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Gender differences in the impact of daily sadness on 24-h heart rate variability

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Abstract

Reduced heart rate variability (HRV) is proposed to mediate the relation between depressive symptoms and cardiovascular health problems. Yet, several studies have found that in women depression is associated with higher HRV levels, whereas in men depression is associated with lower HRV levels. So far, these studies have only examined gender differences in HRV levels using a single assessment. This study aimed to test the interactive effects of gender and sadness on ambulatory-assessed HRV levels. A sample of 60 (41 women) employees participated in an ambulatory study. HRV levels (mean of successive differences; *MSD*) were continuously measured for 24 h. During the daytime, hourly assessments of sadness and other mood states were taken, while depressive symptoms were assessed with the Center for Epidemiologic Studies Depression scale (CES-D). Gender differences were observed when examining the impact of average daily sadness on *MSD*. In women, but not in men, the total amount of sadness experienced during the day was associated with higher circadian *MSD* levels. These findings suggest that researchers need to take gender differences into account when examining the relation between sadness, HRV, and cardiovascular problems.

Descriptors: Heart rate variability, Sadness, Depression, Gender, Ecological momentary assessment

Depressive symptoms, such as severe sadness, have been shown to predict adverse cardiovascular health outcomes (Penninx et al., 2001). Several biological and behavioral pathways have been proposed to mediate this relation, among which is reduced cardiac autonomic control (Kemp et al., 2010). Cardiac autonomic control can be assessed by measuring the variability in the heart rate. Heart rate variability (HRV) arises as a result of the interplay between the parasympathetic branch of the autonomic nervous system, slowing down heart rate, and the sympathetic branch, accelerating heart rate. HRV provides a useful marker of cardiovascular health and is an independent predictor of cardiovascular morbidity and mortality (Thayer & Lane, 2007, 2009).

However, not all studies support the hypothesis that depressive symptoms predict adverse cardiovascular health outcomes. Gender differences have been found to exist, with depression being associated with increased morbidity or mortality rates only in men, but not in women (Ferketich, Schwartzbaum, Frid, & Moeschberger, 2000; Penninx et al., 1998, 1999). Importantly, gender differences have also been found in the proposed mediator of these effects. Thayer, Smith, Rossy, Sollers, and Friedman (1998) showed in college students that depressed men had lower HRV levels compared to nondepressed men. Yet, depressed women showed elevated HRV levels compared to nondepressed women. In another study, Chambers and Allen (2007) found that, in a clinically depressed sample, depressed women had higher HRV levels than depressed men. Most recently, in a random sample of people from the elderly community, Chen and colleagues found that in elderly men depressive symptoms were associated with reduced high frequency power, while this association was not apparent for women (Chen, Yang, Kuo, Su, & Chou, 2010). As higher levels of HRV helps to protect against cardiac events, gender differences in the association between depressive symptoms and HRV might explain the observed gender difference in cardiovascular problems. However, previous studies have only examined the effects of gender and depressive symptoms on HRV levels during short (< 10 min) resting periods. It remains unclear whether these gender differences will also be observed when inspecting HRV levels and depressive symptoms—in particular, experiencing sad mood—in daily life.

The aim of the present study was to examine gender differences in the effects of sadness on ambulatory-assessed HRV levels in a nonclinical sample that we have previously reported (Thayer et al.,

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PSYCHOPHYSIOLOGY

2010). Excessive sadness is one of the two main criterion symptoms (together with anhedonia) that people have to exhibit in order to qualify for a diagnosis of major depressive disorder (American Psychiatric Association, 2013). We focused on one specific aspect of depression, sadness, instead of focusing on the whole diverse cluster of depressive symptoms (leading to 1,030 depression profiles; Fried & Nesse, 2015). Besides being the most commonly reported criterion symptom of depression (Buckner, Joiner, Pettit, Lewinsohn, & Schmidt, 2008; Rakofsky et al., 2013), sadness is the symptom that, of all depressive symptoms, has been associated with the greatest impairment in psychosocial functioning (Fried & Nesse, 2015). In addition, normal variations in sadness and sadness as observed in depression can be considered to lie on a severity continuum, and there is no clear boundary between normal variations in mood and clinical depression (Slade & Andrews, 2005), which makes it reasonable to examine the hypothesized gender by sadness interaction in a nonclinical sample. Theoretically, as mood regulation has been associated with increases in HRV levels in women (Butler, Wilhelm, & Gross, 2006), we specifically expected gender differences in HRV to emerge when examining the mood aspects of depression.

