pii: jc-19-00097 https://dx.doi.org/10.5664/jcsm.7608

JCSM
Journal of Clinical Sleep Medicine

COMMENTARY

# A Meaningful Step Toward Understanding the Cause and Impact of Nightmares

Commentary on Marquis et al. Nightmare severity is inversely related to frontal brain activity during waking state picture viewing. *J Clin Sleep Med.* 2019;15(2):253–264.

Michael R. Nadorff, PhD[1,2]; Caitlin E. Titus, MS[1]; Ashley R. Pate, BS[1]

[1]*Department of Psychology, Mississippi State University, Mississippi State, Mississippi;* [2]*Department of Psychiatry, Baylor College of Medicine, Houston, Texas*

Although it may be thought of as a childhood disorder, with up to 50% of children between ages 3–6 reporting them,[1] nightmares commonly persist into adulthood. In fact, 14% of college students[2] and 4.3% of older adults report frequent nightmares.[3] Nightmares are also very clinically-relevant. For instance, it is known that nightmares are a frequent symptom with posttraumatic stress disorder (PTSD), but some may not realize that the presence of nightmares before a trauma can increase the risk of developing PTSD.[4] Nightmares have also been shown to be comorbid with depression,[5,6] anxiety,[7] schizophrenia,[8,9] and suicide.[10–12] Even when not leading to psychopathology, nightmares are still associated with substantial daytime distress.[13,14] Given its high prevalence and notable comorbidities, there is a great need for theories, and subsequent validation research, to help us understand nightmares, and by proxy to better understand the mechanism by which nightmares contribute to psychopathology and suicide. Levin and Nielsen's Neurocognitive Model[15,16] fits the bill, as it is an exceptionally well thought-out and researched model of dysphoric dreams. The model proposes that nightmares may reflect problematic emotion regulation and highlights several brain structures that are likely implicated in nightmares: the amygdala, medial prefrontal cortex, hippocampus, and anterior cingulate cortex. However, one significant limitation of the model is that there is limited validation work that has been published. The theory was based upon a substantial amount of neurological research, but how do these areas actually respond in those who have nightmares?

Recently, the anterior cingulate cortex[17] and medial prefrontal cortex[18] have been shown to be associated with nightmare frequency, but nightmare distress remains unexamined. Although this may sound inconsequential, it is extremely important as nightmare distress is the component that is most closely associated with psychopathology.[19] It is very possible that understanding nightmare distress will be key in understanding the association between psychopathology and nightmares, and may lead to new clinical breakthroughs to mitigate their effects.

In an innovative new study featured in this issue of the *Journal of Clinical Sleep Medicine*, Marquis and colleagues[20] use SPECT imaging to examine brain activity while looking at neutral and negative pictures among 18 individuals who experience frequent nightmares. The results were consistent with research looking at the neural correlates of nightmare frequency,[17,18] and demonstrated potential overlap between the brain areas involved in disturbing dreams and distress during the day. It lays the groundwork for future studies examining the association between nightmares and psychopathology. It also provides further support for the neurocognitive model of nightmares as the medial prefrontal cortex and anterior cingulate cortex were related with disturbing dreams, but not the hippocampus or amygdala, which in and of itself makes it a novel and meaningful addition to the literature.

In addition to providing important validation work on a leading theory of nightmares, Marquis and colleagues provide a basis for several important future investigations. First, we agree with Marquis and colleagues that more imaging studies, including studies using other imaging strategies, are warranted. Specifically, more research is needed to better understand the role of the anterior cingulate cortex and the medial prefrontal cortex, especially the latter, in disturbing dreams and related psychopathology. Second, given that emotion regulation is implicated with both the anterior cingulate cortex and medial prefrontal cortex, more research is needed examining the role of emotion regulation in nightmares. Recent research has implicated poor emotion regulation in the association between nightmares and suicidal behavior,[21] but the research on emotion regulation explaining the relation between nightmares and psychopathology is still very much in its infancy. Lastly, more nightmare treatment research is needed. In the last year, the research supporting prazosin for treating nightmares has been substantially weakened by large, strong studies that have failed to show effects[22] leaving clinicians with no recommended pharmacological treatments for nightmare disorder. It is possible that treatments that increase activation in the anterior cingulate cortex and medial prefrontal cortex may reduce nightmares, though this is admittedly a very early hypothesis that would require further research. Turning our attention to psychotherapies, research examining the impact of treating emotion regulation on nightmares, as well as the inverse, treating nightmares on emotion regulation, is warranted.

In sum, Marquis and colleagues[20] have provided us with a valuable study that already enhances the literature in a meaningful way, but that also has tremendous potential for shaping the way we study, view, assess, and treat nightmares in the future.

