



Cardiovascular activity during laboratory tasks in women with high and low worry[☆]

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ABSTRACT

Worry has been related to delayed stress recovery and cardiovascular disease risk. Cardiovascular responses to a range of laboratory tasks were examined in this study of high and low worriers. Undergraduate women were recruited with the Penn State Worry Questionnaire to form low ($n = 19$) and high ($n = 22$) worry groups. These individuals engaged in six laboratory tasks (orthostatic stress, supine rest, hand cold pressor, mental arithmetic, and worry and relaxation imagery) while heart rate (HR), HR spectral analysis, impedance cardiography, and blood pressure were acquired. The only significant group difference found was a consistently greater HR across tasks in high worriers ($p < .05$). No group by condition interactions emerged. High trait worry in healthy young women appears to be marked by elevated HR in the absence of autonomic abnormalities. These findings are discussed relative to the literature on worry, with particular reference to its health implications.

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Research on the relationship between personality and cardiovascular disease (CVD) has been historically influenced by the Type A construct (see Friedman and Rosenman, 1971). Accordingly, the literature in this area is dominated by a focus on the Type A-derived traits of anger and hostility (see Everson-Rose and Lewis, 2005, for review). Over the last several decades, anxiety has emerged as another trait that may confer increased CVD risk. A number of earlier epidemiologic studies found anxiety to be an independent risk factor for CVD (Haines et al., 1987; Kawachi et al., 1994a,b). This association has been confirmed in more recent research (Barger and Sydeman, 2005; Shen et al., 2008). In a review of the literature, Kubzansky and Kawachi (2000) concluded that among various manifestations of negative affect, anxiety was a stronger predictor of coronary heart disease than either anger or depression.

Anxiety, as a derivative of the basic emotion fear, is a multifaceted construct with cognitive, behavioral, and physiological components (Plutchik, 1990). From this perspective, fear and anxiety may be viewed on a continuum from state to trait anxiety to anxiety disorders. Indeed, studies of the relationship of anxiety

to CVD have used both trait measures and clinical diagnosis as inclusion criteria. Additionally, the association between anxiety and CVD appears to be less consistent in women than in men (Eaker et al., 1992; Matthews et al., 1998). All of these factors cloud the study of anxiety in relation to CVD, particularly in regard to identification of mechanisms that might account for the relationship between the two.

Systematic laboratory work is needed in conjunction with epidemiologic evidence to uncover the processes that may link anxiety with CVD. This is the process that led from initial development of the Type A construct to testing of the reactivity hypothesis of hostility and CVD (Harbin, 1989; Manuck, 1994). The reactivity hypothesis posits that chronic large increases in CV variables in response to stressors are a risk factor for CVD. Although the literature is not entirely consistent, numerous prospective studies have linked high CV reactivity to stress with the development of CVD (Rozanski et al., 1999; Treiber et al., 2003). Cross-sectional studies have been somewhat more contradictory in demonstrating that trait hostility is associated with high CV reactivity (Suls and Wan, 1993).

Relatively few studies have investigated anxiety within the framework of the CV reactivity model. However, a growing body of literature indicates that anxiety in its clinical, trait, and state forms is associated with low cardiac vagal control, as indexed through the analysis of heart rate variability (HRV) (see Friedman, 2007, for review). Poor parasympathetic “braking” of HR might possibly be a factor in high CV reactivity. Alternatively, low cardiac vagal control might also lead to sustained CV activation and poor stress recovery

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(Mezzacappa et al., 2001). Recovery reflects the magnitude, duration, and frequency of CV responding, and as such may be a more informative measure than CV reactivity, which only yields information on the magnitude of responses (Schwartz et al., 2003).

Delayed CV recovery to stress may be a common factor that links both hostility and anxiety to disease risk. CV recovery has been found to longitudinally predict resting blood pressure (BP) (Stewart and France, 2001), and slow CV recovery has been linked with hypertension and CVD risk factors such as lack of fitness and low socioeconomic status (Schuler and O'Brian, 1997; Steptoe et al., 2002). Moreover, state anxiety has been associated with delayed BP recovery to stress (Vitaliano et al., 1995).

According to the *perseverative cognition hypothesis*, hostility coupled with the tendency to suppress anger may delay CV recovery through rumination (Brosschot and Thayer, 1998). In this hypothesis, some hostile individuals may perseverate on their anger, thereby sustaining CV activation long after a provocative incident has concluded. This model has been extended to include persistent worry, which shares with chronic anger suppression the maintenance of negative affect in consciousness—i.e., rumination (Brosschot et al., 2006). Worry is often viewed as an aspect of anxiety that involves a predominance of verbal thought, the function of which is the cognitive avoidance of threat (Borkovec et al., 1998).

In fact, longitudinal data indicate that men reporting high levels of trait social worry are at increased risk for nonfatal and fatal CVD (Kubzansky et al., 1997). Laboratory manipulated rumination (state condition) has been shown to delay BP recovery to emotional stressors (Gerin et al., 2006; Glynn et al., 2002). Trait worry has also been associated with elevated HR and reduced HRV and cardiac vagal tone during “real-life” ambulatory recording of waking and sleep periods (Brosschot et al., 2007; Pieper et al., 2007) and during laboratory-induced worry (Hofmann et al., 2005). Persistent worry is the defining feature of generalized anxiety disorder (GAD; American Psychiatric Press, 1994), which has been linked with phasic and tonic HR elevations, low HRV, and poor cardiac vagal control (Thayer et al., 1996; Thayer et al., 2000). In contrast, a negative finding was reported in a study that in general revealed no HR or HRV differences between high and low trait worriers in response to various laboratory stressors (Davis et al., 2002).

The aim of the present study was to compare high and low worriers on CV reactivity and recovery to a series of laboratory manipulations. These conditions were selected to represent three task types that have been used widely in stress reactivity and anxiety research. The first task type consisted of body position adjustments that evoke mechanical CV responses. Such tasks are primarily physical in nature and vary little on psychological features. Two tasks were chosen in this category: (1) Orthostatic stress, which involves standing upright from a sitting position, generally eliciting sympathetic CV activation and vagal withdrawal (Friedman and Santucci, 2003; Pagani et al., 1995); (2) Supine rest, which entails lying down in fully reclined position, evoking vagal increases and sympathetic CV withdrawal (Friedman and Santucci, 2003).

