Table 1. *Commentators for special sleep and dreams issue*

	Target article authors				
Commentators	Hobson et al.	Solms	Nielsen	Vertes & Eastman	Revonsuo
Antrobus, J. S.	JAH	MS	TAN		AR
Ardito, R.B.					AR
Bednar, J.A.		MS		RPV	AR
Blagrove, M.	JAH	MS	TAN	RPV	AR
Borbély, A.A. & Wittmann, L.	JAH	MS	TAN	RPV	AR
Born, J. & Gais, S.			TAN	RPV	
Bosinelli, M. & Cicogna, P.C.			TAN		
Cartwright, R.	JAH	MS		RPV	AR
Cavallero, C.			TAN		
Chapman, P. & Underwood, G.	JAH				AR
Cheyne, J.A.					AR
Cipolli, C.				RPV	
Clancey, W.J.	JAH	MS	TAN	RPV	AR
Coenen, A.		MS	TAN	RPV	
Conduit, R., Crewther, S.G. & Coleman, G.	JAH	MS	TAN	RPV	
Domhoff, G.W.	JAH	MS	TAN		AR
Doricchi, F. & Violani, C.		MS			
Feinberg, I.	JAH	MS	TAN	RPV	
Fishbein, W.				RPV	
Flanagan, O.	JAH	MS	TAN		AR
Franzini, C.		MS	TAN		
Germain, A., Nielsen, T.A., Zadra, A. & Montplaisir, J.					AR
Gottesmann, C.	JAH	MS	TAN	RPV	AR
Greenberg, R.	JAH	MS	TAN	RPV	AR
Greene, R.W.	JAH				
Gunderson, K.	JAH				AR
Hartmann, E.	JAH	MS	TAN		
Herman, J.	JAH				
Hobson, J.A.		MS			
Humphrey, N.					AR
Hunt, H.T.	JAH	MS	TAN	RPV	AR
Jones, B.E.	JAH			RPV	
Kahan, T.L.	[JAH]	MS	TAN		
Khambalia, A., Shapiro, C.M.					
Kramer, M.	JAH	MS	TAN		AR
Krieckhaus, E.E.					AR
LaBerge, S.	JAH	MS			
Lehmann, D. & Koukkou, M.	JAH	MS	TAN		

Table 1. *Commentators for special sleep and dreams issue*

	Target article authors				
Commentators	Hobson et al.	Solms	Nielsen	Vertes & Eastman	Revonsuo
Levin, R.					AR
Lydic, R. & Baghdoyan, H.A.	JAH				
Lynch, G., Colgin, L.A. & Palmer, L.				RPV	
Mancia, M.	JAH				
Marczynski, T.J.	JAH	MS	TAN	RPV	AR
Mazzoni, G.				RPV	
Mealey, L.					AR
Montangero, J.					AR
Moorcroft, W.H.	JAH	MS	TAN	RPV	AR
Morgane, P.J. & Mokler, D.J.	JAH	MS		RPV	
Morrison, A.R. & Sanford, L.D.	JAH	MS	TAN	RPV	AR
Nielsen, T.A. & Germain, A.					AR
Nofzinger, E.A.	JAH	MS			
Occhionero, M. & Esposito, M.J.		MS			
Ogilvie, R.D., Takeuchi, T. & Murphy, T.I.	[JAH]	MS	TAN	RPV	
Pace-Schott, E.F.			TAN		
Pagel, J.F.	JAH	MS	TAN	RPV	AR
Panksepp, J.	JAH	MS	TAN	RPV	AR
Perry, E.K. & Piggott, M.A.	JAH	MS			
Peterson, J.B. & DeYoung, C.G.		MS			AR
Portas, C.M.	JAH				
Porte, H.S.			TAN		
Revonsuo, A.	JAH		TAN	RPV	
Rotenberg, V.S.		MS	TAN	RPV	AR
Salin-Pascual, R., Gerashchenko, D. & Shiromani, P.J.		MS			
Salzarulo, P.			TAN		
Schredl, M.	JAH	MS	TAN	RPV	AR
Shackelford, T.K. & Weekes-Shackelford, V.A.					AR
Shapiro		MS			
Shevrin, H. & Eiser, A.S.	JAH	MS	TAN		AR
Siegel, J.M.				RPV	
Smith, C. & Rose, G.M.				RPV	
Solms, M.			TAN		
Steriade, M.	JAH		TAN		
Stickgold, R.			TAN	RPV	
Thompson, N.S.					AR
Vogel, G.W.	JAH	MS	TAN		
Wichlinski, L.J.					AR
Zadra, A. & Donderi, D.C.	JAH				AR

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The case against memory consolidation in REM sleep

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Abstract: We present evidence disputing the hypothesis that memories are processed or consolidated in REM sleep. A review of REM deprivation (REMD) studies in animals shows these reports to be about equally divided in showing that REMD does, or does not, disrupt learning/memory. The studies supporting a relationship between REM sleep and memory have been strongly criticized for the confounding effects of very stressful REM deprivation techniques. The three major classes of antidepressant drugs, monoamine oxidase inhibitors (MAOIs), tricyclic antidepressants (TCAs), and selective serotonin reuptake inhibitors (SSRIs), profoundly suppress REM sleep. The MAOIs virtually abolish REM sleep, and the TCAs and SSRIs have been shown to produce immediate (40–85%) and sustained (30–50%) reductions in REM sleep. Despite marked suppression of REM sleep, these classes of antidepressants on the whole do not disrupt learning/memory. There have been a few reports of patients who have survived bilateral lesions of the pons with few lingering complications. Although these lesions essentially abolished REM sleep, the patients reportedly led normal lives. Recent functional imaging studies in humans have revealed patterns of brain activity in REM sleep that are consistent with dream processes but not with memory consolidation. We propose that the primary function of REM sleep is to provide periodic endogenous stimulation to the brain which serves to maintain requisite levels of central nervous system (CNS) activity throughout sleep. REM is the mechanism used by the brain to promote recovery from sleep. We believe that the cumulative evidence indicates that REM sleep serves no role in the processing or consolidation of memory.

Keywords: antidepressant drugs, brain stem lesions; dreams; functional imaging; memory consolidation; REM deprivation; REM sleep; theta rhythm

1. Introduction

Although its origin is difficult to establish precisely, the view that memories are processed and consolidated in sleep, or specifically in REM sleep, dates back at least to the report of Jenkins and Dallenbach (1924) claiming that human recall improves following an intervening period of sleep. There was intense interest in the possible role of sleep in memory in the late 1960s to the 1980s as evidenced by the wealth of scientific papers on animals (and to lesser extent on humans) devoted to this issue. The position that memories are consolidated in REM has been championed by, among others, Pearlman (Pearlman 1971; 1978; 1979; Pearlman & Becker 1973), Fishbein (Fishbein 1970; 1971; Fishbein & Gutwein 1977; Gutwein & Fishbein 1980a; 1980b); Hennevin and colleagues (Bloch et al. 1979; Hars et al. 1985; Hennevin et al. 1995b; Leconte et al. 1974), and Smith (1985; 1995; 1996; Smith & Butler 1982; Smith & Kelly 1988; Smith & Lapp 1991; Smith & Rose 1996; 1997).

There was a marked decline in the number of studies devoted to this area beginning about the mid-1980s. As discussed below, the principal reason for this fall-off was that on balance the early work failed to convincingly demonstrate a relationship between sleep and memory. There were as many studies that failed to describe a link between sleep and memory as those that claimed such a relationship (Horne 1988; Horne & McGrath 1984; McGrath & Cohen 1978; Smith 1985).

There has been a renewed interest in the role of sleep and memory stemming in part from two complementary articles that appeared in *Science* in 1994: one by Wilson and McNaughton (1994) on rats and the other by Karni et al.

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(1994) on humans. In a follow-up to a study by Pavlides and Winson (1989), Wilson and McNaughton (1994) reported that ensembles of hippocampal "place" cells tend to repeat patterns of activity of waking in subsequent episodes of slow wave sleep (SWS). Karni et al. (1994) showed that improvement on a visual task in humans depended on REM sleep. The two studies supported the view that memories are consolidated in sleep. It is interesting to note that, propelled by these reports, this area reached national public attention in the United States when Jonathan Winson and Matt Wilson appeared on the Charlie Rose television program explaining and promoting their shared belief that sleep is vital for memory consolidation.

This area has recently received a further boost from Allan Hobson and colleagues who have recently come out in favor of the hypothesis that memories are consolidated in REM sleep (Stickgold et al. 2000b). This recent position seems very much at odds with their earlier proposal, termed the activation-synthesis hypothesis (Hobson 1988b; Hobson & McCarley 1977), claiming that dreams (the cognitive component of REM sleep) represent the best cognitive fit (synthesis) to the undifferentiated and random action (activation) of the brain stem on the forebrain, and as such would have little value to the organism and presumably would not need to be remembered.

As indicated by our title, we do not subscribe to the view that memories are consolidated in REM sleep. This target article evolved from an earlier piece by Vertes (1995) which appeared as part of a series in *Sleep Research Society Bulletin* on the topic of sleep and memory. In the same series, Hennevin et al. (1995a), supporters of a role for sleep in memory consolidation, acknowledged why others may be skeptical of this position. They stated:

> The hypothesis of memory processing in sleep has always had to face criticism both from people working in the field of sleep, who predominantly consider that sleeping serves more basic biological functions, and from people in the field of learning and memory, who do not easily accept the idea that information processing can take place in a non-conscious state.

As researchers involved in both sleep (Vertes 1984; 1990) and memory work (Vertes 1986a; Vertes & Kocsis 1997), we remain skeptical on both counts, largely for the reasons put forth by Hennevin et al. (1995a); that is, sleep involves basic biological functions and memory requires consciousness.

2. Background

Memory consolidation refers to neural processing that occurs after information is initially registered, which contributes to its permanent storage in memory (Nadel & Moscovitch 1997). As mentioned, several reports appeared in the 1970s exploring the possible role of sleep in memory consolidation. These studies were of two basic types: (1) examinations of potential increases in REM sleep following heightened experiences in waking; and (2) examinations of the effects of REM sleep deprivation on previously learned tasks. A number of reviews (Dujardin et al. 1990; Fishbein & Gutwein 1977; Horne 1988; Horne & McGrath 1984; McGrath & Cohen 1978; Pearlman 1979; Smith 1985), including recent ones (Hennevin et al. 1995b; Rechtschaffen 1998; Smith 1995; 1996), have been devoted to the topic of sleep and memory. The following is not intended as a review of this area, but rather is meant to serve as a general background and critical assessment of some important issues involving sleep and memory.

2.1. Effects of heightened experiences of waking on subsequent REM sleep

The rationale behind this set of studies is as follows: If REM serves to consolidate learning/memory, then exposure to enhanced learning situations or enriched environments in waking should result in increases in REM to process and consolidate these experiences. We will only briefly discuss this work for we do not believe that it represents a particularly powerful test of the REM consolidation hypothesis owing, among other things, to confounding effects of natural variations in REM sleep and the difficulty of establishing, at least for animals, that enriched experiences represent a significant departure from normal routines. Additionally, there is a certain degree of circularity in this position, in that enhanced learning experiences in waking presumably trigger increases in REM to consolidate them, yet they only become "learning experiences" after being processed and consolidated in REM sleep.

The findings of several reports in animals and humans using this paradigm have been mixed. In general, the majority of studies in animals have reported that heightened learning experiences or enriched conditions in waking produce increases in the amount of REM sleep (Horne 1988; Horne & McGrath 1984; McGrath & Cohen 1978; Smith 1985); on the whole, human studies have not shown this to be the case (Allen et al. 1972; Bowe-Anders et al. 1974; Horne 1976; Horne & Walmsley 1976; Zimmerman et al. 1978).

Horne and McGrath (1984) have raised objections to the animal work, pointing out, for instance, that in many of these reports: (1) increases in REM appeared to be an "artifact" of an overall increase in total sleep time (TST); that is, the proportion of REM to TST was not increased (Gutwein & Fishbein 1980a; 1980b; Kiyono et al. 1981; Krech et al. 1962; Mirmiran et al. 1982; Tagney 1973;); and (2) control animals were generally confined to impoverished environments, raising the possibility that differences between control and experimental animals involved decreases in REM (reflecting decreases in TST) for controls rather than increases for the experimental animals (Gutwein & Fishbein 1980a; 1980b; Krech et al. 1962; Tagney 1973).

McGrath and Cohen (1978) reviewed 15 studies in humans examining the effects of enhanced waking experiences on REM sleep (nondeprivation studies) and reported a lack of effect in 10 of the 15 reports. They concluded: "nondeprivation studies employing humans seemingly provide little support for a relationship between REM sleep and learning."

2.2. REM deprivation (REMD) studies in animals

REM deprivation (REMD) studies in animals and humans are of two types: prior REMD and post (or subsequent) REMD, reflecting whether the REM deprivation period precedes (prior) or follows (post) the learning situation.

2.2.1. Post-REMD studies. Specifically, the post-REMD procedure involves training animals to criterion on a task(s), depriving them of REM sleep for varying periods of time, and then retesting them on the task(s). If REM is critical

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for learning/memory, REMD should severely disrupt these functions; and if REM is not critical, REMD should have no effect on learning/memory.

The most widely used technique for depriving animals of REM sleep is the water tank (or pedestal) technique. In brief, animals are placed on top of a small pedestal (usually a small inverted flower pot) that is surrounded by water. As animals enter REM sleep, they lose postural tone (atonia), partially or fully slip from the pedestal into the water, and awaken. The procedure is thought to fairly selectively deprive animals of REM sleep. Controls are placed on larger diameter pedestals or allowed normal sleep in their home cages.

It is widely acknowledged that the pedestal technique introduces several spurious and uncontrolled variables that are generally recognized to confound results obtained with this method; these include isolation, wetness, heat loss, high levels of stress, muscle fatigue, and a significant loss of slow wave sleep as well as REM (Coenen & van Luijtelaar 1985; Ellman et al. 1978; Fishbein & Gutwein 1977; Grahnstedt & Ursin 1985; Horne & McGrath 1984; Kovalzon & Tsibulsky 1984; Youngblood et al. 1997). The pedestal technique is a severe method for REMD; alternatives are presently used such as the multiple platform and pendulum techniques (van Hulzen & Coenen 1980; 1982; van Luijtelaar & Coenen 1986) as well as the recently developed disk-over-water method of Rechtschaffen and Bergmann (Rechtschaffen 1998; Rechtschaffen & Bergmann 1995).

It appears that the problems inherent in the pedestal technique have significantly clouded findings obtained with it. In fact, Bill Fishbein, an advocate of the REM consolidation hypothesis, recently acknowledged (Fishbein 1995) that he abandoned REMD work in mice because he was not able to respond adequately to criticisms leveled at the technique. He stated that he could not

> have anticipated all the flack that I received, in the years to come, about the "stress factor" produced by the mouse-on-the pedestal technique. I spent a great deal of time trying to prove that there was no stress factor. Despite my efforts to design experiments in a way that training and retention testing were not confounded by the pedestal procedure, it became clear that no matter what control experiment I did, I was never going to convince everyone. Eventually this controversy led me to completely abandon the REM deprivation procedure and look instead at the effects of learning on REM enhancement.

With the caveat, then, that many of the REMD studies supporting a role for REM in memory consolidation may lack validity based on the use of the pedestal technique, a review of the REMD work in animals shows studies to be about equally divided among those showing that REMD disrupted learning/memory (Fishbein 1971; Leconte et al. 1974; Pearlman & Becker 1973; 1974; Smith & Butler 1982; Smith & Kelly 1988) and those showing that this was not the case (Albert et al. 1970; Dodge & Beatty 1980; Joy & Prinz 1969; Miller et al. 1971; Shiromani et al. 1979; Sloan 1972; van Hulzen & Coenen 1979).

As discussed above, it is generally acknowledged that depriving animals of REM sleep with the pedestal technique or other means is debilitating. This has led to the view that the impairments seen following REMD are not true learning/memory deficits but merely performance deficits; that is, animals are simply unable to perform the required task(s), in large part owing to the physically debilitating effects of the deprivation. Attempts to separate learning from performance deficits primarily by looking at short term versus long term effects of REMD have largely shown that impairments are short term or, in effect, performance deficits (Fishbein 1970; 1971; van Hulzen & Coenen 1982).

For example, Fishbein (1971) trained groups of mice on a passive avoidance task, deprived them of REM sleep for 1, 3, 5, or 7 days using the pedestal technique, and then retested them on the task 30 min, 3 h, and 24 h following removal from the pedestal. The results showed that: (1) mice deprived of REM for 1 day showed no impairments at any of the three retest intervals (i.e., 30 min, 3 h, or 24 h) and (2) mice deprived for 3, 5, or 7 days showed marked deficits when retested at 30 min and 3 h but no impairments when retested at 24 h. In essence, mice deprived of REM for 3, 5, or 7 days were very impaired on short term but not on long term retest (i.e., 24 h), indicating that deficits were most likely performance and not learning/memory deficits.

2.2.2. Prior REMD studies. A number of reports (Bueno et al. 1994; Danguir & Nicolaidis 1976; Fishbein 1970; Hartmann & Stern 1972; Linden et al. 1975; Sagales & Domino 1973; Stern 1971; van Hulzen & Coenen 1982; Venkatkrishna-Bhatt et al. 1978) have shown that depriving animals of REM sleep prior to training (prior REMD) impairs acquisition/learning on a variety of tasks. These studies, however, do not seem to test the REM consolidation hypothesis since the deprivation period precedes training/acquisition and there is no potential carryover of information pre to post REMD as in the post-REMD design.

Aside from their intended purpose, we suggest that the prior REMD studies support the position that the deficits seen in post-REMD reports were performance and not memory deficits. With both paradigms (prior and post REMD) animals are impaired to similar degrees on the same types of tasks. In the post-REMD paradigm, however, the claim is made that deficits involve the inability of animals to use information learned prior to deprivation, as a direct result of the loss of REM; that is, animals perform poorly following REM deprivation because without REM they are unable to process, store, and utilize information acquired before deprivation to meet the demands of the task – a memory deficit. Although impairments are similar with the prior REMD paradigm, the claim could not be made that this involves a memory dysfunction. We suggest that in both cases the impairments are mainly performance deficits due in large part to the debilitating effects of deprivation procedures. The following report (van Hulzen & Coenen 1982) is consistent with this view.

van Hulzen and Coenen (1982) deprived two groups of rats of REM sleep for three days – one group with the pedestal (or water tank) technique and the other with the less stressful pendulum technique. Immediately following deprivation, both groups were trained on a two-way shuttle avoidance task (acquisition) and then retested six days later. Rats deprived with the pedestal technique showed severe impairments in acquisition but not on retest; those deprived by the pendulum method showed no deficits on acquisition or retest.

The results show that prior REMD by a stressful technique (pedestal), as opposed to a more moderate procedure (pendulum), affects immediate performance, while neither procedure impairs performance/learning when rats are fully

recovered from REMD – that is, six days after deprivation. The findings suggest that stress (or other factors) associated with REMD and not necessarily the loss of a particular stage of sleep is largely responsible for the disruptive effects of REMD. This was indicated by the authors when they stated:

> shuttle box avoidance performance [was found] to be severely disrupted following 72 hrs of PS [paradoxical sleep] deprivation by means of the water tank technique. Similar effects could not be replicated in using the pendulum technique. Therefore, the possibility that these phenomena are not due to PS deprivation per se must seriously be considered. (van Hulzen & Coenen 1982)

2.2.3. Summary and conclusions. A review of REMD studies in animals shows that they are about equally divided in showing that REMD does or does not disrupt learning/memory. As developed above, it has been argued that reports claiming that REMD disrupts learning/memory are confounded by the use of very stressful deprivation procedures. It appears that stress (and associated factors) rather than the loss of sleep/REM sleep is responsible for the learning/memory deficits seen in these studies. While these reports are open to other interpretations, there appears to be no alternative explanation for studies that fail to show that REMD disrupts learning/memory.

Following a comprehensive review of the REMD literature, Horne (1988) concluded:

> The memory consolidation theories for REM sleep function are having increasing difficulty in handling REM sleep deprivation findings, as it is clear from both animal and human studies that even the longest periods of deprivation do not incapacitate memory, and at best only produce modest decrements.

And further, "In sum, and in relation to the memory consolidation hypothesis for REM sleep, I find the field of REM sleep deprivation and learning in animals unconvincing."

2.3. REM windows

Carlyle Smith, a foremost advocate of the REM consolidation hypothesis and a major contributor to this area, has put forth and provided supporting evidence for the existence of "REM windows"; that is, specific segments of REM sleep that are enhanced following learning and corresponding segments which when disrupted (REMD) impair learning/memory. According to the proposal, memories are selectively consolidated during the period of the REM windows (for review, see Smith 1985; 1995; 1996).

The REMD studies of Smith and coworkers focusing on REM windows appear subject to some of the same problems as other REMD studies, foremost of which is the inability to adequately control for the stress factor associated with the use of the pedestal technique for REMD. However, in defense of Smith and colleagues it should be noted that their work is less vulnerable to this criticism because their REMD periods are generally short, about 4–12 h.

On the other hand, there are difficulties with "REM windows" not encountered by other REMD studies. Of significant concern is the shifting nature of the REM window. As readily acknowledged by Smith (1985; 1996), the precise location of the window in REM varies widely, dependent on such factors as species and even strain of animals, the nature of the training tasks, and the number and distribution (concentrated or dispersed) of training trials per session and/or per day. For instance, in separate reports, the times (post training) of the "REM window(s)" were: 9–12 and 17–20 h (Smith & Butler 1982), 48–72 h (Smith & Kelly 1988), 53–56 h (Smith & MacNeill 1993), 5–8 h (Smith & Rose 1996), and 1–4 h (Smith & Rose 1997). In fact, the last two studies (Smith & Rose 1996; 1997) involved virtually identical conditions (place learning with rats on the Morris water maze) yet the window shifted from 5–8 h in the earlier report to 1–4 h in the later one. Apparently, the only difference was a change from distributed (Smith & Rose 1996) to massed trials (Smith & Rose 1997).

It appears that REM windows (at least as defined for animals) are not present in humans. Smith and Lapp (1991) examined patterns of REM sleep (potential windows) in college students following an intense learning experience (post exams) compared to baseline periods (summer vacation), and reported that aside from an increase in the total number of (rapid) eye movements in test versus control conditions (most prominent in the fifth REM period), there were no changes in sleep/REM sleep under the two conditions. They stated: "No other REM-related measure (minutes of REM sleep, % REM sleep or latency from stage 2 onset to any of the five REM periods) was found to be significant. Further, there were no changes in any of the other sleep parameters measured" (Smith & Lapp 1991).

Finally, although there is some suggestion from recent work in humans that information is differentially processed in distinct phases of SWS and/or REM sleep (Plihal & Born 1997; Stickgold et al. 2000b), to our knowledge "REM windows" has not been independently demonstrated outside of the laboratory of Smith and colleagues (see Smith 1996). It seems that this potentially important phenomenon would be considerably strengthened if confirmed in other laboratories.

2.4. REMD studies in humans: Early reports

Compared to their numbers on animals, relatively few reports on humans have examined the effects of REMD on learning/memory. In contrast to the case with animals in which reports were about equally divided among those showing, or not, that REMD affects learning, the majority of studies in humans have described minimal or no effects of REM deprivation on learning/memory (Castaldo et al. 1974; Chernik 1972; Ekstrand et al. 1971; Lewin & Glaubman 1975; Muzio et al. 1972). If anything, complex tasks (Empson & Clarke 1970; Tilley & Empson 1978), as opposed to simple tasks (Castaldo et al. 1974; Chernik 1972), appear to be affected by REMD.

Following a review of early REMD studies in humans, Horne (1988) concluded:

> It is clear that, given before or after learning, REM sleep deprivation does not lead to any greater learning impairment on simple tasks, but difficult tasks are more affected. Whilst these latter findings can reach statistical significance, the effects are still relatively small, and not convincing enough to support any theory that REM sleep has a crucial role to play in the consolidation of memory.

2.5. REM sleep and memory consolidation in humans: Recent reports

Karni and Sagi (1993) initially showed that improved performance on a perceptual learning task required the pas-

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sage of time; that is, subjects showed no improvement immediately following training but marked improvement 8–10 h following training. As discussed below, they have extended their original findings to sleep: performance was shown to improve not only with an intervening period of waking but also of sleep (Karni et al. 1994).

The task involved identifying the orientation of three diagonal lines (arranged either horizontally or vertically) embedded in a background of horizontal lines. The stimulus (target and background elements) was presented briefly (10 msec) in one quadrant of the visual field followed by a blank screen and then a patterned mask (100 msec). The interval between the onset of the stimulus and onset of the mask (stimulus-to-mask onset asynchrony, SOA) was varied, and the measure of performance was an 80% correct identification (threshold SOA) of the stimulus (horizontal or vertical lines) at a set interval. The index of improved performance was a decrease in threshold SOA (Karni & Sagi 1993; Karni et al. 1994).

In the sleep study, Karni et al. (1994) trained subjects on the task and then tested them after a normal night of sleep, sleep without SWS, or sleep without REM. They described significantly improved performance following a normal night of sleep as well as sleep that included REM but not SWS (SWS deprivation condition), but no gains in performance in the absence of REM sleep (REM deprivation condition). Karni et al. (1994) concluded that learning of this perceptual skill was a slow latent process requiring consolidation over time. The period of consolidation could be in waking or sleep, but if in sleep, it required REM sleep not SWS.

Using the identical visual display, Stickgold et al. (2000b) recently reported, like Karni et al. (1994), that subjects exhibited marked improvement on the task following sleep. Specifically, they reported: (1) no improvement on the task over the course of waking; (2) no improvement unless subjects obtained at least 6 h of sleep; (3) improved performance proportional to the total amount of sleep after 6 h of sleep; and (4) improved performance proportional to the amount of SWS in the first quartile of the night (SWS1) and to the amount of REM in the last quartile (REM4). They proposed that learning was a two-step process requiring both SWS (SWS1) and REM (REM4).

Although there are parallels between the two sets of findings (Karni et al. 1994; Karni & Sagi 1993; Stickgold et al. 2000b), there are several pronounced differences. A major difference involves the performance of subjects during waking. As discussed above, Karni and Sagi (1993) originally showed and subsequently confirmed (Karni et al. 1994) that performance significantly improved over time during waking. By contrast, Stickgold et al. (2000b) reported no improvement during post training waking behavior, even after 12 h, commenting: "12 hours of wake behavior was inadequate to produce reliable improvement while as little as 9 hours of sleep reliably produced improved performance."

Additional differences were as follows: (1) Stickgold et al. (2000b) demonstrated a direct relationship between improved performance and total amounts of SWS, particularly SWS1, whereas Karni et al. (1994) showed that depriving subjects of SWS did not alter performance; and (2) Stickgold et al. (2000b) reported that a minimum amount of sleep (6 h) was required for improved performance, and after 6 h gains were proportional to the total amount of sleep; neither was the case in the report by Karni et al. (1994). Until these discrepancies are resolved, it is difficult to evaluate the reliability of the findings using this perceptual learning paradigm.

2.6. Theta rhythm and REM sleep

In a variation of the REM consolidation hypothesis, Jonathan Winson has proposed and provided supporting documentation for the position that certain types of memory, specifically memories that are critical for the survival of the species, are selectively processed and consolidated in REM sleep (Pavlides & Winson 1989; Winson 1985; 1990; 1993). The theta rhythm of the hippocampus figures prominently in this proposal (Greenstein et al. 1988; Pavlides et al. 1988; Winson 1972; 1978).

Winson (1972) reviewed the behavioral correlates of the theta rhythm of waking in several species and showed that theta was selectively present during certain behaviors characterized as species-specific behaviors that are critical for survival; for example, exploration in rats, defensive behaviors in rabbits, and predation in cats. In addition, theta is present throughout REM sleep (Vanderwolf 1969).

A number of recent reports (including those of Winson and colleagues) have shown that theta is directly involved in mnemonic functions of the hippocampus (for review, Vertes & Kocsis 1997). For example, it has been demonstrated that: (1) long term potentiation (LTP) is optimally elicited in the hippocampus with stimulation at theta frequency (i.e., 5–7 Hz or pulses separated by 170–200 msec) (Diamond et al. 1988; Greenstein et al. 1988; Larson & Lynch 1986; 1988; Larson et al. 1986; Leung et al. 1992; Rose & Dunwiddie 1986; Staubli & Lynch 1987); (2) stimulation delivered in the presence but not in the absence of theta potentiates population responses in the hippocampus (Bramham & Srebro 1989; Huerta & Lisman 1993; Pavlides et al. 1988); and (3) discrete medial septal (MS) lesions that abolish theta produce severe learning/memory deficits, as do MS lesions with unexplored effects on the hippocampal EEG (Berger-Sweeney et al. 1994; Dutar et al. 1995; Hagan et al. 1988; Hepler et al. 1985; Kesner et al. 1986; Leutgeb & Mizumori 1999; M'Harzi & Jarrard 1992; Mizumori et al. 1990; Poucet et al. 1991; Shen et al. 1996; Stackman & Walsh 1995; Walsh et al. 1996; Winson 1978).

In brief, then, Winson's position is that theta serves to encode survival-enhancing information during waking and to consolidate this information during REM sleep. In this scheme, theta is essential for the acquisition of skills for survival.

The primary focus of the research of the senior author is the theta rhythm of the hippocampus. In fact, the senior author was introduced to this area by Jonathan Winson and remains enormously grateful for the opportunity to learn from him. As is evident, however, we do not share Winson's view that theta is instrumental in consolidating memories in REM sleep.

We believe that the case is strong for the involvement of theta in mnemonic functions of waking but not of REM sleep (Vertes 1986a; Vertes & Kocsis 1997). This seeming discrepancy was recently addressed by Fishbein (1996) stating, "Robert Vertes has published a variety of studies that would lead one to assume he would be a leading champion of the theory of memory consolidation in REM sleep. Despite his important contributions he does not believe the collected evidence supports it."

Our position is that theta of REM is a by-product of the intense activation of the pontine region of the brainstem in REM sleep; theta merely reflects this activation and as such may not have any functional significance in REM or at least not the same functional significance as in waking. In a series of studies (Kocsis & Vertes 1994; 1997; Vertes 1979; 1981; 1988; 1992; Vertes & Martin 1988), we have shown that the theta rhythm is generated by a system of connections from the pontine reticular formation (PRF) to the septum-hippocampus. In brief, cells of nucleus pontis oralis of PRF fire tonically with theta and transfer this tonic barrage to the supramammillary nucleus of the hypothalamus where it is converted into a rhythmical pattern of discharge and then relayed to the GABAergic/cholinergic pacemaking cells of the medial septum to drive theta (Vertes & Kocsis 1997).

As previously described (Datta 1995; Jones 1991; Steriade & McCarley 1990a; Vertes 1984; 1990), pontine and lower mesencephalic regions of the brainstem contain discrete populations of cells that control individual events of REM sleep; when activated together these cell groups trigger each of the major indices of REM sleep (cortical EEG desynchronization, hippocampal theta, muscle atonia, PGO spikes, rapid eye movements, myoclonic twitches, and cardiorespiratory fluctuations), and hence the REM state. Part of this orchestration of activity of the pontine RF in REM involves excitation of nucleus pontis oralis and consequently theta. As argued above, theta of REM may simply reflect a highly activated brainstem in REM, and thus bear little functional relationship to its role in waking.

The presence of similar electrophysiological events in waking and sleep does not indicate that they serve the same (or even similar) physiological and/or behavioral function(s). For example, the cortical EEG desynchronization of waking and REM by no means signifies identical processes in the two states; that is, the EEG desynchronization of waking is associated with diverse sensory, motor, emotional, and cognitive processes that are notably absent in REM sleep.

As indicated, we favor the position that theta is critically involved in memory processing functions of waking (Vertes 1986a; Vertes & Kocsis 1997). Specifically, we propose that theta serves to gate and/or encode information reaching the hippocampus simultaneously with it from various external sources (e.g., the entorhinal cortex). In the awake state, the "information arriving with theta" is governed by the behavioral situation (context); that is, the sum of internal and external events relatively time locked to theta. If theta were involved in memory processing functions in REM, it should, in a similar manner, gate information to the hippocampus in that state. Unlike waking, however, in which the information reaching the hippocampus is dictated by behavioral circumstances, there appears to be no mechanism in REM for the selection and orderly transfer of information to the hippocampus from other sources. If the transfer of information in REM is not orderly, or is essentially chaotic, it would seem that there would be no functional value in consolidating or "remembering" this information. In effect, dream-like material might be presented to the hippocampus in REM, but there would be no purpose in storing or consolidating it during REM. This may be the reason that dreams (or other cognitive material of REM) are so poorly remembered.

In sum, the theta rhythm is present in waking and REM; we believe that theta serves a mnemonic function in waking but not in REM sleep.

3. REM sleep and antidepressant drugs

It is well recognized that virtually all major antidepressant drugs suppress REM sleep (for review, Vogel et al. 1990) and it has, in fact, been proposed that the clinical efficacy of these drugs largely derives from their suppressant effects on REM sleep (Vogel 1975; 1983). The major classes of antidepressant drugs are the monoamine oxidase inhibitors (MAOIs), the tricyclic antidepressants (TCAs), and the recently developed and widely used selective serotonin reuptake inhibitors (SSRIs). A review of the actions of several members of these classes of antidepressants shows that they profoundly suppress REM sleep.

3.1. Monoamine oxidase inhibitors (MAOIs)

Of the antidepressants, the MAOIs have the strongest suppressive action on REM sleep. A number of early reports using normal and patient populations showed that MAOIs virtually completely (or completely) suppressed REM sleep for weeks to several months. In an initial study, Wyatt et al. (1969) reported that the MAOIs, isocarboxazid, pargyline hydrochloride, and mebanazine, reduced REM from about 20–25% of TST to 9.7, 8.6, and 0.4% of TST, respectively, and that in one subject REM was virtually eliminated for two weeks.

In a subsequent report in anxious-depressed patients, Wyatt et al. (1971b) described the remarkable findings that the MAOI, phenelzine (Nardil), given at therapeutic doses, completely abolished REM sleep in six patients for periods of 14 to 40 days. There was a gradual decline in amounts of REM sleep for the first two weeks on the drug and a total loss of REM after 3–4 weeks. In a complementary study with narcoleptic patients, Wyatt et al. (1971a) reported that phenelzine completely abolished REM in five of seven patients for the following lengths of time: 14, 19, 93, 102, and 226 days. They stated that: "The complete drug-induced suppression of REM sleep in these patients is longer and more profound than any previously described"; and further that "no adverse psychological effects were noted during the period of total rapid-eye-movement suppression."

Several other studies have similarly shown that MAOIs essentially abolish REM sleep. Akindele et al. (1970) reported that phenelzine completely eliminated REM sleep in four subjects (one normal and three depressed) for 2 to 8 weeks, and addressing possible behavioral consequences stated that, "Far from this leading to disastrous effects on mental functions, as some might have proposed, clinical improvement began." Kupfer and Bowers (1972) showed that phenelzine abolished REM in seven of nine patients, and drastically suppressed it in remaining patients from predrug values of 23.1 and 24.8% of TST to 1.4 and 0.5% of TST, respectively. Finally, Dunleavy and Oswald (1973) reported that phenelzine eliminated REM in 22 depressed patients.

If REM sleep were involved in memory consolidation, it would seem that the total loss of REM with MAOIs for periods of several months to a year (Dunleavy & Oswald 1973; Kupfer & Bowers 1972; Wyatt et al. 1969; 1971a; 1971b) would affect memory. As indicated above, the loss of REM

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did not appear to be associated with any noticeable decline in cognitive functions in these largely patient populations. These studies, however, made no systematic attempt to assess the effects of MAOIs on cognition.

Other reports, however, have examined the actions of MAOIs, primarily phenelzine, on cognition/memory and described an essential lack of impairment (Georgotas et al. 1983; 1989; Raskin et al. 1983; Rothman et al. 1962). For example, Raskin et al. (1983) observed no adverse effects of phenelzine on a battery of 13 psychomotor and cognitive tasks in a heterogeneous population of 29 depressed patients. Similarly, Georgotas et al. (1983; 1989) reported that elderly depressed patients given phenelzine for 2 to 7 weeks showed no alteration in several measures of cognitive function, and concluded that the lack of adverse effects with phenelzine suggests that it is preferable to TCAs (see below) in the treatment of depression in the geriatric population.

3.2. Tricyclic antidepressants (TCAs) and selective serotonin reuptake inhibitors (SSRIs)

As discussed below, virtually all of the commonly used TCAs and SSRIs significantly suppress REM sleep, but unlike the MAOIs, do not eliminate it. Also, the TCAs and SSRIs appear to exert immediate suppressive effects on REM (within the first few days of treatment); by contrast, the MAOIs produce maximal effects on REM about 2–3 weeks following the start of treatment.

An early report by Dunleavy et al. (1972) in normal subjects analyzed the effects on sleep of six TCAs and showed that four of them (imipramine, desipramine, chlorimipramine, and doxepin) markedly depressed REM, beginning with the first night of administration. Chlorimipramine had the strongest suppressive effect on REM sleep, producing a complete loss of REM for the first three nights and an approximate 50% reduction in REM for the remaining four weeks of the study.

Several subsequent examinations of the actions on sleep of these and other TCAs (amitriptyline, amoxapine, nortriptyline, imipramine, maprotiline, clomipramine) have demonstrated that, as a class, TCAs produce an immediate 40–70% reduction in REM and sustained 30–50% decreases in REM sleep (Brebbia et al. 1975; Hartmann & Cravens 1973; Kupfer et al. 1979; 1982; 1991; 1994; Mendlewicz et al. 1991; Nicholson & Pascoe 1986; Passouant et al. 1975; Roth et al. 1982; Shipley et al. 1984; Staner et al. 1995; Ware et al. 1989). Of the TCAs, clomipramine appears to be the strongest REM-suppressant (Passouant et al. 1975; Sharpley & Cowen 1995; Thase 1998).

The SSRIs, like the TCAs, produce an initial marked reduction in REM sleep that slightly abates with time. Examinations of the effects on sleep of several SSRIs (indalpine, fluvoxamine, fluoxetine [Prozac], paroxetine, and zimelidine) show that on average they produce an initial reduction in REM of 40–85% and long term decreases of 30–50% (Kupfer et al. 1991; Nicholson & Pascoe 1986; 1988; Nicholson et al. 1989; Oswald & Adam 1986; Saletu et al. 1991; Sharpley et al. 1996; Shipley et al. 1984; Staner et al. 1995; Vasar et al. 1994; Vogel et al. 1990).

In general, the SSRIs exert stronger suppressive effects on REM than do the TCAs. Staner et al. (1995) compared the actions on sleep of long term treatment with paroxetine (SSRI) and amitriptyline (TCA) in depressed patients, and showed a 42% reduction in REM with paroxetine compared to a 30% reduction with amitriptyline. Similar findings have been described in other comparisons of these classes of antidepressants (Kupfer et al. 1991; Nicholson & Pascoe 1986).

Although the TCAs and SSRIs do not completely eliminate REM sleep, they significantly suppress it by as much as 75–85% in the short term (days) and 40–50% in the long term (weeks/months). As discussed for the MAOIs, if memories are consolidated in REM sleep, it would seem that the sustained reductions in REM with TCAs/SSRIs would alter memory.

There is a substantial literature describing the effects of TCAs and SSRIs on cognitive functions in normal and depressed subjects, including several reviews devoted to the topic (Amado-Boccara et al. 1995; Deptula & Pomara 1990; Knegtering et al. 1994; Thompson 1991; Thompson & Trimble 1982). Because these classes of antidepressants are in such widespread use, it is obviously important to know if they disrupt motor/cognitive functioning.

3.2.1. The effects of TCAs on cognition/memory. Although there is conflicting evidence, mainly related to the diverse procedures used to evaluate the effects of antidepressants on cognition (Amado-Boccara et al. 1995; Deptula & Pomara 1990; Thompson & Trimble 1982), the general consensus is that some TCAs, primarily amitriptyline, impair memory, but most have minor or no effects on memory (for review, Amado-Boccara et al. 1995; Deptula & Pomara 1990; Thompson 1991; Thompson & Trimble 1982). Virtually all TCAs have some sedative and anticholinergic actions (Hardman et al. 1996), and if cognitive dysfunctions are present with TCAs they reportedly involve these properties (Curran et al. 1988; Deptula & Pomara 1990; Spring et al. 1992; Thompson 1991).

A number of studies have shown that amitriptyline disrupts memory – whether given acutely or long term, to the depressed or nondepressed, and across all age groups (Branconnier et al. 1982; Curran et al. 1988; Lamping et al. 1984; Linnoila et al. 1983; Spring et al. 1992; Warot et al. 1996). For instance, Spring et al. (1992) compared the effects of a four-week treatment with amitriptyline and clovoxamine (an SSRI) on psychomotor and memory tests in depressed outpatients, and reported that amitriptyline, despite alleviating depression, significantly impaired performance on the memory tasks. Clovoxamine, on the other hand, had no adverse effects of psychomotor/cognitive performance (see also below).

Spring et al. (1992) attributed the disruptive effects of amitriptyline on cognition to its anticholinergic actions, noting that, in general, anticholinergics (e.g., scopolamine) disrupt memory (Caine et al. 1981; Drachman & Leavitt 1974). They stated: "The decline in memory performance associated with amitriptyline apparently reflects the relatively high anticholinergic action of the drug, rather than a deficiency in its antidepressant action." And further, "Among the tricyclics, amitriptyline has the most pronounced anticholinergic effects, and would, therefore be expected to have the most adverse effect on memory."

Consistent with this interpretation, Curran et al. (1988) compared the effects on memory of four antidepressants (amitriptyline, trazodone, viloxazine, and protriptyline) that varied with respect to their sedative and anticholinergic properties, and showed that the sedating compounds

(amitriptyline and trazodone) but not the nonsedating ones (viloxazine and protriptyline) impaired performance on a battery of memory tests, and that disruptive effects were considerably greater with amitriptyline (an anticholinergic) than with trazodone (no anticholinergic properties) (Gershon & Newton 1980).

In contrast to amitriptyline, most other TCAs have minimal or no adverse effects on memory/cognition. In a well-designed study, Peselow et al. (1991) examined the effects on learning/memory of a four-week treatment with the TCA imipramine (Tofranil) with 50 depressed outpatients, and reported that imipramine improved memory in these patients. Although the improvement in memory was attributed to the clinical efficacy of the compound (not to a memory-enhancing function for imipramine), Peselow et al. (1991) clearly demonstrated, as have several others (Amin et al. 1980; Friedman et al. 1966; Glass et al. 1981; Henry et al. 1973; Raskin et al. 1983; Rothman et al. 1962) that imipramine did not impair memory – even though imipramine is a powerful REM suppressant (Kupfer et al. 1994; Ware et al. 1989). For instance, Kupfer et al. (1994) showed that imipramine produced sustained 35–40% reductions in REM sleep for three years in depressed patients.

Finally, several other TCAs (doxepin, desipramine, nortriptyline, amoxapine, protriptyline, maprotiline, and chlorimipramine) that also suppress REM sleep reportedly produce little or no detrimental effects on memory (Allain et al. 1992; Curran et al. 1988; Georgotas et al. 1989; Liljequist et al. 1974; Linnoila et al. 1983; McNair et al. 1984; Pishkin et al. 1978).

3.2.2. The effects of SSRIs on cognition/memory. As is well recognized, SSRIs are very widely used and currently the most prescribed treatment for depression. As a group the SSRIs do not appear to alter cognitive functions. For instance, there is no indication that any of the following SSRIs have any detrimental effects on psychomotor/cognitive functions in normal or patient populations: fluvoxamine, zimeldine, clovoxamine (an SSRI and partial noradrenergic reuptake inhibitor), sertraline, paroxetine, or fluoxetine (Curran & Lader 1986; Fairweather et al. 1993; 1996; Geretsegger et al. 1994; Hindmarch & Bhatti 1988; Hindmarch et al. 1990; Lamping et al. 1984; Linnoila et al. 1983; Saletu & Grunberger 1988; Saletu et al. 1980; Spring et al. 1992).

Kerr et al. (1992) recently examined the actions of paroxetine, alone or in combination with alcohol, on several psychomotor/cognitive tests in elderly nondepressed subjects with the goal of determining whether SSRIs, unlike compounds with anticholinergic and/or sedative effects, may alter cognitive functions. They speculated that SSRIs "are unlikely to have detrimental cognitive and psychomotor effects because of their unique pharmacological profile," and noted further that "patients often report that treatment with SSRIs leaves them feeling more able to think clearly." It was shown that paroxetine not only had no adverse effects on psychomotor and cognitive functions, but that it slightly ameliorated performance deficits produced by alcohol (Kerr et al. 1992).

Comparisons of the actions of amitriptyline and SSRIs on psychomotor/cognitive performance in healthy or depressed subjects (Curran & Lader 1986; Fairweather et al. 1993; Lamping et al. 1984; Linnoila et al. 1983; Spring et al. 1992) have demonstrated that amitriptyline but not SSRIs produced significant impairments. Lamping et al. (1984) reported that even though amitriptyline and clovoxamine gave rise to comparable relief from depression, the two antidepressants differentially affected memory; that is, "an impairment of memory after chronic amitriptyline administration, as contrasted with an improvement in memory after chronic administration of clovoxamine." Spring et al. (1992) described virtually the identical findings using the same two compounds.

Finally, an early review of this area (Thompson 1991) concluded that: "Newer compounds devoid of antimuscarinic effects, particularly the serotonin reuptake inhibitors, if not sedative, have not been associated with memory impairment. Furthermore, a few more recent studies suggest that these drugs may exert a beneficial influence on memory processes in memory-impaired individuals"; while a recent review (Amado-Boccara et al. 1995) similarly concluded that: "antidepressants which inhibit serotonin reuptake seem to have no deleterious cognitive effects."

3.2.3. Summary of the effects of antidepressants on cognition/memory. In summary, (1) MAOIs virtually abolish REM sleep but have no adverse effects on cognition/memory. (2) TCAs suppress REM by 30–70%. While amitriptyline, a strong anticholinergic and sedative compound, disrupts memory, most other TCAs produce minimal, or generally no, disruptive effects of cognitive/memory. (3) SSRIs suppress REM sleep by 40–85% but do not alter memory or other cognitive functions.

4. Brain stem lesions and REM sleep in humans

Although sizeable lesions at rostral, mesencephalic levels of the brainstem often result in persistent coma or death (Cairns 1952), those located more caudally within the pons are less severe and have been shown to give rise to a condition termed the "locked-in" syndrome. As originally described by Plum and Posner (1966), patients with this syndrome are fully conscious, alert, and responsive, but are quadriplegic and mute. Most of the patients retain the ability to make eye movements and very limited facial/head movements and some can communicate by small facial gestures. For instance, Feldman (1971) described a case of a woman with this syndrome who learned to communicate by Morse code using eye blinks and jaw movements.

A number of reports have examined sleep-wake profiles of these patients, and probably not surprisingly, have shown that most of them (or at least those with bilateral pontine lesions) completely lack REM sleep (Chase et al. 1968; Cummings & Greenberg 1977; Markand & Dyken 1976). For instance, Markand and Dyken (1976) reported that REM sleep was entirely absent in five of seven patients with the "locked-in" syndrome; SWS was present in essentially normal amounts. From case reports, the mental capacities of these patients, including memory for events and people, appear to be intact.

Although rare, there have been a few reports of patients with bilateral pontine lesions who are conscious, ambulatory, and verbally communicative (Lavie et al. 1984; Osorio & Daroff 1980; Valldeoriola et al. 1993). It appears that the lesions in these patients are less extensive than those with the locked-in syndrome. Nonetheless, like patients with the locked-in syndrome, they lack REM sleep (Osorio & Daroff 1980; Valldeoriola et al. 1993). Osorio and Daroff (1980)

described two such patients. Both of them showed similar sleep deficiencies, the most prominent of which was a complete loss of REM sleep. It was further pointed out that aside from minor neurological deficits, the patients led normal lives. The authors stated: "Our two patients are the first awake and ambulatory humans in whom total absence of REM sleep has been demonstrated. These REM deprived patients behaved entirely appropriately and were by no means psychotic." The "psychotic" reference alludes to the early notion, subsequently dispelled (Vogel 1975), that long term REM deprivation produces psychosis.

Lavie et al. (1984) described the interesting case of a man who at the age of twenty suffered damage to the pontine region of the brainstem from shrapnel fragments from a gunshot wound. Following the injury, the man was comatose for 10 days, remained in critical condition for another two weeks and then recovered. An examination of his patterns of sleep at the age of 33 revealed that he essentially lacked REM sleep; that is, REM was absent on most nights and averaged 2.25% of TST on the other nights. Similar to the study by Osorio and Daroff (1980), Lavie et al. (1984) reported that despite the virtually total loss of REM sleep, the man led a normal life. For instance, following the injury the man completed college, then law school, and at the time of the study was a practicing attorney.

Although no systematic attempt was made to examine the cognitive capacities of these patients, the virtual total loss of REM sleep did not seem to result in any apparent cognitive deficits.

5. Functional imaging studies of brain activity in REM sleep

Recent functional imaging studies of human brain activity in REM sleep reveal patterns of activity that are consistent with dream processes but not with memory consolidation.

The mental/cognitive content of REM sleep is dreams. Although dreams are not restricted to REM, they are unquestionably a prominent feature of REM sleep. Dreams are the sole window to cognitive processes of REM sleep. Although the function(s) of dreams have been, and continue to be, strongly debated (see Revonsuo, this issue), a generally agreed-upon feature of dreams is that they are poorly remembered. Similar to its function, diverse explanations have been put forth to account for the amnesic quality of dreams.

Foulkes and coworkers (Foulkes 1982a; 1985; 1999; Foulkes & Fleisher 1975; Foulkes et al. 1989), leading proponents of the view that dreams are a meaningful extension of waking mental life, have suggested that the reason dreams are so easily forgotten is that the brain in REM sleep is in a reflective mode (akin to reminiscing about, or reflecting on, events during waking) rather than in an encoding mode. An important difference, however, between the reflections of dreams and waking is that during waking one can rapidly switch from the reflective to the encoding mode to integrate and possibly store information. This cannot readily be done in REM sleep and as a result the reflections/reminiscences of REM (dreams) are lost to memory (Foulkes 1985; Foulkes & Fleisher 1975).

At the opposite end of the spectrum to the position of Foulkes and others (Domhoff 1969; 1996; Domhoff & Schneider 1998; Hall & Van de Castle 1966; Van de Castle 1994) that dreams are logical and meaningful, Hobson and colleagues (Hobson 1988b; Hobson et al. 1998b) have argued that dreams can be defined by such characteristics as hallucinosis, bizarreness, delusion, and confabulation and have likened dreams to the "delirium of organic brain disease"(Hobson 1997b). Hobson et al. (1998b) have proposed a purely physiological explanation for the amnesia of REM, pointing to the likely correspondence between memory loss and underlying physiological changes in REM, stating: "The loss of memory in REM sleep makes dreaming consciousness much more difficult to recall than waking consciousness. This phenomenological deficit logically implies a physiological deficit: some functional process, present and responsible for memory in waking is absent, or at least greatly diminished, in REM sleep."

Independent of theories of dreams, recent functional imaging studies in humans during sleep have revealed patterns of activity in REM that appear to reflect dream processes, including its amnesic quality. Although differences exist among reports (Braun et al. 1997; 1998; Maquet et al. 1996; Nofzinger et al. 1997), a fairly consistent pattern of brain activity in REM sleep in humans has emerged from these studies. Some important findings are as follows: (1) the pontine reticular formation is highly active in REM sleep; (2) primary sensory areas (e.g., striate cortex for the visual system) are inactive in REM; by contrast, extrastriate (visual) regions (as well as other sensory association sites) are very active in REM; (3) limbic and paralimbic regions, including the lateral hypothalamus, the amygdala and anterior cingulate, and parahippocampal cortices, are intensely activated in REM; and (4) widespread regions of the frontal cortex including the lateral orbital and dorsolateral prefrontal cortices show marked reductions in activity in REM sleep (Braun et al. 1997; 1998; Maquet et al. 1996; Nofzinger et al. 1997).

This general pattern of activity in REM has been viewed as a "closed system" (Braun et al. 1998); essentially, an internal network disconnected from inputs and outputs. For instance, the suppression of activity in the primary visual cortex (input) is consistent with the well-characterized sensory blockade of REM, whereas the deactivation of the prefrontal cortex (output) parallels the failure of dreams to influence executive systems for behavior. With respect to the latter, Braun et al. (1997) stated: "REM sleep may constitute a state of generalized brain activity with the specific exclusion of executive systems which normally participate in the highest order analysis and integration of neural information."

In effect (and not unexpectedly), the brain in REM sleep mirrors the dreaming brain; that is, internally generated visual images are fed to (or recruited by) the limbic system. They are then incorporated into dreams but due to the suppression of activity of the prefrontal cortex dream scenarios are not often recorded and generally do not influence waking behavior. In this regard, in an article on the neural basis of consciousness, Jones (1998) commented that the recent demonstration in imaging studies (Braun et al. 1997; Maquet et al. 1996) that activity in the frontal cortex is depressed in REM suggests "an attenuation of processes important in episodic and working memory and perhaps explaining why unless awakened from a dream, a sleeping person has no memory of the dream."

Finally, if dream material is so readily forgotten in REM sleep (reflecting the state of the brain in REM), it seems

unlikely that other mental phenomena that are not incorporated into dreams would be processed and permanently stored during REM sleep.

In summary, the pattern of brain activity in REM sleep is consistent with dreams but inconsistent with the orderly evaluation, organization, and storage of information which is the domain of attentive, waking consciousness.

6. A proposed function for REM sleep

It appears that the active state of the brain during REM has fueled claims that REM sleep is involved in complex, higher order functions, including memory (for review, Rechtschaffen 1998).

It is tempting to speculate, as several theories do, that magical processes occur during REM sleep; that is, that during the unconscious state of REM sleep some programmed or purposeful reordering of mental events occurs so that a nightly replay of daytime events during REM enhances the storage or consolidation of these events. In contrast to the view that the effects of REM extend beyond sleep to influence waking activities, we propose that REM can be entirely understood within the context of sleep without invoking mental phenomena or quasi-conscious processes (for review, Vertes 1986b). REM is a state of sleep; as such, it would seem that attempts to describe its function should look to sleep and not to waking.

As described in detail in our earlier theoretical paper (see Vertes 1986b), we propose that the primary function of REM sleep is to provide periodic endogenous stimulation to the brain which serves to maintain minimum requisite levels of CNS activity throughout sleep. REM is the mechanism used by the brain to ensure and promote recovery from sleep. We argued that the brain is strongly depressed in SWS, particularly in delta sleep, and incapable of tolerating long continuous periods of relative suppression. REM serves the critical function of periodically activating the brain during sleep without awakening the subject or disturbing the continuity of sleep. By analogy, the process of induction and recovery from general anesthesia is a delicate one requiring the special skills of highly trained medical professionals. The brain performs a very similar function daily and seemingly flawlessly. REM is an integral part of this process.

Our theory is consistent with sleep state organization; the main elements of which are that: (1) the percentage of REM sleep is very high in early infancy (about 50% of total sleep time) and declines sharply at 2–3 months of age; (2) sleep continuously cycles from light to deep sleep and back to lighter stages of sleep as the cycle repeats itself; and (3) REM sleep is quite evenly distributed throughout sleep (occurring about every 90 minutes) and the duration of REM periods become progressively longer throughout sleep.

Regarding this organization, we would suggest that the high percentage of REM sleep in neonates serves to offset equally high amounts of SWS in newborns (see also, Benington & Heller 1994); that sleep cyclically alternates between light and deep sleep to prevent the brain from dwelling too long in deep SWS; and that the progressively longer periods of REM throughout sleep serve to prime the brain for a return to consciousness as waking approaches. With respect to the latter, the disorientation experienced on sudden, unexpected awakenings from sleep (middle of the night), compared to natural awakening, may reflect an inadequate preparation of the brain for waking due to incomplete REM.

In line with the foregoing, reductions in REM, seen particularly with antidepressants, are generally accompanied by a reorganization of sleep; that is, marked increases in light SWS and corresponding decreases in deep SWS as well as frequent awakening (Cohen et al. 1982; Kupfer et al. 1989; 1991; Nicholson & Pascoe 1988; Saletu et al. 1983; 1991; Schenk et al. 1981; Shipley et al. 1984; Staner et al. 1995; Wyatt et al. 1971b). For the SSRIs, this has been referred to as the "alerting" effect on sleep of these antidepressants (Kupfer et al. 1989; 1991; Nicholson & Pascoe 1988; Saletu et al. 1983; 1991; Schenk et al. 1981; Shipley et al. 1984; Staner et al. 1995).

In accord with others (Benington & Heller 1995; Berger & Phillips 1995), we believe that the general purpose of sleep is restitution/recuperation for the CNS, and within this context, the primary function of REM sleep is to prepare the brain/CNS for recovery from sleep.

