



## Cognitive vulnerabilities to the development of PTSD: A review of four vulnerabilities and the proposal of an integrative vulnerability model

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### ARTICLE INFO

#### Article history:

Received 16 September 2007

Received in revised form 7 October 2008

Accepted 13 October 2008

#### Keywords:

Vulnerability

PTSD

Diathesis-stress

Attribution

Rumination

Anxiety sensitivity

Looming cognitive style

### ABSTRACT

While some individuals develop PTSD subsequent to traumatic experiences, many individuals resume prior functioning naturally. Diathesis-stress models suggest that stable individual differences present in individuals prior to trauma may serve as vulnerability factors to symptom development. The high levels of comorbidity and symptom similarity suggest that established vulnerability factors for anxiety and depression may also serve as vulnerability factors for PTSD. The examination of multiple vulnerability factors simultaneously may increase understanding of the etiology of PTSD and comorbid post-trauma symptomatology and account for a greater percentage of variance in PTSD symptoms. In addition, the vulnerability factors may be related to distinct sets of symptoms, with vulnerabilities predicting the PTSD symptoms most similar to their associated disorders. Research examining the relations between attributional style, rumination, anxiety sensitivity, and the looming cognitive style and the development of PTSD after trauma exposure is reviewed and suggestions for future research are provided.

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## 1. Introduction

Posttraumatic Stress Disorder (PTSD) was introduced in the Diagnostic and Statistical Manual for Mental Disorders-III (DSM-III; American Psychological Association [APA], 1980) primarily in response to the large number of Vietnam veterans presenting with symptom presentations that did not cleanly map onto any of the disorders included in the DSM-II (Bloom, 2000). As knowledge about the symptoms comprising PTSD increased, similar symptom presentations were found to be prevalent both in veterans and in survivors of additional traumatic events, such as rape, assault, or natural disasters. The original PTSD diagnostic criteria stated that an individual must have experienced a stressor that would evoke significant symptoms of distress in almost everyone, followed by the experience of three symptom clusters: re-experiencing, numbing, and miscellaneous symptoms (APA, 1980). The DSM-IV definition of PTSD retained the symptom cluster criteria from the previous version, requiring the presence of re-experiencing (e.g., flashbacks, nightmares), avoidance and numbing (e.g., restriction of affect, avoidance of trauma cues), and increased arousal (e.g., exaggerated startle response, difficulty sleeping) symptoms (APA, 1994). However, this version modified the definition of the required precipitating event, changing the focus of the reaction to the event from a normative one to a subjective one by stating that the individual must have experienced, witnessed, or been confronted by an event that involved actual or threatened danger and evoked feelings of helplessness, fear, or horror (APA, 1994).

As consideration of the definitions demonstrates, the original diagnosis and conceptualization of PTSD considered the traumatic event to be the primary causal factor in its development. In its early conceptualization, professionals generally attributed the responsibility for the development of PTSD to the event itself and avoided examining additional factors in an attempt to protect the victims (McNally, 2001). However, increased research on PTSD revealed that even within groups of individuals exposed to severe trauma many do not develop PTSD (e.g., Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). While most victims of traumatic events maintain or return to normal functioning after a short period of time, others develop PTSD, or other psychological disorders, in response to the event. The realization that the development of PTSD was not a normative response to the experience of traumatic events led to research examining risk factors, and later vulnerability factors, for the development of PTSD (McNally, 2001).

## 2. Risk factors

Early research on the influence of individual differences in PTSD development focused on examining and identifying risk factors for PTSD. Recently, two meta-analyses have been conducted to summarize the findings of the extant research (Brewin, Andrews, & Valentine, 2000; Ozer, Best, Lipsey, & Weiss, 2003). The reported findings from the two studies were largely consistent with each other. Both studies indicated that peri- and post-trauma factors were typically stronger predictors of PTSD development than more distal pre-trauma factors. Brewin et al. (2000) identified trauma severity, lack of social support, and subsequent life stress as the strongest predictors, with effect sizes in the small to moderate range. While Ozer et al. (2003) reported peritraumatic dissociation (not included in the Brewin et al.'s meta-analysis; 2000) as the strongest predictor in their study, perceived life threat and perceived support were also listed as relatively strong predictors of PTSD. Both studies suggest that past traumatic

experiences, a personal history of psychological difficulties, and a family history of psychopathology serve as weaker predictors of symptom development. In addition, Brewin et al. (2000) also included female gender, lower SES, less education, lower intelligence, and childhood adversity as weak predictors of PTSD. Finally, younger age at the time of the trauma and minority status were significant, albeit weak, predictors of the development of PTSD in one of the meta-analyses (Brewin et al., 2000). Both studies reported that the strength of the factors varied across samples and trauma type, suggesting that strong predictions about the relations between risk factors and PTSD may be premature. In particular, findings for the influence of gender, age at trauma, and race were inconsistent across samples and trauma type (Brewin et al., 2000). These findings could be the result of many different factors, including variability in measurement, real differences in relations between risk factors and PTSD across trauma types, and the effects of third variables not consistently accounted for in these studies. The prominence of retrospective data was consistently cited as a weakness of the literature and the need for prospective and longitudinal studies was highlighted. Despite the statistical significance and consistent support of the risk factors as predictors of PTSD development, the combined variance accounted for by these risk factors is low (Ozer et al., 2003).

### 2.1. Diathesis-stress model

Diathesis-stress models have recently been applied to psychological disorders in an attempt to identify factors that are causally related to symptom development and would therefore serve as stronger predictors than risk factors alone (Ingram & Price, 2001). The diathesis-stress model was originally used by members of the medical field to describe an underlying pathogenic mechanism that remains latent and harmless until activated by sufficient stress (Ingram, Miranda, & Segal, 1998). Once activated, the pathogenic mechanism influences systemic functioning and may lead to the development of the disorder. The concept and nature of a diathesis-stress model has since been expanded into the psychological realm (Meehl, 1962; Zubin & Spring, 1977; Ingram & Price, 2001). Psychological diatheses are conceptualized as relatively stable individual differences (e.g., personality traits or cognitive styles) that increase one's vulnerability to stress and to the development of psychological disorders. Psychological vulnerabilities are described as stable (without intervention), endogenous (i.e., resides within the person), latent (i.e., not easily observable), and likely to interact with stress (Ingram & Price, 2001).

Within a diathesis-stress model, it is often expected that the list of factors related to the development of a syndrome or disease is open, providing for both multiple known factors and allowing for additional unknown factors (Meehl, 1977). The factors in this list vary in both the strength of their relation with the outcome and their role in development. A factor can be considered specifically related to a disorder when it serves in one of several ways. The strongest type of specificity exists when the factor is both necessary and sufficient for the development of the disorder. Other, although weaker, types of specificity include when a factor is necessary but not sufficient without suitable combinations of other factors and when a change in a factor makes a bigger difference than a change in any other factor under all circumstances (Meehl, 1977). In addition, it is proposed that there are some factors that regardless of the values of the other factors ensure that the disease will be produced. While nonspecific factors can exert a strong influence over the development of a disease, specific factors are assumed to be more strongly related than the other factors.

In the examination of factors related to the development of individual disorders, theoretical differences between vulnerability factors and risk factors in relation to symptom development have been proposed (Ingram & Price, 2001). Namely, risk factors are proposed to be significantly correlated with the symptoms without necessarily being causally related to the symptoms. For example, young age at the time of trauma has been highlighted as a risk factor for PTSD symptom development. It is unlikely that the age of the individual, in and of itself, is strongly related to the development of PTSD. Rather, being young at the time of the trauma may increase the likelihood that the individual will possess or develop other factors strongly related to symptom development, such as the likelihood of developing negative beliefs about the world. Vulnerability factors, on the contrary, are proposed to be more strongly causally related to the development of the symptoms. Specifically, psychological vulnerabilities are assumed to interact with the stressor to lead to the development of psychological symptoms (Zubin & Spring, 1977). In the context of specific and non-specific factors, vulnerability factors are proposed to be more similar to specific factors, while risk factors are considered non-specific influences. Once the vulnerability is activated it is proposed that the individual's experience of distress is prolonged by subsequent biased information processing, impaired performance, and use of poor coping and emotion regulation strategies. Moreover, these vulnerabilities may not only be causally related to symptom development but, once activated, may also serve as maintenance factors for psychological symptoms. The causal nature of psychological vulnerabilities implies that an increased understanding of the vulnerabilities can aid in both the prevention and treatment of the corresponding disorders (Zvolensky, Schmidt, Bernstein, & Keough, 2006).