Another advantage of focusing on a nonclinical sample is that the results cannot be accounted for by use of antidepressant medication, which is often used by clinically depressed patients and has been suggested to account for reduced HRV levels in depression (Licht, de Geus, van Dyck, & Penninx, 2010). Furthermore, we primarily focused on daily assessments of sadness, as symptom questionnaires that have been used in previous laboratory studies correspond only to some extent with momentary assessments of the same construct (Verkuil, Brosschot, & Thayer, 2007), which calls for a replication of the previously obtained findings in an ambulatory study.

We hypothesized that in men higher levels of sadness would be associated with decreased HRV levels, but that in women higher levels of sadness would be associated with higher HRV levels instead. To operationalize sadness, we used three indices. First, we assessed sadness—along with several other mood states—at random times in daily life. Second, given that previous studies focused on prolonged periods of sadness and depressive symptoms, the expected associations might be more prominent when examining accumulated levels of sadness, rather than short-lived moments of sadness, especially since we tested the associations in a nonclinical sample for which these moments might sometimes be rather mild. Therefore, we also examined the interaction between average daytime sadness and gender on average HRV levels. Third, to be able to examine the main and interactive effects of (trait) depressive symptoms on HRV levels, we also assessed depressive symptoms as traditionally done with a questionnaire, the Center for Epidemiologic Studies Depression scale, which is often used to screen for depression (Radloff, 1977). To operationalize HRV, we focused on the mean of successive differences (*MSD*), which is a common measure of HRV in ambulatory studies. *MSD* is derived as the mean of the absolute differences between successive interbeat intervals. It was also the metric used by Thayer et al. (1998) and Chambers and Allen (2007), who consistently found that *MSD* was differentially affected by depression in men and women. We examined both daytime levels, during which sadness was assessed as well as circadian variation in HRV. Reduced circadian variation in HRV has been associated with cardiovascular disease (Malik, Farrell, & Camm, 1990).

Furthermore, we expected that the predicted associations would be independent of possible confounding variables such as physical fitness, smoking and physical activity in daily life. Additionally, we explored whether the findings would be specific for sadness, or would be observed for other negative mood states as well. In sum, we expected to observe the previously found gender difference in the effects of sadness on HRV in a naturalistic setting.

Method

Participants

The present study formed part of a larger study on the effects of work environments on physiological health (Thayer et al., 2010). Participants were employees of a single government facility in the Rocky Mountain region of the United States. Of the 200 workers in the facility, 60 (30%) were able to participate. Participants were enrolled serially after receiving medical clearance. Only participants that were apparently healthy by medical examination and were not on medications that could potentially interfere with HRV levels were included in the final sample. The final sample consisted of 41 women and 19 men. None of them were taking antidepressant or anxiolytic medication. Forty-four participants (31 women, 13 men; $\chi(2)^2 = 1.40$, $p = .50$) agreed to participate in a second measurement day. This study was approved by the National Institute on Aging (MedStar) Institutional Review Board.

Procedure

Volunteers were solicited and gave written informed consent. After receiving consent, the medical exam was scheduled. Those volunteers who were medically cleared for participation were then scheduled for a 24-h ambulatory heart rate recording. To obtain this recording, an experimenter fitted the ambulatory electrocardiogram (ECG) device in the morning before the employees started their regular work activities and instructed them on the use of this device as well as on the use of a handheld computer that contained the hourly diary questions. Participants carried both electronic devices for 24 h during a week working day. For those 47 participants who were measured on a second working day, this same procedure was followed.

Instruments

Diary format. A Palm m100 handheld device (Palm Inc., Santa Clara, CA) was used for the hourly diary. Customized software (Pendragon Forms, version 3.1; Pendragon Software Corporation, Libertyville, IL) was used to implement questions and to transfer responses from the handheld to MS-Access data format. For the hourly diary, an hourly tone ($\pm$ 15 min) was set from 7.00 am to 7.00 pm upon which participants were instructed to fill in the computerized questions. When the participants answered the first question of each entry of the log, the present time was stored to enable comparison between their responses and the cardiac measurements.

