THE ASSOCIATION OF LIMBIC SYSTEM ACTIVATION WITH DREAM, BAD DREAM AND NIGHTMARE GENERATION

by

Warren King

Thesis presented for the degree
Doctor of Philosophy (PhD) in Psychology

Supervisor: Professor Mark Solms, PhD
Co-supervisor: Dr. Kevin Thomas, PhD

Department of Psychology
UNIVERSITY OF CAPE TOWN
April 2018

The financial assistance of the National Research Foundation (NRF) towards this research is hereby acknowledged. Opinions expressed and conclusions arrived at, are those of the author and are not necessarily to be attributed to the NRF.

ABSTRACT

THE ASSOCIATION OF LIMBIC SYSTEM ACTIVATION WITH DREAM, BAD DREAM AND NIGHTMARE GENERATION

Warren King

University of Cape Town

April 2018

Despite the fact that nightmares occur with regularity in the general population, most previous research has focused on clinical samples, and the genesis of idiopathic nightmares remains poorly understood. The aim of the present research was therefore to investigate the neuropsychological mechanisms of idiopathic bad dream and nightmare generation, with a particular focus on the limbic system. High versus low levels of limbic activation and its effect on the frequency of dream, bad dream, and nightmare recall, characteristics, and content were investigated using retrospective and prospective measures. Psychosocial stress – a phenomenon which increases activity in the limbic system – and its relationship to bad dreams and nightmares was also investigated, using questionnaires and a prospective dream diary study. Oral contraceptive use was included as a moderator variable as previous research has indicated that this may temper reactions to stress. The general hypothesis that greater activation of the limbic system results in a greater frequency of recall of bad dreams and nightmares, and also results in more negative dream content, was confirmed. It was also found that external factors which increase limbic activation such as psychosocial stress lead to a greater recall of bad dreams and nightmares. Although oral contraceptive use did not moderate the relationship between stress and bad dream and nightmare recall frequency, more generally positive dream content was found in users of oral contraceptives compared to non-users. Taken together, the results of the studies indicate that similar neuropsychological mechanisms may underlie the formation of idiopathic nightmares and nightmares in clinical conditions, and also that increased levels of limbic activation may result most commonly in negative dream content.

ACKNOWLEDGEMENTS

The research reported in this dissertation would not have been possible without generous funding from the National Research Foundation (NRF) of South Africa. Thank you to Professor Mark Solms for invaluable comments on previous drafts of this dissertation. Thank you also to Professors Martin Teicher, Kathryn Belicki, and Tore Nielsen for providing the Limbic System Checklist-33, Nightmare Distress Questionnaire and Typical Dreams Questionnaire, respectively, for use in some of the studies presented here.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER 1

INTRODUCTION AND OVERVIEW

INTRODUCTION

Despite several decades of clinical and empirical research on nightmares, the etiology of idiopathic nightmares is still unclear (Nielsen, 2017a). Idiopathic nightmares have been found to occur with a prevalence of around 2-5% in the adult population. Otsuka et al. (2018), for example, found nightmares to occur with a prevalence of 3% in a Japanese sample, similar to the prevalence estimate in a representative German sample of 2.4% reported by Schredl (2010). Sandman et al. (2013) reported prevalence estimates of 3.5% for men and 4.8% for women in the Finnish general adult population and war veterans over a more than 30 year period. Li, Zhang, Li, and Wing (2010) reported a prevalence estimate of 5.1% in a Hong Kong sample. Nightmares rise to the level of a clinical problem in approximately 5% of the population (Nielsen, 2017a).

Despite the high prevalence of nightmares in the general population, most research has focused on clinical populations. Nightmares resulting from posttraumatic stress disorder (PTSD) have received the most attention due to the "inseparable association" between the two phenomena (Levin & Nielsen, 2007, p. 494). Criterion B in the *Diagnostic and Statistical Manual of Mental Disorders (5th edition; DSM-5)* (American Psychiatric Association, 2013) even lists nightmares as one of the potential requirements for diagnosis. Prior research has also focused on nightmares in individuals with other clinical disorders (e.g., schizophrenia, anxiety disorders), and on the psychopathological correlates of frequent nightmares (Nielsen & Levin, 2007; Spoormaker, 2005). Since much previous research has eschewed the study of idiopathic nightmares in favour of nightmares in clinical conditions, there is at present a paucity of research on the neuropsychological mechanisms underlying the formation of bad dreams and nightmares in non-clinical samples. As Levin and Fireman (2002b, p. 110) note: "...little is known about the etiology and pathogenesis of frequent idiopathic nightmares or their connection with waking psychological functioning."

The aim of the present set of studies was therefore to investigate the neuropsychological mechanisms of idiopathic nightmare generation, with a particular focus on the limbic system. As nightmares are often emotionally intense and characterized by fear (Zadra, Pilon, & Donderi, 2006), it is likely that the limbic system of the brain is responsible primarily, though not exclusively, for the formation of nightmares. This is due to the relationship of the limbic structures not only with the generation of anxiety with fear conditioning (Levin & Nielsen, 2007), but also with the formation of long-term emotional memories (Devinsky & D'Esposito, 2004). The limbic system has been implicated in the generation of dreaming in general by both neuropsychological lesion studies (e.g., Solms, 1997, 2000a, 2000b) and neuroimaging studies (e.g., Braun et al., 1997; Maquet et al., 1996; Nofzinger, Mintun, Wiseman, Kupfer, & Moore, 1997), as well as specifically in the formation of bad dreams and nightmares in a recent model (Levin & Nielsen, 2007; Nielsen & Levin, 2007).

The present research entailed three studies. The aim of Study 1 was to demonstrate, in a non-clinical sample, that increased activity in the limbic system is related not only with an increased frequency of dream recall in general, but also an increased frequency of bad dream and nightmare recall in particular. A further aim of Study 1 was to demonstrate that increased activity in the limbic system is responsible for affecting dream, bad dream and nightmare characteristics, as well as most recent dream content. Based on promising results from the first study, which used a retrospective questionnaire, a prospective dream diary study was carried out for Study 2, a study which more closely examined the relationship between limbic system activity and dream content. Study 3 investigated a commonly experienced psychological phenomenon which increases activity in the limbic system – psychosocial stress (Herman, Ostrander, Mueller, & Figueiredo, 2005) – and the effect this stress has on producing bad dreams and nightmares. The moderating effect that oral contraceptive use in females may have on the relationship between psychosocial stress and bad dreams and nightmares was also investigated in this study, due to previous research (e.g., Kirschbaum et al., 1995, 1996) that has indicated that oral contraceptive use may temper reactions to stress.

Before considering the neuropsychological mechanisms of nightmares, a definition of the subject under consideration is provided in the following section.

DEFINITIONS OF NIGHTMARES

Both the *DSM-5* (American Psychiatric Association, 2013) and the *International Classification of Sleep Disorders,* 3rd edition; *ICSD-3*) (American Academy of Sleep Medicine, 2014) broadly define nightmares as extremely dysphoric, well-remembered dreams usually involving threat and occurring in the second half of the night (Nielsen & Carr, 2017). The *International Classification of Diseases (10th revision; ICD-10)* defines nightmares as frightening dreams which precipitate awakenings from sleep (World Health Organization, 2016), although emotions other than fear – such as anger or sadness – are included in the *ICSD-3* definition (American Academy of Sleep Medicine, 2014). Predominant emotions other than fear were also reported in both bad dreams and nightmares in Zadra et al. (2006), and later Robert and Zadra (2014), consistent with results from a much earlier study by Belicki, Altay, and Hill (1985), although Belicki et al. investigated only nightmares.

The awakening criterion has often been used as a differentiator of nightmares from what have been termed *bad dreams*, specifically, that nightmares usually result in awakenings from sleep whereas bad dreams do not. In a study using the awakening criteria as a differentiator of the two dream types, Zadra et al. (2006) found that nightmares were rated as significantly more emotionally intense than bad dreams, particularly in terms of fear. This result was later replicated by Robert and Zadra (2014).

Nightmares and bad dreams are also able to be differentiated in terms of content, Robert and Zadra (2014) found that interpersonal conflicts predominated in bad dreams whereas physical aggression was the most common theme in nightmares. Nightmares were also found to be more bizarre and to have more aggressions, failures, and unfortunate endings than bad dreams. Similar results were found by Fireman, Levin, and Pope (2014), who noted that nightmares contained more primary affect such as fear, more death references, and more aggression than bad dreams.

Researchers have also differentiated between bad dreams and nightmares on the basis of the effect these dreams may have on well-being. Zadra and Donderi (2000) found that six of seven different measures of well-being were more highly correlated with nightmare as opposed to bad dream recall frequency, and that well-being accounted for just over three times the amount of variance in nightmare versus bad dream recall frequency (13% and 4%, respectively). In a study of PTSD-affected war veterans, Schreuder, Kleijn, and Rooijmans (2000) found that participants who experienced frequent awakenings from dreams scored higher on psychoneuroticism and general psychopathology on the Symptom Checklist-90 (SCL-90), and also had greater posttraumatic complaints. Blagrove, Farmer, and Williams (2004), however, found that the frequency of recall of *all* dreams with moderate to severe negative affect was a correlate of decreased well-being, regardless of whether or not the dream was a nightmare. That some studies find bad dreams and nightmares to have similar relationships to low well-being does not necessarily imply that the two dream types are continuous, however. For example, if it was found that nightmares and night terrors – two recognizably distinct parasomnias in terms of their characteristics and sleep correlates – have similar relationships to low well-being, this would not imply that these phenomena are unitary (Spoormaker, Schredl, & van den Bout, 2006).

A distinction between bad dreams and nightmares was maintained for the present research for three reasons: (a) it appears that research participants view the distinction as subjectively meaningful; (b) collapsing the two dream types into a single category of 'nightmares' has implications for recall frequency estimates; and (c) bad dreams and nightmares appear to differ with regards to content. With regard to the first point, Zadra et al. (2006) found that only one of the 101 participants in their study had trouble distinguishing between bad dreams and nightmares, as differentiated by the awakening criterion. With regards to the second point, bad dreams have been found to be more frequent than nightmares according to home logs, such that nightmare recall frequency estimates can become inflated by failing to make a distinction between bad dreams and nightmares (Levin & Nielsen, 2007). For example, a study of almost 10,000 prospectively collected dream reports which distinguished between bad dreams and nightmares found that around 3% of dream reports were nightmares whereas around 10% were bad dreams (Robert & Zadra, 2014). Fireman et al. (2014) also found bad

dreams to be four times as frequent as nightmares, consistent with previous research (Levin & Nielsen, 2007; Zadra & Donderi, 2000). With regards to the third point, content differences between bad dreams and nightmares have been reviewed briefly above.

Finally, there is a need to distinguish between the two main types of nightmares: posttraumatic and idiopathic. Posttraumatic nightmares are often associated with PTSD (Nielsen & Levin, 2007) – but not always, as they can occur as part of a posttraumatic stress reaction without complete PTSD (Spoormaker et al., 2006). Levin and Nielsen (2007) have distinguished between trauma-related posttraumatic nightmares – nightmares that may include certain aspects of the traumatic event but do not replicate it in part or whole – and replicative posttraumatic nightmares, which are nightmares that directly reenact parts of the trauma. An increase in the severity of the traumatic event is said to result in the formation of replicative as opposed to trauma-related nightmares. Idiopathic nightmares are nightmares for which no identifiable cause can be found (Nielsen & Levin, 2007; Spoormaker et al., 2006). Levin and Nielsen (2007) further sub-divide these into idiopathic nightmares of low or high distress. This separation of the two types of idiopathic nightmares might be arbitrary, however, as although some studies (e.g., Levin & Fireman, 2002a) have found a relationship between nightmare distress and psychological disturbance, nightmare distress and recall frequency are often not strongly related (Levin & Nielsen, 2007).

OVERVIEW OF THE DISSERTATION

The dissertation is divided into seven chapters. The present chapter has discussed nightmare prevalence and definitions of bad dreams and nightmares, as well as given an overview of the three studies conducted in this research and the reasons for doing so. Chapter 2 presents theories of dreaming that deal with bad dreams and nightmares, and Chapter 3 reviews clinical conditions associated with bad dreams and nightmares that suggest that the limbic system is intimately involved in the production of these phenomena (e.g., PTSD; temporal lobe epilepsy).

Chapters 4, 5, and 6 present the results of the three studies that were conducted for this thesis. Chapter 4 presents the results of the study which looked at the relationship of limbic system activation to bad dream and nightmare recall frequency, and dream

characteristics. Chapter 5 presents the results of the study of dream content in individuals with high versus low levels of limbic system activation. Chapter 6 presents the results of the study on psychosocial stress and related variables, the effect these variables may have on bad dream and nightmare recall frequency, and the potential moderating effect that oral contraceptive use may have on this relationship. Each of these three chapters contains an introduction and literature review, methods and results sections, and concludes with a discussion section. Chapter 7 presents an overall general discussion of the results obtained in the three studies, the links between the three studies, and what the results mean in terms of theories of bad dream and nightmare etiology. It is to these theories that the dissertation now turns.

CHAPTER 2

THEORIES AND NEUROPSYCHOLOGICAL CHARACTERISTICS OF BAD DREAMS AND NIGHTMARES

INTRODUCTION

The current chapter reviews the functional neuroanatomy of dream and nightmare generation, as well as various theories that seek to explain the origin and function of dreams. The past few decades have seen a proliferation of theories on the cause and function of dreaming, as new research from both clinical and neuroimaging studies has delineated the areas of the brain responsible for dream production. The theories reviewed here either deal explicitly with bad dreams and nightmares, or with aspects that are related to bad dreams and nightmares (e.g., mood and affect).

THE FUNCTIONAL NEUROANATOMY OF DREAMING AND NIGHTMARES

Dreaming

Neuroimaging and lesion studies, as well as studies on the neurotransmitters associated with dreaming, have all made valuable contributions to understanding the brain regions associated with the generation of dreams. As it was previously believed that dreaming and REM sleep are synonymous – and as the majority of dreams arise during REM sleep in normal individuals – the majority of neuroimaging studies have investigated brain activation during this sleep period. During REM sleep, increases in regional blood flow or glucose metabolism have been seen in the pontine tegmentum, dorsal mesencephalon, thalamus, Brodmann area 40 of the right parietal cortex, middle temporal cortex, occipital cortex, and mediobasal prefrontal cortex (Agargun & Ozbek, 2006; Hobson, Pace-Schott, & Stickgold, 2000). Near infrared spectroscopy has also shown the visual cortex to be active during REM sleep (Pagel, 2006).

Braun et al. (1997) found increased levels of activation in a number of basal ganglia, specifically the caudate, putamen and ventral striatum (encompassing the nucleus accumbens and substantia innominata). Numerous limbic structures have been found to

be activated during REM sleep, including the amygdala, hippocampus, insula, entorhinal cortex and anterior cingulate cortex (Agargun & Ozbek, 2006; De Gennaro, Marzano, Cipolli, & Ferrara, 2012; Hobson et al., 2000). Amygdala activation has even been found to be higher during both REM and NREM sleep compared to wakefulness (De Gennaro et al, 2012).

Initially, dorsolateral prefrontal cortex (DLPFC) was found to be relatively inactive during sleep and dreaming (Agargun & Ozbek, 2006), although other studies (Maquet et al., 2005) have found higher DLPFC activation in REM relative to NREM sleep. The left supramarginal gyrus and precuneus of the parietal cortex have been found to be relatively inactive during REM sleep (Agargun & Ozbek, 2006), and the motor and sensorimotor cortex is less active during both REM and NREM sleep, along with the opercular cortex and posterior cingulate cortex (Domhoff, 2011).

There is a large degree of overlap between the neuroimaging studies and lesions studies in terms of the brain regions identified as crucial for dreaming. In an analysis of 110 published cases of near or complete cessation of dreaming, Solms (1997, 2000a) found that in 94 cases the lesion causing the dream cessation was situated near the parieto-temporo-occipital junction, a region vital for generating mental imagery – such as the imagery experienced during dreaming. It appears that the temporo-occipital region is the most important for generating visual imagery during dreaming, as lesions there with no parietal involvement can result in total cessation of dreaming (Domhoff, 2011). Although activation of temporo-occipital areas are not commonly reported in neuroimaging studies of brain activity during REM dreaming, the discrepancy between the lesion and neuroimaging studies may be as a result of the fact that "…activity within the visual ventral stream [during REM sleep and dreaming] might be more heterogeneous at any given time than what neuroimaging studies could find until now" (Dang-Vu et al., 2005, p. 422).

In the other 16 cases with cessation of dreaming reported by Solms (1997, 2000a), the lesion was in the ventromesial frontal lobe, a region which contains connecting fibers linking frontal and limbic structures with the ventral tegmentum. The latter two structures are part of a circuit originating in the ventral tegmental areas of Tsai where

the source cells for the mesocortical and mesolimbic dopamine systems are situated. These systems "…ascend through the forebrain bundles of the lateral hypothalamus via basal forebrain areas … and they terminate in the amygdala, anterior cingulate gyrus, and frontal cortex" (Solms, 2000a, p. 846). Along the way these systems synapse on the nucleus accumbens, stria terminalis and nucleus basalis. Lesions in the DLPFC have no effect on dreaming (Solms, 2000a), in keeping with the findings by neuroimaging studies that the DLPFC is relatively inactive during dreaming.

Taken together, the results of the neuroimaging and lesion studies suggest activation of the amygdala, anterior cingulate cortex, and orbitofrontal cortex could be related to the emotional features of dreams, while the occipitotemporal cortex provides visual dream imagery (Cipolli, Ferrara, De Gennaro, & Plazzi, 2017; Palagani & Rosenlicht, 2011). Hypoactivation of the DLPFC is probably associated with the alterations in logical reasoning, working memory, episodic memory and executive functions that occur during REM sleep – where the majority of dreaming takes place – whereas activation of mesiotemporal brain regions – specifically the hippocampus – could be responsible for episodic memories appearing in dream content (Cippoli et al., 2017; Palagani & Rosenlicht, 2011).

Despite the relative consistency of findings across the neuroimaging and lesion studies, theories of dreaming differ in terms of the precise brain areas that the theorists claim to be involved in dream generation. Due to the fact that the majority of dreaming occurs during REM sleep, it was initially thought that dreaming and REM sleep were synonymous. Thus, it was believed that the same mechanisms responsible for generating REM sleep were responsible for generating dreaming (see, for example, Hobson et al., 2000). In the original reciprocal interaction model (McCarley & Hobson, 1975), acetylcholine (ACh) was said to excite the 'REM-on' cells of the pontine reticular formation of the brain stem, while pontine 'REM-off' cells were noradrenergically (NE) or serotonergically (5HT) inhibitory at these sites. The model was later revised such that REM-on was said to be generated by excitatory connections between ACh and glutamate in the pedunculopontine tegmental nucleus (PPT), laterodorsal tegmental nucleus (LDT) and medial pontine reticular formation (mPRF). REM-off is said to be instigated by 5HT (arising from the raphe nucleus) inhibition of the PPT, LDT, and

mPRF and NE (arising from the locus coeruleus complex) (Hobson, 2009; Hobson et al., 2000).

The discovery of the brain stem mechanisms for generating REM sleep led to the activation-synthesis model of dreaming. *Activation* was attributed to brain stem regions, with Hobson and McCarley originally proposing that: "…phasic signals arising in the pontine brain stem during REM sleep and impinging upon the cortex and limbic forebrain lead directly to the visual and motor hallucinations, emotion, and distinctively bizarre cognition that characterize dream mentation" (Hobson et al., 2000, p. 823). Association and language areas in the brain were said to *synthesize* the random and chaotic signals generated by the brain stem during REM sleep in attempt to make sense of these signals, hence the name *activation-synthesis*.

The activation-synthesis model was later revised to include a number of forebrain structures. In the revised model (Hobson et al., 2000; Muzur, Pace-Schott, & Hobson, 2002), forebrain structures are hypothesized to be activated by the ascending reticular activating system (ARAS), the basal forebrain, and possibly the hypothalamus. This forebrain activation is said to allow consciousness to be experienced during dreaming, as well as eye movement and motor pattern information to be transmitted via the ponto-geniculo-occipital (PGO) system. Diencephalic structures (hypothalamus, basal forebrain) are responsible for autonomic and instinctual function and cortical arousal, which provides consciousness and instinctual elements in deams. Anterior limbic structures – which include the amygdala, anterior cingulate, parahippocampal cortex, hippocampus, and medial frontal areas – are said to be responsible for emotional labeling of stimuli, goal directed behaviour, and movement, which provides emotionality, affective salience, and movement in dreams. Other aspects of the revised activation-synthesis model are described immediately below.

The basal ganglia provide the initiation of motor actions, which in dreams is said to translate into the initiation of fictive movement. The cerebellum, responsible for the fine tuning of motor movement, is also said to contribute to fictive movement in dreaming. Thalamic nuclei relay sensory and pseudosensory information to the cortex, and are said to transmit PGO waves to the cortex in dreaming. The inferior parietal cortex,

responsible for spatial integration of heteromodal input, is said to provide spatial organization in dreams. Visual association cortex, which integrates higher order visual information, is said to provide visual hallucinosis during dreaming.

Deactivation of the dorsolateral prefrontal cortex (DLPFC), responsible for executive functions and logic planning, is said to occur via direct inhibition of this brain structure by acetylcholine. This is said to provide loss of volition, logic, orientation, and working memory during dreaming. Primary motor and sensory cortices, which generate sensory percepts and motor commands, are said to provide input/output blockades during dreaming which results in sensorimotor hallucinosis. Overall, dreaming is viewed by Hobson as being similar to a delirium, in which disorientation, visual hallucinations, illogical thinking, loss of recent memory, and confabulation predominate.

The activation-synthesis model was later revised into the AIM (Activation; Input-output gating; Modulation) model, which has been linked to a theory of protoconsciousness (Hobson, 2009). In this theory, the brain generates a virtual reality during sleep (and wakefulness) as a result of AIM (Hobson & Friston, 2012; Hobson, Hong, & Friston, 2014). Factor A is internal activation of the brain by the ARAS (Hobson, 2009). Factor I is input-output gating between internal activation of the brain and external sensory input and motor output (Hobson, 2009; Hobson & Friston, 2014). This gating is mediated by the brainstem, as well as by PGO waves arising from the pontine brainstem (P), lateral geniculate body of the thalamus (G), and occipital cortex (O) (Hobson, 2009; Hobson & Friston, 2014). Factor M is modulation of the shift from predominately external input during waking to predominately internal input during dreaming, facilitated by certain neuromodulatory neurons, primarily aminergic (Hobson, 2009; Hobson & Friston, 2014).

The models of Hobson have been challenged on a number of counts. Firstly, the assumption that the signals generated by the brainstem are chaotic has been challenged by Jones (2000, p. 956), who has argued that she did not "…know of any physiological evidence that the brain stem activation and stimulation of the forebrain is chaotic and would thus impose a chaotic influence on the cortex in dreams." Secondly, a review study of a number of pharmaceutical agents known to affect dreaming (Pagel & Helfter,

2003) found that adrenergic, aminergic, and dopaminergic neuronal populations have the greatest effects on dreaming, with agents affecting DA, 5HT, and NE being consistently associated with reports of altered dreaming. Only weak associations were found between acetylcholinesterase inhibitors affecting the acetylcholine system and reports of altered dreaming. Pagel and Helfter thus concluded: "This study does not provide good support for the theoretical postulates that cholinergic neurons serve as the primary neuroreceptor system involved in dreaming and nightmares" (p. 65).

Finally, the proposal by Hobson and colleagues that the same brain stem structures and neurotransmitters responsible for generating sleep and its stages are also responsible for generating dreaming has been challenged by Solms (1997, 2000a), who has hypothesized a distinct forebrain dream-generation network. Based on his lesion studies (see above), Solms argued that it was impossible to challenge the assertion by Hobson that dreaming was controlled by REM mechanisms as lesions big enough to obliterate the REM-generator would nearly always result in a loss of consciousness. However, it was possible to examine the corollary – that dreaming is not controlled by forebrain mechanisms – if it could be shown that forebrain lesions that spare the brain stem result in cessation of dreaming.

As noted above, Solms (1997, 2000a) reported a number of cases with exclusively forebrain lesions that did in fact result in near or complete cessation of dreaming. A number of studies have also noted that many dream reports from stage 2 NREM late in the sleep period are indistinguishable from REM dream reports (Domhoff, 2005), which is contrary to the assertion by Hobson that dreaming is synonymous with REM sleep. A recent study (Fosse & Domhoff, 2007) in fact found that: "none of the ten expert judges was able to correctly sort a mixed sample of twenty early-night REM and late-night [stage 2] NREM dreams above a chance level, using any criteria they wanted" (p. 61). Moreover, complex partial seizures during NREM (primarily stage 2) sleep in patients with temporal lobe epilepsy can trigger dreams and/or nightmares (Solms, 1997, 2000a).

Studies have found that activation in the midbrain reticular formation and thalamus may be necessary, although not sufficient, to trigger dreaming during stage 2 NREM

sleep (Domhoff, 2011). The intimate connections that the thalamus has with limbic regions, particularly the hippocampus and posterior thalamus, as well as the fact that the amygdala and orbitofrontal cortex may be active enough in stage 2 NREM sleep to sustain dreaming, likely provides the additional impetus sufficient for dreaming to occur during this sleep stage (Domhoff, 2011; Fosse & Domhoff, 2007). Dream reports elicited from stage 1 NREM, in 50-70% of awakenings from this sleep stage, have also been found to be indistinguishable from REM dream reports in every respect except for length (Solms, 2000a). Taken together, these findings indicate that REM sleep may be the *most frequent*, although not the *only* trigger of dreaming (Domhoff, 2005; Solms, 2002). Solms (2000a) thus proposed a "…specific dopaminergic dream-on mechanism that is dissociable from the cholinergic REM-on mechanism" (p. 849).

In his theory, Solms (1997, 2000a, 2000b) proposed that dopaminergic innervation of mesocortical and mesolimbic areas, specifically, the amygdala, hypothalamus, ventral striatum, ventro-mesial frontal lobe, and cingulate gyrus, is said to trigger dreaming in the context of nonspecific cerebral activation, for example, environmental input, arousal during REM sleep, or epileptic activity during NREM sleep in patients with temporal lobe epilepsy (Hobson et al., 2000). Temporal limbic regions provide affective arousal; the bifrontal white matter provides the 'appetitive interest' necessary for dreaming; and the frontal-limbic areas provide selectivity to dream content (Solms, 1997). The parieto-temporo-occipital (PTO) junction is said to support the mental imagery that is inherent in dreaming, with the left parietal region responsible for symbolic mechanisms in the dream process, and the right for concrete spatial mechanisms. Visual association cortex is said to provide visual imagery in dreams.

A number of studies regarding excessive dreaming support Solms' contention that the dopaminergic circuits are crucial for dreaming. One study (Malcolm-Smith, Koopowitz, Pantelis, & Solms, 2012) has found that a majority of dreams can be characterized as 'approach dreams' based on their content, which supports Solms' contention that dreams are characterized by 'seeking' and thus initiated by the dopaminergic 'seeking system.' A recent study (Perogamvros et al., 2015) also supports this contention, as it was found that participants with frequent idiopathic nightmares scored higher than controls on a scale measuring novelty seeking, particularly the exploratory

excitability/curiosity subscale. Also, drugs that chemically activate the dopamine circuit *stimulate* vivid dreaming and nightmares, and there is also evidence for the corollary: drugs that block activity in the dopamine circuit *inhibit* vivid dreaming and nightmares (Solms, 2000a).

Despite the abundance of evidence in support of the theory provided by the neuroimaging and lesion studies, the contention by Solms that dreaming protects sleep has been challenged on two counts. Firstly, developmental studies have indicated that preschool children sleep adequately before they have developed the capacity for dreaming; and secondly, the leucotomized schizophrenic patients mentioned by Solms that do not dream appear to show normal sleep (Domhoff, 2001). However, in that children with under- or un-developed visuospatial skills could be considered to have 'quasi-lesions' of the parietal lobe, the finding that dreaming is a cognitive development that coincides primarily with the development of visuospatial skills can be taken as further evidence in support of Solms' theory of the brain regions implicated in dreaming. As the development of dreaming may be based on the maturation of the forebrain in children (Domhoff, 2001), the developmental studies on dreaming in children also lead credence to the finding that the forebrain is crucial in the dream-generation network.

Finally, Solms' theory has been criticized for focusing too much on the effect of dopamine on dreaming, while not making a complete account of the effect of acetylcholine. Cortical areas such as the amygdala, limbic cortex, and hypothalamus receive both cholinergic and dopaminergic innervations (Perry & Piggott, 2000), although it may be that the release of acetylcholine during sleep "... facilitates dreaming indirectly via the mediation of DA [dopamine] mechanisms" (Solms, 2000b, p. 1037).

Nightmares

The Affect Network Distress (AND) model (Levin & Nielsen, 2007, 2009; Levin, Fireman, & Nielsen, 2010; Nielsen & Levin, 2007, 2009) seeks to specifically explain the formation of bad dreams and nightmares, where most other models explain more general dreaming processes with bad dreams and nightmares being seen as ancillary to these. In the AND model, bad dreams and nightmares are said to "...result from

dysfunction in a network of affective processes that, during normal dreaming, serve the adaptive function of fear memory extinction" (Nielsen & Levin, 2007, p. 300).

Both cognitive and neurophysiological aspects underlie the adaptive fear extinction function of dreaming prescribed in the model. According to the cognitive part of the model, fear-producing stimuli and their physiological responses are repeatedly paired with non-aversive contexts during the dream state in the case of normal dreaming. This paring leads to fear memories gradually being decreased over time (i.e., fear extinction). Fear extinction is accomplished by means of three cognitive processes: (a) memory element activation; (b) memory element recombination; and (c) emotional expression (Levin & Nielsen, 2007, 2009; Levin et al., 2010; Nielsen & Levin, 2007, 2009). Memory element activation refers to the activation during dreaming of basic units of memories, rather than complete memories. Minimal activation of a fear memory is said to trigger an anxiety dream, whereas more extensive, complete or global activation of the memory triggers nightmares of various kinds.

Once activated, isolated memory elements are recombined into virtual simulations that represent novel arrangements of the memory elements, which thus introduce elements that are incompatible with the existing fear memory. The fear memory is extinguished by means of the pairing of such memories with a novel, non-aversive context (i.e., memory element recombination). The reality mimesis of dreaming facilitates the extinction function by ensuring that fear memories are experienced as akin to waking perception, which facilitates emotional regulation. For fear extinction to occur, fear must be emotionally expressed during dreaming, which the third cognitive process proposed by the AND model supposes.

The cognitive part of the model also includes two aspects related to affect: affect load; and affect distress. Affect load is a state factor, and refers to "...the ongoing accumulation of stressful and emotional negative events that impinge on an individual's capacity to effectively regulate emotion" (Levin & Nielsen, 2009, p. 85). Examples would be interpersonal conflicts or daily hassles (Nielsen & Levin, 2009). Increases in affect load result in a concomitant increase in bad dreams and nightmares. Affect distress is a trait factor, and refers to "...a dispositional tendency to experience heightened distress in

response to emotional stimuli" (Levin & Nielsen, 2009, p. 85). Individuals high in affect distress are said to react more negatively and more severely to increases in affect load, and also to be deficient in emotional regulation (Nielsen & Levin, 2009). Such individuals should therefore be more prone to bad dreams and nightmares, and should experience more distress both during and after a nightmare (Nielsen & Levin, 2007).

The neurophysiological aspect of the model comprises the AMPHAC (amygdala, medial prefrontal cortex, hippocampus, and anterior cingulate cortex) network in the human brain. The anterior hippocampus is said to relay dream memory context to the basal nucleus of the amygdala (responsible for activation of fear memories) for further processing by the central nucleus. The central nucleus then activates the brainstem and hypothalamus, which activate the autonomic and behavioural correlates of bad dreams and nightmares, such as increased heart rate. The medial prefrontal cortex (mPFC) together with the dorsal and rostral anterior cingulate cortex (ACC) gate output from the amygdala. More specifically, the mPFC is responsible for control of extinction memories by regulating amygdala activity, and the ACC is seen as the mediator of affect distress (Nielsen & Levin, 2009).

Indirect evidence from studies on the role of the AMPHAC in disturbed dreaming support the AND model. The same brain regions implicated in the AND model are also implicated in REM sleep, PTSD, anxiety disorders, and differences in emotional regulation (Nielsen & Levin, 2007). Also, studies of patients with tempero-limbic hyperactivation in the form of temporal lobe epilepsy (e.g., Solms, 1997) have found an increased frequency of recall of recurring and non-recurring nightmares in these patients (Levin & Nielsen, 2007). Tunbridge and Weinberg (2014) found that individuals experiencing high levels of anxiety in the previous week were more likely to experience recall of bad dreams and nightmares, which supports the contention in the AND model that high affect load should result in an increased recall of bad dreams and nightmares.

In terms of direct evidence, Simor, Pajkossy, Horvath, and Bodizs (2012) found that frequent NM subjects exhibited lower performance on neuropsychological tests of executive function that contained an emotional component, and this impaired

performance was not explained by either trait anxiety or disturbed sleep. This fits with the proposal that impaired prefrontal and fronto-limbic functioning might be associated with frequent nightmares (Levin & Nielsen, 2007; Levin & Nielsen, 2009; Nielsen & Levin, 2007; Simor et al., 2012).

A number of studies also provide evidence that the affect load and distress factors posited in the AND model are related to nightmares (for review see Levin & Nielsen, 2007, 2009). In addition, Levin, Fireman, Spendlove, and Pope (2011) found empirical evidence that affect load was associated with increased nightmare and bad dream frequency, as predicted by the model. Affect distress was found to be associated with heightened distress for both types of dreams, also in keeping with predictions from the model (Levin et al., 2011).

Despite this empirical support, the affect load and affect distress factors are not without problems. The affect distress factor has been said to possess both state and trait properties, despite the contention that it is purely a trait variable (Weiss, 2007), and it is not clear how the concept of affect distress differs from the personality factor of neuroticism. Whether the construct of affect load differs from that of stress is also not clear. Discriminant validity studies on the two affect factors would go some way towards establishing them as unique variables that influence disturbed dreaming, as well as clarifying the relationships between these factors and other factors such as personality variables.

With regards to the neurophysiological aspect of the model, the addition of the right ventral prefrontal cortex (rVPFC) seems warranted considering that the rVPFC can disrupt ACC activity (Weiss, 2007). The model has also been criticized as focusing too narrowly on nightmares and not enough on dreaming in general (Weiss, 2007). Finally, the finding that 55% of bad dreams and 35% of nightmares contain primary emotions other than fear (Robert & Zadra, 2014) suggests that the focus on fear and the fear extinction function of dreaming by the AND model may need to be broadened. Despite these criticisms, the AND model remains the most comprehensive attempt thus far put forward to explain disturbed dreaming.

The AND model has recently been revised into the Nightmare-Affect Network Dysfunction (NM-AND) model (Nielsen, 2017a). In this model, the hippocampus sends excitatory connections to the ventromedial and dorsomedial prefrontal cortex. The PFC in turn sends excitatory connections to the hippocampus via sensory cortices that provide spatiotemporal imagery, and the hippocampus also sends excitatory connections to the amygdala. The amygdala stimulates the brainstem and hypothalamus to activate the autonomic correlates of fear imagery. In terms of inhibitory connections, the vmPFC is said to inhibit fear responses in the amygdala while a dmPFC-hippocampus circuit controls fear renewal by modulating amygdala output according to different contexts. Finally, the ACC – which is central to fear learning and, in particular, fear extinction – also sends inhibitory connections to the amygdala.

The NM-AND model has in turn been linked to the Stress Acceleration Hypothesis of idiopathic nightmares (Nielsen, 2017a, 2017b). According to this theory, a foreshortening of the infantile amnesia period results in early negative or traumatic events that are usually unavailable to memory becoming available, and thus influencing dreams in adulthood in such a way that these dreams become more nightmarish. This early adversity is also said to alter emotional learning and result in long-term dysfunction of the emotion regulation system (the AMPHAC) in such a way that these early memories exert a greater influence over adolescence and adulthood. This dysfunction also leads to increased fear sensitivity and less effective fear extinction. At the time of writing, one study (Nielsen, 2017b) provides direct support for the Stress Acceleration Hypothesis. Other pieces of evidence providing indirect support are reviewed in Nielsen (2017a).

OTHER THEORIES OF DREAMING, BAD DREAMS AND NIGHTMARES

REM-sleep desomatization

Fisher, Byrne, Edwards, and Kahn (1970) originally proposed a model in which anxiety in REM dreams is modulated by reduced physiological responsiveness during REM sleep. In most cases, nightmares experienced during REM sleep are not associated with corresponding autonomic activation, and this lack of correspondence is said to serve a desomatizing function. Since the physiological response to anxiety usually experienced during waking life is absent during REM dreaming, due to the relative physiological

inactivity of this sleep state, the intensity of anxiety experienced during a dream is diminished as a result. This desomatization process is said to serve the function of guarding sleep, specifically REM sleep, as well as to aid in reducing secondary anxiety, and mastering anxiety. When anxiety exceeds a certain threshold, the desomatization process fails, autonomic response is activated, and the dreamer experiences an awakening typical of those experienced after a nightmare.

This model was extended in Perlis and Nielsen (1992), and Perlis, Giles, Fleming, Drummond, and James (1995), with the original model of desomatization during REM sleep linked to the functional consequence of mood regulation. Mood regulation is achieved via a coupling of increased cognitive activation during REM sleep with decreased arousal; mood dysregulation occurs when REM sleep is coupled with *increased* arousal (Perlis et al., 1995). The regulation of mood is said to be achieved through a process akin to behavioural desensitization, in which anxiety-provoking stimuli are counter-conditioned by pairing them with a state of deep muscle relaxation (Nielsen & Levin, 2007; Perlis et al., 1995). This relates to dreaming in that "dreaming may be likened to the process of exposure and REM sleep atonia may be likened to deep muscle relaxation" (Perlis et al., 1995, p. 164).

Support for both the original and extended models comes from studies which have found reduced autonomic activity during REM sleep (Fisher et al., 1970; Perlis & Nielsen, 1992), as well as studies (Perlis et al., 1995) which have found that increased Beck Depression Inventory scores – in other words, greater abnormal moods – are correlated with increased corrugator muscle activity during REM sleep. As desensitization treatment usually requires many sessions (Shapiro, 1989), ascribing a desensitization function to REM dreaming might therefore explain recurrent dreams – which are often negative in content (Zadra, 1994) – and also recurrent bad dreams and nightmares. Nightmares experienced on a single occasion might therefore be considered accelerated desensitization (Nielsen & Levin, 2007).

Despite the empirical support, certain studies (e.g., Shapiro, 1989) suggest that it may in fact be the rapid eye movements experienced during REM sleep that are responsible for any potential desensitization function of this sleep period. Such a view is incompatible

with the hypothesis that it is the muscle atonia of REM sleep that aids desensitization (e.g., Nielsen, 1991; Nielsen, Kuiken, & McGregor, 1989; Perlis & Nielsen, 1992), as Shapiro (1989) was able to demonstration that multi-saccadic eye movements could effectively desensitize even without muscle relaxation. In fact, eye movement desensitization alone eliminated nightmares at both 1 and 3 month follow up in two of Shapiro's patients in which nightmares were the primary presenting complaint.

Assigning the desensitization function to rapid eye movements alone may explain why NREM nightmares have been found to be marked by more anxiety and a stronger physiological response (e.g., Fisher et al., 1970); without rapid eye movements available to desensitize the anxiety response, it occurs with greater magnitude. It may be, however, that rapid eye movements coupled with muscle atonia produces a greater effect than either alone, which may result in accelerated desensitization during REM sleep. Whether it is the rapid eye movements or muscle atonia – or both – that are responsible for any desomatization and/or desensitization that occurs during REM sleep requires further investigation, especially since the desomatization model has not been tested extensively, and is based on a limited number of polysomnographic recordings (Nielsen & Levin, 2009).

Mood regulation

The mood regulation theory of dreaming (Kramer, 1993) posits that dreaming is a problem-solving method, the function of which is to down-regulate negative affect (Cartwright, 2011). Nightmares are seen to be a result of a failure of sleep and dreaming to contain the emotional surge associated with negative dreams (Nielsen & Levin, 2007), with the subsequent interruption of sleep preventing affect reduction and thus resulting in a failure of mood regulation (Cartwright, 2011).

Empirical support for the theory comes from studies that have found an improvement in morning mood compared to pre-sleep mood. Cartwright, Luten, Young, Mercer, and Bears (1998), for example, found that the 10 highest scorers on a depression scale administered pre-sleep had a pattern of decreasing negative and increasing positive affect across the night. A flat distribution of dream affect across the night was noted for low scorers. Divorced individuals with depression who dream of their ex-spouse with

expressed emotion have been found to exhibit a within-the-night negative to positive affect change, and this change correctly classified remission status in 72% of the depressed individuals (Cartwright, 2011; Cartwright, Luten et al., 1998). van der Helm et al. (2011) found overnight reductions in amygdala reactivity in response to emotional stimuli, compared to increases across the day during waking, which would be consistent with the hypothesis that sleep and dreaming may regulate mood and emotion.

Additional support for the theory comes from studies which have found that individuals suffering from a chronic lack of sleep (i.e., insomniacs) – and therefore hypothetically a chronic lack of mood-regulation during sleep – have an increased risk of suicide and suicidal ideation. For example, Chellapa and Araujo (2007) found that depressed individuals with insomnia had significantly higher scores on a suicidal ideation inventory compared to depressed individuals with hypersomnolence. Agargun and Cartwright (2003) found that a reduction in the dream-like quality of REM reports from the first to the second half of the night was associated with suicidal tendency in depressed individuals. This reduced narrative quality of the end-of-night dreams, coupled with these dreams being more negative in affect, was taken to be indicative of a failure to regulate mood in these individuals. Studies have also found that sleep-deprived subjects exhibit less intense and frequent positive mood, as well as more intense negative mood (Kahn, Sheppes, & Sadeh, 2013).

Despite the empirical support for the theory, it is limited in that it seems only to be able to explain dreaming that occurs when moderately negative mood is experienced. Individuals experiencing "...high and low affect in waking, display less mood regulation following sleep. It is those with moderate presleep mood disturbance who show the predicted dream response with progressive downregulation of mood" (Cartwright, 2011, p. 624). At high levels of mood dysfunction (e.g., as in depression) the mood regulation mechanism of dreaming fails; at low levels it is presumably simply not needed. What purpose is served by dreams that occur in individuals with low levels of mood dysfunction is therefore unexplained by this theory, as dreaming should only occur when pre-sleep mood reaches a moderate level. This is not the case, and in fact REM dreams were elicited from a group of participants by Cartwright, Luten, et al. (1998) with levels of pre-sleep depression so low that a floor effect was evident.

The focus of the mood regulation theory on negative mood and a reduction in negative dream affect across the night raises additional issues in that no attempt has been made to account for the existence and function of positive affect in dreams. Dreams marked by positive affect have been found to occur in normal individuals with high and low levels of pre-sleep depression (Cartwright, Luten, et al., 1998), as well as in individuals who are not depressed, who have remitted from depression, and who have not remitted (Cartwright, Young, Mercer, & Bears, 1998). If the function of dreams marked by negative affect is to regulate this affect, the question of what function is served by dreams marked by positive affect is left unanswered. In a similar vein, while proponents of the theory have attempted to explain dreaming in conditions marked by pathologically negative mood, such as depression, no attempt has yet been made to examine changes in dreaming in conditions marked by pathologically positive mood, such as mania. The mood regulation theory of dreaming is therefore currently incomplete. Lastly, Yu (2015) found that the intensities of both positive and negative emotions reach a peak during the third and sixth REM episodes, which is counter to predictions by the mood regulation theory that positive emotions should progressively increase across successive REM dreams while negative emotions decrease.

Threat Simulation Theory

Nightmares are a prototype dream within the Threat Simulation Theory (TST) (Levin, 2000), the central tenet of which is that "...the biological function of human dreaming is the realistic simulation of threatening events during sleep and the repeated rehearsal of the neurocognitive mechanisms involved in threat perception and avoidance" (Valli et al., 2005, p. 190). The simulation of threatening events during dreaming is said to serve an adaptive function (Valli & Revonsuo, 2009) by allowing a rehearsal of the cortical motor programs associated with threat avoidance, in a setting in which the costs of failing to respond appropriately to a threat are low (Malcolm-Smith, Solms, Turnbull, & Tredoux, 2008). In the context of human evolution, the simulation of realistic threats while dreaming would have conferred an adaptive advantage, and the simulation mechanism (i.e., dreaming) would thus have been selected for and transmitted from generation to generation (Valli & Revonsuo, 2009).

Empirical support for TST is mixed at present, with some studies finding in favour of the theory and others not. Consistent with predictions from the TST, Revonsuo & Valli (2000) discovered that 66.4% of 592 dream reports included a threat. A replication of the aforementioned study in a different population, using different judges to score dream reports, obtained an even higher percentage of reports that included a threat (77%) (Valli, Lenasdotter, MacGregor, & Revonsuo, 2007). Valli, Strandholm, Sillanmaki, and Revonsuo (2008) found that students' dream reports contained an inordinately large percentage of threatening events (72.8%) compared to their real-life experiences according to daily logs (15%), which is also consistent with predictions from TST. Robert and Zadra (2014) found that many of the thematic categories found to characterize nightmares –such as physical aggression and being chased – are consistent with TST, although the authors note that "…it remains unclear to what extent nightmares contain realistic threat perceptions and efficient or successful avoidance responses (p. 416).

In a study investigating threat in recurrent dreams, Zadra, Desjardins, and Marcotte (2006) found that 66% of the dream reports contained one or more threats, approximately the same percentage as found in Revonsuo and Valli (2000). It was expected, however, that a greater percentage of threat would be found in recurrent dreams compared to ordinary dreams, considering that recurrent dreams are "...a particularly well-organized form of dream content" (Zadra et al., 2006, p. 452). However, only a small percentage of the dreams (15%) depicted realistic situations critical for survival, and the dreamer rarely succeeded in fleeing the threat, findings that are in contrast to what is predicted by TST.

The finding that threat in the form of emotional difficulties was discovered in almost 8% of the dream reports analyzed in Zadra et al. (2006) is also problematic for TST, as Revonsuo has argued that it is only realistic physical threats which are most important to survival (e.g., Valli, Revonsuo, Palkas, & Punamaki, 2006). Studies on traumatized children from the war-ravaged Kurdish and Palestinian regions (Valli et al., 2005, 2006) (i.e., children exposed to a high degree of waking threat) have found that the dreams of these children differ primarily on 'non-physical aggression' (Valli et al., 2005) and 'psychologically, socially, or financially severe threat' (Valli et al., 2006), compared to

non-traumatized children. Traumatized children who have been exposed to real threatening events thus do not seem to have the pattern of an increased amount of realistic physical threat in their dreams that the TST would predict. Malcolm-Smith et al. (2008) have also found less threat than expected in the dreams of people exposed to more threatening events in real-life. Realistic survival threats in dreams do not appear to occur with great frequency (see, for example, Malcolm-Smith & Solms, 2004; Malcolm-Smith et al., 2008; Zadra et al., 2006), and realistic escapes from such threats occur with even less frequency (Malcolm-Smith & Solms, 2004; Malcolm-Smith et al., 2008; Valli et al., 2005, 2006; Zadra et al., 2006). One study has even suggested that approach (i.e., seeking) is evident in dreams with greater frequency than avoidance (i.e., fear) (Malcolm-Smith, et al., 2012).

Going beyond threat simulation, Revonsuo and colleagues (Revonsuo, Tuominen, & Valli, 2015a, 2015b) have recently put forward the Social Simulation Theory of dreaming. According to this theory, dream experiences do not merely copy social simulation as experienced during wakefulness, but actively stimulates them in ways and proportions that would confer important evolutionary adaptations (Revonsuo et al., 2015a, 2015b). Support for the theory is taken from studies showing that more characters appear in dream reports than in wake reports; that strangers or unfamiliar people are over-represented in dreams compared to waking life; that dreaming specifically simulates Theory-of-Mind or 'mindreading'; and that social interactions frequently occur in dreams (for review see Revonsuo et al., 2015a). Despite this evidence, the theory has been criticized for focusing too much on a narrow function of dreaming, while neglecting other functions that dreaming might have such as memory consolidation and integration, emotion regulation, and creativity and problem-solving (Dresler, 2015).

Hartmann's theories

Boundary permeability. Hartmann (1989, 1991) proposed a theory in which individuals with 'thin' boundaries of the mind are supposedly more susceptible to frequent nightmares, and are also more open, sensitive, and vulnerable to cognitive and emotional intrusions (Nielsen & Levin, 2009). Hartmann, Rosen, and Rand (1998, cf. p. 32) report that these individuals specifically: (1) allow a lot to enter the perceptual field

at once; (2) experience synaesthesia, often merging thoughts and feelings; (3) have a rich fantasy life; (4) lack common psychological defense mechanisms, and have a less definite sense of personal space; and (5) have a tendency to lose themselves in relationships. Individuals with 'thick' boundaries generally display the opposite pattern of tendencies.

Most, but not all, of the studies on boundary permeability and dreaming have supported the contentions that boundary thinness is related to increased dream recall frequency and that individuals with thin boundaries experience more nightmares. Hartmann et al. (1998) found that thin boundaries were positively correlated with the amount of aggressive interactions in dreams, and the nightmare-like-ness of dreams. The amount of detail and amount of emotion in dreams also explained most of the variance in boundary thinness scores on the Boundary Questionnaire (BQ; a questionnaire developed by Hartmann to measure thickness and thinness of boundaries).

A prospective dream diary study that controlled for gender and age (Schredl, Schafer, Hofmann, & Jacob, 1999) found that persons with thin boundaries recalled dreams more often, reported more negative and emotionally intense dreams – including nightmares – and valued their dreams more, especially with regards to the meaningful and creative aspects of dreams. The finding that boundary thinness is related to Schizotypal Personality Disorder (Hartmann et al., 1998), and that nightmares are more common in schizophrenia-spectrum disorders (Levin & Nielsen, 2007), also fits with the relationship of boundary thinness to nightmare recall frequency.

Pietrowsky and Kothe (2003), also using a dream diary study, reported that frequent nightmare sufferers had thinner boundaries, and that number of nightmares over the 4-week dream diary period was significantly correlated with boundary thinness. Physiological, emotional, and cognitive consequences of nightmares were found to be correlated with total scores on the BQ (indicating thinner boundaries), a finding consistent with other research that has found that frequent nightmare suffers experience more distress from nightmares (e.g., Levin & Fireman, 2002a). Previous findings of greater nightmare distress in frequent nightmare sufferers might therefore be explained by virtue of the fact that such individuals may potentially have thinner

boundaries. A more recent study (Kracmarova & Plhakova, 2015) confirmed the finding that frequent nightmare sufferers have thinner boundaries than nonsufferers, and also the finding that nightmare distress correlates with boundary thinness.

Negative findings regarding the relationship of boundary thinness to nightmare recall frequency have also been reported, however (Hartmann, 1989; Spadafora & Hunt, 1990), and one study (Funkhouser, Wurmle, Cornu, & Bahro, 2001) found no relationships between age, boundary thinness, and dream recall in participants over the age of 60. Considering that boundary thickness is said to increase with age (Hartmann, 1991), the sample of Funkhouser et al. may have been restricted in range on that variable, with the authors admitting as much. Finally, despite the empirical support the boundary permeability theory has received, the concept has been criticized for lacking distinction from the personality dimension of openness to experience (Nielsen & Levin, 2007).

Contextualizing images. Another theory of Hartmann explains emotion in dreams as being contextualized by a prominent, central and dominant dream image, known as a Central Image or Contextualizing Image (CI) (Hartmann & Kunzendorf, 2005-2006; Hartmann, Kunzendorf, Rosen, & Grace, 2001; Hartmann, Zborowski, Rosen, & Grace, 2001). Such an image is defined as a "striking, arresting or compelling image – not simply a story – but an image that stands out by virtue of being especially powerful, vivid, bizarre or detailed" (Hartmann, Zborowski, McNamara, Rosen, & Grace, 1999, p. S131). Paradigmatic examples would include being overwhelmed by a huge tidal wave, or being swept away in a whirlwind, and these images provide a picture context for (i.e., contextualize) the dominant emotion(s) in a dream (Hartmann, 1998; Hartmann, Kunzendorf et al., 2001). The theory is linked to the boundary permeability theory, such that individuals with thin boundaries experience more, and also more intense, CIs in their dreams than individuals with thick boundaries (Hartmann & Kunzendorf, 2005-2006; Hartmann, Kunzendorf et al., 2001).

Most empirical research has supported the notion of CIs in dreams. Hartmann, Kunzendorf, et al. (2001) found that, as expected, recent dreams contained more CIs than recent daydreams. The intensity of the CIs was also rated as greater in dreams than

in daydreams, and a greater effect was obtained for intensity than for simply the presence or absence of CIs. The intensity of contextualizing images has also been found to be more intense after trauma and abuse (Hartmann, Zborowski, et al., 2001). Students who reported a history of abuse had higher CI scores compared to matched controls, and 10 individuals who had experienced a traumatic event had higher CI scores than a student control group. The trauma group in fact had the highest CI scores of all samples in the study.

With regard to the specific emotions that are expressed through CIs, Hartmann, Kunzendorf, et al. (2001) found that fear/terror and helplessness/vulnerability were the two emotions contextualized most often in dreams. At least two other studies (Hartmann, Zborowski, & Kunzendorf, 2001; Hartmann, Zborowski, et al., (2001) have found the same result. Positive emotions appear to result in CIs that are less intense than negative emotions (Hartmann, Zborowski, & Kunzendorf, 2001).

Although fear/terror and helplessness/vulnerability are the two emotions which appear to result in CIs most often, the results of some studies suggest that CIs are in fact not tied to specific emotions. For example, Hartmann, Zborowski, and Kunzendorf (2001) found that the abuse versus no abuse and trauma versus no trauma groups in their study were better differentiated by CI scores than by the type of emotion contextualized, a finding replicated by Hartmann, Zborowski, Rose, and Grace (2001). It thus appears that it is emotional activation or arousal in general that produces the powerful contextualizing dream images (Davidson, Lee-Archer, & Sanders, 2005; Hartmann, Zborowski, & Kunzendorf, 2001). More intense emotional activation seems to result in more intense CIs, and such a correlation was in fact found by Davidson et al. (2005). This same study also found that emotion intensity peaked in the CI scene compared to the preceding and succeeding scenes, with a decline in emotion intensity experienced in the latter case.

Despite the empirical support for the CI theory, it has been criticized as lacking in discriminant validity; specifically, that it has not yet been shown that CIs are independent of things such as dream vividness and dysphoric qualities (Nielsen & Levin, 2007). Some of the descriptors used to identify a CI – particularly *bizarre, vivid,* and

detailed – are not clearly related to emotion, so bias may be inherent in attributing the correlation between CI intensity and emotion to the CIs (Davidson et al. 2005). As one of the central tenets of the theory is that emotion in dreams is expressed through CIs, the theory will be weakened if it is found that factors other than emotions can influence the intensity of the images that are contextualized in dreams.

CONCLUSIONS

Neuroimaging and lesion studies largely concur with regard to the brain regions said to be involved in dream generation. Although it was initially believed that REM sleep and dreaming were synonymous, it was later demonstrated that the two are dissociable, and that a significant amount of dreaming occurs during NREM sleep (particularly late-night stage 2). Dream reports from both NREM stage 1 and 2 sleep are also often indistinguishable from REM dream reports.

Although most theories concerning the brain mechanisms of dreaming overlap at least to some extent in terms of the brain regions they implicate in dream generation, there is still a great deal of debate about the precise neurochemical interactions involved in REM sleep and dreaming. Although it is fairly well-established that acetylcholine is crucial to triggering REM sleep, it does not appear to be as critical in triggering dreaming as other neurotransmitters, such as dopamine. The interactions between the neurochemicals responsible for dream generation thus appear to be more complicated than was initially thought (Pagel & Helfter, 2003).

Various functional models to explain dream and nightmare formation have been advanced by a number of different authors over the years. Although the central theoretical postulates of the models often differ substantially, the common thread running through all of them is a focus on affect (particularly fear and other negative emotions). The focus on emotion in the models fits with the limbic and associated areas identified as critical for dreaming in both the neuroimaging and lesion studies reviewed in this chapter, considering that the limbic system is classically considered *the* centre for emotional conditioning in the brain (LeDoux, 1996; Turkington, 1996). Furthermore, a number of clinical disorders affecting the limbic system have been demonstrated to

result in changes in dreaming, particularly an increased frequency of nightmare recall. These disorders are reviewed in the next chapter.

CHAPTER 3

CLINICAL DISORDERS ASSOCIATED WITH LIMBIC SYSTEM DYSFUNCTION AND NIGHTMARES

INTRODUCTION

Clinical disorders associated with bad dreams and nightmares are reviewed in this chapter. Specific emphasis is placed on clinical disorders which affect the limbic system, in keeping with the hypotheses presented in Chapter 1, as well as the functional neuroanatomy of dream and nightmare genesis presented in Chapter 2 which strongly implicates the limbic system (among other brain structures) as crucial to this process. Although a number of other clinical disorders besides the ones listed below are associated with an increased frequency of nightmare recall (for review, see Levin & Nielsen, 2007), emphasis is placed on the following disorders for two reasons: (a) dysfunction of the limbic system has consistently and clearly been associated with these disorders; and (b) alterations in dreaming, particularly an increased frequency of nightmare recall, has been demonstrated in these disorders.

POSTTRAUMATIC STRESS DISORDER (PTSD)

Posttraumatic stress disorder is a condition that develops in response to being involved in or witnessing a severely threatening traumatic event, although it does not develop in all persons experiencing such an event (Mellman & Pigeon, 2010). PTSD is characterized by "...persisting symptoms that include re-experiencing the trauma with intrusive images, flashbacks, or nightmares; emotional numbing and avoidance behaviour; and heightened arousal" (Mellman & Pigeon, 2010, p. 613). Nightmares are virtually synonymous with PTSD, being reported by around 50-70% of individuals with the disorder (Spoormaker & Montgomery, 2008; Wittmann, Schredl, & Kramer, 2007). In a longitudinal study, Pigeon, Campbell, Possemato, and Ouimette (2013) found that the presence of nightmares was associated with significantly higher PTSD severity both at baseline and at a follow up six months later.

Most nightmares in PTSD occur during REM sleep, although a number of sleep laboratory studies have found nightmares in individuals with PTSD to be as likely to occur during NREM (particularly stage 2, but also stage 1) as REM sleep (Mellman & Pigeon, 2010).