The second task pair consisted of hand cold pressor and mental arithmetic, two classic exemplars of the “active” (mental arithmetic) vs. “passive” (cold pressor) distinction between CV stressors (Obrist, 1981; Vella and Friedman, 2007). Hand cold pressor is marked by sympathetic alpha-adrenergic activation (Saab et al., 1993). Reactivity to this task has yielded both positive (e.g., Kasagi et al., 1995) and negative (e.g., Carroll et al., 1996) findings in predicting hypertension. Mental arithmetic, characterized by cardiac sympathetic beta-adrenergic activation and vagal withdrawal, is considered to be a prototypical mental stressor due to its potency and minimal motor demands (Lavallo, 2005).

The final task set was directed at simulating anxious rumination. A worry imagery task was used to elicit focused anxiety about

a subject of great individual concern. This manipulation is marked by vagal withdrawal (Thayer et al., 1996). To contrast this condition, a relaxation imagery task was used to evoke mental calm and vagal activation (Hofmann et al., 2005).

The low and high in worry samples in this study were selected on the basis of the Penn State Worry Questionnaire (PSWQ), an instrument developed and validated for this purpose (Meyer et al., 1990). Worry is the primary feature of GAD, which is about twice as prevalent in women as in men (American Psychiatric Press, 1994). Also, gender differences have been found in previous research on autonomic modulation of cardiovascular activity (Ramaekers et al., 1998). Therefore, for ease of recruitment as well as control of the factor of gender, only women were included in this study.

We hypothesized that women scoring high on worry would generally show increased CV reactivity, delayed CV recovery, and low vagal control of HR in response to these tasks. Additionally, an interaction was predicted in that these effects would be magnified during the worry imagery task. The prediction is based on an extension of the ‘matching hypothesis’ which generally holds that the best fit of person and experimental conditions is likely to reveal individual differences in CV reactivity and recovery (Engelbreton et al., 1989).

Individuals engaged in three sets of task pairs, six conditions total: supine rest and orthostatic stress, mental arithmetic and hand cold pressor, and relaxation and worry imagery. Three dimensions of CV responding were assessed: anticipation (prior to the tasks), reactivity (during the tasks), and recovery (after the task). A montage of measures were used to capture multiple aspects of CV function, including cardiac chronotropic indices obtained from the electrocardiogram (ECG), contractility measures derived from the impedance cardiogram (ICG), and vascular activity as expressed in BP.

1. Method

1.1. Subjects

Forty-one nonsmoking female college students (mean age = 19.7 years) were recruited for the laboratory study through the Virginia Tech Psychology Department's online subject recruitment system. Initially, 472 women completed the online version of the PSWQ. During this selection process, students were screened via a physical and mental health background form included with the PSWQ. One question included in this health form asked for the individual's greatest fear or cause of worry. Information was also collected on medications, prior negative physical and mental health life events, and height and weight to calculate body mass index (BMI). Exclusionary criteria included cardiovascular, pulmonary, or other medical conditions such as hypertension, back pain, diabetes, or neurological disorders, and tobacco use. Those taking anti-depressive or anxiolytic medication were also excluded. Three hundred thirty of the 472 women completing the online screening were deemed eligible for the laboratory part of the study. Extra credit toward psychology courses was earned by participation in both the online and laboratory portions. All portions of the study were approved by the Virginia Tech Institutional Review Board.

Subjects were grouped by worry level after initial screening. Those scoring in the bottom third of 330 eligible individuals on the PSWQ were classified as low worry, and those scoring in the top third were classified as high worry. A total of 180 eligible subjects were invited to participate in the laboratory portion of the study. From these invited individuals, 22 women from the high worry group (M PSWQ = 67.3, $S.D.$ = 5.3) and 19 women from the low worry group (M PSWQ = 37.7, $S.D.$ = 6.5) chose to participate. The Godin Leisure-Time Exercise Questionnaire (Godin and Shephard, 1997) was used to assess physical activity level in the laboratory sample; the worry groups did not differ on this measure. Subjects were asked to abstain from alcohol for 24 h and caffeine for 12 h prior to the laboratory session. The study was double-blind in that neither the experimenter nor the subject knew which worry group the individual belonged to.

1.2. Apparatus and materials

1.2.1. Penn State Worry Questionnaire

The PSWQ is a 16-item instrument that displays good psychometric properties such as high internal consistency, convergent and discriminative validity, and test-retest reliability (Brown et al., 1992; Meyer et al., 1990). Examples of items which

are scored on a 5-point Likert-type scale include “My worries overwhelm me”, and “Many situations make me worry.” The PSWQ has shown discriminant validity in distinguishing individuals meeting DSM criteria for GAD from those with other anxiety disorders or depression (Brown et al., 1992; Fresco et al., 2003; Meyer et al., 1990; Roemer et al., 1995). Use of the PSWQ is supported in application to (a) diagnostic screening and treatment of GAD, (b) the nature of normal and pathological worry, and (c) in identifying analogue GAD subjects for preliminary research on the processes associated with chronic worry and GAD (Brown et al., 1992; Fresco et al., 2003; Roemer et al., 1995).

1.2.2. Recording equipment

The ECG and ICG were recorded with the Ambulatory Monitoring System v4.4 (AMS; Vrije Universiteit, the Netherlands), the validity and reliability of which has been established (Willemsen et al., 1996). Ag–AgCl electrodes were attached to the torso region in accordance with user manual guidelines (Groot et al., 1998). BP was monitored using the IBS SD-700A automated monitor (Industrial & Biomedical Sensors Corp., Waltham, MA).

