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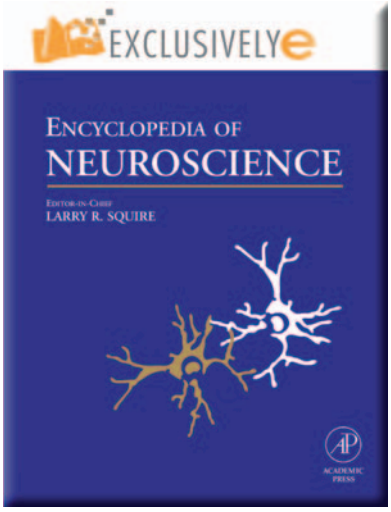

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Dreams, Dreaming Theories and Correlates of Nightmares

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Nightmares Defined

Nightmares are currently defined in both the *Diagnostic and Statistical Manual of Psychiatric Disorders*, *Fourth Edition*, *Text Revision* (DSM-IV-TR) and the *International Classification of Sleep Disorders*, *Second Edition* (ICSD-2) (Table 1). These definitions are consistent in linking nightmares to abrupt rapid eye movement (REM) sleep awakenings with a clear recall of primarily fearful dream content. The more recent ICSD-2 criteria acknowledge that nightmares may involve dysphoric emotions other than fear and anxiety and distinguish idiopathic nightmares from the more severe and highly distressing nightmares associated with posttraumatic stress disorder (PTSD). However, the ICSD-2 does not subscribe to a criterion of subjective distress as does the DSM-IV-TR, even though converging evidence suggests that one's habitual manner of affective responding (nightmare distress) is an important determinant of nightmares as a clinical problem.

Nightmare Prevalence and Correlates

Large community-based epidemiological studies demonstrate a high prevalence of occasional nightmares: about 85% of adults report at least one within the past year, whereas 8–29% report at least one per month. These same studies indicate that 2–6% of respondents report weekly nightmares, a frequency widely considered to be of moderately severe pathology. The 2–6% range is robust cross-culturally, with similar rates reported in Canada, Europe, Japan, the Middle East, and America. This range may nonetheless underestimate the real prevalence of nightmares for a number of reasons, such as reliance on retrospective measures and undergraduate samples, inconsistent operational definitions, and failure to distinguish traumatic from nontraumatic nightmares.

Frequent nightmares are up to three to four times as prevalent in childhood and adolescence as in adulthood. According to the DSM-IV-TR, 10–50% of children aged 3–5 years of age have disturbing dreams, with a prevalence that increases through early adolescence. Nightmare 'problems' (duration >3 months) occur with prevalences in the same order of magnitude: 24%, 41%, and 22% for children between age ranges of 2–5, 6–10, and 11–12 years, respectively. Evidence from the Finnish Twin Cohort, a community-based sample of 1298 monozygotic and 2419 dizygotic twin pairs aged 33–60 years, suggests that nightmares are a common and stable trait from childhood to middle age and are substantially affected by genetic factors. In contrast, nightmare prevalence and frequency in elderly populations are considerably lower than rates found in younger adults.

Females at all ages consistently report nightmares at significantly higher rates than do males, although some portion of this difference may be attributed to trauma-induced nightmares. This consistent gender gap in nightmares appears to first emerge around early adolescence. Nightmares are more frequent and more prevalent in psychiatric populations and are associated with a diversity of symptoms: anxiety, neuroticism, and global symptom reporting, schizophrenia-spectrum symptoms; heightened risk for suicide; dissociative phenomena; health behavioral problems; and sleep disturbances. Nightmares are also linked to a diversity of psychopathological traits. They are particularly prevalent in PTSD, for which they are considered a hallmark symptom. A broad range of traumatic events may trigger nightmares: combat exposure, motor vehicle accidents, natural disasters, crime victimization, and rape.

