

## Pharmacological and non-pharmacological treatments for nightmare disorder

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### Abstract

Interest in the treatment of nightmares has greatly increased over the last several years as research has demonstrated the clinical significance of nightmare disorder. This paper provides an overview of nightmare disorder, its clinical relevance, and the leading treatments that are available. In particular, the paper defines nightmare disorder and then summarizes the recent literature examining the clinical relevance of nightmare disorder, including its relation to post-traumatic stress disorder and other psychiatric conditions. The relation between nightmares and suicidality is also discussed. Recent findings on the treatment of nightmare with imagery rehearsal therapy and prazosin are then summarized. Lastly, the paper comments on potential future uses of nightmare treatment including using imagery rehearsal therapy or prazosin as a first-line intervention for post-traumatic stress disorder and using these treatments as an adjunctive therapy to reduce suicide risk in those at risk of suicide with nightmares.

### Introduction

Nightmares have long been discussed in the context of mental health (Freud, 1955) and this interest in nightmares has re-emerged in recent years as clinically relevant sleep disorders have been identified as a common risk factor for mental disorders. Further, research has demonstrated that the relation between nightmares and negative outcomes are often independent of co-morbid disorders such as post-traumatic stress disorder (PTSD), depression, and anxiety (Nadorff et al., 2011, 2013a; Sjöström et al., 2009). Concurrently to these observations, there has been a substantial growth in the literature on effective nightmare treatments. The current review will provide a brief discussion of nightmare disorder and the relation between nightmares and psychopathology before examining the leading pharmacological and therapy-based treatment options for nightmare disorder. Lastly, we review some potential areas of growth for nightmare treatments as well as potential novel uses for these therapies.

### Definition of nightmares

Nightmares, defined as vivid, disturbing, or frightening dreams that awaken the individual, are a common

form of parasomnia (Levin & Nielsen, 2007). Nightmares primarily occur during rapid eye movement (REM) sleep, and hence are more common during the second half of the night (American Academy of Sleep Medicine, 2006). The fact that nightmares occur in REM sleep differentiates them from night terrors, a parasomnia that occurs in non-REM sleep (APA, 2013).

The DSM-5 (APA, 2013) and ICSD-2 diagnostic criteria for nightmare disorder are similar in many ways. Both diagnostic systems require nightmares to be repeated negative dreams that awaken the individual from the nightmare, making the individual rapidly alert and aware of his or her surroundings. The DSM-5 requires that the nightmares not be better explained by substance use or medication, which is not required by the ICSD-2. On the other hand, the ICSD-2, but not the DSM-5, requires the individual to have difficulty falling back asleep, or for the nightmare to occur in the latter half of the night.

Bad dreams, which are negative dreams that do not lead to a startled awakening, are usually excluded from the definition of nightmares due to their lack of a startled awakening. However, bad dreams and nightmares are quite similar, as both require the recall of a negative dream, and both have been shown to be associated with sleep disruption and adverse

daytime consequences (Duval et al., 2013; Nadorff et al., 2014; Zadra & Donderi, 2000) Due to their similarities, many researchers combine bad dreams and nightmares (Levin & Nielsen, 2007). From a treatment perspective, both nightmares and bad dreams can be effectively reduced with pharmacological and behavioural treatments targeting negative dream content.

Although nightmares are most common in childhood, they are prevalent across the lifespan (Levin & Nielsen, 2007). Research suggests that bad dreams are more prevalent than nightmares (Zadra & Donderi, 2000), though research examining the prevalence of bad dreams throughout the lifespan is lacking. It is estimated that 19% of children experience nightmares at least once per week (Schredl et al., 2008). Although nightmares are often viewed as a childhood sleep disorder, research suggests that nightmares may persist into adulthood, and are a function of age and sex. Studies examining nightmares in adults have found nightmare disorder prevalence rates ranging from 2–6%. This prevalence range is highly consistent across cultures, with similar rates having been found in the USA, Canada, France, Iceland, Sweden, Belgium, Finland, Austria, Japan, and the Middle East (Levin & Nielsen, 2007). Women report more monthly nightmares than men up until age 60, at which point there is no significant gender difference. For women, the rate of nightmares significantly increases from ages 10–19 to ages 20–39, then steadily decreases to ages 50–59, and after age 60 the rate of nightmares stays constant. For men, the rate of nightmares increases from ages 10–19 to 30–39, and then and decreases from 30–39 to 50–59 (Nielsen et al., 2006).

Very little research has examined the prevalence of nightmares among older adults. However, the research that has been done suggests that nightmares may be less common among older adults compared with children and younger adults. In a study comparing older adults and college students, only 4.3% of older adults reported having a problem with nightmares, which was significantly less than the 19.5% of college students in the study reporting having a nightmare problem (Salvio et al., 1992).

