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Impaired executive functions in subjects with frequent nightmares as reflected by performance in different neuropsychological tasks

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ABSTRACT

Nightmare disorder is a prevalent parasomnia characterized by vivid and highly unpleasant dream experiences during night time sleep. The neural background of disturbed dreaming was proposed to be associated with impaired prefrontal and fronto-limbic functioning during REM sleep. We hypothesized that the impaired prefrontal and fronto-limbic functioning in subjects with frequent nightmares would be reflected at the behavioral level during waking tasks as well. 35–35 Subjects with frequent nightmares and matched controls participated in *Study 1*, involving an Emotional Go/NoGo, an Emotional Stroop task, and a Verbal Fluency task. Nightmare subjects exhibited longer reaction times in the Emotional Go/NoGo and Emotional Stroop tasks. Moreover, they committed more perseveration errors and showed less fluent word generation in the Verbal Fluency task. Nightmare subjects showed an overall slowing irrespective of the valence of the stimuli. While the effects of sleep quality and waking anxiety were associated to these deficits in some cases, these factors could not solely explain the difference between the two groups. In *Study 2*, 17 subjects with frequent nightmares and 18 controls were compared by a Color-word and an Emotional, block design Stroop task in order to avoid the slow effects of emotional interference potentially caused by previous items. Nightmare subjects were characterized by an overall slowing in the Emotional Stroop task, irrespective of the valence of the stimuli. In the Color-word Stroop task, nightmare subjects were not significantly slower in comparison with controls. Our results suggest that individuals with frequent nightmares are impaired in executive tasks involving the suppression of task-irrelevant semantic representations.

1. Introduction

Idiopathic nightmare sufferers frequently – at least once a week – experience visually vivid, intense and disturbing dreams that involve fear, anxiety, anger, sadness, disgust or other unpleasant emotions (Nielsen & Zadra, 2010). According to the *International Classification of Sleep Disorders*, 2nd edition (ICSD-II, 2005) these dream disturbances end in abrupt awakenings. However, research on the nature of bad dreams (disturbing dreams that do not awaken the dreamer) suggests that the awakening criterion for nightmare disorder is unnecessarily narrow (Blagrove, Farmer, & Williams, 2004; Spoormaker, Schredl, & van den Bout, 2006; Zadra & Donderi, 2000). Others proposed that *disturbed dreaming* forms a continuum from normal dysphoric dreaming to post-traumatic nightmares where the pressure for awakening varies as a function of situational and dispositional factors as well (Levin, Fireman, Spendlove, & Pope, 2011; Levin & Nielsen, 2007).

Disturbed dreaming is associated with a variety of psychopathological conditions (Agargun et al., 2007; Besiroglu, Agargun, & Inci, 2005; Krakow et al., 2002; Roberts & Lennings, 2006; Semiz, Basoglu, Ebrinc, & Cetin, 2008). These findings provide valuable data on the comorbidity of mental disorders and dream disturbances, but cannot reveal the mechanisms and emergence of disturbed dreaming *per se* because of the confounding effects of waking pathology. While the psychiatric perspective assumes that nightmares and bad dreams are "mere" symptoms of an underlying mental disorder, recent findings suggest the nature of this relationship to be more complex (Lancee, Spoormaker, & van den Bout, 2010; Spoormaker & Montgomery, 2008). For instance, some studies have failed to detect a direct association between psychopathology and nightmare frequency (Levin & Fireman, 2002; Levin & Nielsen, 2007), especially when mental disorders were examined among a sample of frequent nightmare reporters (instead of the inverse, investigating nightmare frequency in psychiatric populations) (Lancee et al., 2010). The association between nightmares and mental complaints seems to be mediated by *nightmare distress*, the affective and cognitive impact of nightmares on daytime

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functioning (Belicki, 1992; Blagrove et al., 2004). Furthermore, nightmare frequency was shown to be a stable disposition with high genetic heritability, which was independent of the genetic influences of general waking anxiety (Coolidge, Segal, Coolidge, Spinath, & Gottschling, 2010).

These findings suggest that instead of the prevailing view of being a symptom of waking dysfunctions, frequent nightmares should be conceptualized as a specific sleep disorder (Spoormaker et al., 2006). Despite their relatively high prevalence (4–5%) in the general population (Nielsen & Zadra, 2000), the underlying mechanisms of nightmare disorder were only scarcely investigated (Germain & Nielsen, 2003; Nielsen, Paquette, Solomonova, Lara-Carrasco, Popova, et al., 2010; Nielsen, Paquette, Solomonova, Lara-Carrasco, Colombo, et al., 2010).

Recent findings indicate that sleep is intimately related to the processing and probably to the regulation of affect-laden memories (Walker, 2009). Studies examining sleep-dependent emotional memory consolidation indicate that emotional processing may especially benefit from REM sleep (Nishida, Pearsall, Buckner, & Walker, 2009; Wagner, Gais, & Born, 2001; Wagner, Hallschmid, Rasch, & Born, 2006). Moreover, REM sleep involves the intense activation of an emotional network comprising the amygdala, the anterior cingulate and the ventromedial prefrontal cortex (Desseilles et al., 2006; Maquet et al., 2005; Muzur, Pace-Schott, & Hobson, 2002). Nightmares generally – but not exclusively – occur during REM sleep (Spoormaker et al., 2006) suggesting that disturbed dreaming is an example of dysfunctional emotional processing during REM sleep. In their integrative model, Levin and Nielsen (2007) proposed that nightmares may be the consequence of the inefficient down-regulation of amygdalar over-activation, and a failure to provide new spatiotemporal contexts for the fearful emotional memories processed during REM sleep. According to the model, the former dysfunction is related to impairments in the ventromedial prefrontal and the anterior cingulate cortex, while the latter is related to impaired hippocampal functioning. Furthermore, these impairments are related to the failure in creating adaptive fear-extinction memories leading to emotional dysregulation during sleep (Levin & Nielsen, 2007; Levin & Nielsen, 2009; Nielsen & Levin, 2007).

