

## 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.

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Although it may be thought of as a childhood disorder, with up to 50% of children between ages 3–6 reporting them,<sup>1</sup> nightmares commonly persist into adulthood. In fact, 14% of college students<sup>2</sup> and 4.3% of older adults report frequent nightmares.<sup>3</sup> 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.<sup>4</sup> Nightmares have also been shown to be comorbid with depression,<sup>5,6</sup> anxiety,<sup>7</sup> schizophrenia,<sup>8,9</sup> and suicide.<sup>10–12</sup> Even when not leading to psychopathology, nightmares are still associated with substantial daytime distress.<sup>13,14</sup> 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<sup>15,16</sup> 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<sup>17</sup> and medial prefrontal cortex<sup>18</sup> 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.<sup>19</sup> 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<sup>20</sup> 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,<sup>17,18</sup> 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,<sup>21</sup> 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<sup>22</sup> 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<sup>20</sup> 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.

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## SUBMISSION &amp; CORRESPONDENCE INFORMATION

Submitted for publication February 7, 2019

Submitted in final revised form February 7, 2019

Accepted for publication February 7, 2019

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## DISCLOSURE STATEMENT

The authors report no conflicts of interest.