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Acute autonomic effects of experimental worry and cognitive problem solving: Why worry about worry?[1]

Bart Verkuil[2] (*Leiden University, The Netherlands*), Jos F. Brosschot (*Leiden University, The Netherlands*), Thomas D. Borkovec (*The Pennsylvania State University, USA*), and Julian F. Thayer (*The Ohio State University, USA*)

ABSTRACT. Worry has been associated with adverse mental and somatic health outcomes, which have been attributed to the pathogenic physiological activity caused by worry. However, experimental evidence is scarce, and existing studies did not address whether the physiological effects of worry do actually exceed those of mere mental load during cognitive problem solving. In the present experiment, heart rate (HR) and heart rate variability (HRV) of fifty-three participants were continuously measured during induced worrying, problem solving concerning issues that were not personally relevant, and relaxation. The results showed that HR was higher and HRV lower during both worrying and problem solving than during relaxation. Differences in emotional responses did not account for these results. This suggests that mere mental load is responsible for - at least a part of - the physiological effects of worry. Consequently, long term health effects of worry might be due to prolonged mental load of worry rather than to its emotional aspects.

KEYWORDS. Worry. Problem solving. Cardiovascular reactivity. Heart rate variability. Experiment.

[2] Correspondence: Leiden University. Institute for Psychological Research. PO Box 9555. 2300 RB Leiden (The Netherlands). E-mail: bverkuil@fsw.leidenuniv.nl

RESUMEN. La preocupación se ha asociado a consecuencias negativas para la salud mental y somática, lo que ha sido atribuido a una actividad fisiológica patogénica causada por la misma. Sin embargo, la evidencia experimental es escasa y los estudios existentes no han analizado si los efectos de la preocupación exceden los de la mera carga mental durante la resolución de problemas. En este experimento se midió de forma continua la tasa cardiaca (TC) y la variabilidad cardiaca (VC) de cincuenta y tres participantes durante una fase de preocupación inducida, una fase de resolución de problemas sobre asuntos no relevantes personalmente y una fase de relajación. Los resultados mostraron que la TC era más alta y la VC más baja tanto durante la fase de preocupación como durante la de resolución de problemas en comparación con la de relajación. Las diferencias en respuestas emocionales no explican estos resultados. Esto sugiere que la mera carga mental es responsable de -al menos- los efectos fisiológicos de la preocupación. En consecuencia, los efectos sobre la salud a largo plazo se podrían deber a carga mental prolongada de la preocupación más que a sus aspectos emocionales.

PALABRAS CLAVE. Preocupación. Resolución de problemas. Reactividad cardiovascular. Variabilidad cardiaca. Estudio experimental.

A large body of research has made clear that stressful events can have profound effects on mental health, such as depression (McEwen, 2003), and somatic health, such as cardiovascular diseases (CVD; Rozanski, Blumenthal, and Kaplan, 1999). However, the exact psychophysiological mechanisms by which stressors have these adverse effects are not well understood (Pieper and Brosschot, 2005). Recently, Brosschot, Gerin, and Thayer (2006) proposed that perseverative thoughts, such as worry, rumination and trauma recall, are important mediators of the health effects of stressors. The recurrent or persistent cognitive representation of stressors, especially their uncontrollability, might prolong physiological activation of several bodily systems, including the endocrine, immune and cardiovascular systems. Prolonged physiological activity, or prolonged arousal, is unequivocally present in the early stress theory of Selye (1950). Also in more recent years, physiological activation that is prolonged beyond the presence of actual stressors has been put forward as a crucial stage in the causal chain from stressors to disease (Pieper and Brosschot, 2005; Schwartz *et al.*, 2003). Prolonged activation of the autonomic nervous system, especially high levels of heart rate (HR) and low levels of heart rate variability (HRV), is a risk factor for CVD and is argued to play an important etiological role in a wide range of other somatic and psychological pathological conditions, including immune dysfunction, diabetes, mood and anxiety disorders and, more generally, self regulatory difficulties (Masi, Hawkley, Rickett, and Cacioppo, 2007; Thayer and Lane, 2000; Thayer and Lane, 2007; Thayer and Sternberg, 2006).