7. Conclusions

We believe that the evidence reviewed in this report disputes the claim that REM sleep serves a role in the consolidation of memory. Numerous studies have shown that depriving animals of REM sleep has no effect on learning/memory. Although other reports have shown that REM deprivation (REMD) disrupts memory, many of them have been questioned based on the use of the stressful pedestal technique for REMD leading to the view that reported deficits were performance and not learning/memory deficits. The majority of REM deprivation studies in humans have failed to show that REMD disrupts memory. Perhaps the strongest evidence against the memory consolidation hypothesis comes from the demonstration that antidepressant drugs or brain stem lesions profoundly suppress, or eliminate, REM sleep, yet neither appears to alter memory/cognitive functions. Finally, recent imaging studies in humans during sleep have described patterns of activity that are consistent with dreams, including their amnesic quality, but inconsistent with the orderly processing, evaluation, and storage of information that characterizes waking consciousness. In conclusion, we believe that the weight of evidence, as reviewed herein, fails to support a role for REM sleep in the processing or consolidation of memory.

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How does the dreaming brain explain the dreaming mind?

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Abstract: Recent work on functional brain architecture during dreaming provides invaluable clues for an understanding of dreaming, but identifying active brain regions during dreaming, together with their waking cognitive and cognitive functions, informs a model that accounts for only the grossest characteristics of dreaming. Improved dreaming models require cross discipline apprehension of what it is we want dreaming models to "explain."

[HOBSON ET AL.; NEILSEN; REVONSUO; SOLMS]

The new brain imaging studies by Braun et al. (1997; 1998) and the new lesion-dreaming research by **SOLMS**, and the solutions to the exclusive REM sleep-dreaming model proposed by **NIELSEN** and **HOBSON ET AL.** afford a timely opportunity to evaluate our neurocognitive conceptions of dreaming sleep. Because it is impossible to use our most powerful experimental methods to study dreaming, models of cognitive processes (ψ) in sleep are more dependent on knowledge of neurobiological processes (Φ) models of waking cognition. But neuroscientists' view of cognitive explanations of dreaming seem woefully simplistic, rather like a Manhattanite's picture of San Francisco from Manhattan in Steinberg's famous cartoon, where California is merely a small undifferentiated smudge on the other side of the Hudson River.

For some neuroscientists, a cognitive explanation is no more than a metaphor located in brain space and time. "Synthesis," "auto-activation," and "back projection" *imply* an explanation of the neural-cognitive process of dream construction that they do not deliver. It is the assumption by neuroscientists such as **HOBSON ET AL.** that these metaphors constitute explanatory models of cognitive processes that is at the heart of the controversy that HOBSON ET AL. attempt to resolve. In inviting us to respond to their revised AIM model, we thank HOBSON ET AL. for the opportunity to comment on this larger conflict between and "explanations" of what is, after all, the same process.

Despite their limitations, cognitive data provide us by far the most detailed information about dreaming. But we need brain scans, not to tell us that dreaming takes place in brain space and time, and not to confirm that dreams have visual imagery, but help us find out about those cognitive and affective characteristics and processes of dreaming that we do not *already* know. And that is a lot. Here, **HOBSON ET AL.** have provided an excellent account of what the recent imaging studies of Braun et al. (1997; 1998), Maquet et al. (1996), and Nofzinger et al. (1997). But as we move from cerebral cortex down to the brain stem, the specificity of the contribution of Φ to ψ processes becomes increasingly diffuse, and correspondingly less informative to both ψ and neurocognitive, $\psi(\Phi)$, models of dreaming. The revised pontine cholinergic-adrenergic model of REM-NREM sleep provided by HOBSON ET AL. provides an account for the location in *time* of most dreaming, and it shows how widespread cortical activation coupled with functional differentiation provides a *general* Φ basis for dreaming.

But the original Activation-synthesis and AIM models also claimed that PGO information and a cognitive "synthesis" process somehow constitute a $\Phi \rightarrow \psi$ explanation of dreaming. Although these proposals were, at the time, altogether plausible, they were found on careful study to be completely without empirical support. The detailed Φ account of the pontine mechanisms of the REM-NREM cycle has created, for neuroscientists and lay persons alike, the impression that the pontine-based Φ model of REM sleep constitutes also a model of dreaming – which it does not.

HOBSON ET AL. start with the thorny epistemological problem concerning what the mentation report can tell us about the actual dream. They review the familiar research literature that shows that the magnitude of every measure of sleep cognition is greater in REM than NREM, and acknowledge Pylyshyn's (1989) argument about the hazards of interpreting *anything* about private experience from verbal reports. These arguments notwithstanding, HOBSON ET AL. note that inasmuch as we all know from our private experience that we do dream, the failure to measure the private experience must be a limitation in psychological methodology. Then they proceed to take verbal report data pretty much at face value.

Several of our studies have successfully separated the dream report from evidence about the private mentation experience. Rosenblatt et al. (1992) showed that both log Total Recall and log Total Visual Imagery word counts of film clips shown before going to sleep were higher following REM than NREM awakenings. This evidence indicates that a portion of the REM-NREM differences in sleep mentation reporting difference may be attributed to superior recall and report processes operating *after* awakening from the respective states. Although I had proposed that the entire dream recall state difference might be attributed to enhanced REM recall in my 1983 REM-NREM word count paper (Antrobus 1983), after further research in our 1992 paper we clearly revised this suggestion, when we measured the magnitude of the recall-memory effect and showed that it was much smaller that the REM-NREM Total Recall effect. We reported that "a substantial part of the dream recall effect is the result of *pre*-awakening processes" (Rosenblatt et al. 1992, p. 223). **HOBSON ET AL.** fail to realize that even though there are real REM-NREM differences in mentation, some part of all REM-NREM differences is owing to a *REM-recall* advantage.

I also proposed in 1983 that the amount of dream content generated within REM sleep was more likely than in NREM "to be influenced by goals or motives established in the waking state" (p. 567), citing as evidence our finding that the dreamlike quality of REM reports dropped over 20 nights in the lab until it was indistinguishable from NREM reports (Antrobus et al. 1991). NREM dream reports were constant over time. This within-REM sleep motive to attend to dreaming appears to extend to the magnitude of visual imagery, holding total dream content constant. In the 1986 word count study (Antrobus 1986), although visual imagery words were strongly associated with the REM-NREM state difference, the visual effect was a subset of the stronger association REM-NREM association with total content count. But in the Antrobus et al. (1995) study where subjects not only gave a verbal mentation report but also matched their reported visual images to one of 16 photographs that varied by brightness and clarity, the proportion of visual imagery to recalled information increased substantially, so that the association of visual imagery with REM-NREM became *superior* to total recalled content. I assume that the emphasis in the report procedure on visual imagery motivated sleepers to attend more strongly to their visual imagery while asleep, and that this attention process operated more strongly during REM than NREM sleep – because as Braun et al. (1998) showed, both the limbic system and extrastriate visual cortex are activated in REM.

From this perspective, visual images and the dreamer's reaction to them may not be simply a set of fully-realized images, produced whether or not the dreamer is moved to notice them – as **HOBSON ET AL.** imply. Rather, the REM dream may be created by the process of attending to poorly structured information automatically generated (Antrobus 1991) in activated extra-pontine brain modules. In REM sleep, the attention process appears to be strongly determined by goal states established in the waking state. The memory of the dream is simply the functional residue of this attentional – construction process. This process of cognitive pen-

etrability may not be, as Pylyshyn suggests, a post-awakening confound, or as Freud suggests, secondary revision of the original dream, but rather the primary process by which the dream is produced. This attentional process is a metaphor for the processes by which activated brain modules attempt to "interpret" or, (make sense of) the noisy activity in neighboring modules. These metaphors are made explicit in my neural network attractor model, DREAMIT2 model (Antrobus 1991). Upon awakening, the interpretive process accelerates as the dormant verbal and meaning modules of the left temporal and prefrontal cortices become active. Objects and persons become named and explained during the sleep-to-waking transition. Some aspects of the dream become more reasonable, while other relationships that were "acknowledged as seen" in the sleep-dream, are now judged bizarre. Conversely, other features that were implicitly understood but not visually imaged in the dream, for example, "I was in my car," are, upon awakening, reported as visual images. That is, reporters cannot recall any visual features.

My original $\Phi \rightarrow \psi$ activation model rested on assumptions about state differences in cortical activation and sensory thresholds that were basically those of Hobson et al. (1978), namely that pontine mechanisms produced widespread cortical activation in REM sleep, but the characteristics of the mentation itself were the best indication of localization of cortical activation. Twenty years later, sharing with **HOBSON ET AL.**, the monist assumption that the state differences in cognitive activation are largely confined to those regions identified by the brain scans, a model of dreaming stands to learn much from maps of regional brain activation in sleep. Of course, locating a cognitive feature, such as image brightness, in time and brain-space does not constitute a Φ explanation of ψ, but as I shall show below, it can certainly help.

But the heart of the controversy lies elsewhere. With the original Activation-synthesis model to the present updated AIM model, Hobson and his colleagues have consistently presented the detailed pontine generator model that accounts for the *when* of REM and NREM sleep as an explanation of *how* the cognitive characteristics of dreaming are produced. But their $\Phi \rightarrow \psi$ causal claims are highly speculative, and indeed, often contradicted by waking $\Phi \rightarrow \psi$ models upon which they are based. Aside from the original and important contribution – that the pons determines, or is at least one determinant of, the widespread activation of the cortex during REM sleep, these assumptions about *how* the pons determines the features of dreaming are completely without empirical support. The most problematic of these models, which I discuss below, concerns the assumption that the "chaotic nature of the pontine auto-activation process" constitutes a basis for the bizarre features of dreaming.

Given the powerful association of the *when* of the REM sleep with the *when* of dreaming, many of us have come to equate the *how* of REM sleep, Φ , with the *how of* dreaming, ψ. **SOLMS** proposes that the temporal REM sleep = dreaming, $\Phi_{\text{REM-NREM}} \rightarrow \psi$, association is only indirectly related to the functional, or causal, $\Phi_{\text{FRONTAL-DOPAMINERGIC}} \rightarrow \psi$ relationship. That is: $\Phi_{\text{REM-NREM}} \rightarrow \Phi_{\text{FRONTAL-DOPAMINERGIC}} \rightarrow \psi_{\text{DREAMING}}$. If he is correct, and a substantial amount of data, including our 1995 diurnal rhythm paper, do support his position, the M = modulation vector of the AIM model loses its causal position in a $\Phi \rightarrow \psi$ model of dreaming.

This leaves us, once again, with a regional cortical-cognitive, $\Phi \rightarrow \psi$, model, where the pontine contribution of AIM is useful only for locating dreaming in time. As **HOBSON ET AL.** show, the accurate brain localization of activity in REM, NREM, and waking states (Braun et al. 1997; 1998), now allows us to attempt to map different features of dreams onto cortical and subeortical regions whose functions in the waking state have been identified – to the extent that these functions are invariant across states. But how far do, and can, these $\Phi \rightarrow \psi$ mappings take us toward an *explanation* of dreaming'? It is noteworthy that almost all $\Phi \rightarrow \psi$ assumptions proposed from brain localization observations in sleep consist of mapping *already known* characteristics of dreams onto well-established functions of brain regions. For brain mapping to tell us something about dreaming, however, it must identify new ψ characteristics of dreaming or force us to change existing ψ models of dreaming. The activation side of the Activation-Synthesis model did just that when it showed that the brain of the motorically-quiet REM sleeper was, in fact, quite active. The Braun et al. papers tell us much more. In particular, they show that REM sleep visual imagery cannot begin in the striate cortex, that many regions that participate cooperatively in waking are dissociated in REM sleep, and that the amygdala and limbic system that create the cognitive and affective characteristics of waking thought appear to contribute to the production of the REM dream.

The next question is, what can future Φ data contribute? Perhaps the most significant questions have to do with the relative influence of one cortical region over its neighbors, the magnitude of their interaction, and the ordinal character of these interactions. Both folk and psychoanalytic "interpretations" of dreams implicitly assume top-down, or what **SOLMS** calls "back projection" of information from meaning to visual-spatial functions, that is frontal to parietal and occipital locations. In 1991, I suggested that several other ordinal activation sequences are possible, particularly, visual-spatial → meaning → conation → motor (Antrobus 1991). That is, occipital-parietal regional may produce images that are interpreted by frontal structures, producing the surprise phenomena (" . . . and suddenly . . . ") of dreams. The subsequent imagined motor responses to these images then follow the same sequence they do in the waking state, except that, being imaged, they do not produce the same feedback as in waking, thereby eliciting additional cognitive and motor responses. Because the construction of the dream experience appears to continue even as the sleeper awakens and constructs a verbal description of his/her sleep experience, one cannot take at face value the ordered information in the report. Although it is well beyond the state of current brain imaging devices, such sequential brain scans could assist in the understanding of the ordinal causal effects in dream construction.

SOLMS takes this ordinal-spatial sequence for granted when he assumes limbic system effects are back projected to the visual cortex. But can, for example, a frontally-created goal – for example to seek out one's parent for protection – actually constrain the visual association and parietal cortices to construct an image of the parent? The assumption that dream motives can be "interpreted" from the sequence of the visual images is implicit and absolutely essential to the interpretation of dreams. Hasegawa et al. (1998) have shown that the retrieval of visual memories – in the temporal cortex – is under prefrontal cortex control. Braun et al. (1997) have shown that while the medial prefrontal cortex is more active in REM than NREM and wakefulness, the lateral orbital, dorsolateral and opercular prefrontal regions are *less* active. The evidence Φ is simply not clear enough at this time to determine whether top-down ψ, or back-projection, assumption is tenable. An equally plausible alternative to the top-down assumption is that image production is initially independent of limbic influence and that it precedes rather than follows the cognitive influence of the limbic structures. The latter might begin with the evaluation of the visual images ("Is that a friendly or unfamiliar face?") and be followed by imagined motor responses ("Shall I stay or run?"). More evidence on the prefrontal location of visual retrieval control, as well as the ordinal relation between these regions during dreaming will help to determine the strength of this key assumption about dream processes.

The next critical $\Phi \rightarrow \psi$ question is whether the pattern of dream features in a given state, such as REM sleep, is rigidly determined by the pattern of brain activation that is supported by subcortical structures, or whether the sleep state supports a general state-specific brain activation architecture that can be modified by the demands of the narrative dream sequence – as it is in waking perception – according to the demands of incoming information. For example, we assume that the dreamer's motor commands to run from an imagined strange man is accomplished in an activated motor cortex. Is the activation level of that motor cortex constant throughout the REM period, or is it *also* modified by the imagined

demands of the dream? Support for the latter position comes from our finding of a decrement in REM dream reporting over 20 nights of lab awakenings, and the increment in visual imagery when photo scales were employed – as reported above.

HOBSON ET AL. show how the $\Phi \leftrightarrow \psi$ relations of a large number of studies can be mapped onto the three dimensions of their AIM model. But as the high dimensionality of these findings, particularly in brain imaging, expands, they acknowledge that to map multidimensional relationships onto three non-orthogonal dimensions tends to weakens the precision of the representation of process. Each of the three AIM "dimensions" is, in fact, an array of multi-dimensional input, Φ, variables linked to another multi-dimensional set of output, ψ, variables that tell us little when they are concatenated into a single dimension. It is like mapping cities by their latitude. Although you can represent each city on a latitude line, you cannot locate it unless you also know its longitude. The real value of a model is to account in a systematic way for all the known evidence, and then use it to suggest new tests of the model until it can no longer represent the evidence and must be replaced by a better model. Although HOBSON ET AL. have indeed used AIM to attempt to account for a large amount of evidence and it has served them and us well, they have shown that it must now be superseded.

There are two problematic pontine-to-cortex issues that have persisted from the Activation-Synthesis to the AIM model. Brain imaging and lesion studies tell us what perceptual and cognitive processes are associated with particular cortical and subcortical regions. Nearly all of the evidence for these relationships comes from responses that are closely linked to antecedent *external* stimuli. Because our F $\Phi \rightarrow \psi$ knowledge is derived from the power of our stimulus-response experimental procedures, we, as scientists, tend to think of the mind-brain as an organ whose every process, every thought and image, is initiated by an external stimulus. This poses a problem for a theory of dreaming, as well increment in visual imagery when photo scales were employed – as reported above as much of waking mentation, such as daydreaming, which is also independent of external stimuli (Antrobus 1991).

HOBSON ET AL. have long maintained that the pontine-generated PGO spikes are the extra-cortical information source whose information constitutes the origin of the dream, and they have proposed far-reaching implications from the fact that this information is generated subcortically. Although, as HOBSON ET AL. point out, I have long and most recently (Antrobus & Conroy 1999) argued that any active cortical region will create organized pattern out of chaotic neural activity so that *no* extra-cortical information is necessary to account for dream imagery (Antrobus 1991), they continue to claim a pontine origin for the dream.

Their PGO claim is also inconsistent with their claim to $\Phi \leftrightarrow \psi$ isomorphism. The assumption that PGO spikes carry eye movement *information* to the brain during REM sleep rests on the assumption that these spikes transmit this information in the waking state, so that having acquired this information in the waking state, the cortex makes the *same* interpretation in REM sleep. But no one claims to *know* the function of PGO spikes in *waking* visual perception, so there is no waking model to apply to sleep. More problematic for the model, the relationships between PGO and REMs is quite different in waking and REM sleep. While PGO spikes are materialized in REM sleep they are not materialized in the waking state. Further, while PGOs mark the *termination* of REMs in the waking state (they may tell the occipital cortex that the foveal image has now stabilized and striate cortex may proceed to analyze it), in REM sleep PGOs are *concurrent* with REMs. Monaco et al. (1984, p. 220) concluded that the dramatic PGO activity of REM sleep PGO seems to be due to "disinhibition resulting from the arrest of firing of diffusely projecting aminergic inhibitory neurons of the dorsal raphe and locus coeruleus." For a fuller statement of this argument, see Antrobus and Conroy (1999). In short, there is no obvious way in which the cortex could use such PGO misinformation. Left without this PGO input, the Activation-Synthesis model, and now the AIM model, leave the cortex with nothing to *synthesize,* so that according to the AIM model, there can be no dream.

In their conclusions, **HOBSON ET AL.** continue to attribute bizarre cognition to chaotic pontine activation despite the fact that no experiments have supported this association, and furthermore, bizarreness mentation is frequently observed in states where PGO activity in minimal (Antrobus et a1. 1995; Reinsel et al. 1992). It is more likely that local chaotic neural activity represents neural attractors that cannot settle on a solution and so communicate nothing to neighboring neural locations, rather than, as HOBSON ET AL. propose, that chaotic neural activity in the pons produces bizarreness in the cortical dream process. Although there may be many ways to produce bizarreness in dream, the Braun et al. (1998) conception of the REM brain as one of dissociated regions of activation suggests a new possibility. Regions that collaborate in waking perception depend on each other for error correction so that when they are forced to function independently, as in acquired deep dyslexia, they produce strange errors, such as naming an apricot, a peach (Hinton & Shallice 1991). It is well known that the individual cerebral hemispheres function differently when separated and fully linked. Caution must be taken in assuming that each of the dissociated regions of the dreaming brain carries out the same functions that it does in waking – especially as they operate without the considerable assistance of the language cortex (Braun et al. 1998).

The dream-no dream dichotomy problem illustrates the rule that a neurocognitive theory cannot be better than the validity of its worst measure. The concept of dream comes to us from the vernacular. It is multi-dimensional: visual features of color, movement; it is thematic, bizarre, conative, and, at times, affective, and, more, verbal with a sense of self reflectiveness and control. Questions about across-sleep-state differences in dreaming carry assumptions about whether the pattern of these features is sustained across states or whether, for example, dreams in some state are more visual and, in other states, more verbal. Even if the underlying *pattern* of features is intact across states, do some features appear at low levels of cortical activation while the rarer features occur only at high levels of cortical activation (Antrobus et al. 1995)?

The **SOLMS** and **NIELSEN** analyses that are based only on the report of dreaming, not even scaled by magnitude, tell us far less about the questions they address than if they had used a multi-dimensional dependent variable scaled by magnitude. Forty years ago, Kamiya (1961) showed that the answer to whether dreaming occurs in both REM and NREM sleep or only in REM was a function of where, on the magnitude of dreamlike mentation scale, one draws the dividing line between dreaming and nondreaming. Because the point is absolutely arbitrary, it prejudices the answer to any question, including those of SOLMS and NIELSEN, about the relation between cognitive and neural processes.

This measurement problem compounds the fallacy of assuming that discretely-defined biological states imply discrete neurocognitive processes. Since the original Aserinsky and Kleitman (1953) discovery that proposed a discrete distinction between REM and NREM sleep, investigators have implicitly assumed that whatever Φ processes produced ψ effects must also operate in an all or none fashion. It is gratifying, therefore, that both **NIELSEN**'s alternate model and **HOBSON ET AL.**, after years of prodding by Foulkes, have agreed that the sources of dreaming in REM and NREM sleep may be regarded as operating respectively in a high and low, rather than on-off mode. This position is consistent with **SOLMS**'s model, except that the underlying Φ source of dreaming , ψ, is only indirectly tied to REM sleep.

Now that Braun et al. (1998) have provided us with evidence about the modular activation of different cortical regions in REM sleep, and **SOLMS** has shown that some forms of dreaming are accomplished in other sleep and waking states, and Antrobus et al. (1995) have shown that dreaming is also associated, during sleep, with the rising phase of the diurnal wake-sleep rhythm, we know that different brain structures may support different features to the cognitive dream. But we cannot determine the role of any

given brain module unless it varies *independently* of the others. Conversely, if the activation of each brain region that participates in dreaming sleep covaries with the activation of the other regions, then even though brain activation is multimodal, it is nevertheless, one dimensional. And if it is one dimensional, we should expect the features of the cognitive dream to also be one dimensional, that is, they should covary – even though they consist of qualitatively different feature classes. For this reason, the lengthy quantitative-qualitative discussion by **HOBSON ET AL.** does not rise to the complexity of the questions they are trying to address. All qualitative differences are ultimately quantitative differences. The questions of interest are how the multidimensional quantitative ψ patterns map onto the multidimensional quantitative Φ patterns – as described by brain state-specific maps.

SOLMS and **NIELSEN** avoid these issues by implicitly assuming that Φ state differences in ψ all lie along a single dimension, namely dreaming or simply recall of any content (NIELSEN). That is, what is called dreaming in one state is assumed to have the same pattern, or profile of features, as dreaming in another. All features rise or fall together so that variation in the magnitude of dreaming describes the joint variation in all features. By restricting their measure of ψ to one dimension their models are insensitive to possible qualitative differences that many vary across states of Φ. For example, suppose that mentation is more verbal in NREM and more visual in REM sleep, but that when sleepers are asked, "Were you dreaming?" they answer in the affirmative in both cases. One would falsely conclude that the different Φ states produce the same quality of mentation, that is dreaming. This criticism is not evidence against their positions, but it renders less convincing SOLMS's conclusion that mentation produced in different Φ states is qualitatively the same.

At this point in time, however, we have little evidence to throw out the one dimensional Φ ↔ ψ model. **HOBSON ET AL.** attempt to address the question but since none of their analyses consider the *relationships among* the cognitive variables, their review of the literature simply does not speak to the issue. Rather, they report whether there are quantitative differences in each variable taken separately. Except for a few studies in our labs, tests for across-Φ state differences in ψ *patterns* have not been carried out by any investigator, and oddly, our tests are not mentioned by HOBSON ET AL. That all variables increase in REM sleep does not speak to the question of whether the *rate of increase* is constant across all. In the absence of that evidence, one can say nothing about the dimensionality of Φ ↔ ψ dreaming relationships.

The only paper I know of that explicitly tests whether the pattern among cognitive relationships differs across two different Φ state *changes,* e.g., REM versus NREM, is our 1995 paper. It shows from the *pattern* of cognitive features that best discriminates between REM and NREM sleep reports is not different from the pattern that discriminates between two points in time along the rising phase of the diurnal activation cycle. That is, the pattern among the cognitive measures that describes the diurnal effect is unchanged in both REM and NREM sleep. It does not differentially magnify the REM or NREMsleep effect; rather it adds *equally* to both REM and NREM sleep mentation. It is important to note that this is not, as **SOLMS** suggests, a NREM effect, but rather an independent pattern of Φ → ψ activation associated with the diurnal sleep-wake cycle.

In closing, I would like to say that the evolutionary hypothesis of the function of dreaming proposed by **REVONSUO** appears compatible with existing knowledge about mammalian evolution. My own disposition is to assume that the evolutionary value of Φ structures is determined by multiple interacting selection factors. We still have no hard evidence that dreaming has, or ever had, any behavioral function. If it did, it remains to be determined whether such value is incidental to some other more direct evolutionary function of REM sleep and others states (**SOLMS**). The study of dreaming has been driven by our curiosity about its dramatic strangeness rather than its function, and I think, many investigators feel that the attention they give to dreaming would be better justified if it had a well documented functional value. REVONSUO reminds us not to lose sight of this question.

Dreaming as an active construction of meaning

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Abstract: Although the work of Revonsuo is commendable for its attempt to use an evolutionary approach to formulate a hypothesis about the adaptive function of dreaming, the conclusions arrived at by this author cannot be fully shared. Particularly questionable is the idea that the specific function of dreaming is to simulate threatening events. I propose here a hypothesis in which the dream can have a different function. **[REVONSUO]**

REVONSUO deserves credit for exploring the possibility that dreams may have a specific adaptive function. His attempt to understand that function, by unifying in an evolutionary approach much of knowledge of both the phenomenological and neurophysiological aspects of our dreams, is laudable. That said, it seems some critical observations must inevitably be voiced.

Beyond the more or less marked differences among the various authors, the scientific debate on the nature of dreams has historically consisted of two opposing positions, one holding that the dream is a mere random byproduct of REM sleep physiology, the other that the dream is an organized subjective experience that performs a specific function. **REVONSUO** reintroduces this opposition in an original way aligning himself with the second position. I will suggest that these two positions need not be opposed.

The dream, notwithstanding the fact that it is based on neurophysiological processes that randomly activate particular cerebral structures, can nevertheless be understood as a structured subjective experience with a specific function. Just as our waking perceptions are amply guided by our expectations and by our interpretative model of the world, so in dreams non-structured stimuli activated by random neurophysiological processes take on meaning for dreamers who superimpose their own interpretative schemes and ways of conferring meaning onto the experiential flux of this stimulation. It is in this sense that the dream is the product of an active construction of meaning.

The dream can be understood as the guardian of sleep, but not in the sense intended by Freud (1900/1950). According to the founder of psychoanalysis, the dream eliminates the emotional stimulus that originates from an unconscious desire of the dreamer, by satisfying it in a hallucinatory manner. In the hypothesis that I am advancing, the processes of dream-construction preserve the sleep from the random cerebral stimulation that accompanies it. If it were not possible to give meaning to this stimulation, thanks to the formation of dreams, it would not be possible to sleep. Without dreams, the experiential state associated with sleep would be similar to a psychotic state that lacks precisely the possibility of assigning meaning to consciously perceived events. The dream can be seen as the solution to an adaptive problem: How to prevent the random neurophysiological stimulation accompanying sleep from impeding the organism in restoring itself. I suggest that this was the adaptive function for which dreaming was selected in our evolutionary history.

Dream images are not random, even while the neurophysiological processes that lead to them are. This position is distinct from that of Hobson (1988b) and Solms (1997a), because unlike them it explains why the brain generates the images that constitute the dream content, and why dreams have particular contents and narrative plots. Dreaming is the way our cognitive and signifying schemes give sense to stimulation that is in itself nonsense. The dream is therefore the reflection of our cognitive organiza-

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tion. Furthermore, this makes the dream a useful clinical and research tool with a specific psychological and cultural function that complements biological function.

From the above, it is apparent that I share with **REVONSUO** both the idea that the contents of a dream are not random, and the hypothesis that the dream has a particular adaptive function. I particularly embrace the idea that dream content is consistent with the original evolutionary environment. My essential divergence is with his idea that the dream has the specific function of simulating threatening events, and of rehearsing threat perception and threat avoidance. In reality, the process of dream construction could have the more general function described above; it would not be surprising that in the exercise of this function, there should sometimes be threat simulations as well, and that reading, writing, and calculating should be absent. As highlighted also by REVONSUO, this depends on the fact that our cognitive architecture, and hence the processes that are basic to the production of dreams as well, are the products of our evolutionary history.

Regarding the experimental studies and data cited by **REVONSUO** in support of his hypothesis, in particular proposition 2: First, the fact that two-thirds or more of the emotions expressed in dreams have negative connotations does not necessarily signify that these emotions are linked to events perceived in the dream as threats. In the waking state, negative emotions (for example, anger, boredom, disgust, and spite) manifest themselves independently of whether or not the one who experiences them is involved in a threatening situation. These emotions may present themselves in contexts very different from those hypothesized by REVONSUO. It is accordingly risky to cite these studies as indirect evidence that dream content is biased towards simulating threatening events, inasmuch as it leads to conclusions that are unjustified in the light of what we know from the psychology of emotions. Regarding "Misfortune" dreams, if it is true that some studies demonstrate the preponderance of these with respect to "Good Fortune" dreams, it is also true that Misfortune dreams often present situations in which events are uncontrollable or inevitable. In what way do dreams of this type help to avoid the real world threatening situations of the waking state?

Dreaming is not exclusively a specialized experience for the simulation of threatening events; it is the active construction of meaning in the state of sleep, for cerebral stimulation that has none. In this light, the simulation of a threat has its place as one aspect of our experiential lives, but not the only one. The alternative approach delineated here takes all dreams into account regardless of their content.

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Internally-generated activity, non-episodic memory, and emotional salience in sleep

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Abstract: (1) Substituting (as Solms does) forebrain for brainstem in the search for a dream "controller" is counterproductive, since a distributed system need have no single controller. (2) Evidence against episodic memory consolidation does not show that REM sleep has no role in other types of memory, contra Vertes & Eastman. (3) A generalization of Revonsuo's "threat simulation" model in reverse is more plausible and is empirically testable.

[HOBSON ET AL.; SOLMS; REVONSUO; VERTES & EASTMAN]

One dream controller is as bad as another [Solms]. The **SOLMS** target article argues persuasively that not all dreaming can be uniquely identified with activity in the REM generating areas of the brainstem (as was proposed in some earlier research). However, substituting the ventromesial forebrain as the single "controller" of dreams, as SOLMS proposes, seems like a step backward, even if lesion studies show that the area is important or even crucial for dreaming. As neuroimaging studies make clear, dreaming is a complex process occurring in a system of multiple interacting units distributed across the brain. In such a distributed system, lesion studies cannot provide any means for deciding on a single location as the controller, because in fact there need be no such clearly-defined module. (Thus in SOLMS's terms, no single brain area need be able to "activate, generate sustain, and terminate" all dreams.)

A more productive approach might be to focus on the essential aspects of dreaming, and only then to consider how the various brain areas might contribute to this process. The most obvious feature that distinguishes dreaming from waking is that dreaming relies on internally-generated inputs (Bednar & Miikkulainen 1998), while waking mentation can be traced, at least in part, to data from the senses. There can be many possible sources of this endogenous activity during sleep, all of which could be considered to "cause" dreaming in some sense. Among these, brainstem REM generators do seem to be "a regular and persistent source of cerebral activation during sleep," as SOLMS himself acknowledges. Thus there is no mistake at all in focusing on the REM state instead of searching for a single anatomical site to unlock the secret of dreaming.

A second minor point from the **SOLMS** article is worth mentioning in passing, because it involves a report most likely published after his article was written. SOLMS speculates that the cortical back-projections which appear to underlie mental imagery do not project as far back as primary visual cortex, which if true could explain why V_i shows decreased activity during REM sleep. However, back projections to V_i certainly seem to be present anatomically, and Kosslyn et al. (1999) have shown that mental imagery can in fact be measured in V_i, albeit only of a certain kind involving specific locations on the retina. Thus a theory of dreaming would have to explain why the back projections to V_i do not typically take part in dreaming; it clearly is not because the connections do not exist.

Non-episodic memory does not require consciousness [Vertes & Eastman]. Strong and valid reasons for discarding the idea that explicit episodic memories from the hippocampus are somehow consolidated during REM sleep are presented by **VERTES & EASTMAN.** However, in several instances they go much further than their cited data would support by concluding that "REM sleep serves no role in the processing or consolidation of memory." They appear to make this claim because of their unusually restrictive definition of memory, in which "sleep involves basic biological functions and memory requires consciousness."

Certainly, some types of memory are intricately linked with consciousness, in particular the episodic memory usually proposed for consolidation. However, memory is a very broad term that is applied to an enormous variety of wonderful phenomena, ranging from the strength of the connection between two Aplysia neurons (or even the state of charge of a certain capacitor in a computer chip), to vastly more complex processes. Rather than being a specific byproduct of consciousness, memory seems to be quite distributed, localized, and ubiquitous in the nervous system (Gilbert 1998). Indeed, it is arguably as much of a "basic biological function" as sleep is.

From this larger view, there is currently no reason to conclude that REM sleep serves no role in non-episodic memory processing, despite the lack of clinical impairments from REM deprivation. Given widespread plasticity combined with the strong activity found in many brain areas during REM sleep, the burden of proof is actually in the other direction: unless one can show a plausible mechanism by which the process of learning has somehow been disabled at each local synapse without abolishing activity, one must assume that the activity has the potential to modify those synapses. Plasticity of this sort would presumably underlie non-

episodic memories, such as procedural/skill memory (Smith 1995) and limbic-system emotional associations (Maquet et al. 1996); such effects could be difficult to measure clinically. It is even possible that the episodic and working memory areas quiet during REM sleep might be suppressed precisely so that they would not undergo plasticity while the rest of the brain is processing other types of memory. Such processing could be very important to proposals such as REVONSUO's.

How solid is the evidence that dreaming is an organized simulation of the world? [Revonsuo] Even though I share with REVONSUO a suspicion that dreams are not as random as Hobson and McCarley (1977) proposed, I do not agree that his particular line of argument "shows beyond any reasonable doubt that dreaming is an organized simulation of the perceptual world." He correctly anticipates most of the argument's weaknesses, but a few important ones have been overlooked. For instance, he cites differences in narrative richness between dream reports and subjective reports from isolated electric stimulation in temporal cortex. However, these conditions are not comparable, since we know that REM sleep activation is a large-scale phenomenon with at least some spatial and temporal structure. An appropriate experimental control would thus require widespread and ongoing artificial brain stimulation, coupled with temporary deactivation of the same frontal-lobe areas suppressed in REM sleep. If such an experiment were practical, it might demonstrate that dream-like mentation could be generated from random activation; meanwhile we must at least consider it possible.

Similarly, when REVONSUO quotes Foulkes as saying "The simulation of what life is like is so nearly perfect, the real question may be, why shouldn't we believe this is real?" (Foulkes 1985), Dennett (1991) would probably point out that (1) there need be no "simulation" of life in dreams separate from the experiencing of the dream, and (2) any brain activity contributing to the experience need not be perfect or realistic at all, as long as the processing machinery treats it as realistic. Indeed, as REVONSUO acknowledges, there are many bizarre, non-realistic features of dreams that are obvious only in retrospect.

What makes threat simulation so special? [Revonsuo] REVONSUO's general hypothesis for mammals, "Dreaming rehearses species-specific survival skills," seems much more defensible than his narrow version for humans: "dream consciousness is essentially a mechanism for simulating threat perception and rehearsing threat avoidance responses and behaviors." (Unfortunately, the version for humans is the one that is most clearly distinguishable from similar earlier theories, such as Winson 1990 and Jouvet 1978.) Threat simulation would seem primarily useful for species which are typically prey rather than predator, and humans clearly serve in both roles. Since REVONSUO acknowledges that "not all dreams are threat simulations," it seems arbitrary to assume that other commonly-cited dreams (such as flying) are mere side-effects.

Given that threats are not the only situations biologically important to humans, ancestral or otherwise, a much more intuitive hypothesis would be that dreams simulate biologically-significant situations in general. In humans that would presumably be approximated as emotionally-salient situations, in the absence of some other internal criterion for what is biologically significant. Threats would just turn out to be a particularly well-represented example of such situations, rather than the primary purpose of the system.

In making the case for threat simulation, REVONSUO dismisses most previous proposals for dream function because they do not systematically analyze dream content. Dream content analysis may be very helpful for formulating hypotheses, but by itself it cannot offer any definitive criterion for preferring one hypothesis over another because of the enormous and largely unknown biases involved in subjective dream content reports. Even during waking life, we focus disproportionately on emotionally salient events when reporting narratives, as a quick glance at the evening news or the movie listings will attest. Given the particular emotional salience of threatening events (again, witness the bizarre popularity of horror films), finding threatening events over-represented in reports does not necessarily indicate that they are over-represented in dreams, and finding them over-represented in dreams does not necessarily mean that they are specifically generated. At a minimum, to use dream content analysis one must compare dream reports with waking reports as opposed to actual waking life.

REVONSUO also seems to go too far in emphasizing the lack of adaptive function for threat avoidance in modern life. Although daily life for many people may be quite dull, certainly those who have served in war, who have lived in the inner city, who have played competitive sports, who have been assaulted, who have encountered a vicious dog, and so on are quite familiar with threatening situations. Despite being relatively safe, people still die every day owing to jealous lovers, natural disasters, and many other causes which have been presumably unchanged for millennia. And thus being alert and ready for quick, decisive action in threatening situations is surely not "obsolete," even if no longer as important as it once was.

Revonsuo's dream model: Why not have emotion precede situation? From data showing only a correlation between emotions in dreams and the situations in which they are experienced, REVONSUO assumes a specific direction of causality, that is that unpleasant dream situations cause the negative emotions through "threat recognition." However, common dream features such as emotional continuity in the face of narrative discontinuity (Seligman & Yellen 1987) would suggest precisely the opposite hypothesis: the brain may somehow activate a certain emotion, which prompts recall of events historically (or perhaps genetically) associated with that emotion.

Reversing the sequence in this way can simplify a key step in REVONSUO's neural model of dream generation. As originally formulated, his model requires some unspecified mechanism for initially selecting memories by their emotional salience. Such a mechanism is difficult to imagine because it would supposedly operate independently of the current emotional state of the brain, since limbic system areas like the amygdala are activated only later in his process. The model also requires another unspecified (and difficult to imagine) mechanism for deciding "when potentially threatening content is present in visual awareness," since no feedback for this judgment is available in the model.

Reversing the sequence leads to a simpler and more concrete approach similar to HOBSON ET AL.'s AIM model. This model would start with activation of an emotional state in the limbic system along with sparse random activation of the visual system. The initial activation would automatically activate (more or less at random) one or more emotionally salient episodic memories of waking experience. Such memories would presumably include specific patterns of activity in the sensory association areas and in motor cortex. In the simplest case, the process of activation could simply strengthen the already-present emotional association between the activated units through a simple connection-specific mechanism like Hebbian learning. This model would merely enhance emotionally salient memories at the expense of others which could be desirable.

Making this model slightly more extravagant to compare with the one proposed by REVONSUO, it could instead generate specific (rather than random) coarsely-determined visual input and/or specific motor cortex activations (Bednar, unpublished research proposal, June 1999). Such a system would amount to supervised training of an association between particular inputs (e.g., threatening situations, as in REVONSUO's model), particular outputs (e.g., fighting or fleeing), and a given emotional state (e.g., fear). The inputs, outputs, and emotional state would all need to be genetically specified somehow, which is what makes this hypothesis more extravagant than the simpler one above. However, the extravagance is no greater than REVONSUO's, and this model does not require the presentation of hypothetical scenarios on the input while hoping for the correct response from a brain that has no proposed feedback signal to guide the "threat recognition" process.

Dreams have meaning but no function

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Abstract: Solms shows the cortical basis for why dreams reflect waking concerns and goals, but with deficient volition. I argue the latter relates to Hobson et al.'s process I as well as M. A memory function for REM sleep is possible, but may be irrelevant to dream characteristics, which, contrary to Revonsuo, mirror the range of waking emotions, positive and negative. **[HOBSON ET AL.; NIELSEN; SOLMS; REVONSUO; VERTES & EASTMAN]**

SOLMS shows how dreams are dependent on cortical areas concerned with appetitive interactions with the world, which accords with previous work on dreams incorporating emotionally meaningful material (Hartmann 2000b), but it is interesting that dreams, which express our goals and concerns, seem also to have attenuated volition. **HOBSON ET AL.** also make the point that there is lowered volitional control in dreams, while referring to there being a current debate on its extent. More detail would be welcome on SOLMS's finding that lesions of the dorsolateral prefrontal cortex, involved with volitional control and self-monitoring in waking, do not have an effect on dreaming, because of the difficulty of measuring any such changes in volition and self-reflectiveness in dreams. Although there is evidence for deficient volition in dreams (e.g. Blagrove 1996; Kahan et al. 1997), Bargh and Chartrand (1999) show that there is a general lack of volition and self-reflection in waking life, and so goal seeking may be activated without conscious volition in both states. Furthermore, for waking life, Kirsch (1998) shows that behavior often follows the automatic activation of response sets, and that volition is often an erroneous attribution after an action. I suggest that in dreams the attenuated volition and self-reflection, and lack of surprise at bizarreness, may be a result of the lack of unexpected stimuli that are contrary to expectations, because, in an extension of their use in waking experience, response expectancies (Kirsch 1985) may be used to produce the successive contents of the dream.

For dreams the attenuated volition and self-reflection may thus be a result of the lack of feedback from an independent environment (which in waking life can cause surprises). That is **HOBSON ET AL.**'s process I, the sensory gating, although with the possibility, as HOBSON ET AL. state, that deficiencies in memory during dreaming are also involved. I would ask whether process M is any more than a measure of memory consolidation (their "memory/amnesia dimension"). For example, from Reinsel et al. (1986), mean total recall count for waking daydreams under minimal stimulation was 68 words, whereas REM recall was 34 words; such differences, although significant, raise the question of whether the low REM recall is a matter of low memory consolidation, memory retrieval in dreams, although usually outside conscious control, being quite resourceful and capable of some complexity.

NIELSEN makes the claim that NREM dreams may be dependent on the next REM sleep phase, as well as on the previous one, and of interest would be whether the window might have a different duration on either side of a REM sleep phase. I am concerned that some psychological REMS/NREMS differences may appear by chance. For example, in Foulkes and Rechtschaffen (1964) the MMPI L scale correlates with REM recall but not NREM, as **NIELSEN** reports, yet the 21 other MMPI scales had no relationship with REM or NREM recall. NIELSEN notes that after dream length is controlled for there are REM/NREM differences in visual imagery word count, number of characters, and self-involvement, and refers to these differences as "qualitative" although these differences are really quantitative, as **HOBSON ET AL.** state, and this would hold whether or not such differences could be eradicated by other methods of controlling for dream length. If the view that there are two systems of dream production arises because of the two physiological stages of sleep, should we be postulating a third system because mentation with dream-like characteristics can also be found briefly in waking daydreams (Foulkes & Fleisher 1975)? Parsimony suggests one system, with dream production turned on more frequently in REMS than NREMS.

VERTES & EASTMAN reason that as dream material is chaotic, and poorly remembered, then "the transfer of information in REM is not orderly," but these two processes may well be entirely independent. They argue that attention is needed for learning, but is absent in REM sleep, but even if attention is required in the first stages of learning, at input, and even that is questionable (cf. Lewicki et al. 1988), it would not be required at later stages of the storage of information, which would occur automatically and outside consciousness, as it does when one is awake. That Wilson and McNaughton (1994) found increased firing in hippocampal place cells during SWS after learning may indicate some effects of sleep on learning, as do the findings of Ambrosini et al. (1995) that SWS and REM sleep both have a memory consolidation function.

In response to their data on the reduction of REM sleep with antidepressants, it may be that if REM sleep is abolished then its functions can be fulfilled when awake; I am not clear whether the position **VERTES & EASTMAN** are attacking is that REM sleep has a function that cannot be fulfilled when awake, or that consolidation of memories can occur in REM sleep as well as when awake. There is also the problem of the alerting effect of REM deprivation (Nykamp et al. 1998) which could hide memory deficits, and in studying the effects of antidepressants, there are also problems if the baseline is the depressed state, during which there may also be memory deficits.

In their theory of REM sleep preparing the sleeping animal for waking, **VERTES & EASTMAN** do not account for the large amount of REM sleep in the fetus, for REM rebound, or for the possibility that REM sleep is evolutionarily earlier than SWS (Siegel 1997). The muscle atonia of REM sleep indicates against this arousal function, as would any confusion of waking up from a REM dream, and any difference in alertness between waking from REM sleep and from deep sleep may be too small to support this hypothesis. The comparison with slow recovery after anesthesia is not helpful, as there are biochemical reasons for this.

REVONSUO's theory of the function of dreaming has similarities to Hartmann's (1996a) theory of the contextualizing of emotional concerns, with the addition of psychomotor practice to Hartmann's emphasis on forming connections in memory. Both authors use the extreme example of nightmares to argue about a function for dreams. REVONSUO asserts that in dreaming we "rehearse threat perception and threat avoidance," with the possibility, when threats are absent, of dreaming of emotionally charged memories, current concerns, or other mundane sources. The article is predicated on pain and fear being adaptive, and holds that dreams depict "ancient concerns" and have as "default values" the simulation of "violent encounters with animals, strangers, and natural forces, and how to escape from such situations." Which explains why REM behavior disorder frequently involves threatening actions, but on evolutionary terms would not positive reinforcement (e.g., dreaming of green fields, flowing water, success or friends) be as important as negative warnings? REVONSUO cites Hartmann's (1998, p. 73) finding that "when people experience a happy event, they are more likely to dream about problems associated with it than the pure happiness of the event itself," but Blagrove and Price (2000) found that happy skilled individuals tend to have happy dreams, and Kallmeyer and Chang (1998) found that particular positive (e.g. joviality, self-assurance) and negative (e.g. fear, sadness) waking emotions were associated with individuals who have positive and negative dreams, respectively.

REVONSUO claims that dream content is biased towards negative elements, yet although Strauch and Meier (1996, pp. 92–93) did find that negative emotions appear twice as often as positive ones, joy was the most common specific dream emotion (followed by anger, fear, interest, and stress), and of 500 REM dreams, as **REVONSUO** cites, general mood was more likely to be positive than negative. Strauch and Meier conclude that dreams are not pre-

dominantly influenced by fears, dismay or stress, but frequently display well-being and pleasant experiences (p. 94). Furthermore, Schredl and Doll (1998) found that although dreams rated independently had a preponderance of negative tone, when rated by the dreamer the ratio of positive to negative moods was balanced.

REVONSUO claims that reading, writing, and arithmetic do not appear in dreams because they are cultural latecomers, but they are usually unemotional activities, and I see no reason why writers would not dream of writer's block, or some other non-reproductive aspect of their professional life. It may thus be too narrow to claim that "the biological standard is the only standard of functionality." Also, **SOLMS** has dreams incorporating emotional and motivating stimuli, as with the traumatic events described by REVONSUO, but this does not show that the incorporation is functional, it may be a by-product of a system that incorporates positive and negative emotional stimuli and motivations into daydreams. However, **HOBSON ET AL.** remind us that dreams are highly penetrable cognitively, so the belief that dreams are concerned with threats may itself lead to such dreams occurring.

The test of this theory of an over-representation of threat simulation in dreams would surely be to find threat themes in people who are not hunter-gatherers or traumatized, because a theory of dreams as incorporating emotional events in general would similarly predict "high levels of survival themes, threat simulation and animal characters" in the dreams of hunter-gatherers. Rather than showing a mechanism of dream function this just shows the effects of being in those conditions. I note, however, that **REVONSUO** does state that today's changed environment may mean that dreams do not now have a function. The author is right however, that some type of selection is going on in the formation of dreams, but to study this selection, the frequency of threatening events in dreams should be compared to their frequency in autobiographical stories, or creative stories, rather than in real life itself.

REVONSUO reports changes in sleep due to PTSD but in the Williamson et al. (1995) paper cited dream variables were not measured, and in Ross et al. (1994) PTSD participants had more REM sleep and greater REM density, but of the 11 PTSD participants just one experienced an anxiety dream. Lavie et al. (1998) found that although PTSD patients had higher awaking thresholds than controls, and more aggressive and hostile dreams, the PTSD and control groups did not differ in dream recall frequency, and Dow et al. (1996) found no differences in dream recall or report length between Vietnam veterans with PTSD and major depression, veterans with depression alone, and veterans with neither PTSD nor depression, and for all groups dream anxiety was no more than mild. Anyway, although stress can increase nightmare frequency (Chivers & Blagrove 1999) and trauma can be represented in dreams and nightmares (Barrett 1996), the correlation of nightmare content with trauma, or even change in nightmare content with recovery from trauma, does not mean that dreaming has a causal role in that recovery (Blagrove 1992a).

REVONSUO states that on average one out of two animals in children's dreams are untamed wild animals, and "the proportion of domestic animals increases and that of wild animals decreases with age." And yet Foulkes (1985, p. 122) found that at ages 3–5 years dream animals "tended most often to come from two classes: domesticated farm animals or relatively familiar and unaggressive undomesticated animals" and Foulkes does not mention that children at ages 5–7 years dream of aggressive animals. Against the claim that children are likely to have infrequent actual experiences of animals, a source of there being so many animals in children's as opposed to adults' dreams may be present-day fairy tales and cartoons, rather than ancestral fears; why children are so interested in animals is then another matter: even if that interest has evolutionary origins dreaming about animals may not do so. Furthermore, Foulkes (1985) gives evidence to interpret strangers in children's dreams as a failed attempt to represent someone who is known, rather than an actual stranger, which is problematic for REVONSUO's claim that strangers in dreams may result from ancestral conditions in which encounters with strangers were potentially life-threatening. **REVONSUO** asks why are male strangers our enemies in dreams, given that "present-day encounters with unfamiliar males in the waking life are not predominantly aggressive," yet surely such a view of male strangers is common in TV and newspapers, and hence is salient to us, even if exaggerated and unrepresentative of reality.

It is unclear why a dreamt simulation should help in the "perceiving and recognizing" of threatening situations. This surely requires real stimulation, and the analogy with flight simulators (n. 17) does not hold, because in using them the operator is highly conscious and attentive. **REVONSUO** argues that lack of consciousness is no problem to the function of dreams in the model, and if dreams have the role of providing practice for actions then this is true, but what of learning flexibility in actions'? Furthermore, the complex movements found to occur in cats during REM sleep without atonia have also been found to occur during wakefulness (Morrison & Bowker 1975), so Jouvet's widely cited result from sleeping cats without atonia may not be evidence for dreaming as motor practice.

REVONSUO is rightly not convinced by theories of "the psychologically adaptive function of dreaming," but dreams could be psychologically expressive of positive and negative emotions, as in work on the measurement of insight due to dream interpretation (e.g. Hill et al. 1993), and on the incorporation by divorcees of their former spouses (Cartwright 1991). It may be that day dreaming and imagery were selected for in evolution, with dreaming being an epiphenomenon. **REVONSUO**'s argument against dreaming having resulted from the evolutionary selection of day dreaming is that dreaming has different features, such as in level of volition and type of moods, but these differences do not show that dreaming is not dependent, physiologically and evolutionarily, on day dreaming and on the ability to imagine and to have imagery.

Sleep, not REM sleep, is the royal road to dreams

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Abstract: The advent of functional imaging has reinforced the attempts to define dreaming as a sleep state-dependent phenomenon. PET scans revealed major differences between nonREM sleep and REM sleep. However, because dreaming occurs throughout sleep, the common features of the two sleep states, rather than the differences, could help define the prerequisite for the occurrence of dreams.

[HOBSON ET AL.; NIELSEN; SOLMS; REVONSUO; VERTES & EASTMAN]

SOLMS provides an excellent summary of evidence that the REM sleep and dreaming are dissociable states. Although all authors in the present issue seem to agree that dreams occur throughout sleep, the temptation to associate them with REM sleep lingers on. Thus **HOBSON ET AL.** attempt to account for dreaming in nonREM sleep by invoking an "admixture of REM-like phenomena within stage 2," **NIELSEN** proposes the existence of "covert REM sleep processes" during nonREM sleep and sleep onset, and **VERTES & EASTMAN** state that "the mental/cognitive content of REM sleep and sleep is dreams." The conceptual dissociation of REM sleep and dreaming is still incomplete.

Dreaming occurs throughout sleep: it may be useful to focus on features that are common to both sleep states and different from waking. In PET scans they consist in the deactivation of hetromodal association areas in frontal and parietal cortex (Andersson et al. 1998; Braun et al. 1997). **REVONSUO** refers to **SOLMS**'s view that dreams are "bizarre hallucinations that weakened frontal reflective systems mistake for real perception." Our recent study confirmed the deactivation of frontal areas in stage 2 and stage 4

of nonREM sleep as well as in REM sleep (Finelli et al. 2000). Another common feature appears to be the relative activation of unimodal cortical areas. In our study, parts of the occipital neocortex were more activated in stage 4 than in stage 2, and unimodal areas in the visual and parietalcortex were activated in REM sleep relative to waking. Hofle et al. (1997) showed for some of these areas a positive covariation with delta activity, an index of REM sleep intensity, and Braun et al. (1997) reported an activation of unimodal visual cortex in REM sleep.

The selective "deactivation" of frontal cortex described in PET studies seems to have an electrophysiological correlate. Thus in the initial nonREM sleep episodes, EEG slow wave activity shows predominance in the fronto-central derivation relative to caudal derivations (Werth et al. 1996; 1997). Moreover, brain mapping during and after prolonged waking revealed that frontal areas exhibit the largest increase of slow-wave activity in nonREM sleep and of theta activity in the waking EEG (Finelli et al., unpublished results; see also Cajochen et al. 1999a; 1999b).

Deactivation of heteromodal association areas, a feature common to nonREM sleep and REM sleep, could be a prerequisite for dreaming. However, its direct association with dream experience would have to be documented by comparing PET scans obtained for sleep periods with and without dreaming. If similar differences would emerge in nonREM sleep and REM sleep, then the pattern would deserve serious consideration as a physiological correlate of the dreaming process. Early studies have attempted to specify dream-related patterns of cerebral glucose metabolism (Gottschalk et al. 1991a; Heiss et al. 1985). However, the poor temporal resolution renders interpretation difficult. PET studies of regional cerebral blood flow using labeled water appear to be more propitious. The comparison of sleep periods with and without dreams would also be a useful approach in quantitative EEG analysis. Such a study would also be useful for testing the activation hypothesis of **SOLMS.**

A final comment pertains to **SOLMS**'s interesting proposition that the mesocortical-mesolimbic dopamine system plays a casual role in the generation of dreams. Neuroleptics such as haloperidol are powerful blockers of dopamine-D2 receptors and would be expected to eliminate dreaming. Awakenings from sleep on nights with and without neuroleptics would be a direct way to test the dopamine-dream hypothesis.

REM sleep deprivation: The wrong paradigm leading to wrong conclusions

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Abstract: There are obvious flaws in REM sleep suppression paradigms that do not allow any conclusion to be drawn either pro or contra the REM sleep-memory hypothesis. However, less intrusive investigations of REM sleep suggest that this sleep stage or its adjunct neuroendocrine characteristics exert a facilitating influence on certain aspects of ongoing memory formation during sleep.
[NIELSEN; VERTES & EASTMAN]

REM sleep facilitates memory formation. Currently this is more a belief than a concept with convincing scientific support. Hence, **VERTES & EASTMAN**'s case against memory consolidation in REM sleep is a very timely contribution reflecting the true and persisting darkness in this area of sleep research. Unfortunately, VERTES & EASTMAN appear to be caught in similar misconceptions to those of researchers supporting a close link between REM sleep and memory consolidation. A great part of VERTES & EASTMAN's review is devoted to studies evaluating recall of memories after a period of REM sleep suppression as compared to control situations, such as arousal from NonREM (NREM) sleep. Such studies did not provide evidence that REM sleep deprivation impairs recall of previously learned materials, under all circumstances, although changes, if they occurred after REM sleep deprivation, were always towards impairment rather than improvement of memory. It is very likely, however, that a stress response induced by REM sleep suppression is the principal factor responsible for recall deficits.

Recently, DeQuervain et al. (1998) demonstrated in rats that glucocorticoids, the release of which is a central marker of the stress response, have a distinctly impairing effect on the retrieval of long-term spatial memories. Hence the REM sleep suppression paradigm is not conclusive about what happens during REM sleep with ongoing consolidation. Moreover, a propensity for REM sleep must be assumed to persist during deprivation conditions, thereby contaminating the outcome of memory retrieval in an unpredictable manner. That is, by inducing nonspecific alterations of cognitive and emotional functions it may disturb or improve recall performance. Even more important, suppression of phenotypic REM sleep may miss those electrophysiological and neurochemical processes mediating memory consolidation.

