



## Commentary on the paper “The heartbeat evoked potential is a questionable biomarker in nightmare disorder: A replication study. By Bogdany, T., Perakakis, P., Bodizs, R., Simor, P., 2021. Neuroimage Clin 33, 102933”

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Dear Editor,

Failing to replicate or failing to be a replication study? We have recently read the article from Bogdany et al. (2021) trying to replicate our previous result of an increased heartbeat evoked potential (HEP) amplitude during REM sleep in patients with nightmare disorder (Perogamvros et al., 2019). While replication is crucial for the continued progress of science (Nosek and Errington, 2020), the study by Bogdany et al. failed to replicate the independent variable used in our original study, namely the populations tested and compared. Therefore, rather than proposing a replication of our previous work, their study provides valuable, new data, as we briefly discuss here.

The authors successfully applied, and even improved, some methodological aspects of the original study. However, they did not use the same inclusion criteria for participant recruitment as we did in our original study. The authors briefly note this limitation at the end of their article. Obviously, this is not a negligible feature for a replication study.

In our study, we compared patients with a clinical diagnosis of nightmare disorder according to the International Classification of Sleep Disorders (American Academy of Sleep Medicine, 2014) to healthy matched controls. By contrast, Bogdany et al. recruited only participants who were not seeking medical help and compared those who reported relatively frequent bad dreams or nightmares to those with fewer such experiences. Although the term ‘nightmare disorder’ appears in the title and the text, no international classification of disorders was used in their studies. As is the case of clinical depression with impairment in one or more domains of functioning (compared for example to the presence of sadness without such daytime dysfunction), clinical diagnosis of nightmare disorder includes the presence of daytime symptoms such as fatigue, mood disturbance (e.g., persistence of nightmare affect) and cognitive impairments (e.g., intrusive nightmare imagery) (American Academy of Sleep Medicine, 2014). The study of pathophysiological mechanisms underlying nightmare disorder thus relies on a rigorous

diagnosis, as does adequate care management.

Daytime dysfunction is a critical criterion for determining whether a sleep or mental complaint is a symptom of a disorder (according to an international classification of diseases) or a normal, adaptive reaction to stress. Indeed, recent experimental evidence suggests that bad dreams of healthy participants benefit emotion regulation processes (Sterpenich et al., 2020), unlike nightmares in patients (Marquis et al., 2016). Whereas, at a first glance, bad dreams and nightmares may fall along a continuum of severity, the functional role of dreaming seems to break down when nightmares are associated with significant daytime emotional distress or impairment in social, occupational, or other important domains of functioning (Nielsen and Levin, 2007). Regarding the link between depressive symptoms and HEP amplitude within the clinical population, we would stress (and agree with Bogdany et al.) that this correlation (based on eleven points) is too preliminary to draw any strong conclusion about the functional role of dreaming in alleviating depressive symptoms in this specific population.

Therefore, the results of Bogdany et al. do not contradict our results. Instead, they indicate that bad dreams in healthy participants who do not seek medical care may reflect some form of emotional homeostasis (Nielsen and Levin, 2007; Sterpenich et al., 2020), whereas nightmares in patients with a clinical diagnosis of nightmare disorder may not. Taken together, the results from both studies stress that a careful differentiation between clinically-diagnosed nightmare disorder patients and healthy participants with functional bad dreams and without daytime distress (Nielsen and Carr, 2017) is a central issue for characterizing the modulation of HEP during emotional processing and (pathological) dreaming.

Kind regards,  
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