Several studies support crucial aspects of this "perseverative cognition hypothesis". Worry has been shown to predict anxiety (Hong, 2007) and cardiovascular disease (Kubzansky *et al.*, 1997), and it was associated with enhanced activation in endocrine, immune and cardiovascular systems (Brosschot, van Dijk, and Thayer, 2007; Gerin, Davidson, Christenfeld, Goyal, and Schwartz, 2006; McCullough, Orsulak, Brandon, and

Akers, 2007; Pieper, Brosschot, van der Leeden, and Thayer, 2007; for a review of earlier studies see Brosschot *et al.*, 2006). Recent diary studies showed that worry in daily life is associated with enhanced HR and lowered HRV, independent of and stronger than actual stressful environmental events (Brosschot *et al.*, 2007; Pieper *et al.*, 2007). However, such momentary assessments yield correlational data, which are not unequivocal with respect to causality. Moreover, they do not allow for more rigorous testing of the mechanisms that are responsible for these physiological effects. More direct evidence of the physiological effects of worry comes from the few studies that have directly manipulated worry and measured its acute cardiac effects (Borkovec and Hu, 1990; Borkovec, Lyonfields, Wiser, and Deihl, 1993; Borkovec, Robinson, Pruzinsky, and DePree, 1983; Davis, Montgomery, and Wilson, 2002; Hofmann *et al.*, 2005; Lyonfields, Borkovec, and Thayer, 1995; Thayer, Friedman, and Borkovec, 1996). Although some of these studies only measured HR and failed to demonstrate differences in cardiac activity between experimentally induced worry and relaxation (Borkovec *et al.*, 1983, Borkovec and Hu, 1990), other studies measured both HR and HRV and found that worry is associated with enhanced HR and with low HRV compared to relaxation (Davis *et al.*, 2002; Hofmann *et al.*, 2005; Lyonfields *et al.*, 1995; Thayer *et al.*, 1996). Still, simple relaxation is not sufficient as a control condition to understand the mechanisms underlying the potentially adverse physiological effects of worry. It is implicitly assumed that its adverse effects are due to the defining characteristics of worry (namely perseveration of negative thoughts) or to anxiety and other negative emotions that are associated with worry (Borkovec *et al.*, 1983). Yet, it is not unlikely that the cardiac effects of worry are at least partly caused by the high cognitive activity or mental effort involved in worrying. Effortful cognitive problem solving has been known for a long time to increase HR and decrease HRV (e.g., Brod, Fencl, Heijl, and Jirka, 1959).

Worry and cognitive problem solving are closely related (Davey, 1994). Both worry and cognitive problem solving involve abstract reasoning about problems, and both can occur without the actual presence of these problems. In fact, worry has been defined as consisting of 'attempts to engage in mental problem solving', albeit thwarted attempts (Borkovec *et al.*, 1983). Thus, it is possible that the effects of worry on HR and HRV that have been found earlier are, at least in part, due to the mental effort associated with the cognitive problem solving component of worry. The crucial difference between worrying and mere cognitive problem solving is the perseveration of negative thoughts and emotions. Worrying involves thinking about topics involving personally relevant threats (Mcintosh, Harlow, and Martin, 1995), thereby increasing and prolonging negative affect and negative thoughts. Mere cognitive problem solving, on the other hand, involves personal topics not associated with threat. Recent neuroimaging studies by Greene *et al.* (Greene, Nystrom, Engell, Darley, and Cohen, 2004; Greene, Sommerville, Nystrom, Darley, and Cohen, 2001) demonstrated that pure cognitive problem solving was associated with activation in the brain areas associated with working memory (the right middle frontal gyrus and bilateral parietal lobes), while only the personal relevance of a problem being solved was associated with additional heightened activation of emotional areas of the brain (the medial frontal gyri, posterior cingulate gyri and the

angular gyri). Thus, to test whether physiological effects of worry are truly due to the perseveration of negative emotions, the latter should be compared with solving problems that are not personally relevant.

