HHS Public Access

Author manuscript

J Mood Anxiety Disord. Author manuscript; available in PMC 2024 December 20.

Published in final edited form as:

J Mood Anxiety Disord. 2024 December ; 8: . doi:10.1016/j.xjmad.2024.100072.

Examining the relationship between emotion regulation, sleep quality, and anxiety disorder diagnosis☆

Bryana L. Schantz[a], **Emma R. Toner**[b], **Mackenzie L. Brown**[c], **Nikki Kaiser**[a], **Alan Chen**[d], **Samrachana Adhikari**[d], **Susanne S. Hoeppner**[e], **Eric Bui**[f], **Naomi M. Simon**[a], **Kristin L. Szuhany**[a,*]

[a]Department of Psychiatry, New York University Grossman School of Medicine, 1 Park Ave, New York, NY, United States

[b]Department of Psychology, University of Virginia, 409 McCormick Rd., Charlottesville, VA, United States

[c]Department of Psychological and Brain Sciences, University of Louisville, 317 Life Sciences Building, Louisville, KY, United States

[d]Department of Population Health, New York University Grossman School of Medicine, 180 Madison Ave., New York, NY, United States

[e]Department of Psychiatry, Massachusetts General Hospital, 55 Fruit Street, Boston, MA, United States

[f]Department of Psychiatry, Caen University & Caen University Hospital, Rue Professeur Edouard Zarifian Ave du 14000, Caen, France

Abstract

Anxiety disorders are highly comorbid with sleep disturbance and have also been associated with deficits in emotion regulation, the ability to control and express emotions. However, the extent to which specific dimensions of sleep disturbance and emotion regulation are associated with anxiety diagnosis is not well-explored. This study examined dimensions of emotion regulation and sleep disturbance that may predict greater likelihood of anxiety diagnosis using novel machine learning techniques. Participants (Mean(SD) age= 28.6(11.3) years, 62.7% female) with primary anxiety disorders (n = 257), including generalized anxiety disorder (n = 122) and social anxiety disorder (n = 135), and healthy controls (n = 89) completed the Difficulties in Emotion Regulation Scale and Pittsburgh Sleep Quality Index. A conditional inference tree was fit to classify likelihood of current anxiety diagnosis based on predictors. The best model fit included 4 split nodes and 5 terminal nodes. Worse scores on two emotion regulation subscales, strategies directed to manage

☆ Members of the Editorial Board are authors of this article. Editorial Board members are not involved in decisions about papers which they have written themselves or have been written by family members or colleagues or which relate to products or services in which the editor has an interest. Any such submission is subject to all of the journal's usual procedures, with peer review handled independently of the relevant editor and their research groups.

*Correspondence to: 1 Park Ave, 8th Floor, New York, NY 10016, United States. Kristin.szuhany@nyulangone.org (K.L. Szuhany).

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.xjmad.2024.100072.

negative emotions and nonacceptance of negative emotions, were the best predictors of current anxiety diagnosis (99.3% probability of diagnosis). For those with better emotion regulation, poor sleep quality and worse daytime functioning due to sleep were important predictors of anxiety diagnosis. Good emotion regulation and non-disturbed sleep predicted high likelihood of being a non-psychiatric control (88.2%). Limitations include cross-sectional design precluding designating directionality of effects of sleep and emotion regulation on anxiety onset; limited sample size; and self-reported sleep. Facets of emotion regulation and sleep disturbance may be important early targets for brief intervention for anxiety disorders.

Keywords

Emotion regulation; Sleep; Anxiety disorders; Machine learning

1. Introduction

Anxiety disorders are common and affect approximately one-third of the US adult population in their lifetimes [1]. Anxiety disorders may co-occur with several health problems, including sleep disturbances [2, 3]. Sleep disturbances are highly prevalent with approximately one in three adults diagnosed with clinical insomnia. Clinical insomnia is defined as difficulty initiating or maintaining sleep, early morning awakening, or poor sleep quality for at least three days per week for three months [4]. Insomnia occurs in 24.9–45.5% of individuals with an anxiety disorder. Specifically, approximately 30.4% and 32.4% of individuals with generalized anxiety disorder (GAD) and social anxiety disorder (SAD), respectively, have comorbid insomnia [5]. Insomnia may cause significant additional impairment in functioning and may also be predictive of future anxiety onset [3,4,6]. The relationship between anxiety and insomnia is likely bidirectional wherein they contribute to the development and are a consequence of each other [7]. Indeed, a recent systematic review found that 60% of included studies supported bidirectionality of anxiety and insomnia and both studies on sleep quality supported this hypothesis [7]. Uniquely, anxiety may precede and predict excessive daytime sleepiness [7].

Meta-analysis suggests that sleep problems in individuals with anxiety disorders are wide-ranging. They include subjective sleep disturbance, and decreased total sleep time, sleep continuity, and sleep depth compared to healthy controls [8]. In this meta-analysis, patients with GAD, which includes sleep disturbance as a diagnostic criterion, demonstrated significant deficits specifically in subjective sleep disturbance and sleep continuity [8]. On the common sleep measure, the Pittsburgh Sleep Quality Index (PSQI), patients with GAD report significantly worse sleep disturbance, habitual sleep efficiency, sleep duration, subjective sleep quality, and daytime dysfunction due to sleep than healthy controls [9]. In patients with GAD in remission, sleep disturbance may persist. For example, compared to healthy controls, patients with GAD reported poorer subjective sleep quality, longer sleep onset latency, and more use of sleep medications on the PSQI [10]. Fewer studies have examined sleep for patients with SAD, precluding ability to examine individual effects in the aforementioned meta-analysis [8]. However, individual studies suggest similar subjective sleep disturbance in SAD[11].

