

## **Testing the Nightmare Cognitive Arousal Processing Model**

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### **Author Note**

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## Abstract

**Objective:** Posttrauma nightmares are recurring nightmares that begin after a traumatic experience. Research has only recently begun to identify variables that predict posttrauma nightmare occurrences. Research has identified presleep cognitive (PSA-C) and physiological (PSA-P) arousal, sleep onset latency (SOL), and sleep disordered breathing (SDB) as potential predictors of posttrauma nightmares. However, previous research includes methodological limitations, such as a lack of physiological measures and a homogeneous sample. To replicate previous findings and increase generalizability, the current study investigated predictors of nightmare occurrences in a sample of male inpatient veterans with mixed-trauma history.

**Method:** Participants ( $n = 15$ ) completed an initial assessment battery and 7 consecutive days of pre and postsleep diaries, including measures of posttrauma nightmare triggers and posttrauma nightmare occurrences. Portable objective measurements of sleep and presleep states were used to examine sleep quality and physical arousal.

**Results:** Analyses revealed that PSA-C and SOL both predicted posttrauma nightmare occurrences and that PSA-P was significantly higher on nights when nightmares occurred.

**Conclusion:** Results replicate earlier research which posits that PSA and SOL play a role in triggering the occurrence of posttrauma nightmares. It should be noted that the sample was relatively small, warranting cautious interpretation of results. However, when taken together with the findings of the replicated study, results could suggest the plausibility of therapies targeting presleep cognitions, sleep onset latency, and presleep arousal in the treatment of posttrauma nightmares.

**Keywords:** Nightmares; veterans; trauma; sleep diaries; multilevel modeling

### **Clinical Impact Statement**

The results of our study highlight the severity and prevalence of nightmares within trauma-exposed populations, such as veterans. Second, our results identify variables that may predict posttrauma nightmare occurrences, which could then be used to inform current interventions focused on treating nightmares within trauma exposed populations. This is particularly important as current treatment options for posttrauma nightmares remain sparse and are less effective than treatments for other sleep disorders, such as insomnia. Thus, our results not only provide novel insights into posttrauma nightmares, but they also produce clinically relevant findings that could be used to directly inform and improve upon current treatment options.

## **Testing the Nightmare Cognitive Arousal Processing Model**

### **Posttrauma Nightmares**

Posttrauma Nightmares are one of the most problematic yet poorly understood symptoms associated with posttraumatic stress disorder (PTSD). Posttrauma nightmares have been commonly operationalized as recurring nightmares that begin after a traumatic event (triggering trauma; Hartmann, 1984). These unique parasomnias are a nearly omnipresent symptom of PTSD, with up to 96% of patients with PTSD reporting frequent nightmares (Germain, 2013; Vermetten et al., 2018). Posttrauma nightmares are quite problematic, with research linking these nightmares to insomnia, depression, anxiety, substance abuse, and suicide (Galatzer-Levy et al., 2013; Nadorff et al., 2013; Pacella et al., 2013; Van der Kolk et al., 1984). Furthermore, existing research demonstrates that both pharmacological and behavioral interventions have mixed efficacy in regards to treating nightmares, with some research reporting positive effects and other research reporting little impact on posttrauma nightmares (El-Solh, 2018; Raskind et al., 2018; Reist et al., 2021). Given the frequency, severity, and resiliency of posttrauma nightmares, clinically relevant information is needed to develop more effective interventions and enhance patient care. One area that may warrant further investigation is the examination of variables that trigger nightmare occurrences.

### **Predictors of Nightmare Occurrences**

One of the first modern scientific nightmare models to explain how nightmares occur was the Affective Network Dysfunction theory (AND; Levin & Nielsen, 2007). The AND theory suggested that several neurological systems related to affect (e.g., amygdala) interact with arousal promoting systems (i.e., HPA-Axis) to produce distressing dreams and nightmares (Levin & Nielsen, 2007; the relationship between these systems is commonly referred to as

AMPHAC). The AMPHAC/AND theory was supported by neuroscientific research which showed that the same affect systems (including the amygdala) and arousal systems (HPA-Axis) are highly active during dreaming, sleep, and fear exposures (Hull, 2002; Morgane et al., 2005). Although more evidence is needed to support this model, the AMPHAD/AND provides a framework for the investigation of arousal and affect, as the main predictors of posttrauma nightmares.

Using the AMPHAC/AND theory as a guiding framework, Youngren and colleagues (2020) investigated the role that affect and arousal play in predicting posttrauma nightmare occurrences. Youngren and colleagues (2020) found that Presleep Cognitive Arousal (such as worry and rumination; PSA-C) and Sleep Onset Latency (SOL) predicted the occurrence of posttrauma nightmares. Post-hoc analyses revealed that posttrauma nightmares were most likely to occur when both SOL and PSA-C were high. This led Youngren and colleagues (2020) to propose the Nightmare Cognitive Arousal Processing Model (NIGHT-CAP). NIGHT-CAP theorizes that presleep negative cognitions (such a rumination or worry) increase the time it takes to fall asleep (SOL). During this prolonged attempt to fall asleep, the dreamer has ample time to continue ruminating or worrying, which ultimately primes the dreamer to have a nightmare once they fall asleep. The NIGHT-CAP model aligns with previous research that examined nightmare occurrences, such as Short et al. (2017) who also found that cognitive factors (such as fear of sleep) predicted posttrauma nightmares. However, the study had limited generalizability because of a homogeneous sample (i.e., females only), a single trauma type, and a lack of physiological measures of both sleep quality and arousal.

