Journal of Anxiety Disorders 26 (2012) 689–695

Contents lists available at SciVerse ScienceDirect

Journal of Anxiety Disorders

Examining the incremental contribution of behavioral inhibition to generalized anxiety disorder relative to other Axis I disorders and cognitive-emotional vulnerabilities

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ARTICLE INFO

Article history:
Received 26 September 2011
Received in revised form 23 March 2012
Accepted 21 May 2012

Keywords:
Keywords: Anxiety disorders
Anxiety sensitivity
Emotion regulation
Major depression
Reinforcement sensitivity theory

ABSTRACT

The goal of the present study was to investigate the incremental contribution of behavioral inhibition system (BIS) sensitivity to the presence of a current generalized anxiety disorder (GAD) diagnosis relative to other Axis I disorders (e.g., major depression, other anxiety disorders) and cognitive-emotional vulnerabilities (e.g., anxiety sensitivity, emotion dysregulation) previously found to be associated with GAD. Participants were 91 individuals recruited from the local community who completed a diagnostic interview and self-report questionnaires. Consistent with expectations, BIS sensitivity emerged as a significant predictor of current GAD status above and beyond major depression, anxiety disorder diagnoses, anxiety sensitivity, emotion dysregulation, and behavioral activation system sensitivity. However, emotion dysregulation also emerged as a significant predictor of GAD status in the final model. Findings speak to the importance of considering BIS sensitivity in models of the development and maintenance of GAD.

1. Introduction

Generalized anxiety disorder (GAD) is a chronic and debilitating anxiety disorder that affects approximately 6% of individuals in the United States at some point in their lifetime (Kessler, Chiu, Demler, Merikanga, & Walters, 2005). GAD is characterized by excessive and uncontrollable worry about a variety of concerns, persisting more days than not over a period of at least 6 months (American Psychiatric Association [APA], 2000). Further, to be diagnosed with GAD, individuals must also experience at least three of the following symptoms: feeling keyed up or on edge, sleep disturbance, muscle tension, being easily fatigued, difficulty concentrating or having one's mind go blank, and irritability (APA, 2000). GAD has historically been the least studied of all the anxiety disorders (Dugas, 2000; Roemer, Orsillo, & Barlow, 2002), and is considered to be the most poorly understood and least effectively treated anxiety disorder (Brown, Barlow, & Liebowitz, 1994; Fisher, 2006). However, the past two decades have seen an increase in research on GAD aimed at elucidating the cognitive-emotional vulnerabilities that may underlie this disorder, with the ultimate goal of improving our understanding of the pathogenesis and treatment of this disorder (Behar, DiMarco, Hekler, Mohlman, & Staples, 2009; Roemer et al., 2002).

Two cognitive-emotional vulnerabilities that have received a substantial amount of attention with regard to the pathogenesis of GAD are emotion dysregulation (defined as maladaptive ways of responding to emotions, including a lack of awareness and understanding of emotions, nonacceptance or avoidance of emotions, difficulties controlling behaviors in the face of emotional distress, and deficits in the modulation of emotional arousal; Gratz & Roemer, 2004) and anxiety sensitivity (AS; defined as the tendency to fear anxiety-related symptoms due to beliefs that their occurrence will have negative somatic, cognitive, or social consequences; Reiss, 1991). AS overlaps with the dimension of emotion dysregulation involving the nonacceptance of emotional experiences. In particular, both AS and emotional nonacceptance involve maladaptive responses to emotional experiences (with emotional nonacceptance pertaining to emotions in general and AS concerning the specific emotion of anxiety) that interfere with the adaptive function of these emotions, contributing to maladaptive behavior. In addition, both emotion dysregulation and AS may encompass a sensitivity to and intolerance of certain emotions, which, in turn, could contribute to avoidant behavior (Zvolensky, Leyro, Bernstein, & Vujanovic, 2011). Nonetheless, despite their overlap, theoretical and empirical literature indicate that AS and emotion dysregulation are distinct constructs, with emotion dysregulation involving