## 2.2. Diathesis-stress models of PTSD

Diathesis-stress models have recently been applied to theories of the development of PTSD (Bowman & Yehuda, 2004; McKeever & Huff, 2003). The diathesis-stress model of PTSD asserts that the traumatic event serves as the primary stressor, and thus activator of PTSD symptoms. However, the model proposes that pre-trauma individual differences contribute to the potential development of PTSD and that individuals who possess higher levels of relevant psychological vulnerabilities before the traumatic experience are at a higher risk for the development of PTSD than those that possess low levels of these diatheses. In diathesis-stress models of PTSD, ecological factors (i.e., relating to the self or the surrounding environment), biological diatheses, and the residual stress of the trauma play a role in the development of PTSD (Bowman & Yehuda, 2004; McKeever & Huff, 2003).

A full diathesis-stress model of PTSD must account for pre-, peri-, and post-trauma factors. For example, risk, vulnerability, and biological factors can all be classified as pre-trauma factors. In addition to risk and vulnerability factors, biological factors, peritraumatic reactions to the trauma, and posttrauma circumstances are likely to influence PTSD symptomatology. While vulnerability factors are just one component of this model, their proposed causal relation to symptom development and tendency to serve as maintenance factors once the disorder has developed suggest that they might play a prominent role in PTSD development. Once understood, vulnerability factors are amenable to change and can be targeted in prevention and intervention.

Several theoretical models of psychopathology have delineated vulnerability as either disorder-general or disorder-specific (Brown, Chorpita, & Barlow, 1998; Clark & Watson, 1991; Barlow, Allen, & Choate, 2004). Barlow et al.'s (2004) unifying theory of emotional disorders describes three levels of vulnerability (i.e., biological/genetic, general, specific), each increasing in its level of disorder specificity. For example, negative affect is considered a general

cognitive vulnerability while low positive affect may be a specific vulnerability for depression. One particular class of psychological vulnerability that has gained considerable theoretical and empirical attention over the past several decades is cognitive vulnerability (e.g., Abramson, Metalsky, & Alloy, 1989; Beck, 1967; Ingram & Price, 2001; Riskind, Williams, Gessner, Chrosniak, & Cortina, 2000). Cognitive models of psychopathology propose that mental processes such as interpretation, attention, and memory mediate the relation between environmental events and emotional responses (Riskind & Alloy, 2006). Cognitive vulnerabilities consist of characteristic possession of biased beliefs or cognitive patterns. Disorder-specific cognitive vulnerabilities are conceptualized as information processing biases regarding threat, harm or safety specific to a particular anxiety disorder (e.g., PTSD-related cognitive biases such as perception of safety of the environment and trustworthiness of others) resulting in related dysfunctional thoughts and behaviors (e.g., perceived ongoing threat and avoidance of trauma-related stimuli; Beck & Clark, 1997). Consistent with the diathesis-stress model described above, cognitive vulnerabilities are assumed to remain latent until activated by sufficient stress or negative life events. Once activated, cognitive vulnerabilities influence individuals' characteristic ways of attending to, interpreting, organizing, mentally representing, and remembering negative life events or anticipated future dangers.

The aim of the present paper is to review the roles of four specific cognitive vulnerabilities in the development and maintenance of PTSD and to provide suggestions for the examination of cognitive vulnerabilities in an integrative manner. The paper will briefly review cognitive theories of PTSD, summarize the extant literature on four cognitive vulnerabilities, and describe a model of potential interactions between the vulnerabilities. Cognitive vulnerabilities were chosen due to the emphasis of cognitive dysfunctions in current theories of PTSD (e.g., Ehlers & Clark, 2000) and the strong empirical support for cognitive-behavioral treatments of PTSD such as Prolonged Exposure and Cognitive Processing Therapy (Foa & Rothbaum, 1999; Resick & Schnicke, 1996). Proposed cognitive vulnerabilities specific to PTSD will be briefly described, but the paper focuses on the role of previously supported vulnerabilities to emotional disorders in relation to PTSD. Specifically, this paper constitutes an empirical review that focuses on the examination of attributional style, rumination, anxiety sensitivity, and looming maladaptive style in relation to PTSD. Attributional style is conceptualized as a characteristic set of interpretations about negative events that increases vulnerability for hopelessness and depressive symptoms (Abramson et al., 1989). Rumination refers to thinking repetitively and passively about psychological symptoms and the causes and consequences of their symptoms (Nolen-Hoeksema, 1991). Anxiety sensitivity is defined as the fear of anxiety and anxiety-related body sensations (Reiss, 1991). Lastly, looming maladaptive style is posited as an attention and interpretation bias causing maladaptive mental representation of intensifying threat (Riskind, 1997). The four vulnerabilities reviewed in the present paper were chosen due to their strong support as vulnerabilities for other emotional disorders and subsequent theoretical and empirical support for their relation with PTSD.

In addition to the description of each vulnerability, an overview of common measurement and a summary of the literature examining the vulnerability in relation to PTSD will be offered. The current review did not restrict by trauma type, sex, or age. In addition, although the present paper highlights the relation between cognitive vulnerabilities and PTSD, studies examining the relation between these cognitive vulnerabilities and Acute Stress Disorder (ASD) are also included. A high degree of symptom overlap exists between the two disorders and research suggests that the majority of individuals that are diagnosed with ASD will later meet criteria for PTSD (e.g., Elklit & Brink, 2004; Kleim, Ehlers, & Glucksman, 2007). Therefore, the two disorders are examined concurrently for the purposes of the current review. Finally, the paper proposes an integrated vulnerability

model of PTSD and suggestions for future directions of PTSD vulnerability research.

### 3. Cognitive theories of PTSD

Early theorists (Epstein, 1991; Janoff-Bulman, 1992; McCann & Pearlman, 1990) proposed that the experience of a traumatic event contradicted the individual's preexisting schemas and that the disruption of the individual's previous beliefs about the self and the world resulted in the development of PTSD. Although the conceptualizations of the set of beliefs differed, these theorists suggested that the experience of a traumatic event leads to the challenging of one or more previously established beliefs. As a result, the entire self-system may be pressured to disorganize, which results in the symptoms of PTSD. McCann and Pearlman (1990) suggested that there were seven areas that could be disrupted after the experience of a traumatic event: dependency, safety, power, independence, esteem, intimacy, and/or frame of reference. Epstein (1991) posited that there were four beliefs central to a personal theory of reality: the beliefs that the world is benign, that the world is meaningful, that the self is worthy, and that people are trustworthy. Janoff-Bulman (1992) similarly argued that when a trauma is experienced, three fundamental assumptions are shattered; the belief in a benevolent world, assumptions that the world is meaningful, and assumptions of the self as worthy.

Theory suggests that after a person experiences a traumatic event he or she is unable to retain previously held beliefs, and the individual is forced to either accept that the world is unjust and unsafe or that he or she did something to evoke the experience (Janoff-Bulman, 1992). Often the victim attempts to evade this dilemma by avoiding stimuli that initiate memories of the event. The attempted avoidance increases the individual's overall levels of anxiety and arousal symptoms. According to this theory, the people with the most positive preexisting assumptions are expected to suffer the highest level of symptoms, due to the presumed largest discrepancy between the event and previously held beliefs. The symptoms are believed to resolve when the individual is able to again maintain belief in a stable, comfortable world (Janoff-Bulman, 1992).