Posttraumatic nightmares are most often reported by combat veterans (see, for example, Kramer, Schoen, & Kinney, 1984) due to the association of combat with PTSD, although such nightmares have also been reported in civilians after experiencing traumatic events (e.g., sexual assault; Krakow et al., 2001). PTSD is also associated with other parasomnias besides nightmares, for example, sleep paralysis and sleep talking (Ohayon & Shapiro, 2000), and sleep disordered breathing and sleep movement disorders (i.e., restless legs syndrome and periodic limb movement disorder) (Krakow et al., 2001). These disorders may be related to a REM sleep mechanism disturbance (Lemarche & De Koninck, 2007), and indeed, a meta-analysis of polysomnographic studies found that patients with PTSD experience more stage 1 sleep, less slow wave sleep, and higher REM density than controls (Spoormaker & Montgomery, 2008).

The content of disturbing dreams and associated sleep disruptions in PTSD may serve to reinforce the disorder, as studies have found that veterans of the Vietnam War with disturbed dreaming often suffer a more severe form of PTSD than those without abnormal dreaming (Agargun et al., 2003). Davis, Byrd, Rhudy, and Wright (2007) found that recall frequency and severity of nightmares in a trauma sample significantly predicted distress outcomes even after controlling for PTSD-related symptomatology, a finding which suggests that nightmares may express or contribute to psychological distress in individuals with PTSD independently of the PTSD symptoms themselves. As another example, Gehrman, Harb, Cook, Barilla, and Ross (2014) reported that nightmare-related distress was correlated with a greater frequency of nightmares in a sample of veterans with PTSD.

Prior research has focused on the replicative nature of nightmares in PTSD (e.g., Ross, Ball, Sullivan, & Caroff, 1989; van der Kolk, Blitz, Burr, Sherry, & Hartmann, 1984), such that the posttraumatic nightmare has been seen as a special class of nightmare. Levin and Nielsen (2007), for example, list replicative posttraumatic nightmares as a separate

class of dream in their typology of dreaming, although they do acknowledge that posttraumatic nightmares may be trauma-related and not exactly replicative. In fact, some recent research has suggested that both replicative and non-replicative nnightmares may occur in the same individuals with PTSD.

For example, Esposito, Benetiz, Barza, and Mellman (1999) found that the majority of dreams in a sample of combat veterans with PTSD contained implausible elements not representative of actual memories, much like normal dreams. Davis et al. (2007) found that the majority of participants with PTSD in their civilian sample reported nightmares that were similar or dissimilar to the traumatic event, rather than exact replications. Similarly, Phelps, Forbes, Hopwood, and Creamer (2011) found that only 45% of their participants reported replay dreams, with 30% reporting mixed dreams, and 25% reporting non-replay dreams. A review (Wittmann et al., 2007) found that only about 50% of posttraumatic dreams comprise exact replications of the traumatic events, indicating that exactly replicative dreams are not as common in PTSD as was once thought. Another review of dream content in PTSD concluded that "...the more comprehensive evaluations of dreams during the chronic phases of PTSD document that salient dream content is not limited to representations of traumatic memories" (Mellman & Pigeon, 2010, p. 614).

However, dreams occurring during PTSD do still contain memories that are less unaltered (i.e., closer to the traumatic event) than is the case in normal dreams (Mellman & Pigeon, 2010). It also appears that more severe concurrent PTSD symptoms are associated with dreams more closely related to the traumatic event (Mellman, David, Bustamante, Torres, & Fins, 2001). Nonetheless, the results of the studies reviewed in this section demonstrate that the categorization of the dreams of individuals with PTSD into any one category in a dream typology is difficult as such individuals may experience more than one category of dreams across the course of the disorder. To this extent, it appears that dreaming in PTSD may be disordered in a more general way, rather than disordered to the extent that replicative nightmares are the sole defining feature of dreams in the disorder.

Numerous neuroimaging studies have documented a link between PTSD and disorder of the limbic system (Liberzon et al., 1998; van der Kolk, 2001), and thus by virtue of the fact that PTSD is strongly associated with nightmares, the link between disorder of the limbic system and an increase in nightmares. Findings between the neuroimaging studies have not been consistent, however. The heterogeneity of findings may be attributable to participants with two distinct sub-types of PTSD being combined in various studies, such that a consistent picture of the brain regions implicated in PTSD has not always emerged. Foa, Riggs, and Gershuny (1995) have suggested that the two distinct forms that PTSD can take are: (a) *a hyperarousal* sub-type, characterized by intrusive memories, re-experiencing/reliving of the trauma, and flashbacks; and (b) *a dissociative* sub-type, characterized primarily by a depersonalization response. These different responses to trauma may even occur across different individuals exposed to the same traumatic event (see, for example, Lanius, Hopper, & Menon, 2003). A review of neuroimaging findings in PTSD by Lanius, Bluhm, Lanius, and Pain (2006) supports the notion of two distinct PTSD sub-types.

Brain areas involved in the hyperarousal sub-type include: the anterior cingulate cortex; medial prefrontal cortex; amygdala; and thalamus (Lanius et al., 2006). According to Arnsten, Raskind, Taylor, and Connor (2015), many symptoms of PTSD as noted in the DSM-V mirror behavior changed associated with weakened prefrontal cortex and strengthened amygdala activity. Disruption of ACC function in PTSD may provide a neural basis for the emotional dysregulation characteristic of the disorder (Lanius et al., 2006), and may explain the increased recall of nightmares in PTSD patients, considering that the ACC may be responsible for regulating fear in dreams (Levin & Nielsen, 2007). The medial prefrontal cortex has been hypothesized to be involved in the retrieval of episodic memories, as well as the extinction of conditioned fear responses (Lanius et al., 2006). Dysfunction of this brain region in PTSD may therefore explain why the dreams of PTSD patients often contain memories that are less unaltered than in the dreams of control participants (Mellman & Pigeon, 2010), and also the increased recall of nightmares. With regard to the latter, this could be as a result of hyporesponsive ventral portions of the medial prefrontal cortex in patients with PTSD failing to inhibit fear expression from the amygdala (Shin & Handwerger, 2009; Kim et al., 2013). The role of the amygdala in fear conditioning is fairly well-established (LeDoux, 2002), as is its role

in fear memory, fear detection, and autonomic activation. Amygdala activation has been implicated in the expression of fear during dreaming (Levin & Nielsen, 2007), and increased amygdala activity in PTSD patients (Lanius et al., 2006) would therefore explain the increased recall of nightmares in this population. It has also been suggested that the intense stress experienced in PTSD could weaken the prefrontal cortex at the same time that dendritic growth in the amygdala is increased (Arnsten et al., 2015).

Disordered thalamic activity has led to PTSD being characterized by some as a disorder marked by thalamocortical dysrhythmia (Lanius et al., 2006). High levels of arousal during traumatic experiences may lead to altered thalamic processing which may disrupt the transmission of sensory information to the frontal cortex, cingulate gyrus, amygdala, and hippocampus. This disruption may in turn lead to a failure to properly integrate memories into the present context, such that traumatic memories remain isolated from normal consciousness (Levin & Nielsen, 2007). Isolation of traumatic memories that are subsequently re-experienced during dreaming may explain why dreams in PTSD are sometimes replicative or contain elements closely related to the traumatic event.

Although not mentioned in the review by Lanius et al. (2006), it is probable that the decreased hippocampal volumes commonly observed in PTSD patients (Levin & Nielsen, 2007; Shin & Handwerger, 2009; van der Kolk, 2001) would be more likely in the hyperarousal sub-type of PTSD. This is because hyperarousal in PTSD may result in increased levels of cortisol, which are known to be toxic to the hippocampus (van der Kolk, 2001). The hippocampus is involved in episodic memory encoding, including emotional episodic memories, as well as playing a role in memory for context during fear conditioning and extinction (Shin & Handwerger, 2009). Some studies have found increased hippocampal activity in PTSD, while others have found diminished activity. It appears that the directionality of hippocampal abnormalities may depend on the type of task used in any particular study, as well as the types of statistical comparisons that are made (Shin & Handwerger, 2009).

Although the precise role of the hippocampus in nightmares has not yet been clarified, it has been hypothesized that the anterior hippocampus may relay dream memory

context to the basal nucleus of the amygdala (Levin & Nielsen, 2007). More specifically, the acquisition and maintenance of fear extinction memories during dreaming may be compromised in PTSD as a result of abnormal hippocampal function, such that fear memories are unable to be properly extinguished, and thus persistently repeat themselves during dreaming in the form of nightmares (Levin & Nielsen, 2007). An alternative possibility is that decreased hippocampal functioning primes incoming stimuli to be interpreted in the direction of the fight or flight response (van der Kolk, 1994), which in dreaming could potentially lead to non-aversive dream content being interpreted as threatening, resulting in a concomitant increase in threatening dreams (i.e., bad dreams and nightmares).

In patients with PTSD of the dissociative sub-type, the most common neuroimaging findings are higher levels of activation in: the superior and middle temporal gyri; the inferior frontal gyrus; the occipital lobe; the parietal lobe; the medial prefrontal cortex; and the anterior cingulate gyrus (Lanius et al., 2006). According to Lanius et al., the activation of the parietal and occipital lobes may reflect visual and somatosensory disturbances described by PTSD patients (for example, patients experiencing looking down on and/or feeling detached from their body during depersonalization experiences).

The finding of increased temporal cortex activation provides a link to patients with temporal lobe epilepsy (who experience chronic temporal lobe hyperactivity due to seizures) who commonly report dissociative experiences during seizures, particularly those with a left-sided focus (Devinsky & D'Esposito, 2004). Penfield and Rasmussen (1957) have also reported symptoms of depersonalization during neurosurgery involving stimulation of the superior and middle temporal gyrus. The finding that frontal brain structures are hyper-activated in patients with PTSD of the dissociative sub-type can be accounted for by a corticolimbic model of dissociation (Lanius et al., 2006). In this model, a reciprocally over-inhibited amygdala due to increased left medial prefrontal activation may lead to decreased emotionality and arousal, which may explain the feelings of depersonalization, derealization, and 'emotional numbing' of patients with the dissociative PTSD sub-type.

What differences in dream and nightmare recall frequency may be attributable to the different PTSD sub-types is not entirely clear, due primarily to a failure to make a distinction between the two sub-types by researchers investigating dreaming in PTSD samples. However, based on the neuroimaging findings reviewed above, it seems likely that an increase in nightmares is more likely in the hyperarousal sub-type than the dissociative sub-type. For the latter, a decrease in dream recall frequency may likely be due to the excessive inhibition of the amygdala and other limbic structures from increased prefrontal and anterior cingulate activation. The results of some studies support this hypothesis. For example, Kaminer and Lavie (1991) found a dramatic decrease in dream recall in a PTSD sample, and Agargun et al. (2003) found a negative correlation between scores on a dissociative experiences scale and duration of nightmares in a sample of individuals who had experienced childhood trauma. More research on differences in dream and nightmare recall frequency and characteristics between the two PTSD sub-types is clearly needed to more closely examine the effects that each sub-type has, and would assist in delineating the brain areas involved in nightmare genesis.

TEMPORAL LOBE EPILEPSY

Patients with temporal lobe epilepsy (TLE) are more likely than the general population to suffer from both frequent non-recurring nightmares, as well as recurring stereotypical nightmares (Penfield & Erickson, 1941; Solms, 1997). Epilepsy is defined as the tendency to have recurrent spontaneous seizures (National Society for Epilepsy (NSE), 1999), and is the second most common neurologic disorder after stroke (Adams & Victor, 1993). Temporal lobe epilepsy can be defined as a collection of disorders in which the primary epileptogenic abnormality is in the temporal lobe (i.e., there exists a focal seizure origin in that region) (Aikia, Salmenpera, Partanen, & Kalviainen, 2000). TLE was previously classified as *complex partial seizure disorder* under the 1989 classification of the epilepsies (Commission on Classification and Terminology of the International League Against Epilepsy (ILAE), 2009) in that consciousness is usually altered as a result of the seizure discharge (Adams & Victor, 1993; Shulman & Devinsky, 2003). This altered consciousness can take the form of altered awareness, impaired responsiveness, or loss of memory for the event, and is in contrast to *simple partial seizures* during which consciousness is not altered (Shulman & Devinsky, 2003).

Alterations of sleep organization have frequently been observed in patients with TLE (Cipolli, Bonnani, Maestri, Mazzetti, & Murri, 2004), with such patients having fragmented and superficial sleep compared to those affected by other epilepsies, including other partial epilepsies (Silvestri & Bromfield, 2004). On the basis of the observation of alterations in sleep organization in patients with TLE, Cipolli et al. (2004) have noted that: "It seems thus legitimate to expect that the effectiveness of cognitive processes underlying dream production and recall varies with respect to the type and severity of epilepsy and presumably according to the brain areas affected by the disease" (p. 407).

Two early studies by Epstein (Epstein, 1964; Epstein & Hill, 1966) linked TLE to dreaming, and in particular, recurrent dreaming. Epstein and Hill (1966) in fact documented a case of TLE in which almost all dream periods in the patient were preceded by temporal spikes on EEG. These spikes then continued throughout the REM period itself, and it was also noted that the dream content experienced by the patient was dysphoric in nature. Epstein (1967) documented that the content of seizures and recurrent dreams in patients with TLE is sometimes highly similar, content which in that particular study involved alterations in body image and position. A much later study (Reami, Silva, Albuquerque, & Campos, 1991) confirmed this earlier finding. Reami et al. documented two cases of TLE in which the epileptic seizures experienced while the patients were awake had similar content to the recurrent dreams experienced by these same patients while asleep. Recording from an EEG demonstrated right anterior temporal discharge in both cases.

Certain studies provide support for the contention that patients with TLE experience a high frequency of nightmare recall. With regards to epilepsy in children and adolescents, a recent review (Schlarb, Christen, Claben, & Bien, 2016) found parasomnias – including nightmares – to be commonly reported by a variety of studies. Regarding epilepsy in adults, Solms (1997) reviewed 24 cases of recurring nightmares in patients with epilepsy reported in the literature from 1915 to 1981, and found that the seizure focus was in the right temporal lobe for 17 of the 19 cases for whom lesion localizing information was available. Solms (1997) also reported nine of his own patients with recurring nightmares, five of whom suffered from complex-partial

seizures (TLE). The recurrent nightmares resolved with use of antiepileptic drugs (AEDs) in two of these cases, and with removal of the irritative epileptogenic focus in one case.

Silvestri and Bromfield (2004) investigated 20 patients diagnosed with TLE using video-polygraphic recording on at least one full night. As with the cases described by Solms (1997), the majority of the patients (14/20) reported recurrent nightmares on a sleep questionnaire and diary distributed to them. These nightmares were reported as déjà vu experiences with intense negative emotional valence. Feelings of unmotivated dread and fear were also common. In addition, no ictal nightmares were recorded during rapid eye movement (REM) sleep, as expected by the fact that most nocturnal seizures occur during non-REM (NREM) sleep (Janz, 1974). This is contrary to what is typically found amongst the normal population, where nightmares are more common in the middle of the night or early morning when REM sleep is more prevalent (Bassetti, Bischof, & Valko, 2005). As in Solms (1997), nightmare prevalence in the sample was found to be inversely proportional to the efficacy of epilepsy treatment, which Silvestri and Bromfield take as evidence in support of the fact that these nightmares are night-time subclinical/EEG ictal equivalents.

Bonanni, Cipolli, Iudice, Mazzetti, and Murri (2002) investigated dream recall frequency in patients with generalized versus complex partial seizures using a dream diary study over a period of 60 days. The results indicated that dream recall ability was unimpaired in both groups of patients, but that dream recall frequency was significantly higher in patients with complex partial seizures, regardless of the side of the epileptic focus. These authors take the results of this study as evidence in support of the assumption that the temporoparietal areas of both hemispheres are involved in the production and recall of dreams. As such, greater activation of the temporoparietal area, as occurs with TLE, may result in a greater number of dreams being generated. This finding is consistent with the finding by Levin and Fireman (2002b) to the effect that normal individuals suffering from frequent nightmares reported not only a greater recall of nightmares, but also a greater recall of dreams in general. The increased NREM sleep associated with epilepsy (Bonnani et al., 2002), and the increased likelihood of nightmares during NREM sleep, may also explain the increased dream recall frequency

of the patients with complex partial seizures observed in this study. As the exact types of dreams experienced by the patients (e.g., normal dreams, dysphoric dreams, nightmares, etc.) were not reported, it is not possible to say for certain if the latter possibility is correct.

Cipolli et al. (2004) collected dream reports from 12 patients with TLE after awakenings from either REM or stage 2 NREM sleep, and analyzed the reports for recall frequency, length, and structural organization. Dream recall frequency was found to be significantly lower after NREM sleep, but did not differ according to seizure focus (left versus right hemisphere), and no interaction between sleep type (REM versus NREM) and seizure focus was found. In addition, no statistically significant difference in terms of dream length with respect to seizure focus, sleep type, or the interaction between the two was found. Likewise, no differences were found in terms of the structural organization of the dream reports with respect to seizure focus, sleep type, and the interaction between these two variables. The fact that all patients in the study were taking AEDs may explain the lack of findings, given the tendency of AEDs to reduce the frequency of nightmare and even normal dream recall in patients with TLE (Silvestri & Bromfield, 2004; Solms, 1997).

Although useful in establishing evidence in support of the fact that dreaming is maintained in both REM and NREM sleep in patients with TLE, only measurements of superficial aspects of the dream reports were used by Cippoli et al., thus precluding any comprehensive analysis of the precise contents of the dream reports and of any differences and/or interactions between seizure focus and sleep type. A further limitation pertains to the fact that no EEG recordings were done, making it impossible to ascertain if the dreams reported after the NREM awakenings were in fact nocturnal seizure equivalents. As with the study by Bonanni et al. (2002), Cipolli et al. also provided no description as to what types of dreams (e.g., normal dreams, dysphoric dreams, nightmares, etc.) were experienced in REM and NREM sleep by their participants.

The fact that temporal lobe epilepsy is associated with dysfunction of the limbic system is well-established. Various studies have found pathological damage involving limbic

and other structures, specifically: the hippocampus; amygdala; fornix; thalamus; mamillary bodies; entorhinal cortex, and olfactory cortex (Bertram, 2013; Hermann et al., 2009). Both hippocampal and amygdala sclerosis has been found in magnetic resonance imaging (MRI) studies of patients with TLE (van Elst, Woermann, Lemieux, Thompson, & Trimble, 2000). Epileptiform discharges in mesial TLE have been found to be initiated by alterations in hippocampus-entorhinal cortex interactions, as well as in the amygdala or entorhinal cortex alone, and seizure-induced cell damage may also result in alterations in limbic network interactions (Avoli et al., 2002). Using EEG-fMRI, Pittau, Grova, Moeller, Dubeau, and Gotman (2012) found that, in unilateral mesial TLE, the amygdala and hippocampus show impaired connectivity on the affected side (and to some extent the healthy side), as well as impaired connectivity with the dopaminergic mesolimbic system and the default mode network. Bonilha et al. (2012) found that aberrant connections can result from altered structural organization of the limbic system in mesial TLE.

A study using single photon emission computed tomography (SPECT) of patients with complex partial seizures found hyperperfusion of cerebral bloodflow in the ispilateral temporal lobe and ipsilateral basal ganglia 60-90s after seizure onset, and in the bilateral medial thalamus, hypothalamus, midbrain, pons, and medial cerebellum more than 90s after seizure onset (Blumenfeld et al., 2004). These results again point to the crucial involvement of the limbic system and associated brain structures in TLE. The intense hypothalamic involvement may play a role in neuroendocrine changes often observed in patients with TLE (Blumenfeld et al., 2004).

Ando, Morimoto, Watanabe, Ninomiya, and Suwaka (2004) found that TLE may be characterized by dopamine hypersensitivity attributable directly to seizure activity, which may explain the nightmare-prone tendency of patients with TLE if the theory of dream generation resulting from dopaminergic innervation (Solms, 1997, 2000a, 2000b) is correct. The fact that reducing seizure activity via AEDs or surgery often reduces nightmare and dream recall frequency fits well with this hypothesis, as reduced seizure activity would result in a concomitant reduction in dopamine sensitivity, thus leading to a reduction in nightmare and dream recall frequency. Put simply, it may be

the case that the relationship between seizure activity and increased dream and nightmare recall frequency in patients with TLE is mediated by dopamine sensitivity.

ANXIETY DISORDERS

The term 'anxiety disorders' refers to several different conditions, such as PTSD, social anxiety disorder, generalized anxiety disorder, and so on (Etkin & Wager, 2007; Marcks, Weisberg, Edelen, & Keller, 2010). The DSM-V currently delineates 12 different types of anxiety disorder in total (American Psychiatric Association, 2013). Mental apprehension, physical tension, physical symptoms, and dissociative anxiety are four primary aspects of the anxiety experience, along with excessive rumination, worrying, and uneasiness (Healy, 2008).

The association of nightmares with anxiety may be genetic, with a study by Coolidge, Segal, Coolidge, Spinath, and Gottschling (2009) having found that "…the covariation between nightmares and anxiety is largely driven by genetic factors" (p. 352), although the amount of covariance was moderate. Coolidge et al. (2009) further discovered that scores on scales of Overanxious Disorder and Separation Anxiety had the highest relationships with nightmare recall frequency, as self-reported by parents for their children.

The relationship between anxiety and dysphoric dreaming is well-documented in children and adolescents, although the relationship in adults is less clear. Mindell and Barrett (2002) found a relationship between a child's level of general anxiety and nightmare recall frequency, as well as the severity of nightmares. The relationship between anxiety and nightmares has been documented in high school students as well (Monroe & Marks, 1977). Nielsen et al. (2000), in a longitudinal study, found a significant association between scores on measures of three different types of anxiety (Separation Anxiety; Overanxious Disorder; and Generalized Anxiety Disorder) and recall of disturbing dreams. The associations were present both at age 13 and 16, for both males and females, with the strongest effect evident for Overanxious Disorder.

It appears that Hersen (1971) was the first to document a positive relationship between nightmare recall frequency and anxiety in adults, as measured by the Taylor Manifest

Anxiety Scale, a result that was later replicated by Haynes and Mooney (1975). In a later study involving frequent nightmare sufferers (defined as individuals who reported an average of two or more nightmares per week), Cellucci and Lawrence (1978) found a correlation not only between state anxiety and nightmare recall frequency, but between anxiety and a number of other sleep-related variables. In general, more anxious participants with more nightmares reported a greater amount of sleep disturbance. A study by Marcks and colleagues (2010) replicated the latter finding, noting that individuals with Generalized Anxiety Disorder were over two times as likely to have sleep problems compared to other individuals in the sample.

Returning to the relationship of anxiety with nightmares, Levin and Hurvich (1995) documented a relationship between nightmare recall frequency and annihilation anxiety in two independent samples, and Zadra and Donderi (2000) documented a moderate-to-large correlation (according to the effect size criteria of Gignac and Szodorai, 2016) between a composite measure of nightmare frequency and scores on the Spielberger State-Trait Anxiety Inventory-Trait and -State. No significant correlations were found between a composite measure of bad dream frequency and the Spielberger measures.

A study by Tunbridge and Weinberg (2014), however, found not only that anxiety predicted bad dream recall frequency, but also that individuals in a 'high anxiety' group experienced more nightmares than individuals in a 'low anxiety' group. Nadorff, Porter, Rhoades, Greisinger, Kunik, and Stanley (2014) reported that patients with a clinical diagnosis of Generalized Anxiety Disorder had more 'bad dreams' than patients without, and also that the frequency of 'bad dreams' decreased in correspondence with Cognitive Behavior Therapy for anxiety. Potvin, Lorrain, Belleville, Grenier, and Preville (2014) also found associations between Generalized Anxiety Disorder and 'bad dreams' but also between bad dreams and unspecified anxiety disorder. Both Nadorff and colleagues (2014) and Potvin and colleagues (2014) used the item "During the past month, how often have you had trouble sleeping because you have bad dreams?" from the Pittsburgh Sleep Quality Index, however, which means that bad dreams and nightmares were conflated in both studies.

A limitation of most previous research is the use of retrospective reports only. One study which did in fact use prospective dream logs (Wood & Bootzin, 1990) found no relationship between nightmare recall frequency and trait anxiety, although no distinction between bad dreams and nightmares was made. In contrast, the study by Celluci and Lawrence (1978) cited earlier, which also used prospective dream logs, did in fact find a positive correlation between nightmare recall frequency and anxiety.

The conflicting results amongst previous research on the relationship between anxiety and bad dreams and nightmares may be due to the term *nightmare* being inconsistently defined in dream research (Zadra & Donderi, 2000). Alternatively, different types of anxiety (e.g., trait, state, generalized, etc.) may be differentially associated with bad dreams and/or nightmares, although it is seldom the case that more than one type of anxiety is evaluated in any one study. The magnitude of the relation also appears to depend on the measure used to assess nightmare frequency (e.g., questionnaire; dream logs; etc.).

The association of anxiety disorders with the limbic system has been established by many neuroimaging studies. A meta-analysis (Etkin & Wager, 2007) of imaging studies in three anxiety disorders (PTSD; social anxiety disorder; and specific phobia) found hyperactivation of the amygdala and insula common across all three disorders. Stein, Simmons, Feinstein, and Paulus (2007) found increased amygdala (predominately left-sided) and insula (bilateral) activation in a group of anxiety-prone students, which included students who met the DSM-IV diagnostic criteria for generalized anxiety disorder (GAD), as well as GAD with social phobia. One participant had GAD, social anxiety disorder, panic disorder, and obsessive-compulsive disorder, and a number of students were prone to anxiety but were sub-threshold for GAD and/or social anxiety disorder.

More recently, Killgore and colleagues (2014) found increased amygdala activation and reduced ventromesial prefrontal cortex activation across a variety of anxiety disorders. Reviews by Adhikari (2014) and Calhoon and Kay (2015) have found that the medial prefrontal cortex and ventral hippocampus act with the amygdala and bed nucleus of the stria terminalis to control anxiety. Given that the same pattern of activation was

observed across a number of different types of anxiety disorders by Stein et al. (2007), Etkin and Wager (2007), Killgore and colleagues (2014), Adhikari (2014) and Calhoon and Kay (2015), it appears that a common neurological substrate underlies most if not all anxiety disorders.

The exaggerated amygdala activity seen in these disorders is likely to be primarily responsible for the increased nightmare recall frequency associated with these disorders, given the link between the involvement of the amygdala in fear and fear as the most commonly experienced emotion in dreams and nightmares. The insula is also known to be active during the processing of negative emotions (Etkin & Wager, 2007). Also of interest is the fact that areas of hypoactivity were found in patients with PTSD in the ventromedial prefrontal cortex and anterior cingulate cortex, but not in social anxiety disorder or specific phobia (Etkin & Wager, 2007). Given that nightmares are generally considered to be the most severe in patients with PTSD compared to other disorders (i.e., the nightmares are sometimes replicative and appear to cause more distress than in anxiety disorders), it appears that amongst other possible factors, amygdala hyperactivity in conjunction with hypoactivity of the brain structures that regulate amygdala output is responsible for the most severe forms of nightmares. Whether hypoactivity of the ventromedial prefrontal cortex and/or anterior cingulate cortex themselves is sufficient to result in an increase in dysphoric dreaming remains to be investigated.

SCHIZOPHRENIA-SPECTRUM DISORDERS

Schizophrenia is a disorder characterized by both positive and negative symptoms. The former refers primarily to delusions, most often of the paranoid type; disordered thought; hallucinations, most often auditory; and psychosis (Barta et al., 1990; Kneisl & Trigoboff, 2009; van Os & Kapur, 2009). Negative symptoms include: blunted affect; impoverished speech; anhedonia; lack of a desire to form social relationships and other impairments in social cognition; and avolition (Brunet-Gouet & Decety, 2006; Carson, 2000; Hirsch & Weinberger, 2003).

Many of the positive psychological symptoms of schizophrenia are predominant during dreaming mental activity, and both REM sleep (from which the majority of dreams and

nightmares emanate) and schizophrenia are characterized by highly similar psychological, electrophysiological, tomographical, and neurochemical disturbances (Gottesman, 2005, 2006a, 2006b). A number of studies have documented the relationship between schizophrenia-spectrum disorders (SSD) and nightmares. The study by Coolidge et al. (2009) (mentioned above in the section on *anxiety disorders*) found the strongest effect size for personality variables to be for the Schizotypal Personality Disorder scale. Hartmann (1984) found that frequent nightmare sufferers had elevated scores on the schizophrenia scale of the Minnesota Multiphasic Personality Inventory, a result that was subsequently replicated by Berquier and Ashton (1992) when frequent nightmare sufferers were compared to a control group.

Levin and Raulin (1991) found positive relationships between three measures of schizotypy and nightmare recall frequency, with the relationships stronger for women than men, as might be expected based on the fact that women are more prone to a greater frequency of nightmare recall than men (Nielsen, Stenstrom, & Levin, 2006). Lusignan et al. (2009) found that patients with schizophrenia experienced a greater frequency of nightmare recall compared to controls, but no differences were found for overall dream recall, number of recurrent dreams recalled, and frequency of different emotions in dreams, although the dreams of the schizophrenics did contain more unknown characters, and the length of their dream reports was shorter compared to controls. In a recent 8-year longitudinal study, Li and colleagues (2016) found that nightmares occurred with a prevalence of 9% in individuals with schizophrenia-spectrum disorders.

In a study examining the relationship of psychosis-proneness to nightmare recall frequency, Claridge, Clark, and Davis (1997) found the greatest predictor to be a scale measuring schizotypal personality, with other aspects of psychosis-proneness unrelated to nightmare recall frequency. Another finding by Claridge et al. was that high schizotypes often reported more enjoyable dreaming, and explain this by appealing to the greater imaginativeness of the schizotype, which may have both positive and negative expressions in dreaming. Hartmann, Russ, van der Kolk, Falke, and Oldfield (1981) also found that many frequent nightmare sufferers had greater imaginativeness

in the form of artistic interests and talents, and that there was a relationship between schizophrenia-spectrum disorders and nightmare recall frequency.

A review (Koffell & Watson, 2009) confirmed that nightmares (along with other unusual sleep experiences) are associated with schizotypy in both clinical and non-clinical samples, although the finding that nightmare recall frequency predicts a number of other subtypes of psychopathology besides schizotypy has challenged the relationship between the two phenomena (Levin & Nielsen, 2007). However, the review by Koffel and Watson (2009) found that unusual sleep experiences and schizotypy are more strongly related to each other than to daytime psychopathological symptoms (e.g., anxiety; depression; or neuroticism) and sleep complaints (e.g., insomnia), indicating that there does indeed appear to be a relationship between nightmare recall frequency and schizotypy. Note though that Michels and colleagues (2014) found that the severity of positive symptoms of schizophrenia was not related to nightmare frequency, even though patients with schizophrenia experienced significantly more nightmares than controls. As a result, they suggest that nightmares in patients with schizophrenia reflect the waking-life distress associated with the disorder and its prodrome, rather than being as a result of the disorder itself.

Stevens (1973) and Torrey and Petersen (1974) were the first to hypothesize that dysfunction of the limbic system may be linked to schizophrenia. According to the limbic theory of schizophrenia "…small structural and functional disturbances of limbic key structures in the medial temporal lobe – especially of the left hippocampal formation and parahippocampal gyrus – play a major role in the pathophysiology of the so-called positive symptoms of schizophrenia: paranoid ideas, delusions, and thought disorder" (Bogerts, 1997, p. 423). Schizophrenia-like reactions and psychosis have been documented in individuals with TLE (Flor-Henry, 1969), also a limbic system disorder, and in fact a recent study found a bi-directional relationship between epilepsy and schizophrenia: participants with epilepsy were eight times more likely to develop schizophrenia, and participants with schizophrenia were nearly six times more likely to develop epilepsy (Chang et al., 2011). Furthermore, chemical activation (e.g., by levodopa or amphetamines) of the ventromesial dopaminergic circuit implicated in the limbic system theory of schizophrenia can not only induce some of the positive

symptoms of schizophrenia, but also excessive, vivid dreaming and nightmares (Solms, 2000a).

Due to the ability of drugs such as levodopa to induce schizophrenia-like symptoms, as well as the fact that antipsychotic medications block dopamine activity in the brain, it was for a time thought that schizophrenia may be due to a hyperdopaminergic state (Grace, 2000). This model later proved to be inadequate, with evidence accumulating that schizophrenia is a corticolimbic disorder, one consequence of which is disruption in the cortical regulation of dopamine (Grace, 2000, 2012). Indeed, both older and more recent volumetric and neuroimaging studies have consistently identified limbic system pathology as central to schizotypal disorders.

With regards to volumetric studies, Bogerts (1984) and Bogerts, Meertz, and Schonfeldt-Bausch (1985) identified reduced volumes in temporolimbic structures – specifically, the amygdala, hippocampal formation, parahippocampal gyrus, and pallidum internum – in postmortem brain measurements of schizophrenic patients compared to controls. Using MRI, Barta et al. (1990) identified reduced superior temporal gyrus and left amygdala volumes in a sample of schizophrenic patients, and noted that shrinkage of the left superior temporal gyrus was strongly correlated with the severity of auditory hallucinations. Both Breier et al. (1992) and Rossi et al. (1994) also found reduced left amygdala volumes in an MRI study of schizophrenics compared to controls, with both studies noting in addition reductions in the left hippocampus, and Breier et al. noting reductions in the right amygdala and prefrontal white matter. Degreef et al. (1992) found that temporal horn enlargements (as noted on MRI) were significantly correlated with schizophrenic symptoms. Other studies have found reduced brain volumes in areas that project to, or receive projections from, mesiotemporal regions (Bogerts, 1997).

With regards to positron emission tomography (PET) studies, Tamminga et al. (1992) identified areas of hypometabolism in the anterior cingulate cortex and hippocampus in a sample of non-medicated schizophrenic patients compared to normal controls. Haznedar et al. (1997) also found hypometabolism in the anterior cingulate cortex in a group of schizophrenic males compared to controls, as well as a reduced metabolic rate

in the medial prefrontal cortex, with reduced gating of affective output from the amygdala as a result of a dysfunctional ACC having been linked to an increased frequency of nightmare recall (Levin & Nielsen, 2007). al-Mousawi et al. (1996) reported hypometabolism in the left amygdala but hypermetabolism in the right temporal cortex, including the right hippocampus, in a sample of medicated schizophrenic patients compared to healthy controls. The authors note that these findings were unlikely to be attributable to medication effects as they were absent in a group of similarly-medicated manic patients. Grace (2012) has also noted that the hippocampus appears to be hyper-active in schizophrenia. He notes that "…an explanation for the pathophysiology of schizophrenia points to a dysfunction of hippocampal interneurons leading to overdrive of tonic dopamine neuron population activity; this results in an abnormally amplified dopamine response to stimuli" (Grace, 2012, p. 1344).

This abnormal dopamine response may contribute to an increase in the number of nightmares experienced by schizophrenics given the ability of dopamine to cause excessive, vivid dreaming and nightmares (Solms, 2000a). Gottesman (2006b) has in fact hypothesized that, during REM sleep, increases in dopamine and decreases in glutamate release observed in the prefrontal cortex and nucleus accumbens reach the same levels at which psychotic disturbances arise during wakefulness, such that the state of the prefrontal cortex and nucleus accumbens is the same during REM sleep dreaming as the state of these structures in schizophrenia.

PARKINSON'S DISEASE

Parkinson's disease (PD) is characterized by four cardinal features: tremor at rest; akinesia or bradykinesia (slowness of movement); rigidity of various body parts; and postural instability (Jankovic, 2008). Cognitive and neuropsychiatric abnormalities such as dementia, as well as sensory abnormalities such as olfactory dysfunction, may also characterize the disease (Jankovic, 2008). Degeneration of the substantia nigra, and the resultant loss of dopamine transmission this causes, was originally thought to be the hallmark of the disease and to cause many of the symptom characteristics of the disorder (Braak et al., 2003).

Although the substantia nigra is indeed affected in PD, nigral damage is almost always accompanied by extensive extranigral pathology (Braak et al., 2003). Research by Braak and colleagues (2003, 2004) has indicated that PD in fact proceeds through six stages, with the pars compacta of the substantia nigra affected only in stage 3. The first two stages of PD involve lesions in the dorsal motor nucleus and/or intermediate reticular zone of the medulla oblongata (stage 1), and lesions in the pontine tegmentum (stage 2). The final three stages involve lesions in various areas of the basal prosencephalon and mesocortex (stage 4) as well as in various regions of the neocortex (stages 5 and 6).

Sleep disorders are often present in patients with PD, including nightmares, vivid dreams, sleep fragmentation, daytime somnolence, sleep apnea, and REM sleep behaviour disorder (RBD) (Borek, Kohn, & Friedman, 2007). Nightmares are reported in approximately 30% of patients with PD (Adler & Thorpy, 2005), although estimates as low as 5.7% have been reported (Borek, Kohn, & Friedman, 2006). A number of studies have documented the relationship between PD and nightmares, with this relationship possibly being due to the dopamine agonists used by PD patients in order to counter the loss of dopamine the disease causes. Dopamine agonists are known to cause vivid dreaming and nightmares (Solms, 2000a), and reducing dopaminergic medications may help reduce nightmares in patients with PD (Adler & Thorpy, 2005).

Kumar, Bhatia, and Behari (2002) found nightmares to be present in 32% of a sample of patients with PD compared to an incidence of 5% in a sample of healthy controls. Frequency of nightmare recall positively correlated with levodopa dose, as well as more severe PD symptoms as measured by two scales (the Hoehn and Yahr scale, and the unified Parkinson's disease rating scale), indicating that it may be both the disease itself and antiparkinsonian drugs that contribute to nightmares in patients with PD. However, patients with more severe PD are likely to be on higher levodopa dosages, meaning it may be that levodopa dose serves as a mediator of the relationship between PD symptoms and nightmare recall frequency. Unfortunately this possibility was not investigated by Kumar et al. (2002).

Iranzo, Valldeoriola, Santamaria, Tolosa, and Rumia (2002) reported that nightmares were experienced by eight of 11 patients in their study. The nightmares, as well as RBD

and period limb movements during sleep, in a number of patients) remained after surgery involving deep brain stimulation of the subthalmic nucleus, even though both Parkinsonian symptoms and other sleep complaints improved post-surgery. Oerlemans and de Weerd (2002) found vivid dreams and nightmares to be experienced by 30% of a large sample (N = 234) of outpatients with PD. Van Hilten et al. (1993) found altered dream experience to occur almost exclusively in a sample of patients with PD compared to age-matched controls.

Borek et al. (2006) found that over half (53.3%) of the PD patients in their study had nightmares. Nightmares (as measured by both prospective dream logs and retrospective self-reports) were also found to be significantly correlated with depressive symptoms, anxiety symptoms, increased anxiety, use of levodopa, psychiatric history, and RBD; although RBD, depressive symptoms, and use of levodopa were the only three significant predictors of nightmare recall frequency in a regression model. Nightmare distress was significantly correlated with depressive symptoms, anxiety symptoms, and increased anxiety. Stage of PD correlated significantly with all three latter variables, which suggests a possible mediating effect of these three variables between stage of PD and nightmare distress, although this possibility was not explicitly tested in the study. A more recent, large-scale study on a sample of 661 patients with PD (Ylikoski, Martikainen, & Partinen, 2014) found that 17.2% of the sample experienced nightmares, approximately half the number reported by Kumar et al. (2002), and much less the number reported by Borek et al. (2006).

Although it is commonly believed that sleep disorders are caused by the use of dopamine agonists in patients with PD, sleep disturbances in PD often present before motor symptoms and initial diagnosis. For example, Gaenslen, Swid, Liepelt-Scarfone, Godau, and Berg (2011) reported that 22.6% of PD patients reported nightmares as a prodromal symptom, compared to 6.5% of age- and gender-matched controls. Pont-Sunyer et al. (2015) also found nightmares to occur up to or exceeding 10 years before the onset of motor symptoms. It has also been suggested that the removal of dopaminergic inhibition of the amygdala in conditions marked by dopamine deficiency (such as PD) may be related to the sleep disorders experienced in these conditions, such as nightmares and RBD (Agargun & Ozbek, 2006). Moreover, nightmares have been

noted in patients not taking dopamine agonists who have been suffering from other Parkinsonian syndromes such as multiple system atrophy (MSA), a neurodegenerative disease that causes any combination of parkinsonism, autonomic and cerebellar dysfunction, and pyramidal signs (Wenning, Ben-Shlomo, Magalhaes, Daniel, & Quinn, 1994). For example, Frumkin (1998) reported a case of olivopontocerebellar atrophy – a form of MSA – due to carbon disulfide poisoning in which the patient complained of severe nightmares when he was first seen by the physician, prior to the administration of any medications.

A number of studies have implicated the involvement of the limbic system in PD. Many subnuclei of the thalamus and amygdala are known to be affected in the later stages of the disease, just prior to the anteromedial temporal cortex becoming affected (Braak et al., 2003; Braak, Ghebremedhin, Rub, Bratzke, & Del Tredici, 2004). For example, Kalaitzakis, Gentleman, and Pearce (2013) found that the amygdala, thalamus, and hippocampus have greater amounts of α-synuclein (the primary structural component of Lewy bodies) in PD patients with disturbed sleep. Tau protein deposits were also found to be higher in the amygdala and hippocampus in PD patients with disturbed sleep. Regarding the role of the hippocampus in PD, studies have suggested "… a key role of the interaction between dopamine transmission and hippocampal synaptic plasticity in memory and behaviour. Any imbalance in this interaction might be implicated in the genesis of not only dementia, but also other neuropsychiatric aspects of Parkinson's disease" (Calabresi, Castrioto, Di Fillipo, & Picconi, 2013, p. 811).

The anterior cingulate cortex is also known to be affected in the final two stages of the disease, as is the hippocampal formation (Braak et al., 2003). Neuroimaging studies reviewed by Emre (2003) have confirmed the involvement of the limbic system in the later stages of PD. MRI studies have identified hippocampal atrophy more severe than that found in Alzheimer's disease (AD), and PET studies have consistently identified hypoperfusion in bilateral temporal and parietal cortices. Hypoperfusion in the frontal areas has also been identified, as with schizophrenic patients. It should be noted that the majority of the PET findings have been based on PD patients with dementia, although the findings have also been replicated in some studies using non-demented patients with PD.

Limbic system pathology in PD is often a result of Lewy-neutrites (LNs) and Lewy-bodies (LBs), which may cause Lewy-body-type degeneration in cortical and limbic structures (Emre, 2003). LNs first appear during stage 3 of PD, with LBs typically first appearing during stage 4 (Braak et al., 2003, 2004). It is known that LBs in the temporal lobe can lead to visual hallucinations. For example, Harding, Broe, and Halliday (2002) found that cases of dementia with Lewy-bodies and also cases with Parkinson's dementia that were experiencing hallucinations had more LBs in the parahippocampus and amygdala than cases without hallucinations. Since dreaming often mimics a hallucinatory state, it is reasonable to assume that LBs may also cause nightmares in patients with PD, particularly those with dementia, due to the irritative effect that LBs may have on the hippocampus and amygdala.

Although nightmares in patients with PD may be caused by dopamine agonists, there is thus a possibility that limbic system dysfunction may cause nightmares in such patients independently of dopamine agonist therapy. In most cases, however, the two effects probably work together to produce the altered dream phenomenology observed in PD. Future research evaluating the dream history of new cases of PD prior to the administration of dopamine agonists, as well as research that more closely tracks changes in dreaming associated with stage progression in PD, would be invaluable in disentangling the relative influences of the disease process itself and the disease treatments on dreaming in PD.

As REM sleep behavior disorder appears as a prodrome to PD (and dementia with Lewy bodies) (Dang-Vu, Gagnon, Vendette, Soucy, Postuma, & Montplaisir, 2012), the neureoanatomical correlates of this disorder may also give some insight into the processes responsible for altered dreaming prior to and after a diagnosis of PD. Hogl, Stefani, and Videnovic (2018) have recently noted that RBD is 'prodromal Parkinsonism'.

RBD is characterized by a loss of REM sleep atonia, nightmares, and dream-enacting behaviours during REM sleep (Fernandez-Arcos, Iranzo, Serradell, Gaig, & Santamaria, 2016). Although it was originally thought to affect men far more than women, recent data has suggested that there is in fact gender parity (Schenck & Mahowald, 2018).

Individuals with RBD often experience frequent dysphoric dreaming and negative dream content. For example, a recent study found that 92.6% of patients with RBD reported unpleasant dream recall (Fernandez-Arcos et al., 2016). The majority of dream content was also marked by physical or verbal aggressions, as well as feelings of being chased or falling. Although dream content in patients with RBD often includes aggressive interactions and confrontations, the dreamer is rarely the instigator (Schenck, 2015).

It has been hypothesized that dopaminergic dysfunction is evident in RBD (Sasai, Inoue, & Matsuura, 2012). In line with this hypothesis, Sasai et al. (2012) found that treatment of patients with RBD with a low dose of a dopamine agonist improved symptoms in 80% of their patients, and Howell and Schenck (2015) remark that neuroimaging studies have demonstrated progressive dopaminergic abnormalities in RBD. In terms of brain regions that are affected in RBD, Hanyu and colleagues (2012) found significant gray matter volume reduction in the right and left cerebellum – specifically the anterior lobes – as well as tegmental portion of the pons and left parahippocampul gyrus. Dang-Vu et al. (2012) also noted hippocampal involvement in RBD, specifically abnormal perfusion, and Howell and Schenck (2015), in a review, have also noted that the pontine tegmentum is implicated in RBD.

The pontine tegmentum is implicated in the generation of REM sleep, during which most dreams – and nightmares – occur (Cipolli et al., 2017), and also in the generation of PGO waves, which precede the onset of REM sleep (Hobson & Friston, 2014). The dopamine system has also been crucially implicated in the generation of dreams and nightmares (De Gennaro et al., 2016; Solms, 2000a), and the hippocampus has been implicated in certain models of dream and dysphoric dream generation (e.g., Levin & Nielsen, 2007; Nielsen & Levin, 2007). The limbic system in general has also been implicated in RBD (Poryazova, Oberholzer, Baumann, & Bassetti, 2013) so it is not surprising that patients with RBD would experience altered dreaming and an increased frequency of nightmare recall.

MIGRAINE

Migraine is typified by intense, unilateral throbbing and pulsatile headache attacks (Gupta et al., 2007). A number of different sub-types of migraine exist, such as migraine with and without aura; familial hemiplegic migraine; abdominal migraine; and basilar migraine (see Headache Classification Subcommittee of the International Headache Society, 2004, for a complete list and description of each sub-type). The association between various migraine disorders and nightmares is not commonly reported, due mainly to the fact that most studies on sleep disorders and migraine fail to include a measure of nightmare recall frequency. Studies that have included such a measure have indeed documented a relationship between the two conditions. Anxiety and epilepsy have also been found to show comorbidity with headache disorders (Lateef, Cui, Nelson, Nakamra, & Merikangas, 2012; Rains & Poceta, 2006), and there is a well-documented relationship between both disorders and nightmares (see the relevant sections on temporal lobe epilepsy and anxiety disorders above).

Nightmares have been found to be related to migraine in both children and adults. With regard to children, Bruni, Galli, and Guidetti (1999) found that the severity of migraine attacks was related to the presence of sleep disorders, particularly nightmares, hypnic jerks, and restless sleep. In a large epidemiological study of adolescents (aged 13-18), Lateef et al. (2012) found that adolescents experiencing any type of headaches reported more persistent nightmares than those without headaches. Adolescents experiencing migraine with aura reported the greatest number of nightmares.

In a large epidemiological study of adults, Ohayon (2004) found a greater recall of nightmares in individuals with morning headache (measured as occurring 'daily,' 'often,' or 'sometimes') compared to individuals without, and the odds ratio was higher when only those reporting 'daily' headaches were compared to individuals without headaches. Vgontzas, Cui, and Merikangas (2008) found a greater than fourfold likelihood that adult migraine sufferers would report nightmares beginning in childhood compared to controls. Sadeghniiat, Rajabzadeh, Ghajarzadeh, and Ghafarpour (2013) reported that the frequency of dysphoric dreams increased as the frequency of migraine attacks increased. Both Levitan (1984) and Heather-Greener, Comstock, and Joyce (1996) have found that dreams characterized by negative affect can often predict

migraine attacks, with Levitan reporting that dreams of terror made up the largest category of dreams reported in that study. Podoll, Topper, Robinson, and Sass (2000) reported that in addition to nightmares, recurrent dreams can occur as migraine aura symptoms, and that the elementary geometric imagery seen in the visual aurae of migraine can be incorporated into dreams preceding awakening with a migraine. A higher frequency of dreams in general has also been found in migraneurs (Giani, Casazza, Mariani, & Lovati, 2017).

With regard to the causal mechanisms of migraine, it has been hypothesized that the limbic system is crucially involved. Migraine has even been conceptualized as being part of a dysfunctional neurolimbic pain network (Maizels, Aurora, & Heinricher, 2012). One suggestion is that cortical hyperexcitability followed by subsequent cortical spreading depression is responsible for triggering migraine attacks (D'Andrea & Leon, 2010; Martin & Behbehani, 2001), and that "sporadic migraine susceptibility may be related to polymorphisms in the same or different excitability molecules" that trigger certain types of epilepsy (Lateef et al., 2012, p. 311). Sleep deprivation can also trigger migraine attacks (Martin & Behbehani, 2001) as well as epilepsy (Malow, 2004), and headache is a common postictal epileptic symptom (Devinsky & D'Esposito, 2004).

It has been hypothesized that the symptoms of migraine are triggered by activation of the trigeminovascular pathway of the limbic system and hypothalamus (Burstein & Jakubowski, 2005; Burstein, Noseda, & Borsook, 2015). Specifically, migraine pain signals that originate in the trigeminovascular pathway alter the activity of the parabrachial complex, periaqueductal gray, hypothalamus, amygdala, septum, nucleus accumbens, bed nucleus of the stria terminalis, and basal ganglia. Hadjikhani and colleagues (2013), for example, found increased amygdala connectivity to the visceroceptive insula in both migraine with and without aura.

The activation of the previously mentioned brain structures is hypothesized to result in the symptoms associated with migraine, such as depression, stress, irritability, fatigue, sleepiness, exaggerated emotional responses, nausea, and loss of appetite. Given the association of hypothalamic and amygdala activation with nightmares, it is not surprising that migraines may also result in an increase in nightmare recall frequency.

An MRI study (Schmitz et al., 2008) found brain abnormalities in the medial frontal lobe in migraine sufferers compared to controls, and also that migraine attack frequency was related to reductions in parahippocampal gyrus density. Both of these brain structures have also been implicated in dream and nightmare generation (Levin & Nielsen, 2007; Solms, 2000a).

There is also evidence to suggest that migraine sufferers have alterations in dopamine function (Dosi, Riccioni, della Corte, Novelli, Ferri, & Bruni, 2013), and the relationship of the dopamine system to the pathogenesis of nightmares is well-established (see, for example, the section of PD and nightmares above). Although a review found little supporting evidence for a *direct* role of the dopaminergic system in migraine pathogenesis (Mascia, Afra, & Schoenen, 1998), high levels of dopamine have been found in migraine sufferers (D'Andrea & Leon, 2010), with numerous studies having documented interictal hypersensitivity of dopamine receptors in such individuals (Mascia et al., 1998). Although the dopaminergic systems may therefore not be directly involved in causing migraines, alterations of these systems are often observed in migraine sufferers and such alterations may possibly contribute to the increased recall of nightmares observed in this clinical population.

CONCLUSIONS

The research reviewed in this chapter on the association of various clinical disorders with nightmares underscores the fact that the limbic system is crucial in dream and nightmare production, and that disorders of this brain system – in particular disorders leading to hyperexcitability of this system – can result in an increase in nightmare recall frequency. Whether the same processes predispose individuals without clinical conditions to a greater propensity of idiopathic nightmares remains to be seen. As Weiss (2007, p. 531) has noted: "there is an implication that because the wide range of clinical conditions are by definition pathological and these conditions are associated with nightmares, nightmares are the result of pathological processes." For the present dissertation, three studies were carried out to investigate the possibility that the same process associated with an increased nightmare recall frequency in clinical conditions – hyperexcitability of the limbic system – is associated with idiopathic nightmares in

normal individuals, but to a lesser extent. It is to these studies that the dissertation now turns.

CHAPTER 4

HIGH VERSUS LOW LEVELS OF LIMBIC SYSTEM ACTIVATION AND ITS ASSOCIATION WITH DREAMS, BAD DREAMS AND NIGHTMARES IN A NORMAL SAMPLE

INTRODUCTION

As noted in Chapter 1, the etiology of idiopathic nightmares is still unclear (Nielsen, 2017a). Nightmares often being viewed as a pathological variant of general dream processes (Weiss, 2007) has led to a focus in previous research on the association of various clinical disorders with nightmares, such that there is a dearth of research on the neuropsychological mechanisms of nightmare genesis in non-clinical samples. Based on the research reviewed in Chapter 3, on the association of various clinical disorders with nightmares, it appears that hyperexcitability of the limbic system and associated brain structures may be responsible for the increased frequency of nightmare recall observed in these disorders. The question remains, however, as to whether the same mechanism is responsible for idiopathic, non-trauma-related nightmares. The studies of various disorders associated with both nightmares and limbic system dysfunction reviewed in Chapter 3 hint at this possibility, and point to this possible common substrate between idiopathic nightmares and nightmares in clinical conditions.

The gender disparity in dream, bad dream, and nightmare recall frequency (Schredl, 2014; Schredl & Reinhard, 2011) also points to the limbic system as being crucially involved in dream and nightmare genesis. These differences begin to appear between the ages of 10 to 15 years old (Gauchat, Seguin, & Zadra, 2014), and continue into adolescence and adulthood. Meta-analyses (Schredl & Reinhard, 2008, 2011) have found that there is a small but substantial gender difference in both dream and nightmare recall frequency, with women reporting more dreams and nightmares than men. The smallest gender effects for both dream (Schredl & Reinhard, 2008) and nightmare (Schredl & Reinhard, 2011) recall frequency are for children (people less than 10 years old) and older adults (people greater than 60 years old), with adolescents and young adults having the largest gender effects. A more recent study (Schredl,

Berres, Klingauf, Schellhaas, & Goritz, 2014) confirmed that women report more current and childhood nightmares, as well as more recurring nightmares and dreams in general than men.

It has been suggested that this gender difference may be attributable to a number of factors, specifically: a greater tendency for women to report more distressing experiences (including nightmares); women being more prone to risk factors that produce nightmares (e.g., physical and sexual abuse; anxiety disorders); and a function of processes that lead women to develop more psychosocial depressogenic risk factors (e.g., ruminative coping; bodily dissatisfaction; low instrumentality) (Gauchet et al., 2014; Levin & Nielsen, 2007). Another possible explanation, however, may be biological differences in emotional brain processes linked to the limbic system (Levin & Nielsen, 2007).

Studies have demonstrated better episodic memory for emotional stimuli as well as greater physiological responses to such stimuli for women compared to men (Bianchin & Angrilli, 2012; Bradley, Codispoti, Sabatinelli, & Lang, 2001). For example, a meta-analysis (Stevens & Hamann, 2012) found that women exhibit greater activation than men in the left amygdala and medial prefrontal cortex in response to negative emotional stimuli. Weisenbach et al. (2014) found that women were better than men at identifying fearful faces, and women showed more widespread brain activation for fear than men. Men, on the other hand, had more widespread activation for anger, happy, and sad emotions.

A study by Lungu et al. (2015) on sex differences in fronto-limbic connectivity found that women have a more affective response to negative emotional stimuli, whereas men have a more evaluative response. Nielsen, Powell, and Kuiken (2013) found, however, that females who experience a high nightmare frequency score higher on a scale of motor skill imitation that is related to mirror behaviors, and that the mirror mechanism involved might be concerned with *motor* rather than *emotional* resonance.

Studies in brain morphology have also identified differences in limbic structures between the brains of males and females. A meta-analysis of gender differences in brain

regions responsible for processing emotion (Wager, Phan, Liberzon, & Taylor, 2003) found that women more frequently activate midline limbic structures including the anterior cingulate, thalamus, midbrain, and cerebellum. Yucel et al. (2001) found differences in anterior cingulate cortex morphology using MRI, with males having greater fissurization of the left ACC. Females had greater symmetry, with less fissurization of the left ACC. Increased fissurization is related to faster processing by the relevant brain area (Huster et al., 2009), most likely due to the increased connectivity such fissurization affords (Yucel et al., 2001). Given that the ACC is partly responsible for regulating emotional output from the amygdala and maintaining appropriate levels of fear (Levin & Nielsen, 2007), these findings may indicate that males are able to regulate emotional output during dreaming with greater effectiveness than females, thus possibly reducing the number of nightmares that they are prone to.

In a voxel-based PET study, Kawachi et al. (2002) found increased glucose metabolism in the hypothalamus for females compared to males. The hypothalamus has been linked to producing the autonomic and behavioural correlates of fear during dreaming (Levin & Nielsen, 2007), as it receives efferent connections from the amygdala (Devinsky & D'Esposito, 2004). Hofer et al. (2006), also using PET, found signal increases in the insula, the amygdala-hippocampal-parahippocampal area and the cerebellar vermis during negative mood induction in women. For men, additional activation during negative mood induction was seen only in bilateral cerebellar cortices. The relationship of the amygdala-hippocampal-parahippocampal area to dreaming and nightmares is well-established (Levin & Nielsen, 2007; Hobson et al., 2000; Solms, 1997, 2000a), and the insula has been associated with the processing of emotional stimuli and the monitoring of the internal emotional state of the individual (Damasio et al., 2000; Stein et al., 2007). Cerebellar vermis activation is explicable by the fact that this structure modulates the physiology of limbic lobe structures (Hofer et al., 2006).

Studies of a possible genetic predisposition towards nightmares also point to potential underlying neurological factors that may explain the tendency of some individuals to experience more frequent idiopathic nightmares compared to others. Hublin and Kaprio (2003) have noted that: "The population-based twin studies…indicate substantial genetic effects in the phenotypic variance of five parasomnias (sleepwalking,

sleeptalking, nightmares, bruxism and enuresis)" (p. 419). In childhood, the estimated proportion of total phenotypic variance attributable to the experience of parasomnias varies between 40-70%, while in adults the variance is between 35-80% (Hublin & Kaprio, 2003).

