January 1st, 2016

Stress and Somatic Symptoms: Rumination and Negative Affect as Moderators

Melissa Joy Garner
Seattle Pacific University

Stress and Somatic Symptoms: Rumination and Negative Affect as Moderators

Melissa J. Garner

A dissertation 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

2016

Approved by:

Amy Mezulis, Ph.D.
Associate Professor of Clinical Psychology
Dissertation Chair

David G. Stewart, Ph.D.
Professor of Clinical Psychology
Committee Member

Thane Erickson, Ph.D.
Associate Professor of Clinical Psychology
Committee Member

Reviewed by:

David G. Stewart, Ph.D.
Chair, Clinical Psychology

Mícheál D. Roe, Ph.D.
Dean, School of Psychology,
Family & Community

Table of Contents

Table of Contents ii
List of Tables iv
List of Figures v
List of Appendices vi
Abstract 1

A. Chapter I: Introduction and Literature Review 3
1. Introduction 3
2. Literature Review 6
2.1. Defining Somatic Symptoms, Syndromes, and Disorders 6
2.2. Current Theories of Somatization 8
2.3. Stress 14
2.4. Negative Affect 16
2.5. Rumination 17
3. Current Study 19
B. Chapter II: Method 21
1. Participants 21
2. Power Analysis 21
3. Procedure 22
4. Measures 22
4.1. Trait Negative Affect 22
4.2. Rumination 23
4.3. Stressors 23
4.4. Somatic Symptoms 24
4.5. Depressive Symptoms 24
5. Data Analytic Plan 25
C. Chapter III: Results 26
1. Data Screening and Analysis 26
1.1. Data Preparation 26
1.2. Participants 26
1.3. Descriptive 27
2. Prospective Analyses 30
2.1. Analysis 30
D. Chapter IV: Discussion 35
1. Was Stress Associated with Greater Somatic Symptoms? 36
2. Do Rumination and/or Negative Affect Moderate the Relationship Between Stress and Somatic Symptoms? 37
2.1. Negative Affect 37
2.2. Rumination 40
2.3. Interaction between NA and Rumination 41
3. Clinical Application 42
4. Limitations and Future Directions 43
4.1. Measurement 43
4.2. Theory 44

4.3. Sample....46
4.4. Next Steps....47
E. References....49

List of Tables

Table 1
Participant Demographics……...… 27
Table 2
Bivariate Correlations and Descriptives among Variables…… 28
Table 3
Descriptive Statistics by Race and Sex……29
Table 4
Final Estimation of Fixed Effects for Stress, Rumination, and Negative Affect to Somatic Symptoms with Baseline Depression…… 32
Table 5
Final Estimation of Fixed Effects for Stress, Rumination, and Negative Affect to Somatic Symptoms without Baseline Depression…… 32
Table 6
Fixed Effects of Stress and Rumination to Somatic Symptoms…… 33
Table 7
Fixed Effects of Stress and Negative Affect to Somatic Symptoms…… 34

List of Figures

Figure 1. Hypothesized model of relationships between stressors, trait negative affect, rumination, and somatic symptoms. 20

Melissa Garner

345 words

Abstract

This study examined the relationships between stress, negative affect, rumination, and somatic symptoms among older adolescents. The following hypotheses were investigated: 1) greater number of life stressors would predict greater somatic symptoms, 2) rumination would moderate the relationship between stressors and somatic symptoms, 3) negative affect would also moderate the relationship between stressors and somatic symptoms, and 4) a three-way interaction between stress, rumination and negative affect would significantly predict somatic symptoms. Participants were 363 (71.1% female) university students with a mean age of 19.06 years (*SD*=2.06 years) who completed eight weekly online questionnaires, assessing levels of rumination and negative affect at baseline, and weekly stressor counts and somatic symptoms. Data were analyzed using hierarchical linear modeling to conduct multilevel moderation analyses, with the dependent variable of weekly somatic symptoms, Level 1 within-subjects predictor variable of weekly stressors, and the Level 2 between-subjects moderator variables of baseline rumination and negative affect. Baseline depression was included as a control variable. As hypothesized, greater weekly stressors significantly predicted greater weekly somatic symptoms (β=.12, t = 4.44, p < .001). However, neither baseline rumination (β = 0.02, t = 0.75, p = .45) nor negative affect (β = 0.01, t = 0.19, p = .85) significantly moderated the relationship between stress and somatic symptoms while controlling for depression. The interaction effect between rumination and negative affect as a moderator was also not significant (β= 0.01, t = 0.25, p = .80). Rumination significantly moderated the relationship between stress and somatic symptoms when depression was not controlled for in the model (β= .04, t = 2.13, p = .03), while negative affect approached significance as a moderator when not controlling for depression (β = 0.04, t = 1.87, p = .06).

Moderation effects were in the expected directions, with the effect of stress on somatic symptoms being stronger for individuals high in rumination or high in negative affect. This study uniquely contributed to the literature by examining the cognitive and affective vulnerabilities that impact stress' effect on somatic symptoms. Clinical implications, limitations, and directions for future research are discussed.

Key words: stress, rumination, negative affect, somatic symptoms

CHAPTER I

Introduction and Literature Review

Purpose

Somatic symptoms refer to physical or bodily symptoms, which may be attributed to a specific disease, or they may be labeled medically unexplained symptoms when there is no discernable medical cause (Fink & Rosendal, 2008). The term 'no discernable medical cause' does not imply that the symptoms are imagined or in any way less real than other physical symptoms. For example, headaches are a common physical symptom that all people experience at some point in their lives, yet in most cases they are not attributed to a specific medical condition such as a brain tumor. Most people are able to tolerate their headaches, yet a subset of individuals finds them intolerable and seeks medical care. Other common somatic symptoms include abdominal pain, nausea, heart palpitations, fatigue, and dizziness (Eriksen & Ursin, 2004). Somatic symptoms rarely occur singly; in fact, one study demonstrated that the mean number of symptoms was 6.6 with over 80% of patients reporting at least mild impairment from their symptoms (Hiller, Rief, & Brähler, 2006). In addition, certain patterns of somatic symptoms are sometimes given diagnoses called functional somatic syndromes, which Barsky and Borus (1999) say are characterized more by symptoms, suffering, and disability than by disease-specific, demonstrable abnormalities of structure or function. These functional somatic syndromes include fibromyalgia, chronic fatigue, and irritable bowel syndrome (IBS). Specific diagnoses, however, do not adequately account for most patients with somatic symptoms and many do not meet criteria for a full diagnosis (Burton, 2003). For the purpose of consistency in this study, the term "somatic symptoms" will be used, for this is the more general term that does not attempt to explain the cause of (or lack thereof) the symptoms.

Individuals with multiple somatic symptoms are a growing health care problem, and currently account for up to half of all primary care visits (Janca, Isaac, & Ventouras, 2006). Somatic symptoms are a costly burden to the healthcare system as well; they are positively associated with sick leave, healthcare use, and disability (De Gucht & Maes, 2006). Not only do primary care patients often present with somatic symptoms, but people with high levels of these symptoms have approximately twice the outpatient and inpatient medical care utilization and twice the annual medical care costs of patients with few to no symptoms (Barsky, Orav, & Bates, 2005). Somatic symptoms affect people of all ages, but the age group found to have the highest number of symptoms is young adults (Nimnuan, Hotopf, & Wessely, 2001). Somatic symptoms are common across ethnocultural groups (Kirmayer & Young, 1998), and they are more prevalent in females than males (Hiller, Rief, & Brähler, 2006).

Most theoretical models of somatic symptoms emphasize the roles of stress and negative emotions in the etiology of somatic symptoms (van Houdenhove & Egle, 2004; Tak & Rosmalen, 2010). Numerous studies have shown that stress predicts somatic symptoms (Burton, Farley, & Rhea, 2009; Haftgoli et al., 2010; Murberg, 2012). However, contemporary vulnerability-stress models of psychopathology suggest that it is important to consider the joint effects of environmental stress and responses to those stressors in understanding the emergence of symptoms. Maladaptive emotional and cognitive responses may exacerbate the effects of stressful events and increase the likelihood that the individual develops somatic symptoms. Negative affect is a well-established emotional vulnerability to negative outcomes when experiencing stress, while rumination is a maladaptive cognitive response to negative mood.

Trait negative affect (NA) is the tendency to experience frequent and intense negative emotions. Studies have indicated that both trait negative affect and stressful events are

associated with somatic symptoms (Thompson, Walz, Croyle, & Pepper, 2007; Burton, Farley, & Rhea, 2009). Therefore, it may be that people who tend to display higher negative affect will have an even stronger relationship between stressors and somatic symptoms. While trait NA has been shown to predict somatic symptoms, no previous studies have examined whether NA moderates the effects of stressful events on somatic symptoms.

Rumination is a cognitive strategy in which one repetitively and passively focuses on one's negative emotions (Nolen-Hoeksema, Wisco, & Lyubomirksy, 2008). Rumination has negatively correlated with self-reported physical health (Thomsen et al., 2003), and positively correlated with distress in young adults (Morrison & O'Connor, 2004). However, no studies to date have examined how rumination may affect the relationship between stressful events and somatic symptoms. People who perseverate on their negative affect may display more somatic symptoms after encountering stressful events than those without this cognitive vulnerability. Thus, the effect of stressors on somatic symptoms may be exacerbated for people who tend to ruminate.

The purpose of this study was to examine somatic symptoms from a vulnerability-stress perspective, specifically the extent to which trait negative affect (emotional vulnerability) and/or rumination (cognitive vulnerability) may exacerbate the effects of stressful events on the presentation of somatic symptoms among young adults. Previous cross sectional studies have established correlations between stressors, negative affect and somatic symptoms, but few studies have utilized a prospective design. Thus, I used an 8-week prospective study design to test study hypotheses.

Defining somatic symptoms, syndromes, and disorders

The conceptualization of somatic symptoms has changed dramatically over the last century. In the early 1900s, the generally accepted theory was that some individuals, after being exposed to a trauma, narrowed their attention to some sensory channels while ignoring others (Brown, 2004). Moreover, it was thought that some memories may become dissociated from the body and certain triggers in the environment can activate them to appear as bodily perceptions rather than memories. Individuals who experienced these dissociations were called 'hysterical.' Later, Lipowski (1968) defined somatization as the tendency to experience or express psychological or emotional distress as the symptoms of physical illness (as cited in Brown, 2004). This idea retreats somewhat from the concept of hysteria, yet still maintains that somatic symptoms are always some manifestation of emotional distress. Currently, somatic symptoms are defined much more neutrally, as "symptoms of physical illness for which no adequate organic basis can be found" (Brown, 2004, p. 793). Another way to describe somatic symptoms is as "persistent, severe, and distressing symptoms that cannot fully be explained by medical knowledge or whose severity cannot be accounted for after medical investigation" (Husain, Browne, & Chalder, 2007, p. 2). These definitions acknowledge that there is no known medical cause, yet do not go beyond that to propose an alternative cause; thus they are also sometimes referred to as "medically unexplained symptoms." The following sections will discuss possible diagnoses for individuals who are experiencing somatic symptoms and the prevalence of these symptoms in young adults.

Diagnosis. In some cases a patient's somatic symptoms can be diagnosed with a DSM-IV or DSM-V disorder. In the DSM-IV, the category known as somatoform disorders examines the nature, number, and duration of symptoms to determine which disorder best describes the pattern of symptoms. Pain disorder describes someone only with various pain symptoms

throughout their body, whereas conversion disorder describes somatic symptoms that suggest a neurological disorder for which no organic cause can be found. The 12-month prevalence rate for pain disorder in the general population is approximately 8%, and this disorder is associated with poor quality of life and high health-care utilization rates (Frohlich, Jacobi, & Wittchen, 2006). A diagnosis of conversion disorder is rarely given (Feinstein, 2011). Finally, somatization disorder refers to someone with a variety of symptoms including pain, gastrointestinal symptoms, sexual symptoms, and at least one pseudoneurological symptom. Somatization disorder is rarely diagnosed (less than 1% of population) because the criteria are so restrictive that many people do not have symptoms in all categories (Hiller, Rief, & Brahler, 2006) and also because physicians may be reluctant to use this diagnostic label. The DSM-V therefore has made some significant changes to the diagnostic criteria for somatoform disorders.

The DSM-V, instead of distinguishing between pain disorder, conversion disorder, and somatization disorder, has combined all these disorders into one called somatic symptom disorder (SSD; American Psychiatric Association, 2013). The criteria for this disorder are much more general. Instead of requiring specific types and numbers of symptoms, SSD is characterized by distressing or disruptive somatic symptoms, as well as disproportionate thoughts, feelings and behaviors surrounding these symptoms. This psychological component was not included in the DSM-IV. Finally, unlike the DSM-IV, these symptoms may or may not be medically unexplained. Someone could have a chronic disease such as cancer, which is often associated with pain, nausea, and fatigue, yet if they are disproportionately distressed by their symptoms they could also be diagnosed with somatic symptom disorder.

