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Examining Physiological Hyperarousal in the Relationship Between Insomnia and Nightmare Proneness

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ABSTRACT

Nightmare proneness has been associated with insomnia beyond nightmare frequency and neuroticism. The present study examined physiological hyperarousal as a potential process contributing to this relationship. Participants were 209 adults recruited from a university participant pool and an online community sample. Participants completed self-report measures of nightmare proneness, insomnia, physiological hyperarousal, nightmare frequency, neuroticism, and trauma symptoms. Hierarchical regression and indirect-effect analyses were conducted. After controlling for recruitment source, age, gender, nightmare frequency, neuroticism, and trauma symptoms, nightmare proneness explained an additional 6.5% of the variance in insomnia. Physiological hyperarousal accounted for an additional 2.3% of variance beyond nightmare proneness. Nightmare proneness showed a significant indirect association with insomnia through physiological hyperarousal but maintained a direct association with insomnia after physiological hyperarousal was considered. Physiological hyperarousal may represent one process contributing to the relationship between nightmare proneness and insomnia but does not fully account for this association. The findings support conceptualizing nightmare proneness and physiological hyperarousal as related but non-redundant factors associated with insomnia.

Keywords: Nightmare proneness; Insomnia; Hyperarousal; Nightmares; Sleep; Neuroticism

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INTRODUCTION

Insomnia is characterized by persistent difficulties initiating or maintaining sleep or early morning awakening and is associated with multiple psychological and physiological processes [1]. Hyperarousal has been particularly prominent in etiological models of insomnia, encompassing elevated physiological, cortical, and cognitive-emotional arousal across the sleep-wake cycle [2]. Another potentially relevant factor is nightmare proneness, a trait-like disposition toward experiencing frequent nightmares [3]. Nightmare proneness has also been examined independently in relation to broader sleep-related characteristics, including sleep quality and chronotype [4]. Importantly, nightmare proneness has been associated with insomnia symptoms beyond nightmare frequency and neuroticism [5]. Thus, its relationship with insomnia does not appear attributable solely to sleep disruption caused by nightmares or general negative emotionality.

One possible explanation for this relationship is hyperarousal. The nightmare proneness model proposes dysregulated hyperarousal and vulnerability as processes through which temperamental and experiential factors may contribute to nightmare proneness [3]. This proposal is consistent with evidence connecting arousal processes with both nightmares and insomnia [6]. Individuals who experience frequent nightmares show evidence of increased arousal during periods preceding rapid eye movement (REM) sleep [7]. More directly, self-reported physiological hyperarousal has been associated with nightmare proneness and remained related to it after accounting for neuroticism [8]. These findings support the possibility that hyperarousal may contribute to the previously observed relationship between nightmare proneness and insomnia.

Hyperarousal is well established in contemporary models of insomnia. Rather than reflecting a single response system, it has been conceptualized as involving interacting physiological, cortical, and cognitive-emotional processes that may extend across waking and sleep [2]. Consistent with this perspective, individuals with insomnia disorder have shown increased high-frequency EEG activity during both nighttime and daytime NREM sleep, suggesting that cortical hyperarousal may extend across the 24-hour sleep-wake cycle [9]. Heightened sleep reactivity – the tendency for stress-related arousal to disrupt sleep – has also prospectively predicted the onset of acute insomnia during periods of increased stress [10]. Hyperarousal may further help explain relationships between broader personality dispositions and sleep; for example, Cellini et al. [11] found that hyperarousal attenuated the association between neuroticism and perceived sleep quality. Together, these findings suggest that physiological hyperarousal may represent one process through which a disposition toward nightmare experiences is associated with insomnia.

Nightmare frequency, neuroticism, and trauma symptoms provide important alternative explanations for these relationships. Nightmares themselves disrupt sleep and are consistently associated with insomnia across diverse populations [12]. Neuroticism is associated with both nightmare proneness and sleep disturbance, while trauma is associated with nightmares, insomnia, and hyperarousal [13,14]. Trauma has also been identified as a potential experiential contributor to nightmare proneness [3]. Accounting for these factors therefore provides a more stringent test of the independent associations of nightmare proneness and physiological hyperarousal with insomnia.

