2008; Pressman & Cohen, 2005; Scheier et al., 1999). Much of the contemporary research on personality and health stems from two literature reviews addressing psychological predictors of coronary heart disease (CHD) conducted in the late 1980s. In the first review, Booth-Kewley and Friedman (1987) came to the conclusion that “modest but reliable associations exist between a number of personality variables and cardiovascular disease” (p. 355). These researchers went on to conduct a meta-analysis investigating whether a particular personality style is associated with increased risk of CHD and other illnesses (Friedman & Booth-Kewley, 1987). They found support for a “disease-prone personality” characterized by the frequent experience of negative mood states such as anxiety, depression, hostility, and anger. This personality type was deemed to be more relevant to health than the specific dispositional and mood factors taken independently.

Although the conclusions arrived at by Friedman and Booth-Kewley (1987) were received with criticism by some (e.g., Matthews, 1988), they did serve to stimulate interest in the study of broad personality variables that link together other factors associated with negative health outcomes. Neuroticism, a higher-order trait possessed by all individuals at varying levels (Eysenck, 1990; McCrae & Costa, 2003), is one such variable. Neuroticism has been conceptualized as a sensitivity to aversive stimuli that affects the propensity to experience negative mood states and cognitions (Watson & Clark, 1984). In their conceptualization of the Five-Factor Model of personality, Costa and McCrae (1992) identify Neuroticism as being composed of a number of lower-order facets. Several of these facets (anxiety, angry hostility, and depression) closely resemble constructs that have been demonstrated to predict the development of cardiovascular disease. The close associations between Neuroticism and the above risk factors suggest that Neuroticism may represent the “disease-prone personality” construct put forth by Friedman and Booth-Kewley (1987).

**Neuroticism as a Risk Factor for Health**

While Neuroticism was once considered to be primarily a marker of somatic complaints (e.g., Costa & McCrae, 1987), there has been renewed interest in the public health significance of Neuroticism in recent years (Lahey, 2009). A number of longitudinal studies have
demonstrated that high Neuroticism levels increase risk of mortality (Christensen et al., 2002; Mroczek & Spiro, 2007; Mroczek, Spiro, & Turiano, 2009; Murberg, Bru, & Aarsland, 2001; Ploubidis & Grundy, 2009; Shipley, Weiss, Der, Taylor, & Deary, 2007; Szekely et al., 2007; Terracciano, Löckenhoff, Zonderman, Ferrucci, & Costa, 2008; Wilson, Mendes de Leon, Bienias, Evans, & Bennett, 2004; Wilson et al., 2005). Findings from several studies suggest that cardiovascular factors are involved in the Neuroticism-mortality relationship. High Neuroticism levels have prospectively predicted death from cardiovascular disease in large community samples (Shipley et al., 2007; Terracciano et al., 2008), and all-cause mortality in cardiac patient populations (Murberg et al., 2001; Szekely et al., 2007). Now that we are at the stage where there is convincing evidence that Neuroticism predicts all-cause and cardiovascular-related mortality, the focus must shift to determining the mechanisms involved.

There are a number of models that outline pathways through which personality traits such as Neuroticism might influence physical health. Prominent among these are health behavior models that address personality effects on health-relevant behaviors such as smoking, exercise, and alcohol consumption. Other frameworks emphasize physiological pathways between personality and disease. Such stress-moderation models propose that personality traits affect the biological stress response and immune functioning via cognitive and behavioral factors such as stress appraisal, emotional reactivity, and frequency of stressor exposure. In support of the health behavior model, Neuroticism is known to relate to negative health behaviors (Booth-Kewley & Vickers, 1994). However, health behavior and stress-moderation models are not mutually exclusive, and it is widely assumed that both are necessary to adequately explain any relationships between personality and health. In a 30-year prospective study, Mroczek et al. (2009) estimated that approximately 60% of the relationship between Neuroticism and mortality could not be explained through two key health behaviors—smoking and excessive alcohol consumption. The authors suggested that physiological pathways may be responsible for a significant portion of the effect of Neuroticism unaccounted for by these established risk factors for cardiovascular disease and mortality. Ploubidis and Grundy (2009) found that Neuroticism had indirect effects on mortality in both men and women that were mediated through psychological distress,
somatic health, and smoking. Thus, it appears that researchers will need to consider both behavioral and physiological pathways when attempting to define the effects of Neuroticism on physical health.

