Perseverative cognition : the impact of worry on health

Verkuil, B.

PERSEVERATIVE COGNITION

THE IMPACT OF WORRY ON HEALTH

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Perseverative cognition: Insights into the impact of worry on health
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Perseverative cognition:

The impact of worry on health

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And when the worrying starts to hurt
and the world feels like graves of dirt
Just close your eyes until
you can imagine this place, yeah, our secret space at will
'Shut your eyes' (Snow Patrol)

CONTENTS

Chapter 1

General Introduction

The perseverative cognition hypothesis

In daily life, many people ponder about things that have happened in the past or about things that might occur in the future. In contrast with animals, human beings are capable of thinking minutes, hours, months and years back and ahead. This ability comes in handy when planning one's holiday or when deciding what jobs one wants to pursue. Also, it can be a pretty enjoyable experience to remember pleasant events or to look forward to such events. Yet, this ability to think back and ahead also has a downside: it gives humans endless opportunities to keep thinking about stressful events that lie either in the past or in the future. For a majority of people worries about upcoming stressful events are indeed a common experience in daily life, also when these events never actually happen.

Frequent worries about the future do not come without costs. Since the early '80s, it has been increasingly recognized that severe, pathological worry is crucial in the onset and maintenance of anxiety and mood disorders (Watkins, 2008). More recently, pathological as well as non-pathological worry has become a growing area of interest in stress research (Brosschot, Gerin, & Thayer, 2006), the major aim of which is to explain why stressful events can make us sick. Stress research in the past fifty years had been dominated by the reactivity model of stress, stating that frequent exaggerated stress-related physiological activity *during* the experience of a stressful event is detrimental for one's health (Linden, Earle, Gerin, & Christenfeld, 1997). For example, people who show a strongly enhanced heart rate during stressful events such as an exam would be vulnerable to develop cardiovascular problems in the long term. Yet, in the last ten years a shift in paradigm could be observed towards prolonged activation models (Linden et al., 1997; Schwartz et al., 2003; Pieper & Brosschot, 2005), stating that stress-related physiological activity that is enhanced *in anticipation of* or *after* stressful events is crucial in the causal chain from stressful events to disease. This focus on the temporal aspects of the stress response was already advocated by early stress researchers (Selye, 1951), but has remained understudied for years. From a prolonged activation model point of view, the total duration of the physiological stress response, and not so much its initial strength, is the most important factor in determining whether stress affects one's health. A central venture for stress researchers is therefore to disentangle why and under what circumstances stress-related physiological activity is prolonged beyond the presence of actual stressful events. Working from within the framework of the prolonged activation model, Brosschot, Gerin and Thayer (2006) proposed that worry is a likely candidate to produce sustained stress-related physiological activity in daily life. Worry can be regarded as a recurrent or persistent cognitive representation of a stressor, termed 'perseverative cognition', and this perseverative cognition could serve as a mechanism that prolongs physiological activation due to stressor. According to the 'perseverative cognition hypothesis' perseverative thoughts, like worry, produce sustained stress-related physiological activity

which in turn leads to health problems. In other words, stressful events cannot affect one's health, unless people worry about these events. Figure 1 depicts the perseverative cognition hypothesis in a model.

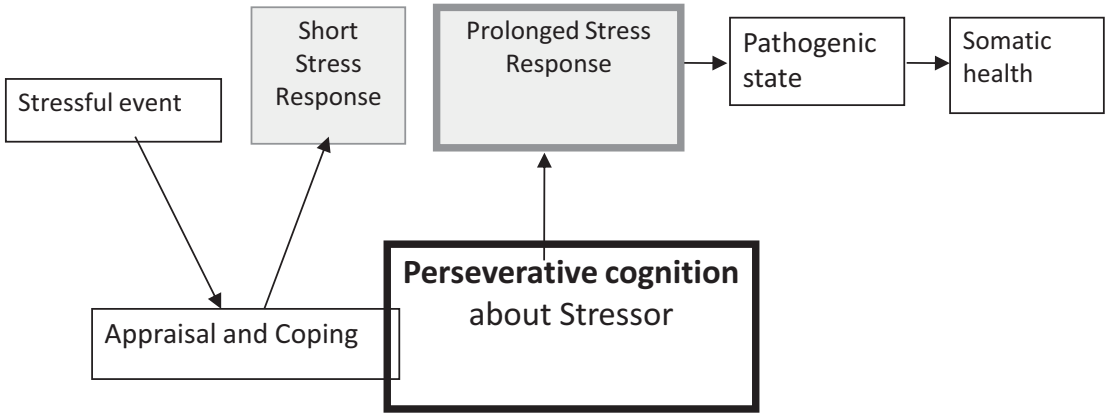

Figure 1. The perseverative cognition model. Only physiological stress responses that are prolonged by worry can lead to a pathogenic state in which people are vulnerable for the development of somatic health problems. Adapted from: Brosschot, Gerin & Thayer (2006).

Evidence for the perseverative cognition hypothesis is accumulating. Recent research shows that worry in daily life is associated with enhanced cardiac activity (in combination with stressful events having similar adverse effects as smoking; Pieper, Brosschot, van der Leeden, & Thayer, 2007; Brosschot, van Dijk, & Thayer, 2007). Furthermore, worry has been found to be predict somatic health complaints like fatigue, neck pain and headache, and this effect could be reduced by a simple worry postponement intervention (Brosschot & Van Der Doef, 2006; Jellesma, Verkuil, & Brosschot, 2009; see chapter 2 for a more thorough review). Yet, the existing studies suffer from limitations that restrict conclusions regarding several crucial aspects of the perseverative cognition model. This thesis addresses several of these aspects.

Firstly, the larger part of studies investigating the effects of worry on somatic health outcomes have relied on self-report trait-worry questionnaires; 15 out of 24 studies reviewed by Brosschot, Gerin and Thayer (2006) solely relied on trait questionnaires. Yet, the extent to which such trait questionnaires are related to the frequency and duration of worry episodes as measured with momentary assessments had not been investigated before this dissertation (but has been now,

as reported in chapter 3). Thus the significance (exact meaning) of the larger part of the current evidence therefore remains unknown. If trait worry questionnaires do not correspond with worry in daily life, the validity and reliability of most current evidence could be questioned. Therefore we used real life, momentary measures of worry to study its prospective relationship with somatic health complaints (chapter 4).

Secondly, only little is known about the relation between daily worry and health outcomes in populations vulnerable to develop stress-related health complaints, such as teachers or nurses (chapter 4), or in people already suffering from severe stress (chapter 5). It is also not known whether the potentially adverse effects of worry can be reduced in these groups. Brosschot and Van der Doef (2006) already showed that a simple worry postponement intervention could reduce the adverse effects of worry on health complaints. Worry plays a pivotal role in the development and maintenance of not only anxious and depressed mood, but likely also in somatic complaints. In a time where a significant amount of employees is suffering from work-related stress and worries (Monsterboard, 2008), and where as yet few evidence-based short and easy to administer stress management interventions are available (Jorm & Griffiths, 2006), it seems of particular interest to test whether such an intervention would also be effective in a clinical population suffering from severe work stress (chapter 5). Showing that a simple worry intervention is effective in reducing somatic complaints, as well as anxious and depressed mood, could potentially provide clinicians, like occupational physicians and general practitioners, with such an evidence-based intervention. Furthermore, it would provide evidence that the perseverative cognition hypothesis holds true in a clinical population too.

Thirdly, whereas ambulatory studies have consistently found associations between *state* (daily) worry and cardiac activity (e.g., Pieper et al., 2007; Brosschot et al., 2007), laboratory studies have yielded inconsistent results (see chapters 6 and 9). Although most studies have shown that *trait* perseverative cognition predicts slowed cardiovascular recovery after stressful events (Glynn, Christenfeld, & Gerin, 2002; Gerin, Davidson, Christenfeld, Goyal, & Schwartz, 2006; Key, Campbell, Bacon, & Gerin, 2008), several of these studies did not find an association between state worry and slowed cardiovascular recovery (Key et al., 2008; Gerin et al., 2006). One possible explanation is that in high trait worriers worrying occurs very automatically and without conscious awareness. If this is true, such implicit worry cannot be assessed via self report methods that rely on information that one can consciously reflect upon and report. According to the definition of perseverative cognition, its pathogenic ingredient is the 'cognitive representation of a stressor', which can theoretically be conscious as well as unconscious (Brosschot et al., 2006). Whereas all studies concerning the perseverative cognition hypothesis have focused on its explicit, conscious forms, no studies have addressed the possibility that implicit or unconscious forms of perseverative cognition slow down

cardiac recovery (see also chapters 2 and 6). If perseverative cognition like worry indeed occurs unconsciously and slows cardiac recovery, this would open up a new and promising venture for stress research, because a large part of cognitive processing in daily life occurs without conscious awareness. This could potentially mean that by focusing on consciously perceived stress, researchers have been focusing on the 'tip of the stress-iceberg'.

Fourthly and finally, as a consequence of worry research only being started recently and the heavy focus on trait-approaches, only very few attempts have been made to study the mechanisms via which worry exerts its detrimental influence on both mental and somatic health. In this thesis, three studies are reported that have been conducted to address these possible mechanisms (chapters 7 – 9).

Thesis outline

The main aim of this thesis was twofold. First, realized in part 1, to further test the perseverative cognition hypothesis, three studies were conducted to test whether worry predicts adverse health-related outcomes, while addressing the above mentioned limitations. In these studies, health-related outcomes were operationalized as either somatic health complaints (chapters 4 and 5) or slowed cardiac recovery (chapter 6). Health complaints and slowed cardiovascular recovery are both associated with morbidity and mortality (Sha et al., 2005; Kivimaki et al., 2006; Jae et al., 2008). The second aim of this thesis, realized in part 2, was to examine possible pathways or mechanisms that could explain or add to the findings from the first part of this thesis (chapters 7 – 9; see below for more details).

The thesis starts with a theoretical review summarizing recent evidence regarding the perseverative cognition hypothesis and providing an integrative theoretical framework on perseverative cognition (chapter 2). In chapter 3, the predictive validity of three widely used trait worry questionnaires is investigated in a large sample of university students. In chapter 4, the effects of momentary assessed stressful events and worry episodes on somatic health complaints is examined in a sample of primary and secondary school teachers. The specific prediction was tested that worry mediates the adverse health effects of stressful events. In chapter 5, the effectiveness of a simple worry postponement and disengagement intervention was tested in a sample of clinical outpatients suffering from severe work stress. In this randomized clinical trial, patients that were awaiting stress management therapy were randomly allocated to either receive the worry postponement and disengagement intervention before the onset of the stress-management therapy or one out of two control conditions, that is the registering of worry episodes or a waitlist control. By experimentally trying to reduce worry, it could be tested whether worry was causally related to somatic health complaints. Furthermore, besides testing the short term effects of this intervention,

the additive effects of this worry intervention on the stress management therapy were investigated. In chapter 6 the effects of explicit and implicit worry on cardiac recovery after a stressful event were investigated. We hypothesized that trait worry would predict slowed cardiac recovery after stress, and that this effect would be mediated by explicit and implicit state worry experienced during the recovery period following the stressful event.

Whereas the first part of this thesis is aimed at examining whether worry is related to health outcomes, the second part of this thesis is aimed at clarifying the mechanisms behind the relation between worry and health (chapters 7 and 9) and also the mechanisms behind the worry process itself (chapter 8). Each of these three studies described in this part corresponds to a study reported earlier in this thesis and targets a specific mechanism that adds to a fuller understanding of how worry affects our health, thereby extending and specifying the perseverative cognition model. Below, the aims or these studies are explained and how they correspond with the part 1 studies. First, although the perseverative cognition hypothesis states that worry can lead to somatic health complaints via prolonged physiological activity, as assumed in the study of chapter 3, an alternative pathway that bypasses the physiological route was suggested there and tested in a study reported in chapter 7. Somatic health complaints could be due to specific worries about one's health. It is possible that in people who frequently experience somatic health complaints bodily sensations trigger cognitive networks related to health, which promote selective cognitive processing and misinterpretations of these bodily sensations. In turn, highly accessible cognitive networks increase the likelihood of reporting somatic health complaints by causing worries about these complaints (Brown, 2004; Brosschot, 2002). To address this issue, we conducted a study in which we examined the extent to which common health complaints were associated with health worry, and with selective cognitive processing of health related information. Second, an implicit assumption in the studies using the worry postponement intervention (Brosschot and Van der Doef, 2006; chapter 5) or mindfulness based intervention aimed to reduce worry, is that worry episodes are prolonged due to difficulties in disengaging attention from threatening information. Indeed, recent work suggests that people who suffer from anxiety and dysphoria especially have trouble with *disengaging* attention from threatening information (Fox, Russo, Bowles, & Dutton, 2001; Fox, Russo, & Dutton, 2002; Goeleven, De Raedt, Baert, & Koster, 2006; Koster, Crombez, Verschuere, Van Damme, & Wiersema, 2006). In chapter 8, it was examined whether this was specifically due to heightened levels of worry. This would provide us with more clues on how to treat worry and provide evidence that interventions indeed do well by focusing especially on the engagement-disengagement dimension in the worry process. Third, concerning the cardiovascular effects of worry (chapter 6), it is not clear which elements of worry actually cause these cardiovascular effects. It might be that this is due to heightened levels of negative emotions, or it might be merely just 'thinking hard', that is, mental load

during cognitive problem solving. We therefore conducted a study in which heart rate (HR) and heart rate variability (HRV) were compared within subjects during induced worrying, problem solving concerning issues that were not personally relevant and relaxation (chapter 8). If mere mental load is responsible for - at least a part of - the physiological effects of worry, this might suggest that long term health effects of worry might be due to the prolonged mental load aspects of worry rather than to its emotional aspects, even though the latter is the most commonly held belief.

Figure 2 represents a more detailed model of how perseverative cognition is hypothesized to influence physiological and subjective health. Stressful events can lead to perseverative cognition, (operationalized as worry in the current thesis), as well as to negative affective responses and changes in information processing ('enhanced processing of threat'), of which the latter could be considered as an unconscious form of perseverative cognition. These three processes interact and enhance each other (chapters 2 and 8). Concerning the content of worry, worry is hypothesized to lead to somatic health complaints via both prolonged stress responses and via specific health worry (chapter 7). Furthermore, worry consists of mental effort and increases negative affect (anxiety), both of which are assumed to be associated with increased stress-related physiological activity (chapter 9).

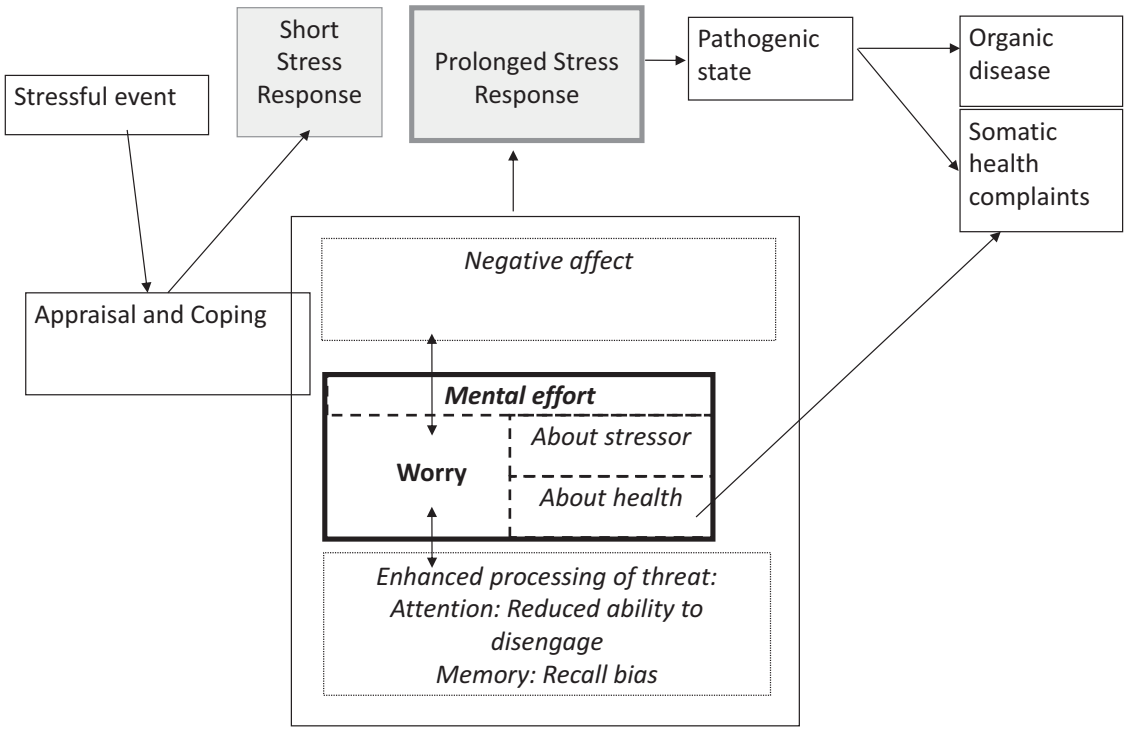

***Figure 2*.** The extended perseverative cognition model.

Summarizing, the studies in this thesis are aimed at showing the effects of worry on several health outcomes, and attempt to address the mechanisms by which these effects are accomplished. These latter studies should be viewed as ‘first attempts’ and were meant to be hypothesis generating and inspiring future research rather than giving definite answers. Expanding knowledge on whether and how worry affects health might bring us a step further in disentangling how ‘stress’ can ultimately lead to ‘disease’.

Chapter 2

Perseverative cognition, psychopathology and somatic health

Bart Verkuil, Jos F. Brosschot, Winifred A. Gebhardt & Julian F. Thayer

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General introduction: the perseverative cognition hypothesis

The idea that stress can make us sick is not new. A long tradition of research into the effects of stressful events has made clear that stressful events can lead to mental (Hammen, 2005) as well as somatic health problems (Cohen & Williamson, 1991; Rozanski, Blumenthal, & Kaplan, 1999). This is especially the case when stressful circumstances are chronic, such as when providing care for a chronically ill spouse (Schulz, 2004; Vitaliano, 2003). It has also been well documented that the way people perceive and appraise events codetermines whether a situation or event is experienced as stressful and that stressful appraisals in turn initiate and activate the physiological stress reaction (Lazarus, 1991). Thus, stress can make us sick and that is in part due to what we think about stressful events. Yet, stress researchers have confined their attention mainly to what we think *during* these events and how that leads to enhanced physiological stress reactions *during* a stressful situation. Little attention has been paid to how these thoughts, when they persevere after (or before, in anticipation of) stressful events, can prolong the stress response. Yet, as we will argue in this chapter, it is the prolongation of stress responses, and not so much acute stress responses that form a crucial link between stressors and later mental (McEwen, 2003; Thayer & Lane, 2000) and somatic problems (Selye, 1951; Ursin & Eriksen, 2004; Linden, Earle, Gerin, & Christenfeld, 1997; Brosschot, Pieper, & Thayer, 2005; Brosschot, Gerin, & Thayer, 2006). Thus, as yet scientists have hardly addressed the important issue of *when, how often* and *how long* we think about stressful events and how 'perseverative thinking' about stressors might prolong the stress response. In psychopathology research though, during the past decade, perseverative cognitive processes have received increasing attention, and have been recognized as core etiological factors in the maintenance of several mental disorders, such as mood and anxiety disorders. We have recently hypothesized perseverative cognition as the mediator of the effects of stressors on not only mental but also somatic illness, because it prolongs not only psychological but also physiological responses to stressors. Brosschot Gerin & Thayer (2006) have stated that stress can only lead to disease when physiological stress responses are prolonged by perseverative cognition. Perseverative cognition refers to mental representations of the stressful events, such as worrisome anticipation before or ruminative thinking after the stressful events. Just as stressful cognitions during stressful events shape the concomitant physiological and emotional stress reaction, perseveration of these representations is hypothesized to prolong this physiological and emotional activity, thereby adding to the total time that stressors can have an impact on our mental and somatic well-being.

Several reasons can be forwarded why insight into how perseverative thoughts prolong physiological activity after or before a stressful event is likely to improve our understanding of how stress influences our health. First, the prolonged cognitive effects of stressful events seem to outlast to a great extent the duration of the stressful events themselves. For example, Gilboa and Revelle

(1994) found that even minor negative daily events can evoke worrisome thoughts that might last up to 11 hours after the stressful event. Second, many, if not most, stress responses are due to stressors that have not yet occurred or will never occur, but are anticipated (i.e. feared) nevertheless. In other words, their anticipation in the form of worrying is in fact their only manifestation. Anticipated stressors - as opposed to actually occurring stressors - is such an extremely common form of stress-related cognition that it is quite surprising that is has received such little attention in stress science. For example, in a large scale survey amongst Dutch employees, one third reported to experience sleeping difficulties every Sunday night due to worries about the upcoming work week (Monsterboard, 2008). In addition, a majority of employees have difficulties in relaxing after work because they keep on worrying about their next stressful workday. Third, studies on the temporal aspects of emotions show that initial emotional reactivity during stressful events is only weakly related to the duration of the emotional response (6 – 14% shared variance) and that even positive changes in the stressful event, such as when a conflict between an employee and their boss is resolved, are only weakly associated with the duration of the emotional responses to it (6% shared variance; Sonnemans & Frijda, 1994). In conclusion then, it seems clear that cognitive, emotional and physiological reactivity during real life stressful events is only one side of the coin, and that investigating the total duration of stress related psychological and physiological activity, even beyond the presence of the real life stressor is of high importance.

In this chapter we will review recent evidence for the perseverative cognition hypothesis, including its effects on mental as well as somatic health. In it we specifically focus on the real life dynamics of perseverative cognition. Furthermore, we will provide a self regulation perspective on perseverative cognition clarifying the notion that perseverative cognition is in fact the default response to stressful situations, a response which is successfully inhibited by most healthy people. We will illustrate how goal directed cognition can lead to pathological perseverative cognition as seen in mood and anxiety disorders. Finally, we will discuss the neurophysiological underpinnings of perseverative cognition. First, the concept of perseverative cognition itself, its manifestation and prevalence will be introduced.

The concept and prevalence of perseverative cognition

Perseverative cognition is defined as "*the repeated or chronic activation of the cognitive representation of one or more psychological stressors*" (Brosschot et al., 2006). A stressor is defined as a situation involving potential harm, without or with low perceived control, that is a *threat* to the psychobiological integrity of oneself or to the attainment of one's higher order goals. Although many terms are used by psychologists to refer to perseverative thinking, such as "rumination", "repetitive thinking", "worry" or "depressive rumination", there are several reasons why we propose to use the

term ‘perseverative cognition’ (Brosschot, van Dijk, & Thayer, 2002; Brosschot et al., 2006) within the framework of psychobiological stress research. These reasons are that (1) related terms involve either too broad or too narrow definitions to be used in a stress model, (2) these terms lack an emphasis on the importance of the perseverative process itself for health, and (3) our definition of perseverative cognition as a “mental representation of psychological stressors" allows to include alternative cognitive processes, for example automatic or unconscious cognitive processes, that may have substantial health-relevant effects. Below, we will discuss each of these arguments in more detail.

First, several concepts involving conscious perseverative thinking exist that are either too broadly defined, such as rumination, defined as “*a class of conscious thoughts that revolve around an instrumental theme and that recur in the absence of immediate environmental demands requiring the thoughts*” (Martin & Tesser, 1996, p. 7) and repetitive thinking, defined as the “*process of thinking attentively, repetitively or frequently about one’s self and one’s world*” (Segerstrom, Stanton, Alden, & Shortridge, 2003, p. 909). Both of these terms can also refer to positive thoughts, whereas perseverative cognition deals with negative thoughts that are specifically concerned with stressors. Other terms are too specifically or narrowly defined, such as depressive rumination: “*behaviors and thoughts that focus one’s attention on one’s depressive symptoms and on the implications of these symptoms*” ((Nolen-Hoeksema, 1991p. 569) and worry: “*a chain of thoughts and images, negatively affect-laden and relatively uncontrollable. The worry process represents an attempt to engage in mental problem-solving on an issue whose outcome is uncertain but contains the possibility of one or more negative outcomes. Consequently, worry relates closely to fear process.*” (Borkovec, Robinson, Pruzinsky, & DePree, 1983, p. 10). Worry as well as depressive rumination deal with stressors, being either future stressors or ones depressive symptoms, and are the most thoroughly investigated types of perseverative cognition. Perseverative cognition encompasses worry as well as rumination, and also other related concepts, such as intrusive thoughts, and negative flashbacks.

Second, the term ‘perseverative' makes clear that the pathological ingredient of mentally representing stressors is their *perseveration*, that is, the *duration of exposure* of the organism to the (cognitive representation of) the stressor (threat). As argued above, the duration of the stress response is its toxic element, for mental as well as for somatic health. Only persistent emotional or physiological responses can lead to problems in either mental or somatic health.

Third, perseverative cognition not only refers to conscious thoughts about stressors, but also to prolonged *automatic* processing of stressor related information, as reflected in for example attentional hypervigilance or enhanced memory retrieval of stress related information (e.g., Rothermund, 2003). The perseverative cognition hypothesis is therefore not strictly limited to conscious thinking about stressors, but also accommodates automatic, or unconscious, stress-related

cognition. Since the greater part of any cognitive processing appears to operate without awareness (Bargh & Ferguson, 2000), a considerable part of perseverative cognition is likely to be unconscious as well. Even minor stressful events cause people to persistently scan the environment for threat and this attentional hypervigilance is only possible when a mental representation, or 'cognition' concerning threat is still present (Wells & Matthews, 1996). This very basic representation of threat is fundamental for survival, and it occurs automatically and without conscious awareness. Although a recent study showed that emotional information reaches conscious access at a lower threshold than neutral information (Gaillard et al., 2006), it is likely that people are not aware of most of their stress-related cognitive processes, as they are not aware of most of the cognitive processes ongoing in daily life.

Perseverative cognition, psychopathology and somatic health problems

Below we provide an overview of the mental and somatic problems that have been associated with perseverative cognition.

Psychopathology: Research into the mental health effects of worry started in the early 1980's with the finding that frequent nighttime worrying is a predictor of the onset and maintenance of insomnia (Borkovec, 1982). Nowadays, perseverative thoughts like worry and rumination are recognized as fundamental characteristics of several psychopathological conditions (Watkins, 2008). For example, worry is a central feature of generalized anxiety disorder (GAD; Borkovec, 1998; Wells & Matthews, 1996) and depressive rumination is recognized as a central feature of depression (Nolen-Hoeksema, 1991). Moreover, perseverative cognition is found in hypochondriasis (Looper & Kirmayer, 2001), social phobia (Abbott & Rapee, 2004) and post traumatic stress disorder (PTSD; Holeva, Tarrier & Wells, 2001).

Perseverative cognition is not just a symptom or epiphenomenon of these pathological conditions. For example, experimental studies show that perseverative cognition is causally related to negative mood. In addition, in prospective studies it has been observed that perseverative thoughts predict the onset and maintenance of anxious and depressed mood (for a review see: Watkins, 2008). Yet, it is unclear at what levels of intensity, i.e., at which frequency and duration, perseverative thinking can be regarded as pathological. Although most research has focused on clinical conditions, perseverative cognitions are not only experienced by people suffering from psychological disorders, but also by healthy people. Recent diary studies among several populations (undergraduates, teachers, and a community sample) showed that, on average, the total duration of worry episodes was 30 minutes a day, while only a minor part (< 4%) of the participants reported no worry episodes at all (Brosschot, van Dijk, & Thayer, 2007; Pieper, Brosschot, van der Leeden, &

Thayer, 2007; Verkuil, Brosschot, & Thayer, 2007b). Unfortunately, ambulatory studies are not regularly conducted with clinical populations and therefore little is known about the exact frequency and duration of perseverative thoughts in these populations. In one of our own studies we found that whereas clinical outpatients suffering from burnout do not seem to worry more than healthy people during the daytime, but that they do worry more during the nighttime (approximately 30 minutes; Verkuil, Brosschot, Korrelboom, Reul-Verlaan & Thayer, submitted). This is in line with the idea that burnout patients have difficulties with disengaging from work. In addition, one ambulatory study conducted with GAD patients showed that they worry approximately 310 minutes per day suggesting that it places a great burden on their daily lives (Dupuy, Beaudoin, Rheaume, Ladouceur, & Dugas, 2001). Indeed, GAD patients even worry about the possible damaging consequences of the worrisome thoughts themselves that they experience (called meta-worry), which adds to the total time these people are in fact worrying. This meta-worry is often taken as a signal that the worry has become pathological and that the person's condition warrants treatment.

Automatic, unconscious or other 'low-level' stress- or threat-related cognitive processes have been associated with mental health in a plethora of studies during the past 30 years (Williams, Watts, MacLeod, & Mathews, 1997; Mineka, 1992). Thus, attentional hypervigilance, for example operationalized as selective attention for threat related stimuli, as well as automatic vigilance, for example operationalized as enhanced memory for threatening stimuli, have been amply documented to play a role in mental health problems. Yet, very few of these studies have actually sought evidence that such forms of perseverative cognition preceded or caused the disorder. A thrilling recent development in experimental psychopathology is that researchers are now attempting to address these automatic processes in order to develop new therapeutic tools. Evidence is now growing that reversing cognitive biases by 'attentional retraining' procedures can reduce symptoms of mood and anxiety disorders suggesting that these biases may have a causal role in the onset and maintenance of such disorders (MacLeod, Rutherford, Campbell, Ebsworthy, & Holker, 2002; Hazen, Vasey, & Schmidt, 2009; Wilson, MacLeod, Mathews, & Rutherford, 2006).

Somatic health: Importantly, for *somatic* outcomes, the perseverative cognition hypothesis is not restricted to *pathological* worry. It is likely that somatic problems can be caused by much less severe, or intense, levels of worry, if only maintained long enough. One of the earliest findings suggesting that perseverative cognition plays an important role in the link between stress and somatic disease was that among elderly men who had a myocardial infarction, those who frequently worried were at heightened risk for experiencing a second myocardial infarction (Kubzansky et al., 1996). In addition, it has been shown that during experimentally induced worry as well as during worry in daily life cardiovascular activity is increased (Lyonfields, Borkovec, & Thayer, 1995; Thayer, Friedman, &

Borkovec, 1996; Verkuil, Brosschot, Borkovec, & Thayer, in press; Pieper et al., 2007). Furthermore, a recent review concluded that perseverative cognition is associated with enhanced activation in endocrine and immune systems (see Brosschot et al., 2006). Finally, more recently it was shown that worry is associated with lowered levels of antibody titers in caregivers (Segerstrom, 2008) and that ruminating after emotional events is linked to enhanced levels of cortisol, in daily life (McCullough, Orsulak, Brandon, & Akers, 2007), as well as in the laboratory (Zoccola, Dickerson, & Zaldivar, 2008).

These findings are in line with the perseverative cognition hypothesis (Brosschot et al., 2006), stating that perseverative cognition prolongs physiological activity which, in turn, leads to a pathogenic state in which one is more vulnerable to developing a somatic disease. However, the discussed studies have mainly focussed on physiological activity, and not so much on disease outcomes. Few studies have suggested that perseverative cognition can indeed lead to somatic disease. Recently, a study by Holman et al. showed that in US citizens suffering from acute stress after the 9/11 attacks, ongoing worries about terrorism predicted cardiovascular health problems up to three years after the attacks (Holman et al., 2008). More indirect evidence for the impact of perseverative cognition on somatic disease is provided by the fact that risk for cardiovascular problems is heightened in people suffering from anxiety disorders and depression (e.g., Wulsin, Vaillant, & Wells, 1999), which are characterized by high perseverative cognition. In addition, studies focusing on work stress have found that reduced mental recovery, or reduced 'unwinding' after work is predictive of cardiovascular mortality (van Amelsvoort, Kant, Bultmann, & Swaen, 2003; Kivimaki et al., 2006). Furthermore, at least two studies among patients suffering from somatic health problems have suggested that perseverative cognition might be an important mediator of the effects of some somatic treatments. In one study it was found that in patients awaiting surgery for their hernia, greater worry about the surgery predicted lower levels of immune cells at the wound site, greater pain, poorer self-rated recovery and a longer recovery time (Broadbent, Petrie, Alley, & Booth, 2003). Furthermore, heightened levels of trait worry in psoriasis patients were predictive of a slowed recovery from photochemotherapy (Fortune et al., 2003). Still, more evidence is needed to test the perseverative cognition hypothesis with respect to disease outcomes. An important venture for future research is to conduct more prospective studies examining whether perseverative cognition is indeed the pathogenic link between stressful events and the onset and maintenance of somatic disease.

It is a highly exciting idea that automatic or unconscious perseverative cognition can cause prolonged stress-related physiological activity, and in the long term even somatic disease. To date, no study has directly addressed the possibility that automatic vigilance or other forms of unconscious perseverative cognition have physiological effects. Three exceptions are studies showing relatively subtle effects of subliminal emotional stimulation on brain activity (Morris, Öhman, & Dolan, 1999),

startle reflex (Ruiz-Padial & Vila, 2007) and skin conductance (Öhman & Mineka, 2001). Moreover, two of our own recent studies have yielded some indirect evidence for the somatic effects of unconscious perseverative cognition. Firstly, in an ambulatory study we found - quite unexpectedly - that worry episodes were not only associated with enhanced heart activity, but that the worry episodes *themselves* also had prolonged cardiac effects, until up to two hours *after* the worry episode had ended (Pieper et al., 2007). This effect was independent of ongoing worry, emotions, health behaviors and physical activity, and therefore we concluded that it must have been due to some unconscious, or at least not verbally reportable form of worry. It is important to note, that this finding can not be due to 'just slow recovery', since such a mild high cardiac increase when caused by a non-emotional stressor such as physical effort normally recovers within a matter of minutes. Secondly, in another ambulatory study we found that conscious daytime worrying predicted heightened heart rate during the subsequent night (Brosschot, van Dijk & Thayer, 2007). During sleep people obviously do not worry consciously, but the hypervigilance that is evoked by stressful events might be prolonged into the night. This was also demonstrated by Hall and colleagues (2004), who found that participants who had to give a speech in the morning, showed decreased levels of parasympathetic activity (low heart rate variability) during the following non-rapid eye movement and rapid eye movement sleep periods (Hall et al., 2004). Although a lot has to be discovered on what exactly happens cognitively during sleep, studies with rats and humans have shown that daytime neuronal activity seems to be repeated or 'replayed' just before or during sleep (Skaggs & McNaughton, 1996; Stickgold, Malia, Maguire, Roddenberry, & O'Connor, 2000). Furthermore, sleep promotes procedural learning and consolidation of memories (Walker & Stickgold, 2004). It is therefore not unlikely that daytime stressful events are mentally represented during sleep in one way or another and that this interferes with physiological recovery during sleep. It is obvious that this will prolong the total amount of physiological 'wear and tear' that stressful events have on the human body, since sleep covers about one third of our lives. Sleep is generally considered to be a basically stress-free recovery period, and therefore the most important restorative period. Future studies are warranted to investigate to what extent and how stressful events and hypervigilance influence physiological recovery during sleep.

Subjective Somatic Health. Ambulatory studies by our group have shown that in several non-clinical populations (students, high school teachers; worry duration of 30 minutes per day on average) non-clinical levels of worry were positively and prospectively associated with subjective health complaints like fatigue, headache and lower backpain (Brosschot & Van Der Doef, 2006). A simple worry intervention, consisting of postponing worry to a daily 30-minute worry period, reduced these health complaints. The effects of the intervention were not restricted to certain types

of complaints, but pertained to a range of different complaints (e.g. cough, palpitations, neck pain). Interestingly, worry *duration* and not or not so much worry *frequency* predicted increases in health complaints and mediated the effect of the worry intervention (Brosschot & Van der Doef, 2006), which is in line with the perseverative cognition hypothesis. Further evidence for the effects of perseverative cognition on subjective health comes from another ambulatory study in which it was found that people who reported to be ruminating a lot about conflicting goals reported heightened levels of somatic complaints (Emmons & King, 1988).

Perseverative cognition specifically related to health problems, that is, health worry or illness worry seems to be of specific importance for subjective somatic health complaints. Being – often even severe - stressors themselves, somatic health problems often give rise to worries. For example, chronic pain patients have been found to worry about pain for 20 minutes per day on average, compared to 17 minutes for non-pain-related topics (Eccleston, Crombez, Aldrich, & Stannard, 2001). Except for having physiological effects just as other worries, these health worries may affect subjective somatic health through an alternative pathway: Enhanced worrisome thinking about somatic signals might promote complaining about them. Indeed, health worry has been found to predict the occurrence of health complaints (Kaptein et al., 2005; Petrie et al., 2005; Devoulyte & Sullivan, 2003) and is associated with increases in pain (Turner, Mancl, & Aaron, 2004). Furthermore, not surprisingly, health worry has been associated with increased doctor consulting (Hay, Buckley, & Ostroff, 2005) and with intensive health care utilization (Looper & Kirmayer, 2001). One mechanism that may underlie these effects is that in people who worry excessively about illness bodily sensations are more likely to trigger illness-related cognitive networks which promote selective cognitive processing and misinterpretations of these bodily sensations as symptoms of illness (Brosschot, 2002; Brown, 2004). In turn, such worries might lower the threshold for actually complaining about these presumed symptoms of illness. Indeed, we recently found that the association between illness-related cognitive bias (increased recall of illness information) and health complaints was mediated by illness worry (Verkuil, Brosschot, & Thayer, 2007a; Brosschot, 2002).

In short, perseverative cognition, even at non-clinical levels, might influence somatic health either via prolonged activity in endocrine, immune and cardiovascular systems or, in the case of specific health worry, via enhanced processing of illness related information.

A self regulation perspective on perseverative cognition

The stress response, and therefore its prolongation via perseverative cognition, can be basically understood as the (default) response to threats to the attainment of a person's goals. The detection of potential threats to one's goals and of signs of failure in attaining these goals is a continuous process. Environmental stimuli are first quickly and briefly – and largely automatically - scanned for

their threat value (i.e. 'fast route', LeDoux, 2000) and once something has been detected that could be a possible threat to one's goals, it immediately leads to an rapid and indiscriminate defensive response, even if it eventually turns out to be only a novel or ambiguous stimulus (LeDoux, 2000; Thayer & Lane, 2000). This defensive response, or motivational state, consists of changes on several levels in the organism: Cognitive changes (attentional hypervigilance and 'higher' perseverative cognition), physiological changes (e.g. increased autonomic nervous activity and release of stress hormones) and behavioral changes (avoidance or approach behavior), known in psychophysiology as the 'defense response' (Lang, 1995). The duration of this defensive response depends on how quickly the system ascertains the safety of the situation. This shutting down of the response because of safety is dependent on a more deliberate cortical processing ('slow route'; LeDoux, 2000) of the potential threat. Thus, the *default* response to any potential threat is this immediate defensive response, served by subcortical networks in the brain, which, under normal circumstances, is under chronic inhibitory control by the prefrontal cortex (Amat et al., 2005; Thayer & Sternberg, 2006; see below for more neurophysiological underpinnings). Interestingly, this default response to threat has been found to be enhanced in females, who, when presented with an equal amount of threat information, showed more persistent activation of subcortical structures in the brain compared to males (Williams et al., 2005). From an evolutionary perspective it makes sense that this defense system initially ‘errs on the side of caution’ by often responding initially to novel or ambiguous neutral information as if it was threatening, and subsequently either continues or stops after a more deliberate appraisal process has taken place. As a consequence when no safety signals can be provided the stress response is prolonged. This seems to be the case in conditions such as anxiety disorders and during chronically stressful situations. There is some evidence that at least in some people, especially high worriers, this is due to a failure to recognize these safety signals. We will return to this possible explanation later in the chapter.

In the remainder of this section the concept of perseverative cognition will be regarded from a self-regulation perspective. More specifically, we will argue that perseverative cognition can be conceptualized as the perseveration of mental representations of goal discrepancies (hypervigilance and 'higher' perseverative thinking), and that perseverative cognition is the cognitive part of the default response to goal discrepancies. We will further contend that this response is enhanced in high worriers because: (1) they have psychological characteristics that make them likely to be more motivated to reduce goal discrepancies; (2) perseverative cognition is reinforced in these people in several possible ways; (3) their perseverative cognition is due to a deficiency in recognizing signals of safety which causes them to respond with the default perseverative cognition response not only when faced with threat, but to any situation containing ambiguity or novelty.

Why people perseverate: perseverative cognition as the default response to potential threat to goal attainment

The essence of perseverative cognition is to keep attention directed towards ones goals, to anticipate threats to goal attainment and, in the case of rumination, to protect oneself from the recurrence of mistakes made in the past (for related theoretical accounts see: Wells & Matthews, 1996; Martin & Tesser, 1996). It has been consistently shown that intensive engagement in the pursuit of a goal has effects on information processing (Johnson, Chang, & Lord, 2006). Goal engagement leads to alterations in pre-attentive processes and the content of thoughts and dreams, and it enhances the perception and processing of goal related stimuli, (Klinger, 1975).