The present study further examined the relationship between nightmare proneness and insomnia, with particular attention to physiological hyperarousal as a potential explanatory process. We first examined whether nightmare proneness was associated with insomnia beyond nightmare frequency, neuroticism, trauma symptoms, and relevant sampling characteristics, and whether physiological hyperarousal contributed additional variance. We then examined whether nightmare proneness was indirectly associated with insomnia through physiological hyperarousal. It was expected that nightmare proneness and physiological hyperarousal would each be associated with insomnia and that nightmare proneness would show a significant indirect association with insomnia through physiological hyperarousal.

MATERIALS AND METHOD

Participants and Procedure

The initial sample consisted of 215 adults recruited from Amazon Mechanical Turk (MTurk; n = 105) and a university psychology participant pool (n = 110). Six participants who failed at least one of three instructed-response attention checks were excluded; no duplicate responses were identified. The final sample consisted of 209 participants ranging from 18 to 76 years (M = 35.11, SD = 16.99), including 137 women and 70 men (two did not report gender). Participants identified as Latinx (43.1%), White/Caucasian (30.1%), Asian (21.1%), Black/African American (2.9%), or another identity/preferred not to respond (2.4%).

Participants completed an online survey of personality, sleep experiences, and sociodemographic characteristics after providing informed consent. University participants received nominal course credit and MTurk participants received $0.25. The study was approved by the university research ethics board (Protocol #2023-12-029).

Measures

Nightmare proneness.

Nightmare proneness was assessed using the 6-item Nightmare Proneness Scale [15]. To avoid criterion overlap, the item referring directly to difficulty initiating sleep (“Many nights I cannot get to sleep because of worry or tension”) was omitted. The resulting five-item score

used responses ranging from 0 (strongly disagree) to 4 (strongly agree), with higher scores indicating greater nightmare proneness (current study α = .86). Nightmare Proneness Scale total scores with and without the removed item were nearly perfectly correlated (r = .99), suggesting that removal of the item did not appreciably alter individual differences in nightmare proneness.

Insomnia

Insomnia was assessed using a 4-item measure of sleep initiation, maintenance, and early awakening difficulties during the past four weeks [5]. Items were rated from 0 (never) to 4 (very often), with higher scores indicating greater insomnia symptoms (current study α = .86).

Physiological hyperarousal

The 6-item Physiological Hyperarousal Scale [16] assessed perceived bothersome physiological arousal experiences during the past month. Items were rated from 0 (not at all) to 3 (severely), with higher scores indicating greater physiological hyperarousal (current study α = .86).

Neuroticism

Neuroticism was assessed using the 6-item Neuroticism scale of the NEO-FFI-30 [17]. Items were rated from 0 (strongly disagree) to 4 (strongly agree), with higher scores indicating greater neuroticism (current study α = .90).

Trauma symptoms

Trauma symptoms were assessed using the 4-item Primary Care PTSD Screen [18], referencing a highly distressing lifetime event and symptoms during the past month. To avoid overlap with nightmare occurrence, one item with nightmare content was modified to read, “Remembered or thought about it when you did not want to?” Items were scored 0 (no) or 1 (yes) and summed, with higher scores indicating more trauma symptoms (current study α = .84).

Nightmare frequency.

Nightmares were assessed using the 3-item Nightmare Frequency Index [19], which defined nightmares as “unpleasant and clearly remembered dreams that awaken you; after waking you quickly become alert.” Items were rated from 0 (strongly disagree) to 4 (strongly agree) and averaged, with higher scores indicating greater nightmare frequency (current study α = .89).

