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Resting heart rate variability moderates the relationship between trait emotional competencies and depression

Elise Batselé[a,b,*], Nicolas Stefaniak[c], Carole Fantini-Hauwel[a]

[a] Université libre de Bruxelles, Center Research of Clinical Psychology, Psychopathology and Psychosomatic
[b] National Fund for Scientific Research, FNRS, Belgium
[c] University of Reims Champagne-Ardenne, Cognition, Health and Society Lab (C2S), France

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ABSTRACT

Objective: A lot of studies have shown that low Emotional Competencies (EC) is associated with depression and anxiety. However, little is known about the psychophysiological processes accounting for these relationships. As heart rate variability (HRV) is thought to be a measure of top-down self-regulatory mechanisms it could impact the link between EC and depression/anxiety. Thus, the aim of this study was to disentangle the interplay between EC and HRV on depression as well as anxiety.
Method: Resting HRV was collected among 97 undergraduate students that filled out EC, depression and anxiety measures.
Results: We observed negatives associations between EC and depression/anxiety. HRV was negatively associated with depression but not with anxiety. There was an interaction effect between EC and HRV showing that EC and depression were associated only at low levels of HRV.
Conclusions: Our study suggests that HRV could be a protective factor against the negative consequences of low EC such as depression.

1. Introduction

All human beings experiment emotions, but individuals differ in how they manage and process these emotions (Petrides & Furnham, 2003). EC – also called "emotional intelligence" (EI) – offer a theoretical conceptualization of this idea and are defined as the way individuals identify, understand, express, regulate and use their own and others' emotions (Mayer & Salovey, 1997; Petrides & Furnham, 2003). Although the term EI is more commonly used in scientific literature, the term EC is preferable because it is more consistent with recent results indicating that EC can be trained and improved (Kotsou, Grégoire, Nelis, & Mikolajczak, 2011). It's also important to highlight the distinction between trait EC, usually measured with self-report questionnaires (i.e. PEC: Brasseur, Grégoire, Bourdu, & Mikolajczak, 2013) and ability EC, assesses with performance-based measures (i.e. MSCEIT: Mayer, Salovey, & Caruso, 2002) as suggested by Petrides and Furnham (2001). Indeed, the former refers to stable dispositions and self-perceived competencies while the latter encompasses actual abilities, in fact what EC people really have and not what they think about their own EC. The current study focused on the trait EC as ability EC is known to overlap with many others cognitive intelligence indices (Webb et al., 2013) and to be less or unrelated with affective disorders (Goldenberg, Matheson, & Mantler, 2006).

EC are essential to cope adequately with the environment. For instance, a recent cumulative meta-analysis showed that EC have a moderate and positive relationship with physical, psychosomatic and mental health suggesting that EC should be an important health predictor (Martins, Ramalho, & Morin, 2010). More specifically, lower levels of trait EC have been associated with internalizing disorders such as depression and anxiety (Lizeretti & Extremera, 2011; Lloyd, Malek-Ahmadi, Barclay, Fernandez, & Chartrand, 2012), even after controlling for age, gender, personality and cognitive abilities (Davis & Humphrey, 2012). Furthermore, recent research suggests that trait EC should be a vulnerability factor to such psychopathological issues (Gomez-Baya, Mendoza, Paino, & de Matos, 2017).

EC is also related to an important concept largely known to be a transdiagnostic marker of psychopathology. Indeed, emotion regulation, defined as the processes by which an individual manages its emotional responses, have been widely investigated in mental health and there are evidences of a broad deficit of emotion regulation in

* Corresponding author at: Center of Clinical Psychology, Psychopathology and Psychosomatic Research, Université Libre de Bruxelles, Av. F. Roosevelt 50, 1050 Brussels, Belgium.
E-mail address: elise.batsele@ulb.ac.be (E. Batselé).

