THEORETICAL REVIEW

Nightmares: A new neurocognitive model

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Review

Summary Nightmares are a prevalent parasomnia associated with a range of psychiatric conditions and pathological symptoms. Current knowledge about how nightmares are produced is still influenced by neo-psychoanalytic speculations as well as by more recent personality, evolutionary and neurobiological models. A majority of these models stipulate some type of emotionally adaptive function for dreaming, e.g., image contextualization, affect desomatization, mood regulation or fear extinction. Nightmares are widely seen to be either an intensified expression of an emotionally adaptive function or, conversely, as evidence of its breakdown. Our recent, affective network dysfunction (AND) model, integrates the tenets of many prior models in proposing that nightmares reflect problems with the fear extinction function of dreaming. This new model accounts for a wide range of dysphoric dream imagery (bad dreams, idiopathic nightmares, post-traumatic nightmares) and incorporates recent findings in the areas of brain imaging, sleep physiology, PTSD, anxiety disorders and the consolidation and extinction of fear memories.
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Introduction

Nightmares, as currently defined in two major nosologies,[1,2] are characterized by awakenings primarily from REM sleep with clear recall of disturbing mentation. The emotional component of nightmares is typically fear-related, although other less frequent emotions such as anger or disgust have also been documented.[3,4] Idiopathic nightmares, for which the cause is unknown, are also now distinguished from post-traumatic nightmares, which are more severe and distressing and often, but not necessarily, associated with post-traumatic stress disorder (PTSD).[1,2] Both types of nightmares are distinguished from sleep terrors, which also involve fear-based arousals but which typically arise from non-REM sleep, are not accompanied by vivid and extensive dreams and do not result in awakenings with clear recall of mentation.

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Nightmares are prevalent and associated with emotional distress

Large community-based epidemiological studies[5–9] indicate that 2–6% of respondents report weekly nightmares, a frequency generally thought to reflect moderately severe pathology.[10] Frequent nightmares are widely thought to be even more prevalent in childhood and adolescence,[1,11–18] however, our recent epidemiological study[19] found relatively low prevalences in pre-schoolers (see also Agargun et al.[20] for similar findings in children age 7–11). Nightmares are also known to be less frequent in elderly populations.[16,21–24] Females at all ages consistently report nightmares more often than do males,[8,9,13,24–31] partly because of higher rates of post-traumatic nightmares but also, possibly, because women report both a greater quantity and a greater intensity of symptoms related to negative emotional disturbances, such as depression and anxiety, than do men.[32,33] Further, the gender difference in nightmare reporting is seen even when controlling for an equally ubiquitous gender difference in dream recall.[24] Evidence from the Finnish Twin Cohort[29] suggests that nightmares are substantially affected by genetic factors.

A large literature for the most part converges in demonstrating that nightmares are associated with problems in the expression and regulation of dysphoric emotions (see reviews in[34,35]). Nightmares are more frequent and more prevalent in psychiatric populations[9,28,31,36–38] and are associated with pathological symptoms such as anxiety,[7,9,13,26,28,29,31,39–47] neuroticism and global symptom reporting,[39,41,44,46,48–50] schizophrenia-spectrum symptoms,[27,38,41,42,51–53] heightened risk for suicide,[26,31,54] dissociative phenomena,[44,55] health behavioral problems,[31,39,56,57] sleep disturbances[7,8,28,49,58–61] and PTSD.[62–65] Links with dysphoric emotional processes are also suggested by nightmares' relationships with both psychopathological traits[38,42,44,46,49,66] and personality variables that implicate waking emotional distress, e.g., heightened physical and emotional reactivity[8,49,67] and maladaptive coping.[68–70] Numerous studies indicate that nightmares are reactive to intense stress[27,47,71–74]; for example, they are more frequently reported during times of increased life stress.[39,41,49,58,67,75–78]

The association of nightmares with this wide spectrum of pathological symptoms and conditions, all of which are marked by considerable emotional distress, supports the contention[34] that nightmare production is related to a general personality style characterized by intense reactive emotional distress.[44,48,79,80] It is also consistent with the suggestion that nightmares are related to a dream function involving the regulation of emotion—fear in particular.

Models of nightmare production

Models of nightmares production are broadly consistent with this large corpus of evidence linking nightmares to processes of emotional regulation. With few exceptions, these models emphasize the modulation or transformation of emotion as a central function of dreaming, and either an augmentation or disruption of this function as central to nightmares. Surprisingly, these theoretical models have been developed in relative isolation and are not well integrated with emerging findings in the neuroscience of sleep and emotions. The present section will summarize existing models of nightmare production, review recent evidence supporting or refuting them, and demonstrate—by outlining a new neurocognitive approach to nightmares—how these models may contribute to a new understanding of the genesis and function of nightmares.

