Cardiovascular reactivity during sadness induction predicts inhibitory control performance

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ABSTRACT

Higher negative affectivity has an association with decreased executive function and cognitive control. Heart rate variability (HRV) serves as an index of cardiac vagal regulation differences in the autonomic nervous system for both cognition and emotion. The current study investigates this association using a classic as well as emotional antisaccade paradigm to study inhibitory control performance. Ninety participants completed affective questionnaires (Beck Depression Inventory-II, and Mood Scale), a 6-minute baseline electrocardiogram, and two different antisaccade tasks. After the baseline, subjects were presented with a video sequence with either neutral, sad, or emotionally arousing content. By subtracting the baseline from the video sequence, we computed HRV reactivity and tested whether the reactivity score could predict inhibitory control performance. We hypothesized that this would be the case in both the sadness and arousal group, but not in the neutral one. Furthermore, we awaited significant performance differences between experimental groups. Contrary to our assumption, inhibitory control performance did not differ between experimental groups. Moreover, there was no significant relation between affective measures and task performance. Nevertheless, cardiovascular reactivity in terms of HRV was predictive of error rates in both antisaccade tasks in the sadness group. We could find this effect neither in the neutral nor in the arousal group. In addition, BDI scores moderated the effect in the emotional task. Results indicate that emotional reactivity to a sad video stimulus as indexed by HRV as well as the interaction with current emotional state predict inhibitory control performance.

1. Introduction

Depression, sadness or negative affect in general are associated with negative consequences on physical health as well as impairments in cognitive functioning in clinical and non-clinical samples [76]. This association has been found across several executive functioning domains [30, 33, 38, 40]. Furthermore, the lack of emotion regulation ability constitutes a core feature of emotional problems, which is part of a broad range of mental disorders, such as depression, anxiety disorders, and others [3]. Xiu et al. [79] found that working memory training not only improves working memory speed, but also emotion regulation ability in terms of better heart rate variability (HRV).

Theoretical frameworks such as the polyvagal theory [58] and the neurovisceral integration model [70] proposed a connection between emotion regulation and HRV. On a central-nervous level, this interaction is emphasized by an overlap of neural networks that are in charge of autonomic and affective regulation. Here, the ventromedial prefrontal cortex, insula, amygdala, and the cingulate cortex are responsible for the autonomic control of heart rate as well as for emotion processing [73]. Connections between emotional state and HRV have been shown recently [30]. Individuals with higher HRV exhibit better emotion regulation [2, 51, 55] and show lower levels of anxiety [46, 60]. Furthermore, it is suggested that higher HRV is beneficial in social interactions [67, 68].

Research has repeatedly shown that there is a positive relationship between HRV and executive functioning [16, 22, 27, 36], including inhibitory control [8, 32], and set shifting [9, 32, 34]. A meta-analysis of 26 studies that applied a large range of cognitive tasks, only showed a small correlation between HRV and self-control performance [80]. Another systematic review on the relationship between HRV and cognitive functioning [21] suggested that increased sympathetic and decreased parasympathetic activity were associated with poorer
executive functioning across several cognitive tasks. Both the polyvagal theory [58] and the neurovisceral integration model [70] further proposed HRV to be connected to the functionality of the prefrontal cortex, which is the neural correlate of executive functioning [47]. Both frameworks received approval by studies using imaging techniques that showed that activation in prefrontal areas is positively correlated with HRV indices [39, 73].

While most studies focused on the investigation of baseline HRV (e.g. [34, 52]), we would like to focus on physiological reactivity, which constitutes short-term changes in physiological states from baseline to changing conditions and thus reflects the adjustment of physical functions to meet situational demands [63]. The ability of individuals to respond flexibly to changing conditions is a strong predictor for physical and mental health; aberrancies in reactivity may weaken the organism, and are further related to several mental disorders [48]. Reactivity analyses are especially useful in the cardiovascular field. Cardiovascular reactivity can be assessed in terms of stimulus-induced changes in heart rate [5] and constitutes a physiological correlate of emotional activation and cognitive effort [16, 24]. As we plan to induce arousal as well as sadness in our experimental groups, cardiovascular reactivity constitutes an appropriate physiological parameter here.

