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## CLINICAL PSYCHOLOGY & NEUROPSYCHOLOGY | RESEARCH ARTICLE

# Understanding depressive symptoms through psychological traits and physiological stress reactivity

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**Abstract:** Understanding the relationship between psychological and physiological factors in depression and depressive symptoms may help us define depression subcategories based on their parasympathetic nervous system activity and reactivity, and perhaps in the future, develop more effective symptom-based treatments. In this study we aimed to shed light on the relationship between selected psychological traits (harm avoidance and self-rumination) and physiological stress (high-frequency heart rate variability, HF-HRV). We recruited 58 females to participate in a laboratory study where they completed a public speech preparation task designed to induce stress. We found that participants with higher scores in self-reported harm avoidance and self-rumination had lower HF-HRV during the stress task, indicating a parasympathetic withdrawal (i.e. more stress). The associations between self-reported depressive symptoms and HF-HRV were not statistically significant. Thus, we linked psychological depression risk factors to specific indices of higher physiological stress.



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### ABOUT THE AUTHORS

Ilmari Määttänen is a postdoctoral researcher at the University of Helsinki, Finland. His research interests include, but are not restricted to, personality and stress. He is currently a principal investigator of an Academy of Finland project involving the Finnish-style persistence or perseverance “sisu”.

### PUBLIC INTEREST STATEMENT

We studied the connection between depressive symptoms and the psychological traits of harm avoidance and rumination, which reflect (among other things) negative emotionality and negative repetitive thinking, respectively. We were specifically interested in high-frequency heart rate variability, which is believed to reflect the withdrawal of a relaxing (parasympathetic) nervous system activity. It is calculated from heart rate values. We placed our participants in a stressful situation (preparation of public speech) in a laboratory and measured their heart rate. We found out that depressive symptoms were associated with both harm avoidance and rumination; and that harm avoidance and rumination were both negatively associated with high-frequency heart rate variability. Surprisingly, depressive symptoms were not statistically significantly associated with high-frequency heart rate variability. This suggests that the pathway from negative emotions and physiological stress to depressive symptoms is not straightforward. More research is needed to better understand this relationship.

**Subjects: Personality; Mood Disorders in Adults - Depression, Mania, Bi-polar; Stress in Adults**

**Keywords: depressive symptoms; heart rate variability; stress; personality; harm avoidance; rumination**

## 1. Introduction

Depression is a state of low mood and aversion to activity, both of which negatively affect an individual's thinking, behaviour and well-being. Depression is characterised by both psychological and physiological features, and the risk factors predisposing people to clinical (i.e. more severe) depression can likewise be either psychological, physiological or both (Monroe & Simons, 1991). Untangling the effects of psychological and physiological risk factors for depression is important for the development of effective medical and psychosocial treatments, and for accurate mental health diagnostics. In this paper we sought to shed light on the relationship between physiological stress, as measured by heart rate variability (hereafter HRV; Porges, 1992), and two specific psychological depression risk factors: harm avoidance and self-rumination, both of which are positively associated with depression (Cruz, Przybeck, Spitznagel, & Cloninger, 2003; Elovainio et al., 2004; Hansenne et al., 1999; Kampman & Poutanen, 2011; Nolen-Hoeksema, 2000, 1991; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008; Nyman et al., 2011; Robinson & Alloy, 2003; Ward, Lyubomirsky, Sousa, & Nolen-Hoeksema, 2003). We also explore the possibility that the route from psychological traits to depression might in part be via stress physiology, that is, high-frequency HRV.

On a physiological level, stress can be viewed as the body's way of reacting to changing environmental conditions, such as external physical threats, which typically involve significant activation and suppression of the sympathetic and parasympathetic parts of the autonomic nervous system, respectively. *Psychological* and *physiological* stress are descriptions of the same phenomenon on different levels of analysis; both involve the body (and mind) reacting or readjusting to meet new environmental (either external or internal) challenges. In an evolutionary context, stress is adaptive and non-pathological; but in modern societies stress reactions can become problematic if they are too strong and/or long lasting. Strong stress reactions were adaptive in the past because they made it easier for our ancestors to avoid and react to external threats (and thus survive), but currently serve little adaptive purpose in the context of work-related deadlines, fear of giving public speeches, or general anxiety about job market uncertainty. A popular conceptualisation of stress emphasises that the environment or situation *per se* does not determine whether or not (negative or maladaptive) stress is experienced, but rather the individual's interpretation of his or her abilities to cope in that situation (Lazarus & Folkman, 1984, pp. 19–20).

Stress can be evaluated by various means, such as measuring electrocardiography and then estimating the heart rate fluctuations caused by breathing, i.e. respiratory sinus arrhythmia (often abbreviated RSA); in the case of this article, the almost interchangeable term HRV is used. In short, HRV is a product of the constant feedback and regulation process between the central and peripheral nervous systems (Porges, 1992; Porges, Doussard-Roosevelt, & Maiti, 1994), and it reflects the relative activity of the autonomic nervous system, especially the parasympathetic branch (vagus nerve, to be precise). Generally, higher HRV may reflect higher physiological potential to adapt to a changing environment (Porges, 1992) and reflects relaxation.

Stress is associated with depression, rumination and harm-avoidance both conceptually and empirically. Depression has been linked to the altered functioning of the autonomic nervous system, particularly to increased sympathetic activity and/or decreased parasympathetic activity (Kemp et al., 2010; Licht et al., 2008; Sgoifo, Carnevali, Alfonso, & Amore, 2015). Cardiac vagal nerve (i.e. parasympathetic) activation has been suggested as a physiological marker for sufficient psychological self-regulation (Rottenberg, Clift, Bolden, & Salomon, 2007). HRV may also be a useful marker for understanding the interaction between psychological depression risk factors and autonomic nervous system functioning (Sgoifo et al., 2015).