Consonant with this view, **NIELSEN** in his target article on mentation in REM and NREM sleep proposes the concept of "covert REM sleep" as a kind of sleep that lacks some of the obvious signs of REM sleep, but shares underlying related processes. Traditional sleep scoring certainly does not focus on the phenomena determining memory during REM sleep which may persist (as reflections of propensity) even in the absence of the phenotypic signs of this sleep stage. This reasoning can be extended to all kinds of REM sleep suppression regardless of whether induced by behavioral techniques such as the pedestal method or by psychopharmacological intervention with antidepressant drugs. In addition, most of the latter work with antidepressant drugs, by focusing on changes during extended periods of treatment, is unable to distinguish drug effects on acquisition, consolidation, and retrieval. Also, the academic achievement of patients who have recovered from bilateral pontine lesions and do not reveal any common signs of REM sleep is clearly impressive. These data show that phenotypic REM sleep is not a prerequisite for memory consolidation, just as the occurrence of EEG desynchronization and theta activity is not restricted to REM sleep. Nevertheless, it cannot be concluded from these patients' performance whether processes are initiated during normal REM sleep, which facilitate certain aspects of a consolidation process.

In light of the apparent shortcomings of experimental procedures relying on suppressed REM sleeps it is amazing how little effort **VERTES & EASTMAN** devote to reviewing experiments that rely on less intrusive manipulations and, indeed, point to a supportive function of REM sleep on memory. VERTES & EASTMAN briefly mention the intriguing work of Stickgold et al. (2000b). The task used there (requiring a preattentive discrimination of visual textures) is remarkable as subjects, performance did not improve unless they obtained some hours of sleep after initial training. This suggests the presence of a slow continuous process of memory formation particularly sensitive to the influence of sleep (Karni et al. 1994; Karni & Sagi 1993). Stickgold and coworkers found that the improvement in texture discrimination was strongly correlated with the amount of slow wave sleep (SWS) in the first quarter of sleep time, and with the amount of REM sleep in the last quarter of sleep time. This pattern is of interest, because it rules out a one-to-one link between REM sleep and memory. Accordingly, Stickgold and coworkers proposed a two-step process of memory formation during sleep, with REM sleep becoming effective in a second step, strengthening associative connections at the neocortical level. However, correlations between the amount of REM sleep and recall performance do not necessarily reflect a relation between cause and effect, which limits respective conclusions.

Another interesting approach was developed by Ekstrands group in the 1970s (Barret & Ekstrand 1972; Ekstrand et al. 1977; Fowler et al. 1973; Yaroush et al. 1971), who compared retention rates across sleep periods of equal length but with different pro-

portions of sleep stages. Ekstrand's group found greater improvement in recall of declarative memories after sleep during the SWS-rich early half of the night than after REM sleep-rich sleep during the late half of the night.

Recent studies in our laboratory have confirmed this (Plihal & Born 1997; 1999a). However, in extending the work of Ekstrand's group to several tasks of procedural memory (mirror tracing, word stem priming), we found greater improvement across late as compared to early sleep. This led us to suggest that some kinds of nondeclarative memory not relying on the integrity of the hippocampus and adjacent temporal lobe structures particularly benefit from late sleep with predominant REM sleep.

In another study (Gais et al. 2000), we examined performance on the visual texture discrimination task mentioned above (Stickgold et al. 2000b; Karni & Sagi 1993). The comparison of retention intervals containing either a 3-hour period of early sleep or a 3-hour period of late sleep indicated early but not late sleep to be primarily necessary for the improvement in texture discrimination skills. It is interesting to note the improvement in task performance after an entire period of undisturbed nocturnal sleep containing early SWS as well as late REM sleep was on average more than 3-fold higher than after a period of early SWS-rich sleep alone. This outcome fits nicely with the two-step model of memory facilitation during sleep proposed by Stickgold et al. (2000b) and others (Giuditta et al. 1995) suggesting that REM sleep plays a role at a later stage of memory processing during sleep.

These experiments together provide evidence that REM sleep and associated processes can enhance memory formation. However, their role probably depends on the type of memory system and prior processing of the materials within this system. There are numerous processes (hormonal concentrations, temperature, etc.) changing in parallel with REM sleep that are candidates for explaining a sleep related memory enhancement as well as REM sleep per se. These processes may interact with cognitive functions in any sleep stage. However, they are neglected in **VERTES & EASTMAN**'s discussion of sleep associated memory formation and, notably, also in **NIELSEN**'s more general discussion of mentation during sleep.

Of utmost importance in this context is the release of corticosteroids from the pituitary-adrenal system, which in humans is at a minimum during early nocturnal sleep and reaches a maximum during late sleep. Glucocorticoids, that is, corticosterone in rodents and cortisol in humans, are potent modulators of ongoing EEG activity and memory function (e.g., DeKloet et al. 1999; Friess et al. 1994; Gronfier et al. 1997; Kirschbaum et al. 1996). In humans, infusion of cortisol during a period of early sleep completely blocked the improvement in declarative memory typically observed over this period (Plihal et al. 1999; Plihal &Born 1999b). Note that in the latter study the blocking effect of cortisol infusion on declarative memory consolidation during early sleep occurred without any concurrent reduction in signs of SWS. Thus, rather than the phenotype of SWS activity, the concurrent suppression of cortisol release turned out to be a crucial prerequisite facilitating declarative memory function during this period of sleep.

Comparable conditions may determine the putative memory process during late sleep when REM sleep prevails and glucocorticoid concentration is elevated. Studies in rodents indicated that memory of events that are emotionally highly arousing and aversive can be enhanced by glucocorticoid administration (Cahill & McGaugh 1996; DeKloet et al. 1999). Experimental improvement of memory ascribed to REM sleep might accordingly turn out to be a result of an accompanying elevation in glucocorticoid levels.

Another well-known example of neurohormonal processes modulating memory is sympathetic activity and the release of catecholamines. Through the activation of central-nervous adrenergic receptors, epinephrine can enhance storage of emotionally arousing events in humans (Cahill et al. 1994; van Stegeren et al. 1998). In humans, concentrations of epinephrine and norepinephrine in the blood are reduced during REM sleep as compared to SWS and wakefulness (Dodt et al. 1997). This could selectively disfacilitate formation of emotional memory. Thus, neurohormonal processes only loosely linked to specific sleep stages may be more relevant for memory consolidation than a specific sleep stage. As an alternative view **VERTES & EASTMAN** propose that REM sleep serves to prime the brain for a return to consciousness as waking approaches. It is noteworthy that exactly the same function has been claimed for the release of pituitary-adrenal stress hormones increasing towards the end of sleep (Born et al. 1999).

REM and NREM mentation: Nielsen's model once again supports the supremacy of REM

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Abstract: Nielsen's model presents a new isomorphic brain-mind viewpoint, according to which the sole dream generator is found in a REM-on (explicit or covert REM) mechanism. Such a model cannot explain the dreamlike activity during SWS (slow wave sleep), SO (sleep onset) and in the last period of sleep. Moreover the hypothesis contrasts with Solms's data, which show that dreaming is present also in case of destruction of the REM generator.
[NIELSEN; SOLMS]

In the fifties and sixties, a target article like the one by **NIELSEN** would not have been imaginable. The identification of REM sleep with dreaming had all the characteristics of an unshakable dogma. Today, NIELSEN documents the existence of rich production of NREM mental experiences; this has led to a divergence in researchers' theoretical views. On the one hand there are the supporters of a one-generator model, in which the REM and NREM dream production would be relatively autonomous from its physiological basis as related to sleep stage. On the other hand, there are the supporters of a two-generator model, according to which REM sleep would be responsible for an oneiric cognitive activity qualitatively different from the one generated by NREM sleep, regardless of stage.

NIELSEN attempts to reconcile the two models, assuming the existence of covert REM sleep processes in NREM sleep responsible for the concomitant NREM mentation. This attempt brings him back to the identification of dreaming and REM sleep, not dissimilar to the positions of Hobson and his group (1998b), in which an isomorphism between physiological background (REM or covert REM) and dream mentation is assumed. In this sense NIELSEN's hypothesis does not seem to be a reconciliation between the two models, but a unique REM-one model of oneiric generation, either in its explicit or covert form. From a theoretical point of view, a unitary explanation is more parsimonious and therefore preferable to the two-generator model. However, NIELSEN's arguments show some weak points that call for further investigation.

REM-sleep related processes would be responsible for NREM mentation, even though activated "in a piecemeal fashion and against an atypical neurophysiological background." (**NIELSEN** target article). Characteristic of this activation would be that it takes place near to the REM phase (10–15 min before or after), even though this is weakly supported by physiological data, making it very difficult to explain data on SWS mentation, as NIELSEN himself admits. SWS dream reports were collected in our lab (Cavallero et al. 1992; Occhionero et al. 1998) in cycle I, 40 min after SO (hence distant from a possible covert REM in SO and distant from REM I), as well as in cycle II 40 min after REM (unpublished data), yielding recall over 60% and differences in comparison with REM dreams with regard to length only. The same holds for spontaneous morning awakenings during NREM (about 70%) which are rich in oneiric activity and not near to any REM

(Cicogna et al. 1998). Moreover, if we were to accept NIELSEN's view, in sleep periods far from REM (when one could not impute covert REM activity) no mental activity would be present: which looks like a very hasty assertion. Only in deep coma, without any cortical activity, is there total absence of thought.

Another datum difficult to explain is oneiric activity in SO, which not only is very bright, but has high recall similar to REM. It seems difficult to maintain that this is covert REM, since physiological factors (transient EMG suppression, REMs, muscle twitches) that could provide evidence of a similarity to REM are almost non existent in SO-St2, the moment of SO in which experimental awakenings usually take place. Without any further data on the presence of REM-on physiological factors in SO-St2, it is difficult to interpret the oneiric richness of SO according to the model.

As to **NIELSEN**'s very broad review of the literature, two remarks: (1) The scheme indicated in Figure 1 (NIELSEN) is not completely convincing with regard to the "apex-dream" typology that should be the most typical expression of a dream-like mentation (REM-like) and it in fact refers to rare situations or even to situations that violate the dream's hallucinatory quality (for example lucid dreams); (2) In our opinion the qualitative differences between REM and NREM mentation are overemphasized in the cases in which controls equating report length showed slight residual differences, to the point of making the authors infer that they were epiphenomena owing to quantitative aspects, explicable in terms of mnemonic spreading activation (Antrobus 1983; Cicogna et al. 1987; 1998; Foulkes & Schmidt 1983). These authors do not deny the influence of an underlying physiological background that may be responsible for the modulation of memory activity (for example EEG differences, differences in sensory thresholds), however they deny that this could be directly involved in the dream production and in the cognitive work typology.

There is also a general problem in research on sleep and dreaming, which is that of handling NREM sleep as a unitary and physiologically similar homogeneous entity, without considering the differences between stages, which are quite remarkable. In terms of radical isomorphism, one can think of as many dreaming generators as sleep stages. The limit of **NIELSEN**'s model can be found in this "isomorphic" brain-mind view, in which the only oneiric generator is found in a REM-on mechanism (but it is unclear why). Among other things, this position contrasts with **SOLMS**'s evidence (see target article) that dreaming continues in case of destruction of the REM generator and is absent in cases of forebrain lesions.

In our view as cognitive psychologists, there are higher cognitive processes which, after having been initiated by REM or NREM subcortical activation mechanisms, follow information processing rules that have no precise correspondence to neurophysiological areas or mechanisms. Even though one may want to find a correlation between cognitive and neurophysiological processes at all costs, the evidence adduced by **NIELSEN** himself as well as by Solms (1997a) shows that the association areas involved in the information processing are equally activated in REM and SWS, whereas the differences in the cerebral blood flow are found precisely in limbic and hippocampal areas affecting memory systems and emotions. The levels of physiologic activation do certainly modulate cognitive processes in terms of "amount of work," but they do not modify their operational modality.

How and why the brain makes dreams: A report card on current research on dreaming

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Abstract: The target articles in this volume address the three major questions about dreaming that have been most responsible for the delay in progress in this field over the past 25 years. These are: (1) Where in the brain is dreaming produced, given that dream reports can be elicited from sleep stages other than REM? (2) Do dream plots have any intrinsic meaning? (3) Does dreaming serve some specialized function? The answers offered here when added together support a new model of dreaming that is testable, and should revitalize this area of study.
[HOBSON ET AL.; NIELSEN; REVONSUO; SOLMS; VERTES & EASTMAN]

Introduction. The reader of these five BBS articles might come away with the impression that they have just witnessed another set of blind men describing an elephant. Although each does bring a different perspective to the problems associated with the phenomenon of dreaming, collectively, they make significant progress in clearing their way for further work. The first three papers (**HOBSON ET AL., NIELSEN,** and **SOLMS**) focus on activity within the sleeping brain to tackle the question of dream construction: How does the brain makes dream experience happen, its dependence on, or independence from, REM sleep? The last two papers (**REVONSUO, VERTES & EASTMAN**) address the function question: Why we do it? They both look into interactions between waking and REM sleep – one challenges the proposal that this sleep stage has any specific role in memory storage and the other champions a different function, that dreams make a contribution to our waking survival from threats.

The articles differ not only in where they look but how. The first two by **HOBSON ET AL.** and by **SOLMS** stay within the sleeping brain, tracing those pathways which are active and which blocked, in order to explain the variations in the cognition reported from sleep. HOBSON ET AL. focuses on the REM sleep system and its activation starting in the pons, SOLMS on the dream system he locates in a dopaminergic system within the forebrain. **NEILSEN** turns the problem the other way around, using the presence of a dream-like report, to predict the presence of REM sleep if only as fragments which previously have escaped traditional scoring. **VERTES & EASTMAN** look at REM sleep as both the independent and dependent variable in turn as they examine the evidence supporting one of its proposed functions: that REM sleep is involved in the storage of newly learned information. Is REM sleep enhanced by intensive pre-sleep learning and is performance post-sleep reduced following REM sleep deprivation? They find these data unconvincing. **REVONSUO** broadens the time frame for exploring a different function of dreaming by hypothesizing that the high proportion of negative affect characteristic of dream reports suggests these were developed from earliest times when waking life was more acutely dangerous. Perhaps they represent a genetically transmitted legacy of survival protocols retained and rehearsed during sleep for use in waking. This is the only paper in this collection that suggests some meaning for dream content.

Background. Following the discovery in the early 1950s of REM sleep, a flood of published reports confirmed that this stage of sleep was strongly associated with the presence of an ongoing dream. Using the reliable external indicators of REM sleep, awakenings were done to capture samples of the immediately preceding mentation. This allowed dreams to be studied systematically as never before possible. Thus the normative characteristics of REM-related dreams, their changes with age, and the effects on them of various manipulations and conditions were mapped out by the mid 1970s. Then came the drought when progress slowed to a trickle.

On reflection, three factors seem to be responsible for this turn of events. The first was the need to modify the initial brain/

behavior models to accommodate the finding that some of the awakenings made from NREM sleep stages also had dream-like characteristics. Before new schemes could be constructed to accommodate these data, the Activation-Synthesis hypothesis of dream construction was published. This effectively dismissed the importance of the dream as an object of study by accounting for its construction as a degraded effort by the sleepy cortex to interpret what were essentially random stimuli initiated from the pons. The third damper on the enthusiasm for the hunt to locate and explain dreaming came from the failure of REM deprivation studies to demonstrate any consistent effect of its loss on waking behavior.

Once dreaming lost its anchor to the REM state, and the questions of its meaning and function went unanswered, it is no wonder most serious investigators turned away. Now the problems raised by these challenges are addressed with new data and more elegant models, which are outlined in these five BBS target articles.

1. The dream construction problem. It was the reports of dream experience outside of' REM sleep, especially those collected from awakenings made shortly after sleep onset, before the criteria for the presence of REM sleep are met, that shook the assumption that REM sleep physiology was necessary for the production of dreams. If dreams can occur in descending Stage 1 when no rapid eye movements are visible, in Stage 2 following periods when REM sleep has been selectively suppressed, and if subjects can identify when they are experiencing dream imagery throughout all sleep stages by use of a signal and do so with more accuracy than experimenters were able to by using the REM markers (Brown & Cartwright 1978), then the activated brain state of REM may represent the best, but not the only, set of conditions under which dreams occupy awareness. The papers by **HOBSON ET AL., NIELSEN,** and **SOLMS** share the view that the relation of the sleeping mind to brain is more complicated than was originally described. No longer can we state with conviction: Every 24 hours we regularly cycle through three distinctively organized states of being: waking, NREM, and REM, each with its own physiological and psychological characteristics. Now we have to qualify this as more or less. Clearly there are differences in how firmly these states are separated from one another both within and between individuals. This permeability of the gates between states helps account for many anomalies in the sleep of some psychiatric and sleep disorder patients and the reports they give of their sleeping mental life.

All three of the papers that address dream production agree on three of the building blocks: (1) There must be a raised threshold for external sensory input. (2) This blockage from the periphery must occur in the presence of an activated brain which stimulates internal sources of stored sensory images, and (3) this source is biased toward the expression of basic motivational drives and negative affect. The three target article authors also call on the evidence from recent brain imaging studies showing the localized activity during REM sleep to differ from the activity level in those areas during NREM and in waking. The evidence of activation in the forebrain of the limbic and paralimbic system including the amygdala and hypothalamus supports that dream construction is emotion-driven. **HOBSON ET AL.** have worked out the conditions under which REM sleep is turned on N starting in the pons. **SOLMS** has traced the dopaminergic mesencephalic tract and demonstrated the necessity of this being intact to sustain the experience of dreaming. **NIELSEN** offered one way to link the two, the pontine activation of REM sleep and that of the emotional-motivational dreaming system in the forebrain, with his concept of "covert REM" for dreams being experienced during periods of NREM sleep.

2. The dream meaning problem. The **HOBSON ET AL.** article is a heroic review of where we are in understanding dreams and how we got here. Having initially denied that these have intrinsic meaning these authors now propose a revision of the activation-synthesis model to account for cognition under many conditions. This new model leaves room for the study of the dream as more than an unplanned epiphenomenon of the "unthinking pons." **SOLMS** suggests that dreams may be experienced independently of REM sleep altogether as when the brain is activated by a seizure. After reviewing the difficulties in studying dreaming in the laboratory setting, **HOBSON ET AL.** build the case for home studies using the Nightcap system. This is a simplified two variable recording device based on the combination of rapid eye movements or no eye movements, in the presence of head movements or no head movements, without the EEG to distinguish sleep from wake and without the EMG of the submental muscle to distinguish quiet wakefulness from REM sleep. Thus, it would be difficult to test **NEILSEN**'s concept of "covert REM" to account for NREM dreaming using this equipment. It would be daunting to distinguish sleep onset dreams in Stage 1 with REM intrusions from Stage 1 dreams with wake intrusions when there are eye movements present in the absence of head movement. Testing predictions based on the AIM model and **NEILSEN**'s proposal will need further development of sensitive equipment to use in the home if experimenter and laboratory effects are to be avoided.

SOLMS's work gives more specificity to the areas of the brain and the connections responsible for the various aspects of the dream and the circuitry necessary to this activity. This suggests the possibility of developing a map of the circuits involved in contributing the various elements required to build a dream, perhaps equivalent to the five outlined to account for waking cognition by Mesulam (1998). This conversion of efforts to understand emotion- driven thought in waking and in sleep is to be applauded, and hopefully rapidly replicated. The next step would be to extend this brain mapping effort to the study of the sleep/wake transition phenomena such as the highly emotional states that are observed during night terrors and episodes of sleep walking with violence. These disorders highlight the difficulty posed by our reliance on the subject's report of the prior sleep mentation; these episodes are followed by nearly complete amnesia. Typically these subjects are unable to give an account of the perceptions responsible for their heightened drive-related emotional behavior. We need both a breakthrough in technology of more objective probes to illuminate what is happening centrally as well as more sensitive inquiry of the observers and patients to describe this experience. This will help to develop the maps of the brain areas that are functioning and not functioning during such episodes of dissociation.

For example, in two of three sleepwalking murder cases (Broughton et al. 1994; and Cartwright 2000) neither attacker recognized their victim. The face recognition pathway was not functioning while other visual pathways were operating that guided the perpetrators' special orientation. One man drove 15 kilometers to his mother-in-law's house, the other walked outside and assembled tools to begin work to repair a pool motor filter. Complex motor behaviors were intact. Neither responded to their victims' screams as did others who were more distant. Both were analgesic for a period following the attack; the first to pain inflicted in the struggle for the knife, the other to the cold water in the pool as he held his wife's head under water. Both had the genetic and personal history of a propensity to arouse abruptly from the delta sleep in the first cycle of sleep into a confused state that aborted REM. They clearly had a NREM to REM transition problem. When challenged during this state, both behaved as if under threat by initiating a fatal attack. Could this be explained as covert REM triggering a basic survival program? More likely it is the stress response of the neuroendocrine system that needs investigation.

NIELSEN's position that there are conditions under which the tight coupling of REM and dreaming is subject to dissociation is confirmed by the NREM dream reports of light sleepers who are in high arousal throughout all sleep and in others when there is a low threshold for arousal following sleep deprivation, and/or during acute stress. Both sleepwalking violence episodes reported above occurred followed periods of extended sleep loss and stress. Dissociation is also seen in REM sleep without atonia of those demonstrating the REM behavior disorder. This also represents a

mixed state when the gates controlling movement during REM are lowered. It is interesting to note that this sleep disorder is sometimes the first symptom of a movement disorder, Parkinson's disease, in which dopamine production is low.

3. The problem of REM function. **VERTES & EASTMAN** deliver a devastating blow to the proposal that one of the functions of REM sleep may be to aid in the transfer of new learning from short term to long term storage. Certainly the evidence supporting this has been meager, and hard to replicate. Because the case is much stronger for an emotional-motivational function for REM/dreams, this is the next place to look.

This point is made when the REM suppression studies of the depressed are examined for their effect on mood rather than on memory consolidation. In the Vogel study (Vogel et al. 1975) REM suppression was carried out not by medication but by voice or hand awakenings at the first signs of REM sleep for six nights, followed by a night of uninterrupted sleep each seventh night for three or more weeks. On re-examination of the sleep in those who responded positively to this manipulation with a remission of depression, Vogel reported that it was not the REM deprivation that was responsible for the difference. The improvement in mood and increased drive behaviors was only seen in those who showed evidence of a build up of "REM pressure" on the intermittent nights without deprivation. Was this due to the appearance of covert REM? Vogel defines "REM pressure" as an increase in REM time and number of REM attempts that occur after the deprivation condition was lifted. This study suggests that depression, a state of low drive behaviors and mood, can be improved if there is rebound following a limitation of REM sleep. The fact that withdrawal from REM suppressing anti-depressants after long-term use can be followed by nightmares suggests an intensified rebound of REM/dreams, a heightening of experienced affect.

If waking mood and drive behaviors improve following the release from a period of REM deprivation, this suggests that there may also be a functional change in the nature of the dreams as well. Those who are severely depressed have little recall following REM awakenings or, at best, dreams with neutral affect. Cartwright et al. (1998b) reported a distinctive dream affect pattern within a night in those depressed volunteers who will later remit without treatment. Waking subjects to collect these dreams creates a night of reduced REM time and so constitutes a minor degree of deprivation. Remission could be successfully predicted when negative affect dreams dominated the first half of the night and more positive dreams were proportionally higher in the second half. This within-night dream affect pattern is also associated with an overnight improvement in depressed mood in normal subjects (Cartwright et al. 1998a).

REVONSUO's hypothesis that dreams involve rehearsal of fight and flight behaviors needed for survival from real life dangers reminds us that these are the behaviors that become dissociated from REM sleep in the sleep walking with violence cases and those with REM behavior disorder. Both these exhibit heightened aggression, the acting out of primitive drives including fighting, fleeing, and even inappropriate sexual behavior. These are expressed overtly in some adult sleep walkers in confusional states following arousal from the first cycle of NREM sleep before the muscle atonia of REM can confine this behavior to the safe expression of dreaming. In the RBD the aggressive behaviors occur when the loss of muscle atonia during REM allow these behaviors to be acted out in response to dreams they recall as having threatening content or which require their aggressive action. This argues that threat-avoidance programs of dreaming, if useful, may become malfunctioning in several ways: either too active or not active enough. In the depressed, this stress response may be attenuated until restimulated by some perturbing treatment. This new conception of dreaming calls for testing the relation of the survival dream scenarios and their adaptive function to the degree and length of prior waking stress and their effectiveness in terms of waking affect and coping behavior.

Conclusion. This group of papers set up a framework for research to fill out the picture of the mind asleep and its relation to pre-sleep and post-sleep waking behavior. After a long delay, we are moving toward a twenty-four hour picture of the brain/behavior relations as they vary around the clock, both in the normal mind and in the various disorders of the mind.

REM sleep = dreaming: The never-ending story

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Abstract: It has been widely demonstrated that dreaming occurs throughout human sleep. However, we once again are facing new variants of the equation "REM sleep = Dreaming." Nielsen proposes a model that assumes covert REM processes in NREM sleep. I argue against this possibility, because dream research has shown that REM sleep is not a necessary condition for dreaming to occur.
[NIELSON]

> Dream researchers face a paradoxical situation: Although a fairly large amount of evidence supports the idea that dreaming occurs during the whole night, irrespective of sleep stages, mental activity in nonrapid eye movement (NREM) sleep is still considered a second rate product in comparison with REM dreaming. And indeed, among scientists and the general public the old-fashioned – and wrong – equation "Dreaming = REM dreaming" is still widely accepted. (Cavallero et al. 1992, p. 562)

This was the start of the 1992 paper comparing REM and SWS (stage 3 and 4) reports and dream memory sources that I had thought might be substantial beyond the REM/NREM dichotomy. But I was wrong. Eight years later, notwithstanding new evidence strengthening the idea that equating REM sleep with dreaming is no longer viable, we are once again facing variants of the old REM = dream isomorphism.

Yet it has been amply demonstrated that dreaming occurs not only in REM but also during ordinary NREM sleep (including delta sleep) during sleep onset, and even during relaxed wakefulness (Foulkes 1985). It has also been shown (Antrobus 1983) that when length of dream report is partialled out, there are few if any qualitative differences between dreams collected in REM and NREM states. Moreover, a number of studies (Cavallero et al. 1990; 1992; Foulkes & Schmidt 1983) have found that when length of dream report is controlled, apparent qualitative differences between REM and NREM reports tend to disappear, suggesting that the same dream production mechanisms are involved across states. When time of night effect is controlled, narrative length is not proportional to time spent in REM prior to awakening; instead, prior sleep duration is a much more potent determinant of narrative length than time in REM (Rosenlicht et al. 1994). A number of studies on the mnemonic sources of dream content suggest that stage differences in dream recall appear more closely related to the level of mnemonic activation and to access to memory traces than to any special dream production mechanism unique to one stage of sleep (Cavallero 1987; Cavallero et al. 1990; Cipolli et al. 1988; Cicogna et al. 1986: 1991).

In general, these results suggest that the same cognitive systems produce mental activity irrespective of EEG sleep stage, as Foulkes proposed in 1985. Moreover, by comparing memory traces from day dreaming and sleep onset dreaming, Cicogna et al. (1986) found a similarity suggesting that "cognitive processes involved in the creation of original narrative sequences may be similar in sleep and waking." Further evidence comes from human neuropsychology, which has established that dreaming is coextensive with competence in mental imaging, a relatively late cognitive acquisition (Kerr 1993); and sleep-laboratory studies of children's dreaming, which indicate that dreaming is absent until ages 3 to 5, and does not assume the form of adult dreaming until ages 7 or 8 (Foulkes 1993c).

Given the above mentioned evidence, one might expect scientists will come to reject the idea, as appealing as it may be, that REM sleep is the brain correlate of the dream. On the contrary, we see a continuous quest for explanations of dreaming in physical events occurring just in REM sleep or just in REM sleep and its immediate temporal surroundings. **NIELSEN**'s new model of covert REM processes in NREM sleep is a good example of these kinds of enterprise. He admits that NREM dreams exist and need to be taken into serious consideration (a good step forward in comparison with old-fashioned theorists who simply dismissed dreaming outside REM sleep as a kind of artifact). But then, instead of trying to develop a model that can account for dreaming as unitary phenomenon in terms of cognitive processes involved in its production, he goes back to the old idea that "real dreams" can be found only in REM sleep and hence one must find hidden REM features in NREM sleep to justify the existence of NREM dreaming. The idea in itself may be fascinating but it is the attempt to reduce dreaming to mechanisms found only in (or around) one state that is doomed to failure because, as I hope the evidence I have reported demonstrates, dreams can occur throughout human sleep and are not confined to a temporal window corresponding to REM sleep and its immediate surroundings. I must confess that I am rather skeptical about the possibility of discovering some covert REM processing underlying SWS dreaming.

Mental states during dreaming and daydreaming: Some methodological loopholes

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Abstract: Relatively poor memory for dreams is important evidence for Hobson et al.'s model of conscious states. We describe the time-gap experience as evidence that everyday memory for waking states may not be as good as they assume. As well as being surprisingly sparse, everyday memories may themselves be systematically distorted in the same manner that Revonsuo attributes uniquely to dreams.
[HOBSON ET AL.; REVONSUO]

HOBSON ET AL. and **REVONSUO** use the difficulty people have remembering their dreams as key evidence in their model of conscious states. We would like to question their assumption that recall of mentation in waking states is so superior to that experienced during sleep. A critical difference in recall between waking and sleeping states may be the existence of an external narrative to which memories for internal events can be tied. Most recall of waking experiences is referenced by external events. When a subject performs any typical laboratory memory task, successful performance is predicated on the subject accepting and using the external temporal structure of the experiment. Recall of items which are internally generated, or of items presented from other learning episodes is regarded as an error (e.g., Roediger & McDermott 1995). If it is accepted that the perception of time during sleep is itself substantially distorted (Stickgold et al. 1997a) then this too may present a rather poor cue for recall. The very predictability of experiences in everyday life may provide both an illusion of memory for mundane events, and a structure with which to enhance memory for exceptions (cf. Reiser et al. 1985).

One of the most dramatic examples of memory failure for routine events is the time-gap experience (Chapman et al. 1999b; Reed 1972). The commonest example of a time-gap experience is when a driver suddenly realises that he or she has no recollection of some considerable part of the journey that is currently underway. The time-gap experience itself is characterised by a surprising failure to recall mentation. The very essence of the experience is this surprisingness. As Reed (1972) observes, a failure to recall significant mentation while spending half an hour sitting in the garden evokes little concern, while a similar failure to recall events during the drive from Bologna to Florence is perceived as a startling anomaly of attention. Two issues arise when considering such experiences – first, the question of whether the failure to recall specific episodic memories from routine, automatized tasks should in fact be surprising, and second, the question of the degree to which the "missing" mentations are internal or external in origin.

The first of these issues is the idea that the predictability of everyday experiences provides an illusion of episodic memory. Because it is clear that the only way to have progressed from Bologna to Florence in half an hour is to have driven along the road, we can confidently say that this has occurred without actually accessing new memory traces laid down by the experience. Moreover, the certainty that any interesting exceptions would have been stored allows us the knowledge that the journey passed in a routine manner in accordance with a general schema for such journeys. In fact, time-gaps may be absolutely routine aspects of most such journeys, the surprise that accompanies the occasional experience is simply brought about by some unanticipated event disrupting the normal flow of experience (Chapman et al. 1999a).

The second issue is the degree to which time-gaps may in fact be populated by internal events, task-related thoughts or daydreams. Our research, and the broader literature on daydreaming (e.g., Giambra 1995; Singer 1993) suggests that if subjects are interrupted during the performance of routine tasks they can often report task- unrelated images and thoughts (TUITs) which may be inaccessible after a delay. The reason that such TUITs or daydreams become inaccessible may simply be that because they are unrelated to the task, there is no retrieval cue available based on the normal structure of the remembered task. Without such an external narrative to impose on a sequence of events, the bizarreness of everyday cognition may itself be increased. Readers may well have had the experience of being engaged in a long and fascinating conversation when one participant suddenly exclaims "How did we ever get onto that topic?" A similar but stronger effect can be observed in one's own thoughts and daydreams – "How did I ever come to be thinking about that?" Without external events to tie previous thoughts to, the only way to answer this question may be a search for random associations between current thoughts and previous ones. We don't have direct access to what we were thinking ten minutes ago.

A common problem here for the investigation of both dreams and daydreams is the provision of any evidence (other than self-report) of their existence. Scientific evidence of any mental state can only come from systematic variation of response as the stimulus is varied. The dreams and daydreams that are hardest to recall may be the very mentations that are stimulus-free. Do they then exist? With time-gaps all we can report experimentally is the balance between internally and externally induced mentations summoned from memory. Although externally induced mentations may be subject to experimental manipulation, the opportunities for control over internally induced mentation are considerably reduced. As we argue below, even when we do know that the to-be-remembered event happened, recall can be notoriously distorted.

REVONSUO suggests that the over-representation of negative emotions, misfortunes, and aggression in dreams supports his hypothesis that dreams are specialised in simulating threatening events. An important issue here is that **REVONSUO** compares dream content with everyday life. Following our argument that memory for everyday life may not be as good as often is assumed, it is perhaps worth reflecting on the degree to which memory for everyday life is itself representative. A growing body of research suggests that autobiographical memory very substantially fails to represent everyday life. A recent study of ours looking at the poor recall of accidents and near-accidents (Chapman & Underwood 2000) not only demonstrates huge levels of forgetting for mun-

dane events, but demonstrates selective retention of particular experiences in memory. Two key factors that determine the likelihood of events being represented in memory are precisely the degree of threat posed and the unpleasantness of the incident (in our study operationalised as the degree to which the participant felt they were to blame in the incident). Unpleasant, traumatic events are routinely over-represented in memory. Such findings are consistent with Wagenaar's (1986; 1994) extended analysis of his own autobiographical memory in which he reports heightened recall of highly unpleasant self-related events. Although we neither dispute nor support REVONSUO's analysis of the content of dreams, we suggest that memory, not real life, is the control condition to which the content of dreams must be compared.

REVONSUO cites Penfield's (1975) claim that random brain stimulation does not produce dreams, but instead produces memory traces. REVONSUO characterises these as short and undramatic excerpts of the patient's previous experiences. It is perhaps worth quoting Neisser's (1967) evaluation of the same data – "in short, the content of these experiences is not surprising in any way. It seems entirely comparable to the content of dreams, which are generally admitted to be synthetic constructions and not literal recalls. Penfield's work tells us nothing new about memory." (p. 169). Deciding how to characterise the reports from Penfield's patients is largely a subjective issue. We note one report of reliving the experience of childbirth (Penfield & Perot 1963). Surely it is not fair to characterise this as a short and undramatic excerpt from that patient's previous experience. More generally we suggest that the reports elicited from such stimulations may share many of the characteristics of dreams, but we stress that these characteristics may also be more representative of autobiographical memories than of real life.

Play, dreams, and simulation

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Abstract: Threat themes are clearly over-represented in dreams. Threat is, however, not the only theme with potential evolutionary significance. Even for hypnagogic and hypnopompic hallucinations during sleep paralysis, for which threat themes are far commoner than for ordinary dreaming, consistent non-threat themes have been reported. Revonsuo's simulation hypothesis represents an encouraging initiative to develop an evolutionary functional approach to dream-related experiences but it could be broadened to include evolutionarily relevant themes beyond threat. It is also suggested that Revonsuo's evolutionary re-interpretation of dreams might profitably be compared to arguments for, and models of, evolutionary functions of play.
[REVONSUO]

The first part of **REVONSUO**'s thesis, that dreams contain a disproportionate number of threat and predation themes, seems quite uncontroversial. As he points out, many studies have reported that a third or more of dreams contain negative emotions (see also, Merritt et al. 1994). Also reasonable is the claim that such figures seem substantially greater than would be likely in the waking lives of the subject populations of these studies, especially given the typical positivity bias (e.g., Cacioppo et al. 1999). Nonetheless, further studies such as those being carried out by REVONSUO and his colleagues are needed to further assess the degree of discrepancy. In particular, REVONSUO may wish to consider not only the relative incidence of threat and fear but also their intensity. Moreover, if simulated threat is what REVONSUO is truly interested in he might consider another common and often intensely frightening sleep-related REM phenomenon: sleep paralysis with hypnagogic and hypnopompic hallucinations (Cheyne et al. 1999a; 1999b). As many as 65% of people with such experiences give the maximum rating to their experienced fear. Although people who suffer from these sorts of nightmares may sometimes be experiencing stress in their waking lives, many volunteer that the level of fear experienced during the episodes exceeds anything they have ever experienced in their waking lives. Fear is often regarded as much too mild a word for the abject terror they experience. Also encouraging for REVONSUO's thesis is our finding of a substantial association between fear and the sense of a malevolent, unseen, threatening presence.

How do we now deal with the substantial remainder of non-threatening dream experiences? Do one-third, or half, or two-thirds of dreams have evolutionary significance and the remainder reflect random error? If the remainder of dreams were an undifferentiated morass, perhaps the narrowness of the threat simulation hypothesis would be less problematic. Dreams, however, are characterized by other themes of, for example, sex and/or flying. Floating and flying are also rather common hypnagogic and hypnopompic sleep-paralysis related experiences and these are more strongly associated with blissful feelings than with fear. We have argued that some of these phenomenal experiences are consistent with attempts to integrate conflicting vestibular and motor program activation during REM (Cheyne et al. 1999b). For example, as in ordinary dreams, activation of pontine vestibular nuclei, in the absence of feedback from compensatory head movements because of inhibition of motoneurons during sleep paralysis, may give rise to experiences of flying during sleep paralysis. Similarly, activation of motor programs, which are inhibited at the base of the spinal cord, continue to generate associated corollary discharge, which produces illusory and somewhat ethereal (because of the absence of feedback from the periphery) movements such as locomotion (Hobson & McCarley 1977; Hobson et al. 1998c).

REVONSUO's raising of the evolutionary thesis does suggest the interesting possibility that these temporary dissociations may also serve important integrative functions relating different aspects of the neural representation of bodily senses – and perhaps even the assembling of neural patterns underlying what Damasio (1999) refers to as the core self. Assembling and integrating neural maps of self representations seem at least as fundamental evolutionary functions as coping with external threats.

The evolutionary claims **REVONSUO** makes for dreaming are very similar to claims that have been made for play since the work of Karl Groos (1896). In one of the more rigorous versions of this sort of account, Fagan (1976) borrowed an interesting notion from engineering, arguing that the difference between practice play and "normal" functional activity was the difference between control and information functions. This analysis might equally be applied to dreaming in light of REVONSUO's suggestions. The information function operates in a manner similar to that suggested for the simulative mode in dreams.

Fagan draws upon aviation for illustrations in which the dynamic properties of aircraft and of their control may be optimized by putting aircraft through "unusual" and "exaggerated" maneuvers that would never be executed in the interest of efficient flight. In the biological example of the cat playing with a captured mouse, variations in amplitude of pouncing, for example, test the limits of the prey's reactions. Indeed, one might even understand that those limits might entail going so far as to permit the prey to escape. Such information may be important for efficient development of strategies that trade off speed, force, and accuracy.

It is intriguing that this way of thinking about dreams suggests that dreaming, as a practice mode, may have some advantages over play. One possible constraint on play (and practice modes more generally) is that it generally requires a "tension free field" or a "secure base." That is, because the informational requirements of practice test the limits of the organism's capacities (i.e., practice play is inherently dangerous), it is best to do this under relatively safe environmental conditions. Even here there are always inherent risks undertaken when one pushes any system to its limits – deliberately or not. Hence, a potentially strong point in favor of

REVONSUO's thesis is that dreaming allows even greater boldness in stretching at least the neural parameters of practice. The motor hallucinations and fictive movements of dreams seldom simply reproduce the mundane movements of everyday life (Hobson et al. 1998b). Rather they often have the unusual and exaggerated features of play. The inhibition of the peripheral motor system in REM would also allow motor programs greater latitude to experiment with (simulations of) extreme maneuvers. Parallel arguments may be made for the range of affect intensity. The attenuation of the somatic body-loop, especially motor reactivity, may allow for less constraint on the neurological components of terror and bliss. Thus arguments for the advantages of play as a practice mode may hold with even greater force for dreams.

Iterative processing of information during sleep may improve consolidation

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Abstract: The relationship between sleep and memory has been controversial since the 1950s. Studies on delayed dream recall and long-term retention of pre-sleep stimuli indicate that sleep may have a positive role in the consolidation of information. This positive indication counterbalances the negative one from the studies on the effects of REM deprivation.
[VERTES & EASTMAN]

Periodically, a number of data are reinterpreted against, rather than in favor of, one or more of the three main hypotheses (interference, decay, consolidation) put forward for the relationship between sleep and memory. Such periodic reexamination is nevertheless useful to establish the value of the arguments brought by the various research strategies. The target article by **VERTES & EASTMAN** states that the evidence so far collected does not support any positive role of REM sleep in the consolidation of recently stored materials. This clear-cut conclusion is supported by two groups of complementary arguments, provided respectively by: (a) a thorough review of the data available on the effects of REM sleep deprivation induced using stressful laboratory techniques in animals and humans or by means of antidepressant drugs in humans; and (b) the interpretation of dream recall failure within the theoretical framework that information cannot be processed and consolidated during sleep as non-conscious state. However, the position of VERTES & EASTMAN cannot be considered conclusive, because the findings taken into account are not representative of the entire bulk of evidence available.

Concerning their first set of arguments, post-sleep retention of pre-sleep stimuli has been investigated by adopting two main strategies comparing respectively: (a) the retention rates after intervals of the same length, but characterized respectively by uninterrupted sleep and by selective sleep deprivation; and (b) the retention rates following sleep periods of similar length, but with different proportions of sleep stages (in particular, of REM and NREM sleep). By using the second strategy it has been shown that the capacity of enhancing retention is not exclusive to REM sleep; in particular, NREM sleep has a more positive effect than REM sleep on retention of simple stimuli such as paired words and sentences. These findings weaken the hypothesis of a superiority of REM sleep in determining long-term retention and also indicate that the sleep effect is influenced also by the characteristics of the materials to be retained.

As far as the second **VERTES & EASTMAN**'s argument is concerned, several items of evidence support the possibility that (a) dream contents obtain a certain level of consolidation during sleep (Cipolli et al. 1992), and (b) stimuli externally delivered during sleep are retained in short-term memory in both REM and NREM sleep (Shimizu et al. 1977). The fate of oblivion of many dream experiences (which are quite ubiquitous during all sleep stages) is only apparent: after failure in spontaneous recall dream: subjects are capable of providing an accurate report if appropriately prompted by means of some content or sort of title they provided after night awakening. This means that dream contents are not decayed from long-term memory, but are not accessible because of interferences between the contents of dreams elaborated over the night.

The retention of stimuli delivered during (both REM and NREM) sleep in short-term memory makes them available for operations which may enhance the degree of consolidation. This possibility is crucial to understand whether consolidation also occurs during sleep for materials stored before sleep. Some data on cued recall (Smith & Weeden 1990) and dream organization (Cipolli 1995) indicate that pre-sleep stimuli can be repeatedly activated and processed in subsequent sleep stages and cycles (as it usually occurs in waking). Repeated auditory stimulation during REM sleep has proved to be capable of enhancing memory of a task previously learned in the presence of the same stimulation. This suggests that external stimulation initiates "recall" of the recently learned material and makes it available for further processing. Moreover, pre-sleep stimuli (such as sentences) are repeatedly incorporated into the contents of dreams elaborated during different stages and cycles of sleep. The similar incorporation rates in REM and NREM sleep and the iterative accessing to pre-sleep stimuli suggest that some processes of implicit memory are at work during all stages of sleep.

Finally, the retention rate of those contents of different dreams which share the same semantic features (the so-called interrelated contents) and, thus, derive from the same materials in memory, is higher than the retention rate of other contents. This suggests that iterative processing during sleep improves consolidation for materials internally accessed for insertion into dreams as well as for materials activated by external stimuli to which they have been associated before sleep. The evidence available, even if not conclusive, makes it plausible that the interactive access and processing during sleep has some consolidative effect for recently stored materials.

Conceptual coordination bridges information processing and neurophysiology

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Abstract: Information processing theories of memory and skills can be reformulated in terms of how categories are physically and temporally related, a process called conceptual coordination. Dreaming can then be understood as a story-understanding process in which two mechanisms found in everyday comprehension are missing: conceiving sequences (chunking categories in time as a higher-order categorization) and coordinating across modalities (e.g., relating the sound of a word and the image of its meaning). On this basis, we can readily identify isomorphisms between dream phenomenology and neurophysiology, and explain the function of dreaming as facilitating future coordination of sequential, cross-modal categorization (i.e., REM sleep lowers activation thresholds, "unlearning").
[HOBSON ET AL.; NIELSEN; SOLMS; REVONSUO; VERTES & EASTMAN]

Now is a good time to bridge the different disciplines of the cognitive and neurosciences on the issue of dreams, with far-reaching implications for future theorizing across disciplines. But relating information processing theories to dream phenomenology and neurophysiology requires understanding the inherent, temporal basis of memory. In turn, a theory of consciousness can be developed that foregrounds how categories are constructed sequentially, cross-modally, and hierarchically in time (Clancey 1999), supported by **HOBSON ET AL.**'s analysis of REM neurophyisology.

From a connectionist perspective, the disinhibition of cross-modal activations suggests "reverse learning' (Crick & Mitchison 1983), by which the neural network is settling down to allow new associations to form or to lower the threshold required to coordinate experience sequentially. Thus procedural memory cannot be coordinated in REM sleep (ruling out any complex rehearsal of survival skills, contra **REVONSUO**). Instead, the function is to facilitate future learning in the awake state (contra **VERTES & EASTMAN**). Attempts to relate dreams to REM and NREM neurophysiology (**SOLMS** and **NIELSEN**) can be improved by characterizing how categories are related in sleep experiences.

What aspects of memory are missing? Dream phenomenology provides striking clues about the neurophysiology of REM sleep as well as aspects of memory and categorization that are essential for everyday consciousness. Perhaps of most importance are scene shifts and multi-modal discoordinations, which are taken for granted by the dreamer (**HOBSON ET AL.**). Freud (1900) characterized this phenomenology in terms of a rebus puzzle. For example in a dream I see a stick in the ground and say to myself "I have a lot at stake" and next am eating a steak. The meaning of the dream to me is revealed by my description, not the literal images or incidents (thus dream structure – the mix of images, sounds, and ideas – is organized by a verbal conceptualization of important concerns in my life, what I have "at stake").

However, to explain dream phenomenology in terms of neurophysiology, we need to characterize and relate both dream content and neurophysiology through an intermediate description of cognitive structure and temporal relationships (Clancey 1999). Finding an isomorphism between dream content and neurophysiology requires reformulating memory and learning in terms of categorization operating upon itself, eschewing notions of a random-access storehouse of beliefs and procedures (Clancey 1997).

Similarly, **HOBSON ET AL.**'s AIM model can be reformulated in terms of a categorization coordinating mechanism. The notion of "information" is characterized in neuropsychological terms as categorization (Edelman 1987); and "processing" is characterized as kinds of constructive operations by which multi-modal categories are physically and temporally related. Thus, I propose a three-layer analysis by which cognitive aspects of dreams and neurophysiology can be related:

1. Dream content (phenomenology).
2. Conceptual coordination analysis (structural and temporal relations of categorizing).
3. Neurophysiology analysis (neural activation between brain areas).

HOBSON ET AL. are right that a deficiency in memory goes a long way toward explaining orientational instability, loss of self-reflective awareness, and failure of directed thought and attention. However, the explanation is incomplete until we say more about what aspect of memory is relevant to these aspects of higher-order consciousness. This is the purpose of Level 2 in my analysis. What specific aspects of memory are missing?

Reformulating cognitive experience in terms of conceptual coordination, we find that higher-order consciousness (e.g., involving directed thought) requires three higher-order categorizing relations that are missing from REM sleep: (1) sequential correlation in multi-modal perceptual categorizing (e.g., relating sound and image), (2) holding a category active so it may be compared, counted, contrasted, etc., (3) categorizing a sequence of experience as a conceptual unit (chunking working memory). (See **HOBSON ET AL.** Figs. 4, 7, 8, 10.)

HOBSON ET AL.'s work shows nicely that the missing aspects of higher-order consciousness are due to aminergic demodulation. Or in conceptual coordination terms, the neurological mechanisms by which associations in different modalities are correlated or made consistent (sounds, images, and meanings correspond), by which categories are deliberately related, and by which episodes are held active (so that they may be objectified, named, and related) are not operating during REM sleep because of failures in aminergic neuromodulation.

In short, by viewing cognitive processes (information processing) in terms of how categories are formed and related (sequentially, hierarchically), the phenomenological structure of dreams can be explained. And by viewing these cognitive processes in terms of neurophysiology, their absence in REM sleep can be explained. This tripartite approach is essential because otherwise the phenomenology of dreams can only be loosely characterized in terms of "thought" or "episodes," and the role of the neurophysiolgical processes in everyday cognition will not be sufficiently articulated. As **HOBSON ET AL.** imply, psychology has heretofore failed to document the differences between waking and dreaming, just as it failed to document different kinds of consciousness among species, let alone between people and machines. This failure is rooted in a storage view of memory (with properties like copying and simultaneous multiple use of categories) and a verbally dominated view of thought (e.g., the assumption that visual thought and analogical reasoning only occurs by representing images as named objects and relationships, cf. Larkin & Simon 1987).

Relying on the information processing perspective of cognitive theory (such as a storage and retrieval view of memory), **HOBSON ET AL.**'s analysis is necessarily limited to talk about information instead of coordinated categorization in time. We can now reformulate AIM in conceptual coordination terms:

Activation (Information Processing Capacity) → perceptual categorization, scenes (coupled or synchronous categorizations), sequencing categorizations (episodes), holding a category active, holding a sequence of recent categorizations active (working memory), substitution within a sequence (e.g., saying chocaholic" by analogy to 'alcoholic"), categorizing a sequence (chunking, proceduralization), hierarchical activation of categories (bottom-up and/or top-down).

Input Source → perceptual categorization driven by: (external) sensory system, emotional correlation (e.g., dramatic theme such as "End of the World fear"), and/or conceptual (higher-order) categorization (e.g., verbal meanings influence imagery).

Modulation (Mode of IP) → how categories are conceptually coordinated, that is, how activation is modulated by other (higher-order) categorizations that are already active: correlating categories across modalities (especially sound, image, and meaning), counting, seeing-as, narrative, logical categorizing (e.g., implication, contradiction, identity), hierarchical goal-directed problem solving.

The changes in AIM during REM sleep involve an inability to hold a category or sequence of categorizations active (Activation), a mostly internally driven perceptual categorization (Input Source), and inabilities to conceptually coordinate across sensory systems and to categorize sequences (Modulation).

In summary, without the persistence enabled by sequential (and hence) temporal categorizing of the aminergic neurons, there is neither primary coordination sequencing required to follow and formulate procedural relations nor, consequently, secondary categorization (and awareness) of coordination that is occurring. Aminergic neurons are not categorizing sensorimotor activity over time (matrixing with cortical neurons is missing). With the shutdown of REM-off neurons, the reticular system is disinherited, contributing to the fantastic cross-modal activations of dreams, in which language, sounds, and images are freely associated. Attentional coordination is lost across systems, facilitated by the lack of feedback from sensorimotor interactions in the world. At the same time, the inability to hold non-sequential or non-synchronous categories active and relate them in time (which occurs in the conceptual coordination of higher-order consciousness) enables wild scene shifts.

How is activation of specific brain areas relevant? As we explicate how dreams are generated (what brain areas and paths are engaged), the conceptual coordination analysis can be mapped in more detail onto specific mechanisms involved in different aspects of categorization.

Insofar as dreaming occurs outside of REM sleep, as **SOLMS** ar-

gues, its story structure may be different from REM dreams. For instance, lucid dreams may combine disorientation and a capability to observe and comment on experience; whether these experiences are simultaneous or sequential is unclear. Building on SOLMS's (sect. 8) analysis, considering the kind of categorizing occurring in the person's experience may provide a clue about which areas are engaged and how they are relating to each other. For example, how are inabilities to hold a category active and to categorize sequences related to the deactivation of dorsolateral prefrontal cortex?

How does conceptual coordination differ during REM and NREM sleep? **NIELSEN**'s effort to characterize the mentation in different forms of sleep may be improved by characterizing the organization of cognitive activity in terms of how categories are related. I suggest the order: perceptual categorization, scenes (simultaneous relation of multiple perceptual categories as in seeing a pen and a knee), sequencing (one scene/event follows another), correlation within a sequence (e.g., a sound is followed by a causally corresponding image), holding a categorization active (e.g., comparing ideas), and categorization of a sequence (consciousness of "what I'm doing now").

Thus, **NIELSEN**'s Figure 1 might be improved by distinguishing the "cognitive processes" (item 4) that are higher-order categorizations missing in dreams (e.g., consolidation, rehearsal, plus forms of discrimination and selective attention) from the simpler relations found in dreams (e.g., perceptual memory activation, orienting/surprise). Aspects of conceptual coordination in sleep mentation can then be reordered (my Fig. 1) according to basic categorization (including NIELSEN's "preconscious precursors"), dreaming (scenes and narrative conceptualization), apex dreaming (protracted conceptualization of dramatic themes), and higher-order consciousness (sequentially coordinated ideas with causal and inferential relations, i.e., thinking). Because different kinds of conceptual coordination are occurring, it is too coarse to characterize NREM sleep as "more conceptual and thoughtlike." The question remains how thinking in NREM sleep and awake cognition differ.

What survival skills can be rehearsed without conceptual coordination? On a different level, **REVONSUO** has provided a broad-ranging, provocative account of the evolutionary function of dreaming. However, we must tighten up the notion of what is learned or reinforced and how what is learned relates to awake performance in the everyday environment. REVONSUO's analysis does not adequately distinguish between stimulus/response association and human inference. How could dreaming experiences, lacking basic aspects of goal-oriented attention, let alone reasoning by analogy and reinterpreting plans, constitute "training episodes" for skilled human performance in threat situations? The structure of dream experience, such as our inability to read text, reveals that conceptual coordination is impaired relative to awake cognitive activity, and hence we can rule out certain evolutionary benefits that require forms of logic, symbolic reference, and analogical reasoning. Although dream content reflects our everyday concerns, the primary function of dreaming, for humans at least, must be neurophysiological.

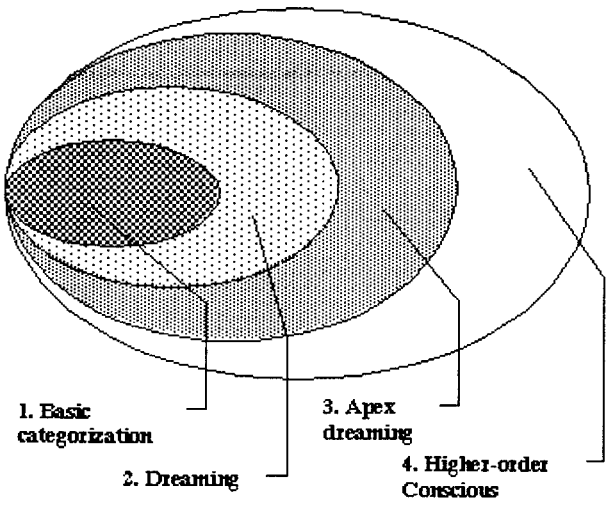

Figure 1 (Clancey). Revision of **NIELSEN**'s "levels of specificity" in terms of increasing conceptual coordination; each oval represents a form of consciousness in which simpler forms of categorization are temporally related in new ways.

However, we must proceed carefully. Conceptualization of meaning occurs in dreams without the associated summarizing and encapsulating statements of meaning by which reasoning occurs when awake. The restricted consciousness of dreaming allows formation of new "dream thoughts," but without the elaborated structure of causally coherent narrative and planning that higher-order consciousness allows. The effect of such experience on the awake planning of humans is unclear. A both-and theory is required: Dream phenomenology is both "the consequence of an active and organized process" and "a passive byproduct of disorganized activation" (sect. 3.3). The coherence of dream drama is most definitely not like the sequence coherence of a narrative story or extended episode of experience. Although a dream may have an overarching theme or setting, the co-presence of dream elements (people, objects, and events) and the shifting story line is fundamentally unlike the coherence a person experiences (and indeed insists upon) when awake. Reading, writing, and calculating are absent because a dreaming person is unable to coordinate imagery and verbalization with a calculus. Such skills require procedural coordination (goal-directed, sequential behavior that is hierarchically organized with categorization "bindings" that may be substituted or generalized as behavior occurs; see Clancey 1999). Dream experiences are indeed multi-modal, but they are not sequentially coordinated and therefore cannot be simulations of real experience. Dream experience lacks higher-order consciousness ("insight into our true condition," sect. 3 .6.l) – precisely what we rely upon to respond flexibly to threats in real life.

Human cognition is not just a stimulus-response system. Response to threats is not merely a matter of fight or flight. People anticipate (imagine what will happen next), plan (imagine what they might do next), make weapons (organize tools and get ready for some action). The complex behaviors involved in hunting and defending a habitat, especially in a social manner, are indeed skills. But they involve a kind of coordinated representation, reification, organization of materials, and behavior sequencing that are not possible during REM consciousness. What kind of simulations might be useful? Logical thinking!

Contrast the dream experience "stung by bees" with the skills of recognizing bee nests or areas where they might gather, methods of killing bees, getting honey from a bee hive, and interpreting how bee behaviors relate to climate and seasons. Aside from merely reinforcing a flight response, a dream about bees could at best reinforce a person's interest to learn more about bees or to attend to associated bee phenomena when awake.