Psychoanalytic models

Early psychoanalytic and neo-psychoanalytic models of nightmare formation (see Table 1) were consistent in hypothesizing that dreaming plays a functional role in emotional development and that nightmares reflect either a modification or a failure of this function. Freud's[81] original dream theory postulated that nightmares are a masochistic variation of the wish-fulfillment function of dreaming which aims to preserve sleep by containing anxiety feelings associated with early libidinal urges. Later models made more specific claims about the function of nightmares as assimilating repressed anxiety,[82] failing to master trauma[83] or transforming shame into fear.[84] More recent theories of nightmares as failures in emotion regulation[85,86] are also informed by and broadly consistent with these neo-psychoanalytic conceptions.

Although much anecdotal clinical evidence has been put forth in support of various psychoanalytic models of nightmares[84,87]—including Freud's own libidinal model[88]—empirical evidence is scarce. Some empirical studies of Freud's dream theory (for reviews see[89–91]) may be pertinent to nightmare models but are beyond the scope of the present review. Two types of recent work can be construed as supporting psychoanalytic nightmare

Table 1 Psychoanalytic and neo-psychoanalytic models of nightmare formation.

Authors	Core mechanism producing nightmares
Freud[81]	Transformation of libidinal urges into anxiety that punishes the self (masochism); analogous to the neurotic anxiety underlying phobias
Jones[88]	Expression of repressed, exclusively incestuous, impulses
Jung[201]	Residues of unresolved psychological conflicts
	Individuation or development of the personality
Fisher et al.[82]	Attempts to assimilate or control repressed anxiety stemming from past or present conflicts (see 'REM sleep desomatization model')
Greenberg[83]	Failure in dream function of mastering traumatic experience
Lansky[84]	Transformation of shame into fear (posttraumatic nightmares only)
Solms[174]	Epileptiform (seizure) activity in the limbic system (recurring nightmares only)
	Activation of dopaminergic appetitive circuits in mediobasal forebrain and hallucinatory representation by occipito-temporal-parietal mechanisms (nonrecurring nightmares and normal dreaming)

models more specifically. First, recent neuroscientific findings consistent with the Freudian view that dreaming expresses affective concerns (e.g., continuing activity of dopaminergic pathways during REM sleep) and that disruption of such expression (e.g., by epileptic seizures or elevated dopaminergic activity) produces nightmares, are broadly consistent with psychoanalytic nightmare models.[92] Second, the demonstration that successful treatment of post-traumatic nightmares is accompanied by an increase in mastery themes in patients' dreams[93] supports Greenberg's mastery failure model[83] to some extent. The mastery model is also supported by the sleep laboratory recordings of nightmare patients conducted by Fisher and colleagues.[82] These findings are reviewed in more detail in a later section. Finally, models by Kramer and Cartwright that were influenced by psychoanalytic models have received some empirical support. However, as these models are not today closely identified with the psychoanalytic approach, this evidence, too, is reviewed in later sections.

In sum, although clinical and, to some extent, empirical evidence supports different psychoanalytic models of nightmare formation, for the most part such models have not been subjected to rigorous empirical scrutiny. Rather, their central tenets have been integrated with more recent nightmare models, where empirical evidence is less scarce.

Personality and evolutionary models

Boundary permeability
Hartmann[27,42,94,95] proposed a personality dimension—"boundary permeability"—that addresses one key emotional aspect of nightmare pathology. Nightmare patients were found to fall at one extreme of a boundary permeability dimension ("thin" boundaries) characterized by a vulnerability to cognitive and emotional intrusions. Thin-boundaried individuals are unusually susceptible to internal events that most individuals do not perceive as threatening or traumatic. Their "thinness" thus consists of a susceptibility to experience spikes of heightened emotional distress which, during dreaming, can lead to nightmares.

A modest body of empirical work supports the validity of the Boundary Questionnaire Hartmann created to measure boundary thin- or thickness. As predicted, thin-boundaried subjects have high levels of dream recall, dream bizarreness and nightmare recall.[70,96–104] Consistent relationships have been found for adolescents[96] and adults, but not for the healthy elderly.[105] However, failures to find relationships between boundary thinness and nightmare recall have also been reported.[95] There is also some uncertainty about how the concept of boundaries is distinct from existing concepts such as openness to experience.

In sum, this model underlines the centrality to nightmare production of *a personality factor*

determining an individual's capacity to resist and manage emotional intrusions across a variety of sleep and wakeful states.

Image contextualization

Hartmann's[90,106,107] more recent contextualization model attempts to identify specific processes underlying emotion regulation during dreaming and nightmares. He proposes that nightmares serve the function of contextualizing, or finding a picture context for, an individual's predominant emotional concerns. Contextualization proceeds by establishing a swath of new associations to the emotion, the result of which is emotionally adaptive.[90] Contextualization is presumed to be a function of dreaming more generally but is more difficult to discern when the underlying emotions are weak or diffuse. However, after exposure to stressful or traumatic events that engender high levels of emotionality, the processes are more evident.