Regardless of how they are specifically diagnosed, patients who suffer from somatic symptoms place a significant burden on the healthcare system because they have more primary

care visits, emergency room visits, and hospital admissions than the normal population, even when controlling for comorbid depression and anxiety (Barsky, Orav, & Bates, 2005). Moreover, Escobar et al. (2010) found that regardless of the presence or absence of a medical explanation, three or more concurrent physical symptoms predicted healthcare use in the general population. Therefore, somatic symptoms, regardless of the particular diagnosis given, are an important problem to address in the general population.

Prevalence in young adults. Somatic symptoms are especially prevalent in young adults. In one study (Nimnuan, Hotopf, & Wessely, 2001), the age range of patients at an outpatient clinic who were most likely to present with somatic symptoms was 16-25, with 72% of those patients having at least one medically unexplained symptom. Another group of researchers studied participants aged 14-24 and found that the lifetime prevalence rate for any specific somatoform disorder was 2.7%, or more than 12% when using less stringent criteria (Lieb, Pfister, Mastaler, & Wittchen, 2000). They also found that somatoform disorders are highly comorbid with depression and anxiety, and are more likely to occur in females. These disorders result in a marked degree of impairment, including missing work or school and higher health care utilization. It is evident that somatic symptoms, whether or not they meet criteria for a DSM disorder, cause disability and dysfunction in the lives of many young adults.

Current theories of somatization

Several theories exist that attempt to explain the meaning and origin of somatic symptoms. Though each theory emphasizes unique aspects of the experience of somatic symptoms, it is clear that they also possess commonalities. For example, all of the current somatization theories take into account the role of stress and negative emotions in the onset and maintenance of somatic symptoms.

Dysfunction of stress response system. One leading theory behind the onset of somatic symptoms is the dysfunction of the stress response system. When an organism encounters a stressful event and perceives it as a physical or emotional threat, the body enacts a physiological stress response neuronally, hormonally, and behaviorally, in order to maintain homeostasis (Tak & Rosmalen, 2010). The body's ability to achieve stability through change is a stress response called allostasis. During allostasis, the immune system, cardiovascular system, and HPA axis, all of which are affected during a stress response, become moderately yet consistently stimulated, leading to various somatic symptoms (Brosschot, Gerin, & Thayer, 2006). Allostasis is normally an adaptive response for people to avoid harm and it dampens after the stress has subsided, yet when the stress system is perpetually activated due to chronic exposure to stressors, symptoms emerge and persist (Linden, Earle, Gerin, & Christenfeld, 1997). The chronic damage to the body from stress is sometimes termed the allostatic load. For example, chronic stress has been implicated in the onset and exacerbation of irritable bowel syndrome because the gastrointestinal system becomes inflamed, resulting in symptoms such as nausea, abdominal pain, heartburn, and loose stools or constipation (Hertig, Cain, Jarrett, Burr, & Heitkemper, 2007). Over time, the individual may also become hypersensitive and hypervigilant to stressful cues in what is known as anticipatory stress, further exacerbating somatic symptoms (van Houdenhove & Egle, 2004).

Psychodynamic model. One of the oldest explanations for the origin of somatic symptoms is rooted in a psychodynamic model. In this model, it is thought that an individual's psychological conflicts are transformed into physical distress (Kirmayer & Young, 1998). Menninger (1947) stated:

> The anxiety is relieved . . . by channeling the originating impulses through the autonomic nervous system into visceral organ symptoms and complaints. These reactions represent

> the visceral expression of the anxiety which is thereby largely prevented from being conscious. The symptom is due to a chronic and exaggerated state of the normal physiology of the emotion, with the feeling or subjective part repressed (p. 96).

In fact, it is often presumed that specific symptoms arise from specific conflicts from a patient's past that they are unable to express but which cause them distress, such as numbness or pain in the pelvic area as a result of childhood sexual abuse (Simon, Gater, Kisely, Piccinelli, 1996). Organs were said to mirror emotional problems, such as cardiovascular symptoms reflecting emotional heartache (Menninger, 1947). It was historically hypothesized that psychic energy that is repressed unconsciously through defenses will ultimately displace itself into the lower order processes of physiological symptoms (Kirmayer & Young, 1998). The patient attributes his or her symptoms to an undiagnosed physical disease and may be unaware of or adamantly deny the presence of emotional distress. Consequently, this theory labels individuals with somatic symptoms as less psychologically-minded because they are unable to talk through or even acknowledge their emotional conflicts and must resort to physical symptoms as a form of expression. Therapy, therefore, attempts to expose unresolved emotional issues and develop greater insight within the patient (Abbass, Kisely, & Kroenke, 2009). Ultimately there is little evidence to support the concept of psychic energy and the idea that greater insight necessarily leads to fewer somatic symptoms (Kirmayer & Young, 1998). However, psychodynamic theory still has some influence today on how clinicians view somatic symptoms by assuming an inherent dualism between body and mind and dismissing some patients with somatic symptoms as merely expressing psychological distress through their body rather than having any 'real' disease.

Traditional learning models. Learning models for somatic symptoms attempt to explain how somatic symptoms become a learned response, often beginning in childhood. In a classical conditioning model, a chronic symptom such as a headache may be accompanied by nausea for example, and nausea becomes a conditioned stimulus that produces distress and heightens sensitivity to pain (Husain, Browne, & Chalder, 2007). In an operant conditioning model, behaviors are positively reinforced through rewarding with a pleasant activity or negatively reinforced through avoiding an unpleasant activity. Thus, a child may learn that physical symptoms result is missing school (negative reinforcement) and receiving more attention from his or her parent (positive reinforcement), and an adult similarly may experience symptoms in order to miss work and/or receive attention from a significant other. Finally, social learning theory states that children learn behaviors and attitudes vicariously through observing and modeling after their parents or siblings (Bandura, 1973). Therefore, if a child observes her parent experiencing frequent stomachaches and receiving attention for those pains, she may consequently imitate that behavior and learn that a stomachache leads to positive attention (Lipowski, 1988). As the child ages, experiencing somatic symptoms may become the primary coping strategy used when under stress. Children also may learn to view certain symptoms as more serious than others depending on how their parents react to them. They may witness a close family member experience a serious illness such as cancer, which can produce pain, nausea, and fatigue among other symptoms. The child may learn to believe that the symptoms themselves are distressing and life threatening rather than the cancer that caused them.

Contemporary cognitive-behavioral model. A more recent elaboration on the learning models to explain the origin and perpetuation of somatic symptoms today is from a cognitive behavioral (CBT) perspective. In cognitive behavioral models, somatic symptoms are thought to

result from the interaction between cognition, behavior, and physiology. The classic CBT model for any disorder, including the problem of somatic symptoms, is to examine the predisposing, precipitating, and perpetuating factors (Deary, Chalder, & Sharpe, 2007). Predisposing factors may include genetic factors, early childhood experience, or personality factors. For instance, neuroticism, a term for the trait-like tendency to experience negative affect, is a stable and heritable personality trait that makes people vulnerable to physical and psychological distress in general as well as medically unexplained symptoms in particular (De Gucht, Fischler, & Heiser, 2004). Early childhood trauma or adversity has also been associated with somatic symptoms later in life (Roelofs & Spinhoven, 2007). Thus, the CBT model suggests that early predisposing factors such as neuroticism and trauma may make certain people more vulnerable to experiencing somatic symptoms after a precipitating event.

Precipitating events are the factors that lead to the onset of somatic symptoms. Numerous researchers have shown that major life events often precede the onset of medically unexplained symptoms (Murberg, 2012; Rozzini, Bianchetti, Carabellese, Inzoli, & Trabucchi, 1988). Researchers hypothesize that the stress of a life event triggers activation of the stress response system, as mentioned above, and for some people that activation is prolonged to the point that unexplained medical symptoms develop. Moreover, a theory called the perseverative cognition hypothesis states that after the stress system is activated, the physiological effects of stress persist only when the individual worries excessively about the stressor (Brosschot, Gerin, & Thayer, 2006). Thus it is not the life event itself that necessarily leads to somatic symptoms, but the individual's response to that event in the form of worry. In the emotional avoidance theory of worry, worry is thought to be a cognitive avoidance strategy that inhibits emotional processing and the affective experience of anxiety, which in the short term is negatively

reinforcing but prolongs anxiety in the long term by triggering physiological (somatic) and behavioral responses in spite of the perception that worry somehow prevents or limits some aversive emotional experiences (Borkovec, Ray, & Stober, 1998; Derakshan, Eysenck, & Myers, 2007). The cognitive-behavioral model highlights the idea that in people who possess such predisposing factors as neuroticism or early exposure to illness, worrying about a stressful event may be all it takes to trigger one or more somatic symptoms.

Finally, the cognitive-behavioral model emphasizes the importance of cognitive and behavioral perpetuating factors that maintain somatic symptoms after they arise. Worry, as mentioned above, may not only precipitate symptoms, but continual worry may also prolong the experience of somatic symptoms. Another way in which symptoms may arise or persist is through sensitization, or the increased likelihood to respond to stimuli because of prior exposure to them (Deary, Chalder, & Sharpe, 2007). In addition to becoming more sensitized to a stimulus, the Symptom Perception Model (Kolk et al., 2003) theorizes that people high in negative affect will selectively attend to internal stimuli and therefore report more physical symptoms. For example, if a person is accustomed to experiencing nausea before a migraine, they may begin to interpret normal bodily sensations as nausea through selective attention. They then may predict an impending migraine, resulting in avoidance of the activity thought to trigger the migraine and consequently perpetuating the symptom. The cognitive-behavioral model shows that the interplay of cognitive processes (such as selective attention) with behaviors (such as avoidance or increased health care use) and physiological factors (such as overactivation of the stress response system) predict the onset and maintenance of somatic symptoms.

Current study: vulnerability-stress model. The current study views somatic symptoms through the lens of a vulnerability-stress model. This model draws from commonalities among

all the aforementioned models, emphasizing the importance of individual predispositional factors (vulnerabilities) in experiencing somatic symptoms, which can include cognitive (rumination) and emotional (negative affect) processes. Vulnerability-stress models also emphasize the interaction of stressors with vulnerabilities to trigger the development of a disorder. Several studies have examined moderators of the relationship between stressors and depression over time using a vulnerability-stress model (Hankin, 2008; Mezulis, Funasaki, Charbonneau, & Hyde, 2010; Seeds & Dozois, 2010), but none to date have explicitly applied this model to somatic symptoms.

Defining stress and its relationship with somatic symptoms

Stress has been defined as any threat to an organism's homeostasis (Chrousos & Gold, 1992). When defining stress, some researchers emphasize the difference between a *stressor* and *stress response*. Selye (1936) referred to the stress response, or *general alarm reaction,* as the response an organism makes when threatened, in an attempt to adapt to new conditions and restore homeostasis. Lazarus and Folkman (1984) differentiated a stressor from a stress response by stating that it is not the stressful event itself that determines a negative outcome within an individual, but rather the individual's appraisal, or perception, of the event. Contemporary models of stress recognize that stress is an interactive relationship between environmental stimuli (stressors) and individual differences in the response to such stimuli (Whitehead, 1994). Additionally, current theories of stress differentiate between primary and secondary appraisal of an event (Lazarus & Folkman, 1984). A primary appraisal is when one determines if an event is relevant to the individual, and if so, if it is stressful or benign. It is thought to be stressful if the demands are believed to exceed available resources. If the event is determined to be stressful, the event may be viewed as a threat, harm, or challenge. Viewing an event as harm indicates a

belief that the event is immediately harmful, viewing an event as a threat indicates possible future harm, and viewing an event as a challenge indicates a belief that the stressful event can be overcome. A secondary appraisal is when an individual determines if and how they should act (or cope) to reduce the stress associated with the event. When an individual cannot adequately cope with a stressor, one possible consequence may be the development of somatic symptoms.

Stress has been identified as a significant contributor toward the development of psychopathology (Ingram & Luxton, 2005), and several studies in particular have demonstrated an association between stressors and somatic symptoms. Burton, Farley, and Rhea (2009) found that spouses of people deployed in the military (a stressful event) had greater levels of somatic symptoms than spouses of nondeployed military members. Additionally, in both of these groups perceived stress was significantly and positively related to levels of somatic symptoms. The researchers concluded that spouses under the most stress endorsed the most symptoms. Another set of researchers found that pain severity was correlated with stress level, and that somatization predicted pain severity (Hwang et al., 2008). Thus, patients who were under greater stress perceived their pain as more severe than those not under stress. Finally, Haftgoli et al. (2010) found that psychosocial stressors were significantly related to somatoform disorders in a primary care setting, and that with each additional stressor the association between them increased 2.2 fold. The previous three studies examined cross sectional data, and therefore it is difficult to know what comes first, stress or somatic symptoms. However, another study by Murberg (2012) prospectively examined the relationship between negative life events (another term for stressful events) and somatic symptoms in adolescents and found that number of negative life events at time one predicted somatic symptoms at time two, controlling for initial levels of somatic symptoms. These findings suggest that stressors predict somatic symptoms, though more studies

should be done to replicate these findings. Few studies on the relationships between stressors and somatic symptoms have been prospective, and while many studies focus on the general adult population or on adolescents, few have specifically examined older adolescents/young adults.