Stress-moderation models emphasize that personality and other psychosocial factors can affect the biological stress response system in a manner that impacts risk of atherosclerosis and related disease development (Black & Garbutt, 2002; Rozanski, Blumenthal, & Kaplan, 1999). Perceived challenges to survival or emotional well-being activate the hypothalamic–pituitary–adrenal (HPA) axis and the sympathetic nervous system, resulting in endocrine, cardiovascular, and immune system changes. It has been hypothesized that frequent stress-related heart rate and blood pressure responses can damage the vascular endothelium, promoting the development of atherosclerotic lesions through the actions of corticosteroids, catecholamines, and inflammatory cytokines. This reactivity hypothesis states that increased frequency, magnitude, and duration of cardiovascular response increase disease risk (Manuck, 1994; Rozanski, 1998).

Cardiovascular reactivity (CVR) is defined as blood pressure and heart rate change in response to acute stressors or external stimuli (Linden, Earle, Gerin, & Christenfeld, 1997). CVR is concurrently associated with carotid atherosclerosis and exercise-induced ischemia (Kamarck et al., 1997; Kral et al., 1997) and has been found to predict coronary artery calcification, hypertension, increased left ventricular mass, and reduced endothelial integrity (Carroll et al., 2001; Jennings et al., 1997; Matthews, Zhu, Tucker, & Whooley, 2006; Pickering & Gerin, 1990; Treiber et al., 2003). Although much attention has been paid to CVR in the past, there is increasing interest in the study of cardiovascular recovery as a health-relevant factor. Cardiovascular recovery refers to the degree to which blood pressure and heart rate responses return to basal levels following a period of evoked response. Delayed recovery contributes to increased cardiovascular burden, and sustained elevations of cardiovascular variables following a stressor are associated with carotid artery atherosclerosis (Heponiemi et al., 2007) and are predictive of hypertension (Devereux & Pickering, 1991; Schuler & O’Brien, 1997). It has been proposed that recovery is superior to reactivity in its ability to predict future blood pressure (Stewart, Janicki, & Kamarck, 2006), and it is now widely accepted that recovery should
be assessed in studies examining the effect of psychosocial factors on acute cardiovascular response.

Neuroticism is associated with greater emotional reactivity to stressors (Bolger & Schilling, 1991; Suls, Green, & Hillis, 1998) and frequency of stressful experience (Hutchinson & Williams, 2007; Vollrath, 2000). However, it is unclear what bearing this has on the cardiovascular system. In a meta-analysis, Chida and Hamer (2008) examined 71 studies addressing the effects of “anxiety, neuroticism, or negative affect” on acute heart rate and blood pressure response. The authors concluded that these factors were associated with decreased cardiovascular reactivity and poorer cardiovascular recovery. However, the decision to lump Neuroticism together with a variety of different constructs (e.g., worry, state anxiety, social anxiety, panic disorder) calls into question the validity of their conclusions. An examination of contemporary health research studies using the more established measures of Neuroticism reveals mixed findings. Some investigations have identified no significant relationships between Neuroticism and cardiovascular reactivity to a Stroop task, a videogame stressor, imagery, a speech task, mental arithmetic, and a social stressor (Girdler, Jamner, & Shapiro, 1997; Schwerdtfeger, 2004; Steptoe, Melville, & Ross, 1984). However, others have shown Neuroticism to predict blood pressure reactivity to a mental arithmetic task and a pain stressor (Kennedy & Hughes, 2004; Rollnik, Schmitz, & Kugler, 1999), and heart rate reactivity to cold pressor and a range of mild physical and cognitive stressors (Kaiser, Beauvale, & Bener, 1997; Muth, Koch, & Stern, 2000; Schwebel & Suls, 1999). Unfortunately, none of the above-mentioned studies examined cardiovascular recovery.

Neuroticism is associated with interpersonal orientation (Smith & Ruiz, 2007), which suggests that people high in Neuroticism are vigilant toward the perception of negative social signals coming from people with whom they interact. This may be reflected in the associations between Neuroticism and social isolation and interpersonal conflict—two psychosocial health risk factors that have strong interpersonal components (Gallo & Smith, 2001). If interpersonal factors are salient for individuals high in Neuroticism, this trait could affect cardiovascular and emotional response during social interaction. Gonzalez-Bono et al. (2002) observed a personality × stressor interaction wherein highly neurotic individuals showed more heart rate reactivity to a speech preparation task than to a written essay.
preparation task. This finding demonstrated the importance of social and interpersonal factors in Neuroticism’s effect on cardiovascular response.

