Prolonged Cardiac Effects of Momentary Assessed Stressful Events and Worry Episodes

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Objectives: To test the hypothesize that increased heart rate (HR) and decreased heart rate variability (HRV) are not only due to concurrent stressful events and worries but also to stressors and worries occurring in the preceding hours or stressors anticipated to occur in the next hour. Worry was expected to mediate at least part of the prolonged effects of stressors. **Methods:** Ambulatory HR and HRV of 73 teachers were recorded for 4 days, during which the participants reported occurrence and duration of worry episodes and stressful events on an hourly basis, using computerized diaries. Multilevel regression models were used, accounting for effects of several biobehavioral variables. **Results:** Stressful events were not associated with changes in HR or HRV. However, worry episodes had effects on concurrent HR and HRV (2.55 beats/minute; −5.76 milliseconds) and HR and HRV in the succeeding hour (3.05 beats/minute; −5.80 milliseconds) and 2 hours later (1.52 beats/minute; −3.14 milliseconds). These findings were independent of emotions, physical activity, posture, and other biobehavioral factors. **Conclusion:** Worry has effects on cardiac activity, and these effects were still visible after 2 hours. The latter finding suggests that a considerable part of prolonged activation may be induced by unconscious stress-related cognition. **Key words:** stressors, worry, prolonged activation, heart rate, heart rate variability.

CV = cardiovascular disease; **HR** = heart rate; **HRV** = heart rate variability; **BP** = blood pressure; **BMI** = body mass index; **ECG** = electrocardiogram.

INTRODUCTION

Prolonged cardiovascular (CV) responses to stressors—and not so much the relatively short responses during stressors—are recognized to strain and wear out the CV system to the extent that they may lead to cardiovascular disease (CVD) (1). Studies (1–3) have shown that delayed cardiac recovery from stressors is predictive of adverse cardiac outcomes. According to a prolonged activation model of the effects of stress on CV health (4), the level of stress-related CV activation during episodes in daily life is not only influenced by psychological stressors occurring during these episodes but also by more "distant" ones, such as stressors in the past and stressors anticipated in the future. These more distant effects are hypothesized to be due to ongoing cognitive representations of stressors called "perseverative cognition" (4,5). For practical reasons, laboratory studies of stress recovery have only tested restricted recovery periods, thereby limiting their ecological validity. Ambulatory studies in natural environments have measured longer time periods and have suggested that CV stress effects may last any period between 5 minutes and the rest of the day and subsequent nocturnal sleep (1,6). Most of these studies, however, failed to indicate where exactly prolonged activation started and how long it exactly lasted after the stressor. Without this information, it is difficult to document precisely prolonged activation and to distinguish it from mere reactivity. The present study's first aim was to compare, in daily life, cardiac effects that occur during stressors with the prolonged effects of these stressors at various temporal distances before and after the stressors.

Its second aim was to test whether worry mediates these prolonged effects. None of these ambulatory studies (Reviewed in 1) investigated a psychological mediator of the prolonged physiological effects. We (4,5) proposed that perseverative cognition, such as worry or rumination, may prolong physiological activation beyond the actual occurrence of a stressor. When a stressor cannot be readily coped with, perseverative cognition will keep the cognitive representation of the stressor active along with its physiological concomitants. On the other hand, a study (6) suggested that daily worry can have prolonged effects, for example, during the subsequent nocturnal sleep. Therefore, we will also test whether worry episodes have prolonged cardiac effects themselves.

Worry and rumination have been shown to increase CV activity in the laboratory as well as in real life (5) and to prolong CV effects after laboratory stressors (7–10). With respect to real life, this was shown only in the above-mentioned study (6), which found that worry mediates the effects of daily stressors on nocturnal heart rate (HR) and heart rate variability (HRV). That study was limited in several ways. No exact beginnings and endings of stressors and worry episodes were measured. Therefore, no short-term prolonged activity during the day, including anticipatory activation, was analyzed. Furthermore, potential confounders of the effects of stressors and worry, such as emotional states and physical activity, were not measured. The current study measured stressors, worry episodes, and anticipated stressors more precisely, including their prolonged effects during daytime. Additionally, we replaced paper-and-pencil diaries by electronic diaries, improving reliability by automatically time-locking the reports.

The current study was partly based on data used in a previous report (11) that concerned only cardiac activity during exactly determined stressful episodes. We extended this work by examining whether the average cardiac activity at any given time period is not only predicted by stressors or worry occurring during that period but also by stressors or worry occurring during several predetermined time periods before that period, and even by stressors expected to occur after that

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period. The advantage of this approach is that these questions can be answered with a single statistical test, using multiple predictors from concurrent as well as the preceding periods (12). For this purpose, we calculated average HR and HRV during the last 15 minutes of measurement periods of approximately 60 minutes. We chose 15 minutes because it is sufficiently short to allow the persons to adequately remember and indicate their emotions and physical posture. It also enabled us to examine the short-term prolonged effects of stressors occurring earlier in the same 60-minute measurement period. In this way, five different durations of prolonged activity were tested: 1) stressors occurring simultaneously with the cardiac assessments (marked "0" in this article); 2) in the same hour but before the measurements ("−1"); 3) in the previous hour ("−2"); 4) in the hour before that ("−3"); and 5) stressors the participant anticipated in the next hour ("+1"). Subsequently, the effect of worry during these periods was measured to investigate whether worry had similar effects and mediated, at least in part, the prolonged effects of these stressors.