Contextualization occurs via contextualizing images (CIs), powerful central dream images whose associated emotions are consistent with a central concern but whose specific contents may portray a quite different event. For example, a dream image of being swept up in a tornado may contextualize an individual's feelings of helplessness, fear and foreboding that stem—not from a previous tornado experience—but from a prior physical assault. In light of the importance of contextualizing images to the hypothesized emotion-guided contextualization process, validation of whether CIs do, in fact, visually picture key emotions is critical. Accumulating evidence by Hartmann's group largely supports this aspect of the validity of CIs.[108] For example, CIs are more frequent among subjects who have been traumatized[90,109] and CI intensity is higher after a recent trauma[110,111] or past physical or sexual abuse[110,112] than it is among healthy control subjects. CI intensity is also higher in dreams subjects consider to be either personally important or especially significant than it is in those considered unimportant or mundane.[113] Independent validation of some features of this model was also recently reported.[114,115] Recent evidence also supports the notion that memory systems become hyper-associative and more flexible during REM sleep.[116,117]

Emphasis on a contextualizing mechanism likens this model to connectionist models of memory[118,119] and emotional processing,[120,121] both of which are thought to be central to REM sleep function.[85,86,122] The focus on context formation during dreaming also presages recent suggestions that sleep-related alterations in context-building functions of the hippocampus influence the consolidation of episodic memories (for review see[123]). For example, the contextualization model anticipates both recent speculations that REM sleep functions to create contextual memories[124,125] and recent research demonstrating that implicit, contextual (hippocampal (Hip)-dependent), learning is facilitated during sleep.[126]

Despite these promising theoretical advances, however, there is still no compelling evidence that the CIs identified in dreaming serve to facilitate emotional adaptation in the manner postulated by Hartmann. Also, the discriminant validity of the CI concept would be bolstered by evidence that such images are independent of factors such as dream vividness and dysphoric quality.

In sum, the contextualization model singles out *emotion as the central instigating force in the formation of dreams and nightmares and emphasizes emotion regulation via contextualization as their central function.*

Threat simulation

The threat simulation model of nightmares[127] is an evolutionary theory that assigns a central role to threat and fear in the production of dreams and nightmares. Revonsuo considers nightmares to be virtual representations of the self engaged in realistic responses to subjectively threatening events. Active 'rehearsal' of such simulated responses enhances threat-avoidance skills in the waking world and confers behavioral and survival advantages. Nightmares are thus exemplary of dreams that fully realize a biologically adaptive function. Children's dreams and nightmares are of particular interest because inherited representations of ancestral threats are thought to be more apparent in their dreams than in those of adults.

This model has generated substantial interest and has been supported to some extent by empirical study. The evolutionary assumption, that nightmares are heritable, has modest support in that one study[29] found persistent genetic effects on the disposition to nightmares both in both childhood and adulthood. However, a functional role for nightmares is not supported by the finding that having nightmares is associated with an increased risk of developing PTSD upon subsequent trauma exposure[128] and that nightmares following trauma are associated with more severe PTSD.[65,129] PTSD itself is associated with several sleep and waking state abnormalities and does not appear to be an adaptive condition in any sense.[130,131]

Assessments of dream content have provided several consistent findings. Revonsuo's group reported that severely traumatized children living in threatening environments report dreams with more

threatening events and dream threats that are more severe than do less traumatized or non-traumatized children.[132] Similarly, adults' earliest remembered dreams (i.e., dreamed in childhood) contain a large proportion of threat themes.[133] Revonsuo[134] also reported that the dreams of college students contain threats that are frequent (66% of reports), severe (39%), realistic (aggression, failures, misfortunes, etc.), directed toward the self (73%), and for which the self responds with relevant defensive behaviors (56%)—all findings consistent with the model. Mixed support comes from a study of recurrent dreams[135,136] in which six of eight predictions from the model were judged to be supported to some extent. 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 undergraduates' most recent dreams[137] contain realistic life-threatening events, a much lower proportion than the 45% of individuals who have experienced severe life-threatening events in real life. It also bears noting 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.[138]

In sum, the threat simulation model provides *an evolutionary context for explaining the realistic representation of fear in nightmares and its potential adaptive function.*

Neurobiological models

REM sleep desomatization
Fisher and colleagues[82] provided psychophysiological evidence consistent with the notion of emotional regulation during nightmares. They found that nightmares occurring spontaneously in the laboratory were characterized by less than expected autonomic activation during REM sleep and, more rarely, during stage 2 sleep. Autonomic activation as reflected in measures of heart rate, respiratory rate and eye movement activity, was low, was limited to the last few minutes of pre-awakening sleep and was, in 60% (12 of 20) of the nightmares recorded, absent altogether. Even lower levels of activation were found in our more recent study.[139] This apparent separation of fearful dream imagery from its expected psychophysiological concomitants prompted Fisher[82] to speculate that REM dreaming possesses 'a mechanism for tempering and modulating anxiety, for desomatizing

the physiological response to it…[for] abolishing or diminishing the physiological concomitants' (p. 770). In line with Freudian notions popular at the time, 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.[82] Nightmares result when the anxiety exceeds a certain threshold and the REM desomatization mechanism breaks down, allowing autonomic activation to occur.