**Current Study**

The first objective of this study was to assess whether Neuroticism affects cardiovascular and emotional reactivity and cardiovascular recovery in response to an interpersonal stressor. Although Neuroticism is linked to emotional reactivity, its relationship to cardiovascular response is inadequately defined. The second aim was to determine whether the affiliative quality of an interpersonal stressor (hostile, neutral, or friendly) is relevant to the effect of Neuroticism on cardiovascular and emotional response. The quality of interpersonal interactions has previously been shown to affect cardiovascular response. For example, verbal harassment during a speech task produces greater blood pressure increase than supportive comments (Gallo, Smith, & Kircher, 2000). Neuroticism is associated with vigilance toward signs of threat or punishment (Derryberry & Reed, 2002; Verhaak, Smeenk, van Minnen, & Kraaimaat, 2004). Thus, for high-Neuroticism individuals, hostile interactions and those providing little information about the attitudes and intentions of the other (i.e., neutral or ambiguous interactions) might be more arousing relative to interactions that are unequivocally friendly in nature.

An acute stress paradigm was implemented in this study. These have a long history of use in behavioral health research, and stressor tasks that contain a social component reliably induce significant levels of CVR (Smith, Nealey, Kircher, & Limon, 1997). Social stressors are also argued to be more ecologically valid than traditional laboratory stressor tasks such as mental arithmetic or cold pressor (Smith, Glazer, Ruiz, & Gallo, 2004). Men were excluded from the investigation because women tend to score higher on several indicators of emotional experience (including physiological reactivity, reported emotional response, and emotional behaviors) in response to emotion-eliciting tasks (Bradley, Codispoti, Sabatinelli, & Lang, 2001; Chentsova-Dutton & Tsai, 2007; LaFrance, Hecht, & Paluck, 2003). This suggested that women would be more reactive to an interpersonal stressor task designed to elicit emotion, and therefore a useful target population for this study.
Predictions

1. The high-Neuroticism group would experience greater cardiovascular and emotional reactivity and less cardiovascular recovery than the low-Neuroticism group.

2. Neuroticism would interact with condition such that the high-Neuroticism group would experience greater cardiovascular and emotional reactivity and less cardiovascular recovery than the low-Neuroticism group in the hostile and neutral conditions only.

**METHOD**

**Participants**

Four hundred and thirty-five women were screened for Neuroticism level through the Washington State University Department of Psychology undergraduate subject pool. Individuals were selected for solicitation if they scored in the upper (high-N) or lower (low-N) tertiles of the Neuroticism distribution. An invitation email message was sent to the 290 prospective participants, with 49 positive responses returned from low-N individuals and 48 returned from high-N individuals. Forty-three low-N and 44 high-N individuals consented to participate. Data from one low-N participant were removed from the analyses following an equipment failure that occurred during the collection of cardiovascular data. This provided a total of 86 participants (42 in the low-N group and 44 in the high-N group). Two participants from the low-N group did not provide sufficient mood-related data to allow for calculation of anxiety and anger variables. The sample was predominantly Caucasian (96%), with the remainder identifying as Hispanic. The mean age of the participants was 19.3 years.

**Design and Procedure**

The six possible presentation orders for the three conditions (hostile, neutral, and friendly confederate behavior) were randomly ordered in blocks. Individuals from the two Neuroticism groups were assigned to the next available condition at the time they arrived to take part in the study. This produced a $2 \times 3$ between-participants design. Participants were directed to refrain from consuming caffeinated beverages or using tobacco products for at least one hour prior to participation. They were informed that the purpose of the study was to examine the effect of cooperation on cardiovascular functioning, and that they would be working on a drawing task with another female participant currently undergoing the same procedure in a separate laboratory room.
Two female confederates were utilized in the study. They were carefully trained to carry out the protocol for all three conditions in a standardized manner. The confederates were informed of the experimental condition in advance in order to allow for the presentation of appropriate affect and demeanor, but they were blind to participant Neuroticism grouping. Individual differences between confederates had no significant effects on any of the cardiovascular, affective, or interpersonal variables.

**Baseline Period**

Participants were taken to the experiment room to complete a 10-minute “vanilla” baseline task that involved the examination and rating of 10 pairs of pictures at 1-minute intervals (Jennings, Kamarck, Stewart, Eddy, & Johnson, 1992). Systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), and heart rate (HR) values were taken once per minute over the last 3 minutes of the period and averaged to give resting values against which change was later assessed. The use of aggregated scores has shown to increase reliability in reactivity research (Kamarck & Lovallo, 2003). At the end of the baseline period, participants completed a brief self-report mood measure that was used to calculate state anger and state anxiety baseline scores.