With specific regard to nightmares, Hublin et al. (1999) found that the probandwise concordance rate in childhood nightmares was 0.55 for monozygotic twin pairs, and 0.41 for dizygotic twin pairs. In adults, the concordance rate was 0.32 for monozygotic twins, and 0.20 for dizygotic twins. Hublin, Jaakko, Markku, and Marrku (2001) found moderate phenotypic covariation and shared genetic effects for the occurrence of nightmares in childhood (R = .50) and adulthood (R = .43). It should be noted, however, that unknown non-genetic effects still play a significant part in causing parasomnias – including nightmares – and that the mechanisms of the genetic effects are not known in detail (Hublin & Kaprio, 2003).

Nonetheless, it is also possible that the response of limbic structures such as the amygdala may be moderated by genetic factors such as functional variation in neurotransmitter transporters (Stein et al., 2007). Certain individuals may thus be genetically predisposed to hyperexcitability of the brain structures responsible for generating dreams and nightmares. This hyperexcitability may in turn predispose certain individuals to some of the clinical disorders discussed in Chapter 3 that are linked to nightmares, such as idiopathic TLE, migraine, and anxiety disorders, as all three of these disorders have been shown to have a genetic etiological component. Alternatively, acquired disorders such as symptomatic TLE or PTSD, for example, may result in limbic hyperexcitability which may increase the frequency of nightmares recalled by individuals with these disorders. The Stress Acceleration Hypothesis (Nielsen, 2017a, 2017b) is another, more recent theory that might explain a predisposition to idiopathic nightmares. According to this theory, early childhood adversity alters emotional learning and results in long-term dysfunction of the emotion regulation system, primarily the limbic system.

In sum, although much previous research points to hyperexcitability of the limbic system as likely to result in an increased frequency of nightmare recall, the link is at

present circumstantial, and is confounded by the presence of pathology when examining dreaming in clinical conditions. It is not known whether this mechanism is likely to result in increased nightmare recall frequency in normal individuals. The primary aim of the present study was therefore to investigate the hypothesis that *higher limbic system activation in normal individuals without clinical conditions is associated with more disturbed dreaming*. Based on the literature reviewed in the preceding and present chapters, four specific hypotheses were proposed:

(a) Given that previous research has shown gender differences in the processing of emotional stimuli, as well as gender differences in brain morphology and activity in limbic structures, *females will have a higher level of limbic system activation than males*.

(b) If hypothesis (a) is correct, *the association of limbic system activation with various sleep and dreaming variables will be greater for females compared to males*, although it may still be evident for the latter group. That is, *an interaction effect between amount of limbic system interaction and gender will be expected for various sleep and dreaming variables*.

(c) Given the importance of limbic structures in generating fear and other negative emotions, *individuals with a higher level of limbic system activation will experience more negative dream content*.

(d) More negative dream themes will predict bad dream and nightmare recall frequency compared to positive dream themes.

METHODS

Participants

The sample comprised undergraduate psychology students who participated in exchange for course credit. Initially, data from 582 students was collected. All students who indicated, by self-report, that they had any psychological or psychiatric condition (e.g., depression; anxiety; obsessive-compulsive disorder, etc.) were excluded from the final sample, as were any students who indicated that they had any serious medical condition, and/or were on any type of medication (e.g., antidepressants; lithium; antiretrovirals, etc.). As the present study aimed to investigate the association of limbic system activation with sleep and dreaming variables in a non-clinical sample, all

outliers on the measure of limbic system activation (the *LSCL-33*; see *Measures* sub-section below) were also deleted. This was to control for the possibility that these participants may have had an undiagnosed clinical condition (e.g., an anxiety disorder) that was inflating their score. Outliers were determined using 1.5 multiplied by the interquartile range as the demarcation line for outliers. The final total sample size was N = 526. The average age was 20.51 years (SD = 3.04), and 81% (n = 425) of the sample was female and 19% male (n = 101).

Measures

A number of questionnaires and a most recent dream collection form were included in an online survey. These measures are described below:

(1) The Limbic System Checklist-33 (LSCL-33) (Teicher et al., 1993)[1]. As this is the main measure for this study, it is discussed in slightly more detail than the others. This is a 33-item self-report questionnaire designed to measure functional features associated with the limbic system (Peterson, Henke, & Hayes, 2002). In addition to sum total scores, four sub-scales can be formed: (a) somatic; (b) sensory; (c) behavioural; and (d) mnemonic. Teicher, Glod, Surrey, and Swett's (1993) validation process for this measure involved comparing a group of normal control subjects to patients with temporal lobe epilepsy. Total scores for normal subjects were all less than 10, whereas those for individuals with TLE were all greater than 23 (range 23-60). Scores on this measure also correlate with other measures of limbic system dysfunction, and individuals with post-traumatic stress disorder score significantly higher on the LSCL-33 than individuals without PTSD (Teicher et al., 1993).

Teicher et al. (1993) evaluated criterion-related concurrent validity by correlating scores on the Dissociative Experiences Scale (DES) and Symptom Checklist-90 (SCL-90) with scores on the LSCL-33, using a sample of psychiatric patients. The correlation between overall LSCL-33 scores and DES scores was strong (r = .81). Good correlations were obtained between the four LSCL-33 subscales and total DES scores, with the lowest being for the *sensory* subscale (r = .69) and the highest for the *behavioural* subscale (r = .81). Teicher et al. (1993) also found test-retest reliability of the LSCL-33

[1] The LSCL-33, NDQ, and TDQ were obtained from and used with permission of Martin Teicher, Kathryn Belicki, and Tore Nielsen, respectively. As these scales are copyright of their respective authors, they are not reproduced in this dissertation.

to be high (r = .92) in a sample of patients with widely different psychiatric diagnoses and backgrounds.

A number of other studies point to the validity of the LSCL-33 in terms of measuring limbic activation. Aycicegi-Dinn, Dinn, and Caldwell-Harris (2008) confirmed, in two different non-clinical samples, that individuals with elevated scores on the LSCL-33 demonstrate a personality profile similar to TLE-related personality features and psychiatric symptoms. Bob et al. (2010) found the LSCL-33 to correlate with Beck Depression Inventory scores, symptoms of traumatic stress, and also symptoms of dissociation. Aycicegi et al. (2003) found that individuals with obsessive-compulsive disorder (OCD) had significantly higher LSCL-33 scores compared to controls, consistent with the hypothesis that dysfunction of an orbitofrontal-limbic network is implicated in OCD. Jasova, Bob, and Fedor-Freybergh (2007) used the LSCL-33 to measure limbic activation in alcohol-dependent patients compared to controls, and reported that "…symptoms of limbic irritability produced by a temporal-limbic seizure-like mechanism are significantly related to alcohol craving…" (p. CR545). Colace et al. (2010) found that drug-addicted individuals who had drug-related dreams also had significantly higher LSCL-33 scores than a control group, consistent with a hypothesis that the mesocortical-mesolimbic dopaminergic system, essential for dreaming (Solms, 2000a), is also responsible for drug-related cravings and dreams.

With specific regards to dreaming, Yu (2010) found LSCL-33 scores to correlate with dream intensity, neuroticism, thin boundaries, and, as with Bob et al. (2010), level of dissociation. Although the LSCL-33 did correlate with neuroticism as measured by the Eysenck Personality Questionnaire Revised-Short Form, the correlation was equal to .416, indicating that there is discriminant validity between the LSCL-33 and neuroticism.

(2) The Dreaming Questionnaire (DRM-Q). A self-report questionnaire developed for this study. The DRM-Q has 11 core items which are repeated for dreams (DRMs), bad dreams (BDs), and nightmares (NMs), thus yielding 33 items in total. These items ask participants to report, for example, how affected they are by their dreams, how vivid they find their dreams, how much aggression is in their dreams, and so forth. Most of the items are 5-point Likert-type ratings. Inspiration for this questionnaire came from

the Sleep and Dreaming Questionnaire (SDQ) (Levin, 1994), with the DRM-Q measuring some of the same dimensions (i.e., vividness, intensity, bizarreness, meaningfulness, and degree of colour, movement, and aggression). Additional items on how affected someone is by their dreams, bad dreams and nightmares were added, as well as items on aggression in these three dream types. Aggression in dreams was further split into aggressions directed at the dreamer by other characters and aggressions that the dreamer directs at other characters.

Additional items on a number of other sleep and dreaming variables were also included, specifically: (a) number of hours of sleep per night; (b) sleep quality; (c) awakenings per night; (d) dreams per week; (e) dreams per night; (f) ease of dream recall; and (g) entirety of dream recall. Seven-point category scale items on bad dream and nightmare distress, as well as 8-point items measuring bad dream and nightmare recall frequency were also added. Additional bad dream and nightmare recall frequency measures asked participants to estimate the number of bad dreams and nightmares experienced in the last month, and in the last year. Three items were included to measure recall of recurrent dreams. These items asked: whether or not a participant has ever had a recurrent dream; whether or not they have had a recurrent dream in the last 12 months; and whether or not they have had a recurrent dream in the last 6 months. In total, the questionnaire consisted of 51 items. See Appendix A for the DRM-Q.

(3) The Nightmare Distress Questionnaire (NDQ) (Belicki, 1992)[1]. A self-report measure that measures the degree of global distress an individual experiences as a result of experiencing nightmares. This scale was included as certain studies (e.g., Levin & Fireman, 2002a) have shown that global and local nightmare distress are two separate constructs that are not necessarily correlated.

(4) The Typical Dreams Questionnaire (TDQ) (Nielsen et al., 2003)[1]. A 56-item self-report checklist scale originally developed by Griffith et al. (1958), and extended by Nielsen et al. (2003). The continuous scale version of this questionnaire was used in the present study, where participants rate on a scale from 0 to 4 how frequently they have experienced each of the 56 dream themes listed on the questionnaire.

(5) The Most Recent Dream Method (Domhoff, 1996). Participants were asked to write down the most recent dream they had experienced. This method is powerful and efficient because it allows large samples of dreams to be collected in a relatively short

period of time, has proven reliability and validity, and yields dream reports that are not significantly different from those reported in REM awakening studies (Domhoff, 2003). See Appendix A for the most recent dream instruction form.

Design and procedure

The study was approved by the Research Ethics Committee of the Department of Psychology, University of Cape Town. An extreme contrasted groups design was used in which participants were split into upper and lower quartiles based on scores from the Limbic System Checklist-33. The primary advantages of using extreme group analysis (EGA) are increased cost-efficiency and increased statistical power, particularly for small sample sizes (Preacher, Rucker, MacCallum, & Nicewander, 2005; Preacher, 2015). From a practical point of view, it was necessary to form groups for the present study in order to test for associations between level of limbic activation and the dream content categories (see below). As the content categories are nominal (Domhoff, 2000a), it was not possible to test for parametric correlations between level of limbic activation and dream content. For reasons of consistency, EGA was maintained for all analyses. *LSCL-33 quartile* was thus the independent variable in all analyses. Dependent variables were scores on the various questionnaire-based measures of dreaming and dream characteristics described above.

For the most recent dream reports, data on how long ago these dreams occurred was noted and reports from dreams that occurred more than one month prior to any one participant completing the survey were excluded. This is due to the finding that retrospective one month reports approximate prospective logs better than retrospective reports obtained from longer past time periods (Zadra & Donderi, 2000). This exclusion criterion led to 6 dreams being excluded. All dream reports less than 50 or greater than 300 words were also excluded. This is due to the fact that dream reports that are too short are likely to have less of most things, whereas dream reports too long tend to have more of most thing in them (Domhoff, 2000a). Failure to control for dream length can thus produce spurious differences when comparing dream content between two groups, if one group happens to have dream reports that are much shorter or longer than the other group (Domhoff, 2000a). This criterion led to an additional 33 dream reports being excluded.

Dream reports were coded using the Hall and Van de Castle system of coding. This is the most widely used empirical system of coding dream content, has proven reliability and validity, and is theory-neutral as well (Domhoff, 1999). Nine of the most commonly used categories were coded for, which were: (a) characters; (b) aggression; (c) friendliness; (d) sexuality; (e) success; (f) failure; (g) misfortune; (h) good fortune; and (i) emotions. Dream report codes were entered into and analyzed using DreamSAT (Schneider, 2001), a Microsoft Excel-based program that determines whether or not the category frequencies depart at a statistically significant level from each other. Statistical significance is determined by means of Cohen's *h*, which determines the significance of differences between two independent proportions (see Domhoff, 1996).

All usable dream reports were coded by the researcher, and a random selection of half the dream reports (approximately half from the lower quartile, and approximately half from the upper quartile) were coded by a second independent coder. Any discrepancies in codes were resolved through discussion between the two coders prior to data analysis. As all identifying information except for participant numbers was removed from the reports prior to coding, both coders were blind as to which LSCL-33 quartile participants' reports came from. The independent coder was also blind as to the design and purpose of the study.

Data collection ran for approximately three months, with all data collected via an online survey. All instructions, including details pertaining to informed consent, were on the survey itself. In order to ensure that informed consent was obtained from every respondent, students were not allowed past the informed consent page of the survey without first giving consent to participate in the study. Participants were free to withdraw from the study at any time. Respondents completed the survey at their own convenience, and participation in the study was voluntary, with students able to choose any other study being run at the same or a later time in order to obtain their course credit.

RESULTS

LSCL-33 quartiles

Quartiles were calculated for the total sample (N = 526) using the *LSCL-33 total* scores (M = 28.82, SD = 14.66). The lower quartile (n = 124) was calculated as individuals having scores less than 18, and the upper quartile (n = 127) as scores greater than 38 (maximum possible score = 132). These cut-offs were similar to those found by Aycicegi-Dinn, Dinn, and Caldwell-Harris (2008) in student samples from the United States and Turkey. In the US, those in the high-scoring LSCL-33 group were students with total scores equal to or greater than 36, and those in the low-scoring group had scores equal to or less than 10. In Turkey, those in the high-scoring group had total scores equal to or greater than 41, with the low-scoring group having total scores equal to or less than 11

Gender differences in LSCL-33 scores

For the total sample, scores on the *LSCL-33 somatic*, *sensory, behavioural,* and *mnemonic* subscales, as well as *total* scores, were entered into a MANOVA as multiple dependent variables, with *gender* (male n = 101; female n = 425) as the between-subjects factor. Descriptive statistics are reported in Table 1. A statistically significant overall effect was detected according to Pillai's trace [V = 0.03, $F(4, 521) = 4.15$, $p = .003$] although the effect size was small ($\eta_p^2 = .031$). Follow-up univariate ANOVAs indicated a statistically significant gender effect for only the *somatic* sub-scale [$F(1, 524) = 12.44$, $p < .001$], with females (M = 9.97, SD = 4.92) scoring higher than males (M = 8.10, SD = 4.28), although the effect was again small ($\eta_p^2 = .023$). No statistically significant differences were evident between genders on the *sensory* [$F(1, 524) = 0.01$, $p = .964$], *behavioural* [$F(1, 524) = 1.17$, $p = .279$], or *mnemonic* [$F(1, 524) = 1.38$, $p = .241$] sub-scales. Contrary to what was hypothesized, no gender differences were evident for *LSCL-33 total* scores [$F(1, 524) = 3.27$, $p = .071$]. A Chi-square test of contingency also indicated no statistically significant association between *gender* and *LSCL-33 quartile* for the total sample [$\chi^2(2; N = 526) = 2.11$, $p = .347$].

As the bulk of all subsequent analyses reported below involved testing group differences between students in the lower and upper quartiles, gender differences using only these two quartiles (male n = 46; female n = 205) were computed. Descriptive

statistics are reported in Table 2. As with the total sample, the four sub-scale scores as well as total scores were entered as multiple dependent variables in a MANOVA, with gender as the between-subjects factor. Again contrary to predictions, no significant overall effect was evident according to Pillai's trace [V = 0.02, $F(4, 246) = 1.16$, $p = .330$]. A Chi-square test of contingency also indicated no association between *gender* and *LSCL-33 quartile* [$\chi^2(1; N = 251) = 1.95$, $p = .163$]. As no gender differences in *LSCL-33 total scores* were evident for the total sample or the lower and upper quartiles, all subsequent between-group differences on relevant variables were investigated between the upper and lower *LSCL-33 quartiles* collapsed across gender.

Psychometric properties of the DRM-Q

Reliability. Reliability for the core 33-item scale was calculated using the total sample of participants who completed all 33 items (N = 396), and was excellent at α = .90. Reliability for the three 11-item *dreams (DRM)*, *bad dreams (BDs)*, and *nightmares (NMs)* sub-scales was acceptable at α = .70 (N = 526), .78 (N = 526), and .80 (N = 396), respectively.

Table 1.
Descriptive Statistics for LSCL-33 Scores by Gender, for the Total Sample

	Male (n = 101)		**Female (n = 425)**	
LSCL-33 Scale	*M*	*SD*	*M*	*SD*
Somatic	8.10	4.28	9.97	4.92
Sensory	6.43	5.37	6.45	5.13
Behavioural	5.64	4.00	6.13	4.06
Mnemonic	6.29	4.02	6.83	4.22
Total	26.46	14.09	29.39	14.76

Table 2.
Descriptive Statistics for LSCL-33 Quartile Scores, by Gender

	Lower Quartile (n = 124)		**Upper Quartile (n = 127)**	
LSCL-33 Scale	Male (n = 27)	Female (n = 97)	Male (n = 19)	Female (n = 108)
Somatic	4.56 (*2.21*)	4.82 (*2.40*)	13.74 (*3.59*)	14.88 (*4.35*)
Sensory	2.11 (*1.87*)	1.82 (*1.60*)	13.74 (*5.64*)	12.77 (*4.56*)
Behavioural	1.78 (*1.50*)	2.41 (*1.84*)	10.95 (*3.41*)	10.56 (*3.67*)
Mnemonic	2.63 (*1.69*)	2.95 (*1.65*)	11.68 (*4.07*)	11.74 (*3.89*)
Total	**11.07 (4.23)**	**12.01 (3.66)**	**50.11 (*6.61*)**	**49.95 (*8.30*)**

Note. Standard deviations are reported in parentheses

Component structure. The 33 core items of the DRM-Q were subjected to principal components analysis via Varimax rotation, using only participants who completed all 33 items (n = 396). The Kaiser-Meyer-Olkin test of sampling adequacy was good at .836, and Bartlett's test of sphericity was statistically significant [$\chi^2(528) = 7041.61$, $p < .001$]. Eight well-defined components emerged with eigenvalues greater than 1, which together accounted for 67.51% of the variance. Component loadings for all individual test items were greater than .50, as were all communalities [range = .57 (*DRM intensity*) to .82 (*BD Colourfulness*)]. All item-component loadings were clear except for the items measuring *BD vividness* and *BD intensity*. These two items loaded on a component with the items measuring *NM vividness* and *intensity*, and also on a component with *DRM vividness* and *intensity*. Given that the loadings of the two *BD* items were greater on the component also comprising *NM vividness* and *intensity*, *DRM vividness* and *intensity* were kept as the only two items loading on the *DRM vividness* component. The full component structure is displayed in Tables 3 and 4. The eigenvalues, percentage of variance explained by each component, and the reliability of each component is displayed in Table 5.

The effect of limbic system activation on dreaming variables

A series of independent samples t tests were run to investigate differences between participants in the upper and lower LSCL-33 quartiles on a number of dream, bad dream, and nightmare variables. All t tests were run as one-tailed, as an effect in the direction of the upper quartile was predicted for all analyses. The level of alpha was set at .003 to control for inflated Type I error rate due to multiple comparisons (i.e., $\alpha = .05/16$). Compared to participants in the lower quartile, participants in the upper quartile rated their quality of sleep as worse ($p < .001$), reported that they wake up more times per night ($p < .001$), and reported having more dreams per night ($p < .001$). The results of these analyses are reported in full in Table 6.

Compared to the lower quartile, individuals in the upper quartile experienced a greater frequency of bad dreams recalled according to an eight-point rating scale ($p < .001$), as well as a greater number of bad dreams in the last month ($p < .001$) and in the last year ($p < .001$). They also indicated experiencing more distress as a result of their bad dreams ($p < .001$). Participants in the upper quartile experienced a greater frequency of

Table 3.

Component Structure of the DRM-Q, Part 1 (Study 1)

Variable	DRM-Q Component	1	2	3	4	5	6	7	8
DRM Aggression to self	1. Aggression to self	**.754**	.054	.122	.027	.060	.057	.014	-.184
DRM Aggression to self intensity	1. Aggression to self	**.769**	.096	.185	-.001	.049	.114	.155	-.052
BD Aggression to self	1. Aggression to self	**.804**	.167	.024	-.036	.005	.069	.089	.146
BD Aggression to self intensity	1. Aggression to self	**.804**	.198	.045	.044	-.023	.127	.038	.242
NM Aggression to self	1. Aggression to self	**.754**	.194	.065	-.072	.091	.109	.006	.257
NM Aggression to self intensity	1. Aggression to self	**.772**	.167	.104	.042	.068	.098	-.019	.257
DRM Aggression to others	2. Aggression to others	.053	**.679**	.059	-.014	.057	.060	.045	-.176
DRM Aggression to others intensity	2. Aggression to others	.115	**.702**	.122	.012	-.047	.119	.109	-.083
BD Aggression to others	2. Aggression to others	.113	**.820**	.009	.008	.024	.134	.017	-.023
BD Aggression to others intensity	2. Aggression to others	.158	**.828**	.075	.033	-.066	.061	-.023	.053
NM Aggression to others	2. Aggression to others	.119	**.769**	.072	-.015	.090	-.019	-.143	.070
NM Aggresssion to others intensity	2. Aggression to others	.228	**.761**	.100	.015	-.024	-.055	.002	.133
DRM Affectedness	3. Meaningfulness-affectedness	.001	.030	**.748**	.013	-.025	.060	.216	.045
DRM Meaningfulness	3. Meaningfulness-affectedness	.084	.200	**.562**	.015	.207	.197	.295	-.190
BD Affectedness	3. Meaningfulness-affectedness	.034	.022	**.818**	-.026	-.041	-.029	.096	.213
BD Meaningfulness	3. Meaningfulness-affectedness	.155	.187	**.768**	.119	.146	.094	.001	-.060
NM Affectedness	3. Meaningfulness-affectedness	.161	.011	**.768**	-.025	.083	-.022	-.030	.310
NM Meaningfulness	3. Meaningfulness-affectedness	.193	.142	**.736**	.181	.228	.095	-.139	.003
DRM Colourfulness	4. Colourfulness	-.013	.026	-.044	**.751**	.087	.058	.265	.023
BD Colourfulness	4. Colourfulness	.001	.007	.091	**.875**	.084	.079	.123	.070
NM Colourfulness	4. Colourfulness	-.013	.018	.101	**.836**	.085	.088	-.025	.199
DRM Realism	5. Realism	-.069	.046	.010	.082	**.735**	.006	.244	-.019
BD Realism	5. Realism	.077	-.042	.177	.087	**.820**	.083	.128	.169
NM Realism	5. Realism	.165	.009	.125	.136	**.779**	.120	-.004	.266

Table 4.

Component Structure of the DRM-Q, Part 2 (Study 1)

Variable	DRM-Q Component	1	2	3	4	5	6	7	8
DRM Movement	6. Movement	.157	.038	.110	.103	.004	**.799**	.169	-.067
BD Movement	6. Movement	.132	.110	.009	.091	.052	**.813**	.063	.163
NM Movement	6. Movement	.191	.090	.095	.053	.213	**.649**	-.123	.321
DRM Vividness	7. Dream vividness-intensity	.037	-.117	.069	.341	.129	-.019	**.715**	.120
DRM Intensity	7. Dream vividness-intensity	.178	.047	.168	.054	.219	.141	**.576**	.161
BD Vividness	8. BD-NM vividness-intensity	.109	-.032	.072	.332	.185	.174	.486	**.515**
BD Intensity	8. BD-NM vividness-intensity	.280	.058	.190	.035	.203	.140	.412	**.525**
NM Vividness	8. BD-NM vividness-intensity	.147	-.040	.122	.269	.272	.128	.172	**.664**
NM Intensity	8. BD-NM vividness-intensity	.389	.079	.207	.103	.083	.131	.030	**.623**

Table 5.

Eigenvalues, Percentage of Variance Explained, and Reliability Values of the DRM-Q Components (Study 1)

DRM-Q Component	Eigenvalue	% Variance	α
1. Aggression to self	2.83	5.06	.74
2. Aggression to others	2.82	5.03	.76
3. Meaningfulness-affectedness	2.64	4.71	.66
4. Colourfulness	2.59	4.62	.73
5. Realism	2.52	4.51	.75
6. Movement	2.41	4.31	.69
7. Dream vividness-intensity	2.35	4.20	.72
8. BD-NM vividness-intensity	2.34	4.17	.69

Table 6.

Descriptive Statistics and t *test Results for Sleep and Dreaming Variables*

	Lower Quartile		Upper Quartile					
Variable	*n*	*M* (*SD*)	*n*	*M* (*SD*)	*t* (*df*)	*p*	*d*	Effect size *r*
Hours of sleep per night	124	7.02 (*1.13*)	127	6.61 (*1.41*)	2.54 (*239.62*)	.011*	0.33	.16
Quality of sleep	124	3.95 (*1.02*)	127	3.35 (*0.97*)	**4.82 (*249*)**	**< .001**	**0.61**	**.29**
Awakenings per night	124	0.88 (*0.98*)	127	1.43 (*1.21*)	**4.00 (*240.78*)**	**< .001***	**0.52**	**.25**
Dreams per week	106	2.42 (*1.65*)	112	2.96 (*1.89*)	2.24 (*216*)	.014	0.30	.15
Dreams per night	122	1.16 (*0.41*)	126	1.48 (*0.64*)	**4.57 (*214.60*)**	**< .001***	**0.62**	**.30**
Ease of dream recall	124	2.75 (*1.08*)	127	2.93 (*1.15*)	1.27 (*249*)	.090	0.16	.08
Entirety of dream recall	124	2.81 (*1.00*)	127	2.83 (*1.00*)	0.22 (*249*)	.410	0.03	.01
BD Frequency	124	4.15 *(1.35)*	127	4.93 *(1.59)*	**4.20 *(244.51)***	**< .001***	**0.53**	**.26**
BDs Last month	121	0.98 *(1.16)*	124	3.05 *(3.31)*	**6.58 *(153.36)***	**< .001***	**1.06**	**.47**
BDs Last year	119	7.25 *(10.42)*	122	21.39 *(35.49)*	**4.22 *(142.19)***	**< .001***	**0.71**	**.33**
BD Distress	124	3.00 *(1.66)*	127	4.10 *(1.66)*	**5.27 *(249)***	**< .001**	**0.67**	**.32**
NM Frequency	124	2.48 *(1.39)*	127	3.61 *(1.74)*	**5.69 *(239.43)***	**< .001***	**0.74**	**.35**
NMs Last month	98	0.40 *(0.80)*	109	1.48 *(2.58)*	**4.15 *(130.45)***	**< .001***	**0.73**	**.34**
NMs Last year	94	2.61 *(4.36)*	110	10.28 *(28.19)*	**2.82 *(115.09)***	**.003***	**0.53**	**.25**
NM Distress	93	3.44 *(2.10)*	109	4.21 *(1.96)*	2.69 *(200)*	.004	0.38	.19
NDQ Total scores	84	24.83 *(7.39)*	100	33.70 *(7.94)*	**7.79 *(182)***	**< .001**	**1.15**	**.50**

Note. All *t* tests were run as one-tailed.

Results marked with an asterix are results for equal variances not assumed, due to a statistically significant Levene's test for homogeneity of variance.

Results in boldface are statistically significant at the level of alpha (α = .003) correcting for inflated Type I error due to multiple comparisons.

nightmares according to the eight-point rating scale ($p < .001$), as well as a greater number of nightmares in the last month ($p < .001$) and in the last year ($p = .003$). Participants in the upper quartile also had greater scores on the NDQ ($p < .001$), but did not indicate greater distress on the category scale item measuring nightmare distress at the corrected level of α ($p = .004$), although this result did trend towards statistical significance.

A Chi-squared test of contingency was performed to examine the association between *LSCL-33 quartile* and participants having had at least one nightmare in the previous month. The results indicated a strong and statistically significant association between *LSCL-33 quartile* and the likelihood of having had at least one nightmare in the previous month [$\chi^2(1, N = 206) = 18.79$, $p < .001$, Cramer's $V = .302$]. More individuals in the upper quartile had at least one nightmare in the previous month than expected, with fewer individuals in the lower quartile having had at least one nightmare in the previous month than expected (see Table 7).

Table 7.
Incidence of At Least One Nightmare in the Last Month by LSCL-33 Quartile

	At least one NM in last month	
LSCL-33 Quartile	No	Yes
Lower quartile		
Observed frequency	73	26
(Expected frequency)	(57.70)	(41.30)
% of Total	35.40	12.60
Std. Residual	2.0	-2.4
Upper quartile		
Observed frequency	47	60
(Expected frequency)	(62.30)	(44.70)
% of Total	22.80	29.10
Std. Residual	-1.9	2.3

For the three items measuring recall of recurrent dreams, Chi-squared tests of contingency indicated a significant association between *LSCL-33 quartile* and participants ever having had a recurrent dream [$\chi^2(2, N = 251) = 6.36$, $p = .042$, Cramer's $V = .159$]. The association was stronger for participants having had a recurrent

dream in the last 12 months [$\chi^2(2, N = 192) = 10.67$, $p = .005$, Cramer's $V = .236$], and in the last 6 months [$\chi^2(2, N = 193) = 9.94$, $p = .007$, Cramer's $V = .227$]. The cell frequencies and standardized residuals indicated that for all three analyses, fewer participants in the lower quartile experienced recurrent dreams than expected, and more participants in the upper quartile experienced recurrent dreams than expected. The results are presented in full in Table 8.

Table 8.
Incidence of Recurrent Dreams (RD) by LSCL-33 Quartile

	Ever had RD?			RD last 12 months?			RD last 6 months?		
LSCL-33 Quartile	Yes	No	Uncertain	Yes	No	Uncertain	Yes	No	Uncertain
Lower quartile									
Observed frequency	61	49	14	24	54	13	19	62	10
(Expected frequency)	(70.60)	(42.50)	(10.90)	(33.70)	(43.10)	(14.20)	(27.80)	(51.40)	(11.80)
% of Total	24.30	19.50	5.60	12.50	28.10	6.80	9.80	32.10	5.20
Std. Residual	-1.1	1.0	0.9	-1.7	1.7	-0.3	-1.7	1.5	-0.5
Upper quartile									
Observed frequency	82	37	8	47	37	17	40	47	15
(Expected frequency)	(72.40)	(43.50)	(11.10)	(37.30)	(47.90)	(15.80)	(31.20)	(57.60)	(13.20)
% of Total	32.70	14.70	3.20	24.50	19.30	8.90	20.70	24.40	7.80
Std. Residual	1.1	-1.0	-0.9	1.6	-1.6	0.3	1.6	-1.4	0.5

The effect of limbic system activation on the DRM-Q variables

The variables comprising each component of the DRM-Q were entered together as multiple dependent variables in eight MANOVAs, one MANOVA for each of the eight components. A significant overall effect for the variables comprising *dream aggression to self* was evident according to Pillai's trace [$V = 0.15$, $F(6, 191) = 5.43$, $p < .001$, $\eta_p^2 = .146$] (lower quartile $n = 88$; upper quartile $n = 110$). Follow-up univariate ANOVAs were used to investigate differences on the individual variables, with $\alpha = .008$ used as the level of significance to control for inflated Type I error (i.e., $\alpha = .05/6$). Significant effects for the items measuring *DRM aggression to self* ($p < .001$), *DRM aggression to self intensity* ($p < .001$), *BD aggression to self* ($p < .001$), *BD aggression to self intensity* ($p < .001$), and *NM aggression to self intensity* ($p = .002$) were evident, although there was no effect for *NM aggression to self* at the corrected level of alpha ($p = .010$). The significant effects favoured the upper quartile in all cases.

A significant overall effect for the *dream aggression to others* component was also evident according to Pillai's trace [$V = 0.09$, $F(6, 190) = 3.04$, $p = .007$, $\eta_p^2 = .087$]

(lower quartile n = 87; upper quartile n = 110). Follow-up univariate ANOVAs were again used to investigate differences on the individual variables, with α = .008 again set as the level of statistical significance to control for inflated Type I error (i.e., α = .05/6). Significant effects were obtained only for *NM aggression to others* (p < .001) and *NM aggression to others intensity* (p < .001). Both significant effects favoured the upper quartile.

For the six *dream affectedness-meaningfulness* variables, a moderate significant overall effect was obtained according to Pillai's trace [V = 0.20, F(6, 191) = 7.41, p < .001, η_p^2 = .189] (lower quartile n = 88; upper quartile n = 110). Follow up univariate ANOVAs, again with α = .008 used as the level of significance to control for inflated Type I error, indicated statistically significant effects for all six individual variables (all p < .001). All effects again favoured the upper quartile.

A significant overall effect was obtained for *BD-NM intensity* [V = 0.05, F(4, 193) = 2.78, p = .028, η_p^2 = .054]. Follow-up univariate ANOVAs indicated a significant effect only for *BD intensity* (p = .004), with the effect in the direction of the upper quartile. A significant overall effect for *dream intensity* was also evident [V = 0.10, F(2, 248) = 13.93, p < .001, η_p^2 = .101], although follow-up univariate ANOVAs indicated a significant effect for only *DRM intensity* (p < .001), which again was in the direction of the upper quartile. No significant overall effects were evident for *dream colourfulness* [V = 0.01, F(3, 194) = 0.66, p = .580], *dream realism* [V = 0.01, F(3, 193) = 0.69, p = .557], or *movement in dreams* [V = 0.01, F(3, 192) = 0.87, p = .457]. The results of all the preceding analyses are presented in full in Table 9.

Component structure of the Typical Dreams Questionnaire (TDQ)

The 56 items of the Typical Dreams Questionnaire (TDQ) were subjected to principal components analysis using data from the total sample (N = 526). The Kaiser-Meyer-Olkin test of sampling adequacy was excellent at .913, and Bartlett's test of sphericity was statistically significant [χ^2(1540) = 10 794.33, p < .001]. As with Nielsen et al. (2003), 16 well-defined components emerged with eigenvalues greater than 1, which in the present study together accounted for 62.93% of the variance. Reliability for the total 56-item questionnaire was excellent at α = .94. The communalities were good, and

ranged from .49 (*seeing a flying object crash*) to .75 (*falling*). The full component structure of the TDQ is displayed in Tables 10 and 11. The component structure has been split into two separate tables due to its large size. The eigenvalues, percentage of variance explained by each component, and the reliability of each component is displayed in Table 12.

The component structure somewhat replicated that found in Nielsen et al. (2003). Exactly the same two items loaded on the *epiphany* component (*52 – Encountering God in some form* and *51 – Seeing an angel*). *Loss of control* and *disasters* were almost entirely identical, and the *alien life, death-murder, inhibition,* and *falling* components were largely identical. The *positive experiences* and *nudity-sex* components were somewhat identical to those same components identified in Nielsen et al. (2003). The remaining seven components – *magic-myth, negative experiences, flying-chase, presence, creatures, panic-embarrassment,* and *abortions* were largely unique to the present study.

Between-quartile differences in typical dream themes

The 16 components of the TDQ were entered into a MANCOVA as multiple dependent variables, with *LSCL-33 quartile* as the between-groups factor (lower quartile n = 105; upper quartile n = 111). This was to test for any differences in the frequency of typical dream themes that may be associated with higher compared to lower levels of limbic activation. Numbers of dreams per week and night as well as BD and NM recall frequency were entered as covariates to control for a potential increase in the frequency of certain dream themes experienced simply due to an increased frequency of dreams, bad dreams, and/or nightmares in general. A significant overall effect for *LSCL-33 quartile* was evident according to Pillai's trace [V = 0.23, $F(16, 195) = 3.73$, $p < .001$, $\eta_p^2 = .234$].

The significant overall effects were investigated further using follow-up univariate ANOVAs, with the level of statistical significance set at $\alpha = .003$ to control for inflated Type I error due to multiple comparisons (i.e., $\alpha = .05/16$). Significant effects were obtained for eight of the 16 components. The greatest effects were obtained for: (a) *presence* ($p < .001$, $\eta_p^2 = .140$); (b) *inhibition* ($p < .001$, $\eta_p^2 = .138$); (c) *loss of control* ($p < .001$, $\eta_p^2 = .113$); and (d) *death murder* ($p < .001$, $\eta_p^2 = .103$).

Table 9.
Summary of Univariate ANOVA Results for DRM-Q Components

DRM-Q Component	Lower Quartile *M* (*SD*)	Upper Quartile *M* (*SD*)	*F*	*df*	*p*	$\eta_p 2$
Aggression to self (ATS)						
DRM ATS	2.05 (*0.86*)	2.61 (*0.94*)	**19.02**	**1, 196**	**< .001**	**.088**
DRM ATS Intensity	2.11 (*0.98*)	2.75 (*1.10*)	**17.73**	**1, 196**	**< .001**	**.083**
BD ATS	2.45 (*1.09*)	3.18 (*1.03*)	**23.02**	**1, 196**	**< .001**	**.105**
BD ATS Intensity	2.61 (*1.20*)	3.17 (*1.15*)	**11.15**	**1, 196**	**< .001**	**.054**
NM ATS	2.67 (*1.27*)	3.12 (*1.14*)	6.84	1, 196	.010	.034
NM ATS Intensity	2.84 (*1.31*)	3.39 (*1.17*)	**9.66**	**1, 196**	**.002**	**.047**
Aggression to others (ATO)						
DRM ATO	1.60 (*0.74*)	1.83 (*0.80*)	4.28	1, 195	.040	.021
DRM ATO Intensity	1.68 (*1.06*)	2.01 (*1.03*)	4.89	1, 195	.028	.024
BD ATO	1.84 (*0.87*)	1.98 (*0.95*)	1.18	1, 195	.279	.006
BD ATO Intensity	1.97 (*1.08*)	2.28 (*1.14*)	3.90	1, 195	.050	.020
NM ATO	1.59 (*0.69*)	2.00 (*0.92*)	**12.19**	**1, 195**	**< .001**	**.059**
NM ATO Intensity	1.86 (*1.05*)	2.36 (*1.11*)	**10.46**	**1, 195**	**< .001**	**.051**
Dream affectedness						
DRM Affectedness	2.27 (*0.80*)	2.95 (*1.04*)	**25.57**	**1, 196**	**< .001**	**.115**
DRM Meaningfulness	2.51 (*0.90*)	3.15 (*1.09*)	**19.78**	**1, 196**	**< .001**	**.092**
BD Affectedness	2.45 (*0.92*)	3.22 (*1.08*)	**27.86**	**1, 196**	**< .001**	**.124**
BD Meaningfulness	2.23 (*0.99*)	2.99 (*1.09*)	**26.06**	**1, 196**	**< .001**	**.117**
NM Affectedness	2.63 (*1.07*)	3.17 (*1.11*)	**12.37**	**1, 196**	**< .001**	**.059**
NM Meaningfulness	2.09 (*0.99*)	2.78 (*1.21*)	**18.74**	**1, 196**	**< .001**	**.087**
BD-NM Vividness						
BD Vividness	4.01 (*1.26*)	4.23 (*1.29*)	1.40	1, 196	.238	.007
BD Intensity	3.32 (*0.90*)	3.70 (*0.93*)	**8.41**	**1, 196**	**.004**	**.041**
NM Vividness	3.99 (*1.44*)	4.01 (*1.49*)	0.01	1, 196	.922	< .001
NM Intensity	3.58 (*1.01*)	3.75 (*1.05*)	1.40	1, 196	.238	.007
DRM Vividness						
DRM Vividness	3.75 (*1.00*)	3.83 (*1.20*)	0.37	1, 249	.545	< .001
DRM Intensity	2.74 (*0.92*)	3.32 (*0.86*)	**26.69**	**1, 249**	**< .001**	**.097**

Note. Results in boldface are statistically significant after corrections to control for inflated Type I error due to multiple comparisons.
The univariate ANOVA results for *dream colourfulness, dream realism,* and *movement in dreams* are not reported as the overall MANOVAs were not statistically significant.

The remaining four statistically significant effects, in descending order of effect size, were: (e) *alien life* ($p < .001$, $\eta_p^2 = .094$); (f) *negative experiences* ($p < .001$, $\eta_p^2 = .069$); (g) *positive themes* ($p < .001$, $\eta_p^2 = .063$); and (h) *epiphany* ($p < .001$, $\eta_p^2 = .057$). All eight effects favoured the upper quartile. The results are presented in Table 13.

Table 10.
Component Structure of the TDQ, Part 1

TDQ Theme	TDQ Component	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
47. Seeing extraterrestrials	1. Alien life	**.722**	-.007	.143	.053	.190	.018	.093	.103	.146	.051	.019	.169	.202	.031	.086	.063
46. Seeing a UFO	1. Alien life	**.706**	.021	.281	.011	.207	-.014	.082	.070	.065	.004	.006	.180	.036	-.013	.092	.132
54. Seeing a flying object crash	1. Alien life	**.563**	.127	.034	.139	.128	.132	.115	.068	.047	.124	.196	-.028	-.062	.112	.025	.095
43. Lunatics or insane people	1. Alien life	**.488**	.213	.086	.071	.058	.120	.080	.178	-.004	.031	.214	-.047	.279	.382	-.064	.014
48. Traveling to another planet	1. Alien life	**.447**	.031	.417	.060	-.024	.044	.010	.171	.097	.026	.147	.278	.076	.106	-.058	-.225
27. Being killed	2. Death-murder	.022	**.725**	.180	.090	.134	.093	.154	.012	.119	.161	.021	.060	.013	.097	-.044	.066
2. Physically attacked	2. Death-murder	.047	**.701**	.014	.120	.072	.054	.189	.063	.124	.114	.052	.003	.210	.090	.062	-.058
28. Seeing yourself as dead	2. Death-murder	-.009	**.683**	.148	.037	.167	-.071	.126	.089	-.007	.004	.241	.106	-.015	-.086	.061	.124
42. Killing someone	2. Death-murder	.330	**.560**	.036	-.050	.012	.184	.110	.105	.268	.014	-.049	.051	.156	.176	.074	-.004
49. Being an animal	3. Magic-myth	.250	.147	**.657**	.075	.078	.127	.007	-.073	.078	.010	.033	.036	.152	.178	.073	.119
26. Being an object	3. Magic-myth	.262	.092	**.596**	-.029	.124	-.016	.170	.039	-.032	-.012	-.156	.056	-.006	-.079	.240	.267
17. Creatures	3. Magic-myth	.009	.113	**.557**	.215	.134	.012	.073	.120	.101	-.030	.179	-.020	.403	.115	.087	-.059
20. Magical powers	3. Magic-myth	.161	.191	**.513**	.049	.094	.375	-.028	.254	.093	.062	.215	.083	.088	.011	-.172	-.177
38. Failing an examination	4. Negative experiences	.077	.034	.050	**.663**	.157	.072	.291	.048	.131	.092	.106	.090	-.009	.011	-.066	.045
31. School, teachers, studying	4. Negative experiences	-.003	.019	.117	**.644**	-.001	.183	-.012	.201	.139	.164	.030	.024	.082	.231	.028	.029
36. A person now alive as dead	4. Negative experiences	.138	.384	.049	**.535**	.126	.064	-.047	-.044	.114	.179	.231	.116	.113	-.003	.137	.100
24. Insects or spiders	4. Negative experiences	.108	.069	.120	**.461**	.107	.014	-.040	.068	.178	.290	.151	.014	.303	-.182	.134	.051
6. Arriving too late	4. Negative experiences	.074	.004	-.130	**.434**	.155	.295	.265	.322	.060	.110	.002	.053	-.085	.164	.231	-.037
22. Tornadoes or strong winds	5. Disasters	.173	.108	.040	.063	**.789**	.069	-.008	.026	.027	.055	.091	.095	.083	.050	.067	.021
21. Floods or tidal waves	5. Disasters	.056	.120	.022	.137	**.691**	.276	.086	.000	.162	-.086	.020	.014	.094	.104	-.013	.086
23. Earthquakes	5. Disasters	.226	.126	.238	.070	**.687**	-.080	.087	.087	-.089	.102	.003	.007	.104	.097	.135	-.087
34. Fire	5. Disasters	.314	.267	.124	.320	**.368**	.105	.070	.141	-.048	.273	.070	.103	.086	.122	-.048	.114
11. Flying...through the air	6. Flying-chase	.144	-.005	.314	.135	-.004	**.641**	.025	-.023	.153	.161	.014	.109	.073	.055	.068	-.163
3. Trying again and again	6. Flying-chase	-.077	.036	.038	.083	.129	**.555**	.265	.135	.026	.226	.116	.015	-.003	.176	.241	.135
7. Swimming	6. Flying-chase	.222	.070	.094	.141	.267	**.502**	.072	.218	.244	.103	.059	-.040	.031	-.096	-.018	.044
1. Chased or pursued, not injured	6. Flying-chase	-.038	.115	-.116	.207	.075	**.488**	.110	.055	-.033	.362	.067	-.054	.279	.080	.233	.021
15. Being tied, unable to move	7. Inhibition	.134	.170	.064	.050	.023	.041	**.725**	.073	.048	.099	.092	-.031	-.005	.108	.049	-.064
39. Smothered, unable to breath	7. Inhibition	.116	.154	.037	.124	.046	-.061	**.596**	.005	.128	.180	.315	.113	.208	-.049	.028	-.046
4. Being frozen with fright	7. Inhibition	-.032	.131	-.008	.043	.098	.409	**.533**	.064	-.010	.209	.024	-.015	.101	-.027	.086	.098
44. Half awake and paralyzed	7. Inhibition	.084	.156	.082	.013	.006	.142	**.491**	-.172	.194	-.109	.233	.162	.220	.221	.186	-.049
8. Being locked up	7. Inhibition	.227	.223	.135	.274	.123	.312	**.439**	.175	-.044	.000	-.077	.082	.060	.056	-.025	.095

Table 11.
Component Structure of the TDQ, Part 2

TDQ Theme	TDQ Component	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
5. Eating delicious foods	8. Positive experiences	.064	.117	.003	.212	.003	.096	.001	**.737**	.048	-.063	.048	.012	.064	.028	.145	.061
10. Finding money	8. Positive experiences	.151	.035	.020	.010	.068	.096	.068	**.683**	.159	.037	.000	.265	.115	.052	.053	.005
16. Superior...mental ability	8. Positive experiences	.106	.136	.412	.022	.028	.151	.000	**.537**	.107	.008	.194	.171	.119	.117	-.092	-.153
41. Being at a movie	8. Positive experiences	.144	-.108	.339	.110	.107	-.183	.111	**.413**	.214	.151	.163	.100	-.112	.282	-.044	.248
14. Being nude	9. Nudity-sex	.136	.192	.056	.133	.038	.024	.033	.121	**.752**	.090	.008	.020	.112	.106	.127	.045
13. Being innapropriately dressed	9. Nudity-sex	.055	.057	.087	.239	.028	.081	.127	.153	**.714**	.040	-.034	.003	.079	.170	.161	-.033
25. Being member of opposite sex	9. Nudity-sex	.016	.144	.371	-.099	.234	.240	.072	.037	**.458**	.020	.116	.097	-.032	-.108	-.025	.206
32. Sexual experiences	9. Nudity-sex	.089	.252	-.020	.222	-.105	.288	.007	.098	**.400**	.228	.090	.049	.187	.125	.035	.010
12. Falling	10. Falling	.050	.135	.028	.195	.034	.194	.127	-.006	.101	**.777**	-.009	.053	.006	.068	.065	-.070
37. Being on the verge of falling	10. Falling	.098	.114	-.009	.151	.034	.206	.143	-.003	.065	**.741**	.107	.104	.070	.156	.039	-.022
29. Vividly sensing a presence	11. Presence	-.007	.119	.043	.068	.138	.115	.193	.069	.009	.088	**.685**	.099	.264	-.011	-.026	.095
45. Seeing a face very close	11. Presence	.238	.049	.140	.129	-.064	.066	.161	.082	-.069	.018	**.604**	.152	.024	.205	.002	.135
35. A person now dead as alive	11. Presence	.112	.264	-.033	.341	.047	.037	.015	.034	.092	-.034	**.465**	.308	-.018	.052	.248	-.051
19. Seeing yourself in a mirror	11. Presence	.234	.052	.075	.017	.269	-.101	.154	.380	.172	.252	**.411**	-.093	-.164	.003	.050	.110
52. Encountering God	12. Epiphany	.146	.055	.013	.043	-.013	.036	.046	.158	.042	.030	.060	**.815**	.121	.004	.020	.040
51. Seeing an angel	12. Epiphany	.089	.106	.138	.101	.128	.006	.034	.087	-.015	.097	.157	**.739**	-.004	.111	-.030	.082
56. Encountering...evil force or demon	13. Violent creatures	.108	.242	.085	-.054	.078	.199	.043	.084	.088	.124	.083	.192	**.660**	.127	.062	.152
40. Wild, violent beasts	13. Violent creatures	.178	.086	.224	.139	.163	.026	.197	.056	.110	-.011	.074	-.012	**.628**	.001	.002	.012
9. Snakes	13. Violent creatures	-.028	-.123	.033	.158	.273	-.018	.201	.212	.207	.149	.080	.260	**.355**	-.282	.048	-.041
53. Discovering a new room	14. Loss of control	.193	.049	.146	-.005	.187	-.006	.054	.108	.194	.285	.097	.039	.114	**.580**	.110	.036
33. Losing control of a vehicle	14. Loss of control	.054	.136	-.022	.280	.153	.160	.211	.103	.211	.084	-.047	.091	.032	**.490**	.102	.237
50. Being a child again	14. Loss of control	.085	.152	.259	.216	.110	.093	.086	.079	.147	.104	.249	.256	-.050	**.474**	-.018	-.069
18. Teeth falling out/losing teeth	15. Panic-embaressment	.113	.076	.019	-.033	.195	.179	.044	.023	.224	-.021	.081	-.007	.001	.054	**.718**	-.130
30. Unable to find...toilette	15. Panic-embaressment	.017	.028	.164	.186	-.050	.051	.147	.183	.077	.231	-.041	.003	.112	.031	**.610**	.173
55. Someone having an abortion	16. Abortions	.200	.108	.087	.112	.022	.001	-.064	.035	.065	-.074	.185	.103	.111	.090	-.007	**.721**

Table 12.
Eigenvalues, Percentage of Variance Explained, and Reliability Values of the TDQ Components

TDQ Component	Eigenvalue	% Variance	α
1. Alien life	2.83	5.06	.74
2. Death-murder	2.82	5.03	.76
3. Magic-myth	2.64	4.71	.66
4. Negative experiences	2.59	4.62	.73
5. Disasters	2.52	4.51	.75
6. Flying-chase	2.41	4.31	.69
7. Inhibition	2.35	4.20	.72
8. Positive experiences	2.34	4.17	.69
9. Nudity-sex	2.25	4.01	.70
10. Falling	2.16	3.86	.80
11. Presence	2.05	3.66	.64
12. Epiphany	1.95	3.49	.70
13. Violent creatures	1.95	3.48	.56
14. Loss of control	1.71	3.04	.60
15. Panic-embarassment	1.49	2.66	.43
16. Abortions	1.19	2.12	n/a

Note. Alpha is not reported for the *abortions* component as only 1 variable comprised this component.

A one-tailed independent samples *t* test on the sleep paralysis subscale proposed by Nielsen et al. (2003), consisting of items 4 (*being frozen with fright*), 15 (*being tied, unable to move*), 29 (*vividly sensing, but not necessarily seeing or hearing, a presence in the room*), 39 (*being smothered, unable to breathe*), and 44 (*being half awake and paralyzed in bed*), indicated a statistically significant difference between the two quartiles. Individuals in the upper quartile (M = 8.71, SD = 4.04) scored higher than individuals in the lower quartile (M = 4.26, SD = 3.87) [$t(249) = 8.91$, $p < .001$, $d = 1.13$, $r = .49$]. The test was run as one-tailed as a direction in the effect of the upper quartile was predicted.

Themes predictive of bad dream and nightmare recall frequency

The 16 components of the TDQ were entered into a forward stepwise multiple regression (probability in: .05; probability out: .10) using the total sample (N = 526), with *BD* and *NM recall frequency* – as measured by the 8-point frequency measures from the DRM-Q – as the dependent variables. Number of BDs and NMs experienced in the

last month and/or year could not be used as dependent variables due to non-normally distributed residuals. Three components were found to positively predict *BD recall frequency* [R^2 = .17, $F(3, 522) = 36.04$, $p < .001$]. In order of entry these were: (1) *death-murder* ($r = .36$, $p < .001$); (2) *flying-chase* ($r = .30$, $p < .001$); and (3) *disasters* ($r = .29$, $p < .001$). Four components were found to positively predict *NM recall frequency* [R^2 = .17, $F(4, 521) = 25.97$, $p < .001$]. These were the three components that predicted BD recall frequency, entered in the same order, as well as *inhibition.* Zero-order correlations of these four components, in order of entry, were: (1) *death-murder* ($r = .33$, $p < .001$); (2) *flying-chase* ($r = .30$, $p < .001$); (3) *disasters* ($r = .29$, $p < .001$); and (4) *inhibition* ($r = .31$, $p < .001$). The results of these regression analyses are presented in full in Appendix C.

Table 13.
Summary of Univariate ANOVA Results for TDQ Components

	Lower Quartile	Upper Quartile			
TDQ Component	*M* (*SD*)	*M* (*SD*)	*F*(1, 210)	*p*	$\eta_p 2$
1. Alien life	1.11 (*2.28*)	3.55 (*3.65*)	**21.88**	**< .001**	**.094**
2. Death-murder	2.43 (*2.61*)	5.45 (*3.99*)	**24.04**	**< .001**	**.103**
3. Magic-myth	1.61 (*2.48*)	3.49 (*3.39*)	8.61	.004	.039
4. Negative experiences	7.01 (*4.58*)	10.06 (*3.98*)	**15.63**	**< .001**	**.069**
5. Disasters	1.75 (*2.55*)	3.59 (*3.03*)	8.56	.004	.039
6. Flying-chase	6.60 (*3.60*)	8.63 (*3.82*)	5.42	.021	.025
7. Inhibition	4.04 (*3.86*)	8.35 (*4.03*)	**33.55**	**< .001**	**.138**
8. Positive experiences	3.59 (*3.58*)	5.98 (*3.87*)	**14.17**	**< .001**	**.063**
9. Nudity-sex	4.38 (*3.51*)	6.05 (*3.55*)	6.64	.011	.031
10. Falling	4.15 (*2.37*)	5.38 (*2.27*)	6.90	.009	.032
11. Presence	2.66 (*3.10*)	5.90 (*3.44*)	**34.21**	**< .001**	**.140**
12. Epiphany	0.81 (*1.39*)	1.83 (*2.08*)	**12.66**	**< .001**	**.057**
13. Violent creatures	2.11 (*2.41*)	3.85 (*2.98*)	8.28	.004	.038
14. Loss of control	2.36 (*2.47*)	4.79 (*2.81*)	**26.83**	**< .001**	**.113**
15. Panic-embaressment	1.44 (*1.62*)	2.26 (*1.99*)	6.02	.015	.028
16. Abortions	0.18 (*0.62*)	0.39 (*0.82*)	3.05	.082	.014

Note. Results in boldface are statistically significant after correction to control for inflated Type I error due to multiple comparisons.

Bad dream and nightmare sub-scales were formed by summing scores on the items that comprised the three components predictive of bad dream recall frequency, and the four components predictive of nightmare recall frequency. Scores on the BD and NM sub-scales were entered as dependent variables in a MANCOVA, with number of dreams per week and night, as well as BD and NM recall frequency again entered as covariates.

LSCL-33 quartile (lower quartile n = 105; upper quartile n = 111) was the independent variable. A significant overall effect was evident [V = 0.15, $F(2, 209)$ = 18.32, $p < .001$, η_p^2 = .150]. Follow-up univariate ANOVAs indicated differences between the LSCL-33 quartiles on both the BD [$F(1, 210)$ = 22.43, $p < .001$] and NM sub-scales [$F(1, 210)$ = 32.76, $p < .001$], with the effect greater on the NM (η_p^2 = .135) compared to the BD (η_p^2 = .097) sub-scale. The upper quartile scored higher on both the BD (M = 17.68, SD = 7.99) and NM (M = 26.03, SD = 10.98) sub-scales compared to the lower quartile (M = 10.78, SD = 6.52 and M = 14.82, SD = 9.34 respectively).

Differences in most recent dream content

Only most recent dream reports from females in the lower (n = 79) and upper quartiles (n = 70) were coded, due to inadequate sample sizes of dream reports from males (lower quartile n = 23; upper quartile n = 12). It was not possible to combine dream reports from females and males as the Hall and Van de Castle norms against which dream reports are statistically compared differ according to gender (i.e., dream reports from males must be compared with the male norms, and dream reports from females must be compared with the female norms as the differences between the two sets of norms are too great to allow combining them into one set). The independent rater coded 34 dream reports from the lower quartile, and 39 dream reports from the upper quartile. Inter-rater reliability was calculated by correlating the frequency percentages obtained for each category from each rater, and was excellent at .91. A two-tailed independent samples t test indicated no difference in the average length of dream reports from the lower (M = 144.65, SD = 66.93) and upper quartiles (M = 138.26, SD = 68.21) [$t(147) = 0.57$, p = .571].

When dream reports from the lower and upper quartiles were compared to each other, three statistically significant differences emerged, all regarding *social interactions*: (1) the dreamer was the aggressor with less frequency in the upper quartile (p = .040); (2) the dreamer was the victim with greater frequency in the upper quartile (p = .045); and (3) the *physical aggression* percent was greater in the upper quartile (p = .020). No other notable differences were evident. The results are presented in full in Table 14, and the h-profile is presented in Figure 1.

Table 14.
Percentages and h-*values of Upper Compared to Lower Quartile Dreams (Study 1)*

Dream Content Category	Lower Quartile (*n* = 79)	Upper Quartile (*n* = 70)	*h*	*p*
Characters				
Male/Female	57%	52%	-.10	.417
Familiarity	73%	71%	-.04	.694
Friends	45%	47%	+.05	.639
Family	26%	22%	-.08	.418
Dead & Imaginary	2%	1%	-.08	.413
Animal	9%	8%	-.02	.827
Social Interaction %				
Aggression/Friendliness	60%	56%	-.08	.558
Befriender	64%	57%	-.14	.543
Aggressor	26%	11%	**-.40**	**.040***
Victimization	74%	89%	**+.39**	**.045***
Physical Aggression	51%	70%	**+.39**	**.020***
Social Interaction Ratios				
A/C Index	.36	.31	-.12	
F/C Index	.24	.22	-.04	
S/C Index	.01	.05	+.09	
Self-Concept %				
Self-negativity	77%	73%	-.10	.467
Bodily misfortunes	26%	24%	-.04	.844
Negative emotions	82%	79%	-.07	.625
Dreamer-involved success	30%	39%	+.20	.504
Dreams with at Least One				
Aggression	53%	49%	-.09	.578
Friendliness	42%	40%	-.03	.844
Sexuality	4%	11%	+.28	.073
Misfortune	30%	37%	+.13	.404
Good Fortune	9%	5%	-.16	.313
Success	8%	12%	+.16	.325
Failure	18%	18%	+.01	.925
Striving	24%	29%	+.12	.454

Note. The lower quartile was used as the baseline.
Statistical significance values are not reported for the *social interaction ratios* as these do not use the exact *h* statistic.
Statistically significant *h* values are marked in boldface
*$p < .05$. **$p < .01$. ***$p < .001$.

