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Cardiac vagal tone predicts inhibited attention to fearful faces

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Abstract

The neurovisceral integration model (Thayer & Lane, 2000) proposes that individual differences in heart rate variability (HRV)—an index of cardiac vagal tone—are associated with attentional and emotional self-regulation. In this paper, we demonstrate that individual differences in resting HRV predict the functioning of the inhibition of return (IOR), an inhibitory attentional mechanism highly adaptive to novelty search, in response to affectively significant face cues. As predicted, participants with *lower* HRV exhibited a smaller IOR effect to fearful versus neutral face cues than participants with *higher* HRV, which shows a failure to inhibit attention from affectively significant cues and instigate novelty search. In contrast, participants with *higher* HRV exhibited similar IOR effects to fearful and neutral face cues, which shows an ability to inhibit attention from cues and instigate novelty search. Their ability to inhibit attention was most pronounced to *high* spatial frequency fearful face cues, suggesting that this effect may be mediated by cortical mechanisms. The current research demonstrates that individual differences in HRV predict attentional inhibition, and suggests that successful inhibition and novelty search may be mediated by cortical inhibitory mechanisms among people with high cardiac vagal tone.

Keywords: vagal tone, inhibition of return, emotion

Cardiac vagal tone predicts inhibited attention to fearful faces

Inhibition plays an important role in emotion regulation, which requires selecting an optimal response from a broad behavioral repertoire, and inhibiting less functional responses (Gross, 1998; Thayer & Lane, 2000). In healthy individuals, the attentional system tends to be reflexively drawn to the location of affectively significant stimuli (Klein, 2000). However, when the location is not relevant and the attentional system has time to disengage, people tend to explore new locations (Klein, 2000). This process—termed the inhibition of return—is considered as a “foraging facilitator” that supports adaptive search by preventing one’s attention from going back to previously examined locations (Klein & MacInnes, 1999; Posner & Cohen, 1984; Posner, Rafal, Choate, & Vaughan, 1985; Stoyanova, Pratt, & Anderson, 2007; Sumner, 2006; Taylor & Therrien, 2005). In the current research, we examine whether individual differences in resting heart rate variability—an index of cardiac vagal tone—predict inhibition of return (IOR; Posner, 1980; Thayer & Lane, 2000). Further, we examine the potential neural mechanisms of this relationship by using spatial frequency filtered faces.

The Neurovisceral Integration Model and Heart Rate Variability

According to the neurovisceral integration model (Thayer & Lane, 2000), neural circuits link the heart with cortical and subcortical brain structures via the vagus nerve (see also Benarroch, 1993; Berntson et al., 1997; Ellis & Thayer, 2010; Levy, 1971; see Figure 1). The vagus nerve provides inhibitory inputs to the heart via the parasympathetic nervous system to regulate metabolic responses to the environment (Thayer & Lane, 2000; see also Porges, 2003). This inhibitory cortical-subcortical circuit is critical for self-regulation (Thayer & Lane, 2000; Thayer, Hansen, Saus-Rose, & Johnsen, 2009). Indeed, there is evidence that the failure of inhibition is linked with various psychopathological disorders such as anxiety, depression,

obsessive-compulsive disorder, and attention-deficit disorder (Thayer et al., 2009). Therefore, robust regulation of the heart via the vagus nerve (i.e., high vagal tone) is associated with a nervous system that responds quickly and flexibly to environmental demands (Thayer & Lane, 2000; Thayer et al., 2009). Indeed, cardiac vagal tone is associated with positive affect, more adaptive patterns of emotional responding and self-regulation (see Friedman, 2007; Ingaldsson, Laberg, & Thayer, 2003; Porges, 1991; Thayer & Friedman, 2004; Thayer et al., 2009). Previous findings suggest that increased vagal tone is associated with prosocial attachment (e.g., Porges, 1995, 1998, 2007), resiliency to stress (Fabes & Eisenberg, 1997), and to trait and state experiences of positive emotion (e.g., DiPietro, Porges, & Uhly, 1992; Oveis et al., 2009). Thus, autonomic flexibility and effective self-regulation associated with high vagal tone promotes social well-being and positive emotions which allows for further enhancement in autonomic flexibility and self-regulation (Kok & Fredrickson, 2010).

Heart rate variability (HRV)—which refers to the differences in beat-to-beat alterations in heart rate—is an index of autonomic, particularly parasympathetic (vagal) activity, influences on the heart (Berntson et al., 1997; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996; Thayer and Lane, 2000). Under resting conditions, the heart is predominately under the control of vagal activity (Levy, 1971). Although the intrinsic heart rate is approximately 105 beat per minute, resting heart rate is only 60-80 beats per minute, indicating that the heart is under the strong vagal control (“vagal dominance”; Brownley et al., 2000; Ellis & Thayer, 2010). HRV can be measured in both the time and frequency domains (Task Force, 1996). Time domain methods include the standard deviation of the interbeat intervals (IBI), the mean square of the successive differences in (rMSSD) in IBIs, and the percentage of IBI differences greater than 50 ms (Thayer & Friedman,

2004; Task force, 1996). In the frequency domain methods, the HR time series is decomposed into its frequency components, which then can be described in terms of a spectral density function that provides the distribution of power as a function of frequency (Berntson et al., 1997; Thayer et al, 2004; Task Force, 1996). The high frequency power (HFP) of HRV ranges from 0.15 Hz to 0.4 Hz and is exclusively mediated by the vagus nerves (Thayer & Friedman, 2004; Task force, 1996). The low frequency band ranges from 0.04 to 0.15 and is thought to reflect both sympathetic and vagal modulation on cardiac activity (Berntson et al., 1997; Thayer & Friedman, 2004; Thayer, Friedman, & Borkovec, 1996; Task force, 1996). High frequency HRV power and root mean square successive differences (rMSSD) are considered to effectively quantify vagal activity (Buchheit, Papelier, Laursen , & Ahmaidi, 2007; Thayer et al, 2006; Task Force, 1996).

