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Examining the relation between respiratory sinus arrhythmia and depressive symptoms in emerging adults: A longitudinal study

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ABSTRACT

Major depressive disorder (MDD) is a debilitating and prevalent disorder associated with lower quality of life and substantial economic burden. Recently, there has been strong interest in respiratory sinus arrhythmia (RSA) as a biological predictor of later depression. Theoretical work suggests that higher resting RSA indexes physiological flexibility and better emotion regulation whereas lower RSA may mark vulnerability for psychopathology. However, empirical findings have varied. This study examined whether lower resting RSA predicted later depressive symptoms in a sample of healthy young adults across one year ($n = 185$). Results indicate that year one (Y1) resting RSA predicted Y2 depressive symptoms. This finding remained significant when accounting for the stability of RSA and depressive symptoms across both time points and when including trait anxiety, body mass index, and medication use in statistical models. Findings provide further support for RSA as a promising biological marker for understanding and predicting depressive symptoms.

1. Introduction

Major depressive disorder (MDD) is a widespread and debilitating disorder with a prevalence of approximately 16.5% in the American population (Kessler & Bromet, 2013). The costs of this disorder, both at the individual and societal level, are significant. Depressed individuals report cognitive and social impairment and substantially lower quality of life (Kessler, 2003; Richards, 2011). Moreover, there is a significant economic burden associated with depression due to functional impairment at work, absenteeism, and comorbid health problems (e.g., Richards, 2011). Further, individuals who experience depression at a younger age have a poorer prognosis, experience greater interpersonal difficulties, and are at higher risk for suicide (Bramesfeld, Platt, & Schwartz, 2006). Depression is also associated with negative physical health outcomes and often indicates future risk for other forms of psychopathology (Lewinsohn, Solomon, Seeley, & Zeiss, 2000; Aalto-Setala, Marttunen, Tuulio-Henriksson, Poikolainen, & Lonnqvist, 2002). Thus, it is important to identify early markers of depression risk in order to provide more targeted prevention of MDD.

1.1. Risk for depression

In the past few decades, research has focused on risk for depression; however, a majority of these studies have focused on individuals currently experiencing or recovering from depression, thus making the study of potential risk factors difficult. Moreover, many of these studies have emphasized environmental factors including early adversity, acute or chronic stress, and poor social support (Paykel and Tanner, 1976; Roca et al., 2013; Wade & Kendler, 2000). Among the studies that have examined potential biological vulnerabilities for depression, several findings have identified elevated risk among children with a parent who is depressed (Joormann, Eugene, & Gotlib, 2008; Weissman et al., 2006). Recent advances in neuroimaging have also allowed researchers to examine brain structures in depressed adults. Findings from these studies suggest that differences in brain function and structure have potential to predict depression in samples of high-risk individuals (Huang, Gundapuneedi, & Rao, 2012; Whalley et al., 2013).

While there has been substantial progress in identifying risk factors, scientists have continued to examine and identify biomark-

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ers of depression and depressive symptoms (Cowen & Wood, 1991; Gentzler, Rottenberg, Kovacs, George, & Morey, 2012; Rottenberg, Clift, Bolden, & Salomon, 2007; Schmidt, Shelton, & Duman, 2011; Yaroslavsky, Rottenberg, & Kovacs, 2013). Among potential biological markers, there has been a strong interest in respiratory sinus arrhythmia (RSA, known alternatively as cardiac vagal control, vagal tone, or high-frequency heart-rate variability; Rottenberg et al., 2007), which is a measure of parasympathetic influence on cardiac activity via the vagus nerve. Because vagal control cannot be measured non-invasively, RSA is used as a common index of parasympathetic regulation. RSA is quantified as high frequency variability in heart rate across the breathing cycle—vagal regulation decreases during inhalation and heart rate increases; during exhalation vagal inhibition increases and heart rate slows. When an individual is at rest (i.e., low challenge), higher resting RSA marks better conservation of resources and is therefore considered adaptive. Research findings also suggest resting RSA may index individual differences in self-regulation abilities (Beauchaine, 2001; Porges, 1995, 2007). Specifically, higher levels of resting RSA indicate increased physiological flexibility, self-regulation capacity, and ability to adapt when faced with environmental stressors (Porges, 1995, 2007).

1.2. RSA as a biological marker of depression

The interest in RSA as a potential marker of depression follows from a growing literature examining the relation between RSA and several physical and mental health outcomes. For example, high RSA is associated with social competence (Eisenberg et al., 1995), resilience among individuals faced with stressors (Gentzler, Santucci, Kovacs, & Fox, 2009), and the ability to respond flexibly to environmental demands (Porges, 2007). Alternatively, low levels of resting RSA are associated with several disorders and problems, including poor impulse control (Beauchaine, 2001), anxiety (Thayer, Friedman, & Borkovec, 1996), as well as negative health conditions, such as cardiovascular disease (Thayer & Lane, 2007) and sudden cardiac death following myocardial infarction (Peltola et al., 2008).

