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Nightmare disorder in light of neuropsychological and polysomnographic investigations

- thesis points-

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Introduction

Nightmares are highly intense and negatively toned mental experiences during REM or light NREM sleep (DSM-IV, 2000). These perceptually vivid, real-like experiences often depict long and complex dream scenarios involving fear, anxiety or other negative emotions (Nielsen & Zadra, 2011), and usually, but not mandatorily, end in abrupt awakenings (Spoormaker, Schredl, & van den Bout, 2006). Nightmare disorder is described as the condition of experiencing nightmares on a weekly basis. Moreover, after awakening from a nightmare, the subject is fully alert and can recall the nightmarish experience. The nightmares cause emotional and cognitive difficulties during waking, and finally, the nightmares do not occur exclusively during the course of another mental disorder (DSM-IV, 2000).

Although the etiology of nightmares is still far from being clarified, recent data has questioned the general view that considers frequent nightmares as secondary symptoms of an underlying mental disorder (Spoormaker et al., 2006). This assumption led to the under-diagnosis of nightmares, and more unfortunately, to the under-treatment of this condition (Schredl, 2010). Nightmares are indeed overrepresented in psychiatric populations, especially in PTSD, where trauma-related recurrent nightmares constitute one of the marked features of the disorder (Swart, van Schagen, Lancee, & van den Bout, 2013). However, the high co-morbidity of mental complaints and nightmares does not implicate a causal relationship between these phenomena. While frequent nightmares and mental complaints may exacerbate the severity of "the waking", as well as of the "the sleep" symptoms, research indicates that nightmare disorders and waking mental disorders stem form different pathological and etiological processes (Coolidge, Segal, Coolidge, Spinath, & Gottschling, 2010; Lancee, Spoormaker, & van den Bout, 2010; Spoormaker & Montgomery, 2008).

A neurocognitive approach to nightmare disorder

Levin & Nielsen (2007) presented a neurocognitive model that describes nightmares as the mental reflections of unsuccessful fear-extinction processes. According to their model, nightmares reflect the intensification of emotional memories supported by amygdalar and subcortical activations. Due to impaired hippocampal functions, the network fails to provide novel spatio-temporal contexts for emotional memories, and because of dysfunctional (medial) prefrontal functions, the amygdalar activity processing emotional reactions is not efficiently inhibited. Levin and Nielsen argue that the failure to provide novel contexts and to reduce the emotional intensity reactivated by emotional memories results in impaired fear-extinction during sleep (Levin & Nielsen, 2007). Levin and Nielsen's neurocognitive model is based mainly on experimental findings and clinical observations regarding subjects with PTSD, and although the authors view nightmarish experiences as a continuum from "normal", non-pathological dysphoric dreaming to the most severe cases of traumatic nightmares, it is still not clarified whether the assumptions of the model can be generalized in relation to nightmare sufferers without PTSD. Since one of the assumptions of the above model is the condition of abnormal fronto-limbic functions in subjects with frequent nightmares, we might expect that nightmare sufferers would exhibit impaired performance in waking behavioral tasks that are dependent on fronto-limbic neural circuitry. Levin and Nielsen's model suggests that nightmare sufferers are characterized by inefficient prefrontal control functions, that do not exert inhibitory control over amygdalar activation. I hypothesized if this is the case during REM-specific emotional information processing, then nightmare sufferers might also exhibit impaired performance in executive tasks that involve emotional information processing.

1. Impaired executive functions in subjects with frequent nightmares

We examined executive functions of nightmare sufferers (NM) and matched healthy controls (CTL) in two consecutive investigations. In the first study we hypothesized that the NM group would exhibit impaired performance in different well-characterized neuropsychological tasks that rely on prefrontal and fronto-limbic (ventromedial prefrontal cortex, inferior frontal, and rostral anterior cingular gyrus) neural circuitry (Baldo, Shimamura, Delis, Kramer, & Kaplan, 2001; Baldo & Shimamura, 1998; Bremner et al., 2004; Phaf & Kan, 2007; Reynolds & Jeeves, 1978). Moreover, in order to control for the influence of waking anxiety and subjective sleep quality on these measures, we measured these constructs by psychometric tests (STAI-T, Groningen Sleep Quality Scale (Sipos, Sipos, & Spielberger, 1994; Simor, Köteles, Bódizs, Bárdos, 2009) and controlled their effects in our statistical models. 35-35 NM and matched CTL subjects completed three different tasks, an emotional Go/noGo task, applying human faces with neutral and emotional (happy, angry) expressions, an emotional stroop task with neutral and emotionally negative words, and the category and letter verbal Fluency task.