The links between stress as measured by HRV, depression, harm avoidance and rumination have been evaluated previously (Bär et al., 2004). Some studies have found a difference in HRV in depressive patients compared with non-depressive control groups (Koschke et al., 2009; van der Kooy et al., 2006; Yaptangco, Crowell, Baucom, Bride, & Hansen, 2015); while others have not (Birkhofer, Schmidt, & Försti, 2006).

Rottenberg et al. (2007) evaluated the link between depression and HRV by differentiating *baseline* HRV from *HRV reactivity*, thereby shedding light on the apparent inconclusive findings on depression and HRV. Baseline HRV is a resting state heart rate measure, reflecting the level of parasympathetic activity, while HRV reactivity measures the change from baseline in reaction to some environmental challenge. Depressive patients often show lower HRV in a resting state, but also less change in HRV. Both may be important indicators for physiological flexibility and self-regulation capacity, although some research indicates a more consistent relationship between depression and HRV reactivity (Rottenberg et al., 2007). Rottenberg (2007) suggests that depression is linked especially to impaired homeostatic regulation capacity, which manifests as weak reactivity to stressful tasks. Moreover, Bylsman et al. (2014) found that depressive patients did not show parasympathetic withdrawal as a reaction to a stressful task as much as the non-depressive or recovered depressive groups; however, the groups did not differ in baseline HRV.

According to *response style theory* (Nolen-Hoeksema, 1991), individual differences in responses to one's depressive symptoms are the key to understanding depression. The way individuals respond to their depressive symptoms influences how long their symptoms last. The core concept in response style theory is *rumination*, which refers to rigid, inflexible and repetitive tendency to dwell on negative thoughts (Nolen-Hoeksema et al., 2008). Response style theory postulates that rumination increases the duration of depression by i) intensifying negative mood via negative “thought-loops”, and ii) disrupting effective, active, and solution-focused problem-solving by making one's thinking pessimistic and fatalistic (Nolen-Hoeksema, 1991; see also Nolen-Hoeksema et al., 2008). Several studies have shown a positive link between rumination and depression severity, duration, and likelihood of onset (Nolen-Hoeksema, 2000, 1991; Nolen-Hoeksema et al., 2008; Robinson & Alloy, 2003, 2003; Ward et al., 2003). Rumination is also more common among females than males, which might partly explain why depression is more common among females (Nolen-Hoeksema, Larson, & Grayson, 1999).

Both rumination and depression are strongly linked to individual differences in harm avoidance, as conceptualised in Cloninger's psychobiological model of temperament (Cloninger, 1987; Cloninger, Svrakic, & Przybeck, 1993). Harm avoidance manifests itself as a pessimistic worry about the future, fear of uncertainty, shyness towards strangers and as a rapid feeling of fatigue (Cloninger et al., 1993). High scores in harm avoidance predispose individuals to both clinical and sub-clinical depression (Crucza et al., 2003; Elovainio et al., 2004; Farmer et al., 2003; Hansenne et al., 1999). In a recent meta-analysis, harm avoidance was found to have a significant role in predicting depression symptoms in 11 out of 12 studies analysed (Kampman & Poutanen, 2011).

Research on temperament traits and HRV is scarce. While some studies have evaluated the associations between the Big Five personality traits and HRV, few studies have focused on temperament traits specifically. Moreover, existing research has typically focused on baseline HRV, but not HRV reactivity. For example, there is a negative association between baseline HRV and the neuroticism subscale *anxiety* (Bleil, Gianaros, Jennings, Flory, & Manuck, 2008; Miu, Heilman, & Miclea, 2009). Puttonen and colleagues (2008) found that high harm avoidance scores were associated with low HRV in a resting state, controlling for age and gender (for similar results, see also Huang et al., 2013; however, see Bleil et al., 2008 for conflicting evidence). However, we have found no previous studies establishing a link between harm avoidance and HRV reactivity.

The association between rumination and HRV has not been extensively studied, and most research has focused on *anxious worry* instead of rumination—albeit the two are similar phenomena. For example, worry is linked to higher baseline heart rate and lower baseline HRV (Brosschot, Van Dijk, & Thayer, 2007). Pieper, Brosschot, van der Leeden, and Thayer, 2007 also found that work-related worry was linked to

higher heart rate and lower high-frequency HRV in teachers, independently of the self-reported objective amount of stress. Given that worry and rumination share about 40 % of variance (Hong, 2007), it is reasonable to presume that rumination is also negatively linked to high-frequency HRV, which reflects parasympathetic activation. Moreover, Ottaviani et al.'s (2016) meta-analysis found that both rumination and worry were linked to a lower baseline high-frequency HRV-component and to higher heart rate, blood pressure and cortisol levels. Rumination itself has also been associated with reduced HRV, and this relationship has been suggested to be a mediator between rumination and depressive symptoms (Carnevali et al. 2018). Rumination-like thinking has been hypothesised to prolong the stress reaction after the concrete stressor (the original cause of stress) has already passed, thereby negatively influencing health (Brosschot, Gerin & Thayer, 2006).

