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Brooding Rumination and Heart Rate Variability in Women at High and Low Risk for Depression: Group Differences and Moderation by *COMT* Genotype

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There is growing evidence that rumination, perhaps specifically brooding rumination, is a core feature of depression and that it contributes to the development and maintenance of the disorder. A separate line of research has highlighted the role played by heart rate variability (HRV). Both brooding rumination and HRV appear to be driven by disruption in the same neural circuit, heightened amygdala reactivity combined with decreased prefrontal control, and both are highlighted in different units of analysis as reflecting the Research Domain Criteria (RDoC) construct of Loss. However, little is known about the relation among these variables. In the current study, we predicted that higher levels of brooding rumination would be associated with lower levels of HRV and that women at high risk for future depression (i.e., those with a history of past major depressive disorder [MDD]) would exhibit higher levels of brooding and lower levels of HRV. We also examined genetic influences on the variables in this model. We predicted that *COMT* Val158Met genotype, which has been linked to heightened amygdala reactivity and deficits in prefrontal functioning, would be associated with brooding rumination and HRV, particularly among women with a history of past MDD. The results largely supported our hypotheses, providing additional support for relations among the different units of analysis for the Loss construct.

Keywords: major depressive disorder, brooding rumination, heart rate variability, *COMT*, RDoC

According to the response styles theory (Nolen-Hoeksema, 1991), the tendency to ruminate, or passively contemplate the causes and consequences of one's negative mood, contributes to the development and maintenance of depression. Supporting this theory, there is growing evidence that rumination predicts prospective increases in depressive symptoms (e.g., Nolen-Hoeksema & Davis, 1999; Sarin, Abela, & Auerbach, 2005), the onset of new depressive episodes (see Abela & Hankin, 2011; Nolen-Hoeksema, 2000), and the severity and duration of depressive episodes (e.g., Nolen-Hoeksema, 1991; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). Research suggests there are two distinct components of rumination termed brooding and reflection (Treynor, Gonzalez, & Nolen-Hoeksema, 2003). Brooding, described as moody pondering, is thought to be a more maladaptive form of rumination than is reflection, a response style that is more oriented toward reappraisal (e.g., Armey et al., 2009; Gibb, Grassia, Stone, Uhrlass, & McGeary, 2012; Grassia & Gibb, 2008, 2009; Schoofs, Hermans, & Raes, 2010; Treynor et al., 2003).

There is growing evidence that rumination may be a key mechanism underlying other depression-relevant information processing biases, including attentional biases (i.e., difficulty disengaging attention from affectively salient stimuli) and deficits in working memory (i.e., removing task-irrelevant, but affectively salient, information from working memory; for reviews, see Joormann, 2010; Whitmer & Gotlib, 2013). These deficits are thought to be driven by disruptions in the corticolimbic circuit, specifically deficits in the ability of prefrontal areas to effectively downregulate heightened amygdala reactivity (cf. Disner, Beevers, Haigh, & Beck, 2011). Supporting the hypothesis for this circuit-level disruption, a number of studies have now documented the link between rumination and altered activity in amygdala and areas of the prefrontal cortex in both clinical and nonclinical samples using a variety of experimental designs (e.g., Cooney, Joormann, Eugène,

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Dennis, & Gotlib, 2010; Johnson, Nolen-Hoeksema, Mitchell, & Levin, 2009; Kühn, Vanderhasselt, De Raedt, & Gallinat, 2012; Ray et al., 2005; Siegle, Steinhauer, Thase, Stenger, & Carter, 2002; Vanderhasselt et al., 2013). Evidence for heightened amygdala reactivity is consistent across studies (e.g., Cooney et al., 2010; Johnson et al., 2009; Ray et al., 2005; Siegle et al., 2002) but whether prefrontal regions exhibit heightened (Cooney et al., 2010; Vanderhasselt et al., 2013) or reduced (Kühn et al., 2012; Johnson et al., 2009; Ray et al., 2005) activity depends on the specific task used. Taken together, these results suggest that rumination is associated with heightened emotional reactivity and less effective cognitive control.