Recent findings on healthy subjects support the idea that sleep and especially REM sleep has an important role in the generalization and consolidation of fear-extinction memories, respectively (Pace-Schott et al., 2009; Spoormaker et al., 2010). Nevertheless, no prior studies have examined fronto-limbic abnormalities and/or related executive functions in subjects with frequent nightmares. We hypothesized that if nightmare subjects were characterized by impaired prefrontal and fronto-limbic functions during REM sleep, alterations in these networks would be reflected during waking tasks as well. Therefore, the aim of our experiments was to test the neurocognitive model of Levin and Nielsen (2007) through different neuropsychological assessments of executive functions.

2. Study 1

In order to examine the behavioral effects of impaired prefrontal and fronto-limbic functioning in subjects with frequent nightmares, we applied a series of neuropsychological tasks that were previously shown to rely on these brain areas. Executive functions or cognitive control processes involving the manipulation of items in working memory or the inhibition of prepotent but inappropriate response tendencies are considered to activate mainly prefrontal and related neural structures (Botvinick, Cohen, & Carter, 2004; Bush, Luu, & Posner, 2000; Dillon & Pizzagalli, 2007; Garavan, Ross, Murphy, Roche, & Stein, 2002; Rueda, Posner, & Rothbart, 2005). In case of emotional information, such functions seem to activate more specifically the ventrolateral and the ventromedial prefrontal, as well as the rostral anterior cingulate cortex (Bremner et al., 2004; Bush et al., 2000; Chiu, Holmes, & Pizzagalli, 2008; Lane et al., 1998; Wingenfeld et al., 2009).

In light of these findings and based on Levin and Nielsen's (2007) neurocognitive model presuming fronto-limbic impairments as the neural background of disturbed dreaming, we anticipated that the nightmare (NM) group – in comparison with the controls (CTL) – would show worse performance in different executive tasks, and especially in those that require the processing of negative emotional information. In order to test this hypothesis we applied three well-characterized paradigms, the Emotional Go/NoGo task, the Emotional Stroop task and the Letter-and Category Fluency task.

The Go/NoGo task is a frequently used paradigm to assay motor response inhibition to perceptual stimuli (Aron et al., 2007). The task involves the presentation of a series of "Go" cues to which subjects have to press a button as quickly as possible, and "NoGo" cues that require the inhibition of this motor response. In the emotional version of this task (Reynolds & Jeeves, 1978), emotionally salient (e.g. happy and/or angry faces) perceptual stimuli are interspersed with emotionally neutral stimuli (neutral faces). The Emotional Go/NoGo task assesses response inhibition in the context of affective information processing, allowing the investigation of perturbations in emotional processing. Previous studies indicate that the Emotional Go/NoGo task activates the ventrolateral prefrontal cortex in relation to response inhibition, while the ventromedial prefrontal cortex and the rostral anterior cingulate cortex are activated in relation to the processing of negative emotional information (Chiu et al., 2008; Dolcos, Kragel, Wang, & McCarthy, 2006; Dolcos & McCarthy, 2006; Hare & Casey, 2005). Since the proper functioning of these networks are reflected by the behavioral measures of reaction time and accuracy (Hare & Casey, 2005; Waters & Valvoi, 2009), *we hypothesized that NM subjects – in comparison with CTLs – would be characterized by longer reaction times and more false alarms, especially in the condition involving the inhibition of negative emotional information (Neutral Go/AngryNoGo).*

The Emotional Stroop task is a widely used tool to investigate attentional bias and emotional interference caused by emotionally salient stimuli (MacLeod, Mathews, & Tata, 1986). The task involves the presentation of neutral and emotionally charged stimuli (e.g. neutral and emotionally negative words) with different colors, and participants are asked to press the button corresponding to the color of the word as quickly as possible. Since the semantic content of the words are irrelevant for the task, subjects may suppress distracting semantic representations, and focus only on the perceptual information (the color) of the presented words. Emotionally charged words may produce stronger interference, since they capture the attention more effectively than neutral words, and may also require additional cognitive processes in order to suppress the semantic content and also to regulate evoked emotional reactions. Consequently, reaction times are generally longer for affect-laden words, especially in subjects who are characterized by emotional dysregulation (Becker, Rinck, Margraf, & Roth, 2001; Bremner et al., 2004; Hope, Rapee, Heimberg, & Dombeck, 1990; Mattia, Heimberg, & Hope, 1993). Brain imaging studies indicate that the Emotional Stroop task is associated with enhanced activation in the amygdala, the anterior cingulate cortex and the middle frontal gyrus (Bremner et al., 2004; Bush et al., 2000; Whalen et al., 1998; Wingenfeld et al., 2009). In light of these findings, *we expected that NM subjects – in contrast to CTLs – would exhibit worse performance in the Emotional Stroop task, reflected by longer reaction times and/or more errors in the trials involving negative emotional stimuli. In other words, we expected enhanced emotional interference in the nightmare in comparison with the control group.*