Disturbed Dreaming, Posttraumatic Stress Disorder, and Affect Distress: A Review and Neurocognitive Model

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Nightmares are common, occurring weekly in 4%–10% of the population, and are associated with female gender, younger age, increased stress, psychopathology, and dispositional traits. Nightmare pathogenesis remains unexplained, as do differences between nontraumatic and posttraumatic nightmares (for those with or without posttraumatic stress disorder) and relations with waking functioning. No models adequately explain nightmares nor have they been reconciled with recent developments in cognitive neuroscience, fear acquisition, and emotional memory. The authors review the recent literature and propose a conceptual framework for understanding a spectrum of dysphoric dreaming. Central to this is the notion that variations in nightmare prevalence, frequency, severity, and psychopathological comorbidity reflect the influence of both *affect load*, a consequence of daily variations in emotional pressure, and *affect distress*, a disposition to experience events with distressing, highly reactive emotions. In a cross-state, multilevel model of dream function and nightmare production, the authors integrate findings on emotional memory structures and the brain correlates of emotion.

Keywords: nightmares, distress, neurophysiology of fear memory, posttraumatic stress disorder, psychopathology

Nightmares are the most common form of disturbed dreaming. Vivid, with emotions escalating at times to the brink of terror, they are manifestations of the dramatic autonomic and cognitive fluctuations that can arise during rapid eye movement (REM) sleep and, under certain circumstances, during Stage 2 sleep. Nightmares can be simple and benign or so emotionally severe that they beg for comparison with psychotic episodes (C. Fisher, Byrne, Edwards, & Kahn, 1970; Hartmann, 1984; Sullivan, 1962). Occasional nightmares are almost ubiquitous in the general population. However, frequent distressing nightmares are more common than generally thought, affecting 4%–10% of individuals by conservative estimates. Nightmares are also extremely common following trauma exposure. Posttraumatic nightmares may depict trauma-related content and, in cases of chronic posttraumatic stress disorder (PTSD), may at times replicate the trauma with great distress (for review, see Mellman & Pigeon, 2005).

There has been a proliferation of experimental work on posttraumatic and nontraumatic nightmares in the past 15 years, the vast majority of which has examined the psychopathological correlates of individuals with frequent nightmares. Furthermore, recent advances in neuroscience have greatly expanded our understanding of dreaming and related brain functions (Maquet, 2000; Maquet et al., 2000) as well as the pathogenic mechanisms implicated in fear conditioning and the development of PTSD (LeDoux, 2000; Ohman & Mineka, 2001). However, despite the clear implications of these developments for understanding nightmares, there are still no comprehensive models of nightmare etiology and no conceptual frameworks for understanding the link between posttraumatic and nontraumatic nightmares. There has not even been a systematic review of the nightmare literature since an article published in *Psychological Bulletin* 35 years ago (Hersen, 1972). Therefore, the present review is designed to provide an update of research published since the last major review, focusing on issues of operationalizing the concept of nightmares, estimating the prevalence of nightmares with diverse measurement techniques, explaining the pathophysiology of nightmares, and systematizing the empirical work on personality and psychopathological correlates of nightmares. We organize this research around a model of nightmare formation that takes into account both cognitive–emotional and neural explanatory concepts and that suggests a distinction between two principal factors in nightmare production: affect load and affect distress. Future research directions are also considered at key junctures in the article.

Purpose and Overview

The basic argument of our review is as follows. First, in distinguishing clinical- from research-inspired nightmare definitions, we suggest that conceptual differences such as emotion range and affect distress have led to an underestimation of nightmares in the general population. Accordingly, we highlight discrepancies in

nightmare definitions from study to study and indicate how these discrepancies are an obstacle to integrating the studies' findings. Second, in examining nightmare prevalence and frequency as a function of age, gender, clinical pathology, and other variables, we underscore additional factors masking the true occurrence of nightmares. We suggest that a dual-process conception of nightmares is emerging by which both the frequency of nightmares and the emotional distress engendered by them determine the severity of nightmare pathology. Third, we support this dual-process conception by demonstrating relationships between a wide spectrum of pathologies (anxiety, schizophrenia, sleep disorders, etc.) on the one hand and measures of both nightmare frequency and nightmare distress on the other. The most recent studies have suggested that nightmare distress may be the more influential determinant of waking psychological distress and may also be associated with the hyperarousal symptom cluster of PTSD.

Finally, we elaborate this dual-process conception within a heuristic multilevel model of nightmare production. This model incorporates recent findings from the literature on fear conditioning and the neurophysiological correlates of sleep and dreaming and proposes a unified framework for explaining a range of disturbed nocturnal imagery. The latter includes normal dysphoric dreaming; occasional idiopathic nightmares and bad dreams; and recurrent, highly distressing nightmares associated with trauma. A key assumption of this model is *cross-state continuity* or the assumption that some structures and processes implicated in nightmare production are also engaged during the expression of pathological signs and symptoms during the waking state. In line with this assumption, we organize our review of the nightmare literature around two key factors, affect load and affect distress, which are presumed to account for nightmare prevalence and nightmare distress respectively. This approach opens the field of nightmares to research that exploits a variety of methodological paradigms heretofore limited in application to patients in an awake state. A second key assumption is *multilevel explanation*, that is, that nightmares are best explained with constructs from both cognitive–emotional and neural levels of discourse. Each of these explanatory levels offers insights into the mystery of nightmare etiology, but the field is still far from achieving a complete synthesis. We conclude the review by offering a number of suggestions for empirical verification of key components of the model.

Nightmare Definitions

Over the past half-century, the nature, correlates, and consequences of nightmares have been progressively clarified. Whereas early investigators confounded nightmares with other sleep disturbances, such as somnambulism, sleep paralysis, and sleep terrors (Jones, 1951; Mack, 1970), subsequent laboratory studies have shown nightmares to be a widespread and relatively distinct phenomenon associated primarily with REM sleep (C. Fisher, Byrne, Edwards, & Kahn, 1970; Hartmann, 1984). Nightmares are now contrasted with somnambulism and sleep terrors, which are considered to be disorders of arousal (Broughton, 1968) that arise exclusively from non-NREM (NREM) sleep (C. Fisher, Kahn, Edwards, Davis, & Fine, 1974; Hartmann, 1984; Joncas, Zadra, Paquet, & Montplaisir, 2002; Kavey, Whyte, Resor, & Gidro-Frank, 1990). Attempts have also been made to distinguish night-

mares that follow trauma exposure (posttraumatic nightmares) and either make reference to the trauma (trauma-related nightmares) or replicate the traumatic event (replicative nightmares) from those that have neither a discernible relationship to trauma (nontraumatic nightmares) nor any other discernible trigger (idiopathic nightmares) (American Sleep Disorders Association, 2005; Mellman & Pigeon, 2005). Advances have been made in developing a standard definition of nightmares, but a wide consensus is still lacking. This difficulty is analogous to the recent failure of a task force to reach consensus on a definition of dreaming (Pagel et al., 2001). In the absence of consensual agreement, we suggest that definitions of nightmares may be conveniently divided into two broad classes: *diagnostic definitions* that apply primarily to clinical practice and *operational definitions* that are used primarily in research settings. Optimally, a standardized definition of nightmares would incorporate the major features of both classes. For our purposes, we have included all forms of disturbed dreaming in the review even while we recognize that differences exist in precisely how the particular form of nightmare was defined across studies. We consider all forms of disturbed dreaming to be phenotypic variants of a common underlying genotype, namely dysphoric imagery produced during sleep, and suggest that the consequences of these variants are largely dictated by waking responses to the imagery (e.g., distress). Later in the article, we offer a typology of disturbed dreaming based on affect load and distress factors.

Nightmares Defined Clinically

Current diagnostic definitions of nightmares can be found in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev.; *DSM–IV–TR*; American Psychiatric Association, 2000) and in the revised edition (*ICSD–R*; American Sleep Disorders Association, 1997) and second edition (*ICSD–2*; American Sleep Disorders Association, 2005) of the *International Classification of Sleep Disorders*. Although these definitions are relatively consistent in linking nightmares to abrupt awakenings from primarily late-night REM sleep with clear recall of fearful dream content, the newer *ICSD–2* criteria deviate from the earlier definitions in a few key respects. First, the *ICSD–2* improves upon past definitions in acknowledging for the first time that nightmares may involve dysphoric emotions of all kinds, in addition to fear and anxiety (K. Belicki & Cuddy, 1991; Zadra & Donderi, 1993). It also specifically distinguishes idiopathic nightmares from the more severe and highly distressing nightmares associated with PTSD. Third, the *ICSD–2* specifically does not subscribe to a criterion of subjective distress as do the *ICSD–R* and *DSM–IV–TR*. This is a decision taken by the *ICSD–2* nosological committee that affects the entire *ICSD–2* spectrum of sleep disorders. It is a particularly unfortunate decision in the diagnosis of nightmares because converging evidence suggests that an individual's habitual manner of affective responding is an important determinant of the nightmare phenomenon. Subjective distress induced by nightmares (nightmare distress) is now appreciated to be a robust correlate of other psychopathological indicators and of a patient's interest in treatment. We consider nightmare distress a cardinal clinical feature of nightmares that is also part of a more general affective profile that we term *affect distress*.

In sum, the *ICSD–R* and *ICSD–2* criteria acknowledge, but do not formalize, the key fact that nightmare-induced suffering or

distress is central to the disorder. Conversely, the distress criterion specified in the *DSM–IV–TR* clearly acknowledges that nightmares cause "clinically significant distress or impairment in social, occupational, or other important areas of function." (American Psychiatric Association, 2000, p. 631). In a later section, affect distress is described in more detail and distinguished from the affect load factor.

Formal diagnostic definitions are seldom used in clinical research settings. In fact, we located only two studies that report having applied the *DSM–IV* (American Psychiatric Association, 1994) criteria for nightmare disorder in participant selection (Agargun et al., 2004; Ohayon, Morselli, & Guilleminault, 1997). These groups reported prevalence estimates for nightmares that were quite low (2%) compared with many other estimates (5%–10%; see nightmare prevalence section below). The relative paucity of studies using clinical nightmare definitions is due, in part, to the fact that a diagnosis of nightmare disorder requires the exclusion of other anxiety disorders which, in turn, requires a more thorough clinical evaluation of participants. Furthermore, although our impression is that, because of the central role of affect distress, nightmare disorder is rarely independent of other anxiety disorders (Levin & Basile, 2003; Nielsen, Laberge, Tremblay, Vitaro, & Montplaisir, 2000), the issue of comorbidity with other psychiatric disorders has never been investigated systematically. Finally, the *ICSD–2* and *DSM–IV–TR* definitions of nightmares no longer require that nightmares originate in REM sleep or that sleep laboratory-confirmed parameters, such as a minimum of 10 min in REM sleep during the nightmare episode, be met for a positive diagnosis of nightmares. This change may well reflect the fact that the clinical definition of nightmares ultimately rests upon subjective evaluations of the nature and distressing influence of dream imagery.

Nightmares Defined in Research

Operational definitions of nightmares vary substantially among research studies, and no consistent definition has emerged. Traditionally, investigators have used retrospective questionnaires that require participants to estimate the frequency of their nightmares over the preceding weeks or months without necessarily defining what nightmares are. As seen in Table 1, a number of different metrics of nightmare and/or bad dream frequencies have been applied. The most common are binary and ordinal scales, and the most statistically sound are interval scales. More recently, daily home logs have supplanted retrospective questionnaires as the gold standard for nightmare frequency estimation. These log-based studies typically include a specific definition of nightmares. Log measures suggest that nightmares are from 3 to 10 times more frequent than would be expected from retrospective questionnaires (Salvio, Wood, Schwartz, & Eichling, 1992; Wood & Bootzin, 1990; Zadra & Donderi, 2000). This rate of underestimation greatly exceeds observed discrepancies in recalling non-nightmare dreams (Zadra & Donderi, 2000). Like other behavioral measures such as coping (Todd, Tennen, Carney, Armeli, & Affleck, 2004), respondents' retrospective reporting may be severely compromised by response biases and memory failures. However, it remains unclear whether the exercise of regularly logging nightmares at home inadvertently increases nightmare frequency. This possibility is supported by the observation that dream recall frequency can be dramatically improved by simple encouragement (Schredl & Montasser, 1996, 1997). However, other evidence (Zadra & Donderi, 2000) demonstrates that the underestimation of nightmare frequency greatly exceeds that of normal dream recall and of other types of dreams.

Investigators have also begun to distinguish disturbing dreams that awaken the sleeper (nightmares) from those that do not (bad dreams; Zadra & Donderi, 2000). Although some studies explicitly require participants to estimate nightmare and bad dream occurrences separately (e.g., Zadra & Donderi, 2000) and some combine the two in a single measure (e.g., Nielsen et al., 2000), most authors make no mention of the distinction. In the latter cases, nightmare prevalence rates may well be increased substantially by the inclusion of bad dreams. Bad dreams are about four times more frequent than nightmares according to home log measures; and, like for nightmares, home logs also estimate bad dream recall to be several times higher than do retrospective self reports (by a factor of about 5:1; Zadra & Donderi, 2000). Although there are many phenomenological similarities between nightmares and bad dreams, it remains unknown whether they are two qualitatively distinct phenomena or a single phenomenon that simply varies in intensity. We suggest that the two are clearly related phenomena that differ in how well they adaptively regulate fluctuations in current levels of affect, or *affect load* as we term it. Because they do not provoke awakenings, bad dreams permit the extinction function of dreaming to continue uninterrupted, producing a decrease in affect load. Nightmares, in contrast, interrupt extinction by provoking an awakening and prevent a concomitant decrease in affect load. In a later section, we review available evidence that supports the implication that bad dreams and nightmares are associated, respectively, with adaptive and maladaptive functioning in this manner.

Clearly, definitions that include bad dreams will lead to prevalence and frequency estimates that are much larger than definitions restricted to nightmares alone. Although this may not be desired in all studies, researchers should at a minimum ensure that participants are made aware of this important operational distinction and report the operational definition that was used.

In the case of research, the evaluation of nightmare distress is used increasingly often. The most widely applied measure of nightmare distress is the retrospective Nightmare Distress Scale (K. Belicki, 1992a, 1992b), a 13-item scale tapping waking attributions about nightmare consequences (e.g., "Do you feel you have a problem with nightmares?") and interest in treatment ("In the past year have you considered getting professional help for your nightmares?"). Several other recent studies have used prospective ratings of nightmare distress (Germain & Nielsen, 2003a; Krakow et al., 2002; Levin & Fireman, 2002a). The latter measures target a specific nightmare event ("How *disturbed* are you by your nightmare *today*?") and may therefore not be tapping the same construct measured by the Nightmare Distress Scale. This point is supported by the finding that these seemingly similar distress measures are significantly correlated ($r = .38$, $p < .001$; Levin & Fireman, 2002a) but account for only about 15% of shared variance. In addition, nightmare distress is often only marginally associated with nightmare frequency ($rs = .20–.35$) and is more strongly associated with measures of psychopathology, personality, and interest in treatment than is nightmare frequency. Thus, interactions between nightmare frequency and distress as well as whether the latter constructs are measured as state or global variables are crucial measurement issues for future research.

Table 1
Common Measures of Nightmare Frequency

Type	Sample questions	Response scale	References	Comments
Binary	Do you suffer from nightmares?	____Yes ____No	(Cernovsky, 1986; Salvio et al., 1992; Salzarulo & Chevalier, 1983)	Parametric statistics not always applicable; easy in large questionnaires; clinically useful but statistically limited
Nominal	How often do you recall a nightmare?	____ never ____ rarely ____ sometimes ____ often	(Cirignotta et al., 1983; Klink & Quan, 1987; Nielsen et al., 2000; Partinen, 1994)	Parametric statistics not typically applicable; easy in large questionnaires
Ordinal	How often do you recall a nightmare?	____ 0 times/month ____ 1–2 times/month ____ 5–10 times/month ____ 11–20 times/month	(Levin, 1994, 1998; Schredl, 2003)	Easy for subjects; often erroneously used with parametric statistics
Interval	How often do you recall a nightmare?	____ 0 nights/year ____ 1 night/year ____ 2 nights/year ____ 3 nights/year ____ 4 nights/year ____ 5 nights/year ____ 6 nights/year ____ 7 nights/year ____ 8 nights/year ____ 9 nights/year ____ 10 nights/year ____ 11 nights/year	(Krakow et al., 2000)	Can be difficult for subjects because of large numbers of categories
Open ended	How often do you recall a nightmare?	____/month	(Nielsen et al., 2003; Zadra & Donderi, 2000)	Easy for subjects; prone to errors and unexpected responses (e.g., 5–12/month)
Content based	Do you have a current or past problem with nightmares?	Any of the previous response scales	(Bixler, Kates, & Soldatos 1979)	May be prone to confounds (e.g., response bias, trait distress)
Third party	Does your child suffer from nightmares?	Any of the previous response scales	(Mindell & Barrett, 2002)	Parental estimates of symptoms are generally less accurate than children's

Summary and Proposed Typology

Several trends in the clinical and research-based definitions of nightmares over the last several decades have influenced our current understanding of nightmare phenomena. Both clinical and research studies call attention to the importance of assessing nightmare distress as integral to defining pathology and of distinguishing the number and severity of traumatic precursors. Research studies further emphasize the importance of distinguishing nightmares, which awaken the sleeper, from bad dreams, which do not. These distinctions, plus developments such as recognizing the diversity of negative emotions in nightmare content and the need to assess nightmares prospectively with daily logs, have contributed to the emerging impression that nightmares are a much more frequent and prevalent phenomenon than was previously thought. This conclusion qualifies the findings of most studies in the following section on nightmare prevalence because these key considerations have not been taken into account by most of them.

We suggest that the affect load and affect distress concepts be incorporated into a more comprehensive typology of disturbed dreaming. In our proposed typology in Figure 1, increasing affect load leads to elevated frequencies of all types of dreams but especially dysphoric dreams, bad dreams, and nightmares low in distress. Increasing affect distress, however, leads to more numerous and more severe nightmares high in distress. This includes trauma precursors, whose increasing numbers and severity will further increase distress. Thus, individuals experiencing height-

ened affect load but who are low in distress may experience nontraumatic nightmares that resolve quickly and are not accompanied by significant waking suffering.

This typology essentially postulates a severity continuum while highlighting factors that contribute to it and that permit us to distinguish among nightmare subtypes. It also underscores the need for evaluating nightmares multidimensionally. As the present review indicates, validated subjective measures are now available for assessing nightmare distress, awakenings from sleep, and trauma precursors, whereas affect load can only be evaluated indirectly (nightmare frequency, daytime emotional concerns). A clearly desirable development in this field would thus be the development of more objective measures of distress (e.g., heart rate variability), awakenings (e.g., electroencephalogram arousals and microarousals), and affect load (e.g., facial electromyogram) which could be applied for both nosological and research purposes. In later sections, specific normal and pathogenic processes contributing to the affect load and distress constructs are discussed in greater detail.

Nightmare Prevalence

Nightmare Prevalence and Frequency Are High in Healthy and Clinical Populations

Occasional nightmares are quite prevalent in the general population. Large community-based epidemiological studies indicate that about 85% of adult respondents report experiencing at least
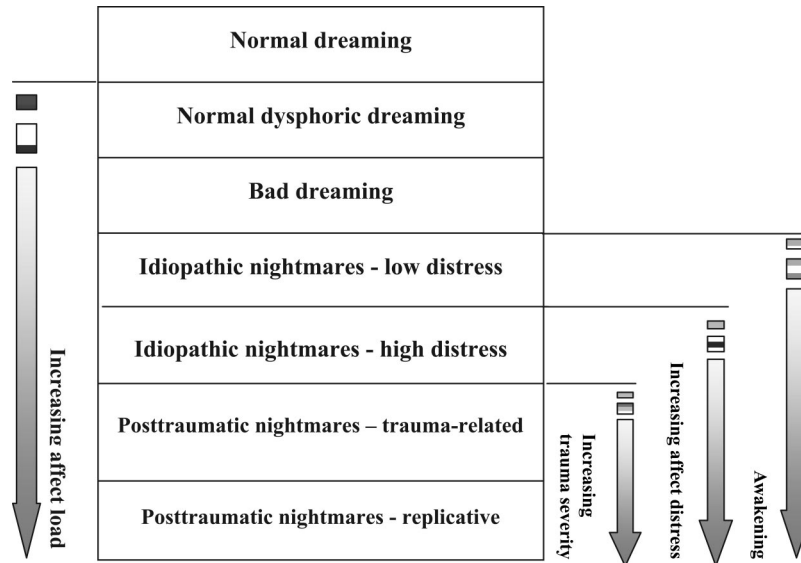
Figure 1. Typology of dreaming organized by increasing affect load, affect distress, and trauma severity. Normal dreaming, dysphoric dreaming, bad dreaming, and idiopathic nightmares with low distress are all considered to be nonpathological in that trauma precursors are absent, and participants report low levels of associated distress. Awakenings from sleep distinguish dysphoric and bad dreaming from idiopathic nightmares with low distress. Idiopathic nightmares with high distress, posttraumatic trauma-related nightmares, and posttraumatic replicative nightmares are all considered pathological because of the subjective affect distress associated with them. Posttraumatic nightmares produce even more distress in step with the number and severity of trauma precursors.

one nightmare within the past year, whereas between 8% and 29% report monthly nightmares (D. Belicki & Belicki, 1982; Bixler, Kales, Soldatos, Kales, & Healy, 1979; Haynes & Mooney, 1975; Levin, 1994; Ohayon et al., 1997). Although relatively infrequent nightmares are not generally considered to reflect clinical pathology, this issue has not been studied empirically.

On the other hand, these same studies indicate that 2% to 6% of respondents report weekly nightmares, a frequency which corresponds to the *ICSD–R* definition of moderately severe nightmares. This estimated range of 2%–6% is highly robust across cultures, with similar rates reported in Canada (D. Belicki & Belicki, 1982; Coren, 1994); France (Ohayon et al., 1997); Iceland, Sweden, and Belgium (Bengtsson, Lennartsson, Lindquist, Noppa, & Sigurdsson, 1980; Janson et al., 1995); Austria (Stepansky et al., 1998), Finland (Hublin, Kaprio, Partinen, & Koskenvuo, 1999b); Japan (Fukuda, Ogilvie, & Takeuchi, 2000); the Middle East (Najam & Malik, 2003); and the United States (Bixler, Kales, Soldatos, et al., 1979; Feldman & Hersen, 1967; Levin, 1994).

Although a clinically significant frequency of nightmares is relatively common cross-culturally, the 2%–6% range reported may considerably underestimate this frequency for several reasons in addition to the definitional issues discussed in the previous section (the more recent *ICSD–2* general population estimate of individuals with "a current problem with nightmares" is 2%–8%; American Sleep Disorders Association, 2005). In the following sections, we describe several of these, including participant age and gender, comorbid clinical conditions, and additional methodological pitfalls that may lead to an underestimation of the daily occurrence of nightmares.

Nightmares Are More Prevalent in the Young and Decrease in the Elderly

Nightmare occurrence is most common in childhood through young adulthood and then declines with increasing age. However, the stability of nightmares within individuals across the life span and the possibility of predispositions, if any, to the initial onset of nightmare experience have not been investigated. Indeed, the test–retest reliability for measures of nightmares across time intervals exceeding 2 months is unknown.

Frequent nightmares are particularly common in childhood and adolescence—perhaps up to three to four times as prevalent as in adulthood. According to the *DSM–IV–TR*, 10%–50% of children ages 3–5 have disturbing dreams (unspecified frequency), with prevalence rates rising through early adolescence (B. E. Fisher et al., 1989; MacFarlane, Allen, & Honzik, 1954; Nielsen et al., 2000; Salzarulo & Chevalier, 1983; Simonds & Parraga, 1982). Partinen (1994) found that between 5% and 30% of children experience frequent nightmares—a range greater than for adults. Similarly, Mindell and Barrett (2002) reported that over 25% of their sample of 60 children ages 5–11 self-reported at least weekly nightmares. Nightmare "problems," defined as persistent nightmares lasting longer than three months, occurred with prevalences in the same order of magnitude: 24%, 41%, and 22% for children in the age ranges of 2–5, 6–10, and 11–12 years respectively (Salzarulo & Chevalier, 1983). Other surveys (Simonds & Parraga, 1982; Vela-Bueno et al., 1985) have demonstrated similarly high levels of self-reported nightmares (20%–30%) for children ages 5–12 but over longer periods of time (at least one nightmare per 6 months).