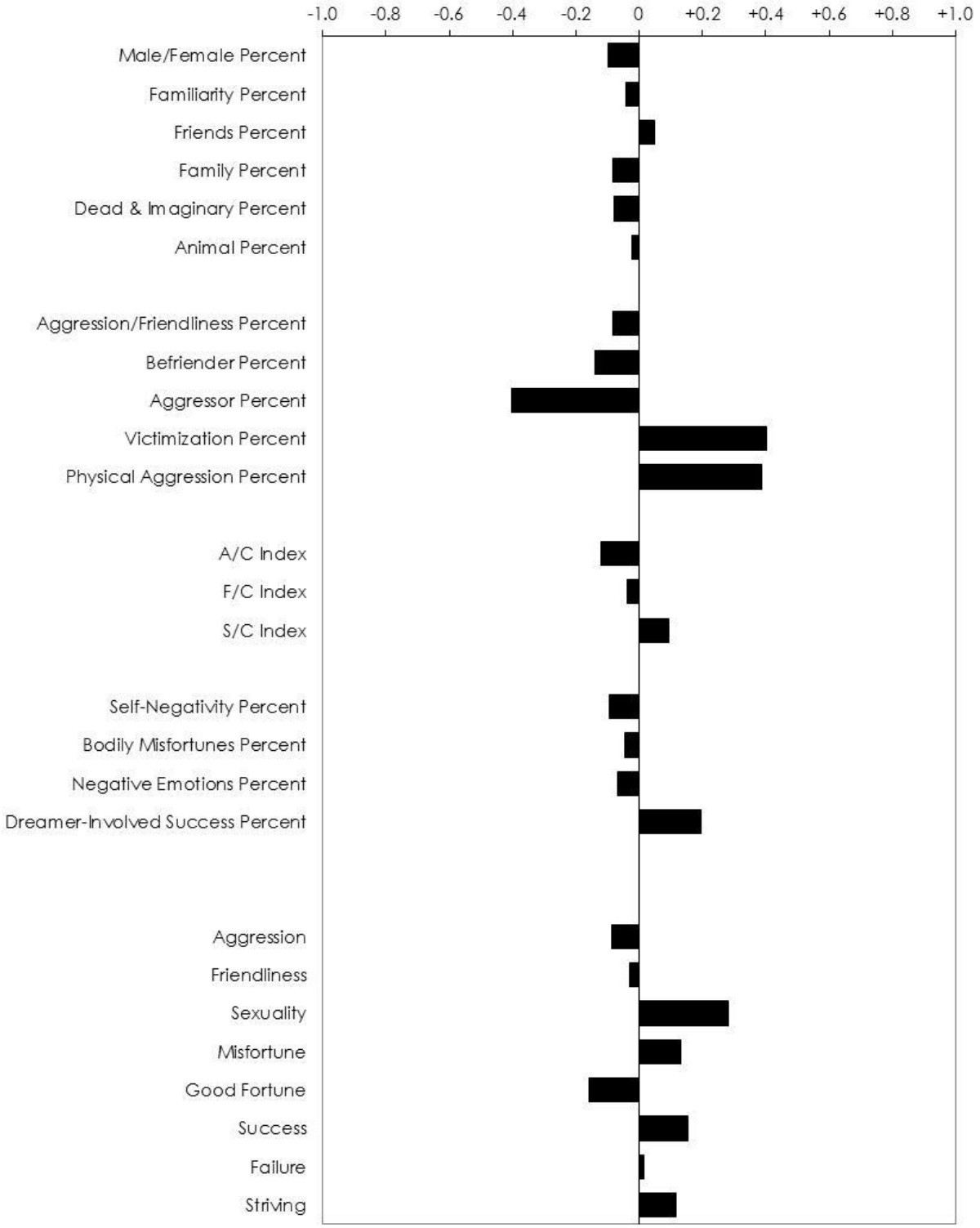

Figure 1. *h*-profile of most recent dream reports of the upper LSCL-33 quartile compared to the lower, with the lower quartile as the baseline.

DISCUSSION

The present study investigated the effect that high versus low levels of limbic system activation have on a number of sleep and dreaming variables, including typical dream themes and dream content. Overall, the results support the contention that *the limbic system plays a crucial role in nightmare generation*, as well as the contention that *increased limbic system activation leads to increased recall frequency of dreams, bad dreams, and nightmares*. Moreover, *increased limbic system activation has effects on dream content*. The hypothesis that woman would have greater levels of limbic activation was not supported. These results are discussed in more detail below.

Gender differences in levels of limbic activation

The hypothesis that females would have a higher overall level of limbic activation compared to males was not supported by the results of the present study, which found no between-gender difference in total scores on the LSCL-33. The one difference in LSCL-33 scores that was found was for the somatic sub-scale, for the total sample, with females scoring higher than males. This result is in line with a study which found that females scored higher than males on a sub-scale measuring somatic complaints, from a larger subjective health complaints questionnaire (Hetland, Torsheim, & Aaro, 2002). Somatic complaints have also been found to be associated with increased levels of anxiety (Egger, Costello, Erkanli, & Angold, 1999), and females have been found to experience greater levels of anxiety compared to males (Chaplin, Gillham, & Seligman, 2009).

Given that the present as well as previous studies have shown limbic activation to be related to dream recall frequency (DRF), and given that there is a well-established gender effect for DRF and nightmare frequency (Schredl & Reinhard, 2008, 2011), it was expected that females would experience overall greater limbic activation than males in the present study. This was especially the case considering that various studies (see the introduction to this chapter) have found morphological differences in limbic structures between males and females.

One possibility is that the gender differences observed in dream, bad dream, and nightmare recall frequency might be attributable to other factors besides innate and/or

acquired gender differences in limbic system activation. Such factors may be linked to gender differences in variables such as verbal intelligence, verbal memory, frequency of talking about emotional matters, recall of emotional experiences, sleep quality, prevalence of insomnia, interest in dreams, coping style, and a higher demand on women's emotional regulation capacities, amongst others (Levin & Nielsen, 2007; Schredl, 2014; Schredl & Reinhard, 2008).

Alternatively, it may be that the LSCL-33 scale itself is not sensitive enough to detect subtle differences in limbic activation. A meta-analysis by Wager et al. (2003) found a number of gender differences in activation levels of limbic structures in response to emotionally arousing stimuli, but most of these differences were in highly specific brain structures. Women did not show more frequent activation overall in response to emotional stimuli compared to men. It may thus be that the level of limbic activation measured by the LSCL-33 is simply too coarse to adequately detect gender differences.

Findings on the sleep and dreaming variables

The hypothesis that higher levels of limbic system activation should be associated with a greater frequency of recall of dreams, bad dreams, nightmares, and also nightmare distress, was confirmed. Regarding raw dream recall frequency, Zadra and Robert (2012) found the mean number of dreams recalled per week to be 4.5, higher than the averages of 2.42 and 2.96 for the lower and upper quartiles on the LSCL-33, respectively, in the present study.

Regarding bad dream recall frequency, the mean number of bad dreams per month on a retrospective measure used by Zadra and Donderi (2000) was 1.60, whereas it was 2.45 per month on prospective dream logs. This compares to mean bad dream frequencies of 0.98 in the last month for the lower quartile, and 3.05 per month for the upper quartile on the LSCL-33 in the present study.

In terms of nightmare frequency, the raw average frequency for number of nightmares in the past month for both the lower (0.40) and upper (1.48) quartiles was less than that reported by Nielsen et al. (2003), which was 1.97 ± 3.40 nightmares/month. Chivers and Blagrove (1999), also using a non-clinical sample, reported that

participants in their study reported an average of 1.60 nightmares over the previous two week period prior to their study, in other words, 3.20 nightmares per month. Levin and Fireman (2002a) reported that participants experienced an average of 3 nightmares over a 21 day dream diary study, although there was considerable variability. Participants in Zadra and Donderi (2000) reported an average of 0.48 nightmares in the previous month according to the retrospective measure, and an average of 0.92 nightmares over a 1-month prospective dream log.

When compared to the results of other studies, the frequency of dreams, bad dreams, and nightmares reported in the present study appears to be lower, although the bad dream and nightmare frequencies are generally more in line with frequencies reported by Zadra and Donderi (2000). This may be because both the present study and that of Zadra and Donderi (2000) explicitly delineated dreams, bad dreams, and nightmares. Even though the awakening criterion might be used to define nightmares to participants, it might be that participants overestimate the amount of nightmares they experience by including bad dreams in their estimates unless dreams, bad dreams, and nightmares are explicitly separated.

The findings that participants with high versus low levels of limbic system activation recalled more bad dreams and nightmares – according to three different recall frequency measures – is in keeping with findings that the limbic system is crucially affected in a number of clinical disorders which are associated with an increased frequency of recall of disturbing dreams (see the review in Chapter 3). The primary advantage of the present study is that this has been demonstrated in a non-clinical sample. It thus appears that certain clinical conditions may amplify the same causal mechanisms responsible for generating normal dreams and idiopathic nightmares in individuals without pathology.

The finding in the present study that participants with a higher level of limbic system activation experienced not only a greater frequency of recall of nightmares, but also bad dreams and dreams in general, supports the contention that nightmares are a more severe expression of the same basic phenomenon (Zadra & Donderi, 2000). Levin and Fireman (2002b) likewise found that individuals who experience frequent nightmares

report a greater recall of dreams in general. More disturbed sleep (in terms of participants in the upper quartile rating their quality of sleep as worse, as well as experiencing more awakenings per night) is in keeping with findings from studies on sleep and dreaming in various clinical conditions which are marked by limbic hyperactivity. Such studies have often discovered more sleep disturbances in patients with these conditions [e.g., migraine (Bruni et al., 1999); PD (Borek et al., 2007); SSDs (Koffel & Watson, 2009); anxiety (Celluci & Lawrence, 1978); and PTSD (Krakow et al., 2001)].

The finding that individuals with higher rates of limbic activation reported more distress from both their nightmares and bad dreams is in keeping with the findings that frequent nightmare sufferers are more affected by both their nightmares and other dreams (Levin, 1994). Although individuals in the upper quartile did not report more nightmare distress on the category scale item measuring this variable, the result trended towards statistical significance, and they did report more distress according to the Nightmare Distress Questionnaire. In fact, the greatest effect size was obtained for the NDQ rating. This finding is similar to that of Levin and Fireman (2002b), who reported that nightmare prevalence was unrelated to a concurrent rating of how disturbing a nightmare itself was. Nightmare prevalence was, however, significantly associated with scores on the same scale as used in the present study (the NDQ), which measures waking distress stimulated by nightmares rather than distress caused as a result of a nightmare itself (i.e., global versus local distress, respectively). Levin et al. (2011) also found affect distress to be associated with heightened distress for both bad dreams and nightmares.

Turning to the results of the dream characteristics, participants scoring in the upper quartile of LSCL-33 scores reported more aggression of other characters towards themselves across all three dream types (normal dreams, bad dreams, and nightmares) compared to the lower quartile. This aggression was also rated as more intense, with the exception of the intensity of the aggression in nightmares. These findings are similar to those of Levin (1994), who found that frequent nightmare sufferers reported more aggression in their dreams compared to low-nightmare controls. Robert and Zadra

(2014) and Fireman et al. (2014) also more recently found physical aggression to be common in nightmares, especially compared to bad dreams.

The higher and more intense rates of dreamer-directed aggression observed in the upper quartile might be expected given that the limbic system has been demonstrated to be highly active in situations that have threatening connotations (Peterson et al., 2002). Higher levels of limbic activation might thus be likely to produce more threatening and/or aggressive situations during dreaming, possibly via a more frequent activation of fear memory elements and/or fear-producing stimuli (Levin & Nielsen, 2007, 2009; Nielsen & Levin, 2007). In keeping with this hypothesis, Peterson et al. found a greater recall of objective threats (defined as events such as death or threat of death, injury or threat of injury, etc.) in individuals with high versus low levels of limbic activation. Alternatively, given that both the hypothalamus and amygdala of the limbic system are involved in the 'fight or flight' response (Devinsky & D'Esposito, 2004), higher levels of limbic activation may lead to the actions of characters in the dream being interpreted as threatening towards the dreamer. Peterson et al. (2002), however, found no difference in subjective threat in the content of dreams reported by individuals with high versus low levels of limbic activity.

Aggression, and the intensity of aggression by the dreamer towards other characters, was also found to be higher in the upper quartile, but only for nightmares. Given the higher rate of dreamer-directed aggression observed in the upper quartile, the higher rates and intensity of aggression to others is most likely to comprise fear-induced defensive aggression, rather than predatory/goal-directed aggression (Blair, 2001; van Elst et al., 2000). This is because nightmares are more likely to be characterized by a high rate of fear and anxiety (Levin & Nielsen, 2007, 2009), as well as a high rate of aggressions, failures, and unfortunate endings compared to bad dreams (Robert & Zadra, 2014), and the effect was only observed for nightmares in the present study.

Despite the findings of greater and more intense dreamer-directed and other-directed aggression on a number of measures, no difference between the quartiles was found for the intensity of dreamer-directed aggression in nightmares. Differences were also found between the quartiles for both dream and bad dream intensity in general, with the

upper quartile rating both as higher, but no difference in nightmare intensity between the quartiles was found. Taken together, these results appear to indicate that although individuals in the upper quartile are prone to experiencing more nightmares – as well as more intense dreams and bad dreams – it appears that once a nightmare occurs it is experienced with equal intensity by the dreamer regardless of level of limbic activation.

Although nightmares were experienced with equal intensity by individuals in both quartiles, individuals in the upper quartile still experienced more distress from their nightmares. Individuals in the upper quartile also rated that they were more affected by their nightmares – as well as their dreams and bad dreams – and also that they found all three dream types more meaningful. The finding that individuals in the upper quartile rated being more affected by their dreams is in keeping with the proposition by Levin and Nielsen (Levin et al., 2011; Levin & Nielsen, 2007; Nielsen & Levin, 2007, 2009) that individuals more prone to frequent nightmares are higher in the trait factor of affect distress, which leads to more distress both during and after a nightmare. Levin (1994) also found that frequent nightmare sufferers indicated that they were more affected by their dreams and nightmares compared to low nightmare sufferers.

The results of the present study indicate that this tendency to experience heightened distress and affectedness may extend to all types of dreams, and not simply nightmares alone. Given that heightened affect distress is characterized by heightened reactivity to emotional stimuli, and given that the limbic system is crucially involved in generating emotional appraisals and reactions to stimuli (Devinsky & D'Esposito, 2004), it is reasonable to assume that individuals with greater activity of the limbic system would experience higher affect distress. Affect distress has also been conceptualized as occurring in a number of conditions characterized by limbic dysfunction (e.g., anxiety, PTSD; Levin & Nielsen, 2007).

The finding that participants in the upper quartile rated all three dream types as more meaningful compared to the lower quartile is in keeping with the theory that individuals with thin boundaries (Hartmann, 1989, 1991) value their dreams more and find them more meaningful (Schredl et al., 1999). Although boundary thinness was not directly assessed in the present study, boundary thinness has been shown to be

positively related to schizophrenia-spectrum disorders, and these disorders have been shown to be positively correlated with increased limbic activation (see Chapter 3). Boundary thinness has also been associated with affect distress (Levin & Nielsen, 2007), and nightmares distress (Kracmarova & Plhakova, 2015) which as noted above may be associated with limbic hyperactivity. It is thus reasonable to assume that individuals in the upper quartile possess thinner boundaries than individuals in the lower quartile, although future studies directly investigating the relationship between limbic activity and boundary thinness would be valuable to confirm this hypothesis.

The final finding from the DRM-Q pertaining to differences between individuals with high versus low levels of limbic activity was of an association between frequency of recall of recurrent dreams and level of limbic system activation. Individuals with high levels of limbic activation reported experiencing more recurrent dreams than expected, and individuals with low levels of limbic activation reported experiencing less recurrent dreams than expected. These findings are not surprising based on the fact that individuals with hyperactivity of the limbic system caused by temporal lobe epilepsy often experience an increase not only in recurrent dreams (Epstein, 1964; Epstein & Hill, 1966; Reami et al., 1991), but also recurrent stereotypical nightmares (Solms, 1997, 2000a). Recurrent dreams have also been documented in individuals suffering from migraine (Nielsen, 2010), and anxiety (Duke & Davidson, 2002; Zadra, 1994), both disorders affecting the limbic system (see Chapter 3).

Findings on the association of limbic activation with typical dream themes

After forming components based on a principal components analysis of the 56 dream themes measured by the TDQ, differences between the LSCL-33 quartiles were found on scores for eight of the 16 components. All differences were in the direction of the upper quartile, and four of the components for which differences were found can reasonably be considered negative (*death-murder*; *negative experiences*; *inhibition*; and *presence*). This would be expected based on the other findings in the present study that individuals with high levels of limbic activation are prone to experience more bad dreams and nightmares, as well as more aggressive dream content. Individuals in the upper quartile also scored higher than the lower quartile on both the *bad dream* and *nightmare* sub-scales formed from the items on the TDQ predictive of bad dream and nightmare recall

frequency, which adds confirmation to the results from the DRM-Q that higher levels of limbic activity are associated with more disturbed dreaming.

Although the results from the DRM-Q indicated that the dreams of individuals with higher levels of limbic activity are characterized by a greater amount and intensity of aggression, the present set of findings from the TDQ reveal that a much larger range of dream content differences between the upper and lower quartiles exists. The greatest effect sizes were for *presence* (vividly sensing a presence; seeing a face very close; a person now dead as alive; seeing oneself in a mirror) and *inhibition*. The finding related to the *presence* component is not surprising given that the limbic system contains neurons that respond selectively in response to images of faces, hands, and eyes (Joseph, 2001), and that the right limbic system has been found to be activated when an individual sees their own face (Kircher et al., 2001). Persinger (2001) was able to generate the feeling of a sensed presence in the room by stimulation of the right temporal lobe, although these findings have been subsequently challenged on a number of grounds (see Castillo, 2011, for review). Seeing a person now dead as alive may be likened to seeing a ghost or spirit, a phenomenon which has been documented in individuals with hyperactivity of the temporal lobe due to epilepsy (Joseph, 2001).

For *inhibition* (being tied, unable to move; being smothered, unable to breathe; being frozen with fright; being half awake and paralyzed; being locked up), this result is also not surprising given that activation of limbic structures has been hypothesized to explain the experiences associated with sleep paralysis (Buzzi & Cirignotta, 2000). Certain aspects of the sleep paralysis phenomenon are reflected in the *inhibition* component (specifically, being smothered and unable to breathe; being frozen with fright; and being half awake and paralyzed). Sleep paralysis has also been found to be associated with migraine and anxiety disorders (Ohayon, Zulley, Guilleminault, & Smirne, 1999), both of which are associated with disorder of the limbic system (see Chapter 3). Moreover, a statistically significant effect in favour of the upper quartile, with a large effect size (d = 1.13), was found in the present study for the *sleep paralysis* sub-scale of the TDQ proposed by Nielsen et al. (2003). Studies have also found that frequent nightmare sufferers rate their dreams as containing more inhibition (Carr, Blanchette-Carriere, Solomonova, Paquette, & Nielsen, 2016).

A higher effect size relative to most of the other comparisons was also obtained for the *loss of control* component, which comprised three diverse items: discovering a new room; losing control of a vehicle; and being a child again. That individuals in the upper quartile would have greater scores on a component consisting of an item measuring losing control of a vehicle can be explained by virtue of the fact that this is a negative and frightening experience. Previous results from the present study have already indicated that individuals in the upper quartile are more prone to negative and frightening dream experiences.

Why individuals in the upper quartile would have higher scores on a component comprising discovering a new room and being a child again is less clear. With regard to the former, this may be likened to what has been termed 'approach' or 'seeking' behaviour in dreams (Malcolm-Smith et al., 2012). Peragamvros et al. (2015) also found that individuals experiencing frequent idiopathic nightmares score higher on scales of waking personality traits associated with reward-related behaviour, particularly a novelty-seeking scale. Such seeking behaviour is triggered by the dopamine system of the brain, a system which has intricate connections with limbic structures (Solms, 1997, 2000a). Furthermore, a meta-analysis by Wager et al. (2003) found that the mPFC was activated in 'approach' responses to emotional stimuli. The mPFC is heavily interconnected with the limbic system (Levin & Nielsen, 2007; Wager et al., 2003) such that individuals with a highly active limbic system might also have a more highly activated mPFC.

With regard to the item of *being a child again,* as the amygdala is partly responsible for forming long-term emotional memories (Devinsky & D'Esposito, 2004), higher scores on this item might be indicative of ingrained emotional memories from childhood resurfacing, or scenes from one's childhood being viewed during dreaming in individuals with high levels of limbic activity. In support of this hypothesis is a study by Vignal, Maillard, McGonigal, and Chauvel (2007) that found that increased limbic activity due to cortical stimulation of the mesial temporal lobe or from spontaneous temporal lobe seizures could trigger recall of autobiographical memories that were 'relived' by the individual.

The finding of a statistically significant effect in the present study in favour of the upper quartile for the *death-murder* component could be expected based on the findings from the DRM-Q of an increased amount of aggression and intensity of this aggression in the dreams of individuals in the upper quartile, relative to the lower. That higher scores for the upper quartile relative to the lower were also obtained for the *negative experiences* component could also be expected considering that this component contains themes which can be considered anxiety-inducing (specifically, failing an examination; school, teachers, studying; a person now alive as dead; insects or spiders; arriving too late). As noted previously, there is a close link between limbic system activity and anxiety disorders as well as anxiety in general.

The finding that individuals in the upper quartile scored higher overall on the *epiphany* component is not surprising considering that this component comprises the items of *encountering God in some form* and *seeing an angel*. This is because limbic system activity has been strongly linked to religious experience, particularly in the case of limbic hyperactivity due to temporal lobe epilepsy. A number of studies have documented patients with this disorder acquiring deep religiosity after the disorder manifests (Bear & Fedio, 1977; Dewhurst & Beard, 1970; Trimble & Freeman, 2006; Waxman and Geschwind, 1975). Moreover, some individuals have reported communing with spirits following amygdala/temporal lobe stimulation (Joseph, 2001). Intense religious experiences have also been documented in patients with schizophrenia (Devinsky & Lai, 2008), another disorder known to be associated with limbic hyperactivity (see Chapter 3).

Two interesting findings from the present study are that individuals in the upper quartile scored higher overall on the *alien life* and also the *positive experiences* components, compared to the lower quartile. With regards to the *alien life* component, individuals with thin boundaries have been found to have a rich fantasy life, thin boundaries have been linked to an increased recall frequency of nightmares, and nightmare recall frequency has been found to be associated with increased limbic activation in the present study. It may thus be that individuals with higher levels of limbic activation have thinner boundaries which therefore results in them experiencing a richer fantasy life. This in turn could result in such individuals experiencing dream

themes such as those comprising the alien life component (seeing extraterrestrials; seeing a UFO; seeing a flying object crash; lunatics or insane people; traveling to another planet). Although no direct link between boundary thinness and limbic activity has yet been established, there is a link between schizotypy and boundary thinness (Hartmann et al., 1998), and between schizotypy and limbic activity (see Chapter 3).

Further regarding schizotypy, individuals scoring high on a scale of Schizotypal Personality Disorder were found by Claridge et al. (1997) to experience more positive and enjoyable dreaming. In the present study, individuals in the upper quartile scored higher on the *positive experiences* component of the TDQ compared to the lower quartile. There thus appears to be an association between limbic hyperactivity and not only more negative dream experiences, but also more positive dream experiences. Some individuals with limbic hyperactivity as a result of temporal lobe epilepsy have in fact been documented to experience elation during the ictal period (Devinsky & D'Esposito, 2004).

Carr et al. (2016) have noted that frequent nightmare sufferers are also more likely to experience intense dreams marked by positive affect, and in fact found in their study that frequent nightmares sufferers reported higher positivity in their daydreams. Nielsen (2017b) also found that participants recalling earlier positive dreams reported more current nightmares, and Robert and Zadra (2014) found that 22% of nightmare and 38% of bad dream narratives contained either a partially positive or entirely positive outcome. It thus appears that positive dream content in individuals with high levels of limbic activation and/or frequent bad dreams and nightmares may be more positive than previously thought.

Findings from the most recent dream content analysis

Firstly, it should be noted that due to the inadequate sample of dream reports from males in the present study, the content analysis results apply solely to females. The sample sizes from each group (lower quartile n = 79; upper quartile n = 70) were also below the size recommended for adequate between-groups comparisons (n = 100-125; Domhoff, 1996).

Despite these limitations, a greater percentage of aggression was found in the most recent dreams of individuals in the upper quartile, compared to the lower. This finding provides objective support for the earlier findings from the subjective self-report DRM-Q scale that the dreams of individuals in the upper quartile are marked by more aggressive content and themes. In the case of the dream content analysis, the type of aggression for which a statistically significant difference was found was *physical aggression*. This finding is similar to that of Petersen et al. (2002), who found differences in the percentage of objective threats in the dreams of individuals with high versus low levels of limbic activity. The concurrence between the two sets of results is not surprising considering that objective threat as defined by Petersen et al. included a number of physically aggressive events such as death or threat of death, injury or threat of injury, and so forth. Greater amounts of physical aggression have also been found to occur in nightmares in a few more recent studies (Fireman et al., 2014; McNamara et al., 2015; Robert & Zadra, 2014).

The finding in the present dream content analysis that the *victimization* percentage was higher for individuals in the upper quartile is similar to the finding that patients with REM sleep behavior disorder – a disorder characterized by dopaminergic dysfunction that may also affect the limbic system (see Chapter 3, present thesis) – are also seldom the instigator in aggressive confrontations, despite the fact that these predominate in their dreams (Schenck, 2015). This finding also supports the contention mentioned earlier that aggression to others is most likely to comprise fear-induced defensive aggression. The content analysis did also indicate, however, that the dreamer was the aggressor with 11% frequency in the upper quartile. Thus, at least some instances of aggression to others are likely to comprise goal-directed aggression for this group.

Surprisingly, no difference was found between the quartiles for the percentage of negative emotions experienced. A difference could be expected based on the fact that the limbic system is responsible for processing and generating emotional responses to stimuli, particularly the amygdala (Devinsky & D'Esposito, 2004). This lack of a between-groups difference in negative emotions could possibly suggest that although increased limbic activity exerts its influence during dreaming in terms of generating frightening and aggressive dream content (as has been shown by the results from the

DRM-Q and TDQ above), that this content is experienced as relatively emotionless during the dream. The emotional influence that negative dreams have might therefore be felt primarily after the dream, during waking hours. A study by Levin and Fireman (2002b) supports this contention, as that study found that nightmare prevalence was related to a global measure of nightmare distress rather than to a concurrent rating of nightmare distress. In other words, nightmare prevalence was more related to waking distress caused by nightmares, rather than to distress experienced during the actual nightmares themselves.

An alternative – and more likely – interpretation may relate to the fact that it was not noted in the present study whether the most recent dream reports collected were from normal dreams, bad dreams, or nightmares. It may thus be that the majority of dream reports were from ordinary dreams, in which intense negative emotions may not have been experienced. Differences in emotional dream content between the two quartiles may therefore exist, but perhaps only for bad dreams and/or nightmares. A future study which examines differences in dream content for dreams differentiated according to type would help to either support or disconfirm this hypothesis.

Another plausible interpretation is that the emotion categories of the Hall and Van de Castle coding system do not make a fine enough distinction to allow the detection of any differences in the emotional content of dream reports from each quartile. The *apprehension* category of the coding system, for example, contains emotions as diverse as *terrified*, *concerned*, and *apologetic*. Moreover, as an emotion is simply coded as present or absent, no allowance is made for intensity of emotion. Thus, a report of feeling *terrified* would be coded the same way as a report of feeling *scared*, even though the former emotion can reasonably be considered to be more intense than the latter. It should also be noted that the scoring of emotions by external raters versus by the dreamer themselves can often yield very different results, for presence versus absence of emotion, their exact nature, as well as intensity (see Sikka et al., 2014). The lack of a difference in emotional content from dream reports collected from each quartile may also be, however, due to a lack of power as a result of the sample sizes being smaller than those recommended.

CONCLUSIONS

Overall, most of the hypotheses of the present study were supported. Limbic system activation was found not only to be associated with certain aspects of sleep, but also to an increased frequency of recall of dreams, bad dreams, and nightmares. High versus low levels of limbic activity were also related to an increase in the level of global distress experienced from nightmares, as well as an increased likelihood of experiencing recurrent dreams. In general, more negative, intense, and aggressive dreaming was observed for individuals in the upper quartile compared to individuals in the lower quartile. This was the case not only for aspects of dreaming as measured by the DRM-Q, but also typical dream themes as measured by the TDQ, and also the content of most recent dreams.

A number of interesting findings regarding positive dream themes being experienced more frequently for individuals in the upper compared to the lower quartile were also noted in the present study. Although these findings were not entirely expected, they are in keeping with previous research on dreaming in certain conditions (especially schizophrenia-spectrum disorders) which has found increased imaginativeness and positivity in dreaming. Thus, increased limbic activity may also be associated with positive as well as negative influences on dreaming, although the latter is more likely to predominate. As noted in the *Discussion* section above, a number of the findings from the present study are similar to, and could be expected based on, research from various clinical conditions marked by limbic hyperactivity.

The lack of an overall statistically significant difference between genders on LSCL-33 scores, and the subsequent collapsing of scores across both males and females, appears to indicate that higher levels of limbic activity are associated with dreaming to a similar extent for both genders. As noted previously, however, the LSCL-33 may not measure limbic activity at a level of detail fine enough to detect any between-gender differences. Furthermore, due to small sample sizes of most recent dreams from males in the present study and the subsequent exclusion of this group from the content analysis, it is not known if limbic activity affects most recent dream content in the same way for both genders. There are also certain limitations inherent in the most recent dream method, where prospective dream diary studies are considered the gold standard in dream

content research (Robert & Zadra, 2008). To more closely examine the influence of limbic activity on dream content, a follow-up prospective dream diary study was therefore undertaken. This study is presented in the next chapter.

CHAPTER 5

DREAM, BAD DREAM, AND NIGHTMARE RECALL FREQUENCY AND CONTENT IN INDIVIDUALS WITH HIGH VERSUS LOW LEVELS OF LIMBIC ACTIVITY – A PROSPECTIVE DREAM DIARY STUDY

INTRODUCTION

Despite the fact that the limbic system has been crucially implicated in dream and nightmare generation, it appears that to date only one study (Petersen et al., 2002) has directly investigated the effect that limbic system activation has on dream content. As discovered in Study 1, it appears that the level of limbic activity does indeed affect dream content to a certain extent, based on most recent dream reports from individuals with high versus low levels of limbic activation. Studies of dream content from individuals with the clinical disorders affecting the limbic system reviewed in Chapter 3 also point to the fact that the level of activation of this brain system may affect dream content.

Due to the close association of nightmares with PTSD, dream content in this disorder has been relatively well-studied. Unfortunately, nearly all the studies have focused almost exclusively on threat in the dreams of PTSD patients, and have eschewed other content indicators such as social interactions, characters, and so forth. Esposito et al. (1999) found themes of threat to be prominent in the dreams of individuals with combat-related PTSD. Mellman et al. (2001) found that participants who reported more distressing dreams with trauma-related content had more severe concurrent PTSD symptoms (and thus presumably higher levels of limbic activity) than participants reporting other dream types. A study by Mellman, David, and Barza (1999) on patients with PTSD being treated with nefazodone found that compared to baseline, dream content evaluated 6 weeks after the beginning of treatment contained elements normally associated with REM sleep mentation such as reality distortion and contemporaneity. PTSD severity had also improved 6 weeks after commencing

treatment. Thus, it appears that improvement in PTSD symptomatology (and thus presumably a reduction in limbic activity) is associated with a normalization of dream content. In a dream diary study, Valli et al. (2006) found that traumatized children reported not only more dreams, but also a greater percentage of dreams including threat, and a greater number of threatening events per dream, compared to a control group. Dale, DeCicco, and Miller (2013) found more aggression, death, threat, intensity of emotions, intensity of aggression, success, and fewer friendly interactions in the dreams of Canadian combat veterans compared to civilians.

Some studies have evaluated dream content in individuals without PTSD, but who have witnessed or experienced a traumatic event. Using a within-subjects design, Hartmann and Basile (2003) found more intense dream images, as well as trends towards more attack imagery and more emotions of fear and terror in the dreams of individuals after the terrorist attacks of September 11, 2001, compared to before the attacks. Propper, Stickgold, Keeley, and Christman (2007) found similar results. Specifically, individuals in that study reported increased levels of stress and trauma after September 11, and dreams collected after the attack contained more instances of threat than dreams collected prior to the attack.

Despite the high occurrence of altered dreaming in patients with temporal lobe epilepsy (TLE), dream content has not been regularly studied in such patients. Most studies on dreaming in TLE have examined aspects such as dream recall frequency (Bonnani et al., 2002), structural organization and length of dream reports (Cipolli et al., 2004), and the relationship of dreaming to seizure activity (Silvestri & Bromfield, 2004). In what appears to be the only dream content study to date with patients with TLE, Bentes et al. (2011) found that the dream reports of patients with pharmacoresistant TLE differed from those of controls on a number of Hall and Van de Castle content categories. Specifically, a higher percentage of familiar characters, family members, and familiar settings were found in the dream reports of patients with TLE. A lower percentage of dreamer-involved success, success in general, sexuality, and striving was found in the dream reports of the TLE patients relative to controls.

The higher rates of familiarity in the dream reports of patients with TLE is likely due to the fact that temporal lobe structures are known to be involved in the production of feelings of familiarity (Bentes et al., 2011), such as the feeling of déjà vu sometimes experienced by TLE patients (Devinsky & D'Esposito, 2004). The lower rates of success may be related to the more negative dreaming that individuals with TLE are prone to (see Chapter 3), and the lower rate of sexuality appears to mirror the hyposexuality that is often observed during the interictal period in patients with TLE (Devinsky & D'Esposito, 2004). The lower frequency of *striving* can be explained by the fact that this indicator is a combination of the *success* and *failure* indicators; thus, lower rates of success in dreams can lead to lower rates of striving given no significant differences in failure, as was the case in Bentes et al. (2011).

Although there have not been a large amount of studies on dream content in patients with anxiety disorders, the findings in the few studies that have been done have been somewhat consistent with the findings from studies of dream content in other disorders affecting the limbic system, such as those reviewed above. An early study by Gentil and Lader (1978) found that the dreams of female anxious neurotic patients contained more aggression relative to the dreams of normal female controls. A more recent study, using the Hall and Van de Castle coding system (Pesant & Zadra, 2006), found a greater amount of aggression in the dreams of individuals high in trait anxiety as measured by the State-Trait Anxiety Inventory. Pesant and Zadra also found more negative emotions, and failures and misfortunes in the dream content of such individuals. More recently, Schredl et al. (2012) found that the dreams of patients with anxiety disorders contained more positive and negative emotions compared to the dreams of a control group.

In a study of a woman with generalized anxiety disorder and panic attacks, Kirschner (1999) found a number of changes on the H/VDC content categories before and after treatment with the antidepressant *sertraline*. Pre- to post-medication, statistically significant increases were found in the percentages of friends, friendly interactions per character, and good fortunes present in the woman's dreams. Statistically significant pre- to post-medication decreases were observed in the percentages of aggressions per character, aggression relative to friendliness, familiar settings, and elements from the past. A decrease in the frequency of physical aggression approached statistical

significance. These results add further support to the previously mentioned findings that the dreams of patients with anxiety are characterized by a high degree of aggression. Moreover, successful treatment of the anxiety appears to reduce the amount of aggressive and negative dream content, and increase the amount of positive dream content. The decreases in the percentages of familiar settings and elements from the past may be indicative of a decrease in the tendency to dwell on the past by the woman in the study, a tendency that has sometimes been noted to occur in individuals with anxiety disorders (Kirschner, 1999).

Turning to dream content in schizophrenia-spectrum disorders, a number of studies have documented that the dreams of schizophrenics are more negative, violent, and unfriendly, compared to healthy controls (Carrington, 1972; Kramer & Roth, 1973; Schnetzel & Carbonnel, 1976). The occurance of victimization also appears to be greater in the dreams of schizophrenics (Carrington, 1972; Stompe et al., 2003). A greater occurrence of total anxiety, and in particular, anxiety regarding death and mutilation, has also been reported in the dreams of schizophrenics compared to controls (Stompe et al., 2003). D'Agostino et al. (2013) found that the dreams of schizophrenic (and other) patients with acute psychosis contained themes of grandiosity and religion. The relationship between religiosity and limbic activation has been noted as well for individuals with temporal lobe epilepsy.

Regarding characters, Kramer & Roth (1973) found that the most frequently occurring characters in the dream reports of schizophrenics were strangers, a result replicated by two more recent studies. Lusignan et al. (2009) found a greater proportion of unknown characters in the dream reports of schizophrenic patients compared to healthy controls, after controlling for report length. Khazaie et al. (2012) reported that the dreams of schizophrenic patients contained fewer friends compared to the dreams of family members of the schizophrenics, and also compared to the dreams of a control group. These same results were also found by Khazaie et al. for a sample of patients with a mental illness other than schizophrenia, however, meaning that the result may be reflective of the dreams of patients with a mental illness in general, rather than schizophrenia in particular.

Regarding Parkinson's disease, in a study of PD patients with REM-sleep behaviour disorder (RBD), Borek et al. (2007) found that PD patients with RBD experienced a greater percentage of dreams characterized by being chased by a person, defense against attack by a person, aggression by the dreamer, and vivid dreams, compared to patients without RBD. Valli et al. (2015) found that there were no major differences in content between the dreams of PD patients with and without RBD, although dreams were more negatively than positively toned in PD patients with RBD. This is not surprising considering that RBD in patients with PD has been linked to activation of limbic structures (Poryazova et al., 2013), and activation of limbic structures has been linked to negative dream content (see Chapters 3 and 4 of the present thesis).

A study which did in fact use a control group (Bugalho & Paiva, 2011) investigated dream content in patients with PD using the H/VDC system of coding dream content. Bugalho and Paiva found that, compared to the controls, the dreams of patients with PD contained a greater percentage of animals, aggressions relative to friendly interactions, and physical aggressions. The patients with PD were found to be the victim in aggressive interactions with a greater frequency compared to controls.

Interestingly, patients with PD were also the befriender in social interactions with a greater frequency compared to controls. This finding may reflect activation of the 'seeking system' of the brain, considering the crucial involvement of dopamine in this system (Solms, 2000a), and also considering that 17 of the 23 patients in the study were on some kind of dopamine agonist treatment. In fact, dopamine equivalent dosage predicted the frequency of the dreamer being the befriender at a level just below the 5% level of statistical significance (p = .054) in a regression model. Bugalho and Paiva note that the increased frequency of animals in the dreams of PD patients relative to controls may be indicative of the activation of less phylogenetically evolved brain regions such as the limbic system. The only dream content category for which PD patients had a lower frequency of occurrence compared to controls was for bodily misfortunes.

A recent study (De Gennaro et al., 2016) also confirms that the dopaminergic amygdala-mPFC system is crucially involved in dream recall. De Gennaro et al. (2016) found that visual vividness in the dream reports of patients with PD taking dopamine agonists

positively correlated with volumes of both the amygdalae and thickness of the left mPFC, as measured by MRI images. Emotional load in the dreams also correlated positively with hippocampal volume.

In terms of migraine, Levitan (1984) and Nielsen (2010) have both noted that nightmares of terror are the most predominant theme in the dreams of migraineurs, although other themes such as frustration, loss, incest, and outsized creatures may also occur. Heather-Greener et al. (1996) found that dreams preceding a migraine attack contained a significantly higher frequency of aggressive interactions, misfortunes, anger, and apprehension than dreams not preceding a migraine attack, in the same individuals. Suzuki et al. (2013) found migraine to be associated with dream enacting behaviour, which is suggestive of RBD. As noted previously, RBD has been associated with activation of limbic structures (Poryazova et al., 2013).

De Angeli et al. (2014) reported that the dreams of migraneurs were characterized by more fear and anguish than controls, and that these emotions were independent of anxiety and depression scores. Ghaffarinejad and Mehdizadeh (2011), in a study using the Hall and Van de Castle scoring system, found the dreams of patients with nocturnal migraine to be characterized by more friendliness, sexuality, and misfortune. Dream contents of sadness, misfortune, confusion, sexuality, and failure accelerated migraine attacks.

In sum, the results of dream content studies involving various disorders affecting the limbic system have been fairly consistent, despite the differing pathologies in each disorder. The most consistent finding across all six disorders reviewed here is a greater occurrence of threat and aggression in the dreams of individuals with disorders resulting in or from hyperactivity of the limbic system. A number of additional findings indicate that, in general, the dream content of individuals with limbic hyperactivity is negative in character. For example, this content may be characterized by a greater occurance of misfortune, a lower occurance of success, and/or more negative emotions. As noted in Chapters 1 and 4 of the present study, it is at present unknown as to whether or not the dream phenomena that occur in patients with disorders of the limbic

system also occur in individuals with higher rates of limbic activity, but without disorder of the limbic system.

The results of the dream content analysis presented in the preceding chapter do suggest that there is a certain degree of overlap between the dream content experienced by individuals with disorders of the limbic system, and individuals with higher rates of limbic activation but without disorder of the limbic system. As noted above, higher rates of aggression appear to be highly characteristic of the dream content of individuals with limbic system disorders, and higher rates of aggression were in fact found in individuals with higher versus lower rates of limbic activity in Study 1 of this dissertation.

The aims of the present study were therefore twofold: firstly, to confirm the results of the dream content analysis conducted in Study 1; and secondly, to confirm the results of Study 1 regarding increased dream, bad dream, and nightmare recall frequency in individuals with higher versus lower levels of limbic activation. Both aims in the present study were achieved *by the use of a dream diary study* as such studies are not only considered superior to retrospective report instruments (as used in Study 1), but also the gold standard in dream research (Zadra & Donderi, 2000). Based on the results of Study 1, and also the literature reviewed in this chapter, two specific hypotheses were proposed:

(a) *The dream reports of individuals with higher levels of limbic system activation will contain in particular more aggressive content, and in general more negative content compared to individuals with lower levels of limbic activation*. It was not possible to hypothesize which specific indicators of negative dream content (e.g., misfortunes; failure; negative emotions, etc.) significant differences would be evident on, as findings on these indicators differ across the studies reviewed above. Based on the results of Bente et al. (2011), differences between the two groups with regard to *sexuality* may also be expected. It was not possible to hypothesize a specific direction, as increased rates of limbic activation may lead to either hypo- or hyper-sexuality (Devinsky & D'Esposito, 2004).

(b) *Individuals with higher rates of limbic system activation will experience a greater frequency of recall of dreams, bad dreams, and nightmares* compared to individuals with lower levels of limbic activation.

METHODS

Participants

Participants were a sub-set from the final sample for Study 1 (after students who were outliers and who met other exclusion criteria were excluded – see Chapter 4), recruited by emailing a request to participate and study information to all students falling in the upper and lower quartiles of LSCL-33 total scores. Ten students from the lower quartile and 13 students from the upper quartile agreed to participate. As only one student (from the upper quartile) was male, this student was excluded from the final set of participants as not enough dream reports from this participant would have been obtained in order to permit adequate statistical power for the content analysis.

The final sample was therefore 22 female undergraduate Psychology students, who participated in exchange for course credit. The average age was 19.63 years (*SD* = 1.18). All students who indicated that they had any psychological or psychiatric condition and/or any students who indicated that they had any serious medical condition, and/or were on any chronic medication were already excluded in Study 1, and thus also from the present study.

Design and procedure

The study was approved by the Research Ethics Committee of the Department of Psychology, University of Cape Town. Participants kept a dream diary for a period of 21 days. This time period was chosen as it was believed that this was long enough to permit an adequate sample of reports from each participant to be obtained, in line with the time period used in previous dream diary studies (e.g., Levin & Fireman, 2002b). A prospective study design was used as retrospective reports typically underestimate average dream recall frequency, and nightmare and bad dream recall frequency are under-estimated to an even greater extent by retrospective reports (Robert & Zadra, 2008; Zadra & Donderi, 2000). It should be noted, however, that not all studies find this.

Lancee, Spoormaker, and van den Bout (2010) found higher nightmare recall frequency estimates on a questionnaire compared to a dream diary, and no differences were found between a questionnaire and diary on an item measuring number of nights with nightmares per week. Van Schagen et al. (2015) also found no differences between retrospective questionnaire estimates and daily nightmare logs.

Participants met with the researcher individually, where they were given the dream diary package. In order to establish a baseline, participants were instructed to begin the dream diary the day that they had their next dream (i.e., Day 1 of the diary had to start with a dream report for all participants). The participants then recorded their dreams for the next 20 consecutive days. They were reminded by weekly telephone calls made by the researcher to record their dreams and answer the questions in the dream diary package. Upon completion of the 21 day period, participants returned the dream diary package to the researcher at their convenience.

Dream reports were coded using the Hall and Van de Castle system of coding dream content (see the *Methods* section of Study 1 for a description of the system). The same nine coding categories and the same coding procedure were employed as in Study 1. Specifically, a random selection of half the dream reports (approximately half from the lower quartile, and approximately half from the upper quartile) were coded by a second independent coder. Any discrepancies in codes were resolved through discussion between the two coders prior to data analysis. As all identifying information except for participant numbers was removed from the dream reports prior to coding, both coders were blind as to which LSCL-33 quartile participants' reports came from. The independent coder was also blind as to the design and purpose of the study. As with Study 1, all dream reports less than 50 or greater than 300 words were excluded. A total of 43 dream reports were excluded according to this criterion.

Measures

The dream diary package contained a consent form, a form for demographic and contact information, and written instructions on how to complete the dream diary. Each package also contained 21 dream record forms, one for each day that participants had to record their dreams. Each record form contained dichotomous (yes/no) questions

asking whether the participant had a dream last night, and whether this was a bad dream or a nightmare. Instructions on what content to include in the dream reports, taken from the most recent dream collection form (see Appendix A), were repeated for each of the 21 days. See Appendix B for the dream diary package that was given to participants.

RESULTS

Differences in dream recall frequency variables

A series of one-tailed independent samples *t* tests were run to investigate differences between participants in the upper (*n* = 12) and lower (*n* = 10) LSCL-33 quartiles on dream, bad dream, and nightmare recall frequency. The frequencies presented include all dreams that were reported over the dream diary period. The *t* tests were run as one-tailed as a trend in the direction of the upper quartile was predicted. A statistically significant effect in the direction of the upper quartile was found for *total number of all dreams* (*p* = .005), and the *total number of normal dreams* (*p* = .007). A significant difference in the direction of the upper quartile was also found for *number of nights with at least one dream of any type* (*p* = .003).

Table 15.
Descriptive Statistics for Dream Recall Frequency Variables

	Lower Quartile (*n* = 10)		**Upper Quartile (*n* = 12)**	
Variable	*M*	*(SD)*	*M*	*(SD)*
TOTAL DREAMS (All types)	8.70	(5.60)	16.50	(7.06)
Normal dreams	6.90	(5.74)	13.33	(5.26)
Bad dreams	1.70	(1.49)	2.50	(2.47)
Nightmares	0.10	(0.31)	0.50	(1.17)
Nights with dreams[a]	7.70	(4.14)	13.33	(4.16)

[a]Number of nights with at least one dream of any type.

Individuals in the upper quartile experienced approximately 1.5 times as many bad dreams, on average, and 5 times as many nightmares than individuals in the lower quartile. Despite this, no statistically significant between-groups differences emerged for average recall frequency of either of these two dream types. Descriptive statistics for the dream diary period are reported in Table 15. The *t* test results are reported in full in Table 16.

Table 16.
Results of t *tests on Dream Recall Frequency Variables Between the Upper and Lower Quartiles*

Variable	*t* (*df* = 20)	*p*	*d*	Effect size *r*
TOTAL DREAMS (All types)	**2.83**	**.005**	**1.27**	**.53**
Normal dreams	**2.74**	**.007**	**1.23**	**.52**
Bad dreams	0.89	.181*	0.40	.20
Nightmares	1.05	.154	0.46	.23
Nights with dreams[a]	**3.17**	**.003**	**1.42**	**.58**

Note. All *t* tests were run as one-tailed.
Results marked with an asterix are results for equal variances not assumed, due to a statistically significant Levene's test for homogeneity of variance
Due to the large standard deviations for some of the variables, the results were checked using the nonparametric Mann-Whitney U test. The exact same pattern of statistical significance was obtained as for that when using the parametric *t* tests reported in the table above. The results are reported in Appendix C.
[a]Number of nights with at least one dream of any type.

When compared to the results of Study 1, slightly more bad dreams over the 21-day dream diary period were recorded on average by the lower quartile compared to the number of bad dreams reported over the last month on the DRM-Q (*M* = 1.70 vs *M* = 0.98 respectively). For nightmares, slightly more nightmares over the 21-day dream diary period were recorded on average by the lower quartile compared to the number of nightmares reported over the last month on the DRM-Q (*M* = 0.40 vs *M* = 0.10 respectively). With regards to the upper quartile, fewer bad dreams were reported over the course of the 21-day dream diary study compared to the number of bad dreams reported over the last month on the DRM-Q (*M* = 2.50 vs *M* = 3.05 respectively). For nightmares, the upper quartile also reported fewer nightmares on average over the course of the dream diary study compared to the number of nightmares reported over the last month on the DRM-Q (*M* = 0.50 vs *M* = 1.48 respectively).

Differences in dream content

The independent rater coded 42 dream reports from the lower quartile, and 79 dream reports from the upper quartile. Inter-rater reliability was calculated by correlating the frequency percentages obtained for each category from each rater, and was excellent at .88. A two-tailed independent samples *t* test indicated no difference in the average length of dream reports from the lower (*M* = 144.65, *SD* = 66.93) and upper quartiles (*M* = 138.26, *SD* = 68.21) [*t*(147) = 0.57, *p* = .571].

When compared to the lower quartile, the upper quartile dreams contained: (a) a lower *familiarity* percentage with regards to characters (p = .043); (b) A lower percentage of *family* characters (p = .002); (c) A higher percentage of *dead and imaginary* characters (p = .002); (d) A higher *aggression/friendliness* percentage (p = .046); and a higher percentage of dreams with at least one: (e) *aggression* (p = .006); (f) *sexuality* (p = .030); and (g) *misfortune* (p = .016). Taken together, these results indicate that the dream content of individuals in the upper quartile can be characterized as generally more negative. The results are presented in full in Table 17, and the *h*-profile in Figure 2.

DISCUSSION

The result from Study 1 of an increased recall frequency of dreams in general by individuals with higher versus lower levels of limbic activation was confirmed by the present study. *Individuals falling into the upper quartile remembered approximately double the number of normal dreams as well as dreams of all types over the 21 day dream diary period*. However, *the findings from Study 1 of the tendency of individuals from the upper quartile to remember an increased frequency of bad dreams and nightmares relative to the lower quartile were not confirmed.* This result is surprising considering that the upper quartile indicated remembering a greater frequency of both bad dreams and nightmares on three different frequency measures used in Study 1 (a Likert-type measure; number of bad dreams/nightmares in the last month; and number of bad dreams/nightmares in the last year). Moreover, retrospective measures have previously been found to underestimate dream, bad dream, and nightmare recall frequency relative to prospective dream logs (Robert & Zadra, 2008; Zadra & Donderi, 2000), although not all studies have found a difference between the two measures (Lancee et al., 2010; van Schagen et al., 2015).

Table 17.
Percentages and h-*values of Upper Compared to Lower Quartile Dreams (Study 2)*

Dream Content Category	Lower Quartile (*n* = 85)	Upper Quartile (*n* = 158)	*h*	*p*
Characters				
Male/Female	47%	52%	+.11	.255
Familiarity	66%	58%	**-.17**	**.043***
Friends	39%	40%	+.02	.845
Family	24%	14%	**-.26**	**.002****
Dead & Imaginary	0%	2%	**+.25**	**.002****
Animal	4%	6%	+.11	.193
Social Interaction %				
Aggression/Friendliness	37%	53%	**+.32**	**.046***
Befriender	61%	47%	-.30	.182
Aggressor	35%	29%	-.14	.617
Victimization	65%	71%	+.13	.636
Physical Aggression	38%	41%	+.06	.779
Social Interaction Ratios				
A/C Index	.12	.17	+.12	
F/C Index	.19	.13	-.14	
S/C Index	.00	.03	+.06	
Self-Concept %				
Self-negativity	60%	69%	+.19	.211
Bodily misfortunes	37%	23%	-.30	.164
Negative emotions	73%	81%	+.19	.240
Dreamer-involved success	50%	51%	+.03	.942
Dreams with at Least One				
Aggression	21%	38%	**+.37**	**.006****
Friendliness	33%	30%	-.06	.682
Sexuality	1%	6%	**+.29**	**.030***
Misfortune	28%	44%	**+.32**	**.016***
Good Fortune	9%	11%	+.06	.629
Success	7%	12%	+.17	.205
Failure	7%	12%	+.17	.205
Striving	13%	20%	+.18	.177

Note. The lower quartile was used as the baseline.
Statistical significance values are not reported for the *social interaction ratios* as these do not use the exact *h* statistic.
Statistically significant *h* values are marked in bold.
*$p < .05$. **$p < .01$. ***$p < .001$.

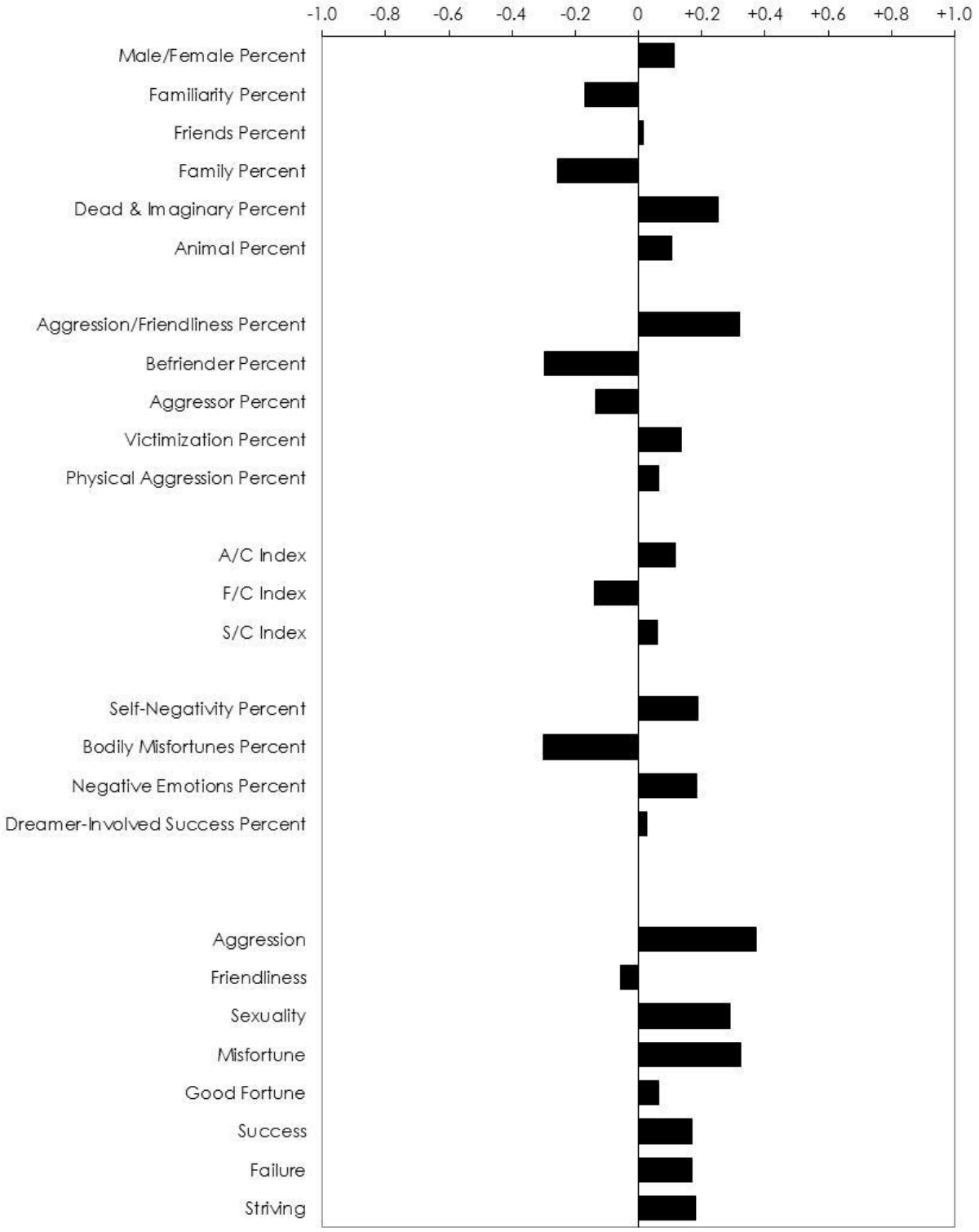

Figure 2. *h*-profile of dream diary dream reports of the upper LSCL-33 quartile compared to the lower, with the lower quartile used as the baseline.

When compared to the results obtained in Study 1, the lower quartile in the present study reported more bad dreams and nightmares compared to what they reported on the DRM-Q, but the upper quartile reported less bad dreams and nightmares, thus reducing the dream recall frequency differences between the two quartiles.

The difference between the results of Study 1 and this study (Study 2) may be due to the finding that a participant's baseline level of dream recall measured retrospectively may influence their level of recall on prospective, log-based measures (Zadra & Robert, 2012). High recallers – for example, the LSCL-33 upper quartile participants in Study 1 – tend to obtain equivalent or even lower log-based dream recall frequency compared to retrospective measures, whereas the opposite is the case for low dream recallers (Zadra & Robert, 2012) – for example, the LSCL-33 lower quartile participants in Study 1. Furthermore, under-reporting of dreams may occur in participants with naturally elevated levels of dream recall (Robert & Zadra, 2008), for example, the participants in the LSCL-33 upper quartile in the present set of studies. These two preceding possibilities explained in this paragraph may thus have resulted in a convergence of bad dream and nightmare recall frequency between the upper and lower quartiles in the present study, and hence a lack of statistically significant results.