In a separate line of research, there is evidence that a physiological mechanism implicated in depression risk—low heart rate variability (HRV)—reflects disruptions in the same circuit (Thayer, Åhs, Fredrickson, Sollers, & Wager, 2012). Cardiac vagal control, measured by the high-frequency component of HRV, is thought to reflect capacity for flexible physiological regulation and is influenced by pathways linking the prefrontal cortex (PFC) with inhibitory medullary cardioacceleratory circuits (Thayer & Lane, 2000). This network includes the anterior cingulate cortex, ventromedial PFC, insular cortex, and amygdala. Amygdala activation is inhibited via PFC vagal pathways, which also inhibit sympathoexcitary neurons in the medulla and activate vagal motor neurons responsible for parasympathetic activity (Saha, 2005). Higher levels of HRV are indicative of more flexible adaptation to environmental demands, while lower levels are associated with risk for a variety of disorders including depression (Beauchaine, 2001; Porges, 2007; Rottenberg, 2007; Taylor, 2010). Supporting the hypothesis that HRV provides a physiological index of prefrontal cognitive control over amygdala driven emotional responding, the results of a recent meta-analysis show that low HRV is associated with increased activation in the amygdala and decreased activation in the ventromedial PFC (Thayer et al., 2012). There is also growing evidence that low HRV is associated with deficits in cognitive control (e.g., Hansen, Johnsen, & Thayer, 2003; Johnsen et al., 2003; Park, Vasey, Van Bavel, & Thayer, 2013) and emotion regulation (e.g., Geisler, Vennewald, Kubiak, & Weber, 2010). Because the vagus nerve communicates bidirectionally with PFC and amygdala, HRV may influence the ease in which an individual is able to effectively regulate negative affect. Preliminary evidence has emerged to support this hypothesis. First, pharmacological interventions (Sandrone et al., 1994; Stein & Kleiger, 1999) and behavioral programs (Nolan, Jong, Barry-Bianchi, Tanaka, & Floras, 2008; Stein, Rottman, Kleiger, & Ehsanin, 1996) associated with improving cognitive control have also been shown to improve HRV. In addition, one study showed when naval personnel went through a 8-week "detraining" program designed to lower HRV, their performance on cognitive control tasks declined from pre- to posttest (Hansen, Johnsen, Sollers, Stenvik, & Thayer, 2004).

Despite common underlying neural influences, no study of which we are aware has examined the link between rumination and HRV. This said both rumination and disruptions in the autonomic nervous system are highlighted as key components of the same construct, Loss, within the Negative Valence Systems Domain of the Research Domain Criteria (RdoC). The construct of Loss was defined by the Negative Valence Systems working group as "a state of deprivation of a motivationally significant con-specific, object or situation . . . The response to loss may be episodic (e.g., grief) or sustained" (http://www.nimh.nih.gov/research-priorities/rdoc/negative-valence-systems-workshop.pdf). There is an obvious overlap with diagnoses of depression but the construct of Loss may cut across current diagnostic boundaries. Each of the RdoC constructs is tied to disruption in a specific neural circuit—disruptions in corticolimbic circuits in the case of Loss—and the explicit goal is to develop a fine-grained, integrated understanding of the constructs across various units of analysis including genes, molecules, circuits, physiology, behavior, and self-report. To advance this goal, research is needed that includes measures of a given construct across multiple units of analysis.

The primary aim of the current study, therefore, was to examine individual variability in the degree to which individuals at risk for depression (i.e., those with a past history of major depressive disorder [MDD]) exhibit brooding rumination and low HRV. Given the significant gender differences in levels of brooding rumination and depression (Nolen-Hoeksema et al., 2008), we chose to focus on women with or without a past history of MDD. We focused on women whose MDD had fully remitted to ensure that any of the results obtained were not attributable simply to current between-groups differences in levels of depressive symptoms. We predicted that currently nondepressed individuals at risk for depression in the future (i.e., those with a history of MDD) would exhibit higher current levels of brooding rumination and lower levels of HRV than never depressed controls. We also predicted that levels of brooding rumination and HRV would be significantly (negatively) correlated.