With the focus on cognitive performance, high emotional arousal leads to deficits in working memory [41, 59, 64], cognitive flexibility [36, 57] and inhibitory control [42, 62]. In inhibitory control, arousal also led to improvement [65, 77]. Research showed that implicit spatial learning as well as sequence learning are disrupted by states of high arousal [43]. Furthermore, working memory performance is impaired in terms of a higher false alarm rate under arousal [14]. As cognitive performance highly depends on current mood and arousal level [43] our goal is to investigate inhibitory control performance in a classic as well as in an emotional antisaccade paradigm in a sample of healthy individuals, where we experimentally manipulate sadness and emotional arousal before task execution. We want to find out whether cardiovascular reactivity to sad or emotionally arousing video stimuli predicts inhibitory control performance.

Based on the connections already shown, one might argue that the confrontation with sad and emotionally arousing stimuli will negatively affect HRV and thus inhibitory control performance. Previous research already examined the influence of mood induction on cognition (e.g., [42, 62]) and autonomic responses (e.g., [66]), executive functioning deficits in mood disorders (e.g., [30]), and the relationship between HRV and affective flexibility [26]. No study to date has investigated the relationship between HRV reactivity, negative affect, and inhibitory control in a psychologically healthy sample, though. Therefore, our research question is concerned whether it is possible to predict inhibitory control performance through cardiovascular reactivity, negative affect and the interaction of both. To address these questions, we are going to investigate the influence of cardiovascular reactivity during sadness and arousal induction on inhibitory control performance in comparison to a neutral control group. Our goal is to show that it is possible to predict performance in the antisaccade paradigm by HRV reactivity. Furthermore, we postulate that individual depression scores will modulate the relationship between HRV reactivity and performance, which has been shown in a study before [34].

## 2. Material and methods

### 2.1. Participants

Ninety volunteers ($M_{\text{age}} = 24.77$, $SD_{\text{age}} = 4.89$) participated with informed consent in the present study, which was conducted in line with the guidelines of the Ethics Committee of the University of the first author. Participants were mainly students as well as acquaintances and co-workers of our lab staff. Thus, we recruited them within seminars and by advertising via Facebook groups and word of mouth. Subjects had to be at least 18 years old, but we set no age limit. They received no compensation for their participation. Exclusion criteria were visual impairment corrected with glasses, any current psychological problem or (psychiatric) drug use. Participants were individually tested in a quiet laboratory. To rule out the presence of mental disorders in our sample, we applied the screening questionnaire from the Diagnostic Expert System for Mental Disorders (DIA-X-SSQ [78]). Moreover, we parallelized experimental groups for age, gender as well as years of education. Within each experimental group, there were 15 men and 15 women. Although we tried to control depressiveness within experimental groups, there was a significant difference between groups in depression scores (measured with the German version of the BDI-II, [28]). Note that those scores are still in the minor range, which means that no clinical depression was present in any of our subjects (see Table 1).

We further controlled some variables that might have an influence on HRV, i.e. BMI (also see Table 1), smoking status, alcohol intake, medication and substance use. None of our subjects used any kind of medication or other substances including alcohol (checked with the DIA-X-SSQ), but 34 subjects were smokers. In addition, we asked our participants to arrive well rested to the lab. Due to bad data quality, the HRV data of two participants had to be excluded, which resulted in a final sample of $N = 88$ for the analysis of HRV data ($M_{\text{age}} = 24.77$, $SD_{\text{age}} = 4.94$).

Ideally, we planned to test a minimum of 66 subjects in three different study groups (22 subjects per group). A power analysis conducted with G*Power [20] determined that a sample size of $N = 65$ is sufficient to detect a medium-sized effect of $f = 0.5$ with a statistical power of $1-\beta = 0.95$ and $\alpha = 0.05$ in an ANOVA model (between subjects, three groups). For the regression analysis, we conducted a separate power analysis, which determined that a sample size of $N = 22$ is sufficient to detect a medium-sized effect of $R^2 = 0.64$ (effect size from [34]) with a statistical power of $1-\beta = 0.95$ and $\alpha = 0.05$ in a hierarchical regression model.

