



Perseverate or decenter? Differential effects of metacognition on the relationship between parasympathetic inflexibility and symptoms of depression in a multi-wave study



Jonathan P. Stange^{a,*}, Jessica L. Hamilton^b, David M. Fresco^c, Lauren B. Alloy^b

^a University of Illinois at Chicago, United States

^b Temple University, United States

^c Kent State University, United States

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ABSTRACT

Depression often is characterized by inflexible autonomic and metacognitive processes that interfere with effective self-regulation. However, few studies have integrated these factors to improve the prediction of which individuals are at greatest risk for depression. Among 134 undergraduates, we evaluated whether parasympathetic inflexibility (a lack of reduction in respiratory sinus arrhythmia) in response to a sadness induction involving loss would prospectively predict symptoms of depression across four waves of follow-up over twelve weeks. Furthermore, we evaluated whether metacognitive components of perseverative cognition (PC) and decentering (identified by a principal component analysis) would moderate this relationship in opposite directions. Multilevel modeling demonstrated that the relationship between parasympathetic inflexibility and prospective symptoms of depression was exacerbated by PC, but attenuated by decentering. Furthermore, individuals with parasympathetic inflexibility, PC, and low decentering were at greatest risk for symptoms of depression across follow-up. These results support the utility of integrating autonomic and metacognitive risk factors to identify individuals at risk for depression.

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Major Depressive Disorder (MDD) is the most common mental disorder (Kessler, Chiu, Demler, Merikangas, & Walters, 2005). It is associated with tremendous impairment and considerable comorbidity with other psychiatric conditions, resulting in major personal, economic, and societal costs (Kessler & Wang, 2009, pp. 5–22; Kessler et al., 2006). Given these debilitating effects, research has aimed to identify possible mechanisms and risk factors for MDD that might serve as targets for prevention or treatment (Alloy, Salk, Stange, & Abramson, 2017). Broadly, MDD is associated with a loss of biological and behavioral flexibility (Kashdan & Rottenberg, 2010; Stange, Alloy, & Fresco, in press). Specifically, MDD is characterized by inflexible physiological responses (Bylsma, Salomon, Taylor-Clift, Morris, & Rottenberg, 2014), difficulty disengaging from perseverative thinking processes such as rumination, and

with mentally distancing oneself from one's negative thinking (Bernstein et al., 2015; Fresco et al., 2007a; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). Furthermore, healthy individuals who are inflexible in response to changes in environmental or emotional context also may be susceptible to developing depression (Stange et al., in press). However, not all individuals who are inflexible in one domain necessarily develop depression, suggesting that the identification of risk factors in isolation may be an overly simplistic representation of risk. Thus, research has sought to integrate biological and behavioral factors that confer risk for depression, and examine how they might interact, in the service of improving the prediction of which individuals are at greatest risk for depression.

One biological index of flexibility relevant to depression is parasympathetic nervous system activity, which can facilitate adaptive behavioral and emotional responses to meet contextual demands (Beauchaine, 2001; Kashdan & Rottenberg, 2010; Porges, 2007; Thayer & Lane, 2009). Parasympathetic flexibility can be measured as the extent to which individuals show contextually-appropriate changes in parasympathetic activity across different

* Corresponding author. Center on Depression and Resilience, Department of Psychiatry, University of Illinois at Chicago, 1601 W. Taylor St., Chicago, IL, 60612, United States.

E-mail address: jstange@psych.uic.edu (J.P. Stange).

environmental or emotional cues. One index of parasympathetic flexibility is respiratory sinus arrhythmia (RSA), a measure of variability in heart rate that occurs over the respiration cycle. During periods of rest, the medial prefrontal cortex (mPFC) typically exerts inhibitory control over the amygdala, indirectly enhancing cardiac control via the vagus nerve, and resulting in elevated resting RSA (Thayer & Lane, 2009; Thayer, Åhs, Fredrikson, Sollers, & Wager, 2012). However, during periods of emotional or environmental challenge (e.g., stressors, sadness, attention to salient stimuli), the parasympathetic nervous system typically withdraws its inhibitory control over heart rate, which results in reductions in RSA, allowing the body to mobilize resources needed to flexibly respond to the challenge (Beauchaine, 2001).

RSA has been proposed as a biological index of the capacity for effective emotion regulation (Beauchaine & Thayer, 2015; Thayer et al., 2012). Indeed, extensive literature has documented that lower levels of RSA at rest are associated with maladaptive emotion regulation and MDD (Kemp et al., 2010; Rottenberg, 2007). However, recent work has suggested that RSA reactivity (vagal withdrawal) in response to sadness might be an index of regulatory flexibility that is particularly relevant to understanding depression and depression risk (for recent reviews, see Hamilton & Alloy, 2016; Stange et al., *in press*). Indeed, MDD appears to be characterized by a lack of RSA reactivity in response to sadness (Bylsma et al., 2014; Rottenberg, Clift, Bolden, & Salomon, 2007), and individuals who have lower RSA reactivity (or vagal withdrawal) may be at risk for the onset of symptoms of depression and a poorer course of MDD (Panaite et al., 2016; Rottenberg, Salomon, Gross, & Gotlib, 2005; Stange, Hamilton, Olino, Fresco, & Alloy, 2017a). However, few such prospective studies have been conducted to evaluate the extent to which low RSA reactivity confers risk for future depression. Furthermore, beyond identifying parasympathetic inflexibility as a general risk factor, there is a need to identify contexts – such as other known risk factors – which could work synergistically with the parasympathetic nervous system in risk for depression (e.g., Aldao, 2013; Stange et al., *in press*).

