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Physiological Activation as a Mediator in the Relationship Between Perseverative Cognition and Somatic Symptoms

Karly M. Murphy
Seattle Pacific University

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Physiological Activation as a Mediator in the Relationship Between Perseverative

Cognition and Somatic Symptoms

Karly M. Murphy

A dissertation proposal submitted in partial fulfillment

of the requirements for the degree of

Doctor of Philosophy

In

Clinical Psychology

Seattle Pacific University

School of Psychology, Family, & Community

Approved by:

Amy Mezulis, PhD
Associate Professor of Clinical Psychology
Dissertation Chair

Jacob Bentley, PhD
Assistant Professor of Clinical Psychology
Committee Member

Bonnie McGregor, PhD
Executive Director and Founder
Orion Center for Integrative Medicine
Committee Member

Reviewed by:

Amy Mezulis, PhD
Chair, Clinical Psychology

Katy Tangenberg, PhD
Dean, School of Psychology,
Family & Community

Acknowledgements

First and foremost, I would like to thank God for providing me with the ability, knowledge, resources, and support to complete this study.

Next, I would like to express my heartfelt thanks to my advisor, Amy Mezulis, Ph.D. for "adopting" me as one of her students. I am extremely grateful for her support, encouragement, and example. Her mentorship has been an inspiration to me both professionally and personally.

I would like to thank the rest of my dissertation committee: Jacob Bentley, Ph.D. and Bonnie McGregor, Ph.D. for their thoughtful comments and challenging questions. Their feedback and encouragement have greatly enhanced the quality of my research. Additionally, I am extremely grateful to Lynnette Bikos, Ph.D., and Deidre Pereira, Ph.D. for their consultation and guidance regarding data analysis and interpretation.

I am also sincerely thankful for my peers including the students in the Mezulis Lab, my graduate school cohort, and my internship cohort. I am particularly grateful for the SASSY team who assisted in the development and implementation of the study, and spent countless hours collecting and cleaning data.

Finally, I would like to thank my family and friends. My parents have always encouraged me to pursue my passions, and throughout graduate school have supported me in doing so both emotionally and financially. My friends have provided immeasurable encouragement, kindness, and prayer throughout the process of this project. I am particularly grateful for support from the people of Bethany Community Church, especially my small group.

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350 words

Abstract

Somatic symptoms are a significant medical and mental health concern that affects healthy adults and places a significant burden on healthcare systems. The perseverative cognition hypothesis posits that perseverative cognition results in prolonged physiological activation that may be interpreted as somatic symptoms. The purpose of this study was to further examine this hypothesis in a sample of young adults. First, I hypothesized that perseverative cognition would prospectively predict somatic symptoms after controlling for anxiety and depression. Second, I hypothesized that parasympathetic nervous system functioning, measured as respiratory sinus arrhythmia (RSA) and sympathetic nervous system functioning, measured as electrodermal responding (EDR) would mediate this relationship. RSA and EDR were measured before, during, and after a stressor task to provide measures of basal levels, reactivity to the stressor, and recovery from the stressor respectively.

These hypotheses were tested in a sample of 220 young adults. Participants ranged from 18 to 39 years old ($M = 19.63$, $SD = 12.10$), 84.5% of the participants were female, and 65.5% were Caucasian. Perseverative cognition predicted somatic symptoms in the unexpected direction in both hypothesized models (RSA: $\beta = -0.23$, $p = 0.001$; EDR: $\beta = -0.23$, p = 0.074). When anxiety and depression were removed as covariates, somatic symptoms were significantly predicted by perseverative cognition in the expected direction in both models (RSA: $\beta = 0.38$, $p < 0.001$; EDR: $\beta = 0.37$, $p = 0.003$). Neither RSA nor EDR at any time point were significantly related to either perseverative

cognition or somatic symptoms, and no indirect effects were observed. Individual

mediations revealed that RSA recovery significantly predicted somatic symptoms (β =

0.16; p = 0.029) such that individuals whose parasympathetic nervous system re-engaged

following the stressor experienced a higher level of somatic symptoms. Additionally,

individuals who reported higher levels of perseverative cognition experienced greater

increases in EDR during the stressor task (β = 0.17; p = 0.041). Overall, the findings of

this study suggest that perseverative cognition is related to sympathetic nervous system

functioning, while parasympathetic nervous system functioning is related to reporting of

somatic symptoms. Future research directions and clinical implications are discussed.

CHAPTER I

Introduction and Literature Review

Purpose

Somatic symptoms are a significant medical and mental health concern that affect American adults. Somatization has been defined as "a tendency to experience and communicate somatic distress in response to psychosocial stress and to seek medical help for it," (Lipowski, 1988). Somatization may manifest in a number of symptoms, such as back pain, indigestion, and fatigue. A number of epidemiological studies have revealed that even healthy adults report more than one somatic symptom each week (Nimnuan, Hotopf, & Wessely, 2001). However, there are also individuals who report and seek medical attention for multiple somatic symptoms, and these individuals may have health care costs that are up to nine times that of the average primary care patient (Smith, Monson, & Ray, 1986). Thus, somatic symptoms place a significant burden on health care systems, accounting for an estimated one-third of all medical visits (Kroenke & Price, 1993; Kroenke et al., 1994)

Many of the current theories of somatization have drawn from the theoretical basis for hypochondriasis (Barsky & Wyshak, 1990; Kirmayer & Taillefer, 1997) and focus on worry about one's health as a key factor in the development and maintenance of somatic symptoms. These cognitive-perceptual models focus on a vicious cycle in which normal somatic perception is amplified, resulting in a physical interpretation of psychological distress. Some of these models also take into account the social and behavioral contexts that reward and maintain the help-seeking behaviors associated with somatic symptoms (Deary, Chalder, & Sharpe, 2007; Kirmayer & Taillefer, 1997).

More recently, both Brown (2004) and more directly Brosschot, Gerin, and Thayer (2006) have broadened this focus on worry about physical symptoms as a perpetuating factor in somatic symptoms to include all perseverative cognition (e.g. worry and rumination). Brosschot, Gerin, and Thayer (2006) posit that the key component in the development of somatic symptoms is not the act of worrying about health specifically, but the perseverative nature of these thoughts that result in prolonged physiological activation that may be interpreted as somatic symptoms. Thus, it may be that perseverative cognition results in somatic symptoms through the mechanism of prolonged physiological activation.

The purpose of the current study is to further examine the perseverative cognition hypothesis proposed by Brosschot, Gerin, and Thayer (2006) in a sample of young adults. First, I hypothesized that perseverative cognition would prospectively predict somatic symptoms. Second, I hypothesized that physiological activation would mediate this relationship. Specifically I examined the mediating role of respiratory sinus arrhythmia (RSA) and electrodermal responding (EDR) in response to induced stress. In the below sections I review the historical and theoretical underpinnings of somatic symptoms, as well as contemporary integrative models of somatic symptoms. I then discuss the theoretical and empirical basis for the hypothesized relationship between perseverative cognition and somatic symptoms. Finally, I discuss RSA and EDR as potential mediators of this relationship.

Conceptualization of Somatic Symptoms

Though earlier definitions of somatization have focused on medically unexplained symptoms (American Psychiatric Association, 2000), the DSM-5 considers somatic

symptoms to be physical manifestations of psychological distress that may or may not be associated with a medical condition (American Psychiatric Association, 2013). Current diagnostic criteria for Somatic Symptom Disorder are (a) one or more somatic symptoms that are distressing and/or result in a significant disruption in daily life; (b) excessive thoughts, feelings, and/or behaviors related to these somatic symptoms or associated health concerns; and (c) the state of being symptomatic persists for at least six months (although any one symptom may not be continuously present). As Somatic Symptom Disorder is a new diagnosis, its prevalence is unknown to date. However, according to the DSM-5 (American Psychiatric Association, 2013), it is likely that the prevalence will be higher than that of the more restrictive DSM-IV diagnosis of somatization disorder, which had a prevalence rate of less than 1%. Similarly, the prevalence of this disorder will likely be lower than that of undifferentiated somatoform disorder, which had a prevalence rate of 19%. Thus the prevalence of somatic symptoms disorder in the general population has been estimated to be approximately 5-7%. Additionally, gender differences in somatic symptom disorder have not yet be studied because of the novelty of this diagnosis. It has previously been demonstrated that women tend to report higher levels of somatic symptoms, so it is likely that the prevalence of somatic symptom disorder will also be higher in females. Additional risk factors for somatic symptom disorder may include trait negative affectivity, low levels of education, low socioeconomic status, and the experience of recent stressful life events (American Psychiatric Association, 2013).

Somatic symptoms such as back pain, indigestion, and fatigue are common and associated with distress and impairment even in sub-clinical populations. In an

epidemiological study of somatization, Hiller, Reif, and Brahler (2006) found that 81.6%
of a representative German sample reported at least one symptom causing at least mild
impairment and 22.1% reported at least one symptom causing severe impairment. This
corroborates the findings of other research on the prevalence and severity of somatic
symptoms in non-clinical populations (Janca, Isaac, & Ventouras, 2006; Rief, Hiller, &
Margraf, 1998). In primary care settings, somatic symptoms account for a quarter to half
of all patient visits, suggesting impairment (Barsky, 1995; Court, 1995). Patients with
somatic symptoms utilize greater than average amounts of health care services, resulting
in substantial direct and indirect costs (Konnopka et al., 2013).

Theoretical Framework of Somatic Symptoms

  Introduction. Psychologists and medical practitioners have recognized somatic
symptoms throughout history. The earliest systematic account of somatic symptoms was
dissociation theory as developed by Janet (1889, 1907) which was later extended into the
concept of conversion (Strachey et al., 1955). More recently, however, contemporary
theories have turned their attention to cognitive-behavioral factors that result in
somatization (Barsky & Wyshak, 1990; Brosschot et al., 2006; Brown, 2004; Kirmayer &
Taillefer, 1997). In the following sections I will first review early theoretical
understandings of somatic symptoms. Next, I will provide an overview of current
integrative models of somatic symptoms. Finally, I will describe the perseverative
cognition hypothesis (Brosschot et al., 2006) a portion of which my dissertation will
examine.

  Early theories of somatic symptoms. Psychologists and medical practitioners
have recognized somatic symptoms throughout history. One of the most well-know and

accepted understandings of dissociation is Freud's theory of conversion. Conversion theory posits that somatic symptoms are a defense against negative emotions activated when the brain attempts to regulate negative affect through suppression of traumatic memories. Though suppression initially protects the individual from overwhelming negative affect, the neural energy associated with this negative affect is not appropriately released, and instead "converted" into somatic symptoms. Thus, an individual can express psychological distress by developing somatic symptoms without conscious awareness of the negative affect, reducing anxiety. Freud also noted that conversion also allowed for secondary gains, such as attention and avoidance of work (Roelofs & Spinhoven, 2007; Strachey et al., 1955).

Though this model has been widely accepted in much of the medical practice, there have not been any systematic studies of conversion, and this model does not seem to adequately account for the findings of the current literature on somatic symptoms (Roelofs & Spinhoven, 2007). Two studies have raised the possibility that somatic symptoms may resolve the experience of negative affect, but their validity has been called into question (Bishop Jr. & Torch, 1979; Raskin, Talbott, & Meyerson, 1966; Roelofs & Spinhoven, 2007). Additionally, if this were the case, we would expect to find lower levels of negative affect and psychological distress among individuals with high levels of somatic symptoms; in fact, several studies have indicated that the opposite is true – somatic symptoms are associated with elevated levels of distress (Kroenke, 2003; Simon, VonKorff, Piccinelli, Fullerton, & Ormel, 1999).

Though Freud's model may be the most well-known early theory of somatic symptoms, the earliest systematic account of somatic symptoms was dissociation theory

as developed by Janet (1889, 1907). Janet considered dissociation to be a state in which

mental operations that are normally integrated with other mental functions operate

outside the sphere of conscious awareness and memory. This definition of dissociation

was based on his observations of individuals with "hysteria," a broad class of

psychopathologies including dissociative disorders, conversion disorders, somatic

symptom disorders, borderline personality disorder, and post-traumatic stress disorder. At

the time, it had already been established that hysteria often followed stressful life events.

Thus, Janus' understanding of dissociation focused on the role of dissociation in

traumatically induced disorders (Roelofs & Spinhoven, 2007).

Janus posited that when individuals experience a traumatic event, they also

experience a spontaneous narrowing of attention resulting in the development of somatic

symptoms through two mechanisms. First, the spontaneous attentional narrowing limits

the number of sensory channels that can be attended to simultaneously, resulting in a loss

of deliberate attentional control over channels that are not attended to. Over time, this

attention style may become a habitual pattern, and though information is processed by the

unattended to channel, this occurs outside of conscious awareness. This dissociated

information processing results in negative dissociation symptoms, including memory

loss, loss of motor control, and loss of somatosensory awareness. Second, according to

Janus, attentional narrowing makes it unlikely that the individual will have full awareness

of the traumatic event, preventing the integration of new memories into the individuals'

existing personal knowledge and sense of identity. Thus, individuals have little control

over the activation of these memories resulting in positive dissociation symptoms,

including re-experiencing, sensory distortions, and pain (Roelofs & Spinhoven, 2007).

Though there is strong empirical evidence to suggest that traumatic experiences may induce dissociative experiences, few studies have examined this relationship among individuals with somatic symptoms (Gershuny & Thayer, 1999; Roelofs & Spinhoven, 2007). There is research that suggests that there tends to be correlation between dissociative experiences and somatic symptoms (R. J. Brown, Schrag, & Trimble, 2005; Nijenhuis, Spinhoven, van Dyck, van der Hart, & Vanderlinden, 1998) but few studies have examined dissociation as an explanatory factor in the relationship between trauma and somatic symptoms (Roelofs & Spinhoven, 2007). In fact, Pribor, Yutzy, Dean, and Wetzel (1993) found that the relationship between dissociation and somatic symptoms disappeared after controlling for trauma. Thus, it seems that trauma, rather than the tendency toward dissociation that may arise from it, is a more important vulnerability with regard to the development of somatic symptoms.

There are a number of studies that have found that a history of trauma such as childhood physical or sexual abuse is a risk factor for somatic symptoms (Fiddler, Jackson, Kapur, Wells, & Creed, 2004; Kimerling & Calhoun, 1994; Kugler, Bloom, Kaercher, Truax, & Storch, 2012; Morrison, 1989; Petkus, Gum, King-Kallimanis, & Wetherell, 2009; Sack, Lahmann, Jaeger, & Henningsen, 2007; Stein et al., 2004). In a review of this literature, Roelofs & Spinhoven (2007) concluded that in subgroups of individuals with clinical levels of somatic symptoms, there are significantly greater levels of abuse. However, all of these studies are retrospective in nature, making it difficult to draw any conclusions about causality. Despite this limitation, it will be important to control for the effect of trauma history on somatic symptoms in order to disentangle the relationship of more proximal variables to the development and maintenance of somatic

symptoms.

Integrative models of somatic symptoms. While nearly all theories of somatic symptoms suggest that they represent emotional distress experienced and expressed as physical symptoms, contemporary theories have examined the mechanisms by which individuals may translate stress or emotional distress into physical symptoms. Barsky & Wyshak (1990) developed one of the earliest integrative models of somatic symptoms, and it has since been quite influential. Their model was first applied to hypochondriasis, and focuses on cognition and perception as a key mechanism. Similar to models of panic and depression, they note that individuals who are prone to hypochondriasis and somatic symptoms amplify normal or benign physical sensations and believe that they are indicative of serious disease. Because the patient is anxious about the possibility of having a serious disease, he or she begins to focus additional attention on his or her own bodily processes and sensations. The individual then begins to experience a broad range of more intense, bothersome, and concerning physical sensations, resulting in a higher level of anxiety and additional focus on physical sensations, and ultimately leading to a vicious circle. This process has since been described as somatosensory amplification. Though this theory has been influential in the literature, it is problematic in that it is a model developed for hypochondriasis and applied to somatic symptoms. Though there is clearly a conceptual connection between these two symptom profiles, only a small portion of patients diagnosed with somatization syndrome also qualify for a diagnosis of hypochondriasis (Rief et al., 1998). Additionally, this model fails to account for external factors that may perpetuate somatic symptoms.