1.3. Dependent physiological measures

HR (in beats per minute; b.p.m.) and HRV indices were derived from the ECG, which was analog filtered (high pass 17 Hz) at acquisition and subjected to online auto trigger level R-wave detection resulting in a resolution of 1 ms. HRV analysis was performed using HRV Analysis Software v1.1 (The Biomedical Signal Analysis Group, University of Kuopio, Finland). Among other HRV measures, this program yields estimates of the ECG power spectrum in the low (LF; 0.04–0.15 Hz) and high (HF; 0.15–0.40 Hz) bandwidths using a Fast Fourier Transform. In the present study, HF power served an index of vagally mediated respiratory influences on HR (Allen et al., 2007; Friedman et al., 2002). LF power served as a measure of slower, baroreceptor mediated influences on HR, for which there is evidence of both vagal and sympathetic β -adrenergic underpinnings (e.g., Friedman et al., 1996; Malliani et al., 1998; cf. Eckberg, 1997; Porges, 2007). LF and HF power were expressed in normalized power spectral density units. The ICG was used to calculate pre-ejection period (PEP in ms), a putative measure of cardiac sympathetic β -adrenergic activity (Sherwood et al., 1990). IBI sequences were examined for validation. IBIs over two standard deviations outside the normal range were removed as were IBIs that differed more than 33% from the previous IBI. Finally, systolic (SBP) and diastolic (DBP) blood pressures (in mmHg) were obtained with the IBS monitor. ECG and ICG were recorded continuously during baseline, task, and recovery periods; SBP and DBP were taken during the first 45 s of these periods.

1.4. Procedure

Subjects were greeted by the experimenter upon arrival at the lab, seated in a comfortable lounge chair, read an explanation of the study, and gave informed consent. Recording equipment was then applied, and individuals were asked to sit quietly for three minutes with eyes closed so that physiological equipment could be checked for accuracy. This initial recording period was used subsequently as an ‘anticipatory baseline’ that reflects state prior to the onset of the experimental procedure. After this period, the experimenter administered a short health questionnaire and gave a verbal description of the experimental protocols. Following this explanation, the task portion of the study began.

1.4.1. Task descriptions

The task portion consisted of six stressors lasting three minutes each. All tasks were performed with eyes closed. Each task was preceded by a 3-min baseline during which a segment of a non-stimulating video portraying daily life scenes and containing no words was shown (see Vella and Friedman, 2007, for details). A three-minute recovery period of quiet sitting with eyes closed followed each task. ECG and ICG were continuously recorded during each epoch. BP recording began at the start of each epoch, with one reading per epoch.

The first task pair involved two body position adjustments. Orthostatic stress involved arising from a lounge chair and standing for 3 min. The instructions were to remain as still as possible while standing without moving, slouching, or opening the eyes. The recovery period began at the end of three minutes upon return to the seated position. In supine rest, the lounge chair was moved from an upright to a fully reclined position, and the subject was instructed to lie quietly still and not sit up for the 3-min period. If any movement was seen, the experimenter asked the subject stay motionless. At the end of the task, the chair was set back to the upright position for the recovery period.

The second task set paired two standard CV stressors. In hand cold pressor, the seated upright individual placed the left hand up to the wrist in a tub of iced water kept at 3–6 °C. A small filter in the tub prevented directed contact between the hand and ice. The instructions were to try to keep the hand in the water for the full three minutes if possible, but the hand could be removed from the water if excessive discomfort was experienced. At the end of the task, the hand was dried off with a towel and the recovery period began. The mental arithmetic task involved serial subtraction beginning at 3000 and decreasing toward zero by

intervals of seven. Instructions were to try to reach the lowest number possible with the fewest errors.

The final set consisted of imagery tasks patterned after those used in Thayer et al. (1996). In one task, the individual was asked to worry about a topic that had been identified in the online questionnaire as their greatest fear, worry or point of concern. The instructions were to sit quietly and focus on the stated concern by picturing the worry and its consequences, concentrating on the details of the worry such as what the worry embodied and its personal effects. Instructions were to clear one’s mind of all thoughts when the recovery period began. The other task in this pair involved relaxation imagery, which was elicited by means of a guided relaxation audiotape that employs soothing, pulsing tones. Instructions were to remain still and relax as much as possible while focusing on the tones and the voice providing the guided relaxation.

After the six tasks were completed, the PSWQ was administered again to verify that subjects remained in their initial worry category. The goals of the study were then disclosed, and the option of seeing one’s CV measures and learning one’s worry group was given. Any remaining questions were addressed, extra credit was assigned, electrodes were removed, and individuals were thanked for participation.

1.4.2. Design and statistical analyses

Tasks were completed in randomized order pairings across subjects that were counterbalanced within but not between task pairings. The body position set always came first, followed by standard and then imagery tasks. The rationale for this strategy was two-fold: (a) full counterbalancing was not feasible within the sample size of the study, and (b) the worry imagery task was placed near the end of the study to avoid contamination of the other tasks by continued rumination among the high worry group. Within each task set, orthostatic stress and supine rest, hand cold pressor and mental arithmetic, and relaxation and worry imagery were counterbalanced across subjects.

The primary hypotheses were analyzed by 2 (group: low vs. high worry) \times 18 (condition: baseline, task, and recovery) repeated measures ANOVAs on the dependent variables of HR, LF and HF power, PEP, SBP and DBP. Within-subjects differences across tasks were examined with a Huynh–Feldt correction for degrees of freedom. Paired *t*-tests were used to compare within-subjects effects between task epochs and baseline/recovery epochs. Covariates used in this study were BMI category, menstrual cycle phase, and time of participation. Subjects with incomplete worry questionnaires in the initial screening were excluded from participation. CV data were missing from one subject in the anticipatory epoch, and in two more during all other conditions due to equipment malfunction. Data from these three individuals were excluded from analysis.