Over the past few years, several neuroimaging and pharmacological studies have provided evidence that heart rate variability (HRV)—an index of cardiac vagal tone—is associated with the functional capacity of the inhibitory neural circuits (Ahern et al., 2001, Lane et al., 2009).¹ For example, the temporary inactivation of cortical structures caused by injection of intracarotid sodium amobarbital (the “Wada” test) increases heart rate and decreases HRV (Ahern et al, 2001). High HRV is associated with highly functional prefrontal inhibitory activity over subcortical structures, which allows the organism to make situationally adaptive emotional and cognitive responses (Thayer & Lane, 2000; Thayer et al., 2009). By contrast, low HRV is associated with reduced prefrontal inhibitory control over subcortical structures such as the amygdala and the failure to recognize safety signals (Thayer et al., 2009). Converging evidence

¹ Heart rate variability (HRV) refers to the differences in beat-to-beat alterations in heart rate (Berntson et al., 1997; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996; Thayer & Lane, 2000).

shows that low HRV is commonly observed in patients with panic disorder, generalized anxiety disorders, and even children of patients with panic disorder (see Friedman, 2007 for a review; Friedman & Thayer, 1998; Srinivasan, Ashok, Vaz, & Yeragani, 2002). Also, low HRV is associated with reduced attentional control, poor emotional regulation, decreased response to various stimuli, and antisocial behavior in adolescents (Friedman, 2007; Mezzacappa et al., 1997; Thayer & Lane, 2000). For example, Johnsen, et al (2003) showed that dental phobics with lower HRV had more difficulty inhibiting prepotent responses to both incongruent color words and dental fear related words in an emotional Stroop task compared to dental phobics with higher HRV. However, there is little empirical evidence on the relationship between HRV and attentional inhibition to affective stimuli in healthy individuals. Based on the neurovisceral integration model (Thayer & Lane, 2000), we predict that individual differences in HRV may be associated with successful performance in a task that requires inhibiting attention to affectively significant stimuli (see Fox, Russo, & Dutton, 2002).

Emotion-Modulated Inhibition of Return

There is extensive evidence that attention is modulated by affectively significant stimuli (MacLeod, Matthews, & Tata, 1986; Mogg & Bradley, 1999; Ohman, Flykt & Esteves, 2001; see also Vuilleumier, 2005). There is, however, debate about whether affectively significant stimuli (e.g., fearful faces) can modulate IOR (Fox et al., 2002; Lange, Heuer, Reinecke, Becker, & Rinck, 2008; Stoyanova et al., 2007; Verkuil, Brosschot, Putman, & Thayer, 2009). For instance, it makes little sense to disengage attention from stimuli vital for survival (Stoyanova et al., 2007). Yet, several studies suggest that IOR is relatively indifferent to affectively important stimuli (Lange et al., 2008; Stoyanova et al., 2007; Taylor & Therrien, 2005). However, research indicates that individual differences in anxiety may moderate the effects of affectively significant

cues on the IOR effect. Specifically, high-trait anxiety participants showed reduced IOR to angry face cues compared to happy or neutral face cues (Fox et al., 2002; Verkuil et al., 2009). In other words, anxious individuals were unable to disengage from negative stimuli and explore their environment. Because of a lack of inhibitory function, individuals with high anxiety may continue to process fear information, which is associated with anxiety-related attentional biases favoring threatening information (Fox et al., 2002; Verkuil et al., 2009). We reasoned that individual differences in vagal tone might have the opposite effect, such that people high in HRV would be able to disengage from potentially threatening stimuli when they interfered with successful task performance.

The Neural Mechanisms of Inhibition of Return

The neural mechanisms of IOR have been extensively explored. Drawing from multiple sources, Sumner (2006) proposed that there are at least two separate neural mechanisms of IOR: (a) *collicular (retinotectal)* and (b) *cortical* mechanisms. Moreover, collicular and cortical mechanisms likely respond differently to affectively significant stimuli. The retinotectal pathway is a phylogenetically old subcortical pathway that routes directly from the retina to the superior colliculus, a brain region linked to eye movements (Rafal, Henik, & Smith, 1991; Sumner, Nachev, Vora, Husain, & Kennard, 2004; Sumner, 2006). The retinotectal pathway appears sensitive to affectively important stimuli (Stoyanova et al., 2007; Vuilleumier, Armony, Driver, & Dolan., 2003). In contrast, the cortical pathway includes several brain regions, such as the temporo-parietal cortex and anterior cingulate cortex, which are typically associated with attentional control (Mayer, Seidenberg, Dorflinger, & Rao, 2004). The cortical pathway allows for more flexible responses to affectively significant stimuli and may help inhibit attention when it is adaptive (see Cunningham, Zelazo, Packer, & Van Bavel., 2007).

To examine the differential role of the collicular and cortical pathways of the IOR to affectively significant cues, we can take advantage of differences in the types of stimuli these two pathways are designed to process. In particular, we present stimuli in different spatial frequency ranges. The spatial frequency is described by the energy distribution in the scale specified as the number of cycles per degree of visual angle and/or the number of cycles per image (Morrison & Schyns, 2001; Parker, Lishman, & Hughes, 1996). Broad spatial frequency (BSF) images which contain all spatial frequency ranges can be filtered to contain either high spatial frequency (HSF)—above 24 cycles per image—or low spatial frequency (LSF)—below eight cycles per image (Holmes, Green, & Vuilleumier, 2005; Vuilleumier et al., 2003). Neuroimaging studies suggests that LSF fearful face are conveyed via the *retinotectal* pathway to the amygdala and may be associated with *collicular* mechanisms, whereas HSF fearful faces conveyed via the *parvocellular* pathway may be associated with *cortical* mechanisms, including the prefrontal cortex (Livingstone & Hubel, 1988; Merigan & Maunsell, 1993; Vuilleumier et al., 2003; Winston, Vuilleumier, & Dolan, 2003). Therefore, HSF fearful faces conveyed via the *parvocellular* pathway may be associated with the *cortical* mechanism of IOR, including the prefrontal cortex. In contrast, LSF fearful faces which benefit from rapid, but coarse, processing by tapping into the *retinotectal* pathway to the amygdala may be associated with the *collicular* mechanism of IOR (Vuilleumier et al., 2003).

Overview

The neurovisceral integration model has proposed that HRV is closely tied to inhibitory function (Thayer et al., 2009). However, relatively few studies have investigated the relationship between individual differences in resting HRV and inhibitory function using affective stimuli. The goal of the current research is to investigate whether individual differences in HRV predict

the inhibition of return to affectively significant and neutral face cues. According to the neurovisceral integration model (Thayer & Lane, 2000), people with high levels of HRV should have highly functional inhibitory responses to affectively significant stimuli and therefore inhibit attention to stimuli when they may interfere with superior performance. In contrast, people with low levels of HRV should have difficulty inhibiting attention to affectively important stimuli. Thus, people with higher levels of HRV should perform better on an IOR task, even when potential distractors are affectively significant.

