Available online at www.sciencedirect.com
ScienceDirect
International Journal of Psychophysiology 63 (2007) 39–47
INTERNATIONAL JOURNAL OF PSYCHOPHYSIOLOGY
www.elsevier.com/locate/ijpsycho

Daily worry is related to low heart rate variability during waking and the subsequent nocturnal sleep period

Jos F. Brosschot [a,*], Eduard Van Dijk [a], Julian F. Thayer [b]

[a] Division of Clinical and Health Psychology, Department of Psychology, Leiden University, Leiden, The Netherlands
[b] Department of Psychology, The Ohio State University Columbus, OH, USA

Received 24 March 2006; received in revised form 5 July 2006; accepted 28 July 2006
Available online 3 October 2006

Abstract

Stress and anxiety are risk factors for cardiovascular (CV) disease. Worry might be a mediator of their risks by prolonging their cognitive representation and concomitant CV activity. We hypothesized that daily stressors and worry, and trait anxiety and trait worry would be associated with high heart rate (HR) and low heart rate variability (HRV) during waking and the subsequent nocturnal sleep period, and that worry would mediate the effects of daily stressors. Low HRV and high HR are physiological risk factors for CV disease. Using an hourly diary, stressors, worry frequency and duration, and biobehavioral variables were measured during one day in 52 healthy subjects. During this time and the subsequent nocturnal sleep period, ambulatory ECG was measured. Stressors, worry and traits were related to higher HR and lower HRV during waking, and the effects of stressors and worry were extended into the sleeping period. Worry duration mediated the effects of stressors. The results were largely independent of biobehavioral variables including sleep quality. The results support the notion that worry, by prolonging CV activity, is a mediator of the CV risks of stress. They also imply a role for unconscious cognitive representation of stress.

Keywords: Stress; Perseverative cognition; Worry; Trait anxiety; Prolonged physiological activity; Heart rate variability; Sleep physiology; Cardiovascular disease

1. Introduction

Overall mortality from organic disease, particularly cardiovascular disease is co-determined by chronic psychosocial stress, including anxiety (Krantz and McCeney, 2002; Kubzansky and Kawachi, 2000; Rosengren et al., 2004; Rozanski et al., 1999; Scheier and Bridges, 1995). Worry has been argued to be an important mediator of these effects (Brosschot et al., 2005, 2006; Friedman and Thayer, 1998). There are several reasons to hypothesize this. First, in order to have substantial effects on health, the physiological effects of stress and anxiety must be prolonged, for example by slow recovery (Brosschot and Thayer, 1998; Linden et al., 1997; McEwen, 1998; Selye, 1950; Sluiter et al., 2000; Ursin, 1980; Ursin and Eriksen, 2004). Brosschot, Thayer and colleagues have hypothesized that worry might be the mediator of the prolonged physiological effects of stress, because worry theoretically may be the primary mechanism by which a person prolongs a stressor's cognitive representation, along with its physiological effects (Brosschot et al., 2005, 2006; Gerin et al., 2001). Some laboratory experiments have already yielded suggestive evidence that slow blood pressure recovery after emotional stress is due to worry or rumination (Gerin et al., 2006; Glynn et al., 2002). However, there have been no studies that tested this hypothesis in real life.

Second, worry is a core mechanism in anxiety disorders. These disorders are associated with an increased risk for cardiovascular (CV) disease. Worry might be responsible for at least a part of this risk by mediating prolonged CV activity related to anxiety. Finally, several studies have shown that trait worry as well as state worry are associated with increased physiological activation, especially CV activation (Brosschot et al., 2002; Dua and King, 1987; Gerin et al., 2006; Glynn et al., 2002; Lyonfields et al., 1995; Roger and Jamieson, 1988; Scheier and Bridges, 1995; Segerstrom et al., 1999; Suchday et al., 2004; Thayer et al., 1996; Vickers and Vogeltanz-Holm, 2003; see for review: Brosschot et al., 2006). At least one study has shown that worry predicts CV disease (myocardial infarct; Kubzansky et al., 1997).

* Corresponding author. Division of Clinical and Health Psychology, Department of Psychology, Leiden University, P.O. Box 9555; 2300 RB Leiden, The Netherlands. Tel.: +31 71 527 3377; fax: +31 71 527 4678.
E-mail address: brosschot@fsw.leidenuniv.nl (J.F. Brosschot).

0167-8760/$ - see front matter © 2006 Elsevier B.V. All rights reserved.
doi:10.1016/j.ijpsycho.2006.07.016

To summarize, worry seems to prolong the cognitive representation of stress, it is a core element in stress and anxiety, and it appears to have substantial physiological effects. These properties of worry make it a likely candidate as a mediator of prolonged activity related to stress and anxiety and a co-determinant of their cardiovascular risk.