In addition to sleep disturbance, emotion regulation may also be disrupted in those with anxiety disorders [12–14]. Emotion regulation is defined as an individual's ability to influence the quality, duration, and intensity of both positive and negative emotions, and their ability to control, express and experience emotions [15], Several facets of emotion regulation have been shown to be disrupted across anxiety disorders. These include use of maladaptive regulation strategies, such as avoidance and emotion suppression, as well as lack of engagement in adaptive regulation strategies, such as problem solving and cognitive reappraisal [16]. Further, maladaptive cognitive emotion regulation strategies, such as catastrophizing and self-blame, are associated with higher levels of trait anxiety and insomnia [17].

Poor use of various emotion regulation strategies is evident in individual anxiety disorders as well. For example, in a meta-analysis of 193 patients with SAD, cognitive reappraisal was used less frequently and effectively and suppression was used more frequently by patients with SAD than controls [12]. Other individual studies support challenges in cognitive reappraisal in patients with SAD [18,19], including imaging studies demonstrating dysfunction in cortical networks responsible for cognitive control [19]. For patients with GAD, similar dysfunction in cortical circuits related to emotion dysregulation were identified in a systematic review of 10 studies [14].

The aforementioned studies and other empirical and conceptual research indicate that emotion regulation is a multidimensional construct [15,20–22]. A common theoretical model of emotion regulation is that of Gratz and Roemer [21]. They suggest six distinct theoretical dimensions of emotion regulation: 1) nonacceptance of emotional responses (e.g., feeling upset or ashamed for experiencing an emotion), 2) difficulties engaging in goal-directed behavior, 3) impulse control difficulties, 4) lack of emotional awareness, 5) limited access to emotion regulation strategies (e.g., cognitive reappraisal), and 6) lack of emotional clarity (e.g., confusion about how one is feeling). This model corresponds to the commonly used and well-validated measure of emotion regulation, the Difficulties in Emotion Regulation Scale (DERS). In a study of 67 patients with SAD, deficits across all DERS subscales, except emotional awareness, were evident compared to healthy controls [23]. In another study of an undergraduate sample with SAD, GAD, or both, impairments in managing negative emotions through emotion regulation strategies (*DERS strategies*) was impaired in all groups compared to healthy controls [24]. Patients with GAD and those with comorbid GAD and SAD demonstrated deficits in accepting negative emotions (*DERS nonacceptance*) and describing emotions (*DERS clarity*) [24].

Identifying which specific components of sleep dysfunction and emotion regulation are disrupted in those with anxiety disorders may be useful to optimize intervention targets. Although the associations between anxiety disorders and both sleep disturbance and emotion dysregulation are well-established, few studies have examined all three constructs simultaneously. In one such study of patients with GAD, deficits in emotion regulation (total score on the DERS) fully mediated several sleep outcomes on the PSQI, including daytime dysfunction, sleep disturbances, excessive daytime sleepiness, and perceived need for more sleep than usual [9]. In a sample of undergraduates, *DERS nonacceptance* was associated poorer sleep in those with higher anxiety symptoms than lower anxiety symptoms [25].

As emotion regulation, sleep disruption, and anxiety are inter-related concepts, it is important to understand their complex relationship. Given that emotion regulation and insomnia are multi-dimensional constructs, it is important to examine specific components associated with anxiety disorder diagnosis to optimize targets for intervention. Therefore, we examined classification of current anxiety disorder diagnosis based on distinct dimensions of emotion regulation and sleep disturbances using machine learning, a novel approach which offers the ability for high validity prediction. Based on previous research, we hypothesized that lack of emotional clarity, lack of emotional awareness, and limited access to emotion regulation strategies as well as poorer subjective sleep quality and sleep duration would be significantly associated with anxiety disorder diagnostic status [8–10,12,24,25].

2. Material and methods

2.1. Participants

A total of 346 participants, with either a primary anxiety disorder (generalized anxiety disorder [GAD] or social anxiety disorder [SAD]) or healthy controls consented to a single time point questionnaire-based protocol after detailed explanation of study procedures. Participants completed questionnaires as part of an ancillary questionnaire-based protocol prior to participating other research studies at Massachusetts General Hospital from 2008 to 2016. All eligible participants were over 18 years old and were excluded if they were pregnant, had a severe unstable medical illness, alcohol/substance abuse within the past six months, history of schizophrenia, psychotic disorders, bipolar disorder, or mental disorder due to medical condition or substance, or were at serious acute suicidal risk. Healthy control participants had no DSM-IV diagnosis within the past six months. All procedures were approved by the Institutional Review Board.

2.2. Measures

2.2.1. Diagnostic screening—Trained doctoral-level clinicians administered either the Mini International Neuropsychiatric Interview [MINI; 26] or Structured Clinical Interview for DSM-IV [SCID-IV; 27] to assess primary, comorbid, and exclusionary diagnoses. Demographic information was also collected. Study clinicians were trained on structured interview measures and participated in weekly review of assigned diagnoses with expert clinicians, supervisors, and the study team.

2.2.2. Pittsburgh sleep quality index (PSQI) [28]—The PSQI is a 24-item self-report scale which assesses subjective sleep disturbance and quality during the past month. The PSQI has seven subscales: *sleep quality, sleep latency, sleep duration, sleep efficiency, sleep disturbance, sleep medication*, and *daytime dysfunction*. PSQI subscales range from 0 to 3, with higher scores indicating poorer sleep. Previous research suggests ability to discriminate between patients with anxiety disorders and healthy controls on PSQI subscales [9,10,24]. Internal reliability was acceptable in this sample (Cronbach's α = .77).