One implication of **REVONSUO**'s theory is that dreaming reinforces an unthinking way of responding to threat situations, merely based on reactive, perceptually, and emotionally driven behavior. If this was indeed an evolutionary advantage, it was originally conferred on other mammals, not Pleistocene man. Such learned associations, if any, are not like skilled human knowledge, because they are not procedurally integrated and flexibly controlled.

The presence of realistic imagery is not sufficient, there must be deliberate behavior, namely sustained attention that holds a goal in mind and orients interpretation and action in a coordinated way to accomplish the goal. In a daydream we can imagine a sequence of events and actions, with controlled behavior. But we lack this capability when dreaming.

Examples of "implicit learning" when awake merely show that

correlations and sequential relations may be learned without reifying them into named objects and relations that are reasoned about (Clancey 1999). Nevertheless, the person is paying attention and performing with higher-order control. Indeed, dream experience lacks the correlation of incidents that is meaningful when awake, so how could a dream sequence produce a useful expectation of how events will unfold in awake life? **REVONSUO** cites evidence that REMD impairs memory for procedural or implicit tasks, which again supports the hypothesis that REM involves a relaxation/unlearning effect that facilitates later learning. So the benefit of REM would be to facilitate future learning in real-life situations, not to rehearse those situations during the dream itself.

At another level, if the average ancestral human were constantly confronted with threatening events (sect. 3.8.1), why would they need to be rehearsed? Everyday experience would surely provide enough practice to develop well-honed, adapted skills. Similarly, REVONSUO views survival skills too narrowly in terms of immediate physical dangers. Aren't "underrepresented peaceful activities," such as working in a forest, as important for survival as appropriate response to threats?

Does REM sleep facilitate future procedural learning? **VERTES & EASTMAN**'s thesis is strongly supported by a conceptual coordination perspective that processing and consolidation of experience requires aspects of consciousness that are missing during REM sleep. But the conclusion that REM sleep could only serve to maintain CNS activity during sleep is not warranted.

VERTES & EASTMAN cite effects of post-REM deprivation on performance of an already practiced task (sect. 2.5) that appear consistent with the hypothesis that REM sleep "settles" the activation level of cross-modal coordination, thus lowering the threshold required for stimulation and hence improving performance. This in itself does not show that consolidation is operating. Instead, performance when awake could be enhanced by the clarity of mind that results from a lowered threshold required to coordinate behavior, and hence an ease in reconstructing practiced skills. For example, sitting down to play the piano in the morning you may find that the passages you labored over the night before are now effortlessly recollected. The practiced behaviors reactivate on an uncluttered path, as irrelevant relations, such as the conceptual context in which the practice sessions occurred, are not salient.

VERTES & EASTMAN cite other REMD studies supporting the hypothesis that REM prepares the brain for future multi-modal coordination learning (sect. 2.2.2). Further experiments might accordingly focus on learning involving multi-modal coordination such as sight-reading music, text comprehension involving visualization and calculation, or navigation involving multisensory cues and spatial orientation. (See also studies of REM sleep integrity and duration cited by **NIELSEN**, sect. 2.2, which provide related support.)

What does dreaming reveal about consciousness? Perhaps the most exciting result of this analysis is what it reveals about consciousness. First, we are conscious when we dream – a major shift from the idea that sleep is an "unconscious" state. Second, story comprehension – making sense of experience through narrative conceptualization – is more fundamental than logical thought (Donald 1991). Third, the essential coordination abilities of awake human consciousness are to hold a category active as a kind of anchor (e.g., to find a correlate and thus to have a basis of a higher-order relational categorization, such as "x is bigger than y") and to hold a sequence active and categorize it (e.g., to conceive of an episode, a procedure or method). These neuropsychological processes enable formulating goals and means for accomplishing them.

Now we may more fruitfully inquire about cognition in other animals. Do all primates have the conceptual coordination capabilities described here? Is counting possible without being able to hold a category active (e.g., scanning objects and incrementing the total)? Do other animals experience in their awake state the rapid scene shifts of human dreams? Does language confer a more stable way of holding a category active, so that cats may be goal directed, but be easily distracted and fooled because they do not name their intentions and reason about shifts in their attention? Can some personality dysfunctions (Rosenfield 1992) be reformulated in terms of inability to coordinate a protracted sequence of "what I'm doing now" (evidenced by Mr. T's rapid categorical shifts of "who I am" [Sacks 1987])?

Such questions are possible only because we no longer take for granted the conceptual coordination capabilities (binding, matching, storing, iterating) that procedural programming languages have given cognitive modelers for free. Reformulating memory, attention, and thought in terms of the neuropsychological mechanisms of consciousness is a dramatic breakthrough – perhaps the most important advance since the information processing revolution in psychology fifty years ago.

The divorce of REM sleep and dreaming

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Abstract: The validity of dream recall is discussed. What is the relation between the actual dream and its later reflection? Nielsen proposes differential sleep mentation, which is probably determined by dream accessibility. Solms argues that REM sleep and dreaming are double dissociable states. Dreaming occurs outside REM sleep when cerebral activation is high enough. That various active sleep states correlate with vivid dream reports implies that REM sleep and dreaming are single dissociable states. Vertes & Eastman reject that REM sleep is involved in memory consolidation. Considerable evidence for this was obtained by REM deprivation studies with the dubious water tank technique.
[NIELSEN; SOLMS; VERTES & EASTMAN]

Introduction. Few discoveries have provoked so much discussion as that of Aserinsky and Kleitman in 1953. An immediate association was established between REM sleep and dreaming: REM sleep was supposed to be the physiological sub-layer of the psychological phenomenon of the dream. A sensation was caused when it was discovered that when people were aroused from REM sleep, they always could recall a vivid dream (Dement & Kleitman 1957a). It was too good to be true! On the physiological side a high brain activity, without actual movements, and on the psychological side, the dream, a clear visual event accompanied by related emotions that simulate reality. After this firm association was established, a method appeared enabling the collection of a large number of dream reports. Researchers needed only to awaken REM sleeping subjects and inquire about their dreams. This dream recall research has been carried out countless times globally with similar results. It thus seemed that everyone undergoes several dream periods per night, and that most dreams concern normal everyday occurrences (Hall & Van de Castle 1966). It is quite striking, however, that during dreaming there is no form of critical awareness. We are not surprised about uncommon or impossible events, at the easy integration of external stimuli into the ongoing dream, such as, for example, the call of an alarm clock, and even less so about the combination of daily events that have no apparent relationship to each other.

The marriage of REM sleep with dreaming was so favored by researchers that facts not confirming this view were ignored as being insignificant. But gradually this intimate relationship was clouded. The papers of **NIELSEN, SOLMS,** and **VERTES & EASTMAN** are taken to discuss three main issues in REM sleep and dreaming research. First, the validity of the dream recall technique. Hypotheses and viewpoints on dreaming are mainly based on the results of this technique of which the validity is unknown. Second, theories on REM sleep are based on the outcomes of REM sleep deprivation, for which, in animals, the controversial water tank technique is often used. The supposed relationship of REM sleep and dreaming caused viewpoints on REM sleep and

dreaming to become completely entangled. Third, and last but not least, the finding that dream mentation also occurred during slow wave sleep was ultimately decisive for the divorce of the two phenomena.

The validity of the dream recall paradigm. There is one point that deserves more attention and is much underexposed, although discussions about this point are not new (Cohen 1977b; Goodenough 1978). **NIELSEN**'s paper only touches upon the validity of the dream recall paradigm. The critical point here is that dreaming occurs during sleep and is not directly observable by researchers. The actual dream cannot be studied, but only its reflection in the real world as told by the subject. No one can directly verify the accuracy of dream reporting. A dream is what someone describes upon awakening and researchers infer a one-to-one relationship between the dream and the way it is reported. But a dream report is once removed from an event or memory. It is therefore impossible to exclude such confounding factors as poor memory, overestimation, suppression or the effects of psychoemotional factors on recall. Another problem can be that distractions at awakening impair dream recall. A further complicating matter is that there is only a short memory period after awakening in which the dream can be immediately recalled, while memory of a dream easily fades away in time. The depth of sleep from which one is aroused can also play a role in this process. Assume that awakening takes place from deep sleep; it takes time before recall can be made. Dreams seem to be recalled with ease only if the sleeper is awakened within seconds after the dream experience occurs. This short memory span for dreams is evidenced by the fact that so few people recall their dreams in the morning.

Thus, a main factor for recall is the accessibility of the actual dream, and among others is determined from the speed of awakening. This is smaller when one is aroused from light sleep compared to deep sleep with its high arousal threshold. In this way one can imagine a gradual course from **NIELSEN**'s four levels of specificity in sleep mentation, running from apex dreaming, to dreaming, to cognitive activity, and finally to cognitive processes; a gradual dream scale ranging from most vivid intense dreams towards vague impressions. Sometimes awakening is facilitated by a frightening or bizarre dream, which then is vividly remembered. And why are dreams so illusory? Could it be that dream recall stories tell us the truth about what actually happened during the dream? So the actual question is what is the relation between the "primary" process (the actual dream) and the "secondary" process (the report or the memory of this experience). How large can the bias be? Is a dream report a reliable enough reflection of the actual dream so that we can base hypotheses on dreaming?

The "marker"-technique, introduced by Dement and Wolpert (1958), is a paradigm that may touch on this problem. These authors tried to mark a point in the dream by inserting an external stimulus into a REM period. The marker was a fine spray of cold water ejected from a syringe on the head of the sleeping subject. If this stimulus did not awaken the person, the subject was then allowed to sleep for a few minutes before being awakened by the experimenter and asked to report his dream recall. In most cases the spray of water was vividly incorporated into the dream story, for example, as a story about a leaking roof. In this way it is possible to get an idea of the relation of what actually happened and the reflection of it afterwards. However, despite the attractiveness of this marker technique, as far as I know, this experiment has not been frequently replicated in the literature. I only found one other experiment in which '"marking" or "tagging" took place. Rechtschaffen et al. (1963a) applied this technique in slow wave sleep, generally with the same results as Dement and Wolpert (1958). Perhaps this paradigm, when systematically applied may be a useful one to gain more insight in the relation of actual dream happenings and the manner in which it is reflected.

NIELSEN postulates an alternative model to explain the finding of dream mentation outside REM sleep. Covert REM sleep processes occur during slow wave sleep and these episodes are closely related to REM sleep in the sense that just before and just following a REM sleep episode such covert process occurs. In the view that I favour and that I explained before, this extra assumption is superfluous. I have to admit, however, that both views are not proven. Nevertheless, by systematically investigating the amount and nature of recall in relation to the nature of sleep, expressed in preceding EEG characteristics, the aforementioned views can perhaps be distinguished. It is now possible to link the complexity of the EEG of a given time period with methods derived from non linear dynamics which give a much better index for complexity than the classic visual analysis and fast Fourier transformation (Pradhan et al. 1995). A positive correlation is then expected between the degree of recall and the dimensional complexity of the foregoing EEG.

The dubious role of the water tank technique of REM sleep deprivation. **VERTES & EASTMAN** dispute the hypothesis that memory is consolidated during REM sleep and that REM sleep has an exclusive function in memory consolidation. In this view REM sleep deprivation should lead to a poor memory consolidation. A main factor in the considerations of **VERTES & EASTMAN** is the effects on cognition obtained when REM sleep deprivation was induced by the water tank technique. This technique is usually applied in animals, and particularly in rats. I agree with **VERTES & EASTMAN** that a thorough review of the literature regarding the cognitive function of REM sleep yields ambiguous results: some supportive, some ambivalent, and some negative. Together with van Hulzen and van Luijtelaar, I have carried out REM sleep deprivation studies for many years and personally experienced the debatable, controversial results of this type of research. Evidence accumulates that the effects of REM sleep deprivation produced by the pedestal technique are merely dependent on the technique of inducing REM sleep deprivation itself, instead of on the genuine effects of the lack of REM sleep. The most disturbing effects on cognitive functions are indeed obtained by using the water tank technique, fewer with the multiple platform technique, and the least with the pendulum and the selective awakening techniques. Furthermore, we determined that the effects on cognition and behavior ran more or less in parallel with the stress accompanying the applied technique (Coenen & van Luijtelaar 1985).

This strongly points in the direction of side effects of the stressful water tank technique as being responsible for the induced effects. Differential effects on behavioral indices were also found by Oniani (1984) using the platform technique together with the selective arousal method. He found behavioral changes only when animals remained on the platforms for the whole period, but not when the last part of the deprivation period was completed with hand-awakenings. Kovalzon and Tsibulsky (1984) replicated the enhanced locomotor activity and the increased intracranial self stimulation found after platform REM sleep deprivation, but could not replicate such changes when deprivation was induced by midbrain reticular formation stimulation, a variant of the selective arousal technique. Van Hulzen and Coenen (1979) demonstrated that consolidation of active avoidance is not reduced after selective deprivation of REM sleep, in contrast to the platform technique. Thus, storage of information acquired during avoidance learning is not dependent on the presence of REM sleep immediately following learning. It is concluded that learning deficiencies obtained after platform deprivation were not owing to REM sleep deprivation per se, but to adverse platform effects. Such a position is now strongly shared by Fishbein (1995).

Unfortunately, despite much research the platform factor responsible for the cognitive and behavioral changes is not yet identified, though the stress factor seems to play a pivotal role (Coenen & van Luijtelaar 1985). After publication of all these results, a drop in the number of animal REM sleep deprivation studies could be observed. Nowadays, the number of studies, however, is again firmly increasing. While the published studies seem to be a tip of the iceberg of unpublished studies, I can easily find more than 50 published studies over the last 5 years. It is likely that the simplicity and cheapness of the technique are the reason for this increase, as well as ignoring the older debates in the literature. A

short survey of these studies shows a lot of diverging facts that are difficult to interpret. Moreover, studies are also directed toward a creation of a version of the classical water tank technique which induces even less stress than the multiple platform by placing more rats in the tank to overcome social isolation (Suchecki & Tufik 2000). Apart from such methodological studies, I would like to raise the question, in fact implicitly raised by **VERTES & EASTMAN**, of the acceptability of the water tank technique in sleep research. Thus, I challenge the international sleep society to thoroughly evaluate whether this technique is still acceptable according to international ethical guidelines, weighing the controversial effects that are difficult to interpret against exposing numerous animals to this technique. The scientific function of the flowerpot seems less adequate than the function for which it was originally designed!

Instead of a cognitive function for REM sleep, **VERTES & EASTMAN** propose a homeostatic function, reminiscent of the classic neural excitability hypothesis of REM sleep (Cohen & Dement 1965). A periodic endogenous stimulation maintains a requisite level of brain activation throughout sleep, and so promotes REM sleep, a faster recovery from sleep to wakefulness. Although I like this theory, the underlying evidence is still far from convincing. Based on this proposal it is now necessary that hypotheses on this proposal are formulated and adequately tested. Nevertheless in looking back on the results of the research of my group, in which all platform effects were disregarded and only effects obtained with the selective hand awakening and pendulum techniques were taken into account, a number of findings went in the same direction as the recent proposal of VERTES & EASTMAN. After deprivation, in some situations more behavioral activity was noticed (van Luijtelaar & Coenen 1985), a decrement in the amplitude of the evoked potential was found (van Hulzen & Coenen 1984), together with an increase in the number of beta-adrenoceptor sites in the cortex of the rat (Mogilnicka et al. 1986). All these effects, which were relatively small but significant, can be interpreted as belonging to a group of changes, all of which point to a small increase in the tonic arousal level as a result of deprivation (Coenen et al. 1986). It is inferred that REM sleep may be involved in regulating the arousal level in the waking state. However, I have to admit that all deprivation effects could also be ascribed to the drive of the brain to trigger REM sleep. To distinguish between these possibilities is a challenge for future research.

REM sleep and dreaming: Double or single dissociable states?

In his review **SOLMS** comes to convincing evidence for a relative independency of both phenomena. Before I comment on the paper of SOLMS, I will first make my own position in this matter clear. To this end I will first quote a passage of my paper (Coenen 1998):

> An important disappointment in dream research was that, now and then, but indeed consistently, non-REM sleeping subjects report dreams upon sudden awakening. This is a fundamental discrepancy that confounds the fixed relationship between REM sleep and dreaming. Although this finding has been abnegated as being insignificant, it cannot be refuted or overlooked. An opinion about dreaming could be that, if there is, for whatever reason, a sufficiently high brain activity during sleep, this may produce a dream. From this perspective, dreams are not the exclusive property of REM sleep; they are only the expression of a high brain activity during sleep. Accepting this explains the fact that an occasional dream recall during non-REM sleep can take place. One can be convinced that high brain activity that always accompanies REM sleep is at the core of dreaming, and causes the observer to mistake dreams rather than brain activity as the essential cause of REM sleep. The various dream-like phenomena that occur while one is falling into sleep, known as hypnagogic hallucinations, can also be declared as a mental expression of high brain activity. This type of dream event that occurs before one dozes off is unexplainable because the person's physiological state is not comparable to the REM sleep condition.

Based on supplementary evidence, **SOLMS**, to a large degree, agrees with the previous hypothesis. Dreaming may be the consequence of various forms of cerebral activation during sleep. He draws the conclusion that this implies a two-stage process. The first is cerebral activation during sleep and the second, the process of construction of a dream. In this respect Hobson and McCarley (1977) suggest that the cortex attempts to create a story from the bombardment from the brainstem and a dream story is the best fit the cortex could provide of this intense activity. They call this the activation-synthesis hypothesis of REM sleep. This view indeed implies a two-generator model. Firstly, cortical activation, which is for unexplained reasons of great importance; and secondly, a generator mechanism that creates a story based on this activation. Nevertheless, another view could also be that the dream is a mere by-product of this cortical activation. Perhaps, cerebral activation is the physiological basis underlying mental activity. Dreams could be merely the mental expression of intense activity in the brain that may be important for other reasons. In the same sense the noise of an automobile engine is merely a by-product of its running.

It is not completely clear what **SOLMS**'s viewpoint is on the previous models. He starts by accepting the statement that REM sleep and dreaming are double dissociable states: REM sleep can occur without dreaming and dreaming can occur without REM sleep. However, in the last part of his paper, in the reconsideration of the relationship between REM sleep and dreaming, SOLMS suggests an alternative explanation for the high correlation between REM sleep and dreaming. He mentions several examples of cerebral activation processes, such as induced by pathological processes and by stimulant drugs and also by REM sleep, and all are associated with dreaming. This thus implies that various brain states, which involve cerebral activation during sleep, are associated with dream reports. He thus shares my view on this, although his explanation of the one generator model (cerebral activation = dreaming) or the two generator model (where the brain itself creates a best fitting story for its own cerebral activity) is still unclear. Thus dreaming is not an intrinsic phenomenon of REM sleep, although dreaming always occurs during REM sleep. But I cannot see his often-mentioned point of the double dissociable states; the complete independency of REM sleep and dreaming. That dreaming can occur without REM sleep is now accepted, but the reverse is hard to accept. I also cannot find proof for this viewpoint. Given the unstable nature of memory for dreams, one can imagine that not every awakening from REM sleep results in a dream recall. In conclusion, rather than proposing, as SOLMS does, that REM sleep and dreaming are double dissociable states, it is perhaps better to regard them now as single dissociable states.

Shedding old assumptions and consolidating what we know: Toward an attention-based model of dreaming

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Abstract: Most current theoretical models of dreaming are built around an assumption that dream reports collected on awakening provide unbiased sampling of previous cognitive activity during sleep. However, such data are retrospective, requiring the recall of previous mental events from sleep on awakening. Thus, it is possible that dreaming occurs throughout sleep and differences in subsequent dream reports are owing to systematic differences in our ability to recall mentation on awakening. For this reason, it cannot be concluded with certainty that sleep cognition is more predominant or in any way different during REM compared to NREM sleep. It is our contention that REM sleep and ponto-geniculo-occipital (PGO) waves do not necessarily represent "pseudosensory" stimulation of the cortex in the generation of dreams, but might rather represent en-

hanced arousal of attention mechanisms during sleep, which results in the subsequent recall of attended mentation on awakening.
[HOBSON ET AL.; NIELSEN; REVONSUO; SOLMS; VERTES & EASTMAN]

Background. In 1953, Aserinsky and Klietman's discovery of a relationship between REM sleep and dream reporting reinforced growing biological reductionist concepts of brain-mind isomorphism. Such concepts also provided researchers with the impetus to study the biological mechanisms underlying REM sleep, with the hope that more general principles of hallucination could be established. This approach is now under threat, as evidence has mounted showing that REM sleep is not the exclusive domain of dreaming (e.g., **SOLMS**). In order to preserve underlying concepts of biological parallelism, researchers have hypothesized that processes underlying REM sleep could exist within NREM sleep (e.g., Pivik 1991; **NIELSEN**). However, a consistent relationship between underlying REM processes and dream reporting has not been found (see Pivik 1991). Regardless, animal-based PGO models have continued to dominate dream theory, despite the fact that a direct test of the relationship between PGO activity and dreaming has not been possible, as PGO activity cannot be directly measured in humans. Recent advances in PET neuroimaging techniques (e.g., Braun et al. 1997; 1998; Maquet et al. 1996), combined with brain lesion studies (Solms 1997a) have forced major modifications to the activation, inputs and modulation (AIM) model (**HOBSON ET AL.**). This model now suggests more modular PGO activation of association areas of the sensory cortex and limbic system in generating "pseudosensory" stimulation, rather than universal cortical activation or the activation of primary sensory areas suggested previously (e.g., Hobson & McCarley 1977; Stickgold et al. 1994a).

HOBSON ET AL. target article: Despite latest revisions to the AIM model, we believe some key aspects of its theoretical framework are still highly controversial for the following reasons:

1. Evidence against the notion that REM sleep is where dream mentation occurs and NREM sleep is predominantly a cognitive void: One central aspect of dream research often neglected is that psychological data regarding dreaming is collected retrospectively, requiring the recall of previous mental events on awakening. Therefore, in terms of strict scientific scrutiny, current evidence does not conclusively show that dreaming is more predominant or in any way different during REM compared to NREM sleep. It is possible that dreaming occurs throughout sleep and differences in subsequent mentation reports are due to differences in recall on awakening (e.g., Koukkou & Lehmann 1993). Most dream researchers assume that awake recall provides equal representation of previous REM and NREM sleep mentation. This may not be correct, as considerable data from sleep inertia research suggests cognitive performance parallels dream report frequency (best performance from REM to poorest performance from SWS; Dinges 1990). It is also interesting that poor dream report frequencies from NREM sleep are often treated as evidence for the absence of NREM dreams (e.g., **HOBSON ET AL.,** sect. 2), yet the absence of dream reports from REM sleep are often considered to be the poor recall of dreams (e.g., **HOBSON ET AL.** target article's sects. 2.2.1, 2.2.2). However, even if HOBSON ET AL.'s assumptions are accepted, there is considerable evidence against the proposal that REM sleep is a state of intense cognitive activity and NREM sleep is a relatively inactive brain state of low cognitive activity.

The results of recent PET studies are not always consistent with the AIM model of **HOBSON ET AL.** For example, EEG delta activity has been found to correlate positively with PET activation of the primary visual and secondary auditory cortex (Hofle et al. 1997). This led Hofle et al. (1997) to interpret this finding as reflecting "the occurrence of visual, auditory and perhaps verbal imagery during SWS" (p. 4806). Braun et al. (1997; 1998) have also demonstrated that the primary visual cortex consistently shows higher levels of activation during NREM sleep than during REM sleep. This led Braun et al. (1997) to observe that "SWS may not, as previously thought, represent a generalized decrease in neuronal activity" (p. 1173).

HOBSON ET AL.'s suggestion that the slow oscillatory rhythms of NREM sleep reflect decreases in brain activation are at odds with recent reports of Steriade and Amzica (1998), finding "frenzied" activity of cortical neurons during the depolarization phase of slow oscillations. "The frenzied activity of cortical neurons during the slow oscillation, occurring in natural sleep or deep anesthesia . . . during which consciousness is conventionally thought to be annihilated, prompts us to consider different roles played by the rhythmic bombardment of thalamic and cortical neurons upon their target" (Steriade & Amzica 1998, pp. 8–9).

HOBSON ET AL. also cite the observation of fast gamma frequency (30–70Hz) EEG and MEG oscillations during REM sleep (Llinas & Ribary 1993) as evidence for intense cognitive processing during REM sleep. However, gamma waves are also observable during NREM sleep (Llinas & Ribary 1993), SWS and deep anesthesia (Steriade & Amzica 1996). Such observations are inconsistent with the lack of PGO activity during these states. In acknowledging these inconsistencies, Kahn et al. (1997) state "the implications of finding the high frequency oscillations in NREM should be further investigated and the findings extended to human psychophysiology" (p. 23).

2. Evidence against the notion that PGO activity provides "pseudo-sensory" stimulation to the visual cortex: The original formulation of the Activation-Synthesis hypothesis was based on findings that pontine activation of eye-movements preceded activation of the cortex (Hobson & McCarley 1977). This hypothesis proposed that eye-movement and visual information was passed to the cortex from the pontine brainstem. This claim was also reinforced by the finding that patterns of PGO wave activity correlated with the direction of REMs during sleep (Nelson et al. 1983). However, this led to the claim that phasic PGO signals "led directly to the visual and motor hallucinations, emotion and distinctively bizarre cognition that characterize dream mentation." (**HOBSON ET AL.**, p. 41). Recently, evidence inconsistent with this "pseudosensory" nature of PGO waves has been derived from human neuroimaging and lesion studies. In fact, there is no consistent evidence supporting the notion that the primary sensory areas show enhanced metabolism during REM compared to NREM sleep (Braun et al. 1998). Nor is there any evidence that lesions to primary sensory areas eliminate dreaming (Solms 1997a). Possibly due to such findings, HOBSON ET AL. have revised their previous "pseudosensory" function of PGO activity. They have proposed that cortical and limbic regions may synthesize their own information when stimulated by PGO waves, claiming a similar induction of imagery to that of Solms's (1997a) concept of limbic back-projection to the visual association cortex. However, such hypotheses are still inconsistent with current imagery models of back projection to the striate cortex, which are based on PET data derived from subjects asked to view and imagine objects (Kosslyn & Thompson 2000). Imagery models based on awake subjects, are more scientifically sound, simply because we know with certainty that the PET data are derived from subjects engaged in visual imagery.

In support of their new version of PGO imagery generation, **HOBSON ET AL.** have cited findings that patterns of lateral geniculate nucleus (LGN) activity in waking cats are sufficient to represent basic elements of natural scenes (Stanley et al. 1999). However, earlier work showed that the occipital aspect of PGO waves was still present after LGN lesions (Hobson et al. 1969). It was then proposed that the thalamic aspect of PGO activity might not be entirely localized in the area of the LGN. In rats, PGO waves cannot be recorded in the LGN (Datta et al. 1998). Also, recent work by Marks et al. (1999) found that PGO innervation of the LGN in cats did not demonstrate the lamina specificity shown by retinal innervation of the LGN in visual processing. Marks et al. (1999) then conclude that the brainstem activation underlying PGO generation in the LGN controls neuronal activity in a different way to that of eye-specific, segregated retinal input to the LGN. In other words, the PGO influence on neuronal activity in the visual system is essentially different from that derived from visual experience.

However, despite **HOBSON ET AL.**'s new version of PGO imagery generation, the current model still reverts to the original activation synthesis concepts. For example: "Internally generated pseudosensory data can be produced by brainstem mechanisms (e.g., via PGO stimulation of visual cortex in REM sleep)" and "eye movement density in REM sleep provides an estimate of the amount of internally generated pseudosensory data because eye movement density reflects brain stem PGO and motor pattern generator activity" (HOBSON ET AL. pp. 55–56). We believe that to be accepted as a viable hypothesis, the "pseudosensory" role of PGO activity during REM sleep requires further clarification and investigation.

PGO activity represents the arousal of attentional awareness during sleep. Based on the initial proposal that dreaming might represent a state of attentional awareness without volitional attentional control (Posner & Rothbart 1998), Conduit (1999) has put forth an attention-based model of dreaming. In the attention-based model, PGO activity is related to the arousal of attention mechanisms during sleep. This arousal produces heightened attentional awareness during sleep, allowing potential recall of attended sleep mentation on awakening. Several lines of evidence support this proposal of arousal of attention mechanisms during sleep.

Orienting, attention, and PGO activity. Bowker and Morrison (1976) first raised the argument that the PGO wave was intimately linked to the startle response. They interpreted behaviors coincident with PGO activity as "alerting or orienting movements in response to some internal discharge, or as we suggest, 'startling' stimuli, that occur with each PGO spike appearance" (p. 188). However, years later, after extensive investigation of PGO and muscular variations in such things as timing, intensity and habituation, these researchers have come to a different conclusion: "neurons (we predict in the reticular formation) identify a signal that requires attention and that this requirement is passed via peribrachial neurons in the dorsal pons that respond to auditory stimuli and also generate PGO waves . . . thus, PGOs in the LGB could reflect a honing of neural mechanisms in the visual system to receive information" (Sanford et al. 1993, p. 443). Consistent with these findings, the pulvinar nucleus of the thalamus receives PGO outputs from the pons (Steriade et al. 1988). Furthermore; there are several lines of evidence showing the pulvinar has a central role in attention processes (Robinson & Peterson 1992). Thus, it is not unreasonable to suggest that phasic PGO activation of various regions of the thalamus could act to enhance the sensitivity and information gathering processes of a variety of sensory relay circuits (Sanford et al. 1994a), hence, heightening attention processes during sleep.

Eye movements and attention. In an approach that we believe is more consistent with an attention-based model than AIM, **HOBSON ET AL.** suggest that the observation of bottom-up control of attentional eye movement (EM) mechanisms during sleep provides evidence in favor of pontine generation of dream imagery, and the observation of top-down control of EM attention mechanisms provides evidence for a scanning hypothesis. Using such an approach, these authors have literally used the activation of attention as an operational definition of dreaming. So, what do we really know? We know that EMs occur during REM. We know that either brainstem or cortical mechanisms can generate and modulate EMs. However, we also know that EMs are usually preceded by a shift in attention (Chelazzi & Corbetta 2000). Therefore, we are fairly certain that attention mechanisms are activated during sleep. We have no conclusive evidence that bottom-up EM control represents the activation of "pseudosensory" imagery.

PET studies of REM sleep and attention. When one considers the possibility that REM may be a state of heightened attentional awareness during sleep, the amount of overlap in PET activation of brain areas during attention tasks in awake individuals and during REM sleep seems more than coincidental. PET studies of subjects during attention tasks have found activation of brain areas common to those activated during REM sleep. These include: the brainstem (particularly the reticular formation), thalamus (particularly the pulvinar nucleus), anterior cingulate, hippocampus, parahippocampal gyri, anterior cingulate, and scattered association areas of the posterior occipital/parieto/temporal neocortex (particularly the parietal and extrastriate areas; Chelazzi & Corbetta 2000; Lockwood et al. 1997; Posner 1994b). Also, shifts in attention have little observable effect in the primary visual cortex (except maybe when the visual field is highly cluttered; Posner & Digirolamo 2000). This result fits well with the specific activation of extrastriate association areas rather than the striate cortex during REM sleep (Braun et al. 1998). The dorsolateral prefrontal cortex is heavily implicated in executive attentional control (Posner & Digirolamo 2000) and in conjunction with attentional awareness is proposed to be necessary for consciousness (Posner & Rothbart 1998). This is compatible with findings that the dorsolateral prefrontal cortex shows little activation during REM sleep (e.g. Braun et al. 1997; 1998).

Electrophysiology during REM and attention. Electrophysiological studies support the proposal that REM is a sleep state of enhanced attentional awareness. A particular component of event related potentials (ERPs), the P300, is elicited in the waking state during the external orientation of attention in response to deviant stimuli or unexpected presentations. Sleep investigations have consistently produced the P300 during REM but not during other sleep stages (e.g., Cote & Campbell 1999). Occipital EEG alpha attenuation is also considered a physiological sign of the activation of visual attention. Recent research has found decreased occipital alpha spectral power during phasic REM periods compared to tonic REM (Cantero et al. 1999a).

Both electrophysiological and metabolic measures of neural activity during sleep can be interpreted as inconsistent with the AIM model. However, we believe such issues can only be resolved once the current temporal resolution of our investigative tools (PET and MRI) and the sleep scoring system we have adopted (Rechtschaffen & Kales 1968) are refined to adequately deal with sleep events lasting less than one second.

SOLMS target article: Some of the most challenging findings for the AIM model recently have been those derived from human lesion studies. Solms (1997a) essentially found that patients with brainstem lesions that eliminated REM sleep could still recall dreams, while patients with cortical lesions to areas such as the parieto-temporo-occipital (PTO) junction reported loss of dream recall with REM sleep intact. Hobson et al. (2000) dismissed the human brainstem lesion findings by stating that any lesion capable of eliminating the pontine REM sleep generator mechanism would eliminate consciousness altogether. After acknowledging this criticism, SOLMS has focused his latest review on the investigation of whether dreaming can be eliminated by forebrain lesions. From this, a large majority of cortical lesions resulting in the cessation of dreaming were located in or near the PTO junction (94/110). The small number of remaining lesions that eliminated dreaming were located near the ventro-mesial quadrant of the frontal lobe. SOLMS then argued that dreaming is driven by cortical back-projection, initiated from frontal DA circuits.

The brain lesion studies reviewed by **SOLMS** are also interpretable in terms of an attention-based model of dreaming. For example, frontal DA circuits have been implicated to play a key role in the regulation of attention processes (Granon et al. 2000), dysfunction of attention in schizophrenia (Swerdlow & Geyer 1998), and attention deficit disorder (Papa et al. 2000). Also, the underlying cognitive disorder of PTO lesions could be a deficit of visuo-spatial attention (see Posner 1994b). Solms's (1997a) findings that damage to extrastriate areas results in similar deficits in dreaming and waking perception is also consistent with an attention model. For example, when attending to the colour, form or motion of visual input, relative increases in neural activity occur within the same extrastriate areas that are believed to process such information (Posner 1994b). Solms (1997a) himself acknowledged his interpretations of the inhibitory function of the anterior cingulate and thalamus during dreaming were compatible with the

proposed functional role of these structures during attention. Generally it is accepted that attention processes act to suppress unattended areas, resulting in a relative enhancement in activity of the cells coding for the attended stimulus (Posner 1994b). PET studies suggest that thalamic and anterior cingulate inhibitory projections enable the selective modulation of posterior parietal and extrastriate areas of the brain during attention (Posner 1994b). SOLMS highlights the finding that lesions to the dorsolateral prefrontal cortex have no effect on dreaming, but are implicated in significant deficiencies of executive control, and hence might explain the executive deficiencies of dream cognition. These findings are consistent with PET findings of dorsolateral prefrontal deactivation during REM (e.g., Braun et al. 1997), and support the proposal that dreaming is an example of heightened attentional awareness with deficient executive attentional control (Posner & Rothbart 1998).

SOLMS's evidence that dreams are cortically initiated is not necessarily conclusive. It could be argued that the functioning of a lesioned brain does not necessarily reflect the full neural circuitry utilized by an intact brain. For example, SOLMS argues that dreaming can only occur if the DA circuits of the ventromesial forebrain are aroused, and thus REM sleep is simply a state that reflects the effects of cerebral activation of this region during sleep. However, in a sleeping, intact brain, cortical arousal is essentially derived from the ascending reticular activating system and/or the PGO generator of the brainstem (Steriade 1996). If normal spontaneous arousal during sleep does not arise from the brainstem, where is its origin?

REVONSUO's target article has put forth a convincing argument that any biological theory regarding the function of dreaming should be accountable through concepts of evolutionary biology. However, the notion that dream consciousness is a unique state providing a mechanism for simulating threat perception and threat avoidance responses currently has inadequate empirical support.

The main evidence cited supporting this proposal has come from the interpretation that dream content shows a significant bias toward representing threatening events. However, most of this evidence comes from dream reports collected from home using dream diaries (e.g., Hall & Van de Castle 1966). Foulkes & Cavallero (1993a) have argued against the assumption that spontaneous dream reports collected from home provide a true representation of the nature of dreams. Human memory research suggests that events attracting attention by being more emotional or unusual are more easily recalled (Brown & Kulik 1977). Thus, vivid, emotional and/or bizarre dreams may be the majority that are reported simply because these are cognitive events we more reliably remember (Cohen & MacNeilage 1974; Van den Hout et al. 1989). If this is indeed the case, such recall biases might persist even in controlled laboratory awakenings. Foulkes and Cavallero (1993a) describe the results of research using systematic REM (and NREM) awakenings as "surprisingly mundane, built around relatively realistic situations" (p. 11). Considering such arguments, it is possible that the "over-representation" of threat in dreams may be due to the way we selectively attend and recall information, especially from spontaneous awakenings at home. This point is worth noting, particularly since **REVONSUO** is continuing to collect home-based dream reports in support of his theoretical claims (sect. 3. 3).

Even if we concede that "threatening" events are over-represented in dreams, rather than more easily remembered, **REVONSUO**'s "threat simulation" proposal is not convincing. For example, if subjects were asked to recall events from their life history, emotionally significant or "threatening" events would probably be most prominent (Brown & Kulik 1977). In other words, all of the events we dream about must have an origin in memory. Therefore, it is the selective nature of attention and memory consolidation during waking that can provide an explanation for the proposed over-representation of "threatening" dream content, rather than a biased dream generation mechanism. This explanation can also be put forth for why adults dream of "current concerns," like for example, divorce. People dreaming of current concerns are also thinking and attending to these problems in their waking life. Hence, their dreams reflect their current psychological state, and do not necessarily provide an overrepresentation of "threat."

Our attention-based model of dreaming is more compatible with Snyder's (1966) "Sentinel" hypothesis of dreaming than **REVONSUO**'s model. Just as PGO mechanisms in an awake animal can heighten sensory awareness to deal with a possible approaching predator (Sanford et al. 1993), PGO activity during sleep might serve a similar function. Thus, it might be that phasic PGO waves act to periodically arouse the attention circuits of the brain enabling potentially threatening stimuli (such as novel or emotionally significant stimuli) to be perceived. If an external stimulus cannot be recognized as a "safe" stimulus (expected or of no emotional significance), the "novelty" or "emotional significance" of the stimulus should induce further attention and arousal to a point where a decision can be made as to whether there is a threat to survival.

VERTES & EASTMAN provide a strong case against the notion that memory consolidation occurs during REM sleep. However, a convincing alternative to the opposing consolidation model is not provided. We believe that if the activation model of VERTES & EASTMAN was developed to support the existence of heightened attentional awareness during sleep, a more consistent account of the existing data could be proposed in opposition to the consolidation model.

VERTES & EASTMAN offers an alternative to the memory consolidation function of REM sleep, arguing that REM provides "periodic endogenous stimulation to the brain" which maintains the "minimum requisite levels of CNS activity throughout sleep without awakening the subject or disturbing the continuity of sleep." If this is the case, what purpose does such stimulation serve? VERTES & EASTMAN begin to address this issue by stating that REM serves to "prime the brain for a return to consciousness as waking approaches" (sect. 6.5). Such interpretations are compatible with an attention-based model of REM sleep and dreaming, as the phasic arousal of attention mechanisms can be interpreted as a form of environmental monitoring in case of attack from predators, equivalent to Snyder's (1966) "sentinel" of sleep.

VERTES & EASTMAN provide a convincing critique of studies involving performance measures taken after REM deprivation, arguing that these studies are confounded by factors such as the stress and physically debilitating effects of deprivation procedures. However, VERTES & EASTMAN do not offer a strong explanation for observed increases in REM sleep after exposure to novel, enriched or enhanced "learning" environments. An attention-based model would predict that any altered environmental conditions will increase perceptions of possible danger or predation during sleep and thus result in REM enhancement and poorer sleep quality.

VERTES & EASTMAN refer to work relating hippocampal theta to long term potentiation and the observation that such activity is highly prominent during REM sleep (Winson 1993). They argue that the theta rhythm is generated as a by-product of the activation of brainstem mechanisms during REM and does not necessarily bear any functional relationship to its role in waking. However, such brainstem activation of the hippocampus is also present in waking during the engagement of attention (Buhusi & Schmajuk 1996). Curiously, VERTES & EASTMAN's proposal that theta "serves to gate and/or encode information reaching the hippocampus" (sect. 2.6) is analogous to other researchers interpretations that theta is involved in attentional processing (Buhusi & Schmajuk 1996). Under an attention-based model, hippocampal theta can be interpreted as a role in attention rather than memory consolidation, thus the unconvincing argument that theta indicates a different function depending on sleep state is not necessary.

An attention-based model of REM sleep and dreaming would maintain that cognition regarding salient memories previously

consolidated during waking is attended to during REM/phasic sleep and thus may be recalled if the subject is awakened. On awakening, if there is any disruption, for example, a delay or distraction, recall will be impaired (Goodenough 1991), as in attentional blanking (Lawson et al. 1999). If consolidation of dream mentation occurs, it must occur following awakening; otherwise, dream mentation would be no more elusive to memory than any other waking event. Thus, from one source of insight into sleep cognition (dreaming), it seems that memory consolidation does not necessarily occur during sleep. Apart from the conditioning of reflexive physiological responses during sleep (e.g., Conduit & Coleman 1998), higher forms of learning requiring memory consolidation do not seem to be possible during sleep (Eich 1990).

NIELSEN's proposal of "covert" REM sleep processes is an important and interesting one. It highlights the problems that modern sleep researchers have when attempting to investigate the possible existence of sleep phenomena which last for seconds (e.g., alpha blocking; Cantero et al. 1999a) or even milliseconds (e.g. PGO waves; **HOBSON ET AL.**), within a definition of sleep which has as its smallest unit, an epoch of 30 seconds (Rechtschaffen & Kales 1968). As new electrophysiology techniques approach submillisecond temporal resolution and PET/MRI scanning resolution is a matter of seconds, the current system of defining sleep must accommodate these advances if our understanding of sleep is to progress.

In many ways, **NIELSEN**'s model parallels previous tonic/phasic models of sleep mentation. Such models proposed the existence of phasic sleep processes (primarily PGO waves) underlying the recall of mentation from sleep (see Pivik 1991). Several lines of evidence presented as supporting NIELSEN's model were originally cited as evidence for tonic/phasic models. These include: the proximity of NREM sleep awakenings to REM sleep (e.g., Stickgold et al. 1994a), REM deprivation effects on NREM recall (Foulkes et al. 1968), drug effects on NREM recall (e.g., Delorme et al. 1965) and sensory stimulation effects (e.g. Conduit et al. 1997).

However, since **NIELSEN**'s covert REM model is not necessarily dependent on the existence of PGO activity, this has allowed the incorporation of previous results relating sleep arousal to imagery reporting, where PGO activity is often absent. Findings such as sleep onset imagery (Vogel 1991), time of night effects (Rosenlicht et al. 1994) and sleep terrors (Broughton 1995) were previously considered inconsistent or incompatible with tonic/phasic PGO models (Pivik 1991).

NIELSEN states that the covert REM model is "similar to the one-generator model in that it assumes commonality of processes for all mentation reports, but it differs in that it extends this commonality to physiological processes" (sect. 3.2). The question that remains is what is this common underlying physiological process?

Conclusions. Previously, we have proposed that PGO activity might be indirectly related to dream reporting through the phasic activation of arousal, which then provides optimum conditions for the recall of ongoing mentation from sleep on awakening (Conduit et al. 1997). However, recent neuroimaging findings from REM sleep (Braun et al. 1997; 1998; Maquet et al. 1996), have shown PET activation of brain regions involved in attention. We now believe that heightened attentional awareness provides the conditions for subsequent recall of dreams on awakening and the unique characteristics of this recalled mentation. Thus, it might be the arousal of attention mechanisms that is the underlying physiological process of **NIELSEN**'s covert REM model, and might also better describe the "A" aspect of the AIM model of **HOBSON ET AL.** It might be damage to the brain mechanisms of attention that underlie the lack of dream reporting in patients suffering lesions to the parieto-temporo-occipital junction or the ventromesial quadrant of the frontal lobe, or the excessive dreaming of patients with damage to the anterior cingulate or thalamus (**SOLMS**). Arousal of attention mechanisms during sleep can be interpreted as supporting the case against memory consolidation during REM sleep, as memory is already consolidated and it is heightened attentional awareness that is present during REM sleep. Finally, there is a sound evolutionary rationale for heightening attentional awareness during REM sleep, as it can be viewed as a mechanism of periodic environmental monitoring. However, if we are to even attempt to begin the testing of such new proposals, we must review our outdated sleep classification methods, so that at least our definition of sleep has the same temporal resolution of our current investigative tools.

Needed: A new theory

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Abstract: Dream content is more coherent, consistent over time, and continuous with waking emotional concerns than most brainstem-driven theories of dreaming allow, but dreaming probably has no adaptive function. A new neurocognitive perspective focusing on the forebrain system of dream generation should begin with the findings on dream content in adults and the developmental nature of dreaming in children.
[HOBSON ET AL.; NIELSEN; REVONSUO; SOLMS; VERTES & EASTMAN]

Introduction. A large body of findings with the Hall and Van de Castle (1966) coding system shows that dreams are more coherent, consistent over time for both individuals and groups, and continuous with past and present waking emotional concerns than **HOBSON ET AL.**'s emphasis on brainstem-driven bizarreness can accommodate (Domhoff 1996). In addition, Foulkes's (1982b; 1999) laboratory discovery of low levels of dreaming until ages 9–11 joins **SOLMS**'s (sect. 6) findings with brain-lesioned patients in demonstrating that REM sleep is insufficient for dreaming. A new neurocognitive theory of dreaming therefore should begin with the hypothesis that Foulkes's developmental findings may correlate with the maturation of the forebrain system of dream generation first uncovered through creative neuropsychological detective work by SOLMS (sect. 8). In addition, the findings with the Hall/Van de Castle system on the lifelong persistence of various kinds of negative dream content suggest there is a "repetition dimension" in people's dream life (Domhoff 1993; 1996) that may relate to the temporal-limbic and frontal-limbic origins of dreaming in SOLMS's (sect. 7) model.

Contrary to **REVONSUO** (sect. 3.2), however, it is doubtful that dreams have any adaptive function. There are too many people, including children and brain-lesioned patients, who sleep adequately without them, and no evidence that either recalled or unremembered dreams have any functions (Antrobus 1993a; Foulkes 1985; 1993a). At best, people in some societies have invented uses for dreams, and in that sense dreams have an "emergent function that develops through culture" (Domhoff 1993, p. 315). Moreover, there is no evidence from systematic psychological studies that supports any psychotherapy-based dream theory claiming one or another function for dreams (Domhoff 1999a; Fisher & Greenberg 1977; 1996; Foulkes 1985).

The brainstem/bizarreness commitment. Both **HOBSON ET AL.** (sect. 4) and **NIELSEN** (sect. 3) present interesting ideas that may explain away much of the "dreaming" in NREM sleep. However, they do concede there is enough dreamlike mental activity in NREM sleep to challenge the strict equation of dreaming and the REM stage of sleep, especially late in the sleep period. The empirical dream psychologists who abandoned the REM sleep/dreaming equation decades ago in the face of contradictory evidence summarized by Berger (1967; 1969), Foulkes (1966; 1967), and Hall (1967) did not ask for much more than what is now granted in these articles. **HOBSON ET AL.** are wrong to chastise psychologists (sect. 2.3.3) for focusing on the cognitive level when the constant changes in their own model show that their comprehensive mind-brain isomorphism is extremely premature (sect. 3).

NIELSEN (sects. 3.1–3.11) nicely demonstrates the arbitrariness of the "stages" of sleep agreed upon by Rechtschaffen and Kales (1968) in the face of great inconsistencies from laboratory to laboratory in analyzing sleep records. Considering the large number of situations that can lead to "missed" REM periods, "intermediate" sleep, stimulation-induced REM sleep, and transitions to Stage II during REM sleep, it would be interesting to know what percentage of a night's sleep is consistent with the scoring manual in a representative sample of uninterrupted nights of sleep from normal participants. A low percentage would strengthen **NIELSEN**'s (sect. 3.14) call for a view of sleep stages as "fluid" and "interactive," which finds echoes in **HOBSON ET AL.**'s (sect. 4) emphasis on "dissociation" and "psychophysiological continua."

It is regrettable that **HOBSON ET AL.** took so long to broaden their theory in the face of contradictory evidence available long ago (Vogel 1978a), but it is possible that the "state" transition at sleep onset (sect. 4.2.2) and the greater activation late in a sleep period (sect. 3.3.4.3) explain much dreamlike NREM mentation. The disappointment is their continuing brainstem commitment, which is also preserved by **NIELSEN** (sect. 3) through his concept of "covert REM sleep." In the face of the new and old findings synthesized by **SOLMS** (sect. 6) to show that brainstem activation is not sufficient for dreaming, and in some unknown percentage of cases may not even be necessary, it would seem that research relating the forebrain system to many different aspects of dream content should now be the primary focus of mind-brain isomorphists.

HOBSON ET AL. (sect. 2.3.3) justify their desire to keep the brainstem at the forefront of their theory on the basis of a commitment to a mind-brain isomorphism. However, this insistence may also be due to their strong belief that dreams are bizarre and discontinuous, although one of their own studies reported "discontinuities" in only 34% of 200 dreams (Rittenhouse et al. 1994). Most others who have studied large samples of dream reports from groups and individuals see dreams as even more realistic (Dorus et al. 1971; Foulkes 1985; Snyder 1970; Strauch & Meier 1996). For example, Hall (1966) concluded that only 10% of 815 home and laboratory reports from 14 adult males had at least one "unusual element," using a scale that can be found in Domhoff (1996). In studies comparing REM reports to samples of waking thought collected from participants reclining in a darkened room, the waking samples were rated as more dreamlike (Reinsel et al. 1986; 1992).

To support their focus on brainstem activation and the bizarre nature of dream content, **HOBSON ET AL.** have to challenge several different sets of impressive findings. First, they reject (sect. 2.3.1) Foulkes's (1982b; 1999) conclusions on the low levels of REM dreaming in young children with the claim that these children are not able to communicate in words about their dreams. But Foulkes's data show that the rate of recall correlates with visuospatial skills, and that there are older children with good communication skills and poor visuospatial skills who do not recall very many dreams in the laboratory. It is more likely that young children do not dream often or well by adult standards, a conclusion favoring a cognitive theory of dreams.

HOBSON ET AL. (sect. 2.3.3) reject Foulkes's findings on the banality of the few dreams his young participants did report by saying the laboratory situation is not conducive to typical dreaming, but Foulkes (1979; 1996b; 1999) already has answered that claim very effectively. More generally, they overstate the differences between home and laboratory dreams. This is shown most recently in a reanalysis using effect sizes (Domhoff & Schneider 1999) with the original codings from the most comprehensive study of this issue, which was carried out by Hall (1966) with 11 young adult male participants who each spent three to four consecutive weeks sleeping in a laboratory bedroom in a house in a residential neighborhood.

HOBSON ET AL. (sect. 2.3.3) denigrate the findings on the everyday nature of most dream content by saying that psychological measurement has not been adequate, but they have not demonstrated that their evolving rating scales for the slippery concept of bizarreness can be used reliably across laboratories. Furthermore, they ignore most of the findings with the Hall/Van de Castle system, which has shown high reliability when used by researchers in many different countries and produced results that have been replicated several times (Domhoff 1996; 1999b) However, **HOBSON ET AL.** (sect. 2.1) do note the Hall/Van de Castle findings on emotion in dreams, which anticipate their own findings of more negative than positive emotions, more reports of emotions in women's dreams, and no gender differences in the distribution of emotions (Merritt et al. 1994).

In their effort to emphasize differences between REM and NREM reports, **HOBSON ET AL.** (sect. 2.2.2) argue against any control for length of report. In so doing they do not seem to realize this problem is handled without loss of data by the indicators based on percentages and ratios that are now standard in the Hall/Van de Castle system (Domhoff 1999b; Schneider & Domhoff 1995).

NIELSEN (sect. 2.9.2) also discusses this issue, but does not come to any conclusion, perhaps because he did not make enough of a study in his laboratory using Hall/Van de Castle indicators with 20 REM and 18 Stage 2 NREM reports (Faucher et al. 1999). It showed the REM reports had higher rates of aggressive social interaction even with this small sample size, which is an impressive result because aggression is more sensitive to age, gender, culture, and home/laboratory comparisons than any other variable (Domhoff & Schneider 1999).

Strong support for the use of the Hall/Van de Castle content indicators in resolving disputes about the nature of REM and NREM reports is provided by a study Hall carried out three decades ago, but that was only recently reported by Domhoff and Schneider (1999). When NREM reports from early and late in the sleep period were compared with REM reports, several of the usual differences appeared. For example, the "cognitive activities percent" (the number of cognitive activities divided by the total number of all activities) was 20% in NREM reports, but only 11 % in REM reports. Conversely, the "verbal activity percent" was 37% in REM reports, but only 22% in REM reports. However, the NREM reports from after the third REM period of the night were more similar to REM reports than early NREM reports on a summary measure for a wide range of Hall/Van de Castle categories. These results are consistent with the recent theorizing by **HOBSON ET AL.** (sect. 3.3.4.3)

HOBSON ET AL. (sect. 2.3.2) call for studies of dreams at home to obtain a more realistic sample of dream content, but they overlook the replicated longitudinal results with the Hall/Van de Castle system, which show that dream content can be constant for individual adults over years and decades, something that might not be expected if dreaming is as chaotic and bizarre as they claim (Domhoff 1996). One of these longitudinal studies showed that the dreams of "the Engine Man," used by Hobson (1988b) to show the bizzareness of dream structure, are highly consistent in content over just a three-month period. His dreams are also below the male norms on key social interactions, and continuous with his waking life in terms of the people and activities in his dreams (Domhoff 1996).

Dream function dream negativism. **REVONSUO** (sect. 2.2) does a convincing job of critiquing rival functional theories, and his "threat simulation" hypothesis draws on an impressive array of ideas from many different kinds of studies (sect. 3.4). Unfortunately, several pieces of his complex argument are highly speculative, including his most crucial sleep/dream claim, the attribution of mental training and implicit learning (sect. 3.7) to REM sleep (**VERTES & EASTMAN**). It also seems unlikely that trauma could stimulate the development of dreaming (sect. 3.5.2), since Foulkes (1982b) found that children with tense home environments or personal problems did not report more dreams, or more negative content, than did other children. Nor is it possible to agree with the idea that the stereotypic movements of decorticated cats could be the acting out of dreams (sect. 4.2) because it is highly doubtful that animals dream (Foulkes 1983). Finally, it is hard to imag-

ine that chase and attack dreams, which rarely contain successful defensive actions in any event, could make human beings any more primed for reacting to threat than they are due to the one-trial fear-conditioning system that is already found in reptiles (Le Doux 1996).

However, **REVONSUO** (sect. 3.5) is on to something when he links negative dreams and the "repetition dimension" (Domhoff 1993; 1996) to the vigilance/fear system centered in the amygdala. If this idea is placed within the context of ontogenetic development and **SOLMS**'s (sect. 7) ideas on the forebrain mechanisms that activate dreaming, then REVONSUO has made a good case that the repetition dimension expresses a person's history of emotional concerns. Just as emotional memories can last a lifetime, so too can posttraumatic stress disorder dreams, recurrent dreams, recurrent themes in dreams, and heightened scores on Hall/Van de Castle indicators. Most generally, then, the available evidence suggests that dreams are both non-adaptive and psychologically revealing (Foulkes 1993a; 1999).

Conclusion. If the methodologically most sound descriptive empirical findings were to be used as the starting point for future dream theorizing, the picture would look like this: (1) dreaming is a cognitive achievement that develops throughout childhood (Foulkes 1999); (2) there is a forebrain network for dream generation that is most often triggered by brainstem activation (Hobson et al. 1998b; Solms 1997a); and (3) much of dream content is coherent, consistent over time, and continuous with past or present waking emotional concerns (Domhoff 1996). None of the papers reviewed in this commentary puts forth a theory that encompasses all three of these well-grounded conclusions. This suggests the need for a new neurocognitive theory of dreaming (Domhoff 2000).

Mesolimbic dopamine and the neuropsychology of dreaming: Some caution and reconsiderations

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Abstract: New findings point to a role for mesolimbic DA circuits in the generation of dreaming. We disagree with Solms about these structures having an exclusive role in generating dreams. We review data suggesting that dreaming can be interrupted at different levels of processing and that anterior-subcortical lesions associated with dream cessation are unlikely to produce selective hypodopaminergic dynamic impairments.
[HOBSON ET AL.; NIELSEN; SOLMS]

The cessation of dreaming after bilateral lesions of the deep white matter surrounding the tip of lateral ventricles is the relevant and original contribution of Solms (1997a) to the neuropsychology of dreaming. Starting from this evidence, which seems corroborated by brain imaging findings showing activation of several limbic structures in the medial basal forebrain, **SOLMS** now ascribes a fundamental and virtually exclusive role to the dopaminergic mesolimbic structures of the "reward-motivational system" in the generation of the dreaming state both within or outside REM sleep (provided a sufficient level of vigilance). But dreaming consists of a variety of concomitant neurocognitive operations; several hindbrain and forebrain mechanisms and several neurochemical systems maybe involved in a active construction and recall of dream experiences. We will critically review some of the arguments raised by SOLMS in favor of his hypothesis.

