



Emotion dysregulation and sleep difficulties in generalized anxiety disorder



Aliona Tsypes^{a,1}, Amelia Aldao^b, Douglas S. Mennin^{a,*}

^a Department of Psychology, Hunter College, City University of New York, 695 Park Avenue, Room 611HN, New York, NY 10065, United States

^b Department of Psychology, The Ohio State University, 1835 Neil Avenue, Room 135, Columbus, OH 43210, United States

ARTICLE INFO

Article history:

Received 11 May 2012

Received in revised form 6 January 2013

Accepted 13 January 2013

Keywords:

Generalized anxiety disorder

GAD

Emotion regulation

Sleep

Anxiety disorders

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.

© 2013 Elsevier Ltd. All rights reserved.

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, Kempnaers, & 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

* Corresponding author. Tel.: +1 212 772 4809; fax: +1 212 772 5620.

E-mail addresses: atsypes@hunter.cuny.edu (A. Tsypes), aldao.1@osu.edu (A. Aldao), dmennin@hunter.cuny.edu (D.S. Mennin).

¹ Tel.: +1 212 772 4809; fax: +1 212 772 5620.

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., Brisette & 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 & Dondori, 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

Table 1
Pearson correlation coefficients between the self-report measures ($N = 125$).

	1	2	3	4	5	6	7	8	9	10	11	12
PSQI												
1. Daytime dysfunction	–											
2. Sleep disturbances	.28**	–										
3. Habitual sleep efficiency	.24**	.35**	–									
4. Sleep duration	.21*	.38**	.70**	–								
5. Sleep latency	.25**	.45**	.54**	.54**	–							
6. Subjective sleep quality	.33**	.45**	.53**	.61**	.54**	–						
Additional sleep questions												
7. Nightmare frequency	.31**	.40**	.15	.17	.30**	.35**	–					
8. Perceived need for more sleep than usual	.45**	.23	.24**	.30**	.19†	.26**	.16	–				
9. Hard time waking up in the morning	.22†	.19†	.12	.06	.14	.20*	.09	.43**	–			
10. Excessive daytime sleepiness	.54**	.24**	.25**	.25**	.22†	.39**	.35**	.55**	.23**	–		
11. Teeth grinding	–.18	–.25**	–.22**	–.24**	–.19†	–.19†	–.22†	–.14	–.01	–.13	–	
12. Average number of naps during the day	.32**	.30**	.29**	.32**	.41**	.37**	.16	.23†	.18†	.37**	–.11	–

* $p < .01$.

** $p < .05$.

GAD and depressive disorders, κ 's from .89 to 1; for other disorders, κ 's from .70 to .85). For the purposes of physiological experimental procedures that were part of the larger investigation, the exclusion criteria consisted of the use of directly affecting cardiac functioning medications (e.g., beta blockers), current substance use or abuse (with the exception of nicotine), or certain medical conditions that affect cardiovascular functioning (e.g., epilepsy, heart condition).

Participants comprised two groups: 59 (74.6% women) who were diagnosed with generalized anxiety disorder (GAD) and 66 (62.1% women) who had no diagnoses. Of those with GAD (average GAD CSR = 5.4), 17 participants had additional diagnoses of major depressive disorder (average MDD CSR = 5) and some of them had secondary diagnoses of anxiety disorders, including social anxiety disorder ($n = 11$; average SAD CSR = 4.2), post traumatic stress disorder ($n = 1$; PTSD CSR = 4), panic disorder ($n = 6$; average PD CSR = 4.5), and specific phobia ($n = 20$, average SP CSR = 4). The mean age of the participants was 28.58 (range 21–55 years), and 85 (68%) of them were women.

2.2. Measures

2.2.1. Emotion regulation

The Difficulties in Emotion Regulation Scale (DERS): The DERS (Gratz & Roemer, 2004) is a 36-item self-report questionnaire that assesses difficulties in emotion regulation under the influence of negative mood. The DERS includes subscales assessing six aspects of emotion dysregulation (nonacceptance, goals, impulse, awareness, strategies, and clarity). Each item is scored on a Likert scale ranging from 1 (*almost never*) to 5 (*almost always*), with higher scores indicating greater problems with emotion regulation. Gratz and Roemer (2004) reported an excellent internal reliability ($\alpha = .93$) for the total score DERS. In this investigation, internal reliability was excellent ($\alpha = .95$).

2.2.2. Sleep (outcome measures)

The Pittsburgh Sleep Quality Index (PSQI): The PSQI (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) is a 19-item self-report measure that assesses sleep quality and sleep disturbances over a one-month interval and includes seven component scores [subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction (i.e., problems staying awake during the day and problems keeping up enough enthusiasm to get things done attributed to poor sleep)]. Although the component score reflecting the use of sleep medication was unintentionally omitted from the present study questionnaire, we had obtained information regarding the participants' self-reported medication use from the

interview conducted as part of the larger study. Each PSQI item is scored on a Likert scale ranging from 1 (*no difficulty*) to 3 (*severe difficulty*), with higher scores indicating greater sleep problems. In the present sample, internal reliability was good ($\alpha = .78$).