In contrast, the few studies that have examined nightmare prevalence and frequency in elderly populations report considerably lower rates than those found in younger adults. In one controlled study comparing healthy elderly participants with college students (Salvio et al., 1992), older adults were only 20% as likely as students to experience problems with nightmares. In a second study of pulmonary patients with and without asthma (Wood, Bootzin, Quan, & Klink, 1993), nightmare frequency was inversely related to age. In a third study (Partinen, 1994), the "often" or "always" occurrence of nightmares among the elderly fell to between 1% and 2% compared with 2% and 5% for young adults (i.e., to about 50% of the young adult levels). In a fourth cross-sectional investigation of 1,300 women, Bengtsson et al. (1980) also found that nightmare prevalence and frequency decreased with increased age. In contrast, Tanskanen et al. (2001) found a slight but anomalous increase in the proportion of participants with frequent nightmares with increasing age, particularly in men, in a large cross-sectional study of over 36,000 respondents from the general population. However, a closer examination of these results revealed a curvilinear relationship between age and percentage of individuals who report nightmares, with peak reporting ages falling between 45 and 54 years of age. However, these findings should be regarded with caution, as they were based on a single questionnaire item with vaguely defined response categories ("never," "occasionally," "frequently"). More recently, our Internet-based study of 24,102 respondents (Nielsen, Stenstrom, & Levin, 2006) found that nightmare frequency (as estimated for a typical month) peaked between the ages of 20 and 29 and declined steadily with increasing age (see Figure 2).

In sum, studies consistently find nightmares to be at their most prevalent in childhood and adolescence, to remain elevated in young and middle adulthood, and subsequently to decline in later adulthood. However, these estimates are based in large part on cross-sectional, retrospective measures that often use nominal response categories, a factor that may introduce substantial variance into the accurate estimation of nightmare changes as a function of age. Longitudinal studies (e.g., Nielsen et al., 2000) would be invaluable in establishing true nightmare lifespan prevalence rates. Moreover, because most studies of nightmares in children are based upon information provided by mothers, which is known to underestimate children's own reporting of nightmares (Lapouse & Monk, 1959), the prevalence of nightmares among children may be even higher than suggested in the literature. In addition, few studies have investigated the role of genetic influences in nightmare occurrence. Evidence from the Finnish Twin Cohort, a community-based sample of 1,298 monozygotic and 2,419 dizygotic twin pairs ages 33–60 years (Hublin et al., 1999b), suggests that nightmares are a common and stable trait from childhood to middle age and are substantially affected by genetic factors. Through structural equation modeling, Hublin et al. (1999b) found the genetic contribution to nightmares to be 44% for men and 45% for women in the case of childhood nightmares and 36% for men and 38% for women in the case of adult nightmares. Hublin et al. also concluded that environmental factors played a role in the expression of the genetic component, although because of extensive variation among populations, the nature of these factors could not be specified. Also, because these results were collected retrospectively from adult participants, recall bias can not be excluded as a confounding factor.

Nightmares Are More Prevalent Among Women

Women at all ages consistently report nightmares at significantly higher rates than do men; however, it is not clear what portion of these nightmares is trauma related (Claridge, Clark, & Davis, 1997; Feldman & Hersen, 1967; Hartmann, 1984; Hersen, 1971; Hublin et al., 1999b; Levin, 1994; Nielsen et al., 2000; Nielsen & Levin, 2005; Ohayon et al., 1997; Schredl & Pallmer, 1998; Tanskanen et al., 2001). For example, in a sample of over 3,433 college students (1,509 men and 1,924 women) collected over 4 years, Levin (1994) found that women were about 50% more likely to report frequent nightmares (once a month or more) than men (12% and 8% respectively). Similarly, as shown in Figure 2, our study of over 24,000 Internet respondents ages

Figure 2. Mean retrospective estimates of nightmare recall in a typical month, log-transformed (± *SEM*; left *y*-axis) and per-month equivalents (right *y*-axis), for a sample of respondents to an internet questionnaire (*n* = 4,627 boys and men, *n* = 19,475 girls and women). Women recall more nightmares than men at ages 10–59 (*p* < .05). Age differences (men and women combined) were significant for all adjacent strata comparisons (*p* < .001). NM = nightmares; log(NM + 1) = base 10 log of nightmares/month + 1. Adapted from "Nightmare Frequency as a Function of Age, Gender, and September 11, 2001: Findings From an Internet Questionnaire," by T. A. Nielsen, P. Stenstrom, & R. Levin, 2006, *Dreaming, 16,* p. 149. Copyright 2006 by the American Psychological Association.

10–59 years (Nielsen et al., 2006) found that women reported more nightmares than men by an overall factor of 1.3:1 (4.4 vs. 3.4 nightmares/month). This difference was highest among 10–19-year-olds (1.39:1) and 20–29-year-olds (1.36:1) and dropped with age (30–39-year-olds = 1.11:1, 40–49-year-olds = 1.05:1, and 50–59-year-olds = 0.96:1). Biases due to individual differences in Internet use were not controlled in these results, however, suggesting caution in their interpretation. Although no study has systematically investigated when this consistent gender gap in nightmares first appears, two investigations (Nielsen et al., 2000; Schredl & Pallmer, 1998) suggest that it may emerge in early adolescence. In a longitudinal investigation of 610 children, Nielsen et al. (2000) reported significant gender differences in prevalence at age 13, the earliest age recorded in the study, with 37% of girls reporting disturbing dreams "sometimes" or "often" compared with 25% of boys. However, the disparity became more accentuated at age 16 with over 40% of girls reporting disturbing dreams compared with 20% of boys. The change over time was due to both an increase in nightmares for girls (more girls than boys reporting nightmares "often") and a decrease for boys (more boys than girls reporting them "never"). In addition, it is noteworthy that the only study to investigate reported nightmare frequency across a broad age range in preadolescence (5–11 years) found no gender differences by age (Mindell & Barrett, 2002). However, in the absence of large, well-controlled longitudinal studies, the precise age of onset for nightmares cannot yet be identified with certainty.

In sum, much converging evidence reveals a robust gender difference in nightmares, with women consistently reporting nightmares with either a higher prevalence or frequency than men. This difference appears to emerge in early adolescence and to be maintained across the lifespan although the magnitude of the difference lessens among older participants.

More Prevalent Nightmares Among Women May Reflect Broader Gender Differences

Although many explanations for the gender difference in nightmares have been proposed, most have in common the possibility that a differential augmentation of either affect load or affect distress is at play for women. Some have suggested that women possess self-report biases and a tendency to report distressing experiences to others to a greater degree than do men (K. Belicki, 1992a; Hartmann, 1984; Levin, 1994). However, such biases may reflect real differences in affect load or distress. In fact, prevalence studies indicate that women report both a greater quantity and a greater intensity of symptoms related to negative emotional disturbances, such as depression and anxiety (Buss, 1988; Nolen-Hoeksema, 1990). Moreover, not only do women have higher rates of dream recall than men, regardless of age (Levin, 1994; Nielsen et al., 2000; Saarenpää-Hekkilä, Rintahaka, Laippala, & Koivikko, 1995; Schredl & Pallmer, 1998), but they rate both their dreams and nightmares as more vivid and meaningful (K. Belicki, 1992a, 1992b; Levin, 1994) and report that their dreams have greater impact on their waking behavior than do men.

Another explanation for the gender difference in nightmare reporting is that women may be more vulnerable to certain risk factors thought to produce nightmares, including higher rates of sexual and physical abuse, anxiety and depressive disorders, and sleep disorders (American Psychiatric Association, 2000). In par-

ticular, PTSD is more prevalent in women than in men, irrespective of age (Breslau, Davis, Andreski, Peterson, & Schultz, 1997; Garrison et al., 1995; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Stein, Walker, Hazen, & Forde, 1997). It is possible that these types of general risk factors facilitate the early development of an underlying pathological factor, such as heightened affect distress, that renders women more prone to experiencing nightmares.

A third possible explanation of the gender difference is that nightmares are a function of processes that also favor the emergence of depression in early adolescent girls. This, too, may reflect a differential increase in affect load or distress among women. Nolen-Hoeksema & Girgus's (1994) review supports the possibility that, although the gender difference in depression emerges only after age 15, preadolescent girls nevertheless are more likely to develop more psychosocial depressiogenic risk factors (e.g., low instrumentality, aggression and interpersonal dominance, high pessimistic attributional styles, ruminative coping, and bodily dissatisfaction) than are boys but that these risk factors lead to depression only when they interact with new challenges and stressors experienced in early adolescence. Thus, even if boys and girls face equal numbers of challenges and stressors in adolescence, the demand on girls' emotional regulation capacities will be greater, and girls will manifest depression more often than will boys. A similar interaction of risk factors and adolescent stressors may lead to more frequent spikes in disturbed dreaming, increased waking distress, and nightmares among girls. Although this question has not been investigated directly, the fact that nightmare frequency at age 5 is one of a number of early signs (e.g., bed wetting at age 5, motor skills at age 5, trait dominance at age 9–11) that predict an earlier age of first intercourse in preadolescent girls (Udry, Kovenock, Morris, & van den Berg, 1995) is consistent with Nolen-Hoeksema and Girgus's version of such a diathesis–stress explanation. In addition, work reviewed earlier suggests that gender differences in nightmare reporting emerge roughly at the same age as has been documented for depression. On the other hand, Cambell and Udry (1995) found no relation between age at menarche and nightmare frequency. Investigating this question in a longitudinal design is an important direction for future research and could further clarify relationships between waking and dreaming as well as the role dreaming may play in emotion regulation.

A fourth explanation is based upon consistently observed gender differences in coping styles (Lyubomirsky & Nolen-Hoeksema, 1995; Nolen-Hoeksema, 1990) and also is linked to possible differences in affect load or distress. Women favor emotion-focused coping, whereas men more commonly use avoidant or disengaged coping. Further, women's tendency to use a ruminative style of coping, which involves a difficulty in distracting one's attention away from the causes and consequences of emotional responses to stress, has been suggested as an explanation for the 2:1 gender difference in depression rates (Nolen-Hoeksema & Girgus, 1994; Orr et al., 2000).

A fifth explanation for the gender difference in nightmares is that of biological differences in emotional brain processes. For example, recent work has shown that women demonstrate better episodic emotional memory and larger physiological responses to emotional stimuli than do men (Bradley, Codispoti, Sabatinelli, & Lang, 2001). Such differences clearly may also include heightened affect distress responses.

To summarize, the well-documented gender difference in nightmare frequency may be due to a number of factors that reflect the differential influence of affect load and/or distress on women. These include biases or readiness to report nightmares, processes shaping the development of depression, vulnerability to nightmare-inducing risk factors (e.g., trauma) and memory for and responding to emotional stimuli.

Nightmares Are Particularly Prevalent Among Clinical Populations

That nightmares appear to be more frequent and more prevalent in psychiatric populations (Berlin, Litovitz, Diaz, & Ahmed, 1984; Cernovsky, 1986; Hersen, 1971; Levin, 1998; Ohayon et al., 1997; Tanskanen et al., 2001) supports a role for affect distress in nightmare production. Historically, psychoanalytic investigators (Federn, 1953; Freud, 1920; Jones, 1951; Mack, 1970) have emphasized the continuity between frequent nightmares and psychosis, two experiences in which the individual struggles to ward off psychological fragmentation and self-annihilation (Stone, 1979; Teixeira, 1984). Furthermore, nightmares have been noted by clinicians to immediately precede the onset of acute psychotic episodes (Arieti, 1974; Bleuler, 1950; Donlon & Blacker, 1973; Fennig, Salganik, & Chayat, 1992; Levin & Daly, 1998). Herz and Melville (1980) found that bad dreams were among the most common symptoms of relapse in two large independent cohorts of schizophrenic patients.

Hersen (1971) reported an increased prevalence of nightmares in a sample of psychiatric inpatients although he did not use a control group for comparison. In addition, both Berlin et al. (1984) and Brylowski (1990) noted high rates of nightmares in psychiatric emergency settings. Although such studies provide consistent results, some methodological problems limit their comparability, for example, failure to report exact frequency rates, information regarding pre-hospitalization nightmares (for comparison), and information regarding categorization by psychiatric diagnosis.

Numerous studies have found that nightmare prevalence and/or frequency is associated with psychopathological traits or symptoms (Hartmann, 1987; e.g., Hartmann, Russ, van der Kolk, Falke, & Oldfield, 1981; Kales et al., 1980; Levin, 1998; Levin & Fireman, 2002a; Zadra & Donderi, 2000). These studies have primarily used nonclinical participants who complete psychopathology measures. Controlled studies investigating prevalence and frequency rates by diagnostic category in established clinical populations are generally lacking. One exception to this has been in the investigation of PTSD in which poor sleep in general and nightmares in particular have been documented in as many as 90% of affected individuals (e.g., Kilpatrick et al., 1998; Woodward, Arsenault, Murray, & Bliwise, 2000). Indeed, nightmares are considered a hallmark symptom of the disorder (Harvey, Jones, & Schmidt, 2003; Ross, Ball, Sullivan, & Caroff, 1989), and disturbed dreaming has been found consistently to be an integral feature of the PTSD re-experiencing/intrusion symptom cluster across a broad range of traumatic events, such as combat exposure, traffic accidents, crime victimization, rape, and natural disasters (Krakow et al., 2002; Kramer & Kinney, 1988; Mellman, David, Kulick-Bell, Hebding, & Nolan, 1995; Neylan et al., 1998; Ohayon & Shapiro, 2000; van der Kolk, Blitz, Burr, Sherry, & Hartmann, 1984).

Nightmares have also been empirically demonstrated to be reactive to intense stress (Cernovsky, 1984c; Coalson, 1995; Cook, Caplan, & Wolowitz, 1990; Hartmann, 1984; Picchioni et al., 2002; Wood, Bootzin, Rosenhan, Nolen-Hoeksema, & Jourden, 1992) and often to accompany a number of chronic health problems, including migraine headaches (Levitan, 1984), bronchitis and asthma (Klink & Quan, 1987), chronic obstructive airways disorder (Krakow, Melendrez, et al., 2001; Wood et al., 1993), and cardiac disease (Asplund, 2003; Parmar & Luque-Coqui, 1998). Among the elderly, nightmares are associated with an increase in irregular heart beats and spasmodic chest pain (Asplund, 2003). Frequent nightmares also afflict substance abusers (Cernovsky, 1985, 1986; Greenberg & Pearlman, 1967; Gross et al., 1966; Lansky & Bley, 1992). More complete descriptions of disturbed dreaming in medical conditions and adverse reactions to drugs can be found in Nielsen's (2005) and Thompson and Pierce's (1999) studies.

Together, these areas of clinical research are consistent with the possibility that increased levels of affect distress are common to both psychiatric and behavioral health symptoms and nightmares. Thus, clarifying relationships between physical health functioning and disturbed dreaming is an avenue of research that may help elucidate the pathogenesis of nightmares. Such research should take into consideration problems with the existing research such as the fact that most patients are seen when in an acute phase of their illness and that confounding variables such as preexisting psychopathology and life stress are usually not controlled.

Other Clinical Conditions Are Marked by Nightmares

The occurrence of nightmares as a central or comorbid symptom of clinical conditions is much more widespread than is generally appreciated (Nielsen, 2005; Nielsen & Zadra, 2000). Globally, nightmares can be viewed as a class of disordered dreams that falls along a continuum of imagery intensification. At one end of this continuum are disorders involving global cessation and impoverishment of dreaming, and at the other end are disorders involving affective intensification, stereotypy, and heightened reality quality (Nielsen, 2005). Intensity appears to vary as a function of concurrent anxiety, stress, and/or sleep disruption. For example, in the case of intensive care unit dream delirium, patients undergo extreme fears of death in addition to the constant disruption of their sleep by medical personnel. Also, in the case of infant peril dreams, mothers in the first few postpartum months bear numerous acute responsibilities for infant care and feeding and are subjected to a constant regimen of sleep interruption. Although a complete discussion of the nosological boundaries of disturbed dreaming is beyond the scope of the present review, we suggest that the processes intrinsic to dysphoric nocturnal imagery are common regardless of the source; that is, the different forms of dreaming are produced by aberrations of processes implicated in the production of normal dreaming (e.g., reality mimesis, emotional activation) and modulated by stress and distress factors. These dynamics are elucidated further in the model of nightmare production described in our model of nightmare production at the conclusion of this review.

Methodological Pitfalls Obscure the Estimation of Nightmare Prevalence

Methodological shortcomings limit the comparability and generalizability of many of the previous observations, and rigorously controlled studies are the exception rather than the rule. Previously, we identified the exclusive focus on nightmare fear, the absence of daily log measures, and the failure to include bad dreams as a type of nightmare as likely contributing factors in underestimating nightmare prevalence. Here we describe some additional, unresolved methodological issues that may influence estimates of nightmare prevalence and frequency. First, variations in retrospective reporting metrics may lead to variance in the estimation of nightmare occurrences. Many researchers (D. Belicki & Belicki, 1982; K. Belicki & Belicki, 1986; K. Belicki & Cuddy, 1991; Feldman & Hersen, 1967; Haynes & Mooney, 1975; Janson et al., 1995; Levin, 1994; Stepansky et al., 1998; Wood & Bootzin, 1990) have categorized nightmare frequency by differing divisions of calendar time (per week, per month, per year) and then prorating these estimates to generate an overall prevalence rate, usually by month or year (e.g., Nielsen et al., 2006). Others have assessed nightmares with general categories, such as "sometimes," "often," and "always" or by dividing respondents into groups that report having a "current" or "past problem" with nightmares (Bixler, Kales, Soldatos, et al., 1979; Cirignotta, Zucconi, Mondini, Lenzi, & Lugaresi, 1983; Klink & Quan, 1987; Partinen, 1994). The latter approaches have been used more often in studies investigating children who may be less able to assess the frequency of their own dream reports per units of time. The effects of such different metrics on estimates of prevalence have not been studied extensively.

Second, like insomnia, disturbed dreaming is not typically assessed in clinical evaluations by primary care physicians and mental health workers. A survey of medical practitioners (Bixler, Kales, & Soldatos, 1979) found that fewer than 4% of patients spontaneously report nightmare complaints to their physicians. Thus, like individuals with other sleep problems, a sizeable proportion of persons with disturbed dreaming do not report their symptoms. This further skews prevalence rates and may result in sampling selection biases. Indeed, there has been no attempt to compare individuals who do and do not spontaneously report nightmare problems to a health care provider.

Third, few studies adequately assess potentially crucial covariates when deriving nightmare prevalence estimates. For example, most studies have used 1st-year college students during the mass testing phase of introductory psychology classes as samples of convenience (D. Belicki & Belicki, 1982; K. Belicki & Belicki, 1986; K. Belicki & Cuddy, 1991; Feldman & Hersen, 1967; Haynes & Mooney, 1975; Levin, 1994; Wood & Bootzin, 1990). Such assessments usually occur within 2 weeks of beginning college, a time of significant life change and adjustment to often novel and stressful situations, conditions that have been demonstrated to increase both overall dream recall (Breger, Hunter, & Lane, 1971) and nightmare prevalence (Wood et al., 1992).

However, although reliance on college students as research participants may bias reporting frequencies and limit the generalizability of results, randomly sampled community surveys (Janson et al., 1995; Stepansky et al., 1998) have largely confirmed prevalence estimates based upon these participants. Specifically, the finding that about 4% of adults from community surveys experience one or more nightmares per week is consistent with the prevalence estimates of 2%–6% found for college students. Three other community surveys (Bixler, Kales, Soldatos, et al., 1979; Cirignotta et al., 1983; Klink & Quan, 1987) assessing prevalence of nightmare problems rather than frequency also confirm the college student-based estimates, indicating that between 5% and 8% of the adult general population report a current problem with nightmares, and about 6% report a past problem.

Fifth, researchers often investigate nightmare prevalence without distinguishing between nontraumatic and posttraumatic nightmares. Given the near ubiquitous recurrence of nightmares following trauma, the failure to exclude posttraumatic nightmares, either by means of self-report, clinical interview or measures of nightmare content, may confound prevalence rates for nontraumatic nightmares. Few studies investigating nightmare prevalence or frequency have assessed lifetime trauma exposure concurrently, a confound that may be crucial in explaining the gender difference in nightmare rates described earlier.

Finally, studies have not distinguished individuals with life-long nightmares (often without any evidence of trauma) from those with recent-onset nightmares, a distinction which may well have implications for several points raised in this review, especially those concerning relationships between nightmares and waking indicators of psychopathology.

Summary

Nightmares have been observed repeatedly to characterize a variety of clinical populations including general and specific psychiatric samples, substance abusers, victims of trauma and severe or chronic stress, patients with several chronic health problems (e.g., migraine, respiratory problems, cardiac disease), and patients under severe stress and sleep disruption. Together, these findings point to the increased salience of nightmares in clinical conditions and suggest fruitful avenues for further study of the pathological dynamics of nightmare formation.

Among the outstanding methodological problems to consider, practice and research over the last several decades have emphasized the frequency of nightmares to the exclusion of other potential subjective and objective measures. Continued improvement to methods of assessing nightmare frequency is certainly still desirable, but recent studies have indicated that frequency is only one determinant of the severity of nightmares as a health problem. Emotional distress is another determinant of pathological severity that likely affects waking psychological functioning at least as much as nightmare frequency. In the following section, we explore the breadth and depth of this distinction between frequency and distress by reviewing evidence that both nightmare dimensions are associated with various clinical pathologies.

Nightmares and Psychopathology: Central Roles for Frequency and Distress

A growing body of research has converged in linking nightmare prevalence, frequency, and/or distress to a broad spectrum of mental health pathologies (Agargun et al., 2003; K. Belicki, 1992a; Bernert, Joiner, Cukrowicz, Schmidt, & Krakow, 2005; Berquier & Ashton, 1992; Blagrove, Farmer, & Williams, 2004; Cellucci &

Lawrence, 1978; Chivers & Blagrove, 1999; Claridge et al., 1997; Feldman & Hersen, 1967; Fennig et al., 1992; Hartmann, 1984; Hartmann & Russ, 1979; Hartmann, Russ, Oldfield, Sivan, & Cooper, 1987; Hartmann, Russ, et al., 1981; Hawkins & Williams, 1992; Haynes & Mooney, 1975; Hersen, 1971; Kales et al., 1980; Kramer, Schoen, & Kinney, 1984b; Lester, 1968; Levin, 1989, 1998; Levin & Fireman, 2002a; Levin, Galin, & Zywiak, 1991; Levin & Hurvich, 1995; Levin & Raulin, 1991; Nielsen et al., 2000; Ohayon et al., 1997; Schredl, Kronenberg, Nonnell, & Heuser, 2001; Tanskanen et al., 2001; Wood & Bootzin, 1990; Zadra & Donderi, 2000). Because most of these pathologies are marked by considerable waking emotional distress, their association with nightmares supports our contention that nightmare production is related to a personality style characterized by intense reactive emotional distress (see especially K. Belicki, 1992a, 1992b; Blagrove et al., 2004; Levin & Fireman, 2002a) However, some caveats should be considered in evaluating the following review of this literature and in attempting to generalize its findings. First, most of these investigations rely solely on retrospective measures of nightmare frequency or prevalence and the overall magnitude of associations identified is often moderate at best (rs = .10–.40). Second, with few exceptions, the studies do not distinguish posttraumatic from nontraumatic nightmares or examine whether the nightmares were of relatively recent origin (within the past year) or of lifelong occurrence. Finally, most of these studies do not quantify nightmare distress.

Six broad psychopathology categories are reviewed for evidence of associations with nightmares: anxiety symptoms, neuroticism and global symptom reporting, schizophrenia-spectrum disorders, other psychiatric disorders, behavioral health problems and sleep disturbances, and PTSD.