2.2.3. Difficulties in emotional regulation scale (DERS) [21]—The DERS is a 36 item self-report derived from the clinical model of emotion regulation. Items are rated on a Likert scale from 1 (almost never) to 5 (almost always) with instructions

to indicate how often the statements apply. Six subscales assess dimensions of emotion regulation, with higher scores indicating more pronounced emotion dysregulation. Previous research suggests ability to differentiate patients with anxiety disorders (including SAD and GAD) from controls on DERS subscales [23,24]. The *nonacceptance* subscale (range: 6–30; Cronbach's α = .94) measures an individual's acceptance (or lack thereof) of their negative emotions (e.g., "*When I'm upset, I feel guilty for feeling that way*"). The *goals* subscale (range: 5–25; Cronbach's α = .92) assesses the ability to direct behavior toward accomplishing goals when experiencing difficult emotions. The *impulse* subscale (range: 6–30; Cronbach's α = .87) assesses the ability to suppress impulsive behavior in the face of negative emotions. The *awareness* subscale (range: 6–30; Cronbach's α = .83) measures awareness and insight into an individual's emotional state. The *clarity* subscale (range: 5–25; Cronbach's α = .84) assesses the ability to understand emotions. The *strategies* subscale (range: 8–40; Cronbach's α = .93) measures the ability to engage in appropriate strategies to effectively moderate negative emotional responses.

2.3. Data analysis

Demographic characteristics for healthy control and anxiety disorder groups were compared using independent samples t-tests and chi-square for continuous and categorical variables.

Only participants with complete data for both DERS and PSQI were included in analyses. Missing values for categorical demographic predictors were coded as a separate category to retain sample size; this simple technique has been shown to be effective for outcome prediction compared to other approaches, including producing lower mean square prediction error [29]. A conditional inference tree was fit to identify categories of DERS and PSQI that best classify the likelihood of anxiety disorder diagnosis (using Partykit package 1.2–6 [30]). The covariates included were the DERS and PSQI subscale scores as well as demographic variables (age, sex, race, education level, income level). Conditional inference trees are a type of decision tree that recursively partition or split the outcome variable based on strength of association with predictors as measured with significant p-values using a permutation test. Compared to standard decision trees, conditional inference trees are less prone to overfitting and selection bias towards predictors with many possible splits, while still generating an interpretable model and heuristics that can be applied for prediction. The conditional inference trees control for biases due to multiple testing in a tree with several splits (nodes) by applying a Bonferroni correction to fix the overall significance level of the test. Therefore, the hyperparameters tuned during the model training were the overall significance level of the permutation test, whether statistical tests are adjusted using Bonferroni correction, and the minimum number of data points in the terminal (final) nodes. Hyperparameter optimization was conducted through grid search, and conditional inference tree models were validated in a 10-fold cross-validation process. Average cross-validated area under the curve for receiver operating curve (AUC) was used to select the hyperparameters for the best model, which was then fit on the full sample to determine splitting rules. All analyses were conducted in R version 4.1.1.

3. Results

3.1. Sample characteristics

The 346 participants (Mean age(SD)= 28.6(11.3) years, 62.7% female) included 122 with a primary diagnosis of GAD (Mean age(SD)=28.4(10.4) years, 73.0% female), 135 with a primary diagnosis of SAD (Mean age(SD)= 27.6(10.4) years, 52.6% female) and 89 non-psychiatric, healthy controls (Mean age(SD)= 30.2(12.4) years, 64.0% female). Demographic characteristics did not significantly differ across control and anxiety disorder groups, with exception of race ($p = .007$; Table 1).

3.2. Conditional inference tree model

Conditional inference trees were trained and validated in a 10-fold cross-validation process. The average cross-validated AUC across out-of-sample predictions was 0.541 (SD=0.124, min=0.286, max= 0.677, 95%CI = (0.482,0.598). While the tree model's average AUC is slightly better than random chance, which corresponds to an AUC of 0.5, the lower limit of the 95%CI is close to 0.5, suggesting only minimal improvement. The hyperparameters were an alpha significance level of 0.01, Bonferroni corrected p-values, and a minimum of 30 data points in each terminal node. The ROC curve of the model fitted on the full data using hyperparameters selected from cross-validation is presented in Fig. 1.

This best model was then fit to the full sample to examine the data splitting heuristics. The model produced a tree with 4 split nodes and 5 terminal nodes (Fig. 2). Worse scores on DERS subscales of *strategies* and *nonacceptance* were the most important predictors of anxiety disorder diagnosis, whereas better scores on PSQI subscales (*sleep quality*, *daytime dysfunction*) were the most important predictors for healthy controls. Demographic variables were not selected as important predictors. The most important predictor overall was DERS *strategies* score, splitting the sample by scores > 12 vs. ≤ 12. Higher DERS *strategies* scores were further split by DERS *nonacceptance*, with 13 being the splitting value. Higher DERS *strategies* (>12), meaning worse perceived access to or ability to implement emotion regulation strategies, and higher *nonacceptance* scores (>13), meaning less ability to accept the presence of negative emotions, almost completely predicted anxiety diagnosis (99.3%).

For lower DERS *strategies* scores (≤12; i.e., better ability to implement strategies to manage negative emotions), worse PSQI *sleep quality* and *daytime dysfunction* scores were the most important predictors of anxiety diagnosis. *Sleep quality* scores of 0 (no impairment in *sleep quality*) were mostly healthy controls (88.2%).

4. Discussion

Using the novel method of conditional inference tree analysis, this study identified facets of emotion regulation and sleep disturbance that predicted the probability of having a current anxiety disorder based on well-validated measures. Few previous studies have examined the interconnection between specific facets of sleep disturbance and emotion regulation and their associations with anxiety, with even fewer utilizing advanced machine learning techniques. Conditional inference tree techniques provide an easily interpretable set of heuristics that can be used in clinical or research settings. When replicated across various

samples, they can be considered for implementation. In our case, with replication across other outpatient settings with expanded data, this type of conditional inference tree may be useful to predict anxiety disorder diagnosis based on disrupted facets of emotion regulation and sleep.

