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Emotion dysregulation and sleep difficulties in generalized anxiety disorder

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ABSTRACT

Diagnostic criteria for generalized anxiety disorder (GAD) include sleep problems, which often persist even after successful treatment of the disorder. The purpose of this study was to examine emotion dysregulation as a potential contributor to sleep problems in GAD patients. Participants comprised two groups: 59 individuals diagnosed with GAD and 66 healthy controls. They were assessed for the presence of mood and anxiety disorders and then completed self-report questionnaires assessing problems with sleep and emotion regulation. Participants in the GAD group scored significantly higher on a number of sleep outcomes than did the control group. Importantly, difficulties with emotion regulation statistically mediated the relationship between GAD and a wide range of outcomes of sleep dysfunction independently of the effects of depression and secondary anxiety diagnoses. Emotion regulation difficulties that characterize GAD mediate the relationship between symptoms of this disorder and a wide range of sleep problems. Implications for treatment and future research directions are discussed.

1. Introduction

Sleep difficulties are included in the diagnostic criteria for both mood and chronic anxiety disorders such as generalized anxiety disorder (GAD) (APA, 2000). The majority of studies have examined sleep dysfunction in the context of major depressive disorder (MDD) despite GAD being one of only two anxiety disorders (along with Posttraumatic Stress Disorder – PTSD) for which the diagnostic criteria include (although not requisite for diagnosis) sleep-related symptoms [i.e., difficulty falling or staying asleep, or restless/unsatisfying sleep (APA, 2000)]. A recent study found that about 74% of primary care patients with anxiety disorders reported sleep disturbance, and those diagnosed with GAD or PTSD were over two times more likely to have sleep problems (Marcks, Weisberg, Edelen, & Keller, 2010). Moreover, anxiety disorders are the most common diagnoses in patients with insomnia symptoms (e.g., Ohayon, 2002).

Research shows that GAD has the highest comorbidity rate with insomnia of the anxiety disorders (e.g., Monti & Monti, 2000). This is not surprising given that the tendency to worry before bed and in bed has been shown to cause sleep interference (e.g., Harvey, 2000), and GAD is characterized by excessive, uncontrollable, and pervasive worry (APA, 2000). Overall, individuals with GAD demonstrate sleep dysfunction in both subjective and objective indices (e.g., Brenes et al., 2009; Fuller, Waters, Binks, & Anderson, 1997; Ohayon, 1997; Roth et al., 2006; Wetherell, Le Roux, & Gatz, 2003). Specifically, compared with healthy controls, GAD patients tend to endorse longer sleep latency (e.g., Akiskal et al., 1984; Fuller et al., 1997; Papadimitriou, Kerkhofs, Kempenaers, & Mendlewicz, 1988; Papadimitriou & Linkowski, 2005), decreased sleep duration (e.g., Papadimitriou & Linkowski, 2005; Saletu-Zyhlarz et al., 1997), decreased total sleep efficiency (e.g., Saletu-Zyhlarz et al., 1997) and increased wake periods throughout total sleep time (e.g., Akiskal et al., 1984; Saletu-Zyhlarz et al., 1997). Sleep maintenance insomnia is one of the most common complaints among individuals with GAD (e.g., Monti & Monti, 2000), reported by 64% of them (Belanger, Morin, Langlois, & Ladouceur, 2004). The majority of individuals with GAD report that their sleep disturbance interferes with daily activities and overall functioning (e.g., Belanger et al., 2004).

Importantly, sleep difficulties in GAD often persist after successful treatment of the disorder. For example, a recent meta-analysis evaluated the impact of CBT for anxiety disorders on associated sleep disturbances and found only a moderate impact of the treatment on sleep (e.g., Belleville, Cousineau, Levrier, St-Pierre-Delorme, & Marchand, 2010). This suggests that there might be dispositional mechanisms that remain present after the improvement of GAD and might therefore account for the continuous presence of sleep difficulties following treatment. One such

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mechanism may be the ability to regulate emotions, which involves the strategies people employ in order to influence the types of emotional responses they have, when these responses occur, and the ways of experiencing and expressing these emotions (Gross, 1998).

According to the emotion dysregulation model of GAD, this disorder is characterized by increased subjective emotional intensity as well as concomitant difficulties in managing emotions (Mennin & Fresco, 2009). Emotional intensity may become dysfunctional when individuals have difficulties knowing when and how to properly regulate it according to the situational demands. Indeed, GAD patients report pervasive difficulties regulating their emotions and endorse more difficulties managing emotions compared to healthy controls and individuals with depression and social anxiety disorder (e.g., Mennin, Holaway, Fresco, Moore, & Heimberg, 2007). Emotion regulation deficits experienced by individuals with GAD also pertain to the implementation of adaptive emotion regulation strategies (e.g., Salters-Pedneault, Roemer, Tull, Rucker, & Mennin, 2006). For example, in one recent study, when individuals with GAD were instructed to implement acceptance and reappraisal to regulate their emotions in response to emotion-eliciting film clips, they demonstrated lower cardiac flexibility (i.e., heart rate variability) relative to when not given specific instructions on how to regulate their emotions. Control participants showed the opposite pattern, that is higher flexibility when accepting and reappraising than when not given instructions, suggesting that they might have benefited from instructions to implement such strategies (Aldao & Mennin, 2012).

