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Nightmares, Bad Dreams, and Emotion Dysregulation

A Review and New Neurocognitive Model of Dreaming

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ABSTRACT—*Nightmares—vivid, emotionally dysphoric dreams—are quite common and are associated with a broad range of psychiatric conditions. However, the origin of such dreams remains largely unexplained, and there have been no attempts to reconcile repetitive traumatic nightmares with nontraumatic nightmares, dysphoric dreams that do not awaken the dreamer, or with more normative dreams. Based on recent research in cognitive neuroscience, sleep physiology, fear conditioning, and emotional-memory regulation, we propose a multilevel neurocognitive model that unites waking and sleeping as a conceptual framework for understanding a wide spectrum of disturbed dreaming. We propose that normal dreaming serves a fear-extinction function and that nightmares reflect failures in emotion regulation. We further suggest that nightmares occur as a result of two processes that we term affect load—a consequence of daily variations in emotional pressures—and affect distress—a disposition to experience events with high levels of negative emotional reactivity.*

KEYWORDS—*nightmares; dreaming; emotion regulation*

Nightmares—vivid and highly emotionally dysphoric dreams that awaken the individual from sleep—are among the most commonly experienced sleep disorders (for extensive reviews of this literature, see Levin & Nielsen, 2007; Nielsen & Levin, 2007). While fear and terror are the predominant emotions associated with nightmares, other emotions such as rage are not uncommon (Zadra, Pilon, & Donderi, 2006). Like most dreams, nightmares typically occur during rapid-eye-movement (REM) sleep.

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Nightmares typically imply nocturnal awakening (Levin & Nielsen, 2007), whereas bad dreams are usually defined as negatively toned dreams that do not awaken the dreamer (Levin & Nielsen, 2007; Zadra & Donderi, 2000; Zadra et al., 2006). Despite phenomenological similarities between nightmares and bad dreams, it remains unknown whether they are two qualitatively distinct phenomena or a single phenomenon varying in intensity. We suggest that bad dreams involve similar processes and merely differ in how effective (or ineffective) they are in regulating shifting surges of current affect levels, a process we refer to as regulating affect load (see below for further discussion). Accordingly, we use the term disturbed dreaming (DD) when referring to both nightmares and bad dreams.

PREVALENCE AND DEMOGRAPHIC CHARACTERISTICS OF DD

Occasional episodes of DD are ubiquitous in the general population. Epidemiological studies indicate that about 85% of adults report experiencing at least one nightmare within the previous year (Levin, 1994), with about 2 to 6% of respondents reporting weekly nightmares. Furthermore, nightmare incidence is reported at significantly higher rates in younger adults starting at age 14, in women after age 14, and in clinical populations (Levin, 1994; Nielsen, Stenstrom, & Levin, 2006). As nightmares are rarely reported spontaneously as clinical problems or inquired about in routine health screenings, true prevalence rates are likely higher. In addition, retrospective reporting significantly underestimates true DD prevalence and incidence rates (Zadra & Donderi, 2000).

Perhaps the most robust finding in the DD literature is the strong association between DD frequency and waking psychopathology (e.g., Berquier & Ashton, 1992; Blagrove, Farmer, & Williams, 2004; Hartmann, Russ, Oldfield, Sivan, & Cooper, 1987; Levin & Fireman, 2002; Levin & Nielsen, 2007; Nielsen, Laberge, Tremblay, Vitaro, & Montplaisir, 2000). Because most

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of these clinical disorders are marked by considerable waking emotional distress, their association with nightmares suggests that nightmare production is related to a personality style characterized by intense reactive emotional distress (Belicki, 1992; Blagrove et al., 2004; Levin & Fireman, 2002; Levin & Nielsen, 2007; Nielsen et al., 2000). Furthermore, it has long been noted that DDs are often precipitated by stressful life events (Berquier & Ashton, 1992; Hartmann et al., 1987). DDs are most commonly associated with trauma exposure and posttraumatic stress disorder (PTSD), and there is a strong link between trauma exposure and subsequent DD (e.g., Mellman, David, Kulick-Bell, Hebding, & Nolan, 1995; Woodward, Arsenault, Murray, & Bliwise, 2000).

