Journal of Traumatic Stress
August 2020, *33,* 511–520

ISTSS International Society for Traumatic Stress Studies

Assessing Triggers of Posttrauma Nightmares

Westley A. Youngren,[1] Nancy A. Hamilton,[1] and Kristopher J. Preacher[2]
[1]Department of Psychology, University of Kansas, Lawrence, Kansas, USA
[2]Department of Psychology and Human Development, Vanderbilt University, Nashville, Tennessee, USA

Posttrauma nightmares are recurring nightmares that begin after a traumatic experience and can occur as often as multiple times per week, often in a seemingly random pattern. Although these nightmares are prevalent in trauma survivors, little is known about the mechanisms underlying their sporadic occurrence. The present study aimed to investigate predictors of posttrauma nightmares. The sample included 146 observations nested within 27 female college students who reported frequent nightmares related to sexual trauma. Participants were recruited from an undergraduate student subject pool ($n = 71$) or were clinical referrals ($n = 75$). Participants completed an initial assessment battery and six consecutive days of pre- and postsleep diaries, which included measures of potential posttrauma nightmare triggers and measures intended to assess sleep quality and posttrauma nightmare occurrence. Descriptive statistics, mean comparisons, and multilevel modeling were used to examine the data. The results showed that both presleep cognitive arousal, $\gamma_{10}\text{SL}_{ij} = 0.58$, $p = .006$, $z(1, N = 146) = -2.61$; and sleep latency (SL), $\gamma_{20}\text{PCA}_{ij} = 0.76$, $p < .001$, $z(1, N = 146) = -2.69$, predicted posttrauma nightmare occurrence. Further investigation suggested that presleep cognitive arousal moderated the relation between SL and posttrauma nightmare occurrence, $\gamma_{30}\text{PCA x SL}_{ij} = 0.67$, $p = .048$ $z(1, N = 146) = 1.98$. The present results are the first to show that the co-occurrence of presleep arousal and delayed sleep onset latency may influence posttrauma nightmare occurrence, suggesting that the time immediately before sleep is crucial to the production of the posttrauma nightmares.

Posttrauma nightmares are among the most persistent and problematic symptoms of posttraumatic stress disorder (PTSD; Germain, 2013). Among trauma survivors, there is a high prevalence of posttrauma nightmares, with some estimates as high as 88% (Forbes, Phelps, & McHugh, 2001; Ohayon & Shapiro, 2000), and they are among the most distressing symptoms to occur following trauma exposure. One of the reasons posttrauma nightmares are so distressing is that they frequently replicate the traumatic event. As such, posttrauma nightmares have been linked to insomnia as well as comorbidities including depression, anxiety, cardiovascular risk factors, alcohol abuse, suicidal ideations, and suicide attempts (Galatzer-Levy, Nickerson, Litz, & Marmar, 2013; Nadorff, Nazem, & Fiske, 2013; Pacella, Hruska, & Delahanty, 2013; Van der Kolk, Blitz, Burr, Sherry, & Hartmann, 1998). Although recent research has shed light on the basic processes related to nightmare vulnerability and production, one of the most basic questions about nightmares has yet to be addressed: Why tonight?

Posttrauma nightmares are recurring nightmares that begin following a traumatic event (Davis, 2008). They are a common but not ubiquitous symptom of PTSD (Germain, 2013) and are classified in the *Diagnostic and Statistical Manual of Mental Disorders* (fifth ed.; *DSM-5*) PTSD diagnostic criteria as a part of the intrusion symptom cluster. Although posttrauma nightmares are not omnipresent with PTSD, previous research has reported high prevalence rates of nightmares, often ranging from 80% to 90%, within trauma-exposed samples (Koffel, Khawaja, & Germain, 2016). Demographic variables, such as gender, age, and past trauma history have also been associated with an increased risk of experiencing nightmares after following a traumatic event (Levin & Nielsen, 2007). However, despite their prevalence and severity, less is known about posttrauma nightmares than daytime intrusion symptoms, such as flashbacks.

The most well-known theoretical model for the development and pathophysiology of the posttrauma nightmare is the affective network dysfunction (AND) model. Central to the AND model is the idea that individual differences in limbic system function explain vulnerability to nightmares; the AND model posits that the production and chronicity of nightmares are related to high levels of arousal in the limbic system (Nielsen & Levin, 2007). Although the AND model potentially explains *why* (i.e., an interaction of experiencing a trauma and specific vulnerability variables) and *how* (i.e., the limbic system) posttrauma nightmares occur, it does not explain *when* they will occur.

Although posttrauma nightmares are common among trauma survivors, they do not occur every night; reported frequencies range from once per week to nearly every night of the week,

Correspondence regarding this article should be addressed to Westley A. Youngren, The University of Kansas, 1415 Jayhawk Blvd, KS 66047. E-mail: way946@ku.edu

DOI: 10.1002/jts.22532

with increased nightmare frequency linked to higher levels of overall PTSD symptom severity (Davis, 2008). Anecdotally, it has been reported that the unpredictable nature of posttrauma nightmares makes these nightmares even more distressing, with many trauma survivors reporting distress because they cannot predict when they will be able to get a good night's sleep. Although the sporadic occurrence of posttrauma nightmares has been recognized, very little research has attempted to identify why these nightmares occur on some nights instead of others. In fact, to date, no study of which we are aware has been specifically designed to identify triggers or antecedents of posttrauma nightmares. The following paragraphs review a likely set of posttrauma nightmare predictors derived from both trauma and sleep research as well as the AND Model (Nielsen & Levin, 2007).

Posttrauma nightmares have typically been conceptualized as intrusion symptoms triggered by the same kinds of reminiscent stimuli as other intrusion symptoms, such as flashbacks (Hartmann, 1998). Reminiscent stimuli are experiences that remind an individual of a traumatic event (i.e., the triggering trauma) and are the most commonly understood predictors of flashbacks and unwanted memories (Michael, Ehlers, Halligan, & Clark, 2005). The association between nightmares and other intrusion symptoms has been supported by confirmatory factor analytic studies in which nightmares and flashbacks load onto a single common factor (Simms, Watson, & Doebbelling, 2002). Given the shared variance between nightmares and flashbacks, it seems plausible that, like flashbacks, reminiscent stimuli may also trigger the production of posttrauma nightmares.