In addition, participants completed supplementary questions about their sleep problems to gather additional information about the nighttime problems and concomitant daytime sleepiness associated with GAD. These questions included: frequency of their nightmares over the past three months, with higher score indicating higher nightmare frequency ["Please estimate on average how often you experience nightmares and disturbing dreams based on number of nights" (from Krakow et al., 2002)]; teeth grinding, with lower score indicating more frequent teeth grinding ("According to you, or your bed partner, do you grind your teeth during your sleep?"), and perceived need for sleep during the past month, with higher score indicating greater perceived need for sleep compared to usual ("Have you felt that you needed more sleep than usual?" "Have you had a hard time waking up in the morning?" "Have you been bothered by daytime sleepiness?" "On average how many naps do you need to take during the day?"). Internal reliability of four questions assessing perceived need for sleep compared to usual during the past month was adequate ($\alpha = .67$). Pearson correlations between all of the self-report measures are shown in Table 1.

3. Results

3.1. Demographic characteristics

Demographic information for the sample is summarized in Table 2. There were no statistically significant differences between GAD and control groups in terms of age, $t(23) = .92, p > .05$; gender, $\chi^2(1, N = 125) = 2.22, p > .05$; or ethnicity, $\chi^2(1, N = 125) = .38, p > .05$.

3.2. Sleep outcomes and emotion dysregulation

We ran a univariate analysis of covariance (ANCOVA), testing the between group differences in emotion dysregulation and sleep problems between participants with GAD and healthy controls. We controlled for the severity of depression and secondary diagnoses of anxiety disorders, by entering MDD CSR and average secondary anxiety disorders CSR as covariates. We found a main effect of GAD diagnosis on sleep outcomes for the daytime dysfunction (as a result of the sleep problems), $F(1) = 6.70, p < .05$; sleep disturbances, $F(1) = 10.24, p < .01$; excessive daytime sleepiness, $F(1) = 7.99, p < .01$; perceived need for more sleep than usual, $F(1) = 18.49, p < .01$; sleep duration, $F(1) = 7.17,$

Table 2
Demographic comparison of patients with GAD and nonanxious controls.

	GAD (n = 59)		Controls (n = 66)		χ^2
	n	%	n	%	
Gender					
Female	44	74.58	41	62.12	2.22
Ethnicity					
Caucasian	39	66.10	47	71.21	.38
	M	SD	M	SD	t
Age (Years)	29.19	6.11	28.05	7.64	.915

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

Table 3
Group differences in the sleep outcomes and emotion dysregulation (ANCOVA analyses with MDD CSR and average anxiety CSR as covariates).

Variable	Group						F	p	d	η^2
	GAD (n = 59)			Control (n = 66)						
	Scores range	M	SD	Scores range	M	SD				
PSQI										
Daytime dysfunction	0–3	1.32	.87	0–2	.78	.63	6.70	.011*	5.18	.05
Sleep disturbances	0–3	1.39	.648	0–2	1.03	.40	10.24	.002**	6.40	.08
Habitual sleep efficiency	0–3	.67	.91	0–3	.17	.53	6.48	.012*	5.09	.05
Sleep duration	0–3	1.21	1.05	0–3	.58	.79	7.17	.008**	5.36	.06
Sleep latency	0–3	1.45	1.025	0–3	.92	.78	2.74	.100	3.31	.02
Subjective sleep quality	0–3	1.36	.84	0–2	.83	.58	8.86	.004**	5.95	.07
Additional sleep questions										
Nightmare frequency	0–17	9.79	6.16	0–17	5.73	5.96	5.92	.017*	4.87	.05
Perceived need for more sleep than usual	1–4	2.82	1.10	1–4	1.97	1.02	18.49	.000**	8.60	.14
Hard time waking up in the morning	1–4	2.40	1.07	1–4	2.00	.88	8.58	.004*	5.86	.07
Excessive daytime sleepiness	1–4	2.91	.97	1–4	2.28	.94	7.99	.006**	5.65	.06
Teeth grinding	1–5	3.96	1.44	1–5	4.52	.92	5.87	.017*	4.85	.05
Average number of naps during the day	1–4	1.61	.70	1–4	1.38	.65	4.12	.045*	4.06	.03
DERS										
DERS total	1.36–3.64	2.54	.65	1.11–2.89	1.83	.47	25.03	.000**	10.01	.18

Note: PSQI, Pittsburg Sleep Quality Index; DERS, Difficulties in Emotion Regulation Scale.

* $p < .05$.

** $p < .01$.

$p < .01$; habitual sleep efficiency, $F(1) = 6.48$, $p < .05$; subjective sleep quality, $F(1) = 8.86$, $p < .01$; hard time waking up in the morning, $F(1) = 8.58$, $p < .01$; average number of naps during the day, $F(1) = 4.12$, $p < .05$; nightmare frequency, $F(1) = 5.92$, $p < .05$; and teeth grinding, $F(1) = 5.87$, $p < .05$ were elevated in the GAD group, such that there were more deficits in the GAD group. Participants in the GAD group reported significantly higher scores on emotion dysregulation as measured by DERS compared to the controls, $F(1) = 25.03$, $p < .01$. All ANCOVA results are presented in Table 3.