Moreover, by using frequency filtered faces in the present study, we seek to examine whether the relationship between HRV and inhibition is related to cortical (high spatial frequency) versus subcortical (low spatial frequency) processes. HSF fearful faces carried via the parvocellular and associated with prefrontal function may be primarily triggering cortically mediated IOR. If so, we would expect that high HRV participants—characterized by highly functional prefrontal activity—may be sensitive to the effect of HSF fearful faces on cortically-mediated IOR. LSF fearful faces which are primarily processed via the retinotectal pathway and connected subcortical structures may be associated with collicularly-mediated IOR. Low HRV participants—characterized by hyperactive subcortical structures—may be better responsive to the effect of LSF fearful faces on collicularly-mediated IOR. Therefore, the ability of people with high HRV to inhibit attention may be more pronounced in response to HSF fearful face cues that are conveyed via the *parvocellular* pathway. In contrast, people with low HRV may have difficulty inhibiting LSF fearful face cues that are conveyed via the *retinotectal* pathway. In addition, similar to highly anxious people, low HRV participants may have difficulty inhibiting BSF fearful faces (Fox et al., 2002; Verkuil et al., 2009).

Methods

Participants

Forty-five undergraduate students (32 females; mean age = 20) participated in the study for partial course credit. All participants were identified as non-smokers and were asked to refrain from alcohol, drug use, and caffeinated beverages for four hours prior to participation (Hansen, Johnsen, & Thayer, 2003). All participants had normal or corrected to normal vision (20/20 visual acuity). People with a history of vision disorders or dysfunctions, neurological or psychiatric disorders, cardiovascular disorders, or medical conditions such as diabetes were excluded from this experiment. Following previous research (Hansen et al., 2003; Thayer, Friedman, Borkovec, Johnsen, & Molina, 2000), participants were divided into two groups—high or low HRV—based on the median split of log high frequency HRV (HF-HRV), which is regarded as reflecting primarily vagal activity, during baseline (Hansen et al., 2003; Task Force, 1996; Thayer and Lane, 2000).² Because males are typically associated with lower HRV than female participants, we set separate criteria for male ($M = 5.97$) and female ($M = 6.44$) participants (Snieder, van Doornen, Boomsma, & Thayer, 2007).

Procedure

All participants were tested individually in a dimly-lit room. They were brought to the lab and three surface electrodes were attached—the negative electrode below the left collar bone, the positive electrode below the right rib cage, and the ground electrode below the left rib cage—to obtain electrocardiographic data. After placement of electrodes and acclimation to the lab during which data quality was checked and task instructions given, resting HRV was recorded for five

² Several previous studies have used a similar approach to compare people with high and low HRV (Hansen, Johnsen, & Thayer, 2003; Hansen, Johnsen, Sollers, Stenvik, & Thayer, 2004; Johnsen et al., 2003; Thayer et al., 2000; Ruiz-Padial, Sollers, Vila, & Thayer, 2003). Nevertheless, repeated-measures multiple regression analyses revealed a similar pattern of results.

minutes, while participants rested quietly. Participants then performed the emotional IOR task (Fox et al., 2002).

Emotional IOR Task. On each trial, a white fixation cross “+” was presented in the middle of the screen and two gray boxes were presented—one on the left and the other on the right of the fixation point (see Figure 2). These boxes measured 6° horizontally and 6° vertically at a viewing distance of 160 cm. The middle of these boxes was located at a distance of 6° from the fixation point. The target that participants had to detect was a black circle, subtending a visual angle of 0.6° across the diameter. The initial fixation display was presented for 1000 ms. Then, a face cue was presented either in a left- or right-side gray box for 300 ms. Then, the initial fixation display reappeared for 200 ms. The central fixation point was enlarged for 300 ms, and then it returned to its original size. After a 160 ms delay, the target circle appeared at the center of either the left- or right-side boxes until the participants responded (or until 2000 ms elapsed). This resulted in a cue-target onset asynchrony (SOA) of 960 ms. There was an intertrial interval of 1000 ms. All stimuli were presented on a black background.

Participants were told that a cue preceding a target did not predict where the target would appear. Therefore, they should ignore the face cues and keep their eyes focused on the fixation point on the center of the screen. They were instructed to indicate where targets appeared by pressing “Z” for a target on the left box or “M” for a target on the right as quickly and accurately as possible. Each participant completed three blocks of trials with different spatial frequencies (BSF, HSF, LSF), and blocks were presented in counterbalanced order. Each block consisted of 12 practice trials and 120 experimental trials (sixty valid and sixty invalid trials) with faces of the same spatial frequency. Fearful and neutral face cues appeared 30 times each on valid and

invalid trials. After each block, participants were allowed a short break. During the task, participants were closely video-monitored.

After the task, participants went through a five minute recovery period. After the recovery period, participants completed the Spielberger State-Trait Anxiety inventory (Spielberger, Gorsuch, & Lushene, 1970) and the Penn State Worry Questionnaire (Meyer, Miller, Metzger, & Borkovec, 1990).

Materials

We selected 132 faces (66 with fearful expressions and 66 with neutral facial expressions; 33 females and 33 males with each expression) from the Karolinska Directed Emotional Faces set (Lundqvist, Flykt, & Ohman, 1998), the NimStim Face Stimulus Set (MacArthur Foundation Research Network on Early Experience and Brain Development), the Cohn-Kanade AU coded Facial Expression Database (Kanade, Cohn, & Tian, 2000) and Pictures of Facial Affect (Ekman & Friesen, 1976). We used 120 faces (60 fearful and 60 neutral faces) for experimental trials and 12 faces for practice trials. All faces were converted to black-and-white (256 grey levels), and contrast and brightness were adjusted to maintain constancy across different face sets. As can be seen in Figure 3, each face was enclosed in a circular frame using Adobe PhotoShop CS3 software (Adobe System, San Jose, CA) to exclude non-facial features (e.g., hair). In order to produce the HSF and LSF stimuli, the original BSF pictures were filtered through a high-pass cut off of > 24 cycles/image for the HSF stimuli and a low-pass cut off of < 6 cycles/image for the LSF stimuli. Average gray-scale values for the BSF, HSF and LSF stimuli were 166, 168, and 158, respectively, and for the neutral and fearful face categories average gray-scale were 164 and 168, respectively, on a 256 gray-level scale. These average gray-scale values did not significantly differ across spatial frequency, $F(2, 357) = 2.02, p = .13, \eta_p^2 = .01$, or

emotional expression, $F(1, 358) = 1.10$, $p = .30$, $\eta_p^2 = .05$. Each stimulus measured 6° horizontally and 6° vertically against a light gray background at a viewing distance of 160 cm and was displayed on a 42 inch high definition plasma television monitor with a resolution of 1024 by 768 pixels.

