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PRACTICE ARTICLE

Cognitive behavioural therapy for anxiety disorders and insomnia: a commentary on future directions

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Abstract. Anxiety disorders and insomnia significantly impair daily functioning. Similar underlying mechanisms may account for the high comorbidity of both disorders, and respective treatments share pharmacological and behavioural features. This review suggests the utility of an integrated CBT approach in the treatment of generalized anxiety, post-traumatic stress, and panic disorders, and comorbid insomnia. Other anxiety disorders were not explored because current data are limited or inconsistent. A comprehensive, non-systematic review of the literature was conducted to evaluate the treatment of comorbid anxiety and insomnia disorders, and data reveal shared pharmacological and behavioural features of insomnia and anxiety disorders treatment. However, research demonstrates that CBT maintains successful treatment results longer than drug therapies. Despite similar treatment approaches, there is a paucity of research that explores integrated CBT approaches for comorbid anxiety and insomnia disorders. This review suggests that future research should assess the impact of combined therapeutic approaches on the simultaneous reduction of anxiety disorders, insomnia, and relapse rates.

Key words: Anxiety disorders, cognitive behavioural therapy, combined treatment, insomnia.

Introduction

> Care keeps his watch in every old man's eye, and where care lodges, sleep will never lie. (William Shakespeare, *Romeo and Juliet*)

Anxiety disorders are the most prevalent mental health problem in the United States and account for more than one third of mental healthcare expenditures (Kessler *et al.* 2005; ADAA, 2012). Left untreated, anxiety disorders may impair daily functioning, increase national healthcare costs, and jeopardize quality of life (Dupont *et al.* 1996; Olatunji *et al.* 2007). Likewise, insomnia increases utilization of healthcare costs, decreases work productivity, and may impair daily functioning (e.g. fatigue, cognition) to the same extent as chronic medical conditions such as congestive heart failure (Simon & Vonkorff, 1997;

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Hajak, 2001; Katz & McHorney, 2002; Baglioni *et al.* 2010). Insomnia exacerbates anxiety disorder symptoms and is a significant risk factor in the development of anxiety and other mental health disorders (Breslau *et al.* 1996; Labbate *et al.* 1997, 1998; Gregory *et al.* 2005; Ramsawh *et al.* 2009). Further, the comorbidity of anxiety disorders (i.e. generalized anxiety, post-traumatic stress, and panic disorders) and insomnia is common likely due to shared cognitive and behavioural mechanisms. Therefore, sleep-focused treatment in conjunction with traditional psychotherapeutic treatment approaches for anxiety disorders may ameliorate comorbid conditions and optimize treatment efficacy. One specific therapeutic approach that has demonstrated effectiveness in the treatment of anxiety disorders and insomnia is cognitive behavioural therapy (CBT).

CBT has overwhelming success for the treatment of anxiety disorders and is recommended as the preliminary and foundational treatment for insomnia (Smith & Perlis, 2006; Schutte-Rodin *et al.* 2008; APA, 2010). Although research also supports similar drug treatments for anxiety disorders and insomnia, such as SSRIs, SNRIs, and benzodiazepines (Mellman, 2006; Schutte-Rodin *et al.* 2008; ADAA, 2012; Baldwin *et al.* 2013), many clinicians are not adequately trained to provide evidence-based pharmacological treatment for anxiety (Baldwin *et al.* 2013). Further, the American Academy of Sleep Medicine (AASM) recommends that short-term drug intervention be used conservatively and be supplemented by CBT (Schutte-Rodin *et al.* 2008). Pharmacological therapy is associated with high costs, daytime sleepiness, poor sleep latency and continuity, amnesia, behavioural disinhibition, drug tolerance, cognitive impairment, anxiety and insomnia rebound, and unknown or harmful long-term outcomes (Morin *et al.* 2006; Kierlin, 2008; Argyropoulos *et al.* 2008; Carney & Edinger, 2010; Baldwin *et al.* 2013). By contrast, CBT sustains improvements over a longer period of time relative to pharmacological intervention and is preferred by patients (Vincent & Lionberg, 2001; Craske *et al.* 2005; Morin *et al.* 2006; Swift *et al.* 2012). Considering therapeutic and financial benefits of CBT, this review focuses on the relationship between anxiety disorders and insomnia and the utility of treating the disorders using CBT.