Nightmares are also reactive to intense stress. More frequent nightmares are reported during increased life stress and are associated with specific stressors, such as anticipated surgery, experimental pain stimulation, menstruation, pregnancy, miscarriage, new motherhood, preparation for exams, sham intelligence testing, stock market downturns, and the viewing of disturbing movies. Their onset is often immediately preceded by stressful events, such as death of someone close, interpersonal conflict, news of a disaster (e.g., an earthquake), and major life events in general.

Nightmares also accompany several chronic health problems, including migraine, bronchitis/asthma, chronic obstructive airways disorder, cardiac disease, and substance abuse. In addition, individuals who report frequent nightmares are characterized by a number of personality variables strongly associated with waking emotional distress: heightened physical and emotional reactivity, imagery vividness, fantasy proneness, maladaptive coping, and thin boundaries.

The association of nightmares with this wide spectrum of pathological symptoms and conditions, all of which are marked by considerable waking emotional

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Table 1 DSM-IV-TR and ICSD-2 diagnostic criteria for nightmare disorder[a]

Component	*DSM-IV-TR nightmare disorder (2000)*	*ICSD-2 nightmare disorder (2005)*
Nature of dream recall	A. Repeated awakenings from the major sleep period or naps with detailed recall of extended and extremely frightening dreams, usually involving threats to survival, security, or self-esteem	A. Recurrent episodes of awakenings from sleep with recall of intensely disturbing dream mentation, usually involving fear or anxiety but also anger, sadness, disgust, and other dysphoric emotions
Nature of awakening	B. On awakening from the frightening dreams, the person rapidly becomes oriented and alert (in contrast to sleep terror disorder and some epilepsies)	C. Alertness is full immediately on awakening, with little confusion or disorientation Recall of sleep mentation is immediate and clear
Nature of associated distress	C. The dream experience, or the sleep disturbance resulting from the awakening, causes clinically significant distress or impairment in social, occupational, or other important areas of function	D. Associated features may include: • Return to sleep after the episodes is typically delayed and not rapid
Timing of event	A. The awakenings generally occur during the second half of the sleep period	D. Associated features may include: • The episodes typically occur in the latter half of the habitual sleep period
Physiological criteria	None	None
Differential diagnosis	D. The nightmares do not occur exclusively during the course of another mental disorder (e.g., a delirium, posttraumatic stress disorder) and are not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition	Nightmares are distinguished from: seizure disorder, arousal disorders (sleep terrors, confusional arousal), REM sleep behavior disorder, isolated sleep paralysis, nocturnal panic, posttraumatic stress disorder, acute stress disorder

[a]DSM-IV-TR, *Diagnostic and Statistical Manual of Mental Disorders*, *Fourth Edition, Text Revision*; ICSD-2, *International Classification of Sleep Disorders, Second Edition*. Items A–D refer to specific definitional criteria described in the manuals; REM, rapid eye movement. Data from American Psychiatric Association (2000) *DSM-IV-TR: Diagnostic and Statistical Manual of Mental Disorders*, 4th edn. Arlington, VA: American psychiatric association; and American Academy of Sleep Medicine (2005) *International Classification of Sleep Disorders: Diagnostic & Coding Manual*, 2nd edn. Westchester, IL: American Academy of Sleep Medicine.

distress, supports the contention that nightmare production is related to a personality style characterized by intense reactive emotional distress.

Models of Nightmare Production

Despite a literature that has proliferated over the past two decades, relatively little is known about the etiology and pathogenesis of nightmares. There is a surprising paucity of working models of nightmare production, with most existing attempts being based on clinical observation but little experimental verification. Many existing models address nightmares only indirectly within the broader context of theories about dream function or personality. Further, some models are concerned almost exclusively with elucidating a nightmare function but not how nightmares are formed.