Somatic symptoms can be conceptualized as a response to stress. Mellner, Krantz, and Lundberg (2005) showed that somatic symptoms are related to higher heart rate and cortisol levels, two physiological responses known to occur when an organism experiences stress. In addition, Whitehead (1992) found (using time-lagged analyses) that stressful life events preceded bowel symptoms, and those with a diagnosis of IBS reported experiencing more stressors than those without IBS. The direct relationship between stress and somatic symptoms is well established, but the individual differences in how one can respond to stress need further research. It is important to target which individuals experience the greatest number of somatic symptoms when facing stressors because of cognitive and/or emotional vulnerabilities. Two individual differences that I explored as potential moderators were trait negative affect and rumination.

Negative Affect

Trait negative affect is the stable temperamental tendency to experience frequent and intense negative emotions (Watson, 1988). NA includes a broad range of emotions such as anger, fear, and sadness, and NA can increase one's attention to internal symptoms (Gendolla, Abele, Andrei, Spurk, & Richter, 2005). Trait NA has been associated with a variety of negative outcomes, including depression, anxiety, and increased physical symptom reporting (Brown, Chorpita, & Barlow, 1998; Gendolla, Abele, Andrei, Spurk, & Richter, 2005; Watson, Gamez, & Simms, 2005).

Negative affect has been shown to predict somatic symptoms in a number of studies. First, Thompson, Waltz, Croyle, and Pepper (2007) found that negative affect correlated with

somatic symptoms in young adults. Second, a prospective study of primary care patients found that NA was the best predictor of changes in somatic symptom reporting over time (De Gucht, Fischler, & Heiser, 2004). This study not only suggests that NA correlates with somatic symptoms, but that NA predicts increases in somatic symptoms. Researchers also demonstrated that NA is related to both physical complaints and perceived stress (Watson, 1988), suggesting that people with high NA who encounter stressors may report more somatic symptoms than those with low NA. It may be that NA, an emotional vulnerability, moderates the relationship between stress and somatic symptoms, as my study will examine. Vulnerability-stress models have posited that NA moderates the relationship between stress and mood outcomes such as depression, therefore it is worth investigating if this same pattern applies with somatic symptoms as the outcome. Another potential moderator (and a cognitive vulnerability) of the relationship between stressors and somatic symptoms is rumination.

Rumination

Rumination was first defined in the depression literature as "thoughts and behaviors that focus one's attention on one's depressive symptoms and the meaning of those symptoms" (Nolen-Hoeksema, Parker, & Larson, 1994, p. 92). Later, rumination was more broadly defined as thinking repetitively about a common theme, not necessarily requiring depressive content (Martin & Tesser, 1996). Rumination is different from worry because it is oriented toward the past and present, whereas worry perseverates on the uncertain future (Watkins, Moulds, & Mackintosh, 2005).

Rumination is a maladaptive coping response to thoughts and feelings related to a negative life event (Garnesfski, Kraaij, & Spinhoven, 2001; Thomsen, 2006). Rumination has correlated with depression in several studies (Koval, Kuppens, Allen, & Sheeber, 2012; Michl,

McLaughlin, Shepherd, Nolen-Hoeksema, 2013), and it is especially powerful in predicting which patients will relapse into depression (Michalak, Hölz, & Teismann, 2011).

There are also several studies establishing a relationship between stress and rumination. For example, one study found that, in young adults, exposure to life stressors correlated positively with rumination. Additionally, rumination mediated the relationship between stressful life events and anxiety/depression (Michl, McLaughlin, Shepherd, & Nolen-Hoeksema, 2013). Moreover, rumination and perceived stress in young adults have been shown to interact to predict the social dysfunction component of psychological distress from a vulnerability-stress model perspective (Morrison & O 'Connor, 2004). Studies on the relationship between rumination and somatic symptoms, however, are scarce.

Thomsen et al. (2004) did find an association between rumination and self-reported physical health, but the study incorporated an overall health measure rather than a list of specific somatic symptoms. In addition, Wilkinson, Croudace, and Goodyer (2013) examined rumination as a predictor for depression and anxiety and found that rumination predicted the somatic symptoms of depression. However, no studies to date have examined the relationship between rumination and somatic symptoms outside the context of depression. Finally, Genet and Siemer (2012) found that rumination moderated the relationship between daily negative events and mood over time in young adults, but, again, no studies have examined whether a relationship exists between stressful events, rumination, and somatic symptoms. As with negative affect, rumination has functioned as a moderator in studies on the relationship between stress and depression, but it has yet to be studied as a moderator of stress and somatic symptoms. In the present study I filled in these gaps by examining whether young adults who experience more stressors and who possess the vulnerabilities of high negative affect and rumination experience

more somatic symptoms. I also addressed the current dearth of prospective studies on these variables by examining their relationships over time.

Current Study

In the present study I examined how rumination and trait negative affect influence the relationship between stressful events and somatic symptoms. I hypothesized that 1) young adults who experience more stressful events would report more somatic symptoms; 2) trait negative affect and rumination would each separately moderate the relationship between number of stressful events and somatic symptoms, specifically that the relationship between stressors and somatic symptoms would be stronger in those individuals with higher negative affect and higher rumination; and 3) the interaction between all three variables would have the strongest prediction of somatic symptoms, such that individuals experiencing a higher number of stressful events, often experience intense negative emotion (high trait NA), and also ruminate on the causes, meaning, and consequences of their negative affect (high trait rumination), would report the greatest number of somatic symptoms. I examined these relationships prospectively to expand upon past researchers' cross-sectional findings on the relationships among these variables.

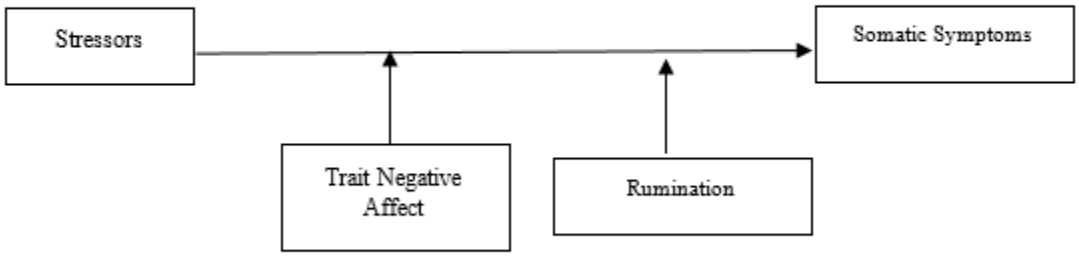

Figure 1. Proposed moderation model. Trait negative affect and trait rumination moderate the relationship between stressors and somatic symptoms.

CHAPTER II

Method

Participants

Participants were 363 undergraduate students recruited from a private university in the Pacific Northwest. Participants ranged from age 18 to 41, with a mean age of 19.06 (*SD* = 2.06). Approximately 70% identified as Caucasian, 3.9% as African American, 15.2% as Asian, 0.6% as Native American, 5.2% as Hispanic/Latino, and 5.2% as other. Approximately 71% were female.

Power analysis

A power analysis for a multiple regression analysis with six predictors was performed using G*Power 3.1 (Faul, Erdfelder, Lang, & Buchner, 2007), which calculated that 55 participants would be required for a medium effect size (.15) with a power of .80. Although multiple regression analyses apply an ordinary least squares approach instead of the maximum likelihood approach of Hierarchical Linear Modeling (HLM) analyses, a G*Power 3 multiple regression analysis was conducted since there is no standard method for HLM power estimation (Castelloe & O'Brien, 2000). G*Power 3 is likely underpowered because the statistical algorithms for HLM are based on large-scale analyses and there is controversy around determining sample size using HLM (Mathieu, Aguinis, Culpepper, & Chen, 2012). My recruitment method allowed for more people than the minimum requirement in G*Power, therefore I aimed to recruit considerably more than 55 participants, and with the hopes of at least 200 participants. I surpassed that goal with a final n of 363.

Procedure

The current study lasted 8 weeks and consisted of two parts. Recruitment took place via classroom presentations and flyers posted around the campus. Informed consent documents notified participants about the potential risks and benefits and the voluntary, confidential nature of the study. Participants first completed the Part 1 baseline set of questionnaires administered through an online survey tool within a 48-hour window after receiving an email with a link to the survey. The baseline questionnaires included measures of baseline somatic symptoms, depressive symptoms, trait NA and trait rumination.

Students who completed Part 1 were invited to participate in Part 2 of the study, in which they were asked to complete weekly diary assessments via an online survey tool across the subsequent 7-week follow-up period. Each week participants responded to questionnaires regarding the previous week's somatic symptoms and stressful events. The diary method provided a way to examine fluctuations in both stressors and symptoms over time and was more accurate than requiring participants to reflect on the entire 7 weeks as a whole. Students had a 48-hour window after receiving an email to complete the weekly questionnaires in order to maintain an equal interval between responses. If they did not complete the first week of Part 2 (week 2 of 8) within the 48 hour window, they were not be able to participate in Part 2. However, if they failed to complete a questionnaire on any of the subsequent weeks (weeks 3-8), they were not penalized and could continue in the study. Participants received research credit for completing the baseline questionnaire and additional research credits for participating in the 7-week diary assessment.

Measures

Trait negative affect. Trait NA was measured at baseline using the Negative Affectivity subscale of the Adult Temperament Questionnaire (ATQ-NA; Evans & Rothbart, 2007). The

ATQ-NA is a 51 item subscale measuring Fear, Sadness, Discomfort, and Frustration. Participants rate on a 7-point Likert scale how true each item is for them, from (1) *Extremely untrue of you*, to (7) *Extremely true of you*. Higher scores on this subscale represent higher temperamental NA. Example items include, "It doesn't take very much to make me feel frustrated or irritated" and "Loud noises sometimes scare me." This measure has demonstrated strong evidence for internal consistency and convergent validity in past studies (Evans & Rothbart, 2007). In the current sample, the internal consistency of the NA subscale was .88.

Rumination. Rumination was measured at baseline using the Ruminative Responses Scale of the Response Style Questionnaire (RRS; RSQ; Nolen-Hoeksema, 1991). This 22-item self-report questionnaire assesses cognitive responses to negative mood that perseverate on the self, one's symptoms, and the causes and consequences of one's mood. Items are rated on a 4-point Likert scale (1 = *almost never*, 4 = *almost always*). Example items from the measure include "When I feel sad or down, I think about recent situations, wishing they had gone better" and "I think about how down I feel." This measure has been used extensively in a college student population and alpha coefficients have ranged from .81 to .85, demonstrating strong internal consistency (Hoff & Muehlenkamp, 2009; Goldstein, Chesir-Teran, & McFaul, 2008). The RRS has also demonstrated convergent validity with other measures of rumination and with measures of depression (Griffith & Raes, 2015). In the current sample, internal consistency was .92.

Stressful events. Stressful events were measured by 25 select items of the Negative Event Scale – University (NES-U; Maybery, 2003) measuring stressful life events and daily hassles appropriate for a college/young adult sample. Example items include "Had a disagreement with parents" and "Not getting the grades you expected." Participants were asked

to indicate whether the event has occurred for them in the past week. A total stressful event score for each week was computed by counting the number of stressful events endorsed.

Somatic symptoms. The Modified Somatic Perception Questionnaire (MSPQ) was used at baseline and each week thereafter to measure somatic symptoms (Main, 1983). The MSPQ is a 13-item self-report scale that was designed to measure somatic symptom perception in a back pain population and has since been used with a variety of other populations. Participants are asked to assess how they have felt throughout the last week by rating the subjective frequency of a variety of somatic symptoms (e.g., nausea, dizziness) on a Likert scale ranging from (0) = *not at all* to (3) = *extremely/could not have been worse*. The range of possible scores is from 0 to 39; a higher score indicates more frequent and severe somatic symptoms. The MSPQ has demonstrated strong reliability and convergent validity with similar measures of somatic symptoms (Deyo, Walsh, Schoenfeld, & Ramamurthy, 1989). Internal consistency as measured by Cronbach's alpha was .84 in this study's sample.

Depressive symptoms. Baseline depressive symptoms (used as a control variable in the model) were measured using the Center for Epidemiological Studies Depression Scale (CES-D; Radloff, 1977), which is a 20-item self-report inventory measuring current depressive symptoms in the past week. Participants rate on a 4-point Likert scale how they felt in the past week, ranging from (0) *Rarely or none of the time* to (3) *All of the time*. Scores range from 0 to 60, with a clinical cutoff of 16. An example item is "I was bothered by things that usually don't bother me." The CES-D measure has been found to have high internal consistency, and convergent validity is supported with other measures of depression (Radloff, 1977). The Cronbach's alpha within the current study was .87.