Summarizing, we expected increased HR and decreased HRV to be related to stressors and worry episodes that occur simultaneously as well as in the preceding 3 hours and also to stressors anticipated to occur in the next hour. Furthermore, we expected worry in these periods to mediate at least part of these effects. We used HR and HRV because both chronic high HR and low HRV are risk factors for CVD, as well as other organic diseases and all-cause mortality (13), and because they are easy to measure in daily life without interfering with natural behavior. Age, gender, body mass index (BMI), bodily motion, time of day, and the consumption of coffee, alcohol, and smoking can influence HR and/or HVR (14–20); we, therefore, controlled for their effects. Due to the hierarchical structure of the data, we used multilevel regression analyses.

METHODS

Participants

Seventy-three teachers were measured between 2001 and 2003, consisting of 49 men and 24 women aged 24 to 69 years (mean, 46.7 years; standard deviation [SD], 9.5), who were employed for an average of 34.0 hours per week (SD, 9.5). Initially, 102 teachers were willing to participate: 29 were excluded due to various reasons (pregnancy, sick leave, allergy to electrodes, use of antidepressants or hypertension medication, or insufficient data recordings). All gave their written informed consent and received a book token worth 20 Euros for participating. The study was approved by the university ethics committee.

Procedure

Soon after recruitment, a preparatory session followed at the each teacher's school, with several tests and questionnaires (not reported here), in which they signed the informed consent. Within the next 2 weeks, an experimenter fitted the ambulatory electrocardiographic (ECG) device (21) in the morning before the teachers started their regular activities. The experimenter instructed the teachers on how to use the ECG device and a handheld computer that contained hourly diary questions, including questions about worry episodes and stressful events (Fig. 1). The teachers carried both devices for two phases of 48 hours each, where one contained two workdays and one contained two nonworkdays. These two phases were on average measured 1 week apart.

Diary Format

A Palm m100 handheld device (Palm Inc., Santa Clara, California) was used for the hourly diary. Additionally, we used customized software (Pendragon Forms, version 3.1, Pendragon Software Corporation, Libertyville, Illinois) to implement questions. An hourly tone (±15 minutes) was set for 8 AM to 10 PM at which time participants were instructed to fill in the computerized questions. When the subjects answered the first question of

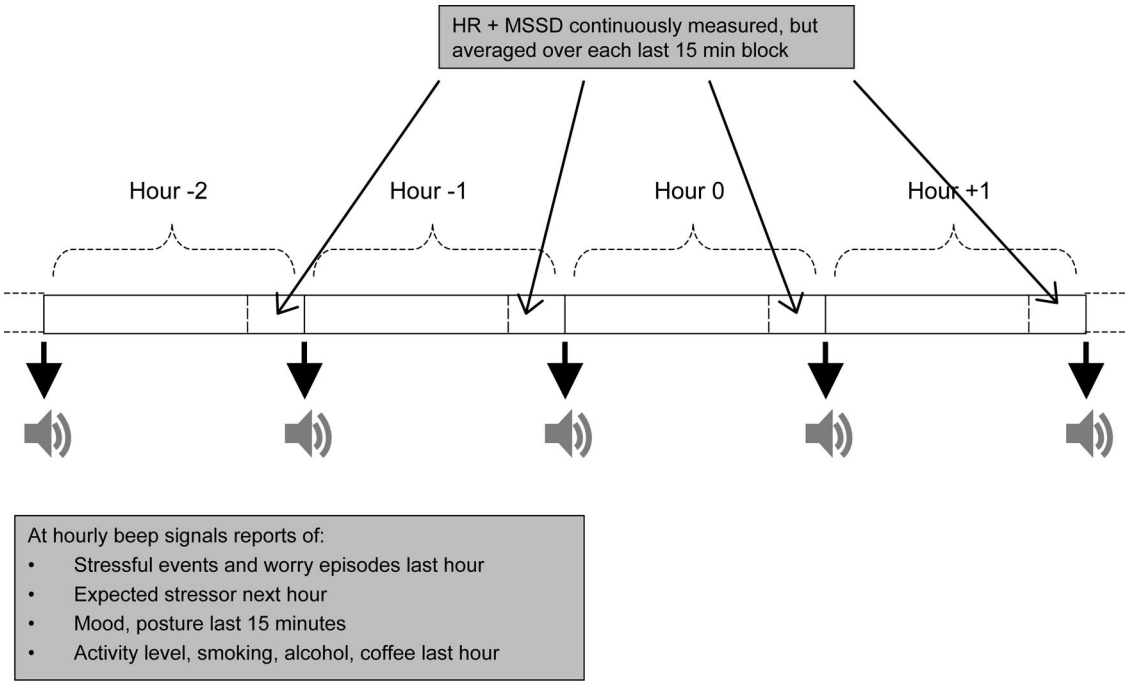

Figure 1. Cardiac measurements and self-reports during ambulatory procedure. HR = heart rate; MSSD = root mean square of successive differences of interbeat intervals.

each log entry, the present time was stored to enable comparison between their responses and cardiac measurements. Due to the 15-minute random prompt, measurement periods were anywhere between 45 minutes and 75 minutes, and "hours" or "hourly measurements" should not be taken too literally in the remainder of this paper.