Worry is increasingly recognized as a pathogenic cognitive process in the link between stressors and disease. However, to date no study has examined whether the physiological effects of worry actually exceed those of mere problem solving. In the present experimental within subjects study (Montero and León, 2007; Ramos-Álvarez, Moreno-Fernádez, Valdés-Conroy, and Catena, 2008), the cardiac effects of experimentally induced worrying were compared with those of a cognitive problem solving task concerning problems that were not personally relevant and with those of a relaxation condition. For this purpose, we employed the operationalization of non-personally relevant problem solving used in the neuroimaging study by Greene and coworkers (Greene *et al.*, 2001; Greene *et al.*, 2004) and shown not to activate emotional brain areas. The main objective of this study was to rule out one of two rival hypotheses: a) the cardiac effects of worry will exceed those of cognitive problem solving, thus ruling out mere mental load as the explanation for the cardiac effects of worry, or b) the two conditions are equal in their cardiac effects, thereby ruling out the hypothesis that negative emotion associated with worrisome thinking is causing the cardiac effects of worry.

Method

Participants

The sample consisted of 18 male and 35 female students, aged 17-50 (mean = 24.4). Seventy-six percent of the sample had Caucasian ethnicity, 4% African, 11% Asian and 9% had mixed ethnicity. Participants were recruited by advertisement at Leiden University.

Procedure

After giving informed consent, all participants took part in three experimental conditions: a worry induction, a cognitive problem solving task, and a relaxation induction. The experimental conditions were presented in counterbalanced order. Each condition lasted 10 minutes. After the experimental conditions, the participants completed questionnaires and were paid 6 euros or received course credits. During the experiment, cardiac activity was recorded.

Worry induction

Following the work of Borkovec and others (Lyonfields *et al.*, 1995; Thayer *et al.*, 1996), participants were asked to write down three personal worry topics before receiving further instructions. To minimize participant's social evaluative concerns about writing down a personal worry topic, they were notified that they could take home or destroy the paper on which they wrote their worry topic. Thereafter, participants were asked 'to worry as you usually do' for ten minutes.

Relaxation induction

In the relaxation condition, participants were asked to relax and to let their minds wander. Some non-arousing magazines were available to read.

Cognitive problem-solving induction

For the cognitive problem solving condition, we selected 10 moral dilemmas from the moral dilemma paradigm that were not personally relevant (for example: 'is it appropriate for your friend to misrepresent his curriculum vitae in order to get a job?' (for more details see Greene *et al.*, 2001). To be comparable with the cognitive activity during worrying (often jumping from one problem to another), participants were presented with 10 dilemmas on a computer screen. Each dilemma was presented on the screen for 1 minute. Participants were asked to judge for themselves the correctness of the actions that were described in the dilemma scenes. To rule out the possibility that participants would feel judged themselves, they were not asked to respond to the dilemmas in any other way.

Assessment of mood

Mood changes due to the experimental manipulations were assessed using visual analog scales, with scales ranging from 0 to 10. After each of the three induction periods, participants rated their level of 'anxiety', 'sadness', 'irritation', 'tension' and 'impatience'. For each rating, the participants were first asked to rate their mood as usual with a vertical line and then to indicate with a cross their mood during the preceding period. To assess the effects of the experimental tasks on mood, we used the change score between mood as usual and mood during the preceding period (Brosschot *et al.*, 1992).

Assessment of cognition

After each induction, participants were asked to rate the intensity with which they worried, felt relaxed, or thought about the moral dilemmas. Additionally, after the worry induction and the cognitive problem solving task, participants were asked to rate the extent to which they had found solutions or had made a decision about the problem(s) they were thinking about.

Physiological recording

HR and HRV were continuously measured, in a non-invasive manner, with the Polar s810i wristwatch and the Polar Wearlink 31 belt band, which has a sampling rate of 1000 Hertz (Polar Electro Nederland BV; Gamelin, Berthoin, and Bosquet, 2006). Before analyzing HR and HRV, the raw interbeat intervals (IBIs) were preprocessed for artifacts using the Polar Precision Software. The corrected IBI series were subsequently processed with the HRV Analysis program, using the smoothness priors based approach which removes the low frequency trend component of the IBIs (Niskanen, Tarvainen, Ranta-Aho, and Karjalainen, 2004). For every 10-minute condition mean HR (in beats per minute, BPM) and mean HRV (root mean squared successive differences, RMSSD, in milliseconds) were calculated.