### **Dream and nightmare theories**

It is often helpful to have a theoretical framework in order to understand a disorder and its effects. However, nightmares have historically received little attention from theorists or researchers. Even Freud, who did a considerable amount of work on dreams, rarely discussed nightmares. In his book *The Interpretation of Dreams*, Freud only mentions nightmares twice, and neither time did he provide a theory as to why individuals have nightmares (Freud, 1955).

Contemporary theorists have put forth ideas about the aetiology of nightmares, although little evidence supporting the theories exists. Despite this fact, a review of the current theories is still warranted, especially given that some of the nightmare treatments are based upon these theories.

After studying the histories of many nightmare sufferers from the psychoanalytic perspective, Hartmann (2001) concluded that nightmares are caused by ‘thin boundaries’. Thin boundaries refer to ‘the lack of separation between areas and processes in the mind, and also a lack of walls and defense’ (Hartmann, 2001). Supporting this theory, Hartmann (1989) found that thin boundaries were positively correlated with remembering one’s dreams, which may lead to remembering one’s nightmares. Similarly, other researchers have also found that thin boundaries are associated with dream recall, more negative and emotionally intense dreams, and regarding one’s dreams to be meaningful (Schredl et al., 1999).

Cartwright (2001) articulated a different view of nightmares, focusing on emotion processing instead of boundaries. She stated that when a traumatic event occurs, an individual may be unable to handle all of the resulting emotions at that time. Therefore, nightmares may emerge in order to help process the emotions caused by the trauma.

Taking a more biological approach, Levin and Nielsen (2007) published the AMPHAC/AND neurocognitive model of disturbed dreaming. The model seeks to explain disturbed dreaming at both physiological and cognitive levels. The model posits that the physiological and cognitive levels serve the function of fear-memory extinction during normal dreaming. In this process, components of fearful events are combined in a new way with non-fearful memories, disarming the memories. However, for individuals with significant distress (especially those who are predisposed to be sensitive to distress), the fearful memories may not be altered, or may be altered in a way even more frightening than the original memory, leading to disturbing dreams (Levin & Nielsen, 2009). The AMPHAC/AND model is consistent with the activation-synthesis hypothesis of dreaming (Hobson & McCarley, 1977), which is one of the most widely-accepted theories of normal dreaming. The activation synthesis hypothesis postulates that dreaming is an event that is physiologically shaped by the sections of the brain that are activated during sleep. In an attempt to make sense of the activation, the brain synthesizes the impulses into a narrative, which we experience as dreams.

Nightmares have also been viewed through a cognitive behavioural lens. The cognitive behavioural theory of nightmares posits that nightmares cause sleep avoidance behaviours (Krakow et al., 2001a;

St-Onge et al., 2009). In this theory the nightmare is considered to be a conditioned stimulus that causes a conditioned avoidance response. The awakening then reinforces the belief that the only way to avoid nightmares is to remain awake, which leads to the nightmare sufferer developing other behaviours to avoid sleep. Put simply, an individual may wake up and thereby escape from having the nightmare, which negatively reinforces the awakenings that are serving as an avoidance response. However, the avoidance of the nightmare increases sleep fragmentation, making an individual more likely to remember their dreams, perpetuating the nightmare problem. Additionally, it is possible that avoiding the nightmare may prevent exposure to the nightmare, potentially explaining why nightmares persist.

### **Negative consequences of nightmare disorder**

Although the most well-known co-morbidity is post-traumatic stress disorder, nightmares are also related to other psychiatric disorders, such as insomnia and borderline personality disorder (APA, 2013). Additionally, nightmares may be a precursor to psychopathology (Mellman et al., 1995; Ohayon & Shapiro, 2000; Sjöström et al., 2009), and have been shown to independently contribute to poor psychiatric outcomes. As such, nightmares have great clinical relevance for both prevention and treatment efforts. Further, nightmares can also affect more than just the nightmare sufferer, it can also affect the sufferer's bed partner. For example, there is a relation between relationship satisfaction and sleep disorders or prolonged sleep disturbances (Troxel et al., 2007), suggesting that the presence of sleep disorders can put significant strain on a relationship.

#### *Post-traumatic stress disorder*

Nightmares have a strong association with PTSD (Harvey et al., 2003; Kilpatrick et al., 1994; Mellman et al., 1995; Ohayon & Shapiro, 2000; Ross et al., 1989). The nightmares observed in PTSD (sometimes referred to as traumatic nightmares) can be re-enactments of the traumatic event or can be thematically or emotionally related to the original trauma, and are consistent with the re-experiencing symptom cluster of PTSD (APA, 2013). Nightmares are often chronic symptoms of PTSD: two studies examining war veterans found that nightmares persisted up to 50 years following the traumatic experience (Guerrero & Crocq, 1994; Kaup et al., 1994). Compared to individuals with idiopathic nightmares, those with PTSD-related nightmares

rated their nightmares as being more distressing (Germain & Nielsen, 2003b).