Data Analytic Plan

Preliminary analyses included calculating bivariate correlations among all study variables. Prospective analysis of the data used Hierarchical Linear Modeling. HLM was appropriate for this design due to its multiple levels of analysis (can assess both within-subject and between-subject variations over multiple time points) and ability to handle missing data. In multiwave modeling, Level 1 equations measure the changes in the dependent variable as a function of any predictor variables measured repeatedly. In this study, somatic symptoms were the dependent variable modeled in Level 1 as a function of weekly stressor count. Level 2 allowed for examination of individual differences that moderate the relationship between stressful events and somatic symptoms. For this study, those variables were trait negative affect and rumination, which were both measured at week 1. Baseline depressive symptoms were added as a control variable at the L2 equation. Level 1 predictor variable (stress) was entered using group mean centering, while Level 2 variables (NA, rumination, and the interaction term) were entered using grand mean centering. Intercepts and slopes were allowed to vary randomly rather than be entered as fixed terms. An example equation can be seen below:

Level 1: $\text{SomSx}_{ij} = \beta_{0j} + \beta_{1j}(\text{Stressors}) + e_{ij}$

Level 2: $\beta_{0i} = \beta_{00} + \beta_{01}(\text{Depression}) + r_{0i}$

$\beta_{1i} = \beta_{10} + \beta_{11}(\text{NA}) + \beta_{12}(\text{Rum}) + \beta_{13}(\text{NAxRum}) + r_{1i}$

CHAPTER III

Results

Data Screening and Analysis

Data preparation. I inspected the data first for missing values, multicollinearity, and normality of distribution using skewness and kurtosis. No variables were significantly skewed or kurtotic, which suggests that the data is normally distributed. I utilized Parent (2013) recommendations to use person-mean imputation for Level 2 equations (baseline variables), which imputes scale scores on an item basis for participants who completed at least 80% of a given scale. This method is advantageous because it imputes a different value for each person depending on that person's mean of completed items, which does not artificially reduce the measure's variability. Also, less than 1% of the data in week 1 was missing, and person-mean imputation does not result in a significantly different interpretation of the data compared with other methods of imputation when there is such a small percentage of data missing. HLM allows for missing data in the level 1 equation for the variables measured in weeks 2-8, therefore person-mean imputation was only used for level 2 variables.

Participants. A total of 371 participants initiated the baseline questionnaire for the present study. Of these, eight participants failed to complete any weekly data. Utilizing simple t-tests, I compared these two groups and found no significant differences regarding gender, age, and all baseline measures. Thus the final N, after excluding those eight participants, was 363. Demographics data are presented in Table 1.

Table 1

Participant Demographics

	N	%
Sex		
Male	105	28.9
Female	258	71.1
Race/Ethnicity		
Caucasian	254	70.0
Asian	55	15.2
Native American	2	0.6
African American	14	3.9
Hispanic/Latino	19	5.2
Other	19	5.2
Year In College		
Freshman	220	60.6
Sophomore	87	24.0
Junior	32	8.8
Senior	21	5.8

Descriptive. Bivariate correlations, means, and standard deviations for study variables are presented in Table 2. Descriptives by race and sex are presented in Table 3. The means for the MSPQ were, as expected, lower than past reported means in clinical samples (Jansson-Fojmark & MacDonald, 2009), as this study used a healthy sample of older adolescents. Means

for the RRS, ATQ-NA, and CESD were comparable to means reported in past studies on college students (Evans & Rothbart, 2007; Johnson, McKenzie, & McMurrich, 2008; Radloff, 1977).

Table 2

Bivariate Correlations and Descriptives among Variables

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	*M (SD)*
1. RRS																		53.26 (1
2. NA	.54**																	197.70 (
3. MQw2	.31**	.36**																18.24 (4
4. MQw3	.35**	.35**	.66**															17.38 (4
5. MQw4	.23**	.28**	.65**	.70**														17.09 (4
6. MQw5	.25**	.30**	.52**	.62**	.66**													16.67 (4
7. MQw6	.22**	.30**	.60**	.71**	.69**	.70**												16.47 (4
8. MQw7	.23**	.29**	.57**	.62**	.66**	.60**	.74**											16.42 (4
9. MQw8	.24**	.26**	.59**	.61**	.70**	.61**	.70**	.73**										16.68 (4
10. NEw2	.18**	.22**	.33**	.33**	.26**	.23**	.30**	.25**	.21**									6.96 (3.1
11. NEw3	.08	.13*	.18**	.31**	.14**	.11	.17**	.12*	.16**	.33**								6.50 (3.3
12. NEw4	.16**	.24**	.30**	.26**	.33**	.24**	.26**	.17**	.24**	.40**	.44**							6.05 (3.5
13. NEw5	.17**	.16**	.16**	.16**	.19**	.19**	.20**	.11	.09	.37**	.39**	.53**						5.40 (3.5
14. NEw6	.09	.16**	.13*	.17**	.24**	.19**	.25**	.13*	.18**	.34**	.33**	.51**	.53**					5.36 (3.7
15. NEw7	.06	.15**	.13*	.19**	.16**	.17**	.21**	.26**	.15**	.39**	.34**	.47**	.40**	.52**				5.07 (3.7
16. NEw8	.06	.20**	.13*	.24**	.21**	.15**	.18**	.16**	.21**	.39**	.35**	.47**	.47**	.49**	.54**			5.45 (3.7
17. CESD	.50**	.56**	.41**	.44**	.43**	.41**	.40**	.44**	.39**	.26**	.15**	.30**	.25**	.22**	.14**	.19**		14.19 (9
18. Sex	.18**	.34**	.11*	.11*	.10	.15**	.11*	.10	.13*	.04	.06	.09	.09	.12*	.12*	.09	.16**	

Notes: MQ = Modified Somatic Perception Questionnaire; NA = Adult Temperament Questionnaire- Negative Affect subscale; RRS = Ruminative Response Scale; NE = Negative Events Scale; CESD = Center for Epidemiological Studies Depression Scale. * $p < .05$, ** $p < .01$

Table 3

Descriptive Statistics by Race and Sex

Sex	Ethnicity		RRS	ATQ NA	MSP Qw2	MSP Qw3	MSP Qw4	MSP Qw5	MSP Qw6	MSP Qw7	MSP Qw8	NE w2	NE w3	NE w4	NE w5	NE w6	NE w7	NE w8	CES D
Male	Caucasian/White	Mean	49.6	3.62	17.47	16.8	15.9	15.6	15.42	15.7	15.4	6.90	6.20	5.56	5.02	4.64	4.62	5.17	11.6
		Std. Deviation	12.6	.630	4.061	4.60	3.79	3.15	3.988	3.69	3.24	2.90	3.10	3.460	3.11	2.96	3.56	3.63	8.32
		Range	53.0	3.61	19.00	26.0	16.0	11.0	20.00	13.0	14.0	15.0	13.0	13.00	12.0	15.0	16.0	18.0	37.9
	Asian	Mean	53.1	3.71	17.38	15.7	16.2	15.2	15.94	15.0	15.1	6.00	5.77	5.388	4.16	4.94	3.94	4.72	13.3
		Std. Deviation	14.4	.680	4.57	3.63	3.93	4.46	4.207	3.46	2.97	3.02	3.22	2.768	2.74	4.03	2.73	3.26	6.73
		Range	53.0	2.88	18.00	11.0	12.0	13.0	15.00	13.0	9.00	12.0	13.0	13.00	10.0	13.0	9.00	10.0	22.0
	African American	Mean	47.5	3.44	21.50	19.7	28.0	16.6	19.66	13.0	20.5	8.75	6.75	4.750	5.25	3.25	.000	3.25	10.5
		Std. Deviation	25.0	.902	9.036	8.30	5.65	6.35	7.637	.	4.94	5.90	3.30	5.852	4.57	3.94	.000	3.94	8.54
		Range	57.0	1.94	19.00	17.0	8.00	11.0	15.00	.00	7.00	13.0	7.00	12.00	10.0	8.00	.00	8.00	18.0
	Hispanic/Latino	Mean	42.3	3.16	15.33	14.5	16.8	15.6	14.40	17.7	19.3	7.33	7.16	6.333	5.66	4.16	3.66	3.83	13.1
		Std. Deviation	9.11	.702	3.011	1.22	4.35	3.57	2.190	6.18	5.13	2.50	3.43	3.32	3.61	3.43	4.13	4.83	8.03
		Range	24.0	2.16	8.00	3.00	11.0	7.00	5.00	13.0	10.0	6.00	9.00	9.00	10.0	9.00	10.0	12.0	20.0
	Other	Mean	43.3	3.62	15.33	15.0	15.0	13.0	15.66	15.6	16.0	4.00	6.33	5.666	4.00	6.66	7.00	4.00	11.6
		Std. Deviation	11.0	.357	3.214	2.64	3.46	.000	4.618	4.61	5.19	2.64	2.08	1.154	3.46	.577	1.73	2.00	3.05
		Range	22.0	.71	6.00	5.00	6.00	.00	8.00	8.00	9.00	5.00	4.00	2.00	6.00	1.00	3.00	4.00	6.00
Female	Caucasian/White	Mean	53.1	4.06	18.72	17.7	17.6	16.7	16.75	16.6	17.0	7.01	6.50	6.316	5.44	5.59	5.11	5.57	14.5
		Std. Deviation	11.3	.584	4.777	4.78	5.12	4.72	4.937	4.80	5.41	2.92	3.52	3.403	3.59	3.73	3.59	3.62	8.97
		Range	58.0	3.04	22.00	25.0	28.0	21.0	28.00	23.0	32.0	17.0	18.0	18.00	18.0	19.0	18.0	19.0	46.0
	Asian	Mean	61.3	4.35	18.51	17.6	16.6	17.0	15.91	17.0	16.9	6.62	6.05	5.540	5.29	5.43	5.40	5.59	19.2
		Std. Deviation	14.5	.659	6.199	5.62	4.63	6.04	5.117	6.13	4.74	3.94	3.41	4.200	3.59	3.57	4.43	3.81	11.3
		Range	54.0	3.06	23.00	23.0	20.0	26.0	25.00	24.0	18.0	17.0	13.0	16.00	16.0	17.0	16.0	17.0	42.0
	Native American	Mean	38.0	3.47	19.50	24.0	22.5	18.0	24.50	18.0	15.5	11.0	13.0	14.50	13.5	13.5	15.0	16.0	17.5
		Std. Deviation	5.65	1.49	9.192	12.7	10.6	2.82	16.26	7.07	3.53	4.24	.00	2.121	7.77	12.0	7.07	8.48	14.8
		Range	8.00	2.12	13.00	18.0	15.0	4.00	23.00	10.0	5.00	6.00	.00	3.00	11.0	17.0	10.0	12.0	21.0
	African American	Mean	55.5	4.12	16.00	17.0	15.3	16.2	15.62	16.5	17.6	7.50	7.50	6.600	4.50	5.30	6.30	4.90	14.1
		Std. Deviation	17.8	.690	4.092	4.52	3.12	3.14	4.172	4.77	4.50	3.92	3.71	4.427	4.62	4.29	4.32	3.72	8.96
		Range	56.0	2.37	12.00	12.0	7.00	7.00	12.00	11.0	12.0	13.0	13.0	12.00	14.0	12.0	12.0	11.0	23.0
	Hispanic/Latino	Mean	63.2	4.21	18.53	19.1	17.5	19.8	18.81	18.0	15.7	8.00	7.07	5.692	7.46	4.69	5.30	5.61	13.5
		Std. Deviation	16.0	.978	4.73	5.44	4.66	6.49	5.723	5.30	3.98	3.74	2.69	3.591	3.38	4.26	3.56	3.73	7.07
		Range	49.0	2.84	15.00	14.0	13.0	20.0	18.00	15.0	11.0	13.0	10.0	12.00	10.0	14.0	12.0	12.0	22.0
	Other	Mean	52.6	3.98	18.33	16.6	16.9	19.2	17.78	15.8	18.0	6.68	7.62	6.312	6.31	6.68	6.31	6.25	13.3
		Std. Deviation	12.3	.549	4.253	4.44	5.11	6.82	6.506	4.61	6.60	3.53	2.57	3.516	3.96	4.52	3.59	4.18	8.30
		Range	45.0	1.82	13.00	15.0	15.0	21.0	18.00	15.0	17.0	12.0	10.0	12.00	13.0	16.0	14.0	13.0	26.0

Baseline. Rumination was positively correlated with baseline somatic symptoms and negative affect. These correlations support the hypotheses that higher rumination and negative affect are associated with more somatic symptoms.