Similar speculations have recurred sporadically in the literature. A short note on a similar desensitization function of dreaming[140] and an empirical study on a speculative anxiety-extinction function of nightmares[7] were published shortly after Fisher et al.'s work but neither demonstrated any awareness of the latter's findings. Haynes and Mooney attributed a specific extinction function to nightmares, i.e., through repeated cognitive exposure to fear-inducing stimuli (akin to implosive therapy), but concluded that their results did not support this hypothesis. Other investigators have also suggested that components of REM sleep may be responsible for desomatization or desensitization. Shapiro[141] suggested that the eye movements of REM sleep may desensitize affect in a way that is similar to eye movement desensitization and reprocessing (EMDR). Nielsen and colleagues[142,143] proposed that the atonia of REM sleep could produce a desensitization effect by repeatedly blocking kinesthetic feedback during traumatic dream imagery so as to extinguish its somatic correlates. Perlis and Nielsen[144] expanded upon this notion in suggesting that anxious dream imagery may be desensitized during REM sleep in a manner analogous to systematic desensitization therapy, i.e., by the pairing of dysphoric dream imagery and atonia of the musculature. Nightmares were considered to be either interruptions of this process (leading to waking sensitization and distress) or accelerated desensitization (analogous with flooding therapy).

Fisher's original findings remain somewhat questionable because an indeterminate number of patients with borderline psychosis, prior trauma[145] and comorbid sleep terrors were included in the study sample. Nonetheless, we replicated some of the findings with a sample of idiopathic nightmare cases[139] and reported some evidence consistent with the notion that dream emotion is inhibited by REM sleep processes related to the orienting response.[142] Little other relevant research with laboratory polysomnography has been conducted.

In sum, the desomatization model proposes a specific, physiologically based, mechanism of emotion regulation during dreaming and nightmares,

i.e., *the coupling of dysphoric imagery with muscle atonia.*

Mood regulation

Kramer's[86,146] mood regulatory theory of dreaming provides an alternative, although potentially compatible, view of emotion regulation during dreaming. The model is premised on findings consistent with the claim that REM sleep is characterized by a 'surge' of affective arousal, i.e., a progressive increase and plateau in limbic system, eye movement, heart and respiratory activity across the REM period. Dream content is thought to adaptively regulate or 'contain' these surges by decreasing the intensity and variability of the associated emotion. Containment is achieved by a longitudinal pattern of dream content that unfolds over successive REM periods of the night and is referred to as 'progressive–sequential' (P–S) in nature. P–S dream patterns enable a form of emotional problem solving that ameliorates mood. The P–S pattern 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 with P–S pattern dreams is exceeded.

While the physiological description of REM sleep as surge-like remains debatable, some other findings support the model. In general, evidence that dreams are influenced by one's immediate pre-sleep thoughts and emotional experiences[86,147,148] and that one's waking state mood is related to the previous night's dreams,[149] 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, especially with the number of dream characters represented.[86] One study by Kramer's group failed to replicate this mood regulatory effect,[86] but consistent findings were reported by Cartwright.[85,150] In the latter case, high pre-sleep depression scores were associated with more dysphoric dreams from the first REM period—but not with sleep physiology variables. This group also reported supporting evidence from studies of divorced women[151–153] and suicidal patients.[154] For example, subjects undergoing marital separation who report more negative dreams at the beginning and fewer at the end of the night are more likely to be in remission a year later than are those with the opposite pattern.[152] Negative dreams early in sleep may thus reflect a within-sleep mood regulation process similar to the

P–S pattern, while negative dreams late in sleep may reflect a failure of this regulation function.

In sum, the mood regulation model posits a specific mechanism for emotion regulation during dreaming, i.e., *the regular coupling of emotional surges with a problem-solving dream structure that unfolds across the night.*

Toward an integrative model of nightmare production

For the most part, existing models of nightmare production have been developed and tested in relative isolation from each other and without much attempted integration with the emerging neuroscientific literature. However, each model has succeeded to some extent in delineating and supporting one or more key components of nightmare production (see Table 2). These components taken together begin to sketch a more comprehensive model of nightmare production. Such a model is presented below and contrasted with existing models in a subsequent section.