Nightmares Are Associated With Anxiety Symptoms

Evidence from a large number of studies is generally consistent with the notion that nightmares are linked to anxiety symptoms. Frequent nightmares are associated with anxiety symptoms in children (B. E. Fisher & McGuire, 1990), adolescents (Nielsen et al., 2000), adults (Berquier & Ashton, 1992; Cellucci & Lawrence, 1978; Cook et al., 1990; Feldman & Hersen, 1967; Hartmann et al., 1987; Haynes & Mooney, 1975; Hublin, Kaprio, Partinen, & Koskenvuo, 1999a; Levin, 1989; Levin & Hurvich, 1995; Ohayon et al., 1997; Tanskanen et al., 2001; Zadra & Donderi, 2000), and psychiatric patients (Hartmann, Russ, et al., 1981; Hersen, 1971). More specifically, nightmare frequency is associated with heightened anxiety about death (Dunn & Barrett, 1988; Feldman & Hersen, 1967; Hersen, 1971; Levin, 1989), fears of annihilation (Levin & Hurvich, 1995), and general neuroticism or trait anxiety (Berquier & Ashton, 1992; Haynes & Mooney, 1975; Hersen, 1971; Levin & Fireman, 2002a; Nielsen et al., 2000; Zadra & Donderi, 2000). In one regional sample of 512 participants at the age of 13, adolescents who reported frequent nightmares were considerably more anxious than their low-nightmare counterparts and were more likely to exhibit *DSM–III* (American Psychiatric Association, 1980) symptoms of anxiety disorders, particularly generalized anxiety disorder, separation anxiety, and overanxious disorder at age 16 (Nielsen et al., 2000). Finally, although one study found a positive association between retrospective measures of nightmares and nocturnal panic attacks (Schredl et al., 2001),

two others failed to observe such a relationship (Craske & Barlow, 1989; Mellman & Uhde, 1990). Additionally, some investigators (Dunn & Barrett, 1988; Lester, 1968; Wood & Bootzin, 1990) reported no significant relationships between nightmare frequency and waking anxiety.

In the case of nightmare distress, recent studies suggest robust associations with anxiety symptoms—possibly even to a greater extent than for nightmare frequency measures. This is demonstrated clearly in Levin and Fireman's (2002a) study in which nightmare frequency was found to be largely independent of trait anxiety, but only when nightmare distress was partialled out. In multiple regression analyses, nightmare distress ratings accounted for most of the unique variance shared with self-report measures of psychopathological symptoms tapping negative affect (i.e., State–Trait Inventory; Speilberger, Gorsuch, & Lushene, 1983; Symptom Checklist-90-Revised [SCL-90-R]; Derogatis, 1994; Beck Depression Inventory; Beck & Steer, 1987). Indeed, nightmare distress was a far stronger predictor of psychopathology scores than was nightmare frequency, correlating highly ($p < .001$) with all but three of twenty psychopathology measures and accounting for more variance than the nightmare frequency measure in almost all cases. Similar findings have been reported by other investigators (Blagrove et al., 2004; Zadra, Germain, Fleury, Raymond, & Nielsen, 1999). Notably, the same relationships did not hold for measures of psychopathology characterized by unusual cognitions such as dissociation and schizotypy, suggesting that the latter class of (schizotypal) symptoms may be independent to some extent from the distress component (see section below on schizophrenia-spectrum disorders).

Relationships between nightmares and psychopathology may therefore be clarified by the inclusion of distress measures as well as more rigorous operationalization that distinguishes these distress measures from frequency measures. In addition, greater efforts should be made to standardize the method of reporting in future investigations. A particular emphasis should be placed on using proximal prospective measurements in light of evidence that the relationship between nightmare frequency and distress diminishes significantly when these variables are assessed prospectively rather than retrospectively (Levin & Fireman, 2002b; Wood & Bootzin, 1990).

Two preliminary conclusions can be drawn from these studies of nightmares and anxiety symptoms. First, distress and frequency overlap but are partially independent components of the nightmare experience, with distress more reliably associated with psychopathology that is characterized by high levels of negative affect. The latter finding supports our assumption of cross-state continuity. Second, very different results are obtained when nightmares are measured at the state and trait levels. Evidence of stronger associations between frequency and distress when measured at the state level is consistent with the possibility that sociocognitive factors increase the self-reporting of nightmares on both measures. In this sense, nightmare distress would be analogous to the symptom-reporting dimension of negative affect, which is closely associated with anxiety/depression but often independent of objective health outcome measures (Watson & Pennebaker, 1989). The absence of any objective nightmare outcome measures in most studies (e.g., polysomnography, actigraphic sleep recordings) continues to hamper the clarification of these relationships. Similarly, the frequently noted fact that nightmares rarely occur in the sleep laboratory

(Hartmann, 1970; Woodward et al., 2000) contributes to this problem but may also be partially explained by it (i.e., patients may be less distressed by their nightmares in a context of clinical recording and surveillance). Further, the observation that some laboratory-recorded nightmares often lack psychophysiological evidence of autonomic activation (H. J. Fisher, 1979; Nielsen & Zadra, 2000) might also be explained by such sociocognitive influences.

Nightmares Are Associated With Neuroticism and Global Symptom Reporting

Individuals who report frequent nightmares also report higher levels of neuroticism, a global personality dimension conceptually and empirically related to propensity for negative emotional reactivity (Eysenck & Eysenck, 1975; Watson & Pennebaker, 1989). Three independent investigations (Berquier & Ashton, 1992; Blagrove et al., 2004; Zadra & Donderi, 2000) demonstrated significantly higher levels of neuroticism as measured by the Eysenck Personality Questionnaire (EPQ; Eysenck & Eysenck, 1975) for individuals with frequent nightmares compared with controls. Similarly, Claridge, Davis, Bellhouse, and Kaptein (1998) reported a relationship between EPQ neuroticism and nightmare distress, although they did not assess nightmare frequency. No comparable relationships were reported for the EPQ Psychoticism scale in these studies, again suggesting that nightmares are associated with global maladjustment based upon symptom reporting rather than a specific pattern of pathology.

Similar findings have been reported consistently for such other broad symptom reporting measures as the Minnesota Multiphasic Personality Inventory (Hathaway & McKinley, 1942; see Hartmann et al., 1987; Kales et al., 1980; Najam & Malik, 2003; Nielsen, 2005), the Cornell Medical Index (Brodman, Erdman, Lorge, & Wolff, 1949; see Hartmann et al., 1987; Kramer, Schoen, & Kinney, 1984a), and the SCL-90-R (Derogatis, 1994; see Blagrove et al., 2004; Kales et al., 1980; Levin & Fireman, 2002a; Zadra & Donderi, 2000). Zadra and Donderi (2000) found both the EPQ Neuroticism and SCL-90-R Symptom indexes to be related significantly to both retrospective and prospective nightmare frequency measures. Similarly, Levin and Fireman (2002a) found both the Symptom index and the Global Distress index of the SCL-90-R to be associated with prospective nightmare frequency and nightmare distress measures. Multiple regressions indicated that these two measures predicted over 36% of the SCL-90-R variance although, as expected, the relationships were substantially stronger for the distress measure (semipartial rs =.25 for frequency and .48 for distress). Comparable findings were reported for both the EPQ Neuroticism scale and the General Health Questionnaire (Goldberg & Williams, 1988) in a recent prospective investigation of 147 university students (Blagrove et al., 2004). Similar, marginal relationships of smaller magnitude were also found between global psychopathology scores and measures of disturbing dreams that did not result in awakenings (Blagrove et al., 2004; Zadra & Donderi, 2000). This may indicate that the postawakening aftermath of a nightmare is a source of distress above and beyond the affect inherent in the nightmare's content. This possibility is also consistent with the finding that nightmare reporting is better predicted by waking attribution factors than by

such self-rated phenomenal qualities as nightmare vividness and intensity (Levin & Fireman, 2002b).

In sum, nightmares are associated with heightened neuroticism and measures of general symptomatology. More recent work suggests that a trait-like, affect distress factor mediates these relationships. Because the distress attributed to nightmares is primarily a waking-state phenomenon, experimental investigations of the cognitive attributional and attentional processes of individuals with and without frequent nightmares are clearly needed to further clarify the nature and role of nightmare distress. Furthermore, studies of the cognitive processes of frequent nightmare individuals who do and do not report high levels of concomitant distress would be of particular interest in this regard.

Nightmares and Schizophrenia-Spectrum Disorders

Phenomenological similarities between nightmare attacks and acute psychotic episodes have long been noted by clinicians and science writers (Bleuler, 1950; Sullivan, 1962). Medieval folklore (Jones, 1951) and Western literature and art contain numerous references to nightmares as precursors to, or analogues of, psychosis (Hartmann, 1984; Levin, 1998; Mack, 1970). For example, the writings of Shakespeare, Coleridge, James, Kafka, and Dostoyevsky, as well as the cinematic work of Bergman and Polanski, all use explicit nightmarish imagery to depict the protagonist's descent into psychotic decompensation (Levin, 1998). Thus, a primary focus of early nightmare studies was the exploration of connections between nightmares and schizophrenia. Hartmann and colleagues (Hartmann, 1984; Hartmann et al., 1987; Hartmann & Russ, 1979; Hartmann, Russ, et al., 1981) stressed a possible relation between frequent nightmares and psychosis. They proposed a biologically based theory that linked the enhanced vividness and dreamlike quality of REM mentation, particularly nightmares, to increased brain levels of dopamine, a neurotransmitter often implicated in schizophrenia. Based in large part on his clinical observations of hundreds of frequent nightmare sufferers solicited through advertisements or undergoing therapy, Hartmann (1984) noted that these individuals demonstrated an unusual degree of openness and vulnerability which he characterized as "thin or permeable boundaries of the mind" (p. 136). According to Hartmann, thin boundaries hinder an individual's ability to distinguish clearly between different intrapsychic states. Hartmann's nightmare theory thus had clear similarities to traditional psychodynamic models of ego boundary impairment in the etiology of schizophrenia (Blatt & Wild, 1976; Bleuler, 1950; Federn, 1953). In a series of empirical investigations, Hartmann (1984, 1989, 1991) found that nightmares were associated with greater boundary disturbances, both on a self-report measure he developed (Boundary Questionnaire; Hartmann, 1991) as well as on the Rorschach (Cooper & Hartmann, 1986; Hartmann, 1984). Corroborating evidence for boundary disturbances in individuals with nightmares was independently reported utilizing the Rorschach (Levin, 1990a) and the self-report boundary measure (Cowen & Levin, 1995; Levin, 1990a; Levin & Raulin, 1991; Pietrowsky & Köthe, 2003; Schredl, Schafer, Hofmann, & Jacob, 1999; see *Thin boundaries* section below for further discussion).

Additional evidence for a link between nightmares and schizophrenia-spectrum disorders was found in a number of clinical investigations. First, nightmare sufferers were found to have

substantially elevated psychotic scales on the Minnesota Multiphasic Personality Inventory (Scales 7–8) as compared with both controls with few nightmares and other clinical populations such as insomniacs (Hartmann, 1984; Hartmann et al., 1987; Hartmann & Russ, 1979; Hartmann, Russ, et al., 1981; Kales et al., 1980). However, these findings have not been uniformly consistent (Berquier & Ashton, 1992). Second, in three independent investigations (Hartmann, Russ, et al., 1981; Kales et al., 1980; Levin, 1998), frequent nightmare individuals were more likely to meet diagnostic criteria for a schizophrenia-spectrum disorder on semi-structured psychiatric interviews than were matched controls. Nightmare sufferers also scored higher than controls on two different self-report measures of psychosis-proneness, the Perceptual Aberration-Magical Ideation scale (Chapman, Chapman, & Raulin, 1976; Levin, 1998; Levin & Raulin, 1991) and the STA (Claridge et al., 1997). Hartmann (1984; Hartmann & Russ, 1979) also reported a greater incidence of mental illness and schizophrenia-spectrum psychopathology in the family members of nightmare sufferers as compared with controls. Third, Levin (1998) found that frequent nightmare participants produced rapid electrodermal habituation patterns to auditory stimuli that were similar to patterns documented for various schizophrenia-spectrum disorders (Dawson, Nuechterlein, Schell, Gitlin, & Ventura, 1994; Ohman et al., 1989). Finally, Watson (2001) reported a strong association between schizotypal and dissociative symptoms on the one hand and frequent nightmares and anomalous sleep experiences on the other, concluding that "dissociation, schizotypy and sleep-related experiences define a common domain that is characterized by unusual cognitions and perceptions" (p. 531) and endorsed Hartmann's thin boundary construct as an explanatory mechanism linking these experiences.

Taken together, these findings suggest that frequent nightmares may be associated with schizophrenia-related psychopathology although it remains unclear whether the relationship is causal or merely correlational. More important, the specificity of these findings was recently questioned by the demonstration that nightmare frequency predicts several other subtypes of psychopathology in addition to schizotypy (Levin & Fireman, 2002a). In the latter study, participants prospectively reporting frequent nightmares (at least 1/week) on home logs scored significantly higher than participants reporting infrequent nightmares on measures of somatization, depression, anxiety, hostility, and dissociation in addition to the schizotypy measures of psychoticism and perceptual aberration/magical ideation. Thus, previous work implicating a specific nightmare–schizotypy relationship may have been hindered by a failure to use other psychopathology variables in addition to schizophrenia-spectrum measures.

Nightmares Are Associated With Other Psychiatric Disorders

Nightmares have also been shown to predict an increased incidence of psychiatric disorders in three large community-based studies. In a second sample of over 3,700 twin pairs, individuals who reported weekly nightmares demonstrated a fivefold elevation in diagnosable psychiatric disorders relative to those with infrequent nightmares (15.7% vs. 3.1%; Hublin et al., 1999b). In a third sample, a community-based survey of over 36,000 respondents,

frequent nightmares were associated with increased psychopathology and death by suicide (Tanskanen et al., 2001). Adjusted hazard ratios indicated a 57% higher suicide rate in individuals reporting occasional nightmares and a 105% higher rate in individuals reporting frequent nightmares compared with those reporting an absence of nightmares. A dose–response relationship between nightmare frequency and suicide risk was also observed.

The latter findings are consistent with results from earlier studies indicating an increased risk of suicide in depressed patients with frequent nightmares (Agargun et al., 1998; Feldman & Hersen, 1967) and with results from a recent study of 176 psychiatric outpatients who exhibited an association between suicidal ideation and nightmares; this was particularly true for female patients (Bernert et al., 2005).

Other studies using vastly different recruitment procedures and samples have also consistently demonstrated higher rates of previous psychiatric treatment in patients with frequent nightmares compared with control participants. Almost 75% of the Hartmann, Russ, et al. (1981) sample of frequent nightmares patients had a history of prior psychiatric care with 10% reporting at least one psychiatric hospitalization. Similarly, 10% of Levin's (1990a) frequent nightmare participants (> 1/week) reported a history of psychiatric hospitalization compared with none for the controls. Kramer et al. (1984a) found that 38% of a sample of frequent nightmare sufferers reported a past psychiatric hospitalization, but no control participants did. Finally, 50% of Berquier and Ashton's (1992) frequent nightmare sample, but none of their control sample, reported a history of psychiatric care. The sources of this variability in prevalence of comorbid psychiatric conditions are unknown, although it is likely that they are due, in part, to differences in the age, gender, and socioeconomic conditions of the participant samples.

Nightmares have also been reported in conjunction with several other indices of psychopathology, thus further bolstering the notion that both phenomena are mediated by a general affect distress factor. As stated earlier, most psychiatric conditions comorbid with nightmares implicate elevated levels of affect distress: borderline personality disorder (Agargun, Kara, Ozer, Selvi, Kiran, & Ozer, 2003; Claridge, Davis, Bellhouse, & Kaptein, 1998), dissociative symptomatology as measured by the Dissociative Experiences Survey (Bernstein & Putnam, 1986; see Agargun, Kara, Ozer, Selvi, Kiran, & Kiran, 2003; Agargun, Kara, Ozer, Selvi, Kiran, & Ozer, 2003; Levin & Fireman, 2002a; Nielsen, 2005; Watson, 2001) and self-reported histories of childhood traumatic events, particularly physical abuse (Agargun, Kara, Ozer, Selvi, Kiran, & Kiran, 2003; Agargun, Kara, Ozer, Selvi, Kiran, & Ozer, 2003).

In sum, frequent nightmares are associated with increased reporting of psychopathological symptoms, in particular, poor psychological well being, high psychopathology scores, heightened suicide risk, and prior psychiatric diagnosis or treatment. Nonetheless, in the absence of controlled longitudinal studies, causal relationships between nightmares and psychopathology have not been established. It is also not yet clear whether nightmares are a generalized indicator of poor overall psychological health. However, the association of nightmares with such a wide range of psychiatric problems is consistent with our suggestion that they reflect a general affect distress factor rather than being associated with any specific psychopathology subtypes. Future research on

this question would be bolstered by the use of behavioral measures and structured diagnostic clinical interviews rather than the self-reporting of symptoms that characterizes most of the existing research.

Nightmares Are Comorbid With Health Behavior Problems and Other Sleep Disturbances

Health-related behavior problems involving elevated affect distress are also associated with increased nightmare frequency and distress. In two investigations (Madrid, Marquez, Nguyen, & Hicks, 1999; Nguyen, 2003), nightmare frequency and distress were associated with such stress-related health problems as allergies, circulatory and gastric problems, pain, and Type A behavior. Nightmares have also been reported in association with morning headaches (Levitan, 1984; Thoman, 1997), although no causal mechanisms for this connection have been suggested. Rates of alcohol and tobacco use are higher (about 50%) among nightmare sufferers than among controls (Berquier & Ashton, 1992), whereas nightmares and disturbed dreams are associated with cigarette smoking only for men (Wetter & Young, 1994). Tanskanen et al. (2001) also found frequent nightmares to be associated with poorer health behaviors (increased use of alcohol and psychotropic medications, higher rates of heavy smoking) and symptoms of depressed mood, life stress, and insomnia. In related findings, several investigations have revealed a frequent occurrence of nightmares among recovering substance abusers during periods of withdrawal (Cernovsky, 1985, 1986; Hershon, 1977). The fact that increased substance usage is a frequent coping strategy for emotion regulation among traumatized individuals further underscores our suggestion that many individuals with frequent nightmares have earlier histories of trauma exposure.

Individuals with frequent nightmares also commonly report greater disruptions of normal sleep patterns than do controls; symptoms include higher rates of sleep-onset and sleep-maintenance insomnia, more frequent awakenings from sleep, and poorer overall sleep quality (Dunn & Barrett, 1988; Haynes & Mooney, 1975; Hersen, 1971; Kales et al., 1980; Krakow, Tandberg, Scriggins, & Barey, 1995; Levin, 1994). High rates of sleep-disordered breathing (Krakow, Melendrez, et al., 2001) and periodic limb movements (Germain & Nielsen, 2003b) have also been reported in individuals with frequent posttraumatic nightmares.

In sum, our notion that a general affect distress factor is central to nightmare production is further supported by studies revealing that a broad range of health behavior problems and sleep symptoms are associated with nightmares. Together with similar work revealing that nightmares are comorbid with anxiety disorders, neuroses, schizophrenia spectrum disorders, and a variety of other psychiatric ailments, the most reasonable conclusion is that all such conditions are marked by a highly negative and reactive emotional response style, a style that also manifests reliably as heightened nightmare distress. Further studies are therefore needed to isolate the nightmare distress component and assess its relationship to psychopathological symptoms independent of other nightmare measures, such as frequency. Such studies, if carefully designed, might succeed in shedding light on possible causal relationships between nightmares and the etiology of other psychopathological symptoms. A possible causal role for nightmares in the etiology of one psychiatric disorder, PTSD, is discussed in the following section.

Nightmares Are Associated With PTSD

Of all psychiatric and health problems, nightmares are most closely associated with PTSD (Harvey et al., 2003; Mellman, Bustamante, Fins, Pigeon, & Nolan, 2002; Pillar, Malhotra, & Lavie, 2000). Nightmares and an accompanying REM sleep dysfunction have been described as a "hallmark symptom" of PTSD (Ross et al., 1989, p. 697), and recent studies have documented frequent sleep disturbances and diminished sleep quality among PTSD patients (Famularo, Kinscherff, & Fenton, 1990; Foa, Riggs, & Gershuny, 1995; Hagstrom, 1995; Krakow, Melendrez, et al., 2001; Kramer & Kinney, 1988; Mellman et al., 2002; Mellman, David, Bustamante, Torres, & Fins, 2001; Mellman, Kulick-Bell, Ashlock, & Nolan, 1995; Mellman, Kumar, Kulick-Bell, Kumar, & Nolan, 1995; Neylan et al., 1998, 2001; Nishith, Resick, & Mueser, 2001; Ohayon & Shapiro, 2000; Ross et al., 1994b, 1989; van der Kolk et al., 1984; Woodward et al., 2000). These disturbances take two predominant forms—intrusive replicative nightmares that reenact parts of the trauma and difficulties initiating or maintaining sleep—and correspond, respectively, to the reexperiencing and hyperarousal symptom clusters defining PTSD (American Psychiatric Association, 2000). The prevalence of nightmares after trauma exposure is extremely high, particularly in the acute phase, with rates as high as 90% of affected individuals (Kilpatrick et al., 1998; Krakow et al., 2002; Neylan et al., 1998; Ross et al., 1989; Woodward et al., 2000), and the frequency of nightmares in PTSD may be as high as 6 nights per week (Krakow et al., 2002). These symptoms may continue unabated for the duration of an afflicted individual's life; at least two studies indicated that frequent, recurrent, trauma-related nightmares still occur 40–50 years after the trauma (Guerrero & Crocq, 1994; Kaup, Ruskin, & Nyman, 1994). This inseparable association between nightmares and PTSD is one of the clearest illustrations of our suggestion that affect distress mediates nightmare production.

Consistent with other studies demonstrating nightmares to be a risk factor for subsequent psychiatric problems (Hublin et al., 1999b; Nielsen et al., 2000; Ohayon et al., 1997), the occurrence of nightmares prior to trauma exposure also predicts the severity of PTSD and other posttraumatic psychiatric symptoms (Mellman, David, et al., 1995). This suggests that nightmares may reflect an occult risk factor for subsequent psychopathology. To illustrate, preexistent sleep disturbances and nightmare complaints were associated with the presence of PTSD in a large epidemiological survey of Toronto residents (Ohayon & Shapiro, 2000). Sleep disturbances in this cohort existed prior to PTSD in great proportions: insomnia (60.9%), excessive daytime sleepiness (71.4%), and parasomnia symptoms including nightmares (40%).

Nightmares and associated sleep disturbances appearing immediately posttrauma are also reliable predictors of subsequent PTSD and comorbid psychopathology. Participants who had distressing dreams immediately after a life-threatening event had more severe PTSD symptoms later than those who did not and had higher levels of acute and chronic PTSD than traumatized individuals without nightmares (Mellman et al., 2001; Mellman, Kulick-Bell, et al., 1995; Mellman & Pigeon, 2005). Similarly, sleep symptoms fre-

quently comorbid with nightmares, such as insomnia and excessive daytime sleepiness, measured as early as 1 month posttrauma robustly predict subsequent PTSD symptoms (Koren, Arnon, Lavie, & Klein, 2002). It may be that frequent posttraumatic nightmares and related sleep symptoms are variable expressions of abnormal REM mechanisms that are causal in the development and maintenance of PTSD (Mellman et al., 2002; Mellman, Kulick-Bell, et al., 1995; Ross et al., 1989, 1994a).

With such strong evidence that nightmares are a distinguishing symptom of PTSD and that they predict the subsequent appearance of PTSD and other psychiatric symptoms, it might be expected that nightmare distress, as part of a more general affect distress style, will play an important and easily detectable role in initiating and maintaining PTSD. Some evidence supports this possibility. First, PTSD patients rate their nightmares as significantly more distressing than non-PTSD patients rate their nontraumatic nightmares (Germain & Nielsen, 2003b). Second, traumatized individuals experience high levels of negative affect in addition to their nightmares, with estimates that up to 80% of PTSD patients meet diagnostic criteria for anxiety, depressive, and substance use disorders (Brady & Clary, 2003). Third, individuals who have in the past experienced the most distressing trauma (i.e., interpersonal violence) are at greater risk of developing PTSD following a current trauma (Breslau, Chilcoat, Kessler, & Davis, 1999). Finally, given that posttraumatic nightmares often repeat a traumatic situation in whole or in part (Mellman & Pigeon, 2005), the remembering of nightmares almost certainly resensitizes patients on a regular basis to their prior trauma and may even induce retraumatization. The latter possibility remains to be studied empirically.