Weekly. Somatic symptoms correlated positively with stressful events from .21 to .33. Somatic symptoms at baseline also correlated positively with negative affect (r = .43) and rumination (r = .36). Somatic symptoms correlated positively with rumination from .24 to .36. Somatic symptoms correlated positively with NA from .26 to .43. Stressful events correlated with rumination from .06 to .18, with weeks 1, 3, and 4 being significant. Stressful events correlated with NA from .13 to .24. Rumination and NA correlated positively at .54. Finally, depression significantly correlated with all other study variables. All correlations, except for certain weeks between stress and rumination, were significant at the .01 level.

Prospective Analyses

Analysis. My core model tested the prospective relationship between stressors and somatic symptoms, as well as examined baseline rumination and negative affect as moderators, using HLM 7. I first analyzed the level 1 model of the relationship between stress and somatic symptoms, then analyzed the level 2 model with the moderators individually and as a 3-way interaction. Baseline depression was controlled for in the model because in the preliminary analyses depression correlated positively with all other variables.

Step one: Does stress predict somatic symptoms? I first examined the hypothesis that higher levels of stressful events would predict higher levels of somatic symptoms across the study period. Weekly fluctuations in stressful/negative events was entered as the predictor variable in Level 1 with weekly somatic symptoms as the outcome variable (see equation below). Depression was controlled for in the analyses by adding it as a level 2 predictor. Results

supported my hypothesis that variability in stress would significantly predict variability in somatic symptoms [unstandardized coefficient = 0.091, SE = 0.021, $t(356) = 4.431$, $p < .001$]. Thus, an individual's variability in the number of stressful events experienced predicted the variability in that individual's somatic symptoms.

Step two: Do negative affect and rumination moderate the relationship between stress and somatic symptoms? I examined my model as a whole, which hypothesized that NA and rumination would moderate the main effect of stress on somatic symptoms. The proposed three-way interaction between stress, NA, and rumination unfortunately was not found to be significant [unstandardized coefficient = -0.005, SE = 0.023, $t(354) = -0.227$, $p = .821$]. Rumination did not significantly moderate the relationship between stress and somatic symptoms [unstandardized coefficient = 0.024, SE = 0.027, $t(354) = 0.866$, $p = .387$], and NA was also a nonsignificant moderator [unstandardized coefficient = 0.004, SE = 0.029, $t(354) = 0.128$, $p = .898$]. A significant predictor of somatic symptoms besides stressful events was the control variable, baseline depressive symptoms [unstandardized coefficient = 0.411, SE = 0.039, $t(356) = 10.441$, $p < .001$]. Thus, while stress was found to significantly predict somatic symptoms, NA and rumination did not moderate the relationship between stress and somatic symptoms as I hypothesized.

$$\text{SomaticSx} = \beta_{0i} + \beta_1(\text{Stress}) + e_{ti}$$

$$\beta_0 = \beta_{00} + \beta_{01}(\text{Depression}) + r_{0i}$$

$$\beta_1 = \beta_{10} + \beta_{11}(\text{Rum}) + \beta_{12}(\text{NA}) + \beta_{13}(\text{NAxRum}) + r_{1j}$$

I also examined my model without controlling for depression, but the hypothesized moderators NA, rumination, and the three-way interaction between stress, NA and rumination remained nonsignificant (see Table 5).

$$\text{SomaticSx} = \beta_{0i} + \beta_1(\text{Stress}) + e_{ti}$$

$$\beta_0 = \beta_{00} + r_{0i}$$

$$\beta_1 = \beta_{10} + \beta_{11}(\text{Rum}) + \beta_{12}(\text{NA}) + \beta_{13}(\text{NAxRum}) + r_{1j}$$

Table 4

Final estimation of Fixed Effects for Stress, Rumination, and Negative Affect to Somatic Symptoms with Baseline Depression

Fixed Effect	Coefficient	Standard error	*t*-ratio	Approx. *d.f.*	*p*-value
For INTRCPT1, π_0					
INTRCPT2, β_{00}	0.008164	0.039685	0.206	356	0.837
CESD, β_{01}	0.410763	0.039336	10.442	356	<0.001
For STRESS slope, π_1					
INTRCPT2, β_{10}	0.088275	0.025285	3.491	354	<0.001
ATQ-NA, β_{11}	0.003700	0.028821	0.128	354	0.898
RRS, β_{12}	0.023578	0.027227	0.866	354	0.387
NAXRRS, β_{13}	-0.005184	0.022837	-0.227	354	0.821

Table 5

Final estimation of Fixed Effects for Stress, Rumination, and Negative Affect to Somatic Symptoms without Baseline Depression

Fixed Effect	Coefficient	Standard error	*t*-ratio	Approx. *d.f.*	*p*-value
For INTRCPT1, π_0					
INTRCPT2, β_{00}	0.010333	0.045407	0.228	357	0.820
For STRESS slope, π_1					
INTRCPT2, β_{10}	0.088781	0.025217	3.521	354	<0.001
ATQ-NA, β_{11}	-0.006432	0.028675	-0.224	354	0.823
RRS, β_{12}	0.017648	0.027081	0.652	354	0.515
NAXRRS, β_{13}	-0.007391	0.022731	-0.325	354	0.745

I then proceeded to examine rumination and NA as single moderators in separate models, and without controlling for depression in either model. When examining rumination alone as a moderator, the model was not found to be significant [unstandardized coefficient = 0.014, SE = 0.024, t(356) = 0.600, p = .549]. NA, also when examined in its own model as a moderator, was not significant [unstandardized coefficient = 0.000, SE = 0.025, t(356) = 0.004, p = .997]. Thus, rumination and NA were not significant even after removing depression as a control variable, however rumination did move closer in the hypothesized direction toward significance when depression was removed while NA appeared to remain unchanged. The three-way interaction between stress, NA, and rumination also did not significantly moderate the relationship between stress and somatic symptoms.

Post hoc power analysis

I then proceeded to conduct a post hoc power analysis to determine if my study was sufficiently powered. I used an online calculator (Soper, n.d.) to find the observed power for a significance test of the addition of interaction effect to the hierarchical model, over and above the independent predictors, which found my power to be .56. This value is well below the suggested power of .80. Therefore, it is possible that my hypotheses were not found to be significant due to insufficient power, and it would be worth repeating these analyses with a higher N in future studies to see if higher power affects the results.

Table 6

Fixed Effects of Stress and Rumination to Somatic Symptoms

Fixed Effect	Coefficient	Standard error	*t*-ratio	Approx. *d.f.*	*p*-value
For INTRCPT1, π_0					
INTRCPT2, β_{00}	0.010334	0.045407	0.228	357	0.820
For STRESS slope, π_1					
INTRCPT2, β_{10}	0.089097	0.025054	3.556	356	<0.001

RRS, β_{11}	0.014220	0.023699	0.600	356	0.549

Table 7

Fixed Effects of Stress and Negative Affect to Somatic Symptoms

Fixed Effect	Coefficient	Standard error	*t*-ratio	Approx. *d.f.*	*p*-value
For INTRCPT1, π_0					
INTRCPT2, β_{00}	0.010332	0.045406	0.228	357	0.820
For STRESS slope, π_1					
INTRCPT2, β_{10}	0.090414	0.025046	3.610	356	<0.001
ZMEAN_AT, β_{11}	0.000094	0.025183	0.004	356	0.997

CHAPTER IV

Discussion

The purpose of my study was to better understand the relationship between stress and somatic symptoms, and specifically to examine whether two individual vulnerability factors (negative affect and rumination) strengthen this relationship. I hypothesized that someone high in negative affect and/or rumination would be more vulnerable to experiencing somatic symptoms when faced with life stressors. I examined my hypotheses prospectively over 8 weeks, in order to see if fluctuations in stress affected fluctuations in somatic symptoms, and to determine if trait negative affect (NA) and rumination impacted the well-documented relationship between stress and somatic symptoms over time. My study examined the overall hypothesis that people who experience more stressors would also experience more somatic symptoms, and that this relationship would be even stronger for those individuals higher in trait NA and/or rumination.

This study makes several unique contributions to the literature on somatic symptoms. First, a vulnerability-stress model has been extensively studied in the context of depression (Hankin, 2008; Mezulis, Funasaki, Charbonneau, & Hyde, 2010; Seeds & Dozois, 2010), but not with somatic symptoms at the outcome. Because somatic complaints are a common reason patients present to primary care (Janca, Isaac, & Ventouras, 2006), it is important to understand the cognitive and affective vulnerabilities that lead some individuals to report more somatic symptoms than others when under stress. In addition, rumination and negative affect, two vulnerabilities studied in the depression literature (Brown, Chorpita, & Barlow, 1998; Koval, Kuppens, Allen, & Sheeber, 2012), had yet to be examined in relation to stress and somatic symptoms prior to my study. Finally, most studies I found on the psychological factors related to

somatic symptoms were cross-sectional analyses, thus my study was novel in using the 8-week diary study design to examine relationships over time.

In the following sections, I will describe the outcomes of each hypothesis beginning with my main effect and then examining each moderator separately and together. First, my hypothesis that variability in stress over time would predict somatic symptoms over time (specifically that greater stress predicts greater somatic symptoms) was supported. Second, my hypothesis that rumination would moderate this relationship was not supported in the original model after controlling for depression. Additionally, my hypothesis that negative affect would moderate this relationship was not supported. Finally, the interaction between rumination and negative affect to moderate the relationship between stress and somatic symptoms was not significant. These hypotheses are discussed in more detail below.

Was Stress Associated with Greater Somatic Symptoms?

The relationship between stress and somatic symptoms is an important area of study because all people experience stress to some degree, and stress affects several of the body's major systems (cardiovascular, gastrointestinal) and can lead to not only physical symptoms but poor physical health (Gianaros & Wager, 2015). For example, people with irritable bowel syndrome (IBS) report more life event stress than healthy controls in part due to chronic inflammation of the GI system (Whitehead, Crowell, & Robinson, 1992), and people experiencing greater stress are also at increased risk for cardiovascular disease in part because stress creates physiological reactions in the body such as increased blood pressure and heart rate as the sympathetic nervous system is chronically activated (Gianaros & Wager, 2015). My study expands upon the literature by examining the relationship between stress and somatic symptoms prospectively over multiple time points. The only study I found that studied number of stressors

predicting somatic symptoms prospectively only used two time points and studied adolescents instead of adults (Murberg, 2012). Thus it is important to examine how fluctuations in stress over time predict fluctuations in somatic symptoms over time.

My hypothesis that fluctuations in stress would predict fluctuations in somatic symptoms, while controlling for week 1 depression, was supported. Specifically, the greater number of stressors an individual experienced, the higher the somatic symptoms that were experienced. These findings support the literature that a greater number of psychosocial stressors predicts greater somatic symptoms, and that despite somatic symptoms' high correlation with depression and anxiety, stressors predict somatic symptoms even while controlling for such variables (Haftgoli et al., 2010). Past studies support the relationship between stress and somatic symptoms within a group, however studies that examined within-individual changes over time have found stress and somatic symptoms to not be significantly correlated, and fluctuations in stress only predicted fluctuations in depression or anxiety (Hertig, Cain, Jarrett, Burr, & Heitkemper, 2007). Thus, my study demonstrates that not only are stress and somatic symptoms correlated, but that stress prospectively predicts somatic symptoms within an individual.

Do Rumination and/or Negative Affect Moderate the Relationship Between Stress and Somatic Symptoms?

Negative affect. Past studies have suggested that higher negative affect predicts the development and persistence of somatic symptoms, with the possible explanation being that NA and depression are highly correlated and people with depression often experience somatic complaints (De Gucht, Fischler, & Heiser, 2004). However, NA had yet to be studied as a moderator of stress and somatic symptoms, therefore my study was unique by examining NA as a vulnerability factor for certain individuals, wherein stressful events may be more likely to

trigger somatic symptoms. It also attempted to separate depression as the reason NA and somatic symptoms might be related by controlling for baseline depression in the model, because when depression was removed I was able to more clearly examine the temperamental tendency to experience negative emotions rather than simply measuring current levels of one type of negative emotion. In this study, negative affect was not found to moderate the relationship between stress and somatic symptoms while controlling for depression; therefore my hypothesis was not supported. I then attempted to examine NA as a single moderator in its own model and without controlling for depression, and it still was not significant. My results do not support the significance of NA as a significant contributor to strengthening the relationship between stress and somatic symptoms in a young adult population. These results do not reflect past literature's findings that NA predicts somatic symptoms (Thompson, Waltz, Croyle, & Pepper, 2007) and correlates with stress (Watson, 1988). I will attempt to explain why this was the case below.

One possibility for why negative affect was not statistically significant at moderating the relationship between stress and somatic symptoms is that NA is too broad a construct. NA includes several negatively valenced emotions including fear, anger, and sadness. The hypothesized model predicted that individuals high in NA, without differentiating between the subtypes of affect, would be more likely to experience somatic symptoms when faced with stress, but it may be that some forms of affect but not others are more related to the other variables (stress, rumination, somatic symptoms) in the model. In addition, trait NA is a stable temperamental tendency to experience frequent and intense negative emotions (Watson, 1988), and depressive symptoms are a state index of current levels of one category of NA, namely sadness (Harding, Willey, Ahles, & Mezulis, 2016) therefore it may be that current symptoms of depression play a larger role in the development of somatic symptoms than does trait NA

because they reflect current distress and a deviation from one's baseline, while one's temperament is fairly stable.