Abnormal levels of autonomic arousal have been directly linked to sleep disturbances in the pathophysiology of PTSD (Bonnet & Arand, 2010; Woodward et al., 2009). For example, arousal-related PTSD symptoms have been linked to reduced amounts of slow-wave sleep (SWS) and fragmentations during rapid eye movement (REM) sleep (Vermetten, Germain, & Neylan, 2018). This apparent entanglement between sleep, PTSD, and physiological arousal led Levin and Nielsen (2007) to propose the previously discussed AND model, which suggests that increased physiological arousal might play a pivotal role in the production of posttrauma nightmares. Following logically from the AND model, if physiological arousal is connected to sleep disruption in people who have been diagnosed with PTSD, it may follow that nightly variations in presleep arousal may predict the occurrence of a posttrauma nightmare.

Along with physiological arousal, cognitive arousal, such as worry and rumination, may also play a role in the production of posttrauma nightmares. This hypothesis was derived from converging bodies of research. First, presleep cognitive arousal (PCA) in the forms of worry and rumination has been found to predict increased sleep latency (SL), or the time it takes to fall asleep, and decreased subjective sleep quality (Borders, Rothman, & McAndrew, 2015). Similarly, rumination about a past traumatic event has been found to predict the persistence of PTSD symptoms (Ehring, Frank, & Ehlers, 2008). Most relevant to dream research is the continuity hypothesis, which theorizes that daily and presleep experiences influence dream content. Consistent with the continuity hypothesis is research by Schredl (2006), which showed that emotions experienced during the day were likely to be represented in dream content. Following logically from research that has shown cognitive arousal to be related to the persistence of PTSD symptoms, longer SL, and poor sleep quality and that cognitive and emotional content prior to sleep may manifest in dreams, it also seems likely that nightly variations in PCA and SL could also predict the occurrence of a posttrauma nightmare.

Poor sleep quality and fatigue may affect sleep architecture by way of altering the proportion of time, in stages, that is conducive to the production of posttrauma nightmares. It is well known that rebound sleep following sleep restriction and partial sleep restriction is associated with changes in sleep architecture, including increased REM and SWS and reduced light sleep (Carskadon & Dement, 2005). It is also well known that both trauma-related and non–trauma-related often occur during REM sleep. Furthermore, individuals with PTSD often have more REM density and interruptions than individuals without PTSD, which may be linked to the frequency of nightmare occurrences (Kobayashi, Boarts, & Delahanty, 2007). Therefore, increasing REM density via sleep rebound, due to poor sleep quality the previous night, may indirectly increase the likelihood of a nightmare during REM sleep (Nielsen & Levin, 2007). However, it should be noted that posttrauma nightmares have been observed to occur during both REM and non-REM sleep (Phelps et al., 2017). Despite occurrences in other stages of sleep, it seems likely that REM rebound caused by a previous night's poor sleep quality might predict the occurrence of posttrauma nightmares that occur during REM.

For people with PTSD, poor sleep quality has also been linked to increased stress reactivity and reports of daytime intrusive symptoms, such as unwanted memories (Dietch et al., 2019; Kerkhof & Van Dongen, 2010; Krizan & Hisler, 2018). Stress reactivity also plays a key role in Levin and Nielsen's AND Model (2007). Briefly summarized, the AND model has proposed that nightmares are formed when dream content activates neural fear pathways, from the amygdala to the hypothalamic–pituitary–adrenal (HPA) axis, increasing autonomic arousal and transforming the cognitive appraisal of dream content into a posttrauma nightmare. The key role of arousal has also been demonstrated by data from a recent study showing that reports of nightmares followed episodes of autonomic arousal caused by sleep apnea events (Phelps et al., 2017). Similar to daytime stress reactivity and consistent with the AND Model, it seems plausible that daytime fatigue due to poor sleep quality may potentiate activation of the amygdala–HPA threat pathways, transforming dream content into a posttrauma nightmare.

Despite increased empirical research on dreams in general and posttrauma nightmares in particular, basic questions about the triggers associated with posttrauma nightmares remain unanswered. It is likely that answering this question has been hindered by the methods used to study posttrauma nightmares, which have relied mainly on retrospective self-report,

the difficulty in capturing a nightmare during a laboratory polysomnographic study (Woodward, Arsenault, Murray, & Bliwise, 2000), and the logistic difficulties of multinight ambulatory polysomnographic (PSG) studies. We addressed this research gap by using at-home morning and evening sleep diaries to identify the triggers of posttrauma nightmares in a sample of college women who reported a history of sexual trauma and frequent posttrauma nightmares. Given the paucity of research on antecedents to posttrauma nightmares, this study drew from theoretical models related to sleep and trauma as well as the AND (Levin & Nielsen, 2007) and sleep continuity models (Schredl, 2006) to identify a list of potential candidate variables that might predict the occurrence of posttrauma nightmares. Derived from those theoretical models, we hypothesized that the following variables would predict the occurrence of posttrauma nightmares: reminiscent stimuli, poor sleep quality, fatigue, SL, presleep arousal, and PCA.

Method

Participants

Participants were recruited through two different methods. Some participants were recruited as part of a larger study, the Prevalence of Traumatic Experiences on Campus (POTEC) study. For POTEC, information was gathered about the prevalence of traumatic experiences and posttrauma nightmares in a sample of students enrolled in an Introduction to Psychology class at a U.S. university. Additional participants were referred from community mental health care providers. Participants in this group were female clients seeking treatment for symptoms caused by sexual trauma who were given the research coordinator's email address and study information. Interested potential study participants contacted the research coordinator and set up a time to determine participation eligibility and enrollment. Inclusionary criteria were as follows: (a) self-reported experience of sexual trauma, (b) experiencing nightmares related to sexual trauma, (c) self-reporting as a female, and (d) over 18 years of age. Participants varied in terms of the language used to describe sexual trauma. For the purposes of this study, sexual trauma was operationally defined as a participant's report of sexual violence; all events met the criteria for a traumatic experience per the Structured Clinical Interview for *DSM-5* (SCID-V; First, Williams, Karg, & Spitzer, 2015).

The analyses included 146 observations nested within 27 female college students who reported experiencing nightmares related to sexual trauma. The median age for the sample was 20.05 years ($SD = 3.08$). This was a moderately diverse sample, with 59.3% of participants identifying as White, 22.2% identifying as Hispanic, 11.1% identifying as Asian, and 7.4% identifying as African American. Approximately half of the participants ($n = 15$, 55.6%) reported having experienced a nonsexual trauma, such as a car wreck or physical abuse, prior to the triggering trauma, whereas 48.2% ($n = 13$) of participants reported having experienced another sexual trauma, such as rape or molestation, prior to the sexual trauma that triggered the nightmares. Nearly all participants met criteria for a PTSD diagnosis ($n = 25$, 92.6%). In total, 13 participants ($n = 71$ observations) were recruited as part of the POTEC study, and 14 participants were recruited from provider referrals ($n = 75$ observations). In general, there were few differences between the POTEC and clinical referral recruitment groups. Participants in both groups reported similar levels of past trauma prevalence, and almost all participants in both samples met the diagnostic criteria for PTSD per the SCID-V and reported distressed sleep. However, there were differences in reported levels of distress, with the clinic sample reporting higher levels of anxiety symptoms (BAI: POTEC $M = 23.7$, $SD = 8.4$; Clinical $M = 31.3$, $SD = 12.1$) and depression symptoms (BDI: POTEC $M = 16.6$, $SD = 7.4$; Clinical: $M = 21.2$, $SD = 4.8$). Information on predictor differences by referral group is presented in Table 2.