Cardiac activity. Ambulatory cardiac measures were acquired continuously by the VU-AMS device (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 (Pieper, Brosschot, van der Leeden, & Thayer, 2007). The mean of the absolute successive differences between normal-to-normal beats (*MSD*) was used to operationalize HRV. The *MSD* is less affected by breathing and is therefore a suitable outcome measure in ambulatory studies (Penttila et al., 2001).

Table 1. *Descriptive Statistics*

		Women ($N = 41$)			Men ($N = 19$)		
		M (/%)	*SD*	Min-Max	*M* (/%)	*SD*	Min-Max
Age		43.73	11.17		46.60	10.06	
Ethnicity	Caucasian	75.6%			84.2%		
	African American	7.3%			5.3%		
	Hispanic/Latino	14.6%			5.3%		
	Asian/Pacific Islander	2.4%					
	Missing				5.3		
Marital status	Single	22%			26.3%		
	Married	53.7%			63.2%		
	Divorced	24.4%			10.5%		
Psychophysiological variables							
MSD		21.05	13.15	5.27–64.36	23.18	12.20	9.51–46.74
Motility		0.05*	0.02	0.01–0.16	0.10	0.08	0.02–0.29
Caffeine intake[a]		2.21	1.96	0–9	2.81	2.02	0–7
Smoking[b]		0.34	1.22	0–7	0.44	1.60	0–7
Alcohol intake[a]		0.12	0.29	0–1	0.23	0.42	0–1
Physical fitness		3.61	1.90	0.50–7.00	3.89	1.74	1–7
CES-D		10.10*	6.37	1.48–27.00	5.11	4.86	0.00–15.00
Momentary mood states							
Sadness		1.21	0.56	1–5	1.20	0.50	1–4
Anger		1.20	0.59	1–4	1.22	0.50	1–4
Agitation		1.90	1.07	1–5	1.86	0.95	1–5
Tiredness		2.23	1.01	1–5	2.10	0.91	1–4
Stress		2.07	1.07	1–5	1.92	0.97	1–4
Pain		1.28	0.63	1–5	1.12	0.37	1–3
Serenity		2.84	1.05	1–5	3.04	0.94	1–5
Interested		3.23	0.98	1–5	3.38	0.89	1–5
Relaxed		2.97	0.97	1–5	3.19	1.02	1–5
Excitement		2.51	1.05	1–5	2.69	0.93	1–5
Happiness		3.20	0.94	1–5	3.22	0.88	1–5
Pleasant		3.20	0.84	1–5	3.32	0.83	1–5
Activated		2.95	0.99	1–5	3.05	0.96	1–5

Note. *MSD* = mean of successive differences; CES-D = Center for Epidemiologic Studies Depression scale.
[a]number of drinks.
[b]number of cigarettes.
*$p < .05$.

The VU-AMS includes an accelerometer sensitive to changes in vertical acceleration. This signal was used to measure motility in daily life.

State sadness. During each measurement period, until 7 pm, participants reported on the handheld computer to what extent they currently experienced the following moods: sadness, anger, stress, pain, serenity, interest, relaxed, excitement, happiness, agitation, tired, pleasant, activated (e.g., "How much sadness do you feel right now?" with answer options ranging from *none* (1) to *a lot* (5). State sadness was operationalized in two ways: as momentary assessed sadness, and the average amount of sadness aggregated per participants, per measurement day.

Depressive symptoms. Symptoms of depression were assessed using the Center for Epidemiologic Studies Depression scale (CES-D; Radloff, 1977). The CES-D is a widely used, valid, and reliable questionnaire to assess depressive symptoms. It consists of 20 depressive symptoms, and participants are asked to rate the extent to which these symptoms were present during the past week on a 4-point scale. A score of 16 or higher on this scale suggests the possible presence of a current depressive episode.

Biobehavioral variables. At each hourly assessment, the participants also reported consumed units of tobacco, coffee, and alcohol (0, 1–2, 2–4, more than 4) in the preceding hour (i.e., since the last assessment). In addition, participants rated their habitual physical activity using the University of Houston Non-Exercise Questionnaire, a seven-point behaviorally anchored rating scale that has been shown to provide valid measures of habitual physical activity (Jackson et al., 1990).