2. Results

2.1. Primary analyses

No significant group differences were initially found for any dependent variable. On further examination, it was clear that the group data were skewed on BMI, with more obese/overweight subjects in the low worry group and more anorexic/underweight subjects in the high worry group. Accordingly, this and all subsequent analyses were run with BMI group as a covariate.¹ There were four BMI groups: 17.0–18.5 (underweight); 18.6–25.0 (normal); 25.1–30.0 (overweight); and >30.0 (obese). The overall group difference in HR was now significant ($F(1, 35) = 4.84, p < .05$). It was also necessary to adjust for two other factors: hormonal fluctuations of the menstrual cycle (Princi et al., 2005; Vallejo et al., 2005) and circadian rhythms (Conrad et al., 2008), both of which can influence HRV. Menstrual phase and time of testing were thus entered as covariates. When all three factors (BMI group, menstrual phase, and time of testing) were controlled, the HR results remained significant ($F(1, 33) = 5.01, p < .05$). As shown in Fig. 1, this difference was robust; the high worry group showed higher HR across all conditions. These differences were significant at $p = .05$ or less for the following conditions: baselines preceding orthostatic stress, supine rest, cold pressor, and worry;

¹ BMI was treated as a categorical rather than as a continuous variable due to a skewed distribution of BMI relative to worry group. Specifically, there were more obese/overweight individuals in the low worry group and more underweight subjects in the high worry group. There was a marginal ($r = -.29, p < .07$) negative association between BMI and PSWQ score (higher BMI group correlated with a lower worry score).

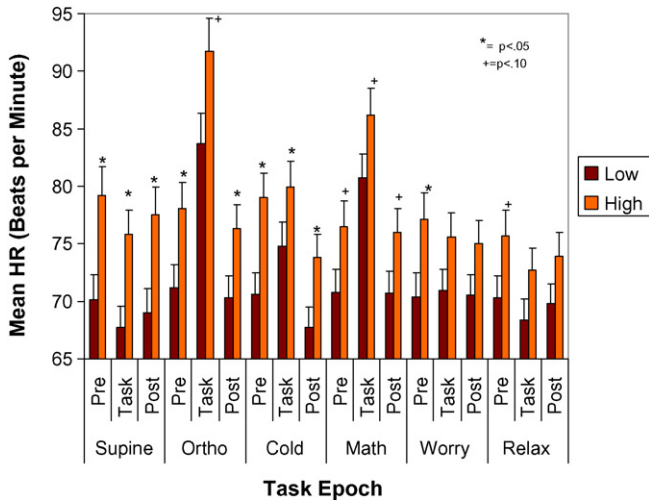


Fig. 1. Comparison of HR between high and low worriers across epochs.

during supine rest; and in recovery following orthostatic stress, supine rest, and cold pressor. The differences were marginally significant ($p < .06 - .1$) for the baseline preceding relaxation and mental arithmetic, during orthostatic stress and mental arithmetic, and in the recovery period after mental arithmetic. There were no other significant main effects for group on any CV variable other than HR.

Significant main condition effects were found for two CV variables on within-subjects differences: HR ($F(8, 275) = 3.26, p < .005$) and HF power ($F(15, 518) = 1.74, p < .05$; see Fig. 2). Each task was then compared to its corresponding baseline and recovery periods. HR was significantly higher during orthostatic stress and mental arithmetic than in their respective baseline (orthostatic stress: $t(37) = 11.9, p < .001$; mental arithmetic: $t(37) = 7.5, p < .001$) and recovery epochs (orthostatic stress: $t(37) = 11.0, p < .001$; mental arithmetic: $t(37) = 9.2, p < .001$). HR was also significantly higher during cold pressor than in its recovery period ($t(37) = 6.8, p < .001$). Similarly, HF power was significantly reduced in orthostatic stress when compared to baseline ($t(37) = 4.6, p < .001$) and recovery ($t(37) = 5.3, p < .001$). HF power was higher in supine rest in comparison to its baseline ($t(37) = 3.1, p < .005$). Other task contrasts with baseline and recovery were not significant for HR and HF power, and there were

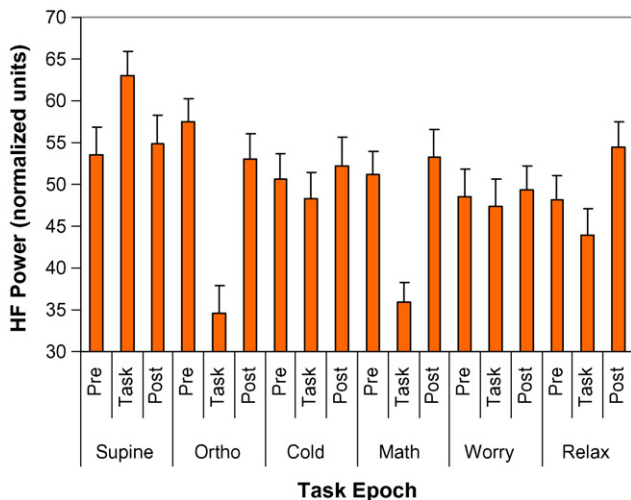


Fig. 2. HF power across epochs in all subjects.

no significant main condition effects or group by condition interactions for LF power, PEP, SBP, and DBP.

2.2. Anticipatory baseline analysis

All dependent variables were examined at the anticipatory baseline while again controlling for BMI, menstrual cycle, and time of testing. Again, HR was found to be higher in the high ($M = 81.0$ b.p.m., $S.E. = 2.3$) than low ($M = 72.5$ b.p.m., $S.E. = 2.2$) worry group ($F(1, 33) = 6.55, p < .02$). No other CV variables differed significantly during this condition.

3. Discussion

The primary finding of this study was a consistent HR elevation in the high worry group when compared to the low worry group. HR was greater in high worriers across all conditions in the study. Moreover, the high worriers showed elevated HR shortly after arrival in the lab, prior to the onset of the experimental protocols (i.e., in the anticipatory baseline). These results are consistent with reported associations between elevated HR and worry in its various forms, such as GAD (Thayer et al., 1996) and state and trait worry and rumination (Dua and King, 1987; Brosschot et al., 2007; Pieper et al., 2007; Roger and Jamieson, 1988). These data do conflict with the one extant negative finding on worry and HR (Davis et al., 2002). No association was found in the present study between worry and reduced HRV, in contrast to other reports of this relationship (Brosschot et al., 2007; Pieper et al., 2007; Thayer et al., 1996). As such, the lack of group differences in HF power bears closer scrutiny.