Nightmares may also influence the development of PTSD following trauma exposure. PTSD symptoms are more severe among individuals who reported having nightmares prior to the traumatic event than among individuals who did not report experiencing nightmares prior to the trauma (Mellman et al., 1995). Similarly, Ohayon and Shapiro (2000) and Bryant and colleagues (2010) found that pre-trauma sleep disturbances increased the risk of developing PTSD (and other psychiatric disorders), among large populations. Mellman and colleagues (2001) found having nightmares of the trauma shortly after the event was related to more severe PTSD symptoms six weeks later. Similar findings have also been reported by Kobayashi and colleagues (2008). These findings suggest that nightmares and other sleep disturbances (e.g. insomnia) may be confer heightened vulnerability for poor psychiatric outcomes following trauma exposure. Thus, nightmares – or more generally, sleep disturbances – and PTSD may arise from common psychophysiological or neural mechanisms (Germain et al., 2008; Levin & Nielsen, 2007).

#### *Anxiety and depression symptoms*

Nightmares have been shown in clinical samples to be related to anxiety and depressive symptoms throughout the lifespan (Levin & Nielsen, 2007; Nadorff et al., 2013a, 2014). However, some community studies have failed to find this result (Lancee et al, 2010b; Spoormaker & van den Bout, 2005), suggesting that the relation may be driven by those with clinically significant anxiety. Nielsen and colleagues (2000) found that disturbing dreams were associated with anxiety among adolescents. Nightmares have also been found to correlate strongly with symptoms of anxiety (0.41) and symptoms of depression (0.37) in a college student sample (Nadorff et al., 2013b) These relations hold true among older adults as well. A recent study of older adults presenting to a family medicine clinic found a very high correlation between nightmares and depressive symptoms (0.70) (Nadorff, 2013a). Additionally, although the prevalence of nightmares among older adults is just above 4% (Salvio et al., 1992), older adults with clinically significant depressive and anxiety symptoms had nightmare prevalence rates of 11.4% and 17.1%, respectively (Mallon et al., 2000). Relatedly, a recent secondary data analysis of a generalized anxiety disorder (GAD) randomized clinical trial found that the presence of a GAD diagnosis was significantly associated with higher levels of bad dream frequency, with 21.6% of those with a GAD diagnosis reporting weekly bad dreams (Nadorff et al., 2014).

*Dissociative disorders*

Dissociative disorders have been linked to acute stress disorder and PTSD (Agargun et al., 2003b), which suggests that they may be associated with nightmares as well. Agarun and colleagues (2003) found that 57% of individuals with a dissociative disorder met criteria for a nightmare disorder. Individuals who reported nightmares also had more dissociative symptoms than did individuals without nightmares. In a sample of 292 undergraduate students those who reported having nightmares scored significantly higher on the Dissociative Experiences Scale than those who did not report having nightmares (Agargun et al., 2003). Similarly, Semiz, et al. (2008) found a significant correlation (0.43) between the Dissociative Experiences Scale and nightmare frequency among individuals with borderline personality disorder.

*Borderline personality disorder*

Researchers have also found a link between nightmares and borderline personality disorder. In Hartmann's (1981) sample of adults with weekly nightmares, he found that 24% met DSM-III criteria for borderline personality disorder. Relatedly, Semiz and colleagues (2008) compared a sample of 88 participants diagnosed with borderline personality disorder with age and sex-matched controls, finding that the patients with borderline personality disorder reported more nightmares, greater dream anxiety, and more disturbed sleep than the control participants. Further, those with borderline personality disorder and a nightmare disorder exhibited more severe psychopathology than those with borderline personality disorder but without nightmare disorder. Nightmares have also been shown to be associated with borderline personality traits. Claridge et al. (1998) found that nightmare distress correlated strongly (0.42) with the Borderline Personality Scale in a sample of 60 undergraduate women.

*Suicidality and suicidal behaviour*

A rapidly growing literature suggests that nightmares increase one's risk of suicidality and suicidal behaviour across the lifespan (Nadorff et al., 2013a; Singareddy et al., 2013). Krakow and colleagues (2000) were the first to mention this association in their study of female sexual assault survivors who took part in a nightmare treatment study, finding that 43% of the sample reported suicidal ideation at intake. Cukrowicz and colleagues (2006) built upon this work through finding that nightmares were associated with suicidal ideation independent of insomnia symptoms.

Nightmares have also been examined in relation to suicide attempts. Sjostrom et al. (2007) studied 165 patients who had been admitted to the hospital following a medically serious suicide attempt. Although insomnia was the most common sleep complaint, two thirds of participants also reported having nightmares. A regression analysis revealed that nightmares were significantly associated with higher scores of suicidality, defined as the risk of attempting suicide, after adjusting for the presence of depression, anxiety, substance use, and PTSD diagnoses. Further, a follow-up study found that persistent nightmares predicted suicide attempts in the next two years in the same sample after controlling for the disorders listed above (Sjöström et al., 2009).