Statistical Analysis

Multilevel regression analyses were conducted to estimate the effects of gender, the mood, and biobehavioral variables on *MSD* levels. The distribution of *MSD* was significantly skewed. This skewness was reduced by logarithmically transforming this variable (lnMSD). The data had a two-level hierarchical structure, in which the 24 hourly assessments of HRV per assessment day were nested within participants. All analyses were performed using the linear mixed model (MIXED) procedure in SPSS 17.0, with maximum likelihood estimation. All numerical variables were centered on their grand mean.

To examine the effects of gender and sadness on HRV levels, we tested whether the three indices of sadness (momentary sadness, average daytime sadness, and CES-D scores) interacted with gender to predict lnMSD levels during the daytime. Biobehavioral variables that were significantly associated with lnMSD levels (time of the day, smoking, motility, and physical fitness) were entered as covariates into the models. In addition to the daytime levels of lnMSD, we also examined whether sadness was differentially

associated with the circadian variations in lnMSD for men and women. We therefore examined the interaction between gender, sadness, and two time variables—a linear time trend (in hours) and a quadratic time trend (time^2). Two-tailed tests with alpha set at .05 were conducted.

Results

Descriptive Statistics

Descriptive statistics for the first measurement day are presented in Table 1. Mean age was 44, and the sample covered a broad age range: 24–62. According to the CES-D, women had more depressive symptoms, ($M = 10.10$, $SD = 6.37$) than men ($M = 5.11$, $SD = 4.86$; $t(58) = 3.03$, $p = .004$), with both scores falling in the normal (nondepressed; < 16) range. Mean motility also differed between men and women, with men showing overall higher levels of movement.

With respect to the momentary assessments, the participants were prompted to answer the hourly forms 1,284 times. At 346 occasions (27%), no response was provided. At 15.3% of all momentary assessments, the participants reported to have experienced at least some sadness. Twenty-three participants (38.8%) did not report any sadness, whereas 37 participants reported at least some sadness during the study. Data from two 24-h recordings from two participants had to be disregarded because of problems with the ECG monitoring. The average amount of hourly assessments per participant per assessment day was 21 ($SD = 2.4$).

Furthermore, the CES-D was moderately associated with momentary assessed sadness, $r(60) = .43$, $p = .001$.

Sadness, Depressive Symptoms, and Heart Rate Variability

The results of the multilevel analyses are presented in Table 2. The models shows that, while controlling for time of the day, smoking, and physical fitness, lnMSD levels were significantly associated with the interaction between gender and average daytime sadness ($B = -.53$, $p = .04$—Models 2 and 4). The interactions between gender and momentary assessed sadness and CES-D were not significant (Models 1, 3, and 4). In addition, to check whether depressive symptoms would aggravate the effect of daytime sadness on lnMSD levels, we examined whether CES-D levels moderated the association between gender, average daytime sadness, and lnMSD levels, but this four-way interaction was not significant.

To examine the interaction between average daytime sadness, gender, and lnMSD, separate multilevel analyses were run for men and women. In women, the average amount of sadness experienced in daily life was associated with higher levels of lnMSD ($B = .61$, $p = .002$). In men, this association was negative and nonsignificant ($B = -.27$, $p = .111$). See Figure 1 for a graphic representation of the associations between gender, average daytime sadness, and lnMSD.

Sadness, Depressive Symptoms, and Circadian Heart Rate Variability

As average daytime sadness was differentially associated with lnMSD for men and women, we subsequently examined differences in circadian variation in lnMSD. Adding the linear and quadratic time trends into Model 2 yielded a significant Gender × Average Daytime Sadness × Time^2 interaction ($B = -.002$, $p = .034$). Figure 2 shows the circadian variation in *MSD*, split by gender and average daytime sadness levels (median split for illustrative