The lack of between-group differences for bad dream and nightmare frequency may also be due to a lack of statistical power resulting from the small sample sizes of the two groups (n = 10 and 12), and/or the dream diary time period not being of sufficient length. A study with larger sample sizes, run over a longer period of time (e.g., 6-8 weeks), may have detected statistically significant between-group differences in bad dream and nightmare frequency. Although no statistically significant frequency differences were obtained in the present study, participants from the upper quartile did in fact experience both more bad dreams and nightmares over the dream diary period than participants from the lower quartile. It may also be that perhaps only people from both quartiles interested in nightmares and dreaming – who may have a tendency to experience more nightmares and bad dreams – were interested in participating in this second study. It should also be noted that as with Study 1, the rate of bad dream and nightmare recall was lower than that reported in most other studies (see Chapter 4, and also Chivers & Blagrove, 1999; Nielsen et al., 2003; and Zadra & Donderi, 2000).

The results of the content analysis confirm the finding in Study 1 that individuals with higher levels of limbic activation are more likely to have dreams characterized by a higher frequency of aggression. Although the exact pattern of results was not replicated, a greater frequency of dreams with at least one instance of aggression was found for individuals from the upper compared to the lower quartile in the present study, and the *aggression/friendliness* ratio was also higher for individuals from the upper quartile in the present study. The findings of a greater amount of aggression in the dream reports of individuals with higher levels of limbic activation is also congruent with the findings of greater amounts of threat and aggression in the dreams of individuals with conditions resulting in hyperactivity of the limbic system, such as PTSD (Valli et al., 2006), anxiety disorders (Gentil & Lader, 1978; Kirschner, 1999; Pesant & Zadra, 2006), schizophrenia (Bugalho & Paiva, 2011; Carrington, 1972; Kramer & Roth, 1973; Schnetzel & Carbonnel, 1976; Stompe, 2003), and migraine (Heather-Greener et al., 1996). People with higher limbic activation may have more aggressive content simply because they are more prone to experience a greater frequency of nightmares, as was found in Study 1.

The findings regarding characters being less familiar, and a lower percentage of family members in the dream reports of individuals with higher compared to lower levels of limbic activation, are also congruent with findings from studies on dream content in individuals with hyperactivation of the limbic system. In particular, these findings are most similar to those of Kramer and Roth (1973), Kazhaie et al. (2012), and Lusignan et al. (2003), who found that the dream reports of schizophrenics contained more strangers, fewer friends, and more unknown characters, respectively. In the present study, the finding of a greater amount of *dead and imaginary* characters in the dream reports of individuals from the upper quartile concurs with the finding from Study 1 that the dreams of individuals with high versus low levels of limbic activation are characterized by a greater amount of death and murder (as noted on the Typical Dreams Questionnaire).

The findings from the present study are however incompatible with the results of the study by Bentes et al. (2011) on the content of dream reports from patients with TLE. Bentes et al. reported higher rates of familiar characters, whereas the results of the

present study indicate that the dreams of individuals with higher levels of limbic activation are characterized by lower rates of familiarity regarding characters. The disparity between the two sets of results may be as a result of the fact that the patients in the study by Bentes et al. (2011) were patients with pharmacoresistant TLE who were candidates for epilepsy surgery. The rate of déjà vu has been found to be the highest (11% to 86%) in individuals with intractable temporal lobe seizures (Brown, 2003); thus, epileptics likely to experience a heightened sense of familiarity may have been over-represented in the sample of Bentes et al.

In addition to a heightened sense of familiarity (déjà vu), patients with TLE may also experience the opposite: a reduced sense of familiarity (jamais vu) (Gloor, Olivier, Quesney, Andermann, & Horowitz, 1982; Sengoku, Toichi, & Murai, 1997). The latter phenomenon is in fact consistent with the results from the present study, suggesting a reduced familiarity of characters in individuals with higher levels of limbic activation. Higher rates of limbic activation (particularly on the right side of the brain) have been associated with the perception of novel as opposed to familiar stimuli (Tulving Markowitsch, Kapur, & Habib, 1994; Tulving, Markowitsch, Craik, Habib, & Houle, 1996), and may thus perhaps be associated with the introduction of more novel characters (i.e., strangers and unknown people) as opposed to more familiar characters (such as family members) in dreams.

In the present study, a greater percentage of misfortune was found in the dream reports of individuals with higher levels of limbic activation. Greater amounts of misfortune have been found in the dream reports of migraineurs (Ghaffarinejad & Mehdizadeh, 2011; Heather-Greener et al., 1996) and individuals high in state anxiety (Pesant & Zadra, 2006), both relative to control groups. As both migraine and anxiety has been related to limbic hyperactivation (see Chapter 3), it thus appears that a greater amount of limbic activity can be associated with more misfortune in dreams in both clinical and non-clinical samples.

The finding of a greater frequency of sexuality in the dreams of individuals in the upper versus the lower quartile could be expected based on the involvement of the limbic system in sexual function (Argiolas & Melis, 2003). Pelin and Yazla (2012), for example,

reported abnormal sexual behavior during sleep in a patient with temporal lobe epilepsy. Ghaffarinejad and Mehdizadeh (2011) also reported greater sexuality in the dreams of patients with migraine, another disorder associated with the limbic system. The neurotransmitter dopamine is also involved in sexual function (Argiolas & Melis, 2003), and this neurotransmitter has been implicated as crucial in dream generation by Solms (2000a). Although a statistically significant between-quartiles difference was not found in the frequency of sexuality in the most recent dream reports collected in Study 1, the difference in frequency did approach statistical significance in that study.

The finding in the present study of increased sexuality in dream reports being associated with higher levels of limbic activity appears incompatible with the finding of Bentes et al. (2011) of a reduced level of sexuality in the dream reports of patients with hyperactivity of the limbic system due to TLE, relative to controls. However, both hypo- and hyper-sexuality have been known to occur in patients with TLE (Devinsky & D'Esposito, 2004; Miller, Cummings, McIntyre, Ebers, & Grode, 1986), thus, the lack of concurrence between the two sets of results is perhaps not surprising.

Lastly, as with Study 1, no significant between-group differences were found in the present study regarding the frequency of emotions in the dream reports. It was noted in Study 1 that the lack of between-groups differences may have been due to the majority of the dream reports in that study comprising normal dreams – in which intense emotions may not be experienced – rather than bad dreams and nightmares. Adding support to this hypothesis is that the majority of dreams reported by participants from both groups in the present study were normal dreams, rather than bad dreams and nightmares.

It was also suggested in Study 1 that the H/VDC coding system may not be sensitive enough to detect differences in emotion between the two groups. Some studies reviewed in the present chapter have, however, found differences in the Hall and Van de Castle emotion categories between individuals with conditions marked by limbic hyperactivity and controls. Pesant and Zadra (2006) found more negative emotions in the dream reports of individuals high in trait anxiety, and Heather-Greener (1996) found more anger in the dreams of migraineurs. In a study of schizophrenic patients

compared to controls, Lusignan et al. found that the dream reports of controls contained more neutral emotions, although no differences were found for either positive or negative emotions. Kirshner (1999) found no change in percentage of negative emotions in the dream reports of a woman with generalized anxiety disorder pre- and post-treatment with an antidepressant. Taken together, these two sets of results indicate that significant findings in the H/VDC emotions categories are not always detected when individuals with high levels of limbic activity are compared to individuals with low levels of limbic activity, as was the case in the present study. As with Study 1, it should again be noted that the scoring of emotions by the dreamer versus by external raters can yield very different results (Sikka et al., 2014).

CONCLUSIONS

The results of the present study confirm the result from Study 1 which found that higher levels of limbic activity are associated with an increased recall frequency of dreaming. Contrary to expectations, higher levels of limbic activation were not associated with an increased recall frequency of bad dreams and nightmares. As noted earlier, the small sample size in each group may have resulted in a lack of statistical power, considering that a greater average recall frequency of bad dreams and nightmares was in fact experienced by individuals with higher levels of limbic activation. Also as noted earlier, conducting the study over a longer time period may also have yielded statistically significant between-group differences in bad dream and nightmare recall frequency.

Overall, the results of the dream content analysis overlap considerably with the results of the dream content analyses performed on dream reports from individuals with clinical conditions affecting the limbic system. Thus, as with the results of Study 1, the results of the present study indicate that dream phenomena that are evident in conditions marked by limbic hyperactivation may also be observed in individuals with greater than usual but non-clinical limbic hyperactivity. The findings of the present study thus add to the body of research which indicates that the limbic system is crucially involved not only in generating dreams, but also shaping dream content. With regards to the latter, increased levels of limbic activity appear to be associated with

increases in aggression and sexuality during dreaming, as well as alterations in the familiarity of characters that are encountered during dreaming.

In order to further examine the effect of increased limbic activity on dreaming, a study was performed investigating a common occurrence that prior research has indicated is related to both the limbic system and dreaming: psychosocial stress. As previous research has indicated that the use of oral contraceptives (OCs) may moderate psychosocial stress, the moderating effect that OC use may have on the relationship between psychosocial stress and dreaming was investigated, along with an examination of whether dream content is affected by OC use. This study is presented in the next chapter.

CHAPTER 6

THE INFLUENCE OF PSYCHOSOCIAL STRESS AND MONOPHASIC ORAL CONTRACEPTIVE USE ON BAD DREAMS, NIGHTMARES, AND DREAM CONTENT

INTRODUCTION

In the past, a number of studies have examined the influence that psychological stress may have on dreaming, particularly with regard to the effect of such stress on nightmare recall frequency. In contrast, studies on the relationship between oral contraceptive (OC) use and dreaming are relatively scarce, despite the fact that OCs are known to influence certain structures in the limbic system, such as the hypothalamus (Cook, 2002) and amygdala (Lifosky et al., 2016). The limbic system is in turn a system which is crucial for dream generation (Levin & Nielsen, 2007; Solms, 2000a). Oral contraceptives affect the limbic system through the hormonal effects these medications exert on the human brain, and studies have documented changes in dreaming as a result of fluctuations in the levels of various hormones (e.g., Natale, Albertazzi, & Cangini, 2003). Oral contraceptive use is related to psychological stress in that a number of studies have documented a decreased stress response in users of OCs compared to non-users (e.g., Bouma, Riese, Ormel, Verhulst, & Odelhinkel, 2009; Hammerfeld et al., 2006; Kirschbaum, Pirke, & Hellhammer, 1995; Nielsen, Segal, Worden, Yim, & Cahill, 2013) Despite the relationship between psychological stress and the use of OCs, the effect of psychological stress on dreaming has not taken into account the potential effect that the use of these medications may have. Studies on the influence of psychological stress on dreaming and of oral contraceptive use on the human stress response are reviewed below.

STRESS, THE LIMBIC SYSTEM, AND DREAMING

Stress and the limbic system

In the human brain, corticotropin-releasing-hormone (CRH) has a functional role in the hypothalamus [particularly the paraventricular nucleus (PVN)] where CRH controls the

pituitary release of adrenocorticotropin hormone (ACTH) (Cook, 2002). ACTH in turn releases glucocorticoids – the main one in humans being cortisol – from the adrenal gland (Herman, McKlveen, Solomon, Carvalho-Netto, & Myers, 2012; Kudielka & Kirschbaum, 2005). Together, these three systems comprise the hypothalamic-pituitary-adrenal (HPA) response to stress. Glucocorticoids also participate in stress activation through feed-forward mechanisms at the level of the amygdala (Herman et al., 2012).

The stress response may comprise two different types: a systemic or a processive response (Herman & Cullinan, 1997). The former involves stressors that pose an immediate physiological threat to the organism, whereas the latter involves stressors that require interpretation by higher brain structures. According to the theory of Herman and Cullinan (1997), immediate physiological threats gain access to the PVN directly. Processive stressors are channeled to the HPA axis as one part of an integrated limbic response to complex stressful stimuli; limbic circuits may then augment or diminish the stress response based on prior experience.

According to Herman et al. (2012), many studies have found the HPA axis stress response to be controlled by the limbic system. The hippocampus has been found to inhibit HPA axis secretion (along with the prefrontal cortex), whereas the amygdala is implicated in the activation of glucocorticoid secretion (Herman et al., 2005). The hippocampus has also been implicated in the interpretation of stressor intensity (Herman et al., 2005). Due to the regulatory function of the medial prefrontal cortex (mPFC), lesions of the anterior cingulate and prelimbic divisions of this brain structure enhance ACTH and corticosterone secretion (Herman et al., 2005). The opposite is also the case – chemical stimulation of the prelimbic mPFC can increase inhibition of the stress response (Herman et al., 2012). It has also been found that everyday psychosocial stress is associated with functional disintegration of ACC/mPFC (Javanovic, Perski, Berglund, & Savic, 2011).

In contrast to the inhibitory role that the hippocampus and mPFC play, the amygdala has an activating function on the HPA axis, such that certain studies have found an increase in CRH levels in the amygdala after presentation of stressful stimuli (Cook,

2002; Herman et al., 2012). In keeping with the hypothesized HPA axis activating function of the amygdala, lesions to the latter brain structure reduce ACTH and/or corticosterone secretion after stress (Herman et al., 2005).

Studies have suggested that there may in fact be two separate types of CRH response in the amygdala: (a) a fear or an alerting response; and (b) an anxiety or generalized stress response (Cook, 2002). Cook (2002) has suggested that these two types of responses are somewhat independent: although the second response often follows the first, the generalized stress response may occur in the absence of the fear or alerting response. In support of the independence of the two responses is a study by Muller et al. (2003), which found that a limbic CRH receptor modulates anxiety-related behaviour, and that this modulating effect was independent of HPA system function. Another limbic structure involved in the stress response is the bed nucleus of the stria terminalis. This region links the amygdala and hippocampus with hypothalamic and brainstem regions, and also conveys excitation of the HPA axis (Herman & Cullinan, 1997).

Stress and dreaming

Given the critical role of the limbic system in both the stress response (as noted above) and dream and nightmare generation (Levin & Nielsen, 2007; Nielsen & Levin, 2007; Solms, 2000a), it is not surprising that previous studies have found a relationship between stress and nightmares. It has even been suggested that nightmares "…may represent not a psychological disorder but a manifestation of the normal dreaming process and its tendency to incorporate recent stress-inducing or threatening experiences" (Wood, Botzin, Rosenham, Nolen-Hoeksema & Jourden, 1992, p. 223).

Cernovsky (1984) examined the relationship between stress and a number of sleep disorders using two scales: the Social Readjustment Rating Scale (SRSS) (Holmes & Rahe, 1967), and the Life Events Inventory (Tennant & Andrews, 1976). Both scales are similar in that they measure stressful life events (e.g., death of a spouse; divorce; marital separation; trouble at work) which are assigned different weightings depending on how stressful they are deemed to be. Positive and statistically significant correlations were found by Cernovsky between both weighted and unweighted scores on the SRSS and nightmare recall frequency. Positive and statistically significant correlations were also

found between nightmare recall frequency and a number of sub-scale scores derived from the Life Events Inventory: the unweighted *life change* score; the weighted *negative events* scores; and the weighted *difference index* (i.e., the difference between positive and negative events) score.

Dunn and Barrett (1988) found that, compared to control participants, frequent nightmare sufferers had more frequent major life stress, although the groups did not differ on minor life stress. In a study that examined the impact of stress on both normal dreams and nightmares, Cook, Caplan, and Wolowitz (1990) found that different kinds of life stressors were associated with the prevalence, recall frequency and content of nightmares, but not dreams. Picchioni (2001) reported that along with life stressors, daily stressors were also associated with higher levels of both nightmare recall frequency and intensity. Moreover, higher levels of social support for stress were associated with lower rates of nightmare recall frequency and intensity. Nightmare recall frequency and intensity was found to be associated with increased levels of coping, which led Picchioni (2001) to hypothesize that nightmares serve the functional role of a stress coping mechanism during sleep. Although intriguing, this hypothesis neglects the possibility that individuals who experience greater daily and life stressors may be forced to engage in greater amounts of coping simply due to experiencing greater amounts of stressful events.

As a type of daily and/or life stress, occupational stress may also be associated with an increased recall frequency of nightmares. In a study of stockbrokers – a traditionally high-stress occupation – Kroth, Thompson, Jackson, Pascali, and Ferreira (2002) found significant correlations between subjective reports of stress and the frequency of recurring nightmares recalled, as well as feelings of being chased during dreams.

Further regarding life stress, Duke and Davidson (2002) found that heightened dream recall in general was associated with more stress. A recent study by Banu, McDuff, and Zadra (2017) found that everyday levels of perceived stress in adults predicted recall of disturbed dreaming, over and above effects related to age, nightmare distress, and general psychopathology. Weinberg, Noble and Hammond (2015) similarly found that individuals who had nightmares or bad dreams reported higher levels of stress

compared to those with no bad dreams or nightmares, as did Antunes-Alves and De Koninck (2012). Hochard, Heym, and Townsend (2016) reported that frequent nightmare sufferers displayed reduced stress tolerance on a cognitive task. An earlier study by Brand et al. (2011), however, found that perceived stress did not predict dream recall in adolescents. Dream recall in Brand et al. (2011) was general dream recall, however, rather than specifically recall of dysphoric dreams, which may explain this discrepancy. High levels of neuroticism and trait anxiety have been found to be associated not only with high levels of cortisol (Tuiten et al., 1995), but also an increased frequency of bad dreams and nightmares (see Chapter 3 of the current dissertation).

One final point to note is that real stressors may increase dream recall frequency in women but decrease recall in men (Belicki, 1986; Duke & Davidson, 2002). The preceding point is important considering the sample in the present study was entirely female (see below). One potential moderating factor of the relationship between stress and dreaming for females may be oral contraceptive use. Oral contraceptives are a promising target as a moderator since a decreased stress response in OCs users compared to non-users has been documented by prior research (see below). Before turning to this research, the mechanism of action of OCs on the human brain is explained.

ORAL CONTRACEPTIVES AND STRESS

Oral contraceptive pills are sub-divided into two types: the combined oral contraceptive pill (COC), containing estrogen and progestogen; and the progestogen-only pill (the 'mini-pill'). Oral contraceptives are manufactured in three different preparations: monophasic OCs provide the same level of hormones across the menstrual cycle; biphasic OCs change the level of hormones once across the cycle; and triphasic OCs change hormone levels three times across the cycle (Oinonen & Mazmanian, 2001).

The estrogens and progestogens are both major classes of steroid hormones (ter Horst, 2010). In addition to both hormones being involved in control of the human female reproductive cycle, certain progestogens are precursors to all other steroids including the estrogens and glucocorticoids (Levin & Hammes, 2011; Xu, Hoebeke, & Bjorntorp,

1990). As discussed above, the glucocorticoids are crucially involved in the human stress response. Use of OCs affects the HPA axis due to the involvement of this axis in hormonal control in the human body. The precise mechanisms of action of OCs on the human brain is, however, too complex to describe adequately here (for overviews, see Levin & Hammes, 2011; Nelson & Cwiak, 2011; and Speroff & Darney, 2011).

Due to the relationship between OC use and the HPA axis, and the HPA axis and stress, it is not surprising that a number of studies have documented an altered stress response in users of OCs. This alteration is primarily in the direction of a reduced response to stress. Kirschbaum, et al. (1995) found attenuated cortisol responses to the Trier Social Stress Test (TSST) (a test which involves performing public speaking and mental arithmetic in front of an audience) in COC users compared to controls. In fact, the cortisol response in COC users was found to be less than half that observed in controls.

The finding of a generally attenuated cortisol response to the TSST in COC users versus controls was later replicated by Kirschbaum, Kudielka, Gaab, Schommer, and Hellhammer (1999), and even later by Hammerfeld et al. (2006). Kirschbaum et al. (1999) also found an attenuated cortisol response in COC users compared to controls after injection with a synthetic analogue of ACTH. Bouma et al. (2009) reported not only a blunted, but also a linearly decreasing free cortisol response during a social stress test. A blunted stress hormone response to emotionally arousing images in women using OCs compared to controls was found in a more recent study too (Nielsen, Segal, et al., 2013).

Kulhman and Wolf (2005) found that memory retrieval was unimpaired in COC users compared to controls following oral administration of hydrocortisone. This is another result that could be expected from the prior findings of an attenuated stress response in users of OCs, considering that cortisol is known to impair retrieval of memories. Finally, Kirschbaum, Platte, Pirke, and Hellhammer (1996) reported attenuated adrenocortical activation following stressful exercise in users of OCs compared to non-users, illustrating that this attenuation can be elicited in response to both physical as well as psychological stress.

How might OC use attenuate the stress response? Some studies have noted a lower rate of ACTH release in users of OCs after stimulation with human CRH (Kirschbaum et al., 1995). ACTH controls the rate of release of cortisol (Cook, 2002); lower rates of ACTH release may subsequently result in lower rates of cortisol release, and thus a subsequent reduction in the level of stress. The study by Kirschbaum et al. (1999), however, found that OC users secrete similar amounts of ACTH and total cortisol in response to psychosocial stress. The attenuated stress response in OC users therefore appears most likely to be due to the enhanced binding of free cortisol by corticosteroid-binding globulin (CBG) in users of OCs ('free' cortisol differs from total cortisol in that the former is the percentage of total cortisol that is not bound to proteins such as CGB). Increased binding of free cortisol by CBG in users of OCs would reduce the amount of biologically active cortisol in the body, thus resulting in a concomitant attenuation of the stress response. This increased CBG binding of cortisol in users of OCs is most likely due to the CBG-enhancing effect of ethinyl-estradiol, which is an estrogen used in many formulations of OCs (Kirschbaum et al., 1999).

Although it does not appear that any previous studies have directly investigated the effect of OCs on dreaming, certain studies on dreaming across the menstrual cycle indirectly indicate that OC use may indeed have such an effect. One of the first of such studies (Swanson & Foulkes, 1967) found that sexuality, dream unpleasantness, and hostility were highest during the menstrual phase. Sirois-Berliss and de Koninck (1982) later replicated the finding of greater hostility during the menstrual period, but found that this also occurred during the premenstrual phase, and also that there was a significant increase in dream anxiety during each of those two phases.

Bucci, Creelman, and Severino (1991) found specificity, concreteness, imagery, and clarity in the content of dream reports to be highest during the early luteal phase, and also that the dominant emotions during this phase were directed towards other people. Themes in the early follicular and late luteal phases were more consistent with passivity and self-care. Natale et al. (2003) found that participant self-ratings of mood were found to be the worst during the pre-menstrual phase. This was reflected in dream content as dream reports from this phase contained predominantly negative emotions. Positive emotions were more common in the pre-ovulatory phase. Taken together, all these

results indicate that hormonal variation throughout the menstrual cycle may have an effect on dreaming.

These findings are related to OC use in that the use of these medications may stabilize mood across the menstrual cycle, due to the suppression of cyclical changes in estradiol and progesterone (Hamstra et al., 2015; Montoya & Bos, 2017). The relationship is quite complex, however. A meta-analysis (Oinonen & Mazmanian, 2002) found that OC-related mood changes are beneficial in most women, but other studies have found increased rates of negative mood symptoms (Gingnell et al., 2013; Pletzer & Kirschbaum, 2014). Combined OCs containing ethinyl-estradiol as an agonist for estradiol receptors may promote positive mood changes, due to the 5-HT (serotonin) enhancing properties of estradiol (Pletzer & Kirschbaum, 2014). Negative mood changes may be as a result of the decline of endogenous estradiol as a result of OC use (Montoya & Bos, 2017; Pletzer & Kirschbaum, 2014). Progesterone may promote positive mood changes at low concentrations and negative mood changes at high concentrations (Pletzer & Kirschbaum, 2014), although the progesterone/estrogen ratio probably correlates the most with the direction of emotional changes (Oinonen & Mazmanian, 2002).

Drospirenone-containing OCs appear to be the most effective at reducing or even improving negative mood in women. Wichianpitaya and Taneepanichskul (2013) found these OCs to be particularly effective at improving premenstrual symptoms. Brown, Ling, and Wan (2002) found improvements in premenstrual and menstrual scores of negative affect relative to baseline in a sample of women taking drospirenone-containing OCs, and Parsey and Pong (2000) found decreases in negative affect in all menstrual cycle phases. Improved quality of life after the use of drospirenone-containing OCs has also been noted (Poromaa & Segebladh, 2012).

Considering that hormonal variation across the menstrual cycle is associated with changes in dreaming (Natale et al., 2003), as well as that OCs may stabilize hormonal variation and mood across the menstrual cycle – or even improve it – there is thus the possibility that users of OCs may experience dreaming differently compared to non-users. Based on prior research which has also documented an attenuation of the stress

response in OC users, it is reasonable to hypothesize that users of OCs would experience more positive dreaming in general compared to non-users, although it appears that no studies have directly investigated this.

The aims of the present study were therefore twofold: firstly, to document whether a relationship exists between psychosocial stress and bad dream and nightmare recall frequency, and whether this relationship is moderated by the use of OCs; and secondly, to investigate if any differences in certain dream characteristics and content exist between users and non-users of OCs. Based on the research reviewed above, four specific hypotheses were derived:

(a) *Greater amounts of psychosocial stress will result in a greater recall frequency of bad dreams and nightmares*, given the association of stress with the limbic system and the association of the limbic system with bad dream and nightmare generation.

(b) *Oral contraceptive use will moderate the relationship between psychosocial stress, and bad dreams and nightmares*. Based on the prior research reviewed above, the use of OCs will attenuate the relationship between stress, and bad dreams and nightmares.

(c) *The content of the most recent dreams of OC users will be more positive than the content of non-users* given the ability of OCs to stabilize mood and hormonal changes across the menstrual cycle, as noted in the literature reviewed above.

(d) *Differences on various sleep and dreaming variables will be evident between users of OCs and non-users.* Given the paucity of research on dreaming in users of OCs this hypothesis is exploratory in nature, and it is difficult to make specific predictions. Based on some of the research reviewed above, however, *dreaming in OC users should generally be more positive*.

METHODS

Participants

Participants were 240 female undergraduate psychology students who participated in exchange for course credit. The average age was 19.42 years (*SD* = 1.71). All students who indicated that they had any psychological or psychiatric condition (e.g., depression;

anxiety; obsessive-compulsive disorder, etc.) were excluded from the final sample, as were any students who indicated that they had any serious medical condition, and/or were on any chronic medication (e.g., antidepressants; lithium; antiretrovirals, etc.).

Of the participants using OCs, the majority (*n* = 59; 96.70%) were using monophasic preparations. Only two participants were using triphasic OCs (3.30%). No participants were currently using biphasic preparations. Due to the small number of triphasic OC users, these two participants were also excluded from the final sample, leaving a sample of exclusively monophasic OC users. All participants in the OCs group who indicated that they had switched from one brand of contraceptive to another were also removed prior to data analysis, based on the recommendation of Oinonen and Mazmanian (2001). All OCs users who had been using for less than 3 months, as well as non-OCs users whom had taken OCs, the contraceptive injection, or any other type of hormonal birth control within 6 months prior to the start of the study were also excluded. This left a final sample of *n* = 45 OC users, and a control group of *n* = 181.

The most common brands of OCs used were *ethinyl estradiol/drospirenone* based OCs (*n* = 31). *Cyproterone/ethinylestradiol* users (*n* = 10) made up the next largest sub-group. Two participants were using *ethinylestradiol/levonorgestrel* based OCs; one participant was using an *ethinylestradiol/norgestrel* based OC; and one participant was using an *ethinylestradiol/gestodene* based OC. In the final sample of OC users, the average length of use of OCs was 18.38 months (*SD* = 12.28), and the median length of use was 14 months.

Measures

A number of questionnaires contained in an online survey were used to measure stress-related variables. Although not all of the questionnaires measure stress *per se*, all variables measured by the questionnaires have been shown to be related to stress by prior research.

(1) The Clinical Outcomes in Routine Evaluation – Short Form A (CORE-SF-A) (Evans et al., 2002)[2]. This is an 18-item Likert-type questionnaire that measures feelings

[2] Obtained from http://www.coreims.co.uk/index.html and used under the Creative Commons Attribution-NoDerivatives 4.0 International (CC BY-ND 4.0) license.

relating to stress and tension. This questionnaire is pan-theoretical and pan-diagnostic, and has demonstrated good reliability and convergent validity (see Evans et al., 2002). Short Form A was used to reduce the total length of the online survey, in an attempt to prevent participant fatigue.

(2) The Perceived Social Stress Scale (PSS) (Cohen, Kamarck, & Mermelstein, 1983)[3]. A 14-item Likert-type questionnaire that measures "…the degree to which situations in one's life are appraised as stressful" (Cohen et al., 1983, p. 385). The scale has demonstrated concurrent and predictive validity, as well as good reliability (see Cohen et al., 1983).

(3) The GAD-7 (Spitzer, Kroenke, Williams, & Lowe, 2006)[4]. A 7-item Likert-type measure of generalized anxiety disorder with demonstrated reliability and validity (see Spitzer, Kroenke, Williams, & Lowe, 2006). This measure was included as previous studies (e,g., Cohen et al., 1983) have shown a relationship between stress and anxiety.

(4) The Coping Strategies Inventory – Short Form (CSI-SF) (Addison et al., 2007)[5]. A 16-item Likert-type questionnaire that measures coping strategies individuals apply to handling stressors and problems. Based on the recommendation by Addison et al. (2007), only the first 15 items were included, as Item 16 has not demonstrated adequate psychometric properties.

This scale measures four types of problem- and emotion-focused coping, as proposed by Lazarus (2007): (a) Problem-focused engagement (PFE); (b) Problem-focused disengagement (PFD); (c) Emotion-focused engagement (EFE); and (d) Emotion-focused disengagement (EFD). All four sub-scales have demonstrated adequate reliability (Addison et al., 2007). The PFE and EFE sub-scale scores are combined to form a second-tier *engagement* sub-scale and the PFD and EFD sub-scale scores are combined to form a second-tier *disengagement* sub-scale, with both sub-scales having adequate reliability (Addison et al., 2007).

---

[3] Obtained from Cohen, S., Kamarck, T., & Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior, 24,* 385-396.

[4] Obtained from Spitzer, R. L., Kroenke, K., Williams, J. B. W., & Lowe, B. (2006). A brief measure for assessing generalized anxiety disorder: The GAD-7. *Archives of Internal Medicine, 166,* 1092-1097.

[5] Obtained from Addison, C. C., Campbell-Jenkins, B. W., Sarpong, D. F., Kibler, J., Singh, M., et al. (2007). Psychometric evaluation of a Coping Strategies Inventory Short-Form (*CSI-SF*) in the Jackson Heart Study cohort. *International Journal of Environmental Research and Public Health, 4,* 289-295.

According to Addison et al. (2007), *engagement* strategies are . . .”approach-related actions that result in confronting stressors, often viewed as a crucial factor in limiting the long-term psychological and physiological sequelae of environmental stressors” (p. 290). *Disengagement* strategies are characterized by avoidance which seeks to limit exposure to stressors. Although disengagement often produces favourable short-term effects, such a strategy can lead to longer-term problems such as depression (Addison et al., 2007). *Emotion-focused* coping efforts emphasize the regulation of the affective response, whereas *problem-focused* efforts emphasize management of the stressful situation (Addison et al., 2007).

(4) The Positive and Negative Affect Scale (PANAS) (Watson, Clark, & Tellegen, 1988)[6]. A 20-item scale in which participants rate on Likert-type items how much they have experienced a certain emotion over a particular time period. In the present study, participants rated how much they had experienced each emotion over the past month. Ten of the items relate to positive emotions, and 10 of the items to negative emotions. This scale was included as the PANAS has shown correlations with the *A-State*, a questionnaire which measures responses to a variety of stressful events (Watson et al., 1988). Both sub-scales of the PANAS have demonstrated adequate reliability and validity (see Watson et al., 1988).

(5) The Dreaming Questionnaire (DRM-Q). A 33-item self-report questionnaire, as used in Study 1. As with Study 1, additional items were included on bad dream and nightmare distress, as well as 8-point items measuring bad dream and nightmare recall frequency. Additional bad dream and nightmare recall frequency measures asked participants to estimate the number of bad dreams and nightmares experienced in the last month. See Appendix A for the DRM-Q.

(6) The Nightmare Distress Questionnaire (NDQ) (Belicki, 1992). A self-report measure that measures the degree of global distress an individual experiences as a result of suffering from nightmares. See the *Methods* section of Study 1 for more information about this questionnaire.

(7) The Most Recent Dream Method (Domhoff, 1996). Participants were asked to write down the most recent dream they had experienced. This method is powerful and

[6] Obtained from Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54,* 1063-1070.

efficient because it allows large samples of dreams to be collected in a relatively short period of time, and has proven reliability and validity (Domhoff, 2010). See Appendix A.

Design and procedure

The study was approved by the Research Ethics Committee of the Department of Psychology, University of Cape Town. A relational design was used to examine the predictive effect of psychosocial stress, moderated by OC use, on bad dream and nightmare recall frequency. To examine differences in sleep and dream characteristics, a between-groups design comparing OCs users with non-users was employed.

All psychology undergraduate students were sent an email informing them of the study. The email contained a link to an online questionnaire, which the students completed in their own time. Data collection ran for approximately three months, and all students were sent a reminder to participate in the study once per month. Participation in the study was entirely voluntary.

All usable most recent dream reports were coded by the researcher. All reports from OCs users were coded by a second independent coder, as well as a random selection of approximately half the dream reports from the OCs non-users. Any discrepancies in codes were resolved through discussion between the two coders prior to data analysis. As all identifying information except for participant numbers was removed from the dream reports prior to coding, both coders were blind as to which group participants' reports came from. The independent coder was also blind as to the design and purpose of the study.

RESULTS

Psychometric properties of the questionnaires

Reliability. Internal consistency reliability was calculated using Cronbach's α, using the total sample (N = 320). Reliabilities for the CORE, PSS, and GAD-7 were very good at .90, .87, and .87, respectively. Overall reliability for the PANAS was good at .74, with the PA and NA sub-scales having very good reliability values of .87 and .86, respectively. The four sub-scales of the CSI demonstrated low-to-moderate reliability, with the values

being: (a) CSI PFE: .79; (b) CSI PFD: .70; (c) CSI EFE: .64; and (d) CSI EFD: .56. Reliability for the NDQ was very good at .87.

Reliability for the core 33-item DRM-Q scale was calculated using the total sample of participants who completed all 33 items (N = 228), and was excellent at α = .91 (the reliability was α = .90 as calculated in Study 1). Reliability for the three 11-item *dreams* (*DRM*), *bad dreams* (*BD*), and *nightmares* (*NM*) sub-scales was moderate-to-good at α = .74, .80, and .80, respectively.

Component Structure of the DRM-Q. In order to test the consistency of the component structure of the DRM-Q across samples, the 33 items of this questionnaire were analyzed using principal components analysis via Varimax rotation, using only responses from participants who completed all 33 items (n = 228). The Kaiser-Meyer-Olkin test of sampling adequacy was good at .823, and Bartlett's test of sphericity was statistically significant [$\chi^2(528) = 4555.25$, $p < .001$].

Eight well-defined components emerged with eigenvalues greater than 1 (as in Study 1), which together accounted for 70.38% of the variance. All communalities were greater than .50 [range = .59 (*DRM Aggression to others*) to .81 (*BD Aggression to self intensity*)]. The full component structure is displayed in Tables 18 and 19. The component structure is displayed in two tables due to its large size. The eigenvalues, percentage of variance explained by each component, and the reliability of each component is displayed in Table 20.

The component structure was largely identical to that identified in Study 1. The one difference in the component structure identified in the present analysis compared to that identified in Study 1 pertained to the *vividness* and *intensity* items. In Study 1, the items measuring *BD vividness* and *intensity* loaded on a component with the items measuring *NM vividness* and *intensity*; and the *DRM vividness* and *DRM intensity* items loaded together on an individual component.

Table 18.

Component Structure of the DRM-Q, Part 1 (Study 3)

Variable	DRM-Q Component	1	2	3	4	5	6	7	8
DRM Aggression to self	1. Aggression to self	**.742**	.123	.193	.154	.025	.092	-.154	.088
DRM Aggression to self intensity	1. Aggression to self	**.785**	.155	.148	.190	.092	.110	-.179	.073
BD Aggression to self	1. Aggression to self	**.852**	.016	.124	.041	.020	.087	.100	.046
BD Aggression to self intensity	1. Aggression to self	**.852**	.180	.095	.077	.009	.119	.100	.094
NM Aggression to self	1. Aggression to self	**.744**	.017	.191	-.014	.059	.093	.349	.003
NM Aggression to self intensity	1. Aggression to self	**.775**	.172	.078	.034	.045	.098	.323	.092
DRM Meaningfulness	2. Meaningfulness-affectedness	.019	**.744**	.027	-.013	.230	.124	-.150	.073
BD Meaningfulness	2. Meaningfulness-affectedness	.125	**.821**	.051	-.015	.217	.091	-.068	.039
NM Meaningfulness	2. Meaningfulness-affectedness	.142	**.755**	.073	.001	.259	.012	.103	-.287
DRM Affectednessfulness	2. Meaningfulness-affectedness	.090	**.711**	.014	.060	-.008	.003	.162	.380
BD Affectedness	2. Meaningfulness-affectedness	.226	**.686**	.030	.060	-.028	-.041	.325	.336
NM Affectedness	2. Meaningfulness-affectedness	.231	**.708**	.043	.118	-.029	-.084	.442	.099
DRM Aggression to others	3. Aggression to others	.000	.133	**.713**	.110	-.067	.056	-.067	.197
DRM Aggression to others intensity	3. Aggression to others	.125	.103	**.747**	.083	-.075	.106	-.075	.124
BD Aggression to others	3. Aggression to others	.029	-.056	**.789**	.138	-.054	.032	-.139	.073
BD Aggression to others intensity	3. Aggression to others	.208	.085	**.723**	.072	-.040	.021	-.021	.051
NM Aggression to others	3. Aggression to others	.134	-.089	**.799**	.030	.016	.061	.204	-.048
NM Aggresssion to others intensity	3. Aggression to others	.217	.009	**.745**	-.112	.001	.026	.207	-.184
DRM Colourfulness	4. Colourfulness	.026	-.080	.097	**.735**	.023	.102	.054	.243
BD Colourfulness	4. Colourfulness	.136	.081	.027	**.850**	.029	.100	-.051	.099
NM Colourfulness	4. Colourfulness	.131	.048	.151	**.762**	-.034	.056	.160	-.213
DRM Realism	5. Realism	.000	.074	-.108	.026	**.800**	.073	-.080	.151
BD Realism	5. Realism	.115	.250	-.119	-.026	**.791**	.039	.123	.134
NM Realism	5. Realism	.094	.239	.003	.111	**.754**	-.066	.327	-.078

Table 19.

Component Structure of the DRM-Q, Part 2 (Study 3)

Variable	DRM-Q Component	1	2	3	4	5	6	7	8
DRM Movement	6. Movement	.132	.161	.058	.093	-.078	**.799**	-.088	.148
BD Movement	6. Movement	.215	-.016	.145	.153	.055	**.794**	.048	.109
NM Movement	6. Movement	.147	-.033	.076	.101	.128	**.754**	.358	-.012
NM Vividness	7. NM vividness-intensity	.088	.171	.035	.435	.272	.129	**.633**	.173
NM Intensity	7. NM vividness-intensity	.301	.221	-.075	.073	.168	.223	**.606**	.215
DRM Vividness	8. DRM-BD vividness-intensity	.066	.046	.143	.421	.051	.168	.210	**.583**
DRM Intensity	8. DRM-BD vividness-intensity	.233	.295	.191	-.007	.185	.132	-.003	**.635**
BD Vividness	8. DRM-BD vividness-intensity	.223	.082	.071	.461	.229	.151	.328	**.466**
BD Intensity	8. DRM-BD vividness-intensity	.480	.227	.003	.047	.225	.165	.250	**.468**

Table 20.

Eigenvalues, Percentage of Variance Explained, and Reliability Values of the DRM-Q Components (Study 3)

DRM-Q Component	Eigenvalue	% Variance	α
1. Aggression to self	4.63	14.02	.91
2. Aggression to others	3.82	11.56	.87
3. Meaningfulness-affectedness	3.70	11.22	.86
4. Colourfulness	2.63	7.98	.77
5. Realism	2.30	6.98	.79
6. Movement	2.15	6.50	.77
7. NM vividness-intensity	2.06	6.25	.70
8. DRM-BD vividness-intensity	1.94	5.87	.74

In the present analysis, the items measuring *BD vividness* and *BD intensity* loaded together on a component with *DRM vividness* and *DRM intensity*; and *NM intensity* and *NM vividness* loaded together on an individual component. The *BD intensity* item loaded slightly higher on the component of *aggression to self*, although as the difference between the two loading values was negligible (.012), this item was counted as part of the *DRM-BD vividness-intensity* component as it made more conceptual sense to keep the item as part of the latter component. Despite this slight difference, the DRM-Q appears to have a highly consistent component structure. With regards to the one difference between the two component structures, it should be noted that in Study 1, the *BD vividness* and *BD intensity* items did in fact load on a component along with the *DRM vividness* and *intensity* items, as these items did here. In Study 1 they were kept as part of a *BD-NM vividness-intensity* component as the loadings on that component were greater.

The relationship between stress, and bad dreams and nightmares, with oral contraceptive use as a moderator

The intercorrelations among variables are displayed in Table 21. All moderation analyses were run using the psychosocial stress variables as predictors, with *OC use* (*yes* or *no*) as the moderator. Number of bad dreams and nightmares in the last month were used as the criterion variables as this time period of measurement was more accurately aligned with the period of time that some of the psychosocial stress scales used in the present study ask participants to report on. For example, the CORE-SF-A asks participants to report how they have felt over the last week, and the PSS asks participants to report how they have felt over the last 1 month.

As the standardized residuals displayed an extremely non-normally distributed pattern for all analyses using *number of BDs over the past month* as the dependent variable, this variable was log-normal transformed (*LogN BDs*). Re-running the analyses with *LogN BDs* as the dependent variable revealed that the transformation correctly adequately for the skewed residuals. No such problems with the residuals were evident using *number of NMs over the past month* as the dependent variable.

Table 21.

Intercorrelation Matrix for Variables Predictive of Bad Dream and Nightmare Recall Frequency

Variable	Scale	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1	**OC Use**[a]	-													
2	**CORE-SF-A**	-.10	-												
3	**PSS**	-.06	.76***	-											
4	**GAD-7**	-.06	.77***	.70***	-										
5	**PANAS PA**	-.01	-.47***	-.61***	-.35***	-									
6	**PANAS NA**	-.05	.74***	.69***	.77***	-.33***	-								
7	**PFE**	.11	-.04	-.08	-.03	.16*	.03	-							
8	**PFD**	.08	-.35***	-.47***	-.26***	.47**	-.33***	.17*	-						
9	**EFE**	-.11	.25***	.35***	.28***	-.19*	.29***	-.02	-.22**	-					
10	**EFD**	.00	.44***	.43***	.42***	-.37***	.45***	-.13	-.29***	.20**	-				
11	**ENGAGEMENT**	.01	.14	.19*	.17*	-.01	.22**	.73***	-.03	.68***	.04	-			
12	**DISENGAGEMENT**	.08	.02	-.10	.08	.15	.04	.05	.68***	-.05	.50***	.01	-		
13	**BDs Past month**	.05	.24**	.24***	.27***	-.15	.19*	.09	-.23**	.13	.24***	.15*	-.02	-	
14	**NMs Past month**	.05	.29***	.29***	.37***	-.08	.29***	.10	-.10	.19*	.16*	.20**	.04	.65***	-

Note. N = 171. Two-tailed correlations are reported. Listwise deletion of missing data was used.

Scale: OC Use = Oral contraceptive use; CORE-SF-A = Clinical Outcomes in Routine Evaluation - Short Form A; PSS = Perceived Social Stress;

GAD-7 = Generalized Anxiety Disorder-7; PFE = Problem-focused engagement; PFD = Problem-focused disengagement

EFE = Emotion-focused engagement; EFD = Emotion-focused disengagement; ENGAGEMENT = Engagement total scores

DISENGAGEMENT = Disengagement total scores; BDs Past month = Total number of bad dreams within the last 1 month

NMs Past month = Total number of nightmares within the last 1 month.

[a]Oral contraceptive use = Yes was coded as 1, and Oral contraceptive use = No was coded as 0.

*p < .05; **p < .01; ***p < .001

The predictors were centered using the mean subtraction method of centering, and the moderator was centered by coding *OC use yes* = 1 and *OC use no* = -1. The moderating effect of OC use was tested using a two-step hierarchical regression. The psychosocial stress predictor and the moderator variable were entered at step 1, and the psychosocial stress predictor, the moderator, and a new variable – the interaction between the moderator and the independent variable – were entered at step 2. A significant change in the amount of variance explained between step 1 and 2 was taken as an indication of a moderating effect.

Separate analyses were run for each of the psychosocial stress predictor variables, which were: **(a)** CORE total score; **(b)** PSS total score; **(c)** GAD-7 total score; **(d)** PANAS PA sub-scale score; **(e)** PANAS NA sub-scale score; **(f)** CSI PFE; **(g)** CSI PFD; **(h)** CSI EFE; **(i)** CSI EFD; **(j)** CSI ENG; and **(k)** CSI DISENG sub-scale scores.

Bad dream recall frequency. Due to some participants not completing the bad dream recall frequency item, the final sample size for this analysis was $N = 171$ (OCs users $n = 38$; OCs non-users $n = 133$). No moderating effect of OCs use on the relationship between any of the psychosocial stress measures and bad dream recall frequency was evident, although a number of the stress variables significantly predicted bad dream recall frequency. These were: **(a)** CORE total scores ($r = .31$, $p < .001$; Step 1: $\beta = .31$, $t = 4.26$, $p < .001$; Step 2: $\beta = .34$, $t = 3.89$, $p < .001$); **(b)** PSS total scores ($r = .29$, $p < .001$; Step 1: $\beta = .30$, $t = 3.97$, $p < .001$; Step 2: $\beta = .30$, $t = 3.34$, $p < .001$); **(c)** GAD-7 total scores ($r = .33$, $p < .001$; Step 1: $\beta = .33$, $t = 4.58$, $p < .001$; Step 2: $\beta = .37$, $t = 3.94$, $p < .001$); **(d)** PANAS NA sub-scale scores ($r = .26$, $p < .001$; Step 1: $\beta = .27$, $t = 3.61$, $p < .001$; Step 2: $\beta = .31$, $t = 3.21$, $p = .002$); **(e)** CSI PFD sub-scale scores ($r = -.17$, $p = .016$; Step 1: $\beta = -.17$, $t = 2.22$, $p = .028$; Step 2: $\beta = -.20$, $t = 2.11$, $p = .036$); and **(f)** CSI EFD sub-scale scores ($r = .23$, $p < .001$; Step 1: $\beta = .23$, $t = 3.03$, $p = .003$; Step 2: $\beta = .26$, $t = 2.26$, $p = .025$).

The PANAS PA sub-scale scores did not predict bad dream recall frequency, and neither did the CSI EFE nor PFE scores. CSI ENG and DISENG sub-scale scores also did not predict bad dream frequency. Due to the number of variables involved and the

subsequent length of the statistical tables, the regression results are reported in full in Appendix C

Nightmare recall frequency. As with the bad dream frequency variable, some participants did not complete the nightmare recall frequency item. As the data from two participants in the OCs non-users group resulted in large standardized residuals (> 5) for all initial analyses, these two participants were removed, and the analyses re-run. The final sample size for all subsequent analyses was therefore $N = 169$ (OCs users $n = 38$; OCs non-users $n = 131$). No moderating effect of OC use on the relationship between any of the psychosocial stress measures and nightmare recall frequency was evident, although a number of the stress variables significantly predicted nightmare frequency. As with bad dream frequency, these were: **(a)** CORE total scores ($r = .34$, $p < .001$; Step 1: $\beta = .35$, $t = 4.74$, $p < .001$; Step 2: $\beta = .36$, $t = 4.27$, $p < .001$); **(b)** PSS total scores ($r = .35$, $p < .001$; $\beta = .35$, $t = 4.83$, $p < .001$; Step 2: $\beta = .38$, $t = 4.43$, $p < .001$); **(c)** GAD-7 total scores ($r = .39$, $p < .001$; Step 1: $\beta = .39$, $t = 5.47$, $p < .001$; Step 2: $\beta = .39$, $t = 4.33$, $p < .001$); and **(d)** PANAS NA sub-scale scores ($r = .35$, $p < .001$; Step 1: $\beta = .35$, $t = 4.86$, $p < .001$; Step 2: $\beta = .34$, $t = 3.76$, $p < .001$).

PANAS PA sub-scale scores did not predict nightmare recall frequency at Step 1, although the scores had a statistically significant negative correlation ($r = -.14$, $p = .034$), trended towards significance (Step 1: $\beta = -.14$, $t = 1.83$, $p = .069$), and did negatively predict nightmare recall frequency at Step 2 ($\beta = -.23$, $t = 2.46$, $p = .015$). It should be noted that there was high multicollinearity between the PANAS PA sub-scale scores and the interaction variable ($r = -.57$), as well as an increase in the size of the partial and part correlations compared to the zero-order correlations for both the PANAS PA sub-scale scores and the interaction variable. Given that the PANAS PA sub-scale scores were not a significant predictor at Step 1, it is likely that the interaction variable was acting as a suppressor variable when entered at Step 2, thus resulting in the PANAS PA scores becoming a significant predictor at the latter compared to the former step.

Whereas CSI PFE sub-scale scores predicted bad dream recall frequency, they did not predict nightmare recall frequency. As with bad dream frequency, CSI PFD sub-scale scores were again a significant negative predictor ($r = -.16$, $p = .019$; Step 1: $\beta = -.16$, $t =$

2.09, p = .038; Step 2: β = -.18, t = 1.99, p = .048). In contrast to the result obtained with bad dream recall frequency as the criterion, CSI EFE sub-scale scores significantly predicted nightmare recall frequency (r = .27, p < .001; Step 1: β = .28, t = 3.67, p < .001; Step 2: β = .26, t = 3.19, p = .002). CSI EFD sub-scale scores significantly predicted nightmare recall frequency at Step 1 (r = .18, p = .009; β = .18, t = 2.37, p = .019), but not at Step 2 (β = .19, t = 1.70, p = .097), although there was large multicollinearity between the CSI EFD scores and interaction variable (r = -.74). Again in contrast to the result obtained with bad dream frequency as the criterion, CSI ENG sub-scale scores significantly predicted nightmare recall frequency (r = .27, p < .001; Step 1: β = .27, t = 3.59, p < .001; Step 2: β = .25, t = 2.85, p = .005). As before, CSI DISENG sub-scale scores were not a significant predictor. Again, due to the number of variables involved and the subsequent length of the statistical tables, the regression results are reported in full in Appendix C.

Differences in dream content between OCs users and non-users

The most recent dream reports were coded using the Hall and Van de Castle system of coding dream content. The same nine coding categories as used in Study 1 and 2 were used for the present study. See the *Methods* section of Study 1 for additional information on the Hall and Van de Castle system of coding. Due to the small sample size, all most recent dreams from OCs users were included in the sample (n = 45). For the non-users of OCs, all dream reports of less than 50 or more than 300 words were removed from the sample, as with Study and Study 2 (see Chapter 4 for the rationale behind this). In total, 28 dreams were excluded according to this criterion. This resulted in a usable sample of n = 153 control dreams.

An independent samples t test indicated no difference in the mean length of dream reports from students using OCs (M = 142.47, SD = 86.99) and students not using OCs (M = 121.69, SD = 59.27) [t(56.53) = 1.50, p = .139]. The t test was run with equal variances not assumed, due to a statistically significant Levene's test for homogeneity of variance. Inter-rater reliability was calculated by correlating the frequency percentages obtained for each category from each rater for the two coders, and was excellent at .91.

Only one between-group difference was evident for *characters*, with non-users of OCs having more animals in their most recent dreams than OCs users (p = .036). For *social interactions*, a lower *aggression/friendliness ratio* was evident for OCs users compared to non-users (p = .003).

Most differences were evident on the *self-concept* percentages, with the differences suggesting that the most recent dreams of OCs users are generally more positive overall than those of non-users. Specifically, less *self-negativity* (p = .007), *bodily misfortunes* (p = .005), and *negative emotions* (p < .001) were present in the dreams of users compared to non-users.

A greater tendency towards *dreamer-involved success* in the dreams of users compared to non-users was also evident, although higher than the .05 level of statistical significance (p = .053). In keeping with the trend of generally more positive content in the dreams of OCs users, a greater percentage of dreams with at least one instance of *good fortune* was also evident compared to non-users (p < .001). No other statistically significant differences in content were found. The results are reported in full in Table 22, and the *h*-profile in Figure 3.

Differences in sleep and dream characteristics of OCs users and non-users

A series of two-tailed independent samples *t* tests were run using the sleep and dreaming variables measured as dependent variables, to test for differences between the OCs users and OCs non-users. Alpha was set at .003 to control for inflated Type I error rate due to multiple comparisons (i.e., $\alpha = .05/16$). At the corrected level of alpha, no statistically significant between-group differences were evident for any of the variables measured. The results of these analyses are presented in full in Table 23.

The variables comprising each factor of the DRM-Q were entered together as multiple dependent variables in eight MANOVAs. Statistical significance for individual variables was computed using follow-up univariate ANOVAs. Only one statistically significant overall effect was detected, which was for the *dream realism* component [V = 0.06, $F(3, 163) = 3.16$, p = .026, η_p^2 = .055] (OCs users n = 38; OCs non-users n = 129).

Table 22.

Percentages and h*-values of OCs Users Compared to OCs Non-users Dreams*

Dream Content Category	OCs Non-users (*n* = 153)	OCs Users (*n* = 45)	*h*	*p*
Characters				
Male/Female	50%	51%	+.03	.822
Familiarity	72%	76%	+.08	.440
Friends	42%	44%	+.04	.714
Family	29%	32%	+.05	.611
Dead & Imaginary	2%	1%	-.06	.539
Animal	8%	3%	**-.22**	**.036***
Social Interaction %				
Aggression/Friendliness	62%	33%	**-.58**	**.003****
Befriender	33%	20%	-.30	.270
Aggressor	26%	10%	-.43	.206
Victimization	74%	90%	+.43	.211
Physical Aggression	50%	69%	+.38	.159
Social Interaction Ratios				
A/C Index	.21	.13	-.18	
F/C Index	.14	.21	+.18	
S/C Index	.03	.02	-.01	
Self-Concept %				
Self-negativity	73%	55%	**-.38**	**.007****
Bodily misfortunes	48%	20%	**-.60**	**.005****
Negative emotions	81%	59%	**-.48**	**< .001*****
Dreamer-involved success	36%	75%	+.80	.053
Dreams with at Least One				
Aggression	35%	22%	-.28	.103
Friendliness	31%	40%	+.19	.251
Sexuality	6%	4%	-.07	.701
Misfortune	31%	42%	+.23	.183
Good Fortune	3%	24%	**+.71**	**< .001*****
Success	6%	13%	+.26	.129
Failure	10%	4%	-.21	.211
Striving	14%	18%	+.09	.585

Note. The Ocs non-users were used as the baseline.

Statistical significance values are not reported for the *social interaction ratios* as these do not use the the exact h statistic.

Statistically significant *h* values are marked in bold.

*$p < .05$. **$p < .01$. ***$p < .001$.

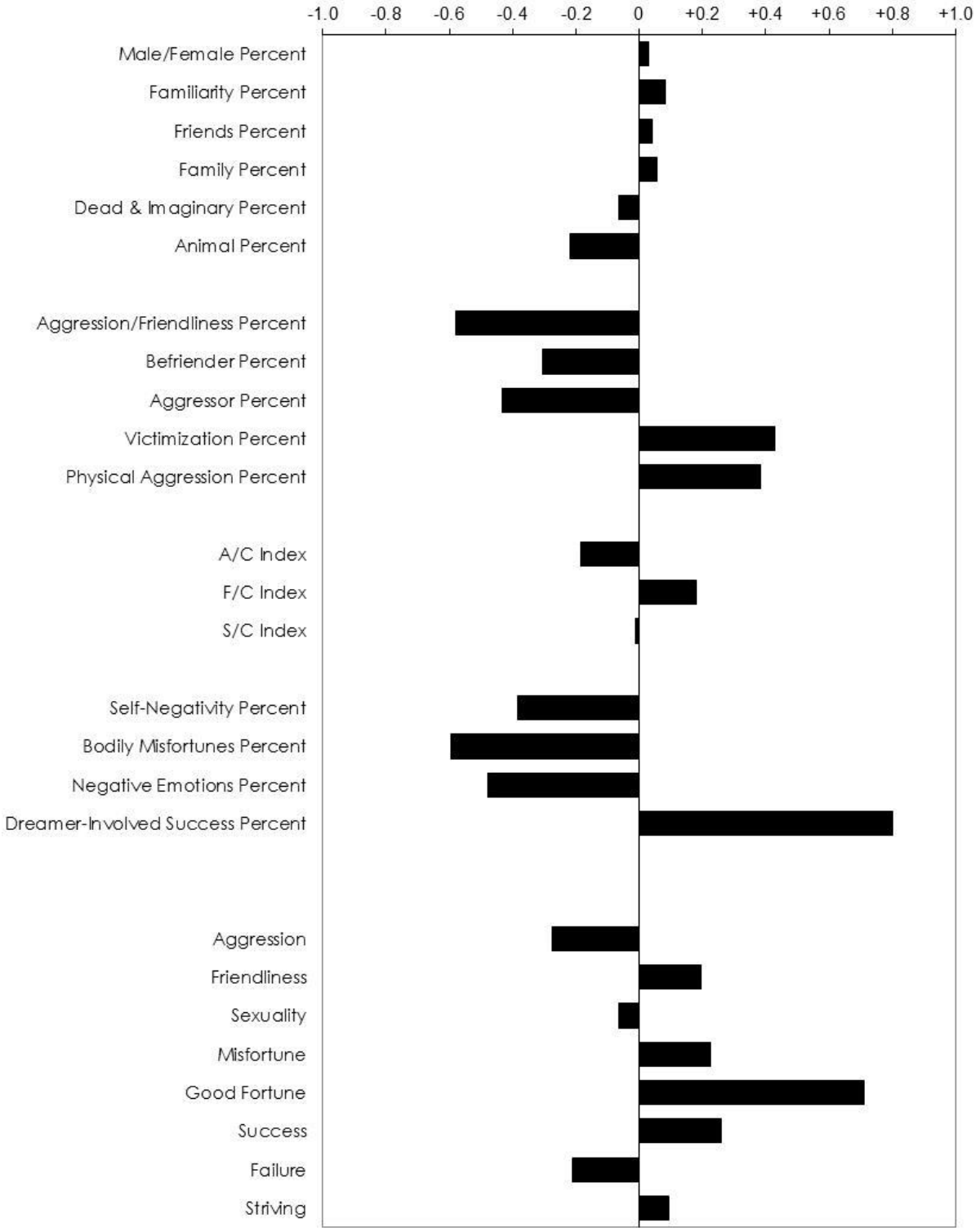

Figure 3. *h*-profile of most recent dream reports of users of oral contraceptives compared to non-users of oral contraceptives, with non-users as the baseline.