First, we discuss previously observed connections between insomnia and generalized anxiety disorder (GAD), post-traumatic stress disorder (PTSD), and panic disorder (PD), because of the salience of insomnia in each disorder. Other anxiety disorders have been excluded because of scarce or inconsistent data related to insomnia, including obsessive-compulsive disorder, social anxiety disorder, specific phobias, and illness anxiety disorder (Clark *et al.* 1995; Smith *et al.* 2005; Buckner *et al.* 2008; Marcks & Weisberg, 2009). Other disorders may share aetiological underpinnings, but research has not conclusively supported a relationship between the disorders and insomnia. Second, we summarize the efficacy of CBT and suggest an integrated approach in the treatment of anxiety disorders and insomnia. Last, we discuss the implications of existent research on the treatment of relevant disorders and provide suggestions for future research.

Anxiety disorders and insomnia

Anxiety disorders are the most prevalent type of psychiatric condition that occurs within individuals primarily affected by insomnia (Ford & Kamerow, 1989; Clark *et al.* 1995; Buckner *et al.* 2008). Researchers emphasize a comprehensive approach to studying insomnia, but it has been regarded previously as a symptom secondary to anxiety disorders (Singh *et al.* 2006). Although the literature suggests that insomnia is a risk factor in the development

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of anxiety disorders and that both disorders share a feedback relationship, few prospective studies have examined the development and trajectory of anxiety and sleep difficulties more generally (Ford & Kamerow, 1989; Breslau *et al.* 1996; Jansson-Frojmark & Lindblom, 2008; Fava *et al.* 2009). Some research suggests that sleep difficulties precede anxiety disorder expression, such as PTSD (Babson & Feldner, 2010). Other prospective research, however, suggests that anxiety is a strong predictor of insomnia (Jansson & Linton, 2006). Drawing a distinction between sleep difficulties and insomnia in relation to anxiety disorders may be irrelevant due to the reciprocal relationships that exist. Directionality aside, several anxiety disorders share strong associations with insomnia and respective sleep difficulties, specifically GAD, PTSD, and PD.

The literature suggests that hypervigilance, impaired arousal regulation (e.g. hypothalamic-pituitary-adrenal axis, circadian rhythm), and other biopsychosocial factors influence the development and maintenance of GAD, PTSD, PD, and insomnia (Abelson *et al.* 2007; Fava *et al.* 2009; Lewis *et al.* 2009; Zambotti *et al.* 2011). Additional impairments relative to specific anxiety disorders have also been explored. Specifically, GAD patients display diminished sleep initiation, sleep maintenance, and stages 3 and 4 non-REM sleep (Reynolds *et al.* 1983; Papadimitriou *et al.* 1988). In addition, emotional memory processing deficits may mediate the relationship between nightmare occurrence and the development of PTSD. Finally, some PD patients experience panic attacks during hypoarousal states, and 25–33% experience nocturnal panic attacks (Mellman & Uhde, 1989; Wells, 1990; APA, 2013). Similar biopsychosocial features may allow for single integrated approaches for the treatment of anxiety disorders and insomnia. Such approaches, most notably CBT, may improve maladaptive cognitions and behaviours relative to each disorder.

CBT for anxiety disorders

Over 325 studies have established the efficacy of CBT, which decreases safety behaviours and utilizes exposure, attention focus modification, and cognitive restructuring to gradually expose patients to increasingly feared stimuli and allow habituation and extinction to occur (Marom & Hermesh, 2003; Butler *et al.* 2006). An effect size of 0.64–0.70 has been observed for the efficacy of CBT for GAD, and CBT sustains treatment effects for at least 2 years (Barlow *et al.* 1992; Gould *et al.* 2004; Haby *et al.* 2006). CBT targets GAD by addressing intolerance of uncertainty and cognitive avoidance through exposure and relaxation techniques (Covin *et al.* 2008). CBT may also be the most effective treatment for PTSD through the use of cognitive restructuring and exposure therapy (Frueh *et al.* 2004; Monson *et al.* 2005). A meta-analytical review by Bradley and colleagues (2005) demonstrated an effect size of 1.01–1.65 of CBT for PTSD. Nearly half of PTSD patients treated with CBT no longer meet diagnostic criteria for the disorder, including those that do not complete treatment (Bradley *et al.* 2005). Finally, CBT has been used to successfully treat PD through the use of psychoeducation, cognitive restructuring, and exposure techniques (Freedman & Adessky, 2009). Research demonstrates a CBT effect size range of 0.57–0.68 in the treatment of PD, and CBT effectively treats daytime and nocturnal panic (Craske & Tsao, 2005; Haby *et al.* 2006). Overall, CBT is a well-established, empirically supported treatment for anxiety disorders (Marom & Hermesh, 2003).