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anxiety and depression (i.e. Aldao, Nolen-Hoeksema, & Schweizer, 2010). To date, most studies have established a positive relationship between EC and functional emotion regulation strategies and a negative link with dysfunctional ones (Peña-Sarrionandia, Mikolajczak, & Gross, 2015). However, these studies mainly rely on self-perceived emotion regulation and rarely account for more objective emotion regulation markers such as parasympathetic nervous system (PNS) activity, which is measured through vagally mediated heart rate variability (HRV[1]), i.e. the change in the time interval between successive heartbeats. Two major theories have led researchers to consider HRV as reflecting top-down self-regulatory processes (i.e. executive functioning, emotion regulation, working memory, attentional regulation) (Holzman & Bridgett, 2017). According to Porge's polyvagal theory (Porges, 1995; Porges, 2001), the ability of the ventral vagal complex to rapidly withdraw its inhibitory influence allows humans to rapidly engage and disengage with their environment without the metabolic cost of activating the slower-responding sympathetic nervous system. Indeed, many social processes (i.e., nonverbal communication, romantic courtship) require this rapid management of metabolic resources (24). Thus, the polyvagal theory emphasizes the relationship of ventral vagal complex activity and the regulation of the emotional processes underlying social behavior. Secondly, Thayer and Lane's neurovisceral integration model (Thayer & Lane, 2000) states that HRV is influenced the central autonomic network (CAN), the stellate ganglia and the vagus nerve. The CAN receives information from internal and sensory external environment (input) and sends output that directly influences heart rate. This is the reason why HRV is considered to be a reflection of the CAN output and more specifically, the index of the physiological responses regulation when emotions arise. As such, HRV's level appears to be a good indicator of the ability to regulate emotions as it assesses the strength of vagal influence to adjust the cardiac reaction when emotions arise (Thayer, Hansen, Saus-Rose, & Johnsen, 2009).

As HRV is thought to be a transdiagnostic biomarker of psychopathology reflecting emotion regulation abilities (Beauchaine & Thayer, 2015), its relationships with depression or anxiety have been widely studied. Decreased HRV has often been associated with those two disorders (Brunoni et al., 2013; Chalmers, Quintana, Abbott, & Kemp, 2014). Interestingly, a longitudinal study has shown that participants free of depression and exhibiting lower HRV at baseline, were more prone to develop depressive symptoms after 10 years, even after controlling for potential cofounding variables (Jandackova, Britton, Malik, & Steptoe, 2016). So, HRV may be a trait biological marker, increasing the vulnerability to develop affective disorders (Brunoni et al., 2013), whose heritability is estimated between 47 and 60% (Golosheykin, Grant, Novak, Heath, & Anokhin, 2017). Although these studies displayed significant relationships between depression/anxiety and HRV, other researchers have found contradictory results (Hammel et al., 2011; Moser et al., 1998).

So, an interesting question arises about the interplay between trait EC and HRV on depression and anxiety as they are both known to have consequences on them. To date, there are only two studies about trait EC and HRV. They evidence inconclusive results but the two studies (Craig et al., 2009; Laborde, Brüll, Weber, & Anders, 2013) rely on HRV markers that reflect a mixture of PNS and sympathetic nervous system (SNS) influences (LF/HF ratio and SDNN). Recent recommendation states to use HRV indices that refer to clearly identified physiological systems (Laborde, Mosley, & Thayer, 2017).

The aim of this study was to disentangle the interplay between trait EC and HRV, as a measure of top-down self-regulatory mechanisms, on depression and anxiety. Indeed, how HRV influence the relation between trait ERC and psychological disorders remains unclear. HRV could be a moderator of the relationship between trait EC and depression/anxiety. For example, neuroticism has less detrimental consequences on life outcomes at higher level of HRV but not at low (Ode, Hilmert, Zielke, & Robinson, 2010). Further, it has also been shown that the consequences of thought suppression efforts on emotional distress are more likely to appear at low HRV level but not at high level (Gillie, Vasey, & Thayer, 2015). Thus, a moderation effect could not be excluded highlighting the flexibility and protective role of HRV. An alternative hypothesis posits that HRV is a mediator between trait EC and depression/anxiety. Indeed, some studies have revealed that the effect of emotion regulation on affective disorders is partially indirect through HRV (Appelhans & Luecken, 2006; Thayer et al., 2009). Emotion regulation difficulties could lead to a lower HRV and then to negative consequences. As trait EC and emotion regulation are partly entangle, this hypothesis needs to be assessed.