An affect network dysfunction (AND) model of nightmare production

The AND model of nightmare production is founded on a synthesis of recent findings in brain imaging, sleep physiology, PTSD, fear memory, anxiety disorders and personality and complements a recently proposed neurophysiological description of nightmare formation referred to as the AMPHAC model (short for amygdala, medial prefrontal cortex, hippocampus and anterior cingulate cortex[34]). It stipulates that nightmares result from dysfunction in a network of affective processes that, during normal dreaming, serves the adaptive function of *fear memory extinction.*

The model combines cognitive and neural levels of explanation. At the cognitive level, dreaming is proposed to facilitate fear memory extinction according to currently accepted notions of fear memory acquisition and extinction. The latter are supported by a vast body of empirical research dating back to Pavlov's[155] studies of classical conditioning (for review see[156,157]). To briefly summarize these findings, fear memory acquisition results when an innocuous stimulus, destined to become a conditioned stimulus (CS), is paired with an unconditioned stimulus (UCS) that induces the reflexive unconditioned response (UCR) of fear. This CS–UCS pairing is learned (i.e., forms a fear memory) and causes subsequent presentations of

Table 2   Key components of nightmare production highlighted by recent models.

Authors	Model	Key component of nightmare production
Hartmann et al.[94,95]	Boundary permeability	Personality factor determining an individual's capacity to resist and manage emotional intrusions across a variety of sleep and wakeful states
Hartmann[90,106]	Image contextualization	Emotion as the central instigating force in the formation of dreaming and nightmares; Emotion regulation via contextualization as central function of nightmares
Revonsuo[127]	Threat simulation	Evolutionary context for explaining the realistic representation of fear in nightmares and its potential adaptive function
Fisher et al.[82]	REM sleep desomatization	Physiologically based mechanism of emotion regulation during dreaming and nightmares: the coupling of dysphoric imagery with muscle atonia
Kramer[86,146]	Mood regulation	Mechanism for emotion regulation during dreaming: regular coupling of emotional surges with a problem-solving dream structure that unfolds across the night

the CS alone to evoke conditioned responses (CR) that are similar to the fearful UCR. Fear memory acquisition is rapid, enduring and adaptive, saving the individual from repeating potentially dangerous errors. Fear memory extinction, in contrast, is not simple forgetting, erasing or undoing of an acquired fear memory. Rather, repeated occurrences of the CS in the absence of the UCS, or repeated pairings of the CS with new, non-fearful contexts, leads to the formation of new extinction memories. Extinction memories override original fear memories whenever CSs are presented, as suggested by the fact that a previously extinguished fear memory will reappear with the passage of time (spontaneous recovery) or if the UCS is presented in the absence of the CS (reinstatement).[158]

Fear extinction memories are suggested to be created or maintained during dreaming by three novel imagery processes that operate repeatedly on the constituent elements of acquired fear memories: (1) *element activation*, or the increased availability during dreaming of features of fear memories that are largely dissociated from their episodic (real-world) contexts; (2) *element recombination,* or the reorganization of these features into novel (virtual-world) 'here-and-now' simulations of reality that present CSs without their pairing with UCSs but rather are paired with new, non-fearful, contexts, and (3) *emotional* expression, or the experience of modified emotional reactions to these recombined features. Engagement of these fear extinction processes may well be an automatic feature of dreaming, with the representation of specific CSs in dream content

being determined by ongoing daytime demands on the emotional memory system. These demands are a function of a hypothetical factor we term *affect load*, i.e., a situational or state factor that reflects the combined influence of stressful and emotionally negative events (e.g., interpersonal conflicts, daily hassles) on an individual's capacity to effectively regulate emotions.

At the neural level, the fear extinction function is supported by a network of limbic, paralimbic and pre-frontal regions that constitute the control center for numerous emotional processes in both sleeping and waking states, including the perception and representation of emotional stimuli and the expression and regulation of emotional responses. Four brain regions in particular operate in a coordinated manner to influence other perceptual, cognitive, memorial, affective and motoric brain events: the amygdala (Am), the medial prefrontal cortex (mPFC), the Hip complex and the anterior cingulate cortex (ACC). While there is some redundancy of function in this network, each of these brain regions corresponds roughly to a particular domain of processing in the fear extinction process, i.e., Am: emotional activation and control of fear memory acquisition; Hip: control of memory context; mPFC: storage and control of extinction memories; ACC: regulation of affect distress. These hypothetical links are supported by a vast literature on animal and human fear learning and the brain correlates of social distress and personality (for reviews see[34,156,157]).