No investigations to date have examined the relationship between emotion dysregulation and associated dysregulated behavior, such as sleep, in individuals with GAD. Research shows that affective aspects of increased mental activity at bedtime are likely to contribute to sleep problems (e.g., Espie, 2002; Schmidt, Harvey, & Van der Linden, 2011). The relationship between disrupted sleep and emotion is likely bidirectional, with sleep affecting emotions and emotions, in turn, affecting sleep. For example, the emotions of guilt, shame, and regret have been shown to be positively associated with self-reported insomnia severity (e.g., Schmidt & Van der Linden, 2009); hostility has been linked to shorter sleep duration even after statistically controlling for psychiatric disorders (e.g., Brissette & Cohen, 2002); and loneliness has been positively associated with lower sleep efficiency and higher wake time after sleep onset (e.g., Cacioppo et al., 2002). There is also evidence suggesting that poor sleep impairs next-day affective functioning, as sleep deprivation or poor sleep have been shown to increase negative affect and decrease positive affect in both clinical (e.g., Kahn-Greene, Killgore, Kamimori, Balkin, & Killgore, 2007; Zohar, Tzischinsky, Epstein, & Lavie, 2005) and healthy samples (e.g., Babson, Trainor, Feldner, & Blumenthal, 2010; McCrae et al., 2008; Rose, Manser, & Ware, 2008).

The negative impact of emotions experienced by individuals both at bedtime and during the day on sleep might be mediated by difficulties effectively regulating these emotions. However, unlike the influence of sleep-interfering cognitive activity (e.g., Harvey, 2002), the role of affective processes and emotion regulation in poor sleep has received very little of the research attention. Indeed, dysfunctional affect control (e.g., inability to downregulate negative and positive affective states) has been linked with sleep disturbances (e.g., Schmidt et al., 2011; Schmidt & Van der Linden, 2009; Talbot, Hariston, Eidelman, Gruber, & Harvey, 2009). For example, difficulties downregulating positive affect in individuals with bipolar disorder have been shown to contribute to sleep-onset insomnia (Johnson, 2005), and individuals with insomnia have been shown to engage in more dysfunctional, emotion-focused, coping compared to good sleepers (LeBlanc et al., 2007). Additionally, a large body of literature suggests that problems with the expression and regulation of dysphoric emotions are associated with nightmares (for a review, see Levin & Nielsen, 2009). Nightmares are associated with anxiety (e.g., Ohayon, Morselli, & Guilleminault, 1997; Zadra & Donderi, 2000) and overall are more prevalent in psychiatric populations (e.g., Ohayon et al., 1997).

Despite the findings demonstrating a strong association between emotion, emotion dysregulation and both GAD (e.g., Mennin et al., 2007) and sleep problems (e.g., Schmidt et al., 2011), no research, to our knowledge, has looked at the role of emotion dysregulation as a potential mediator of the relationship between GAD diagnosis and negative sleep outcomes. The present study sought to address this gap in the literature by examining the role of emotion dysregulation in the sleep problems associated with GAD diagnosis. We hypothesized that GAD participants would experience more sleep problems than the healthy controls (e.g., Saletu-Zyhlarz et al., 1997; Uhde, 2000). We also predicted that emotion dysregulation would statistically mediate the relationship between GAD diagnosis and these poor sleep outcomes. Due to the substantial comorbidity between GAD and MDD (e.g., Watson, 2005), we controlled for severity of depression in all analyses in order to isolate effects of GAD.

2. Materials and methods

2.1. Study participants and diagnostic screening

This study is part of a larger investigation examining the relationship between emotion regulation and GAD. An Institutional Review Board approved the study and informed consent was obtained from each participant prior to inclusion into the study. Participants ($N = 125$) were recruited via flyers placed in an urban community surrounding a large private university in the northeast United States. These flyers invited individuals who self-identified as excessive worries to participate (i.e., "Do you worry excessively?"). A similar set of flyers asked for non-anxious controls. Interested participants emailed the laboratory and were asked a series of questions to determine preliminary eligibility. They had to be aged 21–65, fluent in English, not be college students, and have no history of heart conditions or diabetes (the larger study included psychophysiological measures). After screening approximately 200 participants, the participants meeting the above criteria were invited to come to the laboratory to participate in a diagnostic assessment.

Participants were interviewed by advanced clinical psychology graduate students and post-baccalaureate research assistants using the Structured Clinical Interview for DSM-IV-TR (SCID; First et al., 2002) to assess for the presence of mood and anxiety disorders. All of the interviewers were trained rigorously over a 6-month period in diagnostic interviewing with the SCID. As part of training, they were required to achieve reliability with expert diagnosticians in a departmental clinic.

Reliability of the SCID diagnoses was determined via the clinical severity rating (CSR) from the Anxiety Disorders Interview Schedule for DSM-IV (DiNardo, Brown, & Barlow, 1994). CSR is a 0–8 rating of the severity of symptoms and associated impairment, with scores of 4 or greater representing clinically significant symptom severity. Complete agreement (within one rating point) in diagnosis and CSR between the interviewer and an expert diagnostician (PhD) was necessary for diagnosis to be considered as present. In order for participants to be included in the GAD group, diagnosis of GAD and a CSR for GAD higher than or equal to that of other anxiety disorders were required. CSR scores of 4 or higher were required in order to be diagnosed with GAD. To be included in the control group, participants needed to have no diagnoses of any mood or anxiety disorder. In addition, 25% of interviews were coded for each diagnosis by the second author, who watched a video recording of the interview (for