Generalizability of the NIGHT-CAP model could also be complicated by comorbid sleep disordered breathing. For example, Miller et al. (2018) examined physiological markers of

posttrauma nightmares in inpatient veterans. Using actigraphy and an ambulatory respiratory monitor, Miller and colleagues found that sleep disordered breathing (SDB) and physiological arousal independently predicted the onset of nightmares. These findings were consistent with emerging literature suggesting that there may be two types of trauma-related nightmares: those exacerbated by SDB and those in the absence of SDB (Youngren, Balderas, & Farrell-Higgins, 2021). The aforementioned research serves as a caution to control for SDB as a potential confound in posttrauma nightmare investigations. Regarding physiological arousal, however, Miller et al.'s (2018) results are consistent with the AMPHAC/AND model, which proposes a key role for physical arousal in the generation of nightmares.

### **Purpose**

The purposes of the present study were to replicate the findings of the original Youngren et al. (2020) NIGHT-CAP study (i.e., that nightmares can be predicted by presleep arousal, sleep latency, and the interaction of the two), to investigate the generalizability of the NIGHT-CAP model (via a sample of male veterans), to address measurement-based limitations, and to actively account for the effects of SDB discussed by Miller et al. (2018). By adding a physiological monitor of presleep arousal and sleep quality, using a male population, and actively controlling for SDB, this study aimed to replicate, extend, and strengthen the results of the initial NIGHT-CAP Model.

### **Methods**

#### **Participants**

Male trauma survivors were recruited from a local Veterans Affairs (VA) Hospital's 6-week inpatient PTSD treatment unit. Interested participants were screened for eligibility. Inclusionary criteria were as follows: a) self-reported experience of a trauma, b) experiencing

nightmares related to a trauma, and c) Over the age of 18. Exclusionary criteria: a) currently experiencing PTSD with dissociative/psychotic features. Eligible participants completed the study protocol described below and were not compensated for participation.

## **Procedure**

First, the participants signed a statement of informed consent that explained the purpose and procedure of the study. Immediately following the completion of the informed consent form, a trained clinical researcher reviewed the patient's medical chart and administered a battery of questionnaires used to assess the potential presence of SDB, suicidal ideation, and psychosis. After the initial battery, participants were given the DREEM Headband (DH) and watched a brief video produced by the company that explained how to wear the device and use it to track sleep. After the DH introduction, participants were then instructed to complete paper-pencil surveys every night immediately before going to bed (presleep surveys) and every morning immediately after awakening (postsleep surveys). They were instructed to complete pre and postsleep surveys and wear the DREEM Headband (DH) for seven consecutive days and nights. After completing seven consecutive days and nights of assessment, participants met with the research coordinator for debriefing.

## **Measures**

### ***Diagnostic Interview, Initial Battery, and Chart Reviews***

An assessment of psychological symptoms, demographic variables, and estimated SDB occurred immediately following the informed consent process. The initial interview/battery/chart reviews included assessments of demographic characteristics, SDB symptoms, and PTSD diagnosis.

### ***PTSD Symptomatology***

PTSD symptomology was assessed via electronic medical records. Prior to entering the inpatient unit, all participants were assessed for active PTSD and had their PTSD diagnosis updated on their Electronic Medical Record (EMR).

### ***Sleep Disordered Breathing***

Estimated SDB was assessed using one of two methods. If a current SDB diagnosis (such as obstructive sleep apnea; OSA) was reported in the participant's EMR, this diagnosis served as a measure of SDB. For individuals who did not have a current SDB diagnosis, the Berlin Questionnaire was used to assess for symptoms related to SDB (Netzer et al., 1999). The Berlin Questionnaire is a 9-item scale that comprises 3 categories related to the risk of having SDB. Scores derived from the Berlin Questionnaire can be used to classify *High Risk*, *Low Risk*, and *No Risk* SDB. The Berlin Questionnaire has been demonstrated to have extremely high test-retest reliability ( $r = 0.92$ ) and moderately high sensitivity (68.9–87.2%) for screening SDB cases, such as Obstructive Sleep Apnea (OSA; Chung et al., 2008). Participants were coded as estimated SDB if their scores were considered *High Risk* for SDB ( $2 \geq$  categories with a positive score).

### ***Demographic Variables***

Demographic variables were measured in the initial survey. Demographic variables included measures of age, gender, ethnicity, and trauma history.

### ***Presleep Surveys***

Participants completed a presleep survey each night, immediately before going to bed for seven consecutive days. As part of the survey, participants were asked to report the time and date of completion, which were then used to assess for protocol adherence. Presleep surveys included only a measure of presleep arousal (both cognitive and somatic).



**Presleep Arousal.** Presleep arousal was assessed with the Presleep Arousal (PSA) scale, a 16-item measure with two subscales, somatic (PSA-S) and cognitive (PSA-C) presleep arousal states. Participants responded to items on a scale ranging from 1 (*not at all*) to 5 (*extremely*). Higher scores indicated higher states of presleep arousal (Nicassio et al., 1985). PSA-S was created by totaling items 1-8, while PSA-C was created by totaling items 9-16. A total presleep arousal score was created by totaling items 1-16. The PSA demonstrated acceptable to high levels of reliability in the current study (total  $\alpha = .85$ ; PSA-S,  $\alpha = .79$ ; PSA-C,  $\alpha = .88$ ; Nicassio et al., 1985).

### ***Postsleep Surveys***

Each morning, participants completed a postsleep survey immediately after waking up for seven consecutive days. As part of the survey, participants were asked to report the time and date, which were then used to assess for protocol adherence. Postsleep surveys included measures of recalled presleep cognitions and SOL, as well as reports of dreams/nightmares. Postsleep measures can be found in the Measures Appendices.