The AMPHAC model in Fig. 1 illustrates, for the neural level of explanation and in very simplified
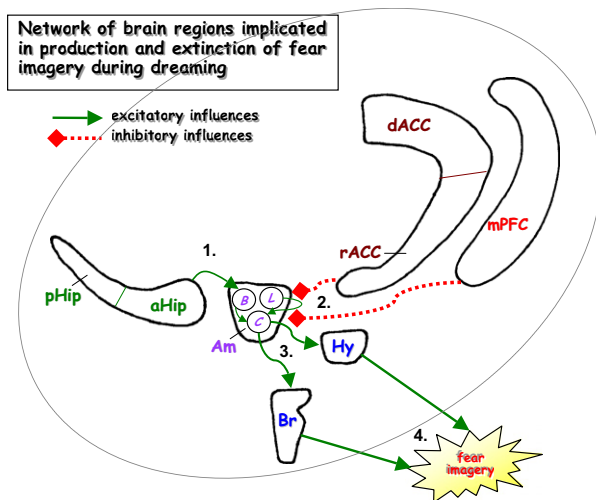
Figure 1 Graphic illustration of a network of brain regions hypothesized to be implicated in the production and extinction of fear imagery during dreaming. Dysfunction in this network is proposed to produce nightmares of varying severity. 1. Dream context is relayed in realistic (virtual) form via anterior hippocampus (aHip) to basal nucleus (*B*) of the amygdala (Am) and further processed by Am central (*C*) nucleus. 2. Pathways from medial prefrontal cortex (mPFC) and dorsal and rostral anterior cingulate cortex (dACC, rACC) regulate Am processing and output to induce fear extinction memories, to signal affect distress and to maintain tolerable levels of fear. 3. The *C* nucleus stimulates brainstem (Br) and hypothalamus (Hy) which 4. Activate the autonomic and behavioral correlates of fear imagery, e.g., increased heart rate and respiration. Many other interconnections among regions are well-documented but only those thought to be most relevant to fear consolidation and fear extinction during dreaming are shown. See text for further explanation. Excitatory influences are depicted with solid green arrows; inhibitory influences with dotted red arrows.

form, how a fear memory might be activated during a mundane bad dream. A context for emotional activation is relayed by the recombination of fragmentary memory elements into a realistic (virtual-world) scenario (e.g., '*I am in a dark alley*') via the anterior hippocampus (aHip). The contextual information is processed by the basal nucleus (B) of the Am and given further affective qualities by the Am central (C) nucleus (e.g., '*I feel increasingly nervous*'). Inhibitory afferents from the mPFC and dorsal and rostral anterior cingulate cortex (dACC, rACC) impinge on the Am, down-regulating its Ce outputs to stabilize the emotion ('*My nervousness seems to plateau*') and signal distress according to the historical propensities of the individual, e.g., a mildly agoraphobic individual ('*I start to feel trapped and panicky*'). The C nucleus signals the brainstem (Br) and hypothalamus (Hy) to express the autonomic and behavioral

correlates of fear ('*I feel my heart starting to beat quickly; I turn around and run from the alley*').

Extinction in this system would be achieved by introducing into the virtual-world simulation non-aversive memory elements that are emotionally incompatible with the activated fear memory and that contribute to production of new fear extinction memories. In the latter example, '*a close friend appeared in the dark alley*' or '*suddenly I found myself in a brightly lit street*' would constitute contextual memory elements that are sufficiently non-aversive and incompatible that the growing fear would be assuaged to some degree. Formation of stable extinction memories likely only emerges gradually, over many iterations of element recombinations occurring in different hippocampally controlled contexts. Extinction memories are, in fact, less robust than original fear memories; the latter can be reinstated if extinction memories are not maintained.

Although there is an absence of longitudinal research on nightmares that might clarify how fear memory extinction occurs over time, findings from cross-sectional samples reveal that subjects who report cessation, at least a year earlier, of prior recurrent dreams (which are usually emotionally negative) score higher on well-being measures than do subjects who currently continue to experience recurrent dreams.[159] Further, increases or decreases in well-being over several years are accompanied by parallel changes in dream content such as emotions or social interactions.[160] These findings are consistent with the suggestion that the negative affect in recurrent dreams is progressively extinguished over time and that this extinction generalizes to daytime affect.

In sum, the AND model stipulates that affect load determines an ongoing need for the development of new fear extinction memories as well as the maintenance of existing fear extinction memories. The latter are dependent upon dream processes that activate, dissociate and recombine attributes of fear memories in new, realistic and potentially fear-extinguishing contexts.

In contrast to the affect load factor, *affect distress*, which is regulated largely by the ACC, is proposed to be a major determinant of whether a nightmare will constitute a clinical problem. It determines the degree of distress an individual will experience both during and after a nightmare. Affect distress is defined as a dispositional or trait-like factor consisting of a long-standing tendency to experience heightened distress and negative affect in response to emotional stimuli. It is influenced by prior abuse, neglect, trauma and other developmental factors and is associated with a wide variety

of psychopathological conditions. Although there is substantial overlap between affect distress and both the negative affect and neuroticism personality dimensions,[161,162] two broad personality dimensions involving heightened emotional reactivity (see[163] for discussion), affect distress is considered to entail a more specific and immediate propensity to reactivity—particularly in response to dysphoric imagery. Affect distress may thus be a temperament subtype in the negative affect hierarchy, much like the case proposed for anxiety sensitivity.[163]

The cognitive and neural explanatory levels of the AND model together define an affective network within which perturbations produce the different types of dysphoric dreaming—from occasional bad dreams to non-traumatic nightmares to replicative post-traumatic nightmares. Mundane bad dreams and nightmares presumably occur when sudden increases in affect load (e.g., loss of job, argument with spouse) lead to a prominent activation of fear memories and the fear extinction mechanism. This may be accompanied by an overactive Am, a failure of the mPFC to adequately regulate the Am output or an inability of the Hip to produce sufficiently incompatible contexts to establish or maintain extinction memories. Furthermore, post-traumatic nightmares may occur as a result of similar conditions but with the added involvement of trauma memories that are particularly resistant to feature recombination (and extinction) and coupled with heightened levels of ACC-mediated affect distress.