We further propose that the hyperarousal symptom cluster of PTSD is an expression of the distress response style suggested by these studies and, thus, an important causal element of PTSD etiology. Consistent with this, cross-lagged panel analyses reveal that hyperarousal strongly influences, but is not generally influenced by, the other symptom clusters, and trajectory analyses demonstrate that respondents for whom hyperarousal is the most pronounced posttraumatic symptom show lower overall symptom improvement than do control participants with less prominent hyperarousal (Schell, Marshall, & Jaycox, 2004). Further, hyperarousal reactions may involve greater defensive reactivity and reduced visual perceptual engagement (Miller & Litz, 2004). Thus, it is also possible that hyperarousal indirectly mediates nightmare production by increasing an individual's sensitivity to threat cues in the waking state.

Consideration of this hyperarousal evidence may help explain the seemingly anomalous finding that patients who adjust well to their PTSD condition over time recall fewer, briefer and less salient dreams than do nontraumatized participants (Kaminer & Lavie, 1991; Kramer et al., 1984b; Pillar et al., 2000). Reduction in dream recall may reflect inhibitory processes that reduce dreaming intensity in order to also suppress nightmares (Kaminer & Lavie, 1991). The mechanism of such inhibition remains unknown, but it has been suggested (Nielsen, 2005) that the hyperarousal symptoms of PTSD lead eventually to an emotional numbing (equivalent to elevated alexithymia; cf. Badura, 2003) that is highly comorbid with PTSD and that may reduce the intensity of dream emotions. Emotional numbing is, in fact, best predicted by the number of hyperarousal symptoms in PTSD patients (Weems,

Saltzman, Reiss, & Carrion, 2003), suggesting that PTSD patients may expend so much cognitive, behavioral, and emotional effort managing hyperarousal and reactivity that they exhaust or deplete their emotional and neurochemical resources, such as catecholamines (Litz et al., 1997).

Taken together, the previous results further support the notion that demonstrated relationships between frequent nightmares and psychopathology are mediated by the factor of nightmare distress. As it is currently measured, *nightmare distress* refers to an individual's reactions to their nightmares while awake. Individuals highly disturbed by their nightmares may therefore suffer from a more generalized condition of waking-state distress linked especially to anxiety and depression reactions. They may also disproportionately attend to and selectively recall mood-congruent cognitions both during waking (Bowers, 1981; Clark & Teasdale, 1982) and during sleep. For this reason, we propose in our nightmare formation model (see the next section) that nightmare distress is one of two general factors. It increases an individual's affective response after awakening from the nightmare but also to some extent during the nightmare's formation. It is conceptualized as a trait factor in contradistinction to affect load, which we view as a state factor.
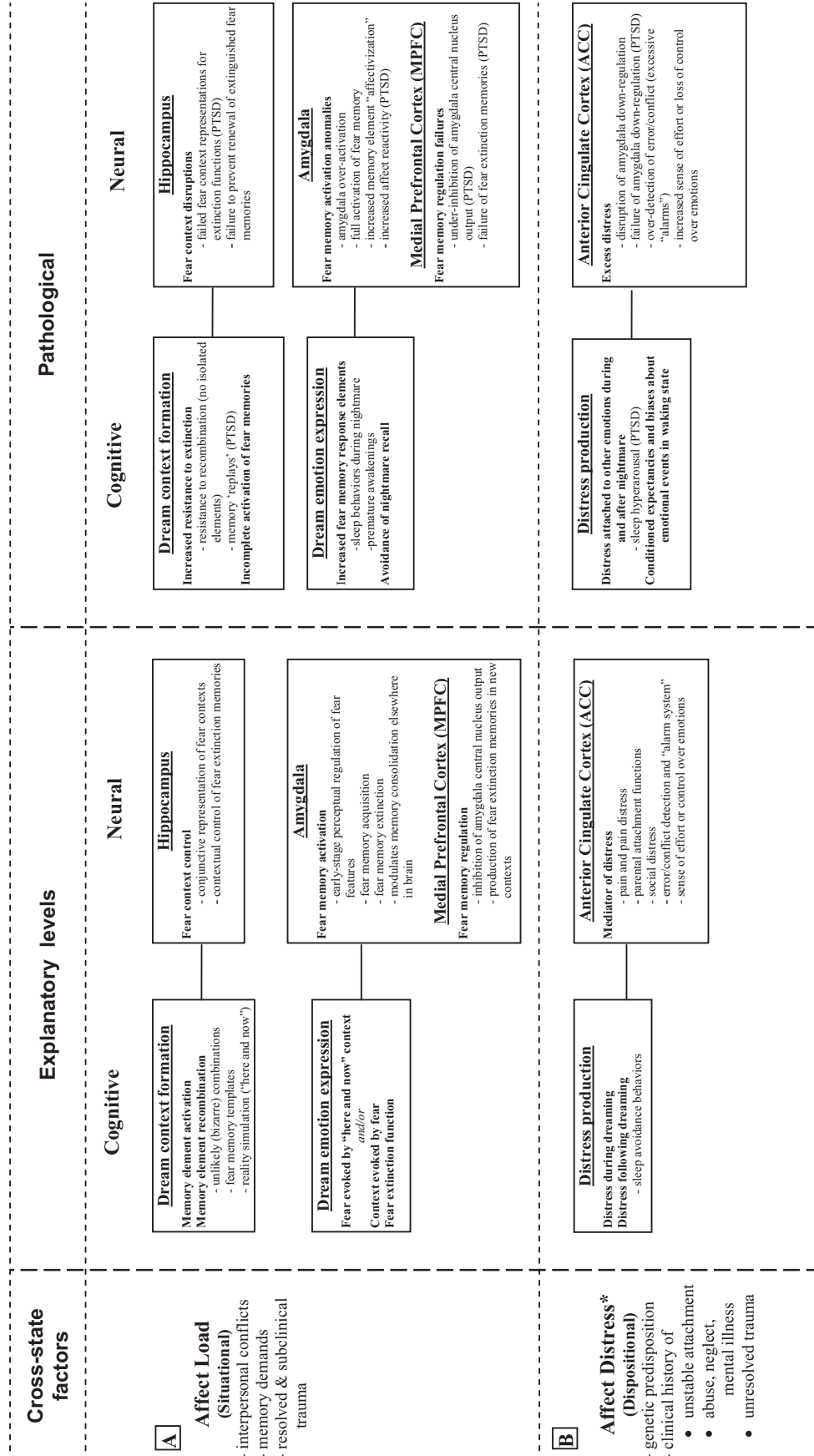
Nightmare Formation: A Neurocognitive Approach

As the previous sections together indicate, a new dual-process conception of nightmares that distinguishes nightmare frequency from nightmare distress promises to parsimoniously explain several established findings about nightmares, including variations in estimates of their prevalence and frequency, robust gender and age differences, and their comorbidity with a wide spectrum of psychopathological symptoms—PTSD in particular. In the present section, we further elaborate this conception in the context of a cross-state, multilevel working model of dreaming and nightmare production (see Figure 3). The objective is to relate the general dual-process framework to more specific cognitive and neural systems that may be critical to the production of dreams and, by disruption of these systems, of nightmares.

Global Assumptions of the Model

The nightmare model is predicated on two global assumptions: *cross-state continuity* and *multilevel explanation*.

Cross-state continuity. The first assumption refers to the possibility that some structures and processes implicated in nightmare production are also engaged during the expression of pathological signs and symptoms during the waking state. The assumption derives from a considerable body of evidence that dreaming mentation is continuous with waking thought (Cartwright & Ratzel, 1972; Cipolli, Baroncini, Fagioli, Fumai, & Salzarulo, 1987; Foulkes, 1985; Foulkes & Fleisher, 1975; Kahan, LaBerge, Levitan, & Zimbardo, 1997; Kuiken, 1995; Levin & Young, 2001–2002; Purcell, Moffitt, & Hoffmann, 1993; Saredi, Baylor, Meier, & Strauch, 1997). Accordingly, some pathogenic mechanisms implicated in nightmare formation should also be identifiable in waking functioning and thus amenable to experimental manipulation with standard waking-state methods. A review of the evidence leads us to propose that the cross-state continuity assumption is

Cross-state factors

Explanatory levels

Pathological

Neural

Hippocampus

Fear context disruptions
- failed fear context representations for extinction functions (PTSD)
- failure to prevent renewal of extinguished fear memories

Amygdala

Fear memory activation anomalies
- amygdala over-activation
- full activation of fear memory
- increased memory element "affectivization"
- increased affect reactivity (PTSD)

Medial Prefrontal Cortex (MPFC)

Fear memory regulation failures
- under-inhibition of amygdala central nucleus output (PTSD)
- failure of fear extinction memories (PTSD)

Anterior Cingulate Cortex (ACC)

Excess distress
- disruption of amygdala down-regulation
- failure of amygdala down-regulation (PTSD)
- over-detection of error/conflict (excessive "alarms")
- increased sense of effort or loss of control over emotions

Cognitive

Dream context formation

Increased resistance to extinction
- resistance to recombination (no isolated elements)
- memory 'replays' (PTSD)
Incomplete activation of fear memories

Dream emotion expression

Increased fear memory response elements
- sleep behaviors during nightmare
- premature awakenings
Avoidance of nightmare recall

Distress production

Distress attached to other emotions during and after nightmare
- sleep hyperarousal (PTSD)
Conditioned expectancies and biases about emotional events in waking state

Neural

Hippocampus

Fear context control
- conjunctive representation of fear contexts
- contextual control of fear extinction memories

Amygdala

Fear memory activation
- early-stage perceptual regulation of fear features
- fear memory acquisition
- fear memory extinction
- modulates memory consolidation elsewhere in brain

Medial Prefrontal Cortex (MPFC)

Fear memory regulation
- inhibition of amygdala central nucleus output
- production of fear extinction memories in new contexts

Anterior Cingulate Cortex (ACC)

Mediator of distress
- pain and pain distress
- parental attachment functions
- social distress
- error/conflict detection and "alarm system"
- sense of effort or control over emotions

Cognitive

Dream context formation

Memory element activation
Memory element recombination
- unlikely (bizarre) combinations
- fear memory templates
- reality simulation ("here and now")

Dream emotion expression

Fear evoked by "here and now" context
and/or
Context evoked by fear
Fear extinction function

Distress production

Distress during dreaming
Distress following dreaming
- sleep avoidance behaviors

A

Affect Load
(Situational)
- interpersonal conflicts
- memory demands
- resolved & subclinical trauma

B

Affect Distress*
(Dispositional)
- genetic predisposition
- clinical history of
  - unstable attachment
  - abuse, neglect, mental illness
  - unresolved trauma

especially likely to hold true for the affect load and affect distress constructs.

Multilevel explanation. The second assumption of the model is that, ultimately, nightmares will be most satisfactorily explained by a combination of hypothetical concepts derived from two different levels of scientific discourse: (a) a cognitive–emotional level composed of a set of imagery processes responsible for the representation of emotional dream imagery—fear imagery in particular—and (b) a neural level consisting of a network of brain regions underlying these imagistic and emotion processes. Although there may exist isomorphic relationships between these levels of discourse, current knowledge does not yet allow us to describe them (for a discussion of the isomorphism problem, see Nielsen, 2000). Some key points of convergence will nevertheless be suggested.

In casting such a wide net for theoretical constructs, we acknowledge that our nightmare model does not yet constitute a satisfactory explanation of nightmare pathology. Rather, at the current state of knowledge, we feel that its strength is as a heuristic tool, a means of synthesizing rapidly emerging empirical findings and theoretical constructs from several fields toward the crafting of testable hypotheses about nightmare formation.

Evidence for Cross-State Continuity

Affect load. We conceptualize affect load, a state factor, as the combined influence of stressful and emotionally negative events on an individual's capacity to effectively regulate emotions. Taxing negative events are not equivalent to trauma in that they are not life threatening (American Psychiatric Association, 2000), although this distinction can be difficult to make, especially in the case of children. We propose that increases in stress or negative emotions constitute an increase in affect load that can influence the individual in both waking and sleeping states. Thus, in susceptible individuals, it can bring about more frequent nontraumatic nightmares. Two types of evidence are consistent with this view.

First, the reporting of more frequent nontraumatic nightmares is associated with increased life stress (Barrett, 1996; Berquier & Ashton, 1992; Cernovsky, 1990; Dunn & Barrett, 1988; Hartmann et al., 1987; Hartmann, Falke, et al., 1981; Husni, Cernovsky, Koye, & Haggarty, 2001; Kales et al., 1980; Kramer et al., 1984b). Dysphoric dreaming is also associated with a variety of specific stressors, such as anticipated surgery (Breger et al., 1971), experimental pain stimulation (Nielsen, McGregor, Zadra, Ilnicki, & Ouellet, 1993), menstruation (Bucci, Creelman, & Severino, 1991), pregnancy (Maybruck, 1989), miscarriage (Van, Cage, & Shannon, 2004), new motherhood (Nielsen & Paquette, 2004), preparing for exams (Duke & Davidson, 2002), taking sham intelligence tests (Koulack, Prevost, & de-Koninck, 1985; Stewart & Koulack, 1993), stock market downturns (Kroth, Thompson, Jackson, Pascali, & Ferreira, 2002), and watching disturbing movies (De Koninck & Koulack, 1975; Greenberg, Pillard, & Pearlman, 1972; Powell, Nielsen, Cheung, & Cervenka, 1995). It is likely that an individual must react subjectively to such seemingly stressful events in order for dream content to be affected; for example, students undergoing exams do not report dysphoric dreams when their self-reported stress levels remain unchanged (Delorme, Lortie-Lussier, & De Koninck, 2002).

Some studies report negative findings for such relationships, for example, between nightmares and poor social adjustment (Cernovsky, 1984a, 1984b, 1984c), Life Events Inventory scores (Paykel & Uhlenluth, 1972; see Zadra & Donderi, 2000) or the number of life events over the past year (Köthe & Pietrowsky, 2001). Nonetheless, the fact that these findings were all obtained from retrospective reports of stressful life events, nightmares, or both suggests a possible basis for these discrepant findings.

Figure 3 (opposite). Cross-state, multilevel model of dreaming and nightmare formation. Normal dreaming is hypothesized to be produced by interactions between, on the one hand, cross-state factors affect load (A) and affect distress (B) and, on the other, emotional imagery processes as explained at both cognitive and neural levels. Nightmares are influenced additionally by pathological mechanisms that may affect these normal dreaming processes. **Cognitive level:** Normal dream processes unfold in two broad, interactive steps—creation of dream context via memory element activation and recombination, and expression of dreamed emotions appropriate to the context. Memory element recombination results in new contexts that facilitate fear extinction but also in the occurrence of bizarre or unusual contents. Fear memories act as templates organizing information about stimulus, response, and meaning components of fear experiences. Dream context is rendered hallucinatory ("reality simulation"), which maximizes involvement of emotional processes associated with the amygdala. Nontraumatic nightmares occur when affect load is elevated by daytime emotional concerns and memory demands, leading to resistance of the fear extinction process, a tendency to reproduce complete fear memories and an excess of response elements during dreaming. Nightmare distress is produced as a result of genetic disposition or prior factors, such as abuse, neglect, or trauma. Distress may be mediated by hyperarousal during sleep and may lead to pathological consequences during and after nightmares (nightmare distress) or during the daytime (e.g., conditioned emotional expectations). **Neural level:** Cognitive-level processes and pathology are paralleled by systems-level neural events. Dream context formation is attributed to the fear context control function described for the hippocampus, whereas dream emotion expression is linked to fear memory activation functions of the amygdala and of the MPFC, which gates the output of the amygdala and is implicated in the production of fear extinction memories. Affect distress is mediated by the ACC, which is linked to a number of distress-related functions: pain, parental attachment, social interactions, error/conflict detection, and a sense of emotional control. Pathological mechanisms may affect any of the proposed neural systems and may include failure to manage fear contexts (hippocampus), anomalies of fear activation (amygdala) and regulation (MPFC) and excess distress responses (ACC). PTSD = posttraumatic stress disorder.

Second, cross-state continuity in affect load is suggested by the observation that stressful life events often precipitate the onset of nightmare problems. Feldman and Hersen (1967) first reported that nightmare frequency in young adulthood was associated with a personal experience of the death of someone close or perceived interpersonal stress, particularly in the domains of work and family life. High interpersonal conflict was also anecdotally cited by Hartmann et al. (1987) as the greatest precursor of adult participants' nightmares. Sixty percent of both Hartmann & Russ (1979) and Kales et al.'s (1980) samples and most of Dunn and Barrett's (1988) participants identified a major life event preceding the onset of their nightmares. Prospective evidence comes from a study (Wood et al., 1992) demonstrating that, immediately after the 1989 San Francisco earthquake, (a) nightmares were twice as common in two San Francisco Bay area student samples than in an Arizona comparison group, even though baseline frequencies of nightmares did not differ; (b) nightmare frequency was related in a dose–response manner to proximity to the earthquake epicenter; and (c) nightmares of those close to the earthquake more often concerned the topic of earthquakes (40%) than nightmares of those more distant from it (5%). The additional fact that nightmare frequency decreased considerably over time suggests that, for most participants, this event did not evoke PTSD but rather a short-term increase in affect load that was processed relatively quickly.

Affect distress. We conceptualize affect distress, a trait factor, as a long-standing disposition to experience heightened distress and negative affect and to react with extreme behavioral expressions. Consistent with recent work emphasizing diathesis-stress modeling in experimental psychopathology (e.g., Abramson, Metalsky, & Alloy, 1989; Chorpita & Barlow, 1998; Mineka & Zimbarg, 2006), we suggest that an affect distress style develops over time as a form of emotional response, originating in the infant's instinctive ability to signal caregivers by expressing distress and its need for food, social contact, and relief from pain. Over time, the expression of distress is progressively regulated and replaced by the normal panoply of emotions. However, to the extent that the infant, child, or young adult has a family history of mental illness or is subjected to early object loss, insecure attachment, neglect, abuse or trauma and perceives himself or herself to be losing emotional control, distress will be felt and expressed more intensely and more frequently. It will come to play an increasingly dominant role in his or her personality and mental health. Evidence does, in fact, indicate that high affect distress is related to early development of a chronic perception of uncontrollability over adverse life events (e.g., early object loss, inconsistent parenting, insecure attachment); this perception acts as a risk factor or cognitive diathesis for emotional dysregulation in the face of subsequent stress (Abramson et al., 1989; Chorpita & Barlow, 1998).

Our cross-state continuity assumption suggests that this trait distress factor may influence both waking and sleeping processes. For the waking state, we propose that affect distress is akin to both the negative affect and negative emotions personality dimensions (Chorpita & Barlow, 1998; Watson & Pennebaker, 1989) and that it is expressed over a wide variety of psychopathological conditions (anxiety disorders, health behaviors, PTSD, etc). In the case of nightmares, affect distress appears in the intense emotional reactions expressed after awakening from a nightmare but also to some extent in the extreme emotions that are expressed within the nightmare itself. Surprisingly few studies have examined the un-

derlying cognitive, affective, and imagistic dimensions of an affect distress personality style in relation to elevated nightmare production and reporting. However, an emerging body of evidence is linking nightmare distress to various constructs associated with waking affect distress, such as increased physiological and psychological reactivity (Kramer et al., 1984b; Levin, 1994), imagery vividness (Levin & Fireman, 2002a), maladaptive coping (Köthe & Pietrowsky, 2001; Levin & Fireman, 2001), and thin boundaries (Hartmann, 1984; Hartmann, Elkin, & Garg, 1991; Levin et al., 1991). We briefly review these literatures to demonstrate the wide swath of findings that converge to support the cross-state continuity assumption as applied to affect distress.

Physiological and psychological reactivity. Individuals high on the negative affect dimension demonstrate heightened internal focus and increased somatic amplification, including a tendency to report somatic symptoms in the absence of objective health problems (Watson & Pennebaker, 1989). In agreement with the cross-state consistency assumption, a number of authors (Hartmann, 1984; Kales et al., 1980; Kramer et al., 1984b; Levin, 1994) have noted that nightmare individuals are more emotionally reactive to their internal states than are nondisturbed dreamers on the basis, at least in part, of evidence of increased physiological arousal (C. Fisher et al., 1970, 1974). Frequent nightmare sufferers are also more affected by both their nightmares and other dreams the next day (Levin, 1994). In one study, 89% of an undergraduate sample reported that their nightmares dramatically affected them the next day, and 26% reported that this effect was lasting (Dunn & Barrett, 1988). Levin & Fireman (2002a) found no such differences between individuals high and low in nightmare frequency when ratings of nightmare intensity and vividness were completed the morning after a nightmare but did find them when individuals were asked to respond globally to the same variables ("In general, how vivid are your nightmares?"). Similar findings were previously reported (Taub, Kramer, Arand, & Jacobs, 1978).

Imagery vividness and fantasy proneness. Evidence for cross-state continuity of affect distress is also found in studies of imagery processes. Affect distress may directly influence imagery processes such as vividness, absorption, or the propensity to engage in fantasy. There is evidence, for example, that imagery vividness is associated with increased fear activation (Lang, Greenwald, Bradley, & Hamm, 1993), that elevated absorption in imagery is associated with increased affective responsiveness, heightened memory clarity for negative events (Destun & Kuiper, 1999; Roche & McConkey, 1990; Wild, Kuiken, & Schopflocher, 1995), and threat-related source-monitoring deficits (Johnson, Hashtroudi, & Lindsay, 1993; Johnson, Kahan, & Raye, 1984; Johnson & Raye, 1981) and that fantasy proneness is associated with poor psychological functioning (Rauschenberger & Lynn, 1995; Waldo & Merritt, 2000).

Relations with nightmares are also clear. Studies have reported that frequent nightmares are associated with fantasy proneness, psychological absorption, and imaginative involvement (Starker, 1974, 1984; Starker & Hasenfeld, 1976). Hypnotic susceptibility, vividness of visual imagery and absorption are also associated with nightmare distress but not with nightmare frequency (K. Belicki, 1992b; K. Belicki & Belicki, 1986). Recently, high levels of fantasy proneness, psychological absorption, and dysphoric imaginative involvement were found to be associated with prospective measures of nightmare frequency and distress, but in an additive

fashion (Levin & Fireman, 2001–2002, 2002a). Further, 13 of the 46 (28%) individuals classified as high in nightmares (one or more per week) met Lynn and Rhue's (1986) criteria for a high fantasy-prone individual (cutoff > 37 on the Inventory of Childhood Memories and Imaginings; S. Wilson & Barber, 1981), a rate far exceeding the population base rate of 4%.

Maladaptive coping. Affect distress may have deleterious effects on an individual's habitual coping style. The ruminative, emotion-focused coping style identified, for example, in depressed individuals (Nolen-Hoeksema & Girgus, 1994) may have analogous expressions in the sleep state. In fact, Levin and Fireman (2001–2002) found dysphoric daydreaming, a variable indicating propensity for uncontrollable and obsessive worrying (Huba, Aneshensel, & Singer, 1981), to be the best predictor of both nightmare frequency and distress. Similarly, Nielsen et al. (2000) found nightmares to be strongly and selectively associated with the obsessive-compulsive scale of the SCL-90. In addition, Pietrowsky and Köthe (2003) reported that half their participants indicated a preoccupation with their nightmares, including compulsive intrusive imagery, the following day.

