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Cardiac vagal reactivity during relived sadness is predicted by affect intensity and emotional intelligence

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ABSTRACT

Background: The induction of one particular emotion – sadness – has shown two different profiles of autonomic nervous system (ANS) response that are characterized by activation, or withdrawal in cardiac parasympathetic activation. We tested whether individual differences in emotion expression predict cardiac vagal reactivity from baseline to autobiographical sadness induction.
Methods: Respiration sinus arrhythmia (RSA_c) was measured in 56 adults (28 men) asked to relive an episode of sadness. Participants completed an emotional intelligence (EI) test, and a measure of trait affect intensity.
Results: Sadness resulted in cardiac vagal activation with concomitant increase in HR suggestive of parasympathetic and sympathetic co-activation. Individual differences were observed in autonomic reactivity during sadness. Higher scores on the affect intensity measure and the emotional intelligence test predicted greater change in RSA_c during sadness and recovery.
Conclusion: The tendency to experience affect intensely and the ability to perceive emotions predict adaptive physiological regulation during sadness.

1. Introduction

1.1. The autonomic nervous system and emotion

Emotions represent one of the most puzzling aspects of human evolution. Emotions provide socially relevant information about how to engage successfully in interactions with others (Keltner and Kring, 1998). In this sense, emotions constitute an adaptive aspect of human behavior. However, emotions such as sadness and fear are accompanied by distress and can become so all-encompassing and absorbing that they interfere with other adaptive life functions. These seemingly contradictory dimensions of emotion – flexible adaptation versus pathological absorption – are difficult to reconcile.

Special attention in research on emotions has been given to cardiac vagal control – the regulation of heart rate (HR) by parasympathetic influences emanating from the vagus nerve. The vagus nerve exhibits tonic parasympathetic influence on HR that can be measured at rest. In addition, cardiac vagal control is responsive to environmental demands and can be rapidly withdrawn or applied to modulate physiological arousal in an efficient manner (Berntson et al., 1997; Porges et al., 1996). Good cardiac vagal control is reflected in high resting levels and greater magnitude of change from rest to task. Throughout this manuscript, we use cardiac vagal control to refer to resting cardiac parasympathetic influence on HR, and cardiac vagal reactivity to refer to change in cardiac parasympathetic influence from rest to task demand. Where the direction of change is meaningful, we use cardiac vagal activation and withdrawal to refer to an increase, or decrease in parasympathetic activation on HR, respectively.

It has been argued that cardiac vagal reactivity underlies the ability to regulate emotions and respond adaptively to emotional provocation, thereby acting as a protective factor against psychopathology (Beauchaine, 2001; Butler et al., 2006; Denver et al., 2007; Porges et al., 1996; Rottenberg et al., 2005). Porges' Polyvagal Theory (1995, 1997) provides a theoretical basis for understanding the role of cardiac vagal reactivity in emotion regulation. Polyvagal theory is based on the idea that two separate vagal nuclei reside in the brain stem – the nucleus ambiguus and the dorsal motor nucleus. The system controlled by the nucleus ambiguus is a more recently evolved branch, unique to mammals. This system terminates on visceral organs that are important in emotions and their communication such as the heart, larynx, and facial muscles (Porges, 2003). The efferent vagal projection is thought to mediate adjustments to emotional states as well as behavioral responses (Rottenberg, 2007).

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Research has generally been consistent with the idea that cardiac vagal reactivity is involved in effective emotion self-regulation and may be involved in psychopathological conditions that involve particular emotions. Studies have reported that, cardiac vagal withdrawal is a physiological correlate of sustained attention (Porges et al., 1996) and state regulation (Calkins and Keane, 2004; Gentzler et al., 2009), and that cardiac vagal activation is a physiological correlate of emotion resilience (Butler et al., 2006). Moreover, in a meta-analysis of 13 studies meeting rigorous standards for methodology, Rottenberg (2007) reported evidence for a small to medium association between the chronic pathological condition of depressive disorder and diminished cardiac vagal control. Further, greater cardiac vagal withdrawal from baseline to sadness during a sadness inducing video has been found to predict fewer future depressive symptoms among children (Gentzler et al., 2009).