3.3. Mediation analyses

Linear regressions confirmed the relationship between GAD diagnosis and emotion dysregulation ($\beta = .43$, $p < .01$) and between GAD diagnosis and sleep outcomes, specifically, daytime dysfunction ($\beta = .25$, $p < .05$), sleep disturbances ($\beta = .32$, $p < .01$), excessive daytime sleepiness ($\beta = .28$, $p < .01$), perceived need for more sleep than usual ($\beta = .42$, $p < .01$), sleep duration ($\beta = .26$, $p < .01$), habitual sleep efficiency ($\beta = .26$, $p < .05$), subjective sleep quality ($\beta = .29$, $p < .01$), hard time waking up in the morning ($\beta = .29$, $p < .01$), nightmare frequency ($\beta = .25$, $p < .05$), average number of naps during the day ($\beta = .21$, $p < .05$), and teeth grinding ($\beta = -.25$, $p < .05$).

Nonparametric bootstrapping procedures that involve random data resampling with replacement (Preacher & Hayes, 2008) were employed to examine whether the association between GAD diagnosis and each of the sleep outcome measures was statistically mediated by the hypothesized mediating variable (DERS) when controlling for the severity of depression (MDD CSR) and average

secondary anxiety disorders CSR. Bootstrapping is more powerful compared to the traditional tests of mediation that assume normality, and the bias-corrected bootstrap method produces more accurate confidence intervals than other bootstrap methods (MacKinnon, Lockwood, & Williams, 2004). In order for mediation to occur, there must be a relationship between a predictor and both a mediator and an outcome variable. Additionally, adjusting for a mediator should decrease the relation between the predictor and outcome variable. Full mediation occurs if such adjustment for the mediator makes the relation between a predictor and an outcome variable lose statistical significance.

In this study, data were resampled with replacement to generate 1000 independent estimates of the total and direct effects of GAD diagnosis on sleep. The indirect effects were considered statistically significant if their 95% bias-corrected and accelerated confidence intervals did not contain zero at α level of .05. Results indicated that DERS fully mediated the relationship between GAD diagnosis and most sleep outcomes, specifically, the daytime dysfunction, $BCa = .1735-.5882$; sleep disturbances, $BCa = .8848-5.4874$; excessive daytime sleepiness, $BCa = .0022-.3955$; and perceived need for more sleep than usual, $BCa = .0354-.4687$.² Sleep duration, habitual sleep efficiency, subjective sleep quality, hard time waking up in the morning, nightmare frequency, number of naps during the day, and teeth grinding were not statistically mediated by DERS. Results of

² Controlling for medication use in the statistical analyses resulted in DERS no longer mediating the relationship between GAD diagnosis and excessive daytime sleepiness. The rest of the analysis did not change.

Table 4
Summary of Mediation Analysis (1000 bootstrap samples).

Outcome variables	Product of coefficients		Bootstrapping	
	SE	p	BCa 95% CI	
			Lower	Upper
PSQI				
Daytime dysfunction	.0996	.0000**	.1735	.5882
Sleep disturbances	.0654	.0006**	.0353	.3096
Habitual sleep efficiency	.0822	.0031**	-.1564	.1864
Sleep duration	.0971	.0010**	-.0281	.3504
Subjective sleep quality	.0819	.0002**	-.0253	.3099
Additional sleep questions				
Nightmare frequency	.6993	.0001**	-.7327	2.0560
Perceived need for more sleep than usual	.1080	.0002**	.0354	.4687
Average number of naps during the day	.0641	.2311	-.0319	.2378
Hard time waking up in the morning	.0979	.0138*	-.1760	.2202
Excessive daytime sleepiness	.0994	.0015**	.0022	.3955
Teeth grinding	.1466	.0300	-.4867	.1109

Note. SE, standard error; p, precise significance value; CI, confidence interval; BCa, bias corrected and accelerated; PSQI, Pittsburg Sleep Quality Index.

* $p < .05$.

** $p < .01$.

the mediation and nonparametric bootstrap analyses are presented in Table 4.

4. Discussion

Although previous research has consistently demonstrated the important role of emotion regulation in GAD (e.g., Mennin et al., 2007) and sleep problems (e.g., Schmidt et al., 2011), emotion dysregulation has not yet been examined as one of the potential mediators of the relationship between GAD diagnosis and specific sleep difficulties. Thus the main purpose of the present study was to address this gap in the literature. First, we found that participants with GAD had significantly higher sleep dysfunction than healthy controls. Second, we found that emotion dysregulation significantly mediated the relationship between GAD diagnosis and a number of sleep outcomes. Our findings extend existing knowledge on specific sleep problems associated with GAD diagnosis and contribute to the literature by examining the relationship between emotion dysregulation and sleep problems in GAD patients.