**Task Period**

The stressor task involved having participants give verbal instructions to the confederate on how to sequentially reproduce three separate two-dimensional figures. The task design was modified from a cooperative paradigm previously used to examine how affiliative quality affects cardiovascular response in interpersonal interactions (Smith & Ruiz, 2007). The confederate was brought to the room, seated across the table from the participant, and applied a blood pressure cuff. Next, a rigged draw was conducted that ensured assignment of the confederate to the “drawer” role and the participant to the “communicator” role. This placed the confederate in a position to provide verbal feedback to the participant.

Easels were given out to each individual and placed at an angle, ostensibly to obstruct visibility of the figures and drawing paper from across the table. The participant easel had three figures presented on separate pages; the confederate easel had three pages of drawing paper. Both easels had interspersed with these pages three copies of the state affect measure. Interpersonal quality of the interaction (the condition) was manipulated by way of scripted lines presented inconspicuously on the confederate’s...
drawing paper. Depending on condition, the lines were hostile/cold (e.g., “you’re making this too difficult”), neutral/ambiguous (e.g., “hmmm”), or friendly/warm (e.g., “you’re really good at this”). To make the confederate’s scripted lines appear credible, it was announced that she was permitted to provide verbal feedback as it related to her ability to follow the drawing instructions. To prevent participants from being diverted from the task by the confederate’s words, the participants were requested not to provide any verbal information other than drawing instructions.

The scripted lines were delivered at the 20-second and 80-second marks of each 2-minute trial, for a total of six comments across all three trials. The confederate was cued remotely at the appropriate time by the researcher via a concealed indicator light connected to a switch. The researcher started both the participant and confederate pressure monitors at the 30-second and 90-second marks of each trial, but only recorded cardiovascular values for the participant. When a trial was completed, the participant and confederate were cued to turn the page on the easel and complete the state affect measure.

Recovery Period

Immediately upon completion of the task period, the confederate was removed from the room, ostensibly to separate the two individuals for the recovery period. Cardiovascular measurements were then taken once every 2 minutes over a 10-minute period. The recovery period interval was set at this length because stressor tasks that induce emotion and feelings of anger tend to require more time for cardiovascular variables to return to baseline levels than do other types of laboratory stressors (Linden et al., 1997). To avoid any effect that mood reporting might have on cardiovascular response post-task, the state affect measure was not administered during the recovery period. At the end of the period, participant perception of the confederate was assessed as a manipulation check.¹

¹ Participants rated the confederate on a polar affiliation axis ranging from “warm and friendly” to “cold and hostile.” Two well-validated interpersonal measures (the Interpersonal Adjective Scales; Wiggins, Trapnell, & Phillips, 1988; and the Impact Message Inventory–Circumplex; Kiesler & Schmidt, 1993) were implemented for this purpose. Consistent with design intentions, confederates in the friendly condition were rated as being most warm, whereas those in the hostile condition were rated as being least warm. Scores for the neutral condition fell in the middle. Details on this analysis are available from the first author upon request.
Self-Report Measures

Individuals completed the 12-item Neuroticism scale of the NEO Five Factor Inventory (NEO-FFI; Costa & McCrae, 1992). The items require individuals to respond on a 5-point Likert scale (1 = strongly disagree, 5 = strongly agree). Scale scores on the NEO-FFI range from 12 to 60 and are interpreted on a continuum. The Neuroticism scale of the NEO-FFI has demonstrated good internal consistency and test-retest reliability. For the current sample, Cronbach’s $\alpha$ was .83. The bottom tertile of the distribution was bounded by a score of 28, whereas the upper tertile was bounded by a score of 34.

State anxiety and state anger were measured using a 12-item instrument derived primarily from the State-Trait Personality Inventory (STPI; Spielberger, 1979). Anxiety was assessed using six items from the State Anxiety scale of the STPI, and anger was assessed using four items from the State Anger scale of the STPI plus two additional previously validated items (Smith, Ruiz, & Uchino, 2004). Instructions ask respondents to indicate “at this moment I feel (e.g., calm, annoyed, tense)” on a 4-point Likert scale (1 = not at all, 4 = extremely). Both scales range from 6 to 24. Cronbach’s $\alpha$ scores for the State Anxiety scale were adequate, ranging from .76 (baseline) to .88 (task). Cronbach’s $\alpha$ scores for the State Anger scale were also adequate, ranging from .59 (baseline) to .93 (task).

Cardiovascular Assessment

The Dinamap Model Pro 100 monitor (General Electric, Miami, Florida) was used to measure blood pressure and heart rate. It utilizes the oscillometric method to estimate blood pressure. Measurements were obtained via a properly sized occluding cuff positioned on the nondominant upper arm, according to the manufacturer’s specifications.