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CBT for insomnia

In addition to anxiety disorders, CBT is also an effective treatment for insomnia (CBT-I), and patients prefer CBT-I relative to drug treatment (Vincent & Lionberg, 2001). A meta-analysis conducted by Okajima and colleagues (2011) reveals that CBT-I is associated with significant improvements in several sleep domains, including sleep onset latency, total sleep time, wake after sleep onset, and sleep efficiency 12 months after treatment (effect sizes of 0.45, 0.42, 0.47, and 0.54, respectively). CBT-I is a multi-component therapy comprised of several core interventions recommended by AASM's practice parameters. CBT-I includes sleep restriction, stimulus control, cognitive restructuring, and sleep hygiene education (Perlis *et al.* 2005; Morin *et al.* 2006). During sleep restriction therapy, insomnia patients attempt to lie in bed only for an amount of time equal to their average sleep time (Perlis *et al.* 2005; Kierlin, 2008). Although counterintuitive, sleep restriction increases sleep exigency, decreases sleep latency, affords better sleep quality by inducing mild distress (Perlis *et al.* 2005), and may ameliorate sleep-related performance anxiety (Kierlin, 2008). Stimulus control therapy, a principal treatment, significantly reduces insomnia (Perlis *et al.* 2005; Morin *et al.* 2006) through sleep environment modification, which alleviates negative associations with the sleep environment (Kierlin, 2008). Modifications include lying in bed only when sleepy, avoiding behaviours in the bedroom other than sleep or sexual activities, and leaving the bedroom if awake after 15 minutes (Perlis *et al.* 2005; Kierlin, 2008; Swift *et al.* 2012). Cognitive-based treatments generally include patients' education of the cognitive model of insomnia and addressing maladaptive thoughts about the effects of insomnia on health in an attempt to ameliorate sleep-related anxiety (Perlis *et al.* 2005; Archer *et al.* 2009; Edinger *et al.* 2009). Finally, sleep hygiene educates patients with empirically sound instructions to improve sleep quality, quantity, and knowledge (Perlis *et al.* 2005).

CBT-I is an effective treatment, but research demonstrates that other practice parameters endorsed by the AASM may also be used to tailor treatment on a case-by-case basis (Morin & Espie, 2004; Morgenthaler *et al.* 2006). Such treatments include paradoxical intention, relaxation training, biofeedback, multi-component therapy without CBT, traditional CBT, and sleep hygiene therapy (Morgenthaler *et al.* 2006). Additional research suggests phototherapy, sleep compression, and other alternative experimental treatments (e.g. neurofeedback; Morin & Espie, 2004). Auxiliary treatment components or alternative delivery methods (e.g. internet-based CBT-I) may be beneficial to specific sleep problems or patients' characteristics (Perlis *et al.* 2005; Morin *et al.* 2006; Vincent & Lewycky, 2009). Yet, little is known about the unique contribution of individual components *vs.* the efficacy of combined psychotherapeutic approaches in the treatment of insomnia (Perlis *et al.* 2005; Morgenthaler *et al.* 2006; Morin *et al.* 2006). Further, there is a paucity of research exploring the efficacy of treatment relative to patient population, disorder, or comorbid conditions.

Integrated CBT approaches

CBT is used to treat both anxiety disorders and insomnia. Similar, but separate, cognitive behavioural models conceptualize the disorders and the treatment approaches underlying CBT. Specifically, the cognitive model of anxiety identifies maladaptive negative cognitions (i.e. automatic thoughts) as the central component of the model (Beck, 1995). The negative automatic thoughts likely stem from negative core beliefs about the self, the world, and