Worry Episodes and Stressful Events

The subjects received definitions of worry episodes and stressful events in print before starting the momentary measurements. The word for worry in Dutch ("piekeren") can also mean "thinking hard" or "pondering." To make sure that the subjects used the right concept, we introduced the word "ruminneren" (rumination) which is a seldomly used Dutch word, and defined a "rumineer" episode or worry episode as "*when you, for a certain period of time, feel worried or agitated about something. It is a summary-term for processes such as worry, ruminating, keeping on about something, fretting or grumbling about some problem or angry brooding etc. Thus, it is about a chain of negative thoughts that is hard to let go of.*" By using this definition, we additionally made sure that the subjects would also report types of perseverative cognitions other than only worry, such as angry brooding and rumination. Stressful events were defined as "*all minor and major events due to which you, to any extent, feel tense, irritated, angry, depressed, disappointed or otherwise negatively affected.*" Subsequently, on the handheld computer, the participants reported at the time of the hourly tone whether a worry episode or a stressful event or both had occurred during the preceding hour and what their approximate starting points and duration were. Additionally, they reported whether they expected a stressful event to occur in the upcoming hour (22).

This procedure allowed us to create the following independent variables: stressors and worry episodes occurring during the last 15-minute period of each hourly measurement (marked $\text{Stressor}^{0,}$ Worry (0)), during the same hour but preceding that last 15-minute period ($\text{Stressor}^{-1,}$ Worry^{-1}), during the preceding hours ($\text{Stressor}^{-2,}$ Worry^{-2}, $\text{Stressor}^{-3,}$ Worry^{-3}), and stressors expected in the next hour (Stressor^{+1}). Some stressors and worry episodes continued across one or even several "hours." To prevent using them multiple times for predicting the same cardiac effect, we counted them only once. Importantly, stressors and worry episodes from hourly diary reports that were >20 minutes earlier than the next report were not used. This yielded a total of 368 sequences of connected measurement periods, with two to 14 measurement periods per sequence; 71.7% of these sequences were between 3 periods and 13 periods long.

Mood, Activity, and Other (Bio)Behavioral Variables

During the last 15 minutes of each hourly measurement period, the subjects reported on the computer to what extent they had felt the following four moods: being angry or irritated; sad or gloomy; tense or restless; and happy or cheerful (not at all, some, a bit, much, very much). They also reported what their main posture had been in those last 15 minutes (lying, sitting, standing, walking, biking, other), and they reported on consumed units of tobacco, coffee, and alcohol (0, 1–2, 2–4, or more) in the preceding hour, and on the level of physical activity in the preceding hour (not at all, some, a bit, much, very much). Because of their skewed distributions, the smoking, alcohol, and coffee variables were dichotomized into yes/no variables. A more objective estimate of high activity was obtained with the ECG device, which includes an accelerometer sensitive to changes in vertical acceleration. This motility signal was used to distinguish periods with high activity from periods with low activity. High physical activity were identified as motility higher than the 48-hour average plus 1 SD (indicating high physical activity) in combination with a visually detected simultaneous increase of HR, which was presumably due to this high activity. The percentage of 30-second periods that were spent in high activity during each 15-minute period was used as a covariate to control for cardiac differences due to intense movement. Additionally, each period got a time code (1 = morning until 12 PM; 2 = afternoon until 6 PM; 3 = evening until sleep), providing the variable "time of day."

Cardiac Activity

Raw interbeat intervals were acquired continuously by the VU-AMS device (version 4.6, TD-FPP, Vrije Universiteit, Amsterdam, Netherlands) (21), and the ECG signal was recorded, using disposable pregelled Ag-AgCL electrodes (ConMed, Utica, New York,) placed at the jugular notch of the sternum, 4 cm under the left nipple and at the lateral right side. From the raw interbeat intervals, the device derives and stores 30-second averages of HR (in beats/minute) and root mean square of successive differences (MSSD) of interbeat intervals (in milliseconds), which we used as an index of HRV. MSSD is recommended for ambulatory studies, as it is less influenced by respiration (23). For the current analyses, only the cardiac measurements of the last 15 minutes of each hourly period were used. Of these last 15 minutes, we calculated mean HR and MSSD after eliminating all parts with outliers in SD, mean, minimum, and maximum values of HR, MSSD, inter-beat-intervals, and motility. Because of the skewed distribution of MSSD (lnMSSD), this variable was log transformed.

Statistical Analysis

To test the effects of predictor variables on the 15-minute averages of HR and MSSD, multilevel regression analysis was used (24,25), which made it possible to account for the different measurement levels and to correct the likely correlations between measurements within individuals. There were three levels: the 15-minute periods of HR and MSSD measurement (*period level*), which were nested within several sequences of connected measurement periods per participant (*sequence level*), which in turn were nested within persons (*person level*). Each of these levels is responsible for variance in the dependent variables HR and MSSD, and the multilevel analysis is used here to test whether variables at the episode level (e.g., stressor, worry) and person level (e.g., gender, BMI) can explain this variance.