Barsky and Wyshak's (1990) model was later extended by Kirmayer and Taillefer (1997) to encompass social and forensic factors in addition to cognitive and perceptual factors. They posited that, initially, physical symptoms might be due to a known medical problem or normal bodily sensations that are a part of daily living. The amount of attention that is focused on the body will result in varying degrees of awareness of these physical sensations. Once specific bodily sensations enter into awareness, the individual evaluates their relative importance. In individuals prone to somatic symptoms, the importance of benign physical sensations are often amplified through attribution to illness, cognitive distortions, and vulnerability schemas based on past illness experiences. These cognitive and emotional reactions to physical sensations prompt illness behaviors such as seeking help and reassurance, often from medical professionals or within the individual's own social context. These interpersonal interactions may have the effect of reinforcing these patterns, or promoting recovery. Amplification and maladaptive attributions of physical symptoms may also result in avoidance behaviors in both social and occupational contexts, resulting in functional limitations and physical deconditioning. The various psychological and social mechanisms may increase physiological reactivity.

Deary, Chalder, and Sharpe (2007) proposed a similar mode of somatic symptoms that places these factors within the cognitive-behavioral framework of predisposing, precipitating, and perpetuating factors. The fundamental assumption of this model is that somatic symptoms are maintained by cognitive, behavioral, and physiological factors that are part of an autopoietic cycle. A genetic vulnerability to somatic symptoms combined with childhood trauma serve as predisposing factors, increasing the likelihood that an

individual will develop somatic symptoms. Stressful life events lead to physiological symptoms of stress, producing somatic symptoms and beginning the process of selective attention and sensitization. The individual then associates stress with somatic symptoms and, through operant conditioning processes of avoidance, begins to avoid activities that may lead to additional symptoms. This becomes a vicious cycle of ever-increasing symptomology.

Brown (2004) proposed an integrative conceptual model of medically unexplained symptoms. Brown notes that somatic symptoms can be explained by research and theory from cognitive psychology, and focuses on the nature of attention mechanisms in the cognitive system. An individual's cognitive system is constantly inundated with information that may influence his or her thoughts and behaviors. According to the hierarchical cognitive model of attentional control proposed by Norman and Shallice (1986), one of the primary tasks of the attention system is to filter this information to determine what requires further processing. The parallel spread of activation within these systems results in the generation of multiple perceptual hypotheses, each of which provides an interpretation of the world based on current perceptions and previous experiences. The "Primary Attentional System" (PAS), which is characterized by intuitive, effortless, and self-evident operation, then selects the most active perceptual hypothesis. The individual then uses this hypothesis to organize relevant information into "primary representations" that provide an understanding of the environment and guides future actions. In contrast, the counterpart of the PAS, the "Secondary Attention System" (SAS), is characterized by effortful and deliberate operation, and is associated with self-awareness. This understanding of the cognitive

system, however, underscores the fact that oftentimes behavior is at least partially determined by the PAS, a system that operates without purposeful control. Thus, on some occasions, there may not be a direct relationship between sensory stimulation and personal experience, and often the generation of perceptual experience can be over-determined by past experience.

According to Brown's (2004) theory, medically unexplained symptoms arise when the PAS selects "rogue representations," which is a general term for information that is inappropriately selected by the PAS. Rogue representations may arise from many sources within the cognitive system, including memories of organic pathology, exposure to physical symptoms in others, sociocultural transmission, and verbal suggestion. The rogue representation selected by the PAS results in activation of the SAS such that the individual begins paying selective attention to physical sensations, disease-confirming information and negative affect, resulting in repeated activation of the rogue representation within the cognitive system. These secondary attention processes facilitate reactivation of the rogue representation in the memory system

The perseverative cognition hypothesis. Though previous theories have taken into account the role of worry about health conditions as an important factor in the development and maintenance of somatic symptoms, these theories have focused on the content rather than the nature of these cognitions. Additionally, current theories do not propose a clear mechanism for the relationship between cognitions and somatic symptoms. Brosschot and colleagues (2006) propose that it is not the health-related content of thoughts that result in the development of somatic symptoms, but rather the repetitive nature of these thoughts, which they term "perseverative cognition."

Perseverative cognition has been defined as "the repeated or chronic activation of the cognitive representation of one or more psychological stressors," (Brosschot et al., 2006). Additionally, they posit that perseverative cognition plays a much broader role in psychopathology, and is likely a crucial factor in somatic health as well. Specifically, they have proposed that perseverative cognition prolongs physiological activation in response to a stressor. This prolonged physiological activation is the mechanism through which they believe that perseverative cognition impacts somatic health (Figure 1). However, no studies have directly examined this explanatory pathway.

Perseverative Cognition

As noted above, perseverative cognition has been defined as "the repeated or chronic activation of the cognitive representation of one or more psychological stressors," (Brosschot et al., 2006). A number of cognitive processes such as anticipatory stress, cognitive intrusions, obsessions, and craving have a common feature in that they are repetitive. However, the two most well studied types of perseverative cognition are worry and rumination.

Worry. Worry has been defined as an uncontrollable chain of fear-laden thoughts and images that plays a role in nearly all anxiety disorders (Borkovec & Ray, 1998; Borkovec, Robinson, Pruzinsky, & DePree, 1983). Generally, when an individual experiences worry, they engage in self-talk about negative events he or she is afraid may happen in the future. Eysenck and Calvo (1992) presented a cognitive model of worry, which posits that it has three major functions: alarm, prompt, and preparation. Initially, worry serves as an alarm, where upon initial detection of an internal or external threat, information about the threat is brought into awareness. Second, threat-related thoughts

and images from long-term memory are then prompted into conscious awareness. The

preparation function finally permits the individual to anticipate negative future scenarios

and initiate anticipatory coping, which may include trying to prevent the anticipated

negative developments or to prepare for the expected negative event or outcome.

Oftentimes, individuals feel as though the reason they worry is because it helps them to

discover ways to avoid negative future events, or prepares them for negative events if

they are unavoidable (Borkovec & Roemer, 1995; G. C. L. Davey, Tallis, & Capuzzo,

1996; Freeston, Rheaume, Letarte, Dugas, & Ladouceur, 1994). It has also been theorized

that worry may serve to suppress somatic anxiety or distract the worrier from more

emotionally laden topics (Borkovec & Ray, 1998).

Though worry may subjectively seem to be a productive mental task, empirical

research has linked worry to a number of disorders and negative outcomes. Worry is a

cardinal feature of Generalized Anxiety Disorder according to the DSM-5 (American

Psychiatric Association, 2013) and is also a common process in other psychological

disorders, including panic disorder, social anxiety disorder, obsessive-compulsive

disorder, post-traumatic stress disorder, and anorexia nervosa (Papageorgiou, 2006). For

example, in a prospective study of the relationship between worry and anxiety and

depression in undergraduate students, researchers found that worry was associated with

both depression and anxiety (Segerstrom, Tsao, Alden, & Craske, 2000).

Worry has also been specifically associated with somatic symptoms (Borkovec,

1994; Brosschot & van der Doef, 2006; Brosschot, 2002; Freeston et al., 1996; Rector &

Roger, 1996). Borkovec (1994) found that worry is associated with pain, and Freeston

and colleagues (1996) found that worry is associated with physical symptoms similar to

those found in generalized anxiety disorder. Brosschot and Van Der Doef (2006) found that daily worrying was predictive of a broad set of somatic complaints after controlling for trait anxiety, sex, and age. Additionally, participants who were instructed to use a worry postponement strategy reported fewer somatic complaints than participants who were not, and this effect was mediated by worry duration.

Rumination. Similar to worry, rumination is when an individual experiences repetitive, intrusive negative cognitions (Papageorgiou & Siegle, 2003). Though a number of definitions have been proposed for rumination, generally ruminative thoughts are characterized by conscious cognitive activity, recurrence, uncontrollability, and negative content (Thomsen, Yung Mehlsen, Christensen, & Zachariae, 2003). Rumination is believed to arise in response to goal discrepancies (Martin & Tesser, 1996) or stressful, uncontrollable events (Clark, 1996; King & Pennebaker, 1996; Segerstrom et al., 2000). Martin and Tesser (1996) have proposed that the goal of rumination for individuals is to reduce discrepancies, though it is not always beneficial or helpful in meeting this goal. Nolen-Hoeksema's (1991) response styles theory suggests that rumination helps individuals to turn their attention inward, evaluate a problematic situation and their emotions about it, and gain insight. In support of this possibility, an experimental study conducted by Lyubomirsky and Nolen-Hoeksema (1993) determined that when dysphoric participants underwent a rumination induction, they believed that they were gaining insight about themselves and their problems However, the solutions they produced were relatively poor.

Research has demonstrated that there are individual differences in levels of rumination, such that some individuals may be more prone to ruminate than others

(Martin & Tesser, 1996; McIntosh, Harlow, & Martin, 1995; Susan Nolen-Hoeksema, Parker, & Larson, 1994). Higher levels of rumination have been well established as a predisposing and perpetuating factor in depression, and has specifically been associated with increased severity and length of depressed mood in major depressive disorder (Martin & Tesser, 1989; Susan Nolen-Hoeksema, 1987, 1991; Segerstrom et al., 2000; Teasdale & Barnard, 1993; Wells & Matthews, 1994). Rumination has also been associated with negative outcomes linked to depression including negatively biased thinking, difficulties with problem solving, decreased motivation, impaired concentration, and increased stress (Lyubomirsky & Tkach, 2008). There is also preliminary evidence to suggest that rumination is an important process in the development and maintenance of anxiety disorders (Blagden & Craske, 1996; Muris, Roelofs, Rassin, Franken, & Mayer, 2005; Nolen-Hoeksema, 2000).

In addition to being associated with depression, and to a lesser extent, anxiety, rumination has also been associated with increased levels of somatic symptoms (Lok & Bishop, 1999; Rector & Roger, 1996; Thomsen et al., 2004). In a study conducted by Rector & Roger (1996) students completed self-report measures of rumination, somatic symptoms, and a range of other coping and personality measures just after starting college, and then eight weeks later. Thus, data were collected during a presumably stressful life period. Rumination was initially associated with somatic symptoms, but the correlation was not significant at the eight-week follow-up. In another study, rumination was associated with somatic symptoms in Asian adults (Lok & Bishop, 1999). This effect was mediated by perceived stress. Neither of these studies controlled for affective symptoms, however. More recently, Thomsen (2004) conducted a longitudinal

examination of the relationship between rumination and self-reported physical health in young and elderly adults. At baseline, rumination significantly predicted somatic symptoms, and this relationship was mediated by negative affect. However, in their longitudinal analyses, rumination predicted somatic symptoms only among the young. The mixed findings across these studies indicate that further research is warranted to clarify the relationship between rumination and somatic symptoms. This is one of the few studies that have controlled for negative affect in addition to sex, baseline somatic symptoms, and life events; however, symptoms of depression and anxiety were not included as covariates.

In sum, the distinction made between worry and rumination is that worry corresponds to domains of future threat, while rumination corresponds to domains of past loss. Similarly, worry has particularly been associated with anxiety disorders while rumination has been associated with depressive disorders. However, there is evidence to suggest that much of worry is over past events (Borkovec et al., 1983; Molina, Borkovec, Peasley, & Person, 1998). Additionally, depressive rumination may include concern about future implications of depressive symptoms (Nolen-Hoeksema, 1991). Elevated levels of worry have been associated not only with anxiety symptoms, but also depressive symptoms (Starcevic, 1995). Thus, it is likely that the recurrent nature of these thoughts is salient in the development of anxiety and depression as proposed by Segerstrom and colleagues (2000). Brosschot, Gerin, and Thayer (2006) have extended this possibility to include not only outcomes of anxiety and depression, but also somatic symptoms, and there is some empirical support for this relationship to date (Brosschot, 2002; Rector & Roger, 1996; Borkovec, 1994; Freeston, Dugas, Letarte & Rheaume, 1996; Brosschot &

Van Der Doef, 2006; Thomsen et al, 2004; Lok & Bishop, 1999; Rector & Roger, 1996).

However, it remains to be seen whether perseverative cognition is prospectively related

to somatic symptoms after controlling for symptoms of depression and anxiety.

Physiological Activation as a Mediator

Brosschot, Gerin, and Thayer have also proposed physiological activation as a

potential mechanism of the relationship between perseverative cognition and somatic

symptoms. A number of studies have indicated that there is an association between

perseverative cognition and a variety of cardiovascular and skin conductance measures,

including heart rate, heart rate variability, skin conductance level, and blood pressure

(Brosschot, van Dijk, & Thayer, 2003; Dua & King, 1987; Glynn, Christenfeld, & Gerin,

2002; Lyonfields, Borkovec, & Thayer, 1995; Neumann, Waldstein, Sollers, Thayer, &

Sorkin, 2001; Roger & Jamieson, 1988; Schwartz, Gerin, Davidson, & Christenfeld,

2000; S C Segerstrom, Glover, Craske, & Fahey, 1999; Suchday, Carter, Ewart, Larkin,

& Desiderato, 2004; Thayer, Friedman, & Borkovec, 1996). These measures are all

examples of psychophysiological markers of autonomic nervous system activity.

The autonomic nervous system is the portion of the peripheral nervous system

responsible for the control of bodily functions that are not consciously directed, such as

heart rate and breathing. The autonomic nervous system consists of both the sympathetic

nervous system and the parasympathetic nervous system. The sympathetic nervous

system is activated during the "fight or flight" response, and serves to prepare an

organism for activity by increasing heart rate, respiration, and blood pressure; in contrast,

the parasympathetic nervous system opposes these effects by slowing heart rate and

respiration, and increasing digestion (often termed "rest and digest; Zisner & Beauchaine, 2015).

Electrodermal responding (EDR). Electrodermal responding (EDR) refers to the phasic changes in skin conductance that is measured by capturing current flow across the surface of the skin while voltage is held constant in areas of the body with high concentrations of eccrine sweat glands. This specific type of sweat gland is primarily found on the palms of the hands and the soles of the feet and is innervated by the sympathetic nervous system. Research has shown that eccrine sweat glands are more reactive to emotion than sweat glands in other parts of the body. Thus, assessing eccrine sweat gland activity and reactivity to stimuli provides psychophysiological information regarding individual differences in emotional reactivity that is specific to the sympathetic nervous system. Specifically, both the number of electrodermal responses and relative magnitude of those responses capture sympathetic nervous system orienting and reactivity (Zisner & Beauchaine, 2015).

Previous research has indicated that EDR is a biomarker for both internalizing and externalizing symptoms of psychopathology. For example, individuals with high levels of trait anxiety also exhibit greater electrodermal activity when compared to a control group. In contrast, individuals with externalizing disorders, low anxiety, or high levels of aggression are more likely to exhibit low levels of electrodermal activity (Zisner & Beauchaine, 2015). Though the literature on the relationship between EDR and somatic symptoms is limited, there is some evidence to suggest an association. For example, Kanbara and colleagues (2004) found that individuals with somatic symptoms had a lower level of physiological reactivity, including electrodermal activity, than a control

group. As somatic symptoms are generally considered an internalizing symptom however, this finding is contrary to the literature suggesting that internalizing disorders are related to increased electrodermal activity. Thus, additional research is needed in order to better understand the relationship between EDR and somatic symptoms.