Based on the above theory and results, we formulated the following hypotheses:

- (1) Depressive symptoms, rumination, and harm avoidance are all positively intercorrelated.
- (2) Depressive symptoms, rumination and harm avoidance are all associated with
  - (a) lower baseline high-frequency HRV.
  - (b) lower high-frequency HRV during *preparation for* and *recovery from* a stressful task.
- (3) Depressive symptoms are associated with lower high-frequency HRV reactivity during a stressful task.

## 2. Methods

### 2.1. Participants

Fifty-eight female participants (N = 58; mean age = 25; age range: 20–39) were recruited from a pool of 588 people (n = 588; 512 females, 72 males, 4 unreported; mean age = 26; age range: 18–60), all of whom had previously completed a web-based survey on depression, stress, and rumination. This survey was advertised on student mailing lists across the University of Helsinki, and in it participants completed the same psychometric measures used in the current study (detailed below). We selectively invited a group of participants whose rumination-scores were visually inspected as the most normally distributed among several possible randomly selected groups. This was done to satisfy the requirements of our statistical models. Only females were recruited because our focus was not on gender differences, and we wanted to simplify our statistical models and conclusions. Individuals with clinically diagnosed depression were excluded.

### 2.2. Materials

Depressive symptoms were measured with Beck's Depression Inventory II (BDI-II; Beck, Steer, & Brown, 1996). BDI-II consists of 21 statements (for example, "I feel that I am complete failure as a person", and "I feel guilty all the time") scored on a scale from 0 to 3 on how well the statements describe the respondent's current state; higher scores indicate more severe depressive symptoms. We used the BDI-II measurement scores as a continuous variable (summed across all statements); participants were not classified into depression severity categories. The range of the scores was 0–63, and the scale had a good inter-item reliability (Cronbach's  $\alpha = 0.89$ ).

Temperament traits were measured by Cloninger's Temperament and Character Inventory (TCI; Cloninger, 1994). TCI has 240 questions, which are answered on a scale of one to five (1 = Does not fit, 5 = Fits perfectly), and 28 subscales, which in turn yield four temperament traits and three character traits. We focused on the 20 questions measuring trait harm avoidance and its four subscales (anticipatory worry, fear of uncertainty, shyness with strangers, and fatigability). The scale had an excellent inter-item reliability (Cronbach's  $\alpha = 0.90$ ).

Rumination tendency was measured with the self-rumination scale (Elliot & Coker, 2008). We used a modified, Finnish version of Elliot's and Coker's (2008) original self-rumination scale (Palomäki, Laakasuo, & Salmela, 2013). Participants were asked to evaluate 10 statements on

a scale of one to seven (1 = Strongly disagree, 7 = Strongly agree; example items: “Sometimes it’s hard for me to shut off thoughts about myself”, and “I often find myself re-evaluating something I have done”). The scale had a good inter-item reliability (Cronbach’s  $\alpha = 0.85$ ).

Physiological stress was measured by electrocardiogram (ECG), blood pressure (BP) and electrodermal activity (skin conductance); but for current purposes, only the relevant ECG and heart rate signal are described here. ECG was measured by modified lead II placement with signal frequency of the recording being 2000 Hz. The measurement device was Biopac and the collection software AcqKnowledge (version 3.8.2). Analysis was conducted using the ECGlab toolbox in Matlab (version 8.1; De Carvalho, da Rocha, de Oliveira Nascimento, Neto, & Junqueira, 2002). Frequency components above 35 Hz and below 0.05 Hz were suppressed from original signal using second order Butterworth filters, along with a bandpass filter at 50 Hz to remove mains noise. After R-spike detection, resultant data were visually inspected according to the instructions provided by Porges and Pyrne (1992). Ectopic values were identified and interpolated for HRV calculations.

The high-frequency (HF) component of HRV was extracted from the ECG using a frequency method. The HF component ranges between 0.15–0.4 hertz, and we defined it according to common practice utilising the time distance between ECG R-wave peaks. The HF component was extracted using half-hanning windows and by performing a fast Fourier transformation from the 512-point signal. This resulted in a numerical sum of squares value that reflects the HF component’s power in a unit of time. A higher numerical value reflects greater HRV.

The HF-component was calculated from the heart rate of the beginning *rest* (“baseline”) period, before the actual task. In addition, the HF-component was calculated from the stressful *speech preparation task* and from the *end rest* phases. Finally, a baseline-standardised variable (*reactivity*), was calculated, where the HF-component during beginning rest was subtracted from the speech preparation HF-component divided by standard deviation of the beginning rest HF-component. Selection of the utilised stress task (*preparation of*, rather than *keeping* an actual speech) was done keeping in mind that the HR data quality, and thus also HRV quality, is often compromised during an actual public speech task compared to the stationary *preparation* of the said task (Beda, Jandre, Phillips, Giannell, Neto, & Simpson, 2007).

### 2.3. Procedure

Participants signed informed consent forms at the beginning of the study, and were instructed they could withdraw at any point if they so wished. They were then instructed to wash their hands before moving into a soundproof, electrically shielded laboratory room, where they sat in front of a 17 inch computer monitor and were attached to physiological stress measurement devices, including ECG. Blood pressure was measured from the non-dominant hand, and skin conductance from the middle joints of the index and middle fingers; however, blood pressure and skin conductance responses are not the focus of this study. Participants were then informed that the researchers were able to monitor them through a webcam.

Next, the participants sat relaxed in the laboratory for five minutes with the laboratory door closed and lights switched off. This was the *beginning rest* (i.e. a baseline) period.