Psychoanalytic and Neopsychoanalytic Models

It is widely accepted that Freud's dream theory failed to deal adequately with nightmares. To incorporate them into his theoretical framework, Freud resorted to the notion of masochistic wish-fulfillment dreams, within which anxiety feelings stemmed from the transformation of libido. Despite the unpopularity of this idea, twentieth-century theories of nightmare production were nonetheless largely informed by Freud's work. For example, various neo-Freudian models proposed that nightmares were expressions of repressed incestuous impulses, were residues of unresolved psychological conflicts, were attempts to transform shame into fear, and so forth. Some of these views (e.g., incestuous impulse theory) have held little currency in contemporary deliberations over processes of nightmare production, whereas others have continued to wield considerable influence. For example, one contemporary model embraces an explicitly psychoanalytic framework in describing a three-stage neurological mechanism responsible for normal dreaming and nonrecurring nightmares. Other, more recent, mastery and adaptation models of dream function (see later) are also broadly consistent with neopsychoanalytic orientations.

Personality and Evolutionary Models

Boundary permeability Based on extensive clinical work with frequent lifelong nightmare sufferers, Hartmann proposed the personality dimension of 'boundary permeability' to explain nightmare pathology. Nightmare sufferers fall to one extreme of this dimension ('thin' boundaries) by virtue of their striking openness, sensitivity, and vulnerability to cognitive and emotional intrusions – and possibly by biologically based differences as well. Hartmann reasoned that thin-boundary individuals were susceptible

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to internal events not usually perceived by most others as threatening or traumatic, that they had difficulty discerning internal fantasy from external reality, and that they were prone to source monitoring disturbances across various states of consciousness. Their boundary 'thinness' thus consists in a vulnerability to spikes of heightened emotional distress; during dreaming this vulnerability leads to nightmares.

A modest body of empirical work supports the validity of the Boundary Questionnaire created to measure an individual's boundary thinness or thickness. As predicted by the model, thin-boundary persons have high levels of dream recall, dream bizarreness, and nightmare recall. Relationships have been found for adolescents as well as for adults, but not for healthy elderly individuals. However, failures to find relationships between boundary thinness and nightmare recall have also been reported. In general, despite the generally positive findings and the publication of a book on the boundary model, this approach has not been elaborated into a complete theory of nightmares and has not enjoyed widespread application as such by the research community.

Image contextualization Unlike most other models that consider nightmares to result from a breakdown in emotional regulation processes, Hartmann's contextualization model proposes that (with the exception of nonrepetitive PTSD nightmares) nightmares serve the function of contextualizing, or finding a picture context for an individual's predominant emotional concerns. Contextualization proceeds by establishing a wide swath of new associations to the emotion, the result of which is emotionally therapeutic. Contextualization is presumed to be a function of dreaming more generally, but the contextualizing processes in normal dreams are often difficult to discern when the underlying emotions are too weak or diffuse. However, after exposure to stressful or traumatic events that engender high levels of emotionality, the processes are more evident. Emotion is thus the central instigating force in the formation of dreaming and nightmares, and integration of this emotion, via the establishment of broad memory associations, is the ultimate goal of dreaming.

Contextualization occurs via contextualizing images, which are powerful central images of a dream whose emotions are consistent with a central concern but whose content may differ from this concern. For example, an image of being swept up in a tornado may contextualize an individual's feelings of helplessness, fear, and foreboding that stem from a prior assault. Hartmann's group has demonstrated that such images are, in fact, more frequent after trauma. Research has also revealed that the presence and intensity of contextualizing images are related to thinness on the Boundary Questionnaire, to a history of trauma, and to the impact of a dream on waking life. Independent validation of some features of this model was also recently reported.

Emphasis on a contextualizing mechanism likens this model to connectionist models of memory and emotional processing; both of which are thought to be central to REM sleep function. Recent evidence does support the notion that memory systems become hyperassociative and more flexible during REM sleep. Hartmann's focus on context formation during dreaming also presages recent theoretical trends in neuroscience – specifically, the view that sleep-related alterations in hippocampal context-building functions are key in determining how dreaming transmogrifies episodic memories into oniric worlds and relationships. Thus, the contextualization model anticipates both recent speculations that an REM sleep function is to create contextual memories and recent research demonstrating sleep-related facilitation of the consolidation of implicit, contextual (hippocampal-dependent) learning.