Respiratory sinus arrhythmia (RSA). Parasympathetic nervous system activity is often measured using vagal tone, which refers to the tonic influence of the vagus nerve on the sino-atrial node of the heart (Porges, 1995). Respiratory sinus arrhythmia (RSA), an indirect measure of vagal tone, refers to the changes in heart rate across the respiratory cycle. The predictable changes in heart rate across the respiratory cycle occur due to increases in inhibitory parasympathetic signaling during exhalation, and decreases in inhibitory parasympathetic signaling during inhalation. Previous research has indicated that RSA likely serves as an important transdiagnostic biomarker of emotion dysregulation difficulties (for a complete review, see Beauchaine, 2015). However, the conditions under which it is measured have important interpretative implications.

When RSA is measured while an individual is at rest, it is often termed basal RSA. Basal RSA serves as an index of resting vagal tone. Under resting conditions, polyvagal theory posits that the influence of the parasympathetic nervous system should be high; therefore, lower resting RSA indicates decreased physiological flexibility and a lower ability to adapt when faced with stressors (Porges, 1995; Porges, 2007). Empirical evidence has validated this theory, as higher basal RSA has been associated with more adaptive emotion regulation strategies (Gentzler, Santucci, & Fox, 2009), while low resting RSA has been associated with both internalizing and externalizing symptoms (Zisner & Beauchaine, 2015).

When RSA is measured during a stressor task, it is often termed RSA reactivity. RSA reactivity refers to the degree to which vagal tone changes during a challenging or stressful experience. Polyvagal theory posits that RSA reactivity is an index of an individual's ability to adapt to environmental demands (Porges, 1995; Porges, 2007). Reductions in vagal tone indicate that the vagus nerve is withdrawing its inhibitory effect on cardiac functioning, indicating a decrease in parasympathetic nervous system activity. This allows the individual to utilize resources in order to meet the environmental demand he or she is being faced with. The relationship between RSA reactivity and psychopathology, however, remains unclear. For example, Beauchaine (2001) found that individuals with excessive RSA reactivity in addition to low basal RSA is indicative of emotion regulation difficulties. In contrast, other studies have shown that low RSA reactivity to stress, which has been theorized to be related to a low level of responsiveness to changing environmental demands, may also indicate poor emotion regulation skills (Zisner & Beauchaine, 2015).

When RSA is measured following a stressor task, it is expected that RSA will return to resting levels. However, if this is not the case, an ongoing stress response may be present. This may be caused by a failure of an individual to employ adaptive emotion regulation strategies (Santucci et al., 2008). However, this measure of RSA is far less researched and understood than basal RSA, and its implications for psychopathology require further exploration. Regardless, investigating all three components of RSA (basal, reactivity, and recovery) measures the temporal course of emotional responsivity to a stressor over time, and provides a more complete understanding of an individual's ability to regulate emotions as evidenced by their physiology (Santucci et al., 2008).

Though basal RSA has been established as a transdiagnostic marker for a number of psychological disorders, no studies that I am aware of have examined the relationship between any measure of RSA and somatic symptoms or related disorders. There is, however, a strong association between somatic symptoms, depressive symptoms, and anxiety symptoms; thus the overlap among these conditions indicates that RSA will likely serve as a psychophysiological marker for somatic symptoms as well. Additionally, there is a significant body of literature to suggest that there is a relationship between somatic symptoms and other measures of physiologic dysregulation, including respiration (Grossman, 1983), hyperventilation (Troosters et al., 1999), and heart rate and end-tidal carbon dioxide pressure (Wientjes & Grossman, 1994). Thus, it is likely that RSA is also an important factor in the development and maintenance of somatic symptoms.

Perseverative cognition and physiological activation. Perseverative cognition has previously been associated with a number of physiological measures of both sympathetic and parasympathetic nervous system activity, including heart rate, heart rate variability, skin conductance level, and blood pressure (Brosschot et al., 2003; Dua & King, 1987; Glynn et al., 2002; Lyonfields et al., 1995; Neumann et al., 2001; Roger & Jamieson, 1988; Schwartz et al., 2000; Segerstrom et al., 1999; Suchday et al., 2004; Thayer et al., 1996). With regard to RSA, a number of studies have indicated that perseverative cognition is associated with lower basal RSA. At least two studies have demonstrated that both experimentally induced and trait measures of worry are associated with decreased RSA (Lyonfields et al., 1995; Thayer et al., 1996). Additionally, Brosschot, van Dijk, and Thayer (2003) found that lower HRV was associated with periods of worry that occurred throughout the day during an ambulatory study. The

relationship between perseverative cognition and skin conductance level, however, is less

well established. In fact, a several studies have reported null effects for the relationship

between perseverative cognition and skin-conductance level (Dua & King, 1987;

Segerstrom et al., 1999; Vickers & Vogeltanz-Holm, 2003). Though the literature in this

area is limited, it is likely that there is an association between perseverative cognition and

both increased sympathetic nervous symptom activity and decreased parasympathetic

nervous system activity (Brosschot et al., 2006).

The Present Study

Brosschot and colleagues (2006) have proposed that perseverative cognition

prolongs physiological activation in response to a stressor. This prolonged physiological

activation is the mechanism through which they believe that perseverative cognition

impacts somatic health. However, no studies have directly examined this explanatory

pathway. The present study will examine this portion of the perseverative cognition

hypothesis. I will first test the hypothesis that perseverative cognition will predict

subsequent somatic symptoms, the way in which I have operationalized a pathogenic

state, above and beyond depression and anxiety. Second, I will examine whether

physiological activation, operationalized as RSA and EDR, will mediate this relationship.

Each of these physiological measures will be collected before, during, and after

participants complete a stressor task so that both tonic and phasic measures of

sympathetic and parasympathetic nervous system activity can be accurately represented

(Figure 2). Specifically, I hypothesized that physiological activation as defined by (a)

lower basal RSA, (b) greater decreases in RSA in response to a stressor, (c) less return to

basal RSA during the recovery period, (d) higher basal EDR, (e) greater increases in EDR

in response to a stressor, and (f) less return to basal EDR during the recovery period may explain the relationship between perseverative cognition and somatic symptoms.

CHAPTER II

Method

Sample and Participant Selection

Sample Size. The flexible nature of SEM allows for examination of complex associations, the use of various types of data, and comparison across models; however this flexibility has also resulted in difficulty in establishing guidelines for the sample size necessary to detect effects that are present in the data (Wolf, Harrington, Clark, & Miller, 2013). A number of rules of thumb have been proposed including a minimum N of 100 or 200 (Boomsma, 1982; Boomsma, 1985), 5 or 10 observations for each parameter to be estimated (Bollen, 1989) and 10 cases per variable (Nunnally, Bernstein, & Berge, 1967). Based on these rules of thumb, estimates of the necessary sample size range from 100 to 200. According to a guide provided by Fritz and MacKinnon (2007), when using bias-corrected bootstrapping, approximately 148 participants are required to detect a small to moderate effect (0.26) for both the alpha and beta path of the mediation model. In the present study, 220 participants were enrolled. Data from five participants were excluded due to missing data on exogenous variables, resulting in a final sample size of 215 for the initially hypothesized models. Exact numbers of participants with data for each measure are included in Tables 3, 4, and 5. In posthoc analyses, 219 participants were included due to the elimination of exogenous variables from the model that had previously limited use of their data.

Recruitment. Participants were recruited from introductory psychology courses at Seattle Pacific University where students receive course credit for enrolling in a research study. All students were eligible for the study if they were enrolled in the introductory psychology course and at least 18 years of age.

Participants. Participant ($N = 220$) demographics per self-report are presented in Table 1. Participants ranged in age from 18 to 39 years old ($M = 19.63$, $SD = 12.10$). For the 2017 Autumn Quarter, the most recent statistics available, Seattle Pacific University (SPU) reported a total population of 2,911 undergraduate students. SPU reports that the average age of these students is 21, females represent 67% of the undergraduate population, and 40% of the undergraduate students fall under the broad category of "ethnic minority." The recruited sample has higher female representation than the undergraduate population. The sample did have a comparable age range and racial and ethnic diversity of the whole undergraduate population sampled.

Procedure

This dissertation was part of a larger study conducted by the Adolescent Cognition and Emotion (ACE) Lab at Seattle Pacific University entitled Stress and Somatic Symptoms in Young Adults (SASSY). The Seattle Pacific University institutional review board approved all study procedures and materials for SASSY. In the following section however, I will outline only those procedures and measures relevant to this dissertation.

Participants were recruited from introductory psychology courses that took place from September, 2015 to March, 2017. Though students were recruited to participate at the beginning of the quarter, they were allowed to begin participation in the study at any

time in the quarter that would allow them to complete all portions of the study by the end

of the quarter. Participants signed up for the research study using the online Sona

platform used by Seattle Pacific University. Once students enrolled in Part 1 of the study,

they completed a baseline questionnaire that included measures of trauma, depressive

symptoms, somatic symptoms, anxiety, rumination, and worry. Following completion of

the baseline questionnaire, participants were invited to enroll in Part 2 of the study by

scheduling a laboratory visit.

During the laboratory visit, researchers first reviewed the informed consent

document with the participant. Once the participant provided consent by signing this

form, the researcher attached eight self-adhering pre-gelled electrodes to the participant's

skin (one on the sternum; one on the lowest left rib; one on the lower back; one on the

back of the neck; one on the collarbone; one on the front of the neck; and two on the

bottom of the left foot). Each of these sensors was attached to a lead that was connected

to a Biopac MP 150 Data Acquisition System (Biopac Systems, Inc., Goleta, CA). ECG

and EDR signals were recorded using AcqKnowledge 4.1 software for the duration of the

laboratory visit.

After being hooked up to the physiological recording equipment, participants

watched a series of nature scenes during which basal physiological measures were

recorded. Next, participants were told that they had two minutes to prepare a speech on

the topic of "Why are you a good friend?" that they may have to present. They were

provided with a screen to type notes on for their speech that also indicated the amount of

time remaining in the preparation period. Following this preparation period, the computer

showed a screen indicating the condition to which the participant was randomized.

However, no participants were ever required to present their speech. After completing

participation in the laboratory visit, participants were debriefed, and told that no

participants ever had to give their speech. They were also provided with information on

the empirical support for the use of this paradigm as a stressor task. Following the

laboratory visit, participants were asked to complete six additional online questionnaires

that included a measure of somatic symptoms. The participant completed one of the

questionnaires every two to three days, providing longitudinal information about their

somatic symptoms over the course of approximately two weeks.

Measures

     Covariates. Previous research has indicated that there are strong correlations

among depression, anxiety, and somatic symptoms (for a review, see Henningsen,

Zimmermann, & Sattel, 2003). For example, Haug, Mykletun, and Dahl (2004) found

significant correlations among anxiety, depressive, and somatic symptoms independent of

age and gender in a sample of over 50,000 Norwegian individuals. As previously

reviewed, there is also substantial evidence to suggest that both rumination and worry are

related to depression and anxiety (Muris et al., 2005; Segerstrom et al., 2000).

Additionally, as discussed above, trauma history is a well-established predictor of

somatic symptoms (Roelofs & Spinhoven, 2007). Thus, baseline measures of depressive

symptoms, anxiety, and trauma history will be included as covariates in all primary

analyses in order to better understand the specific relationships of interest in the current

investigation

     Center for epidemiological studies depression scale (CES-D). Depressive

symptoms were measured at Part 1 with the Center for Epidemiological Studies

Depression scale (CES-D; Radloff, 1977). The CES-D is a measure developed to assess

depressive symptoms over the past week among adults with subclinical levels of

depressive symptoms. The measure consists of 20 statements related to depressive

symptoms. Sample items include: "I had crying spells," "I felt that everything I did was

an effort," and "I felt hopeful about the future" (reverse scored item). Participants

rated the frequency with which they experienced each symptom during the past

week using a 4-point Likert scale ranging from 0 (*rarely or none of the time [less than 1*

day]) to 3 (*most or all of the time [5-7 days]*). Scores were calculated first by reverse-

scoring positively worded items and then summing the items to produce a total

score. Scores on the CES-D range from 0 to 60, with higher scores indicating a greater

degree of depressive symptoms. Scores above 16 suggests a significant level of

depression (Radloff, 1977). The CES-D and is considered a valid measure of depressive

symptoms, as it highly correlates with clinician ratings of depression ($r = .53$; Radloff,

1977). Cronbach's alpha for the CES-D among college student samples ranges from .78

to .87 (Radloff, 1991; Verhaeghen, Joorman, & Khan, 2005). In the present study, the

Cronbac''s alpha was 0.72.

Generalized anxiety disorder questionnaire – IV (GAD-Q-IV). Generalized

anxiety symptoms were measured at Part 1 with the Generalized Anxiety Disorder

Questionnaire – IV (GADQ-IV; Roemer, Posa, & Borkovec, 1995). The measure was

developed as a screening device for generalized anxiety disorder, and has subsequently

been revised based on DSM-IV criteria (Newman et al., 2002). This measure consists of

a number of items designed to determine whether an individual's worry is distressing,

uncontrollable, and often about minor things. Sample items include "Do you experience

excessive worry?" and " Do you find it difficult to control your worry (or stop worrying) once it starts?" The sixth item of the questionnaire acts as a skip out item. It asks "During the last six months have you been bothered by excessive and uncontrollable worries more days than not?" If the individual responds "no" they are instructed to skip the three remaining questions, which further characterize the symptoms and functional impact of the individual's worry. The GAD-Q-IV was scored according to the instructions outlined by Newman and colleagues (2002), yielding a sum total for each participant. This sum score can be compared to a cutoff rate of 5.7 to determine whether the individual may have Generalized Anxiety Disorder. The GAD-Q-IV has been established as an effective screening tool for Generalized Anxiety Disorder with a low false positive rate and a fairly low false negative rate (Newman et al., 2002). Because of the structure of the measure, it is not appropriate to assess internal consistency and item-total correlations for the GAD-Q-IV.

Brief trauma questionnaire (BTQ). Trauma history was measured at Part 1 with the Brief Trauma Questionnaire (BTQ; Schnurr, Vielhauer, Weathers, & Findler, 1999). The BTQ is a 10-item measure of trauma history. The measure consists of a ten prior life experiences that would be extraordinarily stressful or disturbing for almost everyone. Sample items include "Have you ever been in a serious car accident, or a serious accident at work or somewhere else?" and "Has anyone ever made or pressured you into having some type of unwanted sexual contact?" Participants will be asked to indicate "Yes" or "No" in response to each question. A count is taken of the number of items each participant responded yes to, with a potential score ranging from 0 to 10. While there is limited psychometric data for this measure, early evidence for its' reliability and validity

have been promising (Lancaster, Melka, & Rodriguez, 2009; Schnurr et al., 1999). In a

study of the reliability between the BTQ and an interview with a subset of participants,

kappa coefficients for the existence of a history of traumatic events ranged from .60 to

1.00. Kappas for eight of the items fell above .74 (Schnurr et al., 1999).

Perseverative cognition.

Ruminative response scale (RRS). The RRS is a 22-item measure of rumination.

Participants were asked to indicate how often they do each item when they are sad on a

four-point scale from 1 (*almost never*) to 4 (*almost always*). Sample items include "think

about a recent situation, wishing it had gone better," "think about all your shortcomings,

failings, faults, mistakes," and "think about how angry you are with yourself." The

potential total sum scores range from 22 to 88. Additionally, two subscales (brooding and

reflection) can be calculated (Treynor, Gonzalez, & Nolen-Hoeksema, 2003). A

confirmatory factor analysis of the Dutch version of this measure indicated that a two-

factor structure provided adequate fit (Schoofs, Hermans, & Raes, 2010). Additionally,

the RRS has demonstrated high internal consistency, with Cronbach's alpha ranging from

0.90 to 0.91 (Schoofs et al., 2010; Treynor et al., 2003). The internal consistency for each

of the subscales are lower, but acceptable (α= 0.78 for the brooding subscale and α=0.75

for the reflection subscale; Schoofs et al., 2010). In the present study, only the brooding

subscale was used, and the Cronbach's alpha was 0.78.