Emotional processing theory was designed to integrate learning, cognitive, and personality theories of PTSD and attempts to explain why some victims develop PTSD and others do not (Foa, Steketee, & Rothbaum, 1989). The theory is based on the idea that many negative events in life are emotionally re-experienced, but that generally the frequency and intensity decreases (i.e., habituation). The victims that do not experience this decrease in frequency and intensity are believed to be the ones that develop PTSD. Habituation is disrupted when the individual does not allow himself or herself to remember and experience the emotions associated with the event. According to this theory, PTSD is the result of the pathological nature of the memory of the traumatic event that contains erroneous associations and evaluations that lead to avoidance. People with pathological fear are also believed to be reluctant to be in fear provoking situations because they fear that anxiety will persist until they escape, that stimuli present an unrealistic threat of causing harm, and that consequences are particularly aversive, such as going crazy or losing control (Foa & Rothbaum, 1999). The pathological fear of memories of the traumatic event leads to avoidance, resulting in an increase of re-experiencing and arousal symptoms, which reinforce and increase fear (Foa & Rothbaum, 1999). The theory suggests that the victims also develop views of themselves as incapable of self-protection (from future traumatic events) and normal functioning (because of an inability to control symptoms).

Foa and Riggs (1993) suggest that three factors are involved in determining who develops chronic PTSD: the victim's schemas about the world and self prior to the trauma, the victim's memory records of the trauma, and the victim's memory records of posttrauma experiences (Foa & Rothbaum, 1999). Unlike Janoff-Bulman's (1992)

suggestion that people with extremely positive views of the self and world are more vulnerable, Foa and Riggs (1993) suggest instead that people with extreme, rigid views either positive or negative are more vulnerable. This theory is more congruent with research indicating that victims of multiple violent acts are at a high risk for developing PTSD (Rothbaum, Foa, Riggs, Murdock, & Walsh, 1992). An individual that perceives himself or herself as highly competent and the world as extremely safe would find a traumatic event completely contradictory to his or her expectations and may cause him or her to doubt all of his or her previous views. On the other hand, a person who believes he or she is entirely incompetent and the world is completely dangerous would have his or her beliefs reinforced by a traumatic event, and thus increase the fear. The second factor believed to be related to an individual's vulnerability to the development of PTSD is the memory of the traumatic event, which is largely influenced by the severity of the event. Traumatic experiences with higher levels of violence are believed to have a larger number of stimulus-danger associations, which increase the belief of the world as entirely dangerous (Foa & Rothbaum, 1999). Finally, negative evaluations of others and the belief that one's reactions to the event are further signs of incompetence increase an individual's risk (Foa & Rothbaum, 1999). In sum, emotional processing theory posits that a person's pretrauma schemas, memory of the event, and memory of experiences prior to the event can interact and impede the emotional processing of the trauma, which leads to the development of chronic PTSD.

Ehlers and Clark (2000) added that PTSD becomes persistent when individuals process the trauma in a way that leads to the sense of a serious, current threat. An excessively negative evaluation of the event and a disturbance in the memory of the event combine to produce this feeling of constant threat (Ehlers & Clark, 2000). According to these views, being able to accept the trauma as an unfortunate random event, instead of a reflection of the norm, would alleviate the person from the feeling of ever-present fear and belief that it is essential to protect oneself from developing or maintaining PTSD symptoms. While there is not a universally accepted theory of PTSD, it is typically believed that the presence of various schemas about the self, world, or others function as cognitive vulnerabilities to PTSD and play a role in the development and maintenance of symptoms.

#### 3.1. Measurement of PTSD related cognitions

Foa, Ehlers, Clark, Tolin, and Orsillo (1999) attempted to identify the cognitive beliefs most related to the development of PTSD across trauma type. Consideration of theoretical conceptualizations and clinical experience resulted in 110 items under consideration, with 33 items selected for use in the final scale, entitled the Posttraumatic Cognitions Inventory (PTCI). A principal-components analysis suggested a three factor solution: a) negative cognitions about the self, b) negative cognitions about the world, and c) self-blame for the trauma. This three factor solution was also supported by Beck, Coffey, Palyo, Gundmundsdottir, Miller, and Colder (2004) within a motor vehicle accident population. These three cognitive belief domains have been found to be predictive of PTSD severity, depression, and anxiety in traumatized individuals (Foa et al., 1999). Foa et al. (1999) provide evidence for convergent and discriminant validity as well as internal consistency and test-retest reliability. Additional measures used to assess cognitive changes related to PTSD in sexual assault victims include the Personal Beliefs and Reactions Scale and the World Assumptions Scale (as used in Owens & Chard, 2001; Owens, Pike, & Chard, 2001; Wenniger & Ehlers, 1998).

#### 3.2. Structure of PTSD

The diagnostic criteria and symptom presentation of PTSD overlap with other anxiety and mood disorders, including Major Depressive Disorder (MDD), Dysthymic Disorder, Generalized Anxiety Disorder

(GAD), and Panic Disorder (PD; Friedman & Yehuda, 1995). The numbing symptoms of PTSD, such as decreased interest in previously pleasurable activities, are similar to symptoms of MDD and dysthymic disorder. Sleep and concentration difficulties serve as criteria for MDD, dysthymic disorder, GAD, and PTSD. While the specific arousal symptoms included in the PTSD diagnostic criteria do not overlap with the PD criteria, the nature of the symptom cluster is consistent with PD symptoms. In addition, the PTSD re-experiencing symptom cluster includes the experience of physiological reactivity on exposure to internal or external reminders of the event. This physiological reactivity can manifest as or develop into panic attacks. Two studies that examined peri-traumatic trauma reactions found that over half of the civilian trauma survivor participants reported experiencing panic attacks during the trauma (Bryant & Panasetis, 2001; Nixon & Bryant, 2003). A number of symptoms associated with PTSD, though not included in the diagnostic criteria, such as guilt and suicidal ideation are also consistent with symptoms of other anxiety and mood disorders (Friedman & Yehuda, 1995). Given the similarity of the diagnostic criteria, it is not surprising that individuals suffering from PTSD often present with comorbid anxiety and mood disorders (Brady, Killeen, Brewerton, & Lucerini, 2000; Brown, Campbell, Lehman, Grisham, & Mancill, 2001; Friedman & Yehuda, 1995; Keane & Kaloupek, 1997).

The inclusion of depression and other anxiety disorders in the trauma response spectrum and the similarity of symptoms between PTSD and other emotional disorders suggest that examining known diatheses for these disorders could be beneficial when studying potential diatheses for PTSD. Historically, much more research has focused on identifying vulnerability factors to depression, general anxiety, and PD than to PTSD. The examination of these factors in relation to PTSD has indicated that vulnerabilities originally linked with other emotional disorders may be related to PTSD symptomatology as well. The four vulnerabilities reviewed in the present paper were chosen due to their strong support as vulnerabilities for other emotional disorders and the subsequent theoretical and empirical support for their relation with PTSD.

#### 4. Negative attributional style

The hopelessness theory of depression proposes that an individual's interpretations of perceived negative events determine whether or not the individual will develop hopelessness depression – a cognitively mediated subtype of depression characterized by motivational deficits, interpersonal dependency, anergia, insomnia, anhedonia, brooding, suicidal ideation, and psychomotor retardation (Abramson et al., 1989; Alloy et al., 2000). Specifically, the hopelessness theory highlights three types of inferences related to the development of hopelessness: (a) inferences about why the event occurred; (b) inferences about the consequences of the event; and (c) inferences about the self in response to the occurrence of the event (Abramson et al., 1989). The tendency to consistently attribute negative events to internal, stable, and global causes is described as a negative attributional style (NAS) and is proposed to serve as a cognitive vulnerability to depression. The hopelessness theory proposes that individuals high in NAS are vulnerable to feelings of hopelessness and depressive symptoms, due to a tendency to assume responsibility for negative events and believe that negative events will always happen in all areas of their lives. In the absence of negative life events the individuals should display a normal level of functioning, regardless of their level of the NAS. Considerable research suggests that NAS increases an individual's vulnerability to both depressive symptoms and depressive episodes (Abramson et al., 1999; Alloy et al., 2000; Gladstone & Kaslow, 1995; Joiner & Wagner, 1995).