### 2.2. Experimental procedure

At the beginning of the experimental session, subjects gave written informed consent and filled out a set of questionnaires, which comprised of the DIA-X SSQ [78], a mood scale (BFS, [81]) and the BDI-II [28]. Both for the mood scale and BDI-II, participants were asked to rate their mood over the past 2 weeks with focus on the moment of the experimental session (10–15 min, adaptation period). After filling out the questionnaires, we took a 6-minute baseline recording of HRV. After this resting period, subjects were presented with a 90 s video clip (neutral vs. highly arousing vs. sad) including the same physiological recordings as during baseline. Finally, they performed a classic antisaccade task and an emotional antisaccade task; the order of task presentation was

### Table 1

Sample characteristics; mean values and standard deviations as well as statistics of the group comparison.

|                  | Neutral | Arousal | Sadness | F       | p       | $\eta^2$ |
|------------------|---------|---------|---------|---------|---------|----------|
| Age (years)       | 24.83   | 24.03   | 25.27   | 0.48    | 0.618   | 0.011    |
| (5.04)            | (3.21)  | (6.09)  |         |         |         |          |
| Extent of education (years) | 16.97   | 16.75   | 17.08   | 0.16    | 0.852   | 0.004    |
| (2.65)            | (1.95)  | (2.28)  |         |         |         |          |
| BDI-II score      | 2.7     | 3.53    | 5.33    | 4.48    | 0.014   | 0.093    |
| (2.77)            | (4.15)  | (3.39)  |         |         |         |          |
| BMI               | 23.77   | 21.69   | 22.20   | 3.36    | 0.039   | 0.072    |
| (4.43)            | (2.32)  | (2.52)  |         |         |         |          |
| BFS Before        | 45.50   | 48.77   | 46.47   | 1.33    | 0.271   | 0.030    |
| (8.90)            | (6.70)  | (8.08)  |         |         |         |          |
| BFS After         | 45.50   | 43.53   | 45.64   | 0.39    | 0.678   | 0.009    |
| (9.59)            | (9.83)  | (11.49) |         |         |         |          |
| BFS Change        | 0.00    | -5.23   | -0.82   | 3.16    | 0.047   | 0.068    |
| (7.73)            | (7.57)  | (10.41) |         |         |         |          |
balanced across participants. We further applied the mood scale at the end of the experiment to test whether individual mood changed over the course of the experiment. The experimental session took 1.5 h per participant; the testing took place between 9am and 5pm.

2.3. Mood induction procedure

For arousal induction, the application of emotional video clips is a well-established and efficient method (see [23, 25]). Short fragments from existing feature films should induce altered states of arousal and sadness. The chosen scenes in the present study correspond to highly arousing realistic scenes, which are classified as unpredictable and uncontrollable like violent confrontations. As we further strive for measuring physiological reactivity in response to sad scenes, we decided to use film clips instead of other commonly applied techniques for stress induction, e.g. the Montreal Imaging Stress Test or the social-evaluative threat paradigm. Thus, it is possible to induce both arousal and sadness with the same induction technique. The aversive video paradigm has been shown to elicit a strong physical and affective response [49]. Therefore, we intended experimentally inducing three sustained states: (a) a neutral, low arousal state serving as control condition; (b) an aversive, high arousal state; and (c) a sad state. The selected fragment for induction of arousal showed a distressing scene of violence (an aggressive and violent encounter between men during which one is killed by a fire extinguisher from the movie ‘My father and I’). That for induction of sadness showed an explicitly sad scene (a young boy suffering from cancer and his funeral from the movie ‘The fault in our stars’); finally, a social interaction (job application from the movie ‘The intern’) was presented as the control condition. All selected fragments had matched audiovisual characteristics. The arousal scenes have successfully been used in previous studies investigating states of stress (e.g., [29, 43]).

The neutral as well as the sadness video clips have not been used in other studies before. Therefore, we validated them before applying them in our experiment. Both film clips were shown in an online questionnaire (N = 100; Mage = 29.12, SDage = 10.99; 68 females). Then we asked subjects about their current mood after each video clip to check whether those had an impact on subjects’ mood (How do you feel now? How happy / sad are you now?). We then calculated repeated measures ANOVA models, and thus compared the mood values after the neutral video clip with the mood values after the sad video clip and found a significant main effect of video condition. Subjects were significantly sadder (F(1,99) = 157.52, p < 0.01, η²p = 0.61) as well as significantly unhappier (F(1,99) = 78.54, p < 0.01, η²p = 0.44) after watching the sad video clip. The cinematographic material was approved by the Austrian Commission for Media for Youth (JMK) for viewers above 16 years and participants were informed that the scenes they were about to watch might contain offensive or distressing content. Subjects could end their participation in the experiment at any time if desired.