Pennsylvania State worry questionnaire (PSWQ). The PSWQ is a 16-item

measure of worry (Meyer, Miller, Metzger, & Borkovec, 1990). Participants were asked

to indicate how typical each item is of them on a five-point scale from 1 (*not at all typical*

of me) to 5 (*very typical of me*). Five of the items are reverse scored. Sample items

include "Many situations make me worry," "I find it easy to dismiss worrisome

thoughts," (reverse scored), and "My worries overwhelm me." The potential sum score

ranges from 16 to 80. The PSWQ has demonstrated good internal consistency in previous

studies (α =0.93; Brown, Antony, & Barlow, 1992). In the present study, Cronbach's

alpha was 0.73.

Physiological activation.

Electrodermal responding (EDR). A galvanic skin response (GSR) amplifier was

used to measure sweat secretion linked with SNS activity through electrodermal

responding (EDR). The system assesses skin conductance based on the moisture level of

the skin through two disposable electrodermal electrodes. These electrodes were placed

on the left foot directly beneath the big and little toes. The area was cleaned and dried

before attaching the electrodes and then secured with athletic tape around the foot and a

sock. Participants were asked to rest their foot in a stable position and to refrain from

moving it as much as possible. Specific settings on the GSR amplifier included a gain of

5 micromho, along with a low pass filter at 10 Hz. AcqKnowledge 4.1 software will be

used to record EDR, and to apply a low pass filter and waveform math transformation to

the raw data. EDR data will be processed using the Mindware EDA 3.0.20 analysis

program. EDR was scored in 30-second epochs as the number of skin conductance

responses from peak to valley that that exceeded 0.05 microsemens over a duration of at

least one second. The number of responses was averaged across the 4-minute

baseline (nature scene viewing), 2-minute stressor, and 3-minute recovery periods. Each

epoch was visually inspected and corrected as needed by trained research assistants.

More responses indicated increased sympathetic nervous system activity.

Basal EDR was calculated as the mean number of EDR responses across baseline epochs (bEDR). EDR reactivity to the stressor task (sEDR) was calculated by subtracting bEDR from the mean of EDR responses across the stressor task epochs. Positive EDR reactivity scores indicate an increase in the number of responses from baseline while negative EDR reactivity scores indicate a decrease in the number of responses from baseline. EDR recovery (rEDR) was calculated by subtracting bEDR from the mean number of responses across the recovery period epochs. Positive EDR recovery scores indicate an increase in EDR from baseline while negative EDR recovery scores indicate a decrease in EDR from baseline (Zisner & Beauchaine, 2015).

Respiratory sinus arrhythmia (RSA). Basal RSA, RSA reactivity, and RSA recovery data were acquired using ECG signals amplified and sampled continuously at 1000 Hz with a Biopac MP150 Data Acquisition System (Biopac Systems, Inc., Goleta, CA). AcqKnowledge 4.1 software was used to record ECG, which was measured using pre-gelled disposable Ag/AgCL electrodes placed in a Lead II configuration on the chest and abdomen. Data was analyzed within the respiratory range of 0.15 to 0.50 Hz using spectral analysis and normalized with logarithmic transformations. ECG data was processed using the Mindware HRV 3.0.20 analysis program. RSA was scored in 30-second epochs and averaged across the 4-minute baseline (nature scene viewing), 2-minute stressor, and 3-minute recovery periods. Each epoch was visually inspected and corrected as needed by trained research assistants.

Basal RSA was calculated as the mean of RSA responses across baseline epochs (bRSA). RSA reactivity to the stressor task (sRSA) was calculated by subtracting bRSA from the mean of RSA responses across the stressor task epochs.

Positive RSA reactivity scores indicate an increase in RSA from baseline while negative

RSA reactivity scores indicate a decrease in RSA from baseline. RSA recovery (rRSA)

was calculated by subtracting bRSA from the mean of RSA responses across the recovery

period epochs. Positive RSA recovery scores indicate an increase in RSA from baseline

while negative RSA recovery scores indicate a decrease in RSA from baseline (Zisner &

Beauchaine, 2015).

      Somatic symptoms.

      Patient health questionnaire – 15 (PHQ-15). The PHQ-15 (Kroenke, Spitzer, &

Williams, 2002) is a 15-item measure of somatic symptoms. The measure consists of a

list of symptoms including "Stomach pain," "Headaches," and "Shortness of breath."

Participants were asked to indicate the level at which they were bothered by each

symptom on a scale ranging from 0 (*not bothered at all*) to 2 (*bothered a lot*). At

baseline, participants were asked to indicate this for symptoms over the past four weeks.

On the daily surveys, participants were asked to indicate this for symptoms since the last

time they had completed the survey. The potential sum scores range from 0 to 30. The

PHQ-15 has demonstrated adequate internal consistency in previous studies ($\alpha = 0.79$)and

evidence for good validity in non-Hispanic populations (Interian, Allen, Gara, Escobar, &

Díaz-Martínez, 2006). In the present study, Cronbach's alpha values ranged from 0.78 to

0.87.

Data Analytic Plan

      SPSS 24 was used to prepare and examine data prior to testing the primary

hypotheses of this study. Specifically, an analysis of normality and outliers was

conducted and descriptive statistics were calculated. Additionally, correlations among

variables were examined. Mplus 7 was used to conduct confirmatory factor analysis in

order to examine the fit of all latent variables: rumination, worry, perseverative cognition,

and somatic symptoms. Additionally, Mplus 7 was used to examine the mediating role of

six physiological measures (basal EDR, EDR reactivity, EDR recovery, basal RSA, RSA

reactivity, and RSA recovery) between trait perseverative cognition (a latent variable

consisting of both worry and rumination) and subsequent somatic symptoms. The

hypothesized mediational relationships of EDR and RSA were tested separately. Each

model included measures of the physiological mediator at baseline, during the stressor

task, and during the recovery period (Figures 3a and 3b). Because physiological measures

during the stressor task and recovery period were entered as change scores from baseline,

the three mediators were not allowed to covary. Mplus 7 provided tests of overall model

fit, standardized coefficients for each path, and tests of direct and indirect effects for both

models with depression, anxiety, biological, and trauma history included as covariates.

CHAPTER III

Results

Preliminary Analysis

Prior to testing my proposed model, I inspected the data for missing data, normality of distribution and outliers. Descriptive statistics are described below.

Data Preparation. There were 220 participants who enrolled in the study. No data imputation was used at the raw data level. Instead, scale scores were calculated when individuals completed 80% of the items in the measure. Using these guidelines, 215 participants had complete data on all baseline variables and were included in the primary analyses, as Mplus allows for missing data on endogenous variables. All available data is used to estimate the model using full information maximum likelihood; each parameter is estimated directly without first filling in missing data values (Muthén & Muthén, 2017). Though data from 215 participants was included in these analyses, it is important to note that not all participants completed all measures. 164 participants had complete data for RSA variables collected during the laboratory visit and 163 had complete data for EDR variables collected during the laboratory visit. Additionally, 150 participants completed at least five of the six daily questionnaires. Exact numbers of participants with data for each measure are included in Tables 3, 4, and 5. In posthoc analyses, 219 participants were included due to the elimination of exogenous variables from the model that had previously limited use of their data.

Normality and outlier analysis. Outliers were assessed graphically through stem-and-leaf plots as well as through visual inspection of the raw data. There was sufficient evidence to support deleting two problematic outliers. One participant's data

(ID #239) for RSA was coded as missing in all subsequent analyses, as the values for their RSA were far below the normally observed range. Similarly, one participant's data (ID# 228) for EDR was far above the normally observed range, and was coded as missing in all subsequent analyses.

Normality was assessed graphically through histograms (see Table 2), normal curves, P-P plots, Q-Q plots and box plots. It was assessed numerically through skewness and kurtosis output and computed standardized z-scores. Normality was also assessed the Shapiro-Wilk test of normality (S-W test; Field 2005). Scores on Basal RSA and EDR during stressor did not significantly differ from the normal distribution. The distribution of all other scores used for analyses were significantly different than normal (see Table 2). I examined variables that showed skewness and kurtosis (Table 2). Positive skewness values indicate scores lean to the left of the distribution and negative skewness values indicate the scores lean to the right. Positive kurtosis values indicate more weight in the tails while negative kurtosis values indicate less weight in the tails compared to what would be expected with normal distribution (Westfall, 2014). In order to address the non-normal nature of my data, bootstrapping procedures as recommended by Preacher & Hayes (2008) were used. Bootstrap resampling uses the sample as a population from which many random samples are drawn and continuously replaced so that they have an equal likelihood of being randomly selected on all subsequent drawings (Mallinckrodt et al., 2006). The bootstrap resampling procedure increases power by providing non-symmetric confidence intervals, reducing the likelihood of making a Type II error. Additionally, bootstrapping does not assume normality of the sampling distribution.

Descriptive Statistics. Bivariate correlations, means, and standard deviations for baseline study variables are presented in Table 3, descriptive data for the lab visit variables are presented in Table 4, and descriptive data for the average daily measures in the daily questionnaires are presented in Table 5.

Baseline. As expected, both trait worry and trait rumination were positively correlated with depressive symptoms and anxiety symptoms. However, only trait rumination was associated with trauma history. Additionally, sex was associated with both trait worry and trait rumination (see Table 3), and was included as a covariate when fitting the hypothesized model.

Lab Visit. All measures of RSA were positively correlated with one another, and all measures of EDR were positively correlated with one another as expected (see Table 4). Additionally, no RSA measures were correlated with EDR measures. RSA during the stressor was also positively correlated with age.

Weekly. Biological sex correlated with somatic symptoms on days one through four, but not five and six. As expected, all measures of somatic symptoms were positively correlated with one another (see Table 5).

Confirmatory Factor Analyses. Prior to testing the proposed mediation model, I performed confirmatory factor analyses for all latent variables: rumination, worry, perseverative cognition, and somatic symptoms. According to guidelines published by Schreiber, Nora, Stage, Barlow, & King (2006), in order for a model to have adequate fit, relative χ^2 ratios should be less than 2, comparative fit index (CFI) values should be at least 0.95, root mean square error of approximation (RMSEA) values should be less than 0.06, and standardized root mean square residual (SRMR) should be less than 0.08. When

adequate model fit was not observed, changes were made to model fit were determined through examination of modification indices, and evaluation of the consistency of these potential changes with the theoretical underpinnings of the measure. Alternative models were compared using the Akaike information criterion and Bayes information criterion as suggested by Schreiber and colleagues (2006) for non-nested models. For both of these criterions, smaller values indicate improved model fit.

Based on these analyses, worry was modeled as a two-factor structure as this was a better fit for the data (see Table 6, Figure 4b). The two factors were based on the structure of the measure, with reverse-coded items making up a separate factor from items that were not reverse coded. Both rumination and somatic symptoms were modeled as single factors (Figure 4a and 4c respectively). For both worry and rumination, items were allowed to covary based on a combination of modification indices and similarity of item content. Additionally, measures of somatic symptoms were allowed to covary among consecutive days, given that less change in symptom report is expected over a shorter time period (Figure 4).

Primary Analyses

Tests of Model Fit for RSA. I assessed my hypothesized RSA model using MPlus 7. I first evaluated the adequacy of the hypothesized model by examining the fit statistics including the chi-square (χ^2) likelihood ratio statistic, relative χ^2 ratios, CFI, RMSEA, and SRMR. The fit indices were: $\chi^2(505) = 931.42$, $p < 0.001$; χ^2 Ratio = 1.84; CFI = 0.88; RMSEA = 0.06; SRMR = 0.21.

Tests of Direct and Indirect Effects for RSA. Once adequate model fit was established, I assessed the joint effects of multiple mediators using bootstrap resampling

procedures (Mallinckrodt et al., 2006; Preacher & Hayes, 2008). I specified 5000

bootstrap iterations and used 95% bias-corrected confidence intervals (Figure 5a).

Specific direct effects, indirect effects, and total effects are discussed below and reported

in Table 7.

C' path: Did trait perseverative cognition predict daily somatic symptoms? Trait

perseverative cognition significantly predicted daily somatic symptoms ($\beta = -0.23$, $p =$

0.001). However, this relationship was in the unexpected direction, such that individuals

with higher levels of perseverative cognition experienced fewer somatic symptoms, after

controlling for anxiety symptoms, depressive symptoms, trauma history, and biological

sex.

A paths: Did trait perseverative cognition predict RSA? Within the hypothesized

model, perseverative cognition did not significantly predict basal RSA ($\beta = -0.01$, $p =$

0.956), RSA reactivity ($\beta = -0.12$, $p = 0.147$), or RSA recovery ($\beta = -0.11$, $p = 0.269$).

B paths: Did RSA predict daily somatic symptoms? Within the hypothesized

model, basal RSA ($\beta = -0.08$, $p = 0.202$) and RSA recovery ($\beta = 0.067$, $p = 0.267$) did not

significantly predict somatic symptom. RSA reactivity was trending ($\beta = 0.09$, $p = 0.099$)

such that individuals who experienced less of a decrease in RSA during the stressor task

were more likely to experience somatic symptoms.

Indirect effects. Within the full model, RSA did not mediate the relationship

between perseverative cognition and somatic symptoms. This was true for basal RSA (β

$= 0.00$, $p = 0.967$), RSA reactivity ($\beta = -0.01$, $p = 0.343$), and RSA recovery ($\beta = -0.01$, p

$= 0.486$).

Tests of Model Fit for EDR. I assessed my hypothesized EDR model using MPlus 7. I first evaluated the adequacy of the hypothesized model by examining the fit statistics including the chi-square (χ^2) likelihood ratio statistic, relative χ^2 ratios, CFI, RMSEA, and SRMR. The fit indices were: $\chi^2(505) = 1010.20$, $p < 0.001$; χ^2 Ratio = 2.00; CFI = 0.86; RMSEA = 0.07; SRMR = 0.20.

Tests of Direct and Indirect Effects for EDR. Once adequate model fit was established, I assessed the joint effects of multiple mediators using bootstrap resampling procedures (Mallinckrodt et al., 2006; Preacher & Hayes, 2008). I specified 5000 bootstrap iterations and used 95% bias-corrected confidence intervals (Figure 5b). Specific indirect effects, total effects, and direct effects are discussed below and reported in Table 8.

C' path: Did trait perseverative cognition predict daily somatic symptoms? Trait perseverative cognition did not significantly predict daily somatic symptoms ($\beta = -0.23$, $p = 0.074$). Similar to the RSA model, though this relationship was not significant it was trending in the unexpected direction, such that individuals with higher levels of perseverative cognition experienced fewer somatic symptoms, after controlling for anxiety symptoms, depressive symptoms, trauma history, and biological sex.

A paths: Did trait perseverative cognition predict EDR? Within the hypothesized model, perseverative cognition did not significantly predict EDR. This was true for basal EDR ($\beta = -0.10$, $p = 0.430$), EDR reactivity ($\beta = 0.18$, $p = 0.144$), and EDR during the recovery period ($\beta = 0.06$, $p = 0.634$).

B paths: Did EDR predict daily somatic symptoms? Within the full model, EDR did not significantly predict somatic symptoms. This was true for EDR at baseline ($\beta = -$

0.03, $p = 0.688$), EDR reactivity ($\beta = -0.06$, $p = 0.546$), and EDR during the recovery

period ($\beta = 0.07$, $p = 0.341$).

    Indirect effects. Within the full model, EDR did not mediate the relationship

between perseverative cognition and somatic symptoms. This was true for EDR at

baseline ($\beta = 0.00$, $p = 0.926$), EDR reactivity ($\beta = -0.01$, $p = 0.846$), and EDR during the

recovery period ($\beta = 0.00$, $p = 0.876$).