Assessment of individual differences
  – *Penn State Worry Questionnaire* (*PSWQ*; Meyer, Miller, Metzger, and Borkovec, 1990). This trait worry questionnaire consists of 16 self-report items that are directed at the excessiveness, duration, and uncontrollability of worry. The PSWQ has demonstrated high reliability, high temporal stability and substantial validity in the assessment of trait-worry (Meyer *et al.,* 1990; Verkuil, Brosschot, and Thayer, 2007).
  – *State Trait Anxiety Inventory-Trait Form* (*STAI-T*; van der Ploeg, Defares, and Spielberger, 1980). For measuring trait-anxiety we administered the trait version of the State-Trait Anxiety Inventory. It consists of 20 self-report items and earlier use has shown good internal consistency and validity (van der Ploeg *et al*., 1980).

Biobehavioral variables
     Participants' were asked to report the number of cigarettes, the number of cups of coffee, and the number of alcoholic beverages they had consumed before participating in this study, because this could influence their cardiac activity. For the same reason, participants were also asked to report the use of medication and chronic disease of themselves or their family, and their body mass-index (BMI) was measured.

Statistical analyses
     The distributions of the heart rate variability variables were significantly skewed according to the Kolmogorov-Smirnov test, but could successfully be normalized using log transformations. For variables that could not successfully be normalized (the biobehavioral variables and indices of mood change between the conditions), we used the appropriate non-parametric tests (Spearman's rho). To investigate differences in the physiological, cognitive, and mood variables between the conditions, repeated measures MANOVAs with condition (worry, cognitive problem solving, relaxation) as within subjects factor were used. To examine differences between individual means, we conducted pre-planned t-tests. P values and effect sizes for repeated measures designs are reported (Dunlap, Cortina, Vaslow, and Burke, 1996).

Results

Descriptive statistics
     Table 1 shows the means and standard deviations of the cardiac variables and the trait questionnaires for males and females. The mean levels of trait worry and trait anxiety were in line with previous studies conducted with student participants. With respect to gender differences, t-tests yielded no significant differences between women and men (all ps > .05), although inspection of the means suggested that women scored higher on the trait questionnaires and had higher HR and RMSSD than men. Spearman correlations between the biobehavioral variables (number of cups of coffee, cigarettes and alcoholic beverages, BMI, medication and medical history) and HR and RMSSD during the three conditions were not significant (all ps > .05).

TABLE 1. Means and standard deviations of cardiac
variables and trait questionnaires.

|	Males		Females	
	M	*SD*	*M*	*SD*
Heart Rate (bpm)				
Worry	75.28	9.38	78.01	11.77
Relaxation	72.70	11.11	75.69	11.61
Cognitive problem solving	75.22	11.21	78.01	11.86
RMSSD (ms)				
Worry	37.07	15.22	46.21	31.92
Relaxation	42.40	19.04	47.06	30.40
Cognitive problem solving	36.01	13.54	43.74	28.98
Trait worry	43.94	13.96	49.03	13.56
Trait anxiety	39.61	13.95	41.21	9.00

Manipulation check

A repeated measures ANOVA with condition (worry, cognitive problem solving, and relaxation) as a within subjects factor on rated intensity of worry revealed a main effect of condition ($F_{(2,104)}$ = 97.35, p < .0001, η^2 = .65). Pre-planned t-tests showed that the intensity of worry was significantly higher in the worry induction condition (M = 5.43, SD = 2.14) than in the relaxation (M = 0.43, SD = 1.15; $t_{(52)}$ = 15.97, p <.0001, r = .82) and cognitive problem solving conditions (M = 1.87, SD = 2.39; $t_{(52)}$ = 8.52, p < .0001, r = .62). In addition, the intensity with which participants were worrying was equal to the intensity with which participants were thinking about the dilemmas (M = 5.32, SD = 2.13; $t_{(52)}$ = .30, p = .768, r = .02). A paired t-test also showed that participants rated the extent to which they found solutions to their worries to be lower (M = 4.51, SD = 2.33) than to the impersonal moral dilemmas (M = 6.81, SD = 1.81; $t_{(52)}$ = 5.90, p = .000, r = .48).