In total, 30 participants completed the informed consent process. Two participants completed fewer than three pre- and postsleep surveys, so their data were removed from analyses. One participant was heavily medicated for narcolepsy and experienced frequent hallucinations; thus, her data were also removed from data analyses. Seven participants completed fewer than six but more than two continuous pre- and postsleep surveys. Following mixed-model missing data recommendations (Faraway, 2016), these participant's data remained in these analyses. The remaining participants ($N = 20$) completed at least six consecutive pre- and postsleep surveys. The electronic time stamps from night and morning diaries showed that all participants included in the analyses completed their surveys on the appropriate nights and mornings and within 15 min of getting into bed for sleep and awakening, respectively. In summary, the final following data analyses consisted of 146 observations nested within 27 participants.

Procedure

Participants completed an informed consent form that explained the purpose and procedure of the study. Immediately following the completion of this form, a trained clinical researcher administered the PTSD module of the SCID-V as well as a battery of questionnaires used to assess general depression- and anxiety-related symptoms. Participants were then instructed to complete evening (i.e., presleep) and morning (i.e., postsleep) electronic sleep and dream diaries for the next 6 consecutive days. Participants accessed these diaries from a reusable link emailed to their personal email account and completed the questionnaires with their personal electronic devices at their usual place of residency. All hard-copy data, such as the informed consent and SCID-V assessment, were stored in a protected filing cabinet, and all electronic data, such as survey responses, were stored on a firewall-protected and password-encrypted computer. The University of Kansas' Office of Research Institutional Review Board approved all study protocols.

Table 1
Model-Building Process

n		Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	**Model 8**	Model 9
Fixed components										
Intercept	$\hat{\gamma}_{00}$	–3.37**	–3.40**	–3.51**	–3.48**	–3.65**	–3.53**	–3.36**	**–5.18****	–3.71**
Sleep latency	$\hat{\gamma}_{10}$	2.33*	2.34*	2.36*	2.36*	2.45*	2.43*	2.74**	**2.58***	1.53
Presleep cognitive arousal	$\hat{\gamma}_{20}$	1.33	1.40	1.41	1.39	1.41	1.79*	1.79*	**2.40***	
Subjective sleep quality	$\hat{\gamma}_{30}$	–2.35*	–2.37*	–2.37*	–2.41*	–2.38*	–2.24*	–1.63		
Sleep efficiency	$\hat{\gamma}_{40}$	1.33	1.33	1.33	1.45	1.68	1.66			
Fatigue	$\hat{\gamma}_{50}$	0.73	0.72	0.77	0.85	1.06				
Sleep duration	$\hat{\gamma}_{60}$	0.39	0.37	0.37	0.47					
Reminiscent stimuli	$\hat{\gamma}_{70}$	0.35	0.36	0.37						
Presleep physical arousal	$\hat{\gamma}_{80}$	0.78	–0.03							
Daytime dysfunction	$\hat{\gamma}_{90}$	–0.19								
Model components										
AIC		95.7	93.7	91.7	89.8	88.1	87.2	88.0	**93.5**	94.1
BIC		123.3	118.8	114.3	109.9	105.6	102.3	100.5	**113.9**	101.7
Loglik		–36.8	–36.9	–36.9	–36.9	–37	–37.6	–39.0	**–41.47**	44.1
Likelihood ratio test			0.03	0.06	0.13	0.22	1.15	2.76	**2.76**	5.91*

Note. Fixed effects were estimated using maximum likelihood. Deviance and corresponding likelihood ratio test calculated using restricted maximum likelihood tests. The final model is indicated in bold. AIC = Akaike information criterion; BIC = Bayesian information criterion.
*$p < .05$. **$p < .01$.

Measures

PTSD. Symptoms of PTSD were assessed using the PTSD module from the SCID-V (First et al., 2015). The SCID-V is a structured interview used to determine whether individuals meet the *DSM-V* criteria for PTSD; it has demonstrated good psychometric properties in a variety of populations.

Depression. The Beck Depression Inventory–I (BDI-I; Beck, Steer, & Brown, 1996) was used to assess depressive

Table 2
Predictor Descriptives Characteristics

	POTEC ($n = 71$)		Clinical ($n = 75$)		Combined ($N = 146$)	
Variable	*M*	*SD*	*M*	*SD*	*M*	*SD*
Reminiscent stimuli	0.23***	0.43	0.46***	0.50	0.37	0.48
Fatigue	2.23*	1.85	3.81*	2.43	3.16	2.34
Presleep cognitive arousal	13.72	8.74	13.09	6.93	13.35	7.71
Presleep somatic arousal	4.03	2.93	6.10	4.17	5.25	3.83
PSQI subscale score						
Subjective Sleep Quality	1.35	0.80	1.41	0.69	1.38	0.74
Sleep Efficiency	1.38	0.84	1.33	0.77	1.35	0.79
Daytime Dysfunction	1.65***	1.32	2.89***	1.65	2.38	1.64
Sleep Duration	0.63	1.01	0.88	0.70	0.77	0.99
Sleep Latency	1.43	0.85	1.32	0.76	1.37	0.79

Note. POTEC = Prevalence of Traumatic Experiences on Campus study; PSQI = Pittsburgh Sleep Quality Inventory.
*$p < .05$. **$p < .001$.

symptoms. The BDI-I is a 21-item assessment on which individuals use a scale of 0 to 3 to respond to items related to depressive symptoms (possible score range: 0–63). A score range of 0–13 indicates minimal to no depression symptoms, 14–19 indicates mild depression symptoms, 20–28 indicates moderate depression symptoms, and 29–63 indicates severe depression symptoms. In the present sample, Cronbach's alpha was .74, which is lower than previously reported scores (e.g., Cronbach's $\alpha = .86$; Beck, Epstein, Brown, & Steer, 1988).