In terms of self-regulation, a psychological stress response arises when people experience a discrepancy between an expected state and the actual state that they are in (Ursin & Eriksen, 2004). Expected states can either be desired states in the present ('standards', such as being healthy or having enough money to buy food) as well as desired states that lie in the future ('goals', such as becoming a successful employee (Boldero & Francis, 2002). In daily life, one's actual state is continuously monitored, occurring mostly automatically, and compared to these desired states, or, reference values. Discrepancies can be detected between a standard (e.g., being healthy) and one's actual state (e.g., having received a diagnosis of cancer) which will result in the stress response. In the case of goals that lie in the future it is not so much the discrepancy that gives rise to the stress response, as setting a goal automatically implies that one hasn't attained this goal. In this case it is the perceived rate or speed with which one is making progress towards attaining this desired state that is compared to one's actual state (Carver & Scheier, 1990). When this speed is perceived to be too low this will also result in the stress response. In the remainder of this chapter we will refer to both types of discrepancies as goal discrepancies.

When people are confronted with such goal discrepancies, information concerning these discrepancies tends to stay activated in the brain, whereas when goals are attained information about goal discrepancies is inhibited. Again, this suggests that the default response to perceiving a goal discrepancy is cognitive *perseveration*, in the sense that this response is simply maintained as long as the discrepancy is present. This was first shown by Zeigarnik more than 80 years ago, who showed that memory for interrupted tasks is better than for completed tasks (Zeigarnik, 1927). Perseveration of goal directed cognition has been found in several other studies (Marsh, Hicks, & Bink, 1998; Goschke & Kuhl, 1993; Rothermund, 2003). For example, Rothermund (2003) found that failure on a cognitive task was associated with slowed responses in a dual tasking experiment when participants were presented with failure related words, indicative of hypervigilance after failure. In another study, participants who were made to believe that they had failed on an intelligence task reacted faster to concern-relevant information on a lexical decision task (Koole, Smeets, van

Knippenberg, & Dijksterhuis, 1999). In line with these findings on hypervigilance, conscious perseverative cognition has also been found to be associated with experiencing discrepancy, for example between one's actual self and one's ideal self (Roelofs et al., 2007; Jones, Papadakis, Hogan, & Strauman, 2009).

Most studies have taken place in laboratory settings and have measured this perseveration of stressor related cognition immediately after the goal frustration (e.g., Rothermund, 2003) or after six minutes (Koole et al. 1999). Thus, it remains to be established how long this initial perseveration of stressor related cognition lasts. There are some interesting clues however. A study by Zadro et al. suggests that this perseveration can last much longer after a stressful event (Zadro, Boland, & Richardson, 2006). In this study participants that had been socially excluded from an online ball tossing game after 45 minutes still showed a bias towards interpreting ambiguous social situations in a more threatening manner.

Psychological moderators of perseverative cognition duration

In the next sections we will discuss how perseverative cognition can get enhanced, thereby foremost discussing studies that have been conducted with chronic (trait) worriers.

(1) Goal commitment

Although hypervigilance doesn't have to be pathological per se, several authors have proposed that it is the fundamental process that underlies clinical worry and rumination (Martin & Tesser, 1996; Nolen-Hoeksema, 2000). Indeed, most anxiety- and mood disorders are characterized by extreme hypervigilance and perseverative thinking. What determines difficulties to stop perseverating, or – to the same end - stimulates its continuation, to the extent that it starts to have health consequences?

The initial duration of perseverative cognition after one has encountered a threat to goal attainment depends on how strong one is committed to attaining the goal, which differs between individuals and between situations. According to several authors (Shah & Higgins, 1997; Feather, 1963) the strength of goal commitment is a function of the interaction between (1) the importance or value that people attach to their goals, and (2) the expectancy that one can either cope or not cope with the goal discrepancy and is either still able to attain the goal or not any more: both expectancies appear to produce perseverative cognition. Here we will review evidence that shows that high worriers are likely to be more committed to their goals when confronted with threats to attainment.

a) Value of the goals

Several studies have found that the duration of hypervigilance is a function of *higher level goals*. For example, Koole et al. (1999) showed that hypervigilance persisted longer after failure on a task when people believed that completing this task successfully was very important for one of their higher order goals, for example obtaining a good job later in life, in contrast when people had the opportunity to scale down the importance of this task by focusing on other goals. In addition, in a study by Magee et al. (2003) it was shown that women who were over-invested in becoming a parent after recurrent miscarriage had more negative thoughts about the future than women who had other goals to focus on than becoming a parent (Magee, MacLeod, Tata, & Regan, 2003). Furthermore, the tendency to link the (non-) attainment of lower level goals to the (non-)attainment of higher level goals has been related to rumination and depression (Mcintosh, Harlow, & Martin, 1995). In one study we also found that in a sample of single females higher commitment to the goal of finding a partner was associated with higher rumination about not having attained this goal (Gebhardt, Massey, van der Doef, Verhoeven & Verkuil, 2007). Several other studies have shown that perfectionism, the tendency to strive after high level self-set goals that are higher than one's current performance level, is associated with the tendency to ruminate (e.g., O'Connor, O'Connor, & Marshal, 2007).

b) Expectancy of goal attainment: negative outcome expectancies

A second important factor that influences the initial duration of goal directed cognition is the expectancy of the outcome of the stressful event, or goal discrepancy. In their Cognitive Activation model of Stress, Ursin and Eriksen (2004) distinguish three kinds of outcome expectancies that are associated with different kinds of responses: positive outcome expectancies (coping), negative outcome expectancies (hopelessness) and no outcome expectancies (helplessness). As perseverative cognition is the default response to stress, it will arise when people hold negative or no outcome expectancies. Indeed, worry was found to be associated with doubts concerning ones problem-solving skills and the tendency to be pessimistic about the outcome (Robichaud & Dugas, 2005). Likewise, people with low self-esteem are more prone to ruminate (Wood & Dodgson, 1996).

(2) Reinforcement of perseverative cognition: coping

Although people who frequently worry or ruminate might lack confidence in general coping skills, they paradoxically report to be very motivated to use perseverative cognition as a strategy to cope with goal discrepancies. For example, Szabó and Lovibond (2002) asked students to keep a log of their worries for a week and they found that almost 50% of the reported worries consisted of problem solving attempts. Furthermore, cross-sectional studies have shown that people who

frequently worry think that "worrying helps solving problems" or that "ruminating about the problem will help gaining insight" (Roelofs et al., 2007; Papageorgiou & Wells, 2001). In addition, people suffering from GAD report that worry serves as a distraction from more emotionally laden topics (Borkovec & Roemer, 1995), suggesting that in some people worry might serve to cognitively avoid intense negative emotions. Both the motivated use of perseverative cognition as a problem solving strategy and the use of perseverative cognition as a cognitive-avoidance strategy have been proposed to be reinforcing perseverative cognition.

Despite many worriers' beliefs that that worrying is helpful in solving one's problems, research has shown that worry and rumination are ineffective strategies to cope with stressful situations, yielding only more perseverative cognition as a result. First, worry and rumination are characterized by an abstract way of thinking about problems, and "abstract models are unlikely to lead to concrete actions" (cited from: Borkovec, Ray, & Stöber, 1998, p. 566). For example, in the case of rumination, Watkins and Baracaia (2002) found that depressed patients who were led to ask themselves abstract, ruminative problem solving questions ('why do I feel this way?') in a problem solving task came up with less relevant solutions than depressed patients who were led to ask themselves concrete, process focused questions ('how am I deciding what to do next?'). This abstract way of thinking might be due to the fact that trait worriers link the frustration of lower order goals to the frustration of higher order, or abstract goals, and therefore might focus more on solving abstract problems. Second, even if worrying leads to concrete solutions, worriers are not highly likely to come into action and implement their solutions. Worriers have less confidence in their problem solving skills (Davey, 1994), have elevated needs for evidence that a given solution will work (Tallis, Eysenck, & Mathews, 1991) and try to come up with as many solutions as possible before trying out these solutions (Startup & Davey, 2003; Davey, 2006). Behaviorally implementing a solution is also difficult when the problems that people are worrying about have already happened or might happen in the future. Additionally, worry also seems to become reinforced as the worst case scenarios that people worry about almost never happen, which might lead to the superstitious belief worry has been a successful coping strategy (Borkovec et al., 1998).

In sum, although worry itself might be appraised by the individual as helpful, enhancing one's positive outcome expectancy about worry, negative outcome expectancies will likely persist as no concrete action will be taken to reduce or remove the current or future threat, leading to a vicious cycle in which possibly threatening events are coped with by worrying. Furthermore, although the discussed studies have mainly focused on the outcome expectancies associated with *conscious* perseverative cognition, unconscious perseverative cognition (threat related hypervigilance or cognitive bias) is suggested to be part of a worrisome coping style (Wells & Matthews, 1996) and is likely to be subject to the same reinforcing factors. Although the authors are not aware of studies

showing operant conditioning of for example threat-related cognitive biases, indirect evidence for the effects of outcome expectancies on automatic processing comes from a recent study showing that expectancies of reward can modulate saccadic eye movements (Milstein & Dorris, 2007).

Another way in which perseverative cognition is reinforced has been put forward by Borkovec et al. (1998). In studies with non-clinical as well as clinical populations he demonstrated that worry might be a cognitive avoidance response to threat. High worriers are thought to have learned to use worry as an emotion regulation strategy because worry suppresses somatic anxiety, due to the verbal nature of worrying (Borkovec, Lyonfields, Wiser, & Deihl, 1993; Borkovec & Hu, 1990). Indeed, threatening thoughts yield smaller cardiovascular responses than threatening images about the same material (Vrana, Cuthbert, & Lang, 1986). In addition, worry, in contrast to relaxation, has been associated with blunted cardiovascular responses to threatening imagery (Borkovec & Hu, 1990). Furthermore, worry is likely to bias information processing away from threatening images. Several studies have shown that verbally memorizing information impairs the retrieval of the visual memory of this material, called 'verbal overshadowing' (Schooler & Engstler-School, 1990). Importantly, suppressing, blunting and 'verbal overshadowing' result in a reduction of the total time that people are exposed to aversive, possibly traumatic, images and this is thought to negatively reinforce the use of worry as an emotion regulation strategy. Although this might be adaptive in the short term, persistently avoiding threatening information and its associated somatic arousal, by relying on worrisome thought, interferes with the integration and extinction of threatening material in memory (Foa & Kozak, 1986). It increases the risk that threatening information is repetitively retrieved from memory (for example in the form of intrusive thoughts; Holmes, Brewin, & Hennessy, 2004) and warrants further use of worry.

Although most studies have focused on the avoidance function of worry, it is likely that rumination has the same function, which is also a verbal and abstract thinking style. Furthermore, it is unknown whether this avoidance function also pertains to unconscious perseverative cognition, and to date it is unclear to what extent unconscious mental representations consists of verbal or imaginary parts. Yet, one study has shown that being consciously motivated to cognitively avoid certain information, enhances the automatic cognitive bias for this information (Lavy & Van den Hout, 1994), providing some indirect evidence that attempts to avoid threat-related imagery might prolong threat-related unconscious perseverative cognition.

In short, there are several reasons why worrying might become associated with positive outcome expectancies, and as a result might be prolonged and difficult to unlearn (disengage from). Since worry seldom actually helps to solve problems, these positive reinforcers suggest a vicious circle leading to ever more worry.

(3) Not recognizing safety signals

A third important factor that influences the duration of perseverative cognition is the (in)ability to recognize novel and ambiguous stimuli as safe. As mentioned above, the default cognitive response to stressful events will cease once a safety signal is recognized that signals that no goal is currently threatened. In line with this idea, Woody and Rachman (1994; p. 745) stated that: "*Safety signals de-limit the range and duration of threat and, hence, of fear. In the presence of an established safety signal the animal/person is assured of safety from threat in that place at that time. Having attained a safety signal, the person/animal can rest and reduce vigilance for a time*." As mentioned earlier, pathological worriers are strongly engaged in the pursuit of their goals and they require a lot of evidence before they dare to implement a solution to try solve the problem. A consequence of this might be that at least pathological worriers do not easily recognize novel and ambiguous situations as safe. Recent evidence supports this idea. For example, in a study conducted with GAD patients and healthy controls where participants were repeatedly shown cues (colored dots) that signaled either novel neutral or threat-related words while their cardiac responses were recorded simultaneously. It was shown that whereas the cardiac responses of healthy control participants to the neutral words showed habituation to the repeated presentation of neutral words, the GAD patients showed no habituation to these words, suggesting that they have difficulties in disengaging attention from these novel neutral stimuli in the context of threat (cf. Thayer, Friedman, Borkovec, Johnsen, & Molina, 2000). Other authors suggested that GAD patients show subcortical brain activity to neutral information as if it was threatening (Hoehn-Saric, Schlund, & Wong, 2004; Nitschke et al., 2009). Thus, high worriers seem to indiscriminately keep on responding to threatening as well as neutral stimuli, and thereby do not recognize safety signals. Not recognizing safety signals is possibly due to elevated requirements for evidence that has been found in high worriers, which in this case could be enhanced requirements for proof that a signal indeed signifies safety. By not recognizing safety, the fear response and perseverative cognition are therefore prolonged.

Biological vulnerability for perseverative cognition

In the sections above we referred to how the default stress response, and with it perseverative cognition, initiated by sub-cortical brain structures, is under tonic inhibitory control by the prefrontal cortex (Amat, et al., 2005; Thayer et al., 2006). There are several conditions in which this prefrontal inhibition is tuned down, making one vulnerable for perseverative cognition. Conditions involving low prefrontal inhibition include being in a chronic stress situation or being an anxiety patient. In their Neurovisceral model of perseverative thinking Thayer and Lane (2000) and Thayer and Brosschot (2005) explain how low prefrontal inhibition is characterized by low parasympathetic activation, which can be measured by low heart rate variability (HRV), and how low prefrontal inhibition leads to

prolonged and indiscriminate responses to environmental stimuli. We will provide a shortened account below.

Heart rate variability (HRV) is the variability of the time periods between adjacent heart beats, e.g. interbeat intervals (IBI). This variability is the result of the dynamic interplay between the fast acting parasympathetic nervous system and the relatively slower sympathetic nervous system. Cortical and subcortical areas in the brain that are responsible for the integration of internal and environmental information, including emotionally relevant information, are directly linked to HRV. These circuits are referred to as the Central Autonomic Network (CAN). Structurally, the CAN includes the anterior cingulate, insular, and ventromedial prefrontal cortices, the central nucleus of the amygdala, the paraventricular and related nuclei of the hypothalamus, the periaquaductal gray matter, the parabrachial nucleus, the nucleus of the solitary tract (NTS), the nucleus ambiguus, the ventrolateral medulla, the ventromedial medulla, and the medullary tegmental field. The primary output of the CAN is mediated through the preganglionic sympathetic and parasympathetic neurons. Importantly, these neurons innervate the heart via the stellate ganglia and the vagus nerve. The interplay of these inputs to the sino-atrial node of the heart is the source of the complex variability that characterizes the healthy heart rate time series (Saul, 1990). Thus, the output of the CAN is directly linked to HRV. It is logical that systems that control emotion, attention and autonomic nervous system activity are strongly interconnected and even largely overlap. Attention is always in the service of information that pertains to an organism or person's important goals, and hence related to the emotional value of the information. Likewise, when highly relevant goals are at stake autonomic activity is strongly needed to support approach or avoidance behavior. Therefore, these structures are bound to be highly interconnected and often activated in concert.

Low tonic levels of HRV might indicate a predisposition to keep on ‘erring on the side of caution’ when confronted with threat, novelty and ambiguity because such a chronically low HRV represents a breakdown of the inhibitory influences discussed earlier, that allows for efficient self-regulation including the interruption of on-going behavior. As such, an excitatory *positive feedback loop* is allowed to emerge, reflected on the psychological level in hypervigilance and perseverative thinking. As we discussed in the previous section, this response might become ever more enhanced once people implicitly or explicitly start to believe that this response actually helps them in solving their problems. As a consequence, the normally fine-tuned ability to adjust to changing environmental factors becomes a rigid, inflexible response disposition, which is in fact a continuation of the default defense response in the absence of clear threat signals. This is reflected in a failure to recognize safe environmental signals and in responding to them as if they are threatening. In support of this idea patients suffering from GAD have been shown to have lower tonic levels of HRV, when compared to non-anxious controls (Thayer et al., 1996). Furthermore, people low in HRV have been shown to have

an attentional bias for threatening information, and interpret ambiguous situations more negatively (Shook, Peña, Fazio, Soller III, & Thayer, 2007).

Besides in mood- and anxiety disorders, there might be several not so obvious conditions that are also associated with low prefrontal inhibition such as having a low aerobic fitness (Hansen, Johnsen, Sollers, Stenvik, & Thayer, 2004) or suffering from somatic health problems (e.g., diabetes and immune dysfunction (Masi, Hawkley, Rickett, & Cacioppo, 2007; Thayer & Lane, 2007; Thayer & Lane, 2000; Thayer & Sternberg, 2006). These conditions are associated with heightened risks to develop other stress-related mental and somatic problems, which may in fact be associated with the above mentioned excitatory positive feedback loop. For example, normotensive salt-sensitive men, a group that is at risk of developing hypertension, has been shown to have low levels of HRV at rest and during mental challenge (Buchholz, Schachinger, Wagner, Sharma, & Deter, 2003), indicating low prefrontal inhibition. Other studies have also shown that these men, compared to matched healthy controls, are also characterized by enhanced startle responses to negative information (Buchholz et al., 2001) and enhanced cortisol responses during stress (Weber et al., 2008), suggesting that default responding to stress is not only enhanced but also perseverates. It is an intriguing idea, that people that have low HRV for other reasons than chronic stress, i.e. diabetes, obesity, low aerobic fitness etc., would also cognitively perseverate (worry, ruminate) more as a result of the lower prefrontal inhibition associated with their condition. To the authors knowledge, this has never been tested.

In sum, we propose that low prefrontal inhibition reflected in low HRV predisposes people to respond with enhanced cognitive, affective and physiological activity to stressors. This, in combination with the psychological vulnerability factors for perseverative cognition discussed above, causes even seemingly neutral stimuli to trigger the stress response. As a consequence, the total time that people worry about stressful events increases, thereby adding to the total duration of exposure to stress representations, or perseverative cognition, and their physiological effects in daily life.

Conclusions

In this chapter we have provided an overview of the role that perseverative cognition plays in the onset and maintenance of stress-related mental and somatic health problems. Perseverative cognition is a common reaction to stressful events in everyday life, and it can account for stress-related physiological activity that is prolonged beyond the presence of actual real life stressors. This prolonged physiological activity is proposed to be the missing link in the relationship between psycho-social factors and the chronic pathogenic state in which one is more prone to develop mental and somatic problems. Furthermore, we have discussed that perseverative cognition forms part of the default response to threat, novelty and ambiguity, which basically is an adaptive self-regulatory

response. We also outlined which psychological and biological factors enhance this default response. Excessive commitment to one's goals, the motivated but exaggerated use of perseverative cognition as a strategy to cope with possible threats to goal attainment and the inability to recognize signals of safety were forwarded as pathogenic psychological processes that lead to a vicious cycle where one worry episode enhances the likelihood of the occurrence of another episode. This increases the total amount of time that stressful events have a prolonged 'wear and tear' effect on the human body.

Although the pivotal (causal) role of unconscious perseverative cognition in psychopathology has been acknowledged for a long time, its effects on somatic health have remained largely unexplored. However, while evidence is accumulating, numerous studies have already supported the perseverative cognition hypothesis. We have speculated on a role of unconscious perseverative cognition that may be as important or even more important, than that of conscious perseverative cognition. By focusing on conscious perseverative cognition alone we may have been only touching the tip of the iceberg of stress-related causes of mental and somatic problems.

Chapter 3

Capturing worry in daily life: Are trait questionnaires sufficient?

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Abstract

Worry is crucial in the development and maintenance of anxiety disorders and has been associated with several other adverse health outcomes. Yet, little is known about the frequency and perseveration of worry in daily life, and its predictability by widely used trait questionnaires.
In this study 432 students completed the Penn State Worry Questionnaire (PSWQ), the Worry Domains Questionnaire (WDQ) and the State Trait Anxiety Inventory-Trait Version (STAI-T) and kept a log of worry frequency and duration during six consecutive days and nights.
The results showed that worry is a very common phenomenon that is predicted by the two trait worry questionnaires, independent of trait anxiety. The often clinically utilized PSWQ predicted worry duration better than the WDQ, and exclusively predicted night-time worry and several other indices of perseverative and potentially pathogenic worry.
Although this study provides some support for the predictive validity of the PSWQ and the WDQ, these questionnaires did not account for the larger part of variance in daily worry. Future studies of worry and its associated perseverative processes should consider using momentary assessments.

Introduction

Perseverative cognition, such as worry and rumination, is crucial in the development and maintenance of mood disorders, anxiety disorders and insomnia (Borkovec, Ray, & Stöber, 1998; Nolen-Hoeksema, 2000). Moreover, perseverative cognition is attracting growing interest in research concerned with the *somatic* health effects of stress (for a review see: Brosschot, Gerin, & Thayer, 2006). A great deal of research on perseverative cognition has focused on *worry*, which is traditionally studied in relation to anxiety and insomnia (Borkovec, Robinson, Pruzinsky, & DePree, 1983). For example, uncontrollable and excessive worries are the core characteristics of generalized anxiety disorder (GAD; American Psychiatric Association, 1994). Recently, worry has also been found to predict depressive mood (Hong, 2007), post traumatic stress symptoms (Holeva, Tarrier, & Wells, 2001) and subjective health complaints (Brosschot & Van Der Doef, 2006; Petrie et al., 2005).

Worry can be measured by trait questionnaires as well as by ecological momentary assessment methods that measure state worry. The most frequently used trait questionnaires are the Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990) and the Worry Domains Questionnaire (WDQ; Tallis, Eysenck, & Mathews, 1992). The PSWQ is often used to measure pathogenic aspects of worry, for example its uncontrollability. Several studies that have been important in developing and testing theoretical models of GAD have used the PSWQ, for example to link pathological worry to potentially pathogenic phenomena and processes such as negative mood and 'stop rules' (Startup & Davey, 2003; Davey & Levy, 1998), intolerance of uncertainty (Dugas, Gagnon, Ladouceur, & Freeston, 1998) and meta-worry (Wells & Carter, 2001). Moreover, the PSWQ has been used as an outcome measure in several studies evaluating the efficacy of cognitive-behavioral therapies (Borkovec & Costello, 1993; Borkovec, Newman, Pincus, & Lytle, 2002; Dugas et al., 2003) and pharmacological treatments for GAD (Mogg, Bradley, Baldwin, & Brodrick, 2004). Whereas the PSWQ is a measure of pathological worry and the worry process, the WDQ was developed to measure the content of worries. In contrast with the PSWQ, the WDQ has been found to tap into constructive worrying (Davey, 1993). The WDQ has proven useful to differentiate worry topics associated with anxious mood and depressive mood (Diefenbach et al., 2001). Furthermore, in studies concerned with the differences between worry and obsessive thoughts, the WDQ and its short form (Stöber & Joormann, 2001) have proven useful in helping participants identify their most worrisome thoughts (Langlois, Freeston, & Ladouceur, 2000b; Langlois, Freeston, & Ladouceur, 2000a; Lee, Lee, Kim, Kwon, & Telch, 2005).

Several studies have shown that both the PSWQ and the WDQ have promising psychometric properties (Brown, 2003; Davey, 1993; Meyer et al., 1990; Stöber, 1998). However, the hallmark of validity of trait questionnaires is the extent to which they predict the behavior they are supposed to

measure, that is, its frequency and its duration. This issue does not appear to be conclusively addressed for the PSWQ and the WDQ, as we will argue below. Also, it is important to know whether these trait worry questionnaires are better predictors of worry in daily life than the closely related and broadly used phenomenon of trait anxiety (Startup & Erickson, 2006). Finally, it is also essential to know whether the clinically used PSWQ predicts potentially pathogenic aspects of worry in daily life better than the WDQ, for example the perseverance of worry during several days and the continuation of daily worry into the night.

The first validation study of the PSWQ showed that subjects scoring high on the PSWQ estimated that they had spent more time worrying per day during the past week than those scoring in the middle or low range (Meyer et al., 1990). In contrast, in a study with GAD patients, the PSWQ did not significantly correlate with estimates of the percentage of time spent worrying per day during the past month (Brown, Antony, & Barlow, 1992). However, Dupuy et al. (2001), using daily measurements for fourteen days, found that PSWQ scores predicted worry duration (r = .59) in a group of healthy participants and in a group of participants diagnosed with GAD (r = .42). Nevertheless, recent research has shown that pathological worry as observed in GAD patients and normal worry as observed in, for example, healthy students are not separate phenomena but mainly differ in severity, particularly its frequency and duration (Ruscio, Borkovec, & Ruscio, 2001). Therefore, it is important to examine to what extent trait worry questionnaires predict worry on this full severity range. Furthermore, the latter study did not differentiate between the frequency and the duration of worry, while it seems that worry duration is more pathogenic than worry frequency. According to Davey (2006, p. 218): “dysfunctional perseveration is one of the critical defining features of pathological worry”. Short worry episodes may reflect successful problem solving while longer worry episodes may imply potentially pathogenic processes. Duration of worry, and not or much less so frequency, predicted negative health outcomes (Brosschot & Van Der Doef, 2006), and mediated the effects of daily stress on heart activity during subsequent nocturnal sleep (Brosschot, van Dijk, & Thayer, 2007). Only two other diary studies have looked at correlations between the PSWQ and worry frequency as well as worry duration. Szábo and Lovibond (2002) found that the PSWQ predicted worry frequency (r=0.48) during seven days, but not worry duration. In contrast, Brosschot and van der Doef (2006) found that in high school students the PSWQ predicted worry frequency (r = 0.37) and worry duration (r = 0.37) during six days. To our knowledge, no studies have investigated the predictability of worry in daily life by the WDQ. Surprisingly, none of these studies differentiated between daily worry and evening/nightly worry, although a study by Tallis, Davey and Capuzzo (1994) showed that most worrying in students takes place between 9 p.m. and 3 p.m. Furthermore, worry at night is an often reported complaint of people suffering from insomnia (Harvey, Tang, & Browning, 2005) and GAD (Belanger, Morin, Langlois, & Ladouceur, 2004). Also, it

has been suggested to be a predictor of adverse cardiac activity patterns during sleep (Brosschot et al., 2007; Hall et al., 2004).

In short, it has not been shown whether these two broadly used tests of trait worry predict actual worry, whether they do this better than trait anxiety, or whether the PSWQ is better at predicting perseverative (i.e. pathogenic) aspects of worry than the WDQ. These issues were addressed in this study, which is the first to do this by using momentary assessment to investigate worry frequency and duration in the daytime as well as the night-time. In this study we defined the perseverative aspects of worry not only as the prolongation (duration) of individual worry episodes, but also in a exploratory way, as (a) the persistence of daytime worry into the night-time, (b) the persistence of worry into the following day(s), and (c) the total number of days and nights that people reported worrying over the six days. The latter is in line with the DSM-IV that states that a defining feature of pathological worry is that it occurs "more days than not, for at least 6 months" (APA, 2001).

Method

Participants

The sample consisted of 432 first year psychology students. Eighty one percent of the sample was female and 19% was male. The mean age was 21, with a minimum of 17 and a maximum of 59 years. Data were obtained during obligatory courses that were taught during the second semesters of four successive years (1999-2002). Five hundred and eighteen students were invited to participate, however 16.6% of these failed to return their worry logs. Students received course credit for partaking in this course.

Instruments

Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990; Dutch translation: van Rijsoort, Emmelkamp, & Vervaeke, 1999). This questionnaire consists of 16 self-report items. Items are directed at the excessiveness, duration and uncontrollability of worry as experienced in clients diagnosed with GAD, for example: "Once I start worrying, I can't stop". The PSWQ has demonstrated high reliability as well as high temporal stability and substantial validity in the assessment of trait worry (Meyer et al., 1990; van Rijsoort et al., 1999).

Worry Domains Questionnaire (WDQ; Tallis et al., 1992 ; Dutch translation: van Rijsoort et al., 1999). The Worry Domains Questionnaire (WDQ) was administered to assess the content and amount of worry. It consists of 25 items that make up the following five subscales: Relationships (e.g., "that I

will lose close friends"), Lack of Confidence (e.g., "that I lack confidence"), Aimless Future (e.g., "that I'll never achieve my ambitions"), Work Incompetence (e.g., "that I will not keep my workload up to date"), and Financial (e.g., "that I'm not able to afford things"). Internal reliability (van Rijsoort et al., 1999) and 4-week retest reliability of the total score and all subscales are satisfactory (Stöber, 1998).

State Trait Anxiety Inventory-Trait Form (STAI-T; Dutch version: van der Ploeg, Defares, & Spielberger, 1980). To measure trait anxiety we administered the trait version of the State-Trait Anxiety Inventory. The STAI-T is a questionnaire that measures the participants' predisposition to anxiety and has often been used in studies evaluating the effectiveness of treatments for GAD (Fisher & Durham, 1999). It consists of 20 self-report items and earlier use has shown good internal consistency and validity (van der Ploeg et al., 1980).

Worry log. The worry log is a one-page A4 form that has previously been used in a study by Brosschot and van der Doef (2006; see the Appendix for an example). On this form an adapted version of Borkovec's et al.'s (1983) working definition of worry was given (see below). All participants were instructed to register their worries during 6 days by tallying each worry episode. More specifically, they were instructed to register a worry episode whenever they noticed that they were worrying, or immediately after they had been worrying. At the end of each day they were asked to estimate the total number of worry episodes (*daily worry frequency*) and the total *duration* of these episodes (in minutes), based on their tallies. Each morning, they were requested to estimate the frequency and total duration of any nightly worry episodes (*worry frequency and duration in the night-time*).

To operationalize perseveration of worry, three additional and exploratory worry perseveration indices were calculated: (a) the persistence of daytime worry into the night-time, (b) the persistence of worry into the following day(s) and (c) the total number of days and nights that participants worried. These perseveration indices were calculated as follows. For each day and night, the data from the worry log were recoded into dummy variables that indicated whether participants had been worrying ('1') or not at all ('0'). The total number of days and nights that people had been worrying were summed to obtain (c). The dummy variables for the daytime and night-time for each day were added up. A score of 1 was given for daytime and night-time worrying respectively. These scores were added up per day, whereby a score of 2 indicates perseveration of worrying into the night. By counting the total number of perseveration scores we obtained (a). Likewise, the persistence of worry into the following day(s) (b) was calculated by counting perseveration scores for every two consecutive days and nights.

Procedure

The experiment consisted of two group sessions, with an interval of two weeks between them. During the first session the participants completed the questionnaires and received instructions concerning the registration of worry. These instructions were also printed on the back of the worry log. More precisely, they were told that (after the working definition of Borkovec et al., 1983): 'worrying involves thinking about a subject that has or can have negative consequences for you, and for which there is no, or not yet, a solution; it often, but not always, consists of a chain of negative thoughts, about the same or different topics, and often concerns something in the future, and the thought often takes shape as 'Imagine that . . . ' or 'What would happen if . . .? '; The same thoughts often return; when you are engaged in worrying it is difficult to stop or hold. It definitely occupies your mind, and it is often 'disturbing and intensive.' Participants were urged to conform their idea of worry as much as possible to this definition.

At the second group session the participants returned their worry logs and were debriefed.

Statistical analyses

The predictability of worry duration, worry frequency and worry perseveration by trait questionnaires was analyzed with Pearson correlations, hierarchical, and forced entry regression analyses. The distributions of worry duration, worry frequency and worry perseveration (i.e. number of days and nights worrying and number of successive days worrying) were significantly skewed and were transformed into normal distributions using logarithmic transformations. When describing the data (see section 3.1 and table 1), we report the untransformed data. All data were analyzed using the SPSS 11.0 software package.

Results

Descriptive statistics

Table 1 shows the descriptive statistics of the most important variables in this study. On average, participants worried 28 minutes: 22.5 minutes during the daytime, and 5.5 minutes during the night-time. On average, participants worried during 4.69 days (S.D. = 1.62) and 1.93 nights (S.D. = 1.87). Only 2% of the participants indicated that they had not been worrying at all during the six registration days and nights. Thirty-nine participants scored above the suggested clinical cut-point of 62 on the PSWQ (Behar, Alcaine, Zuellig, & Borkovec, 2003). On average, these participants worried 61.81 minutes a day (S.D. = 48.39).

The trait questionnaires showed satisfactory levels of internal consistency (Cronbach's α: PSWQ = .93, WDQ = .90, STAI-T = .90). The mean scores on the PSWQ (M = 43.51; S.D. = 12.97), the WDQ (M = 21.41; S.D. = 14.71) and the STAI-T (M = 37.45; S.D. = 8.77) are within the normal range

for healthy subjects and are comparable with mean scores found in other studies using young adults (Brosschot & van der Doef, 2006; Tallis et al., 1994).

The participants that did not return their worry logs did not differ significantly from those who did return their logs on the PSWQ, WDQ and STAI-T. In line with earlier studies on gender differences in worry (Robichaud, Dugas, & Conway, 2003), women scored significantly higher than men on all worry and anxiety variables.

Table 1. *Descriptive statistics of worry episodes and trait questionnaires*

Measure	*M*	SD	Minimum	Maximum
Worry duration				
Total	28.04	41.62	0.00	524.33
Daytime	22.51	35.61	0.00	481.67
Night-time	5.52	9.42	0.00	72.50
Worry frequency				
Total	3.55	3.81	0.00	30.00
Daytime	3.01	3.35	0.00	29.17
Night-time	0.54	0.84	0.00	7.50
PSWQ	43.51	12.97	16.00	78.00
WDQ	21.41	14.71	0.00	82.00
STAI-T	37.45	8.77	21.00	66.00

Note. PSWQ = Penn State Worry Questionnaire; WDQ = Worry Domains Questionnaire; STAI-T = State Trait Anxiety Inventory-Trait version.

Prediction of worry duration and frequency by trait questionnaires

Pearson correlations between trait questionnaires and worry duration and worry frequency are shown in table 2. Total worry duration correlated moderately with the PSWQ (r=.49, p<.01), the WDQ (r=.41, p<.01) and the STAI-T (r=.43, p<.01). In addition, total worry frequency correlated moderately with the PSWQ (r=.44, p<.01), the WDQ (r=.41, p<.01) and the STAI-T (r=.43, p<.01). To examine the extent to which trait worry questionnaires independently predict worry duration and frequency, forced entry regression analyses were conducted (see table 3 for results). Total worry duration was best predicted, (R^2 = .26; $F(3, 372) = 43.10$, p<.001), by the trait worry questionnaires, the PSWQ, (β = .33, p< .01), and the WDQ, (β = .14, p< .05), but not by the STAI-T, β = .10, ns). Total worry frequency was best predicted (R^2 = .23; $F(3, 383) = 38.36$, p<.001) by the PSWQ (β = .20, p< .01) , the WDQ (β = .19, p< .01) and the STAI-T (β = .16, p< .05) together.

Table 2. *Pearson correlations of trait questionnaires and worry in daily life*

	Worry duration	Worry frequency	PSWQ	WDQ	STAI-T
Worry duration	-				
Worry frequency	.73	-			
PSWQ	.49	.44	-		
WDQ	.41	.41	.63	-	
STAI-T	.43	.43	.76	.64	-

Note. All correlations are significant at the 0.001 level (2-tailed). PSWQ = Penn State Worry Questionnaire; WDQ = Worry Domains Questionnaire; STAI-T = State Trait Anxiety Inventory-Trait version.

Table 3. *Forced entry regression analysis of worry in daily life*

	Total worry duration				Total worry frequency			
Measure	*β*	p	R^2	F	*β*	p	R^2	F
			.26	43.10			.23	38.36
PSWQ	.33	.000			.20	.005		
WDQ	.14	.020			.19	.003		
STAI-T	.10	.195			.16	.030		

Note. PSWQ = Penn State Worry Questionnaire; WDQ = Worry Domains Questionnaire; STAI-T = State Trait Anxiety Inventory-Trait version.

Specific prediction of night-time worry

To assess which trait questionnaire specifically, and exclusively, predicted worry in the night-time *independent* of worry in the *daytime*, a hierarchical regression analysis was conducted. Daytime worry was entered in the analysis in the first block and the three trait questionnaires were entered in the second block. Results are shown in table 4. After controlling for daytime worry, only the PSWQ predicted worry duration (β = .18, p< .01) and worry frequency in the night-time (β = .14, p< .05).

Table 4. *Hierarchical regression analysis of worry in the night-time*

Block	Measure	Worry duration night-time				Worry frequency night-time			
		β	p	ΔR^2	F Change	*β*	p	ΔR^2	F Change
1				.32	182.85			.30	167.61
	Worry Daytime[a]	.47	.000			.48	.000		
2				.03	5.99			.02	4.07
	PSWQ	.18	.008			.14	.036		
	WDQ	.06	.271			.07	.247		
	STAI-T	-.03	.655			-.04	.548		

Note. PSWQ = Penn State Worry Questionnaire; WDQ = Worry Domains Questionnaire; STAI-T = State Trait Anxiety Inventory-Trait version. [a] Worry duration in the daytime was used to predict worry duration in the night-time, worry frequency in the daytime was used to predict worry frequency in the night-time.

Specific prediction of the perseveration of worry

The prediction that the PSWQ is a better predictor of the worry perseveration indices than the WDQ and the STAI-T was explored by conducting forced entry regression analyses. Number of nights worrying and number of days during which daily worry persisted into the night-time were only predicted by the PSWQ, (respectively β = .23 and β = .24, p's< .01). Number of days worrying and number of days worrying in a row were predicted by the PSWQ, (respectively β = .23 and β = .19, *p*'s< .05), and the WDQ, (respectively β = .13 and β = .15, p's< .05), although stronger by the former.

Discussion

This study is the first to investigate the predictive validity of the PSWQ and the WDQ, two widely used trait worry questionnaires, and the superiority of the PSWQ in predicting potential pathogenic worry. Collectively, the findings provide acceptable support for the predictive validity of these questionnaires and for the specific clinical applicability of the PSWQ. Furthermore, the study yielded descriptive data of normal daily worry, portraying worry as a very common phenomenon in non-clinical persons, with more days spent worrying than not.

The PSWQ and the WDQ both predicted worry duration and worry frequency in daily life, independent of each other and of trait anxiety. Together, the trait worry questionnaires accounted for approximately 24% of the variance of worry in daily life. This percentage is comparable with that found in other studies using momentary assessment of worry (Dupuy et al, 2001; Brosschot & van der Doef, 2006) and studies concerned with the prediction of daily negative affect by neuroticism (Eid & Diener, 1999; Mroczek & Almeida, 2004). It is not very high, but may become higher when

periods longer than six days are considered, making the state measurements less situationally determined. However, these figures also imply that the bulk of the daily experience of worry is not predicted by trait measures. This might be an issue to consider when investigating the outcomes of worry, since adverse health outcomes might be independently predicted by trait and state levels of negative emotional variables, such as has been shown for negative affect (Cohen et al., 1995; Tang & Gibson, 2005). It is possible that trait worry and state worry might reveal similar differential effects. At least one recent study showed that while trait worry was cross-sectionally associated with somatic health complaints, daily worry episodes predicted an increase in these complaints (Brosschot & van der Doef, 2006). Somewhat more indirect evidence comes from a GAD treatment study in which it was found that cognitive-behavioral therapy was effective in reducing worry as measured by the PSWQ, whereas applied relaxation therapy was specifically effective in reducing daily reports of anxiety (Borkovec & Costello, 1993). Thus, studies untangling the mechanisms whereby worry affects health should consider combining trait measures with momentary assessments of worry.

This study also provided support for the use of the PSWQ as a measure of pathogenic worry. We found that the PSWQ was a better predictor of total worry duration and frequency than the WDQ and the STAI-T. In addition, the PSWQ was the only trait measure that predicted worry at night. Night-time worry is often observed in insomnia (Harvey et al., 2005) and GAD (Belanger et al., 2004), and is believed to play a crucial etiological role in these disorders. Exploratory analysis showed that the PSWQ was the best predictor of worry perseveration as indexed by total number of days and nights spent worrying and the persistence of worry into the night and into the following days. Thus, the PSWQ and not the WDQ or trait anxiety predicted high levels of perseverative worry in daily life, as operationalized by various indices, including total duration, nocturnal worry, and persistence of worry across consecutive days and nights.