2.4. Antisaccade paradigms

The classic antisaccade paradigm followed suggested methodological standards [1]. Here, a neutral cue (white circle, 1° diameter, line thickness 0.3) was presented in the center of the display for a variable interval of 1000 – 2000 ms (Tobii TX-300 screen-unit; resolution: 1920 × 1080; refresh rate: 60 Hz; viewing distance: 60 cm; Dell Precision T-5610). We kept the viewing distance stable via chin rest. After the variable interval, this cue changed its color to blue or yellow, and moved either 12° left or right from the center. After another 1000 ms, it moved back to the center (no gap or overlap). Each position appeared equally often (randomized order). Subjects were instructed to fixate on the central cue. For blue cues, subjects were asked to perform an antisaccade, which is to fixate on the mirror position of the respective target as fast and accurate as possible. For yellow cues, prosaccades were performed, where subjects had to follow the target as fast as possible and fixate on it. Stimulus presentation was the same as in antisaccades. However, subjects were instructed to follow the stimulus as fast as possible and fixate on it. Thus, pro- and antisaccades were presented in the same block. Instructions were randomized across participants. In addition, we applied the same paradigm with emotional faces (happy, fearful, sad, and neutral). Faces were taken from the FACES database [18]. Non-facial features (e.g., hair, neck) were removed, and faces were resized to a size of 6° x 8°. For each expression, five black and white pictures were used. The stimuli were balanced for gender (50% male faces); faces were Caucasian. Emotions were presented randomized within blocks. Both tasks were preceded by 10 practice trials, the data from which were discarded. The order of the paradigms was randomized across participants. Stimulus presentation was carried out with Open Sesame (Version 3.2.8; [45]). Central performance indicators for pro- and antisaccades in both paradigms were (1) saccade RTs and (2) ERs (saccades in direction of the target cue or corrective saccades). The applicability of those parameters has been validated both in healthy and clinical samples [19]. Saccadic and antisaccadic eye-movements were defined by criteria of amplitude ≥1.5°, velocity ≥30°/s and latency between 100 and 900 ms (e.g. [19]).

For calculating RTs, the beginning of a saccade was used, RTs thus representing the latency between stimulus onset and saccadic initiation. Artifact-affected trials (e.g. blink saccades, measurement failures) were removed from analyses (0.02% of trials in the classic paradigm; 0.02% of trials in the scrambled paradigm; 0.02% of trials in the emotional paradigm). Thus, RTs were calculated only on correct responses (99.08% of trials in the classic paradigm; 99.08% of trials in the scrambled paradigm; 99.08% of trials in the emotional paradigm). Binocular eye-movement registration was carried out using an infrared-based eye-tracking system (Tobii TX-300, Tobii AB, Danderyd, Sweden), which allows for quantification of horizontal eye-movements up to +/-25° with a sampling rate of 300 Hz. Before data acquisition, we calibrated and validated the eye-tracker with a 5-point calibration procedure. Artifact correction and calculation of saccade parameters was performed with a custom developed interactive analysis software (see [31]). This software allowed evaluating the RTs based on a linear regression as well as eye-movement interval classification. Within the tool, the automated detection was adjusted manually and directly exported into excel files for further analyses.

2.5. HRV recordings

In our study, we evaluated HRV in the high-frequency band (HF HRV), which marks the variation in heart rate during a breathing cycle, thus serving as a marker of the parasympathetic nervous system [5, 71]. It is the most commonly applied HRV index in emotion and cognition research (e.g. [30, 79]). In addition, we also had a look at the root mean square of successive differences in ms (RMSSD), as this was done in recent studies [8, 34]. Therefore, we obtained an ECG at a sampling rate of 500 Hz by placing three electrodes at the mid-clavicle, solar plexus and lowest right rib using a variopoort-e data recorder and the biometrics software suite (BiSigma, Freiburg, Germany). ECG recordings were offline filtered using a high pass filter (cut-off frequency 3 Hz). R-R intervals were extracted from ECG by an R-peak detection algorithm using Kubios [69]. For artifact correction, we applied the built-in automatic artifact correction algorithm from Kubios, which applies a time varying threshold to separate ectopic and normal beats within successive R-R intervals. We further screened for artifacts visually, and removed any misplaced or wrongly detected beats. Moreover, a 3 Hz cubic spline interpolation was applied to form equidistantly sampled time series from the IBI data. We then computed HRV parameters. Frequency domain analyses were applied, separating individual R-R intervals into frequency bands, and determining the power of each band. Fast Fourier transformation was further used to transform R-R intervals into
high-frequency bands (HF HRV). Thus, HF HRV was indexed by spectral power density in the frequency range 0.15–0.40 Hz [4]. We selected the smoothing option of the software to remove possible low trends in the data. Moreover, we calculated the RMSSD. For calculating HF HRV scores as well as RTs and ERs of both antisaccade tasks prior to viewing (baseline: 6 min, video viewing: 1.5 min + 3.5 min resting after viewing = 5 min in total).