Post Hoc Analyses

    In both of the models tested, the relationship between perseverative cognition and

somatic symptoms was in the unexpected direction given apriori theory and prior

research. From a theoretical standpoint, one possible explanation for this is that the

variance in somatic symptoms related to the problematic aspects of perseverative

cognition may have significant overlap with the variance accounted for by measures of

symptoms of anxiety and depression. Thus, after depression and anxiety are controlled

for, only adaptive aspects of perseverative cognition (such as problem solving) remain

and have a negative association with somatic symptoms. In order to evaluate this

possibility, MPlus 7 was used to assess the total effect of perseverative cognition on

somatic symptoms while excluding measures of depression and anxiety separately and

together. Model fit was then compared to the initially hypothesized total effect. Results of

these analyses are presented in Table 9. Excluding both the GAD and CES-D (measures

of anxiety and depression, respectively) provided a total effect that indicated adequate

model fit by more indices than the hypothesized model. Additionally, in this model the

relationship between perseverative cognition and somatic symptoms was in the expected

direction. These results are consistent with significant overlap of the variance accounted

for by measures of anxiety, depression, and perseverative cognition. The primary

analyses were run once again without the inclusion of depression and anxiety as

covariates in order to evaluate the role of sympathetic and parasympathetic nervous

system activity as an explanatory factor in the relationship between perseverative

cognition and somatic symptoms.

Tests of Model Fit for RSA. I first evaluated the adequacy of the alternative

model by examining the fit statistics including the chi-square (χ^2) likelihood ratio

statistic, relative χ^2 ratios, CFI, RMSEA, and SRMR. The fit indices were: $\chi^2(447)$

=629.99, $p < 0.001$; χ^2 Ratio = 1.41; CFI = 0.95; RMSEA = 0.06; SRMR = 0.08.

Tests of Direct and Indirect Effects for RSA. Once adequate model fit was

established, I assessed the joint effects of multiple mediators using bootstrap resampling

procedures (Mallinckrodt et al., 2006; Preacher & Hayes, 2008). I specified 5000

bootstrap iterations and used 95% bias-corrected confidence intervals (Figure 6a).

Specific direct effects, indirect effects, and total effects are discussed below and reported

in Table 10.

C' path: Did trait perseverative cognition predict daily somatic symptoms? Trait

perseverative cognition significantly predicted daily somatic symptoms ($\beta = 0.38$, $p <$

0.001) such that individuals with higher trait-level perseverative cognition experienced

more somatic symptoms after controlling for trauma history and biological sex.

A paths: Did trait perseverative cognition predict RSA? Within the alternative

model, perseverative cognition did not significantly predict basal RSA ($\beta = -0.01$, $p =$

0.945), RSA reactivity ($\beta = -0.12$, $p = 0.144$), or RSA recovery ($\beta = -0.11$, $p = 0.265$).

B paths: Did RSA predict daily somatic symptoms? Within the alternative

model, basal RSA (β = -0.10, p = 0.275), RSA reactivity (β = 0.03, p = 0.711), and RSA

recovery (β = 0.12, p = 0.173) did not significantly predict somatic symptom.

Indirect effects. Within the alternative full model, RSA did not mediate the

relationship between perseverative cognition and somatic symptoms. This was true for

basal RSA (β = 0.00, p = 0.961), RSA reactivity (β = -0.00, p = 0.786), and RSA

recovery (β = -0.01, p = 0.433).

Tests of Model Fit for EDR. I assessed my alternative EDR model using MPlus

7. I first evaluated the adequacy of the hypothesized model by examining the fit statistics

including the chi-square (χ^2) likelihood ratio statistic, relative χ^2 ratios, CFI, RMSEA,

and SRMR. The fit indices were: $\chi^2(447) = 702.62$, $p <0.001$; χ^2 Ratio = 1.57; CFI =

0.93; RMSEA = 0.05; SRMR = 0.08.

Tests of Direct and Indirect Effects for EDR. Once adequate model fit was

established, I assessed the joint effects of multiple mediators using bootstrap resampling

procedures were used (Mallinckrodt et al., 2006; Preacher & Hayes, 2008). In the present

study, 5000 bootstrap iterations were specified and 95% bias-corrected confidence

intervals were used (Figure 6b). Specific indirect effects, total effects, and direct effects

are discussed below and reported in Table 11.

C' path: Did trait perseverative cognition predict daily somatic symptoms? Trait

perseverative cognition significantly predicted daily somatic symptoms (β = 0.37, p =

0.003) such that individuals with higher trait-level perseverative cognition experienced

more somatic symptoms after controlling for trauma history and biological sex.

A paths: Did trait perseverative cognition predict EDR? Within the hypothesized

model, perseverative cognition did not significantly predict basal EDR (β = -0.09, p =

0.748) or EDR during the recovery period (β = 0.06, p = 0.591). EDR reactivity was

trending (β = 0.18, p = 0.098) such that individuals with more perseverative cognition

were more likely to experience an increase in EDR during the stressor task.

B paths: Did EDR predict daily somatic symptoms? Within the full model, EDR

did not significantly predict somatic symptoms. This was true for EDR at baseline (**β = -**

0.03, p = 0.748), EDR reactivity (β = -0.11, p = 0.303), and EDR during the recovery

period (β = 0.07, p = 0.514).

Indirect effects. Within the full model, EDR did not mediate the relationship

between perseverative cognition and somatic symptoms. This was true for EDR at

baseline (**β = 0.00**, p = 0.917), EDR reactivity (**β = -0.02**, p = 0.711), and EDR during the

recovery period (**β = 0.00**, p = 0.870).

Individual Mediators. I analyzed prospective mediation models of each mediator

separately while continuing to control for biological sex and trauma history. The results

of these individual mediations are presented in Table 12 (RSA) and Table 13 (EDR). As

expected, perseverative cognition significantly predicted somatic symptoms such that

individuals who engaged in more perseverative cognition were more likely to experience

somatic symptoms in all mediation models. These analyses did not reveal any significant

mediation pathways. However, the relationship between basal RSA and somatic

symptoms was trending toward significance such that individuals with lower basal RSA

reported more somatic symptoms (β = -0.15; p = 0.064). Contrary to our hypotheses, the

relationship between RSA during the stressor and somatic symptoms was trending toward

significance such that individuals who experienced greater increases in RSA were more likely to report a *lower* level of subsequent somatic symptoms ($\beta = 0.11$; $p = 0.099$). Similarly, RSA during the recovery period significantly predicted subsequent somatic symptoms ($\beta = 0.16$; $p = 0.029$) such that individuals whose RSA was elevated above their baseline following the stressor were *less* likely to experience somatic symptoms. With regard to EDR, perseverative cognition significantly predicted EDR such that individuals who were more likely to engage in perseverative cognition experienced a greater increase in EDR during the stressor task ($\beta = 0.17$; $p = 0.041$). However, EDR did not remain elevated during the recovery period as hypothesized, indicating that the physiological arousal associated with the stressor task did not persist beyond the immediate impact of the stressor.

CHAPTER IV

Discussion

Many theories of somatization focus on worry about one's health as a key factor in the development and maintenance of somatic symptoms. Brosschot, Gerin, and Thayer (2006) have broadened this focus on worry about physical symptoms as a perpetuating factor in somatic symptoms to include all perseverative cognition (e.g. worry and rumination). They posit that the key component in the development of somatic symptoms is not the act of worrying about health specifically, but the perseverative nature of these thoughts that result in prolonged physiological activation that may be interpreted as somatic symptoms. The purpose of the present study was to better understand the relationship between perseverative cognition and somatic symptoms. Specifically, I sought to examine whether prolonged physiological activation serves as an explanatory factor in the relationship between perseverative cognition and somatic symptoms. First, I hypothesized that perseverative cognition would prospectively predict somatic symptoms above and beyond anxiety and depression such that individuals who have a greater tendency to engage in perseverative cognition would experience more somatic symptoms. Perseverative cognition was measured prior to the stressor tasks, while somatic symptoms were measured during the two weeks following the stressor task. Second, I hypothesized that physiological activation, operationalized as RSA and EDR, would mediate this relationship. Each of these physiological measures was collected before, during, and after participants completed a stressor task so that both tonic and phasic measures of sympathetic and parasympathetic nervous system activity were accurately represented. Overall, my study examined the hypothesis that the mechanisms through which perseverative cognition is predictive of somatic symptoms are heightened sympathetic

nervous system activity and reduced parasympathetic nervous system activity. With

regard to parasympathetic nervous system activity, I hypothesized that individuals with

higher levels of perseverative cognition would have lower basal RSA, experience greater

decreases in RSA (disengagement of the parasympathetic nervous system) in response to

a stressor, and failure of RSA to return to baseline levels during the recovery period

(indicating failure of the parasympathetic nervous system to re-engage). Additionally, I

predicted that individuals with these patterns of parasympathetic response to a stressor

would experience higher levels of subsequent somatic symptoms. With regard to

sympathetic nervous system activity, I hypothesized that individuals with higher levels of

perseverative cognition would have higher basal EDR, experience greater increases in

EDR during a stressor (indicating over engagement of the sympathetic nervous system),

and failure of EDR to return to baseline levels during the recovery period (indicating

prolonged activation of the sympathetic nervous system).

This study makes a number of unique contributions to the literature on somatic

symptoms. First, because somatic complaints are a common reason patients present for

medical care (Janca, Isaac, & Ventouras, 2006), it is important to understand the

cognitive and physiological vulnerabilities that lead some individuals to report more

somatic symptoms than others. Additionally, though several studies have examined the

relationship between perseverative cognition and somatic symptoms, it has previously

been unclear whether this relationship would remain after controlling for depression and

anxiety. Finally, prior to this study, there has been only very limited evidence for the

relationship between EDR and somatic symptoms, and no published examination of the

relationship between RSA and somatic symptoms. In the following sections, I will describe the outcomes of my hypotheses.

Did perseverative cognition predict subsequent somatic symptoms?

My hypothesis that the positive relationship between perseverative cognition and somatic symptoms would remain after controlling for depression and anxiety was not supported. However, perseverative cognition did significantly predict somatic symptoms in the expected direction when anxiety and depression were not included as covariates. This is consistent with previous studies that have found an association between worry or rumination and somatic symptoms without controlling for anxiety or depression (Borkovec, 1994; Brosschot & Van Der Doef, 2006; Freeston et al, 1996; Lok & Bishop, 1999; Rector & Roger, 1996; Thomsen et al., 2004). Additionally, this finding is not surprising given the overlap of symptomology among anxiety, depression, and somatic symptoms. For example, a population-based analysis of the overlap of symptoms in these categories found that among 2510 individuals who reported a high level of symptoms in one of these areas, 36.4% of cases had a high level of symptoms in another area as well (Kohlmann, Gierk, Hilbert, Brähler, & Löwe, 2016). Similarly, a cross-sectional study comparing somatic symptoms among individuals depressive or anxiety disorder to controls found that somatic symptoms were more prevalent among patients with anxiety or depression than among controls (Bekhuis, Boschloo, Rosmalen, & Schoevers, 2015).

It is also important to note that, in the present study, after controlling for anxiety and depression, the relationship between perseverative cognition and somatic symptoms was significant in the unexpected (negative) direction. It is likely that the variance in somatic symptoms related to the problematic aspects of perseverative cognition may have

significant overlap with the variance accounted for by measures of symptoms of anxiety and depression. Thus, after depression and anxiety are controlled for, only adaptive aspects of perseverative cognition (such as problem solving) remain. These adaptive aspects of perseverative cognition would then be expected to have a negative association with somatic symptoms, as was found in the present study. In the current literature, there are not any studies I am aware of that have directly examined the relationship between perseverative cognition and adaptive cognitive strategies. Similarly, though many studies have examined rumination as a maladaptive cognitive strategy, I am not aware of any studies that have specifically examined the relationship between rumination and more adaptive cognitive strategies. However, there are several studies which suggest that worry does not necessarily preclude adaptive problem-solving strategies. For example, a study of children who reported elevated levels of worry compared to their peers did not display deficits in problem-solving skills (Parkinson & Creswell, 2011). Though worry may be associated with problem solving orientation, it is not necessarily predictive of problem solving skills (Dugas, Letarte, Rhéaume, Freeston, & Ladouceur, 1995). Among individuals with GAD, use of maladaptive problem solving strategies seems to be dependent on emotional state (Pawluk, Koerner, Tallon, & Antony, 2017).

Did physiological activation mediate the relationship between perseverative cognition and somatic symptoms?

First, my hypothesis that basal RSA, RSA reactivity, and RSA recovery would together mediate the relationship between perseverative cognition and somatic symptoms was not supported. This was true regardless of whether anxiety and depression were included as covariates. Post-hoc analysis of individual mediations indicated that when

depression and anxiety were not included in the model, there was not a significant association between perseverative cognition and RSA. Interestingly, this is contrary to the findings of a recent meta-analysis of the physiological concomitants of perseverative cognition (Ottaviani et al., 2016). Specifically, analyses of 18 experimental studies and eight correlational studies both indicated an overall association between perseverative cognition and decreased heart rate variability. One potential explanation for this is that many of the studies that resulted in significant changes in heart rate variability responses to perseverative cognition used state measures of perseverative cognition, while studies using trait measures of perseverative cognition (such as ours) did not yield significant results. Future studies of this relationship should include both state and trait measures of perseverative cognition in order to provide a better understanding of the differential and combined effects of both state and trait perseverative cognition on somatic symptoms.

Post-hoc analyses of individual mediations did indicate that the relationship between basal RSA and somatic symptoms was trending toward significance such that lower basal RSA predicted greater subsequent report of somatic symptoms. This is consistent with previous studies that have found that cardiac autonomic functioning is differentially related to somatic symptoms of depression in patients with stable coronary artery disease (de Jonge, Mangano, & Whooley, 2007), adults over the age of 52 (Tak, Janssens, Dietrich, Slaets, & Rosmalen, 2010) and preadolescents (Bosch, Riese, Ormel, Verhulst, & Oldehinkel, 2009). The present study indicates that this finding could be extended to a novel population, college students. However, the relationship between RSA reactivity was trending toward significance such that greater decreases in RSA predicted lower report of subsequent somatic symptoms. Similarly, RSA recovery

significantly predicted somatic symptoms such that individuals whose RSA remained

farther below their baseline reported higher rates of subsequent somatic symptoms. This

is contrary to our hypotheses that individuals whose parasympathetic nervous system

overreacted to the stressor task and then failed to re-engage following the stressor task

would experience higher levels of subsequent somatic symptoms. In fact, this study

revealed that individuals whose parasympathetic nervous system displayed less

engagement during the stressor period and greater engagement during the recovery period

following the stressor experienced higher levels of subsequent somatic symptoms. One

possible explanation for this is that medical conditions were a confounding factor. As

medical conditions were not measured in the present study, they may account for some of

the symptoms participants reported the PHQ-15. With regard to RSA reactivity, one

possibility is that a blunted RSA response is predictive of subsequent somatic symptoms.

A number of previous studies have suggested that there is a "U-shaped" relationship

between RSA and internalizing psychopathology such as major depressive disorder

(Bylsma, Salomon, Taylor-Clift, Morris, & Rottenberg, 2014; Rottenberg, Clift, Bolden,

& Salomon, 2007; Yaroslavsky, Bylsma, Rottenberg, & Kovacs, 2013). The findings

from the present study indicate that a blunted, rather than elevated, RSA response may be

related to somatic symptoms. With regard to RSA recovery, individuals with blunted

RSA responses to a stressor would have less of a change in parasympathetic nervous

system engagement to recover from. Another possibility is that though the

parasympathetic nervous system re-engages effectively following a stressful event,

individuals who are prone to somatic symptoms may be hypervigilant to any bodily

changes (increased digestion, decreased heart rate, etc.) misinterpret these changes as

somatic symptoms. Additionally, it may be important to explore alternate ways of considering the role of physiological arousal measured via RSA, such as the interaction of RSA measurements across phases of the stressor task. Though RSA reactivity and recovery did not predict somatic symptoms in the expected direction, the pattern of parasympathetic response to a stressor may be an important predictor of somatic symptoms. Overall, the results of the present study indicate that the role of RSA in somatic symptoms warrants further explanation.