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others (Beck, 1995). In response to environmental triggers (i.e. perceived threat cues), negative automatic thoughts result in an increase in negative emotional and physiological responses, and dysfunctional behaviours (Beck, 1995). The increase in negative emotional and physiological responses may serve to reinforce dysfunctional behaviours through the narrowing of attention to threat cues and the promotion of avoidance behaviours, which may also reinforce the negative automatic thought (Beck, 1995). Harvey's (2002) cognitive model of insomnia greatly overlaps with the cognitive model of anxiety (Beck *et al.* 1985). Negative automatic thoughts are central to both approaches (Beck, 1995; Harvey, 2002; Mellman, 2006) and one may even suggest that the only true distinction between the models is the context of sleep difficulties as the threat cues or environmental trigger. The negative automatic thoughts conceptualized in the cognitive model of insomnia are sleep specific; however, anxiety and worry are the predominant negative emotional responses, which are experienced as increased physiological arousal (Harvey, 2002), which is the assertion of the cognitive model of anxiety. Additionally, the negative automatic thoughts about sleep and negative emotional and physiological responses may promote dysfunctional behaviours (i.e. sleep incompatible behaviours; Harvey, 2002) much the same way that dysfunctional behaviours may develop in the cognitive model of anxiety. To the best of our knowledge there has been no examination of an integrated CBT model for comorbid anxiety disorders and insomnia or a model for an integrated CBT approach. However, it may not be necessary to create a new CBT model that incorporates both anxiety- and sleep-related content given the significant conceptual overlap. The comorbid disorders share a theoretical background and similar cognitive behavioural treatment processes are used in the treatment of both (i.e. addressing maladaptive automatic thoughts). Therefore, an integrated CBT approach may be successful in treating both conditions simultaneously by specifically addressing both issues within the same treatment (i.e. addressing both anxiety- and sleep-related content). The integrated approach is especially important given the research evidence to suggest that addressing only one disorder when there is a comorbid anxiety and insomnia presentation is not effective for the treatment of both disorders.

Although no formal integrated treatment approach for comorbid anxiety and insomnia disorders exists, auxiliary treatments have been tailored to address sleep-related difficulties in specific anxiety disorders. Data suggest that imagery rehearsal therapy, consciously and repetitively change the disturbing outcome of a dream, reduces the frequency of nightmares and distress in PTSD patients beyond 2 years (Krakow *et al.* 2001). Another auxiliary treatment approach that decreases nightmares and subjective distress is exposure, relaxation, and rescripting therapy, during which patients recall recurring nightmares and engage in relaxation exercises and sleep hygiene education similar to CBT (Davis & Wright, 2007). Moreover, prazosin, a catecholamine inhibitor, diminishes recurring nightmares in PTSD patients (Raskind *et al.* 2003, 2007). In relation to PD, therapy focused on breathing, relaxation, and abrupt awakening by alarm may be particularly useful in the treatment of nocturnal panic patients (Craske & Tsao, 2005). To our knowledge, no auxiliary treatment targeting GAD-specific sleep difficulties exists. However, participants show improvements in sleep following completion of CBT and applied relaxation therapy for GAD (Monti & Monti, 2000; Belanger *et al.* 2004). Some CBT treatment for anxiety disorders and insomnia overlap, but other components may be specific to each disorder. Yet, little research has thoroughly examined integrated behavioural interventions for comorbid insomnia and specific anxiety disorders (Mellman, 2006).

Conclusions and future directions

Anxiety disorders and insomnia often co-occur; however, current treatments may not adequately address both conditions simultaneously. Therefore, clinician education about adequate treatment may prevent the exacerbation and reduce the severity of anxiety disorders and insomnia, increase efficacy and patients' accessibility to behaviour therapies (Gunter & Whittal, 2010), and aid in relapse prevention (Mellman, 2006). CBT alone is significantly more effective long-term relative to medication or combined treatments, is more affordable to patients, and results in lower healthcare and economic costs due to increased psychosocial functioning, quality of life, productivity, and proper healthcare utilization (Koerner *et al.* 2004; Morin *et al.* 2006; Kierlin, 2008; Swift *et al.* 2012). Further, there is evidence to suggest that CBT-I may be efficacious in reducing insomnia symptoms for patients with comorbid insomnia and other psychiatric conditions; however, CBT-I does not adequately address the comorbid psychiatric conditions (Edinger *et al.* 2009). Similarly, anxiety-focused CBT may not adequately address comorbid insomnia or sleep difficulties either. Specifically, sleep difficulties persist despite attenuated PTSD symptoms following CBT for trauma survivors (Galovski *et al.* 2009; Schoenfeld *et al.* 2012; Margolies *et al.* 2013). Other research indicates that insomnia persists in GAD patients despite successful short-term CBT for GAD (Bush *et al.* 2012). Therefore, integrated treatments that address both disorders simultaneously are necessary because the implementation of the core components of CBT is not enough. The treatment effects do not generalize from anxiety-specific treatment to insomnia-specific content and vice versa. Future research is needed to understand specific treatment effects or strategies that may generalize across conditions. Further, research examining the persistence of sleep difficulties, not just insomnia, despite successful anxiety disorder treatment is necessary and could provide insight into anxiety disorder relapse. This line of research may also highlight important cognitive processes necessary for successful treatment that are disrupted by ongoing sleep difficulties.