2.6. Statistical analysis

Due to violation of the normality assumption, we log-transformed HRV scores as well as RTs and ERs of both antisaccade tasks prior to statistical analysis. To check whether there was a significant difference between baseline and video sequence as well as between groups concerning HF HRV and RMSSD, we performed two repeated measures ANOVA models with condition (baseline vs. video sequence) as within-subjects factor and group (neutral vs. arousal vs. sadness) as between-subjects factor. We further computed an ANOVA model to find out if there were performance differences between different emotional stimuli. As we found no meaningful difference between emotions, we then calculated a mean RT as well as ER score across all emotional stimuli. Furthermore, we checked whether experimental groups differed concerning inhibitory control performance. In case of a significant main effect of condition, we report pairwise comparisons, which we adjusted for multiple comparisons (Bonferroni correction). Alpha levels were set at 0.05. Results are reported with original df, corrected p-values as well as 95% confidence intervals. To investigate the relationship between physiological reactivity and inhibitory control performance, we then ran separate hierarchal regression models for each experimental group. We chose separate regression models rather than multilevel analyses due to the premise that sadness and emotional arousal have different influences on the sympathetic nervous system, with the assumption that there would be no significant effect in the neutral group. Centered predictor variables BDI score, BFS score, HF HRV/RMSSD reactivity, and BDI score*HF HRV reactivity/BDI score*RMSSD reactivity were regressed on inhibitory control performance indices, i.e. RTs and ERs in the classic and emotional antisaccade task. Within the sadness group, we recognized several outliers due to no errors or significantly slower RTs using boxplots. We therefore removed those outliers and computed all regression models without them. In order to reduce the influence of heteroscedasticity, robust standard errors were calculated using the heteroscedasticity consistent estimator 3 (HC3; [11]) in the RLM macro for SPSS [10]; standardized coefficients are reported. For all variables included in our regression models, the tolerance values were above 0.1 and the VIF values were below 10, which showed no indication of multicollinearity. Our full datasets are available on Open Science Framework (https://osf.io/9ftj5/). We analyzed our data using SPSS version 25 (IBM).

3. Results

First, we analyzed the mean RTs for different emotional stimuli (neutral, happy, fearful, and sad) and found no main effect of emotion (F[3267] = 0.22, p = 0.88). The same accounted for ERs within the emotional antisaccade task (F[3267] = 0.71, p = 0.55). Therefore, we decided to calculate a mean RT as well as ER score across all emotional stimuli (RT emotional, ER emotional). Experimental groups did not differ concerning their performance in both tasks. Both ERs and RTs in the emotional and classic antisaccade task were similar across the three experimental groups (see table 2). For HF HRV, we found a significant main effect of condition (F[1,84] = 7.06, p < 0.01, ηp2 = 0.078) as well as a significant group effect (F[2,84] = 5.15, p < 0.01, ηp2 = 0.109); the condition by group interaction was not significant (F[2,85] = 1.58, p = 0.213). Pairwise comparisons showed significant differences between the neutral and arousal group (MD = 0.36, p = 0.02) as well as between the arousal and the sadness group (MD = 0.36, p = 0.02). For RMSSD, we found a significant group effect (F[2,85] = 3.25, p = 0.044, ηp2 = 0.071); neither the condition effect nor the interaction reached significance, though (both p’s > 0.09). Here, pairwise comparisons showed marginally significant differences between the sadness and arousal group (MD = 0.13, p = 0.05).