Second, my hypothesis that basal EDR, EDR reactivity, and EDR recovery would together mediate the relationship between perseverative cognition and somatic symptoms was not supported. This was true regardless of whether anxiety and depression were included as covariates. Post-hoc analysis of individual mediations also indicated that when depression and anxiety were not included in the model, neither basal EDR nor EDR recovery were significantly associated with perseverative cognition. This is consistent with several studies have reported null effects for the relationship between perseverative cognition and skin-conductance level (Dua & King, 1987; Segerstrom et al., 1999; Vickers & Vogeltanz-Holm, 2003). However, perseverative cognition did significantly predict EDR reactivity such that individuals who were more likely to engage in perseverative cognition exhibited greater increases in EDR responses during the stressor task. In the present study, these findings indicate that individuals with a greater tendency to engage in perseverative cognition did so during the stressor task, resulting in elevated sympathetic nervous system responding. This is consistent with previous literature that individuals with symptoms of internalizing disorder (such as depression and anxiety) exhibit greater electrodermal activity when compared to a control group (Zisner &

Beauchaine, 2015). However, EDR reactivity did not significantly predict somatic symptoms. This is contrary to the perseverative cognition hypothesis, which posits that prolonged physiological activation is the mechanism by which stress is translated into a pathogenic state such as somatic symptoms. One potential explanation for this is that the present study used a laboratory stressor of speech preparation in order to provoke a stress reaction from participants. I assumed that individuals have a tendency to perseverate would do so in this context; however, I did not measure this directly. It is possible that even individuals with a tendency to perseverate did not continue to engage in perseverative thoughts about this stressor during the allowed recovery period, and thus did not experience continued physiological arousal. Future studies should seek to examine physiological arousal in response to spontaneous perseveration about authentic stressors. Additionally, a state measure of perseverative cognition may provide additional insight in to the relationship between perseverative cognition and EDR.

Clinical Application

A number of studies, including this one, have demonstrated a strong relationship between perseverative cognition and somatic symptoms. When attempting to treat patients with somatic symptoms, it is important for clinicians to address the relationship between cognition and these symptoms, rather than simply treating the physical ailments the patient is presenting with. Additionally, the results of this study suggest that there is significant overlap between the symptoms of anxiety, depression, and somatic symptoms. Therefore, when a patient presents with a complaint of somatic symptoms, it is also important to screen for and treat any underlying anxiety or depressive disorder that may be contributing to the manifestation of somatic symptoms.

This study also demonstrated that trait perseverative cognition is associated with increased activation of the sympathetic nervous system (as measured by EDR) during a stressor. While not empirically tested in the context of this study, it is likely that people who tend to engage in more perseverative cognition were likely engaging in perseverative cognition during the stressor task, resulting in greater increases in electrodermal responding. From a cognitive behavioral perspective, this underlines the importance of teaching patients more adaptive cognitive strategies to be used during stressors in order to limit activation of the sympathetic nervous system. Additionally, contrary to my hypothesis, re-engagement of the parasympathetic nervous system to after a stressor (as measured by RSA) was associated with higher reports of subsequent somatic symptoms. This indicates that individuals who are prone to somatic symptoms may be hypervigilant to any bodily changes, including those associated with parasympathetic activity (increased digestion, decreased heart rate, etc.), and misinterpret these changes as somatic symptoms. Clinically, patients may benefit from interventions that allow for normalization of bodily sensations such as biofeedback and cognitive restructuring.

Limitations and Future Directions

There are several limitations to consider in the interpretation of the results from this study. First, participants in the present study were college students, who were generally young, healthy, Caucasian females. Thus, the results cannot necessarily be generalized to other populations including those of other races, ages, sexes, or who have medical or psychiatric diagnoses. Future research should examine these variables in clinical populations, as well as among samples that are more diverse with regard to age, race/ethnicity, and sex. Second, the majority of measures in this study were self-report.

Thus, the information provided may not be reliable. Additionally, the self-report measures used in this study have considerable overlap in item content. For example, the PSWQ and GAD-IV both emphasize the role of worry, making it difficult to tease out the difference between anxiety (a planned covariate) and worry (an aspect of perseverative cognition). Future studies of the relationship between somatic symptoms that seek to control for depression and anxiety should consider developing more clearly delineated operational definitions of each construct and choose measures (or items from measures) accordingly. Third, though our study was prospective in nature, the time-frame that measures were administered across was relatively short. Additionally, perseverative cognition was measured only as a trait variable at baseline. Though somatic symptoms were assessed at six time points, we did not examine fluctuations in reporting of these symptoms over time. Therefore, it is possible that fluctuations in perseverative cognition over time are associated with fluctuations in somatic symptoms over time. Future research should continue to utilize prospective experimental designs and data analytic strategies in order to further examine the causal relationship between perseverative cognition and somatic symptoms. Fourth, though the measures of RSA and EDR were successive, in the present study they were treated as concurrent mediators. Future research should examine whether patterns of autonomic responding over time provide an explanation for the established relationship between perseverative cognition and somatic symptoms. Finally, the present study did not examine the combined impact of sympathetic and parasympathetic functioning as a mediator of this relationship. Notably, we found associations between perseverative cognition and EDR, as well as between RSA and somatic symptoms. This indicates that both sympathetic and parasympathetic

responses are important in explaining the relationship between perseverative cognition and somatic symptoms. Future studies should seek to explore the roles of these complementary systems in tandem, taking into account both the reactivity and regulatory functions of the autonomic nervous system.

In conclusion, the present study did not find compelling evidence that physiological activation, as indexed by RSA or EDR, was an explanatory factor in the relationship between perseverative cognition and somatic symptoms. However, in post hoc analyses a pattern emerged which suggested that perseverative cognition is related to sympathetic nervous system functioning, whereas parasympathetic nervous system functioning was related to reporting of somatic symptoms. Therefore, future research could benefit from exploration of the combined impact of sympathetic and parasympathetic functioning on the relationship between perseverative cognition and somatic symptoms.

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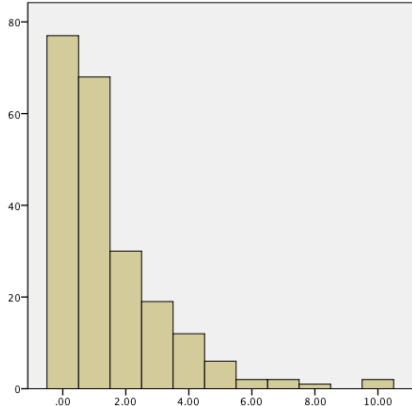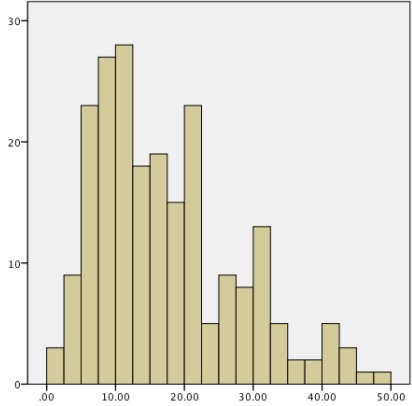
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TABLES

Table 1
Participant Demographics
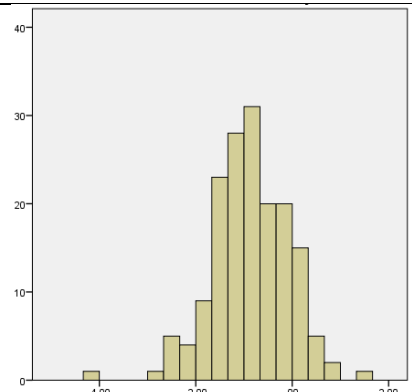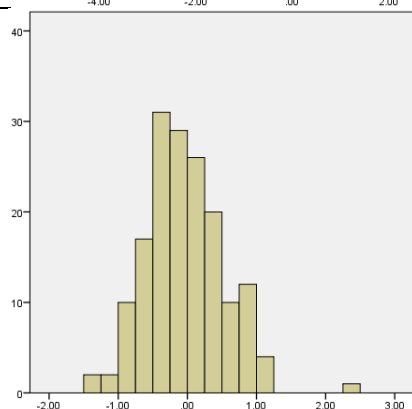
	N	%			N	%			N	%
Biological Sex			*Age*				*Ethnicity*			
Male	34	15.5		18	103	46.8		Caucasian/White	144	65.5
Female	186	84.5		19	57	25.9		Asian	32	14.5
				20	25	11.4		African American	8	3.6
Gender				21	14	6.4		Hispanic/Latino	20	9.1
Male	33	15.0		22	6	2.7		Mixed Ethnicity	13	5.9
Female	183	83.2		23	2	0.9		Other/Missing	3	1.4
Transgender	4	1.8		24	1	0.5		*Race*		
				26	1	0.5		Caucasian	157	71.4
Year in College				27	2	0.9		African American	10	4.5
Freshman	145	65.9		28	1	0.5		Asian American	29	13.2
Sophomore	35	15.9		29	1	0.5		Native American	1	0.5
Junior	25	11.4		39	1	0.5		Pacific Islander	5	2.3
Senior	9	4.1	Missing		6	2.7		Mixed Race	10	4.5
Other	6	2.7						Missing	8	3.6

Table 2
Assessing Univariate Normality of Continuous Variables
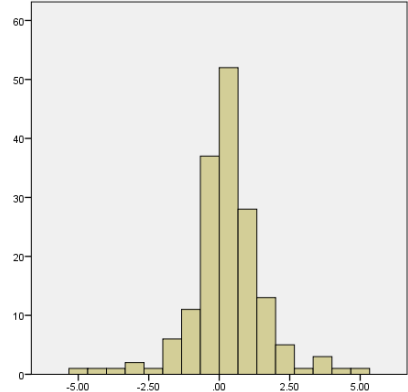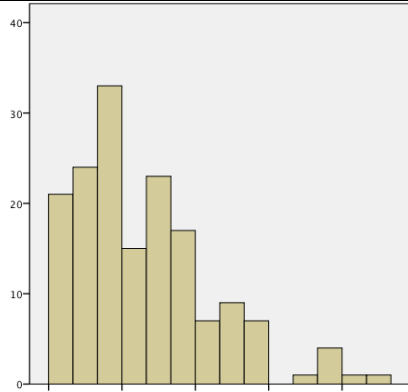
Variables	Histogram	Shapiro-Wilk Test of Normality			Kurtosis		Skewness	
Baseline		R	Df	P	kurtosis	z kurt	skewness	z skew
Trauma History (BTQ)		0.77***	219	<0.001	5.40***	16.51	2.01***	12.24
Depressive Symptoms (CES-D)		0.94***	219	<0.001	0.08	0.24	0.83***	5.06

Anxiety Symptoms (GAD-Q)		0.98**	216	0.002	-0.89**	-2.69	-0.09	-0.54
Trait Brooding Rumination (RRS-B)		0.97***	220	<0.001	-0.81	-2.48	0.09	0.57

		R	Df	P	kurtosis	z kurt	skewness	z skew
Trait Worry (PSWQ)		0.97***	215	<0.001	-0.32	-1.93	-0.78*	-2.35
Stressor Task								
Basal RSA		0.98	160	0.057	0.88*	2.35	0.04	0.21

RSA Reactivity		0.99	160	0.081	1.24***	3.29	-0.38*	-1.98
RSA Recovery		0.98*	160	0.043	0.98	2.60	0.46*	2.43

Basal EDR		0.80***	160	<0.001	6.35***	16.88	2.09***	11.05
EDR Reactivity		0.98*	160	0.035	1.41	3.74	-0.09	-0.49

		R	Df	P	kurtosis	z kurt	skewness	z skew
EDR Recovery		0.92	160	<0.001	3.99***	10.58	-0.30	-1.57
Somatic Symptoms		*R*	*Df*	*P*	kurtosis	*z* kurt	skewness	*z* skew
Day 1		0.93***	163	<0.001	0.91*	2.42	0.97***	5.08

| Day 2 | | 0.92*** | 160 | <0.001 | 0.30 | 0.80 | 0.93*** | 4.84 |

| Day 3 | | 0.94*** | 153 | <0.001 | -0.09 | -0.23 | 0.73*** | 3.72 |

| Day 4 | | 0.88*** | 153 | <0.001 | 3.63*** | 9.31 | 1.53*** | 7.79 |

| Day 5 | | 0.92*** | 157 | <0.001 | 0.84* | 2.18 | 0.94*** | 4.84 |

Day 6   0.89***   146   <0.001   0.54   1.35   1.09***   5.41
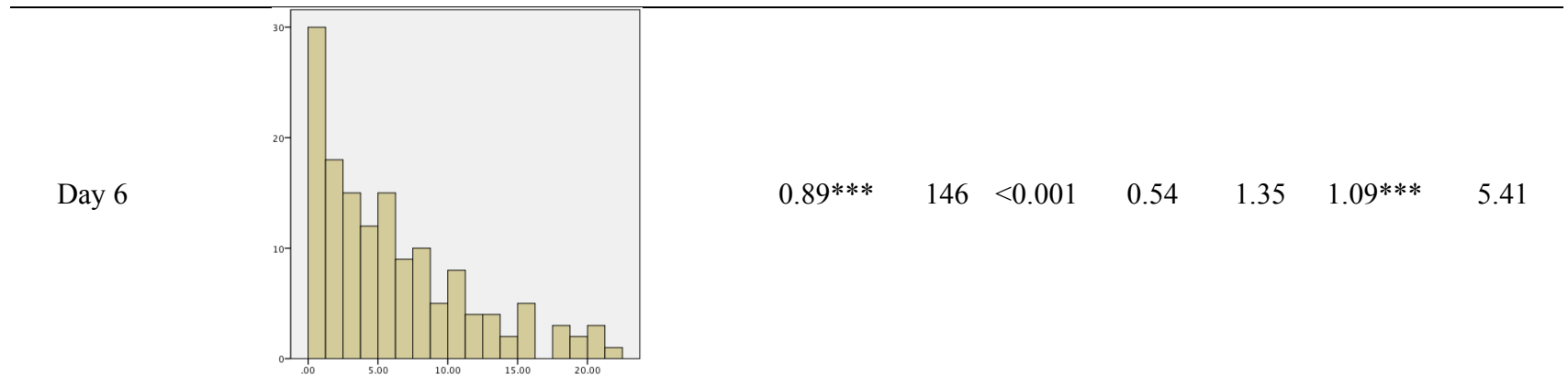
Note. R is the Shapiro-Wilk Test Statistic. To facilitate interpretation, z values for kurtosis and skewness are calculated by dividing by their respective standard error. An absolute value greater than 1.96 is significant $p < .05$, above 2.58 is significant at $p < .01$, and above 3.29 is significance at $p < .001$. * denotes $p < .05$, ** $p < .01$, *** $p <.001$.