Supportive evidence

The AND model tenets are consistent with much current literature.

Converging findings indicate that the same 4 brain regions central to the AMPHAC network are also implicated in REM sleep, PTSD (see reviews in[164,165]), anxiety disorders (see review in[166]) and some individual state and trait differences in emotion regulation (see review in[34]). To elaborate on the evidence for REM sleep, several recent neuroimaging studies indicate that REM sleep is characterized by higher than normal levels of activation in Am,[167–169] mPFC,[169,170] ACC[167–172] and the Hip complex.[168,169,171] Further, a PET study of healthy subjects[173] found that glucose metabolic rates in mPFC during REM sleep was highly correlated with elevated anxiety in the content of dreams sampled during these REM periods. Neuropsychological evidence from brain-lesioned patients[174] also supports the model, demonstrating a link between temporal or fronto-temporal lesions (including instances with Hip, mPFC and ACC lesions) and frequent nightmares of both recurring and non-recurring types.

Findings from studies of traumatized patients are consistent with our suggestion that affect distress can become pathologically linked to nightmare production. Specifically, the heightened affect distress characterizing PTSD is expressed in sleep-related hyperarousal symptoms such as increased awakenings,[59] wake after sleep onset (WASO)[59,175] 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.[176] Hyperarousal is also suggested by the expression of motor activity in sleep, including REM-related twitches in leg muscles, more PLMS in all stages, frequent large body movements, and more REM-related motor activity and vocalization.[62,177–179] Hefez et al.[180] reported that explosive motor activity could be elicited from any stage of sleep in a patient with war-related PTSD while Leopold and Dillon[181] found that some survivors of a maritime disaster expressed motor activity and vocalizations during sleep. In one case a patient appeared to attempt escaping from the bedroom.

Evidence also links the 4 brain regions directly to sub-processes of the proposed fear extinction mechanism. The role of Am activity in controlling fear expression during dreaming is supported by numerous studies demonstrating that Am is robustly related to fear[121,182,183] and mediates the acquisition and expression of conditioned fear memories (for review see[184]). Since Am is activated to a greater extent when stimulation involves visual perception rather than recall or imagery,[182] the virtual-world simulations of dreaming may optimally facilitate its activity at this time. The role of mPFC in controlling fear extinction during dreaming is supported by evidence from animal studies that this region is essential for the extinction of fear memories.[185,186] Some studies,[187,188] but not all,[189] further suggest that establishing multiple extinction memories diminishes the likelihood that a specific fear memory will be renewed. The constantly changing, variegated imagery of dreaming may thus be ideally suited to enabling and maintaining extinction memories. Diminished mPFC activity is also known to prevent the consolidation of extinction memories needed to counteract the overwhelming impact of post-traumatic memories.[186,190–192]

The role of the hippocampus in determining imagery context is supported by evidence that it regulates the expression of fear and fear extinction

memories based upon evaluation of the *context* within which the fear stimuli occur (for reviews see[193,194]). There are presently several theories about how altered Hip activity during REM sleep produces the bizarre yet realistic nature of dreaming.[123] The role of the ACC in mediating affect distress is consistent with many studies implicating ACC in the neural circuitry of pain,[195–197] including the emotional pain produced by social loss[198] and both real and remembered social distress.[199,200]

In sum, the AND model is consistent with a large, recent, scientific literature. The proposed fear extinction function is modeled upon well-established findings on the nature, learning and extinction of fear memories. It should be clear from the preceding that the 'cross-state' assumption of the AND model renders it highly testable in that its emotion regulation processes should be apparent in both waking and sleep states. The fear memories thought to underlie dysphoric dreams are proposed to directly reflect those occurring in waking, fear-based pathological conditions such as phobias or social anxiety. The model is thus amenable to study with a variety of psychophysiological and neuro-cognitive instruments during both sleep and wakefulness. For example, the model would predict that clear links between idiopathic nightmares and an individual's most persisting fear memories could be identified with appropriate measures. Or, that nightmare-induced distress will correlate with affective distress that is associated with other clinical symptoms such as social anxiety or neuroticism. A more thorough review of the clinical and treatment predictions of this model, as well as suggested validational experiments, can be found in Levin and Nielsen.[34]

Relationship to prior models

The AND model incorporates many of the key components of previous models (see above and Table 2) and combines them into a more comprehensive theory of nightmares that is consistent with and integrated into the current scientific literature. The following section describes the main points of agreement and contention between prior models and the current AND model.