HRV Measurement

We recorded electrocardiographic activity via a standard 3-electrode (lead II) setup. The electrocardiogram signals, which were sampled at 1000 Hz (Task Force, 1996), passed through Mindware Technology's BioNex 50-3711-02 two-slot mainframe to an Optiplex GX620 personal computer (Pentium D, 2.80 GHz, 2.00 GB RAM) running Mindware Technology's BioLab 1.11 software and BioLab 1.11 received digital triggers (100 ms pulses) via a parallel port connection with a second Optiplex GX620 running E-Prime 1.1.4.1 (Psychology Software Tools, Inc.), which controlled the experiment. The electrocardiogram signals were inspected offline using Mindware Technology's HRV 2.51 software, which allows for detailed examination of the electrocardiogram trace (plotted in mV against time). Successive R spikes (identified by an automatic beat detection algorithm) were visually inspected and edited for any irregularities. However, less than .1% of the data required cleaning. Successive IBIs (in ms) within the baseline period were written in a single text file and analyzed using the Kubios HRV analysis package 2.0 (<http://basimg.uku.fk/biosignal>) through which time and frequency domain indices of the heart period power spectrum were computed. Time domain indices include estimates of root mean square successive difference in milliseconds and heart rate in beats per minute. For spectral analyses, we used autoregressive estimates following the Task Force of the European Society of Cardiology and the North American Society of Pacing Electrophysiology (1996) guidelines. The root mean square successive difference in milliseconds and the frequency

domain measure of high frequency HRV power are regarded as the primary indices of the cardiac vagal tone. rMSSD and high frequency HRV power were significantly correlated, $r(34) = .89, p < .005$. We used high frequency power as the primary index of the cardiac vagal tone in this study and spectral estimates of high frequency power (in milliseconds squared per hertz) were transformed logarithmically (base 10) to normalize the distribution (Ruiz-Padial et al., 2003). Results were identical when rMSSD was used as our index of HRV (data not shown).

Results

Group characteristics

Demographic and psychological characteristics of the two groups are presented in Table 1. Two groups were significantly different in log high frequency HRV, $t(43) = -7.05, p < .01, d = 2.15$, and mean HR, $t(43) = 2.77, p < .01, d = 0.85$. However, the two groups did not differ in PSWQ, $t(43) = -.34, p = .35, d = .1$, trait version of the STAI (STAI-trait), $t(43) = -.66, p = .51, d = .20$, or state version of the STAI (STAI-state) scores, $t(43) = -1.31, p = .20, d = .40$.

Reaction time data

All analyses on Reaction Times (RT) excluded incorrect trials and outliers (Fox et al., 2002). Reaction times of less than 150 ms (anticipatory responding), or more than 1000 ms (delaying responding) were considered outliers (4% of the data). The trimmed RTs were subjected to a 2 (HRV level: high, low) \times 3 (spatial frequency: broad, high, low) \times 2 (cue emotion: fearful, neutral) \times 2 (cue validity: valid, invalid) mixed factorial analysis of variance (ANOVA). All variables were within-subjects except for HRV level. We hypothesized that: (1) high HRV participants should produce the IOR effect to fearful face cues and (2) their ability to inhibit may be even more pronounced in response to HSF fearful faces, which are associated with the cortical mechanism of IOR, (3) low HRV individuals should show difficulty in

producing the IOR to fearful faces, and (4) their ability to inhibit may be even more disrupted in response to LSF fearful faces, which are associated with the subcortical mechanism of IOR.

Replicating previous research (Fox et al., 2002; Stoyanova et al., 2007), there was a significant main effect of cue validity, $F(1, 43) = 28.72, p < .01, \eta_p^2 = .40$, which provides evidence of the IOR effect: slower RTs to detect targets following valid cues ($M = 440$ ms) than invalid cues ($M = 426$ ms). However, the IOR effect was qualified by a three-way interaction between cue validity, cue emotion, and HRV level, $F(1, 43) = 5.81, p = .01, \eta_p^2 = .12$ (see Table 2). In order to break down the interaction, we collapsed the data across different spatial frequency ranges and examined the data for high- and low- HRV groups separately. The RT data were analyzed in a 2 (cue validity: valid and invalid) \times 2 (cue emotion: fearful and neutral) within-subject ANOVA. For *high* HRV participants, the main effect of cue validity was significant, $F(1, 22) = 13.44, p < .01, \eta_p^2 = .38$, indicating slower responses to valid trials ($M = 431$ ms) than invalid trials ($M = 419$ ms), which reflected the typical IOR effect regardless of cue emotion. For *low* HRV participants, there was no main effect of cue validity ($p > .10$). However, the interaction between cue validity and cue emotion was significant, $F(1, 21) = 14.82, p < .01, \eta_p^2 = .41$. Paired *t*-tests revealed that low HRV participants were slower in valid trials ($M = 444$ ms) than invalid trials ($M = 432$ ms) when cues were neutral, $t(21) = 2.54, p < .02, d = 0.78$, which indicates the typical IOR effect. However, there was no difference in RTs on valid ($M = 440$ ms) and invalid trials ($M = 440$ ms) when fearful faces were used as valid, $t(21) = .15, p = .89, d = .05$, which indicates the reduced IOR effect. In sum, high HRV participants produced the IOR effect to fearful as well as neutral faces. In contrast, low HRV participants only produced IOR to neutral faces. Importantly, they did not produce IOR effects to fearful faces.

Therefore, our data suggest that low HRV participants may exhibit impaired inhibitory function in response to affectively important stimuli.