Moreover, it may be that experiencing negative affect alone is not enough to put someone at risk for increased somatic symptoms, but rather how they choose to cope with that affect. For example, Miers, Rieffe, Terwogt, Cowan, and Linden (2007) found that children and adolescents who ruminated over their anger experienced more somatic complaints than those who coped with their anger in other ways. Thus, while the vulnerability-stress model emphasizes the importance of both cognitive and affective vulnerabilities in moderating negative health outcomes such as somatic symptoms, research on coping with negative affect appears to indicate that emotional vulnerabilities are a greater problem when coupled with a maladaptive cognitive coping strategy such as rumination.

Finally, another hypothesis for why high negative affect did not moderate the relationship between stress and somatic symptoms is that people who experience more somatic symptoms have difficulty recognizing and naming their emotions and therefore would not endorse high NA. Waller and Scheidt (2006) reviewed literature on somatoform disorders in the context of emotion regulation and posited that "somatoform disorders are linked to a diminished capacity to consciously experience and differentiate affects and express them in an adequate or healthy way" (p. 13). In addition, Lilly and Valdez (2012) found that in individuals with a history of trauma (an extreme stressor), emotion regulation difficulties predicted PTSD symptoms and somatic symptoms, but alexithymia, or the inability to identify and describe emotions, moderated the relationship between emotion regulation problems and somatic symptoms (but not PTSD symptoms). Thus, there is evidence that people who have high levels of somatic symptoms have difficulty recognizing their own emotions and may as a result not be able to accurately endorse

their own levels of negative affect, and the NA measure in my study was entirely self-report. Use of self-report measures will be further discussed in limitations and future directions.

Rumination. Few studies have examined rumination's relationship with somatic symptoms, and the few that exist showed that rumination predicted the somatic symptoms of a depression diagnosis (Wilkinson, Croudace, & Goodyer, 2013). My study attempted to examine rumination as a moderator between stress and somatic symptoms in a unique way, that is prospectively and while controlling for depression.

In my original model with negative affect and rumination as co-moderators (after controlling for depression), rumination was not found to be significant and my hypothesis was not supported. However, when I examined rumination as a single moderator between stress and somatic symptoms in its own model, without controlling for depression, rumination was still not significant, though moved toward significance in the hypothesized direction. Thus, it appears that rumination may have a larger impact on the relationship between stress and somatic symptoms than does negative affect. Speculations for this finding will be discussed below.

One reason that rumination was not a significant moderator but seemed to move closer toward significance after depression was removed could be because the measure I used to assess rumination, the Ruminative Response Scale, may also perform as a measure of depressive rumination, meaning repetitively thinking about one's depressive symptoms. Rumination and depression are so highly correlated (Koval, Kuppens, Allen, & Sheeber, 2012; Michl, McLaughlin, Shepherd, Nolen-Hoeksema, 2013), it may be that controlling for the depressive aspect of rumination removed too much of the variance in the measure to be able to purely examine rumination as perseverating on a common theme. Future studies could address this issue by using the Brooding subscale of the RRS, which is a measure of passive and judgmental

pondering of one's mood and does not include depressive items (Whitmey & Gotlib, 2011). It also might be that rumination merely correlates with somatic symptoms as they relate to depression, which often involves a somatic component (Wilkinson, Croudace, & Goodyer, 2013), rather than somatic symptoms overall. Individuals who experience somatic symptoms but not depressive symptoms may be a particular subset of people who have the aforementioned alexithymia and therefore cannot accurately be studied using self-report measures. Measuring vulnerabilities through physiological variables such as heart rate, perspiration, or cortisol levels may be a better way to study people who experience high levels of somatic symptoms yet who do not identify as depressed.

Interaction between NA and rumination. In addition to hypothesizing that rumination and negative affect would each moderate the relationship between stress and somatic symptoms, I also proposed that the interaction between NA and rumination would be the strongest moderator than either variable would be alone. My full hypothesized model with this interactive effect was not supported. Several reasons for why NA and rumination would not serve as significant moderators were hypothesized in the previous sections above. Because the control variable, depression, was the only significant predictor of somatic symptoms in the model besides stress, it appears that depression better explains stress' impact on somatic symptoms than does NA and rumination in this study specifically. Moreover, while the number of stressful events did significantly predict somatic symptoms in the model, level of perceived distress over those stressful events was not measured, and it may be that individuals with higher perceived distress or who appraise life stressors as threatening are more likely to ruminate and experience high negative affect and that this group of individuals experience the most somatic symptoms. Future studies should incorporate both a stressor count and a measure of stress appraisal to

understand how each aspect of the experience of stress impacts physical and psychological functioning.

Clinical Application

Although only parts of my model were found to be significant, research demonstrates significant positive correlations between stress, NA, rumination, and somatic symptoms (Burton, Farley, & Rhea, 2009; Thomsen et al., 2003; Watson, 1988). Therefore, researchers should continue to study how stress, NA, and rumination relate to somatic symptoms, with the addition of depression as a key variable.

One important topic clinicians should address when attempting to treat patients with somatic symptoms is the relationship between stress and physical health. An alternative or complement to simply treating the symptoms themselves, for example providing opiates or muscle relaxants for pain, is psychoeducation on the impact of stress on the body and ways to manage that stress in day to day life. Managing stress is not only essential to subjectively "feeling better" or more relaxed, but stress-management group interventions such as mindfulness-based stress reduction (MBSR) and cognitive-behavioral stress management (CBSM) have been shown to predict a reduction in levels of cortisol (often known as the stress hormone) in the body (Philips et al., 2011; Stefanaki et al., 2014). MBSR has also been implemented in a primary care setting with patients with chronic pain and showed reductions in pain severity and psychological distress and an increase in engagement in life activities (Beaulac & Bailly, 2015). My study not only showed a positive relationship between stress and somatic symptoms, but established a relationship over time such that as stress levels changed week by week, so did levels of somatic symptoms. Thus, while individuals may not be able to predict or control how many stressors they face on a given day or week, they can utilize stress-management

techniques to lessen the impact those stressors have on their physical health and symptoms. In addition, clinicians should focus on the mechanisms through which stress management groups reduce somatic symptoms, because it may be that targeting vulnerabilities such as rumination or negative affect in people undergoing stress leads to reductions in somatic complaints. My study showed that when depression was not controlled for, rumination moved closer to significantly moderating the effect of stressors on somatic symptoms, therefore it is worth continuing to examine cognitive vulnerabilities, along with depressive symptoms, in stress-management interventions.

Limitations and Future Directions

While my study was the first prospective design to examine negative affect and rumination as potential moderators of the relationship between stress and somatic symptoms, my study was not without limitations that should be addressed in order to improve research on these variables in the future. These limitations include issues with measurement, theory, and generalizability of the sample.

Measurement. One limitation of my study was that all my variables were measured using self-report data, due to the ease and relatively low cost of administering these measures. A benefit of my study was the ability to examine relationships among variables over time using multiple data points, however accuracy of measurement would improve if future studies used at least one measure that is not self-report. For example, in addition to getting a self-reported stressor count, studies could experimentally induce stress through a task then measure perceived stress or reactivity through heart rate or perspiration.

In addition, I would have liked to incorporate a biological/physiological measure, such as cortisol levels, in order to more fully represent the biopsychosocial model of stress, but

unfortunately constraints on the study only allowed for the use of psychological moderators. Cruess et al. (2015) found that individuals who engaged in a stress management program not only reported less subjective distress during the intervention, but when faced with a stressful task they exhibited attenuated salivary assays of cortisol (sCORT) when compared with a control group. Future studies should expand upon this research by examining whether an individual's level of negative affect and rumination may moderate the relationship between salivary cortisol levels in response to a stressful task and self-reported somatic symptoms. Ideally research should include disciplines in addition to psychology (such as medicine or biology) to gain a more comprehensive perspective on the impact of stress on the body.

Theory. While rumination and negative affect are both important cognitive and affective vulnerabilities to study in the context of stress' impact on somatic symptoms, future studies should include a more comprehensive examination of potential moderators (or mediators), such as worry and catastrophizing. Rumination involves cognitively dwelling on present and past symptoms (Watkins, Moulds, & Mackintosh, 2005), but it is possible that when faced with stressful life events, people who experience greater somatic symptoms are those who worry about the future. Rumination, as stated in the literature review, is more related to depression, while worry is more related to anxiety (Purdon & Harrington, 2006). Research has shown that people with generalized anxiety disorder (GAD) not only experience excessive worry, but they also experience a high number of somatic complaints (Vijay, Avasthi, & Grover, 2014). In addition, several studies have shown that worry is associated with higher somatic complaints in the general population (Verkuil, Brosschot, Meerman, & Thayer, 2010) and that a reduction in worry predicts a reduction in somatic symptoms (Brosschot & Van der Doef, 2006). In theory rumination should function similarly to worry due to the perseverative cognition hypothesis

(Brosschot, Gerin, & Thayer, 2006), which states that perseverating on stress prolongs the physical health problems associated with stress, and perseverating can include both worry and rumination. However, it may be that worry is a more salient variable to target when aiming to reduce somatic symptoms in adults who are faced with stress, however more research should be done to compare the effects of worry versus rumination on physical symptoms.

Catastrophizing is often studied in the pain literature as excessive worry about one's pain, but it is more broadly defined as "an exaggerated negative orientation toward noxious stimuli" (Sullivan, Bishop & Pivik, 1995, p. 524). Like rumination and worry, it is also viewed as a form of negative repetitive thinking (Flink, Boersma, & Linton, 2013). Chan, Chan, and Kwok (2015) found that in adolescents, catastrophizing mediated the effect of daily hassles on anxiety when controlling for depressive symptoms. Garnefski and Kraaij (2009) found that in response to life stress, people who engaged in rumination or catastrophizing as cognitive coping strategies were more likely to experience depressive symptomatology. Finally, Gautreau, Sherry, Sherry, Birnie, Mackinnon, & Stewart (2015) found that catastrophizing of bodily symptoms maintained health anxiety (worry about perceived health problems) over time in young adults. Thus, future studies should examine all forms of negative repetitive thinking, including catastrophizing, rumination, and worry, as risk factors for developing greater somatic symptoms (not just for anxiety and depression) when faced with life stress.

Another theory-driven limitation in my study was the focus on factors that strengthen the relationship between stress and somatic symptoms, without examining variables that may weaken, or buffer, the relationship. For example, social support, coping style (problem-focused vs emotion-focused), and physical activity levels may all be protective factors in the vulnerability-stress model of somatic symptoms but have yet to be extensively studied in this

way. Stein and Smith (2015) found that social support buffered the relationship between perceived stress and physical symptoms in healthy adult women, therefore future studies should include social support as a moderator in addition to negative affect and rumination when studying women and men. Moreover, Christiansen, Copeland, and Stapert (2008) found that in adolescents daily hassles predicted higher somatic symptoms, and that substance use (a maladaptive coping strategy) positively predicted somatic symptoms while interpersonal coping (an adaptive coping strategy) negatively predicted somatic symptoms. Thus, coping style would be a useful variable to examine as a mediator or moderator between stressful events and somatic symptoms in adults. Finally, Heaney, Carroll, and Phillips (2014) found that older adults who regularly engaged in aerobic exercise exhibited lower cortisol response to high stress conditions than those who did not engage in exercise. However, to date there are no studies exploring physical activity as a buffer to the effect of stress on somatic symptoms. Overall, future studies should incorporate potential protective factors such as social support, certain coping styles, and physical activity in addition to examining the potential vulnerability factors.

Sample. My study examined college students who were primarily Caucasian and middle class, which may limit the generalizability of my findings. Moreover, all participants were volunteers and therefore may have certain characteristics that further limit the extent to which I can generalize the outcome. Because somatic symptoms are of particular concern in a primary care setting due to accounting for nearly half of all primary care visits (Janca, Isaac, & Ventouras, 2006), future studies should seek out participants in primary care to gain a more representative and diverse sample where there can be more room for effects over time rather than only studying health college students where they may be less room for fluctuations in variables.