Aligned with hypotheses, two specific subscales related to emotion regulation were the most valid factors associated with anxiety disorder diagnosis: poor strategies to manage negative emotions and less ability to accept presence of negative emotions. The most important factor associated with anxiety diagnosis was poor strategies to manage negative emotions. Approximately 94% of individuals with higher scores on this scale held an anxiety diagnosis. Those with higher scores on this *strategies* subscale and with less ability to accept the presence of negative emotions (*nonacceptance*) were almost certain to have an anxiety diagnosis (99.3%), regardless of sleep quality. These results align with one known previous study using machine learning techniques (network analysis) to identify relative influence of emotion regulation components on the DERS, daytime sleepiness, and anxiety and depression during the COVID-19 pandemic [31]. They identified the DERS *strategies* subscale to be most influential in terms of interconnections with other symptoms. Results also align with previous studies suggesting deficits in individuals with GAD and SAD in *strategies* as well as *nonacceptance* [23–25]. Contrary to our hypotheses and previous studies, we did not find DERS *clarity* to be an important additional factor predicting current anxiety disorder diagnosis. However, this may be due to the stronger connection between anxiety disorder diagnosis and deficits in emotion regulation strategies, such as cognitive reappraisal, as suggested by other studies [23–25].

Results align with theoretical models of emotion dysregulation in anxiety disorders, such as the process model of emotion regulation [15] and the Acceptance-based Model [32]. The process model highlights the strategies used to manage emotions, whether adaptive or maladaptive. One such strategy is cognitive reappraisal, a cognitive strategy that aims to reinterpret negative thoughts related to anxiety to be more adaptive and modify their emotional impact [15]. Presence of anxiety-related negative thoughts, particularly related to threat appraisal, may be clinically targeted by cognitive behavioral therapy (CBT) for anxiety disorders and are suggested to be a core mechanism of change [33]. The Acceptance-based Model suggests a strong role of negative response to and avoidance of uncomfortable or threatening emotions or physical states [32]. Acceptance and Commitment Therapy (ACT) as well as mindfulness-based therapies, such as Mindfulness-Based Stress Reduction and Mindfulness-Based Cognitive Therapy, have demonstrated efficacy for anxiety disorders and may be useful clinical interventions to target this dimension of emotion regulation [34].

For those in our sample with better emotion regulation abilities, *sleep quality* and *daytime dysfunction* related to sleepiness (e.g., not having enough enthusiasm to get things done; trouble staying awake during activities) emerged as important predictors of anxiety disorder diagnosis versus healthy control. Those with better emotion regulation and no sleep difficulties were more likely to be healthy controls (88.2%). However, those with any reported interference in *sleep quality* (>0) and any *daytime dysfunction* (>0) had a higher likelihood of having an anxiety disorder diagnosis (59.5%), despite better emotion regulation

abilities. The association of poor sleep quality with anxiety disorder diagnosis aligns with our hypothesis and a meta-analysis suggesting large effects on subjective sleep quality (g=2.16) in those with anxiety disorders. We did not originally hypothesize a connection between anxiety disorder diagnosis and daytime dysfunction; however, previous individual studies have identified these associations [35,36]. Our results align with previous findings in patients with GAD demonstrating significant impairment in multiple dimensions of sleep, including sleep quality and daytime dysfunction [8–10]. However, we did not find an association between sleep duration and anxiety disorder diagnostic status as hypothesized. Given parameters of the conditional inference tree for three levels of influence, results appear to indicate higher relative importance of sleep quality and daytime dysfunction in the association with anxiety disorder diagnosis.

The relationship between sleep disturbance and anxiety is likely bidirectional as sleep disturbance, especially when chronic, may increase likelihood of anxiety disorder onset [6,7]. However, presence of an anxiety disorder may also impact sleep [e.g., interference due to nighttime worrying; 8, 37] and leave individuals more vulnerable to emotion dysregulation. Of note, although sleep disturbance is included in the diagnostic criteria of the Diagnostic and Statistical Manual of Mental Disorders [DSM-5-TR; American Psychiatric 38] for GAD as a potential symptom, but not for SAD, both anxiety disorders were associated with sleep disruption in this sample.

Though factors related to emotion dysregulation were most associated with anxiety disorders in our sample, poor sleep quality and daytime dysfunction due to sleep may benefit from supplementary targeted intervention. Consistent with this, a recent meta-analysis reported that improving sleep quality can lead to significant improvements in mental health overall (g=−0.53) and in anxiety [g=−0.51; 39]. Clinically, there are brief, efficacious interventions that can target sleep problems, including poor sleep quality and daytime dysfunction due to sleep. One first-line recommendation for treating insomnia is cognitive behavioral therapy for insomnia (CBT-I), a well-established, efficacious intervention that targets both short-term and long-term sleep problems. It has strong demonstrated efficacy for improving sleep quality [40–42].

This study is not without limitations. First, the cross-sectional nature of the data precludes ability to designate causality in regard to the temporal relationship of sleep and emotion regulation dimensions and onset of anxiety disorder diagnosis. Second, the anxiety population was limited to GAD and SAD. Although research to date indicates links between GAD and SAD with sleep difficulties and emotion regulation [9, 43], future research may expand upon these findings with the full spectrum of anxiety disorders (e.g., panic disorder, agoraphobia). Due to collection of data across multiple ongoing studies for this ancillary self-report database, inter-rater reliability on MINI or SCID-rated diagnoses could not be formally calculated. However, as noted previously, all clinicians reviewed diagnostic entry criteria with supervisors and the study team weekly. Additionally, we did not have information available about comorbid sleep disorders (e.g., obstructive sleep apnea) or concomitant medications which may affect aspects of sleep. For our conditional inference tree, sample size was relatively small, limiting generalizability more broadly to anxiety disorders. Given average AUC (0.54) at slightly better than chance, replicating the analysis

with larger sample sizes and extended predictors would promote broader generalizability and validity. Further, the relatively low proportion of control patients may have biased the model assessment; however oversampling and undersampling techniques to create balanced training data may add further bias by shifting the outcome distribution of the data and potentially introducing bias in our approach of basing decision rules for the conditional inference tree on Bonferroni adjusted p-values [44–46]. Future studies should consider cost sensitive decision trees [47] and consider exhaustive assessment of model performance for varying prediction thresholds [48] to encourage better prediction of minority class. Lastly, given that our analyses relied on participant self-report of emotion regulation and sleep, future research should consider adding a behavioral measurement of emotion regulation and a physiological measurement of sleep quality (e.g., polysomnography) to compare effects when using objective versus self-report measures of these constructs.