Table 3
Bivariate Correlations and Descriptive Statistics among Variables at Baseline

Variable	N	1	2	3	4	5	6	M (SD)
1. Gender (0 = male, 1 = female)	220							0.85 (0.36)
2. Age	214	-0.09						19.63 (2.10)
3. Trauma History (BTQ)	219	-0.13	0.18**					1.45 (1.76)
4. Depressive Symptoms (CES-D)	219	0.11	0.03	0.15*				17.13 (10.22)
5. Anxiety Symptoms (GAD-IV)	216	0.20**	-0.00	0.14*	0.57**			6.36 (2.81)
6. Trait Rumination (RRS-B)	220	0.15*	-0.12	0.17*	0.55**	0.44**		12.04 (3.53)
7. Trait Worry (PSWQ)	215	0.30**	-0.15*	0.03	0.50**	0.75**	0.45**	56.84 (13.59)

*Note. *p < .05, ** p < .01.*

Table 4
Bivariate Correlations and Descriptive Statistics among Variables at Lab Visit

Variable	N	1	2	3	4	5	6	7	M (SD)
1. Gender	220								0.85 (0.36)
2. Age	214	-0.09							19.63 (2.10)
3. Basal RSA	166	-0.09	-0.07						6.80 (0.96)
4. RSA During Stressor	165	-0.09	-0.18*	0.67**					5.94 (0.98)
5. RSA Recovery	164	-0.05	-0.11	0.82**	0.71**				6.79 (0.91)
6. Basal EDR	165	-0.11	0.07	-0.09	-0.03	-0.01			1.58 (1.56)
7. EDR During Stressor	164	-0.09	0.04	0.01	0.10	0.10	0.46**		3.02 (1.44)
8. EDR Recovery	164	-0.13	-0.04	0.03	0.05	0.10	0.63**	0.56**	1.87 (1.46)

*Note. *p < .05, ** p < .01.*

Table 5
Bivariate Correlations and Descriptive Statistics among Daily Somatic Symptoms

Variable	N	1	2	3	4	5	6	7	M (SD)
1. Gender	220								0.85 (0.36)
2. Age	214	-0.09							19.63 (2.09)
3. Day 1 Somatic Symptoms	163	0.20**	0.08						6.60 (4.68)
4. Day 2 Somatic Symptoms	160	0.16*	0.05	0.80**					5.58 (4.41)
5. Day 3 Somatic Symptoms	153	0.20*	0.05	0.70**	0.78**				5.91 (4.52)
6. Day 4 Somatic Symptoms	153	0.17*	0.06	0.56**	0.74**	0.72**			5.34 (4.67)
7. Day 5 Somatic Symptoms	157	0.15	0.05	0.72**	0.81**	0.74**	0.77**		5.90 (4.74)
8. Day 6 Somatic Symptoms	146	0.11	0.10	0.64**	0.74**	0.77**	0.76**	0.85**	5.96 (5.41)

*Note *p < .05, ** p < .01.*

Table 6

Confirmatory Factor Analyses

Measure	Model	AIC	BIC	χ^2 (df)		χ^2 Ratio	RMSEA	CFI	SRMR
Ruminative Response Scale (RRS)	Single factor, no covariation among items	2772.32	2823.23	22.17	(5)	4.43	0.13	**0.95**	**0.05**
	RRS5 with RRS15	**2756.80**	**2811.10**	4.64	(4)	**1.16**	**0.03**	0.99	0.02
Pennsylvania State Worry Questionnaire (PSWQ)	Single factor, no covariations among items	9334.03	9496.04	313.55	(104)	3.02	0.10	0.90	**0.06**
	Two factors, no covariations among items	9287.94	9463.45	259.46	(100)	2.59	0.09	0.92	**0.05**
	Two factors, PSWQ15 with PSWQ14	9254.78	9433.67	224.30	(99)	2.27	0.08	0.94	**0.04**
	Two factors, PSWQ15 with PSWQ7, PSWQ14	9228.86	9411.12	196.38	(98)	**2.00**	0.07	**0.95**	**0.04**
	Two factors, PSWQ15 with PSWQ7, PSWQ14 & PSWQ9 with PSWQ16	**9199.28**	**9384.92**	164.80	(97)	**1.70**	**0.06**	0.97	**0.04**
Somatic Symptoms (PHQ-15)	No covariation among days	4769.26	4826.22	55.29	(9)	5.81	0.17	**0.95**	**0.03**
	Covariation among consecutive days	**4743.14**	**4815.93**	19.17	(4)	4.79	0.15	**0.98**	0.02

Note All CFAs were performed with bootstrap resampling procedures (5000 iterations specified). Statistics indicating best fit (for AIC and BIC) or adequate fit (for χ^2 Ratio, RMSEA, CFI, and SRMR) are **bolded**.

Table 7

Bootstrap Analysis of Direct and Indirect Effects for Hypothesized RSA Model

IV	→ Mediator	→ DV	B (standardized path coefficient and product)						SE	Lower	Upper	Two-tailed significance	
										95% CI			
Indirect Effects													
PC	→ bRSA	→ SS	-0.01	X	-0.08	=	0.00		0.01	-0.02	0.03	0.967	
PC	→ sRSA	→ SS	-0.12	X	0.09	=	-0.01		0.01	-0.05	0.00	0.343	
PC	→ rRSA	→ SS	-0.11	X	0.07	=	-0.01		0.01	-0.04	0.01	0.486	
Sum of indirect effects							-0.02		0.02	-0.07	0.02	0.407	
Direct Effects													
PC	→ SS						-0.23		0.07	-0.36	-0.08	0.001	**
PC	→ bRSA						-0.01		0.09	-0.19	0.18	0.956	
PC	→ sRSA						-0.12		0.08	-0.27	0.05	0.147	
PC	→ rRSA						-0.11		0.10	-0.29	0.09	0.269	
bRSA	→ SS						-0.08		0.06	-0.21	0.04	0.202	
sRSA	→ SS						0.09		0.06	-0.02	0.21	0.099	‡
rRSA	→ SS						0.07		0.06	-0.05	0.19	0.267	

Notes. PC =perseverative cognition, bRSA = basal RSA, sRSA = RSA reactivity during stressor, rRSA = RSA recovery, SS = somatic symptoms. ‡= p<.10, * *p* < .05, ** *p* < 0.01

Table 8

Bootstrap Analysis of Direct and Indirect Effects for Hypothesized EDR Model

IV	→ Mediator	→	DV	β (standardized path coefficient and product)					SE	95% CI Lower	95% CI Upper	Two-tailed significance
Indirect Effects												
PC	→ bEDR	→	SS	-0.10	X	-0.03	=	0.00	0.03	-0.01	0.05	0.926
PC	→ sEDR	→	SS	0.18	X	-0.06	=	-0.01	0.06	-0.06	0.01	0.846
PC	→ rEDR	→	SS	0.06	X	0.07	=	0.00	0.03	-0.01	0.18	0.876
Sum of indirect effects								-0.00	0.10	-0.04	0.03	0.972
Direct Effects												
PC	→ SS							-0.23	0.13	-0.37	-0.06	0.074 ‡
PC	→ bEDR							-0.10	0.12	-0.30	0.11	0.430
PC	→ sEDR							0.18	0.13	-0.04	0.40	0.144
PC	→ rEDR							0.06	0.12	-0.12	0.26	0.634
bEDR	→ SS							-0.03	0.08	-0.16	0.11	0.688
sEDR	→ SS							-0.06	0.10	-0.21	0.10	0.546
rEDR	→ SS							0.07	0.08	-0.07	0.23	0.341

Notes. PC =perseverative cognition, bEDR = EDR at baseline, sEDR = EDR reactivity during stressor, rEDR = EDR during recovery period, SS = somatic symptoms. ‡ = p<.10, * $p < .05$, ** $p < 0.01$

Table 9

Model Comparison for Total Effect

Covariates	AIC	BIC	χ^2 (df)	χ^2 Ratio	RMSEA	CFI	SRMR
Hypothesized Model							
Sex, BTQ, GAD, CESD	**16459.96**	**16790.28**	768.75 (415)	**1.85**	0.063	0.900	0.218
Alternative Models							
Sex, BTQ, GAD	16512.68	16840.98	684.29 (389)	**1.76**	**0.059**	0.915	0.193
Sex, BTQ, CESD	16534.68	16862.08	621.03 (389)	**1.60**	**0.052**	0.932	0.138
Sex, BTQ	16600.94	16926.29	468.39 (363)	**1.29**	**0.036**	**0.968**	**0.072**

Note All regressions were performed with bootstrap resampling procedures (5000 iterations specified). Statistics indicating best fit (for AIC and BIC) or adequate fit (for χ^2 Ratio, RMSEA, CFI, and SRMR) are **bolded**.

Table 10

Bootstrap Analysis of Direct and Indirect Effects for Alternative RSA Model

IV → Mediator → D V				B (standardized path coefficient and product)						SE	95% CI Lower	95% CI Upper	Two-tailed significance	
Indirect Effects														
PC	→	bRSA	→	SS	-0.01	X	-0.10	=	0.00	0.01	-0.02	0.03	0.961	
PC	→	sRSA	→	SS	-0.12	X	0.03	=	-0.00	0.01	-0.04	0.01	0.786	
PC	→	rRSA	→	SS	-0.11	X	0.12	=	-0.01	0.01	-0.07	0.01	0.433	
Sum of indirect effects									-0.02	0.03	-0.08	0.03	0.580	
Direct Effects														
PC	→	SS							0.38	0.08	0.22	0.52	<0.001	**
PC	→	bRSA							-0.01	0.09	-0.18	0.18	0.945	
PC	→	sRSA							-0.12	0.08	-0.27	0.04	0.144	
PC	→	rRSA							-0.11	0.10	-0.29	0.10	0.265	
bRSA	→	SS							-0.10	0.10	-0.29	0.08	0.275	
sRSA	→	SS							0.03	0.08	-0.14	0.18	0.711	
rRSA	→	SS							0.12	0.08	-0.04	0.30	0.173	

Notes. PC =perseverative cognition, bRSA = basal RSA, sRSA = RSA reactivity during stressor, rRSA = RSA recovery, SS = somatic symptoms. [‡]= p<.10, * $p < .05$, ** $p < 0.01$

Table 11
Bootstrap Analysis of Direct and Indirect Effects for Alternative EDR Model

IV	→ Mediator	→ DV	β (standardized path coefficient and product)					SE	95% CI Lower	95% CI Upper	Two-tailed significance	
Indirect Effects												
PC	→ bEDR	→ SS	-0.09	X	-0.03	=	0.00	0.03	-0.02	0.05	0.917	
PC	→ sEDR	→ SS	0.18	X	-0.11	=	-0.02	0.06	-0.11	0.01	0.711	
PC	→ rEDR	→ SS	0.06	X	0.07	=	0.00	0.02	-0.01	0.05	0.870	
Sum of indirect effects							-0.01	0.10	-0.08	0.02	0.886	
Direct Effects												
PC	→ SS						0.37	0.13	0.20	0.51	0.003	**
PC	→ bEDR						-0.09	0.11	-0.27	0.11	0.389	
PC	→ sEDR						0.18	0.11	-0.03	0.37	0.098	‡
PC	→ rEDR						0.06	0.10	-0.13	0.25	0.591	
bEDR	→ SS						-0.03	0.10	-0.23	0.18	0.748	
sEDR	→ SS						-0.11	0.11	-0.31	0.07	0.303	
rEDR	→ SS						0.07	0.11	-0.11	0.28	0.514	

Notes. PC =perseverative cognition, bEDR = EDR at baseline, sEDR = EDR reactivity during stressor, rEDR = EDR during recovery period, SS = somatic symptoms. ‡ = p<.10, * $p < .05$, ** $p < 0.01$

Table 12

Bootstrap Analysis of Direct and Indirect Effects for Individual RSA Mediations

Path							β (standardized path coefficient and product)	SE	95% CI Lower	95% CI Upper	Two-tailed significance	
Basal RSA												
PC	→	SS					0.36	0.07	0.21	0.49	<0.001	**
PC	→	bRSA					-0.02	0.09	-0.17	0.16	0.858	
bRSA	→	SS					-0.15	0.08	-0.31	0.00	0.064	‡
PC	→	bRSA	→	SS	-0.02 X -0.15 =	0.00	0.01	-0.02	0.03	0.873	
RSA Reactivity During Stressor												
PC	→	SS					0.37	0.07	0.21	0.50	<0.001	**
PC	→	sRSA					-0.11	0.07	-0.26	0.03	0.123	
sRSA	→	SS					0.11	0.07	-0.03	0.24	0.099	‡
PC	→	sRSA	→	SS	-0.11 X 0.11 =	-0.01	0.01	-0.05	0.00	0.373	
RSA During Recovery Period												
PC	→	SS					0.37	0.07	0.22	0.50	<0.001	**
PC	→	rRSA					-0.10	0.09	-0.28	0.08	0.259	
rRSA	→	SS					0.16	0.08	0.02	0.31	0.029	*
PC	→	rRSA	→	SS	-0.10 X 0.16 =	-0.02	0.02	-0.07	0.01	0.336	

Notes. PC =perseverative cognition, bRSA = basal RSA, sRSA = RSA reactivity during stressor, rRSA RSA recovery, SS = somatic symptoms. ‡ = p<.10, * $p < .05$, ** $p < 0.01$

Table 13

Bootstrap Analysis of Direct and Indirect Effects for Individual EDR Mediations

Path									β (standardized path coefficient and product)	SE	95% CI Lower	95% CI Upper	Two-tailed significance
Basal EDR													
PC	→	SS							0.36	0.07	0.20	0.49	<0.001 **
PC	→	bEDR							-0.08	0.08	-0.23	0.08	0.313
bEDR	→	SS							-0.01	0.09	-0.17	0.17	0.915
PC	→	bEDR	→	SS	-0.08	X	-0.01	=	0.00	0.01	-0.02	0.03	0.940
EDR Reactivity During Stressor													
PC	→	SS							0.37	0.08	0.21	0.50	<0.001 **
PC	→	sEDR							0.17	0.08	0.00	0.33	0.041 *
sEDR	→	SS							-0.05	0.08	-0.21	0.11	0.539
PC	→	sEDR	→	SS	0.17	X	-0.05	=	-0.01	0.02	-0.06	0.01	0.595
EDR During Recovery Period													
PC	→	SS							0.36	0.07	0.21	0.49	<0.001 **
PC	→	rEDR							0.04	0.08	-0.11	0.20	0.602
rEDR	→	SS							0.03	0.08	-0.13	0.20	0.729
PC	→	rEDR	→	SS	0.04	X	0.03	=	0.00	0.01	-0.01	0.03	0.869

Notes. PC =perseverative cognition, bEDR = EDR at baseline, sEDR = EDR reactivity during stressor, rEDR = EDR during recovery period, SS = somatic symptoms. ‡= p<.10, * $p < .05$, ** $p < 0.01$

FIGURES
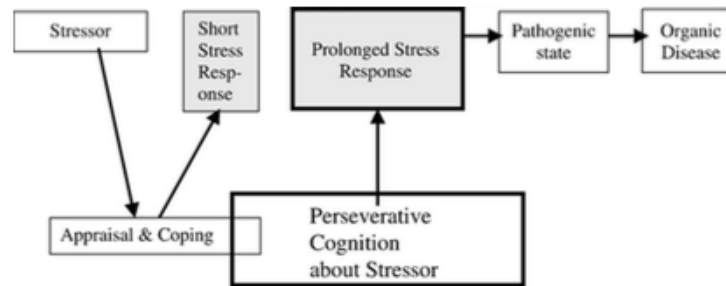
Fig. 1. Basic model of perseverative cognition mediating the effect of stress on organic disease. The difference in size of the two 'stress response' boxes reflects the notion that the hypothesized prolonged response lasts substantially longer than does the response during and immediately following a stressor. It does not reflect the exact relative difference in passage of time.

Figure 1. Brosschot, Gerin, and Thayer's (2006) perseverative cognition hypothesis

(reprinted from Brosschot et al., 2006).