##### 4.1. Measurement of negative attributional style

The most commonly used measure of negative attributional style in PTSD research is the Attributional Style Questionnaire (ASQ;

Peterson et al., 1982). The ASQ is designed to assess individuals' causal explanations for hypothetical positive and negative events. The questionnaire presents the individual with six positive events (e.g., being complimented on your appearance) and six negative events (e.g., a date going badly). Participants are then asked to provide a cause for the event, and then rate the cause on the three attribution dimensions: internality, stability, and globality. The items are summed separately for positive and negative situations; however, studies examining the relation between attributional style and depression often focus on the attributions for negative events (Peterson & Seligman, 1984). The ASQ has demonstrated moderate internal consistency (Peterson et al., 1982; Sweeney, Anderson, & Bailey, 1986; Tennen & Herzberger, 1987), test–retest reliability of the items (Golin, Sweeney, & Schaeffer, 1981), and test–retest reliability of the attribution dimensions (Peterson et al., 1982). A recent confirmatory factor analysis, using a multitrait–multimethod model, of the negative events of the ASQ found support for the proposed three components of negative attributional style (Hewitt, Foxcroft, & MacDonald, 2004). However, results regarding the psychometric properties of the ASQ are equivocal. Other findings have reported low reliability and poor support of a three factor model of the ASQ (e.g., Cutrona, Russell, & Jones, 1985).

Since the development of the ASQ, several alternative variations of the measure have been created for use with additional populations. Measures assessing NAS have been developed for use in both children (e.g., Children's Attributional Style Questionnaire Revised, Thompson, Kaslow, Weiss, & Nolen-Hoeksema, 1998) and adolescents (Adolescent Cognitive Style Questionnaire, Hankin & Abramson, 2002). In addition, an expanded form of the ASQ (Expanded Attributional Style Questionnaire, EASQ; Peterson & Villanova, 1988) was created which included 24 hypothetical negative events instead of the original six. Studies examining the psychometric properties of the modified measures have typically yielded positive results. Thompson et al. (1998) reported that the overall composite score, positive composite score, and negative composite scores on the CASQ-R were fairly stable over a six month period ( $r$ 's ranging from .38 to .53). The ACSQ also demonstrated good test re-test reliability over periods of both two weeks ( $r = .73$ ) and two years ( $r = .51$ ; Hankin & Abramson, 2002). Consistent with the original measure, confirmatory factor analysis results have provided support for three factor models in the children and adolescent versions as well (Hankin & Abramson, 2002; Joiner & Metalsky, 1999).

Providing some support for the convergent validity, measures of NAS have been demonstrated to be positively correlated with depressive symptoms (Conley, Haines, Hilt, & Metalsky, 2001; Hankin & Abramson, 2002; Reijntjes, Dekovic, Vermande, & Telch, 2008; Thompson et al., 1998; Rueger & Malecki, 2007), neuroticism (Corr & Gray, 1996), negative affect (Sanjuán, Pérez, Rueda, & Ruiz, 2008), and learned helplessness (Rueger & Malecki, 2007). As evidence of discriminant validity, NAS has been shown to be negatively correlated with measures of optimism (Rueger & Malecki, 2007), positive affect (Sanjuán et al., 2008) and extraversion (Corr & Gray, 1996). A recent study using the CASQ found that endorsement of NAS was associated with a more negative self performance evaluation in an experimental task, which suggests that self-reported NAS may be representative of real-world experiences (Reijntjes et al., 2008). Finally, consistent with a diathesis-stress model, NAS has significantly interacted with negative life events to predict level of depressive symptoms (Hankin & Abramson, 2002; Metalsky & Joiner, 1992).

##### 4.2. Negative attributional style and PTSD

The experience of sexual assault serves as a negative life event in the cognitive vulnerability-stress model. According to the hopelessness theory, individuals who make negative attributions after the experience of sexual assault will be at an increased vulnerability for

the development of depressive symptoms. Negative attributional style has been linked to various psychological symptoms, including PTSD, depression, and general anxiety, in victims of a variety of traumatic events (Greening, Stoppelbein, & Docter, 2002; Gray, Pumphrey, & Lombardo, 2003; Joseph, Brewin, Yule, & Williams, 1991; Kuyken & Brewin, 1999; Solomon, Mikulincer, & Waysman, 1991; Williams, Evans, Needham, & Wilson, 2002). The relation between abuse, attributional style, and symptom development has been examined using many different populations and methodologies. Researchers differ on whether they have used a composite score for attributional style, general individual attribution dimensions, or attributions for specific events when testing the association with the development of a variety of symptoms (e.g., Falsetti & Resick, 1995; Kuyken & Brewin, 1999; Gray & Lombardo, 2004).

The negative attributional style appears to be consistently associated with the development of PTSD in victims of interpersonal trauma. Positive relations have been reported between composite scores for attributions about negative events and the severity of PTSD in victims of interpersonal trauma and non-interpersonal trauma (Gray et al., 2003; Joseph et al., 1991; Kuyken & Brewin, 1999; Palker-Corell & Marcus, 2004; Runyon & Kenry, 2002; Williams et al., 2002). A longitudinal study of the relation between attributions and PTSD reported that the attribution of negative events to external, stable, and uncontrollable causes was significantly related to an increase in PTSD symptoms (Mikulincer & Solomon, 1988). When individually examining the attribution dimensions, some findings have indicated that the globality and stability dimensions are associated with PTSD symptoms while the causality (i.e., internal vs. external) dimension is unrelated to symptom development (Gray & Lombardo, 2004; Wenniger & Ehlers, 1998). However, other studies examining only causal attribution of traumatic events reported that individuals that attribute the cause of negative events to an external source reported higher levels of PTSD symptoms than those with internal attributions (Hickling, Blanchard, Buckley, & Taylor, 1999; Williams et al., 2002). Support has also been found for a relation between event-specific attributions and severity of PTSD symptoms (Brown & Kolko, 1999; Falsetti & Resick, 1995; Feiring, Taska, & Chen, 2002; Feiring, Taska, & Lewis, 2002). While the relation between PTSD and attributional style is well documented, some studies have failed to find significant relations between attributional style and development of PTSD (Dixon, Howie, & Starling, 2005; Feiring, Taska, & Lewis, 1998; Greening et al., 2002). These inconsistent findings may be explained by differences in study methodologies, including the predictor variables assessed, nature of the trauma, direct vs. indirect exposure to the traumatic event, severity of trauma and consequences, and time between assessment and trauma exposure.

## 5. Rumination

Rumination is typically defined as the tendency to think repetitively and passively about negative emotions, precipitators of negative events, symptoms of distress, and worry about the meaning of distress (Nolen-Hoeksema, 1991). Rumination was initially linked with depressive symptoms and disorders (Nolen-Hoeksema, 2000; Davis & Nolen-Hoeksema, 2000). Longitudinal studies have found that individuals that engage in more ruminative responses when they are sad or depressed report higher levels of depressive symptoms over time, which supports ruminative style as a maintenance factor (Nolen-Hoeksema & Davis, 1999; Nolen-Hoeksema, Larson, & Grayson, 1999). Ruminative style has also received some support as a vulnerability factor for depression (Just & Alloy, 1997; Nolen-Hoeksema, 2000).