We sought to understand how cardiac parasympathetic influence changes between rest and sadness among healthy individuals, and how this change is moderated by individual differences, as this knowledge may inform future research involving emotion regulation and psychopathology. Sadness is commonly described as a low arousal emotion, typified by behavioral withdrawal that occurs when a goal can no longer be achieved (e.g., the loss of a loved one; Ekman, 1999; Lazarus, 1991). Sadness is accompanied by feelings of loneliness, rejection and self-dissatisfaction, and may help maintain group attachment by eliciting empathic responses (Bowlby, 1980). According to this conceptualization, sadness should result in a shift of the ANS toward parasympathetic activation that is characterized by a reduction in HR and concomitant cardiac vagal activation. Research that has examined cardiac vagal reactivity in sadness, however, has shown decidedly mixed effects. Dywan et al. (2008) reported a strong negative correlation between cardiac vagal control and self-report of the intensity with which people felt sadness. Using autobiographical recall, Marci et al. (2007) found that sadness resulted in cardiac vagal withdrawal relative to a neutral emotion state. By contrast, Kreibig et al. (2007), and Rochman and Diamond (2008) found only non-significant cardiac vagal activation during sadness induced by films or autobiographical recall, respectively. Differences in dependent variables (cardiac vagal control vs. cardiac vagal reactivity) and induction procedures (autobiographical recall vs. films) make it difficult to come to definitive conclusions about the link between sadness and cardiac vagal reactivity. A recent review of ANS activity in emotion (Kreibig, 2010) suggested that sadness is associated with two different profiles of autonomic activity – an activation response and a deactivation response. The activation response of sadness bears a relationship to crying and is characterized by increased respiration rate and HR, and an increase, decrease, or no change in cardiac vagal reactivity. The deactivation response is characterized by decreased cardiac activation and electrodermal activity. The activation and deactivation responses to sadness have been difficult to reconcile. Kreibig (2010) has suggested that the method of induction is an important determinant of which response is likely to occur, such that the activation pattern tends to occur with personal recall tasks; whereas the deactivation pattern tends to occur during sadness inductions that employ films. The relationship between sadness and cardiac vagal reactivity may be further complicated by individual differences in the experience and regulation of emotions.

1.2. Individual differences that influence autonomic function and emotion

Individual differences can be observed in emotions (see Stemmler and Wacker, 2010). One relevant variable related to autonomic reactivity to emotions is the intensity with which one tends to experience emotions. Larsen and Diener (1987) have reviewed evidence for the existence of stable individual differences in emotional intensity. These tendencies can be reliably captured by the affect intensity measure (AIM; Larsen et al., 1986), a self-report questionnaire that assesses the frequency and intensity with which people experience both positive and negative emotions. Individuals high in affect intensity experience stronger emotions, engage in more frequent perspective taking, and are more likely to understand another person's perspective (Larsen, 1987; Larsen et al., 1987). If cardiac vagal reactivity is involved in adaptation to emotions, then one would expect indices of cardiac vagal reactivity to covary with individual differences in affect intensity, since those people who tend to experience emotions intensely may invoke regulatory processes to a greater degree.

Another individual difference that plays an important role in adaptation to emotion and may be linked to cardiac vagal reactivity is emotional intelligence (EI, Salovey and Mayer, 1990). Although definitions vary, EI is best conceptualized as a form of intelligence used to process and benefit from emotions (Mathews et al., 2004; Mayer and Salovey, 2000). Specifically, EI involves the ability to be aware of and perceive emotions, to self-generate emotions to assist with thought, understand emotions and the ways in which they are used, and to regulate emotions to promote personal growth (Mayer and Salovey, 1997). Research has reported that high EI is associated with greater acuity in emotion perception and the tendency to experience affect intensely (Engelberg and Sjoberg, 2004). Further, EI is associated with the self-reported use of adaptive emotion regulation strategies to down-regulate the effects of sadness (Mikolajczak et al., 2008). Furthermore, individuals with higher accuracy on an ability-based measure of affect perception were found to experience fewer problematic emotional and somatic states following daily stressors (Robinson et al., 2012). Cardiac vagal reactivity from rest to task demand is theorized to play an important role in responding adaptively to environmental demands by inhibiting or promoting arousal (Beauchaine, 2001; Denver et al., 2007; Porges et al., 1996; Thayer and Lane, 2000). If affect perception abilities support better emotion regulation (see Robinson et al., 2010) then the ability to perceive, interpret, use, and regulate emotions may modify vagal functioning during emotion elicitation.

1.3. The present study

Analysis of heart rate variability (HRV) offers a method for quantifying cardiac vagal control/reactivity (Berntson et al., 1997). Typically, vagal input to the heart is released during inspiration, causing an increase in HR, and is reinstated during expiration resulting in a reduction in HR. The waxing and waning of HR linked to respiration is known as respiratory sinus arrhythmia (RSA). Decomposition of HRV by spectral analysis of cardiac interbeat intervals, identifies a frequency band between .15 and .50 Hz, that is a commonly accepted index of RSA and, correspondingly, parasympathetic influence on HR (Berntson et al., 1997; Cacioppo and Tassinary, 1990; Grossman and Taylor, 2007). This article will follow the commonly accepted practice of quantifying cardiac vagal control/reactivity with RSA.

We were interested in examining the influence of recalled sadness on cardiac vagal reactivity and its modulation by relevant individual differences. If emotional states, like sadness, affect cardiac vagal reactivity and if cardiac vagal reactivity is linked to flexibility in emotional responding, the ability to sustain attention during emotional episodes, emotional self-regulation, and related processes, then individual differences in emotional characteristics are likely to have some influence on cardiac vagal reactivity.

Three general hypotheses were tested. First, we hypothesized that reliving an autobiographical experience of sadness would result in cardiac vagal activation, as indexed by an increase in RSA amplitude from baseline to sadness. Second, we hypothesized that participants with a proclivity to experience affect intensely would