A sequence of three models was tested for HR and MSSD each. First, an intercept-only model was fit containing no predictor variables. This model (Null Model) decomposes the variance of the dependent variable into three independent components, pertaining to the period level, the sequence level, and the person level, and was used as a baseline model. Thereafter (Model 1), we examined the effects of concurrent worry episodes (Worry^{0}) and stressful events (Stressor^{0}), as well as previous stressful events (Stressor^{-1} to Stressor^{-3}), worry episodes (Worry^{-1} to Worry^{-3}), and expectation of stressful events (Stressor^{+1}) by adding these variables to the model. Finally, it was tested whether the effects of stressors and worry episodes were independent of emotional responses and biobehavioral variables, by adding the following variables (Model 2): emotional states, reported posture, percentage of high activity, reported level of activity, time of day, cigarette smoking, consumption of alcohol and coffee, gender, age, BMI, and an autocorrelation parameter.

In general, the multilevel method described here is similar to those commonly used. The main additional feature is that for each measurement period, the impact of observations from earlier measurement periods is accounted for—in the form of stressors and worry episodes from these earlier periods. This means that the same predictor variable was often used more than once. For instance, stressor^{0} predicting HR plays the role of stressor^{-1} in predicting the next adjacent 15-minute value of HR, and so on. As a result, errors in prediction might be correlated. This additional source of dependency in the multilevel regression model is taken into account by explicitly modeling the correlation between successive observations called the autocorrelation. Omitting the autocorrelation would bias the standard errors of the regression coefficients downward and may consequently lead to mistaken rejection of the null hypothesis. Autocorrelation estimates were obtained, using an MLwiN macro similar to that shown in the work of van Eck and colleagues (12). The final model (Model 2) was refined by including the autocorrelation parameter.

To test the hypothesis that the prolonged effects of stressors were mediated by concurrent as well as subsequent worrying, we additionally tested models without worry and compared these models with the models above, including worry. If the prolonged effects of stressors were stronger and more significant without entering the worry episodes, it might be concluded that

worry mediates at least partly the effects of these variables. This was verified with the Sobel statistic (26).

The effects of the predictor variables in all models were considered fixed, because we did not have a specific interest in their random effects. Multilevel regression models were fit, using the program MLwiN, version 2.02 (27). All models were estimated by the method of maximum likelihood. Hypotheses concerning the significance of fixed effects were tested using one-tailed *t* tests, as these hypotheses were explicitly directional. The *t* values were obtained by dividing the estimated model parameter by its standard error.

RESULTS

Descriptive Statistics

A total of 1957 hourly measurement episodes (on average, 26.81 ± 13.12 episodes per participant) were used in the analyses. Of these, 8.7% contained one or more stressors, and 6.1% contained one or more worry episodes, and 2% contained both. Subjects reported a mean of 1.58 stressful events (SD, 1.16) and 1.06 worry episodes per day (SD, 1.69), with most subjects reporting two stressful events ($n = 15$ subjects) and no worry episodes ($n = 35$ subjects) over the total measurement period (adjusted for a differential total number of episodes per person). These frequencies are comparable with findings from other studies, i.e., 1.38 and 1.65 for stressful events (12,22) and 0.96 per day for worry episodes (28).

Prolonged Effects on HR

Concurrent stressful events (Stressor^0), worry episodes (Worry^0), anticipated stressful events in the next hour (Stressor^{+1}). and those reported previously (Stressor^{-1}, Worry^{-1}, Stressor^{-2}, Worry^{-2}) were added as predictors to the intercept-only model (Model 1, Table 1). Stressor^{-1}, Worry^0, and Worry^{-2} showed significant effects on HR ($z = 1.68$, $p < .05$, $z = 2.51$, $p < .01$ and $z = 2.49$, $p < .01$ respectively) and were associated with increases in HR of 2.02 beats/minute (95% confidence interval [CI], 0.82–3.22), 2.86 beats/minute (95% CI, 1.72–4.00) and 2.51 beats/minute (95% CI, 1.50–3.52), respectively. Worry^{-1} was marginally associated with an increase of 2.85 (95% CI, 0.99⁻4.71; $z = 1.53$, $p < .10$) beats/minute. There were no other significant effects. Overall, this model fits well in comparison with the intercept-only model ($\chi^2 = 307.69$, $df = 7$, $p < .001$). To test a possible mediator effect of worry, the analyses were repeated without