Table 2. *Multilevel Models Predicting Log-Transformed MSD Levels*

	Model 1				Model 2				Model 3				Model 4			
	Momentary sadness				Average daytime sadness				CES-D				Final model			
	B	*SE*	*t*	*p*	*B*	*SE*	*t*	*p*	*B*	*SE*	*t*	*p*	*B*	*SE*	*t*	*p*
Intercept	2.46	0.19	12.99	< .001	2.58	0.18	14.47	< .001	2.70	0.19	14.32	< .001	2.48	0.19	13.01	< .001
Time of the day	−0.01	0.00	−4.57	< .001	−0.01	0.00	−13.23	< .001	−0.01	0.00	−12.55	< .001	−0.01	0.00	−4.18	< .001
Smoking	−0.12	0.04	−3.21	< .001	−0.11	0.03	−3.18	< .001	−0.11	0.04	−3.17	< .001	−0.13	0.04	−3.73	< .001
Physical fitness	0.00	0.03	−0.02	0.99	−0.01	0.02	−0.34	.74	−0.03	0.03	−0.99	.33	0.00	0.03	0.10	.92
Motility	−1.05	0.10	−10.19	< .001	−1.66	0.08	−19.81	< .001	−1.60	0.09	−18.71	< .001	−0.99	0.11	−9.16	< .001
Ethnicity—AA vs. H/L	0.62	0.32	1.94	.06	0.61	0.30	2.04	.04	0.60	0.31	1.94	006	0.49	0.31	1.58	.12
Ethnicity—AA vs. C/A	0.44	0.16	2.67	.01	0.45	0.15	2.93	< .001	0.40	0.16	2.48	.02	0.44	0.16	2.75	.01
Age	−0.02	0.00	−5.03	< .001	−0.03	0.00	−5.57	< .001	−0.03	0.00	−5.90	< .001	−0.02	0.00	−4.62	< .001
Gender	0.22	0.10	2.15	.03	0.28	0.10	2.91	< .001	0.32	0.11	2.90	.01	0.19	0.11	1.72	.09
Momentary sadness	0.04	0.02	1.60	.11									0.03	0.03	1.25	.21
Gender × Momentary Sadness	−0.01	0.05	−0.26	.80									0.00	0.05	0.08	.94
Average daytime sadness					0.24	0.14	1.72	.09					0.56	0.18	3.14	< .001
Gender × Average Daytime Sadness					−0.53	0.25	−2.10	.04					−0.85	0.29	−2.90	.01
CES-D									−0.01	0.01	−0.92	.36	−0.03	0.01	−2.64	.01
Gender × CES-D									0.02	0.02	1.06	.29	0.02	0.02	1.27	0.21

Note. AA = African American; H/L = Hispanic/Latino; C/A = Caucasian/Asian/Missing; CES-D = Center for Epidemiologic Studies Depression scale.

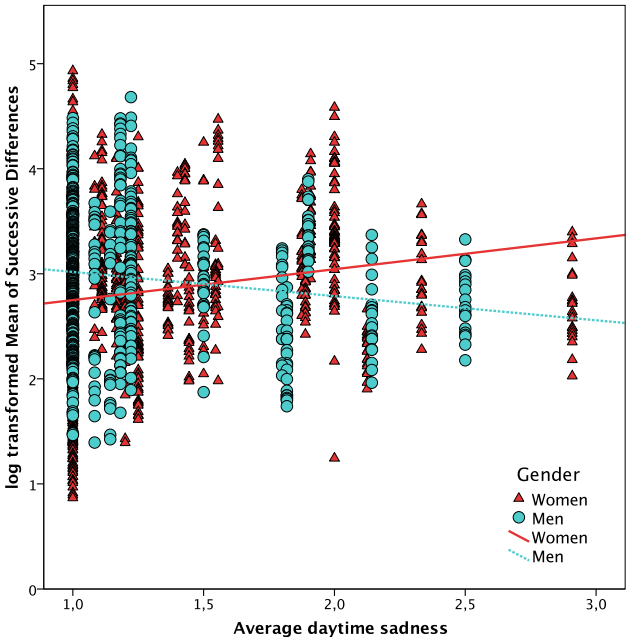

Figure 1. Scatter plot of the associations between log-transformed *MSD* levels and average sadness experienced during the day, split by gender.

purposes only). Overall, women seemed to show less circadian variation in lnMSD levels. In contrast to men, women with higher levels of sadness showed overall higher levels of lnMSD.

Exploratory Analyses

To explore whether the gender difference in the associations between sadness and *MSD* were specific for HRV levels or were also observed when examining heart rate (HR), we also tested the models with HR as dependent variable. The results showed that HR was significantly associated with CES-D scores ($B = .54$ $p = .018$). HR was not significantly associated with the Gender × Sadness interactions ($ps > .09$). In addition, we examined the associations between gender, sadness, and another index of HRV, high frequency power (HF power), which was log transformed to follow a normal distribution (lnHF). LnHF power was significantly associated with the Gender × Average Daytime Sadness interaction. Similar to lnMSD, in women, the average amount of sadness experienced in daily life was associated with higher levels of lnHF power ($B = 1.27$, $p < .001$). In men, this association was negative and nonsignificant ($B = -.35$, $p = .229$).