RESULTS

Baseline Equivalence of Conditions

One-way ANOVA was used to examine baseline equivalence of variables. There were no significant differences between conditions at baseline for any of the four physiological indices (SBP, DBP, MAP, HR), all $F$s(2, 83) ≤ 2.29, $\eta^2$ ≤ .052, $ps$ ≥ .11, or for either of the two affective indices, all $F$s(2, 81) ≤ .37, $\eta^2$ ≤ .009, $ps$ ≥ .69. The high-N group had significantly greater baseline HR, $F(1, 84) = 4.21$, $\eta^2 = .050$, $p = .04$, and state anxiety, $F(1, 82) = 8.74$, $\eta^2 = .10$, $p = .004$, than the low-N group.
Effectiveness of Task

The overall effectiveness of the task in producing cardiovascular and emotional response was assessed using repeated-measures ANOVA. For SBP, DBP, MAP, and HR, there were significant differences in mean baseline, task, and recovery period levels, all $F$s(2, 170) ≥ 114.02, $\eta^2$ ≥ .63, $p$s < .001 (Table 1). Paired-sample $t$ tests showed that for all cardiovascular indices there were significant increases from baseline to task, all $t$s(85) ≥ 8.8, $p$s < .001, and significant decreases from task to recovery, all $t$s(85) ≥ 11.32, $p$s < .001. There were also significant increases in both emotion variables from baseline to task, all $F$s(2, 83) ≥ 7.91, $\eta^2$ ≥ .31, $p$s < .001 (Table 1).

Effects of Condition and Neuroticism

Two-way, repeated-measures ANCOVA was conducted for each cardiovascular and affective dependent measure. ANCOVA has a robust history of use in research investigating the relationship between personality and physiological reactivity. Consistent with prior recommendations (Llabre, Spitzer, Saab, Ironson, & Schneiderman, 1991), task change scores ([mean task value] – [mean baseline value]) were calculated for the cardiovascular and affective variables.

Table 1
Mean Scores for All Cardiovascular and Affective Indices During Baseline, Task, and Recovery Periods

<table>
<thead>
<tr>
<th>Index</th>
<th>Baseline</th>
<th>Task</th>
<th>Recovery</th>
<th>$F$(2, 170)</th>
<th>$\eta^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP (mmHg)</td>
<td>103.8 (0.89)</td>
<td>122.3 (1.17)</td>
<td>109.2 (1.03)</td>
<td>277.47</td>
<td>.77</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>65.8 (0.59)</td>
<td>77.2 (0.72)</td>
<td>68.8 (0.62)</td>
<td>259.48</td>
<td>.75</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>81.7 (0.55)</td>
<td>93.1 (0.70)</td>
<td>84.6 (0.55)</td>
<td>286.61</td>
<td>.77</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>72.8 (1.27)</td>
<td>83.3 (1.65)</td>
<td>71.8 (1.28)</td>
<td>91.23</td>
<td>.63</td>
</tr>
<tr>
<td>State anxiety</td>
<td>10.7 (2.79)</td>
<td>14.5 (0.41)</td>
<td>—</td>
<td>117.07</td>
<td>.59</td>
</tr>
<tr>
<td>State anger</td>
<td>10.0 (0.19)</td>
<td>12.7 (0.45)</td>
<td>—</td>
<td>37.91</td>
<td>.31</td>
</tr>
</tbody>
</table>

Note. Based on repeated-measures analyses of variance. Within rows, all means differ significantly at $p$ < .001. Standard errors are in parentheses. SBP = systolic blood pressure; DBP = diastolic blood pressure; MAP = mean arterial pressure; HR = heart rate; bpm = beats per minute.
measured in each of the three trials. Recovery change scores ([mean
recovery value] – [mean baseline value]) were calculated for the car-
diovascular variables. This approach of using change scores for as-
sessing response has been widely implemented in cardiovascular
research and allows for meaningful comparisons between individuals
to be made (Linden et al., 1997). The baseline value of a dependent
cardiovascular or affective index was included as a covariate to pro-
vide residualized change scores (Benjamin, 1967; Linden et al., 1997).
Although the above strategies have been previously established
within the field, other approaches are also implemented (cf. Chris-
tenfeld, Glynn, & Gerin, 2000; Cohen, Cohen, West, & Aiken, 2003),
and no consensus has yet been reached in the area of cardiovascular
response assessment.