Nightmares are also related to death by suicide. Tanskanen et al. (2001) examined the relation between nightmares and death by suicide in a prospective study conducted in Finland. When compared with individuals without nightmares, those reporting occasional nightmares were at 57% greater risk of death by suicide. Further, participants reporting frequent nightmares were at 107% greater risk of suicide compared to those without nightmares. This study suggests that nightmares are potentially a risk factor for suicide.

In summary, nightmares are a prevalent condition that may confer heightened vulnerability to poor psychiatric outcomes that are often co-morbid with psychiatric conditions, and can independently exacerbate clinical outcomes in affected individuals. Thus, targeting nightmares with effective treatment may be an important component of prevention efforts aimed at high-risk samples, and of intervention for individuals with primary or co-morbid nightmares.

**Nightmare treatment**

Given the strong association between nightmare disorder and subsequent psychiatric complications, nightmares are increasingly being recognized as an important target for treatment, and not only as a 'secondary' symptom of psychopathology. Thus, the remainder of this paper examines the pharmacological and psychotherapeutic treatments for nightmare disorder and nightmares co-morbid with other conditions. Novel uses of nightmare treatments and targets for future research are also proposed.

*Pharmacological treatments*

Recently there has been an increased focus on pharmacological treatments for nightmares, including several recent reviews of this literature (Augedal et al., 2013; Aurora et al., 2010; Kung et al., 2012).

Despite numerous case reports, open-label trials, and randomized controlled clinical trials (see Maher et al., 2006, for review), only prazosin has consistently shown efficacy for the treatment of nightmares and distressed awakenings. Therefore, prazosin was the only medication that was recommended or suggested in the latest best practice guidelines for treating nightmare disorder (Aurora et al., 2010). Thus, our review of pharmacological therapies for nightmare disorder primarily focuses on prazosin, but other medications that may be worth consideration for future research are also mentioned below.

### Prazosin

Prazosin is a sympatholytic medication that is FDA-approved to treat high blood pressure, though it is also used off-label to treat PTSD. Prazosin is an alpha-1 adrenergic receptor antagonist that crosses the blood-brain barrier, and as such is thought to reduce noradrenergic tone during sleep (Feldman & Weidenfeld, 1996; Hilakivi, 1983; Mallick et al., 2005; Raskind et al., 2000; Taylor & Raskind, 2002). Recent best practice guidelines from the American Academy of Sleep Medicine give prazosin a level A recommendation, meaning that prazosin is supported by a great deal of high quality research (American Academy of Sleep Medicine, 2001). Similarly, the Veterans Administration recommends the use of prazosin to improve sleep quality and reduce trauma nightmares (US Department of Veterans Affairs, 2010).

Five randomized controlled trials examining the effect of prazosin on trauma-related nightmares that have been conducted to date (see Table 1 and Kung et al., 2012 for a full review of this literature). Raskind and colleagues (2003) compared prazosin (mean dose = 9.5 mg) to placebo in 10 Vietnam combat veterans with PTSD diagnoses using a 20-week double-blind crossover design. Prazosin was

well tolerated, with only two participants reporting side effects, of mild decreases in blood pressure or dizziness. Further, there was a significantly larger reduction in nightmares during the prazosin trial (severity score on the nightmare item of the Clinician Administered PTSD Scale [CAPS] reduced from 6.9 to 3.6) than was found during the placebo trial (from 7.1 to 6.7 on the nightmare item of the CAPS).

Raskind and colleagues (2007) built upon the previous study in a larger parallel group placebo-controlled study of prazosin. In this study, 40 veterans with chronic PTSD and trauma nightmares were randomized either to prazosin ( $13.3 \pm 3$  mg/day) or placebo conditions for a period of 8 weeks. Prazosin over 8 weeks resulted in a large effect size reduction in distressing dreams and significantly outperformed placebo (Cohen's *d* effect size = 1.68,  $p = 0.02$ ). However, it should be noted that there was no difference between the conditions after 4 weeks of treatment (Cohen's *d* effect size = 1.61,  $p = 0.09$ ). Dizziness upon standing was reported by 15 participants (nine in the prazosin condition, six in the placebo condition). There were no significant changes in blood pressure for participants in the prazosin condition between baseline and the end of the study. Thompson and colleagues (2008) examined a related question: does prazosin reduce non-nightmare distressed awakenings in veterans with PTSD. Utilizing a chart review of 22 veterans, the authors found significant reductions in trauma nightmares on the CAPS nightmare item (Cohen's *d* effect size = 0.56,  $p < 0.05$ ), sleep difficulty on the CAPS D-2 item (Cohen's *d* effect size = 1.80,  $p < 0.01$ ), and non-nightmare distressed awakenings (Cohen's *d* effect size = 1.49,  $p < 0.01$ ).

The literature was expanded to a civilian sample in a study by Taylor and colleagues (2008). The study consisted of 13 civilians with chronic trauma PTSD,

Table 1. Main characteristics of prazosin studies.