First, it should be noted that in general, HF power changed in the expected direction in response to experimental manipulations; i.e., it increased during supine rest and decreased during orthostatic stress and mental arithmetic when compared to baseline and recovery. Hence, it appears that HF power served as a valid vagal indicator during these conditions, and so it can be assumed that lack of group differences in vagal control is a reliable finding of the study.

Discrepancies between this and other studies might stem from the distinction between clinical GAD, in which HRV differences have been found (Thayer et al., 1996), and PSWQ assessed trait worry. Although the convergence of the PSWQ and GAD was noted above, it is possible that the high worry group in this study included significant numbers of sub-clinical GAD individuals. There were no clinical diagnoses made in this study, so this possibility cannot be ruled out. However, the mean PSWQ score in the high worry group ($M = 67.3$) is comparable to those in other studies that found high overlap between PSWQ score and GAD diagnosis (cf. $M = 68.1$ in Fresco et al., 2003; $M = 66.5$ in Roemer et al., 1995). As such, this explanation is plausible but unlikely. Future studies that include GAD diagnosis, PSWQ criteria, and physiological indices will be informative in this regard.

Diminished HRV in state worry has been found in ambulatory studies (Brosschot et al., 2007; Pieper et al., 2007). It is conceivable that HRV differences are more apparent in varied, naturalistic contexts than in more restricted laboratory settings. Moreover, the significant negative relationship between trait worry and HRV found in Brosschot et al. (2007) became marginal after partialing out the effects of variables such as smoking, alcohol, and caffeine. On the other hand, the negative association between trait worry and HR remained significant after being subject to the same analyses. Perhaps HR is a more robust indicator of worry than HRV, and HRV differences are more likely to appear in extreme cases (i.e., clinical GAD) or naturalistic settings. Direct comparisons of PSWQ and clinical worriers and likewise lab and field

studies will be important in assessing the impact of these factors on CV function in worry.

The worry and relaxation imagery tasks appear to have been the least potent elicitor of CV changes in the present study. This task set was designated as the final one to avoid contamination of the other tasks by rumination in high worriers. Unfortunately, this strategy may have diluted the effects of imagery by fatigue. Neither imagery task evoked significant HR or HF power reactivity, in contrast to Thayer et al. (1996). Analogous effects have been noted in clinic settings, in which a progressive decline in HR is observed over the course of a medical visit (Mancia et al., 1983). Hence, the present results are not directly comparable to those of Thayer et al. (1996), since worry and relaxation imagery were the only manipulations used in that study.

It may be that an extended repeated measures design is not optimally matched to the aim of assessing CV responses across a broad range of tasks. Investigators may consider instead a series of experiments involving smaller task sets to avoid potential order and fatigue effects. Inspection of Fig. 1 indicates that the majority of significant group HR differences occurred in the earlier phases of the study. Since tasks in this part of the study also tended to be more reflexive in nature (e.g., orthostatic stress), it is unclear if these HR effects were due to task type or order. That said, similar to Thayer et al. (1996), there were no group by task interactions on HR. This result suggests that excessive worriers are marked by chronic HR elevations, rather than by high reactivity to or delayed recovery from specific stressors. This interpretation is supported by the elevated HR displayed by the high worriers in the initial anticipatory baseline, indicating that they may have been ruminating even before the experiment began.

The lack of group differences in HRV and PEP indicate that worry is associated with tonic HR elevations without concomitant reduction in cardiac vagal tone or increased sympathetic β -adrenergic activity. This result begs the question of what mechanism could support this scenario. A possible explanation can be found in the effects of regular physical exercise on HR, which is associated with resting bradycardia that results from lower intrinsic HR, rather than autonomic adjustments (Bonaduce et al., 1998; de Geus et al., 1996; Goedhart et al., in press; Katona et al., 1982; Kingwell et al., 1992; Uusitalo et al., 1996). The process by which this effect occurs is open to speculation, but it may involve changes in myocardial pacemaker tissue or cell metabolism (Katona et al., 1982). Perhaps a similar mechanism operates in chronic worry, albeit in reverse, resulting in increased intrinsic HR.

Lovallo's (2005) model of individual differences in stress responses may have heuristic value in explaining the results of this study. In this model, individual differences in CV function that in turn show a systematic relationship to personality can be manifested at three physiological levels: the frontal-limbic system, the hypothalamus and brainstem, and the peripheral organs. The HR findings in the absence of HRV and PEP differences suggest that chronic worry may manifest itself at the peripheral level of organ physiology. The lack of BP differences in this study suggests that in young healthy female worriers, this effect is specific to cardiac chronotropic rather than vascular function.

The finding of elevated HR but not diminished vagal or enhanced sympathetic activity does not diminish the potential health risks of worry (e.g., Kubzansky et al., 1997). Chronic high HR is a risk factor for all-cause mortality (Palatini and Julius, 1997). Although altered autonomic tone is posited by these researchers as a potential mechanism linking tachycardia and CV mortality risk, hemodynamic disturbances are also mentioned. Additionally, elevated HR may index "general poor health and lack of vigor" in relation to its association with non-CV mortality (Palatini and Julius, 1997, p. 14). It is noteworthy that the mean HR among the

high worriers, a group of healthy young women, during the anticipatory baseline was 81.3 b.p.m., a figure that approaches the tonic value of 84 b.p.m. which has marked an increase in coronary heart disease incidence in epidemiologic research (Gillum et al., 1991).

Gender is an important factor to consider in studying worry and CV function, and the present findings may not generalize to men. HR tends to be higher in women than men (Palatini and Julius, 1997), and gender differences in HRV have been reported (Thayer et al., 1998). As such, future studies of male worriers may reveal aberrant autonomic activity not found in the present sample. As mentioned above, the link between anxiety and CVD is less consistent in women than in men (Eaker et al., 1992; Matthews et al., 1998). The one extant longitudinal study showing a connection between worry and CVD did not include women (Kubzansky et al., 1997). Moreover, it is not known how the results obtained in a healthy young sample will generalize to older individuals. Clearly, gender and age are important factors for future systematic investigation.