### 5.1. Measurement of rumination

Several measures are commonly used to assess rumination in both PTSD and depressive disorders. The Ruminative Response Scale (RRS) of

the Response Style Questionnaire (RSQ; Nolen-Hoeksema & Morrow, 1991) is the most commonly used self-report measure to examine ruminative processes in major depressive disorder. Specifically, the RRS consists of 22 items on a 4-point Likert scale ranging from 1 (almost never) to 4 (almost always). Questions assess levels of self reflection, symptoms, and causes and consequences of mood. Initial psychometric evaluations demonstrated good 5-month test-retest reliability ( $r = .80$ ; Nolen-Hoeksema, Parker, & Larson, 1994) and good internal consistency ( $\alpha = .89$ , Nolen-Hoeksema & Morrow, 1991;  $\alpha = .89$ , Kasch, Klein, & Lara, 2001). However, one study examining the psychometric properties of the RRS reported modest stability over a six week period ( $r = .48$ ), but low stability over a six month period ( $r = .36$ ; Kasch et al., 2001). Furthermore, findings indicated that the low six month stability was influenced by variation in levels of depressive symptoms, consistent with a state-like nature rather than a trait-like nature (Kasch et al., 2001). Rumination has been shown to be associated with similar constructs, including depressive symptoms (Harris, Pepper, & Maack, 2008; Miranda & Nolen-Hoeksema, 2007; Moulds, Kandris, Starr, & Wong, 2007; Muris, Roelofs, Rassin, Franken, & Mayer, 2005; Nolen-Hoeksema, Stice, Wade, & Bohon, 2007; Roelofs, Muris, Huijbers, Peeters, & Arntz, 2006; Williams & Moulds, 2007), negative affect (Marcus, Hughes, & Arnau, 2008), neuroticism (Muris et al., 2005; Roelofs et al., 2006), trait anxiety (Muris et al., 2005; Roelofs et al., 2006), worry (Muris et al., 2005), avoidance (Moulds et al., 2007) and maladaptive perfectionism (Harris et al., 2008). One study reported that rumination was not significantly related to problem-solving coping, positive temperament, or disinhibition (Kasch et al., 2001). Results examining the predictive validity of the RRS have been equivocal. Higher levels of rumination have significantly predicted the onset of clinically significant major depression over a four year period (Nolen-Hoeksema et al., 2007) and suicidal ideation after one year, while controlling for the influence of depressive symptoms (Miranda & Nolen-Hoeksema, 2007). However, one study reported that baseline rumination was not significantly associated with level of depressive symptoms after six months (Kasch et al., 2001). Inconsistent with theoretical and empirical associations between rumination and depression, one prospective study reported that rumination significantly predicted anxiety, but not depression (Calmes & Roberts, 2007). Concerns have been raised regarding the high content overlap of RRS items with measures of depressive symptoms (e.g., BDI-II; Conway, Csank, Holm, & Blake, 2000; Treynor, Gonzalez, & Nolen-Hoeksema, 2003). However, Treynor et al. (2003) found support for a two-factor model of rumination (i.e., brooding, reflection) independent of depressive covariance.

Less consensus exists in the PTSD literature regarding the main assessment method of rumination. The Response to Intrusion Questionnaire (RIQ; Clohessy & Ehlers, 1999; Murray, Ehlers, & Mayou, 2002) is a self-report measure commonly used to examine cognitive variables that are hypothesized to contribute to the maintenance of intrusive memories. A rumination subscale of the RIQ is typically used to assess rumination ("I dwell on them", "I worry that something like that could happen to me or my family", and "I think about what I could have done differently"). The participants endorse how frequently they experience the items on a Likert scale ranging from 0 (not at all) to 3 (always). Variations of this measure ranging from 3–8 questions have been used (e.g., Clohessy & Ehlers, 1999; Ehling, Ehlers, & Glucksman, 2006; Kleim & Ehlers, 2008; Murray et al., 2002; Schönfeld & Ehlers, 2006). Preliminary investigations of the RIQ have yielded inconsistent internal reliability of the rumination items ( $\alpha = .31$ , Clohessy & Ehlers, 1999;  $\alpha = .77$ , Murray et al., 2002). Therefore, the authors recommend reporting the scores of the individual items in addition to the overall mean score if results yield questionable reliability scores (e.g.,  $\leq .70$ ). However, if results yield adequate reliability estimates then use of the mean score is indicated. Findings indicate that the RIQ is a strong predictor of PTSD severity, providing support for predictive validity (e.g., Murray et al., 2002). Other studies examining rumination and PTSD have used theory driven questions or interviews created or adapted by the authors. For

example, Michael, Halligan, Clark, and Ehlers (2007) created a "Rumination Interview" described as a twenty-five minute, structured interview that contains eight content areas and demonstrated good internal reliability ( $\alpha$ 's = .75–.89). Overall, there is little agreement on a standard assessment of rumination for PTSD. Compared to the measurement of rumination in depression, the assessment of rumination in PTSD largely focuses on reactions to a specific trauma, rather than a generalized ruminative style.

## 5.2. Rumination and PTSD

Rumination has been posited as a cognitive factor that is likely to lead to the perception of ongoing threat and therefore to the maintenance of PTSD symptoms (Ehlers & Clark, 2000). Specifically, trauma-related rumination is likely to involve thinking about the causes and consequences of the trauma and/or the negative consequences of the trauma, without focusing on the events of the trauma itself (Ehlers & Steil, 1995). In fact, rumination has been proposed to be used as a cognitive avoidance strategy, similar to worry, in which trauma-exposed individuals may ruminate in an attempt to avoid memories of the trauma itself (Michael et al., 2007). Rumination, therefore, is believed to prevent the processing of the trauma memories and thereby result in increased re-experiencing symptoms and negative mood (Ehlers, Mayou, & Bryant, 1998).

Associations between rumination and PTSD in cross-sectional examinations have been reported in a variety of samples of trauma populations, including individuals exposed to assault, motor vehicle accidents, and those serving as emergency workers (Clohessy & Ehlers, 1999; Ehlers et al., 1998; Michael et al., 2007; Steil & Ehlers, 2000). Furthermore, longitudinal studies have frequently reported that the endorsement of rumination shortly after a traumatic event serves as a significant predictor of later PTSD symptoms (Ehlers, Mayou, & Bryant, 2003; Kleim et al., 2007; Mayou, Ehlers, & Bryant, 2002). In addition to being associated with PTSD severity, one study found the rumination mediated between PTSD symptoms and anger in a sample of crime victims (Orth, Cahill, Foa, & Maercker, 2008). However, some inconsistent findings have been reported. While several studies reported that rumination remained a significant predictor when controlling for original symptom levels (Michael et al., 2007; Kleim et al., 2007), others have failed to find a significant relation when initial symptom levels are included (Ehlers et al., 1998; Mayou et al., 2002). Although the majority of available studies have used adult samples, Ehlers et al. (2003) found support for a relation between rumination and PTSD in children and adolescents.

## 6. Anxiety sensitivity

Anxiety sensitivity (AS) has been defined as the fear of anxiety and anxiety-related sensations based on the belief that the symptoms have harmful consequences (Reiss, 1991). AS is described as a stable, trait-like characteristic that functions as both a vulnerability and maintenance factor for anxious symptoms. Although AS was traditionally measured as a continuous variable on one dimension, later investigations suggested that there may be three factors within the AS construct (Taylor & Cox, 1998a,b; Taylor et al., 2007; Zinbarg, Mohlman, & Hong, 1999). While the names have varied, the factors typically assess physical, cognitive, and social concerns. When individuals high in AS notice themselves experiencing anxious symptoms they become focused on these symptoms and experience fear, which further increases symptoms. Although AS is most strongly associated with PD, it has also demonstrated significant relations with a variety of other anxiety disorders, including PTSD. For example, a study examining the role of AS across the anxiety disorders found that participants with PD reported significantly higher levels of AS than those with all of the other anxiety disorders except for PTSD (Taylor, Koch, & McNally, 1992). Furthermore, the relation between AS and PTSD remained even when using trait anxiety as a covariate (Taylor et al., 1992).

## 6.1. Measurement of anxiety sensitivity

Anxiety sensitivity has traditionally been measured using the 16-item Anxiety Sensitivity Index (ASI; Reiss, Peterson, Gursky, & McNally, 1986). Each item describes a concern about the experience of anxious symptoms (e.g., "It scares me when I feel faint") and is rated on a 5-point Likert scale (0 = very little to 4 = very much). The ASI has typically yielded internal consistency reliability coefficients between .8 and .9 in subsequent studies (Peterson & Reiss, 1993; Peterson & Plehn, 1999) and has produced adequate test-retest correlations in several studies (Maller & Reiss, 1992; Peterson & Reiss, 1993; Reiss et al., 1986; Schmidt, Lerew, & Jackson, 1997). A longitudinal investigation over a four year period, with assessments once a year, revealed that scores at each time point were significantly correlated with each other (Weems, Hayward, Killen, & Taylor, 2002). Further examination indicated that the majority of participants evidenced predominantly stable levels of anxiety sensitivity, although groups with escalating AS scores and fluctuating AS scores were also found.