Study	Comparison (n)	Dose/session	Outcome measures	Duration of treatment	Military status
Germain et al., 2012	Prazosin (18) BSI (17) Placebo (15)	8.9 mg $8 \times 45$ min 10.4 mg	S-REP: NN	8 weeks	Veterans
Raskind et al., 2003	Prasozin (10) Placebo (10) Crossover design	9.5 mg	CAPS: IN	20 weeks	Vietnam veteran
Raskind et al., 2007	Prasozin (14) Placebo (15)	13.0 mg	CAPS: IN NFQ: NN	8 weeks	Veterans
Raskind et al., 2013	Prasozin (32) Placebo (35)	15.6 mg 7.0 mg 18.8 mg 10.0 mg	CAPS: IN	15 weeks	Active duty
Thompson et al., 2008	Prazosin (22)	9.6 mg 6.0 mg	CAPS: IN	3.4 (1.8)	Veterans
Taylor et al., 2008	Prasozin (13) Placebo (13) Crossover design	3.1 mg	CAPS: IN	7 weeks	Civilians

S-REP, self-report; NN, number of nightmares; IN, intensity; NFQ, nightmare frequency questionnaire; CAPS, clinical administered PTSD scale. Portions of this table are adapted from the table found in Augedal et al. (2013, p. 145).

frequent nightmares, and disturbed sleep. The study spanned 7 weeks and consisted of 3-week trials of both prazosin and placebo with a 1-week washout period in between. The order of the trials was randomly assigned and both the experimenters and participants were blind to the condition. Similar to previous studies (Raskind et al., 2007), prazosin resulted in large effect size reductions on the CAPS nightmare item (Cohen's  $d = 0.96$ ) and also resulted in a large effect size reduction on the PTSD dream rating scale (Cohen's  $d = 1.4$ ). Further, prazosin outperformed placebo on both nightmare measures ( $p = 0.04-0.006$ ).

Recently, a different research team from the previous three studies compared prazosin, a behavioural sleep intervention targeting insomnia and nightmares, and a placebo (Germain et al., 2012). The study consisted of 50 military veterans (mean age 40.9) who were randomly assigned to receive prazosin ( $n = 18$ ), BSI ( $n = 17$ ), or placebo ( $n = 15$ ), with each intervention having an 8-week duration. This study significantly built upon the literature in several significant ways, (1) it is the first study to compare prazosin to a cognitive behavioural therapy, (2) the study incorporated data from polysomnographic studies, and (3) participants with PTSD symptoms but not necessarily with full blown PTSD were included (58% of the sample met DSM-IV criteria for PTSD). Germain and colleagues (2012) found that both prazosin (1.0 to 0.3) and the behavioural sleep intervention (0.9–0.0) significantly reduced nightmare frequency greater than placebo (0.4 to 0.5) on prospective sleep diary measures of nightmare frequency. There were no significant differences between the active treatment groups, though this may have been due to insufficient power to detect this effect. The authors report that there were no significant changes in blood pressure for the prazosin and placebo groups, and that the frequency of side effects did not differ across the groups.

More recently, Raskind and colleagues (2013) recently completed a 15-week randomised controlled trial (RCT) comparing the effects of prazosin to placebo. The study consisted of 67 active duty service members who met DSM-IV criteria for combat-related PTSD. Similar to several previous studies, the outcome measure was the nightmare item on the CAPS. The prazosin group had a significantly greater reduction on the nightmare item between baseline and week 15 (6.0 to 2.9) than placebo (6.6 to 5.4) on the CAPS nightmare item. Similar to previous studies, prazosin was well tolerated and blood pressure did not differ between baseline and the end of the study, nor did it differ by treatment group.

In summary, the literature to date demonstrates the efficacy of prazosin for the treatment of PTSD-related nightmares. However, there are a few

limitations to this literature worth noting. First, most of the RCT studies involve the same research team. Thus, the literature could be strengthened through replication by other research groups. There is also a lack of studies examining the extent to which prazosin may be effective in treating idiopathic nightmares, or nightmares co-morbid with depression or suicidality, for instance. Only the study by Germain and colleagues (2012) included individuals without PTSD, but this study was also limited by the inclusion of military veterans, the majority of whom met criteria for PTSD. Thus, further research is needed to determine whether prazosin is indicated for non-PTSD nightmares, and replications are warranted in larger civilian samples. Lastly, nightmares may recur upon cessation of prazosin, though this was not seen in all samples (Germain et al., 2012) and may be due to differences in chronicity and co-morbidities between samples.

#### *Other pharmacological agents*

Although no other medication has the empirical support of prazosin, there are several other medications that have been examined as potential nightmare treatments (e.g. clonidine, trazodone, risperidone; see Maher (2006) for review). Despite the existence of effective pharmacological treatments, benzodiazepines are often utilized despite having been shown to be ineffective in reducing nightmares (Maher et al., 2006). In a small crossover clinical trial ( $N = 6$ ), clonazepam failed to show significant improvement in any sleep symptom, including nightmare frequency (Cates et al., 2004). Similarly, in another small crossover clinical trial ( $N = 10$ ), alprazolam failed to affect nightmares. Thus, the small literature examining the treatment of benzodiazepines in treating nightmares suggests that they are ineffective.