## CITATION

Nadorff MR, Titus CE, Pate AR. A meaningful step toward understanding the cause and impact of nightmares. *J Clin Sleep Med*. 2019;15(2):179–180.

## REFERENCES

1. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 5th ed. Washington, DC: American Psychiatric Association Publishing; 2013.
2. Nadorff MR, Nazem S, Fiske A. Insomnia symptoms, nightmares, and suicidal ideation in a college student sample. *Sleep.* 2011;34(1):93–98.
3. Salvio M-A, Wood JM, Schwartz J, Eichling PS. Nightmare prevalence in the healthy elderly. *Psychol Aging.* 1992;7(2):324–325.
4. Mellman TA, David D, Kulick-Bell R, Hebding J. Sleep disturbance and its relationship to psychiatric morbidity after Hurricane Andrew. *Am J Psychiatry.* 1995;152(11):1659–1663.
5. Besiroglu L, Agargun MY, Inci R. Nightmares and terminal insomnia in depressed patients with and without melancholic features. *Psychiatry Res.* 2005;133(2–3):285–287.
6. Sandman N, Merikanto I, Määttänen H, et al. Winter is coming: nightmares and sleep problems during seasonal affective disorder. *J Sleep Res.* 2016;25(5):612–619.
7. Nadorff MR, Porter B, Rhoades HM, Kunik ME, Greisinger AJ, Stanley MA. Bad dream frequency in older adults with generalized anxiety disorder: prevalence, correlates, and effect of cognitive behavioral treatment for anxiety. *Behav Sleep Med.* 2014;12(1):28–40.
8. Hartmann E. A preliminary study of the personality of the nightmare sufferer: Relationship to schizophrenia and creativity? *Am J Psychiatry.* 1981;138(6):794–797.
9. Herz MI, Melville C. Relapse in schizophrenia. *Am J Psychiatry.* 1980;137(7):801–805.
10. Sjöström N, Hetta J, Wærn M. Persistent nightmares are associated with repeat suicide attempt: a prospective study. *Psychiatry Res.* 2009;170(2–3):208–211.
11. Tanskanen A, Tuomilehto J, Viinamaki H, Vartiainen E, Lehtonen J, Puska P. Nightmares as predictors of suicide. *Sleep.* 2001;24(7):844–847.
12. Golding S, Nadorff MR, Winer ES, Ward KC. Unpacking sleep and suicide in older adults in a combined online sample. *J Clin Sleep Med.* 2015;11(12):1385–1392.
13. Köthe M, Pietrowsky R. Behavioral effects of nightmares and their correlations to personality patterns. *Dreaming.* 2001;11(1):43–52.
14. Lancee J, Schrijnemaekers NCML. The association between nightmares and daily distress. *Sleep Biol Rhythms.* 2013;11(1):14–19.
15. Levin R, Nielsen TA. Disturbed dreaming, posttraumatic stress disorder, and affect distress: a review and neurocognitive model. *Psychol Bull.* 2007;133(3):482–528.
16. Levin R, Nielsen T. Nightmares, bad dreams, and emotion dysregulation: A review and new neurocognitive model of dreaming. *Curr Dir Psychol Sci.* 2009;18(2):84–88.
17. Shen C, Wang J, Ma G, et al. Waking-hour cerebral activations in nightmare disorder: A resting-state functional magnetic resonance imaging study. *Psychiatry Clin Neurosci.* 2016;70(12):573–581.
18. Marquis L, Blanchette-Carrière C, Carr M, Julien S, Paquette T, Nielsen T. Decreased activity in medial prefrontal cortex and anterior cingulate cortex in idiopathic nightmare sufferers during wakefulness. *Sleep.* 2016;39(Abstract Suppl):A226.
19. Levin R, Fireman G. Nightmare prevalence, nightmare distress, and self-reported psychological disturbance. *Sleep.* 2002;25(2):205–212.
20. Marquis LP, Julien SH, Baril AA, et al. Nightmare severity is inversely related to frontal brain activity during waking state picture viewing. *J Clin Sleep Med.* 2019;15(2):253–264.
21. Ward-Ciesielski EF, Winer ES, Drapeau CW, Nadorff MR. Examining components of emotion regulation in relation to sleep problems and suicide risk. *J Affect Disord.* 2018;241:41–48.
22. Raskind MA, Peskind ER, Chow B, et al. Trial of prazosin for post-traumatic stress disorder in military veterans. *N Engl J Med.* 2018;378(6):507–517.

## SUBMISSION & CORRESPONDENCE INFORMATION

**Submitted for publication Feburary 7, 2019**
**Submitted in final revised form Feburary 7, 2019**
**Accepted for publication Feburary 7, 2019**
Address correspondence to: Michael R. Nadorff, Mississippi State University, PO Box 6161, Mississippi State, MS 39762.

## DISCLOSURE STATEMENT

The authors report no conflicts of interest.