**SOL.** SOL was measured every morning through self-report via an amended version of the Pittsburgh Sleep Quality Inventory (PSQI; Buysse et al., 1989). PSQI instructions were edited to capture the previous night's sleep (compared to the past month's sleep), by replacing references to "past month" with references to "last night". Item #2 was used to assess SOL. Item #2 asks, "Last night, how long (in minutes) did it take to fall asleep?" The PSQI has demonstrated strong validity with past studies reporting significant correlations with actigraphy ( $r = 0.31, p < 0.01$ ) and sleep surveys ( $r = -0.56, p < 0.01$ ; Grandner et al., 2006). Although psychometrics do not exist for item #2 alone, it has become common practice for

nightmare/sleep studies investigating SOL to use item #2 independently as self-report measure of SOL (Davis, 2008; Margolies et al., 2013; Youngren et al., 2020).

**Nightmares and Dreams.** Nightmares and dream content were assessed via procedures similar to the initial NIGHT-CAP study (Youngren et al., 2020). Participants were asked a series of questions about their dreams. First, participants were asked whether they remembered their dreams last night (Y, N). If “Y”, they were then asked whether any of these dreams were nightmares that woke them up and were related to their triggering trauma (Y, N). This scale was based on recommendations from Schneider and Domhoff (2001) and followed a structure similar to the Trauma Related Nightmare Scale (Davis et al., 2001), a larger scale intended to assess posttrauma nightmares. These protocols were used in the initial NIGHT-CAP study to identify the occurrence of 30 posttrauma nightmares.

### ***DREEM Device***

DREEM Headbands (DH) were used to measure physiological aspects of sleep and presleep arousal. The DH is a portable headband that can be used as an ambulatory measure of sleep quality. Embedded within the DH are five dry EEG electrodes, a pulse oximeter, and an accelerometer. These sensors allow the DH to identify sleep, measure SOL, and pulse rate, an index of physiological arousal. The DH signal detection ability has demonstrated criterion validity via comparisons to polysomnography, with an overall accuracy rate of  $83.5 \pm 6.4\%$  (Arnal et al., 2019). DH presleep physiological arousal (PSA-PHYS) was determined by averaging the pulse oximeter scores 15 minutes prior to the onset of sleep (sleep onset was determined by a DREEM algorithm that detected sleep onset; Arnal et al., 2019).

### **Analyses**

Prior to analyses, data were cleaned and transformed into a quantitative Excel .CSV dataset. All the subsequent analyses were conducted using R statistical software version 4.2.2 (R CoreTeam, 2020). Once the data were transformed into a single CSV file, analyses were conducted to examine residuals and identify potential outliers. There were no outliers and missing data handling is discussed in the results section. Next, descriptive and prevalence statistics were calculated to examine the sample's characteristics. Afterward, descriptive statistics, *t*-tests, and chi-square tests were used to examine differences among predictor variables on nights when nightmares did and did not occur. However, given that our predictors were assessed repeatedly for individual participants, participant data were aggregated for *t*-tests and chi-square analyses.

Before hypotheses could be tested, an intraclass correlation (ICC) for participants was calculated to determine if multilevel modeling (MLM) methods were necessary (considering that predictors were assessed repeatedly for individual participants). Following the guidelines discussed in Finch et al. (2016), ICCs were within the range ( $ICC \geq 0.10$ ), indicating that MLM analyses were necessary for testing our hypotheses. After ICCs were calculated, a mixed effects logistic regression analysis using maximum likelihood estimation was used to explore whether our independent variables predicted the occurrence of posttrauma nightmares. The model was:

$$\begin{aligned} \text{logit}(y_{ij}) &= \beta_{0j} + \beta_{1j}X_{ij} \\ \beta_{0j} &= \gamma_{00} + u_{0j} \\ \beta_{1j} &= \gamma_{10} + u_{1j} \end{aligned} \quad \begin{bmatrix} u_{0j} \\ u_{1j} \end{bmatrix} \sim MVN \left( \begin{bmatrix} 0 \\ 0 \end{bmatrix}, \begin{bmatrix} \tau_{00} & \\ \tau_{01} & \tau_{11} \end{bmatrix} \right)$$

where the  $\beta$ s represent random coefficients,  $u$ s are level-2 errors,  $X$ s were fixed predictors (repeated for each IV; cognitive arousal, presleep somatic arousal, presleep thought content,

sleep latency, and SDB),  $y_{ij}$  were posttrauma nightmare occurrences, and  $j_s$  were level-2 unit identifiers (participant code).

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. For post hoc analyses, an interaction term was added to the final model to examine its predictive power (replicating previous analyses; Youngren et al., 2020).

Given limitations of in-person research during Covid-19, not all participants were able to wear a DH. Of the 15 male inpatient participants, only nine wore the DH. Data gathered using DH were further limited because of poor internet quality on the inpatient unit and by participant actions (e.g., not wearing the headband or removing it during night). As such, only  $N = 48$  nights of data were captured from the DH. Given that this was nearly half the amount of the data collected with the daily questionnaires ( $N = 80$ ), the DH data were analyzed separately from the self-report data using nonparametric statistics and repeated measure correlations in place of

MLM analyses. Aggregated chi-square tests and *t*-tests were used to examine differences within the DH data on nightmare and non-nightmare nights.

## Results

### Sample Characteristics

20 participants were enrolled in the research protocol. No participants experienced PTSD with dissociative or psychotic features. Following mixed-model missing data recommendations (Faraway, 2016), five male inpatient participants completed fewer than three pre- and postsleep surveys, so their data were removed from analyses. Six male inpatient participants completed between three and six continuous pre- and postsleep surveys and these data were retained for analyses. The remaining nine male inpatients completed all seven consecutive pre- and postsleep surveys and all of their data were retained for analyses. After the aforementioned data cleaning, our data used in all following analyses included  $N = 80$  repeated measurements collected from  $n = 15$  male inpatient participants.