2. Method

A total of 108 participants took part in the experiment. Due to the exclusion criteria, 7 subjects were excluded and 4 other subjects were removed due to missing data. Thus, the final sample was composed of 97 undergraduate students, who participated in the study in order to validate credits for a psychology course (82 females, mean age = 22.05 ± 3.47). Exclusion criteria were: (1) having a chronic somatic disease, (2) having a psychiatric diagnosis and (3) having difficulty understanding the French language. We have determined the required sample size with an a priori power analysis (G power) with 4 predictors (HRV, CE, HRV X CE and age as control variable). 85 participants were necessary to achieve a power of 0.80, with an alpha of 0.05 and a medium effect size of 0.15. The study was approved by the faculty ethic committee.

2.1. Procedure

Participants were instructed not to smoke, engage in physical exercise or drink coffee/alcohol/energizing drinks 3 h before undergoing the experiment. They were seated in an experimental room during 30 min, which was equipped with a computer. An electrode belt was first attached to the participant's chest to complete a 5-min HRV resting period. The measure started 3–4 min after they seated in front of the screen of the computer (blank screen), just after they were given a detailed explanation of the procedure. Participants were sitting with hands on the knees and bent legs. They were spontaneously breathing and were instructed not to move. After the HRV measure, participants were asked to complete a set of online questionnaires.

Questionnaires

Trait emotional competencies. We used the French version of the PEC (Brasseur et al., 2013), a 50 items scored on a five-point Likert scale (from *strongly disagree* to *strongly agree*). We focused on the two main factor scores: intrapersonal EC (α = 0.85), an interpersonal EC (α = 0.84) and the total EC factor score (α = 0.86).

Depression and anxiety. We used the French validated version of the Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983), a 14-item scale (7 for anxiety and 7 for depression) scored on a 4-point Likert scale ranging from 0 to 3. The two-dimensional structure of the HADS exhibits a good internal consistency (α anxiety = 0.77; α depression = 0.72).

Heart rate variability. HRV was measured by using a Polar® V800 heart rate monitor allowing to extract HRV parameters. The measure taken by the Polar® V800 has been validated as comparable to an electrocardiograph (Giles, Draper, & Neil, 2016). The electrode belt was dampened and placed following Polar's guidelines, tightly but comfortably just below the chest muscles. Measurements were conducted during 5 min (spontaneous breathing and resting state), according to the recommendations of the Task Force of The European Society of Cardiology and The North American Society of Pacing and Electrophysiology (1996). The variability between successive R-spikes

[1] For clarity reasons, we will use the HRV acronym to refer to the vagally mediated HRV.

Table 1
Descriptive statistics.

Variable	Mean ± SD	Min.	Max.
Age	22.05 ± 3.47	19.00	44.00
BMI	22.02 ± 3.22	15.78	37.11
HRV (RMSSD)	37.25 ± 19.98	9.23	105.15
Ln HRV	3.48 ± 0.54	2.22	4.66
Intrapersonal EC	3.13 ± 0.54	1.52	4.24
Interpersonal EC	3.67 ± 0.40	1.84	4.44
Total EC	3.40 ± 0.38	2.22	4.14
Anxiety	8.29 ± 3.83	1.00	21.00
Depression	4.27 ± 3.12	0.00	18.00

was then analyzed using Kubios 3.0.2 premium version (Tarvainen, Niskanen, Lipponen, Ranta-Aho, & Karjalainen, 2014), which allows for the calculation of time- and frequency-domain indices of HRV. Artifacts within the R–R series were visually detected, and we applied an automatic artifact correction level (for more detailed informations, see Tarvainen et al., 2014). Among the HRV indicators, we chose to use the RMSSD (ms2), a time domain measure of HRV reflecting PNS modulation of heart rate (1996). RMSSD is thought to be relatively free of respiratory influences and thus appear as a more reliable measure of HRV than others indices (Hill, Siebenbrock, Sollers, & Thayer, 2009; Task Force of The European Society of Cardiology and The North American Society of Pacing and Electrophysiology, 1996). The HRV index we used in this study has been natural log-transformed (ln) to fit assumptions of linear analyses (Ellis, Sollers, Edelstein, & Thayer, 2008).