Perseverative cognition (PC; Brosschot, Gerin, & Thayer, 2005; Brosschot, Gerin, & Thayer, 2006; Ottaviani et al., 2016) is one factor that may worsen the role of parasympathetic inflexibility in conferring risk for depression. PC refers to metacognitive capacities characterized by repetitive, negatively-valenced, and self-referential mental activity (Mennin & Fresco, 2013; Ottaviani et al., 2016; Watkins, 2008). Although some forms of self-referential thought can promote concrete processing and adaptive engagement with current circumstances (e.g., Mennin & Fresco, 2013; Morin, 2017), PC involves an abstract level of construal that may exacerbate negative affective states (e.g., Segerstrom, Tsao, Alden, & Craske, 2000; Watkins, 2008). Two exemplars of PC that have received considerable empirical attention are depressive rumination, a way of responding to distress that involves repetitively and passively focusing on symptoms of distress and on the possible causes and consequences of these symptoms (Nolen-Hoeksema et al., 2008), and worry, a relatively uncontrollable and negatively-valenced chain of thoughts and images representing an attempt to engage in mental problem-solving of an issue of uncertain outcome (Borkovec, Robinson, Pruzinsky, & DePree, 1983). Although differing in content (e.g., loss vs. threat) and temporal orientation (e.g., past vs. future orientation), rumination and worry are correlated and have many similarities, including mental activity that is self-referential and perseverative, primarily diffuse and abstract in thinking style, and both have been associated with cognitive inflexibility and difficulty disengaging attention from negative stimuli (e.g., Fresco, Frankel, Mennin, Turk, & Heimberg, 2002; Nolen-Hoeksema et al., 2008; Watkins, 2008). Importantly, PC also confers risk for emotional disorders such as MDD (Abela &

Hankin, 2011; Aldao, Nolen-Hoeksema, & Schweizer, 2010; Marchetti, Koster, Klinger, & Alloy, 2016; Nolen-Hoeksema et al., 2008; Olatunji, Naragon-Gainey, & Wolitzky-Taylor, 2013; Roelofs, Huibers, Peeters, Arntz, & van Os, 2008; Spasojevic & Alloy, 2001; Stange et al., 2016). Furthermore, the cognitive representation of stressors embodied in PC may cause a “fight-or-flight” action response, triggering withdrawal of parasympathetic activity that persists during times when it may not be necessary or adaptive for a response to environmental demands (Brosschot, 2010; Brosschot, Van Dijk, & Thayer, 2007, 2006; LeMoult, Yoon, & Joormann, 2016; Ottaviani, Medea, Lonigro, Tarvainen, & Couyoumdjian, 2015). Therefore, individuals who engage in PC who also demonstrate inflexible parasympathetic responses to sadness might have a “double load” of risk for depression in that both sets of characteristics represent inappropriate responses to contextual demands and may interfere with effective self-regulation.

In contrast with PC, decentering is a form of metacognition defined as the ability to “step outside of one’s immediate experience, thereby changing the very nature of that experience” (Safran & Segal, 1990, p. 117). Decentering refers to a set of characteristics involving three related metacognitive processes (Bernstein et al., 2015): meta-awareness, an awareness of one’s subjective experience in consciousness, such as feeling and thinking (e.g., “I am having the thought that I am stupid” rather than “I am stupid”); disidentification from internal experience, the experience of internal states as being separate from one’s self (e.g., “I am having a feeling of sadness” rather than “I am sad”); and reduced effects of thought content on other mental processes, such as attention and emotion. Interestingly, decentering is inversely associated with PC (Fresco et al., 2007a; Kaiser, Andrews-Hanna, Metcalfe, & Dimidjian, 2015), and experimental studies have demonstrated that low decentering mediates the relationship between rumination and negative thinking in depression (Lo, Ho, Nicky, & Siu, 2014). Decentering is attenuated in individuals with MDD relative to healthy individuals (Fresco et al., 2007a) and predicts better outcomes in psychotherapy (Fresco, Segal, Buis, & Kennedy, 2007b; Teasdale et al., 2002, 2001). As meta-awareness is an integral component of mindfulness and decentering (Bernstein et al., 2015), mindfulness-based treatments such as mindfulness-based cognitive therapy (MBCT; Segal, Williams, & Teasdale, 2012) present one avenue of improving the ability to engage in decentering. Furthermore, mindfulness treatments that target meta-awareness also may improve parasympathetic flexibility (Delgado-Pastor, Perakakis, Subramanya, Telles, & Vila, 2013; Krygier et al., 2013), suggesting that decentering and parasympathetic flexibility may work hand-in-hand to facilitate self-regulation and protect against depression. In contrast, it also is possible that decentering would protect against the depressogenic effects of parasympathetic inflexibility. However, few studies have evaluated contextual factors such as decentering or PC that may moderate the degree to which parasympathetic inflexibility confers risk for depression.

At a neurobiological level, the role of the default mode network (DMN) in parasympathetic flexibility, PC, and decentering provides reason to suspect potentially synergistic relationships between these characteristics. For example, Thayer et al. (2012) have proposed that RSA represents an index of how well top-down appraisals of stimulus threat shape parasympathetic responses to the environment. Modulation of RSA with these appraisals is thought to occur via the mPFC, a key node in the default mode network. In addition to modulating RSA, the mPFC (and the DMN more generally) appears to play a role in processing the degree to which beliefs are self-relevant, and for internally-focused thought that is perceived as self-relevant, such as PC (Whitfield-Gabrieli & Ford, 2012; Marchetti, Koster, Sonuga-Barke, & De Raedt, 2012; Hamilton et al., 2011). Relatedly, a recent neuroanatomical and processing

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