Clinical and neurochemical evidence. The clinical correlates of global dream cessation documented by Solms (1997a) are not in themselves evidence that disruption of the reward-motivational system is playing a central role. Adynamia scarcely differentiated dreamers and non dreamers ($p < .1$), whereas measures of frontal function (preservation, $p < .001$) did. This points to the minimal specificity in the cognitive disorder induced by bilateral lesions of the deep white matter anterior to the tip of the lateral ventricular horns. In addition to their adynamia, these patients seem to suffer from a very severe deficit of attentional self monitoring. Solms (1997a) did not provide adequate measures of vigilance for these patients. (i.e., level of arousal was only clinically defined). Dream recall in frontal patients with lack of interest in initiating and sustaining actions should be reinvestigated with more adequate and specific tests to determine whether they suffer a general and diffuse deficit of intensive and/or selective attentional processing or whether their oneiric impairment arises from emotional-motivational deficit. In the latter case it should be further investigated whether the motivational impairment is a low level one (general hypoactivation) or whether it affects higher level processing (motivational learning and discrimination; Gaffan & Murray 1990). From a neuroanatomical point of view, bilateral lesions of the deep white matter anterior and inferior to the lateral ventricles are unlikely to interfere selectively with dopaminergic transmission because, for example, both noradrenegic fibers (Morrison et al. 1981) and cholinergic ones (Selden et al. 1998) traverse the same area to innervate very large sections of the cortical mantle. Hence damage to the deep frontobasal white matter probably has complex clinical and neurochemical effects that cannot be reduced to hypodopaminergic adynamia.

The idea that dopamine agonists and antagonists have opposite effects in increasing and decreasing hallucinatory activity is not completely convincing for at least two reasons: (1) It arbitrarily equates different hallucinatory phenomena endowed with different physiological and phenomenological qualities. Dopaminergic activation certainly plays a role in all these phenomena but the same phenomena cannot be exclusively defined by the level of DA activity. (2) It de-emphasizes the fact that cho1inergic agonists can also induce dream-like activity (Sitaram et al. 1978a).

Brain imaging evidence. In all the published activation studies, subjects underwent prior total sleep deprivation (36–48 hours). In their thorough discussion Braun et al. (1997) acknowledged the potential confounding effects produced by sleep deprivation. Here, we also recall that (1) Sleep deprivation first affects vegetative activities and the emotional section of the anterior cingulate (area 24 in the inferior genual area) is implicated in the regulation of vegetative responses (Devinsky et al. 1995) (2) Sleep deprivation (in particular REM sleep deprivation) enhances DA activity (Brock et al. 1995). Without denying the contribution of motivational-emotional activation to the shaping of dreams (although not all dreams are necessarily endowed with relevant emotional content; see target article by **NIELSEN**), one might suggest caution about the role attributed to dopaminergic activation of mesolimbic structures in the generation of dreams.

Notes and conclusions. The assertion that published cases of loss of dream recall following stroke can be grouped as frontal ones (deep white matter) and parietal ones is incorrect and incomplete. In our (balanced) review of the literature (Doricchi & Violani 1992) we documented a consistent and clinically homogenous body of cases in which total dream cessation accompanied infero-mesial lesions producing visual-verbal disconnection. In the same review, which included 104 cases published in the neurological literature starting from 1883, we reported the nosology of preserved dream recall after frontal lesions with relevant involvement of the underlying white matter, the predominance of dream cessation after posterior lesions, and the loss of the visual component of dreaming. Solms (1997a) confirmed all these findings and provided further informative data on the locus of the lesion that suppresses the visual component of dreaming.

In the same paper, we formulated many specific and testable hypotheses on the relationship between left-hemisphere linguistic-semantic processing impairments and lack of dream recall as well as on the role of posterior parietal – temporal areas in the spatial

shaping of dreams and the modulation of oculomotor activity in REM sleep (see also Doricchi et al. 1993; 1996). Unfortunately Solms (1997a) did not specifically test these hypotheses and his present target article rejects the role of posterior dorsal areas as supporting various processes involved in visual imagery. Citing our 1993 review (since all other relevant literature is reported) would have preserved the originality of SOLMS's contribution and made his review more balanced.

Current neuropsychological evidence clearly indicates that dreaming can be disturbed or interrupted at different levels of cognitive processing. The important developments in neuroimaging techniques and future clinical and experimental research will certainly provide a deeper picture of the various cognitive components leading to the construction of complex oneiric experience and the flow of information in the dreaming brain. At present, no model gives a satisfactory account of the pattern of neural activation and deactivation during dreaming. Some authors (see **HOBSON ET AL.**) simply and cautiously summarize available evidence from many other authors in a list of different neural structures and functions contributing to specific features of the dreaming experience. **SOLMS** (1997a) views mentalistic-psychoanalytic concepts as "censorship" or "hallucinatory backward projection;" in our view, this has little heuristic value and is incompatible with modern neurocognitive and biological approaches to the study of mental processes. Considerably more evidence and reanalysis is needed before we can assign to dopaminergic mesolimbic structures a selective and exclusive role in the generation of dreams.

REM sleep: Desperately seeking isomorphism

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Abstract: If reports given on experimental awakenings validly represent mental activity that was underway before the awakening, REM sleep is neither necessary nor sufficient for dreaming. Another intuitively attractive hypothesis for its function – that REM consolidates or otherwise modifies memory traces acquired while awake – is not supported by the preponderant evidence. There is growing acceptance of the possibility that REM functions to support sleep rather than waking brain processes. **[HOBSON ET AL.; NIELSEN; SOLMS; VERTES & EASTMAN]**

REM sleep and dreaming: Rosetta stone or red herring. As good a case as possible for the REM-dream isomorphism is made by **HOBSON ET AL.,** but one that ultimately fails to convince. It is not possible to review all the contrary evidence but I will cite some significant examples. HOBSON ET AL. note that Hong et al. (1997) found an "impressive" correlation of .8 between visual imagery and REM density and consider this "evidence for a dependence of dream imagery on a qualitative feature of REM sleep" (p. 138). However, this correlation was found in a single subjects (S); further experiments failed to demonstrate this relation in two additional Ss (Antrobus et al. 1995). Moreover, the early hypothesis that the rapid eye movements of REM sleep (REMs) indicate scanning of dream images has not been supported by subsequent, more careful studies (Moskowitz & Berger 1969). REMs are so dramatic a feature of REM physiology that it seemed obvious they must be functionally important. However, my colleagues and I proposed they are adventitious phenomena with no special relation to dream imagery (Feinberg et al. 1987). We suggested that REMs are overt but incidental manifestations of the intense, disinhibited neuronal firing sleep in many motor (and sensory) systems throughout the brain that occurs during REM. Whereas neuronal firing in motor centers that control the limbs must be blocked to prevent movements that would awaken the sleeper, the eyes can move without causing awakening. Nature did not establish an inhibitory pathway from the atonia centers of the brainstem to the oculomotor nuclei simply because none was needed. The physiologically important question then becomes why some brain structures are intensely active in REM sleep. A potential clue is that the structures showing this response tend to be "hard-wired" (Feinberg & March 1995).

HOBSON ET AL. recognize that Antrobus and his colleagues interpreted the eye movement density-visual imagery correlation reported by Hong et al. (1997) not as evidence of brain-behavior isomorphism but as "another example of the simple dependence of dream content on levels of brain activation." I agree with Antrobus's view and think it important to emphasize further the strong albeit circumstantial evidence that REMs density is proportional to within-sleep arousal level (or "activation") (Feinberg et al. 1987). This evidence includes: the reduction of REMs by total sleep deprivation the progressive increase in REMs density across successive REMPs; the further spectacular increase in REMs density when sleep is abnormally extended, becoming extremely "light"; and the strong suppression of REMs density by GABAergic hypnotics, drugs that specifically depress brain arousal.

To further support an isomorphism between REM sleep and dreaming, **HOBSON ET AL.** point to a "positive relationship" between length of preceding REM sleep and word count, citing Stickgold et a1. (1994a). A positive relationship is not fully supported by the cited paper because word counts after 45–60 min of REM were about half as long as those in reports after 30–45 min of REM. Moreover, the results of a simple experiment in our laboratory, better controlled for time of night than that of Stickgold et al., challenge their findings (Rosenlicht et al. 1994). We awoke subjects (Ss) after 5 and 10 min from the second and fourth REMP of the night. Mentation was elicited with a standard protocol. The reports were tape-recorded, transcribed, and scored "blind" for word count by two raters. Word counts did not differ significantly in reports elicited after 5 versus 10 min of REM sleep (325 vs. 413; $p = 0.114$) but there was a highly significant difference between reports from the second fourth REMPs (264 vs. 474; $p. < 001$). We cannot fully explain the discrepancy between the results of the two studies. However, our experiment can easily be repeated and one hopes that it soon will be because its implications are substantial. If our findings are independently confirmed, they would demonstrate that the effects of REM sleep duration on word count are trivial compared to those of time of night. This point gains importance for HOBSON ET AL. because they now accept word count as a measure of "dreaming." So far as the underlying biology is concerned, we and many others have interpreted longer dream narratives later in the night as caused by higher within-sleep arousal ("activation") level. As discussed below, it is still not clear whether higher arousal level produces longer dreams, a wider span of recall for ongoing sleep mentation, or both.

Whether NREM and REM mentation differ qualitatively is the essence of the isomorphism issue. All of the experts in this *BBS* Special Issue on REM sleep and dreaming agree that dreamlike reports, qualitatively indistinguishable from those elicited from REM, can be obtained by awakenings from any stage of NREM sleep. Since the brain physiology of REM is massively different from that of NREM, this rules out a REM-dream isomorphism. At several points in their target article **HOBSON ET AL.** imply that failure to accept the REM-dream isomorphism is tantamount to rejecting the dependence of mental phenomena on the brain. This is hardly the case. One is rather rejecting the claim that a relationship exists between a particular psychological state (dreaming) and a particular physiological state (REM sleep). This rejection is not based on "mentalism" but on the strong contrary evidence. It is past time to accept the failure of this particular isomorphism and look elsewhere for the brain states that underlie dreaming. **SOLMS** does this in his interesting article.

Solms on the neural substrate of dreaming. The isomorphism issue is tackled head on by **SOLMS.** Noting that disagreement remains on the precise frequency NREM dreaming, he emphasizes the general acceptance of "the principle that *REM can occur in*

the absence of dreaming and dreaming in the absence of REM" (sect. 4). While even a rare instance of NREM dreams indistinguishable from those of REM would severely challenge isomorphism, NREM dreams are far from rare. SOLMS cites Hobson's (1988b) comment that "5–10% of NREM dream reports are indistinguishable by any criterion from those obtained from post-REM awakenings" (p. 143) and he points out that, since NREM sleep makes up 75% of total sleep time, "this implies that roughly one-quarter of all REM-like dreams occur outside of REM sleep" (SOLMS sect. 5, his emphasis).

SOLMS goes on to use clinicopathologic correlations to seek the neural substrate required for dreaming. His review finds that patients who have lost the ability to dream have suffered lesions in two forebrain areas: the "parieto-tempero-occipital junction" and the "ventro-mesial quadrant" of the frontal lobe. These observations are especially intriguing because several of these cases showed REM sleep when tested in the laboratory. However, without specific control for the memory impairment likely to accompany such brain lesions, one cannot know whether a patient has lost the ability to dream or the ability (and motivation) to recall and report dreams.

I strongly endorse **SOLMS**'s conclusion that the REM sleep-dreaming relation is in need of so fundamental a revision as to constitute a paradigm shift. This shift is at least 10 years overdue. The REM-dream relation has not been a Rosetta stone but rather a red herring that has led us seriously astray. The failure of REM-dream isomorphism contains an ironic element. The irony lies in the fact that this failure, taken with our knowledge of the brain physiology of REM sleep, tells us a great deal about what the neuronal substrate for dream consciousness is *not*. SOLMS explicitly recognizes this. J. D. March and I arrived at a logically similar conclusion (Feinberg & March 1995). We reasoned that, since brain physiology is qualitatively different in NREM and REM, but the conscious experience of dreaming in the two states is not qualitatively different, "the striking NREM/REM differences in neuronal firing must not involve the neural systems that can affect the quality of conscious experience" (p. 106). Because it is almost certain that marked alterations of the firing patterns in these structures would affect waking consciousness, this conclusion implies that sleep involves disconnections within the brain, as well as a relative disconnection from the environment.

Sleep and memory. Before commenting on **VERTES & EASTMAN,** whose paper deals mainly with this issue, I think it important to emphasize a fact that is too often overlooked: Virtually all modern sleep-dream research is based on the unproved assumption that narratives given by Ss when awakened from sleep represent mental activity that was going on prior to the awakening. Certainly this assumption is consistent with our subjective experiences of dreaming. Nevertheless, there are no data that rigorously exclude the possibility that dream reports are entirely constructed during the process of waking-up. A century ago Goblot (cited by Hall 1981) pointed to this possibility. Some of us who have been present (more recently) as Ss struggled to report their sleep mentation had the impression that a considerable process of reconstruction (construction?) was underway. On some of the infrequent occasions when Ss produced complex and elaborate dream narratives, I thought that the stories were being created *de novo*, while the S was in a fugue state intermediate between sleep and waking. A related point is that any quantitative or qualitative differences in the mentation elicited from the different stages of sleep might be caused by differences in the functional state of memory systems rather than in the mental activity produced during these stages (Feinberg & Evarts 1969).

VERTES & EASTMAN review the experimental literature on whether REM sleep promotes memory consolidation and show it to be unpersuasive. Many of these studies performed REM deprivation with the "flower pot" method that VERTES & EASTMAN rightly emphasize is contaminated by stress. Even in the presence of this stress, there are as many failures to show an impairment of learning and memory by REM deprivation as there are positive studies. Carlyle Smith's evidence that memory "windows" exist during which REM sleep acts to consolidate memories has not been independently confirmed; moreover, the variability in the timing of these windows that Vertes and Eastman extensively document is disconcerting. Using another line of evidence, VERTES & EASTMAN cite data showing that monoamine oxidase inhibitors can virtually eliminate REM sleep without detriment to waking behavior. I agree with this point and, in fact, used it to support my arguments that REM serves a brain function intrinsic to sleep rather than (as does NREM) to waking (Feinberg 1974). Benington and Heller (1994) now also endorse a similar view.

In discussing the stress induced in rats by REM deprivation with the flower pot method, **VERTES & EASTMAN** note in passing that this criticism may not apply to deprivation with Rechtschaffen's yoked control-platform paradigm. This point is not essential to any of their main arguments. Nevertheless, because of the great theoretical importance currently placed on the physiological changes provided by the Rechtschaffen deprivation paradigm, it may be useful to draw the reader's attention to the fact that some investigators believe that these changes are due to stress rather than sleep loss. Thus, I noted (Feinberg 1999) that the pathophysiological changes produced in rats by prolonged total or selective sleep deprivation with the Rechtschaffen technique resemble the non-specific stress responses in Selye's General Adaptation Syndrome (Selye 1937). Rechtschaffen has strongly contested these arguments (Rechtschaffen & Bergmann 1999) and this issue remains controversial.

I would have emphasized more strongly than **VERTES & EASTMAN** the functional implications of the shut-down of memory consolidation systems during sleep. The degree of shut-down is roughly proportional to the level of high amplitude delta EEG, that is, it is maximal in stage 4 and least in stage REM. I have already emphasized that variations in arousal level might explain much of the variance in sleep mentation through its effects on memory function, a view previously proposed by several investigators (Antrobus 1991; Koulack & Goodenough 1976; Zimmerman 1970). Variations in memory function could also explain why Ss can produce non-random estimates of REM but not stage 4 durations (Carlson et al. 1978). If, as many of us assume, one function of sleep is to reverse certain effects of plastic neuronal activity during waking, it seems likely that memory systems would be involved. It makes intuitive sense that the systems being restored would be taken "off-line." The fact that memory consolidation systems are substantially disabled during sleep is therefore consistent with the possibility that one function of sleep is to permit recovery of these systems.

It is in their proposals for the function of REM sleep that I found **VERTES & EASTMAN** disappointing. Hypotheses similar to theirs have been advanced in the past and VERTES & EASTMAN offer no new evidence. As already mentioned, the hypothesis that REM serves a function intrinsic to sleep rather than to waking was advanced a quarter century ago (Feinberg 1974). A proposal similar to VERTES & EASTMAN suggestion that "the primary function of REM sleep is to provide periodic endogenous stimulation to maintain minimum requisite levels of CNS activity throughout sleep" was put forward by Ephron and Carrington (1966). Although VERTES & EASTMAN decry theories that propose "magical" processes for REM sleep, their own proposals seem vulnerable to similar criticism. Notions like "minimum requisite levels of CNS activity" or a brain "incapable of tolerating long continuous periods of relative suppression" could be viewed as vague and metaphorical.

High levels of REM sleep in the neonate. Both **HOBSON ET AL.** and **VERTES & EASTMAN** cite the high neonatal levels of REM sleep to support their differing interpretations of REM's functional significance. But there are reasons to believe that the physiology of neonatal sleep differs fundamentally from that a few months later (Feinberg 1969). Brain wave patterns in the neonate are so rudimentary that one cannot distinguish the NREM from the REM EEG, making it necessary to distinguish sleep states

behaviorally as "quiet" or "active" sleep (cf. Kahn et al. 1996)). Anatomical data can added to earlier arguments against accepting REM in the neonate as physiologically equivalent to that occurring later in infancy. Conel's atlases (Conel 1939) show cortical connectivity in the newborn human brain to be vastly limited compared to that present just a few months later, at which time REM levels are not greatly different from those in the adult. Frank et al. recently reported that "REM" sleep in the newborn rat differs pharmacologically from that in the more mature animal (Frank et al. 1997) and argued that this indicates a different physiological state. For these reasons, it seems hazardous to accept active sleep in the newborn as homologous with the REM of later infancy and adult life and to infer either functional or psychological significance from its high levels.

Nielsen and the hypothesis of covert REM. A masterful and objective review of the experimental literature on dreaming is provided by **NIELSEN** that should be useful for years to come. He accepts the "strong proof that cognitive activity – some of it dreaming – can occur in all sleep stages," and that the physiology of NREM and REM are qualitatively different. Nevertheless, he attempts to preserve an isomorphic REM-dream relation. To do so, he hypothesizes that "sleep mentation is tightly linked to REM sleep processes" and that these processes may dissociate from the REM state and "stimulate mentation in NREM sleep in a covert fashion."

Although I interpret differently much of the evidence **NIELSEN** cites in support of his hypothesis, I strongly agree that one encounters intermediate sleep states that have both NREM and REM features. Such states provoke considerable gnashing of teeth among the unfortunates whose task is to score sleep stages. However, it is a considerable leap from the sporadic occurrence of intermediate states to the notion that these states are invariably but covertly present whenever dreamlike narratives are elicited from NREM awakenings. This hypothesis seems particularly implausible in the case of stage 4 awakenings that give rise to vivid dreams since stage 4 physiology is the polar opposite of that in stage REM. But unless intermediate states with REM characteristics are *always* present when dreamlike narratives are elicited from sleep, covert REM cannot rescue the REM-dream isomorphism.

NIELSEN suggests that his hypothesis can be tested by simple and straightforward experiments. I do not agree that the tests he proposes would give unambiguous answers. NIELSEN proposes that dreamlike mentation reports will occur more frequently when elicited from NREM episodes in close proximity to REMPs, especially those that are lengthy. However, this result need not indicate the presence of covert REM. The one-stimulus model (see below) could parsimoniously interpret such findings as indicating that within-sleep arousal levels are higher at these points. Twenty years of research have shown that NREM sleep is not constant across a sleep cycle but shows the waxing and waning of delta intensity (cf. Fig. 1). Differences in the mentation elicited at the beginning and end of the cycle could reflect differences in the physiology of NREM at these points, independent of proximity to REM. A similar interpretation applies to NIELSEN's prediction that NREM mentation will be increased by sensory stimulation during sleep; such stimulation, already known to increase REM sleep (Drucker-Colin et al. 1983), could alter sleep mentation by raising arousal level. Experiments of the sort NIELSEN proposes would nevertheless be interesting. They could be strengthened if awakenings were performed in relation to points in the computer-quantified delta cycles rather than visually scored sleep stages. For example, it would be interesting if sleep mentation on the ascending limb of these curves differed from that elicited on the corresponding point of the descending limb (which would receive the same sleep stage scores and have similar proximity to REM).

In his discussion of "missing" REM episodes, **NIELSEN** seems unaware of some relevant literature. As NIELSEN notes, the "skipped" first NREMP leads to exceptionally long first NREMPs. This phenomenon is best understood on the level of basic sleep physiology. It has long been known that if one plots total EEG amplitude or spectral power or delta integrated amplitude across sleep, one observes an irregular series of peaks and troughs (Church et al. 1975; Koga 1965; Lubin et al. 1973). The peaks correspond to visually scored stage 3–4, and the troughs are usually scored as stage REM, with stage 2 occupying the intermediate parts of the curve. However, in extremely deep sleep (e.g., in young normal children or young adults after total sleep deprivation – TSD), REM is frequently not scored in the first trough (Fig. 1). In these cases, application of curve smoothing methods

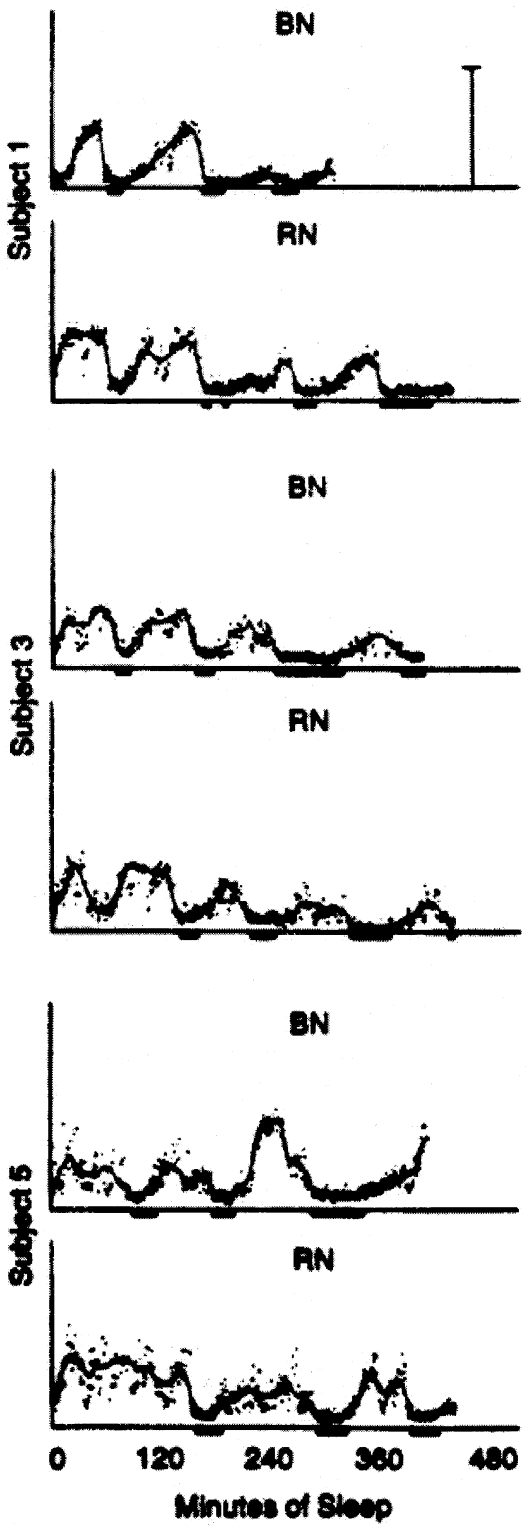

Figure 1 (Feinberg).

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that objectively define successive peaks and troughs reveals that the duration of the first peak (NREMP 1) is not abnormally protracted in either the deep sleep of children (Feinberg et al. 1990) or in young adults after TSD (Feinberg & March 1988).

It will simplify the discussion if we now introduce the one-stimulus model of NREM and REM sleep that March and I proposed in 1988 and expanded in 1995. Briefly, this model holds that NREM and REM sleep occur at different points in the brain's response to a single inhibitory neuroendocrine pulse that occurs recurrently during sleep. This pulse is presumed to originate in the hypothalamus. This stimulus inhibits neuronal activity, reduces cerebral metabolic rate, induces EEG synchrony and depresses arousal level, that is, induces NREM sleep. Functional changes that occur in the inhibitory (NREM) state include relative sensory deafferentation and a shutting down of memory consolidation systems (see above). The intensity of the brain response to the stimulus parallels the waxing and waning of the EEG amplitude curves described above. When the strength of the inhibitory pulse falls below a critical threshold, escape from inhibition occurs. This neuronal escape *is* REM sleep which, as noted above, is characterized by intense, disinhibited firing in many neuronal systems. After a variable duration of REM, another pulse is released and the process repeats. The failures of REM to appear in the first trough of deeply sleeping Ss indicate that the critical arousal threshold for inhibitory escape has not quite been reached.

March and I have shown how the one-stimulus model, along with the homeostatic model of delta (Feinberg 1974) parsimoniously accounts for much of the known phenomenology of human sleep, including sleep architecture patterns and the effects of daytime naps on post-nap sleep (Feinberg et al. 1985; 1992). The model also explains the increased REM produced by (partially arousing) sensory stimulation during sleep, and Datta and Siwek's (1997) findings that low intensity stimulation of brainstem arousal centers converts NREM to REM and more intense stimulation converts REM to waking. Our model would also interpret the increased neuronal firing in cholinergic brainstem centers during REM as components of widespread disinhibition-release phenomena, rather than as specific stimuli for either the REM state or the cognitive events of dreaming.

The case against memory consolidation in REM sleep: Balderdash!

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Abstract: Unfortunately, some researchers think a good scientific theory is one that has been repeatedly confirmed, and a bad theory is one that has not received consistent confirmation. However, confirmation of a theory depends on the extent to which a hypothesis exposes itself to disconfirmation. One confirmation of a highly specific, falsifiable experiment can have a far greater impact than the disconfirmation of twenty experiments that are virtually unfalsifiable. This commentary (1) counteracts misleading biases regarding the REM sleep/memory consolidation theory, and (2) demonstrates how chaotic cerebral activation during sleep is an essential component of long-term memory storage processes.
[VERTES & EASTMAN]

Most theories of the function of sleep – and REM sleep in particular – revolve around the idea that sleep serves an adaptational function for wakefulness. Four of the five authors of the target articles presented in this special journal issue – all except **VERTES & EASTMAN** – are of this view (as it is self-evident to most others). VERTES & EASTMAN's idea is that the functional importance of REM sleep is solely neurobiological. They believe REM sleep is bound to the biological state of sleep itself, to the homeostatically upregulated depressed quietude of SWS (slow-wave sleep), in order to maintain obligatory levels of CNS activity throughout sleep. The brain, they argue, is incapable of sustaining long periods of suppression produced by the delta activity of SWS and as such, requires endogenous stimulation to maintain neuronal homeostasis. However, as it has been for so many other theories ranging from that of Freud in 1900 to the present ones of the post-REM era, VERTES & EASTMAN's theory is unfalsifiable. This is largely the reason the function of sleep (and dreaming) remains unknown. In any case, VERTES & EASTMAN's theory is not under examination in this review; it is the REM-sleep/memory consolidation theory that is. This theory has received considerable attention because there are potential methods for evaluating the evidence relevant to the theory. That is, the theory includes the possibility data can be generated that will falsify it. Therefore some researchers have challenged its basic tenets.

1. Two important caveats. (1) The principle of falsifiability (Popper 1959) has important implications for the way the theory of REM sleep and memory consolidation is evaluated. Many researchers think that a good scientific theory is one that has been repeatedly confirmed, and a bad theory is one that has not received consistent confirmation. They assume that the amount of confirming evidence is the critical factor. However, falsifiability implies that the number of times a theory has been confirmed (or not confirmed) is not the critical element; not all confirmations are equal. Confirmations are more or less important depending on the extent to which a hypothesis exposes itself to potential disconfirmation. One confirmation of a highly specific, potentially falsifiable experiment can have a far greater impact than the disconfirmation of twenty different experiments that are all virtually unfalsifiable. Therefore, it is necessary to look not only at the quantity of the confirming evidence, but also its quality. (2) Even with an earnest attempt at evaluation of the literature, the writer of a review organizes the material and emphasizes certain parts of it so as to persuade the reader to his view. For researchers who do not have a primary interest in the area of the review, and who do not peruse individual experiments reported, the reputation of the reviewer and his steadfast argument might convince the "outsider" the review represents the final word.

The purpose of the present commentary is to counteract the persuasive effects of honest, but nevertheless – what this writer believes – are misleading biases. Unfortunately, space limitations preclude a full critique, and therefore this commentary is limited to a few selected items of **VERTES & EASTMAN**'s target article.

2. REM augmentation studies. One of the most consistent findings in the animal sleep literature (as overwhelmingly consistent as the evidence for cholinergic REM sleep generation) is the augmentation of REM sleep following heightened experiences. Yet, while human studies have not shown the same degree of consistency, the reader is led to think REM sleep augmentation is a bogus finding. Unfortunately, **VERTES & EASTMAN** provide no discussion of the differences between human and animal sleep cycle rhythmicity, nor is there any attempt to reconcile the differences in an effort to point out why REM augmentation may not be easily seen in human sleep. Given the imperfections of the disconfirming experiments in contrast to the consistency of the augmentation phenomenon, it is necessary to evaluate the extent and nature of the flaws in the disconfirming experiments. More emphatically, there is no evidence presented falsifying the confirming evidence that might lead to the theory being modified, or perhaps abandoned for an entirely new theory.

More dismaying is misinterpretation of research report findings. In the effort to build a case against the REM-sleep memory consolidation theory, **VERTES & EASTMAN** make reference to review articles and conclusions drawn by others, in particular the review paper of Horne & McGrath (1984). That paper refers to two publications from my laboratory (Gutwein & Fishbein 1980a; 1980b) in which we examined the effects of enriched and impoverished rearing on REM sleep. The original reports describe findings that are the opposite of what VERTES & EASTMAN state. VERTES & EASTMAN conclude that (1) REM sleep augmentation is an "artifact" of an overall increase in total sleep time (TST), and

(2) the differences between the experimental and control animals comes from a decrease in REM as a result of the controls being reared in impoverished conditions. A reader of the review is led to conclude that the research results were owing to a confounding variable, and therefore should be discarded. In fact, the reported findings in both research reports unambiguously show that REM sleep is significantly augmented as a result of enriched rearing compared to social control (not impoverished) animals and furthermore, the selective REM sleep augmentation in the enriched animals compared to the social control animals is not an artifact of SWS augmentation. Both papers, in considerable detail, report the statistical analyses with significance probability levels of $p < .001$ or greater.

3. REM deprivation studies. **VERTES & EASTMAN** then turn to the REM deprivation research that was so ubiquitously reported during the 1970s and 1980s and again the target article presents misrepresentations and omissions of significant experimental findings. This section begins with a quotation from my 1995 review paper (Fishbein 1995). I abandoned the REM deprivation work because I could not, it says: "adequately respond to criticisms leveled at the (REM deprivation) technique." This is gross distortion. To set the record straight, at the time the REM deprivation experiments were performed, I had reached the point whereby I had come to believe all the crucial experiments to demonstrate the relation between REM sleep and memory consolidation processes using the pedestal technique had been exhausted. To further pursue that line of research would produce experimental results that could only be seen as variations on a theme that, for the most part, had already been established. I believed it was time to move on and employ a new strategy.

3.1. The pedestal technique. The REM deprivation experiments from my laboratory were all designed with extraordinary care, primarily to handle the possible interpretation that the results would be seen as confounded by the "stress" of the pedestal technique. The view had emerged, at the time, that the pedestal technique produces results indistinguishable from experiments employing stress to produce impairments of learning or memory; interestingly, the REM deprivation/memory experiments reporting the effects of stress had all used rats (for a review, see Fishbein & Gutwein 1977). Our experiments, on the other hand, employed mice.

Certainly we knew that confirming evidence, based on reports from many laboratories examining the physiological effects of stress, and particularly those laboratories showing the pedestal technique not to be stressful, would not pacify skeptics wedded to the belief the pedestal technique is stressful. We also did not believe our descriptions of mice freely climbing about the underside of water filled cages, would pacify the skeptics either. Neither did we believe our open field activity experiments, showing no differences between REM deprived and normal animals, would placate the skeptics. However, we did think that if we could perform an experiment that would refute (falsify) the hypothesis that the pedestal technique is stressful by replicating the rat studies (i.e., restricting the activity of the mice while living on a pedestal in the midst of a pool of water), showing that restriction impairs (stresses) the mice, while nonrestriction (non stressed) does not, we would have direct confirmation of our hypothesis that the pedestal technique was not stressful (so long as the mice could climb about – and exercise – in the pedestal cages).

We performed that experiment; it is described in our 1977 paper (Fishbein & Gutwein 1977). The results indicated that the unrestricted mice (living on the pedestals for either 2 or 4 days) had identical activity scores (open field and passive avoidance step-through latencies) compared to controls (animals maintained in standard shoebox cages with wood shavings); whereas restricted animals (also on the pedestals for either 2 or 4 days) were considerably more active than controls and the unrestricted mice. With this experiment we clearly demonstrated why the rat studies had to be discounted as a result of the deprivation procedure, whereas the robustness of the mice demonstrated just the opposite. In short, stress was not a factor we needed to concern ourselves with in our REM deprivation studies. The journal referees of our many manuscripts were convinced we had satisfactorily handled the stress criticism; they would not have allowed our reports to go to press if there was such a gross confound in our research. Yet despite all our efforts, there was always a small number of researchers (perhaps biased by their own theories of the function of REM sleep) who refused to be swayed by the data.

3.2. Prior REMD studies. In the fourth line of their brief discussion of prior REM deprivation studies, **VERTES & EASTMAN** dismiss this research out of hand. "These studies," they say, "do not seem to test the REM consolidation hypothesis since the deprivation period precedes training/acquisition and there is no potential carry over of information pre to post REMD." Period. That's it; and with that said, they select one study (van Hulzen & Coenen 1982) to drive home their point; prior REM deprivation produces only acquisition impairments. Of course, the stress factor is then resurrected and the whole matter is discharged without further ado.

However, this area of research is important and several experiments designed to examine the effects of prior REM deprivation on the conversion phase of memory consolidation have demonstrated that acquisition is unaffected by the prior deprivation. In fact, it is imperative to demonstrate in such experiments that subjects learn and remember normally for at least a brief period before amnesia sets in, otherwise – **VERTES & EASTMAN** would be correct – the experiments would be difficult to interpret because there would be no way to distinguish acquisition from retention impairments. The prior REM deprivation studies are centrally concerned with the role of REM sleep in the formation of newly acquired information.

In two experiments from my laboratory (cited in **VERTES & EASTMAN**'s review, Fishbein 1970; Linden et al. 1975), we unequivocally demonstrate that mice deprived of REM sleep for 3 days prior to learning show perfectly normal retention up to 1 hour after learning compared to non-deprived controls. However, 3, 5, and 7 days after the animals had demonstrated normal retention, they are amnesiac. That is, a memory that had been established was now gone.

In the follow-up to this experiment we reasoned and predicted that if the prior REM deprivation impairs the permanent fixation of a long-term memory trace, the treatment induced its effect by altering the consolidation gradient of the memory fixation process. In this experiment the same experimental design was employed as in the previous one. Animals were deprived of REM sleep for three days and then trained. Electroconvulsive shock (ECS) was then administered at intervals varying from immediately after learning up to 6 hours afterwards. The animals were then tested for retention three days later, long after recovery from the deprivation and ECS. In this experiment we showed that 3 days after training the memory trace remains susceptible to disruption and furthermore a gradient of susceptibility was apparent as the animals recovered from the REM deprivation. The experimental results confirmed our hypothesis. Moreover, the experiment is without any stress confound. All animals were deprived for the same length of time; all animals received training at the same time after the REM deprivation. The only variable manipulated was the time between training and the administration of ECS. The experiment provided indisputable evidence that REM deprivation had sustained the memory trace in a labile form, thereby prolonging permanent consolidation of the memory trace. In short, REM sleep has an important role to play in the fixation phase of the memory consolidation process.

3.3. Post learning REMD studies. **VERTES & EASTMAN** similarly dispose of the REM deprivation studies in which the deprivation is inserted between learning and retention testing; in their view, these are all "performance" deficits. Considering the enormous number of publications that have examined the REM sleep-memory consolidation hypothesis from this perspective, it is hard to believe that so many experiments can be so easily dismissed. There are many confound-free experiments to choose from that

underscore the important role of REM sleep in the mechanisms underlying the storage of long-term memory. One experiment, in particular, from my laboratory can serve as a model example. The experiment was one of our earliest (Fishbein et al. 1971) and is totally free of the "stress" factor. Mice were trained and immediately deprived of REM sleep by the pedestal method for 2 days and then separate groups were administered ECS varying from 5 minutes to 12 hours afterwards. Two days later, after recovery from deprivation and ECS the animals were tested for retention. As in the prior REM deprivation studies, ECS produced retrograde amnesia, with the amnesia gradient occurring two days after training. Important to note, the animals were not under any stress at the time of learning or retention testing, yet REM deprivation induced a brain change that led to a profound amnesia.

Moreover, contrary to **VERTES & EASTMAN**'s suggestion in all the experiments performed in my laboratory, the treatment effects were extremely large. Therefore, these experiments and many more that have followed, have provided exceptionally strong evidence that REM sleep has a major role to play in the processing and consolidation of long-term memories. The task of researchers is not to dismiss these powerful findings because other experiments have not systematically supported them, but to search for a pattern of flaws running through the research literature because the nature of the pattern may then provide reason to modify or possibly abandon the theory. However, that a confounding "stress" factor obscures the interpretation of an experimental result does not mean the theory need be abandoned.

Space constraints preclude further discussion despite other objections to be raised, yet it bears repeating that failure to reject Hebb (the null hypothesis) is not in itself evidence that the theory of REM sleep-memory consolidation should be abandoned. Nevertheless there is one abiding issue central to the REM sleep-memory consolidation hypothesis raised by **VERTES & EASTMAN** that must be addressed. The topic revolves around the role of theta activity involvement in memory processing.

4. Making dreams out of chaos. Central to **VERTES & EASTMAN**'s objections to the REM-sleep-memory consolidation hypothesis is their view that theta waves – which, indisputably, involve memory processing during waking – merely reflect a highly activated brainstem during REM sleep, producing random (chaotic) activation of the cerebral cortex and limbic system (rather than reflecting memory processing activities).

Vertes and I have previously exchanged commentaries about these very points (Fishbein 1996; Vertes 1996). I suggested that the chaos may be the underlying basis of the dream itself. Information comes into the hippocampal system from the cerebral cortex, including the visual, auditory, sensory cortices and the motor cortex. It also receives information from the amygdala concerning odors, unsafe stimuli, and information about the person's emotional state: whether sexually excited, hungry, frightened, and so forth. Recent accumulated evidence suggests that the function of the hippocampus may be to tie together or relate all the things happening at the time the memory is stored. Many experiments suggest that the role of the hippocampus is to construct representational relationships between these various forms of experience, including the order in which events take place. The representations can be likened to a library card-catalogue filing system with essential information stored in neural networks distant from the hippocampus. The filing system performs the necessary work of filing and/or retrieving information. Disruption of the system will of course impair storage (as in Alzheimer's disease) or retrieval processes (as in schizophrenic hallucinations).

4.1. The relation of random excitability to memory. **VERTES & EASTMAN** question whether random, unrelated events can have any functional value in the long-term memory encoding process occurring during REM sleep. They believe that since there is no mechanism in REM to select and orderly transfer the ponto-geniculo-occipital (PGO) spike information to the hippocampus from the brainstem, or for that matter from various cortical regions, the information that does flow will be inherently random and therefore "there would be no functional value in consolidating or 'remembering' this information."

However, chaos may be just the elixir needed to facilitate information storage. Information that is systematic, orderly, and time-locked to the behavioral experience, as **VERTES & EASTMAN** would have it, may not lead to strengthening of memory traces, but to its decline. D.O. Hebb (1949) was the first to point this out in his famous text, *Organization of behavior.* For example, there is no surprise about the fact that hearing a joke for the second time makes for poor entertainment – the better the details are remembered of the first telling, the less interest there is in the second. Similarly, in a prolonged training schedule, there are often periods when practice seems to have a negative effect. The more you try, the worse things get. Such mundane events – yet truisms – might suggest that connections involved in learning might actually be weakened by orderly repetition of the same sequence of events. This behavior is referred to as "habituation." And sometimes it is necessary to have a period of rest before functioning well can occur again ("spontaneous recovery"). In a similar vein, we know that distributed practice is superior to massed practice in learning a skill. Thus, the deteriorative effect of repetition (say, losing interest in repeating the same solution to a problem) leads to habituation.

Hebb's illustrations infer that a memory continually needs to be updated to maintain its organization and persistence. In the same way, perhaps a neuronal memory trace needs to be rearoused to be sustained, but the maintenance of the trace requires new combinations of excitation (Nick & Ribera 2000), which in turn might mean new cognitions or new ideas.

To quote Hebb, the "mere occurrence of a particular 'phase sequence' once, induces changes at the synapse (memory) that make it impossible for exactly the same sequence to occur again, unless the synaptic changes have disappeared with time" (1949, p. 228), or possibly the information needs to be introduced in a different form. In short, the chaos occurring during the REM sleep period that **VERTES & EASTMAN** believes serves no functional value in consolidating information may be the kind of excitation that neural networks require to consolidate and sustain information for prolonged periods.

5. The new findings of Braun and Solms. Finally, **VERTES & EASTMAN**'s objections to the REM sleep-memory consolidation hypothesis must be abandoned in light of the recent brain imaging studies of Braun and colleagues (1997; 1998) and the new lesion-dreaming research of **SOLMS,** that presages renunciation of the brainstem activation-synthesis model of dreaming (and **HOBSON ET AL.**'s AIM update of the model), replacing it with a shared model in which forebrain cerebral activation during sleep and dreaming is either self-activated within the forebrain itself, or activated by either the random orchestration of the ascending brainstem cholinergic system (originating in the pons) primarily during REM sleep, or a non-REM activating system ascending through dopaminergic circuits originating in the midbrain ventral segmental area of Tsai, the origin of the mesolimbic and mesocortical dopamine systems. Therefore it is no longer necessary to look to the pontine brainstem as the sole source of endogenous cerebral activation. In short, (chaotic) activation of forebrain structures throughout the sleep cycle has the potential to reactivate neuronal circuits facilitating the consolidation of memory.

Dreaming is not an adaptation

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Abstract: The five papers in this issue all deal with the proper evolutionary function of sleep and dreams, these being different. To establish that some trait of character is an adaptation in the strict biological sense re-

quires a story about the fitness enhancing function it served when it evolved and possibly a story of how the maintenance of this function is fitness enhancing now. My aim is to evaluate the proposals put forward in these papers. My conclusion is that although sleep is almost certainly an adaptation, dreaming is not.

[HOBSON ET AL.; NIELSEN; REVONSUO; SOLMS; VERTES & EASTMAN]

Evolution and the dissolution of the hard problem. Despite being orthodox naturalists and recommending whole-heartedly the view that mental processes are brain processes, the **HOBSON ET AL.** "dream team" (Flanagan 2000) buys into a bad philosophical idea. This is the idea that there is a "hard problem" of consciousness (Chalmers 1995a), one that they claim they do not treat or resolve with the AIM model, and one that they, like many others, seem to think is beyond current cognitive neuroscience, possibly beyond science, period. I want to convince the dream team that they do treat the hard problem.

What Chalmers (1995a) calls the "hard problem" of consciousness is the problem of explaining how subjectivity can arise from complexly organized material stuff. The hard problem truly exists in one sense, for we have at present nothing remotely approaching a complete theory of how the brain does everything it does, including how it produces consciousness. But Chalmers, as well other "new mysterians" (Flanagan 1991; 1992) like Colin McGinn (1989), have pressed the line that even if we are provided with a complete neurobiological theory of how the brain works, nothing will have been done to erase the intuition that there is an unbridgeable gap between the way the organized objective brain works and the first-person grasp I have of myself as a thinking-feeling creature. Knowing all the facts about how the brain works will fail to explain how the brain gives rise to subjective mental life.

My recommendation for **HOBSON ET AL.** is not to fall into the trap fostered by such intuitions as "It is amazing that my consciousness could emerge from brain processes" and "Thoughts don't feel as if they have neural texture." One falls for the trap when one allows the "gee-whiz" bug to get a grip and thinks that there is any harder, deeper, or further problem than explaining how the mind-brain works for each of the heterogeneous kinds of conscious mental state types. Explaining the mechanisms that give rise to the different types of waking consciousness, NREM, and REM-mentation, is all there is to solving the hard problem. There is no further hard problem that will remain once this labor is completed. It may still seem amazing that by explaining how the brain works we will have explained how the mind works. But so what? It may seem amazing, even incredible, that my solid maple dining table is actually a field in Hilbert space comprised mostly of empty space. But that is what it really is. How things seem, including the powerful intuition that there seems to be an unbridgeable gap between conscious experience and brain processes, has no evidentiary status whatsoever when it comes to how things are.

In fact, we have an evolutionary explanation for why the mind-brain relation seems so mysterious. Philosophers call such explanations "error theories," because they are designed to explain why otherwise intelligent people make grand reasoning errors, such as believing that there is evidence for God or that there are objective moral facts. In the case under consideration, Mother Nature designed us to be in touch first-personally with our brain states at a level of granularity that reveals nothing about their neural texture. But it is an inference to the best explanation that mental events just are brain events. The fact that they don't seem that way is irrelevant. Indeed, it was a wise evolutionary strategy to design us not to have first-personal touch with the deep structure of our mental states and processes, a case where more information would have been too much information. Awake consciousness in the five sensory modalities is an adaptation precisely because it allows us to detect reliably what is going on outside us and to use this information in fitness enhancing ways. There was nothing to gain and everything to lose had Mother Nature designed us to be in touch with our mental states at the level of granularity that neuroscience treats. So the alleged hard problem dissolves when we understand it in evolutionary terms.

What happens to dream consciousness when we think about it in evolutionary terms? **HOBSON ET AL.** once thought that dream consciousness functioned for the sake of memory fixation and consolidation. This hypothesis was motivated by the discovery that acetylcholine is implicated in fixing memories and in the discovery that acetylcholine levels are high during REM sleep. The more recent view that dreaming is probably an evolutionary epiphenomena is motivated by a clearer appreciation of a point that **SOLMS** (1997a) and I (Flanagan 2000) have pressed independently, that sleeping and dreaming are different phenomena, a claim also argued for (or implicit in) **NIELSEN**'s, **VERTES & EASTMAN**'s papers. High levels of acetylcholine may well support the idea that one function of REM-sleep is memory fixation and consolidation, but since we rarely dream about what we need to remember, the hypothesis that dreams themselves serve any memory enhancing function appears unwarranted. Furthermore, **VERTES & EASTMAN** present reason to worry about the memory consolidation hypothesis, favoring instead the view that REM sleep serves to maintain a level of CNS activity during sleep that assists the brain in recovering from sleep. I quite agree that the data on people with fine memories who do not REM is a problem. But one doesn't need to abandon the view (yet) that one normal function accomplished by REM is memory consolidation – perhaps the brain being plastic has other ways to accomplish the task if REM is interfered with, and it is entirely possible, indeed likely that REM sleep, like NREM sleep, serves multiple functions, perhaps including keeping CNS activity at a certain level.

In any case, it seems best, pending resolution of this debate, to think of awake consciousness as an adaptation and sleep as an adaptation (for a reason or reasons in need of further exploration), and to think of dreaming as a nonadaptive side-effect of what sleep produces in a brain designed to be conscious by the light of day. In saying this, however, the proposals of **REVONSUO, SOLMS**, and to some degree, **NIELSEN,** need to be addressed, because they explicitly or implicitly allow a proper evolutionary function for dreaming itself.

A parsimonious account of threat simulation dreams. It is true that in my argument for the thesis that "dreams are the spandrels of sleep" (Flanagan 1995; 1997; 2000), I did not consider any thing like **REVONSUO**'s idea that dreams were selected for the purpose of simulating threatening situations and rehearsing appropriate responses. Three candidates for assigning an adaptive function to dreams that I did consider were Freud's (1900), **HOBSON ET AL.**'s (1988) and Crick and Michison's (1983; 1995). The ideas are, respectively, that dreaming was selected to express socially unacceptable wishes and thereby to preserve sleep, that dreaming functions to consolidate memories, and that dreaming functions to dispose of things not worth remembering.

In each case, my argument against the adaptationist proposal turned on the lack of support for the relevant hypothesis, given the actual content of what we dream about. Most dreams don't express wishes, most dreams don't involve entertaining things worth remembering, nor do they involve entertaining things worth forgetting. Freud aside, I do think that Hobson (1988a) and Crick and Michison (1983; 1995) are probably right that memory consolidation and brain-washing, SAVING and TRASHING, as it were, are one of the things that sleeping, especially REM-sleep, was selected to do (pace **VERTES & EASTMAN**). It is just that the phenomenology of dreams gives no support to the idea that dreaming contributes to this process. Despite their not having an evolutionary proper function, I claim that some dreams express things that our minds activate: emotions, worries, concerns, and memories that we have, and are in this way self-expressive, possibly and in some cases, even worth the effort of interpretation as sources of self-knowledge.

REVONSUO's proposal is that if we think carefully about the original evolutionary situation and at the same time examine the content of dreams, we will see that a plausible case exists for assigning dreams an adaptive evolutionary function. Dreaming was selected to simulate and rehearse threatening situations of the sort

that we probably faced when we evolved tens of thousands of years ago. The argument turns on much interesting data: more dreams are unpleasant than pleasant, nightmares are common, fierce animals and threatening male strangers turn up much more frequently in dreams than they do in current environments, and much more frequently than the run-of-the-mill things we spend our time actually doing or worrying about doing.

These data then form the basis of **REVONSUO**'s proposal that dreaming is not a spandrel after all but an adaptation. Here are some grounds for skepticism. First, we know thanks to work by Darwin (1873/1965) and Paul Ekman (1992) that humans did evolve with certain basic emotions. Ekman's list of basic and universal emotions now extends to seven emotions: fear, anger, sadness, disgust, contempt, surprise, and happiness. It seems utterly plausible to think that these emotions and the affect programs that govern them are adaptations, specifically adaptations that served awake humans who were up and about struggling to survive in harsh and threatening circumstances. It should be said, however, that it is entirely possible that the basic emotions and the affect programs they abide did not evolve among Homo sapiens but rather were handed over to us from earlier hominid ancestors.

This point is relevant because **REVONSUO** has us imagine a selection among Homo sapiens favoring those with heritable skills of simulating and rehearsing threatening events in dreams from those lacking the trait. Once the basic emotions are recognized as adaptations, a more parsimonious explanation for the threat simulation and rehearsal in dreams than the one REVONSUO offers is available. It is well known that the mechanisms activating sleep differentially activate the emotional centers of the brain. One widely accepted explanation is that this has to do with the proximity of the emotional centers to the brainstem from where the ins and outs of sleep are largely, but probably not exclusively, orchestrated. It would not be surprising, therefore, if the basic emotions and the associated affect programs were not thereby differentially activated. Since most of the basic emotions are negative, the associated dreams are also likely to be negative. Since the affect programs are attuned to be activated by stimuli or situations that operated in the original evolutionary context, it would not be at all surprising if minimal experiences with existing animals (canines, especially) and unrelated humans were sewn into the narratives that we know the brain tries hard to construct with the materials it is offered during sleep (probably by the forebrain as **SOLMS** points out).

It is even possible (although I remain agnostic on the matter) that the affect programs governing the basic emotions contain scenarios pre-loaded with content of threatening creatures and situations, so that we are primed to conjure up such scenarios once the relevant affect program is activated. This hypothesis, unlike **REVONSUO**'s, is parsimonious because it requires no special selection pressures to have ever operated on dreaming, while nonetheless explaining why some of the data REVONSUO uses in making his argument might exist as he claims they do. However, one reason for preferring my proposal for explaining threatening dreams in addition to parsimony comes from a problem with thinking that dreams could be useful for practicing for threats that might occur in the light of day.

REVONSUO makes little mention of the fact that most researchers find REM dreams bizarre and disjointed, unlikely sites for realistic rehearsals of threatening scenarios to take place. Also we are not told whether the simulations he claims are the function of dreams occur mostly in NREM dreams, or in REM dreams. If in the former, there is no surprise, since no one, to the best of my knowledge, has denied that in NREM dreams we are often worried and anxious. The trouble is that in realistic NREM dreams we frequently do not think in particularly productive ways about what we are anxious or worried about, instead, we are caught in perseverative ruts. If, on the other hand, the threat simulation and rehearsal dreams are REM dreams then the worry about the bizarre and disjointed nature of REM dreams arises again with the attendant worry that close content analysis of these dreams will not uncover neat simulations and rehearsals conducive to preparation for real world threats.

Freud ex machina. I am extremely grateful to **SOLMS** for his wonderful, pathbreaking book (1997a), and especially for the empirical evidence he provides there and in his target article for distinguishing sleeping from dreaming. My argument (Flanagan 1995; 1997; 2000) was that sleeping is an adaptation, or better, a set of adaptations, whereas dreaming is a free rider on a system designed to be conscious while we are awake, and which is designed to sleep – during which time conscious mental states are serendipitously activated. This argument has been met surprisingly often by the objection that if sleep is an adaptation then so too is dreaming. The basic intuition behind this objection is that sleeping and dreaming are a unity, part of one and the same neurobiological process and thus not suited for separate analyses.

There are two common arguments for not trying to untangle sleeping from dreaming. One is that they universally co-occur. **SOLMS,** thankfully, has provided ample ammunition to quiet those who press this objection. There is a double dissociation. There are people who REM but do not dream and there are people who dream but do not REM. The second argument for not distinguishing sleep from dreams is that they are caused by one mechanism or two, one setting us into NREM sleep and its associated type of mentation, the other doing the same for REM sleep and its associated mentation. The exciting new evidence presented by SOLMS and **NIELSEN** for multiple mechanisms responsible for different stages of sleep and possibly still different, independent ones, for dreaming helps thwart the second argument in favor of identifying sleeping and dreaming. The brainstem may get us REM-ing but it is forebrain activation (probably of the dopaminergic system) that gets us dreaming.

Now the multiple generator models defended by **SOLMS** and **NIELSEN** do cause problems for one who like myself maintains that dreaming is likely not an adaptation, but an evolutionary epiphenomena. The reason is this: in deciding whether some mental process is an adaptation or a free rider on an adaptation (or set of adaptations) much turns on how it is caused. If dreaming is reliably initiated by the forebrain turning on dopaminergic circuits then the process is much more well specified and we can ask why evolution might have selected for a mechanism that reliably sets us to REM dreaming. Often, possibly usually, a reliable mechanism has an adaptationist explanation.

SOLMS, however, is careful to point out that "the biological function of dreaming [I would add, "if any"] remains unknown." But in his book he tentatively endorses an adaptationist proposal that is mentioned in three of the other target articles (**HOBSON ET AL; REVONSUO;** and **NIELSEN**). This is the Freudian view that dreams function as the protectors of sleep. One reason for thinking this is the fact that people who don't dream don't sleep well. It is also worth noting that people who dream but don't REM or suffer some form of REM suppression don't sleep well either. But all these people have other problems – they have suffered strokes, or take drugs that mess with their sleep and/or dreams. So people who sleep abnormally or who have abnormal dreams (excessively vivid dreams, REM-less dreams) don't sleep as well as normals. But I don't see why the abnormal cases SOLMS discusses leads to any view whatsoever on what the function of dreaming is. Especially, how it provides any evidence about what, if any, proper evolutionary function dreaming has.

Another idea that **SOLMS** toys with is that REM dreams actually serve the functions of wish-fulfillment, hallucinatory satisfaction, and involve censorship – essentially the orthodox Freudian view of the way in which dreams protect sleep and thereby preserve mental health (Solms 1997a, p. 174). SOLMS is careful to present this stronger view as a tentative but testable hypothesis. And I agree that it is. But I am happy to bet against it. The reason is this: We need an account of why the forebrain and dopaminergic systems are activated and in being activated produce REM dreams. We already know that there is differential activation of the aminergic and cholinergic systems during different parts of the sleep

cycle and we are in possession of some decent hypotheses for why the brain is producing and/or stockpiling these neurotransmitters.

All these hypotheses – memory consolidation, trash disposal, stockpiling neurochemicals that are needed for attention and learning, even **VERTES & EASTMAN**'s proposal that REM sleep is designed to promote sleep recovery – require no story whatsoever about any biological function for the mentation itself. Meanwhile they can all avail themselves of exactly the same explanation why dreams occur and have the odd phenomenology they have, namely, that as sleep does what it is designed to do, it inevitably activates memories, emotions and so on, that are stored therein. So far, no hypothesis put forward requires that we think of dreaming as more than a side effect of the relevant functions of sleep.