5. Conclusions

Challenges with specific dimensions of emotion regulation, including implementing strategies to cope with negative emotions and difficulty accepting of negative emotions, were highly associated with anxiety disorder diagnostic status. For those with better emotion regulation abilities, sleep disturbances (i.e., poorer sleep quality, worse daytime functioning due to sleep) were moderately associated with anxiety disorder diagnostic status. These results suggest that specific aspects of emotion regulation and sleep disturbance are strongly associated with anxiety disorders. Longitudinal examination of the temporal relationship between these facets of emotion regulation and sleep disturbance and onset of anxiety disorders may help determine if they are useful early intervention targets.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Role of the Funding Source

This work was supported by the Highland Street Foundation. Dr. Szuhany's time was supported by the National Institute of Mental Health (NIMH: K23MH122773) and by a NARSAD Young Investigator Grant from the Brain & Behavior Research Foundation. The funding sources did not have any role in the study design; the collection, analysis, and interpretation of data; in the writing of the report; and in the decision to submit the article for publication.

Declaration of Competing Interest

Dr. Hoeppner reported receiving grants from NIH, the American Cancer Society, the Executive Committee on Research at MGH, Koa Health Inc, and National Institute on Drug Abuse outside the submitted work.

Dr. Bui reported receiving grants from the NIH during the conduct of the study and receiving consulting fees from Cerevel Therapeutics LLC, royalties from Springer and from Wolters Kluwyer, and grants from the US Department of Defense, Osher Center for Integrative Medicine, Patient-Centered Outcomes Research Institute, and Elizabeth Dole Foundation outside the submitted work.

Dr. Simon reports receiving grants from the National Institutes of Health (NIH), US Department of Defense, American Foundation for Suicide Prevention, Patient-Centered Outcomes Research Institute, and support from Cohen Veterans Network; receiving personal fees from Vanda Pharmaceuticals, Praxis Therapeutics, Genomind, Wiley, Bionomics Limited, BehavR LLC, Cerevel, and Engrail Therapeutics Inc; performing grant reviews for American Foundation for Suicide Prevention; receiving royalties for UpToDate contributions from Wolters Kluwyer and APA Publishing; and having spousal stock from G1 Therapeutics and Zentalis outside the submitted work.

Dr. Szuhany reports receiving grants from the National Institutes of Health (NIH) and Brain & Behavior Research Foundation and acting as an Associate Editor for Mental Health and Physical Activity outside of this work.

All other authors report no conflicts of interest.

References

[1]. Kessler RC, et al. Insomnia and the performance of US workers: results from the America insomnia survey. Sleep 2011;34(9):1161–71. [PubMed: 21886353]

[2]. Aernout E, et al. International study of the prevalence and factors associated with insomnia in the general population. Sleep Med 2021;82:186–92. [PubMed: 33957414]

[3]. Soehner AM, Harvey AG. Prevalence and functional consequences of severe insomnia symptoms in mood and anxiety disorders: results from a nationally representative sample. Sleep 2012;35(10):1367–75. [PubMed: 23024435]

[4]. Seow LSE, et al. Evaluating DSM-5 insomnia disorder and the treatment of sleep problems in a psychiatric population. J Clin Sleep Med 2018;14(2):237–44. [PubMed: 29394962]

[5]. Johnson EO, Roth T, Breslau N. The association of insomnia with anxiety disorders and depression: exploration of the direction of risk. J Psychiatr Res 2006;40(8): 700–8. [PubMed: 16978649]

[6]. Hertenstein E, et al. Insomnia as a predictor of mental disorders: a systematic review and meta-analysis. Sleep Med Rev 2019;43:96–105. [PubMed: 30537570]

[7]. Alvaro PK, Roberts RM, Harris JK. A systematic review assessing bidirectionality between sleep disturbances, anxiety, and depression. Sleep 2013;36(7):1059–68. [PubMed: 23814343]

[8]. Cox RC, Olatunji BO. Sleep in the anxiety-related disorders: a meta-analysis of subjective and objective research. Sleep Med Rev 2020;51:101282. [PubMed: 32109832]

[9]. Tsypes A, Aldao A, Mennin DS. Emotion dysregulation and sleep difficulties in generalized anxiety disorder. J Anxiety Disord 2013;27(2):197–203. [PubMed: 23474909]

[10]. Hacimusalar Y, Karaaslan O. Assessment of sleep quality of patients with panic disorder and generalized anxiety disorder during remission: a case-control study. Arch Clin Psychiatry 2020;47:19–24.

[11]. Raffray T, Bond TL, Pelissolo A. Correlates of insomnia in patients with social phobia: role of depression and anxiety. Psychiatry Res 2011;189(2):315–7. [PubMed: 21463903]

[12]. Kivity Y, Huppert JD. Emotion regulation in social anxiety: a systematic investigation and meta-analysis using self-report, subjective, and event-related potentials measures. Cogn Emot 2019;33(2):213–30. [PubMed: 29514588]

[13]. Mennin DS, et al. Preliminary evidence for an emotion dysregulation model of generalized anxiety disorder. Behav Res Ther 2005;43(10):1281–310. [PubMed: 16086981]

[14]. Mochcovitch MD, et al. A systematic review of fMRI studies in generalized anxiety disorder: evaluating its neural and cognitive basis. J Affect Disord 2014;167: 336–42. [PubMed: 25020268]

[15]. Gross JJ. The emerging field of emotion regulation: an integrative review. Rev Gen Psychol 1998;2(3):271–99.