Data Analysis

Descriptive statistics, Pearson correlations, and hierarchical multiple regression analyses were conducted using SPSS for Windows (v. 32). In the first step, recruitment source (MTurk

vs students), age, gender, nightmare frequency, neuroticism, and trauma symptoms were regressed on insomnia; nightmare proneness was added in the second step and physiological hyperarousal in the third. An indirect-effects analysis using PROCESS for SPSS Model 4 [20] subsequently examined whether nightmare proneness was indirectly associated with insomnia through physiological hyperarousal, controlling for recruitment source, age, gender, nightmare frequency, neuroticism, and trauma symptoms. Indirect effects were evaluated using 10,000 bootstrap samples and 95% confidence intervals. Cases with missing covariate data were excluded listwise from multivariate analyses, resulting in an analytic sample of 206 participants. Results were considered significant if $p < .05$ (two-tailed).

RESULTS

Preliminary Analyses

Descriptive statistics and correlations among the primary study variables are presented in Table 1. Preliminary analyses confirmed substantial sociodemographic differences between recruitment sources. The MTurk sample was substantially older (M = 51.45, SD = 8.97) than the student sample (M = 20.55, SD = 4.79), t (206) = 31.46, $p < .001$, and the groups also differed in gender ($\chi^2(1) = 30.80$, $p < .001$). Recruitment source, age, and gender were therefore included as covariates in subsequent analyses.

Table 1: Descriptive Statistics and Correlations Among Primary Study Variables

Variable	M	SD	1	2	3	4	5
1. Nightmare proneness	7.27	5.20	—				
2. Physiological hyperarousal	4.22	3.90	.63**	—			
3. Insomnia	6.82	4.34	.46**	.46**	—		
4. Nightmare frequency	1.11	1.11	.37**	.45**	.40**	—	
5. Neuroticism	10.14	6.53	.65**	.57**	.30**	.19*	—
6. Trauma symptoms	1.74	1.62	.65**	.53**	.29**	.27**	.47**

Note: N = 209. *p < .01; **p < .001. M = mean, SD = standard deviation.

Hierarchical Regression

A hierarchical regression examined whether nightmare proneness and physiological hyperarousal accounted for unique variance in insomnia (Table 2). Recruitment source, age, gender, nightmare frequency, neuroticism, and trauma symptoms accounted for 24.7% of the variance in insomnia on Step 1. Adding Nightmare proneness on Step 2 explained an additional 6.5%, $\Delta F(1, 198) = 18.64$, $p < .001$, and adding physiological hyperarousal on Step 3 explained an additional 2.3%, $\Delta F(1, 197) = 6.73$, $p = .010$. The final model accounted for 33.5% of the variance in insomnia, $F(8, 197) = 12.38$, $p < .001$. VIFs for the primary study predictors were below 3.0; VIFs were higher for age and recruitment source (6.09 and 6.48, respectively), reflecting the substantial age difference between recruitment groups.

Indirect Effect Through Physiological Hyperarousal

PROCESS Model 4 examined the indirect association between nightmare proneness and insomnia through physiological hyperarousal, controlling for recruitment source, age, gender, nightmare frequency, neuroticism, and trauma symptoms. Nightmare proneness was associated with physiological hyperarousal, b = .19, SE = .06, p = .002, which in turn was associated with insomnia, b = .24, SE = .09, p = .010. The indirect effect was significant, ab = .047, BootSE = .026, 95% bootstrap CI [.005, .105]. The direct association between nightmare proneness and insomnia remained significant, b = .30, SE = .08, p < .001.