Explanatory parsimony, and the expectation that dreaming will possess a unified (albeit complex) explanation lead me to expect that once we understand better why the brain needs dopamine, we will see that the activation of the dopaminergic system is just one other causal contributor to mentation that itself serves no fitness enhancing role. The issue is important, for the word on the street is that **SOLMS** has confirmed the orthodox Freudian view of dreams. To see this, consult Mortimer Ostrow' s – President of the Psychoanalytic Research and Development Fund – contribution to the February 24 issue of *The New York Review of Books* (Ostrow 2000, p. 46). It would be good for **SOLMS** to explain where he stands on this important issue, and to explain his reason for not siding with me (assuming he doesn't) in betting that dreaming will, upon closer examination of the reasons for forebrain and dopaminergic activation, be further revealed to be caused by, but not itself be a contributor to, what the sleeping brain is designed to accomplish. Especially in light of the fact that he finds many mentally healthy souls who do not dream, I would have thought that the orthodox Freudian view would, on his own terms, not be thought to be much of a contender. Having read all five contributions to this special issue carefully, I am more rather than less convinced that dreams are the spandrels of sleep.

Sleep, dreaming, and brain activation

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Abstract: Both Solms and Nielsen acknowledge the difficulty of accounting for the similarities between REM and NREM sleep mentation with a two-generator model, and each link dreams, either explicitly (Solms) or implicitly (Nielsen), to brain activation. At present, however, no data indicate that brain activation can be demonstrated whenever vivid dream reports are obtained.
[NIELSEN; SOLMS]

In reading and commenting on this series of articles on sleep and dreaming research, one is first and foremost impressed by the theoretical complexity of the field, a crossroads where different disciplines (epistemology, psychology, neurosciences) intersect, witnessing both the vitality of the research and the difficulty in attempting a unified theory. In fact all the proposed models involve tacit assumptions in each of these fields, which may outweigh the experimental evidence. The only antidote is the effort of spelling out the philosophic options underpinning the rules of the game agreed upon by author and reader; "a priori" implicit paradigms may hinder the theoretical debate. As the authors are neuroscientists, a preponderance of a radical reductionism identifying mind and brain is to be expected. However, the respectable old dualistic approach is still present. This makes for a mix of ideological (metaphysical) and scientific arguments in most theories of sleep and dream research.

SOLMS's target article boldly fits into the thorny framework of the "mind-body problem" (sect. 5). The epistemological problems surface from the Introduction: "REM [sleep] is controlled by cholinergic brainstem mechanism, whereas dreaming seems to be controlled by dopaminergic forebrain mechanisms" where the author claims that different neural circuits (even neurochemically identified) underlie both a physiological state (REM sleep) and complex psychological activities (dreaming). That we inhabit the realm of extreme reductionism is confirmed by the comment on the activation-synthesis model: "the burden of evidence has shifted to the anatomical link between the pontine brainstem and dreaming" (sect. 4, para. 5). Psychology is at last reducible to anatomy. The "localizers" (neo-phrenologists) are alive and well.

SOLMS has the merit of tackling the relevant issues of sleep/dream research with a straightforward logical approach, making it easier for the commentator to explicate points of agreement and disagreement. Despite the suggestive title, the evidence that "Dreaming is preserved with pontine brainstem lesions" (sect. 5) is scanty. On the contrary, the data reported in section 6 are convincing. Common sense strongly supports the notion that mental activity during sleep is affected by forebrain lesions; the focal nature of these lesions is striking however. The disagreement here is confined to the author's open and honest reductionism. Any curious neuroscientist, used to browsing through the various chapters of a neuroscience textbook, must be impressed by the ubiquitous involvement of the dopaminergic system in accounting for diverse and behavioral disturbances. Hence the assertion that "dreaming is generated by this dopamine circuit" (sect. 7, para. 4) may well be correct in a reductionist paradigm, but then in the following statement (sect. 8, para. 1) "dreaming involves concerted activity in a *highly specific* group of forebrain structures" the term "specific" remains puzzling.

The final paragraph (sect. 9) of the **SOLMS** target article addresses the problem of the relationship between dreaming and brain activation. A wary adherent to some form of psychophysical parallelism or correlationism like myself should be content with the proposed correlation between dreaming and brain activation. However, experimental data do not support the conclusion that "dreaming appears to be a *consequence* of various forms of cerebral activation" (sect. 9, para. 3). Brain activation is an ill-defined term, generally implying cortical desynchronization and high levels of cerebral blood flow (CBF) and metabolism. However, Cavallero et al. convincingly demonstrated the existence of slow wave sleep (SWS) dreams indistinguishable from REM sleep dreams (Cavallero et al. 1992). Recent PET studies have shown the reduced metabolic cost of synchronizing modes of operation in the thalamocortical circuits (Maquet et al. 1997), and a negative correlation between delta activity and regional cerebral blood flows was found (Hofle et al. 1997).

On the other hand, continuous Doppler recordings of CBF changes during the night (Hajak et al. 1994) revealed a tonic, continuous drop of CBF, upon which phasic state-dependent changes are inscribed. As a result, late REM sleep episodes occur at lower CBF absolute values than SWS episodes occurring early in the night. Therefore indistinguishable mental activity during sleep can accordingly coexist with different degrees of cortical synchronization and different levels of energy consumption in the cerebral circulatory-metabolic machinery: No combination of the two indexes of brain activation (electroencephalographic or biochemical) can account for differences in mentation during sleep.

Can we draw some preliminary, operational conclusions from these data? The brain seems to be endowed with more degrees of freedom that we had thought possible. Redundancy is a general property of the central nervous system, which makes it extremely flexible in generating similar outputs through different internal operations (as a result, a correlational model is theoretically possible but extremely complex in practice). In the case of dreaming, neither cortical desynchronization nor metabolic level can be taken as obligate indexes for a specific type of mental activity during sleep. The search for the correlation goes on. Finally, I entirely agree with **SOLMS**'s conclusion that "the function of dreaming and

the (equally unknown) function of REM sleep . . . should be uncoupled from one another" (sect. 9, para. 4).

In both the Introduction and in section 4 **SOLMS** states that "not all dreaming is correlated with REM sleep." This may be the starting point to confront his model with **NIELSEN**'s. Nielsen recognizes the difficulty of establishing a rigid correlation between REM/NREM sleep on one hand and different types of mentation on the other hand (the two generator isomorphic model). His proposed solution is straightforward: Whenever we encounter mentation during sleep, REM sleep processes, manifest or covert, must be at work. It is therefore a one-generator model, which identifies in REM sleep processes the unique source of mental activity during sleep.

In my view, the model implicitly assumes that covert REM sleep processes are responsible for some form of brain activation, a feature that is hence shared by the two models. In fact, by disturbing the homeostatic condition of SWS, all physiological variables that connote the covert REM sleep processes may contribute to shifting the level of brain activation; **NIELSEN** specifically mentions cortical EEG desynchronisation in the "atypical NREM sleep episodes" (sect. 3.3) that may depend on covert REM sleep processes. Moreover, in the list of factors that might induce "convert REM sleep to be activated during NREM sleep" (sect. 3.2) quite a few (arousal processes, sensory stimulation, drug effects, sleep deprivation) are known to enhance the energy metabolism of the brain.

The two facets of brain activation (electroencephalographic and metabolic) are therefore prerequisites for the model. **NIELSEN**'s hypothesis is based on well known physiological evidence, and has the merit of being experimentally testable. Polygraphic recordings show that the transition from NREM to REM sleep is not a clear-cut, abrupt event. Rather, different physiological variables change with different, contradictory time courses, and the macroscopic result may be the REM episode or an awakening or a return into NREM sleep. A single physiological variable can change alone, and may anticipate by many seconds, even minutes, the state change. It can be assumed that the complex process ultimately generating the full-blown REM sleep episode may have false starts and aborted outcomes; in this troubled transition (dynamic stage of train stem release in Parmeggiani's model, 1968); many physiological variables (increments in brain temperature and cerebral blood flow, heart rate, and blood pressure, Franzini 2000; motorneuron excitability changes, Nakamura et al. 1978) show a loose temporal link with the REM episode.

All this can be translated, in the terminology of **NIELSEN**'s model, as "covert REM sleep processes" (sect. 3.2). As NIELSEN acknowledges, "evidence of mentation in stage 3 and 4 sleep (Cavallero et al. 1992) is particularly difficult for this model to explain" (sect. 3.14, para. 4). The difficult task of validating the model requires: (1) that some of the physiological markers of "covert REM sleep processes" be identified in the uniform and stable conditions of stage 3 and 4 sleep; and (b) that the apparently "deactivated brain" of SWS may show focal signs of metabolic brain activation linked to the same physiological markers.

The prevalence of typical dream themes challenges the specificity of the threat simulation theory

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Abstract: The evolutionary theory of threat simulation during dreaming indicates that themes appropriate to ancestral survival concerns (threats) should be disproportionately represented in dreams. Our studies of typical dream themes in students and sleep-disordered patients indicate that threatening dreams involving chase and pursuit are indeed among the three most prevalent themes, thus supporting Revonsuo's theory. However, many of the most prevalent themes are of positive, not negative, events (e.g., sex, flying) and of current, not ancestral, threat scenarios (e.g., schoolwork). Moreover, many clearly ancestral themes (e.g., snakes, earthquakes) are not prevalent at all in dreams. Thus, these findings challenge the specificity of the threat simulation theory.
[REVONSUO]

REVONSUO's theory depends largely upon the observation that much dreaming is threatening in nature. But do the scenarios typically dreamed about today reflect the ancestral themes so central to the logic of this theory? Observations by our research group of the typical dream themes remembered by students and sleep-disordered patients are pertinent to evaluating the theory because they afford a global view of the scenarios most readily dreamed about over a lifetime within a given population. Our Typical Dreams Questionnaire (TDQ) includes 55 typical dream themes (cf. Griffith et al. 1958) that subjects check off if they have ever experienced them. We have administered the TDQ to over a thousand undergraduate students at different sites in Canada, the United States, and Japan and to close to a thousand sleep-disordered patients seen at the Sleep Disorders Center in Montreal. Consistencies between the lifetime prevalences of the most common TDQ items and those of Griffith et al. (1958) have been quite remarkable (Zadra & Nielsen 1997). Similarly, consistencies across our various samples have been very high (Nielsen et al. 1998; 1999c; Zadra & Nielsen 1999). In the case of three separate undergraduate student samples from McGill University (M age 20.3 ± 4.5 yrs; 113M; 228F), the most prevalent typical theme, endorsed by 78%, 86%, and 81 % of the student samples respectively (M = 82%) was the threat dream of being chased or pursued, but not physically injured (Zadra & Nielsen 1999). This theme was the second most prevalent typical dream of 233 Japanese undergraduates (M age 18.8 ± 2.3; 112F; 121M), 67% (Nielsen et al. 1999c), as well as the second and third most prevalent typical dream theme of two sleep-disordered patient samples much older in age (M age 44.9 ± 14.3 years; 249M; 235F), that is, 54% and 55% (M = 54%) (Nielsen et al. 1999b).

Such high lifetime prevalences of a threat theme are to some extent consistent with **REVONSUO**'s theory of threat simulation during dreaming. However, the high prevalence of many other typical themes poses problems for the specificity of the theory. Two themes that fall consistently among the "top 4" in both our populations are of sexual experiences (undergraduates M = 76% and patients M = 55%) and falling (M = 72% and 47%). These are not obviously related to the ancestral threats described by REVONSUO. It might be argued that sexual dreams address issues of genetic transmission through sexual reproduction. However, threat is not the principal dynamic of such dreams. Falling dreams may echo long distant threats to the successful evolution of the upright stance in humans, but this is clearly not the type of evolutionary adaptation REVONSUO's model is attempting to explain.

Other highly prevalent themes pose similar problems. Dreams of flying or soaring through the air ranked 9th among both undergraduates (50%) and patients (38%) and typically reflect positive affect, not threat. Other prevalent themes deal less with ancestral sources of threat than with contemporary concerns, in the case of students, schools, teachers, studying (ranked 3rd; 73%), arriving too late, for example, missing a train (5th; 59%), trying again and again to do something (6th; 58%), and failing an examination 10th; 47%). It is not clear why dreaming should so often represent similar positive themes and/or contemporary concerns if its function is still geared toward dealing only with ancestral sources of threat.

Several of our least prevalent themes also do not support the theory because they are ancestral threat themes that occur in very few young or old subjects. Among the undergraduates, tornadoes or strong winds (ranked 45th; scored by 15% of sample) and earthquakes (48th; 12%) are rarely dreamed about. In fact, the likeli-

hood of dreaming of these natural disasters is about the same as dreaming of being a member of the opposite sex (46th; 15%). Other natural disasters, such as fire (33rd; 23%) and threatening animals such as snakes (35th; 21%), wild, violent beasts (40th; 16%), or insects or spiders (23rd; 31%), also have low lifetime prevalences in our samples.

REVONSUO does offer some explanations for why such ancestral themes might be infrequent in dreams. First, some dream contents appear to change over time. For animals and aggressions at least, dreams appear to be more ancestral among children: "The brain has not yet had the chance to adjust the biases in order to better fit the actual environment" (sect. 3.4.2.2). The findings for children's dreams may well fit the threat simulation model, but it is not clear why the same pattern (i.e., high prevalence in the young, decreasing prevalence with age) should not hold true for other categories of threat, such as natural disasters. Nor is it clear why, in the case of children raised in environments relatively free from threat, the brain does not then adjust its simulations so as to be free of threat altogether. The notion of "change over time" in dream content (from ancestral themes to current themes) is problematic because such change would serve no obvious function. As described, it is only ancestral content that serves the (evolutionary) function stipulated by the theory. In sum, consistencies in the prevalences of typical dream themes in multiple study samples offer only limited support for the idea that dreaming is threat simulation. These findings would be more consistent with a less specific version of the theory that postulates simulations of positive, as well as negative, and of current, as well as ancestral, dream themes.

NOTE

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Each distinct type of mental state is supported by specific brain functions

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Abstract: Reflective waking mentation is supported by cortical activating and inhibitory processes. The thought-like mental content of slow wave sleep appears with lower levels of both kinds of influence. During REM sleep, the equation: activation + disinhibition + dopamine may explain the often psychotic-like mode of psychological functioning.
[HOBSON ET AL.; NIELSEN; REVONSUO; SOLMS; VERTES & EASTMAN]

1. Brain support of mentation during sleep-waking cycle [Hobson et al.]. From a general point of view, it is difficult for a neurophysiologist to admit distinct modes of psychological functioning during waking and sleep unless they have different underlying brain states. Each 1/100 second of change, mentation probably involves thousands of variations in neuron activity in numerous complex circuits. This suggests that each mental state has to be sustained by some kind of specific brain state. We must be grateful to **HOBSON ET AL.** for carefully analyzing the psychological data in the literature to show that there are indeed general differences between slow-wave sleep and REM sleep mentation.

It is important to provide a model, as this generates hypotheses for future research. It is certainly bold to propose a unique functional schema to explain mentation during states as different as waking and the various stages of sleep. **HOBSON ET AL.**'s proposal is of high interest; they bring many convincing arguments forward to support different kinds of mental functioning. Nevertheless, two points are questionable. First, it is asserted that there seems to be an opposition between high noradrenergic and serotoninergic levels and a low acetylcholine levels and vice versa. This is indeed true for REM sleep at the cortical level. However, it is not the case for waking mentation where there is simultaneously a high release of noradrenaline (Aston-Jones & Bloom 1981; Hobson et al. 1975), serotonin (McGinty & Harper 1976; Rasmussen et al. 1984) and acetylcholine (Jasper & Tessier 1971; Marrosu et al. 1995). Second, it seems difficult to rule out an involvement of dopamine at least in REM sleep mentation. This transmitter is not taken into account in the "AIM" model.

Our view is that, as is generally accepted, consciousness is mainly generated in the cerebral cortex. Traditional EEG studies, gamma activity, neuron firing, blood flow, glucose uptake, and acetylcholine release all show that the cortex is in a different state during waking, slow wave sleep, and REM sleep, this last stage being defined by criteria very similar to those of attentive waking (for details, see Gottesmann 1999). All these data also demonstrate greater cortical activation in REM sleep than in slow wave sleep. However, inhibitory processes are also involved in cortex functioning. Dopamine, noradrenaline, serotonin (Reader et al. 1979), and histamine (Sastry & Phillis 1976) principally inhibit cortical neurons. The function of dopamine alone cannot explain differences in mentation in normal subjects, because studies in rats (Miller et al. 1983) and cats (Trulson & Preussler 1984) have shown that their neuronal firing rates do not change significantly during the sleep-waking cycle. Histamine neurons become silent as light sleep appears (Vanni-Mercier et al. 1984), and hence could potentially explain differences in cortical functioning during waking and sleep but not between slow wave sleep and REM sleep. In contrast, noradrenergic and serotoninergic neurons fire maximally during waking, decrease their activity during slow wave sleep, and become silent during REM sleep. Thus, they might control cortical functioning during the sleep-waking cycle. The importance of serotonin, at least, is established because decreased release induces the mental distortions associated with depression.

Our hypothesis accordingly is that during waking the cerebral structures involved in mentation are activated and thus able to generate mental activity, but that inhibitory processes in some way control or "normalize" this activation, thereby explaining reflective mentation. During slow wave sleep there is a decrease of both kinds of influence, explaining thought-like mental contents, because some controlled activation does persist. During REM sleep, the strong cortical activation occurs in a context of massive disinhibition, when all monoamines except dopamine are absent. This strong disinhibition alone could explain the original properties of mentation, which partly resemble psychotic symptoms, as described by Hobson et al. (1998b). We suggest that in this original activated and disinhibited state, the release of dopamine, strongly involved in psychosis, would reinforce this often schizophrenic-like mode of functioning. Indeed, an increased release of dopamine induces nightmares (Thompson & Pierce 1999) and psychotic disorders (Buffenstein et al. 1999). Moreover, it is well known that the reduction in the influence of dopamine by induced neuroleptics (Kinon & Lieberman 1996) alleviates schizophrenia.

2. Experimental data to confirm the covert REM sleep hypothesis are still lacking [Nielsen]. The hypothesis of **NIELSEN** is highly important and confirms that dreaming only occurs in the physiological setting of REM sleep (Takeuchi et al. 1999b). This could explain why dreams have been described during slow wave sleep, in addition to thought-like activity. The problem is to determine the crucial physiological criterion to support REM sleep mentation. Dement wrote about thirty years ago (I do not remember where, nor does he) that REM sleep is like an orchestra playing a symphony: Several instruments (criteria of REM sleep) can be absent without suppressing playing (this sleep stage).

The arguments brought forward for sleep-onset dreaming are convincing. I have regularly such vivid life-like dreams currently and have wanted for months to record myself, being neither depressed nor narcoleptic. **NIELSEN** states that in addition to a possibly similar EEG, the same slow eye movements are seen at sleep onset as during REM sleep. To determine whether other criteria of REM sleep are found at sleep onset, particularly those linked

to eye movements, it would be interesting to record phasic integrated potentials (PIPS) (Rechtschaffen et al. 1970), cortical waves (McCarley et al. 1983; Miyauchi et al. 1987), and the ear muscle activity (Pessah & Roffwarg 1972). During slow wave sleep, activation of the visual and, more lightly, of the secondary auditory cortex (Hofle et al. 1997) could be an index of REM sleep and explain dreams recorded during this stage (Foulkes 1962).

Prior to, and sometimes just after REM sleep, mice, rats, and cats show an intermediate stage with hippocampal criterion of REM sleep (for a review see Gottesmann 1996). Several other pieces of experimental evidence show REM sleep premises at this hinge-period, (McCarley & Hobson 1970 for cortical neuron tiring; Morrison & Bowker 1975 for PGO waves; Morales & Chase 1978 for medulla neuron activity; Kanamori et al. 1980 for medulla oblongata neuron firing; Steriade & McCarley 1990a for mesopontine neuron activity; Sei et al. 1994 for blood pressure variations, etc.). In humans, as related by **NIELSEN,** Lairy et al. (1968) described an intermediate phase prior to and after REM sleep. It is characterized by interspersed criteria of slow wave sleep and REM sleep. However, the mental content does not correspond to the description of dreams.

First, it is difficult to establish a psychological contact with the subject behaviorally wakened from this stage. This is in contradiction to the good contact with the outside world observed on awakening from REM sleep, and with the sentry theory of Snyder (1968). Second, the verbal reports do not reveal visual contents but instead "a feeling of indefinable discomfort, anxious perplexity and harrowing worry" (p. 279). Although for Foulkes (personal communication, 1998) this result is debatable, Larson and Foulkes (1969) show that mental contents at this sleep time "are inconsistent with the hypothesis of an intensification of mental activity or cerebral vigilance at pre-REM EMG suppression. They seem, rather, to point to a reduction in reportable mentation and in efficient cognitive reactivity at this point of transition from NREM to REM sleep" (p. 552). These results are not in accordance with vivid visual dreaming activity. Despite its major interest, the hypothesis of **NIELSEN** needs to be confirmed by new psychophysiological studies performed particularly in the period preceding REM sleep.

3. Is Revonsuo so far from Freud? **REVONSUO**'s is a rich hypothesis as it does not imply a systematic phenomenon: Its aim "is not to claim that every single dream of every single individual should realize this (threat simulation) function." This idea is original and supported by many dream contents reported in the literature. It is also true that the historical period of human life represents a ridiculously small part of the lifetime of the human species. Consequently, it is understandable that such ancestral fantasies should persist today above all because "dreaming has no maladaptive consequences, so it has survived." This is generally but not always the case (e.g., Huntington's chorea, which persists probably because reproduction occurs prior to the appearance of the disorder). REVONSUO comes close to Freud's (1900) position when he states "the content of dreams shows far too much organization to be produced by chance." Although he mentions "The Interpretation of Dreams," he overlooks subsequent work in which Freud mentions "primal fantasies" transmitted phylogenetically. More precisely, Freud (1918) described a famous dream "The man and the wolves," which fits with REVONSUO's thinking, for the patient was threatened by wolves. REVONSUO would deduce that "dreaming does have a well-defined and clearly manifested biological function (that is) to simulate threatening events," while Freud's psychological interpretation appeals to a hypothetical observation of parents' sexual "primal scene" and the ancestral danger of castration by the father, which is a threat to the subject's reproduction abilities; a concept also taken up in REVONSUO's theory. In fact, the two interpretations are in some ways complementary.

I am slightly more cautious about **REVONSUO**'s use of physiological arguments. **VERTES** and I (see below) are doubtful about theta rhythm function during REM sleep. Perhaps in opposition to currently accepted ideas (Hobson & McCarley 1977), I am also not totally convinced about the obligatory relation between PGO waves and dreaming. These spikes have to do with short-lasting activating influences (maximum 100 milliseconds, Miyauchi et al. 1987) that transiently reinforce cortical tonic activating processes of REM sleep. Although related eye movements appear in the pontine cat (Jouvet 1962), they are modulated by the cortex (Mouret 1964), which shows an activation of the saccadic eye movement system (Hong et al. 1995). Moreover, the cortical visual projection area is deactivated during REM sleep (Braun et al. 1998; Madsen et al. 1991a). Finally, rats seem to dream, as shown by behavioral characteristics (eye, vibrissae, ear, paws and tail movements) and pontine lesions (Mirmiran 1983). However, they display pontine spikes during REM sleep (Farber et al. 1980; Gottesmann 1967; 1969; Kaufman & Morrison 1981), but no REM-sleep-related spikes in lateral geniculate nucleus and cortex (Gottesmann 1967; 1969; Stern et al. 1974), despite a direct neuronal relationship between pons and visual cortex (Datta et al. 1998). It seems that dreams have a different time scale from that of short-lasting PGO waves, unless we accept that the successive spikes are responsible for the rapid changes of dream content, which currently seems doubtful.

4. Sleep-waking stages are induced and mentation is supported by brain stem structures [Solms]. In his interesting contribution, **SOLMS** is right that dreaming is not initiated in the brain stem, contrary to the old, somewhat naïve theory of Hobson and McCarley (1977), rapidly discarded (Vogel 1978a). All his arguments in favor of the forebrain as generator of dreaming processes, and of mentation more generally, are convincing; today they seem self-evident. Nevertheless, his clinical arguments in support of the assertion that "dreaming is preserved with pontine brainstem lesions," thus that dreaming is able to occur without the brain stem inducing properties of REM sleep, are less convincing. Moreover, in the examples of forebrain-induced dreams it is not always easy to distinguish dreams from hallucinations; and in physiopathological conditions, hallucinations are often mistaken for dreams (Fischer-Perroudon et al. 1974). Through its midbrain and pontine structures the brain stem induces the different sleep-waking states and does not induce but supports correlative mentation.

It is somewhat difficult to agree entirely with **SOLMS**'s hypothesis concerning dopamine's almost exclusive influence on dreaming-generating processes. He is of course right when he emphasizes the role of dopamine. Indeed, as the species evolves this transmitter probably has an increasingly important influence on cortical functioning. Where dopaminergic projections end only at prefrontal level (Hökfelt et al. 1974) in the rat, in primates all cortical areas are concerned (Berger et al. 1991), and this is probably also the case in humans (Smiley & Goldman-Rakic 1993). However, in the normal subject, the absence of noradrenaline and serotonin cortical input seems to be the precondition for the participation of dopamine in the dream mentation of REM sleep. If it were not, we would always be in a dream state, for dopaminergic neurons fire similarly during all stages of the sleep-waking cycle. Moreover, several properties of dreaming seem to be explicable by the disinhibition process alone (Gottesmann 1999).

5. Is it possible to speculate about theta rhythm function during REM sleep? [Vertes & Eastman]. The numerous arguments against the theory of memory consolidation during REM sleep by **VERTES & EASTMAN** are convincing, particularly those obtained in humans by pharmacology. Nevertheless, our own experience shows that it is possible to induce emitted potentials during REM sleep but not during slow wave sleep; this means that during REM sleep there is access to memory processes established during waking (Gauthier et al. 1986), although this result does not demonstrate memory consolidation.

An electrophysiological datum used by **VERTES & EASTMAN,** based on animal studies, appears debatable. They argue that although hippocampal theta rhythm occurs during waking and REM sleep, its properties are different in the two states: only dur-

ing waking would it be involved in mnemonic function. As it has already been clearly shown by Grastyan et al. (1959), learning is associated with theta rhythm in animals. Green and Arduini (1954) were the first to show that this limbic rhythm is induced by activation of the midbrain reticular formation. This is probably owing in part to the stimulation of fibres passing nearby; Vertes (1981) has shown that the nucleus pontis oralis is the main origin of hippocampal synchronized activity. At first sight, it is hard to imagine that when the same (rather crude) basic structure induces similar theta activity during both waking and REM sleep, the target structure could function differently in the presence of the same activity during the two states. However, several pieces of experimental data support this dichotomy. First, the brain stem is not necessary for the induction of the theta rhythm. Acute intercollicular transected rats and cats (Gottesmann et al. 1980; 1984) show virtually continuous theta rhythm and it is difficult to assume that there are integrated functions in the hippocampus of a neocortically comatose animal. Second, serotoninergic innervation of the hippocampus, which is crucial for hippocampal functioning in memory processes (Matsukawa et al. 1997), becomes silent during REM sleep (Rasmussen et al. 1984); the influence of noradrenaline (Segal & Bloom 1974) is also suppressed (Aston-Jones & Bloom 1981). Therefore these two monoamines, which most often have inhibitory influences on higher brain structures, are important for mental processes. Third, by analogy, the neocortical EEG is similar during waking and REM sleep while consciousness is different. Consequently, as suggested by VERTES & EASTMAN, despite nearly identical theta rhythm, the hippocampus is in a different functional state during waking and REM sleep.

The function of REM sleep proposed by **VERTES & EASTMAN** seems to be a revival of Roffwarg et al.'s (1966) theory of the importance of endogenous brain activation in ontogenetic early brain maturation.

Where is the forest? Where is the dream?

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Abstract: In this commentary I discuss the importance of considering the isomorphism between the full richness of dreams and the great body of information about REM sleep that is amply documented in the five target articles. With this inclusive mode I point out the importance of looking at REM sleep as involving both pontine and cortical activity in an integrated network. We cannot have a full appreciation of sleep and dreaming (view of the forest) without taking both physiology and mental activity seriously. **[HOBSON ET AL., NIELSEN, REVONSUO, SOLMS, VERTES & EASTMAN]**

In discussing these five extremely detailed, scholarly, and thoughtful target articles, I will use two organizing principles: isomorphism and the forest and the trees. My commentary will consider some issues that involve all the articles and will also point out some problems that are specific to individual articles.

We are privileged, in this issue, to have extensive and quite complete reviews and summaries of much of the research that has emerged from the sleep research laboratories of the world during the last 50 years. Thus the reader is presented with many of the facts. However, the reader will also notice that different authors give different weight to certain facts in order to bolster their individual theories. I would liken these presentations to a wonderful description of sets of trees in a forest, but I am afraid, as I will try to demonstrate, the forest has been lost. One reason for this is that a major group of trees has been almost totally ignored. That has to do with a discussion of dreams. By this I mean dreams as seen in all their fullness and richness, rather than as collections of single units.

As **REVONSUO** quotes Farthing, "we may define a dream as a subjective experience during sleep, consisting of complex and organized images that show temporal progression" (Farthing 1992). The loss here is as great as that which might occur if we studied Shakespeare by counting and tabulating words and sentences rather than considering the meaning of the units when assembled into story, plot, and poetry. An important reason for raising this issue is that we cannot have a really isomorphic or consistent picture (the forest) without a major part of the picture. These papers present an excellent view of the physiological and the cognitive aspects of sleep and dreaming but leave out what I think is the third leg of a three-legged stool, namely the dynamic and emotional meaning of dreams, which only two of the authors really touch on. Let me give an example of how helpful this can be.

In the 60s, as a psychoanalyst who was also involved in sleep laboratory research, I was troubled by the lack of fit between the total emphasis on the pons for the understanding of REM sleep and the richness and meaningfulness of the dreams that I worked with in the clinical situation. It seemed to me that the cortex must play a significant role in the process. Because of this I chose to study the REM sleep patterns in a group of patients with right parietal lesions and attention deficit visual disorders. The findings were clear and showed that the eye movements during REM sleep were missing in the direction of the affected visual fields (Greenberg 1966; **HOBSON ET AL.** cite this study but fail to note this basic finding. Dorrichi's study 25 years later is a replication of this study). This led to a review of Jouvet's oft cited study showing the persistence of REM sleep in decorticate patients. The REM sleep in these patients was not normal; the eye movements were isolated and lacked the bursts seen in normal sleep. This is certainly consistent with the idea that the pons might be firing away but without a cortex no visual imagery and dreams were being generated.

SOLMS's contention that REM sleep can occur without dreaming and dreaming is separate from REM sleep seeks, on the other hand, to omit the role of the pons in the process. SOLMS can then maintain that it is the cortex alone that is responsible for dreams and that what we know about REM sleep has no relevance to understanding dreams. With Hobson's now partially abandoned but still frequently responded to idea that the pons alone is responsible for REM sleep with dreaming as an epiphenomenon, we have two starkly contrasting views which both fragment the process. In this commentary I wish to underline the fact that, as several of the authors suggest, we are dealing with a process which involves, in nonpathologic cases, the activity of a network of parts of the brain which must be working together to have a fully functional output. This includes the pons as a trigger plus various parts of the cortex which provide affective, imagistic, and motivational components to dreams.

An essential feature of this commentary depends on the idea that dreaming and the physiology of REM sleep are two aspects of the same process and the concept of isomorphism suggests that conclusions in each realm must be consistent with those from the other. Clearly the authors of these target articles disagree amongst themselves about the connection of dreams and REM sleep and the conclusions to be drawn from REM deprivation studies or from the exploration of dreams. I do think, however, that **HOBSON ET AL.** and **NIELSEN** present a very convincing case (the best I've yet seen) for the differences between REM and NREM mentation. I would like to add a few other bits of data. Although Foulkes is a major proponent of the difficulty in distinguishing REM from NREM mentation, a sample of 30 dreams he collected from a student from both REM and NREM awakenings revealed only one dream from NREM (Foulkes 1967). All the rest were from REM. Cartwright (Cartwright et al. 1967 in NIELSEN; and Cartwright 1972) has shown that the subgroup of normal subjects who do seem to generate dream-like material from NREM sleep awakenings are distinguished by various psychologic measures such as a high schizophrenia score on the MMPI. Also, it is mainly with gradual and not abrupt awakenings that mental activity is elicited from NREM awakenings (Goodenough et al. 1965a; Shapiro et al. 1963 and 1965 in NIELSEN), thus raising the question of whether the mentation is truly from NREM sleep. NIELSEN discusses this issue in more detail. Finally, as **REVONSUO** notes, the Penfield

stimulation studies elicit visual flashbacks but these are "dissimilar from dreams" in various respects. Therefore, I will contend that REM sleep and dreaming co-occur and any theory must consider the isomorphism of theories derived from the too different manifestations of a single process.

Thus we have a process that includes well-studied physiologic activities that are associated with a very special type of mental activity. Can this association illuminate our understanding of these two seemingly different phenomena? That is, can what we have learned about REM sleep tell us something about what happens in dreams and can what happen in dreams orient us in our understanding of the physiologic findings? I have noted earlier how clinical experience with dreams led to a study of cortical activity during REM sleep. The role of the cortex is now, 30 some years later, much more clearly elaborated in the imaging studies noted by several of the authors and by the rich picture generated by **SOLMS**'s studies of patients with lesions.

Before considering how the physiologic findings have influenced our understanding of dreams, I must consider and respond to some of the observations the various target articles make about the function of REM sleep and especially the role of information processing. **VERTES & EASTMAN** raise a number of objections to the notion of the role of memory processing in REM sleep. They, in effect, suggest ignoring all the positive studies by proposing that stress is the major "villain." We should note here the very creative studies on cueing during REM sleep by Hars and Hennevin (1987; in **NIELSEN**) which **REVONSUO** fails to include. That nonarousing meaningful cues during REM but not NREM sleep can lead to improved learning indicates clearly that an effective consolidation process is occurring during REM sleep; one cannot invoke stress as issue.

VERTES & EASTMAN readily dismiss the fact that windows for sensitivity to REM deprivation occur, because in relation to different learning tasks or in different species the timing varies. This does not account for the fact that stress occurs equally in the animals deprived of REM during both the window and nonwindow periods, and yet there is the clearly demonstrable effect on memory consolidation only when REMD occurs during the critical period. Furthermore they do not deal with the fact that these time periods correspond to the periods of increase in REM seen after training trials during which there is an increased retention.

Finally, **VERTES & EASTMAN** seem unaware, in their claim that only Smith has shown windows (i.e., not replicated), that Pearlman (1973) had shown this phenomenon many years before. Another set of findings not considered is that different types of learning are or are not sensitive to REMD. We have formulated this as the difference between prepared and unprepared learning (Greenberg & Pearlman 1974). It is unprepared learning or complicated learning that seems sensitive to the REMD impairment of learning or that is followed by an increase in REM pressure after training. This holds for animals and for humans. (See Greenberg & Pearlman 1993 for full discussion.)

This idea is especially important in **VERTES & EASTMAN**'s description of the lack of effect on function of MAOI REM suppression in humans. A brief anecdote may speak most clearly to the kind of memory that is affected. Again it is not cognitive. A colleague was treating one of the subjects in Wyatt's study (personal communication). He did not know any of the sleep data or what drug the patient was taking. For months the patient seemed to be feeling better but also seemed to have no access to past emotionally meaningful experiences. Also no dreams. Then suddenly the patient began to bring in intense dreams and to be much more in touch with her past. The therapist then found out that an MAOI had been administered with complete suppression of REM and that the drug had been discontinued at the time things began to open up. We also have found a disconnection from past meaningful memories in subjects who have been REM deprived (Greenberg et al. 1983). Because of the lack of apparent cognitive deficits, VERTES & EASTMAN dismiss the use of REM suppressant drugs in animals to examine the effects of REM suppression, without stress, on learning. Yet the common denominator for the flower pot method and the drugs is REMD.

If we keep in mind, at this point, the idea of isomorphism, we might see that the observed affect of REMD on the integration of new experiences is consistent with some interesting "new" perspectives about dreams. I use the word "new" because in these target articles references to psychoanalytic ideas about dreams are exclusively references to Freud. Readers should be aware that much has been learned since "The Interpretation of Dreams" was published 100 years ago (see Greenberg & Pearlman 1999). Readers should become familiar with the work of Bonime (1962), French and Fromm (1964), and Reiser (1997) to see how these authors present a picture of dreams that is much more consistent (isomorphic) with much of the sleep lab research presented in these papers.

The picture of dreams that emerges is one in which one can see the current problems in adapting with which the dreamer is struggling and how solutions for problems are searched for. The manifest dream and affects become more important than Freud's theories have suggested. The dreams show the process that can only be inferred from the role seen for REM sleep in the processing of information. The dream can now be understood if one looks for problems rather than just for the categories of or numbers of individual items or events in the dream (see Greenberg et al. 1992).

This approach allows a somewhat different perspective from **REVONSUO**'s evolutionary theory. Yes, REM sleep and dreaming are important in man's and also other mammals' mastery of a threatening environment. By insisting on the separation of REM sleep and dreaming, REVONSUO can allow himself to look only at dreams. He goes even further in reducing his focus to dreams dealing with external dangers, that is, traumatic dreams. This allows him to miss the real import of the Hartmann (1984; 1998) work he cites. This has to do with the evolution of the traumatic dream from a replay of the trauma to one in which there is evidence of integration of the traumatic event into the dreamer's series of life experiences. As this evolution occurs, the clinical manifestations of trauma in both waking and dreaming life abate. The work of the dream is not just to deal with practicing for dealing with external dangers, but rather to enable the dreamer to deal with the complicated and unresolved feelings evoked by external events, be they wild animals, enemies, or other events that feel dangerous, such as abandonment, induced helplessness, humiliation, and so forth.

REVONSUO cites the appearance of threatening animals to support his idea that dreaming evolved to deal with external danger and continues to serve only that purpose. He fails to consider that the appearance of elements like wild animals may be the metaphoric or symbolic language with which the dreamer expresses more internal fears. He claims that dream content is independent of external psychological and physical stimuli. **HOBSON ET AL.** make the same claim. Here there is a failure to consider that dream content shows a great deal of what is emotionally significant. Breger et al. in "The Effect of Stress on Dreams" (1971) presented very clear evidence of the relationship of dream content to the stresses with which the subjects were dealing. In our study (Greenberg & Pearlman 1975) we also showed clear evidence of how the content of the dream was related to emotionally significant waking mental experience and Cartwright (1996; in REVONSUO) also illustrates this point. These studies demonstrate how the dreamer is struggling with life events.

REVONSUO's theory is a special case for the role of dreaming. His idea that it is related to survival is discussed in a much more complete fashion by Rotenberg (1993a). Rotenberg's theory of search activity brings the role of REM sleep and dreaming into a very central position in our consideration of the question of survival. Of interest here is that Rotenberg's studies showed a role for search activity in the maintenance of the immune system. REVONSUO uses the analogy of the immune system to suggest that it is not always called into action. One must consider, however, the importance of it always being ready. One must also consider that ex-

ternal threats are not the only kind of problems that appear in dreams. Our study (Greenberg & Pearlman 1993) demonstrates that almost all dreams show evidence of problems and emotionally meaningful ones at that. What does vary is the appearance of solutions to the problems, varying from successful to unsuccessful. Just as the immune system is not always successful in fighting infection, so too the dream is not always successful, and in the traumatic dream, the system is clearly overwhelmed. What Rotenberg adds to our understanding is the idea of searching for solutions and this is what is of importance for survival. Thus I would suggest that while REVONSUO takes the dream very seriously, his focus is narrowed to a special kind of dream and in part this is the result of his failure to take seriously the idea of an isomorphism between the dream and what occurs in REM sleep in all species that have this process available.

Let us now turn to comments about the individual target articles. **HOBSON ET AL.** present a comprehensive picture in which they give reasons to believe that REM sleep and dreaming indeed go together. They reason that we are dealing, in this process, with a network that includes both the pons and cortical areas interacting. Where they fall short is the failure to include the richness of dreams and the implication that cortical lesions affect both the appearance and the quality of dreams. Thus, dreams provide an opportunity to learn more about the nature of the information that is processed during REM sleep. By taking the position that dreams do not show evidence of the inclusion of stimulating and emotionally important material from waking, they lose the meaningfulness of studies like Breger et al.'s (1971), Cartwright's (1996; in REVONSUO), or ours (Greenberg et al. 1992) to name a few. They also approach the forgetting of dreams from a purely physiologic perspective, rather than considering that the language of a dream is different from awake language and therefore harder to remember. Dreams do not just disappear. Note the frequent experience of the sudden remembering of a dream when a reminder during the day will bring a dream fully to mind. This is a frequent observation in the clinical situation but I suspect most readers have also had this experience. The dream experience is indeed a part of the memory system. How else could one explain the fact that the occurrence of REM sleep plays a role in learning? Something more permanent must be recorded. By continuing to view the forebrain as responding to brainstem input (I in AIM) rather than as a partner in the process, the Hobson group loses the opportunity for a truly isomorphic consideration where the richness of dreams is given a full hearing.

NIELSEN is one author who takes the phenomenology of dreaming into full account and his idea of covert REM mentation deserves consideration. I would raise one question for what is on the whole a plausible and well-documented presentation. Could it be that shifting from one to another kind of mentation could be analogous to the way the mind can wander during wakefulness? Thus, is the appearance of covert REM activity (dreams) during the NREM periods evidence of its constant activity or is there some shifting back and forth (mind wandering) which some subjects are more susceptible to (see Cartwright et al. 1967 and Cartwright 1972)?

SOLMS takes the role of the cortex very seriously and informs us of the special attributes of the cortical areas that are involved in dreaming and also in REM sleep. However, he would like to divorce the pons from any role in dreaming. I think he goes too far in arguing that the pons is not the cause of dreaming and ignores the possibility that it is the trigger for a process that involves networks in the cortex that are involved in dreaming. He then is able to totally ignore the connection between REM sleep and dreams and all the implications for our understanding of dreams which have emerged from studies of the function of REM sleep. For example, he refers to Panksepp's ideas about the "seeking" or "wanting" part of the brain but does not connect this with Rotenberg's ideas about the Search activity function of REM sleep. SOLMS also seems unaware that the "REM" sleep generated in the decorticate subject is not normal REM sleep (see above) and finally, as he notes, the evidence is not in that subjects with pontine lesions abolishing REM sleep can actually still dream.

In conclusion, I have tried to provide a picture of the forest by adding a few trees, or groves, to the excellent descriptions provided by the authors of these articles. I would argue that only by integrating the information we have about dreams with what we know about REM sleep, considering that a network is activated through the whole brain, can we realize the parallels between what we have learned about dreaming and what we have learned about REM sleep. I do not know if this discussion will change any of the authors' minds but I hope that as they write their responses they will read some of the papers and books referred to in this discussion but not included in the target articles. I would also like to note that in a commentary in this journal entitled "The cortex finds its place in REM sleep" (Greenberg 1978), I was premature and only in the last few years has there been growing evidence of the role of the cortex. I hope that the place of the dream will not have to wait so long.

State-dependent modulation of cognitive function

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Abstract: The three introductory questions posed by Hobson et al. point toward further investigations of cellular, circuit, and systems mechanisms involved in cognitive function that include the effect of CNS-state related modulatory systems on these mechanisms.
[HOBSON ET AL.]

Two of three introductory questions posed by **HOBSON ET AL.** concern (1) the major differences between the phenomenological experience of these three physiological states (waking, REM, and NREM); and (2) measures that might establish clear cut differences between these states at the level of brain regions, as well as the cellular and molecular levels. These broad ranging questions might involve measures existing as the average levels of activity of the cholinergic and monoaminergic modulatory systems (Steriade & McCarley 1990a; Siegel 1990) referred to as "M" by the authors, but this has not been established with respect to cognitive function. The traditional view, based on studies reviewed by the authors, is of waking as a CNS state of high cholinergic and monoaminergic tone, of NREM sleep as a CNS state of low cholinergic and monoaminergic tone, and of REM sleep as a CNS state of high cholinergic and absent monoaminergic tone. Whether or not these simple measures are adequate may depend on questions like, "how do these simple changes affect the function of cortical and thalamic circuits and what might result in cognitive function?"

The answers are just beginning to surface, as for example, effects of modulatory tone on EEG activity (Buzsaki 1998; Herculano-Houzel et al. 1999; Steriade et al. 1993) and cholinergic modulation of visual systems interneurons (Xiang et al. 1998). However, the changes in circuit function that can derive from cholinergic and monoaminergic modulation depend on our understanding of the circuit function and its relationship to cognitive function (as for example: Eichenbaum 1999; Goldman-Rakic 1999; Hesselmo 1999; Lisman 1999; Wang 1999). Further, the magnitude of the complexity of this issue is emphasized by a recent study showing 25 different response combinations in CIA interneurons to noradrenaline, serotonin, muscarine, and mGluR agonists (Parra et al. 1998).

It would seem surprising yet it is conceivable that changes in circuit function in CA1 that results from a state specific in modulatory tone (restricted to just the cholinergic and monoaminergic systems) are sufficient to account for the state specific alterations in CA1 information processing. This provided that one also takes into account the effects of the change in cholinergic and mono-

aminergic modulatory tone on other afferent systems to the CA1 (a change in the pattern of septal gabaergic input for example; Dragoi et al. 1999). In other words the author's third question, "Can a tentative integration of the phenomenological and physiological data be made?" implies the challenge of both the investigation of cellular, circuit, and systems mechanisms involved in cognitive function as well as the effects of CNS-state related modulatory systems on these mechanisms.

The dramaturgy of dreams in Pleistocene minds and our own

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Abstract: The notion of simulation in dreaming of threat recognition and avoidance faces difficulties deriving from (1) some typical characteristics of dream artifacts (some "surreal," some not) and (2) metaphysical issues involving the need for some representation in the theory of a perspective subject making use of the artifact.
[HOBSON ET AL.; REVONSUO]

Underlying the conceptual shape and contents of threat recognition and avoidance simulation in **REVONSUO**'s fascinating functional theory of dreams are, I believe, some metaphysical anomalies which make the view somewhat inchoate as it stands. I shall develop this diagnosis via heuristic analogies while drawing on observations contained within the theory of **HOBSON ET AL.**

REVONSUO proceeds on the bold metaphysical assumption that consciousness just *is* the organization of the brain at the phenomenal level. It includes the subjective experience of dreaming (described, quite interestingly as it turns out, as a mechanism) which in slumbering Pleistocene minds (PMs) contained simulations of threat perception and rehearsals of threat avoidance responses and behaviors. By "prepping" the PM for coping with real life counterparts of these simulations, the reproductive success of our ancestors was supposedly enhanced.

But how in general do we picture the contents of another's dreaming mind wherein **REVONSUO**'s simulations and rehearsals are said to have operated, when we reconstruct it using known or plausibly surmised historical facts coupled with seemingly parallel contemporary anthropological data involving dream reports? It will in significant ways depend on how we picture the contents of our own dreams when we recollect them.

Here are two very abbreviated and somewhat "cinematic" examples of how I think dreams often seem to us, and with slight permutations in content can be imagined as having occurred to PMs as well: (1) in our mind's eye, in memory, we imagine an image of our self (the dreamt self) spotting a dangerous animal bearing down on it, the dreamt self picking up a big stick and/or running away; (2) in our mind's eye, in memory, there is no image of oneself any more than there is in waking life when I run from a neighbor's nasty tempered mastiff and don't observe myself doing so.

How, then, might **REVONSUO**'s simulations utilize representations like those pictured, in either (1) or (2) so that we obtain a sense of how dream-production systems churning out thousands of realistic threats during a PM's lifetime is thereby "bound to result in improved threat-avoidance skills"? It is said to turn on "what the dream can do itself' (its "natural functions") where the notion of "the dream itself' (phenomenal or p-dreaming) explicitly excludes reference to any self-conscious recollections or "invented functions," that is, cultural and personal uses of dreams and dream reports (Flanagan 1995).

So how does the dream imaging prove to be functionally efficacious for **REVONSUO** (as it is not for Flanagan, nor "aimless" as in the Activation-Synthesis theory)? How can its utility as simulation rehearsal be depicted at that first person level? My concern is that if it cannot, then the dramatic color and detail of simulation rehearsals as posited by REVONSUO washes off into the purely neurobiological domain, and signals not just a stylistic diminishment but a substantive one.

An analog suggests itself which might prove useful in that its outline parallels in interesting ways with **REVONSUO,** yet contains ingredients which his theory lacks, but may in some form need. This is Aristotle's influential theory that art involves imitation, is instinctual, instructive, and pleasurable. Aristotle, if we focus on the first three attributions and ignore the last, conjures up general, albeit inadvertent, likenesses to REVONSUO's claims that ancestral dream consciousness was biologically functional with adaptive value. (Think of imitation and simulation, as going proxy for each other in numerous contexts, simulations and rehearsals as being instructive, and the species-specific non-conventional nature of the [Aristotelian] instinctual and REVONSUO's biologically functional and adaptive.)

Consider, then, Aristotle and **REVONSUO** with respect to the following examples, any one of which to us looking in at them, as it were, might be called a *transparently instructive simulation* (TIS) of a real world thing or situation: (1) a detailed drawing of the anatomy of a cow for use in a course in veterinary medicine; (2) a video of a black belt karate instructor teaching a student a given kick; (3) a dress rehearsal of a play to iron out the kinks in it before actual production; (4) a cockpit of an airplane (*sans* the plane) wherein pilots can learn to master switches and buttons in preparation for flying a real plane.

In our "looking in" we assume a first person point of view of what it would be like for ourself to see such things or imagine ourself participating in such contexts, and we sense that if we had done so, we would have been edified. (We think of having looked at the cow picture and how that might have helped direct our dissection of a real cow; we think of having memorized the moves in the karate video and using them in self-defense, or, if we had been an actor in the play (the dreamer as other to itself in the dream) we might have noticed mistakes we had made and resolved to, correct.)

Any of these examples might prove friendly to Aristotle's claim that art as imitation (simulation) is instructive. Pursuing the parallel, we ask whether what Aristotle seems to have at hand – a range of TISes – has any kind of counterpart within the medium of dreams? If not, why? And does it matter?

Something *like* TISes seem to me demanded by **REVONSUO** but exactly what or how is so unclear as to make the alleged demand seem gratuitous. Consider some of the "remarkably consistent set of features" listed by **HOBSON ET AL.** as characterizing dreaming: "dream imagery can change rapidly and is often bizarre in nature;" self-reflection, often absent in dreams, "when present, often involves weak, post hoc, and logically flawed explanations of improbable or impossible events and plots"; dreams "lack orientational stability; times and places are fused, plastic, incongruous and discontinuous." Let me call these characteristics *serializing aspects of the dream as artefact of sleep* (or SER).

Dream dramas where it is as if a homuncular Salvador Dali reigned as dramaturge, strike me as unlikely material for TISes in somewhat the same way as a drawing of a cow's anatomy where its organs are distorted and melted into each other would seem less than useful in directing a dissection of a real one. Furthermore, there are other aspects of artefacts that contribute to their potential instructiveness: duration, intersubjective availability, potential for preservation, copying or replication, and so on that seem conspicuously absent from the phenomenal level of dreaming. Such "no shows," the density of SUR, and the exclusion, anyway, of all non-natural, "invented" or "cultural" features from consideration, make the budget of materials available for anything like counterparts to TISes in **REVONSUO**'s theory very skimpy indeed.

The problems I suggest might pose for **REVONSUO** primarily pertain to oddities attending the stuff of dreams and whether certain types of nocturnal "artifacts" could be constructed from it. (Compare: Could a rigid sculpture be made out of feathers and

molasses?) These can be thought of as artifact problems. But there are other rather different difficulties which, though related to the foregoing, seem to me much more threatening to the dramaturgical coherency of REVONSUO's theory. These might be called perspective problems.

Part of what makes Aristotle's idea of instructive imitations (simulations) intelligible is our ability to find a locus of perspective or a subject who engages the simulations either from a standpoint outside of them, or through a presence within them: for example a person viewing or watching something (1 or 2) or being involved in some collective or singular action (3 or 4). But how does any perspective or participating subject inhabit **REVONSUO**'s phenomenal "space" wherein its simulations reside? Which "whos" or "whats" constitute a simulation's dramatic personae and function vis-à-vis each other and the simulation viewed as artifact? It is difficult, indeed, impossible to conceive of either the dreamt self in a dream or the dreaming self (sans body-image) carrying out that role, for they, after all, belong to the "stuff of dreams" and cannot migrate literally and usefully into the real world in spite of anthropological reportage on peoples who may believe they can (Tedlock 1987a; Merrill 1987a). Nor can the real dreamer, in spite of impeccable ontic obduracy be the transporter of instructive dream "text." For this would involve exactly the sort of self-reflective sense of dreaming that REVONSUO regards as unlikely for a PM, and not germane in any case, since the dream's functional utility is attributed wholly to the simulation bearing dream experience itself. So who or what might audition better?

The only viable candidate remaining so far as I can tell is somehow the whole of the subjective experience of dreaming itself, described by **REVONSUO** as a "mechanism." If so, it is this mechanism – and I would suppose qua mechanism, a neurobiological one – that is also a subject with perspective, that is the locus of the dream, and furthermore whatever content the dream is defined by. If this interpretation is correct, REVONSUO's is not only a cognitive functional account of dreaming, but also a ticklish metaphysical position in which the subjective self which dreams is physically objectified as a neurobiological mechanism wherein the artifacts of dreaming coalesce with their artificers, and the content of dreams and the dreamer are one.

It is here (at last) where I believe the underlying metaphysical anomalies alluded to at the outset, to reside. To treat the brain mechanism as itself the needed subjective self with requisite perspective, is to create two more problems for **REVONSUO** (1) the complex and colorful idea of the dreamer-cum-rehearsal simulation is reduced to the very general idea of a person sleeping and, by dreaming about what was hair-raising that day, keeping in tune the neurobiological mechanisms needed to survive the morrow. (2) It assumes that the subjective self can be objectified in terms of a mechanism – a goal, to be sure, shared by virtually any physicalist account of the mind-body relationship. It also shares with these views the burden of coherency in the face of well known challenges from, for example, Nagel (1986), Jackson (1986), McGinn (1991), and many others.

The waking-to-dreaming continuum and the effects of emotion

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Abstract: The three-dimensional "AIM model" proposed by Hobson et al. is imaginative. However, many kinds of data suggest that the "dimensions" are not orthogonal, but closely correlated. An alternative view is presented in which mental functioning is considered as a continuum, or a group of closely linked continua, running from focused waking activity at one end, to dreaming at the other. The effect of emotional state is increasingly evident towards the dreaming end of the continuum. **[HOBSON ET AL.; NIELSEN; SOLMS]**

First of all, **SOLMS**'s target article is about the control of dreaming – in other words, what portions of the brain are necessary for reports of dreaming (or visual dreaming) to occur. In these terms, SOLMS is certainly convincing in his demonstration that portions of the forebrain are involved – specifically the parieto-temporal-occipital junction, probably controlled or activated by a dopaminergic pathway in the ventral-mesial portions of the frontal lobes. SOLMS is right in pointing out that his data disprove the original Hobson and McCarley "Activation-Synthesis" view that dreaming is entirely dependent on REM-sleep activation of the forebrain by brainstem centers. **HOBSON ET AL.** do not exactly acknowledge this, but they do shift their emphasis from dreaming toward a broader attempt to explain the biology of the states of consciousness.

The target article by **HOBSON ET AL.** on the recent AIM model is impressively detailed, up-to-date in its references, and also creative and imaginative. It is a valiant effort to make sense of almost all current information on states of consciousness, organizing it according to three simple-sounding dimensions. The reach of the model, however, exceeds its grasp.

The AIM model makes most sense in terms of its ability to organize or "locate" three basic states – waking, NREM sleep, and REM sleep. I believe that the model is much less useful in its attempts to explain the psychological features of the states – including dreaming – even though **HOBSON ET AL.** use terms such as activation and information flow which are meaningful in cognitive as well as biological terms and thus could provide a bridge. For instance, one problem is that the waking state, which appears in the diagrams as a small, isolated box, actually supports a large array of cognitive activity, including overlap with the other states, as discussed below.

The chief problems I have with the three-dimensional AIM model are the following: First of all, an obvious question when looking at such a dimensional diagram is why *three* dimensions rather than more or less, and why these particular dimensions. The familiar spatial metaphor of a cube or box can delude us into thinking that three dimensions constitute an obvious number and that these three exhaust the possibilities. I do not question that activation (A), direction of information flow (I), and "modulation" (M) are of importance – though "modulation" is a very complex notion, defined at one point as "how information is handled," which is not convincingly a single dimension. I could think of some other relevant dimensions. For instance, do we simply want one dimension of "amount of activation?" What about a dimension of "focus," or "spread of activation," or perhaps a dimension involving velocity of activation, introducing time and measuring the speed with which different portions of the system are activated. I mention these dimensions because, like the others, they can easily be thought of in cognitive as well as purely biological terms. (In fact, although I was not thinking in terms of "dimensions," I made use of such variables years ago in what could be called an early "two-dimensional model." In formulating the principal characteristics of the three biological states we were beginning to recognize – waking, REM sleep, and NREM sleep – I summarized REM sleep overall as a state of "high-activity level," (similar to waking) but with "rough adjustment – poor feedback" (very different from waking) (Hartmann 1967, p. 149). (This formulation in fact may still be helpful in characterizing the physiological and psychological characteristics of REM sleep.)