#### **Psychotherapeutic interventions**

There have been several psychological interventions that have been used to treat nightmares (see Table 2, and for recent reviews see Augedal et al., 2013; Hansen et al., 2013). Due to the presence of these recent reviews, the focus of this review is on the treatments most likely to be used in clinical practice.

#### *Imagery rehearsal therapy*

Imagery rehearsal therapy (IRT) is a cognitive behavioural therapy in which the patient rescripts the nightmare anyway he or she wants and then practises the new dream using imagery. Since the dream can be rescripted in any way, and does not have to contain material from the disturbing dream, IRT is not considered an exposure-based therapy. Rather, IRT

Table 2. Psychotherapeutic treatments for nightmares.

Intervention	Explanation
Imagery rehearsal therapy	Cognitive behavioural therapy in which the patient rescripts the nightmare any way he or she wants and then practises the new dream using imagery.
Exposure, relaxation, and rescripting therapy	Cognitive behavioural therapy that is designed for trauma-related nightmares that combines imagery rehearsal therapy with exposure and relaxation therapy.
Lucid dreaming	Therapy that helps individuals recognize when they are dreaming and then instructs them in how to change their dream while they are having it.
Systematic desensitization	A gradual exposure method where an individual utilizes relaxation techniques while being exposed to a hierarchy of fears.
Exposure	Involves the individual confronting him- or herself with his or her nightmare until it is no longer causing the individual significant distress.

is based on the concept that nightmares are a learned behaviour (Krakow, 2002, p. 18). Therefore, they can be replaced by a less disruptive behaviour, which in this case, is a new dream that does not disrupt sleep or daytime functions.

One of the first investigations of IRT consisted of 20 individuals who were randomly assigned to treatment or nightmare recording (Neidhardt et al., 1992). Three months after treatment the authors found that although nightmare frequency significantly decreased in both groups, there was a significantly greater reduction in the IRT group compared to the recording group. Further, only the IRT group had a significant difference in nightmare severity after 30 months (Krakow et al., 1993). Krakow and colleagues (2000) followed this study up with a large randomized controlled trial in a sample of 169 female survivors of sexual assault with PTSD. This was the first study looking at the effect of IRT in a sample with sub-threshold and full-blown PTSD. At 3-month follow-up nightmare frequency and PTSD symptoms were significantly reduced relative to the control group. Krakow and colleagues (2001a) provided later follow-up results from the previous study. The IRT group evidenced improved sleep quality ( $d = 0.67$ ), and reduced PTSD symptoms ( $d = 1.0$ ) relative to the control group.

Krakow and colleagues (2001b) extended the IRT findings by testing the theory in a sample of adolescent girls who had suffered a sexual assault. The sample consisted of 19 adolescent girls at a treatment facility who were randomly assigned to either the

treatment ( $n = 9$ ) or control ( $n = 10$ ) group. After three months, the treatment group reported a 71% decrease in nightmare frequency ( $d = 1.7$ ) whereas there was no change in the control group. However, there was no significant difference between the groups in PTSD symptoms ( $d = 0.22$  for the treatment group and  $0.27$  for the control group).

Forbes and colleagues (2003) were the first to study IRT in a sample of military veterans. Their sample consisted of 12 Australian Vietnam veterans who had been treated for PTSD in the past but were still having at least one trauma-related nightmare per week. At post-treatment there were significant reductions in nightmare frequency ( $d = 0.70$ ), nightmare intensity ( $d = 0.55$ ), depression symptoms ( $d = 0.43$ ), and anxiety symptoms ( $d = 0.20$ ). All of the significant treatment gains were still present at the 1-year follow-up, showing that IRT had a lasting impact in this sample. Nappi and colleagues (2010) extended the work done by Forbes and colleagues (2003) by testing imagery rehearsal therapy in a larger sample of 58 veterans. At the end of treatment there was a significant decrease in nightmare frequency ( $d = 0.45$ ), nightmare severity ( $d = 0.81$ ), insomnia symptoms ( $d = 0.72$ ), and PTSD symptoms ( $d = 1.03$ ). Lastly, IRT has also been shown to be effective in different types of nightmare sufferers. Three groups of nightmare sufferers – primary nightmare sufferers, nightmare sufferers with major depression, and nightmare sufferers with PTSD – were compared to a waiting-list control condition (Thünker & Pietrowsky, 2012). IRT led to a decrease in nightmare frequency in all three nightmare groups suggesting that IRT can be effective for many different types of nightmare sufferers, though participants with primary nightmares showed the greatest benefit from IRT.