These findings have to be interpreted in the light of several methodological limitations. Our measurement of worry at night could have been prone to retrospective bias as participants were asked to register worry the following morning, instead of during the night, while they were worrying. It might be argued that especially high PSWQ scorers might have overestimated worry at night. On the other hand, overestimations of the time lying awake have been found in both healthy subjects and insomniacs (Nelson & Harvey, 2003; Baker, Maloney, & Driver, 1999), and might thus not be restricted to high PSWQ scorers. In addition, it remains unclear whether worry episodes at night took place while participants were actively engaged in activities or while they were passively lying in bed. Moreover, worry episodes at night could have occurred before or after participants had fallen asleep. This is a potential flaw that necessitates a more rigorous study that includes nocturnal momentary assessments, although such a study will have to solve the problem of how to assess nocturnal worry

without interfering with sleep. Furthermore, one could argue that the percentage of participants that did not return their worry logs was relatively large. However, this was comparable to that found in another study (Brosschot & van der Doef, 2006) and there were no differences between the studies on the scores on the trait questionnaires. In addition, the participants who did not return their worry logs did not differ on the scores on the trait questionnaires from those who did return their logs. Our sample therefore seemed to cover the full worry range. Another concern can be raised about our operationalization of pathological worry. We did not include a measure of GAD to assure whether worry was pathological as defined by the DSM-IV (APA, 1994). However, Ruscio (2002) showed that although high levels of worry are a main characteristic of GAD, a large group of people who show high levels of worry do not receive a full GAD diagnosis. In addition, several studies suggest that student samples are suitable to investigate worry on the full severity range (Borkovec & Roemer, 1995; Ruscio, 2002; Roemer, Borkovec, Posa, & Borkovec, 1995). Our interest was in the prediction of the perseveration of worry, indexed in a variety of ways and not in predicting diagnoses of GAD. Finally, the instructions given to participants need some consideration. Specifically, it is possible that our definition of worry as 'uncontrollable' and 'often disturbing and intensive' led to an *under*-reporting of episodes of 'constructive worry' (Davey, 1993). It is important to note that the superiority of the PSWQ in predicting worry in daily life is likely to be limited to pathological worry. In contrast, the finding of daily worry duration that was twice as long by Dupuy et al. (2001; 60 min versus 30 min by us and by the related study of Brosschot & Van der Doef, 2006) was possibly due to the fact that Dupuy et al. (2001) emphasized that worry was accompanied by anxiety, whereas the definition of worry that we provided laid more emphasis on worry as a process of repetitive negative *thinking*. The former definition could have led participants to report worry as well as anxiety episodes, thereby creating a bias in total worry duration. In addition, Szábo and Lovibond (2002) provided their participants with no definition of worry and found that 11% of the worry episodes were actually rumination episodes. This short outline makes clear that it is essential to consider in great detail what one exactly wants to measure and how one instructs participants.

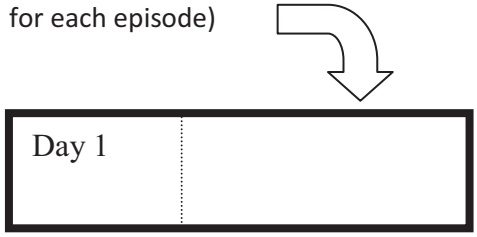
In sum, this study provides reasonable support for the predictive validity of both the PSWQ and the WDQ, their superiority in predicting daily worry over the STAI-T, and their differential predictive validity with respect to perseverative or pathogenic aspects of worry. However, this study also suggests that future studies concerned with the emotional and health outcomes of worry should consider combining trait questionnaires with momentary assessments.

Appendix. Example of the worry log (one day excerpt displayed).

REGISTRATION FORM

Register during daytime: Fill in at the end of the day:

Number of worry episodes (one tally for each episode)

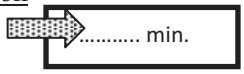


Estimated total number:

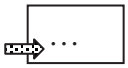
(None?: Fill in: **0**)

Estimated total duration

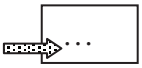
(None?: Fill in: **0**)



Worry episodes at night?: Number:



and duration:



Worry and Somatic Health

Chapter 4

Effects of momentary assessed stressful events and worry episodes on somatic health complaints

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Manuscript under review

Abstract

Somatic health complaints are extremely common and are responsible for a large part of human suffering and health care costs. It has been recognized that psychosocial stress can affect somatic health. Yet, according to the "perseverative cognition hypothesis", stressful events can only affect somatic health when people keep on worrying about them. Worry would prolong stress-related physiological activity that can ultimately lead to health problems. In this ambulatory study we tested whether stressful events and worry predict daily somatic complaints, and whether worry mediates the effects of stressful events. In addition, it was tested whether these effects were independent from negative affect. Using electronic diaries, sixty-nine teachers (age 21 - 60) from Dutch primary and secondary schools reported daily stressful events, worry episodes, negative affect and somatic complaints for a period of six days. Results showed that worry intensity predicted the number of somatic complaints and mediated the effect of stressful events on somatic complaints. Furthermore, these results were independent from biobehavioral variables and daily negative affect. These findings support the perseverative cognition hypothesis proposing that the negative somatic health effects of stressful events are largely due to the worry, that is, to the prolonged cognitive representation of stressors.

Introduction

Somatic health complaints are extremely common. A survey among more than 1 million people in the US revealed that 40% to 55% were subject to headaches, 33% to 46% fatigue, and 15% sore throat at the point in time that they were being examined (Hammond, 1964). In a study in the Nordic European countries (Eriksen, Svendsrod, Ursin, & Ursin, 1998) 75% of the respondents reported one or more subjective health complaints. A recent investigation in a general Dutch population (older than 25 yr) found that low back pain, shoulder pain and neck pain occurred in respectively 26.9%, 20.9% and 20.6% of the subjects (Picavet & Schouten, 2003). Between 33 and 42% of those with complaints consulted their general practitioner about their pain. In addition, most of these complaints concern vague symptoms without a clear medical diagnosis (Kroenke & Mangelsdorff, 1989). A survey in seven outpatient clinics in London, across a variety of specialties, showed that 52% of the patients fulfilled criteria for medically unexplained symptoms. In some specialties, it was the most common diagnosis (Nimnuan, Hotopf, & Wessely, 2001). In addition, the costs for the investigations are frequently high, taking into account the medical actions involved, which come on top of sick leave compensations and the loss of productivity (Eriksen & Ihlebaek, 2002). This short analysis makes clear that it is important to investigate which processes underlie the reporting of somatic complaints.

It has been recognized for a long time that psychosocial stress can affect somatic health. The perception of a stressful event triggers the physiological stress response and when this response is prolonged for too long it can become detrimental for ones somatic health (Mcewen, 1998; Brosschot & Thayer, 1998; Linden, Earle, Gerin, & Christenfeld, 1997; Schwartz et al., 2003; Pieper & Brosschot, 2005). One important feature of stressful events is that they not only trigger the physiological stress response, but also trigger perseverative thoughts about these events. In recent years, this perseverative thinking, such as worrying about upcoming stressful events, has been put forward as the central pathological mechanism mediating between the perception of stressful situations and poor somatic health, including somatic complaints (Brosschot, Pieper, & Thayer, 2005; Brosschot, Gerin, & Thayer, 2006; Watkins, 2008). According to this “perseverative cognition hypothesis” (Brosschot et al., 2006), worrying about stressors prolongs the total amount of time that these stressors adversely affect physiological functioning. Several studies provide evidence for this idea, in the laboratory (see Brosschot et al., 2006, and more recent: Gerin, Davidson, Christenfeld, Goyal, & Schwartz, 2006; Key, Campbell, Bacon, & Gerin, 2008; Zoccola, Dickerson, & Zaldivar, 2008) but also in daily life. With respect to the latter, worry mediated the effect of stressful events on cardiac activity during waking as well as sleeping at night (Brosschot, van Dijk, & Thayer, 2007). Other ambulatory studies have shown that worry about *stressful events at work* is related to heightened cortisol levels (Schlotz, Hellhammer, Schulz, & Stone, 2004) and with heightened heart rate in a

sample of teachers (Pieper, Brosschot, van der Leeden, & Thayer, 2007). In addition, people who keep on worrying about their work after the workday has passed, have difficulties 'unwinding' and suffer from more emotional and somatoform symptoms (Geurts & Sonnentag, 2006; Sonnentag, Binnewies, & Mojza, 2008) than those who do not. Moreover, prospective studies have provided evidence that the tendency to worry about things and the inability to 'unwind' or disengage after work is associated with cardiovascular morbidity (van Amelsvoort, Kant, Bultmann, & Swaen, 2003) and even mortality (Kivimaki et al., 2006).

Furthermore, the perseverative cognition hypothesis predicts that worry – through these prolonged physiological responses - leads to somatic complaints, and mediates the effects of stressful events on these complaints. There is some evidence that stressful events have such an influence on somatic complaints. Cross-sectional studies have shown that worry is associated with several somatic health complaints, especially with (neck) pain (Freeston et al., 1996; Thomsen et al., 2004). Also, Emmons and King (1988) found that individuals who spend a large amount of time ruminating about their conflicting life goals show higher levels of somatic complaints. More recently, in a prospective study, Brosschot and Van der Doef (2006) found in a sample of adolescents that a simple worry intervention was helpful in reducing worry during six consecutive days. This reduction in worry in turn, predicted a decrease in the number of subjective health complaint assessed before and after the six days of the worry intervention. Their study is the first to show that worry was prospectively and therefore perhaps causally related to the number of somatic complaints. The present study was set up to extend the findings from Brosschot and Van der Doef's (2006) study and more precisely test the perseverative cognition hypothesis. Firstly, the effects of (daily) stressful events on somatic complaints were not taken into account in that study. Secondly, only worry episodes were assessed using daily assessments whereas all other variables (including somatic complaints and negative affect) were measured retrospectively. Concerning the first issue, it could still be possible that the worry episodes were an indirect indication of the experience of stressful events. Indeed several studies have shown that stressful events are associated with somatic complaints (see e.g. Joksimovic, Starke, Knesebeck, & Siegrist, 2002; Godin & Kittel, 2004; Lepore, Miles, & Levy, 1997) and the perseverative cognition hypothesis predicts that worry about these events might have mediated their effects. To test this, in the present study we tried to replicate the finding that worry was prospectively related to somatic complaints, while also taking the number of stressful events into account. Concerning the second issue, Brosschot and Van der Doef (2006) only measured the number of somatic complaints before and after a period of six days, during which the worry intervention took place, and asked participants to report on the number of complaints experienced "during the last three days". This latter retrospective way of measuring complaints could have introduced a so called 'retrospective bias'. Houtveen and Oei (2007) recently showed that the

total number of somatic complaints experienced during a week is higher when this is assessed at the end of the week, compared to when these complaints are assessed each single day during a week, suggesting that participants use different memory retrieval strategies for these two kinds of assessments. Thus, by using a retrospective method some participants – especially those who worried a lot during the week - might have overestimated their complaints in the Brosschot and Van der Doef study. We therefore used a daily measurement of somatic complaints in the present study.

Stressful events not only result in worrisome thoughts about these events, but are also accompanied by negative affect. Recent evidence suggests that negative affect is prolonged beyond the presence of actual stressors by worry (Watkins, 2008). Negative affect has also been shown to increase the reporting of somatic symptoms, either by enhancing adverse physiological responding to, for example, infections (Cohen et al., 1995) or by biasing attention towards (threatening) misinterpretations of harmless bodily sensations (Petrie, Moss-Morris, Grey, & Shaw, 2004; Rief & Broadbent, 2007). It is plausible that the effects of worry on somatic complaints are in their turn mediated by negative affect – being an indication of prolonged physiological arousal or prolonged attention towards bodily sensations. Yet, not all studies have found independent effects of negative affect on health-related variables. For example, recent studies by us indicated that negative affect has no or only minor effects on cardiac activity (Pieper et al., 2007; Verkuil, Brosschot, Borkovec, & Thayer, 2009). On the other hand, Thomson et al. (2004) found that negative affect mediated the effect of rumination on poor somatic health in elderly people. However, this finding was restricted to the cross-sectional part of their study and was not apparent in the prospective part. Taken together, it is not yet clear what the role of negative affect is in explaining somatic health complaints, and the effect of stress and worry on them. We therefore examined, in the present study, the role of negative affect by including a daily measurement of negative affectivity.

In short, the present study tested whether (1a) stressful events predict the number of somatic complaints and whether (1b) worry episodes predict the number of and (1c) whether worry mediates the effects of stressful events on the number of somatic complaints. Finally, (2a), we tested whether worry predicts negative affect and (2b) whether the effects of worry on somatic complaints are mediated by negative affect. Importantly, and different from previous studies, all variables were assessed using electronic diaries, either per day or per time block of several hours.

Method

Participants

The managers of fifteen primary schools and seven secondary schools were contacted and were asked whether they gave permission to the researchers to contact their teachers for the purpose of this study. In addition, 57 teachers were contacted individually. The research assistants who helped

carrying out this study (undergraduate students in psychology) were familiar with many of these teachers, which were from their former schools. All teachers received written information about the study and were asked to respond in case they were willing to participate. In total, 102 teachers responded. In the end, 30 of these teachers could not participate in the study, due to sickness or logistic difficulties, resulting in a final sample of 72 participants. All participants gave written informed consent before participating.

Instruments

Electronic diary

Daily reports on the variables of interest were collected using electronic diaries. Participants were handed a Palm-top computer (either a Palm Tungsten E2 or a Palm M100). The usage of the Palmtop was explained by one of the research assistants. For six consecutive days participants were prompted five times a day (from 8.00 until 21.30). The prompts were separated by randomly varying time periods, each lasting at least 2¾ hours, but maximally 3¾ hours. Daily assessments were gathered via Pendragon Forms 5.0 software and prompts were signaled with alarms programmed in Diary Alarms 1.0.

Daily assessments

Subjective health complaints

At every last assessment of the day, the Subjective Health Complaints questionnaire (SHC) was administered (Eriksen, Ihlebaek, & Ursin, 1999). This questionnaire consists of 29-items measuring the presence and severity of health complaints in five different areas of complaints: musculoskeletal pain; pseudo-neurological; gastrointestinal problems; allergic problems; and flu. The SHC is a reliable, easy, and systematic way to score subjective health complaints as they are experienced by the general population (Eriksen et al., 1999). The items ‘anxious’ and ‘depressed’ were removed before the analyses. Due to a programming error, the item ‘migraine’ was not included in the palmtop version of the questionnaire, yet unpublished data by us show that this complaint is very seldom. The item ‘headache’ however, was retained. The total number of the remaining twenty-six SHCs was used as our outcome measure.

Worry episodes

The experience of worry episodes was assessed at each prompt. Participants were provided with a definition of worry based upon Borkovec et al. (1983). This definition was also printed on the case of the PDA. As the Dutch word for worry (‘piekeren’) also refers to ‘thinking hard’, we referred to worry as rumination (‘rumineren’; see also, Pieper et al. 2007) while providing the following description:

"*rumination involves repeatedly and constantly thinking about negative events or situations in the past, present or future. The thoughts are often accompanied by negative tension".* At every prompt, participants had to indicate whether they had been worrying, and if this was the case, then they were asked to indicate for how long (less than 5 minutes; 5 to 30 minutes; 30 – 60 minutes; more than 60 minutes). Subsequently, they were asked to indicate the total intensity of the worry episode(s) by dragging a slider on a scale ranging from 'slightly intense' to 'very intense' (end points ranging from 0-10). Finally, there were asked to indicate whether the worrying concerned private or work related issues. The latter aspects (intensity and content) were also new compared to the Brosschot and Van der Doef study.

Stressful events

Stressful events were also assessed at each prompt. Participants were provided with a definition of stressful events, based upon the definition described in the Daily Hassles Scale (APL, see below): '*Stressful events are minor and major events that have made you feel tense, irritated, angry, sad, disappointed or negative in any other way*'. Participants were asked to indicate the number of stressful events that had experienced ('0', '1', '2', '3', '4', '5 or more') since the last prompt. In case they had experienced one or more stressful events, they were asked to indicate whether the event concerned private or work related issues.

Negative affect

At every last assessment of the day, negative affect was measured with the negative affect subscale of the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988). Example items are 'distressed', 'scared' and 'irritable'. The scale point are: 1 'very slightly or not at all', 2 'a little', 3 'moderately', 4 'quite a bit' and 5 'very much'. Participants were asked to report on the amount of negative affect experienced during the preceding day.

Biobehavioral variables

To be able to control for the effects of biobehavioral variables on somatic complaints, participants were asked at the last assessment of the day to report the number alcoholic beverages, the number of cups of coffee and the number of cigarettes smoked ('0', '1-2', '3-4', '4 or more'), the amount of physical effort (five point scale ranging from 'none' to 'very much'). In addition, to control for the effects of bad sleep on somatic complaints, each morning at the first assessment, participants were asked to report on the sleep quality during the previous night. Sleep quality was measured on a VAS-scale, with score of 0 represented a 'very bad' sleep-quality and a score of 10 a 'very good' sleep-quality.

Baseline questionnaires

Somatic complaints

The number of somatic complaints experienced during the three days before the start of the experiment was assessed with the paper and pencil version of the SHC described above. In order to control for the effects of SHC during the previous day, including the mean number of complaints during the three days before the start of the experiment, a new time lagged variable was created ("Previous SHC").

Trait-worry

The Penn State Worry Questionnaire (PSWQ) was used to measure the level of (pathological) trait-worry. The PSWQ has proven to be a reliable and valid measure (Meyer, Miller, Metzger, & Borkovec, 1990; Verkuil, Brosschot, & Thayer, 2007b). Examples of items are "I am always worrying about something" and "Once I start worrying, I can't stop." The items are scored on a 5-point Likert scale, with a score of 1 representing "Not at all typical" (for me) and a score of 5 representing "Very typical" (for me).

Daily hassles

Stressful events during the past two months were measured with the Daily Problems Checklist (Vingerhoets, Jeninga, & Menges, 1989)). The DPC is based on the Daily Hassle Scale by Lazurus and colleagues(Kanner, Coyne, Schaefer, & Lazarus, 1981) and has been previously used in stress research in teachers (see e.g. Brosschot et al., 1994). Items include "One of your family members had health problems" and "You had a conflict with your partner". Participants were asked to indicate whether they had experienced such an event, and to what extent they were annoyed by these events (five points scale ranging from 'not at all' to 'very much'). The total sum on this questionnaire was used as our measure of experienced stress at baseline.

Trait negative affect

Participants filled in the PANAS, described above, but here the trait version, concerning their mood in general.

Statistical analyses

Multilevel or hierarchical regression analyses were used to analyze the associations between the variables of interest (Singer & Willett, 2003). Multilevel analysis is especially suitable to analyze repeated measures data because it accounts for the dependencies of the different measurements

(level 1) that are nested within individuals (level 2). Another advantage is that multilevel analysis allows for models to be estimated on all available data from each individual and can handle unbalanced datasets that contain irregularly spaced measurement intervals. Missing values on the questionnaires were imputed using the algorithm provided by Van Ginkel & Van der Ark (2005). Multilevel hierarchical regression analyses were performed with SPSS version 14.0.

To test whether worry mediated the effects of stressful events on somatic complaints, and possibly negative affect on turn mediated these effects, a mediation analysis was conducted in line with the guidelines provided by Baron and Kenny (1986).

Results

Descriptive statistics

Of the initial 72 participants, three participants did not complete the study. The final sample consisted of 69 Dutch secondary school teachers, of which 44 were female and 24 male. Age ranged from 21 to 60 years, with an average of 38.88 years (SD = 12.89). Baseline descriptive statistics are shown in Table 1.

Concerning the daily assessments of somatic complaints, fatigue was the most reported somatic complaint, which is in line with previous studies on the prevalence of SHCs (Eriksen et al., 1999); in 59.9% of all daily reports participants complained about fatigue (including missed reports). Other frequently reported complaints were low back pain (27.3%), flatulence / “wind” (25.6%), sleeping difficulties (25.4%) and cold / flu (23.7%).

The mean number of stressful events per day was 2.60 (SD = 1.91), whereas the mean number of worry episodes per day was 1.49 (SD = 1.24), which is slightly higher than previously observed in comparable studies (e.g. 1.58 and 1.06 respectively, by Pieper et al., 2007). The total duration of the worry episodes per day was 37 minutes (SD = 48.56), which is in line with previous studies in non-clinical samples (Verkuil et al., 2007b). The mean intensity score per worry episode was 4.67 out of 10 (SD = 2.57).

Table 1. *Descriptive statistics at baseline.*

	%	M	SD
Gender	64.7% female		
Education	79.4% HBO		
	20.6% MBO		
Age		38.88	12.89
SHC		4.66	3.71
PANAS		16.30	4.66
DHC		33.55	23.17
PSWQ		42.94	11.40

Note. SHC = Subjective Health Complaints; PANAS = Positive Affect Negative Affect Schedule; DHC = Daily Hassles Checklist; PSWQ = Penn State Worry Questionnaire.

Preliminary analyses

To assess whether multilevel analysis would be appropriate to analyze the effects of stressful events and worry episodes on somatic complaints, we first estimated the intra-class correlation in a baseline model with a random intercept and with SHC as the dependent variable, but without any predictors. The results showed that the intraclass correlation was .59, showing that 59% of the variance was due to individual differences between participants, thereby providing evidence for a 2-level hierarchical structure of the data. In addition, since the somatic complaints were measured repeatedly within subjects, we tested whether the error terms of the model would be correlated. Residual error covariance was modeled using the first-order auto-regressive covariance matrix, which showed that the estimated auto-correlation (ρ) was .22 (p = .017). With respect to the model predicting daily negative affect, a baseline random intercept model without predictors showed that the intra-class correlation was .46, that 46% of the variance was due to individual differences between participants. Because residual error covariance using the first-order auto-regressive covariance matrix did not yield stable models, the diagonal covariance matrix was used.

Effects of stressful events and worry episodes on daily somatic complaints

First we examined whether stressful events were associated with the number of SHC, while controlling for SHC the previous day. The effect of stressful events on SHC was significant (B = .191, p < .0001, 95% CI: .087 - .294). When stressful work and private events were entered separately into

the model, the results showed that work related stressors had a larger effect on somatic complaints (B = .366, $p < .01$, 95% CI: .114 - .617) than private stressors (B = .290, $p < .05$, 95% CI: .052 - .527).

Next, we examined the effects of the worry variables, (frequency, duration and intensity) on the number of SHC. The correlations between these variables were high (rs > .87). In a first step, SHC was regressed on worry frequency and worry duration (the variables used in the study by Brosschot & van der Doef [2006]), while controlling for the number of somatic complaints during the previous day. Worry frequency significantly predicted the number of somatic complaints (B = .451, $p < .01$, 95% CI: .152 - .749), and worry duration did this marginally (B = .008, $p = .082$, 95% CI: -.001 - .019). When worry intensity was entered into the model, only worry intensity predicted the number of SHC (B = .094, $p < .01$, 95% CI: .028 - .160), whereas the effects of worry frequency (B = .137, $p = .460$) and worry duration (B = .000, $p = .954$) were not significant anymore.

Mediating effects of worry episodes between stressful events and somatic complaints

To test whether the effect of stressful events on SHC was mediated by worry intensity, worry intensity was regressed on the number of stressful events. The number of stressful events was significantly related to worry intensity (B = 1.165, $p < .0001$, 95% CI: .863 - 1.467). In a subsequent model, stressful events and worry intensity were both added as predictors of SHC. When controlling for the effect of worry intensity, the effect of stressful events on SHC was reduced and became non-significant (B = .053, $p = .337$, 95% CI: -.055 - .161), whereas the effect of worry intensity was still significant (B = .106, $p < .0001$, 95% CI: .071 - .140), suggesting full mediation. Sobel's z-score of this mediated effect was 4.82 showing that the mediation effect was significant ($p < .0001$; Baron & Kenny, 1986).

In a final model we tested whether worry intensity was related to SHC while controlling for biobehavioral variables (age, gender, education level, smoking, alcohol use, sleep quality, baseline traits and daily hassles during the last two months) and SHC during the previous day. The results are presented in Table 2. In the final model, SHC was predicted by worry intensity, time (days) and daily hassles.

Table 2. *Estimates of fixed effects predicting the number of somatic complaints with and without negative affect (baseline and daily measurements)*

	B	SE	*t*	*p*	B	SE	*t*	*p*
Intercept	4.67	0.38	12.18	.00	4.68	0.36	12.97	.00
Stressful events	-0.03	0.06	-0.43	.67	-0.05	0.06	-0.78	.44
Worry intensity	0.11	0.02	5.99	.00	0.10	0.02	4.88	.00
Age	0.00	0.02	0.05	.96	0.00	0.02	-0.01	.99
Gender	0.63	0.59	1.07	.29	0.48	0.55	0.87	.39
Education	-0.68	0.67	-1.02	.32	-0.57	0.61	-0.92	.36
Caffeine	0.04	0.10	0.41	.68	0.05	0.10	0.52	.60
Smoking	0.10	0.06	1.77	.08	0.16	0.06	2.47	.01
Alcohol	0.10	0.10	0.96	.34	0.05	0.10	0.49	.62
Physical effort	-0.10	0.12	-0.85	.40	-0.10	0.12	-0.83	.41
PSWQ	-0.04	0.03	-1.58	.12	-0.04	0.03	-1.54	.13
DHC	0.03	0.01	2.24	.03	0.02	0.01	1.78	.08
Previous SHC	0.03	0.05	0.64	.52	0.06	0.05	1.11	.27
Sleep quality previous night	-0.07	0.15	-0.47	.64	-0.18	0.15	-1.17	.24
Time	-0.26	0.08	-3.43	.00	-0.23	0.08	-3.10	.00
Baseline negative affect					-0.05	0.07	-0.71	.48
Daily Negative affect					0.14	0.04	3.37	.00
Deviance (-2 log likelihood)	1138.435				1103.456			

Note. PSWQ = Penn State Worry Questionnaire; DHC = Daily Hassles Checklist; SHC = Subjective Health Complaints.

Effects of stressful events and worry episodes on negative affect

First, negative affect was regressed on the number of stressful events, while controlling for negative affect during the previous day. Stressful events significantly predicted negative affect (B = 0.520, $p < .0001$, 95% CI: 0.334 - 0.706). Stressful events related to work were a slightly better predictor of negative affect (B = 0.934, $p < .0001$, 95% CI: 0.516 - 1.353) than stressful private events (B = 0.698, $p < .0001$, 95% CI: 0.308 - 1.089). Next, negative affect was regressed on worry frequency and worry duration. Only worry duration predicted negative affect (B = 0.022, $p < .05$, 95% CI: 0.005 - 0.038), whereas worry frequency did not (B = 0.382, $p = .144$). When worry intensity was entered into the model, only worry intensity predicted negative affect (B = .144, $p < .05$, 95% CI: 0.033 - 0.254), whereas the effects of worry frequency (B = -.065, $p = .839$) and worry duration (B = .010, $p = .308$) were not significant anymore. When controlling for the effect of negative affect during the previous day and worry intensity, the effect of stressful events on negative affect was still significant (B = .323,

p < .01, 95% CI: 0.127 - .519), as was the effect of worry intensity (B = .131, *p* < .0001, 95% CI: .077 - .184).

In the final model, negative affect was regressed on stressful events, worry intensity, the biobehavioral variables, negative affect at baseline and negative affect during the previous day (see Table 3). Negative affect was significantly predicted by negative affect at baseline (B = 0.154, *p* = .098, 95% CI: -0.030 - 0.338), worry intensity (B = 0.128, *p* < .0001, 95% CI: 0.069 - 0.187) and, yet marginally, by age (B = 0.057, *p* = .067, 95% CI: -0.064 - 0.366), but not any more by stressful events.

Table 3. *Estimates of fixed effects predicting daily negative affect.*

	B	SE	*t*	*p*
Intercept	13.50	0.54	25.07	.00
Worry intensity	0.13	0.03	4.29	.00
Stressful events	0.15	0.11	1.38	.17
Age	0.06	0.03	1.90	.07
Gender	0.84	0.74	1.13	.27
Education	-0.28	0.85	-0.34	.74
Caffeine	-0.22	0.15	-1.48	.14
Smoking	-0.06	0.09	-0.67	.51
Alcohol	0.09	0.15	0.59	.56
Physical effort	-0.06	0.18	-0.31	.75
PSWQ	0.01	0.04	0.31	.76
DHC	0.02	0.02	0.81	.42
Negative Affect previous day	0.07	0.06	1.05	.30
Sleep quality previous night	-0.05	0.23	-0.20	.84
Time	-0.08	0.12	-0.66	.51
Baseline negative affect	0.15	0.09	1.71	.10

Note. PSWQ = Penn State Worry Questionnaire; DHC = Daily Hassles Checklist.

Mediating effects of negative affect between worry and somatic complaints

To examine whether baseline negative affect and daily negative affect could add to the model predicting the number of SHC, and whether daily negative affect mediated the effect of worry intensity on SHC, these variables were added as predictors in the model discussed above. In this final model, SHC was independently predicted by daily negative affect (B = 0.118, *p* = .010, 95% CI: 0.029 - 0.208), smoking (B = 0.145, *p* = .025, 95% CI: 0.018 - 0.272), and worry intensity (B = 0.095, *p* < .0001, 95% CI: 0.051 - 0.140). A trend towards significance was still apparent for daily hassles at baseline. As

the effect of worry intensity on SHC was not reduced by adding daily negative affect to the model, the hypothesis that negative affect mediates the effect of worry on SHC could not be confirmed. Another possible role of negative affect might be that it interacts with worry and thereby enhances the effects of worry on somatic complaints. However, exploratory analysis showed that the interaction between worry intensity and daily negative affect on SHC was not significant.

Effects on specific complaints

To examine whether worry intensity was associated with the occurrence of specific somatic complaints, multilevel random intercept logistic regression models were fit (using MLwiN software) on the specific complaints. Results showed that worry intensity was significantly associated with the occurrence of 13 of the 26 complaints. These complaints were: neck pain, stomach pains, fatigue, sleeping difficulties, pain in arms, headache, asthma, flatulence / 'wind', chest pains, vertigo, pain in shoulders, stomach discomfort and cold / flu.

Discussion

In this study the effects of stressful events and worry on somatic complaints were examined. In line with previous work, the present study demonstrated that worry was prospectively associated with somatic health (Brosschot & Van Der Doef, 2006; Thomsen et al., 2004). Importantly, this study adds to previous studies the finding that worry mediates the effects of stressful events on somatic health complaints. These effects of worry were independent of negative affect and biobehavioral variables. Moreover we used a more precise measure of worry, that did not contain the connotation of 'thinking hard' of the Dutch word for worry ('piekeren'). Furthermore, health complaints were measured on a daily basis instead of retrospectively as in Brosschot & Van Der Doef (2006). This study therefore provide further evidence for the perseverative cognition hypothesis which states that worry is the crucial link between stressful events and somatic health (Brosschot et al., 2006). The effects of worry were visible on a range of different single complaints, suggesting that there is not one specific biological system involved, but that the effect is the results of a general physiological stress response. This study focused on a group of teachers, workers who are considered to be highly vulnerable for developing work stress related psychological and somatic complaints. As Brosschot & Van der Doef (2006) have found that a relatively simple worry intervention is helpful in reducing worry and somatic complaints in adolescents, a next step might be to test the effectiveness of such a worry intervention in a vulnerable group like this one. Reducing the harmful effects of prolonged stress responses such as worry might be an important addition to existing (preventive) stress management interventions that are aimed at reducing the immediate effects of stressful events, for example assertiveness training. If proven to be effective, this is expected to have large implications

not only for the wellbeing of workers, but, as a consequence, also on the economical costs associated with somatic health complaints.

One reason that worry had stronger effects than stressful events might not have to do with the fact that worry mediated their effects, but with the much larger scope of worry. Worry is always about stressors and by measuring the effects of worry episodes we aggregated the effects of one or many more stressful events at once, including many events outside the time window of this study. Moreover, worries are mostly about very significant personal events, in the (regretted) past as well as in the (feared) future, and these cognitive representations of stressful events are always highly personally relevant. In contrast, by measuring stressful events – that are time-locked - only effects of single stressful events at a time are measured, pertaining only to the here and now. Moreover, stressful events might not necessarily reflect highly personally relevant events, and therefore not lead to any somatic complaints, or, for that matter, worrying.

Furthermore, in line with previous studies, stressful events and worry intensity independently predicted the level of daily negative affect (Watkins, 2008). However, daily negative affect did not mediate or moderate the relation between worry intensity and somatic complaints. Yet, both worry intensity and negative affect were independently associated with the number of somatic complaints. Although worry and negative affect are both signs of prolonged effects of stressful events, the present results suggest that they are associated with the reporting of somatic complaints via separate routes. Worry has been shown to be closely associated with prolonged stress-related physiological activity in daily life and might lead to somatic complaints via such a route. The physiological effects of negative affect, independent of worry and stressors, are less clear (e.g. Pieper et al., 2007). Negative affect might lead to complaints by biasing information processing towards the detection of bodily sensations and interpreting these sensations as threatening or harmful (Petrie et al., 2004; Rief & Broadbent, 2007). This suggestion has to be examined in future studies. What is more, we have previously shown that one specific form of worrying, that is, worrying about ones health, is also associated with enhanced memory for health related information and that this was associated with heightened levels of somatic complaints (Verkuil, Brosschot, & Thayer, 2007a), suggesting that worry itself can lead to somatic complaints via different routes. Future studies should therefore distinguish between more general worries about stressful external events and worry about internal bodily sensations.

Interestingly, of the measures used to capture worry in daily life, worry intensity was most strongly associated with somatic complaints and explained the effects of worry frequency and worry duration on somatic complaints and negative affect. Although worry frequency, duration and intensity were highly correlated (*r*s > .85), we expected that worry duration would be associated with the number of somatic complaints, which was also found by Brosschot and Van der Doef (2006).

Unexpectedly, worry duration was not only related to complaints more weakly than worry intensity, but also than worry frequency, compared to the Brosschot and Van Der Doef study. One explanation may be that in this latter study, total worry duration was estimated by participants at the end of the day, whereas in this study worry duration was measured at random intervals during the day by asking participants to choose from several answer options on their PDA. As the duration of very intense worry episodes might be better recalled at the end of the day, compared to mild worry episodes that possibly occur relatively automatically, the worry duration measure by Brosschot & Van der Doef (2006) might have been more a measure of worry intensity, than a precise measure of worry duration. Yet, this speculative suggestion has to be addressed in future studies on how to best capture worry in daily life.

There are several limitations to this ambulatory study. Although it is prospective, its correlational nature precludes the definite conclusion that worry actually causes somatic complaints. Yet, the intervention study by Brosschot and Van der Doef (2006) has already provided beginning evidence that this is likely the case. Secondly, in this study we measured somatic complaints but did not assess objective illness and absence from work. Thus, it still seems important to investigate what factors predict why people eventually decide to actually not attend work. Thirdly, we only focused on worry in this study, whereas the perseverative cognition hypothesis pertains to a broader range of cognitive representations of stressors, such as negative intrusive thoughts and ruminative thoughts. In addition, as a large part of cognitive processing occurs without conscious awareness, it is likely that stress-related perseverative thoughts occur unconsciously too. It might underlie the effects of negative affect in this study, since it is likely that some form of stress-related cognition must have prolonged negative affect. Unconscious perseveration, such as hypervigilance for threat, cannot be measured using explicit measures such as verbal reports, but warrants the use of implicit measures such as the Implicit Association Test (IAT) or the dot probe task (Mathews & MacLeod, 1994). Future studies could benefit from including portable instruments to measure these types of perseverative cognition. On a related note, previous studies have shown that daytime stressors and worries have prolonged effects during sleep (Hall et al., 2004; Brosschot et al., 2007). This seems to suggest that a part of (unconscious) perseverative cognition takes place during sleeping. This, reduced recovery from stress during sleep might create a vicious cycle in which worry influences recovery during sleep which in turn amplifies the level of experienced stress and worries the next day. Although there were no substantial effects of reduced sleep quality, this might not be very relevant, since these physiological effects during sleep seem also not dependent on sleep quality (Hall et al., 2004; Brosschot et al., 2007). Thus, it is still possible that an even greater portion of somatic complaints can be explained by perseverative cognition that lingers on, together with its physiological effects, during sleep. As still little is known about the role of sleep in the link between stress – worry and somatic

complaints, while sleep is clearly the largest natural restorative period in normal human life, it seems extremely worthwhile to search for ways to investigate the role of perseverative cognition during sleep in future studies.

In sum, in this study evidence was provided that stressful events as well as worry are prospectively associated with somatic complaints and that this effect is mediated by worry intensity. Furthermore, worry intensity was associated with somatic complaints, independently from negative affect. Future studies testing the effectiveness of worry interventions in people at risk for the development of severe somatic complaints are clearly warranted.

Chapter 5

Pretreatment of worry enhances the effects of stress management therapy: a randomized clinical trial

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Manuscript under review

Abstract

In this randomized trial it was tested whether a two-week worry postponement and disengagement intervention (WPD) reduces work stress symptoms and whether it enhances stress management therapy (SMT). WPD effectiveness was investigated in sixty-two outpatients, suffering from adjustment and unspecified somatoform disorders, awaiting SMT provided at a mental health center. Twenty-two patients received WPD two weeks before the onset of SMT. Immediate and additive effects of WPD were compared to "worry registration-only" (N = 15) or to "waiting list control" (N = 25). Although short term effects on somatoform, anxiety and depressive symptoms were not significant, WPD added to SMT effectiveness. Decreases in nighttime worry and work stress symptoms after SMT and at follow-up were substantially more pronounced in the WPD condition. Compared to waiting list, WPD tended to induce decreases in pathological worry during SMT. In conclusion, a brief worry intervention that can be administered by psychologists and occupational physicians may be effective in reducing work stress and may enhance the effects of subsequent SMTs.

Introduction

It has been known for a long time that stressful work situations are associated with huge personal suffering and place a high economical burden on society due to absenteeism, loss of productivity and the use of health care systems (for reviews see: Van Der Doef & Maes, 1999; Michie & Williams, 2003). As has already been proposed by early stress-researchers (Selye, 1951) stress can only affect our health when it is sustained for too long. Prolonged stress responses, and not or not so much acute stress responses are the crucial link between stressors and later mental (McEwen, 2003; Thayer & Lane, 2000) as well as somatic problems (Selye, 1951; Ursin & Eriksen, 2004; Linden, Earle, Gerin, & Christenfeld, 1997; Brosschot, Gerin, & Thayer, 2006). In recent years, worrying about the stressful situations has been proposed to be one of the central pathological mechanisms between the experience of stressful situations and poor mental and somatic health (Brosschot et al., 2006; Watkins, 2008).

According to the perseverative cognition hypothesis (Brosschot et al., 2006), worrying about (work) stressors prolongs the total amount of time that these stressors adversely affect physiological and emotional functioning. Several studies provide evidence for this hypothesis. For example, ambulatory studies have shown that worrying about work stress is related to heightened cortisol levels (Schlotz, Hellhammer, Schulz, & Stone, 2004) and with heightened heart rate (Pieper, Brosschot, van der Leeden & Thayer, 2007). In addition, people who keep on worrying about their work after the workday have more difficulties 'unwinding' and suffer from more emotional and somatoform symptoms (Geurts & Sonnentag, 2006; Sonnentag, Binnewies, & Mojza, 2008) than those who do not. Moreover, prospective studies have provided evidence that the tendency to worry about things and the inability to 'unwind' or disengage after work is associated with cardiovascular morbidity (van Amelsvoort, Kant, Bultmann, & Swaen, 2003; Kubzansky et al., 1997) and even mortality (Kivimaki et al., 2006).

Accordingly, when trying to reduce the negative effects of stressful work situations on psychological and somatic health, interventions seem needed that are able to reduce worry about work in order to minimize the total 'wear and tear' (cf McEwen, 2003) that these situations can have. This focus on preventing prolonged stress responses is in line with Guidance on work related stress by the European Commission which states that: "*Stress is inevitable. What is not inevitable is prolonged, recurrent and/or intense distress*" (p. 79, Levi, 2000). In the present study we tested the effectiveness of a short and easy to administer guided self help intervention aimed at reducing the total time spent worrying in people suffering from work related stress. This intervention might specifically be suited to be administered by occupational physicians and general practitioners, who are the first points of contact for people suffering from work stress and typically have limited time to manage work stress complaints. This intervention, called 'worry postponement and disengagement'

is a so-called 'stimulus control intervention', and requires people to reschedule their day and nighttime worry episodes to a specific moment of the day during which worrying is allowed. This intervention is part of the cognitive behavioral treatment for people suffering from generalized anxiety disorder, of which chronic worry is the main feature, and has previously been found to be effective in reducing worry and its associated tension (Borkovec et al, 1983) and somatoform symptoms (Jellesma, Verkuil, & Brosschot, 2009; Brosschot & Van Der Doef, 2006) in relatively healthy subjects. However, it remains unclear whether this simple intervention is also effective in reducing worry and somatic and emotional complaints in people suffering from work stress.