Some researchers (K. Belicki, 1992a; Wood & Bootzin, 1990) have suggested that dysfunctional coping may mediate nightmare distress. Wood and Bootzin (1990) suggested that individuals suffering nightmare distress are anxious individuals to begin with and thus have greater access to memories of anxiety-related events like nightmares. K. Belicki (1992a) reasoned that distress is related to the evaluative process that individuals make about their nightmares upon awakening and likened the heightened emotional reactivity typical of high nightmare distress individuals to their subsequent difficulty distracting themselves from the memories of such experiences. Although not directly addressing nightmares, a number of studies have demonstrated that maladaptive dispositional coping (namely reduced social support and increased avoidance) in nontraumatized populations is associated with increased distress, which in turn predicts sleep complaints (Paulsen & Shaver, 1991; Shaver et al., 1997; Urponen, Vuori, Hasan, & Partinen, 1988). These findings were most recently confirmed by Germain, Buysse, Ombao, Kupfer, and Hall (2003) who found that the effects of stress exposure on REM architecture were influenced directly by situational factors and indirectly by dispositional factors (coping style and neuroticism).

Thin boundaries. Hartmann and colleagues (Hartmann, 1984, 1989; Hartmann et al., 1991, Hartmann, Mitchell, Brune, & Greenwald, 1984; Hartmann, Falke, et al., 1981) proposed a cross-state personality dimension, "boundary permeability," to help explain nightmare pathology. One extreme of this dimension (thin boundaries) characterized waking and sleeping attributes of frequent nightmare individuals and consisted of a striking openness, sensitivity, and vulnerability to cognitive and emotional intrusions. *Thin* individuals are thus highly susceptible to internal events that are not usually perceived by most individuals as threatening or traumatic. They are also purported to have difficulty discerning internal fantasy from external reality, tend to over-identify self with others, and are prone to source monitoring disturbances across various states of consciousness. Enhanced dream recall, dream bizarreness, cognitive fluidity, and nightmare frequency are all positively related to thin boundary scores as measured by both Hartmann's Ego Boundary Questionnaire (Claridge et al., 1997; Cowen & Levin, 1995; Hartmann, Falke, et al., 1981; Kunzendorf,

Hartmann, Cohen, & Cutler, 1997; Levin, 1990b; Levin et al., 1991; Levin, Gilmartin, & Lamontanaro, 1999; Pietrowsky & Köthe, 2003; Schredl et al., 1999) and the Rorschach (Hartmann, 1984; Hartmann et al., 1987; Levin, 1990a).

Summary. The general assumption of cross-state continuity for the concepts of affect load and affect distress is supported by a convergence of findings from the domains of clinical psychopathology and personality. However, a solid foundation in experimental research is still lacking. It is thus opportune that the continuity assumption specifically implies that the structures and processes underlying nightmares can be investigated during the waking state using brain imaging, cognitive testing, and personality assessment techniques that afford higher levels of experimental control than do the more usual approaches to studying nightmare pathology. A primary goal of our model is therefore to identify structures and processes that are apt to demonstrate continuity across waking and dreaming states and thus to be susceptible to study by methodologies from waking-state paradigms that may be applied in new or innovative ways.

Cognitive-Level Explanation: A System of Emotional Imagery Processes

We consider the formation of nightmares to be equivalent to the formation of dreams more generally and their unique qualities to be shaped by the involvement of one or more sets of pathological influences. This section describes three of the most important sets of imagery processes that we propose are common to both dream and nightmare generation and that are particularly central to the expression of affect load: *memory element activation*, *memory element recombination*, and *emotional activation*. The subsequent section will review pathological influences on these processes that we feel underlie the transformation of dreams into nightmares.

Dreaming Requires Activation of Memories and Emotions

Memory Element Activation

The first set of processes refers to the increased availability during dreaming of a wide range of memory elements. Memory elements, rather than memories per se, are emphasized because complete episodic memories do not typically appear during dreaming (Fosse, Fosse, Hobson, & Stickgold, 2003). Rather, dreaming tends to express memory elements as though original memories had been reduced to more basic units (see reviews in Nielsen & Stenstrom, 2005; Schwartz, 2003). Often, these appear as isolated features, such as an attribute of a familiar place or character (e.g., "there was a stranger who had my mother's style of hair"). Less commonly, several elements may appear together by virtue of their origin in a single past event ("my daughter and I were laughing and eating popcorn, like we did last night at the movie") or their grouping by some other form of organization, such as a script ("I was eating in a restaurant somewhere") or a semantic or phonological category ("there were zoo animals everywhere"). The apparent deconstruction of memories into elements tends to camouflage them, but their presence is nonetheless demonstrable by clinical and experimental observation. Clinically, Hartmann (1998a) has demonstrated that dreams often portray elements of a person's main emotional concerns (e.g., stress, trauma) even if

visual or auditory details of a specific memory are absent. Experimentally, memory elements have been detected as the "day-residues" of previous-day experiences (Cipolli, Bolzani, Tuozzi, & Fagioli, 2001; J. A. Davidson & Kelsey, 1987; Hoelscher, Klinger, & Barta, 1981; Marquardt, Bonato, & Hoffmann, 1996; Saredi et al., 1997) and, with more directed reflection, as residues that are temporally delayed by up to a week (Nielsen, Kuiken, Alain, Stenstrom, & Powell, 2004; Nielsen & Powell, 1989, 1992; Nielsen & Stenstrom, 2005; Powell et al., 1995). It remains unknown why normal dreaming disproportionately favors the partial activation of memory elements. One possibility is that it reflects a more general organizing principle of memory. For instance, declarative memories may be stored as multiple traces in which bits and pieces of a single experience are saved by structurally distinct memory systems (Schacter & Tulving, 1994). In the waking state, episodic memories are then reconstituted when needed from the elements stored in these different systems (Nadel & Moscovitch, 1998). In the dreaming state, the elements appear to be reconstituted in an alternative fashion, perhaps randomly (Hobson, 1988), perhaps linked metaphorically (Hartmann, 1998a), or perhaps combined into composite context memories (Johnson, 2005). All of these possibilities may be true to some extent; elements may be activated as a function of emotional concerns (Klinger, 1990) but with the possible introduction of some pseudorandom and incompatible associations. The net effect of such organization, we suggest, is the creation of novel or nonaversive contexts that facilitate fear extinction when affect load is high.

An early conceptualization of fear memory organization referred to as *fear memory structures* (Foa & Kozak, 1986; Lang, 1979) may be of particular relevance to understanding bad dreams, nightmares, and other forms of disturbed dreaming. Fear memory structures were described as networks of information that unite memory elements about (a) a feared stimulus situation (*stimulus elements*), (b) physiological, verbal, and behavioral responses to that situation (*response elements*), and (c) the meaning of these stimuli and responses (*meaning elements*). During waking, fear memory structures were thought to bias the interpretation of new information by enhancing sensitivity and attention to a structure's stimulus elements, thus ensuring the allocation of more cognitive resources to the processing of this new information. Further, activation of these structures was thought to interfere with access to resources necessary for competing tasks, as exemplified by the response deficits shown by PTSD patients on the emotional Stroop task (Foa, Feske, Murdock, Kozak, & McCarthy, 1991; McNally, 1998).

Today, the hypothetical construct of fear memory structures has been validated in some respects by extensive neurological, cognitive, and learning research demonstrating the existence of fear memories. In other respects, they have not. For example, whereas the internal coherence of fear memory structures was believed to be modified or weakened by the introduction of incompatible elements, thus alleviating associated pathological symptoms (Foa & Kozak, 1986), this notion has now been largely replaced by the more neurologically specific concept of extinction memories which inhibit fear memories (Lang, Davis, & Ohman, 2000; Mineka & Zimbarg, 2006; Ohman & Mineka, 2001).

During dreaming, fear memories likely vary in how completely and coherently their stimulus and response elements are expressed. Minimal activation of fear memory elements may trigger a mild anxiety dream in which a limited number of stimulus or response elements are activated, whereas their wholesale activation would result in a more intense, nontraumatic nightmare. In the former case, only some fear memory elements may be expressed in relative isolation and in no coherent order (i.e., as common residues). In the latter case, more elements may be expressed, and their order may be more veridical, rendering the form of the fear memory more easily identifiable from the nightmare's theme. We would thus expect to observe more recurrent dreams and dream themes among individuals with particularly problematic and coherent fear memories, such as persons with specific phobias, ongoing interpersonal difficulties, or other current sources of stress. In fact, recurrent dreams are more frequent among individuals low in subjective well-being (Brown & Donderi, 1986; Duke & Davidson, 2002), and dream content is known to reflect current emotional concerns (Hartmann, 1998a; Hoelscher et al., 1981; Klinger, 1990; Saredi et al., 1997). However, this hypothesis should be tested more specifically by sampling the nightmare content of specific clinical populations.

More rarely, the elements of a fear memory may be activated globally and in a highly coherent order producing a nightmare that appears to reproduce a past fear experience with appropriate fear context, bodily reactions, and cognitive interpretations. This type of comprehensive activation is illustrated by replicative PTSD nightmares that seem to replay large portions of the original trauma (Mellman & Pigeon, 2005). Between the extremes of mild anxiety dreams and the replicative nightmares of PTSD are various types of dysphoric dreams and nightmares that bear fear memory elements either alone or in combination and with varying degrees of organization. Examples include dreams or nightmares with recurring objects, characters and themes (Domhoff, 1996), typical dream themes (Nielsen et al., 2004), story- and script-like structures (Baylor & Deslauriers, 1985; Cipolli, Bolzani, & Tuozzi, 1998; Nielsen, Kuiken, Hoffmann, & Moffitt, 2001), and others.

In line with the cross-state continuity assumption, we suggest that the ensemble of emotional-imagery processes constituting fear memories underlies a variety of clinical conditions characterized by anxiety and fear, such as panic disorder, phobia, and PTSD (Foa & Meadows, 1997; Mineka & Zimbarg, 2006; VanOyen Witvliet, 1997) as well as dysphoric dreams and nightmares.

Memory Element Recombination

The second set of processes, responsible for the continuous assembly of memory elements into a constant flow of dream imagery, is not well understood but was appreciated by both Freud (1900/1976) and Jung (1974) as the mechanism of *condensation*. Condensation is the merging into a single image of the attributes of several separate (though motivationally linked) images. We propose a similar type of reorganization that produces new image contexts during dreaming much like the remapping of conjunctive representations under control of the hippocampus during the waking state. During dreaming, conjunctive representations are rendered into virtual simulations or "here-and-now" illusions (Nielsen & Stenstrom, 2005) to maximize their impact upon the amygdala, which tends to respond to perceptual, rather than imaginal, stimuli (Phan, Wager, Taylor, & Liberzon, 2002). They are recombined or remapped in order to introduce elements that are incompatible with existing fear memories, thus facilitating (among other functions)

the acquisition or maintenance of extinction memories. The latter inhibit fear memories (see the *Neural Level Explanation: A Brain Network for Fear Imagery* section), and consequently alleviate affect load. Recombinations of memory elements give dreams at once their alien and their familiar quality. In the present discussion, we emphasize three features of recombination that we believe are especially pertinent to the pathology of nightmares.

1. Unlikely combinations. The first is the *de novo* conjunctions of features, many of which produce dream experience that seems bizarre, incongruous, or incompatible with waking life experience. Bizarreness is frequent in dreams (Kunzendorf et al., 1997; Levin & Livingston, 1991; Revonsuo & Salmivalli, 1995), and dreams are significantly more bizarre than waking daydreams (Kunzendorf et al., 1997). A widely accepted explanation for bizarreness is still lacking. One possibility with some empirical support is that REM sleep selectively permits weakly associated (and thus possibly disjunctive) memory elements to become associated (Stickgold, Scott, Rittenhouse, & Hobson, 1999). Another is that bizarreness reflects the relative inactivity of dorsolateral prefrontal executive functioning during REM sleep (Hobson, Pace-Schott, & Stickgold, 2000). We propose that the unlikely combinations of disparate memory elements facilitate acquisition and maintenance of fear extinction memories. Bizarreness may be an inevitable consequence of this mechanism and we would expect to see higher levels of bizarreness in dreams of individuals with high affect load or in emotionally transformative dreams such as nightmares. Some research supports this possibility. Among healthy participants, occurrences of bizarreness are positively correlated with major shifts in dream emotion (Merritt, Stickgold, Pace-Schott, Williams, & Hobson, 1994), whereas among bipolar patients, neutral moods are associated with mundane uneventful dreams and manic moods with bizarre and improbable dreams (Beauchemin & Hays, 1995). Notwithstanding such findings, it remains unknown whether nightmares are more or less bizarre than non-nightmare dreams in this specific sense of recombined elements. To the extent that nightmares replay fear memories or possess recurrent elements they would seem to be less, not more, organizationally bizarre. Further empirical investigation of the organizational coherence of nightmares and normal dreams among individuals suffering from frequent nightmares or from conditions marked by high fear coherence in waking states, such as PTSD or specific phobias, could help elucidate these mechanisms. Prospective research tracking temporal relationships between mood, stress, and perceived coping effectiveness that precede and follow nightmares would also be useful in identifying the sources of such mechanisms.

2. Fear memory templates. A second salient feature of recombination is the organizing influence of fear memory and other emotional memory structures. Although they are not usually expressed fully as memory replays, fear memories may nevertheless act as organizing templates that lend an internal consistency to the dream plot and within which other isolated and frequently incompatible memory elements are ordered and interrelated. As a result, fear-producing stimuli and their physiological responses will be repeatedly paired with alternative, nonaversive contexts and thus extinguished gradually over time. Affect load would be decreased accordingly. Although the specific nature of such a mechanism remains speculative, phenomenological features of dream organization belie their presence. On the one hand, fear memories may

assume an habitual, easily recognized form and express a consistent emotional content in the dream, such as with themes of public nudity, being late, or being pursued (Nielsen et al., 2003). Such themes recur frequently and are associated with diminished psychological well-being until they cease—at which time well-being is high (Brown & Donderi, 1986; Zadra & Donderi, 2000). On the other hand, fear memories may portray relatively novel organizations in which a skeletal structure incorporates many unexpected elements, such as an interpersonal attack scenario which introduces many unanticipated characters and produces many unusual consequences. The latter type of dream has been labeled as *progressive* or even *problem solving* and found to be associated with emotional adaptation (Cartwright, 1986; Kramer, 1993). Fear extinction is more likely to be associated with the latter type of dream and less likely to be associated with the former, although again, there is no research that addresses this issue directly. One useful line of investigation would be to compare organizational coherence of the dreams, nightmares and daytime narratives of frequent nightmare individuals who report high or low levels of accompanying distress to determine whether memory organization corresponds to waking emotional reactivity. Also, because fear memories are purportedly responsible for the nonconscious detection of threat, it would also be informative to investigate whether individuals with high nightmare distress perform similar to individuals with anxiety disorders or PTSD on an affective backwards masking paradigm or the emotional color–word Stroop test. Such research would directly test the cross-state continuity assumption of our model as it applies to fear memory processes.

3. Reality simulation. A third important feature of dream imagery recombination is that the new image sequences consist, for the most part, of lifelike simulations of first-person reality. Memory elements are recombined on various levels of organization (e.g., perceptual, schematic, thematic, symbolic) to produce coherent, continuous simulations of waking life experience. This process was recognized as a central dimension of dreaming phenomenology by Freud (1900/1976), who described it as the hallucinatory, dramatic quality of dreaming, and has been reiterated by many subsequent authors as self-participation (e.g., Bosinelli, Cicogna, & Molinari, 1974), virtual reality (Nielsen, Powell, & Cervenka, 1994; Revonsuo, 2000) and the here-and-now illusion (Nielsen & Stenstrom, 2005). The functional value of reconstituting seemingly unrelated memory elements into virtual simulations may be to strengthen new or weak memory links (Stickgold, Hobson, Fosse, & Fosse, 2001) or to simulate threat to species survival and permit offline rehearsal of behavioral avoidance responses (Revonsuo, 2000). We propose that reality mimesis regulates affect load by ensuring that fear memories are processed in a phenomenological medium similar to that in which they were first formed. Realistic stimulus elements ensure the expression of realistic response elements and thus engage the underlying neural and autonomic apparatus in an ecologically appropriate and consistent manner. This allows for the modification or integration of disturbing emotions during dreaming (Cartwright, 2000; Hartmann, 1998a) in a fashion analogous to that induced by exposure therapy for waking-state, fear-based disorders (Foa & Kozak, 1986). The finding that imagery rehearsal is highly effective in reducing recurrent nightmares in individuals with PTSD (Krakow, Hollifield, et al., 2001; Krakow & Zadra, 2006) is consistent with this formulation. However, additional studies are clearly needed.

Virtual reality-based exposure to personalized nightmare scenarios recombined with nightmare-incompatible memory elements might be a useful clinical approach for examining this feature.

Emotional Expression

There is disagreement over whether emotions drive the selection of dream contents (Hartmann, 1998a; Newell & Cartwright, 2000) or whether they arise later in reaction to these contents (Foulkes, 1982). Our view is that both may be true in the progressive interactive expression of fear memories. Because both stimulus and response elements are encoded in a single fear memory, by the principal of pattern completion (Rudy, Huff, & Matus-Amat, 2004), activation of one type of element would presumably activate the other. The notion of fear extinction implies a mechanism that produces a mimesis of the waking perception of emotional events (i.e., in which stimulus elements preferentially lead to activation of response elements). This ordering maximizes the involvement of the amygdala, which responds preferentially to perceptual stimuli and thus facilitates regulation of affect load. Our model thus differs from Hartmann's (1998a) model, in which stimulus element configurations (contexts) are driven entirely by subjective emotions or response elements and resembles in some respects that of Foulkes (Foulkes, 1982; Foulkes, Sullivan, Kerr, & Brown, 1988) by which context-appropriate emotions are generated in response to the unfolding dream content.

The expression during dreaming of fear and related emotions is considered to be a necessary step in dreaming's achievement of fear extinction. The fact that the emotions appearing in normal dreams are predominantly dysphoric (e.g., two thirds of normal dreams; Domhoff, 2000; Hartmann, 2000), with fear being by far the most prevalent (Nielsen, Deslauriers, & Baylor, 1991), is consistent with this suggestion, as is the frequent occurrence of nondistressing bad dreams and nightmares. As indicated in Figure 1, we consider normal dysphoric dreams, bad dreams and nondistressing, nontraumatic nightmares to be related in this respect. The variable intensity of fear expression in these types of dreams may simply reflect variations in the strength or efficacy of the hypothesized fear extinction function, which is presumed to vary in step with an individual's day-to-day emotional requirements, or affect load. In contrast, more severe nightmares, such as nontraumatic nightmares with high distress, trauma-related nightmares, and replicative PTSD nightmares, are assumed to be shaped additionally by the presence of affect distress and/or prior trauma. The next two sections describe in greater detail how pathogenic alterations in dream formation may underlie these distinctions.

Nightmares Are Pathological Expressions of Fear Memories

Pathogenic Changes Common to All Nightmares

Although fear memories are considered to be a normal phenomenon of human memory, they become pathological when (a) they are highly coherent and resistant to extinction and (b) they contain an excessive number of response elements (Foa & Kozak, 1986; Lang, Kozak, Miller, Levin, & McLean, 1980; Vrana, Cuthbert, & Lang, 1986). We propose that, during nightmares, individual vulnerability (i.e., high levels of affect load and/or affect distress)

interacts with the neurophysiological state of REM sleep so as to favor the activation of highly coherent fear memories—akin to those occurring in waking, fear-based pathological conditions. Accordingly, we propose that 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 affect distress. These assumptions are portrayed graphically in Figure 3.

Increased resistance to extinction. Resistance to extinction is reflected in several possible pathological events. First, during dream formation there may be a marked bias to activate complete fear memories rather than isolated elements of fear memories. Traumatic memories in particular preserve their structural coherence (Lang, 1977), perhaps because of conditions of heightened arousal during encoding, and thereby enter dreams as apparent replays of the original trauma. This replay is accompanied by a sense of perceptual reinstatement and distressing emotions. Novel configurations, including incompatible elements, are thus less likely to be introduced and thus less able to permit acquisition of new extinction memories. Second, the fear memory may simply resist activation altogether. Because availability of a fear memory is a prerequisite for the successful acquisition of an extinction memory (Foa & Kozak, 1986; Lang, 1977), a lack of extinction may occur if the fear memory is not fully activated either during dreaming or later during the waking state. Awakening from a nightmare may cut short fear memory activation and thereby prevent extinction. In addition, the awakening may actually strengthen the fear by serving as an avoidance response. In an extreme case, dream interruption insomnia is thought to result from preemptive awakenings from REM sleep to avoid nightmares altogether (see review in Nielsen & Zadra, 2005). Similarly, avoiding the recall of nightmares or the relating of nightmares to other persons upon awakening may prevent the eventual extinction of its underlying fear memory.

Increase in fear memory response elements. More numerous response elements are revealed by several indicators: an increase in the frequency and intensity of motor imagery in nightmares (e.g., escaping, defending oneself, fighting, attempting to scream), increased activation of the sleep state as signaled by physiological measures (e.g., heightened autonomic arousal), and the overt expression of sleep behaviors (e.g., moving in bed, speaking, emoting; Nielsen & Paquette, 2004). These response elements are often the identifiable correlates of distress that individuals report experiencing during and following their nightmares. A preponderance of response elements may result from a failure of recombinatory processes to limit the number of response elements that are activated and introduced into the narrative.

Influence by affect distress. The production of emotions during dreaming is further complicated by the facilitating influence of the affect distress trait. Affect distress elements may become incorporated into an individual's fear memories and other emotional structures such that, when a fear memory is activated, emotional responses will come to include expressions of distress as well as fear. An individual high in affect distress will therefore experience distress whenever certain fear memories are activated either during a nightmare or later in the day, when the nightmare is recalled. The distress experienced may even lead to further, similar nightmare episodes with recurrent themes. This cyclical process is consistent with the finding that intrusive imagery facil-

itates the release of stress hormones, which heightens affect distress and potentiates further intrusive imagery (Pitman & Orr, 1995). Affect distress may thereby contribute to the pathological portrait of an individual's nightmare disorder, including its cyclical nature.

It may be that all fear memories of an individual high in affect distress are influenced by affect distress in this manner. The establishment of any new fear memories may be similarly affected among individuals high in trait affect distress. Such a situation is consistent with the stress-diathesis approach and may explain common observations such as (a) a clinical history of abuse and trauma predicts future PTSD (Breslau et al., 1999) and (b) past and current traumatic events are often represented together in a single nightmare image (Hartmann, 1998b; Kramer, Schoen, & Kinney, 1987).

Pathogenic Changes in Nontraumatic Nightmares

We propose that occasional or even frequent nightmares (less than 1/week) that are nontraumatic and associated with low waking distress are produced by an intensification of the memory element activation/recombination mechanisms related to normal dreaming and modulated by affect load. As seen in Figure 3, affect load increases with short-term accumulations of interpersonal conflicts, current affective memory demands, and emotional reactions to transitory stressors. Affect load interacts primarily with the stimulus and contextual elements of fear memories such that high affect load may disrupt activation and recombination of dreamed stimuli rather than responses. This may have the effect of producing recurrent, typical, bizarre, or macabre imagery with little affect, mild anxiety, or fear—but not distress. Dreams with little emotional activation may be associated with greater fear memory resistance to extinction than more emotional dreams and nightmares.

Nontraumatic nightmares with high distress involve affect distress mechanisms in addition to fear memories and affect load mechanisms, although it is unlikely that the affect distress in this case is colored by the past occurrence of trauma (although this question has not been sufficiently studied). As mentioned in the previous section, affect distress influences primarily the response elements of fear memories such that participants high on this trait respond with more subjective upset both during and following their nightmares. The activation and recall of nightmare-related fear memories of high affect distress individuals may have inadvertent effects during the waking state, such as the stimulation of various conditioned expectancies and biases for the recall and perception of fear-relevant stimuli. Although these processes have not been investigated directly in frequent nightmare individuals, there is abundant evidence that (a) negatively arousing memories are recalled with greater clarity than neutral ones, particularly for memories of high personal significance (Ochsner, 2000), and (b) individuals with vivid imagistic abilities, a quality that characterizes frequent nightmare individuals, demonstrate heightened autonomic and emotional activation when presented with fear-relevant stimuli (Lang et al., 1993).