Polysomnography has been utilized in a couple studies to assess whether IRT leads to changes in sleep architecture in addition to nightmares. Germain and Nielsen (2003a) examined the impact of one session of IRT on nightmare frequency, psychological distress, and sleep quality (measured by polysomnography) in an uncontrolled study. At follow-up approximately 2 months post-treatment, participants had significantly fewer nightmares ( $d = 1.06$ ) and significantly fewer anxiety symptoms ( $d = 1.01$ ) than prior to treatment. However, reductions in depressive symptoms ( $d = 0.79$ ) and nightmare distress ( $d = 0.9$ ) failed to reach significance. Polysomnography revealed no significant difference in the percentage of sleep that was REM sleep, REM latency, or REM density post-treatment. Building upon this work, Germain and colleagues (2012) compared prazosin, a behavioural sleep intervention consisting of IRT and cognitive behavioural therapy for insomnia, and placebo in a sample of 50 veterans. The authors

found no significant changes over time for sleep latency, wake after sleep onset, or sleep efficiency. However, the authors found that the brief sleep intervention and prazosin both outperformed placebo in global improvements, sleep continuity, and nightmare frequency. Similar to Germain and colleagues, Lancee et al. (2010a) compared IRT to an active treatment. In their study, IRT was compared with an exposure and recording intervention utilizing a self-help format. Additionally, a waiting-list control was recruited. IRT and exposure were found to be equally effective, with both active treatments outperforming a waiting-list control.

Although many studies have found positive results, there are a few studies in which IRT has either not shown an effect, or had a delayed effect. Cook and colleagues (2010) compared IRT to a credible active comparison condition (psycho-education and elements of cognitive behavioural therapy for insomnia) in 124 male Vietnam war veterans with PTSD. Contrary to the previous literature, the authors found that IRT did not outperform the comparison group for nightmare frequency, sleep quality, or PTSD symptoms. Similarly, Lu and colleagues examined IRT in 15 male US veterans with PTSD and nightmares related to traumatic experiences. The authors found that at post-treatment there were no observed treatment benefits. However, treatment gains emerged at both the 3- and 6-month follow-ups with nightmare frequency being significantly reduced at both time points and PTSD symptoms being significantly reduced at 3 months.

In sum, there is a large body of literature suggesting that IRT is effective in treating nightmares. IRT has been shown to decrease nightmare frequency (and in some cases, severity) in children, adolescents, adults, and veterans. Further, several studies suggest that treating nightmares with IRT may also improve sleep quality and reduce symptoms. Recently, Casement and Swanson (2012) conducted a meta-analysis on IRT's effect on PTSD and nightmares, finding that IRT decreased nightmare frequency and PTSD symptoms.

Lastly, since nightmares and insomnia are often co-morbid, the question of whether insomnia treatment should be added to nightmare treatment arises. Casement and Swanson (2012) examined whether a combination of IRT and cognitive behavioural therapy for insomnia was more effective than just IRT alone in helping alleviate sleep disturbances. They found that even combining IRT with insomnia treatment was not more effective for treating nightmares than IRT alone, this treatment combination does show better results in overall sleep quality improvement. Thus, in cases where both nightmares and insomnia are present, a combined treatment approach may be indicated.

### *Exposure, relaxation, and rescripting therapy*

Exposure, relaxation, and rescripting therapy (ERRT) is a cognitive behavioural therapy designed for trauma-related nightmares that combines IRT with exposure and relaxation therapy. ERRT also contains a model aimed at modifying maladaptive sleep habits and educating the patient about trauma. Trauma-related themes are also a focus of ERRT, but are not a main component of IRT (Davis, 2009). For recent reviews please see Augedal and colleagues (2013) and Hansen and colleagues (2013).

In a randomized controlled trial of 43 participants who had experienced a trauma and nightmares were randomly assigned to either EERT or a no-contact waiting-list control, EERT significantly outperformed the control condition in frequency and severity of nightmares and PTSD scores between pre-treatment and 6-month follow-up. In a second randomized controlled trial of 47 participants randomized to either treatment or waitlist control (Davis et al., 2011) there were significant improvements in both frequency and severity of nightmares and symptoms of depression, PTSD, sleep quality, physical health, and dissociation at 6-month follow-up. Thus, there is a small but growing literature supporting the efficacy of EERT in the treatment of trauma-related nightmares. However, further research is needed to determine whether EERT provides any clear benefit above and beyond the effects of IRT.

### *Lucid dreaming*

Lucid dreaming helps individuals recognize when they are dreaming and then instructs them in how to change their dream while they are having it. Two aspects of lucid dreaming are reality testing and dream signs, which are events that do not occur in real life (The Lucidity Institute, 1993). Once the individual is able to recognize when they are dreaming, the individual is then taught to change the dream while experiencing it so that the ending is positive.