[16]. Aldao A, Nolen-Hoeksema S, Schweizer S. Emotion-regulation strategies across psychopathology: a meta-analytic review. Clin Psychol Rev 2010;30(2):217–37. [PubMed: 20015584]

[17]. Zagaria A, et al. Differential associations of cognitive emotion regulation strategies with depression, anxiety, and insomnia in adolescence and early adulthood. Int J Environ Res Public Health 2023;20(10).

[18]. Dryman MT, Heimberg RG. Emotion regulation in social anxiety and depression: a systematic review of expressive suppression and cognitive reappraisal. Clin Psychol Rev 2018;65:17–42. [PubMed: 30064053]

[19]. Picó-Pérez M, et al. Emotion regulation in mood and anxiety disorders: a meta-analysis of fMRI cognitive reappraisal studies. Prog Neuropsychopharmacol Biol Psychiatry 2017;79(Pt B)):96–104. [PubMed: 28579400]

[20]. Cole PM, Michel MK, Teti LO. The development of emotion regulation and dysregulation: a clinical perspective. Monogr Soc Res Child Dev 1994;59(2–3): 73–100. [PubMed: 7984169]

[21]. Gratz KL, Roemer L. Multidimensional assessment of emotion regulation and dysregulation: development, factor structure, and initial validation of the difficulties in emotion regulation scale. J Psychopathol Behav Assess 2004;26(1): 41–54.

[22]. Thompson RA, Lewis MD, Calkins SD. Reassessing emotion regulation. Child Dev Perspect 2008;2(3):124–31.

[23]. Helbig-Lang S, Rusch S, Lincoln TM. Emotion regulation difficulties in social anxiety disorder and their specific contributions to anxious responding. J Clin Psychol 2015;71(3):241–9. [PubMed: 25327520]

[24]. Mennin DS, McLaughlin KA, Flanagan TJ. Emotion regulation deficits in generalized anxiety disorder, social anxiety disorder, and their co-occurrence. J Anxiety Disord 2009;23(7):866–71. [PubMed: 19464142]

[25]. Kirwan M, Pickett SM, Jarrett NL. Emotion regulation as a moderator between anxiety symptoms and insomnia symptom severity. Psychiatry Res 2017;254:40–7. [PubMed: 28448803]

[26]. Sheehan DV, et al. The Mini-International Neuropsychiatric Interview (M.I.N.I.): the development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. J Clin Psychiatry 1998;59(Suppl 20):22–33. quiz 34–57.

[27]. First MD, et al. Structured clinical interview for DSM-IV Axis I disorders, clinician version (SCID-CV). Washington, DC: American Psychiatric Press,; 1996.

[28]. Buysse DJ, et al. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. Psychiatry Res 1989;28(2):193–213. [PubMed: 2748771]

[29]. Manapat D Missing Data in Conditional Inference Trees. Arizona State University,; 2023.

[30]. Hothorn T, Hornik K, Zeileis A. Unbiased recursive partitioning: a conditional inference framework. J Comput Graph Stat 2006;15(3):651–74.

[31]. Sardella A, et al. Anxiety, depression, emotion regulation, and daytime sleepiness: are there links between these factors? network analysis on an italian sample during the Covid-19 pandemic. Clin Neuropsychiatry 2023;20(5):395–404. [PubMed: 38089736]

[32]. Roemer L, Orsillo SM. Expanding our conceptualization of and treatment for generalized anxiety disorder: integrating mindfulness/acceptance-based approaches with existing cognitive-behavioral models. Clin Psychol: Sci Pract 2002;9(1):54.

[33]. Powers MB, de Kleine RA, Smits JAJ. Core mechanisms of cognitive behavioral therapy for anxiety and depression: a review. Psychiatr Clin North Am 2017;40(4): 611–23. [PubMed: 29080589]

[34]. Haller H, et al. A systematic review and meta-analysis of acceptance- and mindfulness-based interventions for DSM-5 anxiety disorders. Sci Rep 2021;11(1): 20385. [PubMed: 34650179]

[35]. Potvin O, et al. Subjective sleep characteristics associated with anxiety and depression in older adults: a population-based study. Int J Geriatr Psychiatry 2014; 29(12):1262–70. [PubMed: 24733621]

[36]. Ramsawh HJ, et al. Relationship of anxiety disorders, sleep quality, and functional impairment in a community sample. J Psychiatr Res 2009;43(10):926–33. [PubMed: 19269650]

[37]. Oh CM, et al. The effect of anxiety and depression on sleep quality of individuals with high risk for insomnia: a population-based study. Front Neurol 2019;10:849. [PubMed: 31456736]

[38]. Association, A.P., Diagnostic and statistical manual of mental disorders. 5th edition, text revision ed. 2022.

[39]. Scott AJ, et al. Improving sleep quality leads to better mental health: a meta-analysis of randomised controlled trials. Sleep Med Rev 2021;60:101556. [PubMed: 34607184]

[40]. Riemann D, Perlis ML. The treatments of chronic insomnia: a review of benzodiazepine receptor agonists and psychological and behavioral therapies. Sleep Med Rev 2009;13(3):205–14. [PubMed: 19201632]

[41]. Trauer JM, et al. Cognitive behavioral therapy for chronic insomnia. SM, & Cunnington, D Ann Intern Med 2015;163(3):191–204.

[42]. van der Zweerde T, et al. Cognitive behavioral therapy for insomnia: a meta-analysis of long-term effects in controlled studies. Sleep Med Rev 2019;48:101208. [PubMed: 31491656]

[43]. Klumpp H, et al. Subjective and objective sleep quality modulate emotion regulatory brain function in anxiety and depression. Depress Anxiety 2017;34(7): 651–60. [PubMed: 28419607]

[44]. Elreedy D, Atiya AF, Kamalov F. A theoretical distribution analysis of synthetic minority oversampling technique (SMOTE) for imbalanced learning. Mach Learn 2023:1–21. [PubMed: 37363047]

[45]. Krawczyk B, Wózniak M, Schaefer G. Cost-sensitive decision tree ensembles for effective imbalanced classification. Appl Softw Comput 2014;14:554–62.