A follow-up univariate ANOVA indicated a significant difference on the *DRM realism* item [$F(1, 165) = 5.68$, $p = .018$, $\eta_p^2 = .033$], with OCs users ($M = 3.34$, $SD = 1.10$) scoring higher than OCs non-users ($M = 2.89$, $SD = 1.00$). Follow-up univariate ANOVAs also indicated significant differences for *BD realism* [$F(1, 165) = 4.12$, $p = .044$, $\eta_p^2 = .024$] and *NM realism* [$F(1, 165) = 8.30$, $p = .004$, $\eta_p^2 = .048$]. In both cases, OCs users (*BD realism:* $M = 3.13$, $SD = 1.36$; *NM realism:* $M = 3.21$, $SD = 1.42$) scored higher than non-users (*BD realism:* $M = 2.69$, $SD = 1.12$; *NM realism:* $M = 2.53$, $SD = 1.23$). No significant overall effects were obtained for any of the other DRM-Q components. The results of this analysis are presented in full in Table 24.

DISCUSSION

The relationship of psychosocial stress-related variables to dysphoric dream recall frequency

The present study examined the predictive effect that psychosocial stress may have on bad dream and nightmare recall frequency, whether or not oral contraceptive use might moderate this effect, as well as differences in dream characteristics and content between users and non-users of OCs. Overall, *the hypothesis that psychosocial stress should positively predict both bad dream and nightmare recall frequency was supported by the results of the present study.* As expected, scores on both the CORE Short Form A and Perceived Social Stress scale positively predicted both the number of bad dreams and nightmares experienced by participants over the last month. These findings are in keeping with those reported by previous research which has documented a relationship between stress and dysphoric dream recall frequency (e.g., Banu et al., 2017; Cernovsky, 1984; Cook et al., 1990; Dunn & Barrett, 1988; Hochard et al., 2016; Kroth et al., 2002; Picchioni, 2001; Weinberg et al., 2015).

This second finding is of importance considering that previous studies have examined the relationship between stress and nightmares using scales such as the SRSS (Holmes & Rahe, 1967) (e.g., Cernvosky, 1984; Picchioni, 2001) which measure stress associated with major life events (e.g., divorce, etc.). The results of the present study extend these findings to include more general psychosocial stress that may or may not be associated with major life events.

Table 23.

Descriptive Statistics and t *test Results for Sleep and Dreaming Variables (Study 3)*

	OCs Users		OCs Non-users					
Variable	*n*	*M* (*SD*)	*n*	*M* (*SD*)	*t* (*df*)	*p*	*d*	Effect size *r*
Hours of sleep per night	45	6.87 (0.99)	181	6.66 (*1.30*)	1.01 (*224*)	.315	0.13	.07
Quality of sleep	45	3.87 (*1.04*)	181	3.73 (*0.98*)	0.83 (*224*)	.407	0.11	.06
Awakenings per night	45	1.29 (*1.25*)	181	1.25 (*1.18*)	0.18 (*224*)	.861	0.02	.01
Dreams per week	44	3.23 (*1.70*)	180	2.57 (*1.78*)	2.23 (*222*)	.027	0.30	.15
Dreams per night	45	1.40 (*0.54*)	181	1.28 (*0.52*)	1.36 (*224*)	.177	0.18	.09
Ease of dream recall	45	2.51 (*1.01*)	181	2.97 (*1.10*)	2.53 (*224*)	.012	0.34	.17
Entirety of dream recall	45	2.93 (0*.99*)	181	2.70 (*1.03*)	1.36 (*224*)	.176	0.18	.09
BD Frequency	45	5.13 (*1.36*)	181	4.62 (*1.61*)	1.95 (*224*)	.052	0.26	.13
BDs Last month	45	2.49 (*2.82*)	181	2.18 (*3.07*)	0.62 (*224*)	.536	0.08	.04
BDs Last year	43	12.14 (*11.98*)	177	13.73 (*27.35*)	0.37 (*218*)	.710	0.05	.02
BD Distress	45	4.04 (*1.78*)	181	3.59 (*1.79*)	1.54 (*224*)	.125	0.21	.10
NM Frequency	45	3.64 (*1.87*)	181	2.92 (*1.77*)	2.44 (*224*)	.016	0.33	.16
NMs Last month	39	1.26 (*2.12*)	133	1.05 (*1.55*)	0.66 (*170*)	.511	0.10	.05
NMs Last year	39	6.56 (*8.18*)	132	5.64 (*9.58*)	0.54 (*169*)	.587	0.08	.04
NM Distress	39	4.79 (*1.69*)	133	3.91 (*2.05*)	2.74 (*73.84*)	.008*	0.64	.30
NDQ Total scores	34	31.59 (*8.76*)	111	30.95 (*9.15*)	0.36 (*143*)	.722	0.06	.03

Note. All *t* tests were run as two-tailed.

Results marked with an asterix are results for equal variances not assumed, due to a statistically significant Levene's test for homogeneity of variance.

An alpha level of $p = .003$ was used to control for inflated Type I error due to multiple comparisons.

Table 24.
Summary of Univariate ANOVA Results for Dream Realism *Component*

	OCs Users	OCs Non-users				
Item	*M* (*SD*)	*M* (*SD*)	*F*	*df*	*p*	$\eta_p 2$
DRM realism	3.34 (*1.10*)	2.89 (*1.00*)	5.68	1, 165	.018	.033
BD realism	3.13 (*1.36*)	2.69 (*1.12*)	4.12	1, 165	.044	.024
NM realism	3.21 (*1.42*)	2.53 (*1.23*)	8.30	1, 165	.004	.048

The finding that scores on the GAD-7 positively predicted both bad dream and nightmare recall frequency was expected based on prior research which has documented a relationship between anxiety and both bad dreams and nightmare (see Chapter 3 of this dissertation for a review of this research).

Although Zadra and Donderi (2000) found a relationship between anxiety and bad dreams, but not nightmares, that study examined trait anxiety whereas the present study measured generalized anxiety. As noted in Chapter 3, it may be that certain types of anxiety are associated differently with bad dream and nightmare recall frequency, when the two types of dreams are differentiated from one another.

Turning to positive and negative affect, the negative affect subscale of the PANAS positively predicted both bad dream and nightmare recall frequency. This relationship could be expected based on the fact that prior research has documented a relationship between stress and bad dream and nightmare recall frequency, as well as a relationship between stress and scores on the PANAS (Watson et al., 1988). This finding is similar to that reported in Norlander, Johansson, & Bood (2005), who found that individuals high in both NA and PA reported the greatest frequency of nightmares, and also the greatest amount of stress.

Where the current results differ from Norlander et al. (2005) is that the positive affect subscale scores did not have any effect on dysphoric dream recall frequency. Although the PANAS positive affect scores did predict the frequency of nightmares recalled at Step 2 of the regression analysis, this is most likely due to a suppressor effect rather than an actual effect, as noted in the *results* section above. It therefore appears that although negative affect is associated with an increase in bad dream and nightmare

recall frequency, positive affect does not appear to have a protective effect against experiencing dysphoric dreams. In other words, *an increase in negative affect increases the frequency of bad dreams and nightmares recalled, but an increase in positive affect does not decrease the frequency of bad dreams and nightmares recalled.*

Turning to the results of the effect of different coping strategies on bad dream and nightmare recall frequency, these results indicate that different types of strategies may have different effects on the recall frequency of each dream type. An emotion-focused disengagement style of coping appears to increase the number of bad dreams recalled (i.e., it is a positive predictor). This result is similar to that reported by Low, Dyster-Aas, Willebrand, Kildal, Gerdin, and Ekselius (2003), who found that coping that corresponds to emotion-focused strategies may lead to a greater frequency of nightmares. The present result extends this finding to include bad dreams.

Emotion-focused disengagement did positively predicted nightmare recall frequency at Step 1 of the regression analysis in the present study. It was not a significant predictor at Step 2, however, and the multicollinearity evident between the CSI EFD scores and the interaction variable complicates the issue further. Emotion-focused *engagement* also positively predicted nightmare recall frequency – a result similar to that of Low et al. (2003) – although not bad dream recall frequency.

Why would an emotion-focused coping style be likely to result in an increased frequency of dysphoric dreams being recalled? This style of coping is concerned with regulation of the affective response to stress (Addison et al., 2007). Attempting to manage the affective response to stress may be likely to engage the parts of the brain responsible for affective regulation, which are primarily the brain structures comprising the limbic system (Devinsky & D'Esposito, 2004; Nielsen, 2017a). As reported in Study 1 and as reviewed in Chapter 3 (both of the present dissertation), increased limbic activation is closely associated with an increase in bad dream and nightmare recall frequency. The ventral tegmental area of the brainstem is also involved in mood and emotion, dopaminergically innervates the mesocorticol and mesolimbic structures, and is crucially involved in the generation of all dream types (including bad dreams and nightmares) (Solms, 1997, 2000a).

Greatly engaging the emotion systems of the brain in order to affectively regulate the stress response would thus be likely to trigger dysphoric dreams; the greater the engagement and subsequent activation of these systems, the more frequent and more severe it could be expected these dreams to be. This is in fact the case, as an *emotion-focused engagement* coping style strongly predicted nightmare recall frequency, but not bad dream recall frequency. Total engagement scores also positively predicted nightmare but not bad dream recall frequency. Considering that engagement strategies result in confronting stressors (Addison et al., 2007), it is reasonable to assume that emotionally confronting a stressful situation would result in the greatest activation of the limbic system, and thus a greater frequency of nightmares being recalled. Emotion-focused coping combined with a disengagement strategy appears to have a somewhat protective effect: although emotion-focused coping may result in activation of the affective systems of the brain, the avoidance component of the disengagement strategy (Addison et al., 2007) might attenuate this activation. Thus, dysphoric dreams of lower intensity (i.e., bad dreams) are recalled more frequently compared to dysphoric dreams of a higher intensity (i.e., nightmares) when emotion-focused coping is paired with a disengagement rather than an engagement strategy.

Considering that an emotion-focused coping style positively predicts the frequency of recall of dysphoric dreams, it is not surprising that the opposite – a problem-focused coping style – appears to serve a protective effect. Problem-focused coping emphasizes management of the stressful situation itself, rather than the emotions involved with the situation (Addison et al., 2007). The protective effect was only apparent, however, when problem-focused coping was paired with a disengagement strategy, as problem-focused engagement neither negatively nor positively predicted either bad dream or nightmare recall frequency. Moreover, total disengagement scores did not predict either bad dream or nightmare recall frequency in either direction. Thus, it appears that any engagement of the affective response, either by emotion-focused coping or an engagement strategy, is likely to increase bad dream and/or nightmare recall frequency.

One caveat to be noted regarding the effects of coping strategies is that the different strategies measured in the present study may have different short- and long-term

sequelae. For example, disengagement may produce more favourable short-term results, but can result in less desirable long-term effects (e.g., depression) (Addison et al., 2007). Considering that the present study measured the relationship between coping strategies and bad dream and nightmare recall frequency in the short-term (i.e., over the last month prior to participants completing the study), different relationships may be uncovered when examining the correspondence between coping strategies and dysphoric dream recall frequency over longer periods of time (e.g., 1 year). Such an examination may prove a fruitful avenue of further study.

Oral contraceptive use as a moderator of the relationship between psychosocial stress-related variables and dysphoric dream recall frequency

Turning to the moderating effect of OC use on the relationship between stress and dysphoric dreams, OC use did not have a moderating effect on the correspondence between any of the stress-related variables and either bad dream or nightmare recall frequency measured in the present study. This was despite the findings that a number of stress-related variables predicted bad dream and/or nightmare recall frequency. Although the lack of a moderating effect was not expected given that previous research has discovered an attenuated stress response in OCs users, there are three explanations which may account for this lack of effect.

Firstly, as most previous studies on the stress response in OC users have examined this response following *acute* psychosocial or physical stress (e.g., Bouma et al., 2009; Kirschbaum et al., 1995, 1996, 1999; Hammerfeld et al., 2006; Nielsen, Segal et al., 2013), it may be that the stress response attenuation induced by OCs is evident only after this type of stress. If this is the case, it would explain the lack of a moderating effect detected in the present study considering that it examined more general psychosocial stress. The second possible explanation is simply that there may not be one at all, regardless of the specific type of stress measured. A future study examining the potential moderating effect of OCs on the stress-dysphoric dreams relationship after acute stress induction with, for example, the Trier Social Stress Test would be invaluable in determining which of these two explanations is correct. Finally, it may be that a moderating effect of OC use on the stress-dysphoric dreams relationship is only

evident during certain phases of the menstrual cycle. A future study examining this would be useful.

The association of oral contraceptive use with dream content

The findings were of generally more positive dream content in the most recent dream reports of users of OCs. The aggression/friendliness ratio was lower, and there was less self-negativity, less bodily misfortunes, less negative emotions, and more good fortune in the MRD reports of OC users compared to non-users. Moreover, the aggression per character ratio was lower, and the friendly interactions per character ratio higher, in the MRD reports of OC users.

These findings could all be expected based on previous research which has documented not only a stabilizing effect on mood and affect (Hamstra et al., 2015; Montoya & Bos, 2017), but also occasionally a positive increase in mood and affect (Oinonen & Mazmanian, 2002; Pletzer & Kirschbaum, 2014) in users of OCs. Drosperinone-containing OCs – of which the majority of the present sample (68.8%) were taking – appear to have the greatest positive effects on mood and affect (Sundstrom-Poromaa & Segabladh, 2012). Thus, the more positive dream content of MRD reports from OC users in the present study is in line with the continuity hypothesis of dreaming which posits that dreams reflect waking-life emotions and experience (Schredl & Hoffman, 2003). Although a difference between the two groups was noted for *negative emotions*, as with Study 2 and 3, it should be noted that self-ratings of emotions by dreamers themselves compared to external raters often yield very different findings (Sikka et al., 2014).

Another point of interest in the present study pertains to a lower frequency of aggressive interactions and a higher frequency of friendly interactions in the MRD reports of users of OCs. This may be indicative of such users experiencing a higher percentage of stage 2 NREM sleep (Baker Mitchell, & Driver, 2001), as noted above. McNamara et al. (2010) found that dreamer-initiated friendliness was more characteristic of NREM dream reports, whereas dreamer-initiated aggressiveness was more characteristic of REM dream reports. The hypothesis that the content of the MRD reports in the present study is indicative of a greater percentage of stage 2 sleep in users of OCs is admittedly highly speculative, as sleep stage was not measured in the

present study. Nonetheless, this hypothesis would be easy to test experimentally, and may prove an interesting avenue of further investigation.

The one finding from the dream content analysis which may at first appear perplexing is that the dream reports of users of OCs contained a lower percentage of animals relative to the dream reports of non-users. The explanation for this is that dream reports characterized by a large frequency of animal characters also often contain a large percentage of aggressions. Aggression and animals are often linked in dream content as physical aggression often involves attacks by animals on the dreamer (Domhoff, 1996, 2000). It is thus not surprising that the dream reports from OC users contained a lower percentage of animals compared to the reports from non-users, given that the dream reports of the former group also contained less aggressive interactions than the latter.

The effect of oral contraceptive use on sleep and dreaming variables

Only one statistically significant difference in the sleep and dreaming variables was obtained in the present study: users of OCs scored higher than non-users in terms of self-ratings of the realism of not only their dreams, but also their bad dreams and nightmares. This may be related to findings by certain studies of changes in sleep structure in women taking OCs. Users of OCs have been found to have increased stage 2 NREM sleep (Baker et al., 2001), and more realistic and thought-like dreams are often (though not always) found to be more characteristic of NREM compared to REM sleep (Mamelak & Hobson, 1989; Solms, 2000a; Nielsen, 2000).

In addition to the statistically significant effect mentioned above, a number of effects trended towards statistical significance. Trends for users of OCs to experience more dreams per week, more nightmares, more nightmare distress, and a lower ease of dream recall were evident. These trends are briefly discussed as they may have reached statistical significance if the sample size of OC users was larger, and if the two groups were more evenly matched in terms of sample size.

The reduced ease of dream recall in users of OCs may be related to the tendency of OCs to increase levels of serotonin as a result of estrogen-mediated inhibition of the monoamine oxidase pathway (Egarter, Topcuoglu, Imhof, & Huber, 1999; Oinonen &

Mazmanian, 2002), and increases in serotonin are known to impair memory (Buhot, Martin, & Segu, 2000; Luciana, Collins, & Depue, 1998). The increased levels of serotonin in users of OCs are also most likely to explain the increase in positive affect in these individuals that some studies find (Oinonen & Mazmanian, 2002; Pletzer & Kirschbaum, 2014). The question remains, however, as to why trends were observed towards OC users having more dreams per week, more nightmares, and more nightmare distress, as such findings seem incompatible with the hypothesized increased levels of serotonin in users of OCs. There are two most likely explanations for this.

First, the increased dream and nightmare recall frequency may be due to a dopamine rebound effect during the 'off'/placebo week in users of OCs, thus increasing dream and nightmare recall frequency during this week. It is known that serotonin and dopamine interact at a neurophysiological level, with the serotonergic system having strong anatomical and functional interactions with the dopaminergic system (Seo, Patrick & Kennealy, 2008), and studies have indicated that frontal dopamine activity is inhibited by the serotonergic system (Millan, Dekeyne & Gobert, 1998). Serotonin withdrawal is also known to result in intense visual dreaming and nightmares (Pagel & Helfter, 2003). The fact that some studies have found less negative affect during the menses period in users of OCs (Parsey & Pong, 2000) is evidence against a withdrawal hypothesis, however.

The second possible explanation is that the trend towards increased dream and nightmare recall frequency observed in users of OCs is due to the ability of OCs to increase not only serotonin, but also dopamine (Fink, Sumner, Rosie, Grace, & Quinn, 1996). Increased levels of dopamine are associated not only with an increase in the dream recall frequency, but also an increase in nightmare recall frequency (Solms, 2000a). The finding of a trend towards OC users experiencing more distress from their nightmares can be explained by the fact that individuals experiencing frequent nightmares are often more affected by these nightmares (Levin, 1994).

CONCLUSIONS

Overall, a number of stress-related variables were found to positively predict bad dream and nightmare recall frequency, as expected by previous research as well as by models on bad dream and nightmare generation (Nielsen, 2017a, 2017b; Levin & Nielsen, 2007; Nielsen & Levin, 2007). OC use did not moderate the relationship between the stress-related variables and dysphoric dream recall frequency, however, which was contrary to expectations.

The findings from the dream content analysis are largely in line with previous research which has indicated that users of OCs experience stabilized and/or increased mood (Pletzer & Kirschbaum, 2014), particularly in users of dropsirenone-containing OCs (Brown et al., 2002; Porooma & Segebladh, 2002; Wichianpitaya & Taneepanichskul, 2013). This increased and/or stabilized mood might possibly be linked to increased levels of serotonin (Pletzer & Kirschbaum, 2014). The findings of generally more positive dream content in the OCs users group is, however, not in line with research documenting an increase in levels of dopamine in users of OCs (e.g., Fink et al., 1996). As noted in Chapters 3, 4, and 5 of this dissertation, increased levels of dopamine are usually associated with more aggressive and negative dream content, and also a greater frequency of nightmares recalled (Solms, 2000a). A trend towards a greater frequency of nightmares being recalled in the OC users group was also noted in the present study, which would be expected if OCs do indeed result in increased levels of dopamine (as noted above). This is a finding which is again contradictory to the findings of generally more positive dream content, however, as frightening dream content is often characteristic of nightmares.

How might these contradictions be explained? One possible explanation is that although users of OCs might be prone to more nightmares, the dream content of their non-nightmare dreams is much more positive than the dream content of the non-nightmare dreams of non-users of OCs. Even though users of OCs might be prone to more nightmares than non-users, it is likely that nightmares would still comprise the minority of dreams experienced by either group. Even 'frequent' nightmare sufferers experience on average as few as one nightmare per month (Levin & Fireman, 2002a, 2002b), and Robert and Zadra (2014) found that only 3% of prospectively-collected dream reports

were from nightmares, with approximately 10% being bad dreams. An alternative explanation may simply be that although the nightmares of users of OCs are characterized by negative content, as might be expected, this content is less negative than that experienced by non-users of OCs. Due to the exploratory nature of the present study it is difficult to say for certain which explanation(s) put forward here are correct. More research on dreaming in users of OCs is clearly needed to elucidate the mechanisms involved in the altered dream phenomenology reported by such users in the present study.

CHAPTER 7

SUMMARY AND CONCLUSIONS

INTRODUCTION

The overall aim of this thesis and the studies reported herein was to add to the existing body of work regarding the etiological and conceptual explanations for bad dream and nightmare formation, particularly regarding non-trauma-related nightmares. Despite the fact that bad dreams and nightmares occur with some regularity in the general population (e.g., Li et al., 2010; Nielsen, 2017a; Otsuka et al., 2018; Robert & Zadra, 2014; Sandman et al., 2013; Schredl, 2010), research on such dreams and their genesis and etiology is more prevalent in clinical populations compared to the former population.

Given the above, three studies were performed with the specific aim of investigating the neuropsychological mechanisms of idiopathic bad dream and nightmare generation. A particular focus was given to the role that the limbic system plays in this regard, due to previous research on clinical and non-clinical samples indicating a crucial involvement of this brain region to the generation of all types of dreams. Using a set of questionnaires, Study 1 investigated differences on a number of dream, bad dream and nightmare-related variables between individuals from a non-clinical sample with either high or low levels of limbic activation. The effect that higher versus lower levels of limbic activation has on most recent dream content was also investigated in Study 1. Building on the results of that study, a prospective dream diary studied was carried out for Study 2, which sought to more closely examine the relationship between levels of limbic activation and dream content.

Having examined limbic activation as an internal factor in Studies 1 and 2, Study 3 examined a common external factor affecting levels of limbic activation – psychosocial stress – and its effect on bad dream and nightmare recall frequency. Oral contraceptive (OC) use was investigated as a potential moderator, due to previous research indicating not only that the use of OCs may attenuate the stress response, but also that oral contraceptives affect certain structures in the limbic system. Given the effect of OCs on

the limbic system, as well as their potential ability to regulate mood across the menstrual cycle (Hamstra et al., 2015; Montoya & Bos, 2017), the effect of OC use on dream content was also investigated.

The three studies yielded the following main findings:

(a) Individuals with higher levels of limbic activation were prone to recall more dreams in general, including normal dreams, compared to individuals with lower levels of limbic activation.
(b) Individuals with higher levels of limbic activation were prone to recalling more bad dreams and nightmares compared to individuals with lower levels of limbic activation. Related to this was the finding that sleep is more generally disturbed in individuals with higher levels of limbic activation.
(c) Individuals with higher levels of limbic activation experienced generally more negative dream content than individuals with lower levels of limbic activation. Some instances of individuals with higher levels of limbic activation experiencing more positive dream content were also found, which could be predicted from previous research on individuals with clinical conditions affecting the limbic system.
(d) Related to Finding (c) above is the finding that acute psychosocial stress – which may increase levels of limbic activation – positively predicted both bad dream and nightmare recall frequency, although oral contraceptive use did not moderate this relationship.
(e) Oral contraceptive use was associated with dreams with generally more positive content and less aggressive interactions, as could be predicted by the stabilizing effect on mood that oral contraceptives may have.

These findings are discussed in turn below.

LIMBIC SYSTEM ACTIVATION AND ITS RELATIONSHIP TO DREAM RECALL FREQUENCY

Given that the limbic system has been implicated in the generation of dreaming by both neuroimaging (Cipolli et al., 2017) and lesion studies (Solms, 1997, 2000a), it is reasonable to suppose that increased levels of limbic activation would result in an

increased frequency of dreams being recalled. This prediction was supported by the results of the first two studies. Study 1 – which investigated differences in dream variables between individuals with high versus low levels of limbic activation – found that individuals with higher levels of limbic activation reported a greater number of dreams per night and per week. In the prospective dream diary study (Study 2), it was found that participants with higher levels of limbic activation recalled not only a greater frequency of dreams of *all* types, but also a greater frequency of normal dreams, and a greater number of nights with at least one dream of any type. In fact, participants with greater limbic activity recalled approximately double the number of normal dreams and dreams of all types over the study period.

These findings support the contention that nightmares are a more severe expression of normal dream phenomena (Nielsen & Levin, 2007; Zadra & Donderi, 2000), and that bad dreams and nightmares may be generated by the same processes that subsume the generation of normal dreams. The present set of findings thus adds to the existing body of research that has found a greater recall of normal dreams in individuals prone to experiencing more frequent nightmares. Levin and Fireman (2002b), for example, found that individuals who reported more frequent nightmares also reported more non-nightmare dreams in a prospective dream diary study over 21 days.

Greater recall of normal dreams in individuals with clinical conditions affecting the limbic system such as TLE has also been found (e.g., Bonanni et al., 2002), along with the corollary that suppression of limbic activation may result in decreased recall of dreams. As an example of the latter, increased prefrontal and anterior cingulate activation in patients with PTSD of the dissociative subtype may result in excessive inhibition of the amygdala, and a concomitant reduction in dream recall (Kaminer & Lavie, 1991). Antiepilectic drugs which suppress limbic activity have also been known to reduce not only the frequency of nightmares recalled but also normal dreams in patients with temporal lobe epilepsy (Silvestri & Bromfield, 2004; Solms, 1997).

The findings from the present studies that individuals with higher levels of limbic activation experience greater recall of normal dreams as well as bad dreams and nightmares are significant given that nightmares have been conceptualized as

pathological dreaming. In other words, nightmares have been thought to have been caused by processes different to those that generate normal dreams, and are thus due to normal dreaming processes gone awry. The results of the studies presented in this thesis lend more credence to a view of dreaming which sees nightmares as "…an intensification of the normative dreaming process" (Levin & Fireman, 2002a, p. 210). The implication of these results is that any theory of dream generation must therefore also account for how bad dreams and nightmares are generated, and likewise, any theory of nightmare generation must also account for the generation of normal dreams.

Three theories of dreaming presented in Chapter 3 do in fact present such an account, with Hartmann's theory of boundary permeability being one such model. Schredl et al. (1999), for example, found that individuals with thin boundaries not only recalled nightmares more often, but normal dreams as well. Solms' theory (Solms, 1997, 2000a, 2000b) is another such model. According to this theory, dopaminergic innervation of mesocortical and mesolimbic areas is said to trigger dreaming in the context of nonspecific cerebral activation. Any greater than average activation of the limbic system would thus, according to this theory, trigger not only more frequent dreaming but also more frequent nightmares.

Finally, Levin and Nielsen's (Levin & Nielsen, 2007, 2009; Nielsen & Levin, 2007, 2009) Affect Network Distress model views different types of dreams as being on a continuum of severity, with normal dreaming at one end and replicative posttraumatic nightmares on the other, and bad dreams and idiopathic nightmares in between. The limbic system and associated brain structures are seen as playing a crucial role in influencing this continuum through the effect that affect load (situational factors) and/or affect distress (dispositional factors) have. Given that the different dream types are seen as being on a continuum in this model, increases in limbic activity as a result of either of these affect factors would result not only in an increased frequency of bad dreams and nightmares being recalled, but normal dreams as well. The findings in Study 3 (present thesis) that stress predicts both bad dream and nightmare recall frequency also fit with the contention of the AND model that high affect load should increase recall of bad dreams and nightmares (Levin et al., 2011; Nielsen & Levin, 2007; Tunbridge & Weinberg, 2014)

LIMBIC SYSTEM ACTIVATION AND ITS RELATIONSHIP TO BAD DREAM AND NIGHTMARE RECALL FREQUENCY

In addition to increased recall of normal dreams, the results of the studies presented in this dissertation indicate that increased levels of limbic activation are also associated with increased recall of bad dreams and nightmares. In Study 1, individuals with greater compared to lower levels of limbic activation reported recalling both more bad dreams and more nightmares. This was the case across a variety of questionnaire measures: not only did individuals with greater limbic activity report recalling more bad dreams and nightmares in general on an 8-point rating scale, but also more of both dream types in the last month and in the last year on a raw frequency measure. They also indicated experiencing more distress as a result of their nightmares.

Individuals with higher levels of limbic activity reported recalling approximately 1.5 times as many bad dreams and 5 times as many nightmares compared to individuals with lower levels of limbic activity in Study 2, a prospective dream diary study. It should be noted, however, that no statistically significant differences in bad dream and nightmare frequency between individuals with high and low levels of limbic activity were found in this study, and that the sample sizes were small. Nonetheless, the trend discovered is in keeping with the findings from Study 1, which appear to be robust, and the lack of statistically significant differences may be due to a lack of statistical power from insufficient sample sizes and/or an insufficient time period over which participants were asked to keep their dream diaries.

The above-mentioned findings are in keeping with previous research on various clinical disorders reviewed in Chapter 3 which indicates a strong link between limbic activation and bad dream and nightmare recall frequency. As with the finding discussed above of a relationship between limbic activation and an increased frequency of dreams recalled, the present set of findings illustrates that this link applies not only in clinical disorders, but in normal individuals as well. A number of the dream theories discussed in Chapter 2 predict the association between limbic activation and an increased recall frequency of bad dreams and nightmares.

Firstly, the Affect Network Distress model of Levin and Nielsen (Levin & Nielsen, 2007, 2009; Levin et al., 2010; Nielsen, 2017a; Nielsen & Levin, 2007, 2009) directly implicates the amygdala, medial prefrontal cortex, hippocampus, and anterior cingulate cortex in the generation of not only normal dreams, but bad dreams and nightmares. Secondly, in that boundary thinness may be related not only to increased levels of limbic activation (see Chapter 2) but also to increased bad dream and nightmare recall frequency, Hartmann's theory of boundary permeability may account for the link between these two phenomena. A number of the dream content findings (discussed below) also support a link between boundary permeability and limbic activation. Finally, Solms's (1997, 2000a) theory that temporal limbic regions of the brain – innervated by dopamine – provide the affective arousal necessary for dreaming would predict that increased affective arousal as a result of increased levels of limbic activation should result in an increase in bad dream and nightmare frequency, as discovered in the present studies.

Related to the increased frequency of bad dreams and nightmares recalled by individuals with higher levels of limbic activation is that such individuals also appear to have more disturbed sleep in general. In Study 1, individuals with greater levels of limbic activation reported having lower quality of sleep, and also waking up more times per night. Considering that such individuals also reported a greater frequency of nightmares, and that by definition nightmares were characterized in the present dissertation as disturbing dreams that awaken the sleeper (see Chapter 1), this finding is not surprising. Previous studies on clinical conditions marked by limbic hyperactivity such as migraine (Bruni et al., 1999), schizophrenia-spectrum disorders (Koffel & Watson, 2009), anxiety (Celluci & Lawrence, 1978), and PTSD (Krakow et al., 2001) have also discovered more disturbed sleep in patients with these disorders.

In addition to more disturbed sleep, individuals in the present study with greater levels of limbic activity reported more distress from their bad dreams and nightmares; being more affected by their dreams, bad dreams and nightmares; and also finding all three dream types more meaningful. Taken together, these findings indicate that waking-life sequelae of dreams, bad dreams, and nightmares are manifested to a greater degree in individuals with higher compared to lower levels of limbic activity.

According to Levin and Nielsen's AND model, frequent nightmare sufferers experience more distress both during and after a nightmare as they are higher on the trait factor of affect distress (Levin & Nielsen, 2007, 2009). Affect distress is also associated with boundary thinness, which has in turn been related to experiencing dreams as more meaningful. Considering that both high affect distress and boundary thinness may be related to higher levels of limbic activity (see Chapter 4), it is not surprising that participants in the present study with such levels of limbic activity rated being more affected by all three dream types; finding them more meaningful; as well as experiencing more distress from their bad dreams and nightmares. The significance of the present findings is that higher levels of limbic activity may not only result in dreamers experiencing more waking-life sequelae of dreaming, but also that these sequelae may possibly extend to all dream types in individuals with high levels of limbic activation.

LIMBIC SYSTEM ACTIVATION AND ITS RELATIONSHIP TO DREAM CONTENT

Overall, greater amounts of aggression as well as more negative dream content were discovered in individuals with higher levels of limbic activation, across a variety of measures and studies. In Study 1, individuals with greater amounts of limbic activation reported more aggression directed at themselves in both bad dreams and nightmares. It was also reported that the dreamer was more aggressive towards others, although only in nightmares.

Analysis of dream reports from Study 2 – a prospective dream diary study – using the Hall and Van de Castle method confirmed a greater percentage of aggression in the most recent dreams of individuals with higher levels of limbic activation, as well as that the dreamer was the victim (i.e., more aggression directed at themselves by others) with greater frequency. A higher ratio of aggression-to-friendliness and dreams with at least one instance of aggression was also noted. Greater instances of misfortune, less familiar characters, less family members, and more dead and imaginary characters in the dreams of individuals with higher levels of limbic activation again confirmed the overall more negative content of their dreams. More overall negative dream themes were also

reported on the Typical Dreams Questionnaire (TDQ) in Study 1 (specifically: more *presence*, *inhibition*, *loss of control*, *death-murder*, and *negative experiences*), and the group with higher levels of limbic activation also scored higher on the bad dream and nightmare sub-scales of this questionnaire suggested by Nielsen et al. (2003).

All of the above-mentioned findings overlap considerably with the results of previous research on dream content in individuals with clinical conditions affecting the limbic system (e.g., PTSD; anxiety disorders; schizophrenia; etc.). Of note is that no differences in dream content regarding emotions were reported between the groups with high versus low levels of limbic activation, in either Study 1 or Study 2. This may be due to the fact that the majority of the dreams comprised normal dreams, in which intense emotions may not be experienced; or that the emotion categories in the Hall and Van de Castle coding system are not sensitive enough to detect differences between the two groups studied in this dissertation.

Of interest in the present thesis is that a number of instances of what could be considered more positive dream content were found for individuals with higher levels of limbic activation. In Study 2, a greater frequency of sexuality was found in the dreams of individuals with higher levels of limbic activation and this same finding approached statistical significance in Study 1. This could be expected based not only on the involvement of the limbic system in sexual function (Argiolas & Melis, 2003), but also based on the involvement of dopamine in both sexual function and dream generation (Argiolas & Melis, 2003; Solms, 2000a).

A number of more positive dream themes for individuals with higher levels of limbic activation were also found on the TDQ in Study 1. Specifically, such individuals were more likely to experience themes of discovering a new room and being a child again, and they also scored higher on the *positive experiences* component. These findings raise the possibility that increased limbic activation is associated not only with more negative dream experiences, but also more positive dream experiences than usual, at least on occasion. Previous research has hinted at this latter possibility in that individuals with Schizotypal Personality Disorder – a disorder affecting the limbic system (see Chapter 3) – have been found to experience more positive and enjoyable dreaming (Claridge et

al., 1997). Individuals with limbic hyperactivity from temporal lobe epilepsy may also experience elation during the ictal period (Devinsky & D'Esposito, 2004). Carr et al. (2016) found that frequent nightmare sufferers are also more likely to experience dreams marked by positive affect, and Robert and Zadra (2014) found that a small-to-moderate percentage of bad dream and nightmare narratives contained positive or partially positive outcomes. There is also the possibility that these positive dream themes are related to approach/seeking behaviour in dreams, triggered by the dopamine system which innervates the mPFC, an area of the brain highly interconnected with the temporal lobe (Levin & Nielsen, 2007; Malcolm-Smith et al., 2012; Perogamvros et al., 2015; Solms, 1997, 2000a; Wager et al., 2003).

More frequent themes of alien life and religious experiences such as encountering God in some form and/or seeing an angel were also found in individuals with higher levels of limbic activation. This again fits with previous research on individuals with clinical conditions resulting in hyperactivation of the limbic system. For example, patients with TLE and/or schizophrenic symptoms are more likely to experience heightened religiosity (Bear & Fedio, 1977; Devinsky & Lai, 2008; Dewhurst & Beard, 1970; Trimble & Freeman, 2006; Waxman and Geschwind, 1975). Taken together, the combination of negative and positive dream content – as well as content such as alien life and religiosity – appears to indicate that the dream life of individuals with high levels of limbic activation may be more nuanced and complicated than previously thought.

PSYCHOSOCIAL STRESS AND ITS RELATIONSHIP TO BAD DREAM AND NIGHTMARE RECALL FREQUENCY

Whereas Study 1 and 2 sought to directly investigate the effect that levels of limbic system activation have on dreaming, bad dreams, and nightmares, Study 3 investigated a common phenomenon that is known to increase levels of limbic activation – psychosocial stress. This study was performed as the limbic system is closely involved in the stress response, particularly via the bed nucleus of the stria terminalis, the hippocampus, amygdala, and the prelimbic divisions of the anterior cingulate (Cook, 2002; Herman & Cullinan, 1997; Herman et al., 2005, 2012).

The same stress variables were found to positively predict both bad dream and nightmare recall frequency: feelings related to stress and tension; perceived social stress (i.e., the degree to which situations are appraised as stressful); tendencies to experience generalized anxiety; and negative affect. Although previous studies have reported relationships between stress and nightmare recall frequency (e.g., Banu et al., 2017; Cernovsky, 1984; Cook et al., 1990; Dunn & Barrett, 1988; Kroth et al., 2002; Picchioni, 2001; Weinberg et al., 2016), the present study extends these findings not only by demonstrating that stress may predict bad dream recall frequency as well, but also that generalized stress – in addition to stress from major life events – may predict both bad dream and nightmare recall frequency.

A unique finding from Study 3 is the effect that different coping mechanisms may have on the relationship between stress and bad dream and nightmare recall frequency. An emotion-focused coping style, concerned with the regulation of the affective response to stress (Addison et al., 2007), was found to positively predict dysphoric dream recall frequency. This again provides evidence that engagement and subsequent activation of the limbic system is related to bad dream and nightmare recall frequency.

At a more specific level, emotion-focused disengagement predicted bad dream but not nightmare recall frequency; and emotion-focused engagement predicted nightmare but not bad dream recall frequency. Taken together, these two results indicate that emotion-focused coping may activate the limbic system and produce dysphoric dreams as a result. The disengagement sub-type (i.e., emotionally avoiding the stressful situation) appears to result in less intense dysphoric dreams in the form of bad dreams, whereas the engagement sub-type (i.e., emotionally confronting the stressful situation) appears to result in more intense dysphoric dreams in the form of nightmares.

This could be expected from Levin and Nielsen's AND model (Levin & Nielsen, 2007; Nielsen & Levin 2007), in which greater engagement of the limbic system would increase the intensity of dysphoric dreaming. The corollary was also noted in the results of Study 3. Specifically, a problem-focused coping style which emphasizes management of the stressful situation and de-emphasizes the emotions involved appears to have a protective effect, as a problem-focused coping style negatively predicted both bad

dream and nightmare recall frequency, although only when paired with the disengagement type of coping. As noted in Chapter 6, it thus appears that any engagement of the affective response, either by emotion-focused coping or an engagement strategy, is likely to increase bad dream and/or nightmare frequency. Considering the relationship between the limbic system, affective responses, and bad dream and nightmare generation, this result is to be expected.

THE EFFECT OF ORAL CONTRACEPTIVE USE ON DREAMING AND DREAM CONTENT

Although oral contraceptive use did not moderate the relationship between stress and bad dream and nightmare recall frequency, it does appear that the use of OCs affects dreaming and dream content. In Study 3, OC users rated their dreams, bad dreams, and nightmares as more realistic compared to non-users. Although it is not possible to identify with certainty the reason as to why, it may be due to OC users having increased Stage 2 NREM sleep (Baker et al., 2001), during which more realistic and thought-like dreams often occur (Mamelak & Hobson, 1989; Solms, 2000a; Nielsen, 2000). Statistically significant trends towards a reduced ease of dream recall, more dreams recalled per week, more nightmares being recalled, and more nightmare distress were also found for users of OCs compared to non-users. The reduced ease of dream recall may be related to impaired memory from increased levels of serotonin in users of OCs (Buhot et al., 2000; Luciana et al., 1998; Pletzer & Kirschbaum, 2014), as a result of estrogen-mediated inhibition of the monoamine oxidase pathway (Egarter, 1999; Oinonen & Mazmanian, 2002). The most likely explanation for the trend towards more dreams per week, more nightmares, and more nightmare distress is due to the tendency of OCs to increase not only serotonin levels, but dopamine levels as well (Fink et al., 1996). Increased levels of dopamine have consistently been documented to result in an increased frequency of dreaming and nightmares (Solms, 2000a), as well as more distress being experienced from nightmares (Levin, 1994).

Turning to dream content, more generally positive dream content was found in users of oral contraceptives compared to non-users. Compared to non-users, the dreams of OC users contained a lower aggression/friendliness ratio; less self-negativity; less bodily

misfortunes; less negative emotions; a greater percentage of dreams with at least one instance of good fortune; and a trend towards more dreamer-involved success. Fewer animals were also found to appear in the dreams of OC users compared to non-users, which could be expected based on that fact that physical aggression often involves attacks by animals on the dreamer (Domhoff, 1996, 2000b). A lower aggression/friendliness ratio and fewer animals in dreams may thus co-occur, as they appear to have done in this study.

The precise reasons as to why users of OCs have more generally positive content compared to non-users remain to be elucidated, although these findings are in keeping with previous research that has found increased levels of serotonin in users of OCs, as well as increased and/or more stabilized mood. Less variability in negative affect and more positive affect across the menstrual cycle in OC users may thus be expressed as more positive dream content in general. Although trends towards more nightmares being recalled and more nightmare distress in users of OCs were noted in Study 3, nightmares may comprise the minority of their dreams; may comprise less negative content compared to the nightmares of non-OC users; or the content of their non-nightmare dreams may be much more positive than the dream content of the non-nightmare dreams of non-OC users. More detailed research in future would help to more closely delineate which of these factors, or the interactions between them, are responsible for the co-occurring trends of more nightmares together with more positive dream content in users of OCs.

LIMITATIONS OF THE PRESENT STUDIES AND SUGGESTIONS FOR FUTURE RESEARCH

Although most findings from the present set of studies are in line with and could be expected based on previous studies, a number of limitations are evident. The limitations for each of the three studies presented in this dissertation are discussed in turn, together with potential future studies arising from the results of the present studies.

Limitations regarding extreme group analysis (EGA) apply to both Study 1 and Study 2. One of these limitations is the tendency of EGA to inflate effect sizes (Preacher et al.,

2005; Preacher, 2015). Effect sizes reported in Studies 1 and 2 should therefore be interpreted with caution. A second limitation of EGA concerns reduced reliability, particularly test-retest reliability (Preacher et al., 2005; Preacher, 2015), although the LSCL-33 was only administered on one occasion here, and demonstrated adequate internal consistency reliability. A third limitation of EGA involves not being able to explore nonlinear relationships (Preacher et al., 2005; Preacher, 2015), although the majority of research regarding the effect of limbic activation on sleep and dreaming variables – including dysphoric dream frequency – points to a purely linear relationship (Nielsen & Levin, 2007). Preacher et al. (2005) and Preacher (2015) also note issues with group assignment, specifically that two underlying categories might not exist, that group assignment might not be accurate, that the proportion of cases in each group might not be equal in the population, and that group constituency might not be stable over samples, contexts, and occasions. The similarity of LSCL-33 cut-off scores for high and low groups in the present studies with those found in both American and Turkish samples by Aycicegi-Dunn et al. (2008) do indicate, however, that group constituency across samples, contexts, and occasions is relatively accurate for the LSCL-33, as well as that two underlying categories might in fact exist, and also that group assignment is relatively accurate.

A second limitation of Study 1 is that level of limbic activation was assessed indirectly using a questionnaire – the LSCL-33. This raises the problem of trying to infer one behavioral level from another. The same issue applies to Study 3, where stress was also measured using questionnaires. Nonetheless, the LSCL-33 has demonstrated good reliability and validity, as noted in Chapter 4 of the present thesis. The stress scales used have also demonstrated good reliability and validity, as noted in Chapter 6.

The greatest limitation from Study 1 is probably that the content analysis was done using only dream reports from females, due to an inadequate sample size of reports from males. The lack of dream reports from males could be due to the fact that not only is dream recall frequency usually higher in females, but also that females – particularly college students – are more interested in dreaming in general (Giambra, Jung, & Grodsky, 1996; Schredl & Piel, 2003, 2008). It was not possible to collapse the data across males and females together – as was done with the quantitative survey data –

due to the fact that there are different norms for males and females regarding the Hall and Van de Castle coding categories (Domhoff, 1996).

Schredl and Piel (2008) found that gender differences in interest in dream interpretation disappear after age 30. A future study on the effect that limbic system activation has on dream content in males and females using a sample with a much wider age range may thus yield more data from males, and in turn allow more generalizations on the relationship between limbic activation and dream content to be made for both genders. Increasing the number of dream reports in total is also needed to address another limitation of Study 1, which is that the sample sizes were slightly below those recommended by Domhoff (1996).

Nonetheless, the results from the dream content analysis were in line with what could be expected based on prior research, and were also in line with the results of the quantitative survey data, indicating that the findings are relatively robust. Future studies on limbic activation and dream content should also distinguish whether the dream reports are from normal dreams, bad dreams, or nightmares, thus allowing a subtler examination of the way in which differing levels of limbic activation impact dream content.

A further limitation that applies to the dream content analysis in Study 1 – and also Study 2 and 3 – is that only percentage of overall agreement was computed for inter-rater reliability. The exact percentage of agreement for each content category is therefore lacking, and agreement for some content categories may have diverged from the overall percentage of agreement. Another limitation pertaining to the coding is that the principal investigator, although blind to which quartile the dream reports came from, knew that the reports could only have come from either the upper or lower quartiles. This may have influenced the coding, although the rate of inter-rater reliability was still high.

One potential future study arising from the results of Study 1 involves the relationship between boundary thinness and limbic activation. In Study 1, it was found that individuals with higher levels of limbic activation rated their normal dreams, and also

bad dreams and nightmares, as more meaningful compared to individuals with lower levels of limbic activation. In Hartmann's theory of boundary thinness (Hartmann, 1989, 1991), individuals with thin boundaries have also been found to value their dreams more and find them more meaningful. Thin boundaries have further been found to be associated with affect distress – which is in turn associated with limbic hyperactivity (Levin & Nielsen, 2007) – as well as schizophrenia-spectrum disorders (SSDs). SSDs have in turn been found to be associated with increased limbic activity and also greater recall frequency of dysphoric dreams. It is thus reasonable to assume that boundary thinness and limbic activity are related and in turn influence dream, bad dream, and nightmare recall frequency, but a study explicitly testing this hypothesis would be valuable.

Similar to the above would be a study investigating the exact contributions that variables such as neuroticism, anxiety, depression, and so on make to level of limbic activation, and how this in turn is associated with sleep and dreaming variables and bad dream and nightmare recall frequency. Path analysis or structural equation modelling would be particularly useful here in investigating the relative contributions and exact associations.

Another interesting potential future study comes from the findings in Study 1 of more positive dream content, and also themes of alien life and religiosity, in individuals with higher compared to lower levels of limbic activation. These findings indicate that high levels of limbic activation may not lead exclusively to an increase in negative dreams and dream content, or at least that the dream life of individuals with higher levels of limbic activation may be more nuanced than might be expected based on previous research. A future study more closely examining positive dream content in individuals with high levels of limbic activation and/or frequent nightmare sufferers may prove a fruitful avenue of further investigation.

Many of the same limitations and suggestions for future studies as discussed here apply also to Study 2, the dream diary study. Specifically, a lack of data from males, lower-than-recommended sample sizes, and a limited age range of the participants. In future, requiring participants to keep dream diaries for a longer period of time would help to

increase dream report sample sizes, although this may lead to lower compliance and completion rates among participants. Directly comparing the content of normal dreams, bad dreams and nightmares between individuals with high versus low levels of limbic activation in a future dream diary study may yield potentially interesting results regarding how these two sample sub-types differ on dream content indicators within the same dream category.

Turning to Study 3, one limitation is that stress and coping strategies were measured only in the short-term. As noted in Chapter 6 of the present dissertation, different coping strategies may have different short- and long-term sequelae, for example, a disengagement strategy may result in less desirable long-term effects (e.g., depression) although it produces good short-term effects (Addison et al., 2007). A future study investigating the relationship between different coping strategies and their effects on dysphoric dream recall frequency in the short- compared to long-term (e.g., 1 year) might yield interesting results.

Further, regarding the relationship between stress and dreaming, Study 3 did not find any moderating effect of OC use on this relationship, although an effect was predicted. This may be due to the fact that no such effect exists, or it may be that such an effect exists only when individuals experience acute psychosocial stress. A future study examining whether such a moderating effect exists after induced acute psychosocial stress using, for example, the Trier Social Stress Test would provide valuable evidence towards which one of these hypotheses are correct.

Turning to the dream report data collected as part of Study 3, a future study collecting dream data over a longer period of time would allow a potential examination of dream content in the 'off'/placebo week of oral contraceptive use, and how this may differ from dream content at other points in the menstrual cycle in users of OCs. Adding a control group of males in this future study would address the lack of such a control group in the present study. The dream data collected in Study 3 also did not ask participants to rate whether the dreams were normal dreams, bad dreams, or nightmares. A future dream diary study investigating this would yield valuable data regarding how many bad dreams and nightmares users of OCs have compared to non-users.

Another interesting potential future study concerns the hypothesis that the content of the MRD reports in the present study is indicative of a greater percentage of stage 2 sleep in users of OCs (see Chapter 6). Although at present a speculative hypothesis, an experimental study measuring sleep stage and its relationship to dream content in users of OCs may provide potentially interesting data in future. Examining any differences in sleep and dreaming that different preparations of OCs might have would also be an interesting future study. As Montoya and Bos (2017) note "…different neural and behavioral effects can be expected on the basis of the type of progestin in the OC, and is an important, yet often ignored factor, to take into account in studies" (p. 126).

FINAL CONCLUSIONS

The results of the three studies presented in this thesis not only largely confirm, but also extend, the findings of previous research regarding limbic system activation and its relationship to dream, bad dream and nightmare genesis, as well as dream content. Higher limbic activation results in a greater recall of dreaming, bad dreams, and nightmares, and is also associated with more negative dream content. External, situational factors such as psychosocial stress which trigger limbic activation may also be associated with a greater recall of bad dreams and nightmares. Interesting findings concerning the potential for increased levels of limbic activation to occasionally result in more positive dream content were also found, raising the possibility that the relationship between the limbic system and dream content may be more complicated than previously thought. Given the effect that oral contraceptive use appears to have on dream content, the role of hormonal factors in the limbic system and their subsequent effect on dream content also appears to be a promising avenue of further investigation. Although dreaming has been studied extensively for decades, the results of the present set of studies indicate that there is still something that can be learnt about the genesis of dreaming, and in particular bad dreams and nightmares.

REFERENCES

Adams, R. D., & Victor, M. (1993). *Principles of neurology* (5th ed.). New York: McGraw-Hill.

Addison, C. C., Campbell-Jenkins, B. W., Sarpong, D. F., Kibler, J., Singh, M., et al. (2007). Psychometric evaluation of a Coping Strategies Inventory Short-Form (*CSI-SF*) in the Jackson Heart Study cohort. *International Journal of Environmental Research and Public Health, 4,* 289-295.

Adhikari, A. (2014). Distributed circuits underlying anxiety. *Frontiers in Behavioral Neuroscience, 8,* 112-118

Adler, C. H., & Thorpy, M. J. (2005). Sleep issues in Parkinson's disease. *Neurology, 64,* S12-S20.

Aikia, M., Salmenpera, T., Partanen, K., & Kalviainen, R. (2001). Verbal memory in newly diagnosed patients and patients with chronic left temporal lobe epilepsy. *Epilepsy & Behavior, 2,* 20-27.

Agargun, M. Y., & Cartwright, R. (2003). REM sleep, dream variables and suicidality in depressed patients. *Psychiatry Research, 119,* 33-39.

Agargun, M. Y., Kara, H., Ozer, O. A., Selvi, Y., Kiran, U., & Kiran, S. (2003). Nightmares and dissociative experiences: The key role of childhood traumatic events. *Psychiatry and Clinical Neurosciences, 57,* 139-145.

Agargun, M. Y., & Ozbek, H. (2006). Drug effects on dreaming. In M. Lader, D. P. Cardinali, & S. R. Pandi-Perumal (Eds.), *Sleep and sleep disorders: A neuropsychopharmacological approach* (pp. 256-261). New York, NY: Springer.

al-Mousawi, H., Evans, N., Ebmeier, K. P., Roeda, D., Chaloner, F., & Ashcroft, G. W. (1996). Limbic dysfunction in schizophrenia and mania. A study using 18F-labelled fluorodeoxyglucose and positron emission tomography. *The British Journal of Psychiatry, 169,* 509-516.

American Academy of Sleep Medicine (2014). *International classification of sleep disorders* (3rd ed.). Darien, IL: American Academy of Sleep Medicine

American Psychiatric Association (2013). *Diagnostic and statistical manual of mental disorders (5th ed.).* Washington, DC: American Psychiatric Association.

Ando, N., Morimoto, K., Watanabe, T., Ninomiya, T., & Suwaki, H. (2004). Enhancement of central dopaminergic activity in the kainate model of temporal lobe epilepsy: Implication for the mechanism of epileptic psychosis. *Neuropsychopharmacology, 29,* 1251-1258.

Antunes-Alves, S., & De Koninck, J. (2012). Pre- and post-sleep stress levels and negative emotions in a sample dream among frequent and non-frequent nightmare sufferers. *Archives of Psychiatry and Psychotherapy, 2,* 11-16.

Argiolas, A., & Melis, M. R. (2003). The neurophysiology of the sexual cycle. *Journal of Endocrinological Investigation, 26,* 20-22.

Arnsten, A. F. T., Raskind, M. A., Taylor, F. B., & Connor, D. F. (2015). The effects of stress exposure on prefrontal cortex: Translating basic research into successful treatments for post-traumatic stress disorder. *Neurobiology of Stress, 1,* 89-99.

Aycicegi-Dinn, A., Dinn, W. M., & Caldwell-Harris, C. L. (2008). The temporolimbic personality: A cross-national study. *European Journal of Psychiatry, 22,* 211-224.

Aycicegi, A., Dinn, W. M., Harris, C. L., & Erkman, H. (2003). Neuropsychological function in obsessive-compulsive disorder: Effects of comorbid conditions on task performance. *European Psychiatry, 18,* 241-248.

Avoli, M., D'Antuono, M., Louvel, J., Kohling, R., Biagini, G., Pumain, R., D'Arcangelo, G., & Tancredi, V. (2002). Network and pharmacological mechanisms leading to epileptiform synchronization in the limbic system in vitro. *Progress in Neurobiology, 68,* 167-207.

Baker, F. C., Mitchell, D., & Driver, H. S. (2001). Oral contraceptives alter sleep and raise body temperature in young women. *Pflugers Archiv: European Journal of Physiology, 442,* 729-37.

Barta, P. E., Pearlson, G. D., Powers, R. E., Richards, S. S., & Tune, L. E. (1990). Auditory hallucinations and smaller superior temporal gyral volume in schizophrenia. *American Journal of Psychiatry, 147,* 1457-1462.

Bassetti, C. L., Bischof, M., & Valko, P. (2005). Dreaming: A neurological view. *Schweizer Archiv Fur Neurologie Und Psychiatrie, 156,* 399-414.

Banu, C., Mcduff, P., & Zadra, A. (2017). Effect of everyday anxiety and psychological well-being on the recall of disturbed dreaming. *Sleep, 40,* A270-271.

Bear, D. M., & Fedio, P. (1977). Quantitative analysis of interictal behavior in temporal lobe epilepsy. *Archives of Neurology, 34,* 454-467.

Belicki, K. (1986). Recalling dreams: An examination of daily variation and individual differences. In J. Gackenbach (Ed.), *Sleep and Dreams: A Sourcebook* (pp. 187-206). London: Garland Publishing Inc.

Belicki, K. (1992). The relationship of nightmare frequency to nightmare suffering with implications for treatment and research. *Dreaming, 2,* 143-148.

Belicki, K., Altay, H., & Hill, C. (1985). Varieties of nightmare experience. *Association for the Study of Dreams Newsletter, 2,* 1-3.

Bentes, C., Costa, J., Peralta, R., Pires, J., Sousa, P., et al. (2011). Dream recall frequency and content in patients with temporal lobe epilepsy. *Epilepsia, 52,* 2022-2027.

Berquier, A., & Ashton, R. (1992). Characteristics of the frequent nightmare sufferer. *Journal of Abnormal Psychology, 101,* 246-250.

Bertram, E. H. (2013). Neuronal circuits in epilepsy: Do they matter? *Experimental Neurology, 244,* 67-74.

Bianchin, M., & Angrilli, A. (2012). Gender differences in emotional responses: A psychophysiological study. *Physiology & Behavior, 105,* 925-923.

Blagrove, M., Farmer, L., & Williams, E. (2004). The relationship of nightmare frequency and nightmare distress to well-being. *Journal of Sleep Research, 13,* 129-136.

Blair, R. J. R. (2001). Neurocognitive models of aggression, the antisocial personality disorders, and psychopathy. *Advances in Neuropsychiatry, 71,* 727-731.

Bob, P., Susta, M., Gregusova, A., Jasova, D., Raboch, J., & Mishara, A. (2010). Traumatic stress, dissociation, and limbic irritability in patients with unipolar depression being treated with SSRIs. *Psychological Reports, 107,* 685-696.

Bogerts, B. (1984). Zur neuropathologie der schizophrenien. *Fortschritte der Neurologie Psychiatrie, 52,* 428-437.

Bogerts, B. (1997). The temporolimbic system theory of positive schizophrenia symptoms. *Schizophrenia Bulletin, 23,* 423-435.

Bogerts, B., Meertz, E., & Schonfeldt-Bausch, R. (1985). Basal ganglia and limbic system pathology in schizophrenia: A morphometric study of brain volume and shrinkage. *Archives of General Psychiatry, 42,* 784-791.