Table 2: Hierarchical Regression Predicting Insomnia Symptoms

Predictor	**β**		
	Step 1	**Step 2**	**Step 3**
Recruitment source	.17	.11	.14
Age	.28	.29*	.31*
Gender	.10	.12	.11
Nightmare frequency	.33**	.24**	.18*
Trauma symptoms	.15	.01	-.02
Neuroticism	.16*	-.01	-.07
Nightmare proneness	—	.41**	.36**
Physiological hyperarousal	—	—	.22*
R^2	.247	.312	.335
ΔR^2	.247**	.065**	.023*

Note. N = 206. Step 1 included recruitment source, age, gender, nightmare frequency, trauma symptoms, and neuroticism; nightmare proneness was added at Step 2 and physiological hyperarousal at Step 3. Recruitment source: 1 = MTurk, 2 = students. Gender: 1 = men, 2 = women. β = standardized beta; R^2 = total variance accounted for; ΔR^2 = change in variance accounted for. *p < .05; **p < 01.

DISCUSSION

The present study examined the relationship between nightmare proneness and insomnia, focusing on physiological hyperarousal as a potential explanatory process. Consistent with previous findings, nightmare proneness was associated with insomnia beyond nightmare frequency and neuroticism [5]. The present results extend this relationship by controlling for trauma symptoms and relevant sociodemographic and sampling characteristics. Physiological hyperarousal accounted for additional variance in insomnia after these factors and nightmare proneness were considered. Further, nightmare proneness showed a significant indirect association with insomnia through physiological hyperarousal. Together, the findings suggest that hyperarousal may represent one process contributing to the relationship between nightmare proneness and insomnia.

These findings are consistent with conceptualizations of nightmare proneness as involving dysregulated arousal and vulnerability [3] and with broader models emphasizing hyperarousal in insomnia [2]. They also converge with physiological evidence linking heightened arousal with both insomnia and nightmares. Individuals with insomnia show evidence of cortical hyperarousal across nighttime and daytime sleep [9], while increased arousal has been observed preceding REM sleep among individuals with frequent nightmares [7]. Moreover, ambulatory psychophysiological monitoring indicates that nightmare episodes themselves are accompanied by increased autonomic activation [21]. Thus, hyperarousal may provide a common process connecting a disposition toward nightmare experiences with broader difficulties initiating and maintaining sleep.

At the same time, the findings do not imply that hyperarousal fully explains the association between nightmare proneness and insomnia. Nightmare proneness remained independently associated with insomnia after physiological hyperarousal was included, and the indirect effect through hyperarousal was relatively modest. This pattern is consistent with nightmare proneness reflecting a broader vulnerability rather than simply autonomic arousal. It is also consistent with evidence that the relationship between nightmares and insomnia is likely multifaceted and potentially reciprocal. Delage et al. [12] for example, concluded from their systematic review that nightmares and insomnia are consistently associated across populations and may mutually aggravate one another. Other processes associated with nightmare proneness, such as cognitive or emotional reactivity, may therefore contribute to insomnia independently of physiological hyperarousal.

Several limitations of the current study warrant consideration. The cross-sectional design precludes conclusions regarding temporal or causal direction; consequently, the indirect effect should be interpreted as consistent with, rather than evidence of, a mediational process. All primary variables were assessed by self-report, including physiological hyperarousal. Future research could incorporate objective indices of autonomic or cortical arousal. Although recruiting both university students and community adults increased sample heterogeneity, the two groups differed substantially on several sociodemographic and study variables. Though recruitment sources were statistically controlled in the current study, replication in more representative community samples would be useful. Finally, although removal of the sleep-related item reduced criterion overlap with insomnia and the resulting five-item measure retained good internal consistency and was strongly related with the 6-item version, modifying the nightmare proneness scale may have affected aspects of its construct validity and warrants further psychometric evaluation.

CONCLUSION

The findings of the present study extend previous work by identifying physiological hyperarousal as one potential contributor to the relationship between nightmare proneness and insomnia. Nightmare proneness remained associated with insomnia after accounting for nightmare frequency, neuroticism, trauma symptoms, and sociodemographic and sampling covariates while physiological hyperarousal provided additional explanatory value and a significant indirect pathway. The results support viewing nightmare proneness and hyperarousal as related but distinct factors in understanding the connection between nightmares and disturbed sleep.

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