Next steps. The ideal next step to expand upon this study is to test a stress management intervention while continuing to examine rumination and negative affect, as well as other possible moderators such as worry or catastrophizing, as moderators. While rumination and negative affect were not found to be significant moderators when controlling for depression in this study, all proposed variables in the study were correlated in the preliminary analyses and previous research does suggest both rumination and negative affect are related to stress levels and somatic symptoms, therefore they are worth continuing to explore, especially in the context of an intervention study. While particular treatments such as MBSR and CBSM have been shown to effectively reduce stress and pain (Philips et al., 2011; Stefanaki et al., 2014), less is known about the mechanisms through which these effects occur, and whether or not these interventions have any impact on somatic symptoms. Once research more firmly establishes which cognitive and affective vulnerabilities place individuals most at risk for somatic symptoms, it is essential that those vulnerabilities be targeted to reduce their negative impact. Additionally, buffering moderators, such as social support or problem-focused coping, may be useful in helping individuals better adapt to stress and subsequently experience less somatic symptoms; stress management interventions should aim to incorporate as many of these positive coping strategies as possible. As mentioned in Limitations, these intervention studies would be best given in a primary care setting to reach patients who seek help for somatic symptoms and could most benefit from managing their stress. Finally, measures of stress response such as salivary cortisol would be more beneficial than simply counting number of stressors, because while all people face life hassles every day, not all experience negative outcomes such as somatic symptoms. It is therefore important to assess how one's body reacts to life stressors via cortisol

not only to measure baseline levels so that vulnerable individuals can be identified, but as a way to track the effectiveness of an intervention in an objective way.

In conclusion, this study served as an important starting point to help direct the research on stress and somatic symptoms toward a better understanding of the cognitive and affective vulnerabilities at play in order to develop treatments to improve outcomes and reduce healthcare costs. At present, the salience of rumination and negative affect as moderators of the relationship between stress and somatic symptoms remains unclear, and more exploration is necessary to clarify their role as well as to explore other possible moderators. Future research expanding upon this study will hopefully lead to a better biopsychosocial conceptualization of somatic symptoms from a vulnerability-stress perspective.

References

Bandura, A. (1973). *Aggression: A social learning analysis*. Englewood Cliffs, NJ: Prentice Hall

Barsky, Arthur J. (2000). The science of self-report: Implications for research and practice. In A. A. Stone, J. S. Turkkan, C. A. Bachrach, J. B. Jobe, H. S. Kurtzman, V. S. Cain (Eds.), Mahwah, NJ, US: Lawrence Erlbaum Associates Publishers.

Barsky, A. J., Orav, E., & Bates, D. W. (2005). Somatization Increases Medical Utilization and Costs Independent of Psychiatric and Medical Comorbidity. *Archives Of General Psychiatry*, *62*(8), 903-910. doi:10.1001/archpsyc.62.8.903

Borkovec, T. D., Ray, W. J., & Stöber, J. (1998). Worry: A Cognitive Phenomenon Intimately Linked to Affective, Physiological, and Interpersonal Behavioral Processes. *Cognitive Therapy & Research*, *22*(6), 561.

Brosschot, J. F., Gerin, W., & Thayer, J. F. (2006). The perseverative cognition hypothesis: A review of worry, prolonged stress-related physiological activation, and health. *Journal Of Psychosomatic Research*, *60*(2), 113-124. doi:10.1016/j.jpsychores.2005.06.074

Brosschot, J.F., & Van Der Doef, M. (2006). Daily worrying and somatic health complaints: Testing the effectiveness of a simple worry reduction intervention. Psychology & Health, 21, 19–31.

Brown, T. A., Chorpita, B. F., & Barlow, D. H. (1998). Structural relationships among dimensions of the DSM-IV anxiety and mood disorders and dimensions of negative affect, positive affect, and autonomic arousal. *Journal Of Abnormal Psychology*, *107*(2), 179-192. doi:10.1037/0021-843X.107.2.179

Burton, C. (2003). Beyond somatisation: a review of the understanding and treatment of medically unexplained physical symptoms (MUPS). *Br J Gen Pract. 53(488*), 231–239

Burton, T., Farley, D., & Rhea, A. (2009). Stress-induced somatization in spouses of deployed and nondeployed servicemen. *Journal Of The American Academy Of Nurse Practitioners*, *21*(6), 332-339. doi:10.1111/j.1745-7599.2009.00411.x

Castelloe, J. M., & O'Brien, R. G. (2000). *Power and sample size determination for linear models*. SUGI Proceedings. Cary, NC: SAS Institute Inc.

Chan, S. M., Chan, S. K., & Kwok, W. W. (2015). Ruminative and catastrophizing cognitive styles mediate the association between daily hassles and high anxiety in Hong Kong adolescents. *Child Psychiatry And Human Development*, *46*(1), 57-66. doi:10.1007/s10578-014-0451-9

Christiansen, L. M., Copeland, E. P., & Stapert, E. B. (2008). Predictors of somatic symptoms in younger rural adolescents. *Adolescence*, *43*(172), 791-806.

Chrousos, G. P., & Gold, P. W. (1992). The concept of stress and stress system disorders. Overview of physical and behavioral homeostasis. *JAMA, 267,* 1244–1252.

Cruess, D. G., Finitsis, D. J., Smith, A., Goshe, B. M., Burnham, K., Burbridge, C., & O'Leary, K. (2015). Brief stress management reduces acute distress and buffers physiological response to a social stress test. *International Journal Of Stress Management*, *22*(3), 270-286. doi:10.1037/a0039130

Deary, V. V., Chalder, T. T., & Sharpe, M. M. (2007). The cognitive behavioural model of medically unexplained symptoms: A theoretical and empirical review. *Clinical Psychology Review*, *27*(7), 781-797. doi:10.1016/j.cpr.2007.07.002

De Gucht, V., Fischler, B., & Heiser, W. (2004). Neuroticism, alexithymia, negative affect, and positive affect as determinants of medically unexplained symptoms. *Personality And Individual Differences*, *36*(7), 1655-1667. doi:10.1016/j.paid.2003.06.012

Derakshan, N., Eysenck, M. W., & Myers, L. B. (2007). Emotional information processing in repressors: The vigilance-avoidance theory. *Cognition & Emotion, 21*(8), 1585-1614. doi:10.1080/02699930701499857

Deyo, R.A., Walsh, N.E., Schoenfeld, L.S., & Ramamurthy, S. (1989). Studies of the Modified Somatic Perceptions Questionnaire (MSPQ) in patients with back pain: psychometric and predictive properties. *Spine, 14,* 507–510.

Eriksen, H. R., & Ursin, H. H. (2004). Subjective health complaints, sensitization, and sustained cognitive activation (stress). *Journal Of Psychosomatic Research*, *56*(4), 445-448. doi:10.1016/S0022-3999(03)00629-9

Escobar, J. I., Cook, B., Chen, C., Gara, M. A., Alegría, M., Interian, A., & Diaz, E. (2010). Whether medically unexplained or not, three or more concurrent somatic symptoms predict psychopathology and service use in community populations. *Journal Of Psychosomatic Research, 69*(1), 1-8. doi:10.1016/j.jpsychores.2010.01.001

Evans, D.E., & Rothbart, M.K. (2007). Developing a model for adult temperament. *Journal of Research in Personality, 41,* 868-888. doi: 10.1016/j.jrp.2006.11.002

Faul, F., Erdfelder, E., Lang, A.-G., & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods, 39,* 175-191

Feinstein, A. (2011). Conversion disorder: Advances in our understanding. *Canadian Medical Association Journal*, *183*(8), 915-919. doi:10.1503/cmaj.110490

Fink, P., & Rosendal, M. (2008). Recent developments in the understanding and management of functional somatic symptoms in primary care. *Current Opinion In Psychiatry*, *21*(2), 182-188. doi:10.1097/YCO.0b013e3282f51254

Flink, I. L., Boersma, K., & Linton, S. J. (2013). Pain catastrophizing as repetitive negative thinking: A development of the conceptualization. *Cognitive Behaviour Therapy*, *42*(3), 215-223. doi:10.1080/16506073.2013.769621

Fröhlich, C., Jacobi, F., & Wittchen, H. (2006). DSM-IV pain disorder in the general population: An exploration of the structure and threshold of medically unexplained pain symptoms. *European Archives Of Psychiatry And Clinical Neuroscience*, *256*(3), 187-196. doi:10.1007/s00406-005-0625-3

Garnefski, N., & Kraaij, V. (2009). Cognitive coping and psychological adjustment in different types of stressful life events. *Individual Differences Research*, *7*(3), 168-181.

Garnefski, N. N., Kraaij, V. V., & Spinhoven, P. P. (2001). Negative life events, cognitive emotion regulation and emotional problems. *Personality And Individual Differences*, *30*(8), 1311-1327. doi:10.1016/S0191-8869(00)00113-6

Gautreau, C. M., Sherry, S. B., Sherry, D. L., Birnie, K. A., Mackinnon, S. P., & Stewart, S. H. (2015). Does catastrophizing of bodily sensations maintain health-related anxiety? A 14-day daily diary study with longitudinal follow-up. *Behavioural And Cognitive Psychotherapy*, *43*(4), 502-512. doi:10.1017/S1352465814000150

Gendolla, G. E., Abele, A. E., Andrei, A., Spurk, D., & Richter, M. (2005). Negative Mood, Self-Focused Attention, and the Experience of Physical Symptoms: The Joint Impact Hypothesis. *Emotion*, *5*(2), 131-144. doi:10.1037/1528-3542.5.2.131

Genet, J. J., & Siemer, M. (2012). Rumination moderates the effects of daily events on negative mood: Results from a diary study. *Emotion*, *12*(6), 1329-1339. doi:10.1037/a0028070

Gianaros, P. J., & Wager, T. D. (2015). Brain-body pathways linking psychological stress and physical health. *Current Directions In Psychological Science, 24*(4), 313-321. doi:10.1177/0963721415581476

Goldstein, S., Chesir-Teran, D., & McFaul, A. (2008). Profiles and correlates of relational aggression in young adults' romantic relationships. *Journal of Youth and Adolescence, 37*(3), 251-265. doi:10.1007/s10964-007-9255-6

Griffith, J. W., & Raes, F. (2015). Factor structure of the Ruminative Responses Scale: A community-sample study. *European Journal Of Psychological Assessment*, *31*(4), 247-253. doi:10.1027/1015-5759/a000231

Haftgoli, N., Favrat, B., Verdon, F., Vaucher, P., Bischoff, T., Burnand, B., & Herzig, L. (2010). Patients presenting with somatic complaints in general practice: depression, anxiety and somatoform disorders are frequent and associated with psychosocial stressors. *BMC Fam Pract***,** *11,* 67

Hankin, B. L. (2008). Cognitive vulnerability-stress model of depression during adolescence: Investigating depressive symptom specificity in a multi-wave prospective study. *Journal Of Abnormal Child Psychology*, *36*(7), 999-1014. doi:10.1007/s10802-008-9228-6

Harding, K., Willey, B., Ahles, J., & Mezulis, A. (2016). Disentangling vulnerabilities from outcomes: Distinctions between trait affect and depressive symptoms in adolescent and adult samples. *Journal of Affective Disorders.* Advance online publication.

Heaney, J. J., Carroll, D., & Phillips, A. C. (2014). Physical activity, life events stress, cortisol, and DHEA: Preliminary findings that physical activity may buffer against the negative effects of stress. *Journal of Aging And Physical Activity*, *22*(4), 465-473. doi:10.1123/JAPA.2012-0082

Hertig, V. L., Cain, K. C., Jarrett, M. E., Burr, R. L., & Heitkemper, M. M. (2007). Daily stress and gastrointestinal symptoms in women with irritable bowel syndrome. *Nursing Research, 56*(6), 399-406. doi:10.1097/01.NNR.0000299855.60053.88

Hiller, W., Rief, W., & Brähler, E. (2006). Somatization in the population: From mild bodily misperceptions to disabling symptoms. *Social Psychiatry And Psychiatric Epidemiology, 41*(9), 704-712. doi:10.1007/s00127-006-0082

Hoff, E., & Muehlenkamp, J. (2009). Nonsuicidal self-injury in college students: The role of perfectionism and rumination. *Suicide and Life-Threatening Behavior, 39*(6), 576-587. doi:10.1521/suli.2009.39.6.576

Husain, K., Browne, T., & Chalder, T. (2007). A Review of Psychological Models and Interventions for Medically Unexplained Somatic Symptoms in Children. *Child And Adolescent Mental Health, 12*(1), 2-7. doi:10.1111/j.1475-3588.2006.00419.x

Hwang, G., Kim, S., Kim, J., Kim, H., Park, S., & Kim, S. (2008). Influence of psychological stress on physical pain. *Stress And Health: Journal Of The International Society For The Investigation Of Stress, 24*(2), 159-164. doi:10.1002/smi.1170

Ingram, R. E., & Luxton, D. D. (2005). Vulnerability-Stress Models. In B. L. Hankin, J. Z. Abela (Eds.) , *Development of psychopathology: A vulnerability-stress perspective* (pp. 32-46). Thousand Oaks, CA US: Sage Publications, Inc.