Consistent with previous research (Saletu-Zyhlarz et al., 1997; Uhde, 2000), GAD participants reported more sleep problems than the healthy controls. Specifically, we found that compared to the controls, GAD patients reported a wide range of sleep disturbances, including higher daytime dysfunction, nightmare frequency, and teeth grinding, more excessive daytime sleepiness, harder time waking up in the morning, higher perceived need for more sleep, lower habitual sleep efficiency, reduced subjective sleep quality, and reduced sleep duration. Indeed, given a high co-occurrence of sleep problems and anxiety disorders, it has been suggested that sleep disturbance might be a bidirectional transdiagnostic process, where poor sleep and the symptoms of psychiatric disorders mutually maintain each other (e.g., Harvey, 2009). Specifically, some insomnia and anxiety researchers (e.g., Morin et al., 2007) have suggested that sleep disturbance in individuals with anxiety disorders is maintained by dysfunctional sleep-related beliefs (e.g., worry about consequences of poor sleep, unrealistic beliefs about increasing the likelihood of falling asleep, misconceptions about the causes of insomnia). These beliefs generate cognitive arousal, which causes emotional and physiological activation and consequently maintains anxiety and further decreases the likelihood of falling asleep. Our findings are also in line with previous investigations showing that GAD patients, compared to healthy controls, tend to experience less efficient sleep with increased wake periods throughout their total sleep time and decreased sleep duration (e.g.,

Saletu-Zyhlarz et al., 1997). Relatedly, a recent study found a strong association between daytime dysfunction attributed to poor sleep and anxiety disorders, particularly GAD (Ramsawh, Stein, Belik, Jacobi, & Sareen, 2009).

Consistent with the emotion dysregulation model of GAD (Mennin & Fresco, in press), participants in the GAD group reported significantly higher levels of emotion dysregulation compared to the controls. Importantly, these difficulties with emotion regulation fully mediated the relationship between GAD diagnosis and sleep disturbances, daytime dysfunction, excessive daytime sleepiness, and perceived need for more sleep than usual, suggesting that problems with emotion regulation possibly contribute to the presentation of specific sleep problems in GAD patients. These findings on the impact of disrupted sleep on both night- and daytime functioning also suggest that the relationship between emotion regulation and sleep is likely bidirectional, where the ways emotions get processed and regulated during the day might impact nighttime emotion regulation and therefore impact sleep quality. Indeed, dysfunctional emotion regulation during the day has been related to sleep disturbances, and these sleep disturbances might, in turn, impair emotion regulation that occurs the following day (e.g., Schmidt et al., 2011).

Interestingly, sleep duration, habitual sleep efficiency, subjective sleep quality, hard time waking up in the morning, nightmare frequency, and teeth grinding were not statistically mediated by DERS, suggesting that these sleep problems might be stronger associated with the effects of GAD diagnosis regardless of emotion dysregulation. Given that almost all of the sleep problems that were not significantly mediated by emotion dysregulation are associated with nighttime functioning, it is possible that these particular problems might be stronger related to the cognitive components of emotion regulation (e.g., thought suppression, worry, rumination) that poor sleepers experience before and during sleep that are not specifically targeted using a more global measure of emotion regulation skills such as the DERS. Indeed, dysfunctional forms of cognitive control have been shown to be associated with sleep disturbance and psychopathology (e.g., Schmidt et al., 2011). Alternatively, consistent with the view of sleep and emotion regulation as a bidirectional process (e.g., Schmidt et al., 2011), it is possible that by mediating daytime functioning, emotion regulation affects sleep indirectly. For example, given that individuals with GAD tend to experience difficulties managing their emotions (Mennin & Fresco, 2009), these difficulties in affect regulation during the day (e.g., inability to effectively downregulate negative emotions as

required by the context) may negatively impact their sleep during the night (e.g., Schmidt & Van der Linden, 2009).

The present study had several limitations. First, the cross-sectional design did not permit drawing conclusions about the causal pathways between the mediating and outcome variables. Thus, while the results suggest that difficulties with emotion regulation might be an important component of understanding how GAD is associated with a number of sleep problems, future research should assess GAD symptoms along with sleep and emotion regulation problems over time in order to further explore potential contributory mechanisms by which GAD patients develop and maintain sleep problems. Another limitation was the reliance on self-report instruments, which have been shown to be subject to a number of biases (Robinson & Clore, 2002). Future investigations should attempt to replicate our findings with more objective measures of sleep dysfunction, for example by recording all-night polysomnographic sleep to measure sleep initiation, the wake time after the sleep onset, and total sleep time along with biophysiological changes that occur during sleep (Monti & Monti, 2000). Additionally, the sample consisted of predominantly younger Caucasian females, which limits generalizability of our findings to a larger population of individuals suffering from GAD. Finally, although we controlled for the depression severity in all of the analyses, we did not have a pure MDD group in order to be able to truly tease apart the effects of MDD versus GAD.