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http://dx.doi.org/10.1016/j.janxdis.2012.05.005

many other dimensions than the nonacceptance or fear of emotions (e.g., access to strategies for modulating emotional arousal and the control of behaviors in the context of emotional distress; Gratz & Roemer, 2004; Gratz & Tull, 2010), and AS including a more specific focus on anxiety and a stronger cognitive component than emotion dysregulation (Taylor & Fedoroff, 1999). Furthermore, previous research has demonstrated that AS and emotion dysregulation are uniquely associated with anxiety-related difficulties, such as panic attacks (Tull, 2006) and GAD (Tull, Stipelman, Salters-Pedneault, & Gratz, 2009). Thus, despite some overlap, emotion dysregulation and AS are distinct constructs that warrant consideration as separate vulnerabilities for GAD.

With regard to emotion dysregulation in GAD, a series of studies by Mennin, Heimberg, Turk, and Fresco (2005) found that individuals with GAD reported higher levels of many dimensions of emotion dysregulation (including a poor understanding of emotions, difficulties self-soothing after the experience of negative emotions, and difficulties managing emotional reactions following mood induction) than individuals without GAD. Further, emotion dysregulation was found to predict the presence of GAD above and beyond both worry and depression. Lending further support to the role of emotional dysregulation in GAD, Salters-Pedneault, Roemer, Tull, Rucker, and Mennin (2006) found that emotion dysregulation (both overall and across the specific dimensions of non-acceptance of emotions, limited access to effective emotion regulation strategies, difficulties engaging in goal-directed behaviors when distressed, difficulties controlling impulsive behaviors when distressed, and lack of emotional clarity) was significantly associated with both worry (a central defining feature of GAD) and analog GAD status. In addition, Roemer, Salters, Raffa, and Orsillo (2005) found that symptoms of GAD and chronic worry are associated with a tendency to avoid and control negatively evaluated internal experiences in both clinical and non-clinical samples.

With regard to the association between AS and GAD, studies have demonstrated that individuals with GAD report higher levels of AS than healthy controls (Deacon & Abromowitz, 2006; Taylor, Koch, & McNally, 1992). Likewise, Cox, Fuentes, Borger, and Taylor (2001) found that individuals endorsing GAD screening questions on the Structured Clinical Interview for DSM-IV (SCID-IV; First, Spitzer, Gibbon, & Williams, 1996) reported higher levels of AS than those who did not endorse the screening questions. Finally, Floyd, Garfield, and LaSota (2005) found that AS was significantly positively associated with worry. Despite growing evidence for the relevance of both emotion dysregulation and AS to GAD, however, there are other cognitive-emotional vulnerabilities theoretically relevant to GAD that warrant investigation as well. One such vulnerability that has yet to be studied in relation to GAD is behavioral inhibition.

Specifically, Gray's revised Reinforcement Sensitivity Theory (rRST) proposes that individual differences in motivational tendencies and personality styles are due to differences in the sensitivity of three basic brain systems that respond to punishing and reinforcing stimuli: the behavioral activation system (BAS), behavioral inhibition system (BIS-rRST), and fight-flight-freeze system (FFFS; Gray & McNaughton, 2000). These differences are thought to underlie the personality dimensions of anxiety and impulsivity, and to have relevance for understanding the development of various forms of psychopathology. According to this theory, the BAS is an appetitive system underlying approach behavior in response to conditioned and unconditioned cues of reward (Corr, 2008). The FFFS is a defensive avoidance system that motivates avoidance and escape behaviors in response to conditioned and unconditioned aversive stimuli and underlies fear and panic (Gray & McNaughton, 2000). Finally, the BIS-rRST is the subsystem underlying anxiety that detects, is activated by, and resolves conflicts that occur between reward (BAS) and punishment (FFFS) contingencies. The BIS-rRST is also activated by approach–approach and avoidance–avoidance conflicts (Gray & McNaughton, 2000). In such situations, depending on the extent of the conflict and resulting arousal, the BIS-rRST may inhibit ongoing or BAS-mediated behavior and direct attention to sources of potential threat, facilitating FFFS-mediated (i.e., defensive or avoidant) behavior (Gray & McNaughton, 2000).