Anxiety. The Beck Anxiety Inventory (BAI; Beck et al., 1988) was used to assess anxiety-related symptoms. The BAI is a 21-item assessment on which respondents rate items related to anxiety symptoms using a scale of 0 (*not at all*) to 3 (*severely/bothered me a lot*). The total possible score range is 0 to 63, whereby scores of 0–21 indicate no to low anxiety, 22–35 indicate moderate anxiety, and 36–63 indicate severe anxiety symptoms (Beck et al., 1988). In the present sample, the observed Cronbach's alpha value was .89, which is similar to previously reported scores (e.g., $\alpha = .94$; Beck et al., 1988).

Presleep variables. Presleep diaries included six daily assessments of putative daytime predictors of nightmares, including reminiscent stimuli, presleep arousal, and fatigue. Participants were instructed to complete these measures immediately before going to bed.

Reminiscent stimuli. To assess whether a participant experienced an event that reminded them of the triggering trauma, participants were asked the following closed-ended question: "Did you experience any event today that reminded you of the trauma associated with your nightmares? This event could have been, but is not limited to, visual reminders, people, places, sounds, smells, thoughts, feelings, life events or anniversaries." If the participant endorsed experiencing a reminiscent stimulus, she was then asked to freely describe the stimulus.

Non–nightmare-related intrusion symptoms. To assess the occurrence of other non–nightmare-related intrusion symptoms, such as flashbacks and unwanted memories, two questions from the PTSD Checklist for *DSM-5* (PCL-5; Weathers, Litz, Keane, Palmieri, Marx, & Schnurr, 2013) were edited to reflect daily experiences and inserted into the nighttime survey. These questions were: "Today, did you experience disturbing and unwanted memories of the traumatic experience" and "Today, did you suddenly feel or act as if the stressful experience were actually happening again (as if you were actually back there reliving it)?" Participants responded with *yes*, *no*, or *unsure*. If a participant endorsed experiencing one of these intrusion symptoms, she was then asked how many times this experience happened during the day.

Presleep arousal. The Presleep Arousal scale (Nicassio, Mendlowitz, Fussell, & Petras, 1985) is a 16-item scale that assesses somatic and cognitive presleep arousal states. Respondents are asked to score items using a scale ranging from 1 (*not at all*) to 5 (*extremely*). Higher scores indicate higher states of presleep arousal. A Somatic Arousal subscale score is created by totaling Items 1–8, whereas a Cognitive Arousal subscale score is created by totaling Items 9–16. A total score for presleep arousal can be created by totaling items 1–18. In the present sample, the Cronbach's alpha value for the total score was .86; the alpha values for the Somatic Arousal and Cognitive Arousal subscales were .67 and .92, respectively. These values are similar to previously reported internal consistency scores (Cronbach's αs = .85, .79, and .88 for the total, Somatic Arousal, and Cognitive Arousal scales, respectively; Nicassio et al., 1985).

Fatigue. The Fatigue Assessment Scale (FAS; Michielsen, De Vries, & Van Heck, 2003) is a 10-item scale intended to assess physical, mental, and motivational fatigue. Participants are asked to respond to items using a scale ranging from 1 (*never*) to 5 (*always*). For the purpose of daily measurement, a statement at the beginning of the study instructed participants to respond to items in reference to how they felt that day. Totaled scores range from 10, representing the lowest level of fatigue, to 50, representing the highest. In the present sample, Cronbach's alpha for the FAS was .91, which is similar to previously reported scores (e.g., $\alpha = .90$; Michielsen et al., 2003).

Postsleep variables. Postsleep diaries included assessments of sleep quality as well as questions about dreams. Participants were asked to complete these measures as soon as possible after they awoke.

Sleep quality. The Pittsburgh Sleep Quality Inventory (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) was used to measure sleep quality. The PSQI instructions were edited to capture the previous night's sleep. This was done by replacing references to "past month" with references to "last night". The PSQI is a nine-item scale that produces a global score for sleep quality (i.e., a summation of all subscales) as well as scores for the following seven subscales: Subjective Sleep Quality (Item 9), Sleep Latency (Item 2), Sleep Duration (Item 4), Sleep Efficiency (Items 1–3), Sleep Disturbances (Item 5), Use of Sleep Medications (Item 6), and Daytime Dysfunction (Items 8 and 9). Each subscale has a possible score of 0 to 3, and the global score is calculated by summing the scores for all subscales. However, the item related to sleep disturbance includes questions about the occurrence of bad dreams. Due to the similarity between this question and the outcome variable (i.e., occurrence of a nightmare), we removed the sleep disturbance subscale from the present analyses. Because of this removal, we also could not include the global score as it is a summation of every subscale. Given that most of the PSQI subscales consist of one item, Cronbach's alpha values could not be calculated for each subscale. However, the PSQI has demonstrated strong validity, with past studies reporting significant correlations with actigraphy ($r = 0.31$, $p < .01$) and

sleep diaries ($r = -0.56$, $p < .01$; Grandner, Kripke, Yoon, & Youngstedt, 2006).

Nightmares and dreams. To capture the occurrence of nightmares and ensure we were assessing posttrauma nightmares rather than other types of dreams, a scale of nightmares and dreams was created for this study. Participants were asked a series of questions about their dreams. First, participants were asked whether they remembered their dreams last night (*yes* or *no*). If they answered yes, they were asked whether one of their dreams was a bad dream (i.e., nightmare) that woke them and was related to their triggering trauma (*yes* or *no*). If they answered yes to this question, they were asked to briefly describe the content of that dream. Other dream categories were (a) bad dreams that woke the dreamer but were not related to the triggering trauma, (b) bad dreams that did not wake the dreamer, (c) stress dreams, (d) vivid dreams, (e) bizarre dreams, (f) joyful dreams, and (g) neutral dreams. Although this measure was devised specifically for the present study, the dream categories were derived from existing dream scales and based on recommendations made by Schneider and Domhoff (2001). The nightmare assessment followed a structure similar to that used in the Trauma-Related Nightmare Scale (Davis, Wright, & Borntrager, 2001), which is a larger scale intended to assess posttrauma nightmares.

Data Analysis

A power analysis calculator was used to detect optimal sample size for a desired statistical power of 0.80 for a one-tailed multilevel logistical regression analysis with two random effects, five fixed effects (all predictors possible), and an error probability of 0.05. The suggested sample size based on this analysis was 118 observations.