Bonanni, E., Cipolli, C., Iudice, A., Mazzetti, M., & Murri, L. (2002). Dream recall frequency in epilepsy patients with partial and generalized seizures: A dream diary study. *Epilepsia, 43,* 889-895.

Bonilha, L., Nesland, T., Martz, G. U., Joseph, J. E., Spampinato, M. V., Edwards, J. C., & Tabesh, A. (2012). Medial temporal lobe epilepsy is associated with neuronal fibre loss and paradoxical increase in structural connectivity of limbic structures. *Journal of Neurology, Neurosurgery, and Psychiatry, 83,* 903-909.

Borek, L. L., Kohn, R., & Friedman, J. H. (2006). Mood and sleep in Parkinson's disease. *Journal of Clinical Psychiatry, 67,* 958-963.

Borek, L. L., Kohn, R., & Friedman, J. H. (2007). Phenomenology of dreams in Parkinson's disease. *Movement Disorders, 22,* 198-202.

Bouma, E. M. C., Riese, H., Ormel, J., Verhulst, F. C., & Oldehinkel, A. J. (2009). Adolescents' cortisol responses to awakening and social stress; Effects of gender, menstrual phase and oral contraceptives. *Psychoneuroendocrinology, 34,* 884-893.

Blumenfeld, H., McNally, K. A., Vanderhill, S. D., Paige, A. L., Chung, R., Davis, K. … Spencer, S. S. (2004). Positive and negative network correlations in temporal lobe epilepsy. *Cerebral Cortex, 14,* 892-902.

Braak, H., Del Tredici, K., Rub, U., de Vos, R. A. I., Jansen Steur, E. N. H., & Braak, E. (2003). Staging of brain pathology related to sporadic Parkinson's disease. *Neurobiology of Aging, 24,* 197-211.

Braak, H., Ghebremedhin, E., Rub, U., Bratzke, H., & Del Tredici, K. (2004). Stages in the development of Parkinson's disease-related pathology. *Cell Tissue Research, 318,* 121-134.

Bradley, M. M., Codispoti, M., Sabatinelli, D., & Lang, P. (2001). Emotion and motivation II: Sex differences in picture processing. *Emotion, 1,* 300-319.

Brand, S., Beck, J., Kalak, N., Gerber, M., Kirov, R., Puhse, U. … Holsboer-Trachsler, E. (2011). Dream recall and its relationship to sleep, perceived stress, and creativity among adolescents. *Journal of Adolescent Health, 49,* 525-531.

Braun, A., Balkin, T., Wesensten, N., Carson, R., Varga, M., Baldwin, P. … Herscovitch, P. (1997). Regional cerebral blood flow throughout the sleep-wake cycle: An (H2O)-O-15 PET study. *Brain, 120,* 1173-1197.

Breier, A., Buchanan, R. W., Elkashef, A., Munson, R. C., Kirkpatrick, B., & Gellad, F. (1992). Brain morphology and schizophrenia: A magnetic resonance imaging study of limbic, prefrontal cortex, and caudate structures. *Archives of General Psychiatry, 49,* 921-926.

Brown, A. S. (2003). A review of the déjà vu experience. *Psychological Bulletin, 129,* 394-413.

Brown, C., Ling, W., & Wan, J. (2002). A new monophasic oral contraceptive containing drosperinone: Effect on premenstrual symptoms. *Journal of Reproductive Medicine, 47,* 14-22.

Brunet-Gouet, E., & Decety, J. (2006). Social brain dysfunctions in schizophrenia: A review of neuroimaging studies. *Psychiatry Research: Neuroimaging, 148,* 75-92.

Bruni, O., Galli, F., & Guidetti, V. (1999). Sleep hygiene and migraine in children and adolescents. *Cephalalgia, 19,* S57-S59.

Bucci, W., Creelman, M., & Severino, S. K. (1991). The effects of menstrual cycle hormones on dreams. *Dreaming, 1,* 263-276.

Bugalho, P., & Paiva, T. (2011). Dream features in the early stages of Parkinson's disease. *Journal of Neural Transmission, 118,* 1613-1619.

Buhot, M., Martin, S., & Segu, L. (2000). Role of serotonin in memory impairment. *Annals of Medicine, 32,* 210-221.

Burstein, R., & Jakubowski, M. (2005). Unitary hypothesis for multiple triggers of the pain and strain of migraine. *The Journal of Comparative Neurology, 493,* 9-14.

Burstein, R., Noseda, R., & Borsook, D. (2015). Migraine: Multiple processes, complex pathophysiology. *The Journal of Neuroscience, 35,* 6619-6629.

Buzzi, G., & Cirignotta, F. (2000). Isolated sleep paralysis: A web survey. *Sleep Research Online, 3,* 61-66.

Calabresi, P., Castrioto, A., Di Fillipo, M., & Picconi, B. (2013). New experimental and clinical links between the hippocampus and the dopaminergic system in Parkinson's disease. *The Lancet: Neurology, 12,* 811-821.

Calhoon, G. G., & Kay, M. T. (2015). Resolving the neural circuits of anxiety. *Nature Neuroscience, 18,* 1394-1404.

Carr, M., Blanchette-Carriere, C., Solomonova, E., Paquette, T., & Nielsen, T. (2016). Intensified daydreams and nap dreams in frequent nightmare sufferers. *Dreaming, 26,* 119-131.

Carrington, P. (1972). Dreams and schizophrenia. *Archives of General Psychiatry, 26,* 343-350.

Carson, V. B. (2000). *Mental health nursing: The nurse-patient journey (2nd ed.).* Philadelphia: W. B. Saunders.

Cartwright, R. (2011). Dreaming as a mood-regulation system. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine (5th ed.)* (pp. 620-627). Philadelphia: Elsevier Saunders.

Cartwright, R., Luten, A., Young, M., Mercer, P., & Bears, M. (1998). Role of REM sleep and dream affect in overnight mood regulation: A study of normal volunteers. *Psychiatry Research, 81,* 1-8.

Cartwright, R., Young, M. A., Mercer, P., & Bears, M. (1998). Role of REM sleep and dream variables in the prediction of remission from depression. *Psychiatry Research, 80,* 249-255.

Castillo, M. (2011). The sixth dimension and God's helmet. *American Journal of Neuroradiology, 32,* 1767-1768.

Cellucci, A. J., & Lawrence, P. S. (1978). Individual differences in self-reported sleep variable correlations among nightmare sufferers. *Journal of Clinical Psychology, 34,* 721-725.

Cernovsky, Z. Z. (1984). Life stress measures and reported frequency of sleep disorders. *Perceptual and Motor Skills, 58,* 39-49.

Chaplin, T. M., Gillham, J. E., & Seligman, M. E. P. (2009). Gender, anxiety, and depressive symptoms. *The Journal of Early Adolescence, 29,* 307-327.

Chang, Y., Chen, P., Tsai, I., Sung, F., Chin, Z., Kuo, H., Tsai, C., & Chou, I. (2011). Bidirectional relationship between schizophrenia and epilepsy: A population-based retrospective cohort study. *Epilepsia, 52,* 2036-2042.

Chellappa, S. L., & Araujo, J. F. (2007). Sleep disorders and suicidal ideation in patients with depressive disorder. *Psychiatry Research, 153,* 131-136.

Chivers, L., & Blagrove, M. (1999). Nightmare frequency, personality and acute psychopathology. *Personality and Individual Differences, 27,* 843-851.

Cipolli, C., Bonnani, E., Maestri, M., Mazzetti, M., & Murri, L. (2004). Dream experience during REM and NREM sleep of patients with complex partial seizures. *Brain Research Bulletin, 63,* 407-413.

Cipolli, C., Ferrara, M., De Gennaro, L., & Plazzi, G. (2017). Beyond the neuropsychology of dreaming: Insights into the neural basis of dreaming with new techniques of sleep recording and analysis. *Sleep Medicine Reviews, 35,* 8-20.

Claridge, G., Clark, K., & Davis, C. (1997). Nightmares, dreams, and schizotypy. *British Journal of Clinical Psychology, 36,* 377-386.

Cohen, S., Kamarck, T., & Mermelstein R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior, 24,* 385-396.

Colace, C., Claps, M., Antognoli, A., Sperandio, R., Sardi, D., & Benedetti, A. (2010). Limbic system activity and drug dreaming in drug-addicted subjects. *Neuropsychoanalysis, 12,* 201-206.

Commission on Classification and Terminology of the International League Against Epilepsy (2009). *Revised terminology and concepts for organization of the epilepsies: Report of the Commission on Classification and Terminology*. Retrieved from http://www.ilae-epilepsy.org/Visitors/Centre/ctf/ctfoverview.cfm

Cook, C. J. (2002). Glucocorticoid feedback increases the sensitivity of the limbic system to stress. *Physiology & Behaviour, 75,* 455-464.

Cook, C. L., Caplan, R. D., & Wolowitz, H. (1990). Nonwaking responses to waking stressors: Dreams and nightmares. *Journal of Applied Social Psychology, 20,* 199-226.

Coolidge, F. L., Segal, D. L., Coolidge, C. M., Spinath, F. M., & Gottschling, J. (2009). Do nightmares and generalized anxiety disorder in childhood and adolescence have a common genetic origin? *Behavioral Genetics, 40,* 349-356.

D'Agostino, A., Aletti, G., Carboni, M., Cavallotti, S., Limosani, I., Manzone, M., & Scarone, S. (2013). Are delusional contents replayed during dreams? *Consciousness and Cognition, 22,* 708-715.

Damasio, A. R., Grabowski, T. J., Bechara, A., Damasio, H., Ponto, L. L. B., Parvizi, J., & Hichwa, R. D. (2000). Subcortical and cortical brain activity during the feeling of self-generated emotions. *Nature Neuroscience, 3,* 1049-1056.

Dale, A. L., DeCicco, T. L., & Miller, N. J. (2013). Exploring the dreams of Canadian soldiers with content analysis. *International Journal of Dream Research, 6,* 22-30.

D'Andrea, G., & Leon, A. (2010). Pathogenesis of migraine: From neurotransmitters to neuromodulators and beyond. *Neurological Sciences, 31,* S1-S7.

Dang-Vu, T. T., Desseilles, M., Albouy, G., Darsaud, A., Gais, S., Rauchs, G. … Maquet, P. (2005). Dreaming: A neuroimaging view. *Schweizer Archiv fur Neruologie und Psychiatrie, 156,* 415-425.

Dang-Vu, T. T., Gagnon, J-F., Vendette, M., Soucy, J-P., Postuma, R. B., & Montplaisir, J. (2012). Hippocampul perfusion predicts impending neurodegeneration in REM sleep behavior disorder. *Neurology, 79,* 2302-2306.

Davidson, J., Lee-Archer, S., & Sanders, G. (2005). Dream imagery and emotion. *Dreaming, 15,* 33-47.

Davis, J. L., Byrd, P., Rhudy, J. L., & Wright, D. C. (2007). Characteristics of chronic nightmares in a trauma-exposed treatment-seeking sample. *Dreaming, 17,* 187-198.

De Angeli, F., Lovati, C., Giani, L., D'Alessandro, M., Raimondi, E., Scaglione, V. … Mariani, C. (2014). Negative emotions in migraineurs dreams: The increased prevalence of oneiric fear and anguish, unrelated to mood disorders. *Behavioral Neurology, 2014,* 919627.

De Gennaro, L., Lanteri, O., Piras, F., Scarpelli, S., Assogna, F., Ferrara, M. … Spalletta, G. (2016). Dopaminergic system and dream recall: An MRI study in Parkinson's disease patients. *Human Brain Mapping, 37,* 1136-1147.

De Gennaro, L., Marzano, C., Cipolli, C., & Ferrara, M. (2012). How we remember the stuff that dreams are made of: Neurobiological approaches to the brain mechanisms of dream recall. *Behavioral Brain Research, 226,* 592-596.

Degreef, G., Ashtari, M., Bogerts, B., Bilder, R. M., Jody, D. N., Alvir, J. M. J., & Lieberman, J. A. (1992). Volumes of ventricular system subdivisions measured from magnetic resonance images in first-episode schizophrenic patients. *Archives of General Psychiatry, 49,* 531-5357.

Devinsky, O., & D'Esposito, M. (2004). *Neurology of cognitive and behavioral disorders.* Oxford, NY: Oxford University Press.

Devinsky, O., & Lai, G. (2008). Spirituality and religion in epilepsy. *Epilepsy & Behavior, 12,* 636-643.

Dewhurst, K., & Beard, A. W. (1970). Sudden religious conversions in temporal lobe epilepsy. *The British Journal of Psychiatry, 117,* 497-507.

Domhoff, G. W. (1996). *Finding meaning in dreams: A quantitative approach.* New York: Plenum Press.

Domhoff, G. W. (1999). New directions in the study of dream content using the Hall/Van de Castle coding system. *Dreaming, 9,* 115-137.

Domhoff, G. W. (2000a). Methods and measures for the study of dream content. In M. Kryger, T. Roth, & W. Dement (Eds.), *Principles and practices of sleep medicine: Volume 3* (pp. 463-471). Philadelphia: W. B. Saunders.

Domhoff, G. W. (2000b). *The repetition principle in dreams: Is it a possible clue to a function of dreams?* Retrieved from http://www.dreamresearch.net/Library/domhoff_2000b.html

Domhoff, G. W. (2003). The scientific study of dreams: Neural networks, cognitive development, and content analysis. Washington, DC: American Psychological Association.

Domhoff, G. W. (2001). A new neurocognitive theory of dreams. *Dreaming, 11,* 13-33.

Domhoff, G. W. (2005). Refocusing the neurocognitive approach to dreams: A critique of the Hobson versus Solms debate. *Dreaming, 15,* 3-20.

Domhoff, G. W. (2011). The neural substrate for dreaming: Is it a subsystem of the default network? *Consciousness and Cognition, 20,* 1163-1174.

Dosi, C., Riccioni, A., della Corte, M., Novelli, L., Ferri, R., & Bruni, O. (2013). Comorbidities of sleep disorders in childhood and adolescence: Focus on migraine. *Nature and Science of Sleep, 5,* 77-85.

Dresler, M. (2015). The multifunctionality of dreaming and the oblivious avatar – A commentary on Revonsuo & Colleagues. In T. Metzinger & J. M. Windt (Eds), *Open MIND, 32*(C). Frankfurt am Main: MIND Group.

Duke, T., & Davidson, J. (2002). Ordinary and recurrent dream recall of active, past and non-recurrent dreamers during and after academic stress. *Dreaming, 12,* 185-197.

Dunn, K. K., & Barrett, D. (1988). Characteristics of nightmare subjects and their nightmares. *Psychiatric Journal of the University of Ottawa, 13,* 91-93.

Egarter, C., Topcuoglu, M. A., Imhof, M., & Huber, J. (1999). Low dose oral contraceptives and quality of life. *Contraception, 59,* 287-291.

Egger, H. L., Costello, E. J., Erkanli, A., & Angold, A. (1999). Somatic complaints and psychopathology in children and adolescents: Stomach aches, musculoskeletal pains, and headaches. *Journal of the American Academy of Child & Adolescent Psychiatry, 38,* 852-860.

Emre, M. (2003). Dementia associated with Parkinson's disease. *The Lancet: Neurology, 2*, 229-230.

Epstein, A. W. (1964). Recurrent dreams: Their relationship to temporal lobe seizures. *Archives of General Psychiatry, 10,* 25-30.

Epstein, A. W. (1967). Body image alterations during seizures and dreams of epileptics. *Archives of Neurology, 16,* 613-619.

Epstein, A. W., & Hill, W. (1966). Ictal phenomena during REM sleep of a temporal lobe epileptic. *Archives of Neurology, 15,* 367-375.

Esposito, K., Benetiz, A., Barza, L., & Mellman, T. (1999). Evaluation of dream content in combat-related PTSD. *Journal of Traumatic Stress, 12*(4), 681-687.

Etkin, A., & Wager, T. D. (2007). Functional neuroanatomy of anxiety: A meta-analysis of emotional processing in PTSD, social anxiety disorder, and specific phobia. *American Journal of Psychiatry, 164,* 1476-1488.

Evans, C., Connell, J., Barkham, M., Margison, F., McGrath, G., et al. (2002). Towards a standardised brief outcome measure: Psychometric properties and utility of the CORE-OM. *The British Journal of Psychiatry, 180,* 51-60.

Fernandez-Arcos, A., Iranzo, A., Serradell, M., Gaig, C., & Santamaria, J. (2016). The clinical phenotype of idiopathic rapid eye movement sleep behavior disorder at presentation: A study in 203 consecutive patients. *Sleep, 39,* 121-132.

Fink, G., Sumner, B. E. H., Rosie, R., Grace, O., & Quinn, J. P. (1996). Estrogen control of central neurotransmission: Effect on mood, mental state, and memory. *Cellular and Molecular Neurobiology, 16,* 325-344.

Fireman, G. D., Levin, R., & Pope, A. W. (2014). Narrative qualities of bad dreams and nightmares. *Dreaming, 24,* 112-124.

Fisher, C., Byrne, J., Edwards, A., & Kahn, E. (1970). A psychophysiological study of nightmares. *Journal of the American Psychoanalytic Association, 18,* 747-782.

Flor-Henry, P. (1969). Schizophrenic-like reactions and affective psychosis associated with temporal lobe epilepsy: Etiological factors. *American Journal of Psychiatry, 126,* 400-404.

Foa, E. B., Riggs, D. S., & Gershuny, B. S. (1995). Arousal, numbing, and intrusion: Symptom structure of PTSD following assault. *American Journal of Psychiatry, 152,* 116-122.

Fosse, R., & Domhoff, G. W. (2007). Dreaming as non-executive orienting: A conceptual framework for consciousness during sleep. In D. Barrett & P. McNamara (Eds.), *The new science of dreaming: Content, recall, and personality correlates (Vol. 2)* (pp. 49-78). Westport, CT: Praeger.

Frumkin, H. (1998). Multiple system atrophy following chronic carbon disulfide exposure. *Environmental Health Perspectives, 106,* 611-613.

Funkhouser, A. T., Wurmle, O., Cornu, C. M., & Bahro, M. (2001). Boundary questionnaire results in the mentally healthy elderly. *Dreaming, 11,* 83-88.

Gaenslen, A., Swid, I., Liepelt-Scarfone, I., Godau, J., & Berg, D. (2011). The patients' perception of prodromal symptoms before the initial diagnosis of Parkinson's disease. *Movement Disorders, 26,* 653-658.

Gauchet, A., Seguin, J. R., & Zadra, A. (2014). Prevalence and correlates of disturbed dreaming in children. *Pathologie Biologie, 62,* 311-318.

Gehrman, P. R., Harb, G. C., Cook, J. M., Barilla, H., & Ross, R. J. (2014). Sleep diaries of Vietnam War veterans with chronic PTSD: The relationships among insomnia symptoms, psychosocial stress, and nightmares. *Behavioral Sleep Medicine, 13,* 255-264.

Gentil, M. L. F., & Lader, M. (1978). Dream content and daytime attitudes in anxious and calm women. *Psychological Medicine, 8,* 297-304.

Ghaffarinejad, A., & Mehdizadeh, A. (2011). Evaluating dream contents in patients with migraine. *European Psychiatry, 26* (Supplement 1), 992-993.

Giambra, L. M., Jung, R. E., & Grodsky, A. (1996). Age changes in dream recall in adulthood. *Dreaming, 6,* 17-31.

Giani, L., Casazza, R., Mariani, C., & Lovati, C. (2017). Sensory modalities during dreams in migraine: Case-control study using a daily questionnaire. *Dreaming, 27,* 260-268.

Gignac, G. E., & Szodorai, E. T. (2016). Effect size guidelines for individual differences researchers. *Personality and Individual Differences, 102,* 74-78.

Gingnell, M., Engman, J., Frick, A., Moby, L., Wikstrom, J., Fredrikson, M., & Sundstrom-Poromaa, I. (2013). Oral contraceptive use changes brain activity and mood in women with previous negative affect on the pill – A double-blinded, placebo-controlled randomized trial of a levonorgestrel-containing combined oral contraceptive. *Psychoneuroendocrinology, 38,* 1133-1144.

Gloor, P., Olivier, A., Quesney, L. F., Andermann, F., & Horowitz, S. (1982). The role of the limbic system in experiential phenomena of temporal lobe epilepsy. *Annals of Neurology, 12,* 129-144.

Gottesman, C. (2005). Dreaming and schizophrenia: A common neurobiological background. *Sleep and Biological Rhythms, 3,* 64-74.

Gottesman, C. (2006a). The dreaming sleep stage: A new neurobiological model of schizophrenia? *Neuroscience, 140,* 1105-1115.

Gottesman, C. (2006b). Dreaming and schizophrenia: A common neurobiological background? *Medecine Sciences (Paris), 22,* 201-205.

Grace, A. A. (2000). Gating of information flow within the limbic system and the pathophysiology of schizophrenia. *Brain Research Reviews, 31,* 330-341.

Grace, A. A. (2012). Dopamine system dysregulation by the hippocampus: Implications for the pathophysiology and treatment of schizophrenia. *Neuropharmacology, 62,* 1342-1348.

Gupta, S., Mehrotra, S., Villalon, C. M., Perusquia, M., Saxena, P. R., & MaassenVanDenBrink, A. (2007). Potential role of female sex hormones in the pathophysiology of migraine. *Pharmacology & Therapeutics, 113,* 321-340.

Hadjikhani, N., Ward, N., Boshyan, J., Napadow, V., Maeda, Y., Truini, A. … Mainero, C. (2013). The missing link: Enhanced functional connectivity between amygdala and visceroceptive cortex in migraine. *Cephalalgia, 33,* 1264-1268.

Hammerfeld, K., Eberle, C., Grau, M., Kinsperger, A., Zimmerman, A., Ehlert, U., & Gaab, J. (2006). Persistent effects of cognitive-behavioral stress management on cortisol responses to acute stress in healthy subjects – A randomized control trial. *Psychoneuroendocrinology, 31,* 333-339.

Hamstra, D. A., de Kloet, E. R., van Hemert, A. M., de Rijk, R. H., & Van der Does, A. J. W. (2015). Mineralcorticoid receptor haplotype, oral contraceptives and emotional information processing. *Neuroscience, 286,* 412-422.

Hanyu, H., Inoue, Y., Sakurai, H., Kanetaka, H., Nakamura, M., Miyamoto, T. … Iwamoto, T. (2012). Voxel-based magnetic resonance imaging study of structural brain changes in patients with idiopathic REM sleep behavior disorder. *Parkinsonism & Related Disorders, 18,* 136-139.

Harding, A. J., Broe, G. A., & Halliday, G. M. (2002). Visual hallucinations in Lewy body disease relate to Lewy bodies in the temporal lobe. *Brain, 125,* 391-403.

Hartmann, E. (1984). *The nightmare: The psychology and biology of terrifying dreams*. New York: Basic Books.

Hartmann, E. (1989). Boundaries of dreams, boundaries of dreamers. Thin and thick boundaries as a new personality measure. *Psychiatric Journal of the University of Ottawa, 14,* 557-560.

Hartmann, E. (1991). *Boundaries in the mind: A new psychology of personality.* New York: Basic Books.

Hartmann, E. (1998). Nightmare after trauma as paradigm for all dreams: A new approach to the nature and functions of dreaming. *Psychiatry: Interpersonal and Biological Processes, 61,* 223-238.

Hartmann, E., & Basile, R. (2003). Dream imagery becomes more intense after 9/11/01. *Dreaming, 13,* 61-66.

Hartmann, E., & Kunzendorf, R. G. (2005-2006). The central image (CI) in recent dreams, dreams that stand out, and earliest dreams: Relationship to boundaries. *Imagination, Cognition and Personality, 25,* 383-392.

Hartmann, E., Kunzendorf, R., Rosen, R., & Grace, N. G. (2001). Contextualizing images in dreams and daydreams. *Dreaming, 11,* 97-104.

Hartmann, E., Rosen, R., & Rand, W. (1998). Personality and dreaming: Boundary structure and dream content. *Dreaming, 8,* 31-39.

Hartmann, E., Russ, D., van der Kolk, B., Falke, R., & Oldfield, M. (1981). A preliminary study of the personality of the nightmare sufferer: Relatioship to schizophrenia and creativity? *American Journal of Psychiatry, 138,* 794-797.

Hartmann, E., Zborowski, M., & Kunzendorf, R. (2001). The emotion pictured by a dream: An examination of emotions contextualized in dreams. *Sleep and Hypnosis, 3,* 33-43.

Hartmann, E., Zborowski, M., Rose, R., & Grace, N. (2001). Contextualizing images in dreams: More intense after abuse and trauma. *Dreaming, 11,* 115-126.

Hartmann, E., Zborowski, M., McNamara, P., Rosen, R., & Grace, N. (1999). Contextualizing images in dreams: Relationship to the emotional state of the dreamer. *Sleep, 22S,* S131.

Haynes, S., & Mooney, D. (1975). Nightmares: Etiological, theoretical, and behavioral treatment considerations. *Psychological Record, 25,* 225-236.

Hazneder, M. M., Buchsbaum, M. S., Luu, C., Hazlett, E. A., Siegel, B. V., et al. (1997). Decreased anterior cingulate gyrus metabolic rate in schizophrenia. *American Journal of Psychiatry, 154,* 682-684.

Headache Classification Subcommittee of the International Headache Society (2004). The international classification of headache disorders: 2nd edition. *Cephalalgia, 24,* S1-S151.

Healy, D. (2008). *Psychiatric drugs explained (5th ed.).* Oxford, UK: Elsevier Health Sciences.

Heather-Greener, G. Q., Comstock, D., & Joyce, R. (1996). An investigation of the manifest dream content associated with migraine headaches: A study of the dreams that precede nocturnal migraines. *Psychotherapy and Psychosomatics, 65,* 216-221.

Herman, J. P., & Cullinan, W. E. (1997). Neurocircuitry of stress: Central control of the hypothalamo-pituitary-adrenocortical axis. *Trends in Neurosciences, 20,* 78-84.

Herman, J. P., McKlveen, J. M., Solomon, M. B., Carvalho-Netto, E., & Myers, B. (2012). Neural regulation of the stress response: Glucocorticoid feedback mechanisms. *Brazilian Journal of Medical and Biological Research, 45,* 292-298.

Herman, J. P., Ostrander, M. M., Mueller, N. K., & Figueiredo, H. (2005). Limbic system mechanisms of stress regulation: Hypothalamo-pituitary-adrenocortical axis. *Progress in Neuro-Psychopharmacology & Biological Psychiatry, 29,* 1201-1213.

Hermann, B., Meador, K. J., Gaillard, W. D., & Cramer, J. A. (2009). Cognition across the lifespan: Antiepileptic drugs, epilepsy, or both? *Epilepsy & Behavior, 17,* 1-5.

Hersen, M. (1971). Personality characteristics of nightmare sufferers. *Journal of Nervous and Mental Disease, 153,* 27-31.

Hetland, J., Torsheim, T., & Aaro, L. E. (2002). Subjective health complaints in adolescence: Dimensional structure and variation across gender and age. *Scandinavian Journal of Public Health, 30,* 223-230.

Hirsch, S. R., & Weinberger, D. R. (2003). *Schizophrenia (2nd ed.).* Malden, Massachusetts: Blackwell Science.

Hobson, J. A. (2009). REM sleep and dreaming: towards a theory of protoconsciousness. *Nature Reviews Neuroscience, 10,* 803-814.

Hobson, J. A., & Friston, K. J. (2012). Waking and dreaming consciousness: Neurobiological and functional considerations. *Progress in Neurobiology, 98,* 82-98.

Hobson, J. A., & Friston, K. J. (2014). Consciousness, dreams, and inference: The Cartesian theatre revisited. *Journal of Consciousness Studies, 21,* 6-32.

Hobson, J. A., Hong, C. C.-H., & Friston, K. J. (2014). Virtual reality and consciousness inference in dreaming. *Frontiers in Psychology, 5,* 1133.

Hobson, J. A., Pace-Schott, E. F., & Stickgold, R. (2000). Dreaming and the brain: Toward a cognitive neuroscience of conscious states. *Behavioral and Brain Sciences, 23,* 793-842.

Hochard, K. D., Heym, N., & Townsend, E. (2016). The behavioral effects of frequent nightmares on objective stress tolerance. *Dreaming, 26,* 42-49.

Hofer, A., Siedentopf, C. M., Ischebeck, A., Rettenbacher, M. A., Felber, S., & Fleischhacker, W. W. (2006). Gender differences in regional cerebral activity during the perception of emotion: A functional MRI study. *Neuroimage, 32,* 854-862.

Hogl, B., Stefani, A., & Videnovic, A. (2018). Idiopathic REM sleep behavior disorder and neurodegeneration – an update. *Nature Reviews Neurology, 14,* 40-55.

Holmes, T. H., & Rahe, R. H. (1967). The social readjustment rating scale. *Journal of Psychosomatic Research, 11,* 213-218.

Howell, M. J., & Schenck, C. H. (2015). Rapid eye movement sleep behavior disorder and neurodegenerative disease. *JAMA Neurology, 72*, 707-712.

Hublin, C., Jaakko, K., Markku, P., & Markku, K. (2001). Parasomnias: Co-occurrence and genetics. *Psychiatric Genetics, 11,* 65-70.

Hublin, C., & Kaprio, J. (2003). Genetic aspects and genetic epidemiology of parasomnias. *Sleep Medicine Reviews, 7,* 413-421.

Hublin, C., Kaprio, J., Partinen, M., & Koskenvuo, M. (1999). Nightmares: Familial aggregation and association with psychiatric disorders in a nationwide twin cohort. *American Journal of Medical Genetics (Neuropsychiatric Genetics), 88,* 329-336.

Huster, R. J., Wolters, C., Wollbrink, A., Schweiger, E., Wittling, W., Pantev, C., & Junghofer, M. (2009). Effects of anterior cingulate fissurization on cognitive control during Stroop interference. *Human Brain Mapping, 30,* 1279-1289.

Iranzo, A., Valldeoriola, F., Santamaria, J., Tolosa, E., & Rumia, J. (2002). Sleep symptoms and polysomnographic architecture in advanced Parkinson's disease after chronic bilateral subthalamic stimulation. *Journal of Neurology, Neurosurgery & Psychiatry, 72,* 661-664.

Jankovic, J. (2008). Parkinson's disease: Clinical features and diagnosis. *Journal of Neurology, Neurosurgery and Psychiatry, 79,* 368-376.

Janz, D. (1974). Epilepsy and the sleep-waking cycle. In G. Vinken & H. Bruyn (Eds.), *Handbook of clinical neurology: Vol. 45* (pp. 457-490). Amsterdam: North Holland.

Jasova, D., Bob, P., & Fedor-Freybergh, P. (2007). Alcohol craving, limbic irritability, and stress. *Medical Science Monitor, 13,* CR543-547.

Jones, B. E. (2000). The interpretation of physiology. *Behavioral and Brain Sciences, 23,* 955-956.

Joseph, R. (2001). The limbic system and the soul: Evolution and the neuroanatomy of religious experience. *Journal of Religion & Science, 36,* 105-136.

Jovanovic, H., Perski, A., Berglund, H., & Savic, I. (2011). Chronic stress is linked to 5-HT1A receptor changes and functional disintegration of limbic networks. *Neuroimage, 55,* 1178-1188.

Kahn, M., Sheppes, G., & Sadeh, A. (2013). Sleep and emotions: Bidirectional links and underlying mechanisms. *International Journal of Psychophysiology, 89,* 218-228.

Kalaitzakis, M. E., Gentleman, S. M., & Pearce, R. K. B. (2013). Disturbed sleep in Parkinson's disease: anatomical and pathological correlates. *Neuropathology and Applied Neurobiology, 39,* 644-653.

Kaminer, H., & Lavie, P. (1991). Sleep and dreaming in Holocaust survivors: Dramatic decrease in dream recall in well-adjusted survivors. *Journal of Nervous and Mental Disease, 179,* 664-669.

Kawachi, T., Ishii, K., Sakamoto, S., Matsui, M., Mori, T., & Sasaki, M. (2002). Gender differences in cerebral glucose metabolism: a PET study. *Journal of the Neurological Sciences, 199,* 79-83.

Khazaie, H., Tahmasian, M., Younesi, G., Schwebel, D. C., Rezaie, M., Rezaie, L. … Ghanbari, A. (2012). Evaluation of dream content among patients with schizophrenia, their siblings, patients with psychiatric diagnoses other than schizophrenia, and healthy controls. *Iranian Journal of Psychiatry, 7,* 26-30.

Killgore, W. D. S., Britton, J. C., Schwab, Z. J., Price, L. M., Weiner, M. R., Gold, A. L. … Rauch, S. L. (2014). Cortico-limbic responses to masked affective faces across PTSD, panic disorder, and specific phobia. *Depression and Anxiety, 31,* 150-159.

Kim, P., Evans, G. W., Angstadt, M., Ho, S. S., Sripada, C. S., Swain, J. E., Liberzon, I., & Phan, K. L. (2013). Effects of childhood poverty and chronic stress on emotion

regulatory brain function in adulthood. *Proceedings of the National Academy of Science USA, 110,* 18442-18447.

Kircher, T. T. J., Senior, C., Philips, M. L., Rabe-Hesketh, S., Benson, P. J., et al. (2001). Recognizing one's own face. *Cognition, 78,* B1-B15.

Kirschbaum, C., Kudielka, B., Gaab, J., Schommer, N. C., & Hellhammer, D. H. (1999). Impact of gender, menstrual cycle phase, and oral contraceptives on the activity of the hypothalamus-pituitary-adrenal axis. *Psychosomatic Medicine, 61,* 154-162.

Kirschbaum, C., Pirke, K., & Hellhammer, D. H. (1995). Preliminary evidence for reduced cortisol responsivity to psychological stress in women using oral contraceptive medication. *Psychoneuroendocrinology, 20,* 509-514.

Kirschbaum, C., Platte, P., Pirke, K. M., & Hellhammer, D. (1996). Adrenocortical activation following stressful exercise: further evidence for attenuated free cortisol response in women using oral contraceptives. *Stress Medicine, 23,* 178-183.

Kirschner, N. T. (1999). Changes in dream content after drug treatment. *Dreaming, 9,* 195-200.

Koffell, E., & Watson, D. (2009). Unusual sleep experiences, dissociation, and schizotypy: Evidence for a common domain. *Clinical Psychology Review, 29,* 548-559.

Kneisl, C. R., & Trigoboff, E. (2009). *Contemporary psychiatric-mental health nursing (2nd ed. Rev.).* New Jersey: Prentice Hall.

Kracmarova, L. K., & Plhakova, A. (2015). Nightmares and their consequences in relation to state factors, absorption, and boundaries. *Dreaming, 25,* 312-320.

Krakow, B., Germain, A., Warner, T. D., Schrader, R., Koss, M., Hollifield, M., Tandberg, D., Melendrez, D., & Johnston, L. (2001). The relationship of sleep quality and posttraumatic stress to potential sleep disorders in sexual assault survivors with nightmares, insomnia, and PTSD. *Journal of Traumatic Stress, 14,* 647-664.

Kramer, M. (1993). The selective mood regulatory function of dreaming: An update and revision. In A. Moffit, M. Kramer, & R. Hoffmann (Eds.), *The functions of dreaming* (pp. 139-196). Albany: State University of New York.

Kramer, M., & Roth, T. (1973). A comparison of dream content in laboratory dream reports of schizophrenic and depressive patient groups. *Comprehensive Psychiatry, 14,* 325-329.

Kramer, M., Schoen, L. S., & Kinney, L. (1984). The dream experience in dream disturbed Vietnam veterans. In B. A. van der Kolk (ed.), *Posttraumatic stress disorders: Psychological and biological sequelae* (pp. 82-95). Washington, DC: American Psychiatric Association.

Kroth, J., Thomspon, L., Jackson, J., Pascali, L., & Ferreira, M. (2002). Dream characteristics of stockbrokers after a major market downturn. *Psychology Reports, 90,* 1097-1100.

Kudielka, B. M., & Kirschbaum, C. (2005). Sex differences in HPA axis response to stress: A review. *Biological Psychology, 69,* 113-132.

Kuhlman, S., & Wolf, O. T. (2005). Cortisol and memory retrieval in women: Influence of menstrual cycle and oral contraceptives. *Psychopharmacology, 183,* 65-71.

Kumar, S., Bhatia, M., & Behari, M. (2002). Sleep disorders in Parkinson's disease. *Movement Disorders, 17,* 775-781.

Lancee, J., Spoormaker, V. I., & van den Bout, J. (2010). Nightmare frequency is associated with subjective sleep quality but not with psychopathology. *Sleep and Biological Rhythms, 8,* 187-193.

Lanius, R. A., Bluhm, R., Lanius, C., & Pain, C. (2006). A review of neuroimaging studies in PTSD: heterogeneity of response to symptom provocation. *Journal of Psychiatric Research, 40,* 709-729.

Lanius, R. A., Hopper, J. W., & Menon, R. S. (2003). Individual differences in a husband and wife who developed PTSD after a motor vehicle accident: A functional MRI case study. *American Journal of Psychiatry, 160,* 667-669.

Lateef, T. M., Cui, L., Nelson, K. B., Nakamura, E. F., & Merikangas, K. R. (2012). Physical comorbidity of migraine and other headaches in US adolescents. *The Journal of Pediatrics, 161,* 308-313.

LeDoux, J. (1996). *The emotional brain.* New York: Simon & Schuster.

LeDoux, J. (2002). *Synaptic self: How our brains become who we are.* New York: Penguin Putnam.

Lemarche, L. J., & De Koninck, J. (2007). Sleep disturbances in adults with posttraumatic stress disorder: A review. *Journal of Clinical Psychiatry, 68,* 1257-1270.

Levin, E. R., & Hammes, S. R. (2011). Estrogens and progestins. In L. L. Brunton, B. A. Chabner, & B. C. Knollman, *Goodman & Gilman's the pharmacological basis of therapeutics (12th ed.)* (pp. 1163-1194). New York: McGraw-Hill Medical.

Levin, R. (1994). Sleep and dreaming characteristics of frequent nightmare subjects in a university population. *Dreaming, 4,* 127-137.

Levin, R. (2000). Nightmares: Friend or foe? *Behavioral and Brain Sciences, 23,* 965-965.

Levin, R., & Hurvich, M. S. (1995). Nightmares and annihilation anxiety. *Psychoanalytic Psychology, 12,* 247-258.

Levin, R., & Fireman, G. (2002a). Nightmare prevalence, nightmare distress, and self-reported psychological disturbance. *Sleep, 25,* 205-212.

Levin, R., & Fireman, G. (2002b). Phenomenal qualities of nightmare experience in a prospective study of college students. *Dreaming, 12*, 109-120.

Levin, R., Fireman, G., & Nielsen, T. (2010). Disturbed dreaming and emotion dysregulation. *Sleep Medicine Clinics, 5,* 229-239.

Levin, R., Fireman, G., Spendlove, S., & Pope, A. (2011). The relative contribution of affect load and affect distress as predictors of disturbed dreaming. *Behavioral Sleep Medicine, 9,* 173-183.

Levin, R., & Nielsen, T. A. (2007). Disturbed dreaming, posttraumatic stress disorder, and affect distress: A review and neurocognitive model. *Psychological Bulletin, 133,* 482-528.

Levin, R., & Nielsen, T. (2009). Nightmares, bad dreams, and emotion dysregulation: A review and new neurocognitive model of dreaming. *Current Directions in Psychological Science, 18,* 84-88.

Levin, R., & Raulin, M. L. (1991). Preliminary evidence for the proposed relationship between frequent nightmares and schizotypal symptomatology. *Journal of Personality Disorders, 5,* 8-14.

Levitan, H. (1984). Dreams which culminate in migraine headaches. *Psychotherapy and Psychosomatics, 4,* 161-166.

Li, S. X., Lam, S. P., Zhang J., Yu, M. W., Chan, J. W., Chan, C. S. … Wing, Y. K. (2016). Sleep disturbances and suicide risk in an 8-year longitudinal study of schizophrenia-spectrum disorders. *Sleep,* 39, 1275–1282.

Li, S. X., Zhang, B., Li, A. M., & Wing, Y. K. (2010). Prevalence and correlates of frequent nightmares: A community-based 2-phase study. *Sleep, 33,* 774-780.

Liberzon, I., Taylor, S. F., Amdur, R., Jung, T. D., Chamberlain, K. R., Minoshima, S., Koeppe, R. A., & Fig, L. M. (1998). Brain activation in PTSD in response to trauma-related stimuli. *Biological Psychiatry, 45,* 817-826.

Lifosky, N., Riedeger, M., Gallinat, J., Lindenburger, U., & Kuhn, S. (2016). Hormonal contraceptive use is associated with neural and affective changes in healthy young women. *Neuroimage, 134,* 597-606.

Low, J. F. A., Dyster-Aas, J., Willebrand, M., Kildal, M., Gerdin, B., & Ekselius, L. (2003). Chronic nightmares after severe burns: Risk factors and implications for treatment. *The Journal of Burn Care & Rehabilitation, 24,* 260-267.

Luciana, M., Collins, P. F., Depue, R. A. (1998). Opposing roles for dopamine and serotonin in the modulation of human spatial working memory functions. *Cerebral Cortex, 8,* 218-226.

Lungu, O., Potvin, S., Tikasz, A., & Mendrek, A. (2015). Sex differences in effective fronto-limbic connectivity during negative emotion processing. *Psychoneuroendocrinology, 62,* 180-188.

Lusignan, F. A., Zadra, A., Dubuc, M. J., Daoust, A. M., Mottard, J. P., & Godbout, R. (2009). Dream content in chronically-treated persons with schizophrenia. *Schizophrenia Research, 112,* 164-173.

Maizels, M., Aurora, S., & Heinricher, M. (2012). Beyond neurovascular: Migraine as a dysfunctional neurolimbic pain network. *Headache, 52,* 1553-1565.

Malcolm-Smith, S., Koopowitz, S., Pantelis, E., & Solms, M. (2012). Approach/avoidance in dreams. *Consciousness and Cognition, 21,* 408-412.

Malcolm-Smith, S., & Solms, M. (2004). Incidence of threat in dreams: A response to Revonsuo's threat simulation theory. *Dreaming, 14,* 220-229.

Malcolm-Smith, S., Solms, M., Turnbull, O., & Tredoux, C. (2008). Threat in dreams: An adaptation? *Consciousness and Cognition, 17,* 1281-1291.

Malow, B. (2004). Sleep deprivation and epilepsy. *Epilepsy Currents: Reviews and Critical Analysis, 4,* 193-195.

Mamelak, A. N., & Hobson, J. A. (1989). Dream bizarreness as the cognitive correlate of altered neuronal behavior in REM sleep. *Journal of Cognitive Neuroscience, 1,* 201-222.

Marcks, B. A., Weisberg, R. B., Edelen, M. O., & Keller, M. B. (2010). The relationship between sleep disturbance and the course of anxiety disorders in primary care patients. *Psychiatry Research, 178,* 487-492.

Martin, V. T., & Behbehani, M. M. (2001). Toward a rational understanding of migraine trigger factors. *Medical Clinics of North America, 85,* 911-941.

Mascia, A., Afra, J., & Schoenen, J. (1998). Dopamine and migraine: A review of pharmacological, biochemical, neurophysiological, and therapeutic data. *Cephalalgia, 18,* 174-182.

Maquet, P., Peters, J-M., Aerts, J., Delfiore, G., Dequerldre, C., Luxen, A., & Franck, G. (1996). Functional neuroanatomy of human rapid-eye-movement sleep and dreaming. *Nature, 383,* 163-166.

McCarley, R. W., & Hobson, J. A. (1975). Neuronal excitability modulation over the sleep cycle: A structural and mathematical model. *Science, 189,* 58-60.

McNamara, P., Johnson, P., McLaren, D., Harris, E., Beauharnais, C., & Auerbach, S. (2010). REM and NREM sleep mentation. *International Review of Neurobiology, 92,* 69-86.

McNamara, P., Minsky, A., Pae, V., Harris, E., Pace-Schott, E., & Auerbach, S. (2015). Aggression in nightmares and unpleasant dreams and in people reporting recurrent nightmares. *Dreaming, 25,* 190-205.

Mellman, T. A., David, D., & Barza, L. (1999). Nefazodone treatment and dream reports in chronic PTSD. *Depression and Anxiety, 9,* 146-148.

Mellman, T. A., David, D., Bustamante, V., Torres, J., & Fins, A. (2001). Dreams in the acute aftermath of trauma and their relationship to PTSD. *Journal of Traumatic Stress, 14,* 241-247.

Mellman, T. A., & Pigeon, W. R. (2010). Dreams and nightmares in posttraumatic stress disorder. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (5th ed.) (pp. 613-619). Philadelphia: Elsevier.

Michels, F., Schilling, C., Rausch, F., Eifler, S., Zink, M., Meyer-Lindenberg, A., & Schredl, M. (2014). Nightmare frequency in schizophrenic patients, healthy relatives of schizophrenic patients, patients at high risk states for psychosis, and healthy controls. *International Journal of Dream Research, 7,* 9-13.

Millan, M. J., Dekeyne, A., & Gobert, A. (1998). Serotonin (5-HT)2C receptors tonically inhibit dopamine (DA) and noradrenaline (NAD), but not 5-HT, release in the frontal cortex in vivo. *Neuropharmacology, 37,* 953-955.

Miller, B. L., Cummings, J. L., McIntyre, H., Ebers, G., & Grodes, M. (1986). Hypersexuality or altered sexual preference following brain injury. *Journal of Neurology, Neurosurgery, and Psychiatry, 49,* 867-873.

Mindell, J. A., & Barrett, K. M. (2002). Nightmares and anxiety in elementary-aged children: Is there a relationship? *Child: Care, Health & Development, 28,* 317-322.

Monroe, L. J., & Marks, P. A. (1977). MMPI differences between adolescent poor and good sleepers. *Journal of Consulting and Clinical Psychology, 45,* 151-152.

Montoya, E. R., & Bos, P. A. (2017). How oral contraceptives impact social-emotional behaviour and brain function. *Trend in Cognitive Sciences, 21,* 125-136.

Muller, M. B., Zimmerman, S., Sillaber, I., Hagemeyer, T. P., Deussing, J. M., et al. (2003). Limbic corticotropin-releasing hormone receptor 1 mediates anxiety-related behavior and hormonal adaptation to stress. *Nature Neuroscience, 6,* 1100-1107.

Muzur, A., Pace-Schott, E. F., & Hobson, J. A. (2002). The prefrontal cortex in sleep. *Trends in Cognitive Sciences, 6,* 475-481.

Nadorff, M. R., Porter, B., Rhoades, H. M., Greisinger, A. J., Kunik, M. E., & Stanley, M. A. (2014). Bad dream frequency in older adults with Generalized Anxiety Disorder: Prevalence, correlates, and effect of cognitive behavioral treatment for anxiety. *Behavioral Sleep Medicine, 12,* 28-40.

Natale, V., Albertazzi, P., & Cangini, A. (2003). The effects of menstrual cycle on dreaming. *Biological Rhythm Research, 34,* 295-303.

National Society for Epilepsy (1999). *Seizures*. Retrieved from http://www.epilepsysociety.org.uk/AboutEpilepsy/Whatisepilepsy/Seizures

Nelson, A. L., & Cwiak, C. (2011). Combined oral contraceptives (COCs). In R. A. Hatcher, J. Trussell, A. L. Nelson, W. Cates Jr., D. Kowal, et al., *Contraceptive technology (20th rev. ed.)* (pp. 249-341). New York: Ardent Media.

Nielsen, S. E., Segal, S. K., Worden, I. V., Yim, I. S., & Cahill, L. (2013). Hormonal contraceptive use alters stress responses and emotional memory. *Biological Psychiatry, 92,* 257-266.

Nielsen, T. A. (1991). Affect desensitization: A possible function of REMs in both waking and sleeping states. *Sleep Research, 20,* 10.

Nielsen, T. A. (2000). A review of mentation in REM and NREM sleep: "Covert" REM sleep as a possible reconciliation of two opposing models. *Behavioral and Brain Sciences, 23,* 793-1121.

Nielsen, T. A. (2010). Disturbed dreaming as a factor in medical conditions. In M. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (5th ed.) (pp. 1116-1127). New York: Elsevier.

Nielsen, T. A. (2017a). The stress acceleration hypothesis of nightmares. *Frontiers in Neurology, 8,* 201-224.

Nielsen, T. A. (2017b). When was your earliest dream? Association of very early dream recall with frequent current nightmares supports a stress-acceleration explanation of nightmares. *Dreaming, 27,* 122-136.

Nielsen, T. A., & Carr, M. (2017). Nightmares and nightmare function. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine (6th ed.)* (pp. 546-554). Philadelphia, PA: Elsevier.

Nielsen, T. A., Kuiken, D. L., & McGregor, D. L. (1989). Effects of dream reflection on waking affect: Awareness of feelings, Rorschach movement, and facial EMG. *Sleep, 12,* 277-286.

Nielsen, T. A., Laberge, L., Paquet, J., Tremblay, R. E., Vitaro, F., & Montplaisir, J. (2000). Development of disturbing dreams during adolescence and their relation to anxiety symptoms. *Sleep, 23,* 1-10.

Nielsen, T. A., & Levin, R. (2007). Nightmares: A new neurocognitive model. *Sleep Medicine Reviews, 11,* 295-310.

Nielsen, T. A., & Levin, R. (2009). Dreams, dreaming theories and correlates of nightmares. In L. R. Squire (Ed.), *Encyclopedia of neuroscience (Vol. 3)* (pp. 663-669). Oxford: Academic Press.

Nielsen, T. A., Powell, R. A., & Kuiken, D. (2013). Nightmare frequency is related to a propensity for mirror behaviors. *Consciousness and Cognition, 22,* 1181-1188.

Nielsen, T. A., Stenstrom, P., & Levin, R. (2006). Nightmare frequency as a function of age, gender, and September 11, 2001: Findings from an internet questionnaire. *Dreaming, 16,* 145-158.

Nielsen, T. A., Zadra, A., L., Simard, V., Saucier, S., Stenstrom, P., Smith, C., & Kuiken, D. (2003). The typical dreams of Canadian university students. *Dreaming, 13,* 211-235.

Nofzinger, E., Mintun, M., Wiseman, M., Kupfer, D., & Moore, R. (1997). Forebrain activation in REM sleep: An FDG PET study. *Brain Research, 770,* 192-201.

Oerlemans, W. G. H., & de Weerd, A. W. (2002). The prevalence of sleep disorders in patients with Parkinson's disease: A self-reported, community-based survey. *Sleep Medicine, 3,* 147-149.

Ohayon, M. M. (2004). Prevalence and risk factors of morning headaches in the general population. *Archives of Internal Medicine, 164,* 97-102.

Ohayon, M. M., & Shapiro, C. M. (2000). Sleep disturbances and psychiatric disorders associated with posttraumatic stress disorder in the general population. *Comprehensive Psychiatry, 41,* 469-478.

Ohayon, M., Zulley, J., Guilleminault, C., & Smirne, S. (1999). Prevalence and pathological associations of sleep paralysis in the general population. *Neurology, 52,* 1194-2000.

Oinonen, K. A., & Mazmanian, D. (2001). Effects of oral contraceptives on daily self-ratings of positive and negative affect. *Journal of Psychosomatic Research, 51,* 647-658.

Oinonen, K. A., & Mazmanian, D. (2002). To what extent do oral contraceptives influence mood and affect? *Journal of Affective Disorders, 70,* 229-240.

Otsuka, Y., Kaneita, Y., Nakagome, S., Jike, M., Itani, O., & Ohida, T. (2018). Nightmares and sleep paralysis among the general Japanese population: A nationwide representative survey. *Sleep and Biological Rhythms, 16,* 187-195.

Pagel, J. F. (2006). The neuropharmacology of nightmares. In M. Lader, D. P. Cardinali, & S. R. Pandi-Perumal (Eds.), *Sleep and sleep disorders: A neuropsychopharmacological approach* (pp. 241-250). New York, NY: Springer.

Pagel, J. F., & Helfter, P. (2003). Drug induced nightmares – an etiology based review. *Human Psychopharmacology: Clinical & Experimental, 18,* 59-67.

Palagani, L., & Rosenlicht, N. (2011). Sleep, dreaming, and mental health: A review of the historical and neurobiological perspectives. *Sleep Medicine Reviews, 15,* 179-186.

Parsey, K. S., & Pong, A. (2000). An open-label, multicenter study to evaluate Yasmin, a low-dose combination oral contraceptive containing drospirenone, a new progestogen. *Contraception, 61,* 105-111.

Pelin, Z., & Yazla, E. (2012). Abnormal sexual behavior during sleep in temporal lobe epilepsy: A case report. *Balkan Medical Journal, 29,* 211-213.

Penfield, W., & Erickson, T. (1941). *Epilepsy and cerebral localization.* Springfield, IL: Thomas.

Penfield, W., & Rasmussen, T. (1957). *The cerebral cortex of man: A clinical study of localization of function (4th ed.)* (pp. 157-181). New York: Macmillan.

Perogamvros, L., Aberg, K., Gex-Fabry, M., Perrig, S., Cloninger, C. R., & Schwartz, S. (2015). Increased reward-related behaviors during sleep and wakefulness in sleepwalking and idiopathic nightmares. *PLoS ONE, 10,* e0134504.

Perlis, M. L., Giles, D. E., Fleming, G. M., Drummond, S. P. A., & James, S. P. (1995). Sustained facial muscle activity during REM sleep and its correlation with depression. *Journal of Affective Disorders, 35,* 163-171.

Perlis, M. L., & Nielsen, T. A. (1993). Mood regulation, dreaming and nightmares: Evaluation of a desensitization function for REM sleep. *Dreaming, 3,* 243-257.

Perry, E. K., & Piggott, M. A. (2000). Neurotransmitter mechanisms of dreaming: Implication of modulatory systems based on dream intensity. *Behavioral and Brain Sciences, 23,* 990-992.

Persinger, M. A. (2001). The neuropsychiatry of paranormal experiences. *Journal of Neuropsychiatry and Clinical Neuroscience, 13,* 515-524.

Pesant, N., & Zadra, A. (2006). Dream content and psychological well-being: A longitudinal study of the continuity hypothesis. *Journal of Clinical Psychology, 62,* 111-121.

Peterson, N. D. J., Henke, P. G., & Hayes, Z. (2002). Limbic system function and dream content in university students. *The Journal of Neuropsychiatry and Clinical Neurosciences, 14,* 283-288.

Phelps, A. J., Forbes, D., & Hopwood, M., & Creamer, M. (2011). Trauma-related dreams of Australian veterans with PTSD: Content, affect and phenomenology. *Australian & New Zealand Journal of Psychiatry, 45,* 853-860.

Picchioni, D. (2001). Nightmares as a coping mechanism for stress. *Master's dissertation, San Jose State University, California, United States.* Retrieved from http://scholarworks.sjsu.edu/etd_theses/2194

Pietrowsky, R., & Kothe, M. (2003). Personal boundaries and nightmare consequences in frequent nightmare sufferers. *Dreaming, 13,* 245-254.

Pigeon, W. R., Campbell, C. E., Possemato, K., & Ouimette, P. (2013). Longitudinal relationships of insomnia, nightmares, and PTSD severity in recent combat veterans. *Journal of Psychosomatic Research, 75,* 546-550.

Pittau, F., Grova, C., Moeller, F., Dubeau, F., & Gotman, J. (2012). Patterns of altered functional connectivity in mesial temporal lobe epilepsy. *Epilepsia, 53,* 1013-1023.

Pletzer, B. A., & Kerschbaum, H. H. (2014). 50 years of hormonal contraception – time to find out, what it does to our brain. *Frontiers in Neuroscience, 8,* 256.

Podoll, K., Topper, R., Robinson, D., & Sass, H. (2000). Recurrent dreams as migraine aura symptoms. *Fortschritte der Neurologie-Psychiatrie, 68,* 145-149.

Pont-Sunyer, C., Hotter, A., Gaig, C., Seppi, K., Compta, Y., Katzenschlager, R. … Tolosa, E. (2015). The onset of nonmotor symptoms in Parkinson's disease (The ONSET PD Study). *Movement Disorders, 30,* 229-237.

Poromaa, P., & Segebladh, B. (2012). Adverse mood symptoms with oral contraceptives. *Acta Obstetricia et Gynecologica Scandinavica, 91,* 420-427.

Poryazova, R., Oberholzer, M., Baumann, C. R., & Bassetti, C. L. (2013). REM sleep behavior disorder in Parkinson's disease: A questionnaire-based survey. *Journal of Clinical Sleep Medicine, 15,* 55-59.

Potvin, O., Lorrain, D., Belleville, G., Grenier, S., & Preville, M. (2014). Subjective sleep characteristics associated with anxiety and depression in older adults: A population-based study. *International Journal of Geriatric Psychiatry, 29,* 1262-1270.

Preacher, K. J. (2015). Extreme groups designs. In R. L. Cautin & S. O. Lilienfeld (Eds.), *The Encyclopedia of Clinical Psychology (Vol. 2)* (pp. 1189-1192). Hoboken, NJ: John Wiley & Sons, Inc.

Preacher, K. J., Rucker, D. D., MacCallum, R. C., & Nicewander, W. A. (2005). Use of the - extreme groups approach: A critical reexamination and new recommendations. *Psychological Methods, 10,* 178-192.

Propper, R. E., Stickgold, R., Keeley, R., & Christman, S. D. (2007). Is television traumatic? Dreams, stress, and media exposure in the aftermath of September 11, 2001. *Psychological Science, 18,* 334-340.

Rains, J., & Poceta, J. (2006). Headache and sleep disorders: Review and clinical implications for headache management. *Headache, 46,* 1344-1363.

Reami, D. O., Silva, D. F., Albuquerque, M., & Campos, C. J. R. (1991). Dreams and epilepsy. *Epilepsia, 32,* 51-53.

Revonsuo, A., Tuominen, J., & Valli, K. (2015a). The avatars in the machine – Dreaming as a simulation of social reality. In T. Metzinger & J. M. Windt (Eds), *Open MIND, 32*(T). Frankfurt am Main: MIND Group.

Revonsuo, A., Tuominene, J., & Valli, K. (2015b). The simulation theories of dreaming: How to make theoretical progress in dream science – A reply to Martin Dresler. In T. Metzinger & J. M. Windt (Eds), *Open MIND, 32*(R). Frankfurt am Main: MIND Group.

Revonsuo, A., & Valli, K. (2000). Dreaming and consciousness: Testing the threat simulation theory of the function of dreaming. *Psyche, 6*. Retrieved February 3, 2012, from http://psyche.cs.monash.edu.au/v6/psyche-6-08-revonsuo.html

Robert, G., & Zadra, A. (2008). Measuring nightmare and bad dream frequency: impact of retrospective and prospective instruments. *Journal of Sleep Research, 17,* 132-139.