Despite these limitations, our findings have important clinical implications. Research shows that cognitive-behavior therapy (CBT) for anxiety disorders has only a moderate effect on co-occurring sleep difficulties (Belleville et al., 2010), and CBT for insomnia has only a moderate effect on anxiety problems (Belleville, Cousineau, Levrier, & St-Pierre-Delorme, 2011). The results of the present study suggest that future research should further explore the impact of the interaction between various emotional and cognitive factors on sleep in anxiety disorders in order to improve their existing treatments and develop new intervention techniques. Indeed, there are potentially promising new insomnia interventions that have not yet been evaluated in those with insomnia and an anxiety disorder. For example, producing solutions to pre-sleep worries (i.e., engaging in constructive worry, or structured problem solving) in the early evening may decrease presleep cognitive arousal and thus decrease sleep latency (Carney & Waters, 2006). Additionally, an exercise (Francis & Pennebaker, 1992) that involves writing about emotional content has been shown to facilitate sleep among those who complained about excessive mental activity in bed (e.g., Harvey & Farrell, 2003). As an alternative to these more cognitive approaches, mindfulness-based practices, such as meditation to manage the emotional reactions to disturbed sleep and daytime fatigue, may also help in alleviating sleep problems and reducing overall arousal (e.g., Ong & Sholtes, 2010).

Additionally, it would be important for future research to elucidate specific emotion regulation strategies that poor sleepers who report high difficulties in emotion regulation in response to distress rely on as well as the ways these strategies interact and impact sleep and psychopathology. For example, although acceptance and reappraisal are considered uniformly adaptive emotion regulation strategies frequently utilized in a number of psychotherapeutic treatments, individuals with GAD might not necessarily benefit from implementing either of these strategies (Aldao & Mennin, 2012). Furthermore, it is important to consider the interaction between adaptive and maladaptive emotion regulation strategies in relation to the psychopathology symptoms (Aldao, in press). Adaptive strategies have been shown to have the highest (negative) association with psychopathology symptoms when maladaptive strategies are also highly utilized (Aldao & Nolen-Hoeksema, 2012).

5. Conclusions

Taken together, the present findings suggest that the difficulties with emotion regulation experienced by individuals with GAD mediate the relationship between symptoms of this disorder and a wide range of sleep disturbances, and these results are independent of the effects of depression and secondary anxiety diagnoses. Individuals' difficulties in emotion regulation might be dispositional characteristics that remain present even after successful treatment of GAD and might potentially account for the lasting presence of certain sleep difficulties in GAD patients. Clinicians who work with anxious patients can expect residual sleep problems that often persist even after successful treatment of GAD (Belleville et al., 2010) and, thus, should specifically target emotion dysregulation in GAD treatment in order to alleviate these lingering sleep problems. A similar approach might be used in relation to other anxiety disorders (e.g., post-traumatic stress disorder, panic disorder, obsessive compulsive disorder) due to the high prevalence of sleep problems in these disorders. Thus, future studies should examine whether emotion dysregulation also mediates the relationship between other types of symptoms and sleep disturbances.

Acknowledgment

The authors would like to thank Lucy Gatchel and Katie McLaughlin for their assistance in earlier stages of this project.