Regarding nightmare occurrences, of the 15 participants included in analyses, two participants reported zero posttrauma nightmares, four participants reported one posttrauma nightmare, two participants reported two posttrauma nightmares, two participants reported three posttrauma nightmares, two participants reported four posttrauma nightmares, two participants reported five posttrauma nightmares, and one participant reported experiencing six posttrauma nightmares. In sum, 36 nightmares were captured between 15 analyzed participants. On average, participants reported experiencing 2.53 posttrauma nightmares over the course of up to 7 days.

The sample included  $N = 15$  male veterans who reported experiencing nightmares related to a traumatic event. Traumatic experiences within the sample were mixed (combat related trauma  $n = 7$ ; military sexual trauma [MST]  $n = 6$ ; non-combat non-MST  $n = 2$ ). The sample was

primarily middle aged,  $M = 48.53$  ( $SD = 12.32$ ) and a majority of the participants were white ( $n = 12$ , 80%; African American  $n = 3$ , 20%). All participants had a diagnosis of PTSD and all participants had at least one traumatic experience prior to the nightmare triggering trauma.

Nearly half of the sample ( $n = 7$ ) met criteria for estimated SDB.

### **Nightmare Nights vs non-Nightmare Nights**

SOL was measured by both self-report and the DH. The self-report data showed that the SOL was significantly longer on nights when nightmares occurred ( $M = 50.66$ ,  $SD = 31.84$ ) compared to nights when nightmares did not occur ( $M = 25.35$ ,  $SD = 16.50$ ;  $t = 11.77$ ,  $p < 0.01$ ). Consistent with self-report, SOL as measured by DHs was significantly longer on nights when nightmares occurred ( $M = 47.65$ ;  $SD = 19.56$ ) compared to nights when nightmares did not occur ( $M = 25.50$ ;  $SD = 18.31$ ;  $t = 11.06$ ,  $p < 0.05$ ). There was a significant correlation between SOL measured by self-report and SOL measured by DHs ( $r = 0.84$ ,  $p < 0.01$ ). For more information, see Tables 2 & 3.

Presleep physiological arousal was measured by both self-report (PSA-S) and the DH pulse oximeter (PSA-PHYS). Self-reported PSA-S was not significantly different on nights when nightmares occurred ( $M = 18.13$ ,  $SD = 3.62$ ) compared to nights when nightmares did not occur ( $M = 15.46$ ,  $SD = 3.61$ ;  $\chi^2 = 22.96$ ,  $p = 0.19$ ). In contrast, PSA-PHYS was significantly higher on nights when nightmares occurred ( $M = 79.87$ ,  $SD = 1.98$ ) compared to nights when nightmares did not occur ( $M = 73.00$ ,  $SD = 3.34$ ;  $t = 21.18$ ,  $p < 0.05$ ). There was not a significant correlation between self-reported PSA-S and PSA-PHYS ( $r = -0.07$ ,  $p = 0.71$ ). For more information, see Tables 2 & 3.

Presleep cognitions were measured with the PSA-C. Self-reported PSA-C was significantly different on nights when nightmares occurred ( $M = 27.21$ ,  $SD = 7.41$ ) compared to

nights when nightmares did not occur ( $M = 22.88$ ,  $SD = 6.29$ ;  $t = 38.74$ ,  $p < 0.05$ ). Descriptive information is presented in Table 3.

### **Predictive Analyses**

The Level 2 grouping variable (participants) had an ICC score of .57, suggesting that 57% of the variance in the results could be explained by differences between participants, thus supporting the use of MLM analyses. Using the top-down model-building procedure (which tested all proposed level 1 fixed effects) and controlling for estimated SDB, the final model included the Level 1 fixed effects of SOL and PSA-C as well as the Level 2 random intercept for participant code (u0j). Model-building results are presented in Table 1. The results of the mixed-effects logistic regressions showed that SOL,  $\gamma_{10\text{SOL}} = 0.05$ ,  $z(N = 79) = 2.64$ ,  $p < 0.01$ ; and PSA-C,  $\gamma_{20\text{PSA-C}} = 0.11$ ,  $z(N = 79) = 1.78$ ,  $p < 0.05$ , significantly predicted the occurrence of nightmares. Odds ratios revealed that for each 1-unit increase in SOL, the odds of a posttrauma nightmare occurring increased by a factor of 1.49, and for each 1-unit increase in PSA-C, the odds of a nightmare occurring increased by a factor of 2.39.

To test moderation, an interaction term SOL x PCA was created and entered into the final model which included SOL and PCA. In this newly created model, the interaction term was not significant,  $\gamma_{30\text{SOL} \times \text{PSA-C}} = 0.04$ ,  $z(1, N = 79) = 0.77$ ,  $p = .46$ .

### **Discussion**

Consistent with the predictions of the NIGHT-CAP model, the current study found that SOL and PSA-C predicted the occurrence of posttrauma nightmares even when controlling for estimated SDB. In addition to our primary outcomes, data gathered using DHs demonstrated that both objectively measured presleep physiological arousal and SOL were significantly higher on nights when nightmares occurred, compared to nights when they did not. Confidence in the

replicability of these results was enhanced by the utilization of mixed method assessment protocols (i.e., self-report and DH) and the high correlation between subjective and objective data. Our results align both with pre-existing research and theory and add support to the NIGHT-CAP Model (Youngren et al., 2020) as a theory that can be used to predict the occurrence of posttrauma nightmares.