TABLE 1. Effects of Stressful Events and Worry Episodes on Heart Rate

	Model 1 Estimate ± SE (*p* value, *t* test one-sided)$^{\text{(two-sided)}}$	Model 2 Estimate ± SE (*p* value, *t* test one-sided)$^{\text{(two-sided)}}$
Fixed effects		
Intercept	77.45 ± 1.02 (<.001)$^{<.001}$	63.08 ± 1.65 (<.001)$^{<.001}$
Stressor^0	1.18 ± 0.95 (.11)$^{.22}$	0.73 ± 0.92 (.45)$^{.90}$
Stressor^{-1}	2.02 ± 1.20 (.046)$^{.09}$	1.45 ± 1.02 (.08)$^{.16}$
Stressor^{-2}	0.15 ± 0.76 (.43)$^{.86}$	0.08 ± 0.66 (.45)$^{.90}$
Stressor^{+1}	−1.15 ± 1.81 (.26)$^{.52}$	−0.23 ± 1.39 (.44)$^{.88}$
Worry^0	2.86 ± 1.14 (.006)$^{.01}$	2.55 ± 0.98 (.004)$^{.01}$
Worry^{-1}	2.85 ± 1.86 (.06)$^{.12}$	3.05 ± 1.56 (.02)$^{.05}$
Worry^{-2}	2.51 ± 1.01 (.006)$^{.01}$	1.52 ± 0.88 (.04)$^{.08}$
Angry		0.43 ± 0.55 (.22)$^{.44}$
Sad		−0.05 ± 0.82 (.48)$^{.96}$
Tense		0.93 ± 0.50 (.03)$^{.06}$
Happy		0.14 ± 0.34 (.34)$^{.68}$
% High activity		10.68 ± 0.67 (<.001)$^{<.001}$
Activity level		3.25 ± 0.34 (<.001)$^{<.001}$
Posture		2.46 ± 0.20 (<.001)$^{<.001}$
Gender		4.36 ± 2.18 (.02)$^{.04}$
Age		−0.15 ± 0.11 (.08)$^{.16}$
BMI		0.35 ± 0.30 (.12)$^{.24}$
Smoking		1.80 ± 1.48 (.11)$^{.22}$
Alcohol consumption		0.99 ± 0.70 (.07)$^{.14}$
Coffee consumption		0.60 ± 0.51 (.12)$^{.24}$
Time of day[a]		−0.03 ± 0.33 (.46)$^{.92}$
Variance components		
Person level		
Intercept (σ^2_v)	64.12 ± 12.24	43.51 ± 9.88
Sequence level		
Intercept (σ^2_u)	29.12 ± 3.77	11.33 ± 3.43
Period level		
Intercept (σ^2_e)	73.08 ± 2.70	6.51 ± 3.51
Deviance	14246.76	8456.58

SE = standard error; BMI = body mass index.

[a] 1 = morning; 2 = afternoon; 3 = evening.

worry variables. The effect of Stressor^{-1} became slightly higher (increase in HR of 2.30 beats/minute (95% CI, 1.10–3.50; z = 1.92, $p < .05$), but a Sobel test showed no evidence for mediation (Sobel = 1.49, $p = .14$). Stressful events and worry episodes that happened earlier (Stressor^{-3}, Worry^{-3}) did not have significant effects. For simplicity's sake, they were left out of the models below.

Next, emotional (being angry, sad, tense, happy) and physical (percentage of high activity, subjective activity level, and posture) variables as well as gender, age, BMI, smoking, coffee and alcohol intake, and time of day were added to the previous model (Model 2, Table 1). There were several independent significant effects. A 1-unit increase of tense emotional states (on a scale of 5 units from "not at all" to "very much") was marginally related to increases in HR of 0.93 beats/minute (95% CI, 0.43–1.42; z = 1.86; $p < .05$). Maximal percentage of high activities was associated with a mean increase in HR of 9.88 beats/minute (95% CI, 9.16–10.60; z = 13.72; $p < .001$). Additionally, 1-unit increase in activity level (on a scale of 5 units from "not at all" to "very much") and posture (on a scale of 6 units from "lying" to "other posture") were related to increases in HR of 3.08 beats/minute (95% CI, 2.71–3.45; z = 8.35; $p < .001$) and 2.73 beats/minute (95% CI, 2.51–2.94; z = 12.62; $p < .001$), respectively. Females displayed a higher mean HR of 4.36 beats/minute (95% CI, 2.18–6.53; z = 2.00; $p < .05$) than males. Alcohol consumption was marginally related to a 0.99-beats/minute increase in HR (95% CI, 0.29–1.68; z = 1.42; $p < .10$). With a best-fitting autocorrelation parameter of 0.17, the overall fit of Model 2 was good in comparison with Model 1 ($\chi^2 = 5790.18$; $df = 15$; $p < .001$). Importantly, the inclusion of these factors in the model did not markedly change the effects of Worry^{0} and Worry^{-2}. The effect of Worry^{-1} changed from marginally significant to significant, being associated with an increase in HR of 3.05 beats/minute (95% CI, 1.49–4.61; z = 1.96; $p < .05$), but the effect of Stressor^{-1} became marginally significant (z = 1.42; $p < .10$). When using two-tailed t tests (Table 1), the effect of Worry^{-2} became marginally significant ($p < .10$).