In addition, methods more sensitive to the time course of recovery might reveal impaired CV recovery in worriers, and should be employed in future research (Linden et al., 1997). Baroreflex control as assessed by HRV is another dimension of cardiac function that been shown to be reduced in trait anxious individuals (Watkins et al., 1999). Such methods may uncover CV features of worry not found in the present study.

3.1. Summary and conclusion

The most prominent finding of this study is that of elevated HR in a sample of female undergraduates classified as high worry by the PSWQ. This effect did not show a particular association with condition, be it baseline, a range of laboratory tasks, or recovery. In contrast, high and low worry groups did not differ on non-invasive measures of cardiac vagal and sympathetic tone, nor on BP. Future studies might focus on task subsets in order to obtain a purer representation of their CV effects. It will also be important to compare these results with those obtained under ambulatory conditions, in clinical samples, and in men to get a better sense of their representation of the CV concomitants of worry.

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References

- Allen, J.B.J., Chambers, A.S., Towers, D.N., 2007. The many metrics of cardiac chronotropy: A pragmatic primer and a brief comparison of metrics. *Biological Psychology* 74, 243–262.
- American Psychiatric Press, 1994. *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)*, 4th ed. APA, Washington, DC.
- Barger, S.D., Sydemann, S.J., 2005. Does generalized anxiety disorder predict coronary heart disease risk factors independently of major depressive disorder? *Journal of Affective Disorders* 88, 87–91.
- Bonaduce, D., Petretta, M., Cavallaro, V., Apicella, C., Iannicello, A., Romano, M., et al., 1998. Intensive training and cardiac autonomic control in high level athletes. *Medicine and Science in Sports and Exercise* 30, 691–696.
- Borkovec, T.D., Ray, W.J., Stober, J., 1998. Worry: a cognitive phenomenon intimately linked to affective, physiological, and interpersonal behavioral processes. *Cognitive Therapy and Research* 22, 561–576.
- Brosschot, J.F., Gerin, W., Thayer, J.F., 2006. The perseverative cognition hypothesis: a review of worry, prolonged stress-related physiological activation, and health. *Journal of Psychosomatic Research* 60, 113–124.
- Brosschot, J.F., Van Dijk, E., Thayer, J.F., 2007. Daily worry is related to low heart rate variability during waking and the subsequent nocturnal sleep period. *International Journal of Psychophysiology* 63, 39–47.