All subsequent analyses were conducted using *R* statistical software (Version 3.4.4). Descriptive and prevalence statistics were calculated. Given that observations were nested within Participants, an intraclass correlation coefficient (ICC) score for participants was calculated to determine whether multilevel modeling (MLM) was necessary, which it was, ICC = .08. For the following analyses, the model treated observations as nested within the participant (i.e., multiple observations for each participant). First, a mixed effects logistic regression analysis utilizing a random-effects model with maximum likelihood estimation (MLE) was used to explore whether our independent variables predicted the occurrence of posttrauma nightmares where ßs represent random coefficients, *u*s are Level 2 errors, *X*s are fixed Level 1 predictors (repeated for each independent variable), y_{ij} is posttrauma nightmare occurrences, and *j*s are level indicators (i.e., participant code).

$$\begin{aligned} \text{logit}(y_{ij}) &= \beta_{0j} + \beta_{1j}X_{ij} + \ euro_{ij} \begin{bmatrix} u_{0j} \\ u_{1,j} \end{bmatrix} \\ \beta_{0j} &= \gamma_{00} + u_{0j} \\ \beta_{1j} &= \gamma_{10} + u_{1j} \end{aligned}$$

$$\sim MVN\left(\begin{bmatrix} 0 \\ 0 \end{bmatrix}, \begin{bmatrix} \tau_{00} & \\ \tau_{01} & \tau_{11} \end{bmatrix}\right)$$

In accordance with Finch et al. (2016), a top-down model-building strategy was used to determine the most parsimonious final model. The top-down model-building strategy uses Akaike information criterion (AIC) and Bayesian information criterion (BIC) scores and the likelihood ratio test to determine the best-fitting model (i.e., the final model). This strategy creates a "top model," which is a model that includes all possible predictors then, one by one, removes a nonsignificant parameter with the lowest weight in the equation and uses the likelihood ratio test to compare the new model, with the removed predictor and 1 degree of freedom, to the previous model. This procedure continues until there is a significant difference between models as determined by the likelihood ratio test. Then, AIC and BIC values are used to determine the best-fitting model, with lower values indicating improved model fit; the best-fitting model then becomes the final model. For an overview of how this procedure was used in the present study, see Table 1.

To further investigate the associations among the interactions between significant predictors of posttrauma nightmares, we conducted post hoc moderation analyses. After examining the first-order associations between significant predictors, we constructed an interaction term to test the conditional association of PCA on the association between SL and posttrauma nightmare occurrence. This choice was informed by prior research that has demonstrated a bidirectional association between SL and cognitive arousal, where cognitive arousal can impact the time it takes to fall asleep, and increased SL can exacerbate anxiety symptoms (Wicklow & Espie, 2000; Zoccola, Dickerson, & Lam, 2009). To test for potential moderating associations, an interaction term was created and added to the final model as a Level 1 predictor.

Results

Posttrauma Nightmare Occurrence

During the course of the study, five participants (POTEC: $n = 3$; clinical: $n = 2$) reported experiencing no posttrauma nightmares, 15 participants (POTEC: $n = 6$; clinical: $n = 9$) reported experiencing one posttrauma nightmare, six participants (POTEC: $n = 3$; clinical: $n = 3$) reported experiencing two posttrauma nightmares, and one participant (clinical group) reported experiencing three posttrauma nightmares. A 2×4 chi-square test of independence with a Yate's correction for continuity revealed no statistical difference between groups regarding nightmare frequency, $s\gamma^2(3, N = 27) = .101$, $p = .992$. Thus, the study captured 30 posttrauma nightmares nested within 27 participants.

Table 3 shows the descriptive statistics for each independent variable, stratified by the occurrence of a posttrauma nightmare. To statistically compare predictors by nightmare occurrence, predictor data were aggregated within each person and

Table 3
Predictors Stratified by Posttrauma Nightmare Occurrence

	Nightmare did not occur		Nightmare occurred	
Variable	*M*	*SD*	*M*	*SD*
Fatigue	3.03	2.24	3.81	2.69
Presleep cognitive arousal	12.57**	7.38	16.96**	8.27
Presleep somatic arousal	5.05	3.82	6.19	3.86
PSQI subscale				
Subjective Sleep Quality	1.41	0.76	1.28	0.66
Sleep Efficiency	1.29	.72	1.61	1.03
Daytime Dysfunction	2.35	1.63	2.54	1.73
Sleep Duration	0.70	0.91	1.06	1.25
Sleep variable				
Total sleep time (min)	462.63**	91.99	388.85**	108.48
Sleep latency	1.28**	0.78	1.77**	0.76
Sleep latency (min)	30.3**	14.91	62.4**	46.76

Note. $N = 27$ (146 observations). p values correspond to t-test results. PSQI = Pittsburgh Sleep Quality Inventory.
*$p < .05$. **$p < .01$.

compared between groups using paired t tests and chi-square analyses. The average time of posttrauma nightmare occurrence was 3 hr 35 min after falling asleep (i.e., 215.04 min). The average sleep-onset latency time on nights when posttrauma nightmares occurred was 62.4 min, which was significantly longer than the average time on nights when posttrauma nightmares did not occur ($M = 30.3$ min), $t(df = 145) = 5.605$ $p < .001$. The average total sleep time on nights when posttrauma nightmares occurred was 388.85 min, which was significantly shorter than the average time on nights when posttrauma nightmares did not occur (462.63 min), $t(df = 145) = 10.22$, $p < .001$.

First-Order Predictors of Posttrauma Nightmare Occurrence

The Level 2 grouping variable (i.e., participants) had an ICC score of .08, suggesting that 8% of the variance in the results could be explained by differences between participants, thus supporting the use of MLM analyses. After utilizing a top-down model-building procedure, the final model included the Level 1 fixed effects of PCA and SL as well as the Level 2 random intercept for participant code (u_{0j}). Model-building results are presented in Table 1. The results of the mixed-effects logistic regressions showed that SL, $\gamma_{10}\text{SL}_{ij} = 0.76$, $p < .001$, $z(1, N = 146) = -2.69$; and PCA, $\gamma_{20}\text{PCA}_{ij} = 0.58$, $p = .006$, $z(1, N = 146) = -2.61$, significantly predicted the occurrence of nightmares. Odds ratios revealed that for each 1-unit increase in PCA, participants were 1.75 times more likely to experience a posttrauma nightmare, and for each 1-unit increase in SL, participants were 2.09 times more likely to experience a posttrauma nightmare.

Moderation

To test moderation, the interaction term PCA x SL was created and entered into a regression equation along with the first-order terms, PCA and SL. In this newly created combined model, only the interaction term was significant, $\gamma_{30}\text{PCA x SL}_{ij} = 0.67$, $p = .048$ $z(1, N = 146) = 1.98$. The analyses revealed that when SL increased and PCA levels were high, participants were most likely to have a nightmare. However, when levels of SL were low, both high and low levels of PCA had similar effects. For a depiction of these results, see Figure 1.