A determinant of anxiety disorders treatment success may be through the reliance that cognitive processes, such as learning and memory, have on normal sleep processes. Learning and memory play an integral role in the development and extinction of anxiety disorders (Barlow, 2000) and anxiety-focused CBT is a treatment rooted in learning-based techniques and the consolidation of emotional memories (Davey, 1997; Barlow, 2000; Bouton *et al.* 2001; Rothbaum & Davis, 2003). Extensive research supports the importance of normal sleep in learning and memory consolidation processes (see Stickgold & Walker, 2005). Specifically, healthy sleep has been identified as an important factor in the consolidation of emotional memories (Wagner *et al.* 2001; Hu *et al.* 2006). Animal studies have shown that sleep facilitates the consolidation of fear-based memory (an important finding for translational studies of anxiety) and sleep disruption impairs this process (Graves *et al.* 2003; Ruskin *et al.* 2004; Silvestri, 2005; Fu *et al.* 2007); however, the empirical literature in human samples is limited. In a recent study conducted by Soehner & Harvey (2012), participants with at least one severe insomnia symptom within a year demonstrated one third of cognitive functioning (e.g. memory) compared to controls. Other data suggest that sleep promotes fear extinction generalization (e.g. Pace-Schott *et al.* 2009), which is analogous to the therapeutic process used in CBT for anxiety disorders and allows individuals to extend the extinction learning to new contexts (e.g. Rothbaum & Davis, 2003). Learning and memory consolidation processes are imperative for the therapeutic process and may be maximized by

addressing sleep difficulties within the context of CBT. Understanding the impact that sleep difficulties have on these therapeutic processes and adequately addressing insomnia symptoms in the context of anxiety disorder treatment with sleep-related treatment modifications may maximize therapeutic gains. Therapeutic gains may also be made through further examination of the simultaneous or sequential treatment of anxiety disorders and comorbid insomnia. The impact of this research may extend beyond anxiety disorders treatment and include other disorders or conditions in which sleep difficulties may impact CBT processes (e.g. depression, continuous positive airway pressure/bi-level positive airway pressure adherence).

The relationship between anxiety disorders and insomnia is complex, and scarce research explores the impact of anxiety disorder treatment on insomnia. As cognitive impairment following insomnia (Soehner & Harvey, 2012) may undermine the efficacy of the primary treatment for anxiety disorders and insomnia (i.e. CBT), additional research may elucidate the relationship between residual sleep difficulties and anxiety disorder symptoms. Although CBT is multi-faceted, few studies have assessed the unique contribution of each component or the efficacy of combined psychotherapeutic approaches to address anxiety disorders and insomnia simultaneously (Perlis *et al.* 2005; Morin *et al.* 2006). Studies exploring the primary *vs.* secondary nature of each diagnosis are also scarce. Research may optimize the results of anxiety disorder and insomnia treatment by assessing various treatment sequences and combinations (Mellman, 2006) as well as relapse rates related to untreated anxiety disorder or insomnia symptoms. Finally, implementing comprehensive prospective studies may aid in identifying risk factors, understanding the course of illness, and developing ideal treatment for the prevention and onset of anxiety disorders and insomnia.

Declaration of Interest

None.

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Learning objectives

(1) Address sleep complaints in patients presenting with anxiety disorders.
(2) Consider CBT as preliminary and foundational treatment relative to drug therapy within the context of anxiety disorder and insomnia treatment.
(3) If considering drug therapy, do so on a short-term basis and educate the patient prior to treatment about short-term goals and potential negative side effects.
(4) If possible, treat insomnia and anxiety disorders simultaneously with CBT.
(5) Tailor components of CBT to optimize treatment of specific anxiety disorders and sleep difficulties.

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