

fear conditioning training selectively suppressed REM for 1 to 2 hours post-training (Sanford et al. in press). Adrien et al. (1991) utilizing a similar procedure reported a significant decrease in REM and no REM rebound during the subsequent 24 hours. From our perspective, then, it seems that a learning paradigm in which REM is selectively suppressed would be problematic for theories that REM is necessary for retaining the same learning. Interesting to note, Adrien et al. (1991) also reported an increase in NREM1 and we found an increase in NREM percent. These findings are consistent with suggestions that NREM may promote memory consolidation (e.g., Fowler et al. 1973; Wilson & McNaughton 1994).

The striking electrophysiological phenomena of REM are especially beguiling, leading researchers to search for special meaning or relevance for their occurrence. This has led to the inbuilt assumption for many theorists that neural activity specific to REM, as opposed to NREM or sleep in general, somehow aids in memory consolidation. That same activity would seem to us to pose potential problems for the processing of previous learning. For reasonably accurate memories to be formed, one would expect that reactivated traces (if such occur) would need to be free from internal and external interruptions. Alterations in hypothalamic function and the highly activated brain, as described in the previous section, would present possible sources of internal interference. In addition, brain processing may be almost as susceptible to external influences during REM as during wakefulness. Evoked potentials are similar during REM and wakefulness. This finding (among others) led to Llinás and Paré's (1991) suggestion that brain processing in REM and wakefulness is the same except for the elevated sensory threshold during REM. Actually, we demonstrated that cats in REM-A may behaviorally orient to simple external auditory stimuli of varying intensities in much the same way they do in wakefulness (Morrison et al. 1995). This suggests even more similarity between the way information is processed in wakefulness and REM. Indeed, these similarities do not rule out the possibility for rudimentary (S-R type) learning during REM itself, but in no way suggest that memory would be promoted. If so, such learning could pose problems for the idea that memory consolidation takes place during REM. According to interference theory, the formation of associations in the interval between learning and recall may be a factor in forgetting (Hulse et al. 1980).

One of the major problems we see with ascribing functional significance to neural activity in REM is the dramatically altered central orchestration of neural events. It seems to us that even theories that deal with specific processes must take into consideration the condition of the organism as a whole. In wakefulness, an extremely activated brain, irregular respiration, bouts of tachycardia, and twitching muscles coupled with potential extraneous interference from the environment would hardly be considered optimal for memory formation. We see no reason to think that some special quality of REM makes this same combination of factors conducive for consolidating information previously learned in another state.

Post-traumatic nightmares as a dysfunctional state

Tore A. Nielsen and Anne Germain

Sleep Research Center, Hôpital de Sacré-Coeur de Montréal, Montréal, Québec, Canada H4J 1C5; ^aPsychiatry Department, Université de Montréal, Québec, Canada. t-nielsen@crhsc.umontreal.ca

Abstract: That PTSD nightmares are highly realistic threat simulations triggered by trauma is difficult to reconcile with the disturbed, sometimes debilitating sleep and waking functioning of PTSD sufferers. A theory that accounts for fundamental forms of imagery other than threat scenarios could explain the selection of many more adaptive human functions – some still pertinent to survival today. For example, interactive characters, a virtually ubiquitous form of dream imagery, could be simulations of at-

tachment relationships that aid species survival in many different ways. [REVONSUO]

PTSD as a dysfunctional dreaming state. The threat simulation theory would appear to suggest that nightmares, as exemplary threat simulations, are highly functional, for example, “nightmarish dreams are not ones that failed to perform their function, but, by contrast, prime examples of the kind of dreams that fully realize their biological function” (REVONSUO, sect. 2.2.8). Such a notion would be clearly at odds with the predominant psychiatric view that considers nightmares to be dysfunctional, as embodied in the Nightmare Disorder and Post-traumatic Stress Disorder categories of the DSM-IV (American Psychiatric Association 1994).

However, nightmare functionality in this model is limited primarily to a past, evolutionary function, not to a current regulatory function. REVONSUO likens nightmares to natural variations in a biological defense system such as the immune system. Like immune responses, which are sometimes overactive in susceptible, hypersensitive, individuals (e.g., allergy sufferers), acute or chronic nightmare sufferers may suffer merely from a “harmful side effect” of the threat simulation system – much like an allergic condition – but a side effect whose evolutionary costs (nightmare distress) nevertheless did not outweigh its benefits (survival). Further, such side effects are likely transmitted genetically, as natural selection of such variations would require. Thus, one cannot necessarily argue that the distress and impairment of Nightmare Disorder constitute evidence against the biological function of nightmares. Rather, they may simply be an inherited “cost” of the evolutionary necessity to avoid threat. This argument holds to the extent that Nightmare Disorder is inherited; there is at present only limited evidence supporting this possibility (Hublin et al. 1999a).