Several researchers have conducted studies to examine the association between resting RSA and depressive symptoms. However, the precise relation between RSA and depressive symptoms is unclear in the literature. In some studies, low resting RSA is associated with higher depression or more depressive symptoms, as would be expected. In one such study, Gentzler et al. (2012) followed children between the ages of 5–14 at high risk for MDD, defined as having a parent with a childhood-onset mood disorder. The high-risk group did not show the same developmental increase in resting RSA as the low risk group. This suggests that young people at risk for depression may have an atypical trajectory of resting RSA over time. In another study of adults diagnosed with MDD, the authors found the expected association between low RSA and depression. However, the authors were not able to rule out a possible effect of medication (Licht et al., 2008).

In contrast, some researchers have found no resting RSA differences between individuals diagnosed with MDD compared with age and sex matched controls (Lehofer et al., 1997). Rottenberg, Wilhelm, Gross, and Gotlib (2002) also found that for a sample of adults who were diagnosed with MDD, resting RSA was not related to depression severity, but was positively associated with sadness and negatively associated with suicidality (i.e., lower RSA correlated with higher suicidality). Paradoxically, higher levels of resting RSA at year one predicted a more malignant course of depression by year two. Given these inconsistent findings, Rottenberg et al. (2007) conducted a meta-analysis to determine the association between depression and RSA across 13 studies. This meta-analysis revealed a small to moderate effect in both healthy participants ($d = 0.33$) and participants with compromised cardiovascular functioning ($d = 0.28$). In both groups, lower RSA corresponded to higher depression.

One explanation for these inconsistent findings may be the presence of comorbid disorders such as anxiety, medication effects, and physical health (Molgaard, Hermansen, & Bjerregaard, 1994; Rottenberg, 2007). Specifically, anxiety, poor physical health (e.g., obesity), and certain medications are also associated with low resting RSA (Watkins, Grossman, Krishnan, & Blumenthal, 1999), leading researchers to question whether the association between resting RSA and depressive symptoms may be driven by other factors. Additionally, most studies have examined the association between RSA and depressive symptoms in smaller clinical samples with diagnosable depression. Examining only one extreme of the distribution neglects the continuum of depressive symptoms and could produce unexpected associations between these variables (Beauchaine, 2009).

In sum, although findings are inconsistent, researchers have typically studied those who have already been diagnosed with MDD making it unclear whether depressive symptoms or lower resting RSA levels occurred first. Therefore, the predictive value of resting RSA as a biological vulnerability for later depressive symptoms is a topic that has not been fully explored. Given the theory proposed by Porges (1995, 2007) and empirical findings of a negative association between RSA and depression, it is possible that resting RSA may be a viable predictor of later depressive symptoms.

1.3. The current study

In the current study, we examine the association between resting RSA and depressive symptoms in a relatively large sample of young adults over a 12-month time period. In contrast to prior research, we examined resting RSA in a healthy sample consisting predominantly of emerging adults (i.e., not yet diagnosed with depression). This developmental stage is of interest given heightened risk for psychopathology during the young adult years. We also assessed several covariates that may have clouded the relation between RSA and depression previously, including anxiety, body mass index (BMI), and use of psychiatric medication. We hypothesized that lower resting RSA would be associated with higher depressive symptoms scores at both time points (Y1 and Y2). Further, we hypothesized that lower levels of Y1 RSA would predict higher Y2 depressive symptoms. Importantly, because both RSA and depression show high stability over time (Beauchaine, Neuhas, Brenner, & Gatzke-Kopp, 2008; Sloan, Shapiro, Bagiella, Gorman, & Bigger, 1995), we tested a model where Y1 RSA predicted Y2 depressive symptoms while accounting for both stability in RSA and depressive symptoms across the two time points and potential confounding variables.

2. Method

2.1. Participants

At year one, 371 college students were recruited through a psychology department participant pool. Of those, 336 provided consent for future contact and were therefore considered eligible for the longitudinal component of the study. These individuals were contacted approximately 12 months after their Y1 visit and invited to participate in the study follow-up. Due to the presence of non-traditional students at the university, we made no age restrictions for the study. However, as expected, the majority of the participants were young adults (mean age = 25; mode = 23). Given the wide age range (18–64), age was also included as a covariate in statistical models.