Further evidence for a link between increased stress and DD comes from a landmark prospective study by Wood, Bootzin, Rosenhan, Nolen-Hoeksema, and Jourden (1992), who found nightmare incidence to be twice as high immediately after the 1989 San Francisco earthquake in two San Francisco Bay-area groups than in an Arizona sample, despite equal baseline frequencies. Importantly, these differences were dose-response specific to proximity to the earthquake epicenter—those who were closer had more nightmares.

THE AMPHAC/AND NEUROCOGNITIVE MODEL OF DISTURBED DREAMING

Despite the proliferation of recent experimental work on DD, nightmare pathogenesis remains largely unexplained. Current work by us (Levin & Nielsen, 2007; Nielsen & Levin, 2007) incorporating recent advances in cognitive neuroscience, sleep neurophysiology, and fear conditioning—particularly in relation to PTSD and sociocognitive-based diathesis (i.e., vulnerability)–stress models of psychopathology—supports a multilevel model of dream function and nightmare production that unites neural and cognitive processes in both waking and sleeping. The neurophysiological branch of this model is termed the AMPHAC network, after its presumed underlying neurophysiological centers: the amygdala (A), the medial prefrontal cortex (MP), the hippocampus (H), and the anterior cingulate cortex (AC). The cognitive branch is termed the affect network dysfunction (AND) model. Together, the two branches integrate explanatory concepts at both a neural level (i.e., a cohesive and interconnected network of limbic and forebrain regions underlying emotional expression and representation) and a cognitive level (i.e., a dream-production system that transforms fear memories into dream and nightmare imagery). Disruption of processes at these levels can account for a variety of features associated with nightmare imagery (lack of emotional control, bizarre features, or replay of traumatic memories).

The AMPHAC/AND model stipulates that DD results from dysfunction in a network of affective processes that, during normal dreaming, are presumed to serve the adaptive function of fear-memory extinction. Indeed, the underlying neurophysiology and biochemistry of REM sleep appears to be primed to activate these very systems. At the cognitive level, dreaming is proposed to facilitate fear-memory extinction by three processes: memory-element activation, memory-element recombination, and emotional expression.

The first process refers to the increased availability of a wide range of memory elements during dreaming. For example, it has long been noted that, with the exception of trauma memories, dreams often do not represent coherent episodic memories; the deconstruction of memories into isolated elements or basic units is considered by most dream and sleep researchers to be a cardinal phenomenal feature of dreaming. The second process, memory-element recombination, is largely responsible for the continuous assembly of isolated memory units into a constant and phenomenologically coherent flow of dream imagery. We propose that this organization occurs during dreaming: New image contexts are produced for highly emotionally arousing memorial elements. We propose that these new memorial components are rendered into virtual simulations that maximize their impact on limbic structures, in a manner functionally identical to that which occurs during waking. Limbic structures respond more readily to perceptual stimuli than to imaginal stimuli. The new representations are then recombined to introduce contextual elements that are incompatible with existing fear memories, thus facilitating emotional processing by providing novel contexts for fear that reinforce the development of new extinction memories. The reality mimesis endemic to dream phenomenology (i.e., that dreams feel real and are experienced as waking perception, not simply as hallucination) ensures that fear memories are processed in a medium similar to that in which they were first encoded, thus facilitating emotion regulation.

We consider the third process, emotional expression, to be a necessary step in dreaming's fear-extinction function, as it maximizes the involvement of neural structures—primarily but not limited to those of the limbic system—to further ensure the adequate deployment of attentional resources in order to downregulate negative emotional arousal.