This study was designed to test the short term effects of this worry intervention on the duration and frequency of worry episodes in people suffering from work stress, diagnosed according to the DSM-IV with adjustment disorder or undifferentiated somatoform disorder. We tested whether the intervention is effective in reducing work stress–related somatoform, anxiety and depressive symptoms which have been shown to be caused by worry (Watkins, 2008). Moreover, we also tested if and to what extent this short intervention - aimed as it is at a crucial prolongator of work stress, i.e. worry - adds to a typical cognitive behavioral based stress management therapy (SMT) of people suffering from work stress. The tendency to worry has been suggested to be an important mediator of the treatment effects of CBT and mindfulness meditation and a reduction of worry might be a prerequisite for CBT to be fully effective. However, empirical evidence is still scarce and previous studies have found mixed results for the mediating role of worry in the context of CBT in general as well as for work stress (Jain et al., 2007; Ciesla & Roberts, 2002). If the worry intervention appears to have positive effects in this study, either on itself or by boosting subsequent SMT, or both, it might offer general practitioners, occupational physicians and psychotherapists an alternative brief and easy-to-use intervention, or at least an enhancer of their standard treatment. In addition, it could make clear that interventions aimed at crucial mediators of CBT, such as worrying or biased attention - which can also be retrained (e.g., Hazen, Vasey, & Schmidt, 2009) enhances the effects of standard, more intensive, CBT.

In short, we tested the following hypotheses: the worry postponement and disengagement intervention will: (1) Be effective in reducing the total time spent worrying; (2) Be effective in decreasing somatoform, anxiety and depression symptoms; (3) Add to the effects of a regular CBT-based SMT on somatoform, anxiety and depression. Finally (4), we expect these latter two effects to be mediated by a reduction in worry.

Method

Subjects

The study took place at PsyQ Business, a psycho medical institution in The Hague, a division of one of the largest organizations for mental health care in the Netherlands. The institution is specialized in the treatment of psychopathology that is due to or affects stress at work. In general, patients are referred to this institution by their general practitioners or occupational physicians in order to follow stress management therapy (SMT). During a first interview with a clinical psychologist, patients are initially screened according to DSM-IV criteria for psychological disorders and a team of clinical psychologists thereafter proposes a subsequent treatment program. Patients were asked to participate in the present study when (1) they were referred to the SMT, based on a DSM-IV axis I diagnosis of either adjustment disorder, unspecified somatoform disorder (burnout), or severe work problems (axis IV) and (2) when they had to wait at least two weeks before starting with this SMT in order to be able to implement the pretreatment intervention. In these patients, we were interested in finding strong effects, that is, d >.80 (Cohen, 1988). To find such an effect we needed 63 patients. Excluded from the SMT, and therefore this study, were participants with substance abuse as the primary axis I diagnosis, serious medical conditions, organic psychiatric disorders, severe suicidality or a history of schizophrenia. All participants gave written informed consent before entering the study. No financial incentives were given and treatment costs were covered by mandatory insurance for mental health or by the employers of the participants. The study was approved by the Medical Ethical Committee of PsyQ.

Procedure

Participants who were willing to participate were invited at the institution for the first baseline measurement. During this session participants provided informed consent and completed the symptom questionnaires. Thereafter, participants were randomly allocated to one of three conditions: the Worry Postponement and Disengagement intervention (WPD) or to one of the two control conditions, that is, the Registering of Worry (WR) or a waitlist control condition, Treatment As Usual (TAU). Randomization was performed by the researchers in separate blocks (each consisting of 2 – 6 participants) by opening blinded envelopes in which the conditions were concealed in advance. Participants in the WPD and WR conditions then received the appropriate intervention. Two weeks after this baseline measurement, the SMT started.

Instruments

As the worry intervention has been previously shown to reduce somatoform symptoms, the primary outcome in this study was the total number of somatoform symptoms, assessed with 27 items of the Dutch version of the Subjective Health Complaints questionnaire (SHC; Eriksen, Ihlebaek, & Ursin, 1999; the original version contains 29 items, but two items measuring anxiety and depression were removed before analysis). Secondary outcome measures were Dutch version of the State Trait Anxiety Inventory - Trait version (van der Ploeg, Defares, & Spielberger, 1980), measuring anxiety symptoms, and the Dutch version of the Beck Depression Inventory – Second version (BDI-II; van der Does, 2002), measuring symptoms of depression. Participants were asked to complete the outcome questionnaires before the start of the SMT (2 weeks after the baseline assessment), at the end of the SMT (14 weeks after the baseline assessment) and after a follow-up period of three months (26 weeks after the baseline assessment). In addition, to test whether changes in worry mediated the direct effects of the worry pretreatment on the outcomes we asked participants in the WPD and WR conditions to keep a log of the frequency and duration of their worry episodes (Verkuil, Brosschot, & Thayer, 2007) and to return these before the start of the SMT. Furthermore, they were asked to report to what extent the worry intervention (worry postponement or worry registration only) had been helpful in reducing worries on a ten-point scale. To be able to test whether the pretreatment of worry also reduced the level of pathological worry during the subsequent SMT and whether this mediated the treatment effect on the outcomes we also administered the Penn State Worry Questionnaire (PSWQ) at baseline and at the end of the SMT. Questionnaires were sent to the participants via mail and could be returned using prepaid envelopes.

Worry intervention conditions

Worry postponement and disengagement (WPD)

Participants in this condition were provided with information on the functions of worry during a meeting that lasted approximately 30 - 45 minutes. More specifically, it was explained to them that worry can be regarded as a thwarted problem solving strategy and that worrisome problem solving while one is supposed to do other things (e.g., job demands) is likely to be unsuccessful. Participants also received a booklet containing the information and exercise described below. The intervention consisted of two parts. The first part concerned managing worry episodes that occurred during daily life. They were asked to deal with these naturally occurring worry episodes by: (a) becoming aware of the fact that they were worrying by keeping a log of their worries, and by (b) disengaging from their worries and postponing them to a moment later that day, a so called worry half-an-hour. The

postponement of worry episodes was based on the protocol developed by Borkovec et al. for the treatment of pathological worrying (Borkovec, Wilkinson, Folensbee, & Lerman, 1983; Brosschot & Van Der Doef, 2006) and forms part of the manual for stress counseling available to occupational physicians (van der Klink & van Dijk, 2003), but is believed to be seldom used by them. The second part of the intervention concerned the worry half-an-hour. During this period participants were instructed to deal with the registered problems that they had been worrying about during the day by (a) deciding whether the problems concerned issues that they could influence or control themselves, or whether the problems concerned issues (temporarily) out of their control. To help guiding this decision, they were asked to write down several problem solving steps (i.e., a problem description, the kind of solutions that they had already tried out, possible alternative solutions and, finally, their decision to either try to solve the problem or disengage from it). Guided writing about worry problems has previously been shown to be effective in reducing anxiety symptoms (Bowman, Scogin, Floyd, Patton, & Gist, 1997). If the problems could be managed (b), they were encouraged to plan when to implement the solution and to subsequently put this into practice. If they decided that a solution could not be implemented and that they had to disengage from the worry topic (c), they were instructed to practice with an exercise in (temporarily) disengaging from the worry problems, which is based on a worry reduction protocol developed by Korrelboom (Competitive Memory Training; Korrelboom, Van der Gaag, Hendriks, Huijbrechts, & Beretty, 2008). For this exercise participants had to recall moments in their lives during which they had realized that they had disengaged from a previously worrisome problem. They then practiced with thinking about the current worrisome problem while simultaneously recalling the disengagement experience from memory. More details on the whole WPD intervention can be provided by the authors on request. After one week, participants were called to remind them about the registration of worry and to inquire about whether they had any trouble with putting the worry intervention into practice. No interventions were given during the calls, which lasted a maximum of five minutes.

Worry registration (WR)

To control for the possible beneficial effects of getting extra attention and of the effects of becoming aware of worry episodes (step 1 as described above), participants in the worry registration condition were asked to keep a log of their worries, based upon the rationale that worry is a habit and that when one wants to change ones behavior, becoming aware of its manifestation is the first step to take. After one week, the participants were also called to remind them about the registration of worry and to inquire about whether participants had any trouble with putting the worry registration into practice.

Treatment as usual (TAU)

To control for effects of time, a third group of participants were told that they had been randomized to wait for the start of the stress management therapy. They did not receive any extra treatment or attention during this period.

Stress management therapy (SMT)

After two weeks, participants started with the stress management group therapy. Each group consisted of about eight people. The therapy consisted of 12 weekly sessions taking two hours each. The therapy groups were led by two out of four experienced clinical psychologists that were blind to the pre-SMT conditions that the participants were allocated to. The therapy consisted of a combination of psycho-education and cognitive behavioral psychotherapy. In their review of work stress interventions, Van der Klink et al. found that such cognitive behavioral SMTs had a moderate effect on symptoms of anxiety and depression, with a mean effect size of Cohen's d = .55 (Van der Klink, Blonk, Schene, & Van Dijk, 2001), whereas effects on somatoform symptoms are mixed (e.g., Eriksen et al., 2002). During the first phase of the therapy participants worked at reducing their complaints by restoring their energy-imbalance, mostly by actively planning relaxing activities and by monitoring of their energy levels. Subsequent phases of the therapy focused on promoting assertiveness skills and practicing cognitive therapy to teach participants how to identify and correct dysfunctional beliefs. During the course of the study, therapists were allowed to provide cognitive interventions aimed to reduce worrying, such as by using cognitive restructuring techniques or by using the Socratic dialogue. However, they were instructed not to provide interventions aimed at the temporal dynamics of worrying, especially not the worry registration or postponement and disengagement interventions. This was done in order to keep the most important manipulation of the pre-SMT worry intervention as pure as possible while at the same time allowing typical and potentially probable effective ingredients of the therapy to remain intact and to keep this group therapy as standard as possible. A protocol of the SMT can be provided by the authors on request.

Statistical analysis

Multilevel growth curve models were used to analyze differences between the pretreatment conditions in the development of the outcome measures over time (Singer & Willett, 2003). Multilevel analysis (MLA) is especially suitable to analyze repeated measures data because it accounts for the dependencies of the different measurements (level 1) that are nested within individuals (level 2). Another advantage is that multilevel growth curve analysis allows for individual time curves to be estimated on all available data from each individual and can handle unbalanced datasets that contain irregularly spaced measurement intervals. Analyses were performed on the

intention to treat sample. Missing values on the questionnaires were imputed using the algorithm provided by Van Ginkel & Van der Ark (2005).

At the first level, the effect of time was examined. When analyzing the worry diary data, time reflected the 14 days of the worry registration period. When analyzing the treatment outcome measures, time reflected the four time points (expressed in number of weeks since start of the experiment) at which the outcome measures were administered, coded as 0 (baseline), 2 (end of experimental phase / start of SMT-treatment), 14 (end of SMT) and 26 (follow up). The second level of measurement was the individual level, as the time series were nested within the different individuals. To test the hypotheses, multilevel regression models were estimated for all outcome measures, allowing for individual variation in regression intercepts and, when it improved the fit of the model as assessed with -2 log likelihood tests, individual variation in regression slopes. In the present study two a priori contrasts tested the following null hypotheses: (1) no difference between Worry Postponement and Disengagement (WPD) and Treatment As Usual (TAU), (2) no difference between WPD and Worry registration (WR). Significant interactions were explored further with t-tests and correlation analyses. Additionally, between condition effect sizes were calculated (Cohen, 1988) as well as the percentages of participants that showed clinically significant and reliable changes (Jacobson & Truax, 1991). Due to our unequivocal expectations we used one-tailed significance tests. The mediation hypothesis was examined using the guidelines provided by Baron and Kenny (1986). Significance of mediation effects were tested with Sobel tests.

Results

Sample characteristics and drop-out

Sixty-three patients decided to participate in the study (for descriptive statistics see Table 1). Several participants did not return their questionnaires at the follow up measurements: ten at T2, twelve others at T3 and two others at T4 (see Figure 1). In addition, one participant in the WR condition stopped with the SMT because another treatment was indicated (marital counseling). Chi square tests showed that there were no significant differences between the conditions in the total number of participants that left the study at T1 ($\chi^2(2) = .39$, $p = .82$), T2 ($\chi^2(2) = 1.33$, $p = .52$) or T3 ($\chi^2(2) = .27$, $p = .88$). There were no significant baseline differences between the treatment groups in scores on the SHC, STAI, BDI-II or PSWQ. There were also no significant differences between the conditions in DSM-IV diagnoses, the number of attended treatment sessions and the subjective rating to what extent the therapy was rated as helpful. Descriptive statistics of the questionnaires are provided in Table 2. To give an impression of the severity of somatoform complaints in this sample: In normative samples symptoms are seldom scored by more than 50% (e.g., Eriksen et al., 1999), while in the current sample the following six symptoms were reported by more than half of the participants,:

fatigue (91.9%), sleeping difficulties (74.2%), lower back pain (64.5%), headache (66.1%), shoulder pain (54.8%) and neck pain (53.2%). Scores on the STAI-T, BDI-II and PSWQ were similar to levels observed in clinically anxious and dysphoric outpatients (Startup & Erickson, 2006; Dozois, Dobson, & Ahnberg, 1998).

Table 1. *Descriptive statistics*

	TAU (N = 25)		*WR (N = 15)*		*WDP (N = 22)*				
	M	*SD*	*M*	*SD*	*M*	*SD*	χ^2 *(df)*	*F (df)*	*p*
Female %	60		40		59.1		1.76 (2)		.414
Marital status, %							3.87 (6)		.695
Married	36.00		21.43		42.86				
Living together	48.00		14.29		19.05				
Divorced	4.00		0.00		4.76				
Unmarried	12.00		64.29		33.33				
Education, %							.60 (6)		.996
Secondary school	8.33		13.33		14.29				
Lower education	54.17		53.33		47.62				
Higher education	29.17		26.67		28.57				
University	8.33		6.67		9.52				
DSM-IV axis I classification (%)									
Adjustment disorder	40.0		73.3		54.4				
Undifferentiated somatoform disorder	12.0		6.7		9.1				
Depressive episode	12.0		-		-				
Anxiety disorder	-		-		4.5				
None reported[a]	12.0		20.0		31.8				
Antidepressant medication at baseline, %	12.00		6.67		22.72		2.07 (2)		.356
Age, years	45.00	8.31	39.80	8.02	40.91	7.54		2.504 (2)	.090
Number of cigarettes p/w	4.32	7.20	5.70	9.02	3.70	7.59		0.296 (2)	.745
Alcoholic beverages p/w	3.48	6.31	6.64	6.50	4.68	7.05		1.022 (2)	.366
Total hours of exercise p/w	1.96	2.32	1.87	2.80	2.50	3.36		.296 (2)	.745

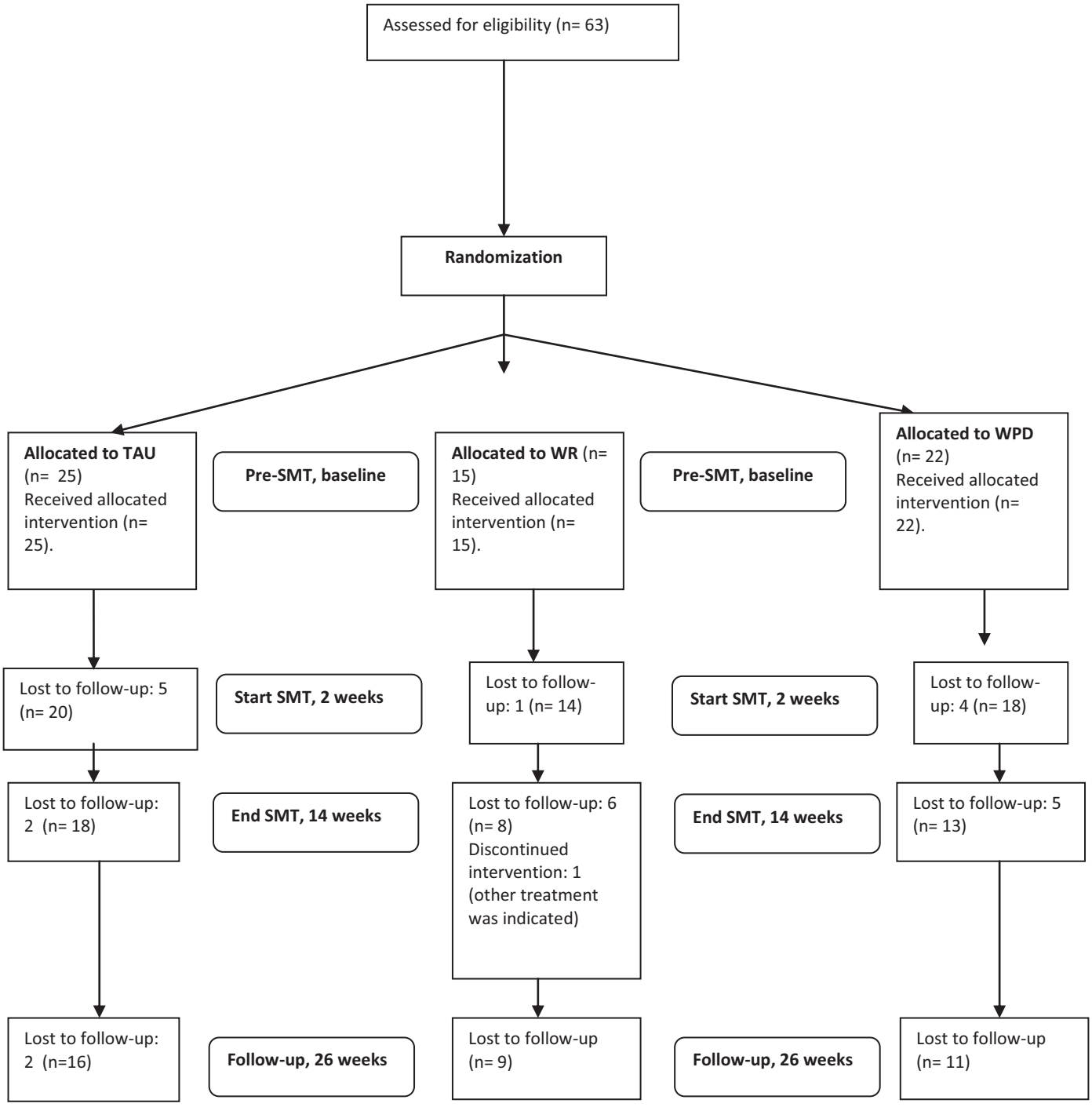
Note: [a] Not all participants had received an axis I diagnosis, and some diagnoses at the start of the stress management therapy were not reported in the patients files. These patients had for example been successfully treated for a DSM-IV axis I diagnosis in a previous treatment but still suffered from work stress symptoms and participated in the SMT due to severe work problems (reported on axis IV); WPD = worry postponement and disengagement intervention; TAU = treatment as usual; WR = worry registration

Table 2. *Primary and secondary outcome variables means and standard deviations at baseline and follow-ups*.

		Time in weeks											
		Baseline			Start therapy (2 weeks)			Follow up (14 weeks)			Follow up (26 weeks)		
		N	M	SD	N	M	SD	N	M	SD	N	M	SD
SHC	TAU	25	9.04	4.36	19	8.53	3.84	18	8.44	4.95	16	8.88	5.11
	WR	15	8.20	4.26	14	8.14	4.02	8	6.50	4.31	9	6.78	5.74
	WPD	22	9.14	4.06	18	8.39	3.96	13	7.15	5.52	13	6.62	4.70
STAI-T	TAU	25	52.56	10.15	20	50.10	10.60	18	43.78	10.03	16	42.19	8.78
	WR	15	54.53	8.98	14	54.21	10.39	8	42.50	9.29	9	43.56	10.14
	WPD	22	52.91	9.53	18	50.89	9.63	13	38.92	9.87	13	40.62	13.12
BDI-II	TAU	24	18.42	9.23	20	17.05	9.66	18	9.89	8.13	16	11.69	7.12
	WR	15	21.67	9.32	14	22.29	10.61	8	11.25	7.36	9	10.89	7.99
	WPD	22	18.82	8.17	18	17.17	10.18	13	8.54	7.53	11	6.82	7.15
PSWQ	TAU	25	53.04	11.47				18	45.83	11.27			
	WR	15	59.33	11.49				8	49.75	12.13			
	WPD	22	56.04	9.11				13	42.85	8.42			

Note: SHC = Subjective Health Complaints; BDI-II = Beck Depression Inventory, Second Edition; STAI = State Trait Anxiety Inventory-Trait version; PSWQ = Penn State Worry Questionnaire; TAU = treatment as usual; WR = worry registration; WPD = worry postponement and disengagement intervention;.

Figure 1. Overview of the study



Effects of worry pretreatment on state and trait worry

Visual inspection of the worry data suggested that these could best be described by quadratic trends, which proved to be significant when tested in a baseline growth curve model. Table 3 shows the results of the MLA on the worry data for WPD and WR conditions (NB. the waitlist control group (TAU) did not yield worry data). In both groups daytime worry duration and worry frequency decreased. Although worry duration seemed to drop more in the WPD condition, there were no significant differences between the two conditions in total daytime worry duration and worry frequency, nor any interactions with the time variables. However, an overall decrease in nighttime worry was apparent and the linear and quadratic time curves for the nighttime worry data were different for the two conditions. For WPD, worry duration and frequency decreased during the first week of the intervention period, while increasing again during the second week. The reverse pattern was apparent for the registration group. T-tests showed less frequent nighttime worry episodes ($t(435) = 2.392$, p = .009; M = 1.32 episodes per night, SD = 1.67) and shorter worry nighttime duration ($t(433) = 2.671$, p = .004; M = 19.66 minutes per night, SD = 42.14) in WPD compared to WR (M = 2.04 episodes, SD = 5.51 and M = 22.24 minutes, SD = 27.91 respectively). The difference between WPD (M = 5.31, SD = 1.38) and WR (M = 4.42, SD = 2.35) in self reported deceases in worry, although in the expected direction, was not significant ($t(23) = 1.167$, $p = .128$).

Pathological worry (PSWQ) was measured at baseline and after the SMT. MLA showed that there was a significant decrease in PSWQ scores before and after SMT for all patients (B = -13.80, p < .0001, 95 % CI: -20.64 - -6.90). The difference between the decreases in PSWQ scores between WPD and TAU was marginally significant (B = 7.063, $p = .063$, 95 % CI = -2.064 – 16.191), whereas the difference between WPD and WR was not significant (B = 5.019, $p = .184$, 95 % CI = -6.113 – 16.152).

Table 3. *The effect of condition (worry registration versus worry postponement and disengagement) on worry duration and – frequency during the pre-SMT period.*

Variables	*Time*		*Condition*	*Time x Condition*	
	B(SE)		B(SE)	B(SE)	
	linear	quadratic		linear	Quadratic
Daytime worry duration	-.1766 (.0605)*	.0122 (.0043)*	.0696 (.4280)	.0392 (.0925)	-.0038 (.0065)
Daytime worry frequency	-.0668 (.0329)*	.0047 (.0023)*	.0586 (.2279)	-.0039 (.0504)	.0001 (.0034)
Nighttime worry duration	-.1414 (.0740)$^{p = .057}$	.0112 (.0053)*	-.1963 (.5220)	.3346 (.1149)*	-.0251 (.0083)*
Nighttime worry frequency	-.0601 (.0299)*	.0045 (.0022)*	-.0309 (.2031)	.1005 (.0464)*	-.0078 (.0034)*

* = $p < .05$, two-tailed

Effects of worry interventions on somatoform, anxiety and depressive symptoms

First, the time course of the somatoform, anxiety and depressive symptoms during the whole study was estimated using multilevel models. Concerning the number of somatoform symptoms (SHC), the MLA random intercept model showed that there was a significant decrease for all participants (B = -.129, $p < .0001$, 95 % CI = -.198 – - .062) in SHC from baseline to follow-up three months after the SMT. The preplanned contrasts showed that the linear decrease in symptoms from baseline to follow-up after the SMT differed significantly between WPD and TAU (B = .099, $p = .017$, 95 % CI = .008 – .190), whereas there was a trend for WPD to be more effective than WR (B = .067, $p = .109$, 95 % CI = -.039 – .173). See also Figure 2 for a graphic representation of the predicted model.

Concerning the levels of anxiety (STAI-T), the MLA random intercept model showed a significant overall decrease in anxiety (B = -.589, $p < .0001$, 95 % CI = -.789 – -.410). In addition, the linear time trend differed significantly between the WPD condition and TAU (B = .203, $p = .048$, 95 % CI = -.037 – .444), whereas the difference between WPD and WR was not significant (B = .105, $p = .229$, 95 % CI = -.176 – .386). The same pattern was found for depressive symptoms. A random intercept - random slope MLA model showed a significant overall decrease in depressive symptoms (BDI-II; B = -.495, $p < .0001$, 95 % CI = -.675 – -.314), Furthermore, the difference between WPD and TAU was marginally significant (B = .195, $p = .054$, 95 % CI = -.044 – .434) whereas the difference between WPD and WR was not significant (B = .032, $p = .409$, 95 % CI = -.245 – .309).

Table 4. *Multilevel models predicting changes in somatoform, depressive and anxiety symptoms*.

Variables	*Time*	*Condition (main effects)*		*Time x condition contrasts*		*Cohen's d*		*Cohen's d*		*Cohen's d*	
		WPD vs TAU	WPD vs WR	WPD vs TAU	WPD vs WR						
	B(SE)	B(SE)	B(SE)	B(SE)	B(SE)	2 weeks (pre-treatment)		3 months (post treatment)		follow up 3 months	
						WPD vs TAU	WPD vs WR	WPD vs TAU	WPD vs WR	WPD vs TAU	WPD vs WR
SHC	-.129 (.034)*	.071 (1.164)	-.669 (1.324)	.099 (.046)*	.067 (.054) p = .109	.13	.40	.54	.47	.65	.59
STAI-T	-.589 (.091)*	.441 (2.576)	2.800 (2.928)	.204 (.122)*	.105 (.142) p = .229	.22	.43	.95	.43	.42	.36
BDI-II	-.495 (.090)*	.070 (2.612)	3.745 (2.979)	.195 (.119) p = .054	.032 (.138) p = .409	.21	.38	.64	.26	.62	.19

Note: * p < .05, one-tailed. WP = worry postponement pretreatment; TAU = treatment as usual; WR = worry registration; SHC = Subjective Health Complaints; BDI-II = Beck Depression Inventory, Second Edition; STAI-T = State Trait Anxiety Inventory.- Trait version

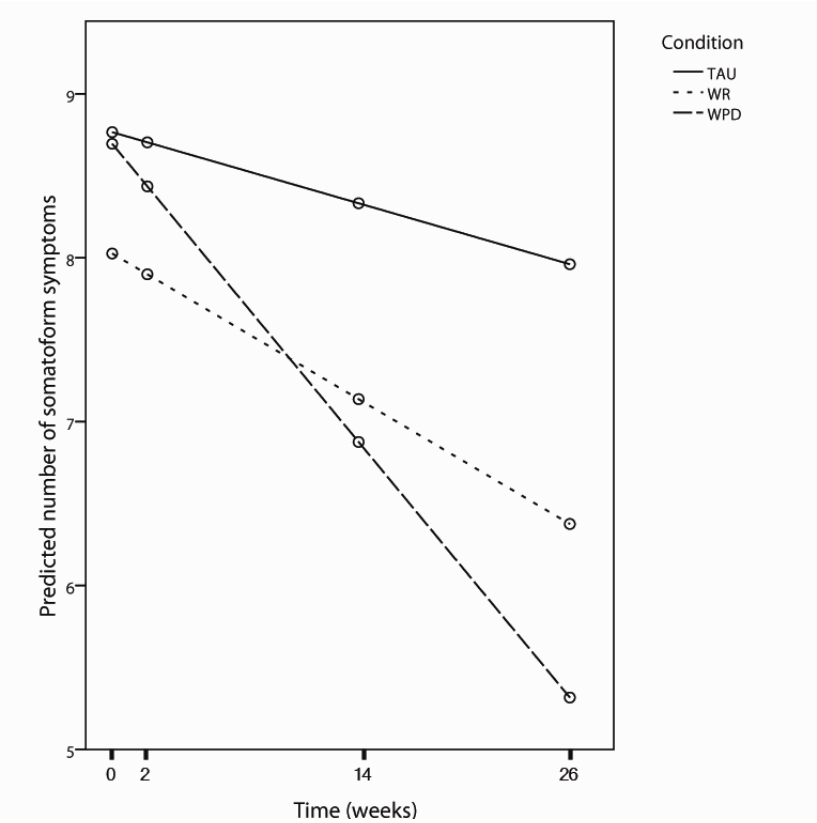


***Figure 2*.** Effects of the worry postponement and disengagement intervention (WPD) versus worry registration (WR) and treatment as usual (TAU) on somatoform symptoms.

Direct effects of worry postponement and disengagement

To examine whether these differences between the time course in symptoms between WPD and TAU were present during the two weeks before SMT, follow-up tests were conducted on data from participants in these two conditions. Concerning the number of SHC, paired t-tests showed that there was a significant decline in SHC during the two weeks of the worry pretreatment period in the WPD condition ($t(17) = 3.12$, $p = .003$), whereas the decline was not significant in TAU ($t(18) = 1.05$, $p = .15$). However, an ANCOVA with SHC at baseline as a covariate and Condition as a between subjects factor did not yield any significant differences between the conditions in SHC at two weeks. Concerning the STAI-T, there was a trend towards a significant decline in anxiety symptoms during the two weeks of the worry pretreatment period in the WPD condition ($t(17) = 1.63$, $p = .06$), which was not apparent for TAU ($t(19) = 0.827$, $p = .209$). There were no significant differences between the conditions in STAI-T scores however. Finally, there was a trend towards a significant decline in BDI-II scores during the two weeks of the worry pretreatment period in the WPD condition ($t(17) = 1.460$, $p = .081$), which was not apparent for TAU ($t(18) = 0.635$, $p = .267$). There were no significant differences between the conditions in BDI-II scores.

Additive effects of the worry postponement and disengagement on SMT effectiveness

To test the third hypothesis, that the WPD intervention would enhance the effects of the SMT on work stress symptoms, a second set of follow-up tests was conducted. Concerning the SHC, directly after the SMT, the number of somatoform symptoms was significantly lower compared to baseline in WPD ($t(12) = 2.086$, $p = .029$), whereas a trend was apparent in TAU ($t(17) = 1.383$, $p = .092$). At follow-up, the decrease in somatoform symptoms compared to baseline was significant for both conditions. ANCOVAs showed that at follow-up, but not directly after the SMT, participants in the WPD condition reported significantly less SHC than participants in TAU ($F(1,26) = 2.950$, $p = .049$). Concerning the STAI-T scores, in both conditions the level of anxiety symptoms directly after the SMT and at follow-up had significantly decreased below baseline levels (all ts > 2.5). Participants in the WPD condition reported lower levels of anxiety directly after the SMT ($F(1,28) = 3.894$, $p = .029$), but not at follow-up, when compared to participants in TAU. Concerning the BDI-II scores, in both conditions the level of depressive symptoms directly after the SMT and at follow-up had significantly decreased below baseline levels (all ts > 3.5). At follow-up but not directly after the SMT, participants in the WPD condition had significantly less depressive symptoms than participants in TAU ($F(1,23) = 2.964$, $p = .049$).

Effect sizes

Raw change scores in symptoms from baseline to the follow up measurements were used to calculate between group effect sizes (Table 4). Effect sizes of .20 indicate small effects, effect sizes of .50 indicate medium effects and effect sizes of .80 show large effects (Cohen, 1988). From Table 4 it can be derived that, while not all differences between the conditions were significant, most effect sizes were small to medium, and there was a large effect for the difference between WPD and TAU in the level of anxiety symptoms directly after the treatment.

The clinical significance of the results was determined by examining the percentage of participants that showed reliable reductions in the outcome variables that went below clinical cut-off points (Jacobson & Truax, 1991). Reliable change was calculated on the basis of the formula provided by Jacobson and Truax (1991) with Cronbach's alphas as the indices of questionnaire reliability. The numbers of participants that showed reliable changes below clinical cut-off points were compared between the conditions with exact chi-square tests. Cut-off points were determined as follows: STAI-T: 46 (Fisher & Durham, 1999); BDI-II: 12 (Dozois et al., 1998), PSWQ: 45 (Behar, Alcaine, Zuellig, & Borkovec, 2003). As there were no such data available for the total number of SHC, clinically significant reductions in SHC scores were determined by outcome scores that fell two standard deviations below baseline scores (i.e. a reduction of 8 complaints). Paired comparisons between the number of clinically significant and reliably changed participants among the three conditions showed

a significant difference between WPD and TAU in clinical change on the STAI scores directly following the SMT. Eight participants (62%) in the WPD condition showed a clinical change, compared to four (29%) in TAU ($\chi^2(1, N = 31)$ 4.918, $p = .032$). The difference between WPD and WR, in which two participants (25%) showed clinical change, was not significant, ($\chi^2(1, N = 21) = 2.651$, $p = .119$). No other differences between the conditions were apparent.

Overall, the percentages of participants realizing clinical and reliable change during the two week pre-SMT period were: 0% (SHC), 12.5% (STAI-T) and 3.1% (BDI-II). Directly after the SMT and at follow-up, the percentages of reliable changes were: 9.5% and 9.1% (SHC), 47% and 50% (STAI-T), 38% and 40% (BDI-II) and 33% (PSWQ measured only after SMT).

Mediating effects of momentary assessed and trait worry on treatment outcome

Our next hypotheses concerned whether changes in worry would mediate the effects of the WPD intervention on changes in anxiety, depressive and somatoform symptoms. As symptoms levels during the course of treatment did not significantly differ between WPD and WR conditions, it was therefore irrelevant to test whether changes in daily worry during the two intervention weeks mediated the differential effects of these worry intervention. Yet, correlation analysis within these conditions indicated that reductions in the number of nighttime worry episodes were associated with reductions in SHC ($r(28) = .35$, $p = .043$), whereas reductions in the duration of the nighttime worry episodes were associated with reductions in BDI-II ($r(24) = .37$, $p = .036$) and STAI ($r(24) = .36$, $p = .043$). When controlling for reductions in the other outcomes, however, these associations became non-significant, suggesting that reductions in daily worry had a relatively small independent contribution to reductions in work stress symptoms. In addition, self reported change in worry due to the worry interventions was associated with changes in SHC ($r(25) = .39$, $p = .027$) and BDI-II ($r(25) = .57$, $p = .001$). Thus, people who reported to have been worrying less during the 2 weeks before the SMT also reported lower levels of somatoform and depressive symptoms.

In addition, although differences between WPD and TAU in PSWQ scores after the SMT were only marginally significant (i.e. the first step of the mediation analysis according to Baron and Kenny (1986)), we decided to explore whether PSWQ-Change mediated the effects of the WPD condition on the outcomes. Although the effects of Time*PSWQ-Change on the outcomes were significant and slightly reduced P levels of the Time*Condition effects, Sobel tests showed no significant mediation effects.

Discussion

This study examined the effectiveness of a simple and easy to administer worry intervention, i.e. worry postponement and disengagement (WPD), in reducing worry and its associated symptoms of work stress. Thus, it was hypothesized that this worry intervention would not only reduce worry but also symptoms of work stress, theoretically because it would reduce the total load on mind and body produced by worrying. It was investigated whether the WPD intervention would be effective by itself at the short term (within two weeks), or whether it would enhance the effectiveness of a subsequent stress management therapy (SMT), or both. In addition, it was hypothesized that reductions in (pathological) worry would mediate reductions in work stress symptoms and the effects of the SMT on work stress symptoms. The results partially confirm our hypotheses.

First of all, we found that the WPD intervention, but also the registering of worry episodes (WR), led to decreases in daytime and nighttime worry. Importantly, the WPD intervention was specifically associated with decreases in the frequency and duration of nighttime worry episodes. With regard to the work stress symptoms, the WPD intervention did not have significant short term effects, although these symptoms tended to deviate from baseline more strongly in the WPD condition. The most important and innovative finding from this study was that during the whole course of the study, participants who had received the WPD intervention showed the largest decreases in somatoform, anxiety, and - to a lesser extent - depressive symptoms. This was most apparent when the effects of WPD were compared to the effects of a waitlist control group who had received no intervention before the SMT, and thus only treatment as usual (TAU). Significant differences between the conditions were found in symptom levels directly after the SMT and at a follow-up of three months. Directly after the SMT, participants in the WPD condition reported less symptoms of anxiety compared to TAU, and at a three month follow-up measurement they also reported less somatoform and depressive symptoms. To our knowledge, this is the first study to show that a 'pretreatment' intervention directed at a crucial pathogenic process, in this case worry, enhances the effectiveness of a standard cognitive-behavioral therapy. This finding is even more important with respect to the somatoform effects, since previous studies showed no effects of SMT on somatoform symptoms (e.g., Eriksen et al., 2002; Tveito & Eriksen, 2009). Since these symptoms are associated with high health care costs and long term sickness absence, is it encouraging that adding a time-limited and simple worry intervention potentially can enhance the somatic effects of such a standard SMT.

Especially nighttime worries could be considered as a pathological feature of worry and were, as mentioned, reduced by the WPD intervention. Nighttime worries likely indicate difficulties in disengaging from work and other stressful events. As such, nighttime worries might play a crucial role in the link between stress / worries and well being, for example by interfering with physiological

recovery during sleep (Brosschot, van Dijk, & Thayer, 2007). Interestingly, reductions in nighttime worry were associated with reductions in work stress symptoms.

A point that requires some future attention is that the difference between the effects of WPD and mere registering of worries (WR) on symptoms was not significant. Although this is most likely due to our small sample size as the differences were in the expected direction, it leaves open the interesting possibility that the effects of WPD could be largely attributed to becoming more aware of one's worries. If this is the case, than this might indicate that an even more simple intervention than WPD could already be effective in enhancing SMT. Another point pertains to our mediation hypothesis. Contrary to our expectations, we did not find any mediating effects of pathological worry and the data did not permit us to test mediating effects of momentary assessed worry, because this variable was not available for participants in the TAU condition. As such, the precise temporal mechanisms underlying reductions in work stress symptoms during the course of the SMT remain indistinct. Follow-up studies are warranted that investigate this issue more thoroughly, for example by measuring worry and work stress symptoms more frequently during the course of treatment.

While the results of this study are encouraging and hopefully stimulate more research into the short term and additive effects of short and easy to administer interventions, there are some limitations. First, the time period for our follow-up measurement, three months after the SMT, was relatively short and it is not clear to what extent the results pertain to longer follow-up periods. With respect to its clinical relevance, one could argue that it is a weakness of this study that participants were not screened on psychopathology using structured clinical interviews like the Structured Clinical Interview for DSM Disorders (SCID). However, the most common DSM-IV classifications to diagnose clinical forms of work stress are 'adjustment disorder' and 'unspecified somatoform disorder', which remain controversial (Mayou, Kirmayer, Simon, Kroenke, & Sharpe, 2005). Moreover, the use of the present convenience sample adds to the generalizability of the results of the study for at least two reasons. First, the inclusion of this sample closely resembles usual clinical practice in which inclusion criteria are often less strict than in randomized controlled trials aimed at specific psychopathologies. Second, the sample represents a large part of the population that suffers from somatoform, anxiety and depressive stress symptoms which are known to be highly comorbid. With respect to the effects of the WPD intervention, an alternative explanation for its effects that cannot be ruled out is that these were partially due to attention. Participants in the WPD and WR conditions received more attention from psychologists before the start of the SMT, as they were called up after one week to check whether there were any problems with the worry registration. Future studies that compare the effectiveness of WPD to interventions not primarily focused on worrying are needed to test these suggestions. Another limitation is that in testing the effects of WPD on worry, we focused merely on

the frequency and duration of worries, but not on the content of worries. It would be interesting to examine whether WPD stimulates another way of thinking about problems. For example, a recent study showed that manipulating the concreteness of worrisome thinking causes changes in depressive symptoms (Watkins & Moberly, 2009). Although the present intervention is mainly aimed at limiting the total amount of wear and tear that worry episodes can have on the body (postponement and disengagement), we also asked participants to write down their worry problems, which might have enhanced a more concrete thinking style that reduced nighttime and pathological worrying. Finally, this study focused on outpatients suffering from work stress and it remains unclear to what extent these findings extend to other populations. For example, it remains unclear whether the findings extend to cognitive behavioral therapies for anxiety and mood disorders, and whether the additional effects of WPD are limited to subsequent *group* interventions. On a broader scale it might be interesting to test whether worry interventions are effective in preventing severe forms of work stress, for example in workers vulnerable for developing work stress (e.g. teachers, nurses). This is in line with recent calls for the promotion of self help strategies to reduce stress among the general population (Jorm & Griffiths, 2006).