Mood effects

Figure 1 illustrates the mood ratings after the three conditions, compared to a baseline of how participants usually feel. Examination of the mood change scores showed that the worry induction led to higher levels of anxiety ($t_{(52)}$ =2.68, p = .010, r = .12) and tension ($t_{(51)}$ =2.57, p = .013, r = .11), whereas the cognitive problem solving task did not lead to any significant changes in mood (ps > .09) and the relaxation condition overall led to lower levels of negative mood than mood as usual (ps <.01). Subsequently, a repeated measures MANOVA with condition (worry, cognitive problem solving, and relaxation) as a within subjects factor was performed on the mood variables (anxiety, sadness, impatience, tension, irritation). The omnibus test yielded a significant effect of condition ($F_{(10,194)}$ = 5.17, p < .0001, η^2 = .21). Pre-planned t-tests showed that overall the worry induction led to more negative mood than relaxation (all ps <.01). In addition, the worry induction led to more anxiety ($t_{(51)}$ = 2.86, p = .003, r = .24), sadness ($t_{(51)}$ = 2.41, p = .021, r = .21), and tension than the cognitive problem solving task ($t_{(51)}$

= 2.67, p = .010, r = .24). No significant difference between the conditions emerged in the amount of irritation and impatience (ps >.35).

FIGURE 1. Effects of worry, relaxation and cognitive problem solving on mood. Bars marked with asterices (*) are significantly different from baseline (mood as usual). Error bars represent +/- 1 standard error.
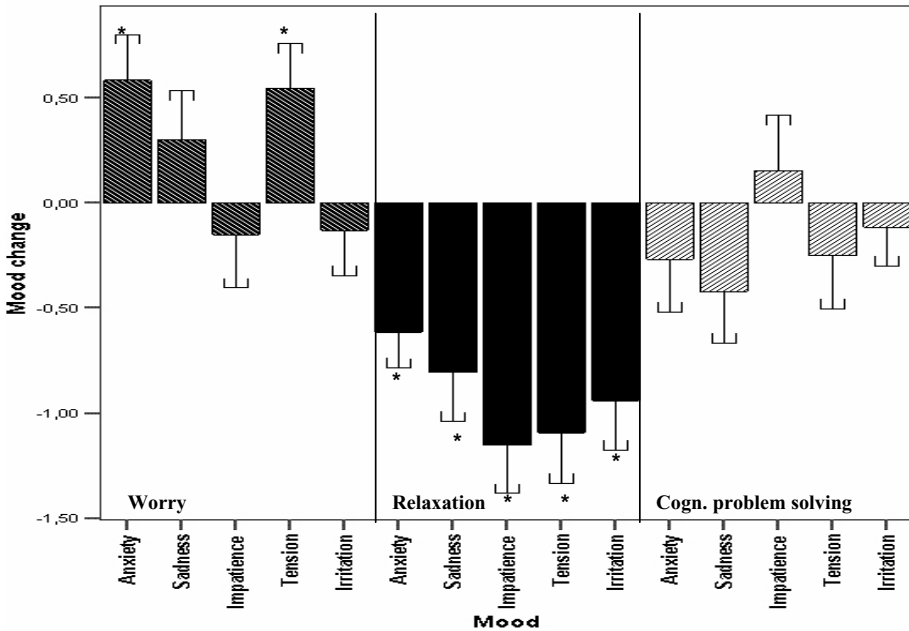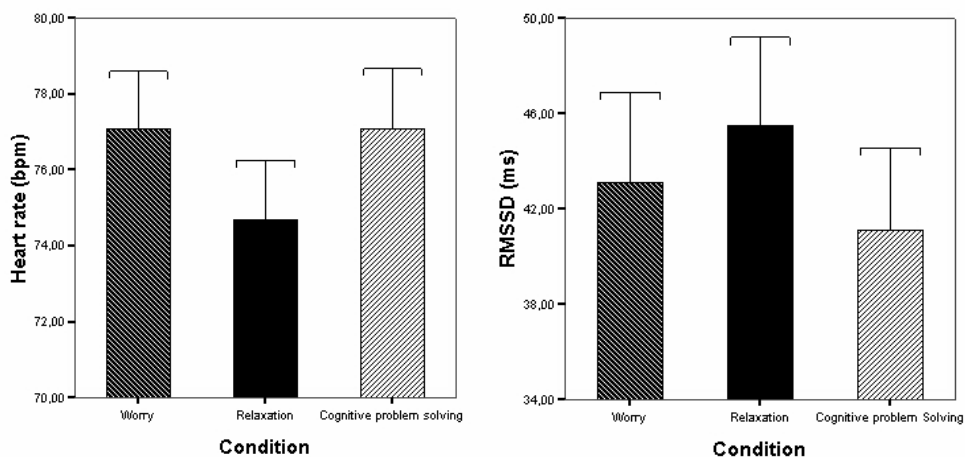