On the other hand, nightmares induced by trauma are much more directly pertinent to the predictions of the theory because they are less likely to be due to genetic dispositions than are idiopathic nightmares and because their severity is more likely to be due to trauma severity than to inherited factors (see Connor & Davidson 1997 for review). Rather, future PTSD susceptibility is increased by past exposures to trauma, particularly violent trauma; the more numerous the past exposures, the higher the likelihood that a future trauma will trigger PTSD (Breslau et al. 1999). Thus, if there is evidence that PTSD nightmares are associated with signs of dysfunctional adaptation to the environment, then the threat simulation theory is weakened.

REVONSUO acknowledges that PTSD nightmares do not necessarily facilitate adaptation to the trauma that incited them. The nightmares of war veterans with PTSD are not adaptive because their content does not deal with the real threats of the battlefield: “There are few such skills among human threat avoidance programs whose rehearsal would be of much help in an environment where one may at any moment get killed by shrapnel, the invisible sniper's bullet, nerve gas, hidden land mines . . . and so on” (REVONSUO, sect. 6.3, para. 3). It appears that only current threats that correspond to ancestral threats may benefit from the “rehearsals” of threat simulation. Nonetheless, one may question this reasoning in the case of war trauma (where a strategy of “combat avoidance at any cost” could well help to save a soldier's life), as well as for rape and assault trauma (where avoidance of the perpetrator and/or the crime scene could well prevent worse injuries), for motor vehicle trauma (where avoidance of driving could enhance survival), or for any number of other, somewhat predictable, trauma. It is not clear why these types of trauma would *not* benefit from the threat simulations proposed by the theory whereas other similar, or even less predictable ancestral types of trauma, such as natural disasters, would.

Furthermore, PTSD may well be a dysfunctional, if not completely debilitating condition, which can hinder rather than facilitate adaptation. REVONSUO does not review a rather large body of evidence describing the dysfunctional aspects of PTSD. He thus leaves the impression that PTSD would not be likely to be an impediment to the goal of survival. It is our impression, however, that the accumulating mass of evidence characterizing PTSD as

dysfunctional supports the notion that it may work counter to the evolutionary pressures described by REVONSUO. First, and perhaps most obviously, the nightmares of PTSD can often disrupt sleep and engender dysfunctional reactions in the daytime. In severe cases, such reactions can be worse than those induced by Nightmare Disorder. Moreover, many studies have found abnormalities in REM sleep latency, REM sleep amount, and REM density (see Benca 1996, for review), evidence favoring the hypothesis that PTSD is a function of disturbed REM sleep (Ross et al. 1989). Studies of PTSD sufferers have also found anomalies of breathing (Krakow et al. 2000), arousal regulation (Mellman 1997), sleep efficiency (Mellman et al. 1997), body and limb movements (Mellman et al. 1995), and NREM sleep awakenings (Kramer & Kinney 1988), among others. These, and numerous studies assessing perturbations in waking state variables as diverse as memory (Moradi et al. 1999; Wolfe & Schlesinger 1997), visual imagery (Bryant & Harvey 1996), startle (Orr et al. 1997), P300 (Metzger et al. 1997), and corticotrophin-releasing hormone (Baker et al. 1999) all indicate severe abnormalities in PTSD sufferers. Such global perturbations of key cognitive and physiological systems would seem to decrease an individual's chances of survival significantly. Whereas the threat simulation theory would predict that PTSD nightmares are evolutionary remnants that are, at worst, non-functional in nature, the evidence together suggests that they reflect a more generally disturbed, dysfunctional state that is induced by traumatic, much more than genetic, factors.

The polyvalence of successful evolution. His limited characterization of dreaming as threat simulation leads REVONSUO to consider only one specific adaptive function pertinent to human evolution. For example, the evolutionary advantage afforded by dreaming dealt with "*behavioral* strategies to avoid contact with such animals and to escape or hide if attacked by them" (sect. 3.4.2.1, para. 4, emphasis added). Presumably, detouring, running fast, hiding, and the like were the behaviors that gave humans a reproductive edge in this case. However, in prehistoric times there were also naturalistic events that led to the selection of highly advanced, cognitive, social, and emotional skills that were not necessarily organized around threat. Why were such skills also not simulated during dreaming so that waking-state adaptation could be facilitated on several fronts at once?