The hallmark of a mediator of prolonged physiological responses to stressors is that it extends these responses even into periods in which stressors are absent. Sleep is perhaps the most important of these types of recuperative periods, because it covers a large part of our life and is the most critical natural episode for psychological and somatic restoration. Thus, the role of worry as a cause of prolonged cardiac activation may be even more convincing if it can be shown that its cardiac effects are prolonged during sleep at night. To date, there have been no studies showing direct effects of worry on cardiac activity during sleep. Indirectly, worry is related to poor sleep quality, which in turn is related to heart disease and general mortality (Dew et al., 2003). Moreover, during poor sleep, low levels of heart rate variability (HRV) and high levels of heart rate (HR) have been found (Hall et al., in press). A recent review (Pieper and Brosschot, 2005) showed that several types of stress were associated with prolonged cardiovascular effects during sleep. Stressful events in the past six months were associated with high sleeping HR (Ituarte et al., 1999), and frequent episodes of negative emotions were related to a higher blood pressure (BP) during sleep (Shapiro et al., 1997). Trait anxiety was also related to higher sleeping BP (Pasic et al., 1998; Raikkonen et al., 1999). There was only one experimental study of stress and sleep, that showed that anticipating an oral speech the next morning was related to low vagally-mediated HRV throughout the whole preceding sleeping period (Hall et al., 2004). The latter effect could not be explained by poor sleep quality. It is therefore possible that stress and perhaps worry can increase physiological levels without necessarily disturbing sleep quality. Studies with work stress yielded less consistent results. Three work stress studies (Schnall et al., 1998; Unden et al., 1991; Vrijkotte et al., 2000) obtained evidence of prolonged cardiovascular activity during sleep while two others did not (Fauvel et al., 2001; Goldstein et al., 1999), and a third study found an effect only when family stress was also high (Brisson et al., 1999). Pieper and Brosschot (2005) concluded that job stressors might be too specific or often not sufficiently distressing to yield effects that extend beyond the working floor.

The present study tested two main hypotheses. First, worry and stressors during the day, and trait anxiety and trait worry, are hypothesized to be associated with high HR and low HRV during both waking and subsequent nocturnal sleep. Second, it was hypothesized that – at least part of – the increased waking and sleeping cardiac levels of daily stress but also of trait anxiety and trait worry are mediated by daily worry. Low HRV is associated with increased risk of cardiovascular morbidity but also all-cause mortality, and has been proposed as a general marker for disease (Palatini and Julius, 1997; Stein and Kleiger, 1999; Task Force Guidelines, 1996; Thayer and Friedman, 2004; Tsuji et al., 1994) and high levels of HR have also been linked with all-cause mortality in several large studies (see Habib, 1999, for a review). The present study measured worry episodes and their duration, daily stressors, and several biobehavioral variables during one day using an hourly diary. Worry duration was measured in addition to the mere frequency of worry, because if worry is shown to be the mediator of prolonged activation, this will be even more the case when worry itself is prolonged. Moreover, it was previously found that worry duration is a better predictor of somatic symptoms than worry frequency (Brosschot and Van den Doef, 2006). HR and HRV (root mean square of successive differences of inter beat intervals, RMSSD) were assessed with ambulatory equipment during the day as well as the subsequent night.

2. Materials and methods

2.1. Subjects and procedure

Subjects were recruited from the general population in the area around the city of Leiden in The Netherlands by way of newspaper advertisements. They received the equivalent of 25 US dollars for their participation. Complete data were available for 52 subjects, who will be the focus of this study. Thirteen of them were men, and thirty-nine were women, aged between 15 and 65 (mean = 33.8; S.D. = 13.9). The subjects came to the laboratory between 8:00 AM and 10:00 AM. They returned questionnaires that measured trait worry, trait anxiety, age and gender that they received via the mail. Next, they were instructed about the use of the diary and an ambulatory physiological measurement device (see below). The latter apparatus signaled the subjects to complete the diary with a short 'beep' approximately every hour (plus or minus 10 min). Each hourly diary entry contained questions about stressors and worry during the preceding measurement period. The electrodes and the apparatus were attached and the subjects left the laboratory. The signals continued until 11 PM, to preclude interference with sleep onset. The next morning, the subjects indicated their sleep quality during the preceding night. Thereafter, they returned the diary and apparatus to the laboratory, were debriefed, and received their monetary compensation.

2.2. Heart rate and heart rate variability during waking and sleeping

HR and HRV were measured by the Ambulatory Monitoring System (AMS; De Geus et al., 1995; version 4.6. TD-FPP, Vrije Universiteit, Amsterdam, the Netherlands). This device has been used extensively and details of its characteristics have been published elsewhere (De Geus et al., 1995). In the present study the electrocardiogram signal was recorded using disposable pre-gelled Ag–AgCL electrodes (ConMed, New York, USA) that were placed at the jugular notch of the sternum, 4 cm under the left nipple and at the lateral right side. Using this three electrode configuration only the inter beat interval time series was available for analysis. The device detects the R-wave of the electrocardiogram and records the time in milliseconds (with one millisecond resolution). From the raw inter beat intervals the device derives and stores 30-second averages of HR (in