Prolonged Effects on MSSD

Stressor^{0}, Worry^{0}, Stressor^{-1}, Worry^{-1}, Stressor^{-2}, Worry^{-2}, as well as Stressor $^{+1}$ were added to the intercept-only model at the same time adjusting the model for possible autocorrelation (Model 1, Table 2). Only Worry^{0} and Worry^{-1} showed significant effects on MSSD (z = 2.80, $p < .01$; z = 2.29, $p < .01$ respectively) and were associated with decreases in natural logarithm lnMSSD of −0.14 (95% CI, −0.19 to −0.09; corresponds with −5.34 milliseconds) and −0.16 (95% CI, −0.23 to −0.09; corresponds with −6.04 milliseconds), respectively. As there was no effect of stressors (Stressor^{+1} and Stressor^{-1} to Stressor^{-3}), testing mediation by worry became irrelevant. The model fits well compared with the intercept-only model ($\chi^2 = 39.62$; $df = 7$; $p < .001$). As with HR, stressful events and worry episodes that happened earlier had no significant effect and were further disregarded.

The emotional, biobehavioral, and person variables were added to the model (Model 2, Table 2). Increases in high activities, activity level, and a more active posture were related to decreases in lnMSSD of −0.13 (95% CI, −0.17 to −0.09; z = 3.36; $p < .001$; corresponds with −4.98 milliseconds), −0.08 (95% CI, −0.10 to −0.06; z = 3.95; $p < .001$; corresponds with −3.14 ms) and −0.04 (95% CI, −0.05 to −0.03; z = 3.67; $p < .001$; corresponds with −1.60 ms), respectively. A 1-unit increase of happy emotional states (on a scale of 5 units from "not at all" to "very much") was related to increases in lnMSSD of 0.03 (95% CI, 0.01–0.05; z = 1.72; $p < .04$; corresponds with 1.24 milliseconds). Smoking was marginally related to decreases in MSSD of −0.11 (95% CI, −0.19 to − 0.03; z = 1.42; $p < .10$; corresponds with −4.26 milliseconds). MSSD tended to decrease as the day progresses −0.03 (95% CI, −0.05 to −0.01; z = 1.72; $p < .05$; corresponds with −1.21 milliseconds). The inclusion of these variables did not noticeably change the previously found effects of Worry^{0} and Worry^{-1}, but the effect of Worry^{-2} became significant: −0.08 (95% CI, −0.13 to −0.03; z = 1.74; $p < .05$; corresponds with −3.14 milliseconds). With a best-fitting autocorrelation of 0.20, the overall fit of Model 2 was good in comparison with Model 1 ($\chi^2 = 784.47$; $df = 15$; $p < .001$). As in the analyses of HR, when using two-tailed t tests (Table 2), the effect of Worry^{-2} became marginally significant ($p < .10$).

DISCUSSION

The present study was designed to examine the prolonged cardiac effects of stressful events and worry episodes. We found marginal evidence that stressful events increased HR for 1 hour. No stressor effects of longer duration were found, and no stressor effects were found on MSSD. Additionally, no effect of anticipating a stressor in the succeeding hour was found. However, there were substantial and independent concurrent and prolonged effects of worry episodes on both HR and MSSD, with durations up to 2 hours. These findings were the most robust findings of this study, and they were largely independent of the effects of emotions, physical activity, posture, circadian rhythm, and biobehavioral factors, such as gender, age, body mass, and negative health behaviors.

The magnitude of prolonged effects of worry, i.e., about 2 to 3 beats/minute (HR) and 3–5 milliseconds (MSSD), were comparable to effects previously found for concurrent worry episodes in laboratory studies (29–31). To qualify our current findings, it is important to emphasize that these previous studies were laboratory studies that concerned reactivity, which is cardiac activity during stress experiences, whereas our current findings concern prolonged activity in real life. Thus, the present study shows that worry episodes affect the heart not only during their occurrence, but that they, along with stressful events, had prolonged effects up to several hours afterward.

The former effect, i.e., during worry, does not indicate a causal relationship; it can still be reasoned that high HR and