In sum, the contextualization model is a relatively new proposal which is still in the process of validation but which is consistent with several emerging trends in the neuroscience of memory.

Threat simulation The threat simulation model of nightmares is an evolutionary theory that considers nightmares to be virtual representations that enable the self to engage in behaviorally realistic responses to subjectively real threatening events. Active 'rehearsal' of responses during such simulations enhances threat-avoidance skills in the waking world and confers behavioral and survival advantages. Thus, Revonsuo suggests that nightmarish dreams are prime examples of dreams that fully realize this biological function. The suggestion that ancestral threats are more apparent in children's dreams than in those of adults underlines the importance of assessing the dreams of these two populations comparatively.

This model has generated substantial interest and empirical test in the scientific community but remains controversial. The evolutionary assumption of the model, that nightmares are heritable, has been supported to some extent. The only available study on this question used structural equation modeling of the responses of subjects from the Finnish Twin Cohort to reveal persistent genetic effects on the disposition to nightmares both in both childhood (44–45% of phenotypic variance) and adulthood (36–38%).

However, heritability *per se* is insufficient to establish function; many psychiatric disorders bear a genetic component. As described earlier, nightmares

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are associated with increased vulnerability, psychopathology, and dysfunctional adaptation to the environment. A functional role for nightmares is not supported by the finding that sleep disturbances (including nightmares) increase the risk of developing PTSD upon subsequent trauma exposure and that nightmares following trauma are associated with more severe PTSD. PTSD is associated with a host of sleep and waking state abnormalities and does not appear to be an adaptive condition in any sense.

While this model has stimulated little research on the content of nightmares *per se*, assessment of dream content has provided several consistent findings. Revonsuo's group reported evidence that severely traumatized children living in environments of threat report dreams with more threatening events and dream threats that are more severe than do less traumatized or nontraumatized children. Similarly, the earliest dreams remembered by adults (i.e., dreamed when they were children) contain a very large proportion of threat themes, also as predicted by the model. Revonsuo also reported that the dreams of college students contained threats that were frequent (66% of reports), severe (39% nontrivial), realistic (aggression, failures, misfortunes, etc.), and directed toward the self (73%), and to which the self responded with relevant defensive behaviors (56%) – all findings consistent with the model. Mixed support comes from an independent study of recurrent dreams in which six of eight predictions from the model were judged to be supported. However, less than 15% of these dreams contained realistic and probable threats critical for physical survival or reproductive success. Further, less than 2% of the dreams supported all of the predictions. Similarly, only 8% of undergraduate students' most recent dreams contain realistic life-threatening events (and escape is depicted in only a third of these), whereas severe life-threatening events are experienced in real life by 45% of them. It bears noting, however, that the presence of threats *per se* in dream content does not necessarily prove that dreams are adaptive in an evolutionary sense; these threats may simply reflect daily reality, as stipulated by the continuity hypothesis of dreaming.

In sum, the threat simulation model remains a provocative but largely unproved explanation of nightmares. As an evolutionary theory, many of its tenets are difficult, if not impossible, to test empirically. However, it provides a context, which places nightmare studies within the broader field of evolutionary biology, and has generated many novel hypotheses for empirical testing.

Neurobiological Models

Neurotransmitter imbalance Hartmann and colleagues early proposed a model of nightmare etiology based upon the effects of pharmacological treatments for chronic nightmare sufferers. An imbalance in neurotransmitter systems, characterized by diminished levels of norepinephrine and/or serotonin and elevated levels of dopamine (or a combination of these), leads to repeated experiences of nightmares. Hartmann reviewed much of the evidence available at the time but appears to have abandoned this work in favor of the boundary and contextualization models. Evaluation of this complex model today would require an extensive review of the side effects and interactions of several classes of drugs and is not attempted here. Interested readers are directed to the 'Further reading' section for relevant reviews of this literature.