In terms of CVR, there was a significant main effect of condition
on SBP, DBP, MAP, and HR change from baseline to task (Table 2).
Tukey-Kramer multiple pairwise comparisons revealed that for SBP,
MAP, and HR, the hostile condition produced greater cardiovascu-
lar reactivity than the friendly condition, all ps ≤ .02, and neutral
condition, all ps ≤ .002. The hostile condition also produced greater
DBP change than the neutral condition, p = .005. In terms of recov-
ery, there was a significant main effect of condition on all four car-
diovascular indices (Table 2). Pairwise comparisons showed that for
SBP, DBP, MAP, and HR, the hostile condition produced greater
elevation from baseline to recovery than the friendly condition, all
ps ≤ .02, and neutral condition, all ps ≤ .005. The overall trend sug-
gested that the hostile condition produced significantly greater car-
diovascular response than both the friendly and neutral conditions.

There was a significant effect of condition on anger and anxiety
reactivity (Table 2). The effect size of condition was significantly
greater for anger than for anxiety. The hostile condition produced
greater anger change from baseline to task than the friendly and
neutral conditions, all ps < .001. The hostile condition also created
greater anxiety change than the friendly condition, p = .004.

Despite the effectiveness of the manipulation, no significant main
effects of Neuroticism were found for cardiovascular reactivity, all
Fs(1, 79) ≤ 0.33, η² ≤ .004, ps ≥ .57, or cardiovascular recovery, all
Fs(1, 79) ≤ 0.14, η² ≤ .002, ps ≥ .71. However, Neuroticism had ex-
pected main effects on change in anger, F(1, 77) = 6.71, η² = .08,
p = .01, and change in anxiety, F(1, 77) = 6.95, η² = .08, p = .01,
from baseline to task.
Neuroticism and Cardiovascular Response

Contrary to expectations, there were no significant interactions between Neuroticism and condition on anger or anxiety response, all $F$s(2, 77) ≤ 2.32, $\eta^2$ ≤ .07, $p$s ≥ .11 (Figure 1), which indicated that the effects of Neuroticism on emotional reactivity did not vary across conditions. Also running counter to predictions, there were no significant interactions between Neuroticism and condition on CVR, all $F$s(2, 79) ≤ .80, $\eta^2$ ≤ .02, $p$s ≥ .45.

Neuroticism interacted with condition to affect cardiovascular recovery in a manner that was partially consistent with predictions.

### Table 2

Effect of Condition on Change in Cardiovascular and Affective Indices From Baseline to Task and Recovery Periods

<table>
<thead>
<tr>
<th>Index</th>
<th>Hostile</th>
<th>Neutral</th>
<th>Friendly</th>
<th>$F$</th>
<th>$\eta^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\Delta$SBP(mmHg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Task</td>
<td>23.0 (1.65)</td>
<td>15.6 (1.68)</td>
<td>16.8 (1.64)</td>
<td>5.79**</td>
<td>.13</td>
</tr>
<tr>
<td>Recovery</td>
<td>9.6 (1.05)</td>
<td>2.9 (1.07)</td>
<td>3.6 (1.04)</td>
<td>12.16***</td>
<td>.24</td>
</tr>
<tr>
<td>$\Delta$DBP(mmHg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Task</td>
<td>13.4 (1.00)</td>
<td>9.2 (1.03)</td>
<td>11.2 (0.97)</td>
<td>4.21*</td>
<td>.10</td>
</tr>
<tr>
<td>Recovery</td>
<td>4.9 (0.65)</td>
<td>2.2 (0.67)</td>
<td>2.2 (0.64)</td>
<td>5.67**</td>
<td>.13</td>
</tr>
<tr>
<td>$\Delta$MAP(mmHg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Task</td>
<td>14.3 (0.99)</td>
<td>9.4 (1.05)</td>
<td>10.8 (0.98)</td>
<td>6.25**</td>
<td>.14</td>
</tr>
<tr>
<td>Recovery</td>
<td>5.2 (0.55)</td>
<td>1.7 (0.56)</td>
<td>2.3 (0.54)</td>
<td>11.69***</td>
<td>.23</td>
</tr>
<tr>
<td>$\Delta$HR(bpm)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Task</td>
<td>17.7 (1.90)</td>
<td>5.2 (1.95)</td>
<td>8.8 (1.88)</td>
<td>11.26***</td>
<td>.23</td>
</tr>
<tr>
<td>Recovery</td>
<td>1.1 (0.77)</td>
<td>-2.3 (0.81)</td>
<td>-1.6 (0.78)</td>
<td>5.31**</td>
<td>.12</td>
</tr>
</tbody>
</table>

$F$(2, 79)

$\Delta$State anxiety
task 5.4 (0.55) 3.1 (0.59) 2.5 (0.56) 7.17*** .16

$\Delta$State anger
task 6.3 (0.56) 1.5 (0.60) 0.3 (0.56) 31.67*** .45

*Note. Based on two-way repeated-measures analyses of covariance. Change values have been adjusted to control for covariate. Standard errors are in parentheses. SBP = systolic blood pressure; DBP = diastolic blood pressure; MAP = mean arterial pressure; HR = heart rate; bpm = beats per minute.