Thus, three dimensions may not be the right number. However, my main concern is that the three dimensions of AIM may not truly be dimensions at all. It makes little sense to discuss in detail what happens in different regions of a three-dimensional cube unless one has reason to believe that the three axes are actually more or less orthogonal (independent). On the contrary, I believe there is considerable evidence suggesting that two of the important dimensions (I and M) and perhaps all three dimensions are intimately correlated.

Table 1 (Hartmann). *A continuum*

	Focused Waking thought	Looser, Less-structured thought	Reverie Free Association Daydreaming	Dreaming
What dealt with?	Percepts Math symbols signs, words		fewer words, signs, more visual-spatial imagery	almost pure imagery
How?	logical relationship – If A then B		less logic, more noting or picturing of similarities, more metaphor	almost pure picture – metaphor
Self-reflection:	highly self-reflective – "I know I am sitting here reading."		less self-reflective, more "caught up" in the process, the imagery	in "typical" (non-lucid) dreams total *thereness*, no self-reflection
Boundaries:	solid divisions, categorizations, thick boundaries		less rigid categorization, thinner boundaries	merging condensation loosening of categories, thin boundaries
Sequence of ideas or images:	A→B→C→D	A → B ⟨C ↕ D⟩	A → B ⟨C ↕ D⟩	A ⟨B ↔ C ↕ D⟩
Processing:		relatively serial; net functions chiefly as a feed-forward net		net fuctions more as an auto-associative net
Subsystems:		activity chiefly *within* structured subsystems		activity less *within*, more across or *outside* of structured subsystems

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For instance, suppose we turn off the lights, close our eyes, and pull a blanket over our heads, but remain awake. Obviously we are moving from chiefly external input to internal input along dimension I, and as we do, we inevitably also move along the dimension M, and begin to process material, produce images, and so on, in a more "dreamlike" manner. A whole literature on "sensory isolation" supports this close association. Antrobus and others have shown that the longer a period of uninterrupted isolation (or internal processing) lasts, the more dreamlike cognitive activity becomes (see Antrobus 1990 for a review). All this occurs even if we are not tired. If we are tired and are "winding down" at the end of a day, our brains are presumably less activated. As "A" is reduced we concomitantly find ourselves paying less attention to the external world and more to the internal world (changes in "I"), and we also begin to think in a more imagistic and dreamlike way (changes in "M"). In fact, a huge body of work on relaxation states, meditation states, and hypnosis demonstrates such changes. This suggests that the "dimensions" are by no means independent, and that perhaps we need another way to conceptualize these changes in mental functioning.

My colleagues and I have in fact found it useful to think in terms of a continuum or a series of related continua, running from focused waking at one end through reverie and daydreaming, to dreaming at the other (Hartmann 1996a;1998; 2000a; Kunzendorf et al. 1997) (see Table 1).

This continuum, which we can call the "focused waking-to-dreaming continuum," obviously refers to states of the system capable of supporting conscious mental processing. It is clear that a certain level of activation is required to support consciousness, and thus all the points on the continuum in Figure 1 can be considered states of relatively high activation. Under these conditions we consider it useful to think of one continuum with many strands, rather than orthogonal dimensions.

This view emphasizes the continuity in mental functioning between the conscious states of waking and dreaming rather than the discontinuities. The work of Antrobus (1991), Foulkes (1990), Klinger (1990), Singer (1975), and others supports this continuum viewpoint. Material indistinguishable from dreams can be obtained from quiet waking, from sleep onset, and from NREM sleep. We have demonstrated that under certain conditions, daydreams and dreams are very similar and that depending on personality characteristics, the daydreams of some students are scored just as dreamlike and "bizarre" as the dreams of other students (Kunzendorf et al. 1997). Dreaming is mental activity (based on cortical activity) at the right end of the continuum that contains very little focused rapid-processing activity. For instance, we have shown that reading, writing, and arithmetic ("the three Rs") are extremely rare in dreams (Hartmann 1996b; 2000b). Activation or spread of activation is less focused and more diffuse, which I have related for many years (Hartmann 1973, p. 138, 155n) to low levels of norepinephrine in the cortex (among other factors).

Furthermore, the "qualitative" differences between dreaming

and waking mental activity are not as clear-cut as sometimes thought. For instance, if we consider the difficulty of recall, which **HOBSON ET AL.** frequently cite as a clear difference, I think it is useful to consider whether it is appreciably more difficult to recall last night's dreams than to recall the waking daydreaming or reverie we experienced this morning while washing or shaving.

In brief, our view is that at the dreaming end of the continuum there is more overlap of cortical activation patterns, or more bringing into conjunction elements often kept separate. Thus, we can say that presumably at the cortical level there is greater connectivity; connections are made more readily at the dreaming end of the continuum. As is well known, this can sometimes be useful artistically or creatively, but it is an everyday phenomenon as well. Five different women have told me a dream very close to the following: "I dreamt of Joe, my boyfriend, but in the dream he looked very much like my father" or "he turned into my father." I am not attempting any deep or Freudian analysis; my point is a simple one. In all five cases the women awakened and said something like: "Isn't that interesting. Of course Joe is like my father in three or four different ways; odd that I had never noticed that before." In other words the similarities were evident if one stopped to consider them but it took a dream – activity at the right hand end of the continuum – to bring the two networks together.

Thus, we consider connections to be made more broadly in dreaming – at the right end of the continuum – than in focused waking, but the process is not at all random; we have shown that it is guided by emotion. (Emotion is hardly mentioned in the models discussed in the target papers, although recent imaging studies – reviewed by **HOBSON ET AL.** and by **SOLMS** – have lent strong support to activation of the limbic system, especially the amygdala, during REM sleep.)

For my group, the paradigmatic dream is what we have come to call the "tidal wave dream." We have studied people – apparently normal adults – who have recently experienced an acute trauma: an escape from a fire in which others were killed, a rape, an attack, a sudden death of someone close. We believe this is an important starting point in the study of dreams, because here – unlike most of the time – we know clearly what is emotionally important in the dreamer's life. The dreamer, after an acute trauma, may or may not have a few dreams that repeat the actual trauma or aspects of it. Then, very frequently, one or more dreams such as the following occur:

> "I was walking along the beach when suddenly a huge tidal wave came and engulfed me. I was tossed around, I tried hard to get to the surface. I woke up terrified."

What is going on here? Obviously the person is not dreaming of his or her actual experience. Rather the dream is contextualizing (producing a picture-context for) the dominant emotion of terror/helplessness/vulnerability. We believe this is a paradigm, that the same process probably occurs in all dreams but is less easy to detect when we do not know of a clear dominant concern (for details, see Hartmann et al. 1998a). We have shown quantitatively that powerful images of the tidal wave type occur more frequently after trauma (Hartmann 1998, Hartmann et al. 1998b).

The view then is that the dream pictures or "contextualizes" the emotional state of the dreamer. Again, the continuum view suggests that emotion always influences our mental activity and imagery, but this is least evident at the left end of the continuum, when we are doing arithmetic or involved in focused thought. It becomes more evident as we move towards the right. There is evidence suggesting that emotion has a greater influence on our cognition and perception when we are relaxed or in a relaxed hypnotic state than in our ordinary waking state (for instance, see Kunzendorf & Maurer 1989; Klinger 1996).

It is of interest that Hobson's group continues its curiously negative view of the process of dreaming. They have previously spoken of dreaming as "delirium," (Hobson 1997b) and as "your brain on drugs" (Kahn & Hobson 1994). In the present target article making an analogy with epilepsy where "activation signals of limbic origin commandeer the cortex and force it to process," they argue that "the cortex of the dreaming brain is compelled to process internal signal rising from the pons." All this is a very "focused-waking-centric" viewpoint. We consider dreaming in a more neutral manner as simply one end of a continuum of mental functioning. Dreaming, after all, is a widespread natural phenomenon, consuming a considerable amount of our time. It stands to natural-selection-based reason that dreaming probably has some function for the organism, and in fact several related functions have been proposed (Moffit et al. 1993; Hartmann 1998). Far from being "compelled," dreaming can be considered the least constrained type of mental activity. I find it natural to think of great portions of the cortex as an image generator. The system can be "constrained" to perform arithmetic or logic problems during focused waking, but it tends to "relax" into daydreaming or dreaming when there are no such constraints.

Viewing mental functioning along a continuum, as in Table 1, may also be useful in resolving the "one-generator versus two-generator" issues raised in the target article by **NIELSEN.** Our model certainly favors the idea of a single generator producing mental activity (thoughts and images) but a single generator whose products can vary along a continuum or a series of related continua as in Table 1. **NIELSEN** does not mention the varieties of waking mentation at all, but if one accepts the evidence that dreaming mental activity is not completely and qualitatively separate from daydreaming activity which in turn is not completely separate from other waking thought, one would hardly want to postulate a separate "generator" producing each of these related states. Nor is evidence presented by **NIELSEN** or others that would lead me to believe that NREM mentation is so qualitatively different from the entire continuum above that it would require a "generator" of its own.

Furthermore, from the point of view of brain anatomy, including the neuropsychological and brain imaging data reviewed, it makes a great deal of sense to consider a single image generating process, which, however, can be activated in a number of different ways, presumably using the cortical and subcortical pathways delineated in the target article by **SOLMS.**

The close relationship between dreaming and other forms of mental activity such as daydreaming or other waking imagery is also supported by ontogenetic studies by Foulkes, demonstrating that the ability to experience and report fully formed visuo-spatial dreams develops gradually, at around age 5–8, at about the same time as the development of full visuo-spatial abilities in the waking state (Foulkes et al. 1991).

The lesion studies by **SOLMS** may also be relevant to these questions, in the sense that it would be of great interest to know the precise status of daydream or reverie activities in the neurological patients who had lost the ability to dream, or to dream visually. **SOLMS**'s study procedures include an extremely detailed dream interview covering 13 different areas (Solms 1997a, pp. 83–86). However, he does not specifically mention interview questions dealing with daydreams or reverie. If indeed **SOLMS** found a well-delineated group of patients who had stopped dreaming (or stopped dreaming visually) but continued to have clear visual daydreams and reverie exactly as before, I would take this as evidence against the continuum view I am discussing here. However, I do not believe this to be the case. No data are presented specifically on loss of daydreaming or reverie; however, **SOLMS** does mention in the present paper that in the large PTO (parietal-temporal-occipital) patient group other deficits were found. For instance, right-sided lesions in PTO were associated with not only cessation of dreaming but "disorders of spatial cognition." Left-hemisphere lesions were associated with disorders of "quasi-spatial (symbolic) operations." He also notes that lesions in visual association areas caused defects in visual dream imagery, "in association with identical deficits of waking imagery." **SOLMS** in fact suggests that "the visual imagery of dreams is produced by activation during sleep of the same structures that generate complex visual imagery in wak-

ing perception." Therefore I believe that SOLMS's results are consistent with a view of a single widespread cortical system or image generator (with several subsystems) generating what we usually call dreams, and that this system similarly generates a whole continuum of waking imagery.

Reflexive and orienting properties of REM sleep dreaming and eye movements

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Abstract: In this manuscript Hobson et al. propose a model exploring qualitative differences between the three states of consciousness, waking, NREM sleep, and REM sleep, in terms of state-related brain activity. The model consists of three factors, each of which varies along a continuum, creating a three-dimensional space: activation (A), information flow (I), and mode of information processing (M). Hobson has described these factors previously (1990; 1992a). Two of the dimensions, activation and modulation, deal directly with subcortical influences upon cortical structures – the reticular activation system, with regard to the activation dimension and the locus coeruleus and the pontine raphe neuclei, with regard to the modulation dimension. The focus of this review is a further exploration of the interaction between dreaming and the cortical and subcortical structures relevant to REM sleep eye movements.
[HOBSON ET AL.]

The question of cortical versus subcortical control of saccadic eye movements during REM sleep is addressed by the target article and previous Hobson publications. **HOBSON ET AL.** review the controversy between brainstem-only models (bottom-up model) of control of REM sleep and combined brainstem-cortical models (top-down model). If REM sleep eye movements are exclusively regulated by brainstem mechanisms, then either they are totally independent of dream images, or both the eye movements and dream images of REM sleep are significantly governed by brainstem mechanisms. If they are under combined brainstem-cortical control, then dreaming could be of cortical origin and still linked to eye movements.

Lesion studies demonstrate that some form of REM-like eye movements occur during REM sleep in the decerebrate preparation: these and other studies demonstrate clearly that noncortical structures are necessary and sufficient for the generation of the eye movements of the REM sleep. The dilemma presented by such findings regards the commonly held assumption that visual dreaming during REM sleep is cortical phenomenon.

In 1975, McCarley and Hobson asserted that the occurrence of REM sleep and its timing are controlled by reciprocal interaction between cells in the pons (FTG cells) and cells in the nucleus locus coeruleus (LC cells). This assertion was instrumental in the neurophysiological and conceptual basis of the current modulatory (M) portion of **HOBSON ET AL.**'s three-dimensional AIM model. In 1977 McCarley and Hobson's activation-synthesis model (the A of the AIM model) literally turned the universally accepted model of dreaming on its head. They contend that brain stem neuronal mechanisms more significantly influence the timing, formal properties, imagery, and content of REM sleep dreaming and that the role of the cortex was secondary, synthesizing incoming volleys of corticofugal excitation (Hobson & McCarley 1977).

HOBSON ET AL. note that it is highly implausible that all REM sleep saccades are concordant with dream imagery, given the presence of eye movements in the congenitally blind. Also, animal studies in cats and monkeys, in which accurate measurements of the direction of both eyes and their positions are possible, indicate that the eyes are moving asymmetrically during REM sleep. HOBSON ET AL. conclude that even brainstem initiated REM sleep eye movements are most likely under the control of a final common pathway which integrates brainstem generation and forebrain modification, a substantive modification of his earlier assertions of brainstem control (Nelson et al. 1983).

Such integration or cortical and non-cortical control of REM sleep eye movements is consistent with Doricchi et al. (1993; 1996), who observed that patients with left visual hemi-inattention, or visual neglect, showed dissociation between the direction of waking saccades and that of REM sleep. In contrast, during waking, exploratory saccades were present in both lateral directions, but confined to the right hemispace. Hence, the neglect patient is capable of executing saccades in both horizontal directions, but does not do so in REM sleep. Following two months of training, neglect patients increase leftward waking saccades, but none appear during REM sleep.

Therefore these authors (Doricchi et al. 1993) propose that two mechanisms control waking saccadic eye movements: one of a more voluntary, cognitive classification, typically employed in visual exploration, and the other more automatic and involuntary, related to reflexive-orienting. The total absence of leftward saccades during REM sleep indicates that *all saccadic eye movements during REM sleep are reflexive-orienting eye movements.*

Morrison and colleagues (Ball et al. 1991a; Bowker & Morrison 1976; Morrison et al. 1995; Sanford et al. 1992b; 1993), propose that REM sleep pontine geniculate occipital (PGO) waves constitute a response of central mechanisms to afferent information which, in the waking state, would be capable of eliciting an orienting response. Spontaneous PGO waves, similar to those accompanying the waking orienting response, are present throughout REM sleep, indicating that PGO waves are the REM sleep equivalent of an orienting response. They propose that *during REM sleep, higher cortical structures are in a state of virtually continuous orientation* (Ball et al. 1991b). This is a similar to Doricchi's conclusion that REM sleep eye movements are functionally equivalent to waking reflexive-orienting saccades.

Pompeiano and Morrison (1965; 1966; Morrison & Pompeiano 1966; 1970; Pompeiano & Valentinuzzi 1976) along with coinvestigators, based upon a series of studies in the decerebrate preparation, have demonstrated a complete cessation of REM eye movements, following bilateral vestibular enucleation. They have identified the anatomical structures responsible for REM bursts as the vestibular nuclei, the oculomotor nuclei, and the oculoorbital system.

Herman et al. (1983) have shown that the characteristic of eye movements during REM sleep in humans is more consistent with those observed with accompanying head movements than with those observed when the head is stationary. These observations, elicited by careful questioning, are not inconsistent with the concept that REM sleep eye movements are related to vestibular activity and reflective-orienting responses (Herman 1992).

It is proposed that a constant property of vivid REM dreaming is the sensation of orientation in the dreamt space, or the hallucinated impression that the dreamer is physically present in the dreamt scene, in three-dimensional space. The dreamer is directionally oriented and aware of a spatial relationship to the persons or objects present in the dream. The sensation of orientation is made possible by cortical-subcortical mechanisms, including the vestibular system. The associated eye movements are related to reflexive and orienting responses to the dreamt surround via feedback loop connecting cortex to vestibular nuclei. *It is postulated that the sense of orientation is the phenomenological equivalent in dreaming to the continuous vestibular activation during REM sleep.* This ubiquitous presence of self in the dream parallels Pompeiano and Morrison's finding that intact vestibular structures are required for the typical occurrence of REM sleep. In the same manner that the vestibular nuclei are necessary for the eye movements of REM sleep, so is the sense of self oriented in space an essential component of human dreaming.

The ghost of Sigmund Freud haunts Mark Solms's dream theory

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Abstract: Recent neuropsychological data indicating that an absence of dreaming follows lesions of frontal subcortical white matter have been interpreted by Solms as supportive of Freud's wish-fulfillment, disguise-censorship dream theory. The purpose of this commentary is to call attention to Solms's commitment to Freud and to challenge and contrast his specific arguments with the simpler and more complete tenets of the activation-synthesis hypothesis.
[HOBSON ET AL.; NIELSEN; SOLMS]

We have recently commented at length on **SOLMS**'s neuropsychological data and dream theory (for scientific details and references see Hobson & Pace-Schott 1999). The following précis is drawn from a paper presented at the American Academy of Arts and Sciences in Boston on January 12, 2000, at which Mark Solms was the invited discussant. This commentary focuses sharply upon SOLMS'S interpretation of his data, which clearly reveals his commitment to psychoanalytic dream theory. Only those data (and they are few) which can be retrofitted onto Freud's dream theory are deemed worthy of SOLMS's theoretical attention. The rest (and they are many) are either discredited or dismissed. While SOLMS does not mention Freud in his target article, it is clear from his book (Solms 1997a) and from his subsequent writings that his major theoretical orientation is psychoanalytic. The following commentary is thus designed to clarify our interpretive position and to contrast it with the Freud-Solms theory.

Solms's attempt to resuscitate Freud's dream theory. Until recently, I would have said that Freud's theory had no scientific status whatsoever and that activation-synthesis was its natural replacement (Hobson 1988b; Hobson & McCarley 1977). But recently **SOLMS** has interpreted his very interesting findings on the effects of brain lesions and brain disease upon dreaming as favoring Freud's dream-as-wish-fulfillment hypothesis (Solms 1997a). In essence, SOLMS has found that strokes or surgical interventions that damage two distinctive areas of the brain in the inferior parietal lobe and in the white matter tracts connecting subcortical structures to the medial prefrontal cortex are often followed by a loss of dreaming. The parietal region subserves spatial cognition and the white matter tracts connect important components of the limbic system that are thought to mediate emotion, motivation, and reward. This finding, interesting enough in itself, is complemented by a raft of brain imaging studies showing selective activation of these same structures and their directly associated cortical and subcortical regions during REM, the phase of sleep most capable of supporting dream consciousness. SOLMS also points out, quite correctly, that temporal lobe seizures produce dream-like states in waking and intensify dreaming in sleep.

Surely the new evidence indicates, as **SOLMS** claims, that Freud was on the right track in postulating wishes as dream instigators especially insofar as wishes are related to emotional arousal in humans. And so it might at first glance seem. But emotions, motivations, and rewards are not wishes in any unconscious, Freudian sense. Furthermore, positive dream emotions, like elation, joy, and erotic excitement do not qualify as unconscious Freudian wishes, and they certainly do not need disguise. More importantly, as we have shown in our formal analysis of dream emotion, they are not disguised in dreams. Why, after all, would we want to be unconscious of these emotions? Because they might wake us up? When I have dreams of flying or sex I hope they won't be censored and that I won't wake up!

What about negative emotions, like anxiety, fear, and anger which constitute a goodly portion of dream emotion? Are they wishes? I think not! I can see why I might want to disguise or censor them, but negative emotions are not disguised in my dreams either! The fact that I do wake up from these dreams is both a relief and proof that both the wish fulfillment and the guardian of sleep ideas that Freud concocted are just plain wrong!

What does activation-synthesis make of all this? Simply that in response to automatic and selective activation in sleep of the brain structures mediating the emotions, we consciously experience them undisguised and uncensored in our dreams. This is exactly what we said in our 1977 activation-synthesis paper.

> The new theory cannot yet account for the emotional aspects of the dream experience, but we assume that they are produced by activation of brain regions subserving affect in parallel with the better known sensorimotor pathways." (Hobson & McCarley 1977, p. 1336)

Recent PET imaging studies have confirmed this prediction and we have further elaborated this notion by suggesting that emotions may have a primary shaping force in determining dream plots.

If you want to interpret your anxiety dreams, or your elation dreams, or your anger dreams, go right ahead. You will find, as I do, that these emotions are associated with the same kind of plot content that they are in daily life (even if the dream plots are more humorously bizarre). For example, anxiety in my dreams is often associated with being lost (fear), with not having appropriate attire or credentials (anxiety), with being chased, or threatened (fear). Can this be read as the product of either wish fulfillment or disguise-censorship?

What I mean is that if you want to understand your dreams, the last person you would want to consult is Sigmund Freud or one of his psychoanalytic protégés! That is because unconscious wishes play little or no part in dream instigation, dream emotion is uncensored and undisguised, sleep is not protected by dreaming, and dream interpretation, via free association, still has no scientific status whatsoever. **SOLMS**'s data-based and eloquent defense of Freud is nonetheless welcome. Why? Because SOLMS is so committed to the original text that he allows us to reframe the activation, synthesis theory in the light of the new neuropsychological findings and to show even more clearly the weakness of Freudian theory.

Support for activation-synthesis by the new neuropsychological data. If it is not unconscious wishes, then what does cause dreaming? Why are dreams bizarre? Why are dreams hyperemotional? And most interesting of all, why are dreams so quickly and thoroughly forgotten?

Here are activation-synthesis answers to these questions. Dreaming is caused by brain activation in sleep. Dreams are bizarre because the activation process differs in important ways from that of waking. Dreams are hyperemotional because the emotional brain is selectively activated in sleep. Dreams are forgotten because recent memory systems are disabled during sleep. Let us look at each of these issues in turn to see how well neurophysiology can explain them.

As soon as we fall asleep, the brain begins a dramatic transition from one activation state (waking) to another (REM sleep). The main features of this transition are the blockade of sensory inputs and motor outputs (which puts the activated brain in a closed-loop mode) and the chemical demodulation of the activated brain (which puts it in a distinctive processing mode). Associated with these changes, all of which are controlled by the brain stem, is the selective activation of internal information sources including pseudo-sensory data, which generate the dream perceptions and emotional data, which generate the dream affects.

Just after sleep onset, the brain is still activated enough to produce micro-dreams and it starts to do so as soon as external data can no longer have easy access to the still appreciably activated brain. Not only are the sensory and motor gates of the brain rapidly closing, but three of the specific chemical systems of the brain stem that support waking consciousness are also beginning to shut down. For these reasons the self-reflective awareness, directed thought, attention, and memory control of waking are rapidly slipping away. This concatenation of events is responsible for sleep onset dreaming.

Sleep onset dreaming is short-lived and evanescent because the activation status of the brain rapidly plummets and it ultimately falls to such low levels (in Stages III and IV of non-REM sleep) that mental activity of any kind is difficult to sustain. As sensory and motor gates continue to close and we become dead to the world, three of the brain stem chemical systems that help support waking consciousness decline even further. PET scans reveal that the lights have gone out in command central. Under these conditions, full-fledged dreaming is practically impossible. However, simple recall of at least some mental content from these stages is around 50 percent (Nielsen 1999).

But after about a 90 minute interval, the cerebral tables are turned and the brain is converted by REM sleep into an all but obligatory dream machine. This is because while two of the brain stem chemical systems regulating consciousness (those producing norepinephrine and serotonin) are completely shut off, the acetylcholine system is released from inhibition. The acetylcholine system not only reactivates the brain, but provides it with powerful, internally generated signals. These signals project to the sensory systems of the thalamus and cortex, triggering vivid dream perceptions, and to the limbic system of the forebrain triggering dream emotions. And, of course, those two forebrain systems communicate with each other. Under these conditions, dreaming is more vivid, more bizarre, and more sustained than in any other state.

Human PET scan studies of REM (see **HOBSON ET AL.** 1998a; 1998b; 2000; and target article, for reviews) confirm the main features of this picture and add an important, unexpected observation: the dorsolateral prefrontal cortex is selectively deactivated in REM sleep. The hallucinating, emotion-drenched brain is thus deprived not only of the chemicals it uses to tame such processes in waking but also loses the direction provided by its key top-down guidance system. The prefrontal cortex is generally considered to be the seat of executive cognitive functions like working memory, volition, self-reflective awareness, critical insight, and judgment, none of which work well in dreaming (see again HOBSON ET AL.)

No wonder that we are so hopelessly unable to get a grip on things in dreams, to know where we are or what exactly we are doing, or even to recognize that we are dreaming! On the contrary, we normally ignore all of the obvious internal evidence and conclude that we are awake! And no wonder we can't remember dream events either as they occur or after we wake up. Dream forgetting is not owing to repression as the Freudians would have us believe. It is simple, organically determined amnesia. No wonder, too, that dreams are bizarre. Improbable or downright impossible incongruities and discontinuities are synthesized by the hyperassociative brain only to pass unnoticed because the censor – far from being hyperactive – is sound asleep! It is in the accounting for dream bizarreness, that Freud's theory is not only incorrect, it is absolutely backwards!

Even **SOLMS** has admitted that disguise-censorship is the weakest part of Freud's theory. But I submit that this characterization of disguise-censorship is a euphemism, at best. Without disguise-censorship, Freud's dream theory is entirely negated because it cannot do what it was intended to do: that is, explain dream bizarreness. Even if one were to admit that dreaming is, in part, motivated by forces that include something like instinctual drives, it is clear on its face that these forces are undisguised and that dream bizarreness is entirely inadequate to the task Freud assigned to it.

Critiques of activation-synthesis, including **SOLMS**'s**,** have pointed to the tendency of late night NREM sleep to support dream mentation almost as well as REM. The obvious reason for this change is that as the night progresses, the general level of brain activation rises toward waking levels and NREM sleep becomes more and more REM-like. The EEG fluctuates between stages I and II (rather than III and IV). The input-output gates are still closed, and aminergic modulation is still weak enough to allow sleep to continue. Under the circumstances, dreaming is quite likely and, in essence, late night NREM dreaming is the other side of the sleep onset dreaming coin. But REM sleep is still the optimal substrate for dreaming and that is why its neurophysiology is so informative. In addition, many REM-related physiological events probably occur in NREM sleep without producing the full complement of signs necessary to score REM sleep in traditional methods (Nielsen 1999).

Can dreaming and REM sleep brain process be dissociated? Hoping to sidestep the devastating impact of modern sleep neurobiology upon Freud's dream theory, **SOLMS** and other critics of activation-synthesis have argued that since the forebrain is the acknowledged seat of dream consciousness and since dream consciousness can occur outside of REM sleep, then REM sleep physiology and its brain stem control systems are of no relevance to dream theory. In this view, the forebrain is free to act alone and can produce dreams independent of the activating, input-output gating and modulatory influences of the brain stem.

This argument is fatally flawed in three important respects:

1. The first fatal flaw is that there is now half a century of solid extensive evidence from laboratories around the world that the major determinant of the forebrain's neurophysiological state and of its propensity to dream is the set of brain stem core systems that run from the medulla up through the pons and midbrain to the basal forebrain, hypothalamus, and thalamus. Indeed **SOLMS**'s own speculative neurophysiological model of dream-instigating wishes invokes the dopamine system, which projects from the ventral tegmental area of the brain stem to the mesolimbic reward circuit in the ventral striatum, thalamus, and medial prefrontal cortex.

2. The second fatal flaw is that the brain is instantiating the neurophysiological conditions of REM sleep from the very moment of sleep onset and varying them in intensity continuously throughout the night until the moment of awakening. As their strength flows and ebbs, dreaming becomes more or less probable, more or less intense, and more or less emotional. In other words, REM physiology is relevant to dream generation even in so-called NREM sleep. I say "so-called" because we have shown that REMs occur in NREM sleep at an average level about one-third that of REM. So NREM sleep is a misnomer. It doesn't really exist! All of sleep is REM sleep (more or less). See **NIELSEN**'s target article.

3. The third fatal flaw is that REM sleep, by everyone's account, provides the optimal neurophysiological conditions for dreaming. The specific conditions of REM include cholinergic forebrain activation and stimulation, aminergic forebrain demodulation, and active input-output gating. These account for all the formal aspects of dreaming in a more economical manner than Freud's implausible theory.

As far as the notion that it is dopamine alone and not the mix of noradrenergic, serotonergic, and cholinergic neuromodulators that are critical to the propensity to dream, **SOLMS** is grasping at straws and the wrong straws at that. The medial forebrain lesions that lead to cessation of dreaming interrupt all of the neuromodulatory systems (and many other pathways) linking the brain stem and basal forebrain to the cortex. So why single out dopamine? Presumably because it is known to mediate motivation and reward. But how we get from motivation and reward to Freud's unconscious wishes is not at all clear. SOLMS's choice of dopamine is all the more surprising because it is the one neuromodulatory substance that shows no tendency to change its output over the sleep-wake cycle being as active in NREM sleep as it is in REM as it is in waking (See Hobson & Pace-Schott 1999)! So, at best, dopamine could be as necessary to dreaming as it is to any other activated mental state but it could not possibly be either sufficient or specific enough to account for the distinctive aspects of dream consciousness.

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Dreaming as play

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Abstract: Dreaming can provide a marvelous opportunity for the "playful" exploration of dramatic events. But the chance to learn to deal with danger is only a small part of it. More important is the chance to discover what it is like to be the subject of strange but humanly significant mental states.

[REVONSUO]

At a time when theories of dreaming are tending to lose touch with psychological and biological reality, **REVONSUO**'s target article comes as a welcome call for a return to common sense. Dreaming, REVONSUO reminds us, is about having dreams. Dreams tell stories in which the dreamer is an active protagonist. These stories can and often do leave lasting traces on the dreamer's mind. Hence, surely, the way to understand the evolutionary function of dreaming must be to consider the relevance of such stories to the kinds of survival problems that ancient humans had to face.

I have no doubt that this is the right way to go. And, as it happens, in the early 1980s I proposed a theory that is quite similar in spirit to the one described here (Humphrey 1980; 1983; 1986). I began by noting, as **REVONSUO** does implicitly, that there is an obvious analogy between dreaming and childhood play. Dreaming, like play, allows the subject to simulate his or her own participation in dramatic or dangerous events, without suffering the consequences these events would have in the real world. One of the chief functions of play is to provide an opportunity for the player to gain practice in exercising the relevant physical, intellectual and social skills. So, there is every reason to suppose this is a major function of dreaming too.

Now, **REVONSUO** picks up on just one aspect of this: he suggests that the main purpose of dreaming is the simulation of environmental threats, so that the dreamer is able to practice making his or her escape. I've no quarrel with this suggestion so far as it goes (and REVONSUO does make a good case for it). But, as a theory of dreams in general, it strikes me as being far too narrow – with regard to what it says both about the kinds of situation that are simulated and about the kinds of learning that take place.

To continue the analogy with play, even though childhood play does of course often centre around imaginary dangers, it is clearly not the case that learning to escape these dangers is play's main, let alone its only, function. Rather, play contributes in a major way to social and psychological development, especially through providing practice in role-playing and empathy.

> Play is a way of experimenting with possible feelings, possible identities without risking the real biological or social consequences. Cut! time for tea, time to go home – and nothing in the real world has changed, except perhaps that the child is not quite the person that he was before, he has extended just a little further his inner knowledge of what it can feel like to be human. (Humphrey 1986, p. 106)

But if this broad-band "sentimental education," as I have called it, is the functional rationale for play, surely we should expect something like it to be the rationale for dreams as well. In my own writings I've stressed in particular the key role that dreams can have in the education of a "natural psychologist" – through introducing him to introspectively observable mental states that are as yet unfamiliar in real life (and possibly beyond the scope of waking play).

> "Dreaming" represents the most audacious and ingenious of nature's tricks for educating her psychologists. In the freedom of sleep the dreamer can invent extraordinary stories about what is happening to his own person, and so, responding to these happenings as if to the real thing, he can discover new realms of inner experience. If I may speak from my own case, I have in my dreams placed myself in situations that have induced feelings of terror and grief, passion and pleasure, of a kind and intensity I have not known in real life. If I did now experience these feelings in real life, I should recognise them as familiar; more important, if I were to come across someone else undergoing what I went through in the dream, I should have a conceptual basis for modeling his behavior. (Humphrey 1983, p. 85)

Neither is this mere arm-chair theorising. My interviews with people in psychologically-taxing situations have shown again and again that dreaming is indeed a recognised and valued resource for gaining insight into what it is like to be in another person's place. A young midwife, for example, revealed: "I think most midwives dream about giving birth when they start working in maternity units, and it was a fairly common experience among the students that I trained with. . . . I've never myself been pregnant. But my dreams have certainly made me more understanding, more relaxed and more confident in talking to mothers" (quoted in Humphrey 1986).

REVONSUO may object that this is all too rosy. It is all very well for me to point to the ways in which dreams can help with empathy-building and interpersonal understanding, in the relatively secure and sociable world that we now live in. But, for him, the true evolutionary context for dreaming was the harsh world of the Pleistocene, where human life was nasty, brutish, and short – and everyone lived in a constant state of post-traumatic stress.

I would answer that this Hobbesian vision of the EEA is simply much too bleak. Studies of contemporary hunter-gatherers such as the Kalahari Bushmen – those whom Sahlins (1977) has with good reason called "affluent savages" – have shown that, on the whole, their life is (and presumably has long been) remarkably easy, unstressful, and free of danger. In fact the main – if not the only – serious challenges these people face are precisely in the area of their human relationships (family politics, love affairs, status battles, jealousies).

Then why, to end with one of the stronger bits of evidence for **REVONSUO**'s narrow view of what dreams are about, are there so many animal characters in children's dreams? And why, for that matter, so many animals in story books, in the play-room, in Walt Disney cartoons, and so on? What can these animals be doing, if it is not that they represent archaic threats? I believe the truth is that these play-animals are usually just what they seem to the child to be: simple, and indeed highly simplified, proxies for human beings – which, as it happens, are peculiarly well suited to the child's first tentative experiments in empathic projection and in applying a theory of mind. As Levi-Strauss (1962) once put it, animals are "good to think with." But this discussion is for another time.

New multiplicities of dreaming and REMing

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Abstract: The five authors vary in the degree to which the recent neuroscience of the REM state leads them towards multiple dimensions and forms of dreaming consciousness (Hobson et al.; Nielsen; Solms) or toward all-explanatory single factor models (Vertes & Eastman, Revonsuo). The view of the REM state as a prolongation of the orientation response to novelty fits best with the former pluralisms but not the latter monisms.

[HOBSON ET AL.; NIELSEN; REVONSUO; SOLMS; VERTES & EASTMAN]

1. Intimations of pluralism. **HOBSON ET AL.**'s overview of REM dreaming as a state specific organization of consciousness represents an important and exciting expansion of Hobsons's previous attempts to synthesize the neuroscience and phenomenology of dreaming, with the potential for a still greater inclusiveness. Their addition of recent brain imaging and lesion studies showing hypothalamic, limbic, right parietal, and secondary visual activation, to his earlier model of pontine generation allows an expanded view of both a bottom-up and top-down REM state. The result is broadly consistent with Freud's suggestion of a "topographical regression" in dreaming, as well as with views of the

dreaming process that emphasize its basis in emotional activation (Lowy, Kuiken) and in cross-modal visual-spatial elaborations intrinsic to imaginative forms of dream bizarreness (Hillman, Hunt). It is also consistent with a specific phenomenology of a prototypical or background dream bizarreness related to delirium syndromes, with their shifting mix of cognitive confusion or clouding, emotionality, and intrusive, predominantly visual, hallucinosis (see also Hunt 1982; 1989), as well as separating that state specificity of REM dreaming from the more cognitive ruminations of NREM mentation.

HOBSON ET AL. make just the use of Hunt et al. (1993) for which I had originally hoped, showing that controlling dream bizarreness measures for report length, when novel events actually require more words for adequate description, will falsely dilute the defining feature of dream experience. Their organization of states of consciousness in terms of a three-dimensional space based on activation, source of input, and neuromodulation pathway is a significant contribution in its own right, and usefully differentiates prototypical REM and NREM mentation, as well as atypical variations in dreaming and sleep onset, although the number and level of specificity of the dimensions ultimately needed to classify discrete states of consciousness and forms of dreaming remains open (see Hunt 1982; 1989; Pekala 1991 for more phenomenologically-based attempts).

HOBSON ET AL.'s expanded approach might be further enriched by including the fuller implications of Morrison's (1983) demonstration that the positive activation of the REM state, with its cholinergic basis, motoric paralysis, and vestibular activation, is a prolonged form of the orientation or postural still response to novelty in wakefulness – Pavlov's "curiosity reflex." First of all, it suggests that pontine activation need not be considered as "random," but potentially already primed and/or self organized in terms of recent unassimilated novelty from wakefulness (Hunt 1989). The orientation response model is also consistent with less executive and volitional (higher forebrain) control, since a primary alerting and precognitive response is required in novel circumstances – which in our symbolic species will include not only intense emotionality (limbic) but also the recombinatory basis of creative symbolic operations (Geschwind's parietal zone of neocortical convergence). From this perspective the proper comparison is not REM state with generalized wakefulness but REM state with the cholinergic based orientation response to waking novelty, which might contradict the degree of REM state specificity posited by HOBSON ET AL.

Finally, the orientation response, as the fuller physiological context of the REM state, allows a more ready inclusion of nonnormative but widely studied alternative forms of dreaming, not as "dissociations," but as augmentations and intensifications of REM dream cognition. In terms of psycho-physiology this seems clearest in lucid dreaming, which can involve an intensification of phasic REM features and parietal activation, consistent with its fully developed phenomenology of heightened bizarreness, enhanced kinesthetic and vestibular effects, and meditation-like self awareness and visual-spatial phenomena (see Hunt 1989; 1995). A prolonged orientation response to novelty is fully consistent not only with a fluctuating delirium as normative dream bizarreness but also with its potential for reorganization into the more specific forms of novelty "metabolism" found in lucid dreams, the imaginative dreams of such interest to Jung, nightmares, and the creative problem solving dreams documented by numerous scientists, writers, and artists. **NIELSEN**'s term "apex dreaming" captures the way those forms of dreaming actually intensify REM dream features rather than dissociate them and reflect the limbic, vestibular, and parietalspatial activations that **HOBSON ET AL.** now include within their activation-synthesis model.

NIELSEN makes a plausible case that NREM mentation, especially in its more overtly dream-like aspect, involves brief phasic manifestations of the REM state. The question becomes whether this explanation works as well for more thought-like NREM mentation.

There may be a danger here of over-generalizing the specific form of cortical activation associated with the REM state, an acetylcholine mediated, pontine based orientation response (Morrison 1983), with other state specific forms of cortical activation. I have argued (Hunt 1989) that thought-like NREM mentation may reflect a dysfunctional cognitive activation in sleep, based on a defensive vigilant response to the artificialities and stress of the sleep laboratory setting (consistent with **NIELSEN**'s recent finding of EEG power differences with versus without mentation) and as a subjectively disruptive response to depressive or obsessional issues. Certainly when we awaken in home settings having been "thinking all night" about ongoing waking concerns, it is associated with a sense of having slept poorly.

Such dysfunctional sleep rumination reflects a milder version of W. R. Bion's (1962) comment on some schizoid patients, that they suffer from a sense of not being able to dream, fully sleep, or fully awaken. Lairy's research on intermittent sleep, Lairy et al. 1967 cited by **NIELSEN,** may constitute a physiological reflection of this transitional state, but the presence of an EEG like that of the REM state, without other phasic REM indicators, need not indicate the actual REM state, since there is more than one pathway of physiological and biochemical activation of the cortex (**HOBSON ET AL.**). Correlations between thought-like NREM mentation, higher EEG activation, and waking conflict and psychopathology (see Hunt 1989) seem most consistent with a defensive hypervigilence, potentially distinct from the phasic bursts and after-effects of the pontine orientation response associated with more vivid, dream-like experience. NIELSEN, consistent with HOBSON ET AL. rejects a one generator model for all sleep mentation, which would be based on an overly generalized model of cortical activation, but then seems to put forward a one generator model for the physiological basis of NREM mentation. State-specific differences in phenomenology, coupled with the existence of different pathways of physiological activation, might be more consistent with a two generator model for the more thought-like and the more bizarre, dream-like patterns of NREM mentation.

SOLMS's evidence of the loss of dreaming with prefrontal damage, also linked to the loss of interest and affect after leukotomy, and with parietal lesions, also the region posited by Geschwind (1965) as the convergence zone for a cross modal translation capacity central to the symbolic capacity, is invaluable, and indirectly supportive of empirical phenomenologies of dreaming that center on affect and creative recombinatory imagery (bizarreness). However, while consistent with those cognitivists who have long sought to decouple a psychology of dreaming (seen as cortical) from the brain-stem physiology of the REM state, his own separation of the two may be premature.

On the one hand, there is the large literature linking dream bizarreness to pontine and related phasic features of the REM state, with both phenomenology and physiology correlated with levels of creative imagination in wakefulness (see Hunt 1989, for review). Similarly, while prefrontal and parietal mediators of REM dreaming can be artificially separated from brain stem features by lesions, it is important to note that they are normally conjoined in what amounts to a state specific patterning of consciousness in the sense posited by **HOBSON ET AL.**

Certainly, with **SOLMS,** dreaming, in the sense of a form of consciousness based on an attenuation of reflective thought and volition and a heightening of narratively organized imagery, can appear in conditions outside the REM state – as in sleep onset dreaming, NREM mentation, suggested dreams in hypnosis, waking daydreaming, and guided imagery. Although all these forms of "dream" will entail higher cortical processes, there would seem to be important differences in their form, consistent with very different pathways of subcortical and cortical activation. While, in one sense, SOLMS can say that the REM state is but one "arousal trigger" for a single dreaming process, the acetylcholine (and possibly dopamine) based pontine orientation response seems to produce a form of dream experience specific to the REM state (vivid and apex dreaming) and to similarly triggered and patterned altered

states of consciousness in wakefulness (certain hallucinogenic drugs, visionary states). These seem, with **HOBSON ET AL.,** quite differently organized than most daydreaming or thoughtlike NREM mentation.

SOLMS is surely well supported in separating the REM state from a more general cognitive dreaming process, but a further equally supported step would be to distinguish within different forms of dream experience, so that prototypical REM dreaming would be a specific organization of cognitive-affective processes, normally entailing the entire circuit of cortical and subcortical activation that SOLMS has so carefully deconstructed through his neurological cases.

2. Assertions of monism

Vertes & Eastman. Why must the REM state, this exceedingly complex phenomenon of nature, have but one function? VERTES & EASTMAN seem to outline all possible skepticisms about memory consolidation research in order to advance their new version of the endogenous stimulation model also held by some of the pioneers of REM state research (Roffwarg) and more specifically developed by Ephron and Carrington (1966). However, Morrison's (1983) research, showing the REM state to be a specially sustained form of the orientation/still response to novelty, affords a view more consistent with multiple overlapping functions for the REM state (and REM dreaming). These include not only the endogenous stimulation model, but the equally plausible evidence for a separate role in fetal and neonatal maturation, as well as memory consolidation – especially for novel stimulation of high emotional significance.

More specifically, **VERTES & EASTMAN**'s demolition efforts with memory consolidation research seem narrowly conceived. The cortical activation of the pontine based orientation response operates from acetylcholine mediated pathways, distinct from the norepinephrine based activation of volitional executive control (Vanderwolf & Robinson 1981). Accordingly, the appropriate comparison for the mnemonic consequences of the REM state would be to the more recognitive encoding functions associated with emotional novelty and stress in wakefulness, which seems more consistent with the actual thrust of much of the mnemonic REM research reviewed, along with the lack of encoding for less vivid dreaming (coupled with life long recall for especially intense dreams; Knudson & Minier 1999), and a diffusely primed background for assimilating recent waking novelty that need not enter dreaming as such, yet is also consistent with empirical research on some unresolved "day residues" appearing in ordinary dreaming (see Hunt 1989).

Finally, to suggest that the high levels of REM sleep in neonates, still higher in the premature, is an endogenous compensation for slow wave sleep is not consistent with the near universality of the REM state late in gestation, the absence of true slow wave sleep until about three months, and the way that facial expressions in the neonatal REM state can be developmentally ahead of those shown in wakefulness – most consistent with a specific cortical maturation function distinct from the ontogenetically later one of endogenous compensation for NREM sleep (see Hunt 1989).

The REM state would seem to be one of the best illustrations for the economy and complex elegance of the natural order in which multiply nested functions can re-use a more basic organismic capacity.

Revonsuo. If the basic rule in scientific theory is still maximum parsimony given an unbiased consideration of the full range of evidence, along with at least potential testability, then **REVONSUO**'s model of threat simulation as the core of the REM state, based on an adaptation to the allegedly overwhelming threat situations of early Hominid evolution, ignores equally strong evidence for other forms and functions of dreaming and is as ad hoc, "extra," and inherently untestable as Freud's analogous hypothesis of the phylogenetic "primal horde."

Closer to home of what is directly researchable, threat simulation or nightmare dreaming does also follow as one major form of dreaming, especially prominent under major organismic stress, from Morrison's (1983) demonstration that the physiology of the REM state is identical to that of the orientation/still response to waking novelty. The orientation response as substrate is certainly consistent with nightmares as the predominant form of dreaming under system overload and with anxiety as the predominant dream affect, but it is equally consistent with other forms of dreaming such as lucidity, creative-imaginative dreaming, and the problem solving dreams attested by scientists, inventors, artists, musicians, and writers (see Hunt 1989). Not all novelty is stressful.

To consider only the most skeptical views on the existence of other dream forms, without a similarly close look at one's own all-explanatory model, bypasses the recent tendency of research in both the physiology and psychology of dreaming towards a new pluralism. Empirically then, **REVONSUO**'s use of anthropological evidence from contemporary hunter-gatherers is especially questionable, since in addition to "threat simulation," the literature here attests to the diverse forms of dreaming described in these complexly "dream centered" societies, including lucid, mythological-archetypal, social and personal problem solving, artistic creativity, and diagnosis and healing of illness (Hunt 1989; Tedlock 1987b).

On more theoretical grounds, if the most direct indication we have today of evolutionary threat simulation dreams are post-traumatic and stress based anxiety dreams, then it is difficult to see how our widely described paralyzed fears, slow motion running, and escape tactics based on absurd reasoning could be a rehearsal or simulation of anything adaptive. Pushing the origins of the model back into mammalian evolution seems even more questionable in terms of testability. However, on analogy to his use of hunter-gatherer evidence, my own domestic dogs have not only shown the expected growling and barking in their REM states, but also movements of drinking, sexuality, and tail wagging. Rapid paw movements (RPMs) can of course be as consistent with chase and play as with flight.

The view that nightmares and stress dreams show the essence of all dreaming is like saying that the underlying purpose of the vestibular system, with its compensatory eye movements restoring postural balance, is nausea and vomiting, because that is what happens when the system is overloaded in extreme dizziness. While vomiting is the extreme response, the predominant functioning of the vestibular system is to restore equilibrium as rapidly as possible. Helpless vomiting is no more "adaptive" to a vertigo sufferer climbing a tree than is the confused, panicked running we can do in our nightmares. Vestibular response, like REM sleep, is a complex multi-layered organismic process that can a final common pathway for potentially very different functions.

Empirically, it seems more and more that there is no single essence or core function to either dreaming or the REM state. They are complex and multiple, though with every possibility of our tracing the interrelations and interactions of their several dimensions once that complementarity is accepted.

The interpretation of physiology

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Abstract: Not at all self-evident, the so-called isomorphisms between the phenomenology and physiology of dreams have been interpreted by Hobson et al. in an arbitrary manner to state that dreams are stimulated by chaotic brainstem stimulation (an assumption also adopted by Vertes & Eastman). I argue that this stimulation is not chaotic at all; nor does it occur in the absence of control from the cerebral cortex, which contributes complexity to brainstem activity as well as meaningful information worth consolidating in the brain during sleep.
[HOBSON ET AL.; VERTES & EASTMAN]

HOBSON ET AL.'s presentation of the isomorphism between REM sleep physiology and dreaming, has over interpreted the physiology. The physiology does not disprove any more than it proves, either Freud's theories (as put forward in *The interpretation of dreams*, 1900) or other more recent interpretations, to the effect that dreams originate in the cortex and contain highly meaningful information. I take issue with two fundamental points.

1. Chaos reigns in the brainstem and determines the dream. I do not know of any physiological evidence that the brainstem activation and stimulation of the forebrain is chaotic and would thus impose a chaotic influence on the cortex in dreams. On the contrary, circuits within the brainstem, as in the spinal cord, are highly ordered, so that specific motor patterns such as locomotion, chewing and vestibulo-ocular nystagmus, are generated there in a repetitive, rhythmic, and highly predictable manner. Complex organized behaviors are also generated in the brainstem, including sexual and rage behaviors that persist in decerebrate animals. Indeed, these are the very behaviors that are often unmasked in REM sleep without atonia and dreams. In addition to being highly organized behaviors, they are also instinctual and highly motivational, perhaps stimulating the wishes that emerge in dreams (see Jones 1991).

2. The cortex has no control over the brainstem and the dream. I do not know of any physiological evidence that the cortex has no control over the brainstem or over the central activities of dreams. On the contrary, corticofugal outputs reach the entire brainstem as well as the spinal cord, influencing the very neurons shown to be critical for the initiation and maintenance of REM sleep in the pontine reticular formation. Moreover, elimination of the corticofugal impulses by lesions of the cortex in cats were shown in Jouvet's laboratory to result in a complete impoverishment of both rapid eye movements and PGO spikes, reducing them to the very stereotypic, highly ordered and hence low-ininformation-content pattern of purely brainstem driven activity (Jouvet 1975). The cortex thus appears both to control and introduce complexity to the brainstem activity and undoubtedly to dreams, which can accordingly also contain highly meaningful information.

Indeed, I might posit that the physiology of REM sleep provides considerable support for Freud's basic assumptions, according to which instinctual and highly motivational impulses arise from the brainstem and are in turn worked upon by the cortex where condensation, displacement, and symbol formation may control the continued activity of the brainstem and provide the complex and seemingly bizarre, though meaningful, content of dreams (Freud 1900).

VERTES & EASTMAN provide overwhelming evidence against memory consolidation in REM sleep. They also go on to provide convincing evidence for a critical role of theta in memory consolidation during waking. Theta occurs during active and attentive waking but also (and in the most robust, continuous way) during REM sleep. Yet these authors interpret this physiology differently during REM and waking, presenting theta as a mere epiphenomenon in REM sleep, because it would have no functional value occurring in association with the chaotic state stimulated by the brainstem in REM sleep. Either theta plays no role in memory consolidation or it does so in REM, as it does in waking.

I would agree with Winson (1990) that theta and REM are associated with species-specific highly motivational and orderly instinctive behaviors and underlying processes that are important for survival (see Jones 1998). In this framework, theta during REM, as during waking, is associated with maximizing, reinforcing, and potentiating the neural links underlying these behaviors as well as with reforming them in relation to the organism's changing world over a lifetime. Evidence to date has indicated that REM sleep is involved in consolidating procedural learning, a process that does not require or usually involve conscious awareness, (e.g., in walking, running or skiing). Such procedural learning can be highly practiced, with more and more efficiency, hence rapid responses and behaviors.

REM sleep in the fetus may provide procedural learning and preparation prior to birth for performing important behaviors, including locomotion and flight, by exercising the specific circuits. Such may be the case in the wildebeest, who begins to walk and then to run minutes after emerging from the womb in the dry season in order to escape predation and to begin a long migration of several hundred miles with its mother in search of a water supply. REM sleep during that migration may then facilitate the learning of speedy adjustments to changing terrain and rapid escape from real predators. Theta would be present during the waking experience and the dreamed replay to consolidate and potentiate the new sensory-motor associations and reactions with the old.

Such a process might also help in the learning and honing of nonessential skills by humans, such as more smoothly and rapidly negotiating the mogulled terrain on the second day down the ski slope following practice during dreaming through the intervening night. The importance of REM sleep for such highly practiced and unconscious processes and skills might be very difficult to document in the laboratory. Yet, as discussed by **VERTES & EASTMAN,** a few studies have recently shown effects of REM sleep on consolidation of procedural tasks. As in these experiments, the challenge has been to devise experiments and measures that are sensitive enough to reveal and confirm the full role of REM sleep in these processes. This role, though it may seem inessential, may provide a considerable advantage to the organism in strengthening associations and perfecting behaviors that have been important for the survival of each species in its evolutionary past.

The "problem" of dreaming in NREM sleep continues to challenge reductionist (two generator) models of dream generation

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Abstract: The "problem" of dreaming in NREM sleep continues to challenge models that propose a causal relationship between REM mechanisms and the psychological features of dreaming. I suggest that, ultimately, efforts to identify correspondences among *multiple* levels of analysis will be more productive for dream theory than attempts to reduce dreaming to *any* one level of analysis.
[HOBSON ET AL. ; NIELSEN]

NIELSON's "position," and a core issue in the debate between 1-gen and 2-gen theorists, turns on whether the differences in REM and NREM dreams are essentially *quantitative* (i.e., "relative"), or *qualitative* (i.e., "absolute"). The basic logic is that if REM and NREM dreams differ quantitatively, then the same qualities should describe both REM and NREM dreams, but to different degrees; this situation is consistent with 1-gen models (also see Foulkes & Cavallero 1993, p. 10). However, if REM and NREM dreams differ qualitatively, in that the defining qualities which characterize REM dreams (e.g., emotionality, vividness, bizarreness) do not characterize NREM dreams, then a 2-gen model is needed to explain these "absolute" differences (e.g., Hobson 1988b; Koukkou & Lehmann 1983).

It is notable that the debate between 2-gen and 1-gen theorists about the proposed differences between REM and NREM dreaming, as well as the extent to which the psychological experience of dreaming can be explained by neurophysiological mechanisms, is mirrored in the debate about whether sleep cognition is "discontinuous" or "continuous" with waking cognition (i.e., 1-gen model); (for recent discussions of this essentially parallel debate, see Purcell et al. 1993). For example, recent efforts to compare the incidence of high-order cognitive skills, such as self-reflection, decision making, and meta-attention, across REM dreaming and waking suggest that although these skills may be more character-

istic of waking than dreaming, these metacognitive skills are, nevertheless, frequently associated with the recall of dream experiences (see, especially, Kahan & LaBerge 1996; Kahan et al. 1997). If one assumes a priori that dreaming involves, for example, a suspension of self-reflection (e.g., Hobson 1988b; Koukkou & Lehmann 1983), then one is not inclined to actively test this hypothesis by comparing the incidence of such skills across sleep and waking (also see Kahan 1994; Kahan & LaBerge 1994; Purcell et al. 1993 for discussion of this issue). In fact, many of the "discontinuity" theorists would claim that differences in cognition across waking and sleep are the result of the same physiological/psychological isomorphism that explains the differences in cognition across REM and NREM sleep (see **HOBSON ET AL.**).

In his balanced review of the evidence for 2-gen versus 1-gen models of dream generation, **NIELSEN** discusses the long and controversial research lineage which, ultimately, shows that REM is *not* required for vivid dreaming to occur, although vivid (and "apex") dreaming may occur *more often* in REM sleep. NIELSEN discusses a number of studies, some of them his own, which demonstrate that Stage 1 sleep at sleep onset, late-night Stage 2, and occasionally Stage 3/4 sleep, are associated with vivid dreaming that *cannot* be distinguished from vivid REM dreaming. Even **HOBSON ET AL.**, the consummate "2-gen" theorists, explicitly noted this fact when describing the early dream research: "Reports *qualitatively* indistinguishable from dreams were obtained from Stage 1 sleep at sleep onset, a phase of sleep without sustained eye movements . . . and some of the reports from non-REM sleep were indistinguishable *by any criterion* from those obtained from post-REM awakenings" (1988b, pp. 142–43, emphasis added). NIELSON also acknowledges the recent and persuasive neuropsychological evidence that shows an absence of dreaming even though REM sleep is intact (Solms 1997a). In other words, REM is neither necessary nor sufficient for vivid dreaming to occur (Cavallero & Cicogna 1993; FouIkes 1985; 1993c; Solms 1997a; this volume). This body of evidence is clearly contrary to the reductionist, 2-gen models (e.g., HOBSON ET AL. target article).