REM sleep desomatization Fisher and colleagues are among very few groups to have recorded polysomnographic variables during spontaneous nightmares. They found nightmares to be associated with smaller than expected levels of autonomic activation during REM sleep and, in some cases, stage 2 sleep. The extent of autonomic activation recorded with measures of heart (HR), respiratory rate (RR), and eye movement (EM) activity was low, was limited to the last few minutes of preawakening sleep, and was, in 60% (12 of 20) of the nightmares recorded, absent altogether. Even lower levels of activation were found in a more recent study. This apparent separation of subjective fear (fearful dream imagery) from its normal psychophysiological concomitants (low or no autonomic arousal) was referred to as 'desomatization,' that is, an REM sleep mechanism for modulating anxiety by abolishing or diminishing its physiological concomitants. Such a mechanism was thought to help preserve REM sleep, to prevent the self-perpetuation of anxiety, and to contribute to the mastery of traumatic experiences. Nightmares result when the anxiety exceeds a certain threshold and the REM desomatization mechanism breaks down, allowing autonomic activation to occur.

Findings from this study, though influential in clinical research, remain somewhat questionable because an indeterminate number of patients with borderline psychosis, prior trauma, and comorbid sleep terrors participated in the study sample. Nonetheless, some of the findings have been replicated with a sample of idiopathic nightmare cases. Little new empirical evidence has been brought to bear on this model, although some laboratory findings are consistent with the possibility that dream emotion is inhibited by REM sleep processes related to the orienting response. Fisher's findings and speculations, however, have had an impact. Several investigators have also suggested that components of REM sleep may be responsible for desomatization or desensitization – that

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is, the eye movements of REM sleep desensitize affect in a way similar to what occurs in eye movement desensitization and reprocessing (EMDR) therapy.

In sum, the desomatization model is based upon a limited number of polysomnographic recordings and has not been tested systematically, but has generated much further speculation. The specificity of its proposed mechanism remains an intriguing explanation of nightmare function that is compatible with other theories of dream and nightmare function.

Mood regulation Kramer's mood regulatory theory of dreaming is premised on laboratory findings consistent with the claim that REM sleep is characterized by a 'surge' of affective arousal (e.g., a progressive increase and plateau in limbic system, eye movement, and heart and respiratory activity across the REM period). Dream content is proposed to serve the adaptive function of containing these surges by decreasing the intensity and variability of the associated emotion. This is achieved by a particular pattern of dream content that unfolds across the night and is referred to as 'progressive-sequential' (P-S) in nature. P-S dream series enable a form of emotional problem solving that ameliorates mood. P-S dreaming is distinguished from a repetitive-traumatic pattern, during which an emotional conflict is simply stated and restated without evidence of adaptive change. Nightmares presumably occur when the capacity of dreaming to assimilate the emotional surge in this fashion is exceeded.

While the physiological description of REM sleep as surgelike remains debatable, Kramer has marshaled some empirical support for the proposed function of dream content. In general, evidence that dreams are influenced by one's immediate presleep thoughts and emotional experiences, and that one's waking state mood is related to the previous night's dreams, is consistent with the notion that intervening dream activity regulates mood. More specific evidence that dreaming mediates this regulation is that pre- to post-decreases in mood scores, the 'unhappiness subscale' especially, are correlated with intervening dream content scores, scales involving the number of dream characters especially. While one study by Kramer's group failed to replicate this relationship, consistent findings have been reported by independent researchers. In this case, presleep depression scores were found to be correlated negatively with emotional tone in the dreams of the first REM period of the night (higher depression associated with more negative dreaming) but not with sleep physiology variables. There is also evidence for a mood regulatory function of dreaming in studies of persons undergoing marital separation, who report more negative dreams at the beginning and fewer at the end of the night and who prove more likely to be in remission a year later, compared to those with the opposite pattern. These findings may indicate that negative dreams early in sleep reflect a within-sleep mood regulation process, while negative dreams late in sleep reflect a failure of regulation.