We utilised the *public speech task*, which is a well-known method used to induce stress (e.g., Määttänen, Ravaja, & Henttonen et al., 2018). Participants were instructed to compose a three-minute speech, in which they were to defend themselves against accusations of shoplifting, and to evaluate how well they expected to perform in this task. They were instructed to make up a story about what had happened, where it had happened, how the staff had behaved towards them and whether they would seek any compensation for the treatment they received. They were also instructed to articulate clearly and consistently while maintaining eye contact with the two researchers, who would be present to evaluate the speech. The participants were not allowed to use paper or pen while creating their speech, and they were instructed to speak exactly for the full three minutes.



The participants spent three minutes preparing their speech, and the time remaining was constantly shown on a countdown clock on the computer screen. After the preparation period, two researchers dressed in white coats (a man and a woman) entered the laboratory to “formally evaluate” the speech prepared by the participants. The participant was reminded that their speech should last exactly three minutes, again indicated by a countdown clock on the computer screen. After the public speech task, the researchers left the test room and participants were asked to fill out a questionnaire about their emotional state.

In addition to the public speech task, participants also completed a verbal memory task; this task involved remembering negative and neutral word lists and retrieving the lists afterwards, but is not covered in this article. The public speech- and verbal memory task order was counter-balanced across subjects, and there was a 3-minute intermediate rest period between the tasks.

After both tasks were completed, participants relaxed for another five minutes with the laboratory door closed and lights switched off (*end rest period*).

After the end rest, participants filled out the BDI-II questionnaire, Cloninger’s TCI questionnaire and the self-rumination scale. The self-report measures were collected after the tasks to avoid participants’ responses affecting the experimental set-up. Finally, the participants were thanked and thoroughly debriefed. In total, it took participants about one hour from start to finish at the laboratory.

#### **2.4. Data analysis**

As per our hypotheses, only the high-frequency (HF) -component of heart rate variability in the ECG was considered relevant, as it is a marker for activation of the parasympathetic nervous system. The HF-component was calculated for the beginning rest period (5 minutes) before any other tasks, the speech preparation period (3 minutes) and the *recovery* (*end rest*) period after the last stress task (5 minutes). In addition, the HF-variable *reactivity* was calculated.

Four participants did not complete the self-rumination questionnaire at the end of the study and were excluded from the analyses. The final sample size used for the analyses was thus 54 participants. One participant did not fully complete the BDI-II scale, and this participant was excluded from the analyses using the BDI-II variable. Outlier diagnostics revealed no significant outliers in any of our variables. Due to positive distribution skew, we log-transformed the BDI-II and HRV -variables.

In our analyses, we focused on Pearson zero-order correlations and multiple regression models (i.e. general linear models). Age was controlled in all analyses. Assumptions for using general linear models were fully met: the sample size was sufficient, the model error terms were close-to normally distributed and there was a linear relationship between the evaluated variables. We did observe some heteroscedasticity in the prediction errors between the variables. Thus, all analyses were also run using both robust regression and by examining bootstrapped confidence intervals. The main results did not change in any meaningful way when doing so, and thus heteroscedasticity was concluded to have no meaningful influence on the linear regression model.

#### **3. Results**

Variable characteristics are shown in Table 1. On average, HF-HRV decreased from the resting period to the preparation period, and then increased again during the recovery period. However, HF-HRV was still lower during the recovery period than during the resting period. The HRV reactivity variable is not shown, but it is logical that HF-HRV decreased from the resting period to the speech task period - a statistically significant negative correlation means higher reactivity.

The correlations between our variables were mainly consistent with the hypotheses. Only the correlation between HRV and depressive symptoms was not statistically significant; see Table 2.

**Table 1. The descriptives of age, depressive symptoms, rumination, harm-avoidance and HRV**

Variable	Mean	Standard deviation	Cronbach's alpha
Age	24.98	5.25	NA
Depressive symptoms (BDI-II)	29.12	7.56	.89
Rumination	43.70	10.76	.90
Harm avoidance	105.51	21.79	.85
HF-HRV: rest	202.73	261.82	NA
HF-HRV: SP	116.77	227.71	NA
HF-HRV: recovery	175.77	211.26	NA

(HF-HRV: rest = Beginning rest or baseline HF-HRV; HF-HRV: SP = Speech Preparation HF-HRV.) N = 54, except in BDI-II (n = 53).

As expected, depressive symptoms, rumination and harm avoidance were strongly positively intercorrelated ( $r_s = .56-.64$ ,  $p_s < .001$ ). The correlations between depressive symptoms and HRV across all HF measures were negative, but not statistically significant ( $r_s = -.1$  to  $-.26$ ,  $p_s > .05$ ).

Rumination was negatively associated with HRV, as was hypothesised. Rumination was most strongly, and statistically significantly, associated with HRV in the speech preparation period ( $r = -.325$ ,  $p = .017$ ), and to HRV reactivity ( $r = -.355$ ,  $p = .008$ ). However, rumination was not statistically significantly associated with neither rest nor recovery period HRV (at the  $p < .05$  level), even though the trends of the associations were consistent with the hypotheses.

Harm avoidance and its subscale scores were also negatively correlated with HRV in the speech preparation period, again supporting our hypotheses ( $r = -.358$ ,  $p = .012$ ). Of the harm avoidance dimensions, all but *shyness* had a statistically significant relationship with HRV during the speech preparation. However, harm avoidance was not statistically significantly associated with HRV reactivity ( $r = -.106$ ,  $p = .446$ ), with the exception of the harm avoidance dimension *anticipatory worry* ( $r = -.316$ ,  $p = .02$ ). The same was true for rest HRV, where the only statistically significant association (at the  $p < .05$  level) was with the harm avoidance subscale *fear of uncertainty* ( $r = .272$ ,  $p = .03$ ). In summary, the link between HRV and harm avoidance was more consistent when evaluating HRV during the preparation period, compared with baseline HRV or HRV reactivity.