Our secondary goal in this study was to examine genetic influences on these relations. Specifically, although there is a clear link between brooding rumination and depression, there is also a well-known heterogeneity of cognitive profiles in depressed individuals (Rose, Abramson, Hodulik, Halberstadt, & Leff, 1994), and there is growing interest in identifying genetic influences on various forms of cognitive vulnerability to depression (see Gibb, Beevers, & McGeary, 2013), which may help to identify specific subgroups of individuals who are most likely to exhibit disruptions in rumination as well as HRV. When seeking to identify specific genetic influences of cognitive vulnerability, a promising approach is to focus on genes known to affect activity in neural circuits thought to underlie the biases (cf. Gibb et al., 2013). With regard to biases reflecting deficits in cognitive control, dopamine-related genes may be important because of their role in prefrontal regulation. Research has found several dopamine-related polymorphisms to be associated with dysregulated PFC activity, such as deficits in working memory performance (Söderqvist et al., 2012), task-switching (Doll, Hutchison, & Frank, 2011), and emotion regulation (Lataster et al., 2011). The *catechol-O-methyltransferase (COMT)* val^{158}met polymorphism is associated with the degradation of catecholamines such as dopamine. A single-nucleotide polymorphism (SNP) located at codon 158 of the *COMT* gene codes for an amino acid valine (val) to methionine (met) substitution. Val alleles are associated with quicker degradation of dopamine in the PFC than are met alleles (Lotta et al., 1995). The PFC relies mainly on the *COMT* enzyme for dopamine catabolism (Chen et al., 2004), making the *COMT* val^{158}met genotype a relevant candidate gene for examining cognitive vulnerabilities related to inefficient cognitive control. A recent meta-analysis concluded that an increasing number of *COMT* met alleles was

associated with increased activation in prefrontal areas (reduced cognitive efficiency) during emotional tasks (Mier, Kirsch, & Meyer-Lindenberg, 2010). Given this, the *COMT* val^{158}met polymorphism appears to be a promising candidate for examining genetic influences on rumination and HRV. Indeed, *COMT* variation is highlighted within the RdoC matrix for Loss as a key influence at the genetic level (http://www.nimh.nih.gov/research-priorities/rdoc/rdoc-constructs.shtml#loss). Although we examined main effects of *COMT* polymorphism on brooding rumination and HRV, our primary goal was to determine whether *COMT* genotype would help to explain some of the heterogeneity observed in depression. Therefore, we predicted that COMT genotype would moderate the link between depression risk (past history of MDD) and current levels of brooding rumination and HRV.

Method

Participants

Participants in this study were 111 women recruited from the community as part of a larger study of the intergenerational transmission of depression. Women in the depression group (n = 55) were required to have a lifetime history of MDD but to currently be in full remission from the disorder. In addition to a past history of MDD, 31% had a history of a past anxiety disorder, 22% had a past substance abuse disorder, and 7% had a past eating disorder. Women in the control group (n = 56) were required to have no lifetime diagnosis of any *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (*DSM–IV*) mood disorder and no current Axis I diagnosis. Among women in the control group, 4% had a history of a past anxiety disorder, 14% had a past substance abuse disorder, and 4% had a past eating disorder. Exclusion criteria for both groups included symptoms of schizophrenia, organic mental disorder, alcohol or substance dependence within the last 6 months, or history of bipolar disorder. The average age of women in our sample was 40.90 years (SD = 6.77, range = 27–55), 71% were currently married, and 90% were Caucasian. The median annual family income was $55,001 to $60,000, and 47% of women had a bachelor's degree or higher.

Measures. The Structured Clinical Interview for *DSM–IV* Axis I Disorders (SCID-I; First, Spitzer, Gibbon, & Williams, 2002) was used to assess for lifetime histories of *DSM–IV* Axis I disorders. This measure is a widely used diagnostic interview with well-established psychometric properties (Lobbestael, Leurgans, & Arntz, 2011; Zanarini & Frankenburg, 2001). To assess interrater reliability, a subset of 21 SCID interviews was coded by a second interviewer. Interrater reliability for diagnoses of MDD was excellent (κ = 1.0).

Women's levels of rumination were assessed with the Ruminative Response Scale (RRS; Treynor et al., 2003), which consists of two subscales, brooding and reflection. The RRS is a 22-item self-report questionnaire assessing the frequency with which thoughts or behaviors occur when the person is feeling sad, down, or depressed (e.g., "Go someplace alone to think about your feelings"). Reponses are rated on a 4-point Likert-type scale from *almost always* to *almost never*. We focused on the 5-item brooding subscale, which exhibited good internal consistency in the current sample (α = .81).