Notwithstanding these limitations, this study is the first to show that a simple guided self help intervention helps reducing worry, especially nighttime worry and that it enhances the effects of a subsequent SMT on both mental and somatic symptoms of work stress. Since work stress symptoms form a major humanitarian and economic burden, and are also a vulnerability factor for the development of severe conditions such as cardiovascular disease and mental health problems, further testing of the effectiveness of simple interventions that aim to target mediators of psychological treatments is recommended.

Chapter 6

Effects of explicit and implicit perseverative cognition on cardiac recovery after cognitive stress

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Abstract

Slow cardiovascular (CV) recovery after stress is a predictor of adverse CV outcomes. Perseverative cognition (PC) about stress has been hypothesized to co-determine slow recovery. In the present study, it was investigated whether two types of trait PC, i.e. trait worry and trait rumination, predicted delayed cardiac recovery after a cognitive stressor. Furthermore, it was examined whether explicit state PC (i.e. negative intrusive thoughts) or implicit state PC (i.e. automatic vigilance) additionally predicted delayed cardiac recovery.

Fifty nine participants performed a stressful task, which consisted of an unsolvable synonym task. After a 6-minute recovery period, participants reported on their level of negative intrusive thoughts (i.e. explicit state PC), and performed a lexical decision task (LDT) to measure automatic vigilance for task-related information (i.e. implicit state PC). Cardiac activity was continuously measured using heart rate (HR) and heart rate variability (HRV). Trait worry and rumination were measured by the Penn State Worry Questionnaire (PSWQ) and the Ruminative Response Scale (RRS), respectively.

The results showed that high trait worriers had a slower HR recovery from the cognitive stressor compared to low trait worriers. They also showed delayed HRV recovery, but only when the tendency to dwell upon ones negative mood (the 'brooding' subscale of the RRS) was low. Slow HR recovery was associated with high levels of negative intrusive thoughts and with automatic vigilance, but in the unexpected direction for the latter. These results provide evidence that delayed cardiac recovery is associated with trait as well as state PC, and suggest that brooding attenuates the HRV suppressing effect of high trait worry.

Introduction

A large body of research has made clear that stressful events can have profound effects on the pathogenesis of cardiovascular diseases (e.g., Rozanski, Blumenthal, & Kaplan, 1999; Black & Garbutt, 2002; Krantz & McCeney, 2002; Rosengren et al., 2004). The investigation of how stressful events can affect cardiovascular health has for a long period focused on adverse cardiovascular activity *during* these stressful events, while in more recent years the insight has grown that stress-related cardiovascular activity that is *prolonged* beyond the presence of these stressors might be much more detrimental (McEwen, 1998; Pieper and Brosschot, 2005; Schwartz et al., 2003). Delayed heart rate recovery has indeed been found to be predictive of coronary events (Pitsavos, 2004), heightened levels of carotid atherosclerosis (Heponiemi, 2007; Jae et al., 2008) and even all-cause mortality (Cole, 2000; Nishime, Cole, Blackstone, Pashkow, & Lauer, 2000). In addition, delayed blood pressure recovery predicted hypertension 3 and 5 years later (Stewart & France, 2001; Borghi, Costa, Boschi, Mussi, & Ambrosioni, 1986, respectively). Clearly, it is important to elucidate the psychological, that is, cognitive-emotional, factors that contribute to this delayed cardiovascular recovery.

It has been suggested that stressful events are associated with delayed cardiovascular recovery particularly because these events evoke negative, worrisome thoughts (Brosschot, Gerin & Thayer, 2006). According to the perseverative cognition (PC) hypothesis (Brosschot et al., 2006), worry and rumination extend the mental representation of a stressful event beyond its actual presence and this is suggested to delay cardiovascular recovery after this event. Perseverative cognition (PC) is defined as "the repeated or chronic activation of the cognitive representation of one or more psychological stressors" (cited from: Brosschot et al., 2006, p 114). This definition of PC is quite broad and as such, previous studies have focused on different operationalizations of PC when testing the PC hypothesis. First, PC can be measured as a trait or personality characteristic – some people are more prone to worry or ruminate than others – or as a state, that is, measuring the actual experience of negative repetitive thoughts during an experiment or in daily life. Trait and state worry appear only marginally related (Verkuil, Brosschot & Thayer, 2007). The second aspect is the content of the stressor that is represented. Whereas 'worry' refers to PC about future stressors, 'depressive rumination' refers to PC about ones current sad mood, while 'angry rumination' refers to PC about anger provocations. Third, all previous studies have focused on explicit forms of PC whereas it likely that implicit forms of PC exist. Below we will discuss to what extent these different operationalizations of PC have yielded supportive evidence for the PC hypothesis.

Several studies have suggested that high trait ruminators recover more slowly from stressful events (Gerin, Davidson, Christenfeld, Goyal, & Schwartz, 2006; Roger & Jamieson, 1988; Key,

Campbell, Bacon, & Gerin, 2008). However, these studies measured trait anger rumination (Gerin et al., 2006) and trait depressive rumination (Key et al., 2008; Roger and Jamieson, 1988) and not trait worry, that is, *anxious* PC. Both trait worry and trait depressive rumination are important risk factors for the onset and maintenance of mood and anxiety disorders (Nolen-Hoeksema, 1991; Borkovec, Ray & Stöber, 1998) which are in turn important risk factors for the development of cardiovascular diseases (CVD) (Kawachi, Sparrow, Vokonas, & Weiss, 1994; Wulsin, Vaillant, & Wells, 1999). For example, worry is one of the central features of Generalized Anxiety Disorder (GAD) and rumination plays an important role in Major Depressive Disorder (MDD). At least two study have shown that trait worry directly predicts cardiovascular health problems, that is myocardial infarction (Kubzansky et al., 1997) and the long term cardiovascular effects of a major stressful event ('9/11'; Holman et al., 2008). Although depressive rumination and anxious worry are related forms of PC, they possess some characteristics that distinguish them. For example, Watkins et al. (2005) found that worrisome thoughts are rated as more upsetting and disturbing than ruminative thoughts. This would imply that worrisome thoughts, which are typically measured with the Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990), might have stronger cardiac effects than ruminative thoughts. On the contrary, depressive rumination, as typically measured by the items of the Ruminative Response Scale (RRS; Treynor, Gonzalez, & Nolen-Hoeksema, 2003) might have somewhat less forthright physiological effects. This idea seems to be supported by empirical evidence. On the basis of their review of the physiological effects of PC, Brosschot et al. (2006) concluded that trait worry, as measured with the PSWQ is a the better predictor of delayed physiological recovery than trait rumination, as measured with the RRS. Moreover, worrisome thoughts were reported to continue for a greater number of years than rumination (Watkins, Moulds, & Mackintosh, 2005), implying that worrying may cause longer 'wear and tear' on the body (cf: McEwen, 2003). In the present study, we addressed this issue and expected trait worry (measured by the PSWQ) to be the stronger predictor of delayed cardiac recovery than trait rumination (measured by the RRS).

State worry has also been suggested to be implicated in slow CV recovery after stressful events (Brosschot et al., 2006). It has been shown that during experimentally induced worry as well as during worry in daily life cardiovascular activity is increased (Lyonfields, Borkovec, & Thayer, 1995; Thayer, Friedman, & Borkovec, 1996; Verkuil, Brosschot, Borkovec, & Thayer, 2009; Pieper, Brosschot, van der Leeden, & Thayer, 2007). Worry has also been shown to mediate the effects of daily stressors on prolonged cardiac activity during waking and sleeping (Brosschot, van Dijk, & Thayer, 2007). Yet, direct evidence that delayed cardiovascular recovery after a stressful event is due to perseverative cognition is still scarce, as most experimental studies have only found an association

between delayed recovery and trait PC, and not, or not consistently, between state PC measured after or during the recovery period (Glynn, Christenfeld, & Gerin, 2002; Key et al., 2008; Gerin et al., 2006). This may be due to several limitations of these latter studies. Firstly, these studies used anger provocation or emotional recall tasks as stressors, after which explicit state worry might just be less likely. For example, only 31% of the participants in a study by Glynn reported anger related thoughts after an anger recall task. The present study aimed to use a more general stressor, i.e. performance on an unsolvable cognitive task within an evaluative context. Such tasks have been previously shown to be experienced as physiologically and psychologically stressful (Brosschot et al., 1992; Weidner, Friend, Ficarrotto, & Mendell, 1989). Accordingly, in the present study we tested whether state PC concerning a previous stressor is associated with slowed cardiac recovery and adds to a model wherein slowed cardiac recovery is predicted by trait PC.

A second limitation of these previous studies pertains to the nature of state PC. Gerin and colleagues found that although delayed cardiovascular recovery after recalling an anger provoking event was predicted by trait angry rumination (Gerin et al., 2006; Glynn et al., 2002), this was not due to state rumination, as measured by thought sampling. However, in a more recent study Key et al. (2008) did find an effect of state rumination on cardiovascular recovery, but this was – unexpectedly - only true for people *low* in trait rumination (Key et al., 2008). As a possible explanation for this finding the authors suggested that perseverative cognition in frequent ruminators occurs largely implicit, without conscious awareness, and would therefore be difficult to report. Thus, delayed cardiac recovery after stressful events might not or not only be caused by explicit PC, but also by implicit or *unconscious* PC related to these events. It is not unlikely that implicit PC exists. In the last decades it has become clear that a large part of our information processing in daily life occurs relatively implicit and without reflective conscious awareness (Bargh & Chartrand, 1999; Bargh & Ferguson, 2000). Thus, there is reason to expect that stressful events not only give rise to explicit PC, but also to implicit PC. One example of implicit PC is 'automatic vigilance' for stressor related information. Automatic vigilance can be regarded as the increased sensitivity of the attentional system for task or stressor related information. This occurs for example after failure on a task (Rothermund, 2003; Smith, Ruiz, & Uchino, 2000). To date, no study has directly addressed the possibility that this type of PC causes prolonged stress-related physiological activity. Automatic vigilance or other forms of implicit or unconscious cognitive processing, such as after subliminal emotional stimulation, have not been tested for their physiological effects with the exception of relatively subtle effects on brain activity (Morris, Öhman, & Dolan, 1999), startle reflex (Ruiz-Padial & Vila, 2007) and skin conductance (Öhman & Mineka, 2001).

Finally, the role of mood in recovery from stressors remains unclear. It seems common sense that state negative mood is associated with cardiac activity. However, in several ambulatory (Brosschot et al., 2007) and laboratory studies (Verkuil et al., in press; Key et al., 2008) state mood was measured and was found to be unrelated to heightened or prolonged cardiovascular activity. In other studies of PC and recovery this was not tested, although effects of trait hostility on slowed blood pressure recovery have been reported (Anderson, Linden, & Habra, 2005). Therefore, in the present study we also investigated the effects of state anxiety and state sadness.

Summarizing, this study tested the hypothesis that slowed cardiac recovery after a stressor is predicted by high trait PC, especially trait worry, and by explicit and implicit state PC (negative intrusive thoughts and automatic vigilance) and negative affect. To test this, we used an unsolvable cognitive task, which consisted of an intelligence test of which the participants were made to believe that it predicted future career success, thereby creating an evaluative environment. This task has been previously used to evoke automatic vigilance (Koole, Smeets, van Knippenberg, & Dijksterhuis, 1999).

Materials and Methods

Participants

Fifty-nine undergraduate students from Leiden University participated in this study (mean age = 22.4 years, SD = 3.66; 12 males, 47 females). The sample was predominantly Caucasian (80%); 12% identified themselves as Black, 5% as Hispanic and 3% as Asian. They received € 4.50 or course-credits for their participation. This study was approved by our Institutional Review Board. All subjects provided written informed consent.

Instruments

Cardiac activity. 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 et al., 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 et al., 2004). For every 6-minute phase of the experiment (baseline, mental challenge, recovery) mean HR (in beats per minute, BPM) was calculated. In addition, to obtain a measure of HRV and vagal activity, spectral analyses using an autoregressive algorithm following the Task Force of the European Society of Cardiology and the North American Society of Pacing Electrophysiology (Task Force of the

European Society of Cardiology the North American Society of Pacing, 1996) guidelines were performed. Mean High Frequency (HF; 0.15 to 0.40 Hz) power (in milliseconds squared), was calculated for every phase of the experiment. In addition, the root mean of squared successive differences, RMSSD, in milliseconds was calculated for every phase.

Trait perseverative cognition (PC)

Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990; Dutch translation; van Rijsoort et al., 1999). This questionnaire consists of 16 self-report items. Items are directed at the excessiveness, duration and uncontrollability of worry, for example: 'Once I start worrying, I can't stop'. The PSWQ has demonstrated high reliability as well as high temporal stability and substantial construct and predictive validity in the assessment of trait worry (Verkuil et al., 2007).

Ruminative Response Scale (RRS; Treynor et al., 2003; Dutch translation: Raes, Hermans & Eelen, 2003). The RRS consists of 22 items measuring ruminative responses to depressed mood. These items form three subscales: Brooding, defined as 'a passive comparison of one's current situation with some unachieved standard', (cf. Treynor et al., 2003); Reflection, defined as 'a purposeful turning inward to engage in cognitive problem solving to alleviate one's depressive symptoms'; and a Depression scale. Only the rumination scales were used in this study: Reflection, consisting of five items (e.g., 'I analyze recent events to try to understand why I am depressed') and Brooding, consisting of three items (e.g., 'I think "What am I doing to deserve this?" '). The RRS possesses good internal consistency and of its subscales, the Brooding subscale correlated most highly with measures of chronic strain, providing evidence for its maladaptive features.

Cognitive stress task

Manipulated IQ task (derived from: Koole et al., 1999). The cognitive stress task consisted of six verbal analogies that were modeled after a normal IQ test. Each analogy consists of two blank spaces that have to be filled after one minute. Example: "____" relates to 'but' as 'however' relates to " ____". For each blank space, four possible answers were given, one of which the participants had to choose, although all analogies were unsolvable and they were sufficiently ambiguous to allow giving bogus (positive and negative) feedback (see Procedure). After the recovery phase the participants were asked to report, on a Likert scale (ranging from 'Not at all' to 'A great deal'), how much effort they had spend on trying to solve the analogies.

State perseverative cognition (PC)

Implicit PC: Implicit PC concerning the cognitive stress task was operationalized as automatic vigilance and measured with a lexical decision task (LTD; cf. Koole et al., 1999). The LDT is typically used to assess implicit activation of cognitive schemata, for example in studies concerning persistent activation of information related to goal discrepancies or intentions (e.g., Forster, Liberman, & Higgins, 2005; Marsh, Hicks, & Bink, 1998) and therefore very well suited to measure implicit activation of cognitive representations related to the stress task.

Participants were seated in front of a computer screen. They were told that on each trial of the task they were about to perform they would be shown a string of letters and that the task was to decide if the string was a word or a non-word. They could indicate their response by pressing one of two buttons on a response box and were asked to do this as quickly and accurately as possible. Each trial started with a fixation cross that lasted 2000 ms. Thereafter the letter strings were presented, with a maximum of 1000 ms per trial. The task started with ten practice trials. Subsequently, sixty-four words and non-words were shown. The order of the presentation of the trials was randomized for each participant. The task was programmed in E-Prime 1.1 software.

We used 8 words that were related to intelligence (e.g., 'smart', 'intelligent'), 8 control words that were unrelated to intelligence but reflected generally positive characteristics (e.g. 'brave', 'tolerant') and 16 neutral distracter words ('piano', 'eyes'). The remaining 32 words were non-words. Intelligence related and control words were matched on word length and word frequency. The index for *Automatic Vigilance* was calculated by subtracting the reaction times (RTs) to intelligence related words from the RTs to control words.

Explicit PC. To measure state PC related to the task, we used 7 items of the Sarason Cognitive Interference Scale (Sarason, 1978) which measures the level of distracting, intrusive thoughts of the participants experienced after the IQ task, during the recovery period (e.g. 'After the IQ task, I was thinking about how bad I had performed on the IQ task'). The CIQ has been used frequently in test anxiety literature, and possesses good psychometric properties with internal consistency (Cronbach's alpha) estimates ranging from .71 to .91. Studies using the CIQ have demonstrated that it is sensitive to changes in intrusive thoughts that are related to individual and situational factors (cf Pierce, et al., 1998).

Mood states. During the baseline and recovery phases the levels of state anxiety and state sadness were assessed using visual analog scales (Brosschot et al., 1992; Johansson, 1976). Participants first indicated how they usually felt on this 100mm scale and thereafter rated their current levels of anxiety and sadness. We used the difference between 'mood as usual' and 'current mood' as indices

of state anxiety and state sadness. This method is relatively insensitive to the shifting of internal standards for reporting ones mood.

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 since awaking on the day of participation, as these factors could influence their cardiac activity. For the same reason, participants were also asked to report their height and weight, use of medication and whether they suffered from a chronic disease.

Procedure

The experiment started with a 6-minute baseline recording of HR and HRV during which participants reported their mood state on the visual analogue scales. Thereafter, participants were instructed to perform an intelligence test. They were told that "this test is a good measure of analytic ability and is a reliable predictor of future success in numerous careers". The six analogies appeared one by one on the computer screen (see 'Instruments'). After every analogy participants received feedback ("that was not the correct answer" or "that was the correct answer"). To manipulate the level of negative feedback (high versus low levels), half of the participants received negative feedback after each analogy, the other half received negative feedback after two analogies and positive feedback after four analogies. In both conditions, the computer informed the participants that 8 percent of the other attendants had the same score, i.e. zero or four respectively. However, we found no significant different effects of this feedback manipulation on cardiac activity or on the state PC variables, nor any interaction effects with trait or state PC and mood states. Given that the task itself was sufficiently stressful to increase cardiac activity independent of feedback (see results), we therefore discarded the latter in the rest of the analyses.

After the cognitive task, participants performed a simple filler task (rate the attractiveness of several paintings) while HR and HRV were measured continuously, to allow a period of cardiac recovery for later analysis. After this 6-minute recovery phase, participants rated their current level of anxiety and sadness and performed the LDT (implicit PC). After these tasks had been completed, participants filled in the questionnaires, (including the explicit PC questionnaires), and were debriefed.

Statistical analysis

HF power and RMSSD were transformed logarithmically to normalize the distributions (consequently we refer to them as lnHF power and lnRMSSD). To test whether trait PC was associated with delayed

cardiac recovery, we used multilevel growth curve modeling (Singer and Willett, 2003) with HR or HF power as dependent variables1, and linear and quadratic time trends, based on visual inspection of the time curves of the cardiac data, and the PSWQ and the RRS subscales as predictors. The interactions of interest were the interactions between the linear Time trend and the PSWQ or the RRS subscales. Significant interactions were further explored by Pearson correlations and by analyses of (co)variance using the median splits of the trait scales. Only the biobehavioral variables that had a significant bivariate correlation with HR or HRV and that significantly improved the fit of the models (cf. Singer & Willett, 2003) were entered as covariates, (i.e. smoking and age in the HR and HRV analyses, respectively). This was also done as the number of measured biobehavioral variables was so large that entering them all would decrease the degrees of freedom too much for the present sample size. To examine whether trait PC was associated with negative intrusive thoughts, anxiety, sadness or automatic vigilance, we conducted analyses of variance and Pearson correlations. To test the hypothesis that these state PC variables and the mood states variables mediated the effect of trait PC on cardiac recovery, we conducted hierarchical multilevel regression analyses (Baron & Kenny, 1986). All independent variables were centered on their grand mean in order to reduce multicollinearity (Aiken & West, 1991). All analyses involved two-tailed tests, with alpha set at .05.

Results

Descriptive statistics

Table 1 shows the means and standard deviations of the variables measured in this study. In line with previous studies females scored higher on Brooding, and had a trend for higher Trait Worry scores. Because of technical problems, cardiac measures of two participants were not available. Therefore the analyses of the cardiac variables are based upon 57 participants. Females had higher baseline HR, but no different lnHF power than men. The amount of effort spent on the task was rated above the scale's midpoint (M = 4.24, SD = 0.68).

[1] The same analyses were also conducted with lnRMSSD as outcome variable and yielded the same pattern of results.

Table 1. *Descriptive statistics*

Gender					
	Female (*N* = 47)		Male (*N* = 12)		
	M	*SD*	*M*	*SD*	*p*
PSWQ	49.17	12.32	41.83	9.60	.07
RRS	40.70	9.28	35.66	11.54	.12
Brooding	6.51	1.77	5.17	1.59	.02
Reflection	11.04	3.76	10.00	3.86	.40
Baseline heart rate (bpm)	79.70	8.60	70.47	9.80	.00
Baseline HF power (ms^2)	579.07	714.39	534.91	654.51	.80
Baseline RMSSD (ms)	55.89	43.93	58.71	33.71	.40
RT intelligence related words	558	73	560	85	.95
RT control	536	69	545	63	.68
Negative Intrusive Thoughts	14.21	6.92	16.50	5.87	.30
State anxiety	1.55	1.20	1.49	1.13	.46
State Sadness	1.57	1.52	1.50	1.75	.88

Trait PC and cardiac recovery

Heart rate (HR). Preliminary inspection of graphs of the HR responses suggested that a quadratic time trend best described the data (see Figures 1 and 2). First, a baseline multilevel growth curve model was fitted with HR as dependent variable, and linear and quadratic time trends as predictor variables. Thereafter, Trait Worry, Brooding, Reflection and smoking were entered into the model as predictor variables. Adding these variables significantly improved the fit of the model (χ^2 = 18.26, df = 7, $p < .05$). It was also apparent that a significant amount of variance in the data was due to correlations between the repeated cardiac measurements (AR rho = -.36, 95% CI: -.60 to -.05). Results showed a significant main effect of Time linear (B = 4.86, 95% CI: 1.91 – 7.82) and Time quadratic (B = -2.89, 95% CI = -4.34 to -1.44). Follow up paired t-tests showed that the cognitive stressor led to an increase in HR (M = 79.56, SD = 10.62) compared to baseline (M = 77.62, SD = 9.60); during the recovery period (M = 76.02, SD = 8.49) HR decreased below the baseline HR level (*p*s < .05). Furthermore, the Time-linear x Trait Worry interaction was significant (B = .09, 95% CI: .03 - .15,

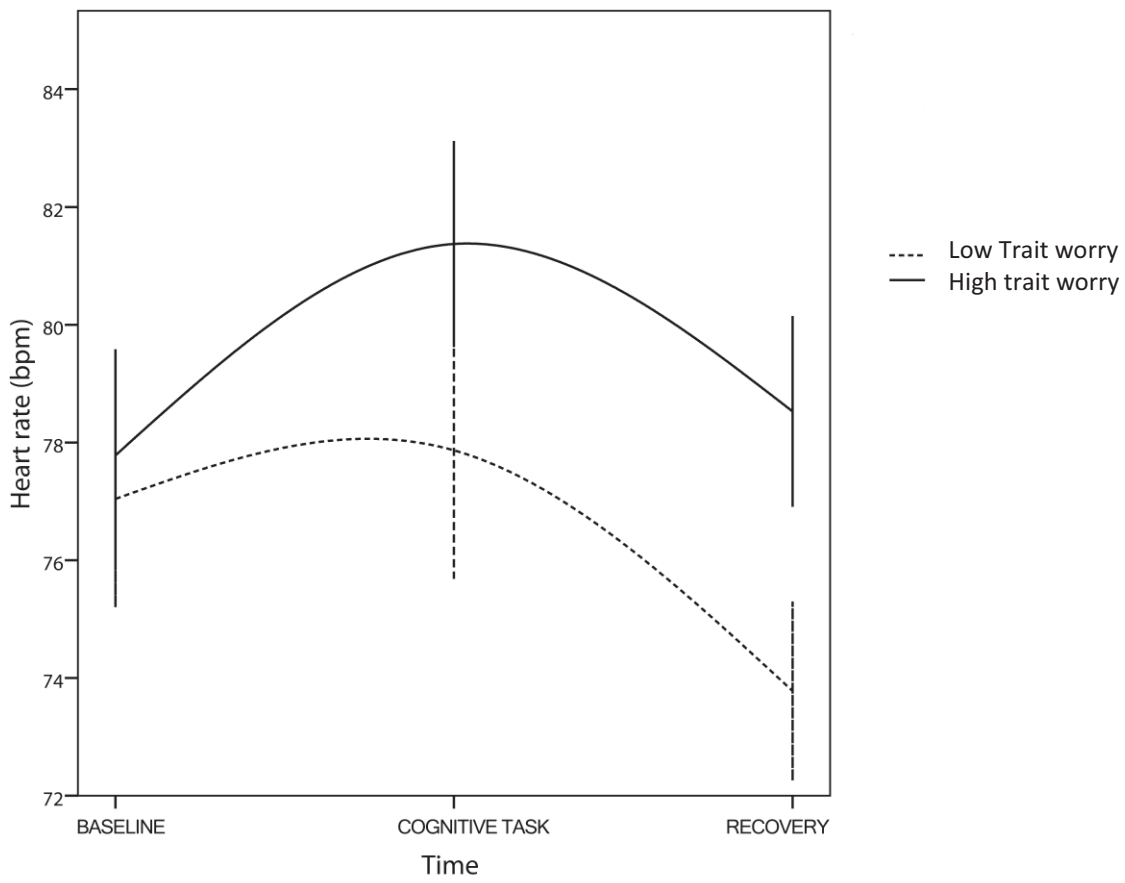

Figure 1. Mean level of heart rate during the experiment for low and high trait worriers. Error bars show mean +/- 1 standard error.

p < .05), as was the Time-linear x Brooding interaction (B = -.66, 95% CI: -1.21 to -.20, *p* < .05). As shown by the sign of the B values, the effects of Trait Worry and Brooding were in the opposite direction. To explore whether and how they influenced each other we tested whether the interaction between Time-linear x Trait Worry x Brooding was significant, which was not the case. No other main or interaction effects were significant. Figure 1 shows the effect of Trait Worry on HR recovery. For illustrative purposes Trait Worry scores were median split. High trait worriers had a higher HR (M = 78.52, SD = 8.27) during the recovery period than low trait worriers (M = 73.77, SD = 8.17), while controlling for HR during baseline and during the cognitive task ($F(1,53) = 4.57$, $p < .05$). Figure 2 suggests that high Brooders had lower HR during the cognitive task, but not during baseline or recovery. Yet, follow up univariate tests did not reveal significant differences between the Brooding groups.

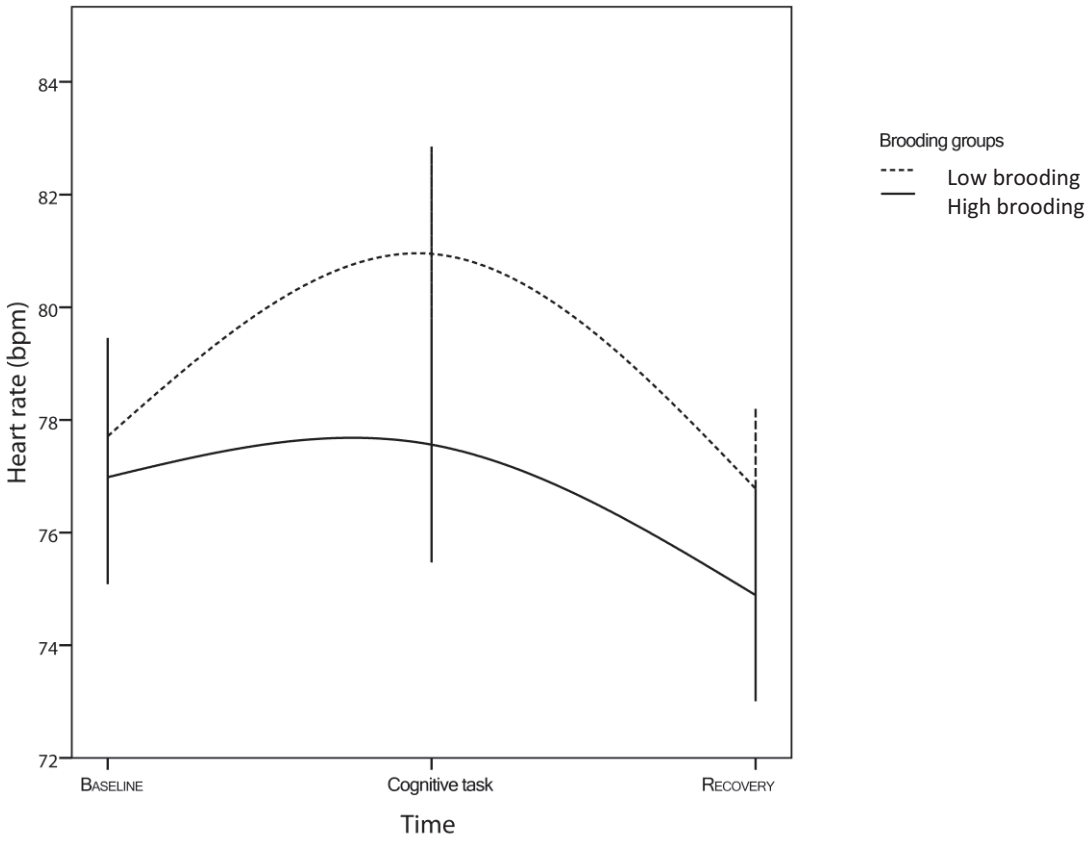

Figure 2. Mean level of heart rate during the experiment for low and high brooders. Error bars show mean +/- 1 standard error.

Heart rate variability (HRV). A baseline multilevel growth curve model was fitted with lnHF power as dependent variable, and linear and quadratic time trends as predictor variables. Adding Trait Worry, Brooding, Reflection and age as predictor variables significantly improved the fit of the model when compared to the baseline growth curve model (χ^2 = 18.57, df = 7, p < .05). The results showed a significant main effect of Time (linear, B = -.68, 95% CI: -1.05 to -0.32; quadratic, B = .32, 95% CI: .14 - .50). Follow up paired t-tests showed that the cognitive task led to an overall decrease in lnHF power (M = 5.34, SD = 1.23) compared to baseline (M = 5.69, SD = 1.21; p < .05). During the recovery period (M = 5.59, SD = 1.29) lnHF power did not differ from baseline. In addition, a main effect of Trait Worry appeared, with high trait worriers showing higher lnHF during the experiment (B = .03, 95% CI: .0001 - .061, p < .05). The expected effects of Time-linear x Trait Worry (B = -.01, 95% CI: -.02 - .00, p < .05) and Time-linear x Brooding (B = .08, 95% CI: .01 - .16, p < .05) were significant. In line with the results from the analyses on HR, the effects of Trait Worry and Brooding on lnHF during the experiment were in the opposite direction. Explorative analysis showed that these two-way interactions were subsumed by a significant three-way interaction between Time-linear x Trait Worry x Brooding (B = .006, 95% CI: .001 - .010, p < .05). Figure 3 shows that whilst Trait Worry was

associated with low lnHF power during recovery, this effect was moderated by the level of Brooding: when Brooding was low, high trait worriers had a significantly lower lnHF during recovery than low trait worriers ($F(1,32) = 4.24$, $p < .05$). On the other hand when Brooding was high (figure 2B), lnHF power was also high during recovery, irrespective of Trait Worry status. No further main or interaction effects were significant.

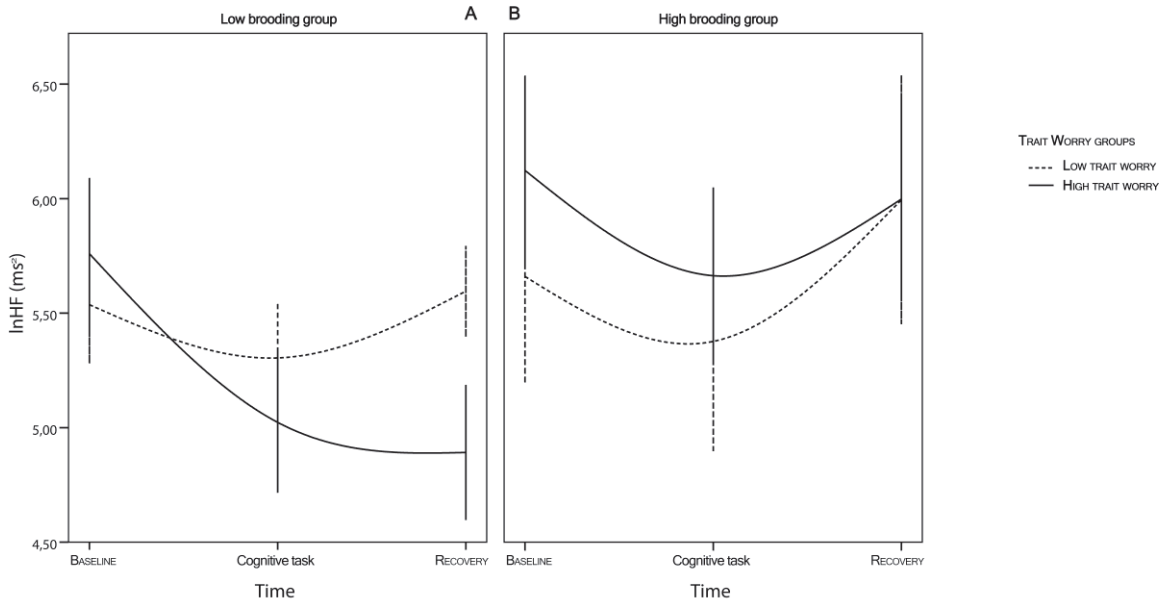

Figure 3. Interaction between Trait Worry and Brooding on lnHF power recovery. Error bars show mean +/- 1 standard error.

State PC and mood states after cognitive stress

First we inspected whether the cognitive task led to implicit PC, as indexed by automatic vigilance. The reaction times (RTs) to the intelligence-related words on the LDT were significantly slower than the RTs to control words (see table 1). The Automatic Vigilance Index (RTs IQ – RTs control = - 21 ms) differed significantly from zero ($t(58) = 3.15$, $p < .05$),. Trait Worry was not associated with different responding to IQ-related versus control words on the LDT. However, an exploratory analysis showed that Trait Worry was associated with a general slowing down on *all* LDT trials (mean $r(58) = .30$, $p < .05$). No associations were found between Brooding or Reflection and LDT reaction latencies. In addition, the Automatic Vigilance index was also not associated with the amount of negative intrusive thoughts during recovery ($r(58) = .05$, ns).

With regard to changes in mood states during the experiment, we found that state anxiety and state sadness during recovery were not significantly different from baseline.

Effects of explicit and implicit PC and mood states on cardiac recovery

Multilevel regression analyses were conducted to test whether state PC and the mood states influenced cardiac activity during recovery, and whether they could add to the effects of the PSWQ and Brooding on HR recovery. The results of these analyses are presented in Table 2. When state PC (Automatic vigilance index and negative intrusive thoughts) and mood states were entered into the model already containing Trait Worry and Brooding (see above), the fit of the model significantly improved ($\chi^2 = 14.46$, df = 6, $p < .05$). As shown in Table 2, in addition to the effects reported above (i.e. of Smoking, Time x Trait Worry, Time x Brooding), significant effects were also found for Time x Automatic Vigilance and, although marginally but still in the predicted direction, for Time x Negative intrusive thoughts. Exploration of the effect of Automatic Vigilance on HR recovery made clear that slower responding to *control* words, instead of IQ words, was associated with increased HR during recovery ($r(55) = .28$, $p = .034$). Importantly, by entering the cognitive emotional variables, the effect of trait worry was not reduced. Both Automatic Vigilance and Negative intrusive thoughts had independent effects on cardiac recovery.

The same steps were repeated for lnHF power. However, entering the PC and mood states variables into the model did not significantly improve the fit of the model, and no significant effects were found for these variables. As females had higher brooding scores and previously also have been shown to have higher baseline HRV (Thayer et al., 1998; Chambers and Allen, 2007), we also checked whether gender could possibly account for these results. This was not the case however.

Table 2. *Multilevel regression analysis predicting HR (bpm)*

Predictor	*B*	*SE*	*P*	*95% Confidence Interval*	
				Lower Bound	Upper Bound
Intercept	77.12	1.23	.00	74.67	79.56
Time, linear	4.58	1.54	.00	1.51	7.66
Time, quadratic	-2.68	0.74	.00	-4.26	-1.28
Smoking	0.62	0.30	.04	0.03	1.21
Trait Worry	0.09	0.12	.46	-0.15	0.30
Brooding	-0.33	0.75	.65	-1.82	1.16
Sadness	0.03	0.49	.95	-0.93	0.99
Anxiety	-0.20	0.48	.67	-1.16	0.74
Automatic Vigilance	-0.01	0.02	.76	-0.05	0.04
Negative Intrusive Thoughts	-0.17	0.18	.34	-0.54	0.19
Time-linear * Trait Worry	0.07	0.03	.04	0.00	0.13
Time-linear * Brooding	-0.52	0.20	.01	-0.91	-0.11
Time-linear * Sadness	-0.35	0.33	.29	-1.02	0.32
Time-linear * Anxiety	0.42	0.38	.27	-0.33	1.19
Time-linear * Automatic Vigilance	0.01	0.01	.03	-0.02	0.00
Time-linear * Negative Intrusive Thoughts	0.10	0.05	.06	-0.00	0.19

Discussion

The present study aimed to test whether cardiac recovery from a cognitive task could be predicted by trait PC, especially worry, and whether the effects of trait PC on delayed cardiac recovery would be due to heightened levels of explicit and implicit PC or negative mood states after this task. The results partially confirm our expectations and are in line with previous studies that have tested the perseverative cognition hypothesis (see Brosschot et al., 2006). As predicted, high trait worriers had a slower HR recovery from the cognitive task compared to low trait worriers. This adds to previous studies showing that trait PC is associated with slowed cardiovascular recovery after stress (Brosschot et al., 2006; Brosschot et al., 2007; Pieper et al., 2007; Glynn et al., 2002; Gerin et al., 2006; Key et al., 2008). The health effects of trait worry were recently also made clear by another study in which it was shown that high trait worriers showed enhanced heart rate, although not reduced HRV, during the anticipation and recovery phases of several laboratory tasks and also during the performance of these tasks (Knepp & Friedman, 2008). Given this accumulating amount of evidence, it is likely that trait worry exerts its damaging health effects (Kubzansky et al., 1997) by

prolonging the total physiological load that stressors have on the human body. The fact that this slowed recovery effect was restricted to trait worry is consistent with the results of a recent review (Brosschot et al., 2006) showing stronger effects of trait worry than other trait PC variables, such as trait depressive rumination. In fact, the effect of one of the rumination scales, Brooding, i.e. tendencies to dwell upon ones negative mood which is considered as a maladaptive aspect of rumination, was even reversed. Even more, trait worry and brooding had an interacting effect on HRV recovery; only when brooding tendencies were low, high trait worry was associated with slow HRV recovery. This suggests that Brooding attenuates the suppressing effect of trait worry on HRV recovery. Although we did not particularly expect these findings for brooding, they seem to be in line with several studies showing enhanced HRV levels in depressed women (Thayer, Smith, Rossy, Sollers, & Friedman, 1998; Chambers & Allen, 2007). Our sample consisted for the most part of women (80%). The increases in HRV in depressed women that have been previously found have been suggested to be a biological 'compensatory response' to counteract the detrimental effects of stress on cardiac activity. More specifically, increased HRV in women might reflect 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 & Lane, 2000). As brooding can be considered an emotion regulation strategy, although often unsuccessful, this idea fits the finding that women who respond to stress with brooding thoughts show enhanced HRV levels during recovery. As high HRV levels are cardioprotective, these speculations may also offer an explanation for the findings that women suffering from (sub-clinical) depression are at reduced risk for cardiovascular health problems when compared with men (Hybels, Pieper, & Blazer, 2002; Wulsin et al., 1999). They may also explain the present findings, and suggest that gender clearly is an important factor to be more systematically investigated in future studies.