Next, we ran a linear regression model predicting HF-HRV during the speech preparation period, since this HRV variable was most consistently linked to both harm avoidance and rumination in the correlational analyses. In the linear regression model, speech preparation HF-HRV was predicted by rumination, harm avoidance and participants' age. The resulting model was statistically significant ( $F(3, 50) = 3.571$ ,  $p = .02$ ), and explained 17.6 per cent of the variance in HF-HRV ( $r^2 = 0.176$ ). Rumination tendency was statistically significant negative predictor of speech preparation HF-HRV when presented alone in the model ( $t = -2.475$ ,  $p = .017$ ), as was harm avoidance ( $t = -2.764$ ,  $p = .008$ ). However, neither rumination ( $t = 0.894$ ), nor harm avoidance ( $t = 1.862$ ), were statistically significant predictors of speech preparation HF-HRV when they were presented in the model together (and with age), although their negative association with HF-HRV remained non-significant. This was most likely due to the high correlation between the rumination and harm-avoidance variables ( $r = .556$ ,  $p < .001$ ), and the mild multicollinearity between the two (Harm avoidance, Tolerance = 0.668, VIF = 1.497. Rumination, Tolerance = 0.661, VIF = 1.513), which led to a suppression effect when the variables were presented together in the model. In the combined model with rumination and harm avoidance, age was positively associated with HF-HRV, but this association was not statistically significant ( $t = 1.245$ ,  $p = n.s.$ ).

Table 2. Pearson correlations between the variables

Variable	1	2	3	4	5	6	7	8	9	10
1. Dep. symptoms										
2. Rumination	<b>0.64**</b>									
3. HA	<b>0.62**</b>	<b>0.56**</b>								
4. HA: Worry	0.60**	0.63**	0.82**							
5. HA: Uncertainty	0.35*	0.32*	0.80**	0.51**						
6. HA: Shyness	0.40**	0.35**	0.71**	0.39**	0.57**					
7. HA: Fatigability	0.57**	0.41**	0.83**	0.61**	0.57**	0.38**				
8. HF-HRV: Rest	-0.10	-0.11	-0.26^	-0.12	<b>-0.27*</b>	-0.25^	-0.20			
9. HF-HRV: SP	-0.17	<b>-0.33*</b>	<b>-0.36*</b>	<b>-0.31*</b>	<b>-0.28*</b>	-0.25^	<b>0.30*</b>	0.75**		
10. HF-HRV: Recovery	-0.20	-0.24^	<b>-0.31*</b>	-0.27^	-0.24^	-0.23^	-0.23^	0.80**	0.76*	
11. HF-HRV: Reactivity	-0.26^	<b>-0.36**</b>	-0.10	<b>-0.32*</b>	0.11	0.08	0.12	-0.01	0.02	0.11

Note. ^ =  $p < 0.1$ , \* =  $p < 0.05$ , \*\* =  $p < 0.01$ ,  $n = 54$ , except in variable 1 ( $n = 53$ )  
(HA = Harm avoidance, HF-HRV: SP = Speech Preparation HF-HRV). Hypotheses-relevant cells are highlighted, and within these significant ( $p < .05$ ) effects are in bold.



#### 4. Discussion

We aimed to shed light on the relationship between self-reported depressive symptoms, and psychological depression risk factors, namely, harm avoidance and rumination, and physiological stress as measured by HF-HRV. We conducted a laboratory study with a stress-inducing public speech task, and performed correlational and regression analyses to evaluate the associations between our variables.

Generally, our results were in line with existing research but we also made novel observations. As expected, harm avoidance, depressive symptoms and rumination were all positively intercorrelated, which is in line with previous research (Crucza et al., 2003; Elovainio et al., 2004; Farmer et al., 2003; Hansenne et al., 1999; Kampman & Poutanen, 2011; Nolen-Hoeksema, 1991, 2000; Nolen-Hoeksema & Davis, 1999; Nolen-Hoeksema et al., 2008; Robinson & Alloy, 2003; Ward et al., 2003). In earlier studies, the link between depressive symptoms and HF-HRV has been inconsistent, but more often than not there has been a negative association between the two (Bär et al., 2004; Kemp et al., 2010; Rottenberg, 2007). According to a review by Hamilton and Alloy (2016), HRV is consistently negatively associated with clinical depression, but there are inconsistencies when HRV is associated with depressive symptoms. In our study, the correlations between depressive symptoms and the HRV measures were also negative, but only the correlation between depressive symptoms and HRV reactivity almost reached statistical significance.

Rottenberg et al. (2007) proposed that decreased (closer to zero) HRV reactivity, compared with rest or baseline HRV, is more strongly associated with depressive symptoms. However, this was not supported by our results, which showed that depressive symptoms are *not* associated with weaker responses to challenges in the environment.

Previous studies on harm avoidance, rumination, and HRV have focused mainly on baseline HRV. In these studies, high harm avoidance scores, and especially high fatigability dimension scores, have been linked to low baseline heart-rate variability (Huang et al., 2013; Puttonen et al., 2008). Our results were somewhat consistent with these findings; however, harm avoidance was even more strongly associated with HRV during the speech preparation period. This underscores the importance of differentiating between various HRV indices when studying temperament- or other individual difference measures and physiological stress. Previous studies have also linked rumination-like thinking to lower baseline HRV (Brosschot et al., 2006, 2007). Our results, however, failed to support these findings. However, we did find support for the link between rumination and HF-HRV reactivity as well as HF-HRV during speech preparation.