### **SOL & Nightmare Occurrences**

SOL was found to be a significant predictor of nightmare occurrences. Although the DH data were limited, DH data were remarkably consistent with self-reports of SOL. Regardless of assessment method, SOL on nights when nightmares occurred was roughly twice as long as on nights when nightmares did not occur. On nights without nightmares, SOL was typically around 30 minutes, whereas on nightmare nights, SOL was roughly an hour. These findings closely replicate observations of SOL in the initial NIGHT-CAP study (Youngren, et al., 2020) and demonstrate that SOL may be a consistent signal of an impending nightmare.

Consistent with the initial NIGHT-CAP Model, SOL may be important in the production of nightmares because lying sleepless in bed offers extended processing time, without daytime distractions. The longer it takes to fall asleep, the greater opportunity to worry and/or think about traumatic events and/or anxiety provoking thoughts. This theory is consistent with broader dream literature which suggests that presleep cognitions play a pivotal role in determining dream content (Schredl & Hofmann, 2003). Thus, it may not be that extended SOL is directly involved with nightmare production. Instead, the extended time offered by increased SOL may simply provide time for negative cognitions to fester which then may lead to nightmare occurrences. However, it should be noted that this study did not find a significant interaction effect between



SOL and presleep arousal. Thus, the current study does not directly support the previous interpretation.

### **Presleep Cognitive Arousal & Nightmare Occurrences**

Our results indicate that presleep cognitions (PSA-C) played a crucial role in predicting nightmare occurrences. These results align theoretically with the Continuity Hypothesis, which posits that presleep cognitions (such as presleep cognitive arousal) play a large role in determining dream content (Hartmann, 2000; Schredl & Hofmann, 2003). Furthermore, our findings also align with the Garcia et al. (2017) study, which also found that daily stress was related to stress in dreams and predicted nightmare occurrences. Thus, our results were consistent with the Continuity Hypothesis and seem to suggest that negative presleep cognitions (captured by PSA-C) prior to falling asleep may prime the sleeping brain to produce a nightmare following sleep onset. This rationale aligns with pre-existing understandings of nightmare occurrences, such as the Vicious Cycle, which proposes the daytime variables such as stress and worry impact nightmare occurrences in a cyclical matter (Davis, 2008). In addition to aligning with preexisting conceptualizations, we also believe that the NIGHT-CAP model uniquely offers evidence of a temporal link between cognitive/affective arousal and nightmares, while the addition of SOL extends theoretical understanding of nightmares and is an easily measured objective biomarker.

Consistent with the Continuity Hypothesis, Fear of Sleep may be an example of how cognitive arousal impacts dream content and nightmare occurrences. This idea was first posed by Neylan et al. (1998) and was even recently supported by an EMA study that found Fear of Sleep to be a predictor of nights with nightmares (Short et al, 2018). When considering Fear of Sleep as a cognitive predictor of nightmare occurrences, future research examining presleep cognitive

arousal's role on nightmare occurrences should assess for arousal type and Fear of Sleep in order to examine specific cognitions related to nightmare occurrences.

### **Presleep Physiological Arousal & Nightmare Occurrences**

Self-reported PSA-S did not significantly predict the occurrence of posttrauma nightmares. However, objectively measured DH-presleep arousal (PSA-PHYS) differed on nights when nightmares occurred compared to nights when they did not (see Table 2). These results support the notion that physiological arousal may play a pivotal role in the manifestation of posttrauma nightmares. Although these results were significant, it must be noted that our sample size for DH measurements was quite small. As such, these results should be interpreted with caution. However, our results align with Miller et al. (2018) and the AMPHAC/AND model (Levin & Nielsen, 2007) which identified physiological arousal as a predictor of nightmares. Considering our findings, the limitations of our findings, and prior research, future research is needed to better understand how presleep physiological arousal impacts posttrauma nightmare occurrences.

### **Nightmare Cognitive Arousal Processing (NIGHT-CAP)**

Our findings both partially replicated, but also extends the NIGHT-CAP theory of nightmare production, which proposes that increased time spent trying to fall asleep (SOL) provides an increased opportunity for negative presleep cognitions (PSA-C), which in turn primes the dreamer to have a nightmare once they are asleep (Youngren et al., 2020). However, it should be noted that SOL and presleep arousal independently predicted nightmare occurrences, while interaction of the two was not significant. The current study also critically improved generalizability via recruitment of a demographically different sample (i.e., middle-aged male veterans) with different trauma types than the original study. Lastly, our study further developed

the NIGHT-CAP model by providing evidence that presleep physiological arousal plays a role in posttrauma nightmare production. Overall, the results of this study help support the NIGHT-CAP model as a theory that aims to explain why nightmares occur when they do.

### **Limitations & Strengths**

Although the results of this study supported the hypotheses generated by the NIGHT-CAP Model, there were several important limitations. One limitation of this study was the sample size. Although some effects were large enough to be detected with the smaller sample size, smaller effects (e.g., the interaction of SOL and PSA-C) were likely to be missed. Future research should be conducted with a larger sample size to more thoroughly examine these results and reexamine the interaction of SOL and PSA-C on nightmare occurrences. Another limitation of this study that impacted data collected was the DH. Due to issues with the DHs (e.g., participants removing the devices/devices falling off during the night), data were limited, which left us unable to add the DH data into the MLM analyses. Future research using the DH should consider some methods to increase the adherence to and utilization of these devices.

In addition to data, another limitation of the study was the study's utilization of only inpatient participants, which may have higher base rates of comorbidities than non-inpatient samples. Thus, future research should measure and account for additional comorbidities in order to ensure comorbid psychopathologies do not confound results. Similarly, patient use of psychiatric medications was not measured. Because of the prevalence of psychiatric medications in inpatient units and their possible impact on dream content, nightmares, and sleep (DeMartinis & Winokur, 2007), future research should assess for medications when using inpatient samples to better account for potential confounding variables. Lastly, patients completed study protocols while on an inpatient PTSD unit. Although participants had bedrooms to themselves, the shared

living space (inpatient unit) could have impacted sleep quality through unmeasured ways. The environment within inpatient settings (such as “lights out” time and nighttime checks) is unlikely to be consistent with participant’s sleeping schedule, thus posing another complication for conducting sleep research within an inpatient setting. Despite these limitations, the limited usage of the DH devices provided some objective measures to the NIGHT-CAP Model.