Our second aim was to examine whether the effects of trait worry and brooding on delayed cardiac recovery would be due to heightened levels of explicit and implicit PC after the cognitive stress task. The expected effect of negative intrusive thoughts on cardiac recovery was only found for HR recovery and was merely a statistical trend when using two tailed significance tests. Still, this finding is in line with the perseverative cognition hypothesis and with studies showing that state worry is associated with enhanced HR (Lyonfields et al., 1995; Thayer et al., 1996; Verkuil et al., in press; Pieper et al., 2007). The results concerning our hypothesis about the cardiac effects of implicit PC were less straightforward and cannot be easily explained. First of all, in contrast to what has been previously found, that is faster responses to concern related words compared to control words, we found that for all participants the responses to the control words were faster than to the IQ-related

words. Second, although we found that slow cardiac recovery was associated with automatic vigilance after the mental challenge, follow up tests showed that delayed cardiac recovery was associated with slowed responses to control words on the LDT. A tentative explanation for these unexpected findings might be the following. Slowed LDT responses have been suggested to reflect an inability to disengage attention from the emotional value of the LDT words at the expense of attending to other aspects of the words (cf the 'affective interference hypothesis' posed by Siegle, Ingram & Matt, 2002). This might explain why for all participants reactions to the IQ words were slower than to the control words. In our LDT the control words reflected positive personality characteristics that were unrelated to intelligence. In this case, those participants that showed a slower HR recovery were those for who the cognitive task had most strongly threatened their *general* self-esteem, reflected specifically in slower responses to positive characteristics. However, these tentative suggestions should be examined in future studies for example by using other tasks measuring implicit cognition, like the Implicit Association Task (IAT) or flanker tasks (Rothermund, 2003). In addition, future studies should include concern related negative words as a limitation of this study was that we only included concern related positive and more general positive words. Studying implicit PC seems especially warranted as a large part of our information processing in daily life occurs relatively automatically. Focusing on explicit reports about stressful experiences could result in an underestimation of the effects that stressful events have on physiological functioning. Investigating whether and how implicit processing of stress-related information has adverse effects on physiological functioning might lead to a better understanding of how stress can eventually lead to somatic disease and therefore seems an essential venture for future studies.

We also explored the role of negative affect in delaying cardiac recovery. Trait worry was associated with heightened negative affect after the cognitive task, but no evidence was found for an association between negative affect and delayed cardiac recovery. This finding is in line with other studies that also have not found effects of negative affect on cardiac recovery (Key et al., 2008; Verkuil et al., in press). However, one possible explanation for not finding effects of negative affect in the present study is that the level of negative affect was relatively low. The cognitive task did not lead to overall significant increases in negative affect. Also, manipulating the type of feedback did not cause differential effects on cardiac activity, mood states or state PC. This is in contrast with previous studies that have provided (bogus) negative and positive feedback after cognitive tasks (Koole et al., 1999; Thompson, Webber & Montgomery, 2002). This limits our results in the sense that our results may pertain to slow cardiac recovery after cognitively stressful tasks as opposed to more emotionally stressful tasks. All participants rated their effort on the task above average and performing the task resulted in the expected cardiac pattern. Thus, the task seems to have required effort and seems to

have been perceived as relatively neutral by most participants. This kind of task might resemble the tasks that people frequently engage in at work. Recent studies have already shown that high trait worriers recover more slowly from emotionally demanding tasks like recalling emotional events (Glynn et al., 2002; Key et al., 2008; Gerin et al., 2006). The present study adds to this the possibility that high trait worriers also recover more slowly from more cognitively demanding tasks that are not particularly emotional for most people. It remains a future challenge to find out why, and by what mechanisms, this is the case. As emphasized, we believe that implicit cognitive mechanisms may play a crucial role.

Future studies could also examine other indices of the cardiovascular system. We focused on cardiac activity, that is, HR and HRV, and although these indices are associated with reduced future cardiovascular health, it remains unclear to what extent trait worry and brooding tendencies have differential effects on hemodynamic functioning, such as blood pressure, cardiac output and total peripheral resistance. Another limitation is that we used a relatively small, young and healthy sample. Although the sample of this study seemed to represent trait worriers on the full severity range, it would be useful to conduct a similar study with older participants and / or patients suffering from mood and anxiety disorders. Another limitation is that during the recovery period, participants performed a filler task that could have interfered with the experience of negative intrusive thoughts (e.g., Gerin et al., 2006) and therefore speeded up recovery. It is also possible that task performance during recovery slowed down recovery for all participants. In any case, it is possible that one or both of these mechanisms might have reduced diminishing the amount of variance in recovery that could be explained by trait and state PC. Thus, the prediction of recovery by worry might have been further improved when we had not used the filler task. Finally, one could argue that the cardiac effects of the current task were relatively small (e.g., 2 heart beats per minute) when compared to anger recall tasks (8 – 10 bpm). Yet, these latter tasks require participants to verbalize their thoughts possibly accounting for a part of these observed differences. More importantly, the increase of 2 heart beats per minute is in line with laboratory studies using a similar task (Weidner et al., 1989) and is in line with laboratory and ambulatory studies showing that stress and worry episodes increase heart rate with approximately 2 beats per minute (Verkuil et al., in press, Pieper et al., 2007). Still, it is possible that larger cardiac effects were obtained when the baseline period lasted longer than the six minutes that we currently used. Six minutes might have been too short to get a good estimation of baseline cardiac levels, especially since heart rate levels at the end of the recovery period were below baseline levels. Future studies should therefore include longer baseline periods.

In sum, the present study provides further evidence that delayed cardiac recovery is associated with PC, and shows that brooding attenuates the HRV suppressing effect of high trait

worry. The results could not confirm a mediating role of several forms of state PC. Trait worry and brooding tendencies are central elements in mood and anxiety disorders which, in turn, are important risk factors for the development of cardiovascular diseases. Therefore, investigating the long term implications of the present findings seems an important goal for future research.

Worry and Health : Mechanisms

Chapter 7

A sensitive body or a sensitive mind? Associations between somatic and cognitive sensitization, health worry and subjective health complaints

Bart Verkuil, Jos F. Brosschot & Julian F. Thayer

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Abstract

Psychobiological sensitization and health worry appear to be involved in the etiology of clinical manifestations of somatic health complaints (SHCs) via amplified processing of health-related information. However, it is not clear whether sensitization and health worry are also associated with *common* SHCs, which are extremely common and are responsible for a large part of both human suffering and health care costs. In this study we investigated whether SHCs are associated with health worry and two types of sensitization: cognitive health-related sensitization and somatic sensitization. We also examined whether health worry mediated the relation between cognitive sensitization and SHCs and whether both levels of sensitization interact.

In this study a non-clinical sample of 47 female students completed questionnaires about recent subjective health and health worry and underwent tests for cognitive sensitization, operationalized as Stroop interference and free recall performance, and somatic sensitization, operationalized as pain tolerance and pain threshold in a Cold Pressor Task.

Results showed that severity of health complaints was positively related with recall of health-related stimuli, but not with Stroop interference, and with worrying about health complaints. In addition, worry mediated the relationship between recall bias and severity of health complaints. Both the number and severity of recent health complaints were associated with pain tolerance. Pain threshold was associated with Stroop interference for health related information.

The results suggest that specific types of cognitive and somatic sensitization are associated with common health complaints, and that worrying about one's complaints might play a role by enhancing biased memory of health-related information.

Introduction

Somatic health complaints (SHCs) are extremely common and are responsible for a large part of both human suffering and health care costs (Ihlebaek, Eriksen, & Ursin, 2002; Eriksen, Svendsrod, Ursin, & Ursin, 1998; Picavet & Schouten, 2003). Moreover, SHC as well as self-rated health significantly predict mortality over and above objective measurements of health (Idler & Benyamini, 1997; Sha et al., 2005). Most SHCs concern difficult to diagnose vague symptoms - such as low back pain, headache or fatigue - and they are responsible for the majority of visits to general and other medical practitioners (Khan, Khan, Harezlak, Tu, & Kroenke, 2003). Typically, physicians can only find an organic basis for 10-20% of the most common symptoms, while only a small number receive a psychiatric diagnosis, for example somatoform disorder (Kroenke & Mangelsdorff, 1989). Clearly, it is essential to elucidate the processes underlying the reporting of health complaints.

Research concerned with clinical manifestations of SHCs - somatoform or functional syndromes - has suggested that these syndromes are characterized by *sensitization,* operating at somatic, cognitive and even at behavioral and social levels (Ursin & Eriksen, 2001; Brosschot, 2002; Eriksen & Ursin, 2004; Ursin, 2005). Sensitization is the increased reactivity of a single neuron or neural systems, caused by their repeated usage, and is thought to be a basic mechanism underlying the formation of memory (Bailey & Chen, 1991). More recently, it has been put forward as a process that could explain how somatic sensations develop into somatoform or functional syndromes levels (Ursin & Eriksen, 2001; Brosschot, 2002; Eriksen & Ursin, 2004; Ursin, 2005). *Somatic sensitization* is manifested as the amplification of somatic sensations, especially the lowering of pain thresholds and reduced tolerance for pain. It appears to be implied in chronic conditions such as irritable bowel syndrome (Bouin, Meunier, Riberdy-Poitras, & Poitras, 2001; Rodrigues, Nicholas Verne, Schmidt, & Mauderli, 2005), whiplash (Kasch, Qerama, Bach, & Jensen, 2005), and fibromyalgia (Marques, Ferreira, Matsutani, Pereira, & Assumpção, 2005; Lautenbacher, Rollman, & Mccain, 1994; Stevens, Batra, Kotter, Bartels, & Schwarz, 2000). Furthermore, Edwards (2005) suggested that heightened pain somatic sensitization, combined with reduced pain-inhibitory capacity, may predict chronic pain syndromes in initially healthy pain-free people. At a higher, *cognitive* level, sensitization is manifested as cognitive *bias* (Brosschot, 2002), that is, selective processing of information that is of high relevance for individuals (Rosen & Schulkin, 1998). Cognitive biases for information related to complaints, including pain, have been found in several clinical groups that are difficult to diagnose and treat, including somatoform patients, chronic pain patients, fibromyalgia patients and persons with high health anxiety (Keogh, Ellery, Hunt, & Hannent, 2001; Pauli, Schwenzer, Brody, Rau, & Birbaumer, 1993; Snider, Asmundson, & Wiese, 2000; Pauli & Alpers, 2002; Lim & Kim, 2005; Pincus & Morley, 2001; Roelofs, Peters, Zeegers, & Vlaeyen, 2002; Montoya, Pauli, Batra, & Wiedemann,

2005) as well as in patients with medically explained conditions that are influenced by stress such as psoriasis (Fortune et al., 2003) and asthma (Jessop, Rutter, Sharma, & Albery, 2004).

These clinical conditions however only represent 'the tip of the iceberg' of SHCs. Only recently have studies begun to address whether sensitization is implicated in *common* SHCs, that is, SHCs that are experienced by most of us (Ursin & Eriksen, 2001; Brosschot, 2002; Eriksen & Ursin, 2004; Ursin, 2005). One study (Williams, Wasserman, & Lotto, 2003) showed an attentional bias for health related information in students scoring high on a 14-item SHC checklist and low self-rated health. However, this study has some limitations, including a failure to control for the possibility that the bias was in fact a general negative emotional bias. In another study (Buchgreitz, Lyngberg, Bendtsen, & Jensen, 2006), somatic sensitization, as indicated by pain intensity ratings during pressure controlled palpation, was found to be related to the frequency of tension-type headache in the general population. The first purpose of the present study was to replicate and extend these studies by examining whether the number and severity of SHCs are associated with somatic and cognitive sensitization, while controlling for a general negative emotional bias (see Method section for further details). Furthermore, two further elementary propositions from the sensitization theory (Brosschot, 2002) will be tested.

Firstly, the occurrence and severity of common SHC might also be influenced by health-related worry (Brosschot, 2002; Brown, 2004c; Looper & Kirmayer, 2002). Health worry has been found to predict the occurrence of health complaints (Kaptein et al., 2005; Petrie et al., 2005) and a particularly intense form of health worry, catastrophic thinking, has been associated with increases in pain (Turner, Mancl, & Aaron, 2004) and other somatic complaints (Devoulyte & Sullivan, 2003). Furthermore, health worry has been associated with consulting a physician (Hay, Buckley, & Ostroff, 2005) and with intensive health care utilization (Looper & Kirmayer, 2001; Martin & Jacobi, 2006), suggesting that health worry is closely associated with the reporting of complaints. It is possible that bodily sensations trigger cognitive networks related to health, which promote selective cognitive processing and misinterpretations of these bodily sensations (Brosschot, 2002; Brown, 2004; Looper & Kirmayer, 2002). In turn, highly accessible cognitive networks increase the likelihood of reporting SHC by causing worries about these complaints. Thus, a second aim of the present study is to investigate whether health worry is related to SHCs and whether health worry mediates – at least in part – the relationship between cognitive sensitization and SHCs.

Secondly, it has been proposed that the effects of somatic and cognitive sensitization and health worry on SHCs are closely related and add up or even strengthen each other (Brosschot, 2002). It seems quite adaptive that frequent and intense bodily signals are not only enhanced by somatic sensitization, but are also given priority at higher levels of information processing, and are thus accompanied by cognitive sensitization (Brosschot, 2002). A possible downside, however, is that

paying more attention to bodily sensations and worrying about them could result in increased reporting of symptoms (Brosschot, 2002; Brown, 2004). Indeed, the widespread pain complaints of fibromyalgia patients are associated with both reduced pain thresholds and tolerance (indicating somatic sensitization (Marques et al., 2005; Lautenbacher et al., 1994; Stevens et al., 2000)) and hypervigilance (indicating cognitive sensitization (McDermid, Rollman, & McCain, 1996; Carrillo-de-la-Pena, Vallet, Perez, & Gomez-Perretta, 2006)). Additional support for the multilevel view of sensitization comes from findings that show that sensitization of the spinal cord is under cognitive control (Matre, Casey, & Knardahl, 2006). Still, the multilevel theory has not been tested directly by showing that cognitive and somatic sensitization are related *to each other* and have additive or interacting effects on common SHC. Showing such evidence was therefore the third aim of the present study.

In summary, the present study was designed to test the following hypotheses: (1) SHCs are associated with somatic and cognitive sensitization, as well as with health worry (2a) health worry is associated with cognitive and somatic sensitization and (2b) the relationship between cognitive sensitization and SHCs is mediated by health worry, and (3), somatic sensitization is related to cognitive sensitization and their effects on SHCs interact.

Materials and Method

Subjects and procedure

Fifty-one female students at Leiden University were invited to participate in the study. Four participants who indicated that they suffer from a chronic medical condition were removed from the analyses. The age of the final 47 subjects ranged from 18 to 33 with a mean of 20.5.

After being introduced to the laboratory, participants gave informed consent and performed three tasks in the following order: a Cold Pressor Task (CPT), a modified Stroop task and an incidental recall task (see task descriptions below). Subsequently, they completed questionnaires and were debriefed and paid. They received € 6 or course credits as compensation for their 45 minute participation.

Somatic sensitization: CPT

Indices of somatic sensitization that are most often used are pain tolerance and pain threshold assessed using a CPT. Moreover, since these indices have been differentially related to clinical subjective somatic complaints (13-19), it seems necessary to use both of them. The CPT consisted of a water tank which was, on the surface of the water, divided into two sections, one filled with ice and another kept free of ice to allow a hand to be immersed in the water without direct ice contact. A

pump kept the water flowing continuously to prevent a build up of warmer water around the hand. Mean water temperature was 2.5˚C (SD = .33). Participants were asked to immerse their dominant hand into the water and to indicate when it started to hurt, but to leave it there until the pain became intolerable. *Pain threshold* was taken as the time (in seconds) elapsed when it started to hurt, and *pain tolerance* was the time elapsed between the pain threshold and the moment the hand was withdrawn.

Cognitive sensitization

Cognitive sensitization can be operationalized in several ways, of which attentional and memory bias are the most common ones. Evidence from *clinical* populations suggests that at least *clinical* complaints are differentially associated with these operationalizations. For example, chronic pain seems to be associated mainly with recall bias, whereas somatoform disorders seem to be associated with attentional bias (for reviews see: 9 and 26). For these reasons, we employed a test for attentional bias (a modified Stroop task) as well as a memory task (incidental free recall task).

Modified Stroop task

The modified Stroop was presented on a Dell computer with a 17" LCD monitor. Latency in color naming was measured with a voice-key. Four categories of words were used (see Appendix): 7 health-related words, 7 negative emotional words and 7 neutral words and 7 specific cold-pressor related words. The health words were based on studies of the most common health complaints in the general population such as 'tired', 'back pain' and 'flu' (Eriksen et al., 1998). The negative emotional words were added to control for a negative emotional cognitive bias. They were based on word familiarity ratings in the Dutch language area (Hermans & De Houwer, 1994; Crombez, Hermans, & Adriaensen, 2000) and included words related to angry, sad as well as anxious moods such as 'scary', 'weak' and 'cruel'. Neutral words were vehicle related words (for example: 'cylinder' and 'passenger'). We choose words that were semantically interrelated, instead of unrelated words (Williams et al., 2003), to make the properties of the categories more comparable. We also included CPT-related words to check for the possibility that the association between somatic and cognitive sensitisation is very specific, that is, restricted to words directly related to the somatic sensations during the CPT, instead of health in general. The CPT-related words were related to sensations experienced during a CPT (von Baeyer, Piira, Chambers, Trapanotto, & Zeltzer, 2005), and included 'wet', 'stinging' and 'cold'. The four categories of words were matched with regard to word length, number of syllables and word frequency according to the Integrated Language Database of Dutch (Institute for Dutch Lexicology, 1996). We tested whether the word categories had the expected different emotional valence ratings by asking participants to rate the valence of the words at the end

of the experiment on a scale from -2 (extremely negative) to 2 (extremely positive). Overall, the negative (M = -1.34, SD = .37] and health-related words (M = -1.31, SD = .41) were rated as more negative than the CPT-related (M = -.90, SD = .35) and neutral words (M = .16, SD = .32); [$F(3, 144) = 246{,}74$, $p<.01$]. Words of each category were presented in blocks and the order of the blocks and order of the words within the blocks were randomized across participants.

Inaccurate responses (3.8%) due to voice key failures or incorrect responses were excluded from the analyses. Response latencies faster than 150 ms or slower than 2000 ms and individual mean latencies deviating more than 3 standard deviations (3.4%) were removed. An interference score was calculated by subtracting mean latencies to neutral words from mean latencies to negative, health and cold pressor words.

Incidental free recall

After the Stroop task participants received a blank A4 sheet. They were asked to write down as many words as they could remember from the Stroop task. No time limit was given, and after four minutes the task was ended by the experimenter (Williams et al., 2003; Russo et al., 2006). Recall performance was indexed by the total number of words that people could recall within each category. As the words were presented in clustered blocks it was possible that recall performance was confounded by order of appearance of the blocks. However, no association between the number of recalled health related words and order of appearance was found (Kruskal-Wallis $\chi^2 = 21.06$, df = 23, $P > .05$).

Subjective Health

Following the literature, subjective health was measured in a specific (discrete complaints) and general ways.

Discrete SHCs were measured with the Subjective Health Complaints questionnaire (Brosschot & Van Der Doef, 2006; Eriksen, Ihlebaek, & Ursin, 1999). The SHC is a 29-item self-report questionnaire concerning severity and duration in days of subjective health complaints experienced during the last month from four different areas of complaints: musculoskeletal pain, pseudoneurology, gastrointestinal problems, allergy and flu. Example items are: ‘low back pain’, ‘cough’ and ‘headache’. Severity of each complaint is rated on a 4-point scale. Total number of complaints as well as a total severity score were used.

In addition, we also measured general *self-rated health* (Idler & Benyamini, 1997). Participants were asked to rate their health in comparison with people of the same age. They could respond with "worse", "the same" or "better".

Health worry

Analogous with subjective health, health worry was also measured in specific (discrete complaints) and general ways. *Complaint specific worry* was assessed by counting the number of times participants indicated that they had been worrying about that complaint for each of the 29 complaints on the SHC questionnaire.

In addition, general tendency to worry about health was measured with three dichotomous items (e.g., 'Do you worry a lot about your health?') derived from the Whitely Index. These items have previously been confirmed to measure *general illness worry* (Fink et al., 1999).

Statistical analyses

All analyses were conducted using SPSS 14.0 software. The data were screened for normality using the Kolmogorov-Smirnov Test. The distributions of most variables were skewed and after transformations these variables were still skewed. Therefore, we used non-parametric tests (Spearman's Rho, Mann Whitney's *U* and Kruskal-Wallis tests) to test the hypothesized associations. To test the hypothesis that cognitive bias was a mediator – as defined by Baron and Kenny (Baron & Kenny, 1986) - between health worry and SHC, we used regression analysis (the assumption that residuals should be normally distributed (Tabachnick & Fidell, 2001) was met, Kolmogorov-Smirnov $Z = 0.75$, $p < .05$). Because of the specific direction of our hypotheses we used one-tailed tests.

Results

Descriptive statistics

The mean number of complaints that participants had experienced during the last month was 7.91 (SD = 3.37). The three most frequent complaints were "tiredness" (80.9% of the subjects; mean duration = 4.08 days, SD = 4.11), "cold, flu" (74.5%; M = 4.24 days, SD = 6.42) and "headache" (68.1%; M = 2.54 days, SD = 4.33), which is generally in line with the outcomes of other studies involving young females, although these percentages are somewhat above average (Eriksen et al., 1998; Haugland, Wold, Stevenson, Aaroe, & Woynarowska, 2001). The mean duration of the complaints was 4.54 days (SD = 3.60). With respect to self-rated health, 4.3% of the participants rated their health as "worse", 82.3% as "the same," and 8.5% as "better", in comparison to peers. On average, participants reported to have been worrying about 1.53 of their health complaints (SD = 1.70) with

36.2% reporting no complaint specific worry. The mean on the general illness worry scale was 0.61 (SD = .83) with 56% reporting no general illness worry. Thirteen percent of the sample had visited their general practitioner during the past month with a maximum of two visits. These participants also reported more worry about their complaints (Mann Whitney's *U* = 35.50, p<. 05).

Subjective health and health worry

Spearman correlations between subjective health indices and the health worry indices are shown in Table 1. Number and severity of SHCs were positively correlated with complaint specific worry (*r*s = .48 and .49, respectively; *P*s < .05). There were no significant correlations between the health worry measures and SRH. Finally, number and severity of SHC were significantly associated with self-rated health (*r*s = -.35 and -.37, *P*s < .05), with high levels of SHC associated with poor self-rated health.

Table 1. *Correlations between somatic health complaints, self-rated health, complaint specific and general illness worry*

	SHC		SRH	Worry	
	Severity	Number		Complaint specific	General illness
SHC[1]					
Severity	-				
Number	.92*	-			
SRH[2]	-.37*	-.35*	-		
Worry					
Complaint specific	.49*	.48*	-.19	-	
General illness	.00	.05	.00	.09	-

* Correlation is significant at the 0.01 level (1-tailed). [1] SHC = Subjective health complaints; [2] SRH = Self-rated health

Subjective health, health worry and cognitive sensitization

Modified Stroop task

Figure 1 presents the response latencies on the modified Stroop task. There were no significant associations between subjective health indices and health worry indices on the one hand and any of the three Stroop interference scores on the other hand.

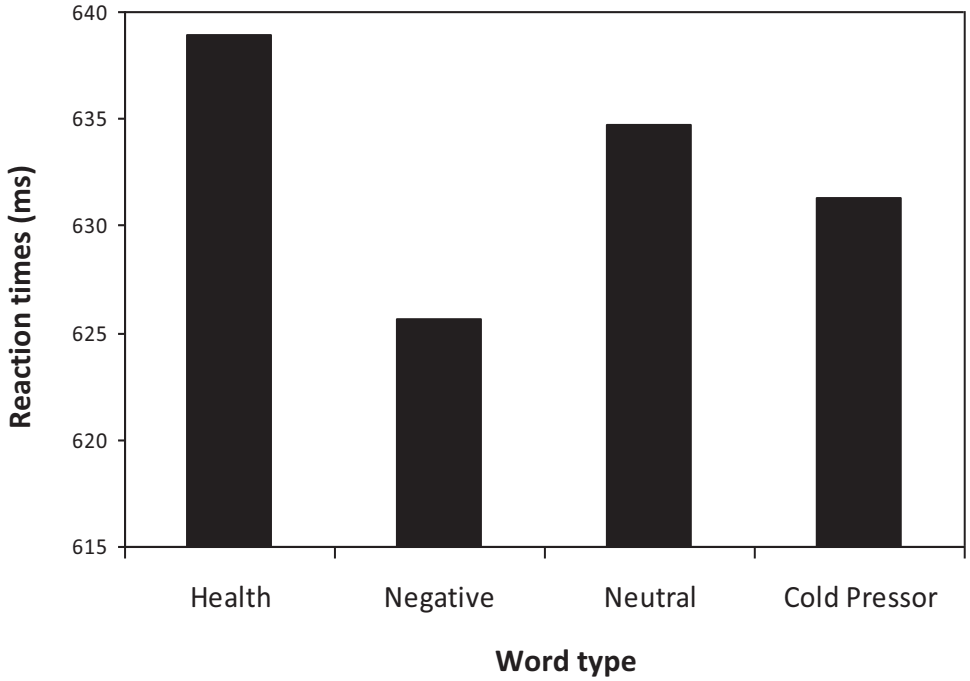

Figure 1. Mean response latencies per word category on the modified Stroop task

Incidental free recall

Results of the correlation analyses are shown in Table 2. Severity of SHCs (r = .29, P < .05), self- rated health (r = -.31, P < .05) and complaint specific worry (r = .34, P < .05) were all significantly associated with a recall for health related words, in the expected directions. No such associations were apparent for number of SHC and general illness worry.

The number of recalled negative words was positively related to Stroop interference on trials with negative words (r = .34, p < .05). No further significant correlations were found between Stroop performance and recall for the different words.

Table 2. *Correlations between somatic health complaints, self-rated health, health worry and free recall performance*

		Health words	Negative words	Neutral words	Cold Pressor words
SHC[1]					
	Number	.20	.17	.07	-.02
	Severity	.29*	.15	.06	-.10
SRH[2]		-.31*	-.04	.06	-.00
Worry					
	Complaint specific	.34*	.24	.00	-.20
	General illness	.14	-.19	-.08	-.17

* $p < .05$; [1] SHC = Subjective health complaints; [2] SRH = Self-rated health

Mediating effects of health worry

Table 3 shows the results of the mediation analysis. In line with the suggestions by Baron and Kenny (1986), severity of SHCs was first regressed on complaint specific worry and, subsequently, complaint specific worry was regressed on recall bias. These two basic requirements for establishing a mediation effect were met. In the final step, the mediator (complaint specific worry) was entered first in the regression analysis, followed by recall bias. The relationship between recall bias for health related words and severity of SHCs was mediated by complaint specific worry.

Table 3. *Mediating effect of complaint specific worry*

Step and variables	B	SE B	p
Regression 1			
Criterion: SHC[1] (severity)			
1. Recall for health words	.55	.33	.05
Regression 2			
Criterion: Complaint specific worry			
1. Recall for health words	.28	.12	.02
Regression 3			
Criterion: SHC (severity)			
1. Complaint specific worry	1.26	.37	.00
2. Recall for health words	.20	.32	.53

[1] SHC = Subjective health complaints

Subjective health, health worry and somatic sensitization

There were no significant associations between subjective health indices and health worry on the one hand and pain threshold on the other hand. Pain thresholds were significantly, but moderately, (r = .40, P < .05) associated with pain tolerance, our second measure of somatic sensitization. Inspection of pain tolerance scores yielded a clear distinction into two groups of subjects: those who removed their hands from the water after less than 77 seconds ("low tolerance"; N = 33) and those who kept their hands in the water until 131 seconds or for the total four minutes ("high tolerance"; N = 14) (see Figure 1). The low pain tolerance group had significantly more health complaints (M = 8.55 versus M = 6.42; Mann-Whitney U = 153.50, P < .05) and more severe health complaints (M = 11.30 versus M = 7.86; Mann-Whitney U = 133.00, P < .05) than participants with high pain tolerance. There were no differences between the groups on self-rated health or on the two health worry indices.

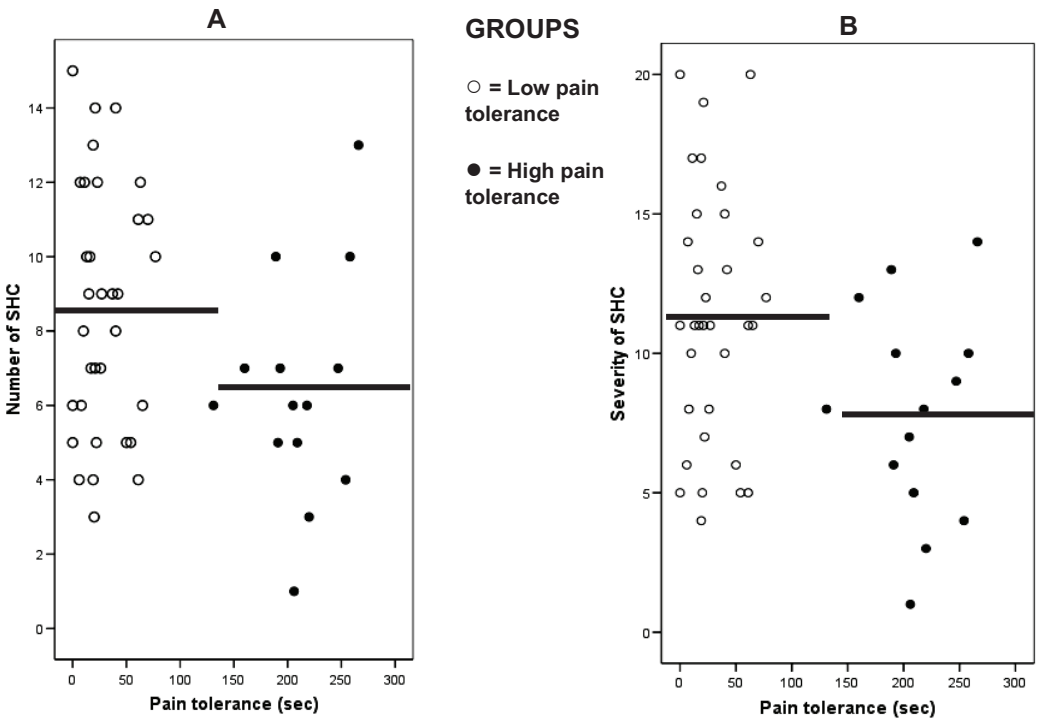

Figure 2. Panel A shows the difference in number of SHC between the pain tolerance groups. Panel B shows the difference in severity of SHC between the pain tolerance and the severity of SHC (the bold lines represent the means on the SHC for the pain tolerance groups).

Somatic sensitization and cognitive sensitization

To test the hypothesis that the different levels of sensitization are related, we tested whether subjects who had low pain thresholds and low tolerance for pain showed cognitive bias towards CPT related and health related words during the Stroop task and the recall test. People with low pain thresholds exhibited more Stroop interference when presented with health-related words (r = -.27, P < .05), but showed no better recall for these words. There were no associations between pain tolerance and both kinds of cognitive sensitization, or between the two kinds of somatic sensitization and bias for CPT-related words.

Discussion

The present study aimed to test whether subjective health is associated with cognitive and somatic sensitization, and whether health worry mediated the link between cognitive sensitization and somatic health complaints (SHCs). It was also expected that the two types of sensitization would be related and interact, thereby yielding evidence of the multilevel nature of sensitization. The results partly support the hypotheses: Subjective health is related to both types of sensitization and to health worry, and the association between subjective health and cognitive sensitization is mediated by health worry. Furthermore, we found that somatic sensitization and cognitive sensitization for health related information were related. However, based on the paradigm used by Williams et al. (2003), several different indices were used for each of these factors, and these associations were not always found for each of the two indices per factor. Subjective health was measured by SHCs in the last month and by general self-reported health. Cognitive sensitisation was measured by attentional bias and free recall for health related words, and somatic sensitisation was measured by pain threshold and tolerance. Finally, two indices for health worry were employed: worrying about recent complaints and general illness worry. The results showed that the associations between the two indices per factor were moderate, suggesting that the indices were tapping into the same construct, but were not measuring exactly the same. We will discuss the results for each hypothesis in more detail below.

With respect to cognitive sensitization, there was an association between SHCs and self-rated health on the one hand and cognitive sensitization on the other hand, but the latter was found for health related *recall* and not for attentional bias (Stroop interference). This suggests that even non-clinical individuals with common SHCs possess highly accessible cognitive networks related to health. When retrieving information from memory, health related information is given priority over neutral and negative information. The fact that this recall bias was found for severity rather than the number of SHCs, suggests that it may depend on the cognitive appraisal of complaints, that is, the meaning, suffering and threat involved in the complaints. This would be in line with studies that found recall

biases in conditions that are characterized by extreme emotional appraisals such as excessive worry (catastrophizing), chronic pain (Pincus & Morley, 2001b) and in emotional disorders such as depression and anxiety (Russo et al., 2006). In other words, the link between recall bias and SHCs may be related to emotional appraisals such as worry. In line with this, we found that the association between SHCs severity and recall bias was mediated by complaint specific worry. These results seem to indicate that a highly accessible health-related cognitive network, as indexed by a recall bias, may increase the likelihood of reporting SHCs by causing worries about these complaints. This might also explain why there were no effects of general illness worry. The effect of a trait-like measure of health worry on actual SHCs might be much less substantial than worries linked to these very complaints. In a recent study we have found that general measures of worry correspond only to a small extent with worry in daily life (Verkuil, Brosschot, & Thayer, 2007). Future studies should consider using additional momentary assessments of worry and recall bias to be able to uncover the dynamic process by which worry and recall bias interact and possibly enhance SHCs (Brosschot & Van Der Doef, 2006).

The lack of a relationship between subjective health and attentional bias is in partial discordance with Williams et al. (2003), who found an association of SHCs and Stroop interference for health related words. This disparity is not likely to be caused by differences in the Stroop tasks, because the blocked design we used usually results in larger Stroop effects (Waters, Sayette, Franken, & Schwartz, 2005) than the random design Williams et al. used. In line with models of pain which propose that pain demands attention (Eccleston & Crombez, 1999), it is more likely that the pain task, the CPT, led to an attentional bias that could have overruled the more subtle association between SHCs and attentional bias. Indeed, we found that people with low pain thresholds showed an attentional bias for health related words.

For somatic sensitization we also found a relationship with subjective health: The less participants tolerated pain, the more numerous and severe their health complaints were. Interestingly, until now, reduced CPT pain tolerance has only been found in somatoform disorders (i.e. in studies comparing people experiencing clinical levels of SHC with healthy controls ; Kasch et al., 2005; Stevens et al., 2000; Gramling, Clawson, & McDonald, 1996; Bouin et al., 2001). This study is the first to show this for common health complaints, and offers some support for our sensitization theory of these complaints (Ursin & Eriksen, 2001; Brosschot, 2002; Eriksen & Ursin, 2004; Ursin, 2005).

Finally, we found the expected association between the two types of sensitization. This too, however, was restricted to specific types of sensitization: pain threshold and attentional bias for health related words. This can be understood when the detection of thresholds is viewed as an interaction between somatic sensitization and attention. An early detection of pain is more likely to

be related to attentional bias for health-related information such as in the Stroop task we used, and less so to recall of this information.

There are several methodological limitations. First of all, the study was conducted in a young and female sample. As several studies suggest that mechanisms contributing to somatic complaints differ between males and females (Lee, Mayer, Schmulson, Chang, & Naliboff, 2001; Staud, Robinson, Vierck, & Price, 2003) it is unclear how the findings might generalize to older and male populations. Second, one could argue that compared to the tests we used for cognitive sensitization, our test for somatic sensitization, the CPT, allows more conscious cognitive strategies to deal with the pain, such as distraction, acceptance or catastrophizing, which could have contaminated the measure of sensitization (Masedo & Esteve, 2007; but see Hodes et al. (Hodes, Rowland, Lightfoot, & Cleeland, 1990) who found no effects of distraction on pain tolerance). This possibility can not be completely ruled out and future studies could consider instructing participants to use the same coping strategy. Another limitation is that our measures of somatic sensitization were restricted to cold-induced pain. Other measures of somatic sensitization, for example, thermal or electrical pain induction might have yielded different findings. A further limitation is that a general tendency to experience negative emotions could have caused high SHC scores and influenced our tests of sensitization. However, the lack of effect on the general negative emotional words in our tasks ruled out such a general negative emotional bias. This is in line with recent studies which have shown that associations of selective attention for pain, SHCs and worry and catastrophizing exist independent of negative affect (Vervoort, Goubert, Eccleston, Bijttebier, & Crombez, 2005; Brosschot & Van Der Doef, 2006; Crombez, Eccleston, van den Broeck, Van Houdenhove, & Goubert, 2002). Finally, an obvious limitation of a study such as the present one is its correlational nature, which allows no clear conclusion as to causal directions of the relationships found. Although useful as a first investigation of these relationships, it is clear that future studies should use prospective designs or interventions directed at worry or cognitive sensitization.

In summary, this study has focused on SHCs in a non-clinical female sample and has provided preliminary support for the sensitization theory of SHC (Ursin & Eriksen, 2001; Brosschot, 2002; Eriksen & Ursin, 2004; Ursin, 2005). It suggests that a large variety of common health complaints are associated with specific types of cognitive and somatic sensitization, that is, enhanced recall of health related information and lowered tolerance for pain. The results also seemed to imply that this relationship, at least for cognitive sensitization, is mediated by worries about these complaints. One possible implication of this is that it might be more fruitful to focus interventions at cognitive sensitization rather than worry.

APPENDIX

Words used as stimuli

English Translated Word Sets			
Health Complaint Words	Negative Words	Neutral Words	Cold Pressor Words
Tired	Scary	Brake	Wet
Back Pain	Concerned	Cylinder	Numbed
Flu	Cruel	Mirror	Stinging
Migraine	Hateful	Passenger	Cold
Cough	Jealous	Seat	Prick
Pain	Worthless	Turbo	Insensitive
Nausea	Weak	Door	'Pins and needles'

Chapter 8

Interacting effects of worry and anxiety on attentional disengagement from threat

Bart Verkuil, Jos F. Brosschot, Peter L. J. Putman & Julian F. Thayer

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Abstract

Recent work suggests that the ability to disengage attention from threatening information is impaired in people who suffer from anxiety and dysphoria. It has been suggested that this impaired ability to disengage from threat might specifically be associated with the tendency to perseverate about threat (i.e. worry), which is a main characteristic of anxiety disorders and a wide range of other psychopathologies. However, no studies have yet addressed this issue. The present study examined whether trait worry as well as worry intensity after experimental worry induction are associated with impaired ability to disengage attention from threatening cues (angry faces), independently from or in conjunction with anxiety. Sixty-one participants performed a visual cueing experiment that required detection of a target stimulus at one of two possible locations. Prior to the target neutral, happy or angry facial cues appeared at one of these two locations; An overall faster responding to invalidly cued trials relative to validly cued trials is believed to indicate inhibition of return (IOR) to a recently attended location, or, in other words, attentional disengagement. Lower disengagement from angry faces was only found when both trait worry and anxiety were high. When anxiety was kept constant, both trait worry and state worry was associated with reduced attention allocation to neutral faces instead. The results seem to suggest that specific threat-related deficiencies in disengagement may be a function of the co-occurrence of worry and anxiety.

Introduction

Perseverative thinking such as worry is a central feature of a wide range of psychopathologies and has been proposed to be an important transdiagnostic process (Harvey, Watkins, Mansell, & Shafran, 2004). Worry predicts anxiety and depressive affect (Hong, 2007), and it is the main characteristic of generalized anxiety disorder (GAD; American Psychiatric Association, 1994). In addition, it is found in social phobia (Mellings & Alden, 2000), panic disorder (Casey, Oei, & Newcombe, 2004), obsessive compulsive disorder (Comer, Kendall, Franklin, Hudson, & Pimentel, 2004), eating disorders (Sassaroli et al., 2005) and in depression (Diefenbach et al., 2001). More recently, it has been suggested that worry prolongs physiological stress responses beyond the actual presence of stressors, thereby contributing to the total wear and tear of stressors on the human body (Brosschot et al., 2006; Pieper et al., 2007; Brosschot et al., 2007). Given this seemingly broad importance of worry in the development and maintenance of mental and somatic health problems, studies that investigate its cognitive underpinnings are warranted.

A large number of studies conducted with extreme worriers, that is, people suffering from GAD, have shown that they show biased processing of threat-related information that is associated with the excessiveness of their worrying. For example, they interpret ambivalent information in a more negative way (Hazlett-Stevens & Borkovec, 2004), have biased explicit memory (Friedman, Thayer, & Borkovec, 2000) and selectively attend to concern-related threatening information (Mathews & MacLeod, 1985; Mathews, Mogg, Kentish, & Eysenck, 1995; Mogg, Mathews, & Weinman, 1989; Mogg, Bradley, Millar, & White, 1995). These biases in the processing of threat are thought to contribute to the prolongation of worry episodes in GAD.