Interestingly, in our study the psychological vulnerability factors of depression—harm avoidance and rumination—rather than depressive symptoms *per se*, were more consistently associated with HRV. However, this might be a logical outcome given that we excluded clinically depressed participants. It would be beneficial to follow the same individuals with multiple measurements to fully understand the possible causal route from psychological traits to HRV to depression.

Both harm avoidance and rumination were strongly correlated with HF-HRV during the speech preparation period. A multiple regression model with harm avoidance and rumination as predictors explained 17.6% of the variance in HF-HRV during preparation for a stressful task. Compared with typical studies in psychology, this model has high explanatory power; thus, harm avoidance and rumination together seem to form a prominent vulnerability factor predicting physiological stress in an ecologically valid task environment.

The harm avoidance subscale of *shyness* was not significantly associated with HF-HRV, despite the fact that the task itself most likely created a stressful social context (or an anticipation thereof). There was a trend, however, in the logical direction. The harm avoidance subscale of *fatigability* was *positively* associated with HF-HRV during the speech preparation task, which is contrary to the other subscale associations and previous research. Unlike previous research, we did

not find fatigability to be significantly associated with rest or baseline HF-HRV. Moreover, only rumination and the harm avoidance subscale of *worry*, but not harm avoidance itself, were associated with HF-HRV recovery.

In previous studies HRV was proposed to be a practical measure for understanding the link between depression and autonomic nervous system dysfunction, as well as a physiological measure of depression-related deficits in psychological self-regulation (Rottenberg et al., 2007; Sgoifo et al., 2015). HRV could potentially be used to create more accurate and efficient mental health diagnostics when used side by side with other physiological indicators; two of which, harm avoidance and rumination, were identified in our study to be strongly associated with HF-HRV. However, it is also important to measure HRV in a specific situation, rather than focusing merely on its baseline. Moreover, it might be fruitful to complement subjective symptom categorisation with physiological ones in depression diagnostics. This is in line with Rottenberg (2007), who proposed that depression affecting HRV might be a specific subtype of depression. Both our current study and that of Keen and colleagues (Keen, Turner, Mwendwa, Callender, & Campbell, 2015) suggest that different physiological changes are behind the different symptoms. If diagnostic systems could be developed to the point where they are able to take into account each patient's specific psychological and physiological symptoms, it might be possible to develop psychosocial and medical treatments that focus purely on those specific symptoms. This, in turn, might help to create more efficient mental health treatments.

There are some limitations in our study. The study was correlational and cross-sectional, and thus we could not infer directions of causality between our measures over extended periods of time. The study population does not represent the whole population (participants were young females), and the depressive symptom scores were higher than would be expected from a general population.

In conclusion, we demonstrated that specific psychological risk factors for depression, namely, harm avoidance and rumination, are significantly associated with physiological stress, as measured by HF-HRV during stressful and non-stressful situations. Our results contribute to extant literature by showing clear intercorrelations between multiple psychological and physiological measures associated with stress and depression.

## Abbreviations

HRV	Heart rate variability
HF-HRV	High-frequency component of heart rate variability

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## Competing Interest

The authors declare no competing interests.

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## References

- Bär, K., Greiner, W., Jochum, T., Friedrich, M., Wagner, G., & Sauer, H. (2004). The influence of major depression and its treatment on heart rate variability and pupillary light reflex parameters. *Journal of Affective Disorders*, 82, 245–252. doi:10.1016/j.jad.2003.11.009
- Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Manual for the beck depression inventory- II*. San Antonio, TX: Psychological Association.
- Beda, A., Jandre, F. C., Phillips, D. I., Giannella-Neto, A., & Simpson, D. M. (2007). Heart rate and blood pressure