TABLE 2. Effects of Stressful Events and Worry Episodes on lnMSSD

	Model 1 Estimate ± SE (*p* value *t* test one-sided)$^{(\text{two-sided})}$	Model 2 Estimate ± SE (*p* value *t* test one-sided)$^{(\text{two-sided})}$
Fixed effects		
Intercept	3.36 ± 0.05 (<.001)$^{<.001}$	3.71 ± 0.09 (<.001)$^{<.001}$
Stressor^{0}	−0.01 ± 0.04 (.40)$^{.80}$	−0.04 ± 0.05 (.23)$^{.46}$
Stressor^{-1}	−0.05 ± 0.05 (.16)$^{.32}$	−0.06 ± 0.06 (.15)$^{.30}$
Stressor^{-2}	0.01 ± 0.03 (.42)$^{.84}$	0.03 ± 0.04 (.19)$^{.38}$
Stressor^{+1}	−0.04 ± 0.07 (.28)$^{.56}$	−0.07 ± 0.08 (.19)$^{.38}$
Worry^{0}	−0.14 ± 0.05 (.001)$^{.002}$	−0.15 ± 0.05 (.002)$^{.004}$
Worry^{-1}	−0.16 ± 0.07 (.02)$^{.04}$	−0.15 ± 0.08 (.03)$^{.06}$
Worry^{-2}	−0.04 ± 0.04 (.16)$^{.32}$	−0.08 ± 0.05 (.04)$^{.08}$
Angry		−0.02 ± 0.03 (.24)$^{.48}$
Sad		−0.03 ± 0.04 (.28)$^{.56}$
Tense		−0.002 ± 0.03 (.47)$^{.93}$
Happy		0.03 ± 0.02 (.04)$^{.08}$
% High activity		−0.13 ± 0.04 (<.001)$^{<.001}$
Activity level		−0.08 ± 0.02 (<.001)$^{<.001}$
Posture		−0.04 ± 0.01 (<.001)$^{<.001}$
Gender		−0.004 ± 0.13 (.49)$^{.98}$
Age		−0.002 ± 0.006 (.37)$^{.74}$
BMI		−0.02 ± 0.02 (.13)$^{.26}$
Smoking		−0.11 ± 0.08 (.08)$^{.16}$
Alcohol consumption		−0.05 ± 0.04 (.11)$^{.22}$
Coffee consumption		0.03 ± 0.03 (.13)$^{.26}$
Time of day[a]		−0.03 ± 0.02 (.04)$^{.08}$
Variance components		
Person level		
Intercept (σ^2_{u0})	0.19 ± 0.03	0.16 ± 0.03
Sequence level		
Intercept (σ^2_{u0})	0.03 ± 0.01	0.002 ± 0.01
Period level		
Intercept (σ^2_{e})	0.12 ± 0.004	0.03 ± 0.01
Deviance	1901.18	1116.71

SE = standard error; lnMSSD = natural logarithm root mean square of successive differences of interbeat intervals; BMI = body mass index.

[a] 1 = morning; 2 = afternoon; 3 = evening.

low MSSD cause worry and stress perceptions, instead of the other way around. The latter finding, however, that worry and stress are related to cardiac levels up to 2 hours after it, clearly supports the perseverative cognition model, as it is a prospective finding, indicating that worry episodes precede, and thus likely induce, high HR and low MSSD. Both chronic high HR and low MSSD are shown to be independent risk factors for CVD (32,33), and these findings therefore offer support for the notion that daily worry is a possible factor in generating potentially pathogenic CV activity. Furthermore, the prolonged cardiac effects of worry episodes are independent. This implies that they will accumulate when, for example, a person starts to worry again 1 hour or 2 hours after a worry episode: The possible effects of previous and later worry episodes add up (Fig. 2). In this way, a person experiencing chronic worries or stressful events or both might often suffer from accumulated cardiac effects that are well above the cardiac rest levels that reflect CVD risks (e.g., for HR, >83 beats/minute) (32,33).

It is intriguing that not stressful events but worry episodes seems to be most clearly associated with prolonged cardiac activation. There are several possible explanations. First, worry is not something completely different from stressful events. Worry is always about stressful events in the past, the present, or the future. Thus, by measuring the effects of worry episodes, we aggregated the effects of one or many more unsolved stressful events from the (regretted) past as well as expected in the (feared) future (5). Moreover, these worries typically involve events that are the most emotionally relevant for the person. In contrast, the effects of stressful events found in this study were confined to those from a limited time period (within the time frame from 4 hours before to 1 hour after a cardiac measurement period), and are not restricted to the emotionally most relevant. This may explain two findings in this study. First, the cardiac effects of worry were independent of the stressful events measured in this study. Second, the cardiac effects of worry are much greater than those of stressful events, because they pertain to many more events and much more intense— past and future—stressful events.

The finding that worry itself can have prolonged effects corroborates the finding that daily worry has prolonged effects into nocturnal sleep (6) but also poses a theoretical problem.