Such a notion seems more consistent with the wide variety of very common themes and structures seen in dream reports (see commentary by Germain et al. this issue). In fact, it could be argued that any dream content with a high overall prevalence is a candidate for supporting a biological function analogous to that of threat simulation. For instance, the observation that interactive character imagery is virtually universal to dreaming could lead forthright to a theory of dreaming as simulation of attachment relationships. Attachment relationships (Bowlby 1969) are also fundamental to survival and may have been as essential to threat mitigation as were the behavioral strategies of running from predators and disasters. Strong interpersonal bonds could have ensured strong tribal structures which, in turn, could have enabled organized defenses against predators and cooperative problem-solving skills more generally. Perhaps more important, such a socio-emotional function for dreaming would still have clear adaptive significance for dreams occurring today. For example, family and group cohesion remain essential ingredients in many aspects of health and survival (e.g., Albert et al. 1998; King 1997).

Similar arguments might be made for different ubiquitous classes of dream imagery such as self-imagery and place-imagery. For example, self-imagery may facilitate functions related to ego and self-state development (Fiss 1986) or the learning of new motor competencies; place-imagery may facilitate functions related to spatial learning and orientation (Winson 1993). All such functions may have evolved much in the way that REVONSUO describes for threat perception and avoidance, with the important difference that these are more polyvalent cognitive and socio-emotional functions that are pertinent to the continuing evolution of our species today.

Insights from functional neuroimaging studies of behavioral state regulation in healthy and depressed subjects

Eric A. Nofzinger

Western Psychiatric Institute and Clinic, Pittsburgh, PA 15213.
nofzinger@msx.upmc.edu

Abstract: New data are presented showing excellent replicability and test-retest reliability of REM sleep findings from functional brain imaging studies in healthy subjects on which newer brain-based models of human dreaming have been constructed. Preliminary region-of-interest findings related to bottom-up versus dissociable brain systems mediating REM sleep and dreaming are also presented.

[HOBSON ET AL.; SOLMS]

The field of dream research is indebted to the efforts of each of these groups of investigators in their tireless efforts to formulate synthetic models of brain function that underlie the experience of dreaming. SOLMS has provided an intriguing challenge to the basic conceptualization of dreaming as a bottom-up phenomena and the work of HOBSON ET AL. reviews an astonishing array of preclinical, experiential, and cognitive neuroscience data in their most recent formulation of a brain-state model of consciousness. I can only add a few observations from our functional brain imaging studies across the behavioral states of waking, NREM, and REM sleep in healthy and depressed subjects that may have relevance to these areas of inquiry (Nofzinger et al. 1997; 1998; 1999; 2000).

A concern in human brain imaging studies of sleep is whether the findings are replicable both across and within subjects. This is important, since isolated disparate findings should not direct models of brain function as conceptualized by each of these groups of authors. This is an appropriate concern, since most studies have relied on statistical methods involving thousands of statistical comparisons across all brain pixels in relatively small sample sizes. Our group has now replicated in an independent group of four subjects our original findings of brain structures that have increased relative glucose metabolism in REM sleep when compared with waking. Additionally, in the new sample, we performed a test-retest reliability study in which the waking to REM sleep

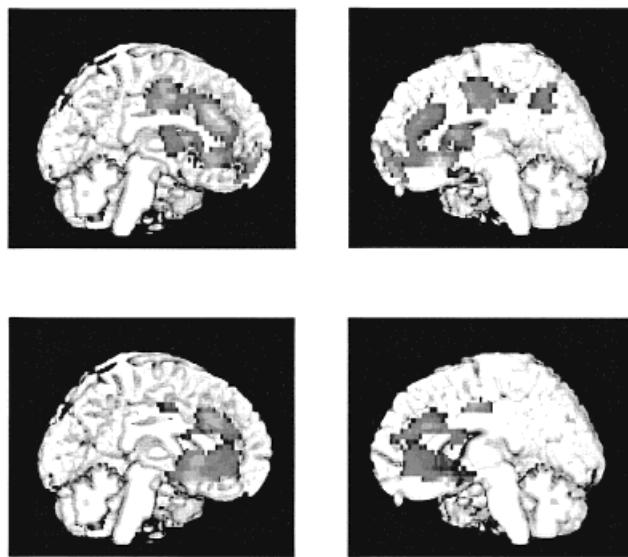


Figure 1 (Nofzinger). Bilateral mid-sagittal sections showing REM sleep minus wake activations. Two figures on top demonstrate regions activated in four healthy controls at each of two time-points separated by 12 weeks. Two figures on bottom demonstrate regions activated in six independent healthy subjects from a prior study.