Conversely, a strength of this study was controlling for the presence of SDB following recommendations from recent research (Miller et al., 2018; Mysliwiec et al., 2018). Here we show that even in the presence of SDB symptoms, extended sleep latency and cognitive arousal were the most robust predictors of posttrauma nightmares. Given the results of our study and the intricate relationship between SDB and posttrauma nightmares, additional research is needed to understand how these variables interact and to replicate our novel findings.

### **Summary**

In conclusion, the results of this study documented that the time it takes to fall asleep (SOL), presleep cognitions (PSA-C), and potentially presleep physiological arousal all play a role in whether a posttrauma nightmare occurs. These findings were consistent with predictions informed by the NIGHT-CAP Model (Youngren et al., 2020), which proposed that presleep processing time and presleep cognitions can be used to predict whether posttrauma nightmares occur. These results also extend the generalizability of the NIGHT-CAP Model by demonstrating the predictive utility of SOL and PSA-C in another trauma exposed population with high prevalence of SDB. Furthermore, these findings offer evidence that presleep physiological arousal may also play a role in nightmare occurrences. Although the results of this study offer meaningful contributions to scientific theory, their translation to clinical practice may offer greater real-world impact. The results have highlighted two variables (SOL and PSA-C) that

could be specifically targeted by interventions with the intent of reducing nightmare frequency. Posttrauma nightmares have been linked to a host of negative psychosocial outcomes (e.g., suicide and substance abuse; Galatzer-Levy et al., 2013; Nadorff et al., 2013). Thus, increasing the mechanistic understanding of nightmare generation may have significant impact on public health. Overall, we view these results as the next laid bricks in the path towards understanding the nature of posttrauma nightmares.

## References

- Arnal, P. J., Thorey, V., Ballard, M. E., Hernandez, A. B., Guillot, A., Jourde, H., ... and Sauvet, F. (2019). *The Dreem Headband as an Alternative to Polysomnography for EEG Signal Acquisition and Sleep Staging*. bioRxiv, 662734.
- Bahammam, A. S., and Almeneessier, A. S. (2019). Dreams and nightmares in patients with obstructive sleep apnea: a review. *Frontiers in Neurology*, 10, 1127.
- Beck, A. T., Steer, R. A., and Brown, G. K. (1996). *Beck depression inventory-II*. San Antonio, 78(2), 490-8.
- Beck, A. T., Epstein, N., Brown, G., and Steer, R. A. (1988). An inventory for measuring clinical anxiety: psychometric properties. *Journal of Consulting and Clinical Psychology*, 56(6), 893.
- Blevins, C. A., Weathers, F. W., Davis, M. T., Witte, T. K., and Domino, J. L. (2015). The posttraumatic stress disorder checklist for DSM-5 (PCL-5): Development and initial psychometric evaluation. *Journal of Traumatic Stress*, 28(6), 489-498.
- Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., and Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Research*, 28(2), 193-213.
- Cacioppo, J. T., Tassinary, L. G., Stonebraker, T. B., and Petty, R. E. (1987). Self-report and cardiovascular measures of arousal: Fractionation during residual arousal. *Biological Psychology*, 25(2), 135-151.
- Chambon, S., Galtier, M. N., Arnal, P. J., Wainrib, G., and Gramfort, A. (2018). A deep learning

- architecture for temporal sleep stage classification using multivariate and multimodal time series. *IEEE Transactions on Neural Systems and Rehabilitation Engineering*, 26(4), 758-769.
- Chung, F., Yegneswaran, B., Liao, P., Chung, S. A., Vairavanathan, S., Islam, S., ... and Shapiro, C. M. (2008). Validation of the Berlin questionnaire and American Society of Anesthesiologists checklist as screening tools for obstructive sleep apnea in surgical patients. *Anesthesiology: The Journal of the American Society of Anesthesiologists*, 108(5), 822-830.
- Colvonen, P. J., Straus, L. D., Stepnowsky, C., McCarthy, M. J., Goldstein, L. A., and Norman, S. B. (2018). Recent advancements in treating sleep disorders in co-occurring PTSD. *Current Psychiatry Reports*, 20(7), 48
- Davis, J. L. (2008). *Treating post-trauma nightmares: A cognitive behavioral approach*. Springer Publishing Company.
- Debellemaniere, E., Chambon, S., Pinaud, C., Thorey, V., Dehaene, D., Léger, D., ... and Galtier, M. N. (2018). Performance of an ambulatory dry-EEG device for auditory closed-loop stimulation of sleep slow oscillations in the home environment. *Frontiers in Human Neuroscience*, 12, 88.
- DeMartinis, N. A., & Winokur, A. (2007). Effects of psychiatric medications on sleep and sleep disorders. *CNS & Neurological Disorders-Drug Targets (Formerly Current Drug Targets-CNS & Neurological Disorders)*, 6(1), 17-29.
- El-Solh, A. A., Vermont, L., Homish, G. G., and Kufel, T. (2017). The effect of continuous positive airway pressure on post-traumatic stress disorder symptoms in veterans with