The three-way interaction between HRV level, spatial frequency, and cue validity was significant, $F(2, 86) = 4.48, p < .02, \eta_p^2 = .09$. In order to break down the interaction, we collapsed the RT data across cue emotion and conducted a 2 (cue validity: valid, invalid) \times 2 (HRV level: high, low) mixed factorial ANOVA for broad, high, and low spatial frequency stimuli separately. At broad, high and low spatial frequency ranges, main effects of cue validity were significant, $F(1, 43) = 30.03, p < .01, \eta_p^2 = .41$ for BSF, $F(1, 43) = 10.92, p < .01, \eta_p^2 = .20$ for HSF, and $F(1, 43) = 19.41, p < .01, \eta_p^2 = .31$ for LSF, with slower responses to valid trials ($M = 442$ ms for BSF, $M = 442$ ms for HSF, and $M = 439$ ms for LSF) than invalid trials ($M = 432$ ms for BSF, $M = 432$ ms for HSF, and $M = 427$ ms for LSF). Therefore, all three types of spatial frequency stimuli produced the IOR effect, thereby supporting that parovellular and magnocellular, as well as the retinotectal, pathways may be involved in producing the IOR (Sumner, 2006).

Although we did not observe a four-way interaction, we compared the cue validity effect of fearful and neutral face cues at BSF, HSF, and LSF in high and low HRV participants to test our hypotheses. To simplify the analyses, we obtained indices for the cue validity effect ($CV = RT_{\text{invalid cue}} - RT_{\text{valid cue}}$) for fearful and neutral cues of different spatial frequency separately (Waters, Nitz, Craske, & Johnson, 2007; see Table 2). A positive score indicates faster responses to valid than invalid trials, suggesting that participants maintain attention to where a preceding cue is presented. A negative score indicates faster responses to invalid than valid trials, suggesting that the IOR effect (Posner & Cohen, 1984; Waters et al., 2007). The cue validity effects were compared with Bonferroni correction to control against the accumulation of alpha

error due to multiple comparisons. The level was set at .05, one-tailed, for all analyses because we had a directional hypothesis. As expected, high HRV participants produced significantly more negative cue validity scores ($M = -21$ ms) than low HRV participants ($M = -4$ ms) when cues were HSF fearful, $t(43) = 2.47, p = .017, d = 0.75$, indicating enhanced IOR effects in response to HSF fearful faces (see Figure 4). Furthermore, HRV was negatively correlated with the cue validity effect for HSF fearful face cues, $r = -.32, p = .03$, 2-tailed (see Figure 5). However, the two groups were not different from each other for other cues ($p > .08$). Thus, high HRV participants showed the significantly enhanced IOR effect for HSF fearful cues. Contrary to our hypothesis, there was no HRV difference in the IOR effect for LSF fearful face cues.

Discussion

According to the neurovisceral integration model (Thayer, 2006; Thayer et al., 2009), HRV may be associated with the functional capacity of attentional inhibitory mechanisms vital for cognitive and emotional regulation. In this study, we examined effects of individual differences in resting HRV on the inhibition of return (IOR) to fearful and neutral face cues at different spatial frequency ranges. High HRV participants demonstrated typical IOR effects in the service of superior task performance. Their ability to inhibit attention to affectively significant stimuli was even more pronounced in response to HSF fearful faces. Given that HSF fearful faces are conveyed via the parvocellular pathway to the primary visual cortex and mediated by prefrontal activity (Vuilleumier et al., 2003; Winston et al., 2003), HSF fearful faces may be closely associated with the cortically mediated IOR. Interestingly, our data showed that only high HRV individuals demonstrated IOR for HSF visual cues. This may indicate that HSF cues triggered the cortically mediated IOR so that only high HRV participants—associated with highly functional prefrontal activity—were capable of producing the IOR. In contrast, low HRV

participants—characterized by poor prefrontal activity—could not produce IOR when cues were presented at high spatial frequency. Therefore, high HRV participants may be particularly efficient at generating cortically mediated inhibitory effects to affectively important stimuli. This superior inhibitory ability may be advantageous for inhibiting the unnecessary processing of affectively significant information and initiating novelty search. These results are consistent with other research from our group which showed that individuals with higher resting HRV were more likely to approach novel stimuli and thus receive feedback concerning the affective value of stimuli in their environment (Shook, Pena, Fazio, Sollers, & Thayer, 2007).

In contrast, low HRV participants showed difficulty producing IOR to fearful face cues in general. Low HRV participants have been characterized as having a lack of prefrontal inhibition and hyperactive amygdala (Thayer et al., 2009). This failure to inhibit fearful stimuli in low HRV participants may be dysfunctional from the emotion-regulatory perspective. Once threat is detected, it is important for an organism to make responses appropriate for the situation, which may require them to inhibit further processing of threat information, to make additional visual search to find an escape or additional threat sources, and to decide what actions should be taken based on the sum of the collected information. However, because of insufficient inhibitory function, low HRV individuals may continue to process fear information, which may cause them to prolong defensive behavior patterns and fail to search for an escape or additional threat. Consistent with this perspective, low HRV has been linked with psychiatric disorders such as panic disorder and generalized anxiety disorders (see Friedman, 2007 for a review; Friedman et al., 1993). However in the present study high and low HRV participants did not differ on self-report measures of anxiety or worry. Therefore it is possible that low HRV may be a predisposing factor that may lead to prolonged processing of threat related information. Over

time this may lead to the development of at least certain anxiety disorders (cf. Melzig, Weike, Hamm, & Thayer, 2009).

The impaired ability to inhibit the processing of affectively important stimuli may lead to perseverative cognition, such as chronic worry or rumination (Brosschot, Gerin, & Thayer, 2006; Friedman, 2007; Verkuil et al., 2009). Perseverative cognition can be defined as the constant activation of the cognitive representation of stress-evoking contents, and is considered to be a characteristic of various affective disorders, such as depression, anxiety disorders, and posttraumatic stress disorder (Brosschot, Pieper, & Thayer, 2005; Thayer & Friedman, 2004). Perseverative cognition, often accompanying negative emotion, in turn constantly activates physiological stress defense mechanisms, such as an increase in HR and a decrease in HRV, which can eventually ‘wear and tear’ the system down (Brosschot et al., 2005). A recent review of the literature (Brosschot et al., 2006) provided considerable evidence that perseverative cognition may cause a variety of diseases.