Cardiac effects

Mean HR and RMSSD are shown in Table 1 and Figure 2. A repeated measures MANOVA with condition as a within subjects factor was performed on HR and RMSSD. A significant main effect for condition emerged ($F_{(4, 208)}$ = 6.67, p < .0001, η^2 = .11). Pre-planned t-tests showed that HR was significantly higher during worrying ($t_{(52)}$ = 4.54, p < .0001, r = .11) and cognitive problem solving ($t_{(52)}$ = 3.68, p = .001, r = .10) than during relaxation. HR did not differ between the worrying and cognitive problem solving conditions ($t_{(52)}$ = 0.02, p = .978). A complimentary pattern was found for RMSSD. RMSSD was significantly higher during relaxation than during worrying ($t_{(52)}$ = 3.07, p = .003, r = .06) and cognitive problem solving ($t_{(52)}$ = 3.63, p = .001, r = .09). RMSSD did not differ between the worrying and cognitive problem solving conditions ($t_{(52)}$ = 1.49, p = .143).

To test whether the cardiac difference between worry and cognitive problem solving on the one hand and relaxation on the other could be due to changes in mood, change scores were calculated for mood, HR, and RMSSD by subtracting mean scores during relaxation from either worry or cognitive problem solving. Spearman correlations showed

that changes in mood were not related to changes in HR and RMSSD ($ps > .05$). In addition, visual inspection of the graphs did not suggest different temporal patterns between the conditions, and these temporal patterns were therefore not further analyzed.

FIGURE 2. Effects of worry, relaxation and cognitive problem solving on heart rate and RMSSD. Error bars represent +/- 1 standard error.


Individual differences in cardiac activity

As previous studies have suggested that gender, trait worry, and trait anxiety can account for differences between the cardiac effects of the conditions, we conducted two subsequent analyses to examine whether these individual differences moderated the cardiac effects of the conditions.

Therefore, the means of HR and RMSSD of the *combination* of worry and problem solving cognitions were calculated, because as shown above there was no difference in their cardiac effects. A repeated measures MANCOVA with HR and RMSSD as within subjects dependent variables, gender as a between subjects factor, and trait anxiety as covariate did not yield significant results. The same analysis for trait worry showed that the Condition x Gender interaction approached significance ($F_{(2, 48)} = 3.14$, $p = .052$, $\eta^2 = .12$) and that the interaction effect Condition x Gender x Trait worry was significant ($F_{(2, 48)} = 4.56$, $p = .015$, $\eta^2 = .16$). To examine this three-way interaction effect, we performed a median split on the trait worry questionnaire (PSWQ scores: low trait worriers $M = 36.03$ ($SD = 6.41$); high trait worriers $M = 59.00$ ($SD = 8.54$)). Pre-planned t-tests showed that trait worry and gender influenced the mean difference in RMSSD during the relaxation condition and the cognitive tasks, but not the observed difference in HR. Figure 3 shows the mean RMSSD separately for males and females, and for low and high trait worriers. Paired t-tests show what is clearly visible in Figure 3, that is, that RMSSD was higher during relaxation than during worry and cognitive problem solving ($ps < .05$), except for female high trait worriers ($t_{(19)} = 0.58$, $p = .57$) who showed equally high RMSSD during both cognitive tasks and during relaxation.