Discussion

The present study was the first, to our knowledge, to investigate the predictors of posttrauma nightmares in a sample of college women with a reported history of sexual trauma. The results showed that the PCA × SL interaction predicted the occurrence of posttrauma nightmares. Specifically, the results revealed that when both PCA and SL levels were high, individuals were most likely to experience a posttrauma nightmare. However, before theorizing about the nature of this interaction, we will first consider the first-order predictors PCA and SL,

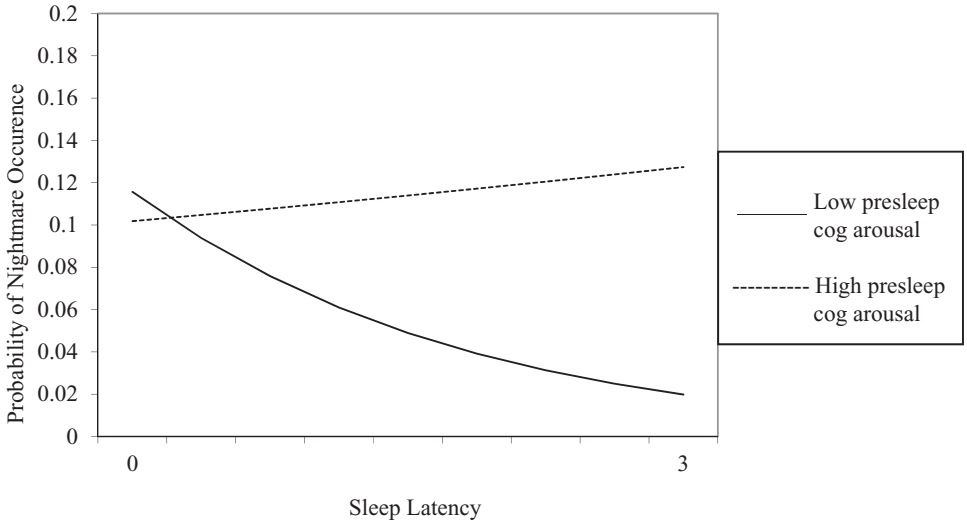

Figure 1. Interaction of presleep cognitive arousal and sleep latency. Cog arousal = cognitive arousal.

both of which significantly predicted posttrauma nightmare occurrences in the absence of the interaction term, independently as a stepping stone to understanding their combined effect.

The present findings showed that PCA, reported before bedtime, significantly predicted the occurrence of posttrauma nightmares. The mechanism that links PCA to the production of posttrauma nightmares cannot be precisely known given the limitations of our data. Consistent with the continuity hypothesis, the exact thoughts involved during the bouts of increased PCA, such as rumination about the traumatic event or nightmare apprehension, may have primed the cognitions that were later transformed into a posttrauma nightmare. In other words, reviewing or pondering posttrauma nightmare content before falling asleep might have been part of the mechanistic process for the production of a posttrauma nightmare. The continuity hypothesis has been supported by research on dreams showing that thinking of a stimulus prior to falling asleep makes it more likely for the "thought-of" stimuli to manifest in dream content (Graveline & Wamsley, 2015). Strauch and Meier (1996) demonstrated that after REM sleep awakenings, participants reported that up to 60% of dream content consisted of stimuli experienced prior to falling asleep. Following the continuity hypothesis, PCA may predict the occurrence of posttrauma nightmares because presleep cognitions prime the brain to create similar dream content during sleep.

Although SL was found to be a significant predictor of nightmares, our study was descriptive and based on self-report; thus, we could not fully determine the precise role of SL in the production of nightmares. One possible explanation is that a longer SL period might have directly influenced sleep architecture. The inability to fall asleep might have led to increased sleep pressure, which could have then resulted in REM rebound and led to faster and more direct entry to REM sleep (Borbely & Neuhaus, 1979; Cajochen, Knoblauch, Kräuchi, Renz, & Wirz-Justice, 2001). Given that at least some posttrauma nightmares occur during REM sleep, if SL did alter sleep architecture, it is possible that SL propelled participants into a stage of sleep during which posttrauma nightmares were more likely to occur.

Although PCA and SL were found to be first-order predictors of nightmares, their predictive power was qualified by the second-order interaction term. The explanation for this finding may lie in the summation of the independent processes. The concurrent occurrence of PCA and SL may have simultaneously primed the dreamer's dream content (i.e., the continuity hypothesis); provided a prolonged and uninterrupted period for PCA, potentially in the form of rumination or worry about the trauma or nightmare; and, as a result of sleep debt, made the dreamer more likely to experience a stage of sleep (i.e., REM) during which posttrauma nightmares and dreams commonly occur. Thus, the combination of PCA, which presumably reflects a focus on trauma- or nightmare-related content, and extended processing time during SL may have created the optimal environment for the production of a posttrauma nightmare. This rationale is consistent with a large body of literature that suggests a bidirectional relation between PCA and SL, whereby SL has been demonstrated to provide extended time for cognitive processing, and high levels of cognitive arousal have been reported to increase the difficulty of falling asleep (Zoccola et al., 2009). With this theoretical explanation of the interaction results, we propose a model that suggests that the co-occurrence of PCA and SL may be crucial to the production of a posttrauma nightmare.

Accordingly, our Nightmare Cognitive Arousal Processing (Night-CAP) model suggests that it was the intersection of PCA and SL that jointly influenced the occurrence of posttrauma nightmares in this sample of highly distressed female college students who reported a history of sexual trauma. The Night-CAP model supplements extant research that has identified individual differences that may make some people (i.e., the *who*) more vulnerable to posttraumatic symptoms following

the experience of a traumatic event and the neurological mechanisms involved in their occurrence (i.e., the *how*) by specifying the cognitive and behavioral indications of *when* posttrauma nightmares are likely to occur. The Night-CAP model proposes that posttrauma nightmares occur when PCA delays sleep onset, which also provides extended time for increased negative cognitions that can prime dream content.

This was the first study of which we are aware that utilized mixed-effects methods to identify night-to-night predictors of posttrauma nightmares. Empirical research on dreaming and nightmares is challenging because nightmares are elusive and have often failed to manifest during periods of measurement (Woodward et al., 2000). Although PSG measurement would have allowed us to more precisely characterize the posttrauma nightmares in terms of the stage of sleep during which they occurred, it seems likely that the minimally intrusive sleep measurement methodology used in the present study enabled us to capture nightmares more reliably than if we had used more intrusive measurement techniques. However, it should be noted that the study relied entirely on self-report measures, which are less accurate assessments of sleep onset than polysomnography. Thus, in order to address the middle ground between invasive tools and self-report measures, future research should attempt to use less invasive physiological sleep monitors.