Pathogenic Changes in Posttraumatic Nightmares

The previous considerations allow us to better distinguish posttraumatic from nontraumatic nightmares. In the former variant, trauma causes an underlying fear memory to become firmly entrenched and highly resistant to extinction. This may mean that there is a diminution of recombinatory dream elements that is inversely proportional to the degree of fear memory coherence; degree of fear memory coherence is thought to underlie the severity of PTSD symptoms (Foa, Stekete, & Rothbaum, 1989). We suggest that fear memory resistance to extinction is responsible for both the finding that trauma-related nightmares incorporate many recognizable elements of the original trauma and the finding that replicative PTSD nightmares appear to replay the original trauma more completely (Mellman & Pigeon, 2005). A similar process may underlie memory more generally. For example, highly specific autobiographical memories are often associated with higher levels of emotional distress (Lang, 1979; Lang & O'Connor, 1984). Thus, in contrast to the variable progression of dysphoric imagery seen in nontraumatic nightmares, posttraumatic nightmares are more likely to be realistic and predictable because of the activation of structurally coherent fear memories.

Second, in the case of posttraumatic nightmares, the response elements of underlying fear memories may be especially salient and amplified by affect distress. This is suggested by the presence of several sleep-related hyperarousal symptoms, including increased awakenings, wake after sleep onset and insomnia, as well as nightmares in stages other than REM sleep and at times other than the habitual last third of the night, for example, Stage 2 nightmares occurring early in the sleep episode (van der Kolk et al., 1984). It is also suggested by the expression of motor activity in sleep, including more frequent REM-related twitches in leg muscles, more periodic leg movements in sleep in all stages, more frequent gross body movements, and more REM-related motor activity and vocalizations (Lavie, 2001). Hefez, Metz, and Lavie (1987) reported that explosive motor activity could be elicited from any stage of sleep in a patient with war-related PTSD. Similarly, Leopold and Dillon (1963) found that some traumatized survivors of a sea disaster expressed motor activity and vocalizations during sleep; in one case a patient appeared to attempt escaping from the bedroom. The finding that PTSD is comorbid with many cases of REM sleep behavior disorder (Husain, Miller, & Carwile, 2001), which is characterized by motorically active dreams and diminished REM sleep muscle atonia, further implicates excessive response elements and distress in posttraumatic nightmare formation.

In sum, the spectrum of dysphoric dreams that includes both nontraumatic and posttraumatic nightmares may be attributed to interactions between fear memories, short-term accumulations of affect load, and a pathogenic distress diathesis in vulnerable individuals. Fear memory coherence and resistance to extinction may be a factor common to all types of dysphoric dreaming, whereas affect distress distinguishes pathological from nonpathological nightmares. In addition to the pathological sleep changes described above, it is also highly likely that these processes interact in various ways during waking states and that sleep- and waking-state interactions among processes also occur. As an illustration, phobic individuals who selectively process phobic threat cues and focus narrowly on stimuli that activate their underlying phobia-relevant fear memories may apply the same acquired encoding biases to selectively scan their dream imagery for threats and to reflect upon their recalled nightmares with a similar narrow focus. As a result, such individuals may experience nightmares as more threatening

and distressing than do other types of individuals and may be more likely to misattribute their endogenous imagery to actual environmental threats—a type of source-monitoring deficit documented for normal dreaming (Johnson et al., 1984). Further, the physiological conditions of REM sleep may facilitate this process. Thus, nightmares can be likened to false alarm responses in a manner similar to the false alarm responses of panic disorder (Clark, 1988).

Neural-Level Explanation: A Brain Network for Fear Imagery

The pathophysiology of nightmares has received relatively little study and remains poorly understood. In contrast, a recent surge in research on the brain correlates of emotion, fear memory, PTSD, and to a lesser extent, normal human sleep and dreaming, has begun to clarify the nature of normal and posttraumatic emotional processing during sleep. This new research may be the best available source of information for modeling possible neural mechanisms of nightmare frequency and distress in both posttraumatic and nontraumatic cases. In this section, we review recent findings concerning brain and autonomic nervous system correlates of

emotions, fear conditioning, fear extinction, PTSD and REM sleep physiology to demonstrate that both posttraumatic and nontraumatic nightmares may arise from disturbances in a network of brain regions controlling the processing of fear and distress.

These findings support the view that nightmares and related pathology (e.g., PTSD) are associated with disruptions in a brain network of limbic, paralimbic, and prefrontal regions that constitutes 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. The full extent of this network is still not known and includes several brain regions not covered in the present review (e.g., insula, hypothalamus, nucleus accumbens; see Morgane, Galler, & Mokler, 2005), but the pathological changes that have been identified in recent years appear to affect two principal limbic structures as well as their corresponding prefrontal regulatory extensions (see Figure 4). These structures include the amygdala (A) and its medial prefrontal cortex (MPFC) extension, as well as the hippocampal (H) complex and its anterior cingulate cortex (ACC) extension (Hull, 2002; for reviews see Nutt & Malizia, 2004; Rauch, Shin, & Wright, 2003). A convenient acronym for this ensemble of struc-
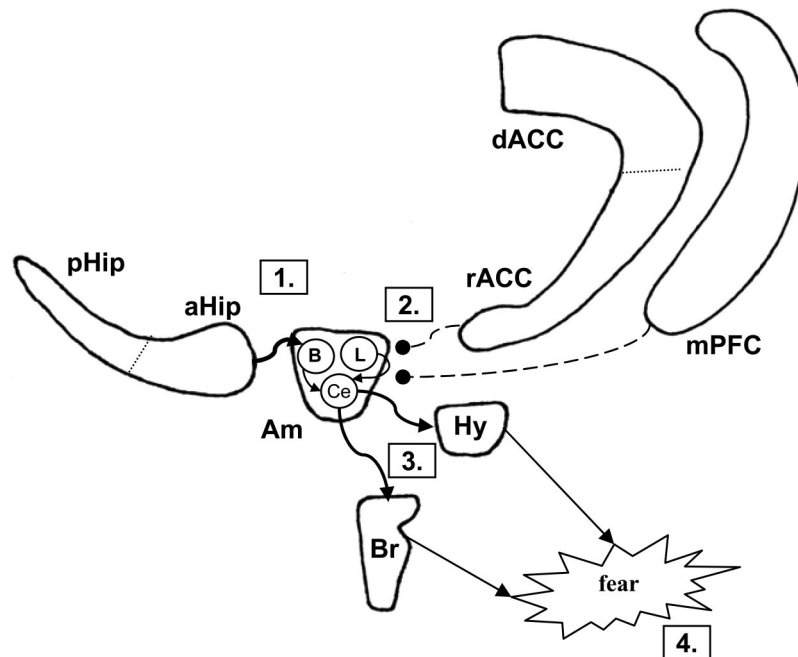

Figure 4. Schematic representation of one possible set of neural interactions in the AMPHAC (A = Amygdala, MP = MPFC, H = Hippocampus, AC = ACC) network that are implicated in the production of fear during normal dreaming. (1) Hippocampal contextual information is relayed in realistic (virtual) form via anterior hippocampus (aHip) to basal nucleus (B) of the amygdala (Am), where it is further processed by central (Ce) nucleus. (2) Medial prefrontal cortex (mPFC) and dorsal (dACC) and rostral (rACC) anterior cingulate cortex afferents to lateral (L) and Ce amygdalar nuclei regulate the output of Ce neurons to induce extinction, to signal distress, and to maintain appropriate levels of fear. (3) The Ce nucleus signals brainstem (Br) and hypothalamus (Hy) circuits, producing (4) the autonomic and behavioral correlates of fear within the dream. Excitatory connections are in solid lines; inhibitory connections are in dashed lines. Although most components of this network are reciprocally connected, for readability only connections judged most pertinent to the consolidation and extinction of fear are displayed. The model is not intended to account for all aspects of dreaming, only the production of negative affect. Fear at nightmare intensities may be produced by perturbations in any or all of these interconnected components as explained in the text.

tures is the AMPHAC (A = amygdala, MP = MPFC, H = hippocampus, AC = ACC) network. Unless otherwise indicated, the research cited refers to results from human participants.

General Qualities of the AMPHAC Network

We suggest that the four designated brain regions, although serving several different emotional functions, operate in a coordinated manner as part of a larger emotional control structure. This control structure, in turn, influences other perceptual, cognitive, memorial, and affective brain events. We single out these regions on the basis of several anatomical and functional considerations:

1. There is ample evidence of anatomical connections between the regions (for reviews, see Aggleton, 1992; Faw, 2003; Morgane et al., 2005). Amygdala, in particular, is massively connected to the other regions in a reciprocal fashion (Aggleton, 1992). The four regions are also robustly connected to sensory, motor, and autonomic brain regions and thus are well suited to mediate higher cognitive functions, behaviors, and affective responses. Functional connections between these regions have been reported. The amygdala's influence on other brain regions has particularly strong and convergent support (Gilboa et al., 2004; McGaugh, 2004; Roozendaal, McReynolds, & McGaugh, 2004), and roles for the hippocampus and amygdala in basic dream production are now widely accepted (for review, see Nielsen & Stenstrom, 2005).

2. All four regions have been implicated in the detection, generation, maintenance, and remembering of normal emotions, fear in particular (Maren & Quirk, 2004; Phan et al., 2002; Phan, Liberzon, Welsh, Britton, & Taylor, 2003). The amygdala is central in mediating a number of visceral and autonomic reactions such as blood pressure, heart rate, and respiration, likely by virtue of its connections with hypothalamic structures (Aggleton, 1992). The MFPC and hippocampus are critical for the acquisition and memory of conditioned fear and fear extinction. The ACC is central in mediating affect distress (Eisenberger & Lieberman, 2004).

3. These brain regions are associated with both state and trait individual differences in emotional responding, thus supporting the cross-state continuity assumption that state and trait differences (vis-à-vis affect load and affect distress) influence nightmare frequency and nightmare distress differentially. Among the individual differences related to affect distress that imaging studies have found most consistently to be linked to the AMPHAC network are fear conditionability (Furmark, Fischer, Wik, Larsson, & Fredrikson, 1997), response inhibition (Langenecker & Nielson, 2003), novelty seeking (Kabbaj & Akil, 2001), and fear disposition/anticipatory worry (Pujol et al., 2002).

4. All of the regions are implicated in emotion-based disorders including, but not limited to, anxiety disorders (generalized anxiety, social anxiety, phobia, panic, obsessive-compulsive disorder), mood disorders (depression, bipolar disorder), personality disorders (borderline, psychopathy), and other psychiatric conditions (schizophrenia). These studies are reviewed in a later section.

5. All of the brain regions have been implicated in PTSD. Several studies have demonstrated that PTSD patients have reduced volumes of one or another of the structures (for reviews, see Nemeroff et al., 2006; Nutt & Malizia, 2004). Most work has demonstrated reduced hippocampal volumes (e.g., Bremner, Mletzko, et al., 2005; Lindauer et al., 2004, 2005; Lindauer, Olff,

Meijel, Carlier, & Gersons, 2006; Vermetten, Vythilingam, Southwick, Charney, & Bremner, 2003; Vythilingam et al., 2005). Two meta-analyses (Kitayama, Vaccarino, Kutner, Weiss, & Bremner, 2005; Smith, 2005) of 9 and 13 studies comprising 138 and 215 PTSD patients and 201 and 325 controls, respectively, both reported bilateral hippocampal volume reductions. A few studies have reported volume reductions for the ACC (Rauch, Shin, et al., 2003; Woodward et al., 2006; Yamasue et al., 2003), and two have reported reduced amygdalar and hippocampal volumes (Matsuoka, Yamawaki, Inagaki, Akechi, & Uchitomi, 2003; Nakano et al., 2002, respectively). Failures to find reduced volumes have also been reported (Bonne et al., 2001; Bonne, Grillon, Vythilingam, Neumeister, & Charney, 2004; Golier et al., 2005), raising the question of whether reduced volumes reflect risk factors for PTSD, such as alcohol abuse or dependence, rather than consequences of PTSD per se (Jelicic & Merckelbach, 2004).

6. Imaging studies have also shown that activity levels in the four AMPHAC regions increase during REM sleep above levels seen in either wakefulness or NREM sleep (Braun et al., 1997, 1998; Hobson, Stickgold, & Pace-Schott, 1998; Maquet et al., 1996, 1997; Nofzinger, 2004; Nofzinger, Mintun, Wiseman, Kupfer, & Moore, 1997). The network is thus a vital component of the physiological infrastructure of normal dreaming and is likely influential in shaping emotional imagery during both normal and disturbed dreaming.

The preceding anatomical and functional considerations clearly implicate the AMPHAC neural network in a multiplicity of emotion-based personality attributes and anxiety disorders in addition to nightmares. Of the many findings that have emerged recently, observed changes associated with PTSD are particularly useful for understanding nightmare production more generally. These changes include increased amygdala activity, increased hippocampal activity, decreased MPFC activity and either increased or decreased ACC activity (Bremner, 2003; see reviews in Liberzon & Phan, 2003; Pitman, Shin, & Rauch, 2001). A predominant hypothesis (Nutt & Malizia, 2004; Rauchs et al., 2004) is that PTSD symptoms result from a hyper-responsivity of the amygdala to threat stimuli, leading to exaggerated symptoms of arousal and distress, coupled with a failure of the other brain regions (hippocampus, MPFC, ACC) to adequately dampen this activation. We suggest that a similar pathological mechanism may explain both posttraumatic and nontraumatic nightmares. That is, during the formation of nightmares, the amygdala may become increasingly responsive to fear-related memory elements portrayed in the dream, while its regulation by MPFC, hippocampus and ACC is disturbed in some way. Severe and posttraumatic nightmares are particularly affected by disturbance of the ACC, which allows the amplification of distress.

Brain imaging studies have demonstrated that dysfunctions of these brain regions are often rendered more apparent under specific experimental situations such as viewing aversive stimuli or generating emotional imagery. Dreaming is a naturally occurring situation that may amplify the dysfunctions in a similar manner. The nature and quality of sleep, and of REM sleep in particular, likely interacts with these dysfunctional regions to sculpt the affective and cognitive contours of nightmare experience and render it distinct from other anxiety states. If so, the extensive literature on brain function can be mined for indicators of which network components fail during sleep and result in nightmares of

different types and intensities. One goal of the present section is thus to describe some possible ways in which REM sleep-related disturbances in the AMPHAC network could produce an increase in the frequency of nightmares and account for some of the diversity in their experiential makeup, intensity, and associated distress (for reviews of nightmare diversity, see Nielsen, 2005; Nielsen & Zadra, 2005).

The following sections describe some views on how each of the network components may be implicated in cognitive–emotional functions, in fear conditioning and extinction, in PTSD symptomatology, and more speculatively, in the formation of posttraumatic and nontraumatic nightmares. A brief summary of the general roles of each of the four components is as follows:

1. Amygdala: control center for expressing affect load; critical to conditioned fear, fear memory, fear detection, and autonomic activation;

2. MPFC: down-regulates emotional activity in the amygdala, in part, by producing extinction memories to inhibit conditioned fear;

3. Hippocampus: regulates the extinction and reexpression of conditioned fear via the amygdala and MPFC, in part, by control over fear memory context;

4. Anterior cingulate cortex (ACC): regulates the degree of affect distress expressed during emotional activation.

Amygdala: Control Center for Affect Load

Amygdala activity is robustly related to fear (LeDoux, 2000; Ohman & Mineka, 2001; Phan et al., 2002), but its component structures may mediate a variety of dysphoric emotions. The amygdala is not usually activated when stimulation involves recall or imagery but is reliably active when visual perception is required (Phan et al., 2002). It is extremely responsive to emotional facial expressions of many kinds (Bishop, Duncan, & Lawrence, 2004; Nelson et al., 2003; Somerville, Kim, Johnstone, Alexander, & Whalen, 2004) and can react rapidly to fearful stimuli outside of awareness (Morris, Ohman, & Dolan, 1998; Pegna, Khateb, Lazeyras, & Seghier, 2005; Whalen et al., 2004; M. A. Williams & Mattingley, 2004). Basolateral amygdala is responsive to the masked presentation of fearful eyes against a black background (Whalen et al., 2004). Among highly trait-anxious participants, basolateral amygdala responds to masked fearful faces, whereas dorsal amygdala responds selectively to consciously perceived fearful faces, regardless of trait anxiety status (Etkin et al., 2004). An association between high state anxiety and amygdala activity in response to fearful stimuli has been reported (Bishop, Duncan, & Lawrence, 2004), as has an association between trait anxiety and resting state amygdala activity among depressed patients (Abercrombie et al., 1998). These findings of interactions with anxiety status may well explain some failures to find amygdala activation with masked fearful stimuli (Phillips et al., 2004). The findings together suggest that the amygdala can automatically regulate brain responses to subtle perceptual signs of fear at very early stages of perception (R. J. Davidson, Putnam, & Larson, 2000). One of its normal functions may be to impart danger warnings when threatening signals are visually detected (Davis & Whalen,

2001). Amygdala oversensitivity may therefore underlie the enhanced threat-sensitivity that is characteristic of anxious participants (Etkin et al., 2004). We suggest that the amygdala's normal role during dreaming is similar to its role during wakefulness, except that it responds to threatening perception-like (virtual) stimuli, rather than to perceptual stimuli per se. Specifically, its responsivity to dreamed stimuli may be rendered equivalent to that of waking perceptual stimuli by REM sleep's production of imagery that mimics perception, that is, imagery that has a here-and-now or virtual reality quality (Nielsen & Stenstrom, 2005). Furthermore, amygdala oversensitivity during dreaming may produce an enhanced sensitivity to threat and thus lead to atypical fear intensification during a nightmare.

Amygdala mediates conditioned fear. Of particular interest for the present review is that the lateral nucleus of the amygdala in a variety of animal species mediates a form of memory whose hallmark is the acquisition and expression of conditioned fear (for review, see Maren & Quirk, 2004), that is, the process by which a neutral stimulus, such as a tone, becomes fear inducing once it has been coupled with an aversive event, such as a footshock (e.g., Pavlov, 1927). Memories of such conditioned fear (fear memories) can be acquired quickly, sometimes after only a single pairing of the neutral and aversive events (Armony & LeDoux, 1997), and they are quite durable. It is feasible that amygdala-mediated fear memories are expressed to a limited degree during normal dreaming and to a fuller extent during nightmares.

The context in which fear conditioning takes place can also modulate the strength of fear memory expression. For example, a remembered fear response may be weakened or even extinguished if the fear-provoking stimulus occurs in a context different from the original learning context. Context itself may even become a learned aversive stimulus, as when darkness becomes a contextual trigger of nightmares in traumatized individuals (Grillon, Pellowski, & Merikangas, 1966). Such contextual conditioning variations depend upon amygdala activity but also require hippocampal involvement (Armony & LeDoux, 1997), as described in a later section. Even when a fear memory has been successfully extinguished, presumably by involvement of the MPFC (see the next section), studies on nonhuman animals indicate that stimulation of the amygdala can reinstate it (Kellett & Kokkinidis, 2004).

Amygdala role in emotional memory. Beyond its role in fear conditioning and extinction, there is compelling evidence that the amygdala modulates other forms of emotional memory, such as inhibitory avoidance learning (McGaugh, 2004) and declarative emotional memory (Canli, Zhao, Brewer, Gabrieli, & Cahill, 2000; Phelps, 2004). This evidence suggests that the amygdala may be integral to the various memory consolidation functions that have been proposed for sleep and dreaming (for review see Stickgold, 2005). We suggest one possible memory mechanism for dreaming is that the amygdala controls the degree of emotional activation that accompanies the pseudoperceptual elements expressed during dreaming, thus increasing or decreasing the memorability of these elements. This possibility is consistent with the fact that amygdala activation enhances memory for emotionally intense stimuli (Kensinger & Corkin, 2004; Medford et al., 2005), especially negative stimuli (Canli et al., 2000). It enhances memory by selectively facilitating or impeding processes underway in other parts of the AMPHAC network, for example, hippocampus and MPFC (in rats; Nathan, Griffith, McReynolds, Hahn, &

Roozendaal, 2004) and elsewhere, for example, caudate nucleus and cortex (in humans; see review in McGaugh, 2004). Because emotional memories are more persistent and vivid than other types of memories (Bradley, Greenwald, Petry, & Lang, 1992; Canli et al., 2000; Ochsner, 2000), the amygdala's addition of affective salience to hippocampally mediated memories may enhance both the encoding and the consolidation of these memories (Phelps, 2004).

This type of controlled affectivization of memory elements by amygdala activity may explain why nightmares are recalled so clearly and so often. However, in the present review, we focus on the amygdala's role in fear conditioning as more central to an explanation of nightmare production and therefore do not further pursue this and other memory theories of dream function. Readers are directed to Stickgold, Fosse, and Walker (2002) for a more detailed neurobiological hypothesis of declarative memory processes during sleep in relation to PTSD and to McGaugh (2004) and Maren and Quirk (2004) for thorough discussions of basolateral and lateral amygdala modulation of emotional memory more generally.

Amygdala and PTSD. In PTSD patients, amygdala activity appears insufficiently regulated. It is hyper-reactive to trauma-related stimuli (for a review, see Hull, 2002; Nutt & Malizia, 2004; Pissiota et al., 2002), even when such stimuli are below recognition threshold (Hendler et al., 2003). This exaggerated reactivity correlates positively with the severity of PTSD symptoms but not with the severity of either depression or trauma exposure (Rauch et al., 2000; Shin et al., 2004). During repeated symptom provocation trials, amygdala activation progressively increases (Gilboa et al., 2004). The fact that amygdala, MPFC, and ACC are differentially activated during fear stimulation trials suggests that amygdala function may be uncoupled from its prefrontal control mechanisms when PTSD patients process emotionally negative stimuli (Liberzon & Phan, 2003; Rauch et al., 2000; L. M. Williams et al., 2006). This uncoupling appears to be exacerbated over time, suggesting that the PTSD vulnerability may become more apparent with demands for sustained attention (L. M. Williams et al., 2006).

Implications for nightmares. Amygdala activity very likely underlies the expression of fear during dreaming as it does during waking states. The predominance of fear in dream reports (Merritt et al., 1994; Nielsen et al., 1991) and the activation of amygdala during REM sleep beyond waking-state levels (Braun et al., 1997; Maquet et al., 1996) support this possibility. Moreover, during nightmares, an overactivated amygdala may underlie the full expression of fear memories. This possibility is consistent with facts reviewed earlier to the effect that nightmares of all types depict an individual's current concerns, whereas posttraumatic nightmares (trauma related or replicative) often clearly depict either partial or complete episodic memories of conditioned fear responses.

At least one function of dreaming may be to adaptively respond to such expressions of conditioned fear, that is, to attempt to regulate or even extinguish them. However, as the following sections will demonstrate, this view may be too simplistic. Fear in humans is a complex phenomenon that blends processes of fear conditioning with those of acquiring and managing fear contexts, building fear extinction memories, and managing emotional distress. Consideration of the amygdala's interactions with MPFC, hippocampus, and ACC is necessary to provide a more complete portrait of fear and its possible regulation during sleep.

MPFC: Mediator of Extinction

The MPFC is believed to regulate impulsive emotional expression stemming from amygdala activity (Bonne et al., 2004). One meta-analysis of 55 positron emission tomography (PET) and functional magnetic resonance activation studies using emotional tasks (Phan et al., 2002) suggests that its regulatory role may extend to processing all types of emotions. It is particularly essential for the extinction of fear memories (Santini, Ge, Ren, Pena de, & Quirk, 2004) which occurs when a fear memory is repeatedly activated in a context different from the one in which it was acquired. Extinction occurs not through the unlearning or disappearance of an acquired fear memory but through the production of a new extinction memory that inhibits its expression (see review in Bouton, 2002). Nonhuman animal studies have indicated that extinction memories are more labile than fear memories (Maren, 2005) and are disrupted by inactivation of the MPFC (Milad & Quirk, 2002). With disruption of an extinction memory, the original fear memory resurfaces. In fact, fear memories may resurface for several reasons: the passage of time (spontaneous recovery), reexposure to the original aversive stimulus (reinstatement), reexposure to the original learning context (renewal; see review in Bouton, 2002) and exposure to trauma (Armony & LeDoux, 1997). Some studies (Chelonis, Calton, Hart, & Shachtman, 1999; Gunther, Denniston, & Miller, 1998), but not all (Bouton, Garcia-Gutierrez, Zilski, & Moody, 2006), have suggested that establishing multiple extinction memories diminishes the likelihood that a specific fear memory will be renewed. If true, this implies that the regulation of fear memories requires considerable sustained maintenance to counteract the many factors favoring their return. MPFC, likely in concert with the context-regulation processes of the hippocampus (see next section), appears critically implicated in this function. The state of dreaming, which occupies from 20%–25% of every normal sleep episode, may be ideally suited to facilitate such a long-term, process-intensive function. Nontraumatic nightmares may reflect an acceleration of this function, whereas severe and posttraumatic nightmares may indicate that a breakdown of this function has occurred, that is, that one or more extinction mechanisms have ceased to function properly.