FIGURE 3. Individual differences moderating the effects of the cognitive tasks
(worry and cognitive problem solving) and relaxation on RMSSD. Error bars
represent +/- 1 standard error.
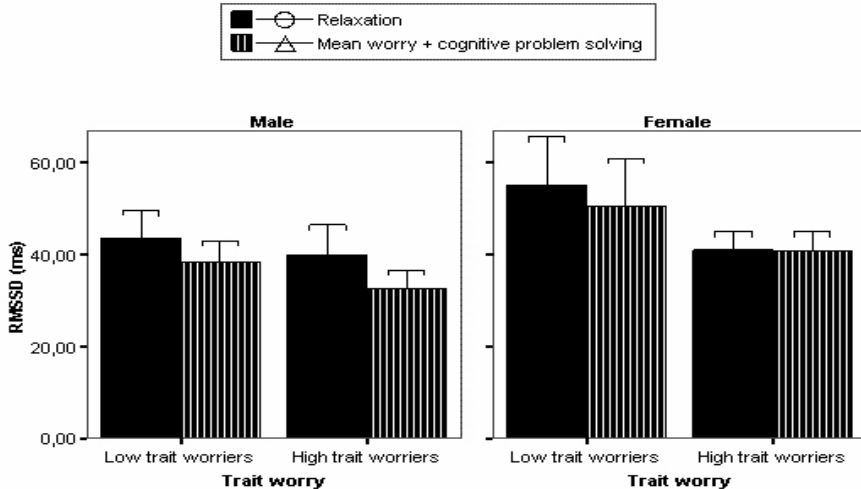
Discussion

The aim of this study was to investigate whether the cardiac effects of experimentally induced worry would differ from cognitive problem solving and relaxation. As expected, participants had higher HR and lower HRV during worry and cognitive problem solving compared to relaxation. Crucially for the goals of this investigation, the cardiac effects of induced worry were not different from cognitive problem solving per se.

The cardiac effects of experimental worry compared to relaxation that we found in this study are in line with previous experimental studies (Davis *et al.*, 2002; Hofmann *et al.*, 2005; Lyonfields *et al.*, 1995; Thayer *et al.*, 1996). Together, these results add important experimental evidence to the findings from real life studies that worry is associated with changes in cardiovascular, immunological, and endocrinological activation (Brosschot *et al.*, 2006; Brosschot *et al.*, 2007; McCullough *et al.*, 2007; Pieper *et al.*, 2007). These studies offer support for the theory that worry or more generally 'perseverative negative cognition' may represent an important risk factor for somatic disease, either on its own or by mediating the effects of other stress factors (Brosschot *et al.*, 2006).

However, the results of this study also suggest that the cardiac effects of induced worry are not different from cognitive problem solving per se. This might imply that mere mental load is responsible for at least a part of the physiological effects of worry, irrespective of the personal relevance of worrisome problem solving. Because it is unlikely that common daily cognitive problems can have substantial adverse health effects, it seems logical to infer that the adverse health effects of worry might be at least partly due to the prolonged mental load of worry, prolonged because worries tend to last longer than common cognitive problems and perhaps occur more often as well.