[46]. Blagus R, Lusa L. SMOTE for high-dimensional class-imbalanced data. BMC Bioinforma 2013;14(1):16.

[47]. Siers MJ, Islam MZ. Class imbalance and cost-sensitive decision trees: a unified survey based on a core similarity. ACM Trans Knowl Discov Data 2020;15(1):1–31.

[48]. Adhikari S, et al. Revisiting performance metrics for prediction with rare outcomes. Stat Methods Med Res 2021;30(10):2352–66. [PubMed: 34468239]

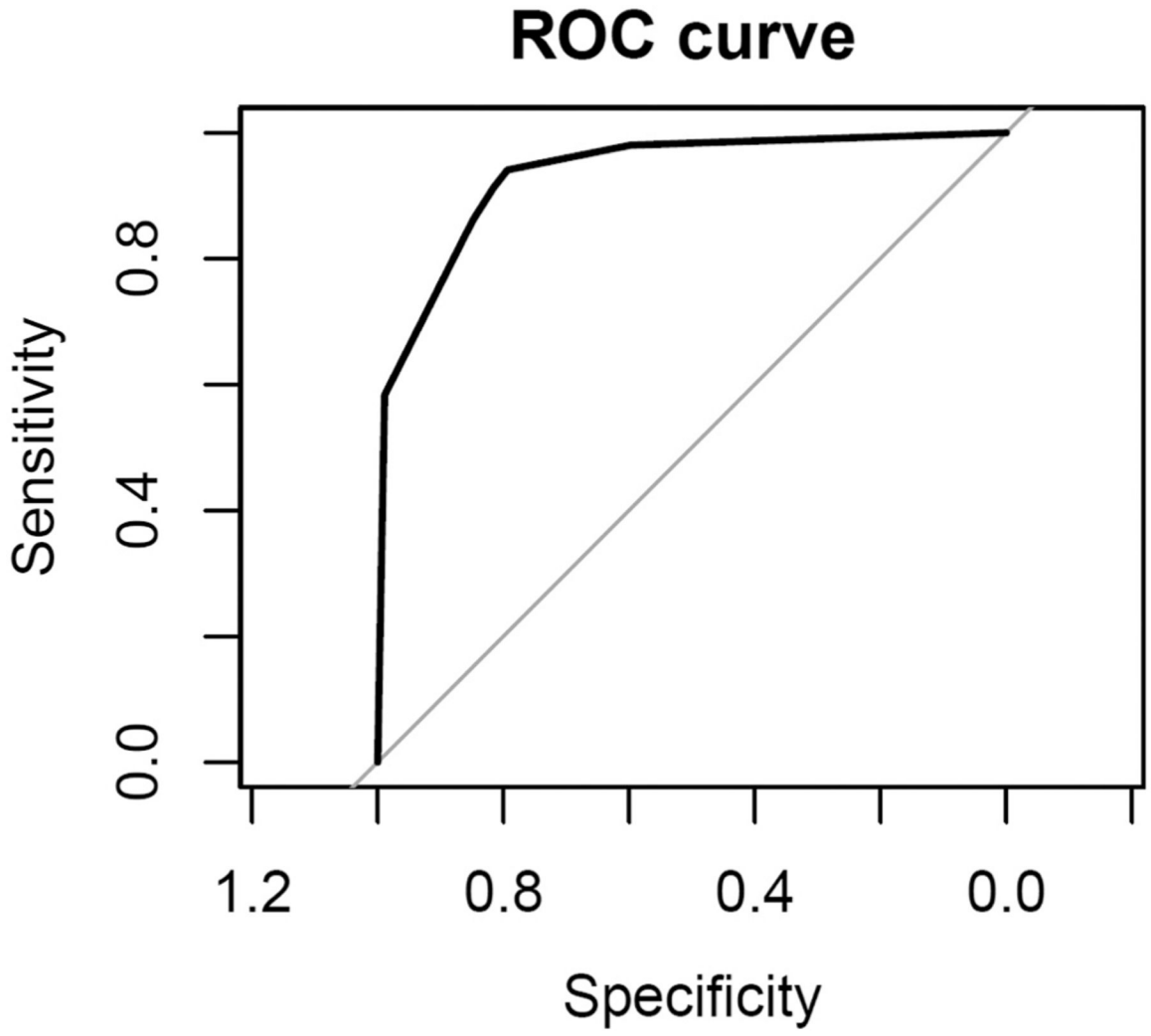

Fig. 1.
Receiver operating characteristic (ROC) for the full sample using conditional inference tree hyperparameters found through cross-validation.