2.2. Statistical analyses

First, we used the Bayes factor to decide what covariates should be taken into account by using R and the Bayes factor package (R Core Team, 2017, Morey & Rouder, 2015). We have examined Pearson's correlations and proceed to robust (HC3) moderation and mediation analysis using the process macro (Hayes, 2017). Thirdly, all interaction terms were centered prior to compute interaction effect following Aiken and West (1991) recommendations.

3. Results

Descriptive statistics are provided in Table 1.

Model selection. In order to determine which covariates needed to be included in subsequent analyses, we used Bayes Factor (BF) to decide if covariates add significant information to our model (Rouder & Morey, 2012). The BF is a ratio of two likelihood estimation. Larger values for BF reveal that the model is more probable while smaller values reveal that the model is less probable. The BF can be compared together by computing the ratio between two BF. We estimated the BF from the

Table 2
Bayes Factors of the retained models.

Models	BF
1. HRV + EC + HRV × EC	216.45
2. Age + HRV + EC + HRV × EC	622.65
3. Age + Gender + HRV + ERD + HRV × ERD	528.00
4. Age + Gender + Coffee + Alcohol + Smoking + BMI + HRV + ERD + HRV × ERD	13.31

Table 3
Correlation analyses.

	HRV	Anxiety	Depression	Intrapersonal EC	Interpersonal EC	Total score EC
Age	−0.16	0.18	0.24*	0.04	−0.17	−0.06
HRV	–	−0.08	−0.32**	0.16	0.18	0.21*
Anxiety	–	–	0.42**	−0.39**	0.10	−0.22*
Depression	–	–	–	−0.36**	−0.15	−0.33**

* $p < .05$.
** $p \leq .01$.

Table 4
Moderation analyses for depression.

	(1)			(2)			(3)		
	ß	Robust SE	p	ß	Robust SE	p	ß	Robust SE	p
Age	0.21*	(0.08)	0.013	0.27***	(0.07)	0.000	0.18*	(0.08)	0.035
HRV	−0.25*	(0.73)	0.046	−0.25*	(0.61)	0.017	−0.27*	(0.75)	0.039
Total EC	−0.26**	(0.72)	0.004						
HRV × Total EC	0.18	(1.64)	0.06						
Intrapersonal EC				−0.29***	(0.47)	0.001			
HRV × Intrapersonal EC				0.28**	(1.19)	0.011			
Interpersonal EC							−0.09	(0.82)	0.39
HRV × Interpersonal EC							−0.12	(1.77)	0.34
R^2	0.24			0.32			0.16		
F	6.15***			8.70***			4.01**		

ß = Standardized beta coefficients; Standard errors in parentheses.
* $p < .05$.
** $p < .01$.
*** $p < .001$.

simplest (without covariates) to the full covariates model. The description of each model and their associated BF are reported in Table 2. It appears that the model in which age has been added is 2.87 (i.e., 622.65/216.45) more probable than the model without age. However, the others models are not only less parsimonious but also deteriorates the likelihood since the Bayes Factors decrease when other variables are added in the model. In other words, the most likely model is the model in which only age is added in the model. So we retained age as covariate in order to be conservative.