Worry problems are by definition less easy to solve, if not unsolvable, compared to common problems. Indeed, participants came up with fewer solutions to their worry topics than to the moral dilemmas. Thoughts concerned with issues that have not been dealt with successfully are more accessible in memory (Zeigarnik, 1927). Furthermore, although we found that negative mood could not directly account for altered cardiac functioning, several studies have shown that negative mood experienced during worrying adds to the total time spent thinking about problems because it informs us that a problem has not been dealt with effectively (e.g., Startup and Davey, 2001). In addition, as worry often involves highly valued personal goals (Mcintosh *et al*., 1995), it is possible that people require more evidence before implementing solutions to personally relevant problems, which again adds to the time spent worrying (Tallis, Eysenck, and Mathews, 1991). Consequently, worries intrude more often into awareness and are cognitively processed for much longer periods of time than non personal problems. Importantly, we have repeatedly shown that worry duration more so than worry frequency is associated with health complaints and prolonged cardiac effects (Brosschot and Van Der Doef, 2006; Brosschot *et al*., 2007; Pieper *et al*., 2007). As the duration of worry episodes may be longer lasting than the duration of cognitive problem solving episodes, the cardiac effects of worry may produce more sustained wear and tear on the system compared to cognitive problem solving [cf., the Neurovisceral Integration Model (Thayer and Lane, 2000) and the Allostatic Load Model of McEwen (McEwen, 2003)]. It is interesting to note in this context that we have found that the cardiac effects of positive and negative emotions initially do not differ (Jacob *et al*., 1999) but that negative emotions are associated with more sustained cardiac effects than positive emotions (Brosschot and Thayer, 2003). Future studies should further investigate the naturally occurring time course of the cardiac effects associated with worrying and with cognitive problem solving. It is possible that, in parallel with the different time course of the cardiac effects of positive versus negative emotions, worry mainly differs from cognitive problem solving in having sustained cardiac effects. These different temporal effects may account for differential health consequences of worry versus problem solving. These speculations should be addressed in future momentary assessment studies that examine the cardiac effects of worry and cognitive problem solving in daily life.

The results also showed that the HRV of women that were high in trait worry was equally high during the cognitive tasks as during relaxation. This result extends earlier findings showing that women high in depressive symptoms, including rumination, had higher baseline HRV compared to depressed men (Chambers and Allen, 2007; Thayer, Smith, Rossy, Sollers, and Friedman, 1998). These findings have been taken as evidence that the increased HRV in women reflects a compensatory response which counteracts the perseveration of negative thoughts and mood: a higher HRV is positively associated with emotion regulation and frontal cortical activity which are thought to modulate the subcortical activity involved in sustained emotional reactivity (Thayer and Lane, 2000). This compensatory response may explain why women with subclinical depression have decreased mortality (Hybels, Pieper, and Blazer, 2002).

Several limitations of this study need to be addressed. First, we only examined indices of the cardiovascular system at the cardiac level, e.g., HR and HRV, but not at

the peripheral vascular level. It remains unclear to what extent worry and cognitive problem solving have different effects on hemodynamic functioning, such as blood pressure, cardiac output, and total peripheral resistance. As several studies suggest that different mental and emotional tasks are associated with differential cardiovascular reactivity patterns (e.g., Lawler *et al.*, 2001), it is warranted to conduct more studies that manipulate worry and compare its cardio*vascular* effects with relevant control conditions, such as cognitive problem solving. Second, although changes in self reported mood could not account for cardiac differences between conditions, it is possible that these differences could be accounted for by differences in emotionality that participants were not aware of. Several studies suggest that processing of emotional information that is presented subliminally can have autonomic effects independent of the conscious experience of negative affect (e.g., Levy, Hausdorff, Hencke, and Wei, 2000). A final limitation is that a relatively small, young and healthy sample was used. Although the sample of this study seemed to represent worriers on the full severity range, it would be useful to conduct a similar study with a larger sample including older participants and / or patients suffering from GAD. Null findings always raise questions about statistical power. The current sample size was sufficient to detect small, medium, and strong effect sizes, according to Cohen (1988). It is possible that a larger sample size will yield small but significant differences between the conditions. It is quite possible that other participants than the healthy students in this study would worry about other, perhaps more severe, topics that would show stronger cardiac effects than during neutral problem solving. In addition, it is known that cardiovascular diseases, such as hypertension, develop over time, and it is possible that the immediate cardiac effects of different cognitive tasks are dissimilar when tested in an older sample.

In sum, the results suggest that although worry enhances HR and reduces HRV, these effects are not different from engaging in mental problem solving. In addition, female high trait worriers showed a compensatory HRV response when involved in cognitively demanding tasks.

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