- variability during psychophysiological tasks involving speech: Influence of respiration. *Psychophysiology*, 44(5), 767–778. doi:10.1111/j.1469-8986.2007.00542.x
- Birkhofer, A., Schmidt, G., & Förstl, H. (2006). Heart rate variability and depression [Letter to the editor]. *Archives of General Psychiatry*, 63 (9).
- Bleil, M. E., Gianaros, P. J., Jennings, J. R., Flory, J. D., & Manuck, S. B. (2008). Trait negative affect: Toward an integrated model of understanding psychological risk for impairment in cardiac autonomic function. *Psychosomatic Medicine*, 70(3), 328–337. doi:10.1097/PSY.0b013e31816baefa
- Brosschot, J. F., Gerin, W., & Thayer, J. F. (2006). The perseverative cognition hypothesis: A review of worry, prolonged stress-related physiological activation, and health. *Journal of Psychosomatic Research*, 60 (2), 113–124. doi:10.1016/j.jpsychores.2005.06.074
- Brosschot, J. F., Van Dijk, E., & Thayer, J. F. (2007). Daily worry is related to low heart rate variability during waking and the subsequent nocturnal sleep period. *International Journal of Psychophysiology*, 63, 39–47. doi:10.1016/j.ijpsycho.2006.07.016
- Bylsma, L. M., Salomon, K., Taylor-Clift, A., Morris, B. H., & Rottenberg, J. (2014). RSA reactivity in current and remitted major depressive disorder. *Psychosomatic Medicine*, 76(1), 66–73. doi:10.1097/PSY.000000000000019
- Carnevali, L., Thayer, J. F., Brosschot, J. F., & Ottaviani, C. (2018). Heart rate variability mediates the link between rumination and depressive symptoms: A longitudinal study. *International Journal of Psychophysiology*, 131, 131–138. doi:10.1016/j.ijpsycho.2017.11.002
- Cloninger, C. R. (1987). A systematic method for clinical description and classification of personality variants. *Archives of General Psychiatry*, 44, 573–588. doi:10.1001/archpsyc.1987.01800180093014
- Cloninger, C. R. (1994). *The temperament and character inventory (TCI): A guide to its development and use*. St. Louis, MO: Center for Psychobiology of Personality, Washington University.
- Cloninger, C. R., Svrakic, D. M., & Przybeck, T. R. (1993). A psychobiological model of temperament and character. *Archives of General Psychiatry*, 50, 975–990.
- Crucza, R. A., Przybeck, T. R., Spitznagel, E. L., & Cloninger, C. R. (2003). Personality and depressive symptoms: A multi-dimensional analysis. *Journal of Affective Disorders*, 74, 123–130. doi:10.1016/S0165-0327(02)00303-8
- De Carvalho, J. L. A., da Rocha, A. F., de Oliveira Nascimento, F. A., Neto, J. S., & Junqueira, L. F., Jr. (2002). *Development of a Matlab software for analysis of heart rate variability*. 6th international conference on signal processing, Beijing, China, pp. 1488–491, 26–30.
- Elliott, I., & Coker, S. (2008). Independent self-construal, self-reflection and self-rumination: A path model for predicting happiness. *Australian Journal of Psychology*, 60, 127–134. doi:10.1080/00049530701447368
- Elovainio, M., Kivimäki, M., Puttunen, S., Heponiemi, T., Pulkki, L., & Keltikangas-Järvinen, L. (2004). Temperament and depressive symptoms: A population-based longitudinal study on Cloninger's psychobiological temperament model. *Journal of Affective Disorders*, 83, 227–232. doi:10.1016/j.jad.2004.06.005
- Farmer, A., Mahmood, A., Redman, K., Harris, T., Sadler, S., & McGuffin, P. (2003). A sib-pair study of the temperament and character inventory scales in major depression. *Archives of General Psychiatry*, 60, 490–496. doi:10.1001/archpsyc.60.5.490
- Hamilton, J. L., & Alloy, L. B. (2016). Atypical reactivity of heart rate variability to stress and depression across development: systematic review of the literature and directions for future research. *Clinical Psychology Review*, 50, 67–79.
- Hansenne, M., Reggers, J., Pinto, E., Kijiri, K., Ajamier, A., & Anssea, M. (1999). Temperament and character inventory (TCI) and depression. *Journal of Psychiatric Research*, 33, 31–36. doi:10.1016/S0022-3956(98)00036-3
- Hong, R. Y. (2007). Worry and rumination: Differential association with anxious and depressive symptoms and coping behavior. *Behaviour Research and Therapy*, 45, 277–290. doi:10.1016/j.brat.2006.03.006
- Huang, W., Chang, L., Kuo, B. T. N. J., Lin, Y., Chen, Y., & Yang, C. C. H. (2013). Gender differences in personality and heart rate variability. *Psychiatry Research*, 209(3), 652–657. doi:10.1016/j.psychres.2013.01.031
- Kampman, O., & Poutanen, O. (2011). Can onset and recovery in depression be predicted by temperament? A systematic review and meta-analysis. *Journal of Affective Disorders*, 135, 20–27. doi:10.1016/j.jad.2010.12.021
- Keen, L., Turner, A. D., Mwendwa, D., Callender, C., & Campbell, A. (2015). Depressive symptomatology and respiratory sinus arrhythmia in a non-clinical sample of middle-aged African Americans. *Biological Psychology*, 108, 56–61. doi:10.1016/j.biopsycho.2015.03.008
- Kemp, A. H., Quintana, D. S., Gray, M. A., Felmingham, K. L., Brown, K., & Gatt, J. M. (2010). Impact of depression and antidepressant treatment on heart rate variability: A review and meta-analysis. *Biological Psychiatry*, 67(11), 1067–1074. doi:10.1016/j.biopsycho.2009.12.012
- Koschke, M., Boettger, K., Schulz, S., Berger, S., Terhaar, J., Voss, A., ... Bär, K. J. (2009). Autonomy of autonomic dysfunction in major depression. *Psychosomatic Medicine*, 71, 852–860. doi:10.1097/PSY.0b013e3181b8bb7a
- Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal and coping*. New York, NY: Springer Publishing.
- Licht, C. M. M., de Geus, E. J. C., Zitman, F. G., Hoogendijk, W. J. G., van Dyck, R., & Penninx, B. W. J. H. (2008). Association between major depressive disorder and heart rate variability in the Netherlands study of depression and anxiety (NESDA). *Archives of General Psychiatry*, 65, 1358–1367. doi:10.1001/archpsyc.65.12.1358
- Määttänen, I., Ravaja, N., Henttonen, P., Puttunen, S., Paavonen, K., Swan, H., ... Hintsala, T. (2018). Type 1 long QT syndrome and psychological stress in a laboratory setting. *Journal of Health Psychology First Published January*, 22, 2018. doi:10.1177/1359105317751617
- Miu, A. C., Heilman, R. M., & Miclea, M. (2009). Reduced heart rate variability and vagal tone in anxiety: Trait versus state, and the effects of autogenic training. *Autonomic Neuroscience*, 145, 99–103. doi:10.1016/j.autneu.2008.11.007
- Monroe, S. M., & Simons, A. D. (1991). Diathesis-stress theories in the context of life stress research: Implications for the depressive disorders. *Psychological Bulletin*, 110, 406–425. doi:10.1037/0033-2909.110.3.406