Correlation analyses (Table 3) revealed negatives and significant relationships between total trait EC and depression ($r = -0.33$, $p = .001$) as well as anxiety ($r = -0.22$, $p = .029$). Intrapersonal trait EC was negatively correlated to depression and anxiety. We also found a positive and significant relationship between total trait EC and HRV ($r = 0.21$, $p = .042$). Depression was negatively and significantly correlated to HRV ($r = -0.32$, $p = .001$). All other variables' relations were not significant.

Moderation analysis: As shown in Tables 4, 24% of the variance of depression was accounted by Age, HRV and total trait EC. The more age increase, the more depression increase. However, the lower trait EC and HRV are, the higher is depression. We didn't find a significant total trait EC x HRV interaction on depression.

Considering intrapersonal trait EC, 32% of the depression scores' variance was explained by age, HRV and intrapersonal trait EC, as well as by the interaction between intrapersonal trait EC and HRV (Fig. 1). For high HRV people (+1 SD), there was no significant relationship between intrapersonal trait EC and depression (B = 0.02, $t = 0.03$, $p = .97$, 95%CI [−1.36 to 1.40]). However, people exhibiting lower levels of HRV (−1 SD) reported a significant and negative relationship between intrapersonal trait EC and depression (B = −3.33, $t = -3.75$, $p \leq .005$, 95%CI [−5.09 to −1.56]). The interaction contributes to 8% of the total variance ($F[1, 92] = 13.16$, $p < .001$).

bootstrap confidence intervals (10.000 samples). All indirect effect were non-significant, both for depression and anxiety, whatever the trait EC used as independent variable (total, intrapersonal or interpersonal), indicating that a mediation model did not provide a better fit to the data structure (see Table A.2 in supplementary files).

4. Discussion

The purpose of this study was to disentangle the interplay between trait EC and HRV on depression and anxiety. Correlations analyses revealed negative associations between trait EC and depression/anxiety as previously reported in the literature (Lizeretti & Extremera, 2011; Lloyd et al., 2012). Specifically, intrapersonal trait EC, but not interpersonal trait EC, was related to depression and anxiety. Thus, our results confirm and emphasize the strength of the relationship between trait EC and mental health (Martins et al., 2010). We also observed a positive association between HRV and trait EC. To our knowledge, only two studies have explored these relationships but failed to show any relation (Craig et al., 2009; Laborde et al., 2013). However, it's difficult to compare these previous findings with our results as they used EI questionnaires instead of trait EC measures. Further, they used the LF/HF ratio, which is a controversial index of HRV that evaluates both PNS and SNS (Billman, 2013). Anyway, even if the size of the correlation we observed in our study between trait EC and HRV is small regarding Cohen's classification, it should be noted that trait EC is probably more linked to the PNS than the SNS. Furthermore, most of the past research has found associations between HRV and clinical depression (Brunoni et al., 2013). Our results indicate that this link also exists in non-clinical populations. It is quite surprising that we didn't observe the same association for anxiety, as it seems also to be well established in the literature but on clinical population (Chalmers et al., 2014). Future studies on non-clinical populations (and not trait or diagnosed clinical

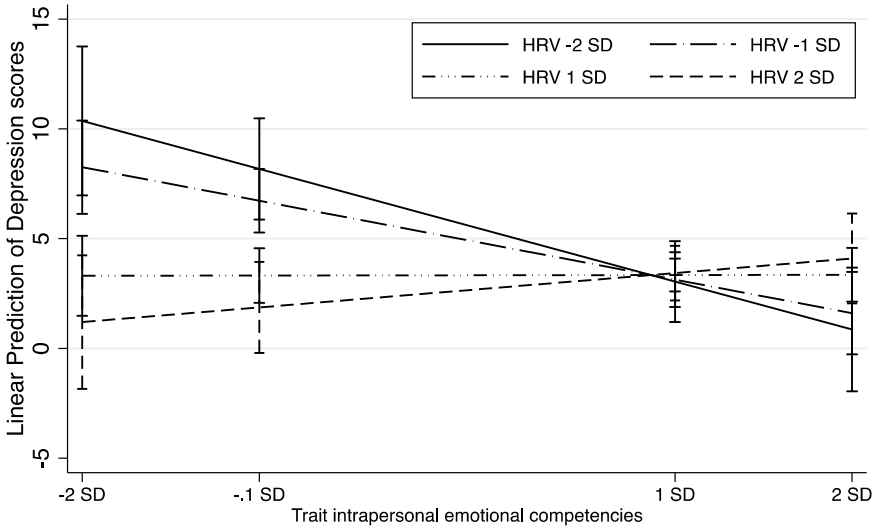

Fig. 1. Interaction effect between intrapersonal EC and HRV on depression.