Women's symptoms of depression were assessed using the Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996). It is a 21-item questionnaire that assesses the severity of current depressive symptoms in the past 2 weeks. The measure has demonstrated good internal consistency and validity in previous research (Dozois, Dobson, & Ahnberg, 1998) and exhibited excellent internal consistency in the current sample (α = .91).

Genomic DNA was collected and isolated from buccal cells/saliva samples using published procedures (Freeman et al., 1997; Lench, Stanier, & Williamson, 1988). The *COMT* gene, which maps to 22q 19951271, has a single nucleotide polymorphism (rs4680) known as the Val158Met polymorphism. The rs4680 SNP (met/met, val/met and met/met) genotype was obtained using the fluorogenic 5′ nuclease (Taqman, Applied Biosystems, Foster City, CA) method using reagents (VIC(tm) and FAM(tm) labeled probes and TaqMan Universal PCR Master Mix without AMPerase UNG) obtained from Applied Biosystems (ABI). Genotype determination was performed using primers purchased from ABI or Integrated DNA Technologies (Coralville, IA). Genotypes were obtained using an ABI Prism 7300 Sequence Detection System using both absolute quantification and allelic discrimination modes (Livak, Flood, Marmaro, Giusti, & Deetz, 1995). Within our sample, 35 women were met/met carriers, 55 were val/met, and 21 were val/val. Genotype frequencies did not vary significantly from Hardy Weinberg Equilibrium (χ^2 = .01, p = .94).

Electrocardiogram (ECG) data were obtained using a Biopac MP150 wireless system and recorded with Acqknowledge v4.2 software (Biopac Systems, Inc., Santa Barbara, CA). ECG was recorded via a standard 3-electrode (lead II) set-up and sampled at 1,000 Hz. During the ECG assessment, participants were instructed to sit quietly and complete questionnaires for 15 min and then were asked to sit without speaking or moving and to breathe regularly for a 5-min rest period. MindWare HRV 3.0.12 was used to inspect, transform, and analyze the ECG signal. Data were visually inspected for artifacts (e.g., an unusual R-R interval), and the experimenter corrected artifacts manually. Women with a suspected or known heart disorder were excluded. After inspection, a power spectral analysis was done sequentially with a fast Fourier transformation. Finally, the power density in the high frequency (HF; .15–.50 Hz) band of HRV was calculated for each 60-s section of the 5-min rest period, as recommended by the Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology (1996).

Procedure

Potential participants were recruited from the community through a variety of means (e.g., TV, newspaper and bus ads, flyers). Participants responding to the recruitment advertisements were initially screened over the phone to determine potential eligibility. Upon arrival at the laboratory at Phase 1, participants were asked to provide informed consent. Next, a research assistant administered the SCID-I. The participants then completed a series of questionnaires and provided buccal cells for DNA analysis. Following this baseline assessment, participants completed follow-up appointments every 6 months for 2 years. At each of these assessments, a research assistant assessed for any depressive episodes that may have occurred in the previous 6 months using

the SCID-I, and participants completed questionnaires. In addition, participants completed an ECG assessment during one of these follow-up appointments.

Results

A preliminary inspection of the data revealed the presence of some missing data, with 0% to 9% missing for any given variable because of participant nonresponse. Little's Missing Completely at Random (MCAR) test was nonsignificant ($\chi^2 = 24.74$, $p = .21$), suggesting that the pattern of missing data was missing at random. Given this, multiple imputation was used to generate 20 imputed datasets, which were used in all subsequent analyses. The results presented reflect the pooled estimates across these data sets. This approach yields more reliable parameter estimates than other methods of dealing with missing data, including single imputation methods (see Schafer & Graham, 2002). Demographic and clinical characteristics of the sample are shown in Table 1. Although the two groups differed significantly in terms of depressive symptoms, the average symptom level in both groups was minimal.

First, we examined the link between women's levels of brooding rumination and HRV. As predicted, higher levels of brooding were significantly correlated with lower levels of HRV, $r = -.19$, $p = .049$. Next, we examined the main and interactive effects of MDD history and *COMT* genotype on brooding rumination and HRV. Both MDD history (yes, no) and *COMT* genotype (met/met, val/met, val/val) were treated as fixed effects in each univariate ANOVA. For brooding, the MDD main effect was significant, $F(1, 105) = 30.03$, $p < .001$, $\eta_p^2 = .22$, showing that women with a history of MDD, compared with those with no history of depressive diagnoses, exhibited higher levels of brooding rumination. Contrary to our hypothesis, however, the *COMT* main effect, $F(2, 105) = .18$, $p = .84$, $\eta_p^2 = .003$, and MDD × *COMT* interaction, $F(2, 105) = .55$, $p = .59$, $\eta_p^2 = .01$, were nonsignificant.