- post-traumatic stress disorder and obstructive sleep apnea: a prospective study. *Sleep Medicine*, 33, 145-150.
- El-Solh, A. A. (2018). Management of nightmares in patients with posttraumatic stress disorder: current perspectives. *Nature and Science of Sleep*, 10, 409.
- Faraway, J. J. (2016). *Extending the linear model with R: generalized linear, mixed effects and nonparametric regression models*. Chapman and Hall/CRC.
- Finch, W. H., Bolin, J. E., and Kelley, K. (2016). *Multilevel modeling using R*. Crc Press.
- First, M. B., Williams, J. B. W., Karg, R. S., and Spitzer, R. (2015). Structured clinical interview for DMS-5, Research Version. *Arlington, VA: American Psychiatric Association*.
- Freud, S., and Cronin, A. J. (2013). *The interpretation of dreams*. Read Books Ltd.
- Galatzer-Levy, I. R., Nickerson, A., Litz, B. T., and Marmar, C. R. (2013). Patterns of lifetime PTSD comorbidity: A latent class analysis. *Depression and Anxiety*.  
<https://doi.org/10.1002/da.22048>
- Garcia, O., Slavish, D. C., Dietch, J. R., Messman, B. A., Contractor, A. A., Haynes, P. L., ... & Taylor, D. J. (2021). What goes around comes around: Nightmares and daily stress are bidirectionally associated in nurses. *Stress and Health*, 37(5), 1035-1042.
- George, K. C., Kebejian, L., Ruth, L. J., Miller, C. W., and Himelhoch, S. (2016). Meta-analysis of the efficacy and safety of prazosin versus placebo for the treatment of nightmares and sleep disturbances in adults with posttraumatic stress disorder. *Journal of Trauma and Dissociation*, 17(4), 494-510.
- Germain, A. (2013). Sleep disturbances as the hallmark of PTSD: where are we now. *American Journal of Psychiatry*. <https://doi.org/10.1176/appi.ajp.2012.12040432>
- Hartmann, E. (1984). *The nightmare: The psychology and biology of terrifying dreams*.



New York: Basic Books.

- Hartmann, E. (2000). We do not dream of the 3 R's: Implications for the nature of dreaming mentation. *Dreaming*, 10(2), 103-110.
- Hull, A. M. (2002). Neuroimaging findings in post-traumatic stress disorder: Systematic review. *British Journal of Psychiatry*, 181, 102–110.
- Kapsimalis, F., and Kryger, M. (2009). Sleep breathing disorders in the US female population. *Journal of Women's Health*, 18(8), 1211-1219.
- Levin, R., and Nielsen, T. A. (2007). Disturbed dreaming, posttraumatic stress disorder, and affect distress: a review and neurocognitive model. *Psychological Bulletin*.  
<https://doi.org/10.1037/0033-2909.133.3.482>
- Levin, R., and Nielsen, T. (2009). Nightmares, bad dreams, and emotion dysregulation: A review and new neurocognitive model of dreaming. *Current Directions in Psychological Science*. <https://doi.org/10.1111/j.1467-8721.2009.01614.x>
- Margolies, S. O., Rybarczyk, B., Vrana, S. R., Leszczyszyn, D. J., and Lynch, J. (2013). Efficacy of a cognitive-behavioral treatment for insomnia and nightmares in Afghanistan and Iraq veterans with PTSD. *Journal of Clinical Psychology*, 69(10), 1026-1042.
- Miller, K. E., Jamison, A. L., Gala, S., and Woodward, S. H. (2018). Two independent predictors of nightmares in posttraumatic stress disorder. *Journal of Clinical Sleep Medicine*, 14(11), 1921-1927.
- Morgane, P. J., Galler, J. R., and Mokler, D. J. (2005). A review of systems and networks of the limbic forebrain/limbic midbrain. *Progress in Neurobiology*, 75, 143–160.
- Mysliwicz, V., Brock, M. S., Creamer, J. L., O'Reilly, B. M., Germain, A., and Roth, B. J.

- (2018). Trauma associated sleep disorder: a parasomnia induced by trauma. *Sleep Medicine Reviews*, 37, 94-104.
- Nadorff, M., Nazem, S., and Fiske, A. (2013). Insomnia symptoms, nightmares, and suicide risk: duration of sleep disturbance matters. *Suicide and Life-Threatening Behavior*.  
<https://doi.org/10.1111/sltb.12003>
- Netzer, N. C., Stoohs, R. A., Netzer, C. M., Clark, K., and Strohl, K. P. (1999). Using the Berlin Questionnaire to identify patients at risk for the sleep apnea syndrome. *Annals of Internal Medicine*, 131(7), 485-491
- Neylan, T. C., Marmar, C. R., Metzler, T. J., Weiss, D. S., Zatzick, D. F., Delucchi, K. L., ... & Schoenfeld, F. B. (1998). Sleep disturbances in the Vietnam generation: findings from a nationally representative sample of male Vietnam veterans. *American Journal of Psychiatry*, 155(7), 929-933.
- Nicassio, P. M., Mendlowitz, D. R., Fussell, J. J., and Petras, L. (1985). The phenomenology of the pre-sleep state: the development of the pre-sleep arousal scale. *Behaviour Research and Therapy*, 23(3), 263-271.
- Pacella, M. L., Hruska, B., and Delahanty, D. L. (2013). The physical health consequences of PTSD and PTSD symptoms: a meta-analytic review. *Journal of Anxiety Disorders*.  
<https://doi.org/10.1016/j.janxdis.2012.08.004>
- Pruiksma, K. E., Taylor, D. J., Wachen, J. S., Mintz, J., Young-McCaughan, S., Peterson, A. L., ... and Hembree, E. A. (2016). Residual sleep disturbances following PTSD treatment in active duty military personnel. *Psychological Trauma: Theory, Research, Practice, and Policy*, 8(6), 697.