Other limitations limit the generalizability and the precision with which we can interpret our findings. The present study's sample consisted of college-aged female sexual trauma survivors. The results may not be generalizable to other genders, ages, or individuals who have experienced traumatic events other than sexual traumas. For example, given the common association between sex and beds, it is not unfathomable to reason that sexual assault survivors may be more triggered and experience more symptoms while they are in bed compared with survivors of nonsexual trauma. Thus, future research should attempt to replicate these results within populations of survivors of different traumatic events. Although there was no significant difference in posttrauma nightmare occurrence rates between referral groups, it should also be noted that the clinical group reported significantly higher levels of anxiety and depression symptoms. Thus, the difference in depression and anxiety symptoms did not act as a statistical confound. Lastly, the limited sample size should be considered when interpreting the results. It is possible that with more power, we could have detected additional significant predictors. Despite these limitations, the present results represent a first step and provide a methodological model for identifying triggers of posttrauma nightmares.

In summary, our results and the Night-CAP model demonstrate that PCA and SL individually and together can significantly predict the occurrence of a posttrauma nightmare. These results provide meaningful knowledge that can be translated to treatment by suggesting that treatment of SL and PCA may indirectly reduce the chance of posttrauma nightmare occurrences. In fact, current posttrauma nightmare treatments, such as exposure, relaxation, and rescripting therapy (ERRT) that successfully decrease rumination and SL have been shown to decrease the frequency of posttrauma nightmare occurrence and PTSD severity (Balliett, Davis, & Miller, 2015), supporting the idea that these variables may be crucial not only to the manifestation of posttrauma nightmares but to treatment as well.

References

Balliett, N. E., Davis, J. L., & Miller, K. E. (2015). Efficacy of a brief treatment for nightmares and sleep disturbances for veterans. *Psychological trauma: Theory, Research, Practice, and Policy*, *7*, 507–515. https://doi.org/10.1037/tra0000055

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Beck Depression Inventory–II*. San Antonio, TX: Psychological Corporation. https://doi.org/10.1037/t00742-000

Beck, A. T., Epstein, N., Brown, G., & Steer, R. A. (1988). An inventory for measuring clinical anxiety: Psychometric properties. *Journal of Consulting and Clinical Psychology*, *56*, 6. https://doi.org/10.1037//0022-006x.56.6.893

Bonnet, H., & Arand, D. L. (2010). Hyperarousal and insomnia: State of the science. *Sleep Medicine Reviews*, *1*, 97–108. https://doi.org/10.1016/j.smrv.2009.05.002

Borbely, A. A., & Neuhaus, H. U. (1979). Sleep-deprivation: Effects on sleep and EEG in the rat. *Journal of Comparative Physiology*, *133*, 71–87. https://doi.org/10.1007/bf00663111

Borders, A., Rothman, D. J., & McAndrew, L. M. (2015). Sleep problems may mediate associations between rumination and PTSD and depressive symptoms among OIF/OEF veterans. *Psychological Trauma: Theory, Research, Practice, and Policy*, *7*, 76. https://doi.org/10.1037/a0036937

Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: Aa new instrument for psychiatric practice and research. *Psychiatry Research*, *11*, 735–742. https://doi.org/10.1016/0165-1781(89)90047-4

Cajochen, C., Knoblauch, V., Kräuchi, K., Renz, C., & Wirz-Justice, A. (2001). Dynamics of frontal EEG activity, sleepiness and body temperature under high and low sleep pressure. *Neuroreport*, *12*, 2277–2281. https://doi.org/10.1097/00001756-200107200-00046

Carskadon, M. A., & Dement, W. C. (2005). Normal human sleep: An overview. *Principles and Practice of Sleep Medicine*, *4*, 13–23. https://doi.org/10.1016/b0-72-160797-7/50009-4

Davis, J. L., Wright, D. C., & Borntrager, C. (2001*). The Trauma-Related Nightmare Scale*. Tulsa, OK: University of Tulsa.

Davis, J. L. (2008). *Treating post-trauma nightmares: A cognitive behavioral approach*. New York, NY: Springer Publishing Company.

Dietch, J. R., Ruggero, C. J., Schuler, K., Taylor, D. J., Luft, B. J., & Kotov, R. (2019). Posttraumatic stress disorder symptoms and sleep in the daily lives of World Trade Center responders. *Journal of Occupational Health Psychology*, *24*, 689–702. https://doi.org/10.1037/ocp0000158

Ehring, T., Frank, S., & Ehlers, A. (2008). The role of rumination and reduced concreteness in the maintenance of posttraumatic stress disorder and depression following trauma. *Cognitive Therapy and Research*, *32*, 488–506. https://doi.org/10.1007/s10608-006-9089-7

Faraway, J. J. (2016). *Extending the linear model with R: Generalized linear, mixed effects and nonparametric regression models* (Vol. 124). Boca Raton, FL: CRC press.

Finch, W. H., Bolin, J. E., & Kelley, K. (2016). *Multilevel modeling using R*. Boca Raton, FL: CRC Press.

First, M. B., Williams, J. B. W., Karg, R. S., & Spitzer, R. (2015). *Structured Clinical Interview for DSM-5, research version*. Arlington, VA: American Psychiatric Association.

Forbes, D., Phelps, A., & McHugh, T. (2001). Treatment of combat related nightmares using imagery rehearsal: A pilot study. *Journal of Traumatic Stress*, *14*, 433–442. https://doi.org/10.1023/a:1011133422340

Galatzer-Levy, I. R., Nickerson, A., Litz, B. T., & Marmar, C. R. (2013). Patterns of lifetime PTSD comorbidity: A latent class analysis. *Depression and Anxiety*, *30*, 489–496. https://doi.org/10.1002/da.22048

Germain, A. (2013). Sleep disturbances as the hallmark of PTSD: Where are we now. *American Journal of Psychiatry*, *170*, 372–382. https://doi.org/10.1176/appi.ajp.2012.12040432

Grandner, M. A., Kripke, D. F., Yoon, I. Y., & Youngstedt, S. D. (2006). Criterion validity of the Pittsburgh Sleep Quality Index: Investigation in a non-clinical sample. *Sleep and Biological Rhythms*, *4*, 129–136. https://doi.org/10.1111/j.1479-8425.2006.00207.x

Graveline, Y. M., & Wamsley, E. J. (2015). Dreaming and waking cognition. *Translational Issues in Psychological Science*, *1*, 97–105. https://doi.org/10.1037/tps0000018

Hartmann, E. (1998). *Dreams and nightmares: The new theory on the origin and meaning of dreams*. New York, NY: Plenum Trade.