Focusing next on HRV, the main effects of MDD, $F(1, 105) = .44$, $p = .52$, $\eta_p^2 = .004$, and *COMT*, $F(2, 105) = .21$, $p = .82$, $\eta_p^2 = .004$, were nonsignificant. However, we did find a significant MDD × *COMT* interaction, $F(2, 105) = 3.49$, $p = .04$, $\eta_p^2 = .06$. Examining the form of this interaction, we found that, among women homozygous for the *COMT* met allele, those with a history of MDD displayed significantly lower levels of HRV than control women, $M_{MDD} = 5.48$ ($SD = .77$) $M_{control} = 6.39$ ($SD = 1.03$), $F(1, 33) = 7.46$, $p = .01$, $\eta_p^2 = .18$. There were no group differences for val/met heterozygotes, $F(1, 53) = .02$, $p = .91$, $\eta_p^2 < .001$ or val homozygotes, $F(1, 19) = 3.28$, $p = .10$, $\eta_p^2 = .14$.

Table 1
Demographic and Clinical Characteristics of the Sample

Variable	MDD	Control	$r_{effect\ size}$
Age, *M* (*SD*)	40.23 (7.47)	41.55 (6.01)	−.10
% White	85%	95%	−.16
RRS-B, *M* (*SD*)	10.98 (3.03)	8.07 (2.27)	.49*
HRV, *M* (*SD*)	5.80 (1.11)	6.00 (.99)	−.10
COMT, % met/met	24%	39%	.15
BDI-II, *M* (*SD*)	8.27 (6.62)	3.59 (4.50)	.38*

Note. MDD = major depressive disorder; RRS-B = Ruminative Response Scale–Brooding Subscale; HRV = high-frequency heart rate variability; BDI-II =Beck Depression Inventory-II.
* $p \leq .001$.

Discussion

The primary goal of this study was to examine individual variability in the degree to which individuals at risk for depression (i.e., women with a past history of MDD) exhibit vulnerabilities thought to underlie the RDoC Negative Valence Systems construct of Loss. Building theoretically from neural models, we explored multiple units of analysis at the behavioral, physiological, and genetic levels. We predicted that women at risk for depression would display higher levels of brooding rumination and lower levels of HRV, and that these two measures would be related. Finally, we predicted that variation in a specific dopaminergic gene associated with deficits in cognitive control (*COMT* met/met genotype), would help to identify which at-risk women were most likely to exhibit higher levels of brooding rumination and lower levels of HRV. Supporting our hypotheses, we found a significant relation between brooding rumination and HRV such that women reporting higher levels of brooding exhibited lower levels of HRV. In addition, women with a history of MDD displayed significantly greater brooding rumination than women without a history of depression, though this link was independent of the *COMT* genotype. Finally, the link between depression history and low HRV was stronger among women homozygous for the *COMT* met allele than among carriers of the val allele.

The current study provides an example of how the integration of multiple units of analysis thought to underlie one construct can lead to a more nuanced understanding of the construct. For example, the current study demonstrated that brooding and HRV, two measures thought to reflect similar deficits in the same corticolimbic circuit, were significantly correlated with one another. However, despite this relation, different factors predicted how at-risk individuals exhibited vulnerability on both measures. Specifically, a history of past MDD predicted brooding rumination independent of the *COMT* genotype. In contrast, the link between MDD history and low HRV was only significant among women homozygous for the *COMT* met allele.

Our findings introduce interesting questions about the relation between HRV and emotion regulation. Although our study is the first to show a link between brooding rumination and HRV, several other studies have linked low HRV to other forms of maladaptive emotion regulation strategies, such as increased worry (Brosschot, Van Dijk, & Thayer, 2007), increased cognitive reactivity (Beevers, Ellis, & Reid, 2011), and decreased executive emotion regulation, (Geisler et al., 2010). These studies and ours suggest that HRV plays an important role in predicting individual variability in emotion regulation. More important, these findings suggest the potential transdiagnostic nature of both brooding and HRV. In a recent study, McLaughlin and Nolen-Hoeksema (2011) found that rumination accounts for a significant proportion of comorbidity between depression and anxiety in adolescents and adults. Given this, future studies should examine the role of brooding and HRV in other forms of psychopathology characterized by deficits in the corticolimbic circuit (e.g., anxiety and substance abuse disorders).