- Brosschot, J.F., Thayer, J.F., 1998. Anger inhibition, cardiovascular recovery, and vagal function: a model of the link between hostility and cardiovascular disease. *Annals of Behavioral Medicine* 4, 326–332.
- Brown, T.A., Antony, M.M., Barlow, D.H., 1992. Psychometric properties of the Penn state worry questionnaire in a clinical anxiety disorders sample. *Behaviour Research and Therapy* 30, 33–37.
- Carroll, D., Smith, G.D., Sheffield, D., Willemsen, G., Sweetnam, P.M., Gallacher, J.E., Elwood, P.C., 1996. Blood pressure reactions to the cold pressor test and the prediction of future blood pressure status: data from the Caerphilly study. *Journal of Human Hypertension* 10, 777–780.
- Conrad, A., Wilhelm, F.H., Roth, W.T., Spiegel, D., Taylor, C.B., 2008. Circadian affective, cardiopulmonary, and cortisol variability in depressed and nondepressed individuals at risk for cardiovascular disease. *Journal of Psychiatric Research* 42, 769–777.
- Davis, M., Montgomery, I., Wilson, G., 2002. Worry and heart rate variables: autonomic rigidity under challenge. *Journal of Anxiety Disorders* 16, 639–659.
- de Geus, E.J.C., Karsdorp, R., Boer, B., de Regt, G., Orlebeke, J.F., van Doornen, L.J.P., 1996. Effect of aerobic fitness training on heart rate variability and cardiac baroreflex sensitivity. *Homeostasis* 37, 28–51.
- Dua, J.K., King, D.A., 1987. Heart rate and skin conductance as measures of worrying. *Behaviour Change* 4, 26–32.
- Eaker, E.D., Pinsky, J., Castelli, W.P., 1992. Myocardial infarction and coronary death among women: psychosocial predictors from a 20-year follow-up of women in the Framingham Study. *American Journal of Epidemiology* 135, 854–864.
- Eckberg, D.L., 1997. Sympathovagal balance: a critical appraisal. *Circulation* 96, 3224–3232.
- Engelbreton, T.O., Matthews, K.A., Scheier, M.F., 1989. Relations between anger expression and cardiovascular reactivity: reconciling inconsistent findings through a matching hypothesis. *Journal of Personality and Social Psychology* 57, 513–521.
- Everson-Rose, S.A., Lewis, T.T., 2005. Psychosocial factors and cardiovascular diseases. *Annual Review of Public Health* 26, 469–500.
- Fresco, D.M., Mennin, D.S., Heimberg, R.G., Turk, C.L., 2003. Using the Penn State Worry Questionnaire to identify individuals with generalized anxiety disorder: a receiver operating characteristic analysis. *Journal of Behavior Therapy and Experimental Psychiatry* 34, 283–291.
- Friedman, B.H., 2007. An autonomic flexibility-neurovisceral integration model of anxiety and cardiac vagal tone. *Biological Psychology* 74, 185–199.
- Friedman, B.H., Allen, M.T., Christie, I.C., Santucci, A.K., 2002. Validity concerns of common heart-rate variability indices: addressing quantification issues in time- and frequency-domain measures of HRV. *IEEE Engineering in Medicine and Biology* 21 (4), 35–40.
- Friedman, B.H., Santucci, A.K., 2003. Idiodynamic profiles of cardiovascular activity: a p-technique approach. *Integrative Physiological and Behavioral Science* 38, 295–315.
- Friedman, B.H., Thayer, J.F., Tyrrell, R.A., 1996. Spectral characteristics of heart period variability in shock avoidance and cold face stress in normal subjects. *Clinical Autonomic Research* 6, 147–152.
- Friedman, M., Rosenman, R.H., 1971. Type A behavior: its association with coronary heart disease. *Annals of Clinical Research* 3, 300–312.
- Gerin, W., Davidson, K.W., Christenfeld, N.J.S., Goyal, T., Schwartz, J.E., 2006. The role of angry rumination and distraction in blood pressure recovery from emotional arousal. *Psychosomatic Medicine* 68, 64–72.
- Gillum, R.F., Makuc, D.M., Feldman, J.J., 1991. Pulse rate, coronary heart disease, and death: The NHANES I epidemiologic follow-up study. *American Heart Journal* 121, 172–177.
- Glynn, L.M., Christenfeld, N., Gerin, W., 2002. The role of rumination in recovery from reactivity: cardiovascular consequences of emotional states. *Psychosomatic Medicine* 64, 714–726.
- Godin, G., Shephard, R.J., 1997. Godin Leisure-Time Exercise Questionnaire. *Medicine and Science in Sports and Exercise* (29 June Supplement), S36–S38.
- Goedhart, A.D., de Vries, M., Kreft, J., Bakker, F.C., de Geus, E.J.C. No effect of training state on ambulatory measures of cardiac autonomic control. *Journal of Psychophysiology*, in press.
- Groot, P.F.C., de Geus, E.J.C., de Vries, J. (1998). Ambulatory Monitoring System. User Manual v1.2. Vrije Universiteit, Department of Psychophysiology, Amsterdam, The Netherlands. www.psy.vu.nl/ams.
- Haines, A.P., Imeson, J.D., Meade, T.W., 1987. Phobic anxiety and ischaemic heart disease. *British Medical Journal* 295, 297–299.
- Harbin, T.J., 1989. The relationship between type A behavior pattern and physiological responsivity: a quantitative review. *Psychophysiology* 26, 110–119.
- Hofmann, S.G., Moscovitch, D.A., Litz, B.T., Kim, H.-J., Davis, L.L., Pizzagalli, D.A., 2005. The worried mind: autonomic and prefrontal activation during worrying. *Emotion* 5, 464–475.
- Katona, P.G., Mclean, M., Dighton, D.H., Guz, A., 1982. Sympathetic and parasympathetic cardiac control in athletes and non-athletes at rest. *Journal of Applied Physiology* 52, 1652–1657.
- Kasagi, F., Akahoshi, M., Shimaoka, K., 1995. Relationship between cold pressor test and development of hypertension on 28-year follow-up. *Hypertension* 25, 71–76.
- Kawachi, I., Colditz, G.A., Ascherio, A., Rimm, E.B., Giovannucci, E., Stampfer, M.J., Willett, W.C., 1994a. Prospective study of phobic anxiety and risk of coronary heart disease in men. *Circulation* 89, 1992–1997.
- Kawachi, I., Sparrow, D., Vokonas, P.S., Weiss, S.T., 1994b. Symptoms of anxiety and risk of coronary heart disease. The Normative Aging Study. *Circulation* 90, 2225–2229.
- Kingwell, B.A., Dart, A.M., Jennings, G.L., Korner, P.I., 1992. Exercise training reduces the sympathetic component of the blood-pressure heart-rate baroreflex in man. *Clinical Science* 82, 357–362.
- Kubzansky, L.D., Kawachi, I., 2000. Going to the heart of the matter: do negative emotions cause coronary heart disease? *Journal of Psychosomatic Research* 48, 323–337.
- Kubzansky, L.D., Kawachi, I., Spiro, A., Weiss, S.T., Vokonas, P.S., Sparrow, D., 1997. Is worrying bad for your heart? A prospective study of worry and coronary heart disease in the Normative Aging Study. *Circulation* 95, 818–824.
- Linden, T.L.W., Gerin, W., Christenfeld, N., 1997. Physiological stress reactivity and recovery: conceptual siblings separated at birth? *Journal of Psychosomatic Research* 42, 117–135.
- Lovallo, W.R., 2005. *Stress & Health: Biological and Psychological Interactions*, 2nd ed. Sage, Thousand Oaks, CA.
- Malliani, A., Pagani, M., Montano, N., 1998. Sympathovagal balance: a reappraisal. *Circulation* 98, 2640–2643.
- Mancia, G., Bertinieri, G., Grassi, G., Parati, G., Pomidossi, G., Ferrari, A., et al., 1983. Effects of blood pressure measurement by the doctor on patients blood pressure and heart rate. *Lancet* ii, 695–697.
- Manuck, S.B., 1994. Cardiovascular reactivity and cardiovascular disease: "Once more unto the breach". *International Journal of Behavioral Medicine* 1, 4–31.
- Matthews, K.A., Owens, J.F., Kuller, L.H., Sutton-Tyrrell, K., Jansen-McWilliams, L., 1998. Are hostility and anxiety associated with carotid atherosclerosis in healthy postmenopausal women? *Psychosomatic Medicine* 60, 633–638.
- Meyer, T.J., Miller, M.L., Metzger, R.L., Borkovec, T.D., 1990. Development and validation of the Penn State Worry Questionnaire. *Behaviour Research and Therapy* 28, 487–495.
- Mezzacappa, E.S., Kelsey, R.M., Katkin, E.S., Sloan, R.P., 2001. Vagal rebound and recovery from psychological stress. *Psychosomatic Medicine* 63, 650–657.
- Obrist, P.A., 1981. *Cardiovascular Psychophysiology*. Plenum Press, New York.
- Pagani, M., Lucini, D., Rimoldi, O., Furlan, R., Piazza, S., Biancardi, L., 1995. Effects of physical and mental exercise on heart rate variability. In: Malik, M., Camm, A.J. (Eds.), *Heart Rate Variability*. Futura Publishing Co., Armonk, NY, pp. 245–266.
- Palatini, P., Julius, S., 1997. Heart rate and the cardiovascular risk. *Journal of Hypertension* 15, 3–17.
- Pieper, S., Brosschot, J.F., van der Leeden, R., Thayer, J.F., 2007. Cardiac effects of momentary assessed worry episodes and stressful events. *Psychosomatic Medicine* 69, 901–909.
- Plutchik, R., 1990. Emotions and psychotherapy: a psychoevolutionary perspective. In: Plutchik, R., Kellerman, H. (Eds.), *Emotion: Theory, research, and experience: Vol. 5. Emotion, Psychopathology and Psychotherapy*, Academic Press, San Diego, CA, pp. 3–41.
- Porges, S.W., 2007. The polyvagal perspective. *Biological Psychology* 74, 116–143.
- Princi, T., Parco, S., Accardo, A., Radillo, O., De Seta, F., Guaschino, S., 2005. Parametric evaluation of heart rate variability during the menstrual cycle in young women. *Biomedical Sciences Instrumentation* 41, 340–345.
- Ramaekers, D., Ector, H., Aubert, A.E., Rubens, A., Van de Werf, F., 1998. Heart rate variability and heart rate in healthy volunteers. Is the female autonomic nervous system cardioprotective? *European Heart Journal* 19 (9), 1334–1341.
- Roemer, L., Borkovec, M., Posa, S., Borkovec, T.D., 1995. A self-report diagnostic measure of generalized anxiety disorder. *Journal of Behavior Therapy and Experimental Psychiatry* 26, 345–350.
- Roger, D., Jamieson, J., 1988. Individual differences in delayed heart-rate recovery following stress: the role of extraversion, neuroticism and emotional control. *Personality and Individual Differences* 9, 721–726.
- Rozanski, A., Blumenthal, J.A., Kaplan, J., 1999. Impact of psychological factors on the pathogenesis of cardiovascular disease and implications for therapy. *Circulation* 99, 2192–2217.
- Saab, P.G., Llabre, M.M., Hurwitz, B.E., Schneiderman, N., Wohlgenuth, W., Durel, L., Massie, C., Nagel, J., 1993. The cold pressor test: vascular and myocardial response patterns and their stability. *Psychophysiology* 30, 366–373.
- Schuler, J.L., O'Brian, W.H., 1997. Cardiovascular recovery from stress and hypertension risk factors: a meta-analytic review. *Psychophysiology* 34, 649–659.
- Schwartz, A.R., Gerin, W., Davidson, K., Pickering, T.G., Brosschot, J.F., Thayer, J.F., Christenfeld, N., Linden, W., 2003. Toward a causal model of cardiovascular responses to stress and the development of cardiovascular disease. *Psychosomatic Medicine* 65, 22–35.
- Shen, B.-J., Avivi, Y.E., Todaro, J.F., Spiro III, A., Laurenceau, J.-P., Ward, K.-D., Niaura, R., 2008. Anxiety characteristics independently and prospectively predict myocardial infarction in men. *Journal of the American College of Cardiology* 51, 113–119.
- Sherwood, A., Allen, M.T., Fahrenberg, J., Kelsey, R.M., Lovallo, W.R., van Doornen, L.J.P., 1990. Methodological guidelines for impedance cardiography. *Psychophysiology* 27, 1–23.
- Suls, J., Wan, C.K., 1993. The relationship between trait hostility and cardiovascular reactivity: a quantitative review and analysis. *Psychophysiology* 30, 615–626.
- Stephoe, A., Feldman, P.J., Kunz, S., Owen, N., Willemsen, G., Marmot, M., 2002. Stress responsivity and socioeconomic status. A mechanism for increased cardiovascular disease risk? *European Heart Journal* 23, 1757–1763.
- Stewart, J.C., France, C.R., 2001. Cardiovascular recovery from stress predicts longitudinal changes in blood pressure. *Biological Psychology* 58, 105–120.
- Thayer, J.F., Friedman, B.H., Borkovec, T.D., 1996. Autonomic characteristics of generalized anxiety disorder and worry. *Biological Psychiatry* 39, 255–266.