Figure 2. Theoretical model for the present study. Perseverative cognition will predict the presence of somatic symptoms. This relationship will be mediated by physiological activation.
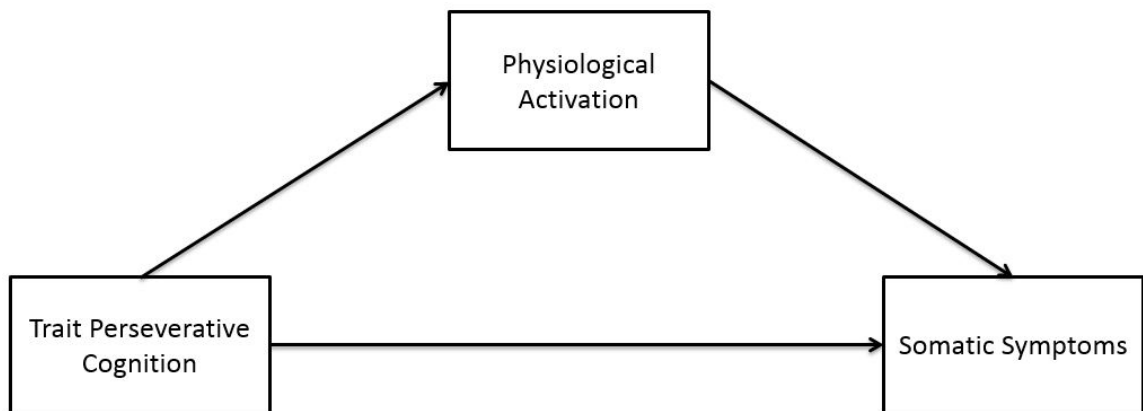
Figure 3. Hypothesized measurement models for examination of the mediating

relationship of physiological activation on the association between perseverative

cognition and somatic symptoms.

Figure 3a. Hypothesized measurement model for the mediating relationship of RSA.
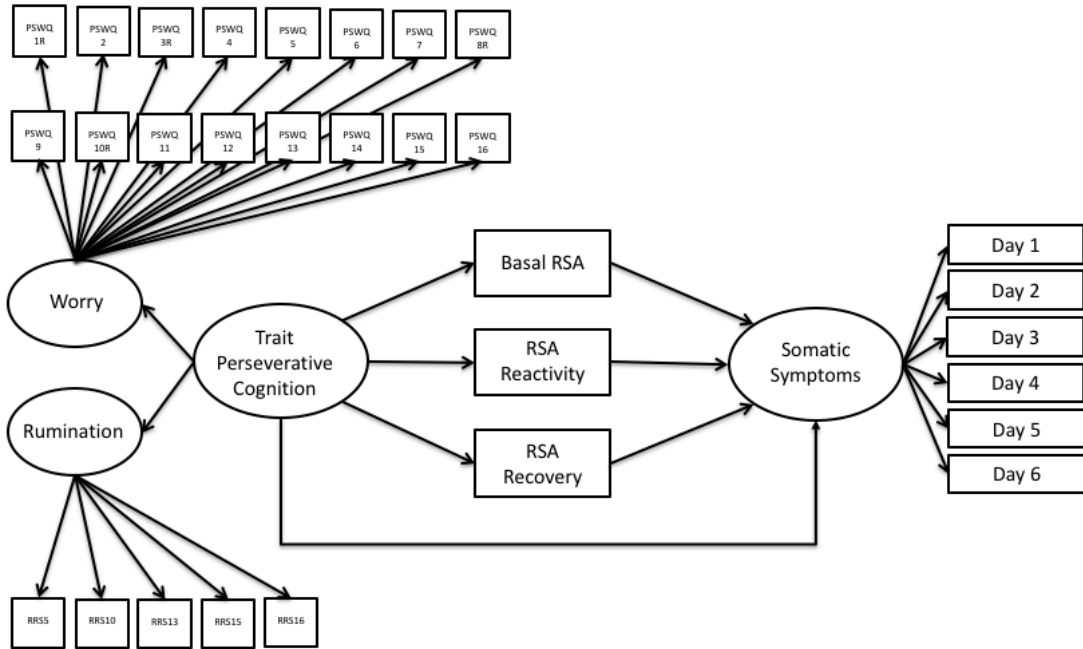
Figure 3b. Hypothesized measurement model for the mediating relationship of EDR.
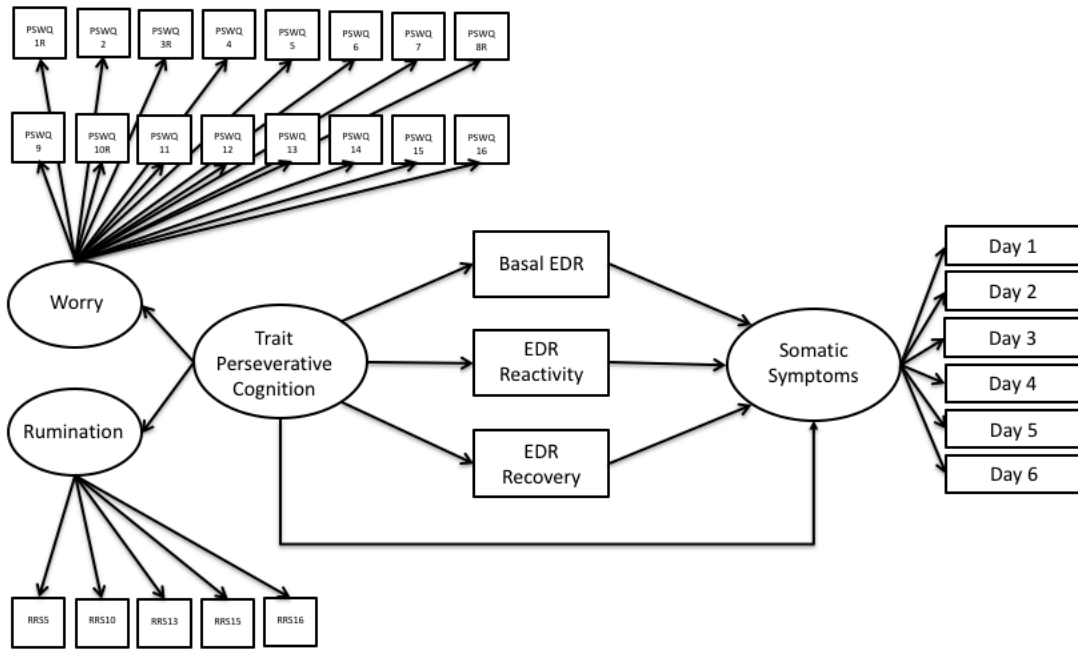
Figure 4. Measurement models of latent variables based on confirmatory factor analyses.

Figure 4a. Measurement model of rumination.
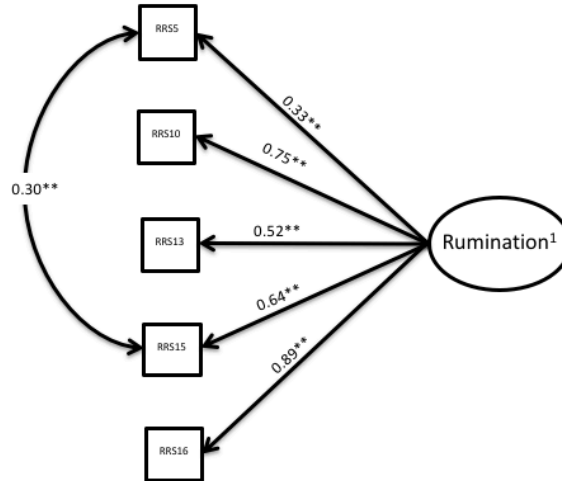
Figure 4b. Measurement model of worry.
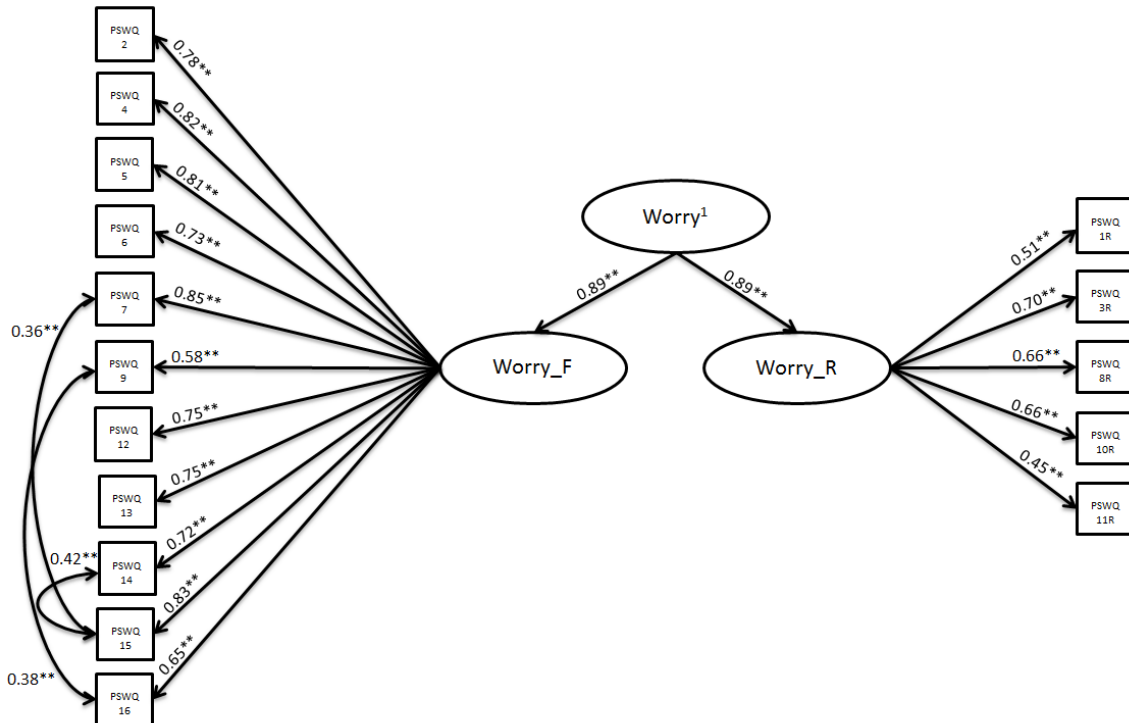
Figure 4c. Measurement model of daily somatic symptoms.
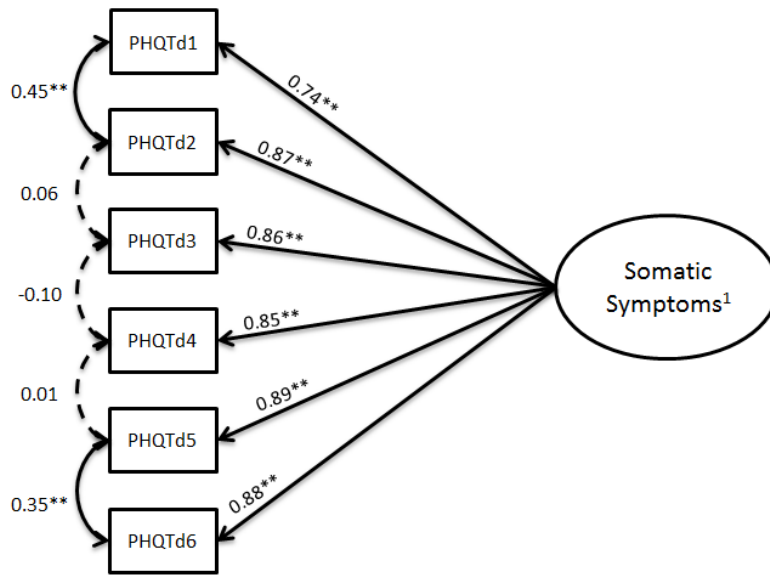
Figure 5. Test of hypothesized mediation models including gender, trauma history, anxiety, and depression as covariates.

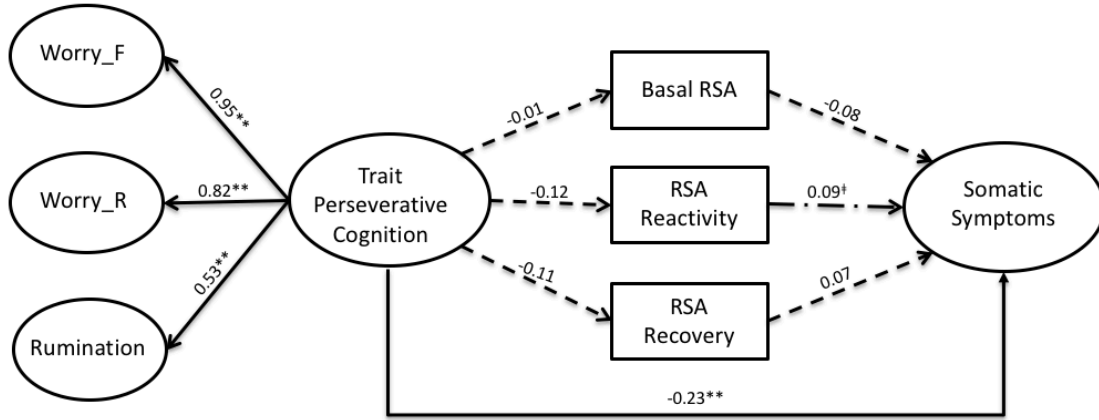*Figure 5a.* Test of hypothesized RSA mediation model.

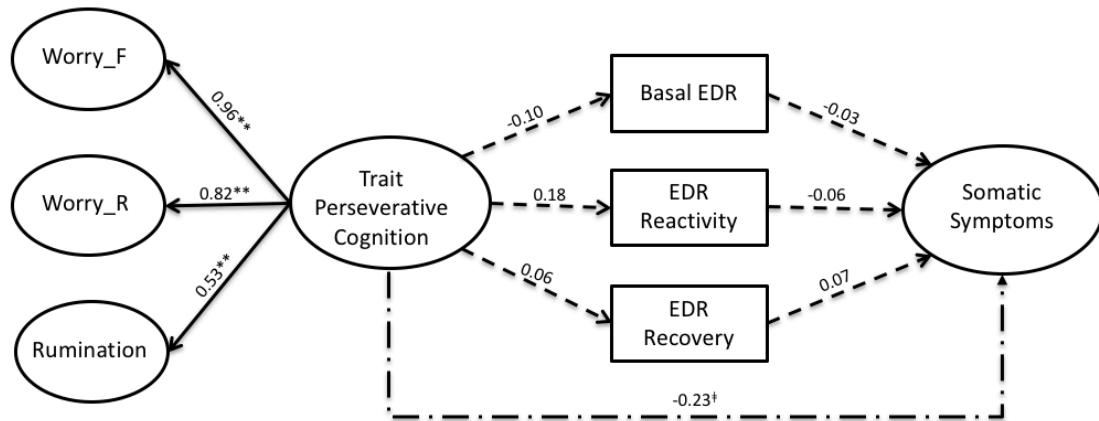*Figure 5b.* Test of hypothesized EDR mediation model.

Figure 6. Test of mediation models including gender and trauma history covariates.
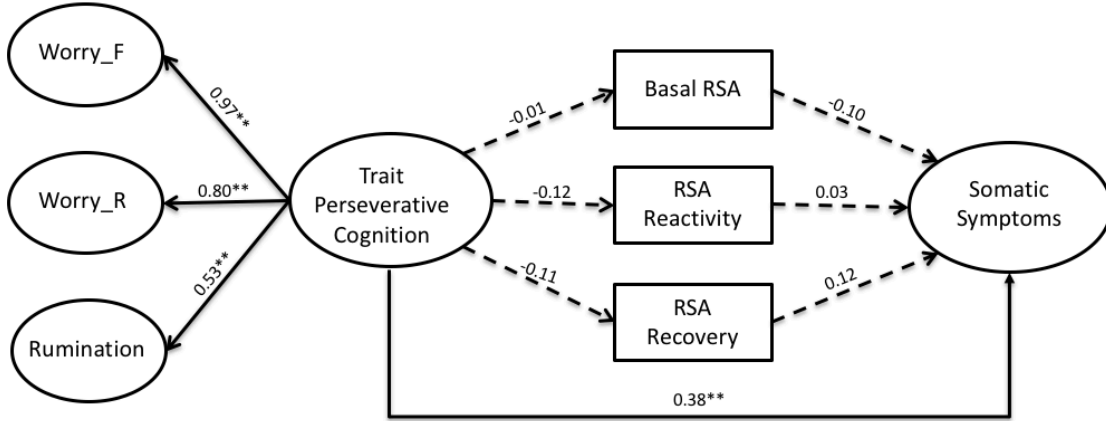
Figure 6a. Test of RSA mediation model.

Figure 6b. Test of EDR mediation model.