Although the current rRST separates the functions of the BIS-rRST and FFFS, available measures of RST (based on the original version of this theory, which distinguished between only BAS and BIS) do not. These measures (including the most widely used measure of RST, the BIS/BAS Scales; Carver & White, 1994) do not include separate subscales to measure FFFS and BIS-rRST sensitivity, but assess combined BIS-FFFS sensitivity within the rRST framework (Corr, 2004; Smillie, Pickering, & Jackson, 2006). Thus, from this point forward, when specifically referring to the BIS-rRST as proposed by rRST (i.e., as a subsystem separate from the FFFS), the term BIS-rRST will be used. The term BIS will be used when referring to studies or findings that stem from measures that incorporate both BIS-rRST and FFFS functioning.

In-line with rRST, there is a substantial body of empirical literature suggesting an association between BIS and anxiety in general. For example, anxiety symptoms generally demonstrate significant positive associations with BIS sensitivity, and an absence of significant associations (or very weak associations) with BAS sensitivity (Beevers & Meyer, 2002; Campbell-Sills, Liverant, & Brown, 2004; Johnson, Turner, & Iwata, 2003; Segarra et al., 2007). BIS sensitivity has also been found to be associated with specific anxiety disorders and anxiety disorder symptoms. For instance, BIS sensitivity has been found to be positively related to social anxiety symptoms (e.g., fear of negative evaluation, social avoidance, distress in response to new people and situations) and social anxiety disorder (Coplan & Arbeau, 2008; see also Kimbrel, 2008, for a review). Likewise, Fullana et al. (2004) found higher BIS sensitivity among individuals with elevated levels of obsessive compulsive symptoms (compared to healthy controls). Finally, Maack, Tull, and Gratz (in press) found that BIS sensitivity was significantly uniquely associated with the severity of posttraumatic stress disorder (PTSD) symptoms within a trauma-exposed undergraduate sample. Notably, however, no studies to date have examined the relationship between BIS sensitivity and GAD specifically.[1]

The absence of research in this area notwithstanding, there is reason to expect that BIS sensitivity may be particularly relevant to GAD. First, BIS sensitivity is strongly associated with the personality trait of neuroticism (e.g., Jorm et al., 1998; Mitchell et al., 2007), a trait that has been found to be more strongly associated with GAD than any other anxiety disorder (Kotov, Watson, Robles, & Schmidt, 2007). In addition, BIS-rRST sensitivity is theorized to motivate avoidance behaviors, and BIS sensitivity has been found to be associated with heightened tendencies to avoid internal experiences (Maack et al., in press), consistent with the proposed emotionally avoidant function of worry (the central defining feature of GAD; Borkovec, Alcaine, & Behar, 2004; Roemer et al., 2005).

Thus, the goal of the present study was to explore the unique association between BIS sensitivity and GAD. To this end, we examined the incremental contribution of BIS sensitivity to the presence of a current GAD diagnosis relative to both other Axis I disorders that have been found to be associated with GAD (i.e., major

[1] It warrants mention that Johnson et al. (2003) did investigate BIS levels among individuals with an anxiety disorder diagnosis, including GAD. However, anxiety disorder diagnoses were not examined separately, but as a group. Consequently, although Johnson et al. (2003) did find higher levels of BIS sensitivity among individuals with an anxiety disorder diagnosis, it is not clear whether people with a diagnosis of GAD exhibited significantly higher levels of BIS sensitivity than those without this diagnosis.