It has been suggested that perseverative cognition may result from a failure of inhibitory prefrontal control over subcortical structures, which can be indexed by HRV (Thayer & Friedman, 2004). A previous study suggested that exposure to threat-related stimuli entails a reduction of vagal tone that continues even after the threat-related stimuli disappears, possibly reflecting a form of perseverative cognition (Johnsen et al., 2003). Our study provides additional evidence that low HRV individuals show impaired ability to inhibit fearful faces, which represents an attentional mechanism for perseverative cognition. In contrast, high HRV participants, who are characterized as having superior inhibitory attentional mechanisms, may be less likely to perseverate negative contents and therefore be better able to instigate new search.

We expected low HRV participants to have particular difficulty inhibiting LSF fearful face cues which would then magnify or prolong the effect of fear information on the amygdala via the collicular-pulvinar pathways (Anderson, Christoff, Anitz, De Rosa, & Gabrieli, 2003; Liddell et al., 2005; Vuilleumier et al., 2003; Vuilleumier, 2005). Although low HRV participants showed reduced IOR effect for LSF fearful face cues compared to high HRV participants, the difference did not reach statistical significance. Timely detection and monitor of affectively significant stimuli are important for survival. LSF fearful cues may trigger the collicularly mediated IOR—closely connected to the amygdala— and people in general may show collicularly mediated IOR that is highly sensitive to and easily modulated by affectively important stimuli (Stoyanova et al., 2007). Additionally, low spatial frequency information is conveyed not only via the retinotectal pathway, but also via the magnocellular pathway (Merigan & Maunsell, 1993; Sumner, 2006; Vuilleumier et al., 2003). Therefore, although LSF fearful information may primarily trigger collicularly mediated IOR and be sensitive to emotional modulation, some LSF information can be conveyed via the magnocellular pathway and be relatively robust to emotional modulation (Vuilleumier et al., 2003).

Conclusion

The current research suggests that individual differences in cardiac vagal tone, indexed by HRV, predict the inhibition of return to affectively significant face cues. People with high cardiac vagal tone are capable of inhibiting attention to affectively significant stimuli. The ability of people with high cardiac vagal to inhibit attention is even more pronounced in response to HSF fearful face cues primarily mediated by the cortical mechanism. In contrast, people with low cardiac vagal tone have difficulty inhibiting attention to affectively significant stimuli. People with both high and low cardiac vagal tone appear to be sensitive to affectively significant

stimuli conveyed via the collicular-pulvinar pathways to the amygdala and produce relatively reduced collicularly mediated inhibitory effect. Thus, successful inhibition to affectively significant stimuli may depend on cortical inhibitory mechanisms among people with high cardiac vagal tone.

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Table 1

Demographic and Psychological characteristics of High and Low HRV participants. Standard Deviations and the Number of Subjects in Parentheses. HRV: Heart rate variability; PSWQ: Penn State Worry Questionnaire; STAI-T: Trait version of the State-Trait Anxiety Inventory; STAI-S: State version of the State-Trait Anxiety Inventory. * $P < .05$.

Variable	HRV Level	
	High (N = 23)	Low (N = 22)
HRV (log high frequency)*	7.0 (0.7)	5.5 (0.7)
Mean heart rate (HR)*	72.2 (10.0)	81.3 (11.9)
Gender ratio (male: female)	7:16	6:16
Age (mean years)	21.0 (4.5)	20.3 (1.0)
PSWQ	51.4 (11.7)	47.4 (17.1)
STAI-T	40.9 (5.7)	39.0 (12.6)
STAI-S	41.0 (8.9)	37.0 (11.5)

Table 2

Mean Correct Reaction Times (in milliseconds) and Mean Cue Validity (in milliseconds) as a Function of Emotion, and Heart Rate Variability (HRV) level. Standard Deviations and the Number of Subjects in Parentheses.

Emotion	Cue Validity	High HRV (N=23)	Low HRV (N=22)
Fearful	Valid	431.5 (51.4)	440.2 (65.2)
	Invalid	419.4 (57.7)	439.8 (61.0)
Neutral	Valid	430.2 (56.4)	443.7 (71.5)
	Invalid	418.96 (57.9)	431.7 (59.3)

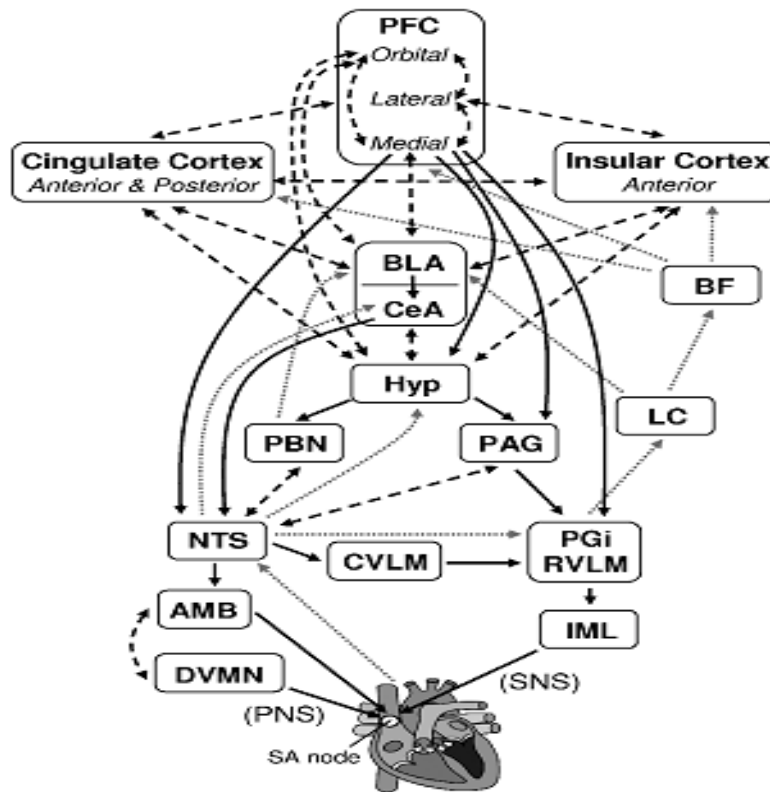


Figure 1. Brain structures associated with the control of heart rate. Solid black arrows indicate efferent pathways to the heart, including right vagus nerve (PNS) and stellate ganglion (SNS) inputs to the SA node. Dotted gray arrows indicate afferent pathways to medullary structures via aortic baroreceptor signals carried through the vagus. Dashed black arrows indicate bidirectional connections. AMB: nucleus ambiguus; BF: basal forebrain; BLA: baso-lateral amygdala; CeA: central nucleus of the amygdala; CVLM: caudal ventrolateral medullary neurons; DVMN: dorsal vagal motor nuclei; Hyp: hypothalamus (lateral and paraventricular); IML: intermediolateral cell column of the spinal cord LC: locus coeruleus; NTS: nucleus of the solitary tract; PAG: periaqueductal gray; PBN: parabrachial nuclei; PFC: prefrontal cortex; PGI: nucleus paragigantocellularis; RVLM: rostral ventrolateral medullary neurons. We adapted and modified the diagram from Ellis and Thayer (2010; with permission of authors and publisher)