References

- Akiskal, H. S., Lemmi, H., Dickson, H., King, D., Yerevanian, B., & Van Valkenburg, C. (1984). Part 2 Sleep EEG differentiation of primary dysthymic disorders from anxious depressions. *Journal of Affective Disorders*, 6, 287–295. [http://dx.doi.org/10.1016/S0165-0327\(84\)80007-5](http://dx.doi.org/10.1016/S0165-0327(84)80007-5)
- Aldao, A. The future of emotion regulation research: capturing context. *Perspectives on Psychological Science*, <http://dx.doi.org/10.1177/1745691612459518>, in press.
- Aldao, A., & Mennin, D. S. (2012). Paradoxical cardiovascular effects of implementing adaptive emotion regulation strategies in generalized anxiety disorder. *Behaviour Research and Therapy*, 50, 122–130. <http://dx.doi.org/10.1016/j.brat.2011.12.004>
- Aldao, A., & Nolen-Hoeksema, S. (2012). When are adaptive strategies most predictive of psychopathology? *Journal of Abnormal Psychology*, 121, 276–281. <http://dx.doi.org/10.1037/a0023598>
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed, text revision). Washington, DC: Author. <http://dx.doi.org/10.1176/appi.books.9780890423349>
- Babson, K. A., Trainor, C. D., Feldner, M. T., & Blumenthal, H. A. (2010). Test of the effects of acute sleep deprivation on general and specific self-reported anxiety and depressive symptoms: an experimental extension. *Journal of Behavior Therapy and Experimental Psychiatry*, 41, 297–303. <http://dx.doi.org/10.1016/j.jbtep.2010.02.008>
- Belanger, L., Morin, C. M., Langlois, F., & Ladouceur, R. (2004). Insomnia and generalized anxiety disorder: effects of cognitive behavior therapy for GAD on insomnia symptoms. *Journal of Anxiety Disorders*, 18, 561–571. [http://dx.doi.org/10.1016/S0887-6185\(03\)00031-8](http://dx.doi.org/10.1016/S0887-6185(03)00031-8)
- Belleville, G., Cousineau, H., Levrier, K., St-Pierre-Delorme, M., & Marchand, A. (2010). The impact of cognitive-behavior therapy for anxiety disorders on concomitant sleep disturbances: a meta-analysis. *Journal of Anxiety Disorders*, 24, 379–386. <http://dx.doi.org/10.1016/j.janxdis.2010.02.010>
- Belleville, G., Cousineau, H., Levrier, K., & St-Pierre-Delorme, M. (2011). Meta-analytic review of the impact of cognitive-behavior therapy for insomnia on concomitant anxiety. *Clinical Psychology Review*, 31, 638–652. <http://dx.doi.org/10.1016/j.cpr.2011.02.004>
- Brenes, G. A., Miller, M. E., Stanley, M. A., Williamson, J. D., Knudson, M., & McCall, W. V. (2009). Insomnia in older adults with generalized anxiety disorder. *American Journal of Geriatric Psychiatry*, 17, 465–472. <http://dx.doi.org/10.1097/JGP.0b013e3181987747>
- Brissette, I., & Cohen, S. (2002). The contribution of individual differences in hostility to the associations between daily interpersonal conflict, affect, and sleep. *Personality and Social Psychology Bulletin*, 28, 1265–1274. <http://dx.doi.org/10.1177/01461672022812011>
- Buysse, D. J., Reynolds, C. F., III, Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Research*, 28, 193–213. [http://dx.doi.org/10.1016/0165-1781\(89\)90047-4](http://dx.doi.org/10.1016/0165-1781(89)90047-4)