NM subjects exhibited worse performance (increased reaction times) in the emotional go/noGo task in case of neutral targets and emotional distractors. This finding is in line with our previous assumption of impaired inhibitory control in NM subjects in case of emotional information processing. Nevertheless, the effect of group (NM vs. CTL) was no more significant when we controlled for the effects of anxiety. While subjective sleep quality did not, anxiety scores seemed to mediate group differences in this specific task.

Regarding the emotional stroop task a main effect of group emerged, due to generally slower reaction times in the NM in comparison with the CTL group. This finding resembled our hypothesis expecting impaired performance in NM subjects. Nevertheless, the NM group

exhibited longer reaction times in the neutral and the emotional conditions as well. Therefore, the emotional stroop effect (Phaf & Kan, 2007) (the so-called emotional interference: longer reaction times in response to emotionally charged words) was only evident in the CTL group. This finding was contrary to our hypothesis, and we speculated that the lack of emotional interference in the NM group was due to the design of our stroop protocol that applied a random presentation of neutral and emotional words. We assumed that the unexpected pattern of reaction times in the NM group was the result of a "slow" effect of emotional interference (McKenna & Sharma, 2004) that biased reaction times of the neutral words as well.

Therefore, we conducted an other experiment with a relatively reduced sample size for both groups, where we presented the same words in a block-design, aiming to avoid the "slow" effects of emotional interference. In this experiment we also included a non-emotional, classical stroop task with incongruent, congruent and control trials. In this experiment we replicated the findings of the firsts study, showing generally increased reaction times in the NM group in response to emotional and neutral words; however, in this case emotional interference was evident in both groups. In contrast to the results of the emotional stroop task, NM subjects did not show lower performance in either condition of the classical stroop task.

And finally, NM subjects exhibited worse performance in the fluency task, committing significantly more perseveration errors. Our results supported the assumption of impaired executive functions in NM sufferers. Nevertheless, NM subjects exhibited lower performance in the neutral condition of the emotional stroop task, questioning the specificity of executive dysfunctions for cases of emotional information processing. Although these studies are the first to examine executive functions in NM subjects, several questions, such as the specificity of these dysfunctions and the causal relationships between our variables warrant further investigations. The detailed description of these findings and our further conclusions and

speculations are presented in **Study 1.** *(Simor, P., Pajkossy, P., Horváth, K., & Bódizs, R. (2012). Impaired executive functions in subjects with frequent nightmares as reflected by performance in different neuropsychological tasks. Brain and Cognition, 78(3), 274–283).*

2. Impaired executive functions in NMs are not unequivocally mediated by waking anxiety.

Although increased reaction times in the NM group in response to neutral targets and emotionally charged distractors in the emotional Go/noGo task seemed to be the function of increased trait anxiety levels in NM subjects in comparison with CTLs; differences in the emotional stroop and the fluency tasks were independent of the levels of anxiety. These findings indicate that executive dysfunctions in NMs are not unequivocally mediated by waking anxiety. These results also corroborate research data suggesting that nightmares are not necessarily the results of an underlying mental disorders (Spoormaker et al., 2006). In contrast nightmare disorder seem to be a specific sleep complaint that might be co-morbid but independent form mental disorders in its origins (Coolidge et al., 2010; Lancee et al., 2010; Spoormaker & Montgomery, 2008). The detailed description of these findings and our further conclusions and speculations are presented in **Study 1.** *(Simor, P., Pajkossy, P., Horváth, K., & Bódizs, R. (2012). Impaired executive functions in subjects with frequent nightmares as reflected by performance in different neuropsychological tasks. Brain and Cognition, 78(3), 274–283).*

Altered sleep in nightmare disorder

Another important feature of nightmare disorder is altered subjective sleep quality (Lancee et al., 2010). Levin and Nielsen's above model would implicate that sleep disruption is caused by emotional over-activation. The activation of limbic and paralimbic structures during sleep were suggested to be related to arousals and sleep disruption (Nofzinger and Maquet, 2011). Questionnaire-based studies indicate that frequent nightmares are associated with impaired subjective sleep quality (Krakow, 2006; Li et al., 2010). Moreover, different sleep disorders are characterized by increased nightmare frequency and negatively toned dream experiences (Nadorff, Nazem, & Fiske, 2011; Schredl, Shafer, Weber, & Heuser, 1998; Uguccioni et al., 2013). However, the extent and nature of sleep disruption in nightmare sufferers was only scarcely elucidated by objective sleep assessments (Fisher, Byrne, Edwards, & Kahn, 1970; Germain & Nielsen, 2003). This is a considerable shortcoming in the field of sleep research in light of the extensive literature regarding macrostructural, as well as more fine-grained polysomnographic analyses of different sleep disorders (Bruni et al., 2008; Chou et al., 2011; Guilleminault, Kirisoglu, da Rosa, Lopes, & Chan, 2006; Riemann et al., 2010; Terzano et al., 2006).