The functional claims of the mood regulation model parallel those of several other nightmare models (e.g., image contextualization, REM sleep desomatization) which claim that dreaming functions to adaptively modify emotions. This claim is supported by a small but growing body of experimental evidence. However, there is as yet no convincing evidence that the P-S dream pattern *per se* is the active agent of mood regulation.

Affective network dysfunction Our neurocognitive model of nightmare formation is based upon a synthesis of findings in the areas of sleep physiology, PTSD, and anxiety disorders. It stipulates that nightmares reflect dysfunction in a network of processes that, during normal dreaming, serves the adaptive function of fear memory extinction. At the cognitive level, this extinction function depends upon three imagery processes that operate on the constituent elements of fear memories: (1) element activation, or the increased availability of isolated features of fear memories removed from their episodic (real-world) contexts, (2) element recombination, or the reorganization of these features into novel, virtual, 'here-and-now' simulations of reality, and (3) emotional expression in reaction to the recombined features, which allows new, fear extinction, memories to be formed and maintained.

At the neural level (see **Figure 1**), the fear extinction function is supported by a network of four limbic, paralimbic, and prefrontal regions that constitute the control center for a number of emotional processes, including the perception and representation of emotional stimuli and the expression and regulation of emotional responses. These four brain regions, which we refer to as the 'AMPHAC model,' include the amygdala (Am), the medial prefrontal cortex (mPFC), the hippocampal (Hip) complex, and the anterior cingulate cortex (ACC). The AMPHAC regions operate in a coordinated manner to influence other perceptual, cognitive, memorial, and affective brain events. However, general correspondences between each region and cognitive processes are also postulated (e.g., Am, emotional activation; Hip, control of memory context; mPFC, control of extinction memories; ACC, regulator of affect distress).

The cognitive and neural explanatory levels together define an affective network within which

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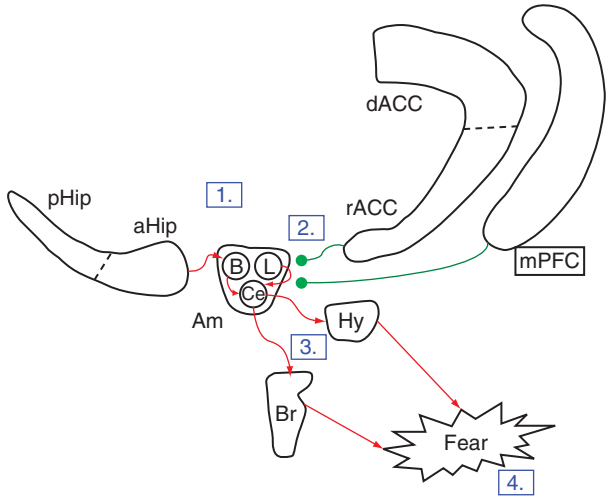

Figure 1 Schematic representation of a network of brain regions hypothesized to be implicated in the production and extinction of fear during normal dreaming; dysfunction in this network is responsible for nightmares. (1) Context is relayed in realistic (virtual) form via anterior hippocampus (aHip) to basal nucleus (B) of the amygdala (Am) and is further processed by the central (Ce) nucleus. (2) Medial prefrontal cortex (mPFC) and dorsal and rostral anterior cingulate cortex (dACC, rACC) afferents to the amygdala regulate the output of Ce neurons to induce extinction and signal distress and maintain appropriate levels of fear. (3) Ce nucleus signals brain stem (Br) and hypothalamus (Hy) to produce (4) the autonomic and behavioral correlates of fear. Excitatory connections are shown in red, inhibitory connections in green. For readability, only connections judged most pertinent to the consolidation and extinction of fear are indicated. Reproduced from Levin R and Nielsen TA (2007) Disturbing dreams, posttraumatic stress disorder, and affect distress: A review and neurocognitive model. *Psychological Bulletin* 133(3): 482–528, with permission from APA.

perturbations may produce a variety of dysphoric dreaming types – from occasional bad dreams to nontraumatic nightmares to recurring posttraumatic nightmares. Further, network activity is 'cross-state,' meaning that it is active during both sleep and wakefulness, which means that its functions – and especially its dysfunctions – should be measurable with various psychophysiological instruments both during sleep and waking states.