Janca, A., Isaac, M., & Ventouras, J. (2006). Towards better understanding and management of somatoform disorders. *International Review Of Psychiatry, 18*(1), 5-12. doi:10.1080/09540260500466766

Jansson-Fröjmark, M., & MacDonald, S. (2009). Exploratory factor analysis of the Modified Somatic Perception Questionnaire on a sample with insomnia symptoms. *Psychology, Health & Medicine*, *14*(1), 62-72. doi:10.1080/13548500802001793

Johnson, S. L., McKenzie, G., & McMurrich, S. (2008). Ruminative responses to negative and positive affect among students diagnosed with bipolar disorder and major depressive disorder. *Cognitive Therapy And Research*, *32*(5), 702-713. doi:10.1007/s10608-007-9158-6

Kirkwood, C. R., Clure, H. R., Brodsky, R., Gould, G. H., Knaak, R., Metcalf, M., & Romeo, S. (1982). The diagnostic content of family practice: 50 most common diagnoses recorded in the WAMI community practices. *Journal of Family Practice, 15,* 485–492.

Kirmayer, L. J., & Young, A. (1998). Culture and somatization: Clinical, epidemiological, and ethnographic perspectives. *Psychosomatic Medicine*, *60*(4), 420-430.

Kolk, A. M., Hanewald, G. P., Schagen, S., & van Wijk, C. (2003). A symptom perception approach to common physical symptoms. *Social Science & Medicine*, *57*(12), 2343-2354. doi:10.1016/S0277-9536(02)00451-3

Koval, P., Kuppens, P., Allen, N. B., & Sheeber, L. (2012). Getting stuck in depression: The roles of rumination and emotional inertia. *Cognition And Emotion*, *26*(8), 1412-1427. doi:10.1080/02699931.2012.667392

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. Springer: New York

Lieb, R., Pfister, H., Mastaler, M., & Wittchen, H. (2000). Somatoform syndromes and disorders in a representative population sample of adolescents and young adults: Prevalence, comorbidity and impairments. *Acta Psychiatrica Scandinavica,101*(3), 194-208. doi:10.1046/j.0902-4441.2000.ap90071.x

Lilly, M., & Valdez, C. (2012). The unique relationship of emotion regulation and alexithymia in predicting somatization versus PTSD symptoms. *Journal Of Aggression, Maltreatment & Trauma*, *21*(6), 609-625. doi:10.1080/10926771.2012.686964

Linden, W., Earle, T. L., Gerin, W. W., & Christenfeld, N. N. (1997). Physiological stress reactivity and recovery: Conceptual siblings separated at birth?. *Journal Of Psychosomatic Research*, *42*(2), 117-135. doi:10.1016/S0022-3999(96)00240-1

Main, C. J. (1983). The modified somatic perception questionnaire (MSPQ). *Journal of Psychosomatic Research, 27*(6), 503-514. doi:10.1016/0022-3999(83)90040-5

Martin, L. L., & Tesser, A. (1996). Some ruminative thoughts. *Advances in social cognition*, *9*, 1-47

Mathieu, J. E., Aguinis, H., Culpepper, S. A., & Chen, G. (2012). Understanding and estimating the power to detect cross-level interaction effects in multilevel modeling. *Journal Of Applied Psychology*, *97*(5), 951-966. doi:10.1037/a0028380

Maybery, D. J. (2003). Incorporating interpersonal events within hassle measurement. *Stress and Health, 19,* 97-110

Maybery, D. J., Neale, J., Arentz, A., & Jones-Ellis, J. (2007). The Negative Event Scale: Measuring frequency and intensity of adult hassles. *Anxiety, Stress & Coping: An International Journal*, *20*(2), 163-176. doi:10.1080/10615800701217654

Mellner, C., Krantz, G., & Lundberg, U. (2005). Medically unexplained symptoms in women as related to physiological stress responses. *Stress & Health: Journal Of The International Society For The Investigation Of Stress*, *21*(1), 45-52. doi:10.1002/smi.1037

Menninger, W. C. (1947). Psychosomatic medicine: somatization reactions. *Psychosomatic Medicine*, 92-97

Mezulis, A. H., Funasaki, K. S., Charbonneau, A. M., & Hyde, J. (2010). Gender differences in the cognitive vulnerability-stress model of depression in the transition to adolescence. *Cognitive Therapy And Research*, *34*(6), 501-513. doi:10.1007/s10608-009-9281-7

Michalak, J., Hölz, A., & Teismann, T. (2011). Rumination as a predictor of relapse in mindfulness-based cognitive therapy for depression. *Psychology And Psychotherapy: Theory, Research And Practice*, *84*(2), 230-236. doi:10.1348/147608310X520166

Michl, L. C., McLaughlin, K. A., Shepherd, K., & Nolen-Hoeksema, S. (2013). Rumination as a mechanism linking stressful life events to symptoms of depression and anxiety: Longitudinal evidence in early adolescents and adults. *Journal Of Abnormal Psychology*, *122*(2), 339-352. doi:10.1037/a0031994

Miers, A. C., Rieffe, C., Terwogt, M. M., Cowan, R., & Linden, W. (2007). The relation between anger coping strategies, anger mood and somatic complaints in children and adolescents. *Journal Of Abnormal Child Psychology*, *35*(4), 653-664. doi:10.1007/s10802-007-9120-9

Morrison, R., & O'Connor, R. C. (2005). Predicting Psychological Distress in College Students: The Role of Rumination and Stress. *Journal Of Clinical Psychology*, *61*(4), 447-460. doi:10.1002/jclp.20021

Murberg, T. (2012). The influence of optimistic expectations and negative life events on somatic symptoms among adolescents: A one-year prospective study. *Psychology*, *3*(2), 123-127. doi:10.4236/psych.2012.32018

Nimnuan, C., Hotopf, M., & Wessely, S. (2001). Medically unexplained symptoms: An epidemiological study in seven specialities. *Journal Of Psychosomatic Research*, *51*(1), 361-367. doi:10.1016/S0022-3999(01)00223-9

Nolen-Hoeksema, S. (1991). Responses to depression and their effects on the duration of depressive episodes. *Journal of Abnormal Psychology, 100*(4), 569-582. doi:10.1037/0021-843X.100.4.569

Nolen-Hoeksema, S., Wisco, B.E., & Lyubomirksy, S. (2008). Rethinking rumination. *Perspectives on Psychological Science, 3,* 400-424.

Parent, M. C. (2013). Handling item-level missing data: Simpler is just as good. *The Counseling Psychologist, 41,* 568–600. doi:10.1177/0011000012445176

Phillips, K. M., Antoni, M. H., Carver, C. S., Lechner, S. C., Penedo, F. J., McCullough, M. E., & ... Blomberg, B. B. (2011). Stress management skills and reductions in serum cortisol across the year after surgery for non-metastatic breast cancer. *Cognitive Therapy And Research*, *35*(6), 595-600. doi:10.1007/s10608-011-9398-3

Purdon, C., & Harrington, J. (2006). Worry in psychopathology. In G. C. L. Davies & A. Wells (Eds.), Worry and its psychological disorders: Theory, assessment and treatment (pp. 41–50). Chichester, UK: John Wiley & Sons.

Radloff, L. (1977). The CES-D Scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement, 1,* 385-401. doi:10.1177/014662167700100306

Rief, W., & Broadbent, E. (2007). Explaining medically unexplained symptoms-models and mechanisms. *Clinical Psychology Review*, *27*(7), 821-841. doi:10.1016/j.cpr.2007.07.005

Roelofs, K., & Spinhoven, P. (2007). Trauma and medically unexplained symptoms: Towards an integration of cognitive and neuro-biological accounts. *Clinical Psychology Review*, *27*(7), 798-820. doi:10.1016/j.cpr.2007.07.004

Rozzini, R., Bianchetti, A., Carabellese, C., Inzoli, M., & Trabucchi, M. (1988). Depression, life events and somatic symptoms. *The Gerontologist*, *28*(2), 229-232. doi:10.1093/geront/28.2.229

Seeds, P. M., & Dozois, D. A. (2010). Prospective evaluation of a cognitive vulnerability-stress model for depression: The interaction of schema self-structures and negative life events. *Journal Of Clinical Psychology*, *66*(12), 1307-1323. doi:10.1002/jclp.20723

Selye, H. (1936). Syndrome produced by diverse nocuous agents. *Nature*, *138*, 32

Soper, D. (n.d.). CALCULATOR: POST-HOC STATISTICAL POWER FOR HIERARCHICAL MULTIPLE REGRESSION. Retrieved from http://www.danielsoper.com/statcalc/calculator.aspx?id=17

Stefanaki, C., Bacopoulou, F., Livadas, S., Kandaraki, A., Karachalios, A., Chrousos, G. P., & Diamanti-Kandarakis, E. (2015). Impact of a mindfulness stress management program on stress, anxiety, depression and quality of life in women with polycystic ovary syndrome: A randomized controlled trial. *Stress: The International Journal On The Biology Of Stress*, *18*(1), 57-66. doi:10.3109/10253890.2014.974030

Stein, E. R., & Smith, B. W. (2015). Social support attenuates the harmful effects of stress in healthy adult women. *Social Science & Medicine*, *146*129-136. doi:10.1016/j.socscimed.2015.10.038

Sullivan, M. L., Bishop, S. R., & Pivik, J. (1995). The Pain Catastrophizing Scale: Development and validation. *Psychological Assessment*, *7*(4), 524-532. doi:10.1037/1040-3590.7.4.524

Tak, L. M., & Rosmalen, J. M. (2010). Dysfunction of stress responsive systems as a risk factor for functional somatic syndromes. *Journal Of Psychosomatic Research*, *68*(5), 461-468. doi:10.1016/j.jpsychores.2009.12.004

Thompson, B. L., Waltz, J., Croyle, K., & Pepper, A. C. (2007). Trait meta-mood and affect as predictors of somatic symptoms and life satisfaction. *Personality And Individual Differences*, *43*(7), 1786-1795. doi:10.1016/j.paid.2007.05.017

Thomsen, D. (2006). The association between rumination and negative affect: A review. *Cognition And Emotion*, *20*(8), 1216-1235. doi:10.1080/02699930500473533

Thomsen, D., Mehlsen, M., Olesen, F., Hokland, M., Viidik, A., Avlund, K., & Zachariae, R. (2004). Is there an association between rumination and self-reported physical health? A one-year follow-up in a young and an elderly sample. *Journal Of Behavioral Medicine*, *27*(3), 215-231. doi:10.1023/B:JOBM.0000028496.41492.34

Van Houdenhove, B., & Egle, U. T. (2004). Fibromyalgia: A Stress Disorder? Piecing the Biopsychosocial Puzzle Together. *Psychotherapy And Psychosomatics*, *73*(5), 267-275. doi:10.1159/000078843

Verkuil, B., Brosschot, J. F., Meerman, E. E., & Thayer, J. F. (2012). Effects of momentary assessed stressful events and worry episodes on somatic health complaints. *Psychology & Health*, *27*(2), 141-158. doi:10.1080/08870441003653470

Vijay, K. G., Avasthi, A., & Grover, S. (2014). A study of worry and functional somatic symptoms in generalized anxiety disorder. *Asian Journal Of Psychiatry*, *11*50-52. doi:10.1016/j.ajp.2014.05.011

Waller, E., & Scheidt, C. E. (2006). Somatoform disorders are disorders of affect regulation: A development perspective. *International Review of Psychiatry*, *18*(1), 13–24. doi:10.1080/0954026050046674

Watkins, E., Moulds, M., & Mackintosh, B. (2005). Comparisons between rumination and worry in a non-clinical population. *Behaviour Research And Therapy*, *43*(12), 1577-1585. doi:10.1016/j.brat.2004.11.008

Watson, D. (1988). Intraindividual and interindividual analyses of positive and negative affect: Their relation to health complaints, perceived stress, and daily activities. *Journal Of Personality And Social Psychology*, *54*(6), 1020-1030. doi:10.1037/0022-3514.54.6.1020

Watson, D., Gamez, W., & Simms, L. J. (2005). Basic dimensions of temperament and their relation to anxiety and depression: A symptom-based perspective. *Journal Of Research In Personality*, *39*(1), 46-66. doi:10.1016/j.jrp.2004.09.006

Whitehead, W. E. (1994). Assessing the effects of stress on physical symptoms. *Health Psychology*, *13*(2), 99-102. doi:10.1037/0278-6133.13.2.99

Whitehead, W. E., Crowell, M. D., Robinson, J. C., Heller, B. R., & Schuster, M. M. (1992). Effects of stressful life events on bowel symptoms: Subjects with irritable bowel syndrome compared with subjects without bowel dysfunction. *Gut, 33*, 825-83

Whitmer, A., & Gotlib, I. H. (2011). Brooding and reflection reconsidered: A factor analytic examination of rumination in currently depressed, formerly depressed, and never depressed individuals. *Cognitive Therapy And Research*, *35*(2), 99-107. doi:10.1007/s10608-011-9361-3

Wilkinson, P. O., Croudace, T. J., & Goodyer, I. M. (2013). Rumination, anxiety, depressive symptoms and subsequent depression in adolescents at risk for psychopathology: A longitudinal cohort study. *BMC Psychiatry*, *13*