To test whether cardiovascular reactivity in HF HRV and RMSSD during video exposure is related to antisaccade performance in the experimental groups (sadness, arousal), we computed a reactivity score (video sequence - baseline) and then calculated hierarchical regression models for experimental groups separately. As predicted, regression models for the neutral control group failed to reach significance (R2

### Table 2

Performance in the emotional and classic antisaccade paradigm (RTs and ERs) for the three experimental groups (neutral, arousal, and sadness), statistics of the group comparisons.

|                | M(SD) | F(2,87) | p    | ηp2 |
|----------------|-------|---------|------|-----|
| **RT emotional** |       |         |      |     |
| Neutral        | 2.40(0.09) | 1.47 | 0.235 | 0.033 |
| Arousal        | 2.37(0.07) |    |       |     |
| Sadness        | 2.41(0.07) |    |       |     |
| **ER emotional** |       |         |      |     |
| Neutral        | 1.14(0.27) | 1.29 | 0.279 | 0.029 |
| Arousal        | 1.23(0.32) |    |       |     |
| Sadness        | 1.10(0.40) |    |       |     |
| **RT classic** |       |         |      |     |
| Neutral        | 2.44(0.08) | 1.65 | 0.199 | 0.036 |
| Arousal        | 2.41(0.06) |    |       |     |
| Sadness        | 2.42(0.08) |    |       |     |
| **ER classic** |       |         |      |     |
| Neutral        | 1.36(0.28) | 0.03 | 0.971 | 0.001 |
| Arousal        | 1.35(0.29) |    |       |     |
| Sadness        | 1.37(0.30) |    |       |     |

### Table 3

Pearson correlations between reactivity scores, the interaction between BDI scores and reactivity scores, as well as performance indices of the two antisaccade tasks within the sadness group.

|       | 1    | 2    | 3    | 4    | 5    | 6    | 7    | 8    | 9    |
|-------|------|------|------|------|------|------|------|------|------|
| 1. BDI | —    | —    | —    | —    | —    | —    | —    | —    | —    |
| 2. BFS | −0.24| —    | —    | —    | —    | —    | —    | —    | —    |
| 3. HF HRV reactivity | −0.12| 0.22 | —    | —    | —    | —    | —    | —    | —    |
| 4. BDI*HF HRV reactivity | −0.06| 0.09 | 0.86**| —    | —    | —    | —    | —    | —    |
| 5. RMSSD reactivity | −0.10| 0.29 | 0.80**| 0.63**| —    | —    | —    | —    | —    |
| 6. BDI*RMSSD reactivity | −0.03| 0.13 | 0.66**| 0.80**| 0.81**| —    | —    | —    | —    |
| 7. RT classic | 0.12 | 0.28 | 0.52**| 0.34 | 0.45*| 0.22 | —    | —    | —    |
| 8. ER classic | 0.06 | −0.25| −0.38*| −0.40*| −0.37*| −0.40*| −0.32 | —    | —    |
| 9. RT emotional | 0.06 | 0.13 | 0.54**| 0.59**| 0.27 | 0.34 | 0.40*| −0.19| —    |
| 10. ER emotional | 0.16 | −0.11| −0.29 | −0.38*| −0.39*| −0.50*| 0.02 | 0.76**| −0.12 |

Note. N = 29.

*p < 0.05, **p < 0.01."
in the experimental group receiving a sadness induction.

More specifically, the first model encompassing only mood and depression scores did not predict the ER towards emotional faces ($\Delta R^2 = 0.03$, $SE = 0.32$, $p = 0.49$). When adding the interaction of BDI score and RMSSD reactivity the explained variance rose up to 40% ($\Delta R^2 = 0.25$, $SE = 0.25$, $p < 0.01$). For the classic antisaccade task, the first model including mood and BDI score did not predict ERs ($\Delta R^2 = 0.11$, $SE = 0.30$, $p = 0.23$). When adding the RMSSD reactivity score in a second step, 20% of variance in the ER could be explained ($\Delta R^2 = 0.18$, $SE = 0.26$, $p = 0.02$). Adding the interaction of BDI and RMSSD reactivity in a third step, did not result in a significant change of explained variance, though ($\Delta R^2 = 0.07$, $SE = 0.26$, $p = 0.12$).

For RTs, regression models did not yield significant results, neither for HF HRV nor for RMSSD reactivity ($R^2$ ranging from $-0.08$ to $0.12$, all $p's > 0.05$). In summary, our results show that HRV reactivity predicted ERs in the antisaccade task, while current affective state moderated this relationship in the emotional version of the task (see Fig. 1).