Still, although biased attention seems to be associated with worry, it is not known what aspects of attention are specifically associated with worry. Attention can be divided into three processes (Posner & Petersen, 1990): orienting towards a stimulus, engaging attention and eventually disengaging from it. Especially the delayed disengagement from threatening information, or prolonged dwell time, is believed to lead to worry and rumination (Georgiou et al., 2005; Fox et al., 2001). This makes sense from a phenomenological point of view: A main characteristic of pathological worry is that high worriers find it extremely difficult to disengage from their worry topics, and the same threatening thoughts occur over and over again. They find it extremely difficult to stop worrying and to mentally disengage (or 'decenter') from their sorrows.

The inability to disengage attention from neutral or threatening information has mainly been studied with regard to enduring negative affect such as in dysphoria (Koster, De Raedt, Goeleven, Franck, & Crombez, 2005) and in trait anxiety (Yiend & Mathews, 2001; Fox et al., 2002; Koster, Crombez, Verschuere, & De Houwer, 2004; Waters, Nitz, Craske, & Johnson, 2007; Derryberry & Reed, 2002). These studies showed that negative affect is especially associated with reduced ability

to disengage attention from threatening information. Although these studies did not directly address whether delayed disengagement from threat was particularly associated with worry, they all focused on emotions that are likely to be caused by perseverative cognition such as worry (Hong, 2007) or rumination (Nolen-Hoeksema, 2000). Tentatively, it might be that delayed disengagement from threat seen across several psychopathologies is due to an association between attention processes and the transdiagnostic process of worrying. We therefore conducted the present study to investigate whether worry is associated with delayed attentional disengagement from threatening information.

To investigate the association between worry and attentional disengagement, we used an emotional modification of Posner's exogenous cueing task (Posner, 1980), which is often used in studies concerned with attentional disengagement. In this task, participants have to respond to a target presented at one of two locations, which is preceded by a cue that has either been presented at the same location as the target (a valid trial) or at the opposite location (an invalid trial). When there is a short period of time between the cue and the target (stimulus onset asynchrony (SOA) < 300 ms), responses appear to be faster to the valid trials. At longer SOAs (> 300 ms) responses to the valid trials are instead slower, which is thought to be due to inhibition of attention to the location on the screen that has previously been attended to (because a cue was presented), a phenomenon called inhibition of return (IOR; Posner & Cohen, 1984). Recent clinical studies of the emotional modulation of this phenomenon however prefer to use the term 'disengagement' instead of 'inhibition of return', since the debate is still ongoing whether these findings can best be explained by an attentional *inhibitory* mechanism, or by a biased attentional shifting mechanism (for a detailed account of the attentional mechanisms explaining the IOR effect see: MacLeod, Dodd, Sheard, Wilson, & Bibi, 2003; Spalek & Hammad, 2005). This spatial cueing task is made an emotional one by presenting as cues either schematic or realistic pictures of threatening (angry), neutral or happy faces (e.g., Fox et al., 2002; Fox et al., 2001), IAPS pictures (e.g., Yiend & Mathews, 2001) or arrows indicating wins and losses in a game (Derryberry & Reed, 2002). In the present study, we tested whether people with a strong tendency to worry (high trait worry) show a diminished disengagement, that is, a lower IOR effect, to angry faces, as compared with happy or neutral faces.

Trait worry is only one way to measure the tendency to worry, and tests for trait worry actually only predict behavior partially (Verkuil, Brosschot, & Thayer, 2007). Therefore, we also used a worry induction procedure, to test whether induced worry intensity is also associated with a reduced ability to disengage attention from angry faces.

Additionally, we wanted to examine whether the role of anxiety is important in these hypothesized relationships of worry with disengagement. Although worry and anxiety are closely related, several studies have made clear that worry and anxiety have independent associations with

health outcomes (e.g., Brosschot & Van Der Doef, 2006) and stress management strategies (Davey, Hampton, Farrell, & Davidson, 1992). We therefore also examined whether the hypothesized association between worry and attentional disengagement from threat was independent of the previously found association for anxiety (Yiend & Mathews, 2001; Koster et al., 2004; Fox et al., 2002; Waters et al., 2007; Derryberry & Reed, 2002), or whether it was the interaction between worry and anxiety that reduces attentional disengagement from threat.

In short, the present study was conducted to examine the following hypotheses: (1) trait worry is associated with decreased attentional disengagement from angry faces, relative to neutral and happy faces, independent of or in interaction with trait anxiety; (2) This association is also found for worry intensity after an experimental worry induction.

Method

Participants

Data were gathered from sixty-one student participants (mean age = 24.61, range 17 – 50). Sixty-seven percent of the sample was female. This study formed part of a larger study of the cognitive and physiological associates of worry and parts of this larger study have been reported elsewhere (Verkuil, Brosschot, Borkovec, & Thayer, in press). Participants were asked to perform several tasks for this experiment among which were the exogenous cueing task (see paragraphs 2.2 and 2.3) and the experimental worry induction (see paragraph 2.5). The order of these tasks was counterbalanced.

Apparatus and stimuli

To measure attentional disengagement, we used a task that was highly similar to the one used by Fox et al. (2002; experiment 2). Three schematic face types: 'angry', 'happy', and 'neutral' faces were used as cues. Each of the faces was 2 cm in diameter on the computer screen. The target that the participants had to localise was a black dot with a diameter of 0.5 cm. The cue and target stimuli were presented inside two light grey boxes that were continuously present on the computer screen. These boxes were 5 cm high by 3.0 cm wide and were displayed 2.25 cm to the left and the right of a central fixation point (shape: +). All stimuli were presented on a Dell computer with a 17" Dell LCD monitor (resolution: 1280 * 1024).

Procedure

Each trial started with a fixation point which was presented at the centre of the screen for 800 ms. A schematic face cue was then presented for 300 ms in either the left or the right box. This cue was then blanked out and 200 ms later the central cross was presented in bold type for 300 ms. The initial fixation display was then presented for 160 ms. Following this, the target was presented in the lower

half of either the left or the right box for 33 ms (Lupianez et al, 1997). Subsequently, the initial fixation display was presented until the participant responded (or until 2000 ms elapsed). This resulted in a cue-target onset asynchrony (SOA) of 960 ms. We used an intertrial interval of 1000 ms. Similar to the procedure used by Fox et al. (2002), each participant completed 16 practice trials, followed by 360 experimental trials, divided into five blocks of 72 trials. Fifty percent (180) of the experimental trials were valid (i.e., the target appeared in the same box as the cue), and 50% (180) were invalid (i.e., the target appeared in the opposite box to the cue). Angry, happy and neutral face cues appeared 60 times each on valid trials and 60 times each on invalid trials. The probability of any particular cue appearing in the left- and right-hand side boxes was equal, as was that of the types of faces.

All participants were seated 50 cm from the computer screen. They were told that the position of the cue did not predict the location of the target and therefore they should ignore the cue and keep their eyes focused on the centre of the screen and respond as quickly and as accurately as possible (Fox et al., 2002). The participant's task was to respond to the target which appeared either on the left or the right hand location by pressing the "Z" on the keyboard when the target was located on the left hand side of the screen and the "M" when the target was located on the right hand side of the screen. A standard QWERTY keyboard was used.

Trait questionnaires

Penn State Worry Questionnaire (Meyer, Miller, Metzger, & Borkovec, 1990; Dutch version; van Rijsoort, Emmelkamp, & Vervaeke, 1999). 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 as well as high temporal stability and substantial validity in the assessment of trait-worry (Meyer et al., 1990; van Rijsoort et al., 1999; Verkuil et al., 2007).

State Trait Anxiety Inventory-Trait Form (STAI-T; Dutch version: van der Ploeg, Defares, & Spielberger, 1980). For measuring trait-anxiety we administered the trait version of the State-Trait Anxiety Inventory. The STAI-T is a questionnaire that measures the participants' predispositions to anxiety. It consists of 20 self-report items and earlier use has shown good internal consistency and validity (van der Ploeg et al., 1980).

State measures

Experimental worry induction. Following the work of Borkovec and others (Lyonfields, Borkovec, & Thayer, 1995; Thayer, Friedman, & Borkovec, 1996; McLaughlin, Borkovec, & Sibrava, 2007), 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' (Lyonfields et al., 1995; Thayer et al., 1996; McLaughlin et al., 2007). After the worry induction, participants were asked to indicate on a ten point scale (1) the intensity with which they were able to worry, (2) the extent to which one negative thought led to another negative thought and (3) the extent to which the same thoughts occurred over and over again. The scores on these items were combined into a short state worry scale (Cronbach's alpha = .71).

State anxiety. The amount of state anxiety after the worry induction was assessed using visual analog scales (Brosschot et al., 1992; Johansson, 1976). Participants rated their level of anxiety at the start of the experiment (baseline) and after the worry induction. For this rating the participants was 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'. The change between 'mood as usual' and 'mood during the preceding period' was used to address the second hypothesis.

Statistical analyses

To investigate whether trait worry, trait anxiety or their interaction were associated with reduced disengagement from angry faces, but not from neutral and happy faces, we conducted a repeated measures ANOVA with Valence and Validity as within subjects factors, and trait worry, trait anxiety, and their interaction as continuous between subjects variables. To be able to examine significant interactions, we calculated cue validity effects for each of the three valences (Waters et al., 2007). Cue validity effects were obtained by subtracting the response latencies to valid trials from the response latencies to invalid trials (CV = RT invalid – RT valid). Negative values therefore indicated faster responses to invalid trials (suggesting IOR, i.e. attention away from the cue), whereas positive values indicated faster responses to valid trials (i.e. no IOR, but attention towards the cue). Relative cue validity effects were calculated by subtracting the CV effect for neutral faces from the CV effect for angry or happy faces. To test whether trait worry, trait anxiety and the worry induction measures were associated with the (relative) cue validity effects we calculated partial correlations and conducted simple slopes analysis in order to examine significant interactions. The predictor variables were centered in order to reduce multicollinearity (Aiken & West, 1991; (Frazier, Tix, & Barron, 2004). Because of our specific hypotheses we used one-tailed tests.

Results

Descriptive statistics

Table 1 shows the means and standard deviations for scores on the trait questionnaires and on the state measures. The mean levels of trait worry and trait anxiety were in line with previous studies

conducted with student participants (Startup & Erickson, 2006). There were no gender differences in trait anxiety, intensity of induced worry, state anxiety and the response latencies on the spatial cueing task (*p*s > .05). Women (M = 47.67, SD = 12.50) had a slightly higher score on the PSWQ than men (M = 41.58, SD = 12.71; *t*(34) = 1.74, p = .091). Reaction times on the different trials are presented in figure 1.

Table 1 *Means and standard deviations of and Pearson correlations between the trait questionnaires, induced worry intensity and state anxiety*

	M	SD	PSWQ	STAI-T	Induced worry intensity
PSWQ [1]	46.11	12.96			
STAI-T [2]	39.69	10.07	.73**		
Worry intensity	15.79	5.09	.49**	.30*	
State anxiety	0.55	1.79	.06	.18	.21

Note: [1] PSWQ = Penn State Worry Questionnaire; [2] STAI-T = State Trait Anxiety Inventory – Trait Version; ** Correlation significant at the .001 level (1-tailed); * correlation significant at the .05 level (1-tailed).

Spatial cueing task

Errors

The percentage of errors was 3.44%. No significant difference between the percentage rates of errors was found between the conditions. Trials in which the responses were incorrect were excluded from the analyses. In addition, trials on which the RTs were faster than 150 ms (anticipatory responding) and trials on which the RTs were longer than 2000 ms (misses) were excluded from the analyses (1.33%).

Reaction times

Mean reaction times were submitted to a repeated measures ANOVA with Valence and Validity as within subjects factors. This analysis showed a significant main effect of Validity ($F(1,62) = 57.39$, $p < .0001$, $\eta^2 = .48$), indicating a general IOR effect (invalid trials M = 297.81; valid trials M = 313.62). There was no significant effect of Valence, and no interaction between Validity and Valence.

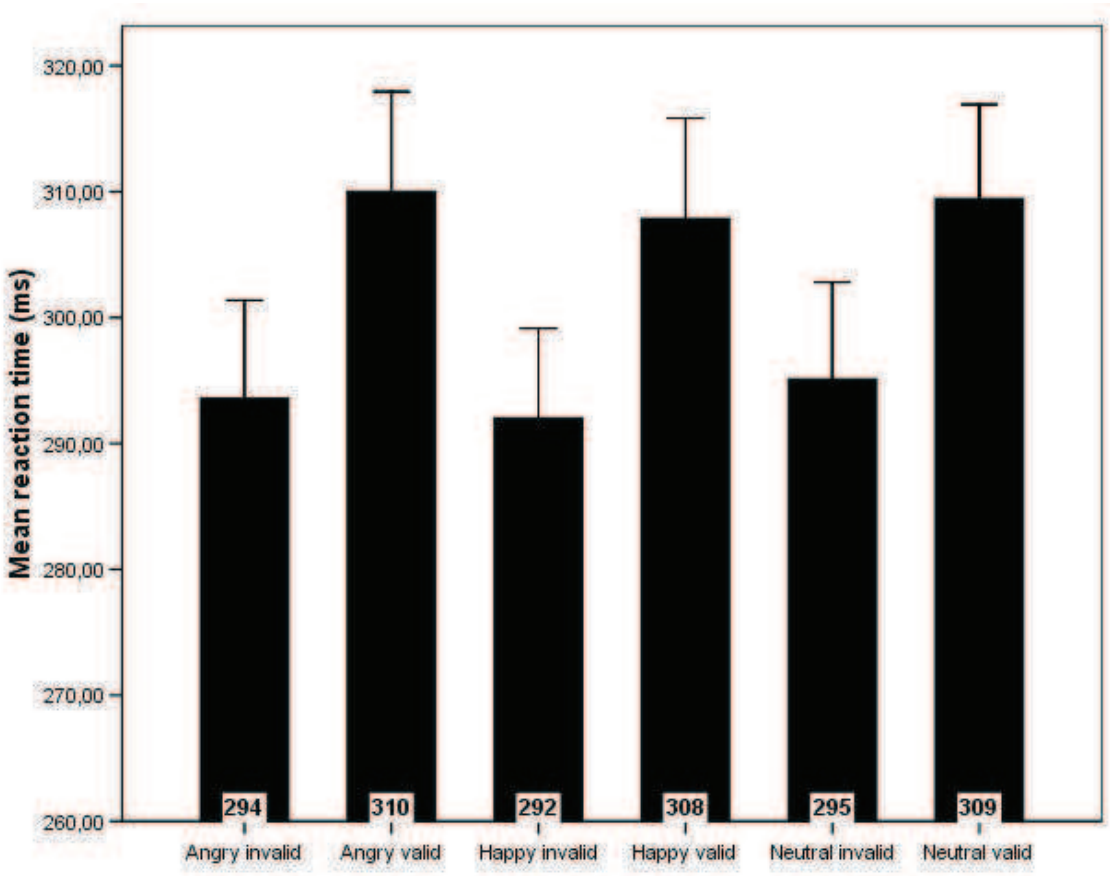

Figure 1. Mean reaction times on the spatial cueing task.

Association between traits and attentional disengagement

The results showed a significant interaction between Trait Worry and Valence x Validity ($F(2,116) = 2.63$, $p < .05$, $\eta^2 = .04$), and a marginally significant effect of Trait Anxiety x Valence x Validity $F(2,116) = 2.37$, $p < .06$, $\eta^2 = .04$). In addition, the four-way interaction of Trait Worry x Trait Anxiety x Valence x Validity was significant ($F(2,112) = 2.81$, $p < .05$, $\eta^2 = .05$). This four way interaction was further examined by inspecting the partial correlations between the cue validity effects and the interaction between trait worry and trait anxiety.

Partial correlation analyses on the CV effect for *angry* faces relative to neutral faces yielded a significant association with the interaction between trait worry x trait anxiety ($r(58) = .23$, $p < .05$). This association was due to the association between trait worry x trait anxiety and the cue validity effect for angry faces ($r(58) = .24$, $p < .05$), while no association was present for the CV effect for neutral faces. A simple slopes regression analysis on the cue attentional bias index for angry faces confirmed that attentional allocation to angry faces was associated with the interaction between trait worry and trait anxiety ($\beta = .26$, $p < .05$), while there were no main effects of trait worry and

trait anxiety. Figure 2 indicates that only when both trait worry and trait anxiety were high, disengagement from angry faces was reduced. Significance tests on the separate regression slopes showed that the slope of the high trait anxiety line was significant (β = .36, p < .05).

Partial correlations analyses on the CV effect for *happy* faces relative to neutral faces yielded a significant association with trait worry (r(58) = .31, p < .05), and, in the opposite direction, with trait anxiety (r(58) = -.29, p < .05). Yet, there were no significant associations between the cue validity effect for happy faces with trait worry or trait anxiety. Analyses on the CV effect for *neutral* faces however showed an association with trait worry (r(58) = -.21, p < .06), and an association with trait anxiety, again in the opposite direction (r(58) = .24, p < .06). This suggests that trait worry, independent of trait anxiety, is associated with reduced attention to neutral faces, whereas trait anxiety, independent of trait worry, is associated with prolonged attention to neutral faces.

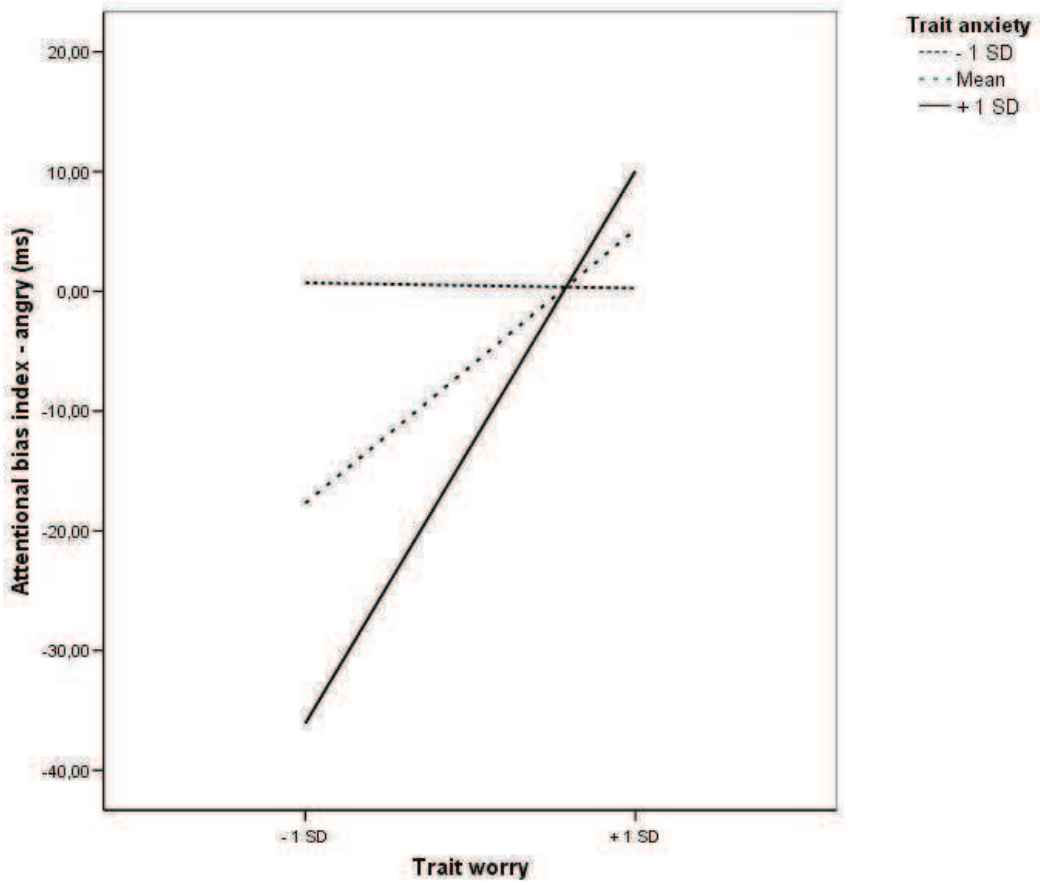

Figure 2. Attentional disengagement from angry faces relative to neutral faces as a function of trait worry and trait anxiety.

Association between induced worry intensity, state anxiety and disengagement

A repeated measures ANOVA with Valence and Validity as within subjects factors and induced worry intensity and state anxiety as continuous between subjects variables yielded a significant Induced Worry Intensity x Valence x Validity interaction ($F(2,114) = 4.61$, $p < .05$, $\eta^2 = .07$).

To examine this interaction, we calculated partial correlations between induced worry intensity and the attentional indices, while controlling for state anxiety and the induced worry intensity x state anxiety interaction. The results showed that induced worry intensity was significantly associated with the cue validity effect of angry faces, relative to neutral faces ($r(57) = .25$, $p < .05$) and with those of happy faces ($r(57) = .39$, $p < .05$). Yet, further inspection of this association showed that induced worry intensity was negatively associated with the CV effect for neutral faces ($r(57) = -.31$, $p < .05$), but not with the CV effects for angry or happy faces.

Trait and state predictors of attentional disengagement from threat

As trait and state variables are often found to have independent associations with performance on attentional tasks (e.g. Fox et al, 2001) and with health outcomes (Cohen et al), we assessed which of the trait and state variables was most directly associated with the attentional bias indices (Table 2). A forced entry regression analysis showed that the cue validity effect of angry faces was best predicted by the interaction between trait worry and trait anxiety ($\beta = .28$, $p < .05$), and by state anxiety ($\beta = .26$, $p < .05$). No significant predictors were found for the CV effect for happy faces. The CV effect for neutral faces was negatively predicted by induced worry intensity ($\beta = -.33$, $p < .05$).

Table 2. *Hierarchical regression analyses on the CV effects for angry, happy and neutral faces*

	CV angry faces				CV happy faces				CV neutral faces			
	β	t	*p*	R^2	*β*	t	*p*	R^2	*β*	t	*p*	R^2
Predictors				.18				.11				.14
(Constant)		-6.77	.00			-5.21	.00			-3.31	.00	
PSWQ	-.11	-0.52	.30		.06	0.29	.38		-.12	-0.54	.29	
STAI-T	.03	0.15	.44		-.15	-0.73	.23		.27	1.34	.09	
Ind. worry int.	-.02	-0.17	.43		.09	0.59	.28		-.33	-2.19	.02	
State anxiety	.26	1.97	.03		.16	1.12	.13		.07	0.51	.31	
PSWQ x STAI-T	.28	2.02	.02		-.09	-0.59	.28		-.10	-0.68	.25	
Ind. worry int. x State anxiety	.15	1.12	.13		.22	1.56	.06		.06	0.44	.33	

Note: PSWQ = Penn State Worry Questionnaire; [2] STAI-T = State Trait Anxiety Inventory – Trait Version; Ind. worry. int. = Induced worry intensity.

Discussion

The main aim of this study was to investigate whether trait worry and induced worry intensity were associated with lower attentional disengagement from threatening stimuli, i.e. angry faces and whether this was independent of anxiety or interacted with it. The results showed that trait worry was associated with lower disengagement from angry faces, but only when trait anxiety was also high. In addition, we found that both trait worry and worry during a worry induction were associated with reduced attention to neutral faces, an effect that was not dependent on anxiety. In contrast, trait anxiety was associated with prolonged attention to neutral faces.

The finding that at high anxiety levels worry is associated with reduced disengagement from angry faces provides further evidence for the proposal that pathological worry (worry that is associated with anxiety) is associated with enhanced elaboration of threatening information, as found in previous studies (Hazlett-Stevens & Borkovec, 2004; Friedman et al., 2000; Mathews & MacLeod, 1985; Mathews et al., 1995; Mogg et al., 1989; Mogg et al., 1995). However, this study adds to this previous work evidence that enhanced elaboration of threatening information may be particularly due to a reduced ability to *disengage* from it. A weak or deficient ability to disengage from threatening information will easily lead to the prolongation of worry episodes, which, in the long term, might exacerbate worry as a core psychological problem, and instigate meta-worry, the core problem of GAD. According to the perseverative cognition hypothesis (Brosschot, Gerin & Thayer, 2006), this prolonged worrying can also lead to *somatic* pathology because it adds to the total time that mental representations of threatening information provoke stress related physiological activation, which could eventually affect somatic health (Brosschot, Gerin & Thayer, 2006).

In addition, the correlation analyses suggested that trait worry and trait anxiety had independent opposing relations with the cue validity effect of neutral faces. Whereas trait worry was associated with reduced attentional disengagement from neutral faces, trait anxiety was associated with prolonged attention to neutral faces. Since it is unclear how participants interpreted these neutral schematic faces the main effects of worry and anxiety have to be interpreted cautiously. Still, the finding that worry was associated with reduced attention to neutral faces was also found when perseverative worry was measured after a proxy for a real-life worry bout (which usually also lasts 8 minutes on average; Brosschot, van Dijk & Thayer, 2007). Moreover, in the regression analysis induced worry intensity was the only significant predictor of attention to neutral faces. This might suggest that worry in essence is associated with an enhanced inhibition of return when neutral information is present, thereby biasing the attentional system away from neutral information, towards more salient information. Yet, this suggestion is tentative and should be addressed in future

studies. How worriers attend to and interpret neutral information certainly deserves more attention given the findings that GAD patients show smaller cardiac orienting responses and impaired habituation of cardiac orienting to neutral information (Thayer, Friedman, Borkovec, Johnsen, & Molina, 2000) and show equally enhanced BOLD responses when presented with neutral information as well as worry related information (Hoehn-Saric, Schlund, & Wong, 2004).

There are several limitations that have to be addressed. Foremost, we used a relatively young and non-clinical group. Although worry might even have adverse effects on health at non clinical levels and student samples are suitable to measure worry on the full severity range (Ruscio, 2002), it remains unclear to what extent our findings extend to pathological worry as observed in GAD. In addition, one could argue that the use of the STAI-T as a measure of trait anxiety has its limitations, as several items of the STAI-T seem to tap into depression (Bieling, Antony, & Swinson, 1998). However, worry has been found to be not only associated with anxiety, but also with sad mood and it could be that reduced attentional disengagement from threat found in anxiety and depression is mainly associated with the transdiagnostic process of worrying. Therefore the confounding between anxiety and depression in the STAI-T actually might add to the generalizability of the present results. Finally, we only examined reduced attentional disengagement at one stimulus onset asynchrony (SOA of 960 milliseconds between cue and target onset) and future studies should use more SOAs to be able to more specifically address the temporal aspects of this reduced attentional disengagement (Samuel & Kat, 2003).

A possible implication of the present findings might be that interventions might do well to focus more on the engagement-disengagement dimension in the worry process. The success of some novel therapies, such as mindfulness-based cognitive behavioral therapy, may be fruitful because they treat this dimension as an important first target (Ortner, Kilner, & Zelazo, 2007). For example, mindfulness based cognitive therapy is aimed less on changing the content, or threat value of worrisome cognitions, as in traditional CBT, but instead aims at disengaging or decentering from these thoughts through the use of meditation or breathing exercises. From a research standpoint a next step might be to conduct studies that investigate how these findings obtained in a laboratory setting transfer to the experience of worry episodes in daily life, for example by linking reduced attentional disengagement from threat to the frequency and duration of worry episodes as captured by momentary assessments. It might be that people that are high in trait worry, but who do not report anxiety, experience other kinds of worry episodes in their daily lives, relative to people high in both trait worry and trait anxiety. It might be that the former experience frequent but short lasting worry episodes, possibly indicating successful problem solving, whereas the latter experience frequent and long lasting episodes that are characteristic of pathological worry.

All in all, the results of this study suggest that specific threat-related deficiencies in disengagement may be a function of the co-occurrence of worry and anxiety.

Chapter 9

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

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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.

Introduction

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 & Kaplan, 1999). However, the exact psychophysiological mechanisms by which stressors have these adverse effects are not well understood (Pieper & 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 & 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 & Cacioppo, 2007; Thayer & Lane, 2000; Thayer & Lane, 2007; Thayer & 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, & Thayer, 2007; Gerin, Davidson, Christenfeld, Goyal & Schwartz, 2006; McCullough, Orsulak, Brandon & Akers, 2007; Pieper, Brosschot, van der Leeden & 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 & Deihl, 1993; Borkovec, Robinson, Pruzinsky & DePree, 1983; Davis, Montgomery & Wilson, 2002; Hofmann et al., 2005; Lyonfields, Borkovec & Thayer, 1995; Thayer, Friedman & 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 et al., 1993; 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 & 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 & 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 & Cohen, 2004; Greene, Sommerville, Nystrom, Darley & 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 & Leon, 2007; Ramos-Álvarez, Moreno-Fernádez, Valdés-Conroy & 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 & 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 & 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 & 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 & Thayer, 2007).

State Trait Anxiety Inventory-Trait Form (*STAI-T*; van der Ploeg, Defares & 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 & 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 *p*s > .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 *p*s > .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).

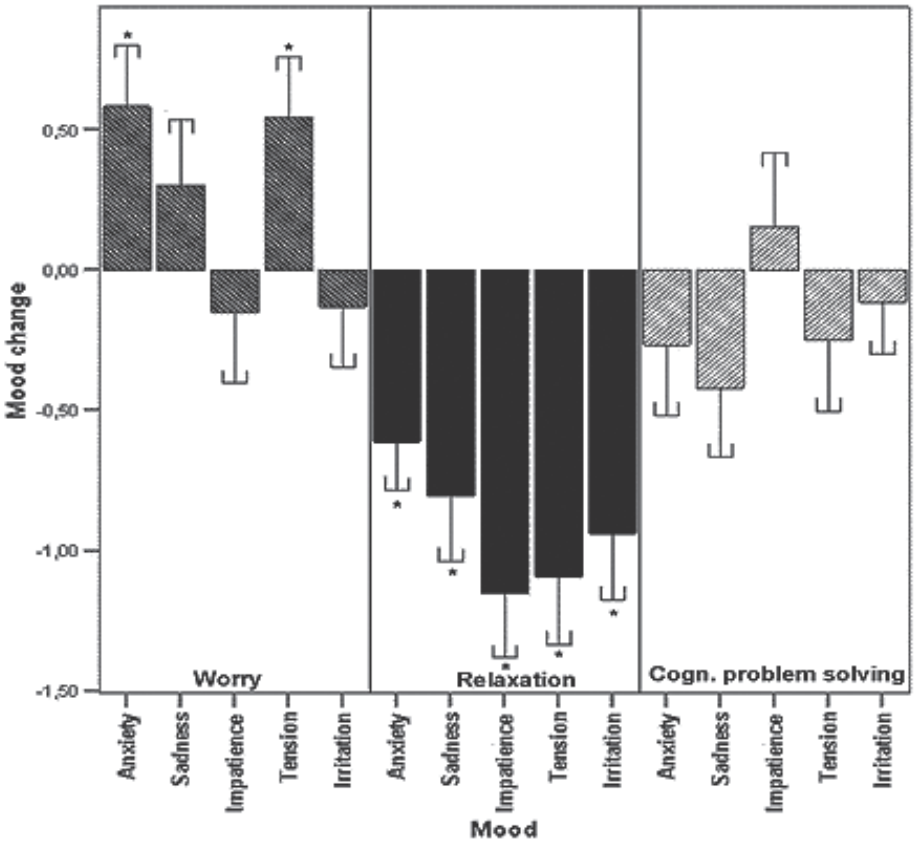

Figure 1. Effects of worry, relaxation and cognitive problem solving on mood. Bars marked with asterices (*) are significantly different from baseline (mood as usual). Error bars represent +/- 1 standard error.

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.

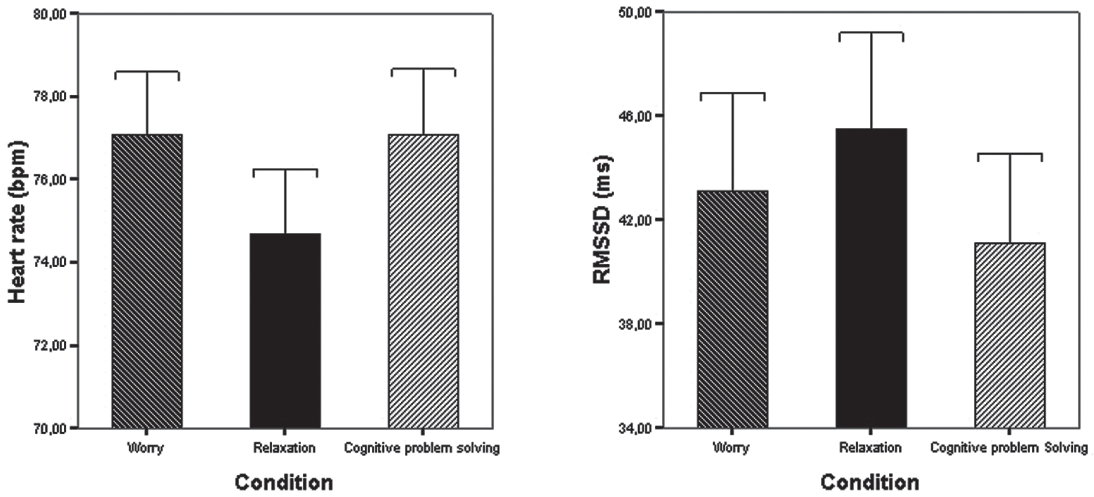

Figure 2. Effects of worry, relaxation and cognitive problem solving on heart rate and RMSSD. Error bars represent +/- 1 standard error.

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, η^2 = .12) and that the interaction effect Condition x Gender x Trait worry was significant ($F_{(2,48)}$ = 4.56, p = .015, η^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.

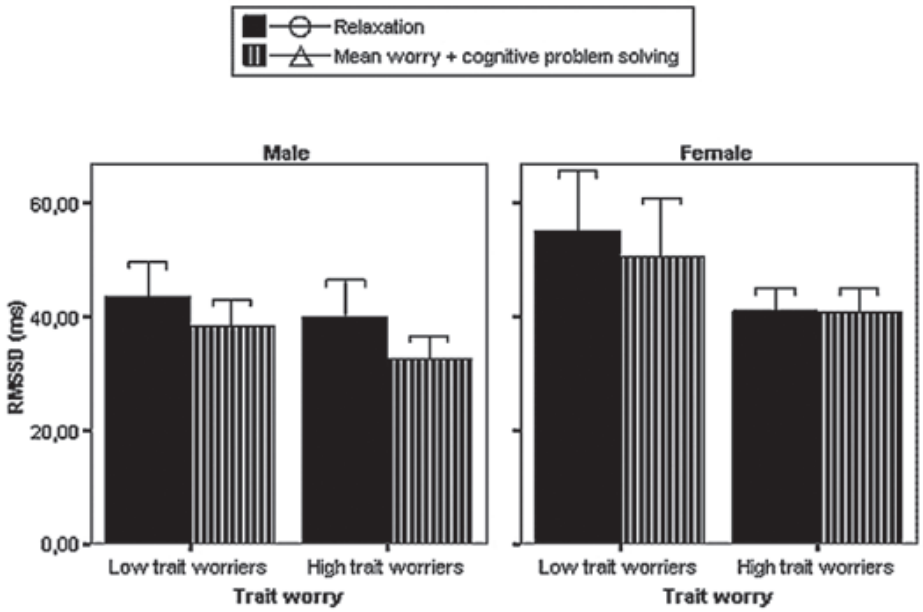

Figure 3. Individual differences moderating the effects of the cognitive tasks (worry and cognitive problem solving) and relaxation on RMSSD. Error bars represent +/- 1 standard error.

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 & 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 & 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 & 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 & 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 & 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 & Allen, 2007; Thayer, Smith, Rossy, Sollers & 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 & Lane, 2000). This compensatory response may explain why women with subclinical depression have decreased mortality (Hybels, Pieper & 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 & 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.

Chapter 10

General Discussion

General discussion

For decades the question that has been the focus of stress research is how stressful events can ultimately lead to disease. The main aim of this thesis was to add knowledge to this quest by examining the effects of worry about stressful events on somatic health, operationalized as somatic health complaints and slowed cardiac recovery after stress, both of which are associated with morbidity and mortality (Cole, 2000; Idler & Benyamini, 1997; Sha et al., 2005). Furthermore, the mechanisms by which worry was hypothesized to affect somatic health were examined. The studies reported in this thesis add to the accumulating number of studies testing the perseverative cognition hypothesis (Brosschot, Gerin, & Thayer, 2006), which is described in chapter 2. This hypothesis states that only prolonged cognitive representations of stressful events (perseverative cognition) lead to sustained or prolonged stress-related physiological activity which, in turn, can lead to somatic disease.

Taken together, the studies reported in this thesis provide evidence for the perseverative cognition hypothesis, that is, worry predicted adverse somatic health outcomes. In addition, the results of the studies presented in the second part of this thesis yielded several new and more specific hypotheses concerning the mechanisms behind the health effects of worry. These results warrant future studies that systematically test these hypotheses, or examine the robustness of the results already found. Below, the main findings from the studies reported in this thesis will be discussed, along with their limitations and their potential implications.

Measurement of worry

The majority of the studies testing the perseverative cognition hypothesis have relied solely on trait questionnaires measuring the tendency to worry or ruminate (Brosschot, Gerin & Thayer, 2006). It is commonly believed that these trait worry questionnaires are a good reflection of what people experience in their daily lives, but this had never been tested. Therefore, before testing the effects of perseverative cognition on somatic health, we examined the extent to which trait worry questionnaires correspond to worry in daily life. This study (see chapter 3) showed that trait worry questionnaires, like the well validated and reliable Penn State Worry Questionnaire (PSWQ), and momentary assessed real-life worry only have 24% shared variance. We therefore decided to include measures of state worry in our studies, either by using ecological momentary assessments (chapters 4 and 5), by measuring the occurrence of worrisome thoughts using retrospective state questionnaires (chapters 6 and 7), or by experimentally inducing a worry episode (chapters 8 and 9). The fact that we not only found additional but also differential effects of trait and state measures of worry has important implications for future studies on worry. The decision about which measure to use to assess worry deserves far more considerate thought than it has hitherto been given.

Worry and somatic health

We examined the relation between worry and somatic health complaints, such as fatigue, headache and neck pain . Momentary assessed (state) worry in a sample of teachers was found to predict the occurrence of daily somatic complaints (Chapter 4). Furthermore, in line with the perseverative cognition hypothesis, this study was the first to show that worry mediated the effects that stressful events had on the occurrence of daily somatic complaints. This provides evidence for the idea that only prolonged effects of stressful events, in other words, worry, is the pathogenic ingredient in the link between stressful events and somatic health problems.

In addition to these prospective effects, we performed a randomized clinical trial in which we aimed to reduce the effects of worry on somatic and mental health in clinical outpatients suffering from severe work stress. The results (see chapter 5) showed that a simple worry intervention was effective in reducing the frequency and duration of nighttime worries, which in turn were associated with decreases in somatic complaints. Moreover, the intervention added to the effectiveness of a subsequent stress management therapy. In another recent study (not included in this dissertation) we tested the effectiveness of the worry postponement intervention in children and found that it was helpful in reducing worry and somatic complaints in seventh grade children (Jellesma, Verkuil, & Brosschot, 2009). In sum, three studies now provide evidence that reductions in worry are associated with reductions in somatic complaints (i.e. chapter 5; Brosschot & Van der Doef, 2006; Jellesma, Verkuil, & Brosschot, 2009). Yet, the selectivity of the samples used (people suffering from severe work stress, children from primary and high schools) still leaves unexplored how the effects of reductions in worry generalize to the larger populations or other patient groups.

Finally, as a third type of evidence supporting the PC hypothesis, we found that induced state worry increased cardiac activity (chapter 9) and that trait as well as state worry were associated with slowed cardiac recovery after stress (chapter 6). In the latter study, state worry was operationalized as explicit worry (the amount of self-reported negative intrusive thoughts) as well as implicit worry (automatic vigilance measured with a lexical decision task). However, the results concerning implicit state worry were less straightforward than those from explicit state worry. Slowed recovery of the heart rate associated with slowed responses to control words depicting positive personality traits, and not with speeded responses to target words depicting task relevant / intelligence related information. Still, the results from the study provide further evidence for the perseverative cognition hypothesis and suggest that future studies should examine how implicit worry can be measured in more adequate ways.

All in all, evidence, in real life as well as in the laboratory, now makes clear that worry might play a substantial role in threatening somatic health. In addition, it was shown that a simple

intervention focused on retraining the ability of worriers to disengage from worrisome thoughts can be helpful in reducing worries and somatic complaints.