- Nolen-Hoeksema, S. (1991). Responses to depression and their effect on the duration of depressive episodes. *Journal of Abnormal Psychology*, 100, 569–582. doi:10.1037/0021-843X.100.4.569
- Nolen-Hoeksema, S. (2000). The role of rumination in depressive disorders and mixed anxiety/depressive symptoms. *Journal of Abnormal Psychology*, 109, 504–511. doi:10.1037/0021-843X.109.3.504
- Nolen-Hoeksema, S., & Davis, C. G. (1999). “Thanks for sharing that”: Ruminators and their social support networks. *Journal of Personality and Social Psychology*, 77 (4), 801–814. doi:10.1037/0022-3514.77.4.801
- Nolen-Hoeksema, S., Larson, J., & Grayson, C. (1999). Explaining the gender difference in depressive symptoms. *Journal of Personality and Social Psychology*, 77, 1061–1072. doi:10.1037/0022-3514.77.5.1061
- Nolen-Hoeksema, S., Wisco, B. E., & Lyubomirsky, S. (2008). Rethinking rumination. *Perspectives on Psychological Science*, 3(5), 400–424. doi:10.1111/j.1745-6924.2008.00088.x
- Nyman, E., Miettunen, J., Freimer, N., Joukamaa, M., Mäki, P., Ekelund, J., ... Paunio, T. (2011). Impact of temperament on depression and anxiety symptoms and depressive disorder in a population-based birth cohort. *Journal of Affective Disorders*, 131(1–3), 393–397. doi:10.1016/j.jad.2010.12.008
- Ottaviani, C., Verkuil, B., Medea, B., Couyoumdjian, A., Thayer, J., Lonigro, A., & Brosschot, J. F. (2016). Physiological concomitants of perseverative cognition: A systematic review and meta-analysis. *Psychological Bulletin*, 142(3), 231–259. doi:10.1037/bul0000036
- Palomäki, J., Laakasuo, M., & Salmela, M. (2013). “Don’t worry, it’s just Poker!”—Experience, self-rumination and self-reflection as determinants of decision-making in on-line Poker. *Journal of Gambling Studies*, 29(3), 491–505. doi:10.1007/s10899-012-9311-3
- Pieper, S., Brosschot, J. F., van der Leeden, R., & Thayer, J. F. (2007). Cardiac effects of momentary assessed worry episodic and stressful events. *Psychosomatic Medicine*, 69, 901–909. doi:10.1097/PSY.0b013e31815a9230
- Porges, S. W. (1992). Vagal tone: A physiological marker to stress and vulnerability. *Pediatrics*, 90, 498–504.
- Porges, S. W., & Byrne, E. A. (1992). Research methods for measurement of heart rate and respiration. *Biological Psychology*, 34, 93–130. doi:10.1016/0301-0511(92)90012-J
- Porges, S. W., Doussard-Roosevelt, J. A., & Maiti, A. K. (1994). Vagal tone and the physiological regulation of emotion. *Monographs of the Society for Research in Child Development*, 59, 167–186. doi:10.2307/1166144
- Puttonen, S., Elovainio, M., Kivimäki, M., Koskinen, T., Pulkki-Räback, L., Viikari, J. S. A., ... Keltikangas-Järvinen, L. (2008). Temperament, health-related behaviors, and autonomic cardiac regulation: The cardiovascular risk in young Finns study. *Biological Psychology*, 78(2), 204–210. doi:10.1016/j.biopsycho.2008.03.003
- Robinson, L. A., & Alloy, L. B. (2003). Negative cognitive styles and stress-reactive rumination interact to predict depression: A prospective study. *Cognitive Therapy and Research*, 27, 275–292. doi:10.1023/A:1023914416469
- Rottenberg, J. (2007). Cardiac vagal tone in depression: A critical analysis. *Biological Psychology*, 74, 200–211. doi:10.1016/j.biopsycho.2005.08.010
- Rottenberg, J., Clift, A., Bolden, S., & Salomon, K. (2007). Rsa fluctuation in major depressive disorder. *Psychophysiology*, 44, 450–458. doi: 10.1111/psyp.2007.44.issue-3
- Sgoifo, A., Carnevali, A., Alfonso, M. P., & Amore, M. (2015). Autonomic dysfunction and heart rate variability in depression. *Stress: International Journal of the Biology of Stress*, 18(3), 343–352. doi:10.3109/10253890.2015.1045868
- van der Kooy, K. G., van Hout, H. P., van Marwijk, H. W., de Haan, M., Stehouwer, C. D., & Beekman, A. T. (2006). Differences in heart rate variability between depressed and non-depressed elderly. *International Journal of Geriatric Psychiatry*, 21, 147–150. doi:10.1002/gps.1439
- Ward, A., Lyubomirsky, S., Sousa, L., & Nolen-Hoeksema, S. (2003). Can’t quite commit: Rumination and uncertainty. *Personality and Social Psychology Bulletin*, 29, 96–107. doi:10.1177/0146167202238375
- Yaptangco, M., Crowell, S., Baucom, B. R., Bride, D. L., & Hansen, E. J. (2015). Examining the relation between respiratory sinus arrhythmia and depressive symptoms in emerging adults: A longitudinal study. *Biological Psychology*, 110, 34–41. doi:10.1016/j.biopsycho.2015.06.004



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