- Cacioppo, J. T., Hawley, L. C., Berntson, G. G., Ernst, J. M., Gibbs, A. C., Stickgold, R., et al. (2002). Do lonely days invade the nights? Potential social modulation of sleep efficiency. *Psychological Science*, 13, 384–387. <http://dx.doi.org/10.1111/1467-9280.00469>
- Carney, C. E., & Waters, W. F. (2006). Effects of a structured problem-solving procedure on pre-sleep cognitive arousal in college students with insomnia. *Behavioral Sleep Medicine*, 4, 13–28. http://dx.doi.org/10.1207/s15402010bsm0401_2
- DiNardo, P. A., Brown, T. A., & Barlow, D. H. (1994). *Anxiety disorders interview schedule for DSM-IV*. Albany, NY: Graywind.
- Espie, C. A. (2002). Insomnia: conceptual issues in the development, persistence, and treatment of sleep disorder in adults. *Annual Review of Psychology*, 53, 215–243. <http://dx.doi.org/10.1146/annurev.psych.53.100901.135243>
- First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (2002). *Structured clinical interview for DSM-IV-TR Axis I disorders, research version, patient edition with psychotic screen*. New York: Biometrics Research, New York State Psychiatric Institute.
- Francis, M. E., & Pennebaker, J. W. (1992). Putting stress into words: the impact of writing on physiological, absentee, and self-reported emotional well-being measures. *American Journal of Health Promotion*, 6, 280–287. <http://dx.doi.org/10.4278/0890-1171-6.4.280>
- Fuller, K. H., Waters, W. F., Binks, P. G., & Anderson, T. (1997). Generalized anxiety and sleep architecture: a polysomnographic investigation. *Sleep*, 20, 370–376. Retrieved from: <http://www.journalsleep.org/ViewAbstract.aspx?pid=24300>
- Gratz, K. L., & Roemer, L. (2004). Multidimensional assessment of emotion regulation and dysregulation: development, factor structure, and initial validation of the difficulties in emotion regulation scale. *Journal of Psychopathology and Behavioral Assessment*, 26, 41–54. <http://dx.doi.org/10.1023/B:JOBA.0000007455.08539.94>
- Gross, J. (1998). The emerging field of emotion regulation: an integrative review. *Review of General Psychology*, 2, 271–299. <http://dx.doi.org/10.1037//1089-2680.2.3.271>
- Harvey, A. G. (2000). Pre-sleep cognitive activity: a comparison of sleep-onset insomniacs and good sleepers. *British Journal of Clinical Psychology*, 39, 275–286. <http://dx.doi.org/10.1348/014466500163284>
- Harvey, A. G. (2002). A cognitive model of insomnia. *Behaviour Research and Therapy*, 40, 869–893. [http://dx.doi.org/10.1016/S0005-7967\(01\)00061-4](http://dx.doi.org/10.1016/S0005-7967(01)00061-4)
- Harvey, A. G. (2009). A transdiagnostic approach to treating sleep disturbance in psychiatric disorders. *Cognitive Behaviour Therapy*, 38, 35–42. <http://dx.doi.org/10.1080/16506070903033825>
- Harvey, A. G., & Farrell, C. (2003). The efficacy of a Pennebaker-like writing intervention for poor sleepers. *Behavioral Sleep Medicine*, 1, 115–123. http://dx.doi.org/10.1207/S15402010BSM0102_4
- Johnson, S. L. (2005). Mania and dysregulation in goal pursuit: a review. *Clinical Psychology Review*, 25, 241–262. <http://dx.doi.org/10.1016/j.cpr.2004.11.002>
- Kahn-Greene, E. T., Killgore, D. B., Kamimori, G. H., Balkin, T. J., & Killgore, W. D. S. (2007). The effects of sleep deprivation on symptoms of psychopathology in healthy adults. *Sleep Medicine*, 8, 215–221. <http://dx.doi.org/10.1016/j.sleep.2006.08.007>
- Krakow, B., Schrader, R., Tandberg, D., Hollifield, M., Koss, M. P., Yau, L. C., et al. (2002). Nightmare frequency in sexual assault survivors with PTSD. *Journal of Anxiety Disorders*, 16, 175–190. [http://dx.doi.org/10.1016/S0887-6185\(02\)00093-2](http://dx.doi.org/10.1016/S0887-6185(02)00093-2)
- LeBlanc, M., Beaulieu-Bonneau, S., Mérette, C., Savard, J., Ivers, H., & Morin, C. M. (2007). Psychological and health-related quality of life factors associated with insomnia in a population-based sample. *Journal of Psychosomatic Research*, 63, 157–166. <http://dx.doi.org/10.1016/j.jpsychores.2007.03.004>
- Levin, R., & Nielsen, T. (2009). Nightmares, bad dreams, and emotion dysregulation: a review and new neurocognitive model of dreaming. *Current Directions in Psychological Science*, 18, 84–88. <http://dx.doi.org/10.1111/j.1467-8721.2009.01614.x>
- MacKinnon, D. P., Lockwood, C. M., & Williams, C. M. (2004). Confidence limits for the indirect effect: distribution of the product and resampling methods. *Multivariate Behavioral Research*, 39, 99–128. http://dx.doi.org/10.1207/s15327906mbr3901_4
- Marcks, B. A., Weisberg, R. B., Edelen, M. O., & Keller, M. B. (2010). The relationship between sleep disturbance and the course of anxiety disorders in primary care patients. *Psychiatry Research*, 178, 487–492. <http://dx.doi.org/10.1016/j.psychres.2009.07.004>
- McCrae, C. S., McNamara, J. P., Rowe, M. A., Dzierzewski, J. M., Dirk, J., Marsiske, M., et al. (2008). Sleep and affect in older adults: using multilevel modeling to examine daily associations. *Journal of Sleep Research*, 17, 42–53. http://dx.doi.org/10.1207/s15327906mbr3901_4
- Mennin, D. S., & Fresco, D. M. (2009). Emotion regulation as an integrative framework for understanding and treating psychopathology. In: A. M. Kring, & D. M. Sloan (Eds.), *Emotion regulation in psychopathology: a transdiagnostic approach to etiology and treatment* (pp. 356–379). New York: Guilford.
- Mennin, D. S., & Fresco, D. M. Emotion regulation therapy. In: J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed.). New York: Guilford Press, in press.