Therefore, our second aim was to characterize the polysomnographic features of nightmare disorder based on full-night, undisturbed laboratory sleep recordings. In our subsequent studies we examined the objective sleep parameters of a group of NM and CTL subjects with different methods, involving standard sleep scoring, sleep-microstructure and quantitative EEG analyses. Since important findings regarding the relationship between nightmare disorder and our dependent variables of interest might be masked by the confounding effects of waking psychopathological symptoms, we selected groups of nightmare subjects without prior or present history of waking psychopathology. Furthermore, we controlled the effects of sub-clinical psychopathological symptoms with statistical methods in all of the studies presented below.

3. NMs, in comparison with CTLs, are characterized by altered sleep architecture reflecting impaired sleep continuity.

We analyzed the sleep-macrostructural characteristics of 17 NM and 23 CTL subjects that spent two consecutive nights in the sleep laboratory. The distribution of sleep stages as well as the number of awakenings of the second nights' sleep recordings was examined following the procedure described by Rechtschaffen and Kales (Rechtschaffen & Kales, 1968).

NMs exhibited impaired objective sleep quality in comparison with CTLs. Reduced sleep efficiency, increased wakefulness after falling asleep, and reduced percentage of SWS characterized the sleep macrostructure of NMs. NMs showed a trend of increased sleep latency and a higher rate of Stage 1 sleep. Furthermore, NMs were characterized by an increased number of nocturnal awakenings in Stage 2 sleep. Furthermore, these differences were independent of the effects of higher levels of waking anxious and depressive symptoms in the NM group.

These findings corroborate questionnaire-based data regarding the relationship between frequent nightmares and poor sleep quality (Li, Zhang, Li, & Wing, 2010; Schredl, 2003) , and replicates earlier findings of polysomnographic investigations (Fisher et al., 1970; Newell, Padamadan, & Drake, 1992). Nevertheless, the present findings are based on a relatively larger sample size and controlled for the confounding effects of waking affective symptoms. The detailed description of these findings and our further conclusions and speculations are presented in **Study 2. (***Simor, P., Horváth, K., Gombos, F., Takács, K. P., & Bódizs, R. (2012). Disturbed dreaming and sleep quality: altered sleep architecture in*

subjects with frequent nightmares. European archives of psychiatry and clinical neuroscience, 262(8), 687–696).

4. Increased REM percentage in NMs is mediated by enhanced negative emotionality.

NMs, in comparison with controls, exhibited increased rates of REM sleep, but this difference turned out to be the function of heightened trait anxiety and depressive symptoms in the nightmare group. This finding is in line with the hypothesized role of REM sleep in emotional information processing (Walker & van Der Helm, 2009), and with clinical research data showing the association of altered REM sleep and affective disorders (Benca et al., 1997). The detailed description of these findings and our further conclusions and speculations are presented in **Study 2.** ***(****Simor, P., Horváth, K., Gombos, F., Takács, K. P., & Bódizs, R. (2012). Disturbed dreaming and sleep quality: altered sleep architecture in subjects with frequent nightmares. European archives of psychiatry and clinical neuroscience, 262(8), 687–696).*

5. NMs are characterized by abnormal arousal processes during NREM sleep.

Frequent awakenings in the NM group suggested that NM subjects were characterized by the imbalance of sleep-and wake-promoting mechanisms, that might be reflected in more frequent arousals during sleep. We examined the frequency and specificity of arousals during sleep based on the scoring method of the Cyclic Alternating Pattern (CAP) (Terzano et al., 2002).