Kerkhof, G. A., & Van Dongen, H. P. A. (2010). Effects of sleep deprivation on cognition. *Progress in Brain Research*, *185*, 105–129. https://doi.org/10.1016/b978-0-444-53702-7.00007-5

Kobayashi, I., Boarts, J. M., & Delahanty, D. L. (2007). Polysomnographically measured sleep abnormalities in PTSD: A meta-analytic review. *Psychophysiology*, *44*, 660–669. https://doi.org/10.1111/j.1469-8986.2007.00537.x

Koffel, E., Khawaja, I. S., & Germain, A. (2016). Sleep disturbances in posttraumatic stress disorder: Updated review and implications for treatment. *Psychiatric Annals*, *46*, 173–176. https://doi.org/10.3928/00485713-20160125-01

Krizan, Z., & Hisler, G. (2018). Sleepy anger: Restricted sleep amplifies angry feelings. *Journal of Experimental Psychology*, *148*, 1239–1250. https://doi.org/10.1037/xge0000522

Levin, R., & Nielsen, T. A. (2007). Disturbed dreaming, posttraumatic stress disorder, and affect distress: a review and neurocognitive model. *Psychological Bulletin*, *133*, 482–528. https://doi.org/10.1037/0033-2909.133.3.482

Levin, R., & Nielsen, T. (2009). Nightmares, bad dreams, and emotion dysregulation: A review and new neurocognitive model of dreaming. *Current Directions in Psychological Science*, *18*, 84–88. https://doi.org/10.1111/j.1467-8721.2009.01614.x

Michael, T., Ehlers, A., Halligan, S. L., & Clark, D. M. (2005). Unwanted memories of assault: What intrusion characteristics are associated with PTSD. *Behavior Research and Therapy*, *43*, 613–628. https://doi.org/10.1016/j.brat.2004.04.006

Michielsen, H. J., De Vries, J., & Van Heck, G. L. (2003). Psychometric qualities of a brief self-rated fatigue measure: The Fatigue Assessment Scale. *Journal of Psychosomatic Research*, *54*, 345–352. https://doi.org/10.1016/s0022-3999(02)00392-6

Nadorff, M., Nazem, S., & Fiske, A. (2013). Insomnia symptoms, nightmares, and suicide risk: Duration of sleep disturbance matters. *Suicide and Life-Threatening Behavior*, *43*, 139–149. https://doi.org/10.1111/sltb.12003

Nicassio, P. M., Mendlowitz, D. R., Fussell, J. J., & Petras, L. (1985). The phenomenology of the presleep state: The development of the presleep arousal scale. *Behaviour Research and Therapy*, *23*, 263–271. https://doi.org/10.1037/t11747-000

Nielsen, T., & Levin, R. (2007). Nightmares: A new neurocognitive model. *Sleep Medicine Reviews*, *11*, 295–310. https://doi.org/10.1016/j.smrv.2007.03.004

Ohayon, M. M., & Shapiro, C. M. (2000). Sleep disturbances in psychiatric disorders associated with posttraumatic stress disorder in the general population. *Comprehensive Psychiatry*, *41*, 469–478. https://doi.org/10.1053/comp.2000.16568

Pacella, M. L., Hruska, B., & Delahanty, D. L. (2013). The physical health consequences of PTSD and PTSD symptoms: A meta-analytic review. *Journal of Anxiety Disorders*, *27*, 33–46. https://doi.org/10.1016/j.janxdis.2012.08.004

Phelps, A. J., Kanaan, R. A., Worsnop, C., Redston, S., Ralph, N., & Forbes, D. (2017). An ambulatory polysomnography study of the posttraumatic nightmares of post-traumatic stress disorder. *Sleep*, *41*, zsx188. https://doi.org/10.1093/sleep/zsx188

Schneider, A., & Domhoff, G. W. (2001). *Quantitative study of dreams*. Santa Cruz, CA: University of California, Santa Cruz.

Schredl, M. (2006). Factors affecting the continuity between waking and dreaming: Emotional intensity and emotional tone of the waking-life event. *Sleep and Hypnosis*, *8*, 1.

Simms, L. J., Watson, D., & Doebbelling, B. N. (2002). Confirmatory factor analyses of posttraumatic stress symptoms in deployed and nondeployed veterans of the Gulf War. *Journal of Abnormal Psychology*, *111*, 637–647. https://doi.org/10.1037/0021-843x.111.4.637

Strauch, I., & Meier, B. (1996). *In search of dreams: Results of experimental dream research*. New York, NY: SUNY Press. https://doi.org/10.5860/choice.33-5995

Van der Kolk, B., Blitz, R., Burr, W., Sherry, S., & Hartmann, E. (1984). Nightmares and trauma: A comparison of nightmares after combat with lifelong nightmares in veterans. *American Journal of Psychiatry*, *141*, 187–190. https://doi.org/10.1176/ajp.141.2.187

Vermetten, E., Germain, A., & Neylan, T. C. (2018). *Sleep and combat-related post traumatic stress disorder*. New York, NY: Springer. https://doi.org/10.1007/978-1-4939-7148-0

Weathers, F. W., Litz, B. T., Keane, T. M., Palmieri, P. A., Marx, B. P., & Schnurr, P. P. (2013). *The PTSD Checklist for DSM-5 (PCL-5)*. Scale available from http://ptsd.va.gov

Wicklow, A., & Espie, C. A. (2000). Intrusive thoughts and their relationship to actigraphic measurement of sleep: Towards a cognitive model of insomnia. *Behaviour Research and Therapy*, *38*, 679–693. https://doi.org/10.1016/s0005-7967(99)00136-9

Woodward, S. H., Arsenault, N. J., Murray, C., & Bliwise, D. L. (2000). Laboratory sleep correlates of nightmare complaint in PTSD in patients. *Biological Psychiatry*, *48*, 1081–1087. https://doi.org/10.1016/s0006-3223(00)00917-3

Woodward, S. H., Arsenault, N. J., Voelker, K., Nguyen, T., Lynch, J., Skultety, K., & Sheikh, J. I. (2009). Autonomic activation during sleep in posttraumatic stress disorder and panic: A mattress actigraphic study. *Biological Psychiatry*, *66*, 41–46. https://doi.org/10.1016/j.biopsych.2009.01.005

Zoccola, P. M., Dickerson, S. S., & Lam, S. (2009). Rumination predicts longer sleep onset latency after an acute psychosocial stressor. *Psychosomatic Medicine*, *71*, 771–775. https://doi.org/10.1097/psy.0b013e3181ae58e8