4. Discussion

In the present study, we found cardiovascular reactivity during a sad video sequence to be a strong predictor of inhibitory control performance, i.e. ERs in the classic as well as emotional antisaccade paradigm. For the classic antisaccade task, there was a significant negative relationship between cardiovascular reactivity and ERs, while for the emotional version of the task, the interaction between depression and cardiovascular reactivity was an even stronger predictor for ERs. This effect indicates that current affective state strongly influences inhibitory control of emotional face stimuli. Contrary to our hypothesis, these effects were only found in individuals, who were confronted with a sad video sequence before task execution, but not in the arousal group. As predicted, no effects of cardiovascular reactivity on inhibitory control performance have been obtained in the neutral control group.

Previous research has shown a relationship between HRV and...
executive functioning across several domains [21]. In clinical populations, higher HRV has previously been found to be associated with better inhibitory control performance, using the stop signal task [38]. Others have found correlations between baseline HRV and inhibitory control performance in a healthy sample [52]. A recent paper further reported higher baseline HRV to be predictive of set-shifting performance, while negative affect moderated this effect [38]. As not only executive functioning, but also emotion regulation are strongly related to HRV [58, 70], our aim was to show that cardiovascular reactivity during sadness and arousal induction as a marker of emotion regulation is a predictor of cognitive performance in healthy individuals. Furthermore, we included negative affect as a moderator. The present study found evidence of HRV reactivity being significantly associated with inhibitory control performance during two different antisaccade tasks, i.e., ERs in both tasks. Interestingly, this effect was only found for ERs, but not for processing speed in terms of RTs.

Both the Polyvagal Theory [58] and the Neurovisceral Integration Model [70] support cardiac vagal tone to be an index of cognitive control. Parasympathetic influences seem to be essential for the adaptation to changing environmental demands ([58, 70, 72]; Reyes del [61]). The results of our study highlight the fact that cardiovascular reactivity as a response to sad content might be of great importance for predicting cognitive performance, especially when including emotional content. As this effect was specific to the sadness group and could not be found in another group with emotionally arousing video content, the importance of negative affect as a state is emphasized. While an earlier study found effects of trait negative affect on cognitive flexibility in healthy individuals [34], our study shows that not only trait, but also state negative affect might play a key role here. Furthermore, the interaction between depression scores and HRV reactivity underlines this assumption. For the ERs in the emotional antisaccade task, this interaction was the strongest predictor, explaining about 20% of variance in addition to HRV reactivity. There is evidence in the literature of a negative relationship between negative affectivity and HRV in both clinical and healthy samples [6, 17, 35]. While we found no direct associations between negative affect and HRV reactivity in the present study, a significant interaction between BDI scores and HRV reactivity predicted inhibitory control performance in terms of ERs in the emotional antisaccade task. This effect is interesting insofar that it suggests that emotional stimuli play an important role here in the interaction between affective state and HRV reactivity. Other studies showed that the interaction between baseline HRV and negative affect specifically influenced a certain domain of set-shifting, namely extra-dimensional shifting [33, 34].

Contrary to our hypothesis, cardiovascular reactivity during emotional arousal did not predict inhibitory control performance. Although the induction of arousal worked pretty well in terms of a change in HRV as well as in mood, it did not influence performance. This effect was specific to the sadness group and could not be found in another group with emotionally arousing video content, the importance of negative affect as a state is emphasized. While an earlier study found effects of trait negative affect on cognitive flexibility in healthy individuals [34], our study shows that not only trait, but also state negative affect might play a key role here. Furthermore, the interaction between depression scores and HRV reactivity underlines this assumption. For the ERs in the emotional antisaccade task, this interaction was the strongest predictor, explaining about 20% of variance in addition to HRV reactivity. There is evidence in the literature of a negative relationship between negative affectivity and HRV in both clinical and healthy samples [6, 17, 35]. While we found no direct associations between negative affect and HRV reactivity in the present study, a significant interaction between BDI scores and HRV reactivity predicted inhibitory control performance in terms of ERs in the emotional antisaccade task. This effect is interesting insofar that it suggests that emotional stimuli play an important role here in the interaction between affective state and HRV reactivity. Other studies showed that the interaction between baseline HRV and negative affect specifically influenced a certain domain of set-shifting, namely extra-dimensional shifting [33, 34].