$p$<.05. **$p$<.01. ***$p$<.001.
Significant interactions between Neuroticism and condition were identified for SBP recovery, $F(2, 79) = 3.77$, $\eta^2 = .09$, $p = .03$, and MAP recovery, $F(2, 79) = 3.18$, $\eta^2 = .08$, $p = .05$ (Figure 2). These

Figure 1
Effects of Neuroticism on mean change in state affect from baseline period to task period across interpersonal conditions. Panel A shows state anxiety change; panel B shows state anger change.
Figure 2
Effects of Neuroticism on mean change in blood pressure from baseline period to recovery period across interpersonal conditions. Panel A shows systolic blood pressure (SBP) change; panel B shows mean arterial pressure (MAP) change.
interactions were probed further by running the ANCOVA with only two of the three conditions selected to allow for pairwise comparisons. In the case of SBP recovery, there was a significant interaction when the hostile and friendly conditions were selected, $F(1, 52) = 6.65, \eta^2 = .11, p = .01$, but not when the hostile and neutral conditions or friendly and neutral conditions were selected, all $Fs(1, 52) \leq 1.36, \eta^2 \leq .03, ps \geq .12$. For MAP recovery the pattern was the same, revealing a significant interaction when the hostile and friendly conditions were selected, $F(1, 52) = 4.91, \eta^2 = .09, p = .03$, but not when the hostile and neutral conditions or friendly and neutral conditions were selected, all $Fs(1, 52) \leq 2.83, \eta^2 \leq .05, ps \geq .10$. These results showed that high Neuroticism produced less SBP and MAP recovery in the hostile condition relative to the friendly condition, and greater SBP and MAP recovery in the friendly condition relative to the hostile condition.

**DISCUSSION**

The current investigation examined the effects of Neuroticism on cardiovascular and affective response to a stressor with three interpersonal conditions. The stressor task proved to be effective in arousing cardiovascular and emotional response, and the experimental manipulation had the intended effects on participant perception of the confederate.

The primary aim of this study was to determine whether Neuroticism had effects on cardiovascular and emotional response to the interpersonal stressor. In terms of emotion, Neuroticism had significant main effects on change in state anger and state anxiety—findings that were consistent with predictions and past research. Neuroticism is characterized as a tendency to experience negative mood states, and thus it was not surprising to find that the high-N group experienced greater affective reactivity to the stressor task. Contrary to predictions, there were no significant main effects of Neuroticism on cardiovascular reactivity or recovery. These results are consistent with the findings of some studies, but inconsistent with others. The absence of an effect of Neuroticism on CVR may have been due to a response “ceiling effect” that overshadowed any subtle influences of personality on blood pressure and heart rate. The interpersonal interactions that occur in daily life are generally less arousing than this laboratory stressor, and using a different stressor
task more comparable to a typical daily hassle might permit identification of a link between Neuroticism and CVR.

The second aim of this study was to assess whether the interpersonal quality of the interaction would moderate the effects of Neuroticism on cardiovascular and emotional reactivity and cardiovascular recovery. Contrary to predictions, there were no significant interaction effects for Neuroticism and condition on reactivity. Neuroticism had no effect on CVR and fairly uniform main effects on anxiety and anger change across conditions. However, there was a significant interaction between Neuroticism and condition such that the high-N group showed less SBP and MAP recovery in the hostile condition and greater SBP and MAP recovery in the friendly condition. This was an important finding, and one that was partially consistent with predictions. The effects within the friendly condition were not expected, and there were not the anticipated differences between Neuroticism groups in the neutral condition. It appears that, despite the negative bias that is associated with Neuroticism, ambiguity during the interpersonal stressor task was not relevant to Neuroticism in terms of cardiovascular or emotional response.