- Thayer, J.F., Friedman, B.H., Borkovec, T.D., Johnsen, B.H., Molina, S., 2000. Phasic heart period reactions to cued threat and nonthreat stimuli in generalized anxiety disorder. *Journal of Psychophysiology* 37, 361–368.
- Thayer, J.F., Smith, M., Rossy, L.A., 1998. Heart period variability and depressive symptoms: gender differences. *Biological Psychiatry* 44, 304–306.
- Treiber, F.A., Kamarck, T., Schneiderman, N., Sheffield, D., Kapuku, G., Taylor, T., 2003. Cardiovascular reactivity and development of preclinical and clinical disease states. *Psychosomatic Medicine* 65, 46–62.
- Uusitalo, A.L.T., Tahvanainen, K.U.O., Uusitalo, A.J., Rusko, H.K., 1996. Non-invasive evaluation of sympathovagal balance in athletes by time and frequency domain analyses of heart rate and blood pressure variability. *Clinical Physiology* 16, 575–588.
- Vallejo, M., Márquez, M.F., Borja-Abrto, V.H., Cárdenas, M., Hermsillo, A.G., 2005. Age, body mass index, and menstrual cycle influence young women's heart rate variability – a multivariate analysis. *Clinical Autonomic Research* 15 (4), 292–298.
- Vella, E.J., Friedman, B.H., 2007. Autonomic characteristics of defensive hostility: reactivity and recovery to active and passive stressors. *International Journal of Psychophysiology* 66, 95–101.
- Vitaliano, P.P., Russo, J., Paulsen, V.M., Bailey, S.L., 1995. Cardiovascular recovery from laboratory stress: biopsychosocial concomitants in older adults. *Journal of Psychosomatic Research* 39, 361–377.
- Watkins, L.L., Grossman, P., Krishnan, R., Blumenthal, J.A., 1999. Anxiety reduces baroreflex cardiac control in older adults with major depression. *Psychosomatic Medicine* 61, 334–340.
- Willemsen, G.H.M., De Geus, E.J.C., Klaver, C.H.A.M., Van Doornen, L.J.P., Carroll, D., 1996. Ambulatory monitoring of the impedance cardiogram. *Psychophysiology* 33, 184–193.