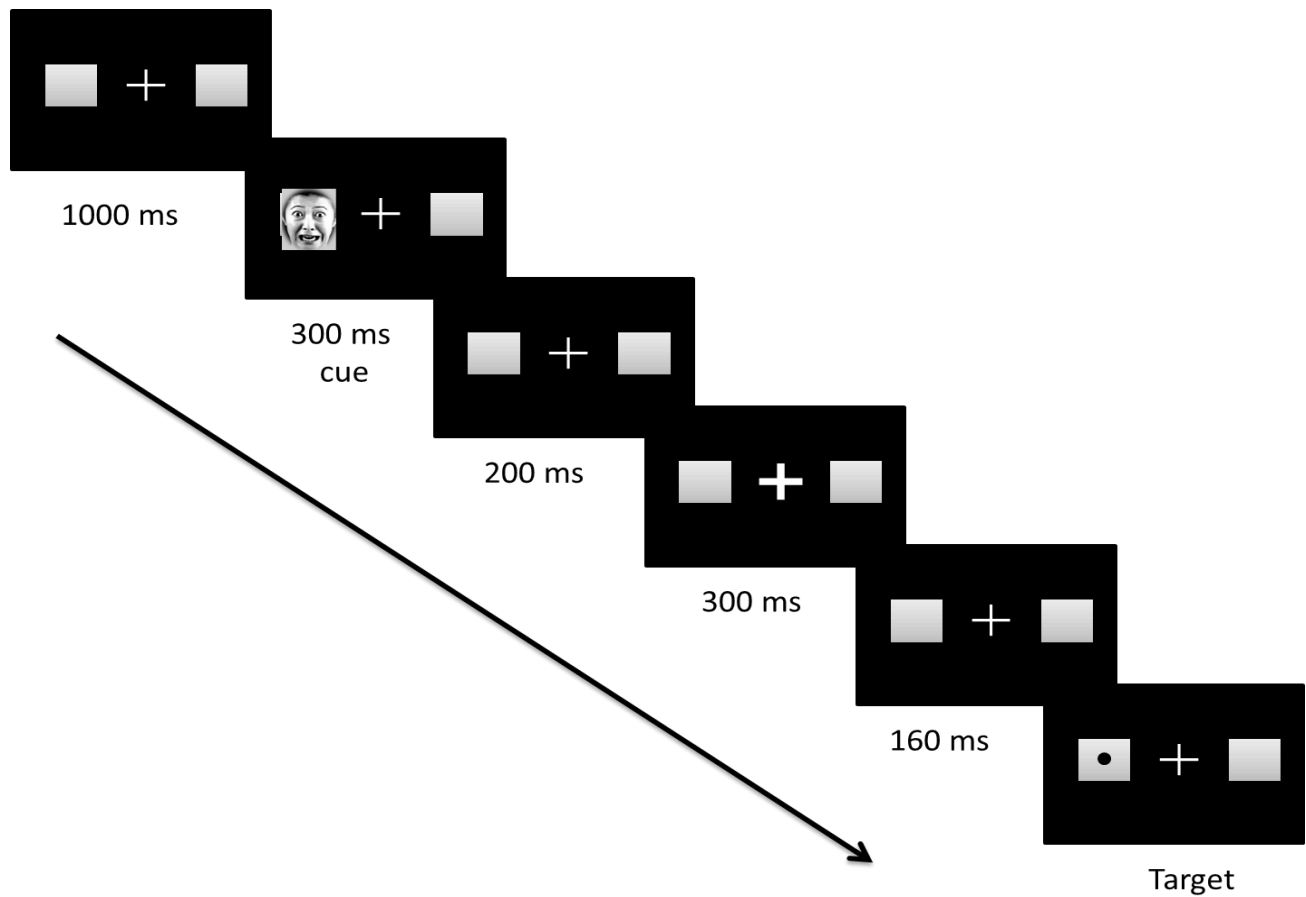


Figure 2. Sample trial of the Inhibition of Return (IOR) task. The cues and targets were equally likely to appear on the right or left of fixation. Cues were either fearful or neutral faces created at broad, high, low spatial frequency and appeared at the beginning of each trial for 300 ms and the initial fixation display appeared for 200 ms. The central fixation point was enlarged for 300 ms, and then returned to its original size for 160 ms. The target circle appeared at the center of either the left or right boxes until the participants responded (or until 2,000 ms elapsed). Stimuli are not drawn to scale.



Figure 3. Example of male (top row) and female (bottom row) stimuli. Normal broad spatial frequency (BSF) fearful and neutral faces (left column), high spatial frequency (HSF) faces (middle column), low spatial frequency (LSF) faces (right column).