Finally, when considering interpersonal trait EC, we found that 16% of the depression scores' variance was explained by all variables with only a significant and positive main effects of age and a significant and negative one for HRV.

For anxiety, moderation analysis didn't reach the significance level (see Table A.1 in supplementary material). We just observed a significant effect of intrapersonal trait EC ($b = -0.38$, $p \leq .001$) with $F(4, 92) = 5.47$, $p \leq .001$, $R^2 = 0.20$.

Mediation analysis: We examined the indirect effect of EC on depression and anxiety through HRV, controlled for age with a robust (HC3) mediation analyze. This indirect effect was computed using 95%

disorder) would be necessary in order to understand if HRV is only linked to diagnosed anxiety disorders or if this relationship also exists for people experiencing some symptoms in everyday life.

Our study aimed to compare two hypotheses. The first hypothesis was that HRV would be a moderator of the relationship between trait EC and depression as well as anxiety. The second and alternative hypothesis was that HRV could be a mediator between trait EC and depression/anxiety. Our results seem to confirm the litterature regarding the link between trait EC and depression/anxiety (Lizeretti & Extremera, 2011; Lloyd et al., 2012). However, the moderating effect of HRV on the relationship between trait EC and depression nuances the

consequences of low trait EC trait. At low levels of HRV there was a negative association between trait intrapersonal EC and depression. But on the contrary, at high levels of HRV this association was no longer significant. Thus, high levels of HRV could be a protective factor against the consequences of low trait intrapersonal EC. It can be explained by the fact that HRV is a bio-marker of top-down self-regulatory mechanisms including executive functioning, working memory, attentional regulation, inhibitory control as well as emotional regulation (Holzman & Bridgett, 2017; Porges, 1995; Thayer et al., 2009) and all those flexibility processes are involved in depression (De Raed, Koster, & Joormann, 2010; Joormann & Gotlib, 2008; Monteiro et al., 2016). Supporting this idea, some studies have already underlined the moderating, and therefore protective, role of HRV against negative consequences of neuroticism and emotional distress following thought suppression (Gillie, Vasey, & Thayer, 2014; Ode et al., 2010). Our study suggests that the actual regulation's resources of a person, indexed by HRV, might vary independently of how bad they perceive their EC. We should note that we didn't find any moderation effect analyses regarding anxiety. Finally, we didn't confirm our mediation hypothesis as the analyses were not significant. Thus, our study suggests that HRV has more a moderator role in the relationship between trait EC and depression than a mediator one.

Several limitations have to be acknowledged. The first one resides in the sample composition: the participants were mainly females. This could impact the generalizability to both genders (Martins et al., 2010). Also, participants were young and university-instructed, which raises direct concerns about the generalizability of the results to other populations. The last main limitation pertains to the direction of causality. Namely, it is probable that the causality is not unidirectional (i.e., trait EC predicting depression/anxiety) but is rather bidirectional. It is indeed likely that low trait EC could lead to the development depression/anxiety, as it reveals behavioral dispositions that lead to difficulties in managing emotions, but this condition could also affect the way people perceive their EC.

Further studies investigating the links between trait EC, but also ability EC, HRV and depression as well as anxiety would be necessary in order to replicate our findings. Plus, it seems important to identify what is, if any, the specific processes indexed by HRV (attention, inhibitory control, executive functions, …) that explain its protective effect.

Conflicts of interest

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article. For the remaining authors, none were declared.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.paid.2018.09.020.

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