- Mennin, D. S., Holaway, R., Fresco, D. M., Moore, M. T., & Heimberg, R. G. (2007). Delineating components of emotion and its dysregulation in anxiety and mood psychopathology. *Behaviour Therapy*, 38, 284–302. <http://dx.doi.org/10.1016/j.beth.2006.09.001>
- Monti, J. M., & Monti, D. (2000). Sleep disturbance in generalized anxiety disorder and its treatment. *Sleep Medicine Reviews*, 4, 263–276. <http://dx.doi.org/10.1053/smr.1999.0096>
- Morin, C. M., Vallières, A., & Ivers, H. (2007). Dysfunctional beliefs and attitudes about sleep (DBAS). Validation of a brief version (DBAS-16). *Sleep: Journal of Sleep and Sleep Disorders Research*, 30, 1547–1554. Retrieved from: <http://www.journalsleep.org/ViewAbstract.aspx?pid=26994>
- Ohayon, M. M. (2002). Epidemiology of insomnia: what we know and what we still need to learn. *Sleep Medicine Reviews*, 6, 97–111. <http://dx.doi.org/10.1053/smr.2002.0186>
- Ohayon, M. M. (1997). Prevalence of DSM-IV diagnostic criteria of insomnia: distinguishing insomnia related to mental disorders from sleep disorders. *Journal of Psychiatric Research*, 31, 333–346. [http://dx.doi.org/10.1016/S0022-3956\(97\)00002-2](http://dx.doi.org/10.1016/S0022-3956(97)00002-2)
- Ohayon, M. M., Morselli, P. L., & Guilleminault, C. (1997). Prevalence of nightmares and their relationship to psychopathology and daytime functioning in insomnia subjects. *Sleep*, 20, 340–348. Retrieved from: <http://www.journalsleep.org/ViewAbstract.aspx?pid=24307>
- Ong, J., & Sholtes, D. (2010). A mindfulness-based approach to the treatment of insomnia. *Journal of Clinical Psychology*, 66, 1175–1184. <http://dx.doi.org/10.1002/jclp.20736>
- Papadimitriou, G. N., Kerkhofs, M., Kempnaers, C., & Mendlewicz, J. (1988). EEG sleep studies in patients with generalized anxiety disorder. *Psychiatry Research*, 26, 183–190.
- Papadimitriou, G. N., & Linkowski, P. (2005). Sleep disturbance in anxiety disorders. *International Review of Psychiatry*, 17, 229–236. <http://dx.doi.org/10.1080/09540260500104524>
- Preacher, K. J., & Hayes, A. F. (2008). Asymptotic and resampling strategies for assessing and comparing indirect effects in simple and multiple mediator models. *Behavior Research Methods, Instruments, and Computers*, 40, 879–891. Retrieved from: <http://www.springerlink.com/content/6xp1676n382p53k1/>
- Ramsawh, H. J., Stein, M. B., Belik, S. L., Jacobi, F., & Sareen, J. (2009). Relationship of anxiety disorders, sleep quality, and functional impairment in a community sample. *Journal of Psychiatric Research*, 43, 926–933. <http://dx.doi.org/10.1016/j.jpsychores.2009.01.009>
- Robinson, M. D., & Clore, G. L. (2002). Belief and feeling: evidence for the accessibility model of emotional self-report. *Psychological Bulletin*, 6, 934–960. <http://dx.doi.org/10.1037//0033-2909.128.6.934>
- Rose, M., Manser, T., & Ware, C. J. (2008). Effects of call on sleep and mood in internal medicine residents. *Behavioral Sleep Medicine*, 6, 75–88. <http://dx.doi.org/10.1080/15402000801952914>
- Roth, T., Jaeger, S., Jin, R., Kalsekar, A., Stang, P. E., & Kessler, R. C. (2006). Sleep problems, comorbid mental disorders, and role functioning in the national comorbidity survey replication. *Biological Psychiatry*, 60, 1364–1371. <http://dx.doi.org/10.1016/j.biopsych.2006.05.039>
- Saletu-Zylharz, G., Saletu, B., Anserer, P., Brandstätter, N., Frey, R., Gruber, G., et al. (1997). Nonorganic insomnia in generalized anxiety disorder 1. Controlled studies on sleep, awakening and daytime vigilance utilizing polysomnography and EEG mapping. *Neuropsychobiology*, 36, 117–129. <http://dx.doi.org/10.1159/00019373>
- Salteras-Pedneault, K., Roemer, L., Tull, M. T., Rucker, L., & Mennin, D. S. (2006). Evidence of broad deficits in emotion regulation associated with chronic worry and generalized anxiety disorder. *Cognitive Therapy and Research*, 30, 469–480. <http://dx.doi.org/10.1007/s10608-006-9055-4>
- Schmidt, R. E., Harvey, A. G., & Van der Linden, M. (2011). Cognitive and affective control in insomnia. *Frontiers in Psychology*, 349, 1–12. <http://dx.doi.org/10.3389/fpsyg.2011.00349>
- Schmidt, R. E., & Van der Linden, M. (2009). The aftermath of rash action: sleep interfering counterfactual thoughts and emotions. *Emotion*, 9, 549–553. <http://dx.doi.org/10.1037/a0015856>
- Talbot, L. S., Hariston, I. S., Eidelman, P., Gruber, J., & Harvey, A. G. (2009). The effect of mood on sleep onset latency and REM sleep in interepisode bipolar disorder. *Journal of Abnormal Psychology*, 118, 448–458. <http://dx.doi.org/10.1037/a0016605>
- Uhde, T. W. (2000). Anxiety disorders. In: M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (pp. 1123–1139). Philadelphia: W.B. Saunders Company.
- Watson, D. (2005). Rethinking the mood and anxiety disorders: a quantitative hierarchical model for DSM-V. *Journal of Abnormal Psychology*, 114, 522–536. <http://dx.doi.org/10.1037/0021-843X.114.4.522>
- Wetherell, J. L., Le Roux, H., & Gatz, M. (2003). DSM-IV criteria for generalized anxiety disorder in older adults: distinguishing the worried from the well. *Psychology and Aging*, 18, 622–627. <http://dx.doi.org/10.1037/0882-7974.18.3.622>
- Zadra, A., & Donderi, D. C. (2000). Nightmares and bad dreams: their prevalence and relationship to well-being. *Journal of Abnormal Psychology*, 109, 273–281. <http://dx.doi.org/10.1037/0021-843X.109.2.273>
- Zohar, D., Tzischinsky, O., Epstein, R., & Lavie, P. (2005). The effects of sleep loss on medical residents' emotional reactions to work events: a cognitive-energy model. *Sleep*, 28, 47–54. Retrieved from: <http://www.journalsleep.org/ViewAbstract.aspx?pid=26094>