Next, we explored whether the gender difference in the sadness–*MSD* association would also be apparent for the other mood states that we assessed (anger, stress, pain, agitation, tired, serenity, interest, relaxed, excitement, happiness, pleasant, activated). This led to two indices of these items (momentary mood and average daily mood). Thereafter, we inspected the Gender × Mood interactions, while controlling for the covariates. For momentary feelings of relaxation, a significant interaction with gender was observed ($B = .07$, $p = .001$). In men, momentary relaxation was associated with increased lnMSD ($B = .07$, $p < .001$), while this association was not significant in women ($B = .002$, $p = .83$).

Discussion

The aim of the present study was to investigate whether sadness affected HRV levels in daily life differently for men and women. HRV levels, indexed by lnMSD, were indeed differentially associated with sadness for men and women, but only when sadness was measured as the average sadness across 1 day. There was no direct association between momentary assessed sadness and HRV. We found that only in women the average amount of sadness experienced during the day was associated with higher 24-h HRV levels. Furthermore, these results were not apparent when focusing on HR, possibly suggesting that the observed gender difference is specific for parasympathetic nervous system activity. In addition, an explorative analysis showed that gender moderated the association between momentary relaxation and HRV.

These findings are in line with studies using single assessments of HRV in more clinical samples, showing that women with depressive symptoms have increased HRV during rest, in contrast to men with depressive symptoms (Chambers & Allen, 2007; Chen et al., 2010; Thayer et al., 1998). Furthermore, in this study we not

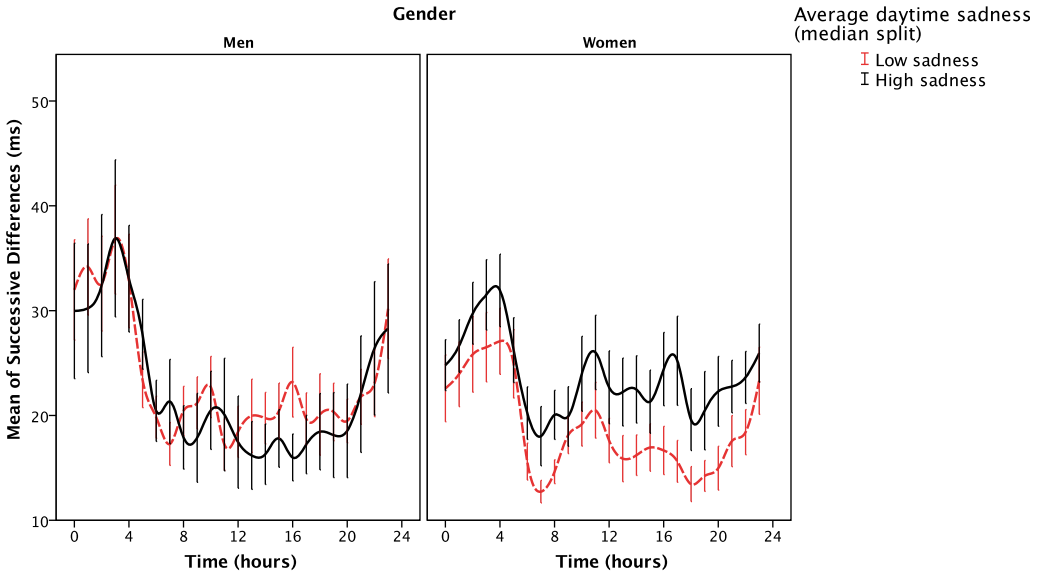

Figure 2. *MSD* levels during the study, split by gender and average sadness experienced during the day (median split).

only observed gender differences in the relation between sadness and HRV, but in the exploratory analyses we also observed gender differences in the relation between ambulatory-assessed feelings of relaxation and HRV, showing that relaxation was associated with increased HRV, but only for men. These different associations for men and women between cardiac activity and mood states warrants further research, as these differences have only been scarcely examined, and mainly in relation to feelings of sadness, anxiety, and stress.