Traditional interpretations of AS produce a sum score representative of one factor. However, subsequent analyses suggested that AS is multidimensional (see Blais et al., 2001 and Zinbarg et al., 1999 for a review). Proposed alternative structures of the ASI have included two, three, and four factors of AS (Blais et al., 2001; Zinbarg et al., 1999). In contrast to the traditional continuous measurement of AS, support has been found for an AS taxon (using 8 items from the ASI; Bernstein, Zvolensky, Feldner, Lewis, & Leen-Feldner, 2005; Bernstein et al., 2005). A taxonic conceptualization of AS suggests that AS is categorical in nature, and that individuals with high levels of AS are qualitatively different than those with low levels. Although initial studies supported the taxonic nature of AS, discrepant findings have also been reported (Broman-Fulks et al., 2008). Consistent with the multiple interpretations of the AS factor structure, the ASI has been revised several times since its inception including the Anxiety Sensitivity Index-Revised, the Anxiety Sensitivity Index-3, and the Anxiety Sensitivity Profile (Taylor & Cox, 1998a; Taylor et al., 2007; Taylor & Cox, 1998b). Cross-cultural examination has suggested that anxiety sensitivity is present and largely similar in structure, consistent with a two factor model (using the ASI-R), across six countries (Zvolensky et al., 2003).

Anxiety sensitivity has displayed significant correlations with anxious and depressive symptoms (Grant, Beck, & Davila, 2007; Smári, Erlendsdóttir, Björgvinsdóttir, & Ágústssdóttir, 2003), negative affect (Kilic, Kilic, & Yilmaz, 2008; Leen-Feldner, Feldner, Reardon, Babson, & Dixon, 2008; McKee, Zvolensky, Solomon, Bernstein, & Leen-Feldner, 2007), neuroticism (Zvolensky et al., 2003), and trait anxiety (Plehn & Peterson, 2002; Schmidt, Zvolensky, & Maner, 2006; Smári et al., 2003). Anxiety sensitivity has been demonstrated to be negatively correlated with mindfulness skills (McKee et al., 2007) and uncorrelated with extraversion and social desirability (Zvolensky et al., 2003). Supportive of criterion validity, patients with agoraphobia endorsed higher levels of AS than individuals with other anxiety disorders and controls, while individuals with other anxiety disorders yielded higher levels than controls (Reiss et al., 1986). The authors reported that ASI scores predicted level of fearfulness above and beyond anxiety and anxiety frequency, and cited this as support of discriminant validity (Reiss et al., 1986). A more recent study reported that anxiety sensitivity served as a significant predictor of spontaneous panic attacks and incidence of anxiety diagnoses over a two year period, while neither trait anxiety nor the interaction between trait anxiety and anxiety sensitivity served as significant predictors (Schmidt et al., 2006). When the relation between AS and panic was examined over an eleven year period, AS served as a significant predictor of panic symptoms and panic attacks, while controlling for trait anxiety, but only trait anxiety served as a significant predictor of panic disorder (Plehn & Peterson, 2002).

## 6.2. Anxiety sensitivity and PTSD

Anxiety sensitivity has since been proposed as a vulnerability and maintenance factor for PTSD (Fedoroff, Taylor, Asmundson, & Koch, 2000; Taylor, 2003, 2004). AS may exacerbate PTSD symptoms by causing the individual to view the PTSD symptoms as signs of impending death, insanity, rejection by others, or indicative of other harmful consequences (Fedoroff et al., 2000). This creates a cycle similar to PD, in which the beliefs that the symptoms are harmful lead to increased fear and attempted avoidance of threat-related stimuli, which further increases the level of PTSD symptoms. Findings in past research have consistently reported an association between AS and PTSD symptoms (Asmundson & Stapleton, 2008; Collimore, McCabe, Carleton, & Asmundson, 2008; Fedoroff et al., 2000; Feldner, Lewis, Leen-Feldner, Schnurr, & Zvolensky, 2006; Keogh, Ayers, & Francis, 2002; Lang, Kennedy, & Stein, 2002; Leen-Feldner et al., 2008; Stewart, Conrod, Samoluk, Pihl, & Dongier, 2000; Wald & Taylor, 2008). Several studies have yielded findings indicating that individuals with PTSD or ASD report higher levels of AS than non-PTSD or non-ASD groups (Asmundson & Carleton, 2005; Bonin, Norton, Asmundson, Dicurzio, & Pidubney, 2000; Bryant & Panasetis, 2001; Kilic et al., 2008; Nixon & Bryant, 2005; Nixon & Bryant, 2003; Lang et al., 2002). AS has served as a significant predictor of PTSD symptoms over time (average of 18 months), with the strength of this relation stronger in females than males (Feldner, Zvolensky, Schmidt, & Smith, 2008). Unfortunately, the majority of studies examining the relation between AS and PTSD have failed to account for the influence of more general vulnerabilities, such as negative affect or trait anxiety. While initial studies that have examined both AS and one of these general vulnerabilities have continued to find significant relations between AS and trauma symptoms (Feldner, Leen-Feldner, Trainor, Blanchard, & Monson, 2008; Feldner et al., 2006; Feldner, Zvolensky et al., 2008; Leen-Feldner et al., 2008; Stewart et al., 2000; Vujanovic, Zvolensky, & Bernstein, 2008), additional research demonstrating independent effects of AS is needed.

Discriminant function analysis has shown that AS successfully discriminated between participants endorsing PTSD and participants not endorsing PTSD (Asmundson, Norton, Allardings, Norton, & Larsen, 1998; Bonin et al., 2000; Hinton, Pich, Safren, Pollack, & McNally, 2005). When using regression methods, AS has emerged as a significant predictor of level of PTSD symptomatology (Asmundson et al., 1998; Feldner et al., 2006) and a moderator between trauma exposure frequency and posttraumatic stress symptomatology (Feldner et al., 2006). Furthermore, reductions in AS has been reported to be associated with reductions in PTSD symptoms after cognitive-behavioral treatment (Fedoroff et al., 2000). AS also mediates PTSD symptoms and heavy drinking (Stewart et al., 2000) and moderates between smoking and PTSD symptoms (Feldner, Babson, Zvolensky, Monson, Bonn-Miller, & Gibson, 2008).

Studies examining the relation between the three (i.e., fears of psychological dyscontrol, fears of physical symptoms, and social concerns) dimensions of AS and level of PTSD symptoms have yielded equivocal findings. While two studies (Lang et al., 2002; Vujanovic et al., 2008) reported that psychological concerns showed the strongest relation with PTSD symptoms, Keogh et al. (2002) found that the social concerns subscale was the most strongly related to PTSD symptoms. Evidence has also suggested that an AS taxon may serve as a vulnerability to the development of PTSD after the experience of traumatic events (Bernstein, Zvolensky, Feldner, Lewis, & Leen-Feldner, 2005; Bernstein et al., 2005). Continued investigation between AS and PTSD is needed to determine which components of AS are most related to PTSD and the most appropriate manner of measuring and testing the relation.

## 7. Looming cognitive style

The Looming Cognitive Style (LCS; formerly the Looming Maladaptive Style) examines individuals' characteristic styles of interpreting and making predictions about present and future potential threat. The looming vulnerability model of anxiety describes a danger schema that places one at risk for the development of anxiety as a result of cross-situationally generating mental representations of rapidly intensifying threat or danger (Riskind, 1997; Riskind et al., 2000; Williams, Shahar, Riskind, & Joiner, 2005). Central to the looming vulnerability construct is the human ability of autoegetic consciousness (Wheeler, Stuss, & Tulving, 1997) or the ability to generate mental representations of past, present, and future events. Mental representation allows for the ability to identify, and subsequently prepare for or avoid, potentially threatening situations. When used appropriately, representational abilities serve a protective function and decrease short-term stress by allowing individuals to prevent or avoid future threats.