We suggest that engagement of these fear-extinction processes may be the default function of REM sleep, with dreaming representing the experienced result of these mechanisms. Representation of specific memorial components in dream content is then determined by ongoing daytime demands on the emotional-memory system—in other words, we dream about what we are emotionally preoccupied by in waking.

We use the term affect load (AL) to refer to the ongoing accumulation of stressful and emotional negative events that impinge on an individual's capacity to effectively regulate emotion. AL, in our model, is a state (i.e., transitory) factor considered to be a primary determinant of DD incidence. Thus, as AL increases, so does the probability of DD. In contrast, affect distress (AD), defined as a dispositional tendency to experience heightened distress in response to emotional stimuli, is proposed to be a major determinant of whether DDs will become clinical

waking problems such as anxiety or fear. AD is akin to the negative-affect dimension recently proposed for distress-based disorders, in that all such disorders involve heightened emotional activation. Individuals high in AD are particularly reactive to both fearful and disturbing visual stimuli, and they report creating more vivid images than do those low in AD, suggesting that reality mimesis greatly facilitates emotion activation.

At the neural level, the fear-extinction function is supported by a network of limbic, paralimbic, and prefrontal regions that constitute the control center for emotion expression and regulation during both sleeping and waking. At the broadest level, the amygdala is the control center for AL and is strongly implicated in fear conditioning. The medial prefrontal cortex serves as the mediator of extinction by regulating impulsive emotional expression via selective gating within the amygdala. The hippocampus plays a crucial role in the encoding and consolidation of episodic memories, as well the representation of stimuli in novel contexts—a crucial mechanism for emotion processing. Last, the anterior cingulate mediates AD; this region has been implicated in pain distress, social exclusion, and separation anxiety and in processing negative emotional stimuli.

Taken together, the cognitive and neural explanatory levels constitute an emotion network within which disruptions produce increasing DD, beginning with occasional bad dreams and proceeding to mildly distressing idiopathic nightmares and, finally, to repetitive and highly disturbing nightmares. Occasional bad dreams and nightmares without much accompanying distress the following day often occur in response to increasing levels of AL and usually remain isolated incidents. However, in vulnerable individuals primed for selective emotional reactivity (i.e., those with high AD), these dreams may serve as activators for previously encoded fear-memory structures and lead to enhanced waking distress—and, subsequently, to more frequent and disturbing nightmares. Thus, we suggest that individuals high in AD utilize encoding biases to selectively scan their dream imagery for threats and may experience their nightmares as more threatening and distressing than individuals low in AD, leading to a preponderance of false alarms of impending danger. Thus, for these individuals, nightmares may well be likened to the same false-alarm responses that have been noted to occur in panic disorder.

STRENGTHS OF THE MODEL

The AMPHAC/AND model is consistent with current literature from cognitive neuroscience, sleep physiology, and fear conditioning. Furthermore, the fact that the model unites waking and sleeping processes renders it highly amenable to empirical investigation, in that emotion-regulation processes should be reflected in convergence across the waking–dreaming continuum. One of the central components of the model is that while AL is proposed to directly affect the incidence of DD, it is the AD component that is responsible for waking dysregulation of emotions and the connection to psychopathology. Thus, AD is presumed to mediate the commonly observed relationship between nightmare incidence and waking distress-based psychopathology; current work being conducted in our laboratory is directly testing these assumptions.

EMPIRICAL EVIDENCE FOR THE MODEL

While broadly speculative at this stage, empirical evidence from the neurophysiology of sleep and dreaming and the affective-neuroscience literature are consistent with these formulations. For example, the work of Foa (Foa & Kozak, 1986), Lang (Lang, Davis, & Ohman, 2000), and LeDoux (2000) on fear-memory structures and fear conditioning highlights fear's automaticity, its disproportionate emphasis on response elements ("running away from a monster"), and its resistance to extinction. Research has demonstrated that frequent nightmares are associated with a number of personality characteristics (heightened imagery involvement, fantasy proneness, psychological absorption, and increased emotional activation to internal states) that are broadly consistent with our AD component (Levin & Nielsen, 2007). Further, imagery vividness is associated with increased fear activation, heightened memorial clarity for perceived negative events, and increased difficulty monitoring the sources of threats. In the recurrent nightmares of PTSD, fear-memory elements may be globally activated in a highly coherent manner, producing nightmares that consistently reproduce past fearful experiences.