An anatomical circuit has been described that clarifies the role of MPFC in regulating amygdala activity (Quirk, Likhtik, Pelletier, & Pare, 2003). MPFC appears to gate relays within the amygdala, especially at the level of lateral amygdala (where sensory afferents are processed) connections to the central nucleus (where emotional efferents are selected). In animal models, MPFC stimulation activates inhibitory projections to both of these subregions (Maren & Quirk, 2004). Such inhibition may delay habitual emotional responses and allow the acquisition and expression of new responses, as when instructions to cognitively reappraise highly negative stimuli or to down-regulate negative affect simultaneously increases MPFC activity and decreases amygdala activity (Ochsner, Bunge, Gross, & Gabrieli, 2002; Ochsner et al., 2004).

MPFC and PTSD. Consistent with this, PTSD veterans exhibit decreased MPFC activity during script-driven traumatic (vs. neutral) imagery that is inversely correlated with both amygdala activation and PTSD symptom severity (Shin et al., 2004). Also, treatment response to the selective serotonin reuptake inhibitor citalopram in PTSD patients is correlated with degree of left

MPFC activity; that is, as Clinician Administered PTSD Scale—Symptom Status Version severity scores decrease, posttreatment MPFC values increase (Seedat et al., 2004). A salient possibility that remains to be investigated, and that finds ample support in nonhuman animal studies (Kellett & Kokkinidis, 2004; Quirk et al., 2003; Quirk & Gehlert, 2003; Santini et al., 2004), is that diminished MPFC activity prevents the consolidation of extinction memories needed to counteract the overwhelming affective impact of posttraumatic memories.

Some studies have also implicated MPFC in the regulation of affect distress, suggesting that MPFC circuits are further modulated by the ACC (see the *ACC: Mediator of Affect Distress* section below). Cerebral blood flow studies demonstrate that MPFC activity is elevated in highly anxious participants and is correlated with both trait (but not state) anxiety (Paulus, Feinstein, Simmons, & Stein, 2004) and the negative affect personality dimension (Zald, Mattson, & Pardo, 2002).

Implications for nightmares. MPFC activation during REM sleep (Braun et al., 1997; Maquet et al., 1996) may play a role in normal dreaming, preventing the excessive expression of all types of affect but of fear in particular. MPFC activity may explain why emotions during dreaming are often muted or unclear and, in many cases, completely and inappropriately absent (Foulkes et al., 1988). Further, heightened MPFC activity during dreaming may contribute to the maintenance or acquisition of extinction memories in response to negative emotions of different types. To optimize emotional adaptation, processes of extinction maintenance may be applied to mild negative emotions as well as to more extreme, sometimes posttraumatic emotions. Such optimization may prevent the long-term accumulation of affect and serve to down-regulate anxiety and distress.

It remains unknown how MPFC reacts during actual nightmares, although brain imaging assessments could provide critical clues to MPFC function. If MPFC activity is found to be diminished, this may signal a disinhibited expression of amygdala-related fear responses and even a breakdown in the maintenance or acquisition of extinction memories. If MPFC activity is increased, it may signal adaptive attempts at building such fear-suppressing extinction memories. The diminution of MPFC activity in PTSD patients during script-driven imagery suggests that its activity during posttraumatic nightmares may be diminished, which would suggest failures in fear regulation and in the expression of extinction memories. Such failures are even suggested by the content of posttraumatic nightmares, replicative nightmares in particular: Patients are often both completely overwhelmed by their emotions and unable to find any effective means to assuage them within the story line of the dream.

As the following section will reveal, the regulation of fear memories during dreaming and nightmares requires consideration of yet another level of complexity that involves the hippocampus' role in mediating both fear memories and fear extinction through the processing of memory for context.

Hippocampus: Mediator of Fear Memory Context

An essential role for the hippocampus in encoding and consolidating episodic memories is now widely accepted (Eichenbaum, 2001; Squire, 2004) and its role in representing stimuli that occur in novel contexts is well known (Nyberg, 2005). A more specific hippocampal role in the processing of fear memories is also the subject of much study (for reviews, see Hamm & Weike, 2005; Maren, 2005; Sanders, Wiltgen, & Fanselow, 2003). Several results from human and nonhuman animal experimentation support the existence of a network in which the hippocampus regulates the expression of fear and fear extinction memories (acquired by the amygdala and MPFC; see the two previous sections) on the basis of evaluation of the context within which the fear stimuli occur (Corcoran & Maren, 2004; for a review, see Maren, 2005). In nonhuman species, such hippocampal influences on the amygdala are demonstrated by evidence that stimulation of the hippocampus activates memory plasticity in the amygdala (Maren, 2005; Ribeiro et al., 2002), and its inactivation impairs contextual fear conditioning (Bannerman et al., 2004; Corcoran, Desmond, Frey, & Maren, 2005; Trivedi & Coover, 2004). In humans, the influences are demonstrated by evidence that anterior hippocampal pathology predicts amygdala activity during the encoding of emotional stimuli (Richardson, Strange, & Dolan, 2004).

The centrality of context. The claim that these hippocampal influences on the amygdala depend upon the evaluation of (and memory for) context is also supported by converging evidence. Animal studies have revealed, first, that memory representations for novel contexts are distributed throughout the longitudinal extent of the hippocampus and that these support contextual fear memories (Rudy & Matus-Amat, 2005). The latter are memories of a context (e.g., training cage) in which an animal has acquired a conditioned fear memory (e.g., tone-footshock association). Exposure to the context alone will also evoke fear, but the latter effect can be eliminated by deactivating the hippocampus—without disrupting the fear memory per se (for review see Eichenbaum, 2004). Second, the hippocampus supports the context preexposure facilitation effect, which refers to the fact that an animal shocked immediately after being placed in a new context does not learn to fear that context unless it is preexposed to it a day before the shock. Disabling the ventral hippocampus reduces this facilitation effect, presumably by preventing the hippocampus from preparing a coherent representation of the context to which the fear can later be associated (Matus-Amat, Higgins, Barrientos, & Rudy, 2004). Third, (dorsal) hippocampus facilitates the acquisition of fear extinction memories, that is, the learned inhibition of fear in new contexts (Corcoran et al., 2005; see the previous section). Fourth, and in a related vein, several studies (Cammarota, Bevilaqua, Kerr, Medina, & Izquierdo, 2003; Corcoran et al., 2005; Corcoran & Maren, 2001, 2004), but not all (Frohardt, Guarraci, & Bouton, 2000; A. Wilson, Brooks, & Bouton, 1995), have suggested that the hippocampus regulates the return of fear responses after the latter have been extinguished in new contexts; inactivation of the hippocampus prevents this return of fear.

In humans, demonstrations of hippocampally mediated contextual influences on the amygdala are less numerous (see review in Phelps & LeDoux, 2005), although the contextual mediation of fear acquisition, extinction, and reinstatement demonstrated in humans is similar to that of nonhuman animals (LaBar & Phelps, 2005). A specific hippocampal link to fear extinction was reported for 2 amnesic patients (1 with MRI-verified hippocampal atrophy) who failed to recover fear responses despite showing normal acquisition and extinction of fear memories; these patients responded as if their extinction training in the original learning context had occurred in a novel context (LaBar & Phelps, 2005).

More generally, humans can normally acquire representations of an event's emotional significance through fear instructions or other symbolic means rather than through direct aversive experiences (Hamm & Weike, 2005). For example, one may come to fear a dog after hearing about how it viciously attacked someone else (Phelps, 2004). Healthy participants who received instructions to expect electrical shocks during presentation of neutral (blue square) stimuli demonstrated both affective arousal and amygdala activation in response to the stimulus—even though they never received any actual shocks (Phelps et al., 2001). Context surrounding facial expressions can also alter amygdala activity. If a face depicting surprise is preceded by a sentence suggesting a negative reaction, such as "She just lost $500 dollars," a greater amygdala response is evoked than if the context to the same face suggests a happy reaction ("She just won $500 dollars"); a very large response in the parahippocampal gyrus is also observed (Kim et al., 2004). In a similar vein, instructions to reappraise or down-regulate emotional responses to negative photographs produce diminutions in amygdala activity, whereas instructions to maintain emotion produce increases in such activity (Ochsner et al., 2004).

Although studies have not consistently demonstrated neural connections between the amygdala and hippocampus, one theory that may explain the discrepancies implicates a mediating role for the MPFC (Maren, 2005). When fear stimuli are encountered in a context different from an extinction context, the hippocampus is thought to inhibit the MPFC, which in turn fails in its normal role of suppressing amygdala-induced fear.

Hippocampal changes in PTSD. As described earlier, numerous studies have reported that bilateral hippocampal volume in PTSD patients is lower than in either traumatized patients without PTSD or normal controls (see reviews in Kitayama et al., 2005; Smith, 2005). Treatment of PTSD with phenytoin produces an increase in right hippocampal volume, which is correlated with a reduction in PTSD symptoms (Bremner, Mletzko, et al., 2005). Treatment with psychotherapy has no similar effect (Lindauer et al., 2005). There is also evidence that hippocampal neuronal integrity, as measured by metabolite ratios (*N*-acetylaspartate/creatine, or NAA/Cr, and choline/creatine, or Cho/Cr), is reduced (Mahmutyazicioglu et al., 2005). These findings suggest that trauma reduces hippocampal activity and diminishes the cognitive functions attributed to it (see review in Nemeroff et al., 2006). In fact, PTSD patients demonstrate decreased hippocampal activity during personalized trauma scripts (Bremner et al., 1999), during emotionally valenced declarative memory tasks (Bremner et al., 2003), during verbal and visual memory tasks (Lindauer et al., 2006) and during verbal declarative memory tasks more generally (Bremner et al., 1995; Shin et al., 2004).

Implications for nightmares. These findings and concepts help to clarify the role that the hippocampus may play within the larger AMPHAC network to regulate emotions in dreams and nightmares and to facilitate a possible fear extinction function. The hippocampus, in conjunction with the amygdala and MPFC, appears to play a central role in regulating multiple aspects of fear memory expression: conditioned fear memories, contextual fear, context preexposure, fear extinction memories, and the renewal of conditioned fear. The hippocampus' capacity for handling vast numbers of declarative memories probably allows it to process the large volume of context representations implicated in an individual's personal fear history.

Imaging studies indicate that hippocampal activity during REM sleep meets or exceeds its activity during either NREM sleep or the waking state (Braun et al., 1997; Maquet et al., 1996). It is thus at its peak of activity when the brain is producing its most visually and emotionally intense dream imagery. Phenomenological evidence is quite clear that vivid dreaming is experienced as subjectively real (i.e., as if the person were acting and interacting with real characters in real places). This subjective realism has been identified by many authors as dramatic quality (Freud, 1900/1976), self-participation (Bosinelli et al., 1974), and virtual reality quality (Nielsen et al., 1994; Revonsuo, 2000). Revonsuo went so far as to suggest that the virtual reality quality of dreams, and especially nightmares, serves the evolutionarily adaptive function of rehearsing survival responses in the face of threat. These observations and speculations indicate the extent to which heightened hippocampal activity is associated with a mode of awareness that involves the consistent reproduction of realistic waking contexts—fear contexts in particular.

Clarifying the precise nature of these contexts may be key to understanding how the hippocampus is implicated in dreaming. Despite its subjective realism, dreaming is clearly not a video-like reproduction of waking episodic memories. As discussed earlier, dreams only rarely portray past experiences accurately (Fosse et al., 2003), except in the case of trauma. Rather, they create new, unexpected, and sometimes bizarre contexts out of a combination of cognitive materials: episodic memory fragments, semantic knowledge (including self-knowledge), symbols, and completely novel configurations. Current opinion is mixed on precisely how the hippocampus may contribute to this paradoxical representation of realistic, albeit new, contexts (see review in Nielsen & Stenstrom, 2005). Some have suggested that diminished hippocampal-to-neocortical communication during REM sleep activates memory fragments without the spatiotemporal contexts that the hippocampus would normally furnish (Payne & Nadel, 2004; Stickgold et al., 2001). Others have hypothesized that the hippocampus either integrates recent memories into new context memories (Johnson, 2005) or structures them into narratives around an individual's goals, desires, and problems (Paller & Voss, 2004). Still others (Nielsen & Stenstrom, 2005) have suggested that the hippocampus' implication in the spatial and temporal binding of memory elements (Luo & Niki, 2005; Newman & Grace, 1999; Wallenstein, Eichenbaum, & Hasselmo, 1998) coordinates reality quality itself. By this account, the hippocampus controls an ongoing synthesis of memory fragments to produce a continuous here-and-now illusion of contextually appropriate activity, that is, the perception-like illusion of being and acting in a particular place ("here") in the continuous present ("now"). All of these new models raise the importance of contextual processing during dreaming and concur to some extent in suggesting that altered hippocampal functioning during REM sleep facilitates memory consolidation. However, none of them addresses the important possibility that the hippocampus contributes in a central way to the regulation of fear memories.

The reviewed literature does suggest several concrete ways in which hippocampal contextual processes may mediate different aspects of fear memory during dreaming and nightmares. Two possibilities, the regulation of contextual fear and context preexposure facilitation, implicate the hippocampus in the acquisition of new fear memories under certain conditions. Although it is con-

ceivable that dreaming facilitates fear learning by binding existing fear-eliciting stimuli and new contexts (fear context learning) or by anticipating future fear-learning contexts (context preexposure facilitation), such possibilities are less compelling and will not be further considered here. Interested readers might wish to pursue the threat simulation model of nightmares (Revonsuo, 2000) as an instance of either of these two conceptualizations.

Rather, we find it more likely that the hippocampus is implicated in the acquisition and long-term maintenance of fear extinction memories during dreaming and, further, that a failure of this role is reflected in highly distressing and posttraumatic nightmares. In the case of normal dreaming, fear memories may be activated, in whole or in part, in the presence of numerous nonaversive settings (contexts) such that many extinction memories are either formed or strengthened. These will inhibit the future expression of associated fear memories and, consistent with some studies (Chelonis et al., 1999; Gunther et al., 1998), prevent their renewal even in the original learning context. Dreaming, by its relentless presentation of novel, unexpected, unfamiliar, and/or nonthreatening contexts, provides a consistent, ever-changing sequence of contexts to supply the formation of extinction memories. Note that by this account, a depiction of successful avoidance behaviors in the face of fear stimuli is not necessary to facilitate an adaptive response (cf. Revonsuo, 2000). Rather, the coupling of the fear memory and the nonaversive context alone is sufficient for extinction. However, two conditions are likely necessary to optimize extinction during dreaming. First, the nonaversive contexts must occur while an extant fear memory is active. A representation of either the eliciting stimulus, the fear response, or both the stimulus and response should be simultaneously active for the new context to promote or strengthen extinction memories. The fact that fear is the predominant emotion in dreams (Merritt et al., 1994; Nielsen et al., 1991) suggests that such memories are easily and frequently activated. Further, clinical studies have documented how stimulus elements related to existing fears are often depicted in dreams and have revealed how these are usually embedded in novel or unrelated contexts (Cartwright, 2005; Hartmann, 1998a). More empirical studies of dream content are needed to assess these attributes of dream experience, especially the simultaneous representation of activating and inhibiting stimulus elements.

Second, nonaversive contexts would be more likely to influence extinction if they are portrayed in a subjectively realistic form. This would ensure that the amygdala's implication is maximized, as it responds preferably to sensory as opposed to imagined stimuli (Phan et al., 2002). As described above, dream imagery is consistently realistic in nature.

Some commonplace, albeit curious, observations about dream experience are consistent with this view of dream function, for example, the inexplicable occurrence in dreams of very old fear memories, even those experienced in childhood (Grenier et al., 2005). This may indicate that even old memories require periodic maintenance and must be activated during dreaming for extinction processes to be effective.

With the more serious disruptions of hippocampal function seen in PTSD, the acquisition and/or maintenance of fear extinction memories in dreams may be severely compromised. In replicative PTSD nightmares, representations of the original traumatizing context as well as the person's original fear responses are more or less intact and thus less likely to benefit from extinction memories.

Because trauma exposure is one of several factors able to induce renewal of long-extinguished fear memories, a debilitating consequence of PTSD may be that hippocampal processes lose their capacity to prevent the renewal of other, unrelated fear memories. Such a possibility is consistent with the frequent observation that trauma-related nightmares intermix traumatic elements with elements of long-past fearful experiences (Hartmann, 1998b; Kramer et al., 1987). This phenomenon has been hypothesized to reflect some kind of adaptive assimilation or working through of a current trauma. However, the presence of such old elements may also signify renewal of previously extinguished fears. Such speculations are amenable to many types of empirical tests.

Implication of the amygdala, MPFC, and hippocampus in fear conditioning, fear extinction, and fear reinstatement together provides a strong basis for explaining variations in the frequency and composition of nightmares and a possible fear extinction function of dreaming. However, this network still falls short in explaining the dominant role of affect distress in the lives of severe and posttraumatic nightmare sufferers. Consideration of the ACC system for mediating distress will clarify this important dimension.

ACC: Mediator of Affect Distress

The ACC is implicated in nightmare formation by virtue of its proposed capacity to mediate affect distress. We demonstrate the functional importance of this role by enumerating close links that have been established between ACC, pain, pain-related distress, and social distress. Further, we highlight key relationships between ACC and gender, personality, and trauma that may help explain some of the differences in nightmare distress reviewed earlier.

Pain, anticipated pain, and pain distress. Numerous reports have implicated the ACC in the neural circuitry of pain (de Leeuw, Albuquerque, Okeson, & Carlson, 2005; Koyama, McHaffie, Laurienti, & Coghill, 2005; Peyron, Laurent, & Garcia-Larrea, 2000; Porro et al., 2002; Qiu et al., 2006). The ACC appears to facilitate the transmission of pain sensation from spinal sources (Zhang, Zhang, & Zhao, 2005) through structures in the midbrain periaqueductal grey (Kupers, Faymonville, & Laureys, 2005). ACC activity is abnormally elevated in chronic pain syndromes such as orofacial pain (de Leeuw et al., 2005). Several lines of evidence have indicated that ACC activity is particularly implicated in the emotional accompaniments to pain, among which distress is primary. This is demonstrated by the fact that the anticipation of pain reliably activates a subset of the same brain areas that are activated during actual sensations of pain, albeit to a lesser intensity; ACC and anterior insula are the most commonly implicated regions of overlap (Botvinick et al., 2005; Jackson, Brunet, Meltzoff, & Decety, 2006; Koyama et al., 2005; Kupers et al., 2005; Morrison, Lloyd, di Pellegrino, & Roberts, 2004; Petrovic et al., 2005; Porro et al., 2002; Singer et al., 2004; Ushida et al., 2005; Vollm et al., 2006). In fact, the intensity of experienced pain can be significantly diminished, as can accompanying ACC activity, by experimental manipulation of cognitive–affective processes such as detachment (Kalisch et al., 2005), lowered pain expectations (Koyama et al., 2005), adopting of an "other" perspective (Jackson et al., 2006), hypnosis (Rainville, Duncan, Price, Carrier, & Bushnell, 1997), and placebo (Kupers et al., 2005). The close association between changes in ACC activity and changes in pain-related affect are consistent with the proposal that ACC activity during

pain is directly linked to the subjective experience of that affect (Rainville, 2002). If this is the case, then we would expect nightmare distress to be associated with intense ACC activity. We might also even predict that the vivid images of nightmares could in some circumstance induce subjective distress to the point of subjective pain (e.g., Raymond, Nielsen, Lavigne, & Choinière, 2002) or that cognitive–behavioral interventions would be effective in alleviating such distress, via a similar ACC mechanism. All of these hypotheses are amenable to direct empirical verification using brain imaging techniques.

Social exclusion and separation distress. Early animal studies established that the same neurochemicals controlling physical pain (opioids, oxytocin, etc.) also regulate the emotional pain produced by social loss (Panksepp, Herman, Vilberg, Bishop, & DeEskinazi, 1980). For example, both morphine and endogenous endorphins alleviate separation distress in a variety of nonhuman animal species as measured by distress vocalizations (see review in Panksepp, 2003). In humans, many studies (see review in Eisenberger & Lieberman, 2004) demonstrated that enhanced sensitivity to either physical or social types of pain is accompanied by enhanced sensitivity to the other type and that augmenting or inhibiting one type can exacerbate or assuage the other type respectively. Recent brain imaging studies have supported and extended these findings by demonstrating that the neural circuitry implicated in pain sensation is also activated during both real and remembered social distress. Eisenberger and colleagues (Eisenberger & Lieberman, 2004; Eisenberger, Lieberman, & Williams, 2003) conducted brain scans while participants were deliberately excluded from participating with two other players in a collaborative computer game. ACC activity, in particular, was heightened in this exclusion condition in step with the intensity of subjectively reported distress. ACC is similarly activated when personalized scripts are used to guide healthy participants through memories of personally distressing past situations (Sinha, Lacadie, Skudlarski, & Wexler, 2004) but is paradoxically inhibited when participants in the protocol are abstinent, cocaine-dependent patients (Sinha et al., 2005). The ACC is also activated by the presentation of aversive pictures (Phan et al., 2003), facial displays of disgust (Amir et al., 2005), and threat-related distracters (Bishop, Duncan, Brett, & Lawrence, 2004; Bishop, Duncan, & Lawrence, 2004), although such static stimuli likely produce much less intense distress and ACC activation than personalized scripts (Panksepp, 2003). Although the ACC may be only one of several brain regions implicated in distress reactions, its central role is further highlighted by findings that it mediates several social and parental attachment functions, such as affiliative behavior (Hadland, Rushworth, Gaffan, & Passingham, 2003), grief following romantic separation (Najib, Lorberbaum, Kose, Bohning, & George, 2004) or death (Gundel, O'Connor, Littrell, Fort, & Lane, 2003), distress vocalizations during separation from caregivers (see review in Eisenberger & Lieberman, 2004) and maternal responses to distress vocalizations by infants (Lorberbaum et al., 2002). A recent imaging study demonstrated ACC activation during stimulation with negative social (vs. nonsocial) emotional stimuli (Britton et al., 2006). Together, these findings clearly link ACC activity to the most severe intensities of human distress that regularly surface in the themes and emotions of nightmares. Accordingly, one model (Eisenberger & Lieberman, 2004) describes ACC function as an "alarm system" that monitors discrepancies (error/conflict detec-

tion) and signals an alarm equivalent (subjective distress) when any are detected. A somewhat related model of normal dreaming (Stickgold, 2003) stipulates that the ACC monitors and detects errors during dream formation and registers these, not as distress, but as negative emotional responses.