The main findings are consistent with previous studies in which we showed gender differences in HRV responses to worry, anxiety, and rumination—which are all strongly related to feelings of sadness (Verkuil, Brosschot, Borkovec, & Thayer, 2009; Verkuil, Brosschot, de Beurs, & Thayer, 2009; Verkuil, Brosschot, & Thayer, 2014). Furthermore, the results are in line with current views on gender differences in stress responses. Men are believed to show the typical fight-or-flight response to stressful events, whereas women might be more likely to show a "tend-and-befriend" response, of which the latter has been suggested to be accompanied by an increase in vagal tone (Taylor et al., 2000). Women might be more likely to regulate their emotions and to talk about their feelings. It is possible that, in women, feelings of sadness are followed by attempts to regulate these feelings, for example, by suppressing or reappraising them. Both of these strategies have been found to increase levels of HRV in women (Butler et al., 2006). Future research is warranted to examine more precisely what aspects of emotional experiences (e.g., emotion intensity or emotion regulation) can account for these results. Furthermore, it remains unclear why gender differences were only found for sadness when using average daily sadness, and not in the moment-to-moment associations with HRV (which was the case for stress). It might be that the physiological effects of sadness only develop after several hours of sadness. In line with this idea, we found that the average sadness levels were also associated with increased HRV levels during the nighttime in women. Given that daily sadness has also been shown to predict cortisol levels the next day (Adam, Hawkley, Kudielka, & Cacioppo, 2006), it is not unlikely that the effects of sadness remain sustained for several hours, even during sleep. Future studies that specifically focus on the temporal dynamics between sadness and HRV are clearly warranted.

The observed gender differences in the relation between sadness and HRV and relaxation and HRV challenge the common implicit assumption that emotions and mood states are associated with similar physiological activity in men and women. In line with this, gender differences have also been reported in the neural concomitants of emotion and emotion regulation (see review by Whittle, Yücel, Yap, & Allen, 2011). Specifically, gender differences are apparent in brain function during sadness. In a study using a sadness induction, self-reported sadness was associated with increased right amygdala activity in men, but not in women (Schneider, Habel, Kessler, Salloum, & Posse, 2000). Interestingly, we have previously reported that amygdala activity was negatively correlated with HRV in men but positively correlated with HRV in women (Nugent, Bain, Thayer, Sollers, & Drevets, 2011). Higher HRV is positively associated with self- and emotion regulation and with prefrontal cortical activity, which are thought to modulate the subcortical activity, in areas such as the amygdala, involved in sustained emotional reactivity (Thayer & Lane, 2007, 2009). Thus, gender differences in a network of neural structures that include the medial prefrontal cortex and the amygdala may represent the neural concomitants of the reported gender differences in the relationship between sadness and HRV found in the present study. Clearly, additional studies that combine neuroimaging and HRV during emotional tasks that include sadness are needed to help explicate these associations.

Several limitations to this study should be acknowledged. First, the results from this study are correlational, and no definite conclusion can be drawn regarding the direction of the observed associations. Second, several remarks can be made about the current sample. Most notably, this convenience sample was small ($N = 60$), nonclinical, and 68% were women. Overall, the levels of sadness and depressive symptoms were low, and it remains to be examined how higher levels of daily sadness would affect HRV levels in men and women. Yet, the main aim of the present study was to provide a "proof of principle," namely, that gender differences in the HRV response to sadness would be apparent in daily life. A next step might be to replicate the current findings in a clinical sample with a more balanced gender distribution, which would have to take into account confounding effects such as use of antidepressants. Additionally, here we only focused on sadness, and for its assessment only one item was used, which is a limitation. Future studies could benefit from using a questionnaire that measures more aspects of depressive symptoms in daily life. Finally, this study only measured mood states and did not focus on other symptoms of depression that could be associated with cardiovascular functioning. Neither did we assess anxiety levels. Cognitive biases, rumination, anxiety, and worrying are typically observed in depression and in nonclinical samples, and are also associated with altered cardiovascular functioning (Verkuil, Brosschot, Gebhardt, & Thayer, 2010). Future studies could benefit from measuring these aspects of depression as they could provide more direct insight into the observed gender differences.

In conclusion, higher levels of HRV are thought to be cardioprotective and were previously observed in depressed women, albeit only in laboratory settings. This study is the first to use an ambulatory design to study gender differences in cardiac autonomic control in response to sadness, the most pertinent feature of depression. The results added to previous laboratory studies that higher daily levels of sadness are associated with heightened HRV levels in women, but not in men. Further examination of the underlying mechanisms that contribute to higher HRV levels in women seems worthwhile.

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