In addition, the current results provide further evidence for the importance of the *COMT* genotype in examining measures of Loss, particularly among physiological measures. Given HRV's role in

the stress response, it may be that *COMT* is a relevant candidate gene for studies of HRV. Specifically, Porges (2007) posited that HRV is responsible for increasing inhibitory vagal control of the heart, which allows the body to return to homeostasis after stress. However, when individuals are unable to disengage from negative stimuli, inhibition is less likely to occur because the perseverative attention maintains the perception of an ongoing stressor.

Our findings may also have implications for the utility of candidate gene studies of different cognitive vulnerabilities. Findings from a recent twin study suggest that the heritability of brooding rumination is relatively small (h^2 = .22; Moore et al., 2013) though there was some evidence that brooding may mediate the impact of genetic vulnerabilities on depression. Given that genetic influences explain up to 60% of the variability in HRV, candidate gene studies of HRV may be more promising (Wang et al., 2009). In fact, several candidate genes associated with cognitive function have also been associated with HRV, such as the angiotensin-converting enzyme insertion/deletion gene (Gard, 2002) and 5-HTTLPR (Ellis, Beevers, Hixon, & McGeary, 2011). These findings, in addition to ours, suggest the promise of examining genetic influences on HRV.

The current study demonstrated a number of strengths, including the novel, multiple units of analysis approach inspired by the RDoC matrix of Negative Valence Systems construct of Loss, In addition, we sought to integrate two literatures that have until now been separate—brooding rumination and HRV—that appear to share common neural substrates. Finally, the proposed role of *COMT* genotype was based on a strong foundation of previous research and theory. This said, however, there were some limitations that highlight areas for future research. First, our study focused on women and future research is needed to determine the generalizability of the results to men. Second, the study was cross sectional and unable to test the temporal relations between depression history, rumination, and HRV. Future research will be essential to clarify these relations and should include a multiwave assessment of both brooding rumination and HRV to allow a better understanding of potential temporal influences between them as well as how both may influence risk for the onset of future depressive episodes. Third, the current study did not examine any task-based measures of HRV. Future research should examine measures of HRV and HRV reactivity together (see Yaroslavsky, Rottenberg, & Kovacs, 2013). Fourth, there is always the possibility in any genetic association study of an unmeasured genetic or nongenetic third variable accounting for the associations reported (e.g., population stratification or linkage disequilibrium between measured variant and actual functional variant). Future studies, therefore, would benefit from the inclusion of a genomic control. A final limitation is that we focused exclusively on one genetic risk factor—*COMT*—and it is unlikely that the observed genotype effects are unique to *COMT*. It should be noted, however, that our decision to focus on *COMT* was based on evidence of its impact on key neural regions thought to underlie both rumination and HRV. Future research is needed to determine whether other dopaminergic genes also influence rumination or HRV either alone or as part of a broader polygenic aggregate.

In summary, the current results provide important information about individual variability in the degree women at-risk for future depression (i.e., those with a prior history of MDD) exhibit brooding rumination and HRV. Importantly, this study is the first to our knowledge to provide evidence that the link between a past history of MDD and low HRV is moderated by genetic influences and that lower HRV is associated with higher brooding rumination. Therefore, this study provides an examination of two mechanisms, brooding rumination and HRV, that may place some women at a heightened risk for future depressive episodes. If replicated and examined longitudinally, these results could contribute to the development of clinical intervention programs that utilize assessments of cognitive measures, physiology, and genetics to identify at-risk populations. These programs could be modeled after current programs that employ this approach through self-report measures (e.g., Watkins, Baeyens, & Read, 2009; Watkins & Moberly, 2009). In addition to traditional cognitive–behavioral approaches, intervention and prevention programs could focus on methods shown to increase HRV, such as diet, exercise, biofeedback, and stress reduction techniques (Thayer & Lane, 2009). Programs such as these may be the key to reducing the occurrence of depression among at-risk populations.

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