To date, other than case reports, there have only been a handful of studies investigating the efficacy of lucid dreaming therapy on nightmares (Spoomaker & van den Bout, 2006). In the first study, 23 nightmare sufferers were randomly assigned to either a 2-hour individual lucid dreaming treatment session, a 2-hour group lucid dreaming treatment session, or a waiting-list control group. At post-test (12 weeks after treatment), participants receiving both the individual and group treatment reported significantly fewer nightmares when compared to pre-treatment baseline. However, there was no significant difference between the treatment and control groups, and no significant change in sleep quality or PTSD symptom severity. In the second study, Lancee and colleagues



(2010c) compared IRT, IRT and sleep hygiene, IRT and lucid dreaming, and waiting-list control. Surprisingly, IRT alone was shown to be more effective than the other two treatment conditions, and was the only treatment condition that resulted in a significant improvement in nightmares compared to the control group.

The lucid dreaming approach differs significantly from the other nightmare treatments. Instead of trying to reduce the frequency or severity of nightmares, it attempts to stop nightmares mid-way through. Thus, it is possible that lucid dreaming may be better tolerated than other nightmare treatments. However, more research is needed before lucid dreaming can be considered for treating nightmare disorder.

#### *Systematic desensitization*

Systematic desensitization is a gradual exposure method where an individual utilizes relaxation techniques while being exposed to a hierarchy of fears, and was one of the first nightmare treatments (Cellucci & Lawrence, 1978; Miller & DiPilato, 1983). Cellucci and Lawrence (1978) recruited 29 undergraduate students who reported having at least two nightmares per week. The participants were randomly assigned to either five sessions of systematic desensitization, placebo (a nightmare discussion group), or continuous tracking of nightmares. The systematic desensitization treatment group had a significantly greater improvement in nightmare frequency when compared to the placebo. Miller and DiPilato (1983) used systematic desensitization to treat 32 self-referred nightmare sufferers, who reported suffering an average of nine nightmares per month. Immediately after treatment there were significant decreases in nightmare frequency for both systematic desensitization and relaxation training, when compared to the waiting-list group. After 25 weeks nightmare intensity was found to be significantly reduced in the systematic desensitization group.

These studies support the use of systematic desensitization for nightmare treatment. However, more research is needed before systematic desensitization can be considered empirically supported for treating nightmare disorder.

#### *Exposure*

In addition to being a component of other nightmare treatments, exposure therapy has also been examined on its own as a treatment for nightmare disorder. Treating nightmares through exposure involves confronting an individual with his or her nightmare until it is no longer causing the individual significant distress. Burgess and colleagues (1998) investigated

the efficacy of using a self-administered exposure treatment in 107 nightmare sufferers who met DSM-III-R criteria for recurrent nightmares. The participants were randomized into either a self-exposure, self-relaxation, or waiting-list control group. Participants in the self-exposure group had a significantly greater reduction in nightmares ( $d = 1.18$ ) at one and six months post-treatment when compared with the self-relaxation and waiting-list groups. Additionally, those who received the self-exposure treatment also experienced a significant reduction in their depressive symptoms ( $d = 0.76$ ). Grandi and colleagues (2006) also investigated the efficacy of a self-exposure treatment for nightmares, finding that the exposure treatment significantly reduced not only nightmare frequency and intensity, but also symptoms of anxiety, depression, and sleeplessness at the post-treatment follow-up. Remarkably, most of the gains were maintained over 4 years.

Although more research is needed, both studies that examined use of self-administered exposure found strong positive results. However, both relied on participants doing a significant amount of homework each day (30–60 min), which may affect compliance and may even be impossible for some nightmare sufferers.

#### **Conclusions and future directions**

In summary, both prazosin and IRT are empirically supported treatments for nightmares. However, additional research is needed to enhance response and remission rates following treatment completion or discontinuation, and to examine the extent to which nightmare treatments are effective in treating nightmares that are co-morbid with other forms of psychopathology.

To the best of our knowledge no study has directly compared prazosin and IRT, but a recent study by Germain and colleagues (2012) that compared prazosin to a brief behavioural intervention that included IRT found no significant differences between the treatments. Similarly, a recent meta-analysis (Augedal et al., 2013) found no significant difference between IRT and prazosin. This literature suggests that both prazosin and IRT are effective in treating nightmares in individuals with PTSD. However, unlike IRT, prazosin has not been studied in treating idiopathic nightmares. Thus, randomized controlled trials are warranted to evaluate the efficacy of prazosin for idiopathic nightmares. Further, and given that psychological nightmare treatments seem to have more durable effects than prazosin or other agents that require long-term use (Augedal et al., 2013), psychological treatments may be a better first-line treatment for nightmares. Fewer side effects have also been reported in psychotherapeutic trials, although side

effects tend to be less closely monitored in these trials. Finally, the efficacy of combined IRT and prazosin remains unknown, but may be especially promising for reducing chronic, treatment-resistant nightmares.

There are several promising directions for future investigations. First, as discussed previously, there is a need for studies examining whether prazosin is effective in treating idiopathic nightmares and nightmares co-morbid with other conditions such as depression or suicidality. For instance, the treatment of nightmares in those at elevated suicidal risk or with past attempts may improve clinical outcomes over time. In a related manner, nightmare treatments combined with other first-line pharmacological or psychological treatments of PTSD may facilitate adherence and improve clinical outcomes.

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