- RStudio Team (2020). RStudio: Integrated Development for R. RStudio, PBC, Boston, MA URL <http://www.rstudio.com/>.
- Raskind, M. A., Peskind, E. R., Chow, B., Harris, C., Davis-Karim, A., Holmes, H. A., ... & Huang, G. D. (2018). Trial of prazosin for post-traumatic stress disorder in military veterans. *New England Journal of Medicine*, 378(6), 507-517.
- Reist, C., Streja, E., Tang, C. C., Shapiro, B., Mintz, J., & Hollifield, M. (2021). Prazosin for treatment of post-traumatic stress disorder: a systematic review and meta-analysis. *CNS spectrums*, 26(4), 338-344.
- Schneider, A., and Domhoff, G. W. (2001). *Quantitative Study of Dreams*. University of California, Santa Cruz.
- Schredl, M., and Hofmann, F. (2003). Continuity between waking activities and dream activities. *Consciousness and Cognition*, 12(2), 298-308.
- Shankman, S. A., Funkhouser, C. J., Klein, D. N., Davila, J., Lerner, D., and Hee, D. (2018). Reliability and validity of severity dimensions of psychopathology assessed using the Structured Clinical Interview for DSM-5 (SCID). *International Journal of Methods in Psychiatric Research*, 27(1),
- Short, N. A., Allan, N. P., Stentz, L., Portero, A. K., & Schmidt, N. B. (2018). Predictors of insomnia symptoms and nightmares among individuals with post-traumatic stress disorder: An ecological momentary assessment study. *Journal of sleep research*, 27(1), 64-72.
- Van der Kolk, B., Blitz, R., Burr, W., Sherry, S., and Hartmann, E. (1984). Nightmares and trauma: A comparison of nightmares after combat with lifelong nightmares in veterans. *American Journal of Psychiatry*. <https://doi.org/10.1176/ajp.141.2.187>

- Vermetten, E., Germain, A., and Neylan, T. C. (2018). *Sleep and combat-related post traumatic stress disorder*. New York, NY; Springer.
- Weathers, F.W., Litz, B.T., Keane, T.M., Palmieri, P.A., Marx, B.P., and Schnurr, P.P. (2013). *The PTSD Checklist for DSM-5 (PCL-5)*.
- Youngren, W. A., Hamilton, N. A., and Preacher, K. J. (2020). Assessing Triggers of Posttrauma Nightmares. *Journal of Traumatic Stress*
- Youngren, W., Balderas, J., & Farrell-Higgins, J. (2021). How sleep disordered breathing impacts posttrauma nightmares and rescripting therapies. *Dreaming*, 31(1), 20.

Table 1  
*Model Building*

		Model 1	Model 2	Model 3	<b>Model 4</b>	Model 5
<b>Fixed Components</b>						
Intercept	$\hat{\gamma}_{00}$	-4.49*	-4.76**	-4.77**	<b>-4.21**</b>	-1.76*
SOL	$\hat{\gamma}_{10}$	0.05**	0.05**	0.05**	<b>0.05***</b>	0.05**
PSA-C	$\hat{\gamma}_{20}$	0.14*	0.11*	0.11*	<b>0.11*</b>	
SDB	$\hat{\gamma}_{30}$	1.07	1.01	1.01		
PTC	$\hat{\gamma}_{40}$	0.18	0.18			
PSA-S	$\hat{\gamma}_{50}$	-0.06				
<b>Model Components</b>						
AIC		92.1	90.5	88.5	<b>87.8</b>	88.7
BIC		108.7	104.7	100.4	<b>97.3</b>	95.9
loglik		-39.1	-39.2	-39.3	<b>-39.9</b>	-41.4
Likelihood Ratio test			0.34	0.05	<b>1.30</b>	2.91*

*Note.* \* < 0.05, \*\*  $\leq$  0.01; PTC = postsleep recall of presleep thought content related to trauma, SOL = Sleep Onset Latency, PSA-S Presleep Arousal-Somatic, PSA-C = Presleep Arousal-Cognitive; Fixed effects estimated using maximum likelihood; Deviance and corresponding likelihood ratio test calculated using REML; **Bolded** model = final model.

Table 2  
*Objective Sleep Data Stratified by Posttrauma Nightmare Occurrence*

	Nightmare Did Not Occur ( <i>N</i> = 42)		Nightmare Occurred ( <i>N</i> = 38)	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Total Sleep Time (minutes)	324.33*	38.73	290.25*	55.79
Sleep Score	66.67*	7.09	56.57*	11.32
Non-REM (minutes)	253.29*	45.76	205.25*	44.42
Non-REM Percentage	79.95%*	22.14%	60.04%*	3.01%
REM (minutes)	89.01*	47.09	70.12*	40.16
REM Percentage	26.18%	16.65%	26.74%	26.74%

*Note.* \*  $p < 0.05$ .

Table 3  
*Presleep Arousal and Sleep Latency Stratified by Posttrauma Nightmare Occurrence*

	<u>Nightmare Did Not Occur (N = 42)</u>		<u>Nightmare Occurred (N = 38)</u>	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
<b>Cognitive Arousal</b>				
PSA-C (SR)	22.88*	6.29	27.21*	7.41
<b>Physical Arousal</b>				
PSA-S (SR)	15.46	3.61	18.13	3.62
PSA-PHYS (DH)	73.00**	3.34	79.87**	1.98
<b>Sleep Onset Latency</b>				
SOL (SR)	25.35 minutes**	16.50 minutes	50.66 minutes**	31.84 minutes
SOL (DH)	25.50 minutes**	18.31 minutes	47.65 minutes**	19.56 minutes

*Note.* \*  $p < 0.01$ ; SR = Self-report measure; DH = DREEM Headband; Pulse rate was the average pulse rate score over the 15 minutes of time prior to sleep onset (which was determined by the DH).