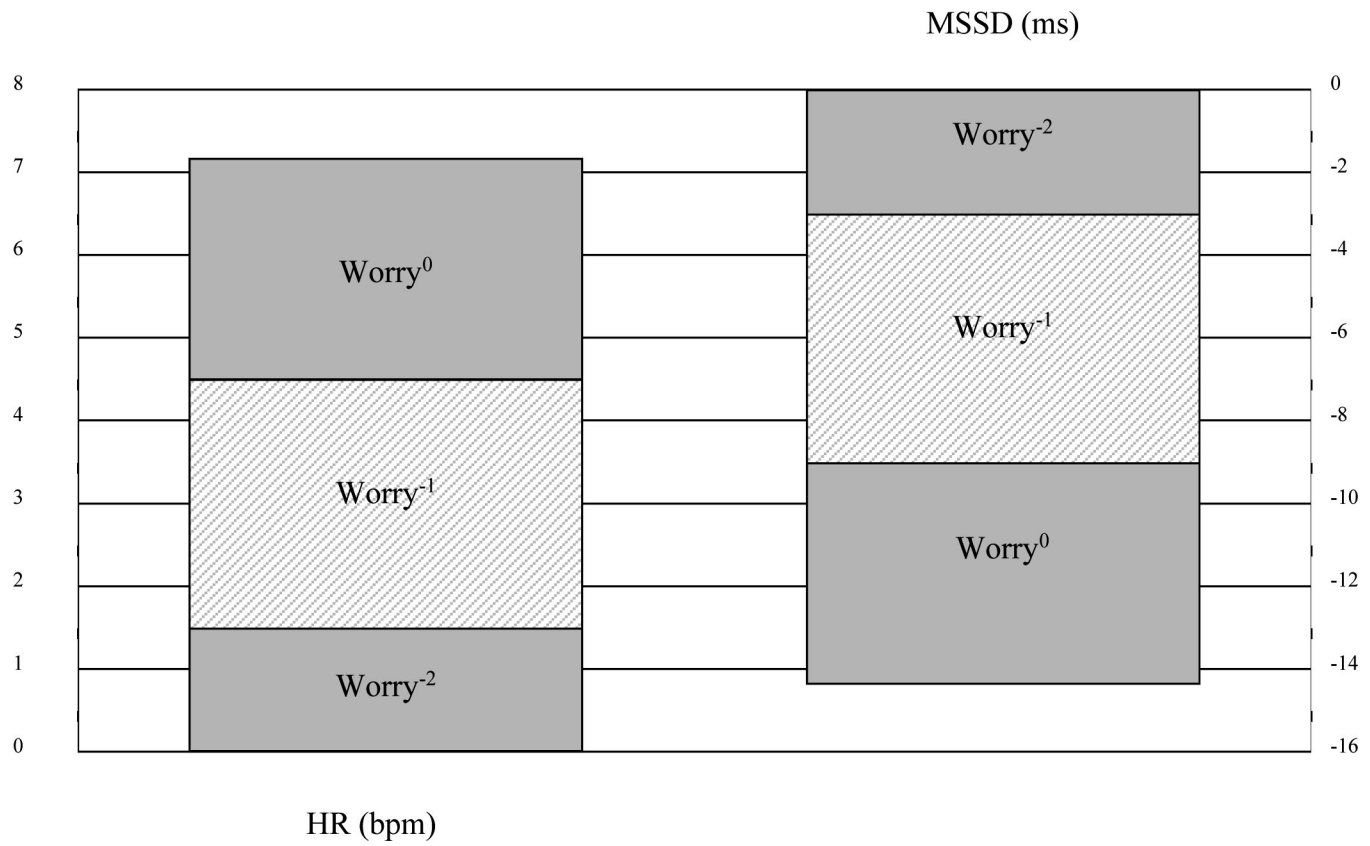

Figure 2. Cumulative effects of worry episodes at different durations on heart rate (*HR*) and root mean square of successive differences of interbeat intervals (*MSSD*). Only significant effects from the models are reported in the figure.

The cardiac worry effects of different durations were independent of each other. This means that none of these effects can be mediated by worry at a later time point. If worry has prolonged cardiac effects on its own, and of a duration up to 2 hours, what is mediating these effects? The finding that prolonged effects of worry are independent of the effects of emotions, biobehavioral and life-style variables, excludes these factors as candidates. There are some possible clues in previous publications indicating that a part of perseverative cognition may act in an unconscious fashion and is not reported by the individual. For example, prolonged low HRV during sleep (in which no conscious worry is possible) was not only found as a result of preceding worry (6) but also—during nonrapid eye movement as well as rapid eye movement sleep—as a result of anticipating a stressful oral speech that had to be performed the following morning after waking up (34). Thus, it is possible that unconscious perseverative processes might—during sleeping as well as waking—result in prolonged physiological effects after termination of conscious worry episodes and might occur perhaps even completely independent of conscious worry (35).

This study has several limitations. The limitations regarding the sample were previously discussed (11). A specific limitation of the current study pertains to the smaller time window of 15 minutes of cardiac measurements, required for the current analyses of prolonged activation. This, and the further reduction in data caused by using only subsequent and adjacent hourly periods, probably made the study less sensitive. This may, for example, explain that the cardiac effects during stressors found earlier (11) were smaller and not significant in this report. There were also too few cases now to test whether the specific cardiac effects of characteristics during stressors and worry episodes, i.e., work- and future-relatedness, were also true for the prolonged effects. Additionally, one might argue that effects of the present study are limited, because some become nonsignificant trends when tested with two-tailed *t* tests. For a discussion of this point, we also refer to our previous paper (11). Additionally, more men than women were observed, and although there were no significant differences in stressors and worries between the sexes, it urges for replication in a sample of women.

In conclusion, the findings of this study extend the results of previous studies by showing worry to have prolonged cardiac effects of itself, for up to 2 hours, and independent of effects of emotions, physical activity, posture, and biobehavioral factors, such as gender, age, body mass, and negative health behaviors. Our findings emphasize the importance of worry as a source of potentially toxic cardiac elevations in daily life. They also imply that still other—probably automatic or unconscious—cognitive perseverative processes may operate to mediate the prolonged cardiac effects of conscious worry. Given that elevated HR and decreased HRV are predictors of morbidity and all-cause mortality, these results indicate that worry—and yet unidentified other perseverative

cognitive processes—may play a considerable role in the effect of psychosocial stress on risk for cardiovascular disease (36). At least two studies so far suggested that worry predicts CVD, for example, in myocardial infarction (37), and the long-term CV effects of a major stressor ("9/11") (38).

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