Contrary to our hypothesis, cardiovascular reactivity during emotional arousal did not predict inhibitory control performance. Although the induction of arousal worked pretty well in terms of a change in HRV as well as in mood, it did not influence performance. This
stands in contrast to earlier studies showing that high emotional arousal leads to deficits in inhibitory control [42, 62]. Nonetheless, arousal also led to improvement in inhibitory control [65, 77]. As we found no performance differences of any kind between the arousal and the neutral group, our results thus do not confirm results of earlier studies. This might be due to the arousal induction technique, which we applied. Other studies applied a socially evaluated cold pressor test [62] or included a warm signal within the task [77]. From the psychophysiological perspective, cardiovascular reactivity and HRV are suitable biomarkers for emotion regulation, which has been applied in a wide range of studies (e.g. [7, 12, 44]). Another appropriate biomarker for emotional arousal might be electro dermal activity, which constitutes an index of the sympathetic nervous system. It has been applied in dyadic interaction studies (e.g. [13]) and for measuring emotional arousal in response to sad or emotionally distressing films (e.g. [15, 37]).

The significant difference of depression scores between experimental groups might be a reason to assume that this difference might drive our results. Nonetheless, BDI scores in the sadness group were still in the minor range, which makes them clinically not relevant. Moreover, as our models suggest, BDI scores do not have a direct effect on task performance, but interact with cardiovascular reactivity during the sad video sequence. This interaction only has a significant influence on emotional stimuli, but not on regular circle stimuli, which means that negative affect mainly interacts with cardiovascular reactivity when emotional stimuli are present. This moderating effect of BDI scores on the connection between HRV reactivity and inhibitory control in the current study supports the Neurovisceral Integration Model [70], which first considered solely emotion regulation, then was extended to cognitive functioning. The model postulated that brain areas involved in the regulation of cognition and emotion, are also involved in cardiac autonomic activity. Studies have found significant positive associations between baseline HRV and activation of executive brain regions of the prefrontal cortex [73]. Furthermore, hypo-activation in these regions is related to diminished baseline HRV [54, 74]. Cardiac vagal control promotes effective functioning of neural circuits involved in self-regulation to promote cognitive as well as behavioral flexibility. The current study finds that the inability to regulate negative affect may interfere with this relationship. The neurovisceral perspective provides an explanation for this interaction.

4.1. Limitations

As our sample mainly consisted of undergraduate students, which will graduate soon and must apply for a job, the neutral clip displaying a job interview might be arousing to some extent. Therefore, this might not be the best control condition. Moreover, our neutral and sadness video-clips have not been applied or validated in other studies before. Therefore, it is difficult to assume that the sadness induction worked. We found no significant differences concerning mood change between the neutral and sadness group, which points towards the fact that the induction did not work properly. One possible explanation for this might be the fact that we did not ask our subjects to rate their mood right after the sad video clip, but at the end of the experimental session. Nonetheless, when validating the video clips, we found that the sad video clip led to significantly worse mood than the neutral one.

5. Conclusion

In the current study, we investigated whether we could extend findings from a previous study investigating the effects of trait negative affect on HRV and set-shifting [34] to state negative affect and inhibitory control. Mood induction has previously been shown to be impactful in several cognitive functions e.g., memory, executive functioning, and attention [50, 53, 75]. As mentioned by Howell & Hamilton [34], neural indices of this relationship should also be investigated applying EEG or fMRI to investigate the effect of negative affect on the connection between inhibitory control and HRV in the central nervous system.

The current findings support and extend on previous research on the role of negative affect in the connection between cognitive functioning and the autonomic nervous system. We found a strong connection between the ERs in the classic as well as emotional antisaccade paradigm and HRV reactivity. During both antisaccade tasks, there was a significant negative relationship between HRV reactivity and ERs. These effects were only found in individuals, who were confronted with a sad video sequence before task execution. BDI scores moderated the effect of HRV reactivity on ERs in the emotional antisaccade task, indicating that current affective state had a great proportion of influence here. Our results extend the neurovisceral integration model to inhibitory control of emotional stimuli, as well state affect influences; this might further improve the understanding of the relationship between the autonomic nervous system and cognitive functioning.

Declaration of Competing Interest

None.

Data Availability

The data that support the findings of this study are openly available in the open science framework at https://osf.io/9kht5/.

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