Boundary permeability
The AND model accounts for the dispositional link to nightmares identified by the boundary model by proposing affect distress as a trait factor implicated in the suffering induced by nightmares—and post-traumatic nightmares in particular. Affect distress is viewed as primarily influenced by an individual's

developmental history. A state factor, affect load, is also proposed to interact with affect distress in the production of nightmares and nightmare distress.

Image contextualization
The AND model recognizes the primacy of dysphoric emotion, fear in particular, in the formation of dreams and nightmares as well as the functional importance of contextualization. The model stipulates that the continuous activation of emotion (via features of fear memories) is necessary for fear extinction to be achieved and that Hip control over contextual fear learning and extinction is central to this process. Whereas Hartmann's contextualization process involves the building of numerous associations between memory elements, the current model stipulates that the incompatibility between fear memory elements and their context is what facilitates extinction.

Threat simulation
The AND model does not deny a possible evolutionary function for dreaming and nightmares although this function is not elaborated here. Nor is it a necessary component of the model's utility. The AND model agrees with the threat simulation model in that virtual-world simulation is seen as an essential component of the fear extinction mechanism. However, whereas Revonsuo proposes that threat simulations serve to incite behavioral rehearsals of adaptive responses, the current model suggests that such simulations serve to maximize activation of fear memory structures (via Am activity) so that these structures may be recombined in the service of fear extinction.

REM sleep desomatization
The AND model proposes a function akin to desomatization (fear memory extinction) but does not assume that a coupling of dysphoric dream imagery with muscle atonia is necessary for this function. Rather, the extinction is achieved by the embedding of dysphoric stimuli in new, emotionally incompatible, contexts. It is possible, however, that this recombination of elements requires the inhibition of muscle action as well as the suspension of several other cognitive capacities (e.g., self-reflection) to be successful.

Mood regulation
The current model and other mood regulation models are quite compatible in postulating similar emotionally adaptive functions for dreaming. Kramer's notion of affective surge is reflected to some extent in the notion of fear memory activation in the AND model, except that such activation may

conform to patterns other than surge-like. Further, whereas the AND model does not hypothesize that a specific problem-solving structure is necessary for adaptive fear memory extinction to take place, it does parallel the mood regulation model in stipulating that the adaptive function takes place gradually over time, i.e., over successive iterations of the same emotion within different contexts occurring across the night (or across several nights).

Conclusion

The neurocognitive model of nightmare production presented here brings together a large research literature on the mechanisms of fear memory, sleep, PTSD, anxiety disorders and personality, as well as key components of many prior nightmare models. The model can account for a wide range of dysphoric dream imagery (bad dreams, idiopathic nightmares, post-traumatic nightmares) and the ubiquitous association between nightmares and numerous pathological conditions. Perhaps most importantly, the new model is amenable to empirical testing using methodologies adapted to both sleep and waking states.

Practice points

Nightmares are a ubiquitous parasomnia with clinically significant frequencies (1/week or more) occurring in 2–10% of the population. They are also more frequent among children, women and a wide range of patients with psychiatric and personality problems. Several models of nightmare production suggest that nightmares may be implicated in an emotional adaptation function. In dealing with complaints of nightmares, clinicians can optimize their treatment recommendations by collecting a variety of information about common precipitating influences. They should:

- Ascertain whether the nightmares are post-traumatic or idiopathic (no known cause).
- Assess the patients psychiatric and personality histories, especially evidence of anxiety-related disorders, to determine whether they are prone to distress reactions.
- Assess the patients day-to-day levels of stress and emotional pressure over the last few weeks to determine whether a high affect load may be inducing a temporary period of bad dreams or nightmares.

Research agenda

Progress in the study of nightmares is hindered by several methodological difficulties. Previous models of nightmare production offer limited experimental designs for surmounting these difficulties and properly testing the putative adaptive functions of dreaming and nightmares. The proposed neurocognitive model offers several new options for advancing nightmare research. Future research could attempt to:

- Develop new measures of, and clarify relationships between, affect load and affect distress; assess relationships with nightmare frequency and other psychopathological indicators.
- Find new means of recording nightmare episodes (laboratory or ambulatory) with polysomnographic and psychophysiological measures.
- Comparatively assess the sleep architecture and dream content of patients with a range of nightmare complaints: bad dreams, idiopathic nightmares, trauma-related nightmares (but no PTSD), replicative nightmares (with PTSD).
- Investigate links between waking and dreaming expressions of nightmare production mechanisms by conducting waking-state psychophysiological, cognitive and neuroimaging studies of different nightmare populations.
- Assess individual differences in personality and psychopathology associated with nightmares and a range of other fear-based symptoms.

Acknowledgments

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