CAP sequences are characterized by phasic electrocortical activities that are different from background EEG and recur in quasi-periodic burst sequences with inter-burst intervals

up to 1 minute. CAP sequences, apart from changes in EEG, exhibit alterations in autonomic activity reflecting sympathetic activations (Ferri et al., 2000). These transient bursts of phasic events are categorized into different subtypes based on their frequency distributions. The subtype A1 is a burst of synchronized delta waves generated at anterior sites with frequency components predominantly between 0.25-2.5 Hz (Ferri, Bruni, Miano, & Terzano, 2005). Subtype A2 is a mixture of synchronized delta oscillations and higher (alpha and beta) frequency activity, while in A3, desynchronized high frequency oscillations are the predominant activities. Higher frequency components of A2 and A3 are generated in posterior sites, and reflect the transient instability of sleep depth in response to afferent inputs, while A1 and the slow component of A2 are characterized by anterior predominance. Although the vegetative components indicate a clear activation during A1 episodes these events seem to be strongly related to sleep-promotion (anti-arousals), reflecting the effort of the cortex to preserve the stability of sleep (Parrino, Ferri, Bruni, & Terzano, 2012). The characterization of the microstructure of sleep proved to be a comprehensive tool in studying developmental changes and pathological alterations, as well as pharmacological treatments of sleep instability (Parrino et al., 2012; Terzano & Parrino, 2000).

Here we showed that the NMs group (n=17) exhibited reduced amounts of synchronized CAP A1 subtype and increased amounts of A2 and A3 subtypes, as well as longer duration of CAP A phases in comparison with CTLs (n=23). These differences were not influenced by the confounding factors of anxious and depressive symptoms. REM arousals did not differentiate the two groups. These findings indicate abnormal arousal patterns in NM subjects reflecting the imbalance of sleep-promoting and arousing mechanisms during sleep. Our study is the first to investigate sleep microstructure in NM subjects and contributes to the understanding of the pathophysiology of this disorder. The detailed description of these findings and our further conclusions and speculations are

presented in **Study 3.** *(Simor, P., Bódizs, R., Horváth, K., & Ferri, R. (2013). Disturbed dreaming and the instability of sleep: altered nonrapid eye movement sleep microstructure in individuals with frequent nightmares as revealed by the cyclic alternating pattern. Sleep, 36(3), 413–419.)*

6. NMs are characterized by wake-like oscillatory activity during sleep, especially in REM sleep.

As far as we have seen in the previous thesis point NM subjects did not exhibit increased arousals during REM sleep. Nevertheless, the lack of significant differences in REM sleep might be related to the constraints of the visual scoring method of CAP. During NREM sleep, phasic activities are clearly visible, however, during REM periods, characterized by reduced muscle tone and low amplitude EEG, the short bursts of activation involving alpha oscillations might not be easily observable. Therefore, we have decided to examine the electroencephalographic features of NREM and REM periods by quantitative EEG analyses.

Power spectral analysis provides a fine-grained and sensitive examination of the electrophysiological oscillations during sleep, which seems to be an efficient tool to detect sleep alterations in different pathological conditions (Armitage, 1995; De la Fuente, Tugendhaft, & Mavroudakis, 1998; Feige, Scaal, Hornyak, Gann, & Riemann, 2007; Krystal, Edinger, Wohlgemuth, & Marsh, 2002; Lindberg et al., 2003; Moritz et al., 2002; Philipsen et al., 2005; Poulin, Stip, & Godbout, 2008). To the best of our knowledge, no previous studies have investigated the quantitative electrophysiological features of a whole night sleep in nightmare disorder.

Our data shows that NMs (n=19), in comparison with CTLs (n=21), exhibited increased relative high alpha (10-14.5 Hz) power in REM, and a trend of enhanced fronto-central low alpha (7.75-9 Hz) power in NREM sleep. High REM alpha and low NREM alpha were strongly an positively correlated in the nightmare-, but not in the control group, suggesting that enhanced alpha activity is a characteristic feature of the pathophysiology of nightmare disorder modulated in a sleep-state dependent manner, exhibiting higher frequency components in REM, and lower ones in NREM sleep. High REM alpha activity peaked at posterior locations, resembling the topographical distribution and frequency range of wake-like alpha activity.

We propose that the appearance of wake-type alpha oscillations during sleep – especially during the activated cortical state of REM sleep – might promote the intensification of sensorial, emotional and cognitive processes shaping the *oneiric* experience and result in perceptually vivid, realistic and emotionally absorbing dream images. The detailed description of these findings and our further conclusions and speculations are presented in **Study 4.** *(Simor, P., Horváth, K.,Ujma, P., Gombos, F., Bódizs, R. (2013) Fluctuations between sleep and wakefulness: wake-like features indicated by increased EEG alpha power during different sleep stages in nightmare disorder. Biological Psychology (in press) doi: 10.1016/j.biopsycho.2013.05.022.)*

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