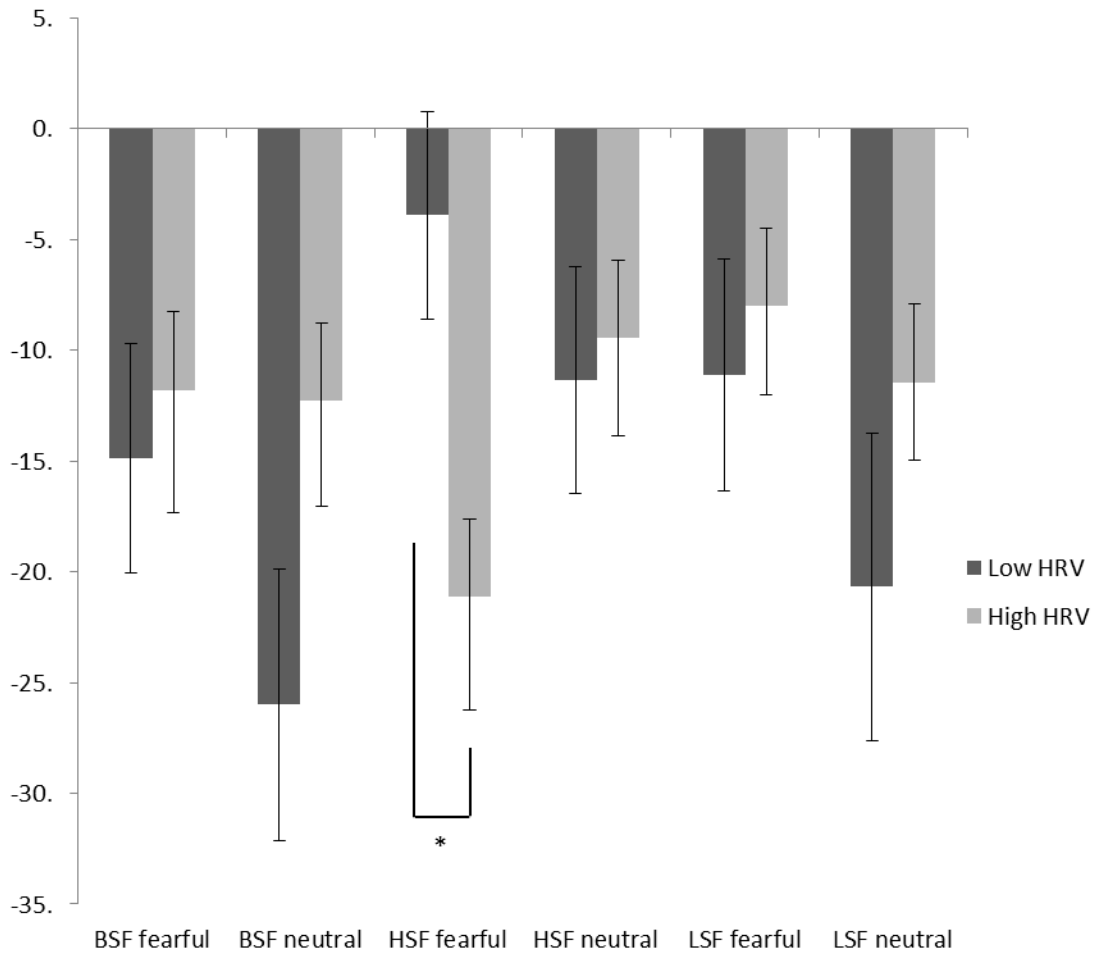


Figure 4. Mean cue validity index (in milliseconds) and standard errors of high and low HRV participants in response to cues of fearful faces at broad spatial frequency (BSF), high spatial frequency (HSF) and low spatial frequency (LSF). Note: * $p \leq .05$.

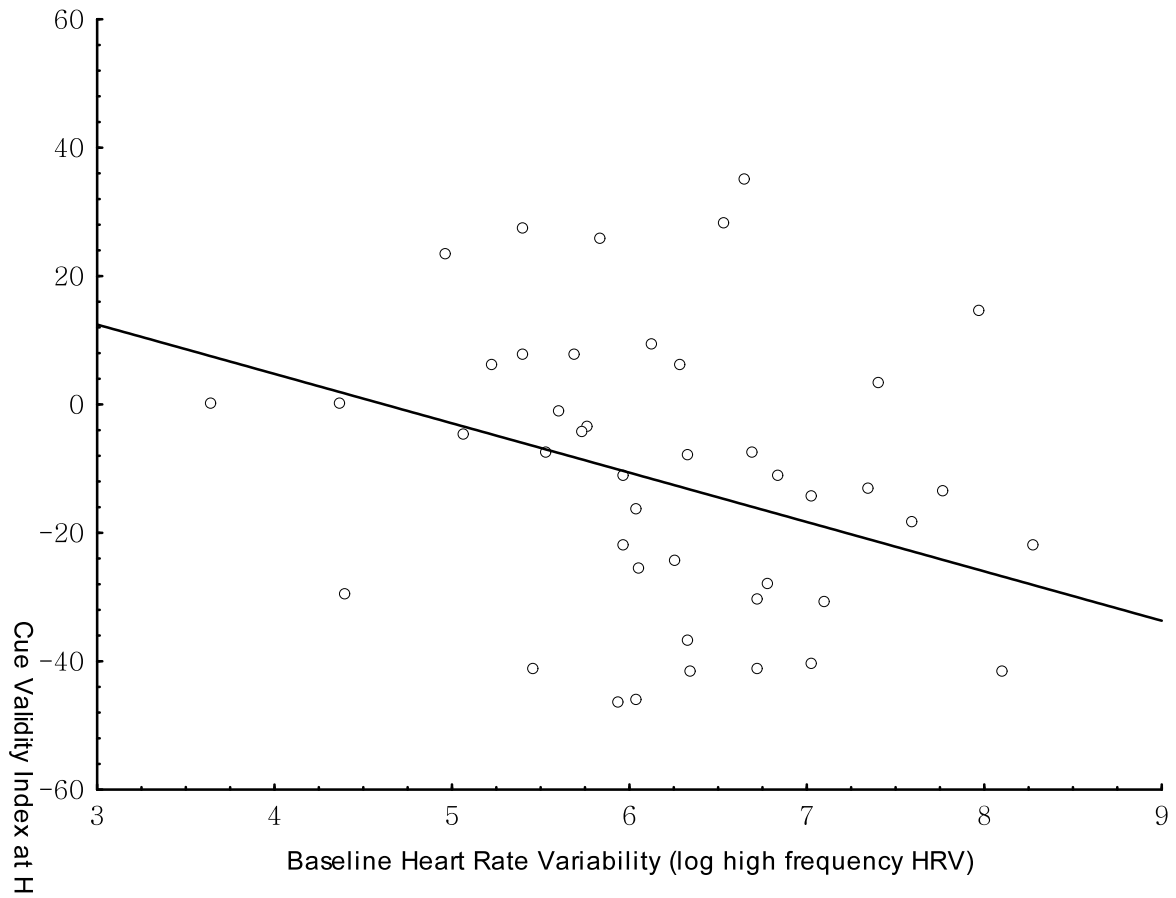


Figure 5. A scatterplot indicating the negative correlation between HRV (*x*-axis) and cue validity index at HSF faces (*y*-axis). $r = -.32$, $p = .03$.