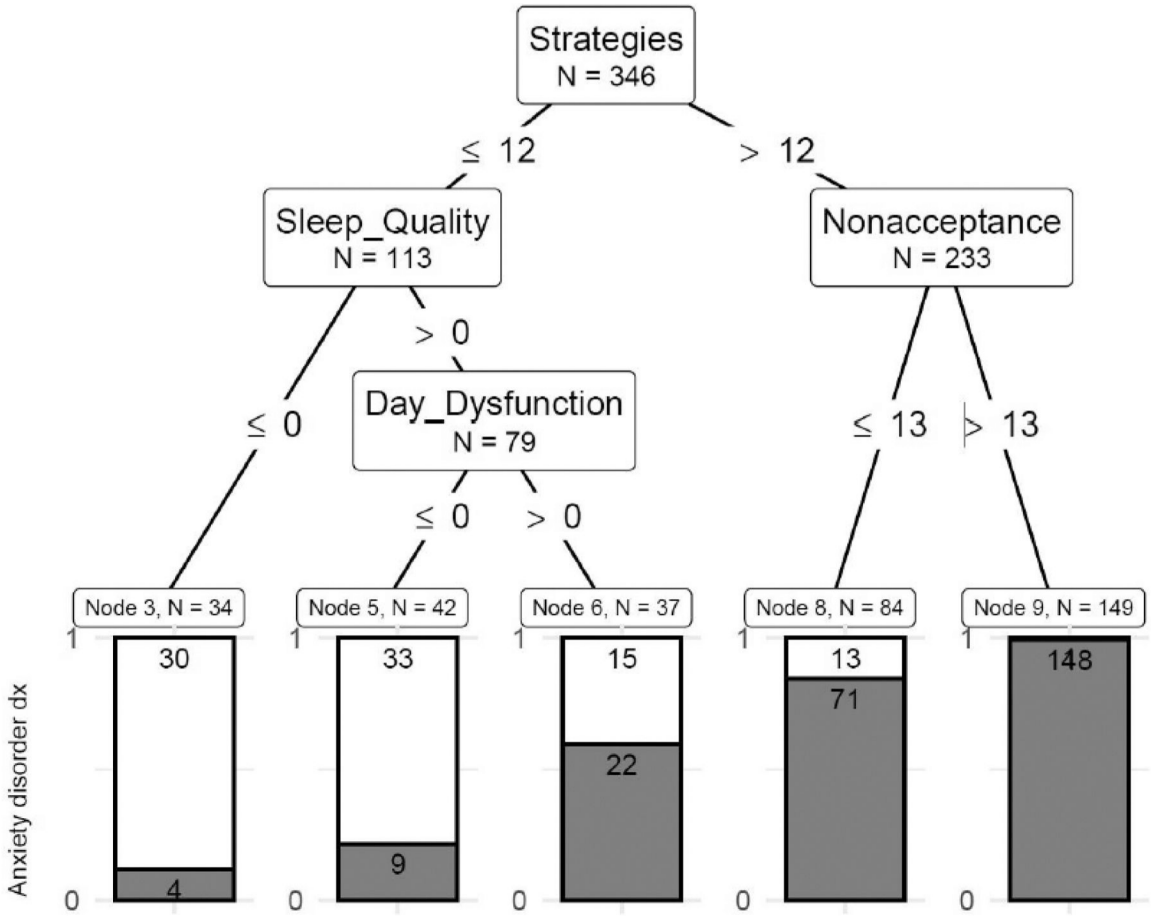

Fig. 2.

Conditional inference model predicting likelihood of current anxiety disorder based on facets of emotion regulation and sleep disturbance. Description: Worse scores on Difficulties in Emotion Regulation (DERS) subscales of strategies and nonacceptance highly predict anxiety disorder diagnosis (gray) in a Conditional Inference Tree. Better scores on DERS strategies as well as better sleep quality and no daytime dysfunction due to sleep predict likelihood of being a healthy control (white).

Table 1

Demographic and clinical characteristics.

Characteristics	Total (*n* = 346)	Anxiety Disorder (SAD and GAD) (*n* = 257)	Healthy Controls (*n* = 89)	p-value
Mean (*SD*) age in years	28.6 (11.3)	28.0 (10.8)	30.2 (12.4)	p = .12
Sex (%, *n*)				
Female	62.7 (217)	62.3 (160)	64.0 (57)	p = .82
Male	32.7 (113)	31.9 (82)	34.8 (31)	
Missing	4.6 (16)	5.8 (15)	1.1 (1)	
Primary Diagnosis				
SAD (%, *n*)	39.0 (135)	52.5 (135)	N/A	N/A
GAD (%, *n*)	35.3 (122)	47.5 (122)	N/A	
Healthy Controls (%, *n*)	25.7 (89)	N/A	100 (92)	
Current Comorbid Diagnoses (%, *n*) *	N/A		N/A	N/A
SAD (only comorbid for GAD primary sample)		21.4 (55)		
GAD (only including SAD primary sample)		15.2 (39)		
Panic Disorder		8.2 (21)		
Agoraphobia		6.2 (16)		
OCD		3.1 (8)		
Specific Phobia		21.8 (56)		
MDD		10.9 (28)		
Ethnicity (%, *n*)				
Non-Hispanic	83.5 (289)	82.5 (212)	86.5 (77)	p = .91
Hispanic	11.6 (40)	11.3 (29)	12.4 (11)	
Missing	4.9 (17)	6.2 (16)	1.1 (1)	
Race (%, *n*)				
White	66.5 (230)	70.0 (180)	56.2 (50)	p = .007
Asian	12.7 (44)	10.5 (27)	19.1 (17)	
African American	6.4 (22)	4.7 (12)	11.2 (10)	
Native American	0.6 (2)	0 (0)	2.2 (2)	
Other	9.0 (31)	8.9 (23)	9.0 (8)	
Missing	4.9 (17)	5.8 (15)	2.2 (2)	
Education (%, *n*)				
Graduate School	28.6 (99)	27.6 (71)	31.5 (28)	p = .74
College Graduate	35.5 (123)	37.0 (95)	31.5 (28)	
Partial College	24.3 (84)	22.6 (58)	29.1 (26)	
High School Graduate	5.8 (20)	5.8 (15)	5.6 (5)	
Partial High School	0.9 (3)	0.8 (2)	1.1 (1)	
Missing	4.9 (17)	6.2 (16)	1.1 (1)	
Mean (*SD*) DERS total score	83.0 (25.7)	85.4 (25.7)	75.9 (24.8)	N/A
Mean (*SD*) global PSQI	6.0 (3.6)	6.2 (3.4)	5.3 (4.0)	N/A

Note.

[*] n = 12–16 missing current comorbid diagnoses (% out of total participants in subset); SAD = social anxiety disorder; GAD = generalized anxiety disorder; OCD = obsessive compulsive disorder; MDD = major depressive disorder; SD = standard deviation; DERS = Difficulties in Emotion Regulation Scale; PSQI = Pittsburgh Sleep Quality Index