It is proposed that for some individuals this defensive representational function develops into a relatively enduring, cross-situational tendency to mentally represent potential threats as rapidly intensifying in risk or danger (Riskind, 1997; Riskind et al., 2000). An individual develops a LCS when the adaptive function of the looming mental representation evolves into a cognitive bias that permeates across situations and leads to overestimation of the magnitude, severity, and progression of potential threat. Individuals with a high LCS appear to be sensitive to signs of threat movement and intensifying danger in their environments and to exhibit biased cognitive processing, regardless of their current levels of anxiety (e.g., Riskind et al., 2000). As a result of these biased threat appraisals, individuals high in the LCS feel a sense of urgency and imperative need for action that often leads to rigid avoidance coping and therefore maintains their anxiety through negative reinforcement processes. Support for the validity of the LCS has been demonstrated in a variety of populations (from clinical to non-clinical) with a variety of methodologies (See Riskind & Williams, 2005). The LCS has been demonstrated relations with anxiety and not depression, thus functioning as a specific vulnerability to anxiety. The LCS has predicted shared variance across a variety of anxiety disorders, including obsessive-compulsive disorder, post-traumatic stress disorder, social phobia, specific phobic fears, and panic disorder (Williams et al., 2005).

### 7.1. Measurement of LCS

The Looming Maladaptive Style Questionnaire (LMSQ; Riskind et al., 2000) was created to measure the extent to which a person appraises threat as rapidly approaching through time and space. The LMSQ presents participants with six vignettes describing potentially anxiety provoking situations (e.g., public speaking, risk of physical injury, romantic rejection) and asks them to answer three questions about the projected outcome (e.g., likelihood of increase in risk) of each vignette based on a five-point Likert scale. The responses across dimensions and vignettes are summed to provide a single total score. Higher scores indicate higher levels of engaging in the LCS.

The LMSQ has demonstrated empirical support for convergent, predictive, and discriminant validity across multiple studies. In the initial validation study, Riskind et al. (2000) found support for the convergent validity of the LMSQ. Specifically, higher scores on the LMSQ correlated positively with higher levels of anxiety symptoms. Although the LMSQ correlates with other anxiety related constructs, research using structural equation modeling has also demonstrated that LCS can be distinguished from measures of anxiety sensitivity, neuroticism, negative affect, and negative life events while predicting distinct variance in anxiety beyond that predicted by those measures (Riskind et al., 2000; Williams et al., 2005; Reardon & Williams, 2007). The LMSQ has significantly predicted residual gains in anxiety and

anxiety related symptoms after controlling for base level anxiety in several longitudinal studies (e.g., Riskind & Williams, 1999; Riskind et al., 2000).

### 7.2. Looming cognitive style and PTSD

Research examining the role of the LCS in PTSD is limited. However, when compared to victims of interpersonal trauma with low levels of the LCS, victims high in the LCS are predicted to view more situations or stimuli as threatening, rate situations as more threatening due to the perceived escalation of danger, spend less time and resources evaluating the situation and determining how to cope with the situation, and use more passive, avoidance-oriented coping techniques. Specifically, trauma victims with high levels of the LCS are predicted to perceive ambiguous situations with a bias toward overestimating the magnitude, severity, and progression of risk and danger. The biased processing associated with the LCS is presumed to lead to longer lasting, more severe, and more frequent post-trauma reactions in victims of interpersonal trauma that are higher in this cognitive style. When asked about their interpretations of film clips displaying ambiguous interpersonal situations, a recent study did find that victims that interpreted positive scenes as rapidly rising in risk endorsed the highest levels of PTSD symptoms (Elwood, Williams, Olatunji, & Lohr, 2007). LCS may function as a vulnerability to the development of psychopathology after sexual assault either independently or in conjunction with other cognitive vulnerabilities.

### 8. Additional vulnerabilities

Several other vulnerabilities may also serve as vulnerability and maintenance factors in PTSD. Considered more as a general vulnerability, personality or temperament traits have received substantial empirical attention as potential vulnerability or maintenance factors for PTSD. Neuroticism (Eysenck, 1967) and negative affect (Watson & Clark, 1984) are often associated with PTSD and anxiety disorders in general. Elevated levels of negative affectivity/neuroticism are common across anxiety disorders regardless of the environmental situation. However, these traits have been found to be elevated in individuals with PTSD, even compared to individuals suffering from other anxiety disorders (Trull & Sher, 1994). Several studies have empirically investigated the relation between neuroticism and higher PTSD symptoms across multiple trauma populations (e.g., Engelhard, Huijding, van den Hout, & de Jong, 2007; Engelhard, van den Hout, & Kindt, 2003; Engelhard, & van den Hout, 2007; Engelhard, van den Hout, & Schouten, 2006; van den Hout & Engelhard, 2004). For example, Engelhard et al. (2003) reported that pre-trauma neuroticism strongly predicted PTSD symptoms, particularly PTSD arousal symptoms, after pregnancy loss. However, the relationship was no longer significant after controlling for pre-trauma arousal. Using a different conceptualization of personality, Miller et al. (Miller, 2003; Miller, Kaloupek, Dillon, & Keane, 2004; Miller & Resick, 2007) have demonstrated that extreme scores for externalizing (i.e., disinhibition and negative temperament) or internalizing (i.e., low positive emotions and negative temperament) personality types were associated with more severe PTSD symptomatology compared to moderate personality counterparts. Together these studies suggest that personality and temperament may be a promising avenue for future research.

Several other vulnerability factors may also be implicated in the development of PTSD. Disgust sensitivity has been indirectly theorized as potentially influencing emotional processing that occurs both at the time of the trauma or by shaping retrospective memories (Dalgleish & Power, 2004). In addition, traumatized individuals who experience high levels of disgust may be more likely to experience self-disgust or mental pollution (Fairbrother & Rachman, 2004; Olatunji, Elwood, Williams, & Lohr, 2008; Power & Dalgleish, 1999).

Emotion regulation difficulties have also recently garnered increasing empirical attention. A recent study found that post-traumatic symptom severity was directly related with emotion dysregulation: specifically a lack of emotional acceptance, difficulty engaging in goal-directed behavior when experiencing negative mood, difficulties with impulse control, and lack of emotional clarity, even after controlling for negative affect (Tull, Barrett, McMillan, & Roemer, 2007). Kashdan, Uswatte, Steger, and Julian (2006) compared affect instability and fragile self-esteem between Veterans who were diagnosed and non-diagnosed with PTSD for a 14 day period and found that Veterans with PTSD reported significantly greater instability (more fluctuation) in both self-esteem and negative affect than those without PTSD. Finally, genetics research has offered evidence implicating specific gene expressions or receptors that may contribute to the onset of PTSD symptoms in animals and humans (e.g., Harada, Yamaji, & Matsuoka, 2008; Kozlovsky et al., 2008). While acknowledging the potential function of these additional vulnerabilities, the present paper focuses on the role of cognitive vulnerabilities to PTSD, and therefore extended review is beyond the scope of this paper.

### 9. Integrated model

As research examining vulnerabilities to psychopathology advances, it has been suggested that studies focus on increasing the understanding of how vulnerabilities may interact in the development of disorders, rather than examining vulnerabilities independently (Ingram & Price, 2001). The wide array of symptoms included in the criteria for and associated with PTSD make it unlikely that any single vulnerability factor will account for a large amount of variance in the prediction of this complex disorder. Rather, it is possible that the examination of multiple vulnerability factors using an integrative diathesis approach could explicate the causal processes through which individuals develop PTSD. An integrative diathesis approach would compare multiple vulnerability factors simultaneously and examine the interactions between the vulnerabilities to allow for a more comprehensive account of PTSD development. Studies utilizing this approach could provide information about which vulnerabilities can uniquely contribute to the development of the disorder and how the vulnerabilities function in relation to each other. An integrative diathesis model should consider both the relation between each diathesis and total level of PTSD symptoms as well as the relation between the diatheses and specific symptom clusters. If multiple diatheses were considered that each shared a strong relation with a specific cluster or subset of PTSD symptoms, together the diatheses could account for a greater total amount of variance. Once vulnerabilities are identified that have independent relations with PTSD symptoms, the influence of the possession of multiple vulnerabilities on the individual can be examined as well. High levels of multiple vulnerabilities could interact with each other and leave the individual at an increasingly greater level of risk. Or, if the vulnerabilities have an additive effect on risk, perhaps a total cognitive risk assessment inventory could be created that could assess cognitive vulnerability in multiple areas.