From a health perspective, perhaps most important was the finding that blood pressure recovery was poorer for the high-N group in the hostile condition. This suggested that the combination of high Neuroticism and frequent hostile interactions might over time contribute to the development of hypertension and cardiovascular disease. Overall, the evidence of an interaction between Neuroticism and condition on recovery lends support to the premise that there is a stress moderation component to Neuroticism’s effect on physical health and mortality. Although the observed interaction effect sizes were relatively small in this sample of university students, they might be significantly greater in populations of older adults or the medically ill. Some of the key investigations linking Neuroticism to mortality have used samples of older adults and disease patients, and the effects of Neuroticism on cardiovascular recovery might be greater in these populations that tend to be less resilient in the face of stress.

This study was not designed to identify the pathways by which Neuroticism affects recovery following hostile and friendly interpersonal interactions, but based on past research we may speculate that cognitive factors are involved. Neuroticism is associated with negative rumination and worry (Muris, Roelofs, Rassin, Franken, & Maver, 2005; Roberts, Gilboa, & Gotlib, 1998), and a study by Key,
Campbell, Bacon, and Gerin (2008) found that state rumination predicted poorer blood pressure recovery following an emotional recall stressor task. If highly neurotic individuals tend to focus their thoughts on the negative aspects of hostile interactions, they may experience an extended stress response and slowed return of blood pressure to baseline. By the same token, neurotic individuals may discount, underestimate, or be less inclined to orient toward stimuli indicative of reward, in this case behavioral indicators of positive evaluation and interpersonal warmth. Such a negative bias could reduce engagement in friendly interpersonal interactions, shorten activation time, and hasten return of blood pressure to baseline. It is even possible that high Neuroticism levels are protective in their cardiovascular effects as they relate to friendly interactions.

It is worth noting that the opposing effects of Neuroticism on cardiovascular recovery in the hostile and friendly conditions essentially “cancelled each other out,” resulting in no net effect. This observation of mirrored effects of Neuroticism is useful, as it will help guide future research in the area of personality and cardiovascular health. However, separate use of hostile or friendly interpersonal stressors might allow for the identification of main effects of Neuroticism on recovery. Neutral and ambiguous interactions do not appear to be particularly relevant to Neuroticism, and as such should be disregarded in future research in this area.

Thus far, investigations into the relationships between psychosocial factors and cardiovascular response have tended to focus more on reactivity than on recovery. One important implication of the current study is that it supports a turn of attention away from reactivity and toward recovery in the examination of connections between personality and cardiovascular health. In terms of clinical implications, the findings outlined here suggest that highly neurotic individuals and patients at risk for developing cardiovascular disease should be taught skills to circumvent hostile conflict, mitigate appraisals of hostility in others, and cope more adaptively with the negative cognitions and emotions that can arise following hostile interpersonal interactions.

This study has some limitations that relate primarily to the characteristics and size of the sample. The exclusion of men from participation leaves questions unanswered about how gender may moderate relationships between Neuroticism and cardiovascular recovery. It has been widely demonstrated that women tend to score
higher than men on measures of Neuroticism (Costa, Terracciano, & McCrae, 2001; Lynn & Martin, 1997), and lower demonstrated effect sizes for the Neuroticism × condition interactions might be observed if men were examined separately using the same experimental procedure. It is also possible that there are other gender-related variables (social or biological) that mitigate or exacerbate the influence of personality on cardiovascular response. Holt-Lunstad, Clayton, & Uchino (2001) found that men and women who completed a paired competition task displayed differing patterns of blood pressure reactivity depending on the sex of their partner—a result indicating that gender can play a role in cardiovascular response to interpersonal stressors. Another limitation of note about the sample is that exclusion of the middle third of the Neuroticism distribution precluded application of the findings to those who score close to the mean on this trait. A larger sample size would allow for utilization of continuous Neuroticism scores from the entire distribution, thus providing more confidence for making inferences from the data.

Future investigations in this area should draw participants from middle-aged, geriatric, and clinical populations in order to determine whether the effects of Neuroticism differ with age and health status. Also, the mechanisms by which Neuroticism interacts with interpersonal factors to affect blood pressure recovery must be probed further. In particular, cognitive and emotional events associated with Neuroticism (e.g., negative rumination, threat appraisal) should be measured along with anger to assess for links to cardiovascular recovery. Path modeling could be used to answer complex questions about relationships between relevant variables. Do neurotic individuals appraise more threat and experience more anger from hostile interpersonal interactions, leading to rumination and slowed recovery? Do they appraise less potential reward from friendly interactions, attenuating positive mood and thereby hastening recovery? Gaining answers to questions such as these will help us understand further how Neuroticism exerts its effects on physical health.

REFERENCES