Two cross-state factors are proposed for which substantial supporting evidence exists. One is a situational or state factor, affect load, and reflects the combined influence of stressful and emotional negative events (e.g., interpersonal conflicts, daily hassles) on an individual's capacity to effectively regulate emotions. Increases in affect load result in intensified dysphoric dreaming in general. A second factor is a dispositional or trait-like factor, affect distress, and is characterized as a long-standing tendency to experience heightened distress and negative affect in response to increases in affect load, and to react. Affect distress is influenced by events such as prior neglect and trauma, is akin to both the negative affect and negative emotions (personality dimensions), and is associated with a wide variety of psychopathological conditions (anxiety disorders, health behaviors, PTSD, etc.). Affect distress is related to early development and acts as a risk factor diathesis for emotional dysregulation in the face of subsequent stress.

Thus, during nightmares, high levels of affect load and/or affect distress interact with the neurophysiological state of REM sleep so as to favor the activation of fear memories that are coherent (i.e., that resist feature recombination). These memories are akin to those occurring in waking, fear-based pathological conditions such as phobias or social anxiety. Nightmare-related fear memories are highly resistant to extinction, overly weighted with response elements (usually involving escape or avoidance), and, in more severe instances, corrupted by elevated affect distress. A fuller delineation of this model can be found in recent work by Levin and Nielsen.

As a new model of nightmare formation, the affective network dysfunction model has not yet been tested empirically. However, its tenets are consistent with much literature; its neural propositions, especially, are consistent with recent neuroimaging studies of REM sleep, PTSD, anxiety disorders, and personality factors. To illustrate, several studies indicate that REM sleep is characterized by high levels of activation in Am, mPFC, ACC, and the hippocampal complex. Additionally, PET studies have found that glucose metabolic rates in mPFC during REM sleep are highly correlated with elevated anxiety in the content of dreams sampled during these REM periods. Neuropsychological evidence from brain-lesioned patients also supports the model, demonstrating a link between temporolimbic brain regions and frequent nightmares of both recurring and nonrecurring types. Eight of nine patients who reported recurring nightmares were found to have temporal or frontotemporal lesions, including, in some cases, the Hip or ACC. Nightmares and 'dream–reality confusions' (DRCs) were also habitually associated with limbic lesions (nine of ten patients); six of these patients had lesions affecting the mPFC or ACC or both.

Findings from PTSD patients also support this model. All four of the designated brain regions are affected in PTSD patients. Further, heightened affect distress may be expressed in the form of several sleep-related hyperarousal symptoms, including increased awakenings, wake after sleep onset (WASO), and insomnia, as well as nightmares in stages other than REM sleep and at times other than the habitual last third of the night (e.g., stage 2 nightmares occurring early in the sleep episode). Hyperarousal is also suggested by the expression of motor activity in sleep,

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including REM-related twitches in leg muscles, more periodic leg movement during sleep in all stages, frequent large body movements, and more REM-related motor activity and vocalization. Explosive motor activity can be elicited from any stage of sleep in some patients with war-related PTSD.

In sum, the affective network dysfunction model is new and still untested but consistent with a large and recent empirical literature. The proposed fear extinction function, while novel, is nonetheless modeled after a well-established research literature on the nature, learning, and unlearning of fear memories. This function is compatible with adaptive emotional functions proposed by other dream and nightmare theorists and may serve as an integrative foundation for resolving these diverse theories.

See also: Dream Function; Dreams and Nightmares in PTSD; Dreams and Dreaming: Incorporation of Waking Events; Nightmares; Parasomnias; Sleep Mentation in REM and NREM: A Neurocognitive Perspective; Sleep and Sleep States: Phylogeny and Ontogeny; Sleep Research and Sleep Medicine in Historical Perspective; The AIM Model of Dreaming, Sleeping, and Waking Consciousness.

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