The identification and increased understanding of vulnerability factors independently related to PTSD and vulnerability interactions influential to the development of PTSD would have implications for future prevention and treatment procedures for PTSD. Research examining prevention of PTSD is in its infancy, and thus far few procedures have received empirical support (see Feldner, Zvolensky, & Schmidt, 2004 for a review). However, research suggests that interventions that focus on cognitive vulnerabilities, such as anxiety sensitivity, are feasible and are linked with reductions in the cognitive vulnerability and subsequent anxiety symptomatology (e.g., Schmidt et al., 2007). Once specific cognitive vulnerabilities receive support in relation to PTSD, the knowledge could be used to individualize prevention and treatment interventions. For prevention, trauma exposed individuals could be screened and individuals high in related

vulnerabilities could be identified and psychoeducation and/or prevention techniques designed to correct the cognitive distortions could be provided. Similarly, cognitive treatments for PTSD could identify cognitive techniques and treatment sections related to particular areas of concern for individuals with PTSD. Clinicians could then utilize only the sections relevant to the individual patient and/or vary the order of presentation according to the distress associated with specific symptoms.

An example of an integrative vulnerability model can be designed using the cognitive vulnerabilities reviewed above. When considering cognitive vulnerabilities previously linked with other disorders, such as the ones included in the above review, it is likely that the individual cognitive vulnerabilities are each associated most strongly with the subset of symptoms closest in resemblance to the disorder originally linked with the vulnerability. As NAS is most strongly linked with depressive and hopelessness symptoms, it would be proposed that it would be most strongly linked with the avoidance and numbing symptoms of PTSD. Similarly, rumination has been posited to be related to increased negative mood and re-experiencing symptoms, and would therefore be expected to be related to both the avoidance and numbing cluster and re-experiencing symptoms. Given the similarities to panic disorder, AS would be predicted to be related to the hyper-arousal symptoms. Finally, LCS could be proposed to be linked with re-experiencing and hypervigilance symptoms. If these relations were supported, the information could be used in treatment. For example, if an individual seeking treatment for PTSD described his or her arousal symptoms as the most frequent and distressing, his or her level of AS could be assessed and relevant techniques could be integrated into treatment.

## 10. Discussion

The consideration of integrative diathesis-stress models has several implications for future research. Meaningful integrative models will include multiple vulnerability factors that are each strongly and specifically associated with PTSD. In order for these models to be examined, research must first identify which of the proposed vulnerabilities have the strongest influences on PTSD development and continue to account for variance even with the inclusion of other variables in the model. In addition, research should aim to differentiate between specific and non-specific factors in relation to PTSD. This can be accomplished using several methods. First, studies examining vulnerabilities for PTSD should consider assessing multiple vulnerabilities simultaneously. It is hoped that as vulnerabilities are examined simultaneously, certain vulnerabilities will emerge as the most strongly related to PTSD. As strong vulnerabilities are identified, studies of newly examined vulnerabilities should consider controlling for the previously established vulnerabilities. This would allow the researchers to determine if the new vulnerabilities can account for additional variance. Studies could compare to previously established strong predictors in a manner similar to a best available treatment in treatment outcome research. In addition, the comparison of effect sizes and meta-analytic procedures can be used to consider the strength of the relations between vulnerabilities and PTSD across studies.

After strong associations have been identified between potential vulnerability factors and PTSD symptoms, the nature of the relation still needs to be defined. One of the key characteristics of vulnerability theory is the presence of the vulnerability prior to the activating event. Longitudinal research is needed to investigate the temporal relationship between the proposed vulnerability factor and the resulting symptoms. Although many of the studies above utilized longitudinal methodology, the studies typically assessed the individual at the time of or shortly after trauma exposure and then at a designated time later. Many characteristics of these potential vulnerability factors could develop alongside or subsequent to the development of the disorder,

which, due to inconsistency with diathesis-stress theory, would render the factors ineligible for the role of a vulnerability factor. For example, the nature and the direction of the relation between AS and PTSD has been questioned and multiple possible relations between the two have been described (Taylor, 2004). Consistent with a vulnerability model, if AS was present in an individual before the experience of a traumatic event, then it could serve as a vulnerability factor by increasing the fear of anxiety symptoms that accompany exposure to trauma related stimuli, therefore increasing avoidance and leading to the development and maintenance of PTSD. Alternatively, an individual could have average levels of AS prior to the traumatic event, but experience panic symptoms during the event resulting in the individual associating anxious symptoms with the trauma. In this scenario, it is likely that the individual concurrently develops high levels of AS and PTSD symptoms, and therefore AS would not have served as a vulnerability. Although even supportive findings would not prove that there is a causal relation between the vulnerabilities and symptoms, longitudinal studies could provide valuable information about the temporal relations between the two and could provide suggestions for future research.

The diathesis-stress model and research examining cognitive vulnerabilities may assist in the elucidation of the conceptualization of PTSD even if findings suggest that proposed cognitive vulnerabilities are not present prior to the experience of traumatic events. As previously mentioned, cognitive vulnerabilities may also serve as maintenance factors of PTSD symptoms. It is likely that findings regarding cognitive vulnerabilities would have similar treatment implications regardless of whether the cognitive vulnerability serves as a traditional vulnerability or if it serves primarily as a maintenance factor. If the vulnerabilities were discovered to serve only as maintenance factors, knowledge gained regarding their relation with PTSD symptoms would be primarily useful for treatment, while true vulnerabilities could prove useful for prevention as well. A second possibility to consider is that a cognitive vulnerability that is developed as a consequence of a traumatic event could maintain a vulnerability function from that point forward in the individual's life. If this is the case, cognitive vulnerabilities could interact with additional life stressors to influence fluctuation in level of PTSD symptoms or relapses.

As previously mentioned, a proposed strength of examining cognitive vulnerabilities is the expectation that knowledge gained will be applicable for treatment and prevention. Research examining the relation between cognitive vulnerabilities and treatment outcome is needed to examine this assumption. Initial research examining the relation between anxiety sensitivity and PTSD treatment is promising, suggesting that anxiety sensitivity does decrease with treatment of PTSD (Fedoroff et al., 2000). However, this research examined the change in levels of AS after individuals participated in standard treatment. While these findings are promising, diathesis-stress models propose that modification of the cognitive vulnerability would result in symptom reduction. Research examining the impact of treatment specifically addressing themes specific to the related cognitive vulnerabilities, once identified, will provide important information about the utility of cognitive vulnerabilities for treatment of PTSD.

One limitation of the extant literature is the heavy reliance on self-report, retrospective measurement of the vulnerabilities. The reviewed studies typically rely on the assumption that an individual is able to accurately report on his or her cognitive patterns; however the validity of this assumption is unknown. Moreover, cognitive patterns are considered valuable largely because of their proposed relation to behaviors, but the reviewed studies typically do not assess the relation between the cognitive styles and behaviors. Furthermore, the measures used typically rely on the individual's retrospective reports or projected reactions to hypothetical events which introduce greater complications. It is

ence with diathesis-stress theory, would  
le for the role of a vulnerability factor. For  
re direction of the relation between AS and  
l and multiple possible relations between  
ribed (Taylor, 2004). Consistent with a  
was present in an individual before the  
event, then it could serve as a vulnerability  
ar of anxiety symptoms that accompany  
d stimuli, therefore increasing avoidance  
pment and maintenance of PTSD. Alter-  
d have average levels of AS prior to the  
ience panic symptoms during the event  
associating anxious symptoms with the  
is likely that the individual concurrently  
and PTSD symptoms, and therefore AS  
vulnerability. Although even supportive  
at there is a causal relation between the  
ms, longitudinal studies could provide  
the temporal relations between the two  
ns for future research.

del and research examining cognitive  
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itive vulnerabilities may also serve as  
D symptoms. It is likely that findings  
bilities would have similar treatment  
whether the cognitive vulnerability serves  
if it serves primarily as a maintenance  
were discovered to serve only as  
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narily useful for treatment, while true  
prevention as well. A second  
vulnerability that is  
maintain a