Trait distress and gender. We suggest that an affect distress personality style underlies nightmares and other pathologies, is linked to ACC activity, and helps to explain both the gender difference (favoring women) and diverse personality differences in nightmare recall. This suggestion is supported by several types of findings. First, participants high in neuroticism, a trait characterized by the tendency to experience negative affect (Costa & McCrae, 1980) and which is more prevalent in women (Costa, Terracciano, & McCrae, 2001), demonstrate elevated ACC activation during an oddball task compared with participants who are high in either extraversion or self-consciousness (Eisenberger, Lieberman, & Satpute, 2005). Second, interindividual differences in ACC morphology are related to an affect distress personality style as well as gender (Butler et al., 2005; de Leeuw et al., 2005). As an illustration, functional magnetic resonance imaging assessment of 100 healthy young participants (Pujol et al., 2002) demonstrated not only that patterns of gyral and sulcal convolutions in the ACC are highly variable between individuals and between the left and right hemispheres within individuals but also that left–right asymmetries are very common (83% of cases) and that a right-side ACC prominence is more common in women. Further, right-side ACC surface area in both men and women is significantly related to all four components of the Harm Avoidance scale of the Temperament and Character Inventory (Cloninger, Svrakic, & Przybeck, 1993): anticipatory worry, fear of uncertainty, shyness with strangers, and fatigability. Women report overall higher Harm Avoidance scores than men, a difference that is satisfactorily explained by the observed anatomical differences in ACC surface area (Pujol et al., 2002). A similar gender difference in ACC activity is found when participants are threatened by the possibility of unannounced electrodermal stimulation; women show an increase in ACC activity, whereas men show a decrease (Butler et al., 2005). In fact, a recent meta-analysis of 65 functional neuroimaging studies found that subgenual (rostral) ACC and medial brainstem are the two brain regions most frequently activated preferentially in women during emotional stimulation (Wager, Phan, Liberzon, & Taylor, 2003). Third, ACC activation (together with amygdala activation) characterizes fear-potentiated startle (Pissiota et al., 2003), a reflex which is elevated among participants with a trait sensitivity to social rejection who are viewing stimuli portraying rejection themes (Downey, Mougios, Ayduk, London, & Shoda, 2004). This reflex is also reliably elevated among healthy participants who are viewing aversive stimuli; more intense stimuli produce the greatest level of startle potentiation (for a review, see Lang et al., 2000).

Fourth, abnormal ACC activity is linked with other emotional disorders for which affect distress is a correlate. ACC hyperactivity is observed in obsessive–compulsive patients during a neutral state; it increases during symptom provocation, and it decreases with successful treatment (see review in Talbot, 2004). Reduced dorsal ACC activity is seen in socially phobic individuals when they are anticipating delivering a public speech (Lorberbaum et al., 2004), and rostral ACC activity is diminished in highly anxious participants (Bishop, Duncan, & Lawrence, 2004), especially

when they are distracted by threat-related cues (Bishop, Duncan, Brett, & Lawrence, 2004). Associations have also been identified between ACC size and/or activity and alexithymia (Berthoz et al., 2002; Gundel et al., 2004), borderline personality disorder (Tebartz van Elst et al., 2003; Vollm et al., 2004), substance addiction, intoxication, and craving (Brody et al., 2004; see review in Goldstein & Volkow, 2002; Kilts, Gross, Ely, & Drexler, 2004) and psychopathy (Birbaumer et al., 2005). For many of these findings, gender differences in both neurophysiological and behavioral measures are again evident.

Fifth, ACC activity has been implicated in a neural circuit that is hyper-responsive to stress and mediates stress-sensitive illnesses such as asthma through immune system responsiveness (Rosenkranz et al., 2005; Tashiro et al., 2003). ACC activity during evaluation of asthma-relevant emotional (vs. neutral) stimuli is strongly correlated with inflammation responses in asthmatics after they are experimentally exposed to antigen (Rosenkranz et al., 2005). ACC activity is also correlated with both anxiety and natural killer cell activity in patients with malignant diseases (Tashiro et al., 2001).

This convergence of findings for an ACC-linked personality style characterized by fear, worry, and negative affect supports our own suggestion that abnormal ACC activity is implicated in nightmare distress. The gender differences in ACC morphology and activity observed also clearly parallel the findings described earlier demonstrating that nightmare frequency and distress are more prevalent among female participants.

Despite these findings, questions still remain as to the significance of increased vs. decreased ACC activity in different contexts. This uncertainty is due largely to the fact that functional relationships between the ACC, amygdala, and other regions have not been thoroughly evaluated. As the subsequent section demonstrates, one emerging hypothesis about ACC-amygdala interactions in PTSD is that a normal down-regulation of amygdala activity by the ACC is disrupted in PTSD patients.

PTSD distress. Evidence for the precise nature of ACC involvement in PTSD remains somewhat inconsistent. Most studies have demonstrated decreased rostral ACC activity relative to controls when patients are presented with trauma-related emotional stimuli (Bremner, 1999; Bremner et al., 1999; Lanius et al., 2001, 2003; Shin et al., 1999, 2001; L. M. Williams et al., 2006). For example, PTSD patients' ACC activity decreases when they view facial expressions of fear—a decrease that is correlated with increased trauma impact and symptomatology (L. M. Williams et al., 2006). Such findings are further supported by evidence of smaller rostral ACC volumes (Rauch et al., 2003) and hypodensity of ACC grey matter (Yamasue et al., 2003) in PTSD patients. A recent large-scale imaging study of 99 Vietnam and Gulf War combat veterans confirmed the smaller ACC volumes in PTSD patients, even when history of alcoholism, body size, cranial volume, and verbal IQ were controlled (Woodward et al., 2006). In this study, inverse correlations between PTSD symptom severity indices and ACC volumes in the order of $-.33$ to $-.34$ ($p < .001$) were reported.

On the other hand, some studies demur by demonstrating that ACC activity increases during some types of emotional stimulation (Gilboa et al., 2004; Rauch et al., 1996; Shin et al., 1997). For example, ACC activity increases when PTSD patients generate combat images but not when they view combat scenes (Shin et al.,

1997). It also increases when patients are presented with nonthreatening stimuli in an oddball paradigm (Bryant et al., 2005), prompting the latter authors to suggest that ACC hyperactivity reflects PTSD hypervigilance, whereas ACC hypoactivity occurs only when the ACC mechanism is overwhelmed by more engaging, traumatic stimuli.

On the basis of this mixed literature, some researchers have postulated that the ACC down-regulates emotion-related amygdala activity and, in the case of PTSD, fails in this regulatory function (Bremner et al., 1999; Hamner, Lorberbaum, & George, 1999; Liberzon & Phan, 2003; Pitman et al., 2001; Shin et al., 1999, 2001). The ACC hypoactivity seen in PTSD patients thus reflects an abnormal release of amygdala-mediated emotional responses in the form of hyperarousal symptoms. This suggested mechanism has been supported by many, but not all, studies (see review in Liberzon & Phan, 2003) and by a variety of evidence: (a) reduced ACC volumes in PTSD patients (Woodward et al., 2006), (b) abnormal functional connectivity between ACC and other brain regions in PTSD patients (Liberzon & Phan, 2003), (c) increased amygdala activation during fear acquisition and decreased ACC function during fear extinction in PTSD patients (Bremner, Vermetten, et al., 2005), and more generally, (d) ACC activation in healthy participants that is associated with inhibition of amygdala responses to generic threat cues (Hariri, Mattay, Tessitore, Fera, & Weinberger, 2003). Note that the amygdala down-regulation hypothesis links production of subjective distress more closely to the amygdala than to the ACC.

Nonetheless, studies demonstrating increases in ACC activation in PTSD patients seems inconsistent with the latter view (Gilboa et al., 2004; Liberzon et al., 1999; Rauch et al., 1996; Shin et al., 1997). Similarly, evidence of no functional connectivity between ACC and amygdala activity during script-driven trauma imagery questions the down-regulation hypothesis (Gilboa et al., 2004). These findings support an alternative view that ACC, rather than the amygdala, plays a primary role in producing subjective distress. Such a view is consistent with the substantial evidence reviewed above supporting an alarm function for the ACC, that is, a function that links ACC activity to pain, anticipated pain, and separation distress.

Neither hypothesis satisfactorily accounts for the divergence of findings at the present time. Consideration of three key factors in future studies may lead to a quicker resolution of the inconsistencies. One factor is the observation, reviewed briefly above, that ACC morphology and associated affect distress vary substantially as a function of gender and personality styles (e.g., harm avoidance). Because PTSD symptomatology, too, varies with gender and is typically comorbid with other personality factors and pathologies such as depression and substance abuse (which themselves are linked to ACC activity and interact with gender; e.g., Li, Kosten, & Sinha, 2005; Wu et al., 2001), more careful assessment of personality and comorbidity could clarify the great variability in PTSD findings.

A second factor concerns potential differences in function among subregions of the ACC. Several meta-analyses indicate that ACC function is sustained by rostral and caudal ACC subdivisions that are responsible, respectively, for the cognitive and emotional processing of stimuli (see review in Bush, Luu, & Posner, 2000; Vogt, Finch, & Olson, 1992). These two subdivisions mediate activity in amygdala pathways differentially during the perceptual

processing of fear stimuli (Das et al., 2005) and during the perception versus the anticipation of pain (Singer et al., 2004). A review of 107 PET activation studies (Paus, Koski, Caramanos, & Westbury, 1998) also revealed important differences in blood flow for different task parameters (e.g., auditory, visual) and response requirements (e.g., arm or eye movements) as a function of rostral/caudal, subcallosal/supracallosal, and limbic/paralimbic divisions of the ACC. As the resolution of brain scanners is increasingly enhanced, the relevance of these distinctions is increasingly recognized. However, the anatomical distinctions are still not reported in all studies. In the modeling of nightmare formation, these ACC anatomical distinctions may prove key to identifying the complex cognitive and affective processes responsible for frequent and/or distressing nightmares. The rostral–caudal distinction, in particular, provides a neurophysiological basis for considering nightmare distress as both a cognitive and an affective phenomenon and for explaining the apparent success of cognitive–behavioral treatments for nightmares (Germain & Nielsen, 2003a; Krakow, Hollifield, et al., 2001).

A third key factor is evidence linking ACC activity to a variety of other processes that may seem only indirectly pertinent to distress and PTSD symptoms but that may nevertheless overlap in various ways. For example, the ACC is implicated in executive and attentional functions such as the detection of conflicting stimuli (Weissman, Gopalakrishnan, Hazlett, & Woldorff, 2005)—especially unexpected conflicts caused by emotional stimuli (Bishop, Duncan, & Lawrence, 2004)—and the linking of reward-related information to appropriate response alternatives (Z. M. Williams, Bush, Rauch, Cosgrove, & Eskandar, 2004). Accordingly, researchers have speculated that ACC activity underlies a variety of subjective experiential qualities such as a sense of mental effort (Botvinick, Cohen, & Carter, 2004), effort associated with the cognitive induction of emotions (Phan et al., 2002), a sense of voluntary control over thoughts and feelings (Posner & DiGirolamo, 2000), and the adaptive signaling of when to try something new in the face of previously unproductive behaviors (M. A. Williams & Mattingley, 2004). The relation of such features to affect distress, to the distress personality, and to the multiplicity of ACC subregions requires much further experimentation.

Implications for nightmares. In sum, the ACC is integral to the AMPHAC network by virtue of its mediation of affect distress. This role is part of a more general personality factor that is expressed during both nightmares and waking-state symptoms. Over the long term, ACC dysfunction may gradually amplify subjectively felt distress, ladening fear memories with distressing response elements and contributing to our postulated affect distress personality trait—a process roughly equivalent to emotional kindling. Any of a number of alterations in rostral or caudal ACC activity may shape this progressive increase in trait distress during nightmares. See the following as illustration.

1. ACC activity may produce excessive activity in the alarm system as a result of elevated detection of stimulus or response discrepancies. Nightmares might therefore contain emotions linked to anomalous stimulus elements.

2. Dorsal ACC dysfunction might increase subjective distress by decreasing the strength and functionality of attachment images. In nightmares, distress might thus occur as a result of attachment figures rejecting, abandoning, or neglecting the self-figure.

3. Rostral ACC dysfunction might facilitate the development of distress by failing to maintain focused attention on fearful emotions. Nightmares might thus be characterized by emotional avoidance scenarios.

4. Dysregulation of the ACC's role in regulating physical and social pain distress may lead to nightmares in which pain is prominent (Nielsen et al., 1993); this has been observed in the case of traumatic burn patients (Raymond et al., 2002).

5. The ACC may fail to resolve attentional conflicts inherent in the activation of fear memories, perhaps by failing to direct attention to incompatible stimulus elements that could modify the structure. Within nightmares, this could manifest as a chronic attention on one's own behaviors and reactions and a disregard for novel stimulus elements of the nightmare scene. Alternatively, altered ACC activation could produce a sense of heightened effort in, or loss of control over, emotional events. In nightmares, this could produce feelings of struggle (e.g., unable to escape, ineffectual fighting) and/or helplessness (e.g., being chased) that are so familiar to some typical nightmare scenarios. Dorsal ACC dysfunction could also prevent the initiation of new, more productive response elements that are presumably enabled by MPFC gating. Nightmares might thus be dominated by intense, recurrent, and unsuccessful action sequences.

6. Any of the preceding changes in ACC function may ultimately impede what is widely thought to be the emotion-regulation function of dreaming (Braun et al., 1997; Cartwright, 1991; Kramer, 1993; Maquet et al., 1996; Nofzinger et al., 1997), leading to an exacerbation of daytime emotional distress.

Other Pertinent PTSD Findings: Hyperarousal

Converging findings also suggest that disruptions of the AMPHAC network underlie different types of anxiety disorders, such as obsessive–compulsive disorder (implicating primarily ACC), panic disorder (implicating hippocampus), social anxiety disorder (implicating amygdala), and others (see review in Rauch, Shin, & Wright, 2003). It is thus feasible that a single, integrated model may emerge that could account for a wider spectrum of anxiety disorders spanning waking and sleep states.

One cross-state concept that much research supports is that a trauma-induced disturbance in the AMPHAC network is the expression of PTSD hyperarousal symptoms during both wakefulness and sleep. Waking-state hyperarousal symptoms are relatively easily detected by various self-report instruments such as the Impact of Event Scale (Weiss & Marmar, 1997), by 24-hr monitoring of elevated heart rate and diastolic blood pressure (Muraoka, Carlson, & Chemtob, 1998), or by measures of elevated autonomic and electromyographic responses to trauma-related cues and to startling stimuli more generally (for reviews, see Harvey et al., 2003; Orr & Roth, 2000; Pitman, Orr, Shalev, Metzger, & Mellman, 1999). Hyperarousal symptoms during sleep are less obvious. PTSD-related cardiovascular anomalies have been observed in the form of elevated ratios of low over high frequencies of spectral power in the electrocardiogram during REM sleep (Mellman, Knorr, Pigeon, Leiter, & Akay, 2004). Heightened physiological reactivity is also apparent in the form of more frequent awakenings (Germain & Nielsen, 2003b), longer wake time after sleep onset scores (Germain & Nielsen, 2003b; Woodward et al., 2000), and increased motor activity and rapid eye movement activity during

REM sleep (Harvey et al., 2003; Orr & Roth, 2000; Pitman et al., 1999). PTSD patients with trauma-related nightmare complaints also exhibit higher REM and NREM sleep respiration rates than do non-PTSD controls (Woodward, Leskin, & Sheikh, 2003). On the other hand, the significantly lower levels of movement time during sleep in PTSD patients (Woodward, Leskin, & Sheikh, 2002) seem anomalous. However, reduced movement time may be analogous to the defensive freezing response seen in other species (Woodward et al., 2002), where it is a strongly conserved behavioral response of the amygdala (LeDoux, 1998).

In sum, perturbations in the components of the AMPHAC network likely produce the various hyperarousal symptoms observed during wakefulness and sleep. These perturbations may be critical in the formation of both posttraumatic and nontraumatic nightmares.

Neuropathology of Nontraumatic Nightmares

Although few studies can be brought directly to bear on our suggestion that nontraumatic nightmares depend upon dysfunctions in the same emotion-processing brain network that underlies posttraumatic nightmares, the available findings are consistent with this suggestion. The study upon which most current thinking about nontraumatic nightmares is based (C. Fisher et al., 1970)—which was published 35 years ago and which remains questionable because of the purported inclusion of some traumatized and borderline psychotic patients (Keefauver & Guilleminault, 1994)—nevertheless demonstrates that nightmares are generally associated with varying levels of autonomic activation during REM sleep (primarily) and Stage 2 sleep (secondarily). Such activation is consistent with the notion that the amygdala is as hyper-responsive and under-regulated during nontraumatic nightmares as it is during posttraumatic nightmares. However, the autonomic increases in heart rate and blood pressure reported in C. Fisher et al.'s (1970) study and in a subsequent study (Nielsen & Zadra, 2005) are not prominent. For example, Fisher et al. found minimal heart rate increases in only a subset of participants, and Nielsen & Zadra (2005) noted only a mean of 6 beats/min increase over the last 3 min of physiologically recorded nightmares and no significant increases in respiration or REM density. Thus, if the amygdala is abnormally activated during nontraumatic nightmares, the consequences of this activation must be modest compared with that seen in PTSD.

Although no brain imaging studies of nontraumatic nightmare sufferers could be located, several studies indicate that amygdala activation is highly characteristic of REM sleep (Maquet et al., 1996; Nofzinger et al., 1997, 2004), the stage from which most nightmares arise. Imaging studies also indicate that REM sleep is characterized by increases in the other components of the AMPHAC network including MPFC (Buchsbaum, Hazlett, Wu, & Bunney, 2001; Nofzinger et al., 2004), ACC (Braun et al., 1997; Buchsbaum et al., 1989, 2001; Maquet et al., 1996; Nofzinger et al., 1997, 2004), and the hippocampal complex (Braun et al., 1997; Nofzinger et al., 1997, 2004). Additionally, an early PET study of healthy participants (Gottschalk et al., 1991) 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. That elements of the proposed network are active during normal REM sleep indicates that a physiological

infrastructure for emotional experience is available during normal dreaming and may account for much of the (normal) dysphoric emotion occurring at this time. It is also likely that disruption of activity in these regions is even greater when either nontraumatic or posttraumatic nightmares are present. The finding that REM density is positively correlated with diurnal affect intensity (Monroe, Simons, & Thase, 1992; Nofzinger et al., 1994) as well as the converse finding that attenuated REM activity is associated with down-regulated waking emotional arousal (Buysse et al., 2001; Nofzinger et al., 1994; Thase et al., 1994) strongly suggest that phasic REM activity may be a marker of stress adaptation and emotional processing (Germain et al., 2003). Although these studies did not specifically examine dreams, they are nonetheless consistent with this notion.

Neuropsychological evidence from brain-lesioned patients (Solms, 1997) also supports this contention, demonstrating a link between tempero-limbic brain regions and frequent nightmares of both recurring and nonrecurring types. Eight of Solms's (1997) 9 patients who reported recurring nightmares were found to have temporal or fronto-temporal lesions, including in some cases, the hippocampus or ACC. The incidence of epilepsy was also higher in this group. Substantial overlap was also noted between the group of recurrent nightmare sufferers without epilepsy and those experiencing a disturbing type of dream referred to as "dream–reality" with frequent nightmares commonly reported by the latter group. Cases of dream–reality confusion were also habitually associated with limbic lesions (9 of 10 patients). Furthermore, 6 of these 9 patients had lesions affecting MPFC or ACC or both. Finally, for 17 patients in Solms' cohort who reported nonrecurrent nightmares, a significant association with limbic system involvement was also noted. Thus, neuropsychological evidence supports the notion that disruption of the AMPHAC network facilitates nightmare production.

Summary and Conclusion

Over the past 3 decades, scientific understanding of nightmares has progressed in several respects. Nightmares are the most commonly experienced parasomnia and frequent nightmares, usually defined as once per week or more, are quite prevalent in nonclinical populations, particularly children and adolescents. Both nightmare prevalence and frequency are associated with psychological dysfunction, trauma exposure, and waking stressors of various kinds. Nightmares are also more commonly reported by women than by men at all ages. Although this consistent gender difference is still not well understood, several explanations (symptom reporting, recall bias, sensitivity to inner experience, ruminatory thinking, emotion regulation) are consistent with the suggestion that gender differences in affect load or affect distress factors are involved.

Prevalence studies are hampered by a number of methodological pitfalls, including the use of retrospective measures, an over-reliance on college student samples, imprecise definitions, a failure to use structured clinical interviews or discriminate among nightmare subtypes, and neglecting to assess such critical covariates as current life stress, age, and history of trauma exposure. In the last review on nightmares, written over 35 years ago, Hersen (1972) noted that confounds inherent in the almost exclusive reliance on questionnaire and self-report measures of nightmare behavior

greatly limited the knowledge base and warranted the need for more objective measures in future work "if a viable nomological network for the nightmare personality is to be established" (p. 41). Although much progress has been made in using prospective measurements, this goal has not yet been fully realized. Hersen's lament remains particularly salient with respect to the near absence of carefully controlled clinical investigations utilizing behavioral and physiological measures. In addition, longitudinal studies that track waking and sleep behaviors over time would be invaluable for clarifying the pathogenesis of nightmares, particularly in populations at increased risk for disturbed dreaming, such as individuals who are high in trait negative affect, display schizotypal features, were recently traumatized, are under considerable life stress, are young, or are female. Similarly, shorter term, repeated measures studies may help to uncover important chronobiological patterns implicated in nightmare production.

Considerable debate still exists over the nosological boundaries of what constitutes a nightmare. The *DSM–IV* and *ICSD–2* differ in whether they consider nightmares to be fear predominant (*DSM–IV*) or whether dysphoric emotions of many kinds may be implicated (*ICSD–2*). Although both manuals consider nightmares to provoke abrupt awakenings, considerable phenomenological similarities exist between disturbing dreams that awaken the sleeper and those that do not but are recalled at a later time (bad dreams). Studies have begun to investigate this issue, but considerable work remains to determine whether these are basically similar subtypes of disturbed dreaming or whether bad dreams may belie a more successful biological strategy for emotion regulation. Finally, although a distinction has been drawn between posttraumatic and nontraumatic nightmares, it remains largely unknown whether these subtypes share common pathogenic mechanisms or even co-occur in the same individuals.

Perhaps the most robust finding in the experimental literature is that nightmare frequency is associated with elevated psychopathology, including higher rates of neuroticism, anxiety, depression, dissociation, substance usage and withdrawal, behavioral health problems, schizophrenia-spectrum disorders, and PTSD. With the exception of the latter category, the mechanisms linking nightmares with poorer psychological functioning are not well understood. Nightmares do not appear to predict specific psychopathology subtypes; rather, they seem to be associated with poor psychological well-being more generally. It is difficult to determine cause and effect relationships in this domain, especially in the absence of controlled longitudinal studies. One promising development is the association of nightmares with affect distress. Recent evidence suggests that links between nightmares and psychopathology may be largely mediated by an affect distress dispositional dimension rather than by nightmare frequency per se, particularly for psychological disorders marked by high levels of negative affect (e.g., anxiety and depression). More research is needed to clarify the nature and mechanisms of affect distress and its links to such waking psychological factors as coping, cognitive attributional style, ruminative thinking, cognitive biases, and other personality factors. Conversely, more research is also needed to validate the hypothetical construct of affect load, which currently cannot be measured other than in its indirect link to nightmare frequency. This problem also implicates more careful assessment of our proposed fear extinction function of dreaming, which is postulated to regulate affect load.

A chronic lack of research attention to pathophysiological features of nightmares has hindered the development of nightmare production models. Nonetheless, a recent surge of research on the brain correlates of emotion, PTSD, and normal human sleep provides a solid foundation for the neurocognitive modeling of nightmare formation. We propose a model that unites explanatory concepts at a neural level (i.e., a network of limbic and forebrain regions underlying emotion expression and representation) and a cognitive level (i.e., a dream production system that transforms fear memories into nightmare imagery). Disruption of specific processes at these levels can account for a variety of specific features commonly observed in nightmare imagery, such as, lack of emotional control, bizarre features, or replay of traumatic memories. In emphasizing that nightmares reflect perturbations of a presumed fear extinction function of dreaming, the model unites normal dreaming with both posttraumatic and nontraumatic nightmares. In specifying additional situational and dispositional factors (affect load and affect distress respectively), the model is able to distinguish among nightmare subtypes, for example, bad dreams versus nightmares, low versus high distress nontraumatic nightmares, posttraumatic versus nontraumatic nightmares. Finally, in specifying neurocognitive mechanisms of emotion regulation, such as amygdala involvement and affect distress, the model integrates emotional processes from the waking state with those of sleep, thus opening the door to future research investigating links between waking and dreaming mechanisms of nightmare production and the individual differences that contribute to both nightmares and a range of other fear-based symptoms.

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