Robert, G., & Zadra, A. (2014). Thematic and content analysis of idiopathic nightmares and bad dreams. *Sleep, 37,* 409-417.

Ross, R. J., Ball, W. A., Sullivan, K. A., Caroff, S. N. (1989). Sleep disturbance as the hallmark of posttraumatic stress disorder. *American Journal of Psychiatry, 146,* 697-707.

Rossi, A., Stratta, P., Mancini, F., Gallucci, M., Mattei, P., Core, L., Di Michele, V., & Casacchia, M. (1994). Magnetic resonance imaging findings of amygdala-anterior hippocampus shrinkage in male patients with schizophrenia. *Psychiatry Research, 52,* 43-53.

Sadeghniiat, K., Rajabzadeh, A., Ghajarzadeh, M., & Ghafarpour, M. (2013). Sleep quality and depression among patients with migraine. *Acta Medica Iranica, 51,* 784-788.

Sandman, N., Valli, K., Kronholm, E., Ollila, H. M., Revonsuo, A., Laatikainen, T., & Paunio, T. (2013). Nightmares: Prevalence among the Finnish general adult population and war veterans during 1972-2007. *Sleep, 36,* 1041-1050.

Sasai, T., Inoue, Y., & Matsuura, M. (2012). Effectiveness of Pramipexole, a dopamine agonist, on rapid eye movement sleep behavior disorder. *The Tohoku Journal of Experimental Medicine, 226,* 177-181.

Schenck, C. H. (2015). REM sleep behavior disorder. In S. Chokroverty, & M. Billiard (Eds), *Sleep Medicine* (pp. 391-405). New York: Springer.

Schenck, C. H., & Mahowald, M. W. (2018). Insights into polysomnography-confirmed rapid eye movement sleep behavior disorder from new prevalence and gender data in the general population. *Sleep, 41,* zsy020.

Schlarb, A. A., Christen, R., Claben, M., & Bien, C. G. (2016). Sleep and dreaming in children and adolescents with epilepsy. *Somnologie, 20,* 242-250.

Schmitz, N., Admiraal-Behloul, F., Arkink, E., Kruit, M. C., Schoonman, G. G., Ferrari, M. D., & van Buchem, M. A. (2008). Attack frequency and disease duration as indicators for brain damage in migraine. *Headache, 48,* 1044-1055.

Schneider, A. (2001). DreamSAT: Automated dream data entry system and Statistical Analysis Tool. Retrieved from https://www2.ucsc.edu/dreams/DreamSAT/DreamSAT_docs.pdf

Schnetzler, J. P., & Carbonnel, B. (1976). Thematic study of the narration of dreams of normal, and of schizophrenic and other psychotic subjects. *Annales Medico-psychologiques (Paris), 1,* 367-380.

Schredl, M. (2010). Nightmare frequency and nightmare topics in a representative German sample. *European Archives of Psychiatry and Clinical Neuroscience, 260,* 565-570.

Schredl, M. (2014). Explaining the gender difference in nightmare frequency. *The American Journal of Psychology, 127,* 205-213.

Schredl, M., Berres, S., Klingauf, A., Schellhaus, S., & Goritz, A. S. (2014). The Mannheim Dream Questionnaire (MADRE): Retest reliability, age and gender effects. *International Journal of Dream Research, 7,* 141-147.

Schredl, M., Binder, R., Feldmann, S., Goder, R., Hoppe, J., Schmitt, J. … Steinig, J. (2012). Dreaming in patients with sleep disorders. *Somnologie - Schlafforschung und Schlafmedizin, 16*, 32-42.

Schredl, M., & Piel, E. (2003). Gender differences in dreams: Data from four representative German samples. *Personality and Individual Differences, 35,* 1185-1189.

Schredl, M., & Piel, E. (2008). Interest in dream interpretation: A gender difference. *Dreaming, 18,* 11-15.

Schredl, M., & Reinhard, I. (2008). Gender differences in dream recall: A meta-analysis. *Journal of Sleep Research, 17,* 125-131.

Schredl, M., & Reinhard, I. (2011). Gender differences in nightmare frequency: A meta-analysis. *Sleep Medicine Reviews, 15,* 115-121.

Schredl, M., Schafer, G., Hofmann, F., & Jacob, S. (1999). Dream content and personality: Thick vs. thin boundaries. *Dreaming, 9,* 257-263.

Schreuder, B. J., Klein, W. C., & Rooijmans, H. G. (2000). Nocturnal re-experiencing more than forty years after war trauma. *Journal of Traumatic Stress, 13,* 453-463.

Sengoku, A., Toichi, M., & Murai, T. (1997). Dreamy states and psychoses in temporal lobe epilepsy: Mediating role of affect. *Psychiatry and Clinical Neurosciences, 51,* 23-26.

Seo, D., Patrick, C. J., & Kennealy, P. J. (2008). Role of serotonin and dopamine system interactions in the neurobiology of impulsive aggression and its comorbidity with other clinical disorders. *Aggressive and Violent Behavior, 13,* 383-395.

Shapiro, F. (1989). Efficacy of the eye movement desensitization procedure in the treatment of traumatic memories. *Journal of Traumatic Stress, 2,* 199-223.

Shin, L. M., & Handwerger, K. (2009). Is posttraumatic stress disorder a stress-induced fear circuitry disorder? *Journal of Traumatic Stress, 22,* 409-415.

Shulman, M., & Devinsky, O. (2003). Cognitive and behavioral aspects of epilepsy. In T. E. Feinberg & M. J. Farah (Eds.), *Behavioral neurology* (2nd ed.) (pp. 675-693). USA: McGraw-Hill.

Sikka, P., Valli, K., Virta, T., & Revonsuo, A. (2014). I know how you felt last night, or do I? Self- and external ratings of emotions in REM sleep dreams. *Consciousness and Cognition, 25,* 51-66.

Silvestri, R., & Bromfield, E. (2004). Recurrent nightmares and disorders of arousal in temporal lobe epilepsy. *Brain Research Bulletin, 63,* 369-376.

Simor, P., Pajkossy, P., Horvath, K., & Bodisz, R. (2012). Impaired executive functions in subjects with frequent nightmares as reflected by performance in different neuropsychological tasks. *Brain and Cognition, 78,* 274-283.

Sirois-Berliss, M., & de Koninck, J. (1982). Menstrual stress and dreams: Adaptation or interference? *Psychiatric Journal of the University of Ottawa, 7,* 77-86.

Solms, M. (1997). *The neuropsychology of dreams: A clinico-anatomical study*. Hillsdale, NJ: Lawrence-Erlbaum.

Solms, M. (2000a). Dreaming and REM sleep are controlled by different brain mechanisms. *Behavioral and Brain Sciences, 23,* 843-850.

Solms, M. (2000b). Forebrain mechanisms of dreaming are activated from a variety of sources. *Behavioral and Brain Sciences, 23,* 1035-1040.

Solms, M. (2002). Dreaming: Cholinergic and dopaminergic hypotheses. In E. Perry, H. Ashton, & A. Young (Eds.), *Neurochemistry of consciousness* (pp. 123-131). Philadelphia: Benjamins.

Spadafora, A., & Hunt, H. T. (1990). The multiplicity of dreams: Cognitive-affective correlates of lucid, archetypal and nightmare dreaming. *Perceptual and Motor Skills, 71,* 627-644.

Speroff, L., & Darney, P. D. (2011). *A clinical guide for contraception.* Philadelphia: Lippincott Williams & Wilkins.

Spitzer, R. L., Kroenke, K., Williams, J. B. W., & Lowe, B. (2006). A brief measure for assessing generalized anxiety disorder: The GAD-7. *Archives of Internal Medicine, 166,* 1092-1097.

Spoormaker, V. I., & Montgomery, P. (2008). Disturbed sleep in post-traumatic stress disorder: Secondary symptom or core feature? *Sleep Medicine Reviews, 12,* 169-184.

Spoormaker, V. I., Schredl, M., & van den Bout, J. (2006). Nightmares: From anxiety symptom to sleep disorder. *Sleep Medicine Reviews, 10,* 19-31.

Stein, M. B., Simmons, A. N., Feinstein, J. S., Paulus, M. P. (2007). Increased amygdala and insula activation during emotion processing in anxiety-prone subjects. *American Journal of Psychiatry, 164,* 318-327.

Stevens, J. R. (1973). An anatomy of schizophrenia? *Archives of General Psychiatry, 29,* 177-189.

Stevens, J. S., & Hamann, S. (2012). Sex differences in brain activation to emotional stimuli: A meta-analysis of neuroimaging studies. *Neuropsychologia, 50,* 1578-1593.

Stompe, T., Ritter, K., Ortwein-Swoboda, G., Schmid-Seigel, B., Zitterl, W., Strobl, R., & Schanda, H. (2003). Anxiety and hostility in the manifest dreams of schizophrenic patients. *Journal of Nervous and Mental Disease, 191,* 806-812.

Sundstrom-Poromaa, I., & Segebladh, B. (2012). Adverse mood symptoms with oral contraceptives. *Acta Obstetrica et Gynecologica Scandinavica, 91,* 420-427.

Suzuki, K., Miyamoto, T., Miyamoto, M., Suzuki, S., Watanabe, Y., Takashima, R., & Hirata, K. (2013). Dream-enacting behaviour is associated with impaired sleep and severe headache-related disability in migraine patients. *Cephalalgia, 33,* 868-878.

Swanson, E. M., & Foulkes, D. (1967). Dream content and the menstrual cycle. *Journal of Nervous and Mental Disease, 145,* 358-363.

Tamminga, C. A., Thaker, G. K., Buchanan, R., Kirkpatrick, B., Alphs, L. D., Chase, T. N., & Carpenter, W. T. (1992). Limbic system abnormalities identified in schizophrenia using positron emission tomography with fluorodeoxyglucose and neocortical alterations with deficit syndrome. *Archives of General Psychiatry, 49,* 522-530.

Teicher, M. H., Glod, C. A., Surrey, J., & Swett C. Jr. (1993). Early childhood abuse and limbic system ratings in adult psychiatric outpatients. *Journal of Neuropsychiatry and Clinical Neuroscience, 5,* 301-306.

Tennant, C., & Andrews, G. (1976). A scale to measure the stress of life events. *Australian and New Zealand Journal of Psychiatry, 10,* 27-32.

ter Horst, G. J. (2010). Estrogen in the limbic system. *Vitamins and Hormones, 82,* 319-338.

Torrey, E. F., & Peterson, M. R. (1974). Schizophrenia and the limbic system. *Lancet, II,* 942-946.

Trimble, M., & Freeman, A. (2006). An investigation of religiosity and the Gestaut-Geschwind syndrome in patients with temporal lobe epilepsy. *Epilepsy & Behavior, 9,* 407-414.

Tuiten, A., Panhuysen, G., Koppeschaar, H., Fekkes, D., Pijl, H., et al. (1995). Stress, serotonergic function, and mood in users of oral contraceptives. *Psychoneuroendocrinology, 20,* 323-334.

Tulving, E., Markowitsch, H. J., Craik, F. I. M., Habib, R., & Houle, S. (1996). Novelty and familiarity activations in PET studies of memory encoding and retrieval. *Cerebral Cortex, 6,* 71-79.

Tulving, E., Markowitsch, H. J., Kapur, S., & Habib, R. (1994). Novelty encoding networks in the human brain: Positron emission tomography data. *Neuroreport, 5,* 2525-2528.

Tunbridge, L., & Weinberg, M. (2014). Nightmares and homeostatis: When bad dreams fail to protect HPMood from anxiety. *International Journal of Dream Research, 7,* 14-22.

Turkington, C. (1996). *The brain encyclopedia.* New York, NY: Facts on File, Inc.

Valli, K., Frauscher, B., Peltomaa, T., Gschliesser, V., Revonsuo, A., & Hogl, B. (2015). Dreaming furiously? A sleep laboratory study on the dream content of people

with Parkinson's disease and with or without rapid eye movement sleep behavior disorder. *Sleep Medicine, 16,* 419-427.

Valli, K., Lenasdotter, S., MacGregor, O., & Revonsuo, A. (2007). A test of the threat simulation theory: Replication of results in an independent sample. *Sleep and Hypnosis, 9,* 30-46.

Valli, K., & Revonsuo, A. (2009). The threat simulation theory in light of recent empirical evidence: A review. *American Journal of Psychology, 122,* 17-38.

Valli, K., Revonsuo, A., Palkas, O., Ismail, K., Ali, K., & Punamaki, R. (2005). The threat simulation theory of the evolutionary function of dreaming: Evidence from dreams of traumatized children. *Consciousness and Cognition, 14,* 188-218.

Valli, K., Revonsuo, A., Palkas, O., & Punamaki, R. (2006). The effect of trauma on dream content – A field study of Palestinian children. *Dreaming, 16,* 63-87.

Valli, K., Strandholm, T., Sillanmaki, L., & Revonsuo, A. (2008). Dreams are more negative than real life: Implications for the function of dreaming. *Cognition & Emotion, 22,* 833-861.

van Elst, L. T., Woermann, F. G., Lemieux, L., Thompson, P. J., & Trimble, M. R. (2000). Affective aggression in patients with temporal lobe epilepsy: A quantitative MRI study of the amygdala. *Brain, 123,* 234-243.

van der Helm, E., Yao, J., Dutt, S., Rao, V., Saletin, J. M., & Walker, M. P. (2011). REM sleep depotentiates amydala activity to previous emotional experiences. *Current Biology, 21,* 2029-2032.

van der Kolk, B. A. (1994). The body keeps the score: Memory and the evolving psychobiology of posttraumatic stress. *Harvard Review of Psychiatry, 1,* 253-265.

van der Kolk, B. A. (2001). The psychobiology and psychopharmacology of PTSD. *Human Psychopharmacology: Clinical and Experimental, 16,* S49-S64.

van der Kolk, B., Blitz, R., Burr, W., Sherry, S., & Hartmann, E. (1984). Nightmares and trauma: A comparison of nightmares after combat with lifelong nightmares in veterans. *American Journal of Psychiatry, 141,* 187-190.

Van Hilten, J. J., Weggeman, M., Velde, E. A., van der Kerkhof, G. A., Dijk, J. C., & van Roos, R. A. C. (1993). Sleep, excessive daytime sleepiness and fatigue in Parkinson's disease. *Journal of Neural Transmission, 5,* 235-244.

van Os, J., & Kapur, S. (2009). Schizophrenia. *Lancet, 374,* 635-645.

van Schagen, A. M., Lancee, J., de Groot, I. W., Spoormaker, V. I., & van den Bout, J. (2015). Imagery rehearsal therapy in addition to treatment as usual for patients with diverse psychiatric diagnoses suffering from nightmares: A randomized controlled trial. *The Journal of Clinical Psychiatry, 76,* e1105-e1113.

Vgontzas, A., Cui, L., & Merikangas, K. R. (2008). Are sleep difficulties associated with migraine attributable to anxiety and depression? *Headache, 48,* 1451-1459.

Vignal, J., Maillard, L., McGonigal, A., & Chauvel, P. (2007). The dreamy state: Hallucinations of autobiographical memory evoked by temporal lobe stimulation and seizures. *Brain, 130,* 88-99.

Wager, T. D., Phan, K. L., Liberzon, I., & Taylor, S. F. (2003). Valence, gender, and lateralization of functional brain anatomy in emotion: A meta-analysis of findings from neuroimaging. *NeuroImage, 19,* 513-531.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54,* 1063-1070.

Waxman, S. G., & Geschwind, N. (1975). The interictal behavior syndrome of temporal lobe epilepsy. *Archives of General Psychiatry, 32,* 1580-1586.

Weinberg, M. K., Noble, J. M., & Hammond, T. G. (2015). Sleep well feel well: An investigation into the predictive value of sleep quality on subjective well-being. *Australian Journal of Psychology, 68,* 91-97.

Weisenbach, S. L., Rapport, L. J., Briceno, E. M., Haase, B. D., Vederman, A. C., Bieliauskas, L. A. … Langenecker, S. A. (2014). Reduced emotion processing efficiency in healthy males relative to females. *Social Cognitive and Affective Neuroscience, 9,* 316-325.

Weiss, D. S. (2007). Conundrums in a theory of disturbed dreaming: Comment on Levin and Nielsen (2007). *Psychological Bulletin, 133,* 529-532.

Wenning, G. K., Ben Shlomo, Y., Magalhaes, M., Daniel, S. E., & Quinn, N. P. (1994). Clinical features and natural history of multiple system atrophy: An analysis of 100 cases. *Brain, 117*(Pt 4), 835-845.

Wichianpitaya, J., & Taneepanichskul, S. (2013). A comparative efficacy of low-dose combined oral contraceptives containing desogestrel and drospirenone in premenstrual symptoms. *Obstetrics and Gynecology International, 2013,* 487143.

Wittmann, L., Schredl, M., & Kramer, M. (2007). Dreaming in posttraumatic stress disorder: A critical review of phenomenology, psychophysiology and treatment. *Psychotherapy and Psychosomatics, 76,* 25-39.

Wood, J. M., & Bootzin, R. R. (1990). The prevalence of nightmares and their independence from anxiety. *Journal of Abnormal Psychology, 99,* 64-68.

Wood, J. M., Bootzin, R. R., Rosenhan, D., Nolen-Hoeksema, S., & Jourden, F. (1992). Effects of the 1989 San Francisco earthquake on frequency and content of nightmares. *Journal of Abnormal Psychology, 101,* 219-224.

World Health Organization (2016). *International classification of diseases* (10th revision). Geneva, Switzerland: World Health Organization.

Xu, X., Hoebeke, J., & Bjorntorp, P. (1990). Progestin binds to the glucocorticoid receptor and mediates antiglucocorticoid effect in rat adipose precursor cells. *Journal of Steroid Biochemistry, 36,* 465-471.

Ylikoski, A., Martikainen, K., & Partinen, M. (2014). Parasomnias and isolated sleep symptoms in Parkinson's disease: A questionnaire study on 661 patients. *Journal of the Neurological Sciences, 346,* 204-208.

Yu, C. K.-C. (2010). Dream intensity profile as an indicator of the hysterical tendencies to dissociation and conversion. *Dreaming, 20,* 184-198.

Yu, C. K.-C. (2015). The vicissitudes of affective valence across the night: A high-density electroencephalographic study. *Dreaming, 25,* 274-290.

Yucel, M., Stuart, G. W., Maruff, P., Velakoulis, D., Crowe, S. F., Savage, G., & Pantelis, C. (2001). Hemispheric and gender-related differences in the gross morphology of the anterior cingulate/paracingulate cortex in normal volunteers: An MRI morphometric study. *Cerebral Cortex, 11,* 17-25.

Zadra, A. L. (1994). *Dimensions of repetition and negative affect in dreams and their relation to psychological well-being*. PhD dissertation, Department of Psychology, McGill University, Montreal, Canada.

Zadra, A., Desjardins, S., & Marcotte, E. (2006). Evolutionary function of dreams: A test of the threat simulation theory in recurrent dreams. *Consciousness & Cognition, 15,* 450-463.

Zadra, A. L., & Donderi, D. C. (2000). Nightmares and bad dreams: Their prevalence and relationship to well-being. *Journal of Abnormal Psychology, 109,* 273-281.

Zadra, A., Pilon, M., & Donderi, D. C. (2006). Variety and intensity of emotions in nightmares and bad dreams. *Journal of Nervous and Mental Disease, 194*(4), 249-254.

Zadra, A., & Robert, G. (2012). Dream recall frequency: Impact of prospective measures and motivational factors. *Consciousness & Cognition, 21,* 1695-1702.

APPENDIX A

DREAMING QUESTIONNAIRE (DRM-Q)

AND DREAM REPORT FORM

DREAMING QUESTIONNAIRE (DRM-Q)

1.) How many hours of sleep do you usually get a night?

_____hours

2.) How would you rate the quality of your sleep? "Quality of sleep" means how well you think you sleep at night.

1) Very bad [ ]
2) Bad [ ]
3) Average [ ]
4) Good [ ]
5) Very good [ ]

3.) About how many times do you usually wake up during the night?

1) Once [ ]
2) Twice [ ]
3) Three times [ ]
4) Four times [ ]
5) Five times [ ]
6) More than five times. Please say how many times: _____

4.) About how many dreams a week do you usually remember per week (e.g., 3 dreams a week)?

_____ dreams a week

5.) When you wake up in the morning, can you usually remember having:

1) 1 dream a night [ ]
2) 2 dreams a night [ ]
3) 3 dreams a night [ ]
4) 4 dreams a night [ ]
5) More than 4 dreams a night: Please say how many: _____

6.) When you wake up can you usually remember your dreams:

1) Easily [ ]

2) Without much effort [ ]

3) With some effort [ ]

4) With much effort [ ]

5) With great effort [ ]

7.) When you wake up do you usually remember your dreams:

1) Only as a fragment [ ]

2) With many missing parts [ ]

3) With some missing parts [ ]

4) Almost as a whole [ ]

5) Entirely [ ]

8.) How much colour would you say your dreams have?

1) No colour at all [ ]

2) A little bit of colour [ ]

3) A medium amount of colour [ ]

4) A lot of colour [ ]

5) A very large amount of colour [ ]

9.) How vivid would you say your dreams usually are?

1) Very unclear [ ]

2) Unclear [ ]

3) Average [ ]

4) Vivid [ ]

5) Very vivid [ ]

6) Extremely vivid [ ]

10.) How realistic would you say your dreams usually are?

1) Not at all realistic [ ]

2) A little bit realistic [ ]

3) Moderately realistic [ ]

4) Very realistic [ ]

5) Extremely realistic [ ]

11.) How intense would you say your dreams usually are?

1) Not at all intense [ ]

2) Slightly intense [ ]

3) Moderately intense [ ]

4) Very intense [ ]

5) Extremely intense [ ]

12.) How meaningful do you think your dreams usually are?

1) I think my dreams don't have any meaning at all [ ]

2) I think my dreams have a little bit of meaning to them [ ]

3) I think my dreams have a moderate amount of meaning to them [ ]

4) I think my dreams have a lot of meaning to them [ ]

5) I think my dreams have a very large amount of meaning to them [ ]

13.) How much movement is usually in your dreams?

1) There is usually no movement at all in my dreams [ ]

2) There is a little bit of movement in my dreams [ ]

3) There is a moderate amount of movement in my dreams [ ]

4) There is a lot of movement in my dreams [ ]

5) There is a very large amount of movement in my dreams [ ]

14.) How affected are you by your dreams after you have them?

1) My dreams do not affect me at all [ ]

2) My dreams affect me a little bit [ ]

3) My dreams affect me a moderate amount [ ]

4) My dreams affect me quite a lot [ ]

5) My dreams affect me to a very large amount [ ]

15.) About *how often* would you say you are aggressive towards people, animals, or other characters in your dreams?

1) Never [ ]

2) Sometimes [ ]

3) Often [ ]

4) Very often [ ]

5) Always [ ]

16.) About *how intense* would you say that your aggression towards people, animals, or other characters in your dreams is – regardless of how often you are aggressive in your dreams?

1) Not at all intense [ ]

2) Slightly intense [ ]

3) Moderately intense [ ]

4) Very intense [ ]

5) Extremely intense [ ]

17.) About how often would you say that *other characters* (e.g., people, animals, etc.) are aggressive *towards you* in your dreams?

1) Never [ ]

2) Sometimes [ ]

3) Often [ ]

4) Very often [ ]

5) Always [ ]

18.) About how intense would you say that the aggression of other characters (e.g., people, animals, etc.) is towards you in your dreams - regardless of how often other characters are aggressive in your dreams?

1) Not at all intense [ ]
2) Slightly intense [ ]
3) Moderately intense [ ]
4) Very intense [ ]
5) Extremely intense [ ]

19.) "Bad dreams" are disturbing dreams (e.g., frightening, anxious, distressing) in which the unpleasant visual imagery and/or emotions *do not cause you to wake up*. How often do you have bad dreams?

1) Never [ ]
2) Once every few years [ ]
3) Once a year [ ]
4) Once every several months [ ]
5) Once a month [ ]
6) More than once a month [ ]
7) Once a week [ ]
8) More than once a week [ ]

20.) Estimate the number of "bad dreams" you have had in the past month:

_____

21.) Estimate the number of "bad dreams" you have had in the past year:

_____

22.) How concerned or distressed are you over your "bad dreams?" (circle one of the numbers below, from a scale of 1 = very little to 7 = very much)

Very little.....Very much

1 2 3 4 5 6 7

23.) How much colour would you say your "bad dreams" have?

1) No colour at all [ ]

2) A little bit of colour [ ]

3) A medium amount of colour [ ]

4) A lot of colour [ ]

5) A very large amount of colour [ ]

24.) How vivid would you say your "bad dreams" usually are?

1) Very unclear [ ]

2) Unclear [ ]

3) Moderately vivid [ ]

4) Vivid [ ]

5) Very vivid [ ]

6) Extremely vivid [ ]

25.) How realistic would you say your "bad dreams" usually are?

1) Not at all realistic [ ]

2) A little bit realistic [ ]

3) Moderately realistic [ ]

4) Very realistic [ ]

5) Extremely realistic [ ]

26.) How intense would you say your "bad dreams" usually are?

1) Not at all intense [ ]

2) Slightly intense [ ]

3) Moderately intense [ ]

4) Very intense [ ]

5) Extremely intense [ ]

27.) How meaningful do you think your "bad dreams" usually are?

1) I think my bad dreams don't have any meaning at all [ ]

2) I think my bad dreams have a little bit of meaning to them [ ]

3) I think my bad dreams have a moderate amount of meaning to them [ ]

4) I think my bad dreams have a lot of meaning to them [ ]

5) I think my bad dreams have a very large amount of meaning to them [ ]

28.) How much movement is usually in your "bad dreams"?

1) There is usually no movement at all in my bad dreams [ ]

2) There is a little bit of movement in my bad dreams [ ]

3) There is a moderate amount of movement in my bad dreams [ ]

4) There is a lot of movement in my bad dreams [ ]

5) There is a very large amount of movement in my bad dreams [ ]

29.) How affected are you by your "bad dreams" after you have them?

1) My bad dreams do not affect me at all [ ]

2) My bad dreams affect me a little bit [ ]

3) My bad dreams affect me to a moderate extent [ ]

4) My bad dreams affect me quite a lot [ ]

5) My bad dreams affect me to a very large extent [ ]

30.) About *how often* would you say you are aggressive towards people, animals, or other characters in your bad dreams?

1) Never [ ]

2) Sometimes [ ]

3) Often [ ]

4) Very often [ ]

5) Always [ ]

31.) About *how intense* would you say that your aggression towards people, animals, or other characters in your bad dreams is – regardless of how often you are aggressive in your dreams?

1) Not at all intense [ ]
2) Slightly intense [ ]
3) Moderately intense [ ]
4) Very intense [ ]
5) Extremely intense [ ]

32.) About how often would you say that *other characters* (e.g., people, animals, etc.) are aggressive *towards you* in your bad dreams?

1) Never [ ]
2) Sometimes [ ]
3) Often [ ]
4) Very often [ ]
5) Always [ ]

33.) About how intense would you say that the aggression of other characters (e.g., people, animals, etc.) is towards you in your bad dreams - regardless of how often other characters are aggressive in your bad dreams?

1) Not at all intense [ ]
2) Slightly intense [ ]
3) Moderately intense [ ]
4) Very intense [ ]
5) Extremely intense [ ]

34.) "Nightmares" are disturbing dreams (e.g., frightening, anxious, distressing) in which the unpleasant visual imagery and/or emotions *wake you up*. How often do you have nightmares?

1) Never [ ]
2) Once every few years [ ]
3) Once a year [ ]
4) Once every several months [ ]
5) Once a month [ ]
6) More than once a month [ ]
7) Once a week [ ]
8) More than once a week [ ]

35.) Estimate the number of nightmares you have had in the past month:

_____

36.) Estimate the number of "nightmares" you have had in the past year:

_____

37.) How concerned or distressed are you over your nightmares (circle one of the numbers below, from a scale of 1 = very little to 7 = very much)

Very little.....Very much

1 2 3 4 5 6 7

38.) How much colour would you say your nightmares have?

1) No colour at all [ ]
2) A little bit of colour [ ]
3) A medium amount of colour [ ]
4) A lot of colour [ ]
5) A very large amount of colour [ ]

39.) How vivid would you say your nightmares usually are?

1) Very unclear [ ]

2) Unclear[ ]

3) Average [ ]

4) Vivid [ ]

5) Very vivid [ ]

6) Extremely vivid [ ]

40.) How realistic would you say your nightmares usually are?

1) Not at all realistic [ ]

2) A little bit realistic [ ]

3) Moderately realistic [ ]

4) Very realistic [ ]

5) Extremely realistic [ ]

41.) How intense would you say your nightmares usually are?

1) Not at all intense [ ]

2) Slightly intense [ ]

3) Moderately intense [ ]

4) Very intense [ ]

5) Extremely intense [ ]

42.) How meaningful do you think your nightmares usually are?

1) I think my nightmares don't have any meaning at all [ ]

2) I think my nightmares have a little bit of meaning to them [ ]

3) I think my nightmares have a moderate amount of meaning to them [ ]

4) I think my nightmares have a lot of meaning to them [ ]

5) I think my nightmares have a very large amount of meaning to them [ ]

43.) How much movement is usually in your nightmares?

1) There is usually no movement at all in my nightmares [ ]

2) There is a little bit of movement in my nightmares [ ]

3) There is a moderate amount of movement in my nightmares [ ]

4) There is a lot of movement in my nightmares [ ]

5) There is a very large amount of movement in my nightmares [ ]

44.) How affected are you by your nightmares after you have them?

1) My nightmares do not affect me at all [ ]

2) My nightmares affect me a little bit [ ]

3) My nightmares affect me to a moderate extent [ ]

4) My nightmares affect me quite a lot [ ]

5) My nightmares affect me to a very large extent [ ]

45.) About *how often* would you say you are aggressive towards people, animals, or other characters in your nightmares?

1) Never [ ]

2) Sometimes [ ]

3) Often [ ]

4) Very often [ ]

5) Always [ ]

46.) About *how intense* would you say that your aggression towards people, animals, or other characters in your nightmares is – regardless of how often you are aggressive in your dreams?

1) Not at all intense [ ]

2) Slightly intense [ ]

3) Moderately intense [ ]

4) Very intense [ ]

5) Extremely intense [ ]

47.) About how often would you say that *other characters* (e.g., people, animals, etc.) are aggressive *towards you* in your nightmares?

1) Never [ ]
2) Sometimes [ ]
3) Often [ ]
4) Very often [ ]
5) Always [ ]

48.) About how intense would you say that the aggression of other characters (e.g., people, animals, etc.) is towards you in your bad dreams - regardless of how often other characters are aggressive in your nightmares?

1) Not at all intense [ ]
2) Slightly intense [ ]
3) Moderately intense [ ]
4) Very intense [ ]
5) Extremely intense [ ]

49.) Have you ever had a recurrent dream? A recurrent dream is a dream that when you remember it leaves you with the subjective feeling of having had it before.

1) No [ ]
2) Yes [ ]
3) Uncertain [ ]

50.) Have you had a recurrent dream in the past twelve months?

1) No [ ]
2) Yes [ ]
3) Uncertain [ ]

51.) Have you had a recurrent dream in the past six months?

1) No [ ]
2) Yes [ ]
3) Uncertain [ ]

DREAM REPORT FORM

Date Today: _____

Time that you woke up:_____

Time of writing this report:_____

If you had any dreams last night, please describe the dreams exactly and as fully as you remember it. Your report should contain, whenever possible: A description of the setting of the dream, whether it was familiar to you or not; A description of the people, their age, sex, and relationship to you; And any animals that appeared in the dream. If possible describe your feelings during the dream and whether it was pleasant or unpleasant. Be sure to tell exactly what happened during the dream to you and the other characters. Continue your report on the other side and on additional sheets if necessary.

APPENDIX B

DREAM DIARY STUDY INSTRUCTIONS AND DREAM RECORD FORM

INSTRUCTION SHEET FOR PARTICIPANTS
DREAM DIARY STUDY

Dear participant

Thank you for taking the time and interest to take part in the dream diary study. Below are the instructions for how to fill out the dream diary, and what to do once you have completed the diary. If you have any questions regarding the instructions or any other aspect of the study, please feel free to contact Warren King, at warren.g.king@gmail.com.

Confidentiality

Each participant in the study is assigned a participant number, which is how you will be identified in the dataset. All responses and/or materials that you submit for this study will be filed under this number. If you are a UCT student, we need to know your name and student number only so that we can assign you your SRPP points, and so that we can match your responses to your name and student number when entering the data. If you are a person with epilepsy, we need to know your name only so that we can match your responses to the information contained in your medical folder when entering the data. After the data is entered, you will be identified only by your participant number. All responses and medical information will be treated with strict confidentiality, will be used only for the purposes of this study, and will be known only to the principal investigator, Warren King.

Instructions for the dream diary study

For this study, you will be provided with materials for you to write down your dreams over a period of 21 days. There are also a few simple questions you need to answer before and after writing down your dream on any particular day. It is very important that you begin recording your dreams on the first day after you have a dream once you have been given the materials, and not on the day you are given the materials. For example, if you are given the materials on a Tuesday, and the next time you have a dream is the Thursday night after this, you will start writing down your dreams for a period of 21 days starting from the Friday morning.

The number of dreams a person has and remembers varies a lot from time to time, and from person to person. If you only have or remember a few dreams during the 21 day period, do not worry – just write down as many dreams as you have or can remember, only if it is only 3 or 4 (or even less). If you think you have had more than one dream on any one night, please write down as many of these dreams that you remember the following morning, with the headings: "DREAM 1, DREAM2," etc.

Things that can enhance your dream recall include keeping the dream record forms at your bedside, where they are easily accessible during the night or when you wake up in the morning. Any distractions from the outside world can get in the way of you remembering your dreams. It may help if when you first wake up in the morning, you lie quietly for a few minutes with your eyes closed and concentrate on any images or feelings that come to you. If you don't remember a dream right away, often parts of it will come back to you if you keep your attention focused on inner imagery and avoid thinking about what you have to do that day.

Take enough time in the morning to complete the dream record form right away, even if all you remember is a vague image. You can always add to the record form later. If you don't write down your dream right away, the image may fade rapidly and you might not be able to recall it later. If you recall having a dream the night before but cannot remember it or did not write it down for whatever reason, please indicate this on the question on the record form asking this, and then write down the reason. For example, if you remember having a dream but did not write it down because you forgot to and could not remember the details of the dream later, please write something like: "Forgot to write down dream in the morning, could not remember it later on."

Finally, do not worry if you do not have or cannot remember even one dream over the 21 day period. If you do not have any dreams over this period, this information is as important to us as if you had dreamt 50 dreams over this period – SO PLEASE DO NOT MAKE UP DREAMS!

Recording your dreams

For each day of the 21 day period, you will need to at least answer the question about whether or not you have had a dream the previous night. If you have had a dream the previous night, there are a few additional simple questions you will need to answer. Finally, you will need to write down a report of your dream (or dreams, if you have had more than one). There are specific instructions about how to write down your dream report, so please follow these closely. These instructions are repeated on every sheet of the dream diary materials.

Returning the research materials

At the end of the 21 days these can be dropped off with Warren King in Room 4.28 of the Psychology Department at UCT, on the 4th floor of the Graduate School of Humanities Building.

Contact details

For any queries please contact the principal researcher, Warren King.

Email: warren.g.king@gmail.com.

Tel.: 083 777 2508

DREAM DIARY STUDY – DEMOGRAPHIC INFORMATION

PARTICIPANT NUMBER: _____

TEL: _____

AGE: _____ **GENDER: Male/Female**

RACE: _____

STUDENT NUMBER: _____

DREAM RECORD FORM

Date today: _____

1.) Did you have a dream last night (please circle)?

1. Yes 2. No

If you had a dream last night, please write down this dream in the space below. But first please tell us the following information:

Time that you woke up: _____

Time of writing this report: _____

Please describe the dream exactly and as fully as you remember it. Your report should contain, whenever possible: A description of the setting of the dream, whether it was familiar to you or not; A description of the people, their age, sex, and relationship to you; And any animals that appeared in the dream. If possible describe your feelings during the dream and whether it was pleasant or unpleasant. Be sure to tell exactly what happened during the dream to you and the other characters. Continue your report on the other side and on additional sheets if necessary.

Thank you for writing down your report! If you had a dream last night, please now answer the following additional questions:

1.) Was this dream a bad dream (please circle)? A bad dream is a very disturbing dream which, although being unpleasant, *does NOT cause you to wake up.*

1. Yes 2. No

2.) Was this dream a nightmare (please circle)? A nightmare is a very disturbing dream in which the unpleasant visual imagery and/or emotions *wake you up*.

1. Yes 2. No

APPENDIX C

ADDITIONAL STATISTICAL RESULTS

Table C1.

Stepwise Regression Results for TDQ Components Predictive of Bad Dream and Nightmare Recall Frequency

				Change Statistics				Model Statistics		
Step	**Model Variables**	R^2	***SEE***	ΔR^2	***ΔF***	***df***	***p***	***F***	***df***	***p***
	BD Frequency									
1	Death-murder	.13	1.37	.13	76.73	1, 524	< .001	76.73	1, 524	< .001
2	Death-murder; Flying-chase	.16	1.34	.03	21.15	1, 523	< .001	50.41	2, 523	< .001
3	Death-murder; Flying-chase; Disasters	.17	1.33	.01	6.28	1, 522	.013	36.04	3, 522	< .001
	NM Frequency									
1	Death-murder	.11	0.11	.11	63.54	1, 524	< .001	63.54	1, 524	< .001
2	Death-murder; Flying-chase	.15	0.14	.04	22.34	1, 523	<. 001	44.23	2, 523	< .001
3	Death-murder; Flying-chase; Disasters	.16	0.15	.01	7.96	1, 522	.005	32.53	3, 522	< .001
4	Death-murder; Flying-chase; Disasters; Inhibition	.17	0.16	.01	5.44	1, 521	.020	25.97	4, 521	< .001

Table C2.

Model Coefficients for TDQ Components Predictive of Bad Dream and Nightmare Recall Frequency

							95% CIs	
Model	**Model Variables**	***b***	***SE***	**β**	***t***	***p***	Lower	Upper
	BD Frequency							
1	Death-murder	0.15	0.02	0.36	8.76	< .001	0.12	0.19
2	Death-murder	0.12	0.02	0.29	6.70	< .001	0.09	0.16
	Flying-chase	0.08	0.02	0.20	4.60	< .001	0.05	0.11
3	Death-murder	0.11	0.02	0.25	5.59	< .001	0.07	0.14
	Flying-chase	0.07	0.02	0.16	3.65	< .001	0.03	0.10
	Disasters	0.06	0.02	0.12	2.51	.013	0.01	0.11
	NM Frequency							
1	Death-murder	0.16	0.02	0.33	7.97	< .001	0.12	0.20
2	Death-murder	0.12	0.02	0.26	5.91	< .001	0.08	0.16
	Flying-chase	0.09	0.02	0.21	4.73	< .001	0.05	0.13
3	Death-murder	0.10	0.02	0.22	4.74	< .001	0.06	0.15
	Flying-chase	0.08	0.02	0.17	3.68	< .001	0.04	0.12
	Disasters	0.08	0.03	0.13	2.82	.005	0.02	0.13
4	Death-murder	0.09	0.02	0.18	3.68	< .001	0.04	0.13
	Flying-chase	0.06	0.02	0.13	2.66	.008	0.02	0.10
	Disasters	0.07	0.03	0.12	2.51	.012	0.02	0.12
	Inhibition	0.05	0.02	0.12	2.33	.020	0.01	0.08

Table C3.

Summary of Analyses of the Moderating Effect of Oral Contraceptives on Bad Dream Recall Frequency

				Change Statistics				Model Statistics		
Step	**Model Variables**	R^2	***SEE***	ΔR^2	ΔF	***df***	Δp	***F***	***df***	***p***
	CORE									
1	OCs use; CORE	.10	0.72	.10	9.29	2, 168	< .001***	9.29	2, 168	< .001***
2	OCs use; CORE; OCs use X CORE	.10	0.72	< .01	0.23	1, 167	.630	6.24	3, 167	< .001***
	PSS									
1	OCs use; PSS	.09	0.72	.09	8.08	2, 168	< .001***	8.08	2, 168	< .001***
2	OCs use; PSS; OCs use X PSS	.09	0.73	< .01	0.05	1, 167	.828	5.37	3, 167	< .001***
	GAD-7									
1	OCs use; GAD-7	.11	0.71	.11	10.70	2, 168	< .001***	10.70	2, 168	< .001***
2	OCs use; GAD-7; OCs use X GAD-7	.12	0.72	< .01	0.34	1, 167	.561	7.22	3, 167	< .001***
	PANAS PA									
1	OCs use; PANAS PA	.02	0.75	.02	1.61	2, 168	.203	1.61	2, 168	.203
2	OCs use; PANAS PA; OCs use X PANAS PA	.02	0.75	< .01	0.04	1, 167	.839	1.08	3, 167	.358
	PANAS NA									
1	OCs use; PANAS NA	.07	0.73	.07	6.70	2, 168	.002**	6.70	2, 168	.002**
2	OCs use; PANAS NA; OCs use X PANAS NA	.08	0.73	< .01	0.47	1, 167	.493	4.61	3, 167	.004**
	CSI PFE									
1	OCs use; CSI PFE	.01	0.76	.01	0.45	2, 168	.640	0.45	2, 168	.640
2	OCs use; CSI EFE; OCs use X CSI EFE	.01	0.76	< .01	0.43	1, 167	.511	0.44	3, 167	.724
	CSI PFD									
1	OCs use; CSI PFD	.03	0.74	.03	2.65	2, 168	.074	2.65	2, 168	.074
2	OCs use; CSI PFD; OCs use X CSI PFD	.03	0.75	< .01	0.32	1, 167	.576	1.86	3, 167	.138
	CSI EFE									
1	OCs use; CSI EFE	.01	0.75	.01	0.96	2, 168	.385	0.96	2, 168	.385
2	OCs use; CSI EFE; OCs use X CSI EFE	.01	0.75	< .01	< 0.01	1, 167	.957	0.64	3, 167	.592
	CSI EFD									
1	OCs use; CSI EFD	.05	0.74	.05	4.78	2, 168	.010**	4.78	2, 168	.010**
2	OCs use; CSI EFD; OCs use X CSI EFD	.05	0.74	< .01	0.11	1, 167	.747	3.21	3, 167	.025*
	CSI ENG									
1	OCs use; CSI ENG	.01	0.75	.01	1.14	2, 168	.322	1.14	2, 168	.322
2	OCs use; CSI ENG; OCs use X CSI ENG	.02	0.75	< .01	0.30	1, 167	.583	0.86	3, 167	.464
	CSI DISENG									
1	OCs use; CSI ENG	< .01	0.76	< .01	0.22	2, 168	.806	0.22	2, 168	.806
2	OCs use; CSI ENG; OCs use X CSI ENG	< .01	0.76	< .01	0.55	1, 167	.460	0.33	3, 167	.806

*P < .05; **P < .01; ***P < .001

Table C4.
Model Coefficients for Variables Predictive of Bad Dream Recall Frequency, Part 1

							95% CIs	
Model	**Model Variables**	***b***	***SE***	**β**	***t***	***p***	Lower	Upper
	CORE							
1	OCs use	0.07	0.07	0.08	1.02	.310	-0.06	0.20
	CORE	0.02	0.01	0.31	4.26	< .001***	0.01	0.03
2	OCs use	0.07	0.07	0.08	1.02	.309	-0.06	0.20
	CORE	0.02	0.01	0.36	3.89	< .001***	0.01	0.03
	OCs use X CORE	0.00	0.01	0.04	0.48	.630	-0.01	0.01
	PSS							
1	OCs use	0.06	0.07	0.06	0.82	.413	-0.08	0.19
	PSS	0.03	0.01	0.29	3.97	< .001***	0.02	0.04
2	OCs use	0.05	0.07	0.06	0.81	.420	-0.08	0.19
	PSS	0.03	0.01	0.30	3.34	< .001***	0.01	0.05
	OCs use X PSS	0.00	0.01	0.02	0.22	.828	-0.02	0.02
	GAD-7							
1	OCs use	0.06	0.07	0.06	0.89	.373	-0.07	0.19
	GAD-7	0.05	0.01	0.33	4.58	< .001***	0.03	0.07
2	OCs use	0.06	0.07	0.06	0.88	.378	-0.07	0.19
	GAD-7	0.06	0.01	0.37	3.94	< .001***	0.03	0.09
	OCs use X GAD-7	0.01	0.01	0.05	0.58	.561	-0.02	0.04
	PANAS PA							
1	OCs use	0.04	0.07	0.04	0.55	.581	-0.10	0.17
	PANAS PA	-0.01	0.01	-0.13	-1.69	.093	-0.03	0.00
2	OCs use	0.04	0.07	0.04	0.52	.601	-0.10	0.17
	PANAS PA	-0.01	0.01	-0.14	-1.44	.151	-0.03	0.01
	OCs use X PANAS PA	0.00	0.01	-0.02	-0.20	.839	-0.02	0.02
	PANAS NA							
1	OCs use	0.07	0.07	0.07	0.97	.335	-0.07	0.20
	PANAS NA	0.03	0.01	0.27	3.61	< .001***	0.01	0.04
2	OCs use	0.06	0.07	0.07	0.92	.357	-0.07	0.20
	PANAS NA	0.03	0.01	0.31	3.21	.002**	0.01	0.05
	OCs use X PANAS NA	0.01	0.01	0.07	0.69	.493	-0.01	0.02

*P < .05; **P < .01; ***P < .001

Table C5.
Model Coefficients for Variables Predictive of Bad Dream Recall Frequency, Part 2

Model	Model Variables	*b*	*SE*	β	*t*	*p*	95% CIs	
							Lower	Upper
	CSI PFE							
1	OCs use	0.04	0.07	0.04	0.52	.605	-0.10	0.17
	CSI PFE	0.01	0.02	0.06	0.73	.467	-0.02	0.05
2	OCs use	0.03	0.07	0.03	0.40	.688	-0.11	0.17
	CSI PFE	0.02	0.02	0.10	0.98	.328	-0.02	0.07
	OCs use X CSI PFE	0.02	0.02	0.07	0.66	.511	-0.03	0.06
	CSI PFD							
1	OCs use	0.05	0.07	0.06	0.76	.446	-0.08	0.19
	CSI PFD	-0.05	0.02	-0.17	-2.22	.028*	-0.09	-0.01
2	OCs use	0.05	0.07	0.06	0.79	.433	-0.08	0.19
	CSI PFD	-0.06	0.03	-0.20	-2.11	.036*	-0.11	0.00
	OCs use X CSI PFD	-0.01	0.03	-0.05	-0.56	.576	-0.07	0.04
	CSI EFE							
1	OCs use	0.05	0.07	0.06	0.72	.474	-0.09	0.19
	CSI EFE	0.02	0.02	0.10	1.25	.214	-0.01	0.06
2	OCs use	0.05	0.07	0.06	0.71	.477	-0.09	0.19
	CSI EFE	0.02	0.02	0.09	1.14	.258	-0.02	0.07
	OCs use X CSI EFE	0.00	0.02	0.00	-0.05	.957	-0.04	0.04
	CSI EFD							
1	OCs use	0.05	0.07	0.05	0.72	.472	-0.09	0.18
	CSI EFD	0.08	0.03	0.23	3.03	.003**	0.03	0.13
2	OCs use	0.05	0.07	0.05	0.70	.485	-0.09	0.18
	CSI EFD	0.08	0.04	0.25	2.26	.025*	0.01	0.16
	OCs use X CSI EFD	0.01	0.04	0.04	0.32	.747	-0.06	0.09
	CSI ENG							
1	OCs use	0.04	0.07	0.04	0.57	.568	-0.10	0.18
	CSI ENG	0.02	0.01	0.11	1.39	.168	-0.01	0.04
2	OCs use	0.04	0.07	0.04	0.53	.594	-0.10	0.17
	CSI ENG	0.02	0.02	0.14	1.45	.149	-0.01	0.05
	OCs use X CSI ENG	0.01	0.02	0.05	0.55	.583	-0.02	0.04
	CSI DISENG							
1	OCs use	0.04	0.07	0.05	0.59	.556	-0.10	0.18
	CSI DISENG	0.01	0.02	0.02	0.27	.790	-0.03	0.05
2	OCs use	0.05	0.07	0.05	0.65	.518	-0.09	0.18
	CSI DISENG	-0.01	0.03	-0.02	-0.24	.807	-0.06	0.04
	OCs use X CSI DISENG	-0.02	0.03	-0.07	-0.74	.460	-0.07	0.03

*P < .05; **P < .01; ***P < .001

Table C6.

Summary of Analyses of the Moderating Effect of Oral Contraceptives on Nightmare Recall Frequency.

				Change Statistics				Model Statistics		
Step	**Model Variables**	R^2	***SEE***	ΔR^2	ΔF	***df***	Δp	***F***	***df***	***p***
	CORE									
1	OCs use; CORE	.12	1.20	.12	11.15	2, 166	< .001***	11.25	2, 166	< .001***
2	OCs use; CORE; OCs use X CORE	.12	1.20	< .01	0.06	1, 165	.805	6.24	3, 165	< .001***
	PSS									
1	OCs use; PSS	.12	1.20	.12	11.66	2, 166	< .001***	11.66	2, 166	< .001***
2	OCs use; PSS; OCs use X PSS	.13	1.20	< .01	0.36	1, 165	.552	7.86	3, 165	< .001***
	GAD-7									
1	OCs use; GAD-7	.15	1.18	.15	14.94	2, 166	< .001***	14.94	2, 166	< .001***
2	OCs use; GAD-7; OCs use X GAD-7	.15	1.18	< .01	< .01	1, 165	.977	9.90	3, 165	< .001***
	PANAS PA									
1	OCs use; PANAS PA	.02	1.27	.02	1.68	2, 166	.190	1.68	2, 166	.190
2	OCs use; PANAS PA; OCs use X PANAS PA	.04	1.26	.02	2.78	1, 165	.097	2.06	3, 165	.108
	PANAS NA									
1	OCs use; PANAS NA	.11	1.20	.12	11.79	2, 166	< .001***	11.79	2, 166	< .001***
2	OCs use; PANAS NA; OCs use X PANAS NA	.11	1.20	< .01	0.03	1, 165	.854	7.83	3, 165	< .001***
	CSI PFE									
1	OCs use; CSI PFE	.01	1.27	.01	1.02	2, 166	.364	1.02	2, 166	.364
2	OCs use; CSI EFE; OCs use X CSI EFE	.01	1.28	< .01	0.18	1, 165	.669	0.74	3, 165	.533
	CSI PFD									
1	OCs use; CSI PFD	.03	1.26	.03	2.19	2, 166	.115	2.19	2, 166	.115
2	OCs use; CSI PFD; OCs use X CSI PFD	.03	1.27	< .01	0.18	1, 165	.676	1.51	3, 165	.213
	CSI EFE									
1	OCs use; CSI EFE	.08	1.23	.08	6.72	2, 166	.002**	6.72	2, 166	.002**
2	OCs use; CSI EFE; OCs use X CSI EFE	.08	1.23	< .01	0.19	1, 165	.660	4.52	3, 165	.004**
	CSI EFD									
1	OCs use; CSI EFD	.03	1.26	.03	2.80	2, 166	.064	2.80	2, 166	.064
2	OCs use; CSI EFD; OCs use X CSI EFD	.03	1.26	< .01	0.10	1, 165	.919	1.86	3, 165	.138
	CSI ENG									
1	OCs use; CSI ENG	.07	1.23	.07	6.44	2, 166	.002**	6.44	2, 166	.002**
2	OCs use; CSI ENG; OCs use X CSI ENG	.07	1.24	< .01	0.10	1, 165	.753	4.30	3, 165	.006**
	CSI DISENG									
1	OCs use; CSI ENG	< .01	1.28	< .01	0.00	2, 166	.998	0.00	2, 166	.998
2	OCs use; CSI ENG; OCs use X CSI ENG	< .01	1.28	< .01	0.55	1, 165	.459	0.19	3, 165	.907

$^*P < .05$; $^{**}P < .01$; $^{***}P < .001$

Table C8.
Model Coefficients for Variables Predictive of Nightmare Recall Frequency, Part 2

							95% CIs	
Model	**Model Variables**	***b***	***SE***	**β**	***t***	***p***	Lower	Upper
	CSI PFE							
1	OCs use	-0.02	0.12	-0.01	-0.19	.852	-0.26	0.21
	CSI PFE	0.04	0.03	0.11	1.43	.156	-0.02	0.10
2	OCs use	-0.01	0.12	-0.01	-0.10	.918	-0.25	0.23
	CSI PFE	0.03	0.04	0.08	0.84	.400	-0.04	0.11
	OCs use X CSI PFE	-0.02	0.04	-0.04	-0.43	.669	-0.09	0.06
	CSI PFD							
1	OCs use	0.03	0.12	0.02	0.22	.829	-0.21	0.26
	CSI PFD	-0.08	0.04	-0.16	-2.09	.038*	-0.15	0.00
2	OCs use	0.03	0.12	0.02	0.29	.774	-0.20	0.27
	CSI PFD	-0.09	0.04	-0.18	-1.99	.048*	-0.18	0.00
	OCs use X CSI PFD	-0.02	0.04	-0.04	-0.42	.676	-0.11	0.07
	CSI EFE							
1	OCs use	0.04	0.11	0.02	0.32	.746	-0.19	0.26
	CSI EFE	0.12	0.03	0.27	3.67	< .001***	0.05	0.18
2	OCs use	0.03	0.11	0.02	0.30	.765	-0.19	0.26
	CSI EFE	0.11	0.03	0.26	3.19	.002**	0.04	0.18
	OCs use X CSI EFE	-0.02	0.03	-0.04	-0.44	.660	-0.08	0.05
	CSI EFD							
1	OCs use	0.00	0.12	0.00	0.00	.997	-0.23	0.23
	CSI EFD	0.10	0.04	0.18	2.37	.019*	0.02	0.19
2	OCs use	0.00	0.12	0.00	0.00	1.000	-0.23	0.23
	CSI EFD	0.11	0.06	0.19	1.67	.097	-0.02	0.23
	OCs use X CSI EFD	0.01	0.06	0.01	0.10	.919	-0.12	0.13
	CSI ENG							
1	OCs use	-0.01	0.11	-0.01	-0.11	.912	-0.24	0.21
	CSI ENG	0.08	0.02	0.27	3.59	< .001***	0.03	0.12
2	OCs use	-0.01	0.11	-0.01	-0.09	.929	-0.24	0.22
	CSI ENG	0.07	0.03	0.25	2.85	.004**	0.02	0.12
	OCs use X CSI ENG	-0.01	0.03	-0.03	-0.32	.753	-0.06	0.04
	CSI DISENG							
1	OCs use	0.00	0.12	0.00	-0.01	.994	-0.23	0.23
	CSI DISENG	0.00	0.03	-0.01	-0.07	.948	-0.07	0.06
2	OCs use	0.01	0.12	0.01	0.10	.920	-0.22	0.25
	CSI DISENG	-0.02	0.04	-0.04	-0.45	.650	-0.10	0.06
	OCs use X CSI DISENG	-0.03	0.04	-0.07	-0.74	.459	-0.11	0.05

*P < .05; **P < .01; ***P < .001

Table C7.
Model Coefficients for Variables Predictive of Nightmare Recall Frequency, Part 1

							95% CIs	
Model	**Model Variables**	***b***	***SE***	**β**	***t***	***p***	Lower	Upper
	CORE							
1	OCs use	0.06	0.11	0.04	0.50	.619	-0.16	0.28
	CORE	0.04	0.01	0.35	4.74	< .001***	0.02	0.05
2	OCs use	0.06	0.11	0.04	0.52	.607	-0.16	0.28
	CORE	0.04	0.01	0.36	4.27	< .001***	0.02	0.06
	OCs use X CORE	0.00	0.01	0.02	0.25	.805	-0.02	0.02
	PSS							
1	OCs use	0.03	0.11	0.02	0.28	.778	-0.19	0.25
	PSS	0.06	0.01	0.35	4.83	< .001***	0.03	0.08
2	OCs use	0.04	0.11	0.02	0.32	.752	-0.18	0.25
	PSS	0.06	0.01	0.38	4.42	< .001***	0.03	0.09
	OCs use X PSS	0.01	0.01	0.05	0.60	.552	-0.02	0.04
	GAD-7							
1	OCs use	0.04	0.11	0.03	0.39	.701	-0.17	0.26
	GAD-7	0.11	0.02	0.39	5.47	< .001***	0.07	0.14
2	OCs use	0.04	0.11	0.03	0.38	.705	-0.18	0.26
	GAD-7	0.11	0.02	0.39	4.33	< .001***	0.06	0.15
	OCs use X GAD-7	0.00	0.02	0.00	-0.03	.977	-0.05	0.05
	PANAS PA							
1	OCs use	0.00	0.12	0.00	-0.02	.983	-0.23	0.23
	PANAS PA	-0.03	0.01	-0.14	-1.83	.069	-0.05	0.00
2	OCs use	-0.01	0.12	-0.01	-0.08	.940	-0.24	0.22
	PANAS PA	-0.04	0.02	-0.23	-2.46	.015**	-0.07	-0.01
	OCs use X PANAS PA	-0.03	0.02	-0.16	-1.67	.097	-0.06	0.01
	PANAS NA							
1	OCs use	0.03	0.11	0.02	0.27	.791	-0.19	0.25
	PANAS NA	0.06	0.01	0.35	4.86	< .001***	0.03	0.08
2	OCs use	0.03	0.11	0.02	0.25	.799	-0.19	0.25
	PANAS NA	0.06	0.01	0.34	3.76	< .001***	0.03	0.09
	OCs use X PANAS NA	0.00	0.01	-0.02	-0.18	.854	-0.03	0.03

*P < .05; **P < .01; ***P < .001

Table C9.

Results of Mann-Whitney U *Tests on Dream Recall Frequency Variables Between the Upper and Lower Quartiles*

	Mean rank			
Variable	Lower Quartile (n = 10)	Upper Quartile (n = 12)	U	p (exact)
TOTAL DREAMS (All types)	**7.80**	**14.58**	**97.00**	**.014**
Normal dreams	**7.75**	**14.62**	**97.50**	**.011**
Bad dreams	10.80	12.08	67.00	.674
Nightmares	10.55	12.29	69.50	.539
Nights with dreams[a]	**7.60**	**14.75**	**99.00**	**.009**

[a]Number of nights with at least one dream of any type.

END