Worry and health: mechanisms

In the second part of this thesis, three mechanisms behind worry and its health effects were examined. The effect of worry on the occurrence of somatic health complaints that was shown in chapters 4 and 5 posed the important question how these effects are brought about. As mentioned above, one mechanism might be the one proposed by the perseverative cognition hypothesis, that is, that worry induces prolonged activity that may lead to somatic problems. Another mechanism was tested in chapter 7, namely whether somatic complaints are caused by worry about specific health complaints. The theoretical rationale for this study was based on the work of Ursin and Eriksen (2001) and Brosschot (2002), who proposed that somatic health complaints are produced by sensitization ('amplified processing') of neural networks that are involved in the processing of pain signals, both at somatic as well as cognitive levels. Sensitization would be reflected in lowered tolerance of pain (somatic sensitization) and enhanced elaboration of health information (cognitive sensitization). The results of the study indeed showed that the severity of somatic health complaints was associated with lowered tolerance of cold-pressor pain and with enhanced recall of health related information. Importantly for the context of this thesis, worry about specific health complaints mediated the association between recall of health related information and somatic complaints. This suggests that at least part of the somatic complaints can be explained by the fact that people keep on worrying about them. This also increases the chances that people decide to consult their general practitioners (Looper & Kirmayer, 2001), which adds to the economical costs that are associated with these somatic complaints. The other route, the prolonged physiological activation one, was not extensively tested (see the next section for some reasons). However, we tested and found support for one important part of that hypothesis, namely that worry is associated with increased physiological activity. In doing this, we corroborated existing empirical evidence but also extended this by testing whether the physiological (i.e. cardiac) effects of worry (chapter 6) were due to its emotional components, rather than to pure mental load (chapter 9). The amount of worry after a stressful task can not be easily controlled experimentally and leaves much room for individual differences. Therefore, instead of focusing on worry measured after stressful events, as done in chapter 6, we decided to use an often used worry induction procedure. Furthermore, we used a within-subjects design in which the cardiac effects of induced worry were compared to the cardiac effects of relaxation and cognitive problem solving. As expected worry clearly increased cardiac activity compared to relaxation, a finding that corroborated a still limited set of previous studies' findings (Lyonfields, Borkovec, & Thayer, 1995; Thayer, Friedman, & Borkovec, 1996; Davis,

Montgomery, & Wilson, 2002; Hofmann et al., 2005). However, worry did this to nearly the same extent as non-worry problem solving, which pointed towards the interesting possibility that the cardiac effects of worry are mainly due to mental load, in contrast with the largely held position that worry enhances heart rate due to its emotional components.

The success of the interventions (chapter 5) in reducing worry and health complaints suggest that it is possible to reverse some of the processes that cause worry. Since the interventions explicitly focused on disengaging from the worrisome thoughts, by observing them from a distanced perspective (chapter 5), it is tempting to believe that this was the process that was indeed changed. However, there was no proof of reduced disengagement in worriers in the literature, and it was difficult to test in these real life studies. Therefore we did this in a laboratory setting (chapter 8) and found evidence that worriers indeed have trouble disengaging from threatening information. More specifically, we showed that people who reported high worry as well as high anxious mood showed prolonged attention (sometimes referred to as 'dwell time' (Fox, Russo, & Dutton, 2002)) to threatening stimuli in an exogenous cueing task. This was taken as evidence that these people have difficulties in disengaging attention from threat. Therefore, training anxious worriers to disengage from such information might be beneficial. Interestingly, it might explain the finding in chapter 5 that worry registering on itself was already beneficial in terms of reducing daytime worry. It is possible that registering facilitates disengaging.

Below we will provide a theoretical integration of the main findings and will discuss these findings more in depth.

Theoretical integration of the main findings

The results of this thesis provide evidence for the extended perseverative cognition model that was presented in the introduction (see figure 1). The combined studies of the thesis suggest that perseverative cognition influences somatic health (chapter 4 and 5), either via prolonged physiological activity (chapters 6 and 9), or via a 'purely' cognitive route (chapter 7). In this thesis we found evidence for both these pathways, and it is interesting to speculate how they may be related.

It is likely that worry about health, just like worry about other stressful events, affects somatic health by prolonging physiological responses. Prolonged physiological activity may not only be detrimental for health in itself, but may also produce bodily sensations that can be interpreted by individuals who worry about their health as additional signs that one is suffering from a serious disease. In an extreme form this vicious cycle is observed in people suffering from hypochondriasis. It is a similar vicious cycle as the one observed in panic disorder in which catastrophic interpretations of harmless bodily sensations trigger physiological responses that evoke panic attacks (cf. Olatunji, Deacon, & Abramowitz, 2009).

A special comment should be made about the role of negative affect that is included as a factor in the perseverative cognition model. As stressful events evoke worry as well as negative affect, it was examined if the effects of worry depended on negative affect or exceeded its effects. In line with previous studies (Watkins, 2008), we found that momentary assessed state worry predicted state negative affect (chapter 4). In turn, state negative affect predicted somatic health complaints independently from worry (chapter 4). State negative affect (anxiety) was also positively associated with prolonged attention towards threat, independently of the interaction between trait worry and trait anxiety (chapter 8). Yet, in chapter 9, heightened negative affect did not explain the cardiac effects of worry. As negative affect was associated with subjective health and prolonged attention to threat, but not with objective indicators of health, these studies together suggest that negative affect might affect health mainly via the cognitive pathway, that is, by prolonging the attention that is paid to (the detection of) bodily signals and towards their catastrophic misinterpretation. In addition, negative affect enhances the prolongation of worry episodes by signaling that a problem hasn't been dealt with effectively, called mood-as-input (Startup & Davey, 2001; Davey, 2006). This prolonged worrying is likely to be accompanied by prolonged increases in physiological activity that, via the physiological route, also threaten somatic health – via increased mental load rather than negative affect.

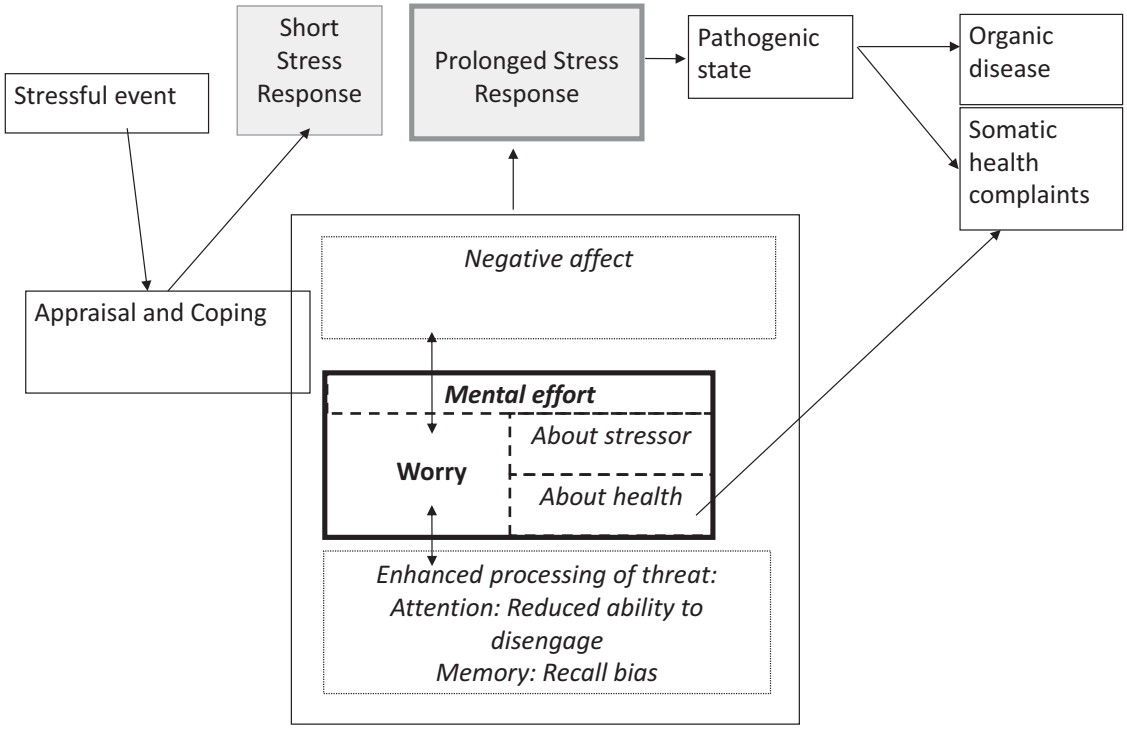

Figure 1. The extend perseverative cognition model

Worry, prolonged physiological activity and somatic complaints

In this thesis somatic health was operationalized as somatic health complaints and slowed cardiac recovery. The latter is actually an intermediate step between the experience of stressful events and ill health. Our main interest was in examining the role of worry in explaining these two adverse health outcomes, and therefore we did not address whether slow cardiac recovery mediated the effect of stressful events on somatic health complaints, nor did we test the cognitive pathway leading from daily worries about ones health to daily somatic complaints. A practical reason not to test these mediating pathways was that this would have required an elaborated real life study that would have covered a whole thesis (e.g., Pieper, 2008). Thus, future research should certainly consider investigating the link between somatic complaints and prolonged physiological activity. In fact, the cardiac effects of worry in real life as found by Brosschot et al., (2007) and Pieper et al. (2007) makes it tempting to speculate that at least a part of the health complaint effects of worry found in the current studies is due to prolonged physiological activity in real life. According to the perseverative cognition model, prolonged physiological activity should lead to increased somatic, organic problems. These organic problems should eventually be accompanied by bodily signals that can eventually be reported as somatic complaints. Yet, to date, relatively little is known about how prolonged activity of several bodily (e.g., cardiovascular, immune, endocrine) systems leads to somatic complaints, although several promising theories exist that are being currently investigated. In the context of chronic fatigue, Wyller et al. (2009) recently proposed that severe fatigue is due to prolonged physiological activity. Correlational evidence supported this idea by showing that people suffering from the chronic fatigue syndrome had elevated blood pressure and heightened body temperature when compared to controls (cf. Wyller, Eriksen, & Malterud, 2009). Yet, other studies found no differences in stress-related physiological activity between people suffering from multiple somatic complaints and healthy controls (Houtveen & van Doornen, 2007). In addition, another model for the link between prolonged physiological activity and somatic complaints was provided by Dantzer (2004; 2005) who proposed that pro-inflammatory cytokines, which can be induced by immune as well as non-immune (e.g., psychological) stressors, trigger sickness behavior, including somatic complaints, without necessarily inducing substantial activity in other physiological systems, such as the autonomic systems.

Future studies are clearly warranted to test both the physiological and the cognitive pathway in real life and also to gain more insight into how worry affects other physiological systems and how they are related to somatic complaints.

Specific components of worry, cardiac activity, and the moderating role of gender

The results from chapter 9 indicated that the cardiac effects of worry are mainly due to mental load. This could possibly mean that people who are continuously mentally occupied with problems, even

when they are non-emotional, are at heightened risk of developing health problems (although it is likely that it is easier to disengage from non-emotional topics than from emotional ones). However, the results from this study are in contrast with another recent study. In this study, the physiological difference between worry and mental load, that is, mental arithmetic (Oathes, Bruce, & Nitschke, 2008) was tested in a sample of men. Transcranial magnetic stimulation of the primary motor cortex yielded larger corticospinal motor responses during worry than during mental load. This was taken as evidence that in contrast with pure mental load, worry, due to its emotional component, is more closely associated with action preparation to escape from a threatening situation (the so called 'fight-flight' response). Yet, one could cast some doubt on the successfulness of the worry induction used in this study as differences in motor responses between the worry induction and a resting baseline were only marginally significant and only so for just one of the used indices of motor preparation. Inspection of the exact instructions provided to participants showed that during the worry induction, participants were instructed to relax their bodies, in order to allow an uncontaminated measurement of the motor responses. This instruction to 'relax while worrying' questions the validity of the procedure to induce worry. Nevertheless, it seems imperative to use a combination of physiological indices to further elucidate which components of worry are responsible for its physiological effects.

Another interesting finding from the studies described in chapters 6 and 9 pertains to gender differences. In line with animal studies showing gender differences in physiological responses to stress (Taylor et al., 2000), the studies reported in chapters 6 and 9 suggest that differences exist between men and women in cardiac vagal control during or after task performance. These differences were moderated by the tendency to ruminate (chapter 6) or worry (chapter 9). This gender difference was most clearly shown in chapter 9, in which we found that only female high trait worriers responded with increased heart rate variability during both cognitive problem solving and worrying compared to relaxation. Males and female low trait worriers showed reduced HRV during these mental tasks when compared to relaxation. In chapter 6 we found that in a largely female sample (80%), the tendency to brood about ones negative mood predicted increases in HRV during recovery after a stressful task. However, this effect was not found for the tendency to worry, which predicted decreases in HRV during recovery. It remains unclear why we did not consistently find that trait worry was associated with increased HRV during or after mental stress in women. Furthermore, a firm empirical basis for gender differences in physiological stress reactions is still lacking. Yet, as proposed in chapter 6, it is likely that trait worry and trait rumination exert their influence on cardiac activity via prolonged explicit and implicit cognitive representations of stressful events that arise in the anticipation or aftermath of these events. Recent work on the content of these representations showed that thinking about recent experiences of anger from a 'self-distanced' rather than a 'self-immersed' perspective reduced blood pressure reactivity (Ayduk & Kross, 2008). If we assume that

some cognitive representations of stressful events more easily trigger physiological responses than others, and men and women differ in the amount of these thoughts, this could possibly explain the inconsistency in the current findings. Furthermore, it could suggest that the function of perseverative cognitions differs by gender. For example, men might be more prone to show a prolonged fight-flight response in reaction to prolonged worry, while worry might encourage women to engage in tend-and-befriend behavior (Taylor et al., 2000), like communicating about their sorrows and fears with others. This latter form of perseveration is termed co-rumination (Rose, Carlson, & Waller, 2007) and might facilitate a self-distanced perspective on stressful events. It might explain several rather puzzling findings, not only the enhanced HRV in high trait female worriers in this study, but also enhanced HRV in depressed women (Thayer, Smith, Rossy, Sollers, & Friedman, 1998; Chambers & Allen, 2007) and even the lower cardiovascular risk of depressed women as opposed to depressed men (Hybels, Pieper, & Blazer, 2002).

The above makes clear that asking how frequent or long participants were thinking about a stressful event is not enough, and that more information is needed on *in what way* they where thinking about this event (that is, self-distanced versus self-immersed; concrete versus abstract; verbal thoughts versus images). Future studies that manipulate the exact content of the cognitive representations of stressful events among males and females are clearly needed to clarify gender differences in stress-related physiological responses.

Worry and the processing of concern-relevant information

Although investigating the cognitive correlates of worry was not the main aim of this thesis was per se, the cognitive elaboration of concern-relevant information was measured in chapters 6, 7 and 8. One perspective on how worry might be associated with information processing was offered in chapter 2. In general, information pertaining to a discrepancy between ones current state and ones goals (goal discrepancy) tends to stay activated until this discrepancy has been resolved, making it likely that people will start worrying about the discrepancy. We also proposed that in extreme worriers this discrepancy is persistently activated because they interpret even neutral or safety information as a threat to their current concerns (cf. Nitschke et al., 2009). Although methodological differences between the studies presented in this thesis limit firm conclusions, it might be interesting to summarize and discuss what this thesis adds to the current knowledge on worry and information processing.

First, in contrast with Koole et al. (1999) and Forster et al. (2005), we did not find that inducing a goal discrepancy (failure versus mixed failure and success) in chapter 6 led to perseveration of concern relevant information, that is, either explicit worry or implicit worry (automatic vigilance), which was assessed with a lexical decision task. Nor did we find that

performing an unsolvable task predicted speeded reactions to concern-relevant information (i.e. words related to intelligence) in the lexical decision task. The data of the lexical decision task made clear that the interpretation of this task is difficult as one can both expect speeded as well as slowed reactions to concern-relevant words (see also: Siegle, Ingram, & Matt, 2002; Algom, Chajut, & Lev, 2004). Yet, the slowing down of responses to concern-relevant words would be in line with the idea that worry and its associated emotional states (anxiety and sadness) are associated with prolonged attention towards concern-related information (chapter 8). As these effects have not yet been systematically examined, we do not recommend using the lexical decision task to measure implicit perseverative cognition.

In a second study (chapter 7) we found that after having exposed participants to a cold-pressor task, health worry was associated with enhanced recall of concern-relevant information (measured with an incidental free recall task). This finding is in line with studies showing that people suffering from generalized anxiety disorder and other anxiety disorders have biases in explicit memory (Friedman, Thayer, & Borkovec, 2000; Russo et al., 2006; Mitte, 2008). However, we did not find that health worry was associated with enhanced attention towards health information (measured with a modified Stroop task), which is in contrast with studies finding Stroop interference in patients suffering from generalized anxiety disorder (Mogg, Bradley, Millar, & White, 1995; Taghavi, Dalgleish, Moradi, Neshat-Doost, & Yule, 2003) We already proposed that this might have been due to the cold-pressor task that preceded the modified Stroop task which might have overruled the association between health worry and biased attention. Another explanation for not finding an association between health worry and biased attention might be that it is just not present in a healthy sample, or too subtle to be captured with a modified Stroop task. Health worry involves enhanced processing of current internal bodily sensations, and information about one's own health that is stored in memory ('I am a person who gets a cold easily, having a cold is terrible'). This attentional focus inwards might not be captured by a Stroop task that presents information externally on a computer screen. Attention towards external health information might only be observed when people have more severe complaints such as asthma or psoriasis (e.g., Fortune et al., 2003; Jessop, Rutter, Sharma, & Albery, 2004), or when they have read about and communicated with others about these complaints.

Still, worry in general, not limited to health, was associated with attentional processes in chapter 8. In this study we were the first to show that worry and anxiety interact and together predict prolonged dwell time on threat (angry schematic faces). In contrast with the modified Stroop task used in chapter 7, the task used in chapter 8 (exogenous cueing task) assessed *spatial* attention, that is, attention towards objects that are not presented at the location where one is currently looking at (e.g., a fixation point), but at peripheral locations that require a shift in attention. This

makes it possible to distinguish between the components of attention (orienting, engaging and disengaging), whereas the results from a modified Stroop task do not permit this distinction. Given that the interpretation of modified Stroop tasks are also under debate (Algom et al., 2004) and exogenous cueing tasks have yielded consistent results (e.g., Goeleven, De Raedt, Baert, & Koster, 2006; Koster, Crombez, Verschuere, Van Damme, & Wiersema, 2006), we advise future studies into the attentional correlates of worry to use exogenous cueing tasks.

In sum, worry was most strongly related to biases in explicit memory and to prolonged attention for threat in this thesis. This knowledge is of particular importance since cognitive biases now can be modified which might reduce emotional distress (MacLeod, Rutherford, Campbell, Ebsworthy, & Holker, 2002). Furthermore, as we proposed in chapter 2, these cognitive biases might have adverse physiological consequences that have to be examined in future studies.

Strengths and limitations

One of the major strengths of the studies presented in this thesis is that worry was not only measured using trait questionnaires. Besides being a more sensitive and better reflection of current real-life experiences of people, the advantage of using these state measures is that they can provide more information about the temporal dynamics (daytime versus nighttime worry) and the pathogenic ingredients of worrying (frequency, duration or intensity). For example, in chapter 5 we showed that especially nighttime worry was associated with health outcomes. Furthermore, daily worry predicted somatic complaints in chapter 4, while a trait measure of worry that provided a more global estimate of daily worries, the PSWQ, did not. However, a complication of the use of more than one index of daily worry is that they did not yield a consistent pattern of results concerning what aspects of worry were most detrimental for one's health. In chapter 4 we found that somatic complaints were predicted by worry duration and to a lesser extent by worry frequency, although these effects diminished when worry intensity was entered into the model. In chapter 5 we found that somatic complaints were best predicted instead by the frequency of worry episodes in the nighttime, and not by worry duration. These contrasting findings could likely be explained by differences in the methodology used (time based sampling in chapter 4 versus event based sampling of worry frequency and daily retrospective assessments of worry duration in chapter 5), but limit firm conclusions and warrant studies that test this explanation.

A second strength of this thesis is that the findings are not limited to one specific population, but can be easily generalized to a broader Western population. We studied the health effects of worry in relatively young and healthy student (chapters 3, 6, 7, 8 and 9) who are well suited to study worry on the full severity range (Ruscio, Borkovec, & Ruscio, 2001). However, we also studied the effects of worry on somatic health complaints in a population at risk for the development of work

stress (that is, teachers, chapter 4) and in people suffering from severe work stress, diagnosed with adjustment disorders (chapter 5). This latter population has only received little attention from clinical psychologists who are merely focused on more severe clinical populations. Non- or sub-clinical worry and somatic health complaints are very common and are responsible for huge personal suffering and high economical costs. More work on how to reduce the detrimental effects of worry is clearly required.

A third strength is that we did not solely rely on self-report questionnaires in the operationalization of our outcome measure, somatic health. Although subjective evaluations of one's health are the main determinant of whether one will visit a doctor or will call sick from work, the combination of subjective and objective measurements of ones health status results in more convincing evidence in support of the perseverative cognition hypothesis.

One could argue that the correlational / prospective nature of some of the findings is a limitation of this thesis. Yet, the cross-sectional and prospective findings that worry is associated with somatic complaints and cardiac activity (chapters 4 and 6) were followed up by studies using experimental designs that were aimed to directly manipulate worry (chapter 5: between subjects design; and chapter 9; within subjects design), thereby aiming to provide information of the causal relation between worry and health. Furthermore, in the introduction we already mentioned that the studies concerning the mechanisms behind worry (chapter 8) and its health effects (chapter 7) were set up to provide initial evidence for the proposed hypotheses, and to stimulate further research. We therefore believe that in order to test these hypotheses, it was justified to start off with cross-sectional designs. Yet, since the hypotheses tested were confirmed, experimental studies should further examine their tenability.

Another limitation to the results is that we can not provide a definite answer to the question at what level worry becomes detrimental to one's health. As described in chapter 2, worry can be regarded as a primarily adaptive response to threat. Only when people have repeated or continuous difficulties inhibiting worrisome thoughts, it is usually regarded as a clinical mental problem. Normative data exist for scores on trait worry questionnaires that facilitate decisions made by general practitioners and other clinicians on whether someone worries too much (and likely suffers from generalized anxiety disorder) and needs psychological treatment, such criteria have to be regarded with caution and cannot replace clinical interviews. With respect to somatic health, studies have shown that even non-clinical worry can be detrimental to one's somatic health (e.g. chapter 4). Yet, it is highly questionable whether a precise dose-response relationship between worry and somatic complaints will ever be established. Other factors that were not the main focus of the present studies also co-determine whether people will be more or less vulnerable for the development of somatic complaints or, in the long run, cardiac problems. For example, genetic

differences exist that predispose people for the development of somatic complaints (Gillespie, Zhu, Heath, Hickie & Martin, 2000), making it likely that individual differences exist in the total physiological load that people can handle before their health is affected.

Practical implications

This thesis resulted in the formulation of innovative hypotheses concerning the effectiveness of costly and time consuming stress management therapies (SMTs). The results of the study reported in chapter 5 suggest that SMTs can be enhanced if specific attention is being paid to the reduction of worry. Part of this intervention already forms part of the manual of occupational physicians on how to deal with work related stress (van der Klink & van Dijk, 2003) and the results of chapter 5 evidently advocate its use.

Besides the worry postponement and disengagement intervention, several other worry interventions exist, such as Competitive Memory Training (Korrelboom, Van der Gaag, Hendriks, Huijbrechts, & Beretty, 2008); Mindfulness Meditation (Jain et al., 2007), Attentional Bias Retraining (Hazen, Vasey, & Schmidt, 2009) and Concreteness Training (Watkins & Moberly, 2009). These interventions are aimed at different aspects of the worry process. As such, it is recommendable to compare the effectiveness of several of these interventions and to examine for who a specific intervention works best, which likely depends on factors like the severity of one's complaints, personality characteristics and co-morbid problems.

Conclusions and future directions

We can conclude from the studies presented in this thesis that when people keep on worrying about stressful events they report more somatic complaints and that they physiologically recover more slowly from such events compared to people who do not worry. Several ventures for future research have already been mentioned in the foregoing, here we will summarize the most important suggestions.

One important next step would be the replication of the studies presented in the second part of this thesis that examined the mechanisms behind worry and its health effects (chapters 7 – 9). Studies using ecological momentary assessments should be conducted to test whether worry about specific health complaints indeed predict somatic health complaints ('the cognitive pathway'). Furthermore, the cardiac effects of worry in daily life should be compared to that of non-emotional problem solving. In these studies gender differences should also be a main aim of investigation, given that the results of these studies suggest that men and women respond differently to stress.

A second venture would be to study the effects of implicit perseverative cognition. As mentioned in chapters 2 and 6, it is likely that cognitive representations of stressful events persevere

without conscious awareness and these might have physiological consequences. A first aim of studies should be to investigate how to capture implicit perseverative cognition. Thereafter, the physiological effects of implicit perseverative cognition during the daytime as well as during the nighttime, during sleep, should be examined.

Third, the effectiveness of relatively easy to administer worry interventions should be examined in larger populations. Dissemination of effective worry interventions can help preventing severe forms of work stress, anxiety, depression and somatic health problems. Besides being beneficial for individuals suffering from sub-clinical forms of these complaints, such interventions might also help to reduce the negative effects that these complaints have on job performance or absenteeism. Furthermore, they might as well help reducing the burden that clinical forms of these complaints currently place on health care systems (Jorm & Griffiths, 2006).

In sum, since its publication in 2006 (Brosschot et al., 2006), the perseverative cognition hypothesis has received gaining interest from researchers and has received support from several new studies (Pieper, Brosschot, van der Leeden, & Thayer, 2007; Zoccola, Dickerson, & Zaldivar, 2008; Holman et al., 2008; Brosschot, van Dijk, & Thayer, 2007). The studies presented in this thesis yielded additional support that worry is associated with somatic health complaints and prolonged cardiac activity, and extended the perseverative cognition model by pointing toward several new cognitive mechanisms underlying these relationships. Notably, two commonly held beliefs were challenged: (1) trait questionnaires are not sufficient to measure worry as they only predict a small extent of worry experiences in daily life and (2) the cardiac effects of worry are not due to its negative emotional experiences as we found that non-emotional problem solving yielded similar cardiac effects. All in all, this thesis further strengthens and extends the evidence for the view that prolonged cognitive representations of stress (worry) are crucial in the link between stressful events and somatic health and provide new avenues for future research.

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Nederlandse samenvatting

Perseveratieve cognitie: de invloed van piekeren op gezondheid

Iedereen maakt zich wel eens zorgen. Piekergedachten over werk, gezondheid of financiële problemen kunnen op zichzelf al als storend en vervelend worden ervaren en gaan vaak gepaard met gevoelens van angst. In dit proefschrift is er gekeken of piekeren een negatieve invloed heeft op onze lichamelijke gezondheid. Hoewel veel onderzoek al heeft laten zien dat stressvolle gebeurtenissen een negatieve invloed hebben op onze lichamelijke gezondheid, doordat ze bijvoorbeeld de kans op hart- en vaatziekten vergroten, is er nog weinig bekend over de invloed van het *piekeren over* stressvolle gebeurtenissen op de lichamelijke gezondheid.

Centraal in het proefschrift staat de 'perseveratieve cognitie hypothese', welke in de hoofdstukken 1 en 2 wordt geïntroduceerd. Perseveratieve cognitie is een term die sinds 2006 gebruikt wordt voor verschillende vormen van persisterende of herhaalde mentale, cognitieve representaties van stressvolle gebeurtenissen. Dit kunnen bijvoorbeeld angstige piekergedachten zijn over gebeurtenissen die nog moeten of zouden kunnen plaatsvinden, maar ook sombere gedachten over dingen die in het verleden zijn gebeurd. Volgens de perseveratieve cognitie hypothese hebben stressvolle gebeurtenissen een negatieve invloed op onze lichamelijke gezondheid omdat mensen over deze gebeurtenissen blijven piekeren. De fysiologische activiteit die optreedt tijdens een stressvolle gebeurtenis zou verlengd worden doordat mensen in gedachten met de stressvolle situatie bezig blijven. Wanneer deze fysiologische activiteit te lang doorgaat zou dit ten koste kunnen gaan van de lichamelijke gezondheid. Mensen zouden hierdoor meer lichamelijke klachten rapporteren en uiteindelijk ook hart- en vaatziekten kunnen ontwikkelen.

In hoofdstuk 2 wordt een overzicht gegeven van de studies waarin de perseveratieve cognitie hypothese is onderzocht. Dit overzicht maakt duidelijk dat onderzoeken in laboratoriumsettings laten zien dat piekeren na een stressvolle gebeurtenis zorgt voor een vertraagd herstel van de bloeddruk en het niveau van stresshormoon cortisol. Studies in het dagelijks leven laten zien dat de hartslag tijdens piekerperiodes is verhoogd, vergeleken met periodes van rust. Prospectieve studies laten bovendien zien dat piekeren is geassocieerd met het ontwikkelen van cardiovasculaire gezondheidsproblemen. Daarnaast wordt in dit hoofdstuk een model gepresenteerd waarin piekeren wordt gezien als een logische eerste reactie op bedreiging, nieuwigheid en onzekerheid. Drie psychologische factoren worden besproken die ervoor zorgen dat deze reactie bij chronische piekeraars blijft voortduren: het extreem veel waarde hechten aan het behalen van bepaalde doelen, het gebruiken van piekeren als een manier om met problemen om te gaan, en als laatste een

verminderd vermogen om in te zien dat er geen onmiddellijk gevaar dreigt, oftewel tekenen van veiligheid te herkennen.

In hoofdstuk 3 wordt onderzocht of veelgebruikte vragenlijsten over piekeren als individuele eigenschap ('dispositioneel piekeren') kunnen voorspellen hoe vaak en hoe lang iemand in het dagelijkse leven piekert. Dit is van belang omdat een groot deel van het onderzoek naar piekeren gebruikt maakt van deze vragenlijsten, terwijl het niet bekend is of ze wel bruikbaar zijn om daadwerkelijk piekergedrag te voorspellen, laat staan dat ze bij kunnen dragen aan het onderzoek naar de lichamelijke effecten van piekeren in het dagelijkse leven. De resultaten van de studie uit dit hoofdstuk wijzen uit dat slechts 24% van het piekeren in het dagelijkse leven wordt voorspeld door deze veelgebruikte piekervragenlijsten. Het is dus aan te raden om naast vragenlijsten over dispositioneel piekeren ook gebruik te maken van zogeheten ambulante meetmethoden, waar de hoeveelheid piekeren in het dagelijkse leven mee in kaart kan worden gebracht.

Om de perseveratieve cognitie hypothese te toetsen worden in de hoofdstukken 4, 5 en 6 de effecten van piekeren op lichamelijke gezondheid onderzocht. In hoofdstuk 4 wordt bekeken of piekeren het aantal lichamelijke klachten (bijvoorbeeld nekpijn, buikpijn, en verkoudheid) dat mensen dagelijks rapporteren kan voorspellen. Bovendien wordt bekeken of piekeren het effect van stressvolle gebeurtenissen op het aantal lichamelijke klachten kan verklaren. Voor deze studie hielden 69 docenten gedurende een week, met behulp van palmtops, dagelijkse registraties bij van stressvolle gebeurtenissen, piekeren en lichamelijke klachten bij. De resultaten lieten zien dat de vooral de intensiteit van het piekeren het aantal lichamelijke klachten voorspelde en bovendien het effect van stressvolle gebeurtenissen verklaarde. Deze studie ondersteunt hiermee de perseveratieve cognitie hypothese: stressvolle gebeurtenissen hebben een negatieve invloed op onze lichamelijke gezondheid omdat mensen over deze gebeurtenissen blijven piekeren.

Om te bekijken of een vermindering van piekeren ook zou leiden tot een vermindering van lichamelijke klachten is een interventieonderzoek gedaan, welke in hoofdstuk 5 is beschreven. Eerder onderzoek liet namelijk al zien dat een korte piekerinterventie, het opschorten van piekeren naar een dagelijks piekerhalfuur, effectief was in het verminderen van piekeren en lichamelijke klachten bij adolescenten. Het onderzoek in hoofdstuk 5 richtte zich op mensen die kampten met werkstress en die op de wachtlijst stonden voor een stress-management-therapie bij PsyQ. Omdat piekeren ook een centrale rol speelt bij het in stand houden van stressgerelateerde psychische klachten zoals angst en depressie, is in dit onderzoek ook bekeken of een vermindering van piekeren tevens een effect heeft op deze psychische klachten. Het tweede doel van dit onderzoek is te bekijken of een korte piekerinterventie (opschorten en leren loslaten van piekergedachten) aangeboden voorafgaand aan de stress-management-therapie effectief is in het versterken van deze therapie. Het idee hierachter is dat piekeren een centraal proces is bij het in stand houden van psychische en lichamelijke klachten

en dat het aanpakken van een dergelijk proces noodzakelijk is voor een effectieve behandeling. De resultaten van deze studie waren dat de piekerinterventie, vergeleken met het registreren van piekeren, leidde tot minder, en minder lange, piekerepisoden gedurende de nacht, maar niet overdag. Een vermindering van het aantal piekergedachten in de nacht was ook gerelateerd aan een vermindering aan het aantal lichamelijke klachten dat de deelnemers rapporteerden. Bovendien hadden – conform de verwachtingen – de deelnemers die voorafgaand aan de stress-management-therapie iets hadden gedaan aan het piekeren meer profijt van deze therapie; ze rapporteerden drie maanden na afloop van de therapie minder lichamelijke en depressieve klachten dan de mensen die de piekerinterventie niet hadden gekregen. Aangezien vorige studies geen duidelijke effecten vonden van stress management therapieën op lichamelijke klachten, is dit een relevante bevinding die zeker gerepliceerd dient te worden.

In hoofdstuk 6 wordt het effect van piekeren op het herstel van de hartslag en de hartslagvariabiliteit na een inspannende cognitieve taak (het moeten oplossen van onoplosbare analogieën) onderzocht. Vertraagd herstel van de hartslag is een voorspeller van cardiovasculaire problemen. Voorgaand onderzoek suggereerde al dat piekeren het herstel van de hartslag na emotionele gebeurtenissen kon vertragen, maar de resultaten waren echter niet eenduidig. In dit onderzoek is zowel naar de rol van piekeren als persoonlijkheidskenmerk gekeken als naar de hoeveelheid gerapporteerde negatieve gedachten na afloop van de cognitieve taak. Geheel nieuw was het meten van ‘onbewust piekeren’ na afloop van de cognitieve taak door middel van een lexicale-decisie-taak. Het is goed mogelijk dat mensen zich niet bewust zijn van een groot gedeelte van hun stressgerelateerde piekergedachten. Een heel groot gedeelte van onze dagelijkse informatieverwerking vindt plaats zonder dat we ons daar bewust van zijn, en het is mogelijk dat dit ook voor het verwerken van stressvolle gebeurtenissen geldt. De resultaten van dit onderzoek lieten zien dat zowel de neiging om te piekeren als het daadwerkelijke aantal negatieve piekergedachten geassocieerd zijn met een vertraagd herstel van de hartslag na de cognitieve taak. De resultaten van de meting van het ‘onbewuste piekeren’ waren echter lastig te interpreteren en vervolgonderzoek zou zich moeten richten op het onderzoeken van welke testen er geschikt zijn om ‘onbewust piekeren’ mee te kunnen meten.

In het kort gezegd ondersteunen de resultaten van de studies uit hoofdstukken 4, 5 en 6 in grote lijnen de perseveratieve cognitie hypothese. In de hoofdstukken 7, 8 en 9 zijn enkele mechanismen onderzocht die mogelijk verklaren waarom piekeren een effect heeft op lichamelijke gezondheid.

Hoewel de perseveratieve cognitie veronderstelt dat piekeren tot lichamelijke klachten leidt doordat piekeren stressgerelateerde fysiologische activiteit verlengd, is er nog een alternatieve route mogelijk, die getest is in hoofdstuk 7. Lichamelijke klachten kunnen te wijten zijn aan specifieke

piekergedachten over gezondheid. Volgens het zogenaamde sensitizatie-model ontstaan en verergeren lichamelijke klachten doordat pijn- en andere negatieve signalen uit het lichaam op alle niveaus (o.a. perifeer, ruggenmerg, hersenstam, limbische structuren, cortex) versterkt worden weergegeven. Mensen met extreem veel lichamelijke klachten zonder medische verklaring zouden lichamelijk gevoeliger zijn voor pijn, meer aandacht schenken aan gezondheidsinformatie in het dagelijkse leven, deze beter onthouden en bovendien ook vaker piekeren over hun gezondheid. In deze studie is onderzocht of dit ook opgaat voor meer alledaagse lichamelijke klachten, zoals hoofdpijn en rugpijn in verder gezonde vrouwelijke deelnemers. De resultaten van de studie in hoofdstuk 7 laten zien dat de ernst van verschillende lichamelijke klachten inderdaad geassocieerd zijn met een verlaagde tolerantie voor pijn en een beter geheugen voor gezondheidsgerelateerde informatie. Bovendien was de ernst van de klachten geassocieerd met de mate waarin de deelnemers piekerden over de lichamelijke klachten. Dit piekeren verklaarde ook de relatie tussen het versterkte geheugen voor gezondheidsinformatie en de ernst van de lichamelijke klachten. Hoewel dit correlaties zijn, en de richting van de oorzakelijkheid hiermee niet aangetoond, is, lijkt dit te suggereren dat piekeren ook via andere wegen dan de fysiologische een belangrijke invloed kan hebben op onze gezondheid.

Het gebruik van een piekerinterventie gericht op het leren opschorten en loslaten van piekergedachten (hoofdstuk 5) werd ondersteund door de resultaten van de studie beschreven in hoofdstuk 8. In deze studie werd aangetoond dat mensen die zich angstig voelen èn bovendien veel piekeren, moeite hebben om hun aandacht los te maken van bedreigende informatie.

In hoofdstuk 9 wordt een studie beschreven waarin werd bekeken of de effecten van piekeren op de hartslag en hartslagvariabiliteit toe te schrijven zijn aan de emotionele componenten van piekeren (zoals vaak wordt aangenomen), of aan het feit dat mensen tijdens het piekeren ook cognitief heel actief bezig zijn. Vergeleken met een periode waarin de deelnemers werd gevraagd om zich te ontspannen, bleken zowel piekeren als hard nadenken over niet persoonlijk relevante problemen dezelfde effecten te hebben op de hartslag en op de hartslagvariabiliteit. De effecten van piekeren op hartactiviteit lijken dus niet veel hoger te zijn dan die van neutrale problemen oplossen. Ligt dat effect van piekeren dan vooral aan de mentale inspanning ervan? Vervolgonderzoek zal meer duidelijkheid moeten geven over de consequenties van deze bevinding.

In het laatste hoofdstuk worden alle bevindingen nog eens op een rijtje gezet en besproken in het kader van de houdbaarheid van de perseveratieve cognitie hypothese. Een belangrijke conclusie is dat deze hypothese wordt ondersteund door de bevindingen in dit proefschrift. Piekeren heeft een negatieve invloed op lichamelijke klachten en op het herstel van de hartslag na een inspannende taak. Bovendien wordt het perseveratieve cognitie model uitgebreid doordat de specifieke mechanismen achter deze effecten in dit proefschrift zijn onderzocht. Voor de praktijk

levert het proefschrift bewijs op dat een korte interventie gericht op piekeren een daarop volgende stress management therapie versterkt. Van de vele vragen die nog resten zijn misschien wel de belangrijkste of piekeren ook onbewust of automatisch kan plaatsvinden, hoe dit het beste gemeten kan worden en of dit 'onbewuste piekeren' fysiologische effecten heeft.

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I don't quite know
How to say
How I feel
Those three words
Are said too much
They're not enough
Chasing Cars – Snow Patrol

Curriculum vitae

Bart Verkuil was born on September 23rd 1980 in Delft, the Netherlands. In 1998, after completing secondary school, Marnix Gymnasium in Rotterdam, he studied Clinical and Health Psychology at Leiden University. He graduated with honors in 2002. Thereafter he worked for two years as a clinician at mental health centre Parnassia, Voorburg. In 2004 he started his PhD project while continuing working part-time as a clinician at mental health centre PsyQ Business, The Hague. His PhD project was made possible by grants provided to Dr. Jos Brosschot (Leiden University) and Prof. dr. Julian Thayer (National Institute on Aging and the Ohio State University, USA). The studies conducted as part of this project focused on the effects of worry on somatic health.

Besides working on his PhD project, he published a book on severe work stress and burnout, together with Dr. Arnold van Emmerik (University of Amsterdam). In addition, he is involved in research on the effects of the work environment on physiological measures of stress, in collaboration with Prof. dr. Esther Sternberg (National Institutes on Mental Health, USA).

Next to working part-time at mental health centre Skils, The Hague, Bart Verkuil currently holds a position as Assistant Professor at the Department of Clinical Psychology of Leiden University.

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