Support for the crucial role of AD in mediating the relation between nightmare frequency and subsequent psychopathology comes from studies by Belicki (1992) and Levin and Fireman (2002) demonstrating that DD frequency is largely independent from waking psychopathology when AD is controlled for, a finding subsequently confirmed by at least three independent investigations.

Empirical support for the role of AL in the generation of DD is abundant. Heightened life stress is associated with increased overall dream recall and with DD in particular, and at least three studies have demonstrated that individuals who have frequent nightmares report that major distressing life events frequently precipitate their nightmares. That nightmares are a ubiquitous feature of trauma exposure also underscores this point.

On the neural level, there is ample evidence of anatomical connections between the four designated brain regions, and all have been implicated in emotional expression and regulation. Further, these brain regions are associated with both state and trait differences in emotional responding and in distress-based emotional disorders, particularly PTSD. Last and perhaps most crucial, imaging studies in both animal and human samples have

found that activity in all four brain regions increases in REM sleep above levels seen in wakefulness or non-REM sleep. Thus, the network is a vital component of normal dreaming and is likely influential in shaping emotional imagery during dreaming (see Nofzinger, 2004, for a review of brain-imaging studies and REM sleep, and McGaugh, 2004, for a review on the neural underpinning of heightened emotional processing).

FUTURE DIRECTIONS

Study of the neurophysiology of dreaming is still in its infancy, and any models explaining dreaming (as opposed to REM sleep) are likely to remain speculative for some time. As other brain components are likely to be integral in generating and shaping dream imagery, our neural model is not meant to be all-inclusive. Thus, while we believe that the anterior limbic system is central to nightmares, by no means do we believe that it is the sole seat of dreaming.

Similarly, despite our emphasis here on fear extinction, that should not be taken as the sole function of dreaming. While other established dreaming models purport similar functions (i.e., threat detection, memory consolidation, mood regulation), the question of dream function has befuddled brain scientists and philosophers alike for some time and is not likely to be answered soon. In addition, the proposition that DD serves an ongoing fear-extinction function in individuals low in AD has not been directly subjected to empirical inquiry and remains an important area for future investigation. In addition, our model does not directly address the question of adaptive versus nonadaptive fears in an evolutionary context, although we presume that fear extinction is highly adaptive despite its predilection for excessive false positives (e.g., nightmares, panic attacks).

For these reasons, our proposed model is meant to serve as a heuristic to generate further research into these mechanisms. For example, as activated fear memory structures are presumed to have an organizing (albeit costly) effect on dream content, empirical investigation of the organizational coherence of both the nightmares and normal dreams of individuals with frequent nightmares would help to elucidate the mechanisms. Similarly, if fear memories are responsible for the nonconscious detection of threat, it would be informative to investigate whether individuals high in AD perform similarly to individuals with anxiety disorders or PTSD on an affective backward-masking paradigm or the emotional color-word Stroop test. It would also be interesting to determine if individuals with high AD who have nightmares demonstrate more readily conditioned fear responses while awake than do those with low AD and nightmares. Finally, prospective research tracking relations among mood, stress, and perceived coping effectiveness both before and after nightmares would be invaluable in determining how nightmares originate.

Recommended Reading

Levin, R., & Fireman, G. (2002). (See References). A representative study of recent empirical research on nightmares.

Levin, R., & Nielsen, T.A. (2007). (See References). A comprehensive and state-of-the-art review of dream and nightmare pathogenesis, discussing the AMPHAC branch of the model in considerably greater detail than the current paper.

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