In light of his discussion of the dissociability of REM and dreaming, it is rather curious that **NIELSEN** ultimately concludes that the evidence supports a 2-gen model because of "residual *qualitative* differences" across REM and NREM dreams (sect. 2.8.4, emphasis added). Unquestionably, vivid dreaming occurs more often during REM than NREM sleep. Also, the reported incidence and qualities of dreaming sampled from NREM sleep is more unstable, in that the similarity between NREM and REM dreaming is influenced by the sleep stage and time of night when mentation is sampled (Kondo et al. 1989), by whether the participants are light/heavy sleepers (Zimmerman 1970) or high/low dream-recallers (Moffitt et al. 1972), and even by the theoretical predisposition of the investigators (Hermann et al. 1978). Critically, however, and as **NIELSEN** aptly points out, the reported frequency of NREM dreaming is intimately tied to how dreams are defined. As yet, there is no consensus among dream theorists on the formal features that define dreaming, and this seriously complicates the task of comparing across studies of purported sleep-stage differences in dreaming. It is not unusual for investigators to provide their own preferred list of the defining features and then proceed to compare REM and NREM mentation on these dimensions (e.g., Hobson 1988b; Koukkou & Lehmann 1983). Clearly, *which* qualities are considered the defining (or "formal") characteristics are often theoretically motivated and drive the comparison between REM and NREM dreaming, as well as, for that matter, comparisons between dreaming and waking cognition.

This question of what features are necessary to judge sleep mentation as a "dream" is central not only to comparisons of dreaming across different sleep stages, but also to theories claiming a formal isomorphism between sleep neurophysiology and the "formal" features of dreaming (e.g., **HOBSON ET AL.,** this volume; Koukkou & Lehmann 1983). If particular features of dreaming (e.g., bizarreness, emotionality, self-representation) are assumed to be *determined by* particular REM mechanisms, then agreement is needed on just what features of dreaming are the consequence of REM neurophysiology. Further, if a formal isomorphism exists between REM and these dream features, then NREM mentation should not evidence these features; in other words, mentation during REM and NREM should differ in *kind* (i.e., qualitatively) rather than simply in *amount* (i.e., quantitatively)!

Unfortunately, **NIELSEN**'s efforts to "reconcile" the 1-gen and 2-gen models by adding to a 1-gen model the assumption of physiological isomorphism only complicates matters. The "one" system that NIELSEN proposes is the REM "system." His hypothesis is that variations in the incidence of dreaming occur in direct proportion to the involvement of REM mechanisms; whether "overt" in unequivocal REM sleep or "covert" in NREM sleep that seems to "mix" in some features of REM. NREM dreaming, then, is due to a (covert) carry-over of REM features into NREM sleep. However, I found this logic suspect; if a *subset* of REM mechanisms is observed in NREM sleep (a "mixed state"), and these REM mechanisms are responsible for NREM dreaming, then doesn't this first call into question the entire enterprise of sleep staging and, second, precisely *which* REM mechanisms are "responsible" for dreaming? NIELSEN does offer persuasive evidence that sleep stages are not always as discriminable as the literature sometimes implies; rather, sleep staging can be difficult and sometimes results in "mixed" states not clearly identifiable as REM or NREM. NIELSON's "mixed states" model could account for some of the variability in the purported frequency of dream recall from NREM sleep, but this variability could also be accounted for by the sampling and individual differences factors mentioned earlier. More importantly, NIELSEN's model does not offer a full accounting of the proposed *qualitative* differences that he considers pivotal to supporting 2-gen over 1-gen models. It is noteworthy that **HOBSON ET AL.** (this issue) offer a similar argument about lucid dreaming; that it occurs in a REM state that "mixes" in waking. However, Stephen LaBerge's work is contrary to this claim; he and his colleagues found that lucid dreaming tends to be associated with *intensified* REM, in which there is heightened brain activation and heightened muscle suppression (of the H-reflex)(Brylowski et al. 1989; LaBerge 1990).

Throughout this *BBS* special issue, an implicit question is: at what "level" dreaming is to be investigated and, eventually, explained: psychological (in terms of the cognitive skills of dreaming and the psychological or developmental conditions under which these skills manifest); neuropsychological (in terms of the consequences of brain damage for dreaming and other complex cognition); or neurobiological or evolutionary (in terms of the neurobiological evolutionary mechanisms that subserve the construction of complex cognitive experiences). Ultimately, we will need all of these – and other – levels of analysis to adequately account for what Harry Hunt called the "multiplicity" of dreaming (Hunt 1989).

At this point, it seems most constructive to consider all of the correlates of dreaming (and waking) cognition. Certainly, our neurophysiology and developmental stage constrain our dreaming and other cognitive experiences, but it seems to me it is the potential of the human cognitive system to generate *meaning* and to represent the self-in-world (e.g., Globus 1987) that makes "dreaming" possible. We thus need to understand the phenomenology of dreaming experience and, thus, must be willing to admit as data first-person, self-reports that describe not only the narrative of the dream events, but also the qualitative aspects and the personal or transpersonal meanings ascribed to that experience. In addition, we need to understand the psychological circumstances associated with dreaming, including the cognitive, developmental, motivational, and cultural conditions. And, we need to understand the neurobiological correlates of dreaming as well as what happens to dreaming (and its component cognitive skills such as memory, attention, imagery, language, organization, consciousness) when the brain is damaged. The puzzle of dreaming – how dreams are generated, why we dream, and how dreaming experience is related to waking experience – is complex and multidimensional, rather like a 3-D chess game; no one level of analy-

sis is sufficient to solve the entire puzzle and all levels are inter-related. To attempt to reduce the multiplicity of dreaming to one "ultimate" level of analysis, whether neurobiological or phenomenological or psychological is to miss crucial puzzle pieces provided by the other levels of analysis and, hence, never to solve the puzzle and understand its intricacies.

A new approach for explaining dreaming and REM sleep mechanisms

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Abstract: The following review summarizes and examines Mark Solms's article Dreaming and REM Sleep are controlled by different brain mechanisms, which argues why the understanding of REM sleep as the physiological equivalent of dreaming needs to be re-analyzed. An analysis of Solms's article demonstrates that he makes a convincing argument against the paradigmatic activation-synthesis model proposed by Hobson and McCarley and provides provocative evidence to support his claim that REM and dreaming are dissociable states. In addition, to situate Solms's findings in concurrent research, other studies are mentioned that are further elucidated by his argument.
[SOLMS]

SOLMS argues against the activation-synthesis model proposed by Hobson and McCarley. Refuting their theory that dream imagery is stimulated from the pontine brainstem, SOLMS claims that REM and dreaming are dissociable states controlled by different brain mechanisms. According to SOLMS, there is a separate forebrain mechanism responsible for turning on dreaming aside from that of the "REM-on" mechanism. Throughout the paper SOLMS cites many studies, including his own, as counter evidence to the Hobson and McCarley model. For instance, in section four, SOLMS refers to Vogel's "An alternative view of the neurobiology of dreaming." Vogel's paper is a direct response against Hobson's and McCarley's non-Freudian theory of dreaming, the activation-synthesis model. In his paper Vogel states that "all the evidence which modern neurophysiology can provide does not and cannot refute the Freudian dream hypothesis" (Vogel 1978a, p. 1531). In terms of Freudian scholarship, SOLMS himself has compiled several of Freud's articles, and has also published *The neuropsychology of dreams: A clinico-anatomical study,* which he frequently cites in his article (Solms 1997a).

In a well-organized format, **SOLMS** directs the reader through his developing argument. In his abstract, SOLMS's statement that the present assumption that REM sleep and dreaming are "physiologically synonymous" is false and in need of revision. Although the forebrain dopaminergic mechanism induces dreaming, the part of the brainstem controlling REM is only one of many activation pathways. According to SOLMS, the assumption that dreaming is an "epiphenomenon of REM sleep" was based on the finding that most dreaming occurs in REM sleep. This observation led to the assumption of dreaming and REM sleep being associable states; a conception that SOLMS intends to prove is incorrect.

Writing in a concise and logical manner, **SOLMS** develops his argument by reviewing past models and theories, and then suggesting new evidence, which challenges previous understandings and theoretical conclusions. For the past two decades Hobson and McCarley's reciprocal interaction model that states "cholinergic brainstem mechanisms cause REM sleep and dreaming" has been accepted by the neurobiology community at large. However, the hypothesis relies on the presupposition that REM sleep is not controlled by forebrain mechanisms, a point which proves essential to SOLMS's main argument, especially in section 6 where he discusses how forebrain lesions cause dreaming cessation and yet "spare the brainstem." Discussion in section 4 is dedicated towards presenting contradictory evidence that "dreaming is generated by the unique physiology of the REM state." SOLMS's suspicion of the activation-synthesis model is evident in section 3 when he states that Hobson's latest developments of the model are "admittedly speculative." Furthermore, SOLMS's confusion stems from the virtually unrecognized discovery that not all dreams are produced by the same brainstem mechanisms as REM sleep. SOLMS believes that the lack of acknowledgment of the latter is due to Hobson modifying his model. Although Hobson disclaims the previous physiological link between REM sleep and dreaming, he maintains an anatomical link between the brainstem and dreaming, a postulate, which, SOLMS feels, confirms the false conception of REM sleep and dreaming as associable states.

Further evidence suggesting that REM sleep and dreaming are controlled by different mechanisms is provided in section 4 which gives examples of when dreaming occurs in NREM sleep, and in section 5, which discusses research that demonstrates dreaming is unaffected by brainstem lesions. Section 5 however, as **SOLMS** points out, has unconvincing data because brainstem lesions large enough to "obliterate REM usually render the patient unconscious," a fact also mentioned by Hobson et al. (1998b). Consequently, it is unlikely that information refuting a correlation between dreaming and REM brainstem mechanism can be obtained using lesion data, a problem that SOLMS solves in section 6 by presenting evidence of forebrain lesions that eliminate dreaming without affecting the pontine brainstem. Citing a long list of references supporting his claim, SOLMS mentions that 108 cases have demonstrated a correlation between dreaming cessation and forebrain lesions confirmed by research using the REM awakening method and morning recall questions. In addition, the lesion site is precisely the same region as targeted in modified (orbitomesial) prefrontal leukotomy, a surgical process that results in complete or nearly complete loss of dreaming in 70–90% of recorded cases.

In section 7, **SOLMS** continues to argue that dreaming is actively generated by a forebrain mechanism. He states that the target zone of prefrontal leukotomy is the white matter of a ventral mesial quadrant of the frontal lobe. Transection of this area causes lack of initiative, or adynamia, reduced imagination or ability to plan ahead, and reduces positive symptoms of schizophrenia. In answer to the question of how any of this relates to dreaming, SOLMS cites three observations: features of schizophrenia have long been equated with dreaming, adynamia is a typical correlate of loss of dreaming, and the chemical activation of this circuit stimulates excessive, vivid, and unusually frequent dreaming. The chemical activation does not affect the intensity, duration or frequency of REM sleep, whereas drugs blocking the circuit eliminate excessive, vivid, and particularly unusual dreams. Further evidence is also mentioned relating nocturnal seizures to dreaming, events associated with the forebrain independently of REM sleep. SOLMS presents interesting findings that definitely support a fresh hypothesis that is still relatively speculative and in need of more evidence. In section 8, SOLMS returns to the activation-synthesis model and its explanation of dream imagery as a product of passive forebrain synthesis of brainstem impulses. SOLMS argues against the postulate by citing clinicoanatomical studies performed by himself and Braun et al. (1997). These findings suggest dreaming is caused by specific forebrain mechanisms, therefore the activation-synthesis model is incorrect because dreaming is not "isomorphically correlated with non-specific brainstem activation of perceptual and motor cortex during REM sleep." However, SOLMS does point out a disparity in the results between Braun et al.'s new functional imagery and his own clinicoanatomical data. In some functional imaging studies there is an involvement of the pontine brainstem during dreaming (Braun et al. 1997; 1998). SOLMS attributes the fact that "REM sleep was equated with dreaming in the imaging studies," owing to the focus on a comparison between dreaming and non-dreaming in NREM only. Hence, SOLMS feels the inclusion in some of the functional imagery studies of the pontine brainstem in dreaming accounts for the disparity between the imagery studies and the clinicoanatomical data.

Although **SOLMS** presents interesting findings that definitely support his relatively fresh hypothesis, his argument is still fairly speculative and in need of more subsidiary evidence. In his favor, SOLMS does suggest a further study to increase understanding: examining the dreaming brain during sleep onset, or during awakening. Most importantly, SOLMS's article represents an alternative argument to the long accepted activation-synthesis model. Thus, SOLMS reminds his readers that we must constantly question and re-evaluate our knowledge and challenge our theories. Models by definition are simply representations and are liable to change and constant improvement. In effect, SOLMS's article not only presents provocative information but it also encourages the reader to invite new ideas and consider changing what we presently accept as the truth. In considering SOLMS's cogent argument, other findings related to dreaming, REM, and brain mechanisms are further elucidated. There are three studies concerned with sleep, which can be related to dreaming and REM as dissociable states. First, the review by Razmy and Shapiro (2000) discusses how dreams appear to occur outside of sleep in Parkinson's disease patients. The paper states that although less time was spent in sleep, "48% of PD patients presented altered dream phenomenon, while 26% displayed hallucinations" (Razmy and Shapiro 2000, p. 5). The distinction between dreaming and sleep in PD patients supports SOLMS's claim that dreaming and REM can function independently. Second, a paper by McCarley and Sinton (2000) suggests there are different types of sleepiness in REM versus NREM. Moreover, efforts are being made to develop clearer neuropsychiatric elements of fatigue, "the possibility of multiple forms of fatigue being identified and defined seems likely" (Shapiro 1998). The plausible inference that different neurological circuits account for the distinctions between REM and NREM sleepiness, and different types of fatigue, makes it easier to conceptualize REM and dreaming as separate states.

A third clinical study was carried out on the airway resistance in adult asthmatics and controls in REM and non-REM sleep demonstrated that the "REM (dreaming) stage of sleep may be associated with bronchoconstriction" (Shapiro et al. 1986). In view of these findings, which suggest REM sleep "has a direct effect" on airway tone, and the observation that the content of the dream was significant in this regard, the existence of independent pathways and brain mechanisms performing various functions is conceivable.

Reference to these studies in relation to the differentiation between REM and dreaming as separate functioning states is both informative and enlightening. We anticipate that future investigations may further substantiate **SOLMS**'s argument and provide greater impetus to clarifying the neurology of dreaming.

Dreaming has content and meaning not just form

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Abstract: The biological theories of dreaming provide no explanation for the transduction from neuronal discharge to dreaming or waking consciousness. They cannot account for the variability in dream content between individuals or within individuals. Mind-brain isomorphism is poorly supported, as is dreaming's link to REM sleep. Biological theories of dreaming do not provide a function for dreaming nor a meaning for dreams. Evolutionary views of dreaming do not relate dream content to the current concerns of the dreamer and using the nightmare as the paradigm dream minimizes the impact of poor sleep on adaptations.
[HOBSON ET AL.; NIELSEN; REVONSUO; SOLMS]

The current debates in the study of dreaming are reviewed from one perspective in this interesting series of articles. The focus is on the form and not the content of the dream with only one exception. The explanations for the form of the dream are biological rather than psychological. The explanations are essentially of a physiologically reductive nature and, in one case, in the form of an extreme brain-mind isomorphism. If a function for the dream experience is entertained, it is a biological or evolutionary one rather than a current concern or immediate psychological one. The possibility that the dream has meaning is either not addressed or rejected.

There is much to applaud and even more with which to agree in these highly informative essays, but the best use, I believe, that I can make of the space available to me is to focus, so to speak, on the other side of the debate and to raise question about various points raised in these reports.

HOBSON ET AL. present a magisterial integration of evidence attempting to explain the neuroscience basis for conscious states; based on demonstrating that waking, REM, and non-REM are physiologically different and that dreaming is REM based. As they appropriately point out, they do not address the "hard problem" of how consciousness could arise from a neuronal system. It is in this transduction that the explanation for consciousness lies. It is this crucial step that McGuinn (1999) believes is unlikely to be solved. A correlation, even if it could be shown between brain state and mental state, would not be an explanation. How does one go from neurons firing to the dream experience of being attacked, and what are the mechanisms to achieve this transformation? This crucial step is missing and seriously undermines the attempt to explain the mind in terms of the brain and even further raises questions about mind-brain isomorphism, even if it could be demonstrated.

There certainly are problems with trying to capture an experience such as dreaming in a verbal report, particularly as the experience occurs during one state and is reported in another (Winget & Kramer 1979). At this point in time, is there a meaningful substitute? That a picture maybe worth a thousand words does not necessarily negate the value of even a 7-word dream report. Certainly, picture drawing has its own limitations. The demand characteristics of probing for additional dream content adds another layer of problems to establishing what a dream experience was "really like." Arguing by analogy that if the physiology is the same in the adult and the infant, the experience must be the same is questionable. Because the authors can imagine the infant experiencing hallucinated emotions and fictive kinesthetic sensations does not demonstrate the relationship, it postulates the answer instead.

Does changing the setting by collecting dreams at home somehow move us closer to the " natural" dream experience? Granted we (Piccione et al. 1976) found little evidence of adaptation to the laboratory over 20 nights of dream collection from each REM period, the study, however, illustrates that extended laboratory studies are indeed feasible, have been done, and provide a wide ranging sample of the reported dream experience. The question remains whether evidence of the experimental situation appears in the dream collection experience at home? And if it does initially, does it adapt out over time? **HOBSON ET AL.** are of the opinion that state specific changes in brain function REM to non-REM virtually guarantee concomitant changes in mental functioning, and that the difficulty in demonstrating such changes is because of the inadequacy of our psychological measurements to identify the changes. The conclusion that the mind and brain have nothing to do with each other they find unacceptable. In searching for an explanation for aspects of the dream experience in a psychological framework, it does not follow that one has to deny a relationship between mind and brain, only that it may not be useful for a particular task.

The so-called inadequate psychological methodologies are more predictive night to night than are the more adequate, by implication, physiological ones (Kramer & Roth 1979). And the dream reports of a given night are related one to another so that one night's dreams are distinguishable from another (Kramer et al. 1976) which has not been shown for the physiological processes REM and non-REM). Conclusions are not a priori acceptable or unac-

ceptable, but might better be based on the evidence available. It is not that mind and brain have nothing to do with each other but rather that the one to one correspondence may not be present and to assume it without evidence begs the question.

That the differences between REM and non-REM are not absolute raises serious question about the centrality of the state differences and the hypothetical mind-brain isomorphism. If the answer is that the significant aspects of the REM non-REM difference are not contained within the boundaries of their current definitions for example, PGO (ponto-geniculate occipital) waves in non-REM precede REM and continue after REM into non-REM and this then accounts for dream content in non-REM sleep. Then the shortcoming in demonstrating isomorphism is limited to the physiologic measurements. Foulkes may have early on attempted to show mind-brain isomorphism (Molinari & Foulkes 1969) but he was unable to replicate the work (Foulkes & Pope 1973). His efforts offer little or no support to the isomorphic position.

HOBSON ET AL. are surprised that direct representations of pre-sleep experiences are not incorporated into the dream. The expectation is that such representation should occur. They see the same being true even for emotionally salient events not being incorporated. We found, as noted above (Piccione et al. 1976), that references to the laboratory continue to appear in the dream reports over 20 nights without much of an overall decrease. We found a significant relationship between the content of verbal samples before and after sleep and the, content of the intervening night's dreams (Kramer et al. 1981). We found that the interpersonal situation in which the dream was experienced and reported influenced the content of the dream report (Whitman et al. 1963a; 1963b; Fox et al. 1968). Pre-sleep themes are connected to the dreams that follow and the night's dreams are connected to the themes obtained after waking in the morning (Kramer et al. 1982). Traumatic experiences are reflected in nightmares but not as simple reproductions (Kramer et al. 1984). As memory is constructive rather than a reproductive enterprise, the expectation that waking events will be simply reproduced in dreams may be unrealistic.

It is unfortunate that a work that Freud never published and never wished to have published (a project for a scientific psychology; Freud 1895) is used as a source for his scientific thinking. Freud never declared brain science off limits as is reflected in *The interpretation of dreams* (Freud 1900/1955) in his discussing the stimuli and sources of dreams:

> Even when investigation shows that the primary exciting cause of a phenomena is psychical, deeper research will one day trace the path further and discover an organic basis for the mental event. But if at the moment we cannot see beyond the mental, that is no reason for denying its existence" (pp. 41–42).

The position that the biology sets limits on the psychology needs to be expanded so the psychology sets limits on the biology as the physiological reductionist is trying to explain a psychological experience. They need to account not just for the formal aspects of dreaming but the content as well. Whether the subject of the hallucination is an animal or my father needs to be explained.

The critique that **HOBSON ET AL.** provide of lesion and functional neuroimaging studies is most helpful. It serves to dampen the view that these studies provide the answer to understanding dreaming. The recognition that subjective states, emotion, may be the primary shaper of dream plot lines links their work to content focused dream studies (Kramer 1993). It may not be the emotion in the dream, but rather the emotional state prior to sleep which relates to the content of the night's dreams. Waking mood is related to the content of dreams, not to the latency to REM or the amount of REM sleep (Kramer et al. 1972). The non-REM aspects of sleep are related to the sleepy aspects of subjective state and the unhappy aspect of mood is related most particularly to the content of the dream.

Brain state modulation by various neurotransmitters contributes significantly to our understanding of REM-non-REM waking differences, but changes in norepinephrine, serotonin, and acetylcholine have not been linked to the experience of dreaming and their role in dream formation is not universally accepted (see target article by **SOLMS** who focuses on the role of dopamine in dreaming). The excessive emphasis on activation from the brain stem in earlier versions of the activation-synthesis hypothesis neglected the role of the higher centers for example, the cortex, in dream formation. The recognition that dreaming is not just a bottom-up process but has a top-down component was, I believe, a necessary corrective. The recall of dreaming is impaired by brain damage and aging in which the index of the damage is a gross measure of impairment (Kramer et al. 1975). Recall of night-reported dreams the next day follows the rules of classical memory theory; primacy, recency, and dramatic intensity are predictive of recallability (Trinder & Kramer 1975).

In their current version of the activation-synthesis hypothesis the brain fits the image to the affect. We have suggested that in the activated state the brain-mind responds to the pre-sleep affective state with an emotionally determined image (Kramer 1993). It is this view that is closest to this version of the activation-synthesis hypothesis. As dream emotion is not an inevitable accompaniment of dream reports (Strauch & Meier 1996), it is unlikely that dream emotion is the shaper of dream plots however the pre-sleep subjective state may be the shaper. The emotional numbing in dreaming fits well with the Freudian idea of the dream as an emotional dampener in its sleep protecting function. Patients who have had an insult to the brain and report no longer dreaming also report poorer sleep (Solms 1997a) and to my view of the dream as a selective affective regulator (Kramer 1993). The consequence of the dream experience is emotional numbing and not the cause of the experience or the dream plot.

In accounting for the formal features of the dream experience, **HOBSON ET AL.** neither address nor do they provide a way of understanding the gender, age, race, social status and marital status, differences in dream content (Kramer et al. 1971; Winget et al. 1972; Winget & Kramer 1979) or the dream content differences in various psychopathologic conditions (Kramer 2000). These differences are granted content but not form differences, and biologic approaches have not been invoked to account for these differences. The recognition of the need for a top-down aspect for the activation-synthesis hypothesis and recognizing the role of emotion in dreaming provides an overlap that may serve as a link between biological and psychological approaches to dreaming.

The burden remains on investigators, like **HOBSON ET AL.** to demonstrate the isomorphism they postulate and provide the transduction mechanisms that translate neural activity into dream consciousness. An advance in techniques and in conceptualizations of a high order will have to take place first. Whether the suggestion that REM sleep as currently defined needs to be redone in order to account for dreaming outside of REM as **NIELSEN** suggests remains to be tested. Without a marker for PGO waves in the intact human this may not yet be possible. The scanning hypothesis, as they note, remains an open question.

The AIM model calls attention to the three interacting aspects of the theory, activation, input, and modulation. These are the three aspects of the biology of dreaming that they feel are necessary to understand the dreaming process. The visual representation of AIM is interesting but I have trouble using it in a predictively testable manner, particularly as it does not deal with the content of the dream or the meaning of the dream. The model is used post-dictively and the multiplication of concepts that occurs when aspects of the model need to be split to fit the data weakens its possible explanatory power.

SOLMS's elegant clinicoanatomical analysis of the relationship between REM sleep and dreaming, the hypothesis central to the activation synthesis theory of dreaming, points out that REM can occur without dreaming, and dreaming can occur without REM. He rejects the idea that dream imagery is isomorphically the consequence of activation of the perceptual and motor cortex. Dreaming is a response to activation, but not specifically from the brain stem; patients with forebrain lesions that spare the brain

stem report the cessation of dreaming but continue to have REM sleep.

In addition to delineating the brain areas needed to generate dreaming, **SOLMS** is trying to explain the formal characteristics of dreaming from a biologic framework, but he does not deal with the specific content of dreams. Dreaming is the consequence of various forms of cerebral activation and occurs only if the activation engages the dopaminergic circuits of the ventromedial forebrain. He makes no statement as to the content of dreams or the function of dreaming either psychological or biological, although he does note that those who no longer report dreaming do not sleep as well as those who do. His contribution is a central critique of the REM = Dream equation and an elaboration of a dream formation mechanism that is not brain stem based but is cortical in nature, completely top down. Nevertheless, it is one in which the biology drives or determines the psychology and is therefore reductionistic in nature. As with all biological theorists SOLMS is unable to explain the transduction from neural activity to mental activity, nor can he account for the content of dreams, nor does he provide a basis to establish either a function for or the meaning of the dream experience. He does note that the loss of dreaming supports the Freudian position that dreaming protects sleep, however, he does not pursue this as a function of dreaming.

NIELSEN attempts to resolve the difference between those who see dreaming as independent of REM sleep and those who see it as the inevitable accompaniment of REM sleep. As the definition of the dream experience expanded to include so called cognitive mentation, the report of the experience is labeled sleep mentation rather than dreaming. There is no widely accepted standardized definition of dreaming because we are still doing the phenomenology of dreaming (Kramer 2000). There is no necessary reason why there should be. The standards applied to what will be counted as a dream, of course, alter the results. As we have no external criteria by which to judge what is and what is not a dream, all reports need to considered. Freud chose not to make a distinction reporting one word dreams such as the "Autodidasker" dream and a dream that took several hundred words in the telling "The Dream of Irma's Injection." Is there yet any necessary reason to chose one over the other? We found (Kramer et al. 1984), as did Fisher (Fisher et al. 1970a) that stage 2 nightmares were not different from REM nightmares in patients with post-traumatic stress disorder.

NIELSEN sees the evidence for neurobiological isomorphism as slim. He argues that the mental content in non-REM sleep is the result of the phasic aspects of sleep occurring in non-REM just prior to and immediately after REM sleep. These suggestions have not been tested and raise questions about the REM non-REM separation as it would attribute the mental phenomena to some sub-aspect of events occurring in REM sleep but not limited to REM sleep. The techniques are not available to test this hypothesis at the present time because we have no index in humans for the PGO waves. If this suggestion indeed is the case, NIELSEN would be confirming a biological explanation for the mental events in sleep and providing support for isomorphism. This position cannot account for the specific contents of the dream experience, nor is this its intent. It also does not deal with the transduction problem.

REVONSUO describes the biological function of dreaming as simulating threatening events and rehearsing threat perception and threat avoidance, which contributes to survival while awake and thereby increases the likelihood of reproductive success. The system operates in and out of awareness and is an alarm response so that all dreams at all times need not show evidence of threat perception and avoidance activity.

The nightmare is the paradigm for dreaming as it represents a threat perception and usually an avoidance. **REVONSUO** dismisses the poor sleep that accompanies this survival response. Nevertheless the 1991 National Sleep Foundation survey points out the negative consequences of insomnia (Roth & Ancoli-Israel 1999), an insomnia that would certainly accompany any traumatic nightmare. The dreams of patients with post-traumatic stress disorder (PTSD) on the same night have only half their dreams related to the trauma (Kramer et al. 1984). The better adjusted patients with PTSD have decreased dream recall (Kamminer & Lavie 1991; Kramer et al. 1984) and an elevated arousal threshold (Schoen et al. 1984) which would make them more vulnerable to predators at night. If the dreaming system is threat perception sensitive, one might expect that strangers would be incorporated into dreams more easily than familiar persons, but the opposite is the case (Kinney et al. 1981). This view of an evolutionary function is rather limited and others have offered a broader, more encompassing way to think about evolutionary functions (Moffitt et al. 1993). An evolutionary theory of the sort proposed would not account for the demographically related content differences described above (Kramer et al. 1971; Winget et al. 1972).

The view **REVONSUO** offers of the emotionally focused dream theories is too narrow in its understanding. For example, in the mood regulatory theory of dreaming, it is the consequence of the improved mood in the morning that is the issue, not just having achieved a less unhappy state. This improved mood state has been shown to covary with a subsequent improvement in psychomotor performance (Johnson et al. 1990). It is the consequence of the threat perception that explains its alleged function.

Papez dreams: Mechanism and phenomenology of dreaming

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Abstract: I agree with Revonsuo that dreaming, particularly about risky scenes, has a great selective advantage. Although the paleoamygdala system generally facilitates stress and alarm, the system which inhibits stress and alarm, initiates bold actions, and mediates learning in risky scenes is the arche, hippocampal system (Papez circuit). Because all thalamic nuclei are inhibited during sleep except arche, Papez probably also dreams in risky scenes.
[REVONSUO]

The mammillary bodies (MBs) which are the most ventral, caudal, and medial mammalian diencephalon, are unique among neurons whose axons constitute the chief input into one of the various thalamic nuclei, and thence to their corresponding cortices. The target of the MBs, anterior thalamic nucleus (ATN), is alone in not receiving inhibitory fibers from the thalamic reticular nucleus, which is usually active in sleep (Alonso & Llinas; Pare et al. 1991). Given that the backbone of the Papez circuit is the massive unidirectional mammillary projection to ATN, this lack of inhibition of ATN in sleep is the basis of my first thesis: Papez plays a critical role in dreaming.

I agree with **REVONSUO** who argues persuasively that:

1. Dreams are critical for survival;
2. There is considerable selective pressure to make the correct decision in threatening or risky real world contexts or scenes (Sparks 1998); and,
3. Dreams are imagined rehearsals for real scenes. This important idea is an extension of Tolman's vicarious trial and error (VTE) at the choice point (online) (Tolman 1938) to dreaming (offline), a complex sequence of events as scenes. But the same process is probably operative in both to let a physically possible sequence of events unfold and see what happens. If that doesn't work, then try another.
4. Given 1–3, dream VTE Dream vicarious trial and error (VTED) is thus presumably particularly critical in preparing for risky scenes.

I disagree with **REVONSUO** that:

1. The origin of risky VTED is to reduce the risk of predation in our earlier hunter-gatherer period; both the phenomenon of dreaming and its neural substrates are present throughout mammalia.

2. The mechanism of risk VTED is important primarily against predators; it is perhaps more import within a species, or within a tribe (e.g., when deciding whether to risk taking on the dominant male). Thus VTED learning in risky scenes plays a comprehensive and critical role within mammalia.

Neurophysiology. My second thesis is that two generally opposing forebrain functions are operative in VTED, just as in waking attention. During phylogeny, mammalian neocortex develops between the two allo pallia arche (hippocampus [HF]) and paleo (amygdala) (Nauta & Feirtag 1986). The allo system most discussed in mediation of action and affect in risky scenes is the paleo amygdala system which elicits specie-specific responses to threats (e.g. freezing and autonomic arousal) (LeDoux 1998). Amygdala also facilitates both stress (as excessive glucocorticoids) (Cullinan et al. 1993) and alarm (as excessive serotonin [5-HT]) (Maier et al. 1993).

However, Metcalfe and Mischel (1999) (discussed only too briefly by **REVONSUO**) introduce the opposite functions of a "cool" rational system (arche) which opposes the "hot" emotional system (paleo). Arche HF inhibits the activation of the stress cascade that amygdala excites (Cullinan et al. 1993; Krieckhaus 1999) and HF reduces alarm via mammillary inhibition of medial raphe (Kriekhaus 1999; submitted) that amygdala facilitates via excitation of dorsal raphe (Maier et al. 1993).

Given that arche inhibits stress and alarm, and arche is well known to mediate explicit learning (Aggleton & Brown 1999; Krieckhaus 1988; Squire et al. 1990) then it would be expected that arche lesions would uniquely interfere with learning to initiate bold action in threatening or risky scenes just the deficit seen following arche (MBs) lesions (Gabriel et al. 1995; Krieckhaus 1988; 1999). Finally, if **REVONSUO** is correct that dreaming is risky VTED, then arche should be critically involved in risky dreams just as it is in risky actions. Indeed, as we saw earlier: (1) Thalamic reticular nucleus can, in night dreams, inhibit all thalamic nuclei except arche ATN. (2) HF lesions severely disrupt choice point VTE and commensurate learning (Hu et al. 1997). Although VTE may be a different process from VTED, it is likely that HF mediates VTED as well. (3) Finally, given that dreams are for learning, then the neural system mediating learning should play a major role in dreaming; and since arche uniquely mediates explicit learning, arche Papez probably dreams explicitly.

Discussion. The diametrically opposite functions of fearful paleo and confident arche are fundamental to the mechanism of VTED or any other forebrain function. Whereas both paleo and arche receive high level invariant information (e.g., faces, grimaces) from posterior association cortex via entorhinal cortex (Krieckhaus et al. 1992; Ungerleider & Mishkin 1982), this information is consistently put to the opposite use (Cullinan et al. 1993; Graeff et al. 1996; Krieckhaus 1999; Metcalfe & Jacobs 1998). These two systems, arche and paleo, are the pallial two of four loops, which together with the two subpallial loops (somatic motor and visceral motor both worked out by Nauta 1966) constitute roughly half of the forebrain.

Because arche encourages rational explicit actions and can control negative affect, its functions are presumably what Freud characterized as secondary process thought (Freud 1911). The no less efficient and sophisticated functions of the paleo loop presumably correspond to what he called primary process thought. With this formulation of VTED, the survival value of Freud's "wish fulfillment" in dreaming is not to fulfill wishes but to instill in us hope and confidence by the responsible, reality oriented, arche dominating the scared and withdrawing paleo. Because these arche dominant dreams are rewarding they are more likely to lead to similar bold actions in later similar waking risky scenes, depending on the acumen of the organism's reality testing. Thus my third thesis: We mammals strive for a healthy equilibrium between arche and paleo, probably realized in a complete, continuous hegemony of arche over paleo.

Finally, as our understanding of neural mechanisms mediating the function of VTED increases, we are better able to distinguish the functions of experience in general, whether awake or dreaming, from the functions of the neural mechanisms that support these experiences. The general issue of the function of experience (given the sufficiency of its underlying neural substrate) reduces to the qualia problem of how neural (physical) activity can cause or be "mental" experience, an issue not pertinent here. More recently, the phenomenon of "blind sight" (relatively accurate adaptive actions but no commensurate experience) though still controversial, raises concrete questions about the functionality of experience, and makes more likely its emergence as epiphenomenal. Thus understanding of selective pressures for dreaming reduces to understanding the structure and function of the forebrain loops using verbal reports of experience simply as proxies for brain states.

Conclusion. Papez dreams, and, as argued by **REVONSUO,** dreaming is VTED learning, predominantly of risky scenes. The diametrically opposite functions of fearful paleo and confident arche determine the functions of dreaming and cognitive processes in general. The desired state of an adult mammal is for primary process paleo to be modulated by rational secondary process arche.

Lucid dreaming: Evidence and methodology

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Abstract: Lucid dreaming provides a test case for theories of dreaming. For example, whether or not "loss of self-reflective awareness" is characteristic of dreaming, it is not necessary to dreaming. The fact that lucid dreamers can remember to perform predetermined actions and signal to the laboratory allows them to mark the exact time of particular dream events, allowing experiments to establish precise correlations between physiology and subjective reports, and enabling the methodical testing of hypotheses.

[HOBSON ET AL.; SOLMS]

Just as dreaming provides a test case for theories of consciousness, lucid dreaming provides a test case for theories of dreaming. Although one is not usually explicitly aware that one is dreaming while in a dream, a remarkable exception sometimes occurs in which one possesses clear cognizance that one is dreaming. During such "lucid" dreams, one can reason clearly, remember the conditions of waking life, and act upon reflection or in accordance with plans decided upon before sleep. These cognitive functions, commonly associated only with waking consciousness, occur while one remains soundly asleep and vividly experiencing a dream world that is often nearly indistinguishable from the "real world" (LaBerge 1985).

Although lucid dreams have been reported since Aristotle, until recently many researchers doubted that the dreaming brain was capable of such a high degree of mental functioning and consciousness. Based on earlier studies showing that some of the eye movements of REM sleep corresponded to the reported direction of the dreamer's gaze (e.g., Roffwarg et al. 1962), we asked subjects to carry out distinctive patterns of voluntary eye movements when they realized they were dreaming. The prearranged eye movement signals appeared on the polygraph records during REM, proving that the subjects had indeed been lucid during uninterrupted REM sleep (LaBerge 1990; LaBerge et al. 1981).

Our studies of the physiology of lucid dreaming fit within the psychophysiological paradigm of dream research that Hobson has helped establish. Therefore, I naturally agree with **HOBSON ET AL.** in believing it worthwhile to attempt to relate phenomenological and physiological data across a range of states including waking,

NREM, and REM sleep. I also share HOBSON's view that REM sleep is unique in many ways; for example, stable lucid dreams appear to be nearly exclusively found in REM. As for the AIM model on which the HOBSON ET AL. article focuses, I regard it as an improvement on the earlier Activation-Synthesis model. The AIM model makes many plausible and interesting connections, but still doesn't do justice to the full range and complexity of the varieties of dreaming consciousness accompanying REM sleep.

One of the problems with AIM is that its three "dimensions" are actually each multidimensional. For example, from which brain area is "Activation" (A) measured? Obviously, A varies as a function of brain location. **HOBSON ET AL.** admit as much when they propose to locate lucid dreaming in a dissociated ATM space with PFC more activated than it usually is (see Fig. 12). If this is true, then non-lucid dreaming would have to be characterized by a low value of A. Incidentally, there is no evidence to support the idea that lucid dreaming is in any sense a dissociated state (LaBerge 1990). Still, the need for multiple A dimensions seems inescapable.

Similarly, the "Information flow" (I) dimension is more complex than at first appears. Experimental evidence suggests that it is possible for one sense to remain awake, while others fall asleep (LaBerge 1990). A further problem with the I "dimension" is the confounding of sensory input and motor output, as can be seen in several of **HOBSON ET AL.**'s examples (e.g., compare Figs.15, 16B, 19). Finally, "Mode of information processing" (M) attempts to reduce the vast neurochemical complexity of the brain to the global ratio of discharge rates of aminergic to cholinergic neurons. Is that really all there is to say about the neurochemical basis of consciousness? What about regional differences of function? What about the scores of other putative neurotransmitters and neuromodulators?

Perhaps due in part to the over-simplifications necessary to fit these multiple dimensions into an easy-to-visualize three, certain features of dreaming consciousness are misunderstood or exaggerated. For example, **HOBSON ET AL.** say "self-reflection in dreams is generally found to be absent (Rechtschaffen 1978) or greatly reduced (Bradley et al. 1992) relative to waking" However the two studies cited suffered from weak design and extremely small sample sizes. Neither in fact actually compared frequencies of dreaming reflection to equivalent measures of waking reflection. A study that did make direct comparisons between dreaming and waking (LaBerge et al. 1995) found nearly identical frequencies of reflection in dreaming (81%) as in waking (79%), clearly contradicting the characterization of dreams as non-reflective. Replications found similar results (Kahan & LaBerge 1996; Kahan et al. 1997). These studies were cited in Hobson's article but otherwise ignored.

Another unsubstantiated claim of **HOBSON ET AL.** is that "volitional control is greatly attenuated in dreams." Of course, during non-lucid dreams people rarely attempt to control the course of the dream by magic. The same is true, one hopes, for waking. But likewise, during dreams and waking, one has similar control over one's body and is able to choose, for example to walk in one direction or in another. Such trivial choice is probably as ubiquitous in dreams as waking and, as measured by the question "At any time did you choose between alternative actions after consideration of the options?" 49% of dream samples had voluntary choice, compared to 74% of waking samples (LaBerge et al. 1995). The lower amount of choice in dreams may be an artifact of poorer recall or a real difference, but choice is by no means "greatly attenuated."

While making the above claim, **HOBSON ET AL.** incorrectly attribute to me the false statement that "the dreamer can only gain lucidity with its concomitant control of dream events for a few seconds (LaBerge 1990)." In fact, lucid dreams as verified in the laboratory by eye-movement signalling last up to 50 minutes in length, with the average being about 2 minutes (LaBerge 1990). The relatively low average is partially due to the fact that subjects were carrying out short experiments and wanted to awaken with full recall. At the onset of lucid dreams there is an increased tendency to awaken, probably due to the fact that lucid dreamers are thinking at that point, which withdraws attention from the dream, causing awakening (LaBerge 1985).

The eye-movement signalling methodology mentioned above forms the basis for a powerful approach to dream research: Lucid dreamers can remember pre-sleep instructions to carry out experiments marking the exact time of particular dream events with eye movement signals, allowing precise correlations between the dreamer's subjective reports and recorded physiology, and enabling the methodical testing of hypotheses. We have used this strategy in a series of studies demonstrating a higher degree of isomorphism between dreamed actions and physiological responses than had been found previously using less effective methodologies For example, we found that time intervals estimated in lucid dreams are very close to actual clock time (see Fig. 2); that dreamed breathing corresponds to actual respiration (Fig. 3); that dreamed movements result in corresponding patterns of muscle twitching (Fig. 4); and that dreamed sexual activity is associated with physiological responses very similar to those that accompany actual sexual activity (see LaBerge 1985; 1990 for details).

These and related studies show clearly that in REM sleep, dreamed bodily movements generate motor output equivalent at the supraspinal level to the patterns of neuronal activity that would be generated if the corresponding movements were actually executed. Most voluntary muscles are, of course, paralyzed during REM, with the notable exceptions of the ocular and respiratory muscles. Hence, the perfect correspondence between dreamed and actual movements for these two systems (Figs. 1–3), and the attenuated intensity (but preserved spatio-temporal pattern) of movements observed in Figure 4.

These results support the isomorphism hypothesis (**HOBSON ET AL.**) but contradict **SOLMS**'s notion of the "deflection" of motor output away from the usual pathways, and his speculation that it isn't only the musculo-skeletal system that is deactivated during dreams, but "the entire motor system, including its highest psychological components which control goal-directed thought and voluntary action" (Solms 1995, p. 58). I believe Occam's Razor favors the simpler hypothesis that the motor system is working in REM essentially as it is in waking, except for the spinal paralysis; just as the only essential difference between the constructive processes of consciousness in dreaming and waking is the degree of sensory input. See LaBerge (1998) for details.

Oddly, **HOBSON ET AL.** ignore the data on eye movements while appealing that we keep open the question of relationship between eye movement and dream imagery "until methods more adequate to its investigation are developed." There is no need to wait. Adequate methods have already been developed, as shown above (Figs. 1–4), and in our recent study showing smooth tracking eye movements during dreaming (LaBerge & Zimbardo 2000).

Memory is another area of inquiry upon which lucid dreaming can shed light. **HOBSON ET AL.** argue that memory during dreaming may be as deficient as it is upon awakening. They give the example of comparing one's memory of a night's dreaming to the memory of a corresponding interval of waking; unless it was a night of drinking being remembered, the dream will yield much less memory. But this is an example comparing episodic memory from waking and dreaming after awakening, and thus is not only unconvincing and vague, but irrelevant. Nobody disagrees that waking memory for dreams is sometimes extremely poor.

In the same vein, **HOBSON ET AL.** write that it is common for dreams to have scene shifts of which the dreamer takes little note. "If such orientational translocations occurred in waking, memory would immediately note the discontinuity and seek an explanation for it." Note the unquestioned assumption. In fact, recent studies suggest that people are less likely to detect environmental changes than commonly assumed (Mack & Rock 1998). For example, a significant number of normal adults watching a video failed to notice changes when the only actor in a scene transformed into another person across an instantaneous change in camera angle (Levin & Simons 1997).

Likewise, **HOBSON ET AL.** assert that "there is also strong evidence of deficient memory for prior waking experience in subsequent sleep." However, the evidence offered is always extremely indirect and unconvincing. A direct test requires lucid dreamers to attempt memory tasks while dreaming, as was done in a pilot study (Levitan & LaBerge 1993) showing that about 95% of the subjects could remember in their lucid dreams a key word learned before bed, the time they went to bed, and where they were sleeping. Subjects forgot to do the memory tasks in about 20% of their lucid dreams. That may or may not represent a relative deficit in memory for intentions.

A major methodological difficulty presented by dreaming is poor recall on awakening. The fact that recall for lucid dreams is more complete than for non-lucid dreams (LaBerge 1985) presents another argument in favor of using lucid dreamers as subjects. Not only can they carry out specific experiments in their dreams, but they are also more likely to be able to report them accurately. That our knowledge of the phenomenology of dreaming is severely limited by recall is not always sufficiently appreciated. For example, **HOBSON ET AL.** repeatedly substitute "dreaming" for "dream recall" (e.g., sect. 2.3.1). **SOLMS** (1997a) makes the same mistake, which in my view, is fatal to his argument. So when he writes "of the 111 published cases . . . in which focal cerebral lesions caused cessation or near cessation of dreaming" he is really saying "in which lesions caused cessation of dreaming or dream recall." To think otherwise would be to suppose that the dream is the report.

All brain work – including recall – is state-dependent

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Abstract: The continuous ongoing mentation is experienced as dreams in some functional states. Mentation occurs with high speed, is driven by individual memory, and uses state-dependent processing strategies, context material, storage options, and retrieval access. Retrieval deserves more attention. Multiple state-shifts owing to individual meaning as extracted also during sleep concatenate dream narratives and define access to segments for awake recall.
[HOBSON ET AL.; NIELSON; SOLMS]

Since the late 50s, dreams have been assumed to be the product of REM sleep. For dreams reported out of other sleep stages, **HOBSON ET AL.** argue now that there are "possible dissociations of state characteristics" that might permit "states in which some parameters match their canonical NREM values while others match canonical REM or wake values." **NIELSEN** makes a somewhat related proposal when speaking of "covert REM sleep processes during NREM sleep." **SOLMS** also postulates a specific dream state that in his view, however, can be independent of REM physiology.

We welcome this blurring of the earlier sharp distinction between two classes of sleep stages and between two classes of experiences. It opens the way for dream studies to take into account three major properties of the continuously ongoing mentation: its high speed, in the subsecond range (otherwise one couldn't even drive a car), its construction from individual memory, and its state-dependency (otherwise one would think in the same way when sober and inebriated, when awake or drowsy or asleep, as child or as adult) that is finely grained. Different modes of momentary mentation have been shown to be associated with distinctly different brain electric characteristics in the second time range (Lehmann et al. 1983; 1995) as well as in the mentation-relevant sub-second time range where the time trajectory of brain momentary states through state space consists of dwell times ("microstates") and shifts: that is, it is discontinuous (Lehmann et al. 1998). Mentation leads to the extraction of individual meaning that, if needed, initiates a state shift to optimize the conditions for the next step of brain work (Koukkou & Lehmann 1983; 1987); hence, the shifts of brain functional state during sleep need to be examined at a much higher time resolution (Cantero et al. 1999b) than in classical approaches to sleep psychophysiology.

The issue of retrieval is approached by **HOBSON ET AL.,** who say that "subjects assert that much antecedent dreaming could not be recalled," but he continues with "one reason for the neglect of this robust phenomenon is that memory isn't there!" Nobody can know for certain whether there was something – but there are compelling reasons to assume that there is always something in the mind during sleep, just as in wakefulness. The brain must continuously process information, from external and internal sources, not only during wakefulness, but also during sleep: otherwise sleeping people couldn't distinguish between relevant stimuli that require awakening or not (the sleeping mother who will awake at the whimper of her baby and sleep right on while the traffic roars by in front of the house; the subject who awakens to his name but not to names without biographical relevance). And, as in waking, if there are no external inputs that require state-shifts, the biographically generated memory has abundant material in waiting for review.

Well, maybe the current mind stuff is not stored for some reason? Again, nobody knows. But, absence of recall does not prove that nothing happened, it primarily shows that there is no recall. Experiences can be stored in a state too remote from the state during later attempts to recall (Koukkou & Lehmann 1983); but, when the original state is re-installed, the experiences become recallable again. There are numerous accounts portraying the effects of this mechanism, a very famous one described by Marcel Proust in "*La recherche du temps perdu*"; flashbacks in drug addicts are its modern version. A family classic is the no recall condition of the candidate when highly excited during an examination, and his "but I knew it all!" surprise when leaving the examination and relaxing. Events experienced in one state are optimally available for recall when the same state is reinstalled (Eich 1986).

We proposed the basic EEG measure of dominant "wave frequency" as first approximation of a metric for the state-dependency of type of dream mentation and in particular, of quality of dream recall (Koukkou & Lehmann 1983): the slower the dominant frequency and thus the further away the brain state at information experience is from the state at attempted information retrieval (i.e., wake), the poorer will be the recall – while arguing that this must zoom in on very brief time epochs (possibly "single waves"), not conventional EEG sleep stages. (But many more measurement dimensions of electric data should be added for comprehensive assessments of brain functional state, e.g., dimensionality of the embedding state trajectory, momentary coherency between intracerebral generator processes, or momentary spatial distribution Lehmann et al. 1998.)

A prime example of state-dependent non-availability of recall is sleepwalking with its goal-directed behavior that occurs in slow wave sleep (Jacobson et al. 1965). Not only is external input treated at high levels, also behavior is selected and implemented. But, the events that happen during sleep walking typically are not recallable after awakening. So-called childhood amnesia is another example: few events before the age of about 4 years are recallable by awake adults: because toddlers' EEGs are dominated by slower frequencies, and adults' EEGs are much faster. From pathological examples: behavior under scopolamine (that is associated with "slowing" of the EEG) cannot be recalled after the drug wore off (Bradley & Elkes 1957). Other classical, pathological conditions with EEG slow wave activity and unavailability of later recall of events are temporal seizures and head trauma.

In sum, we suggest that there is continual mentation during wakefulness and sleep, implemented always with the same basic,

biography-driven brain machinery which leads to varying final products depending on the momentary global functional state of the brain: the momentary state is the fate of the information. The momentary state defines access to state-dependent processing strategies, context memory, storage procedures, and recall options; the numerous shifts of functional state during sleep accordingly concatenate very different mentation characteristics as segments of dreams (Koukkou & Lehmann 1983). Considering the state-dependency of all brain work including recall, and considering that the continual mentations and emotions are implemented in a split-second time range will help to clarify the ever-intriguing experiences during sleep.

Nightmares: Friend or foe?

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Abstract: Revonsuo's evolution-based theory places the nightmare as a prototype dream, which fully realizes its biological function. However, individuals who experience both repetitive (PTSD) nightmares and/or lifelong nontraumatic nightmares demonstrate impaired psychological functioning and attenuated information-processing. The importance of reconciling these discrepancies are addressed and ideas for providing stronger empirical tests of the model are presented.
[REVONSUO]

REVONSUO's evolution-based theory of dreaming is cogently presented in six empirically verifiable propositions and in many respects is quite plausible despite its tendency to overreach at certain points. revonsuo should be commended for drawing attention to the wealth of data indicating that dreaming is an organized and internally consistent simulation of the perceptual world. In addition, REVONSUO effectively supports his central assertion that dreaming is highly specialized in its selection of affect-charged memorial content, particularly of a threatening or dysphoric nature. However, his contention that nightmares are a prime example of dreams that fully realize their biological function is problematic and will be the focus of my comments.

If dreaming evolved to serve a threat simulation function, and if experiences of real threats which approximate the "primitive" human ancestral environment are the only ecologically valid cues for the activation of this system, why then are individuals who suffer from post-traumatic stress disorder (PTSD) so chronically overwhelmed by the very intrusive symptoms which should be helping them successfully resolve their problem? PTSD individuals demonstrate a marked hypersensitivity to overly activated fear-arousal networks (Foa & Kozak 1986) that interfere with their ability to process information effectively. Thus, it is difficult to understand how intrusive traumatic symptoms, whether they be repetitive nightmares or waking flashbacks, aid in successful coping, particularly as they may continue unabated for up to 50 years post-trauma with an absence of accompanying mastery or reduction in psychological distress (Lansky 1995). **REVONSUO** does address this tricky issue but only cursorily, concluding with the questionable assumption that modern-day trauma is too removed from the pre-technological daily traumas in which the dreaming system evolved to be adequately generalizable. While this may be true, modern living still provides numerous approximates of traumas that should activate threat simulator programs (e.g., incest, rape, physical beatings, natural disasters). REVONSUO's argument would be bolstered by data demonstrating that individuals who experience the latter types of psychological trauma recover quicker than those who are exposed to traumas involving insults for which the evolved threat-avoidance programs have not yet been incorporated.

REVONSUO also maintains that dreaming serves a similar biological defense mechanism function as do antigens in the pathoimmune system. We should then expect that exposure to early environmental threats should mobilize the dreaming immunological system and provide a psychological vaccine to deal more effectively with subsequent trauma. In fact, research has repeatedly demonstrated that exposure to early aversive environmental events is a primary pathogenic pathway to later psychopathology (Gershuny & Thayer 1999). Of course it could be argued that these experiences (parental loss, divorce, child abuse) are too disruptive to function effectively as a low dose for subsequent immunological functioning. It can also be argued that dreaming functions at a far more nomothetic and trans-species level and that its presumed protective qualities do not apply to individual cases in either of the above examples. In any case, these discrepancies should be fleshed out in greater detail.

REVONSUO does not address the issue of significant individual variation in the experience of lifelong non-trauma related nightmares. I believe this is crucial, as nightmares are a prototype dream within REVONSUO's model. Frequent lifelong nightmare sufferers demonstrate considerable psychological dysfunction and may be at increased risk for the development of schizophrenia-spectrum disorders (Hartmann 1984; Hartmann et al. 1987; Levin 1990a; 1994, 1998; Levin & Raulin 1991). It would be important to reconcile this data with his contention that such dreams were ancestrally selected for their adaptive function in preparing for fight-flight responses. It is interesting to note that in his influential paper on the development of schizophrenia, Meehl (1989) suggested that humans evolved from a schizotaxic genetic background. Could it be that nightmare sufferers retain a closer link to their ancestral vestige of the early fight-flight patterns than do other individuals? While highly conjectural, understanding better how these pieces fall together could develop our understanding of why nightmares occur.

Last, I believe that the author is too quick to dismiss alternative theories of dream function, particularly the problem-solving model. **REVONSUO**'s claim that "there is no convincing" evidence that dreaming would casually contribute to the solving of either intellectual or emotional problems" runs counter to a large body of empirical data demonstrating otherwise (Cartwright 1986; Fiss 1993; Hartmann 1998; Koulack 1991; Moffitt et al. 1993; Levin 1990b; **VERTES & EASTMAN** 2000; Blagrove 1996 for opposing views). Furthermore, REVONSUO's claim that "the brain's dream production system selects traumatic contents not because they represent unsolved emotional problems but because such experiences mark situations for physical survival and reproductive success" is highly speculative and not directly supported by any empiric evidence. While it is difficult to predict the content of future dream production (Cartwright 1974b; Nikles et al. 1998), numerous studies by Cartwright (1986) and Kramer (1993) indicate that, "the dream is a selective affective regulator which functions as an 'emotional thermostat'" (Kramer, p. 182). In addition, REVONSUO fails to consider the literature on creative dreams (Dave 1978; Dreistadt 1971; Krippner 1981; Livingston & Levin 1991).

If dreams truly simulate phylogenetic threats, an important test of this model would be to determine what effect the presentation of stimuli that have known fear-relevant properties (those that meet the criteria for biological-preparedness for phobia acquisition such as spiders, snakes, angry human faces; McNally 1987) have on subsequent dream content. Furthermore, given **REVONSUO'S** claim that the function of dreams may be optimally realized through implicit processing, such material could also be presented at subliminal activation levels and compared to supraliminal activation conditions. In order to provide a stronger test of his theory, levels of incorporation of the target stimuli in subsequent dream production could be compared to known current concerns of the dreamer (Nikles et al. 1998) to directly determine which stimuli have greater predictive utility. Further studies along these lines would be most helpful in providing new clues into the investigation of dream function.

Koch's postulates confirm cholinergic modulation of REM sleep

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Abstract: Robert Koch (1843–1910) discovered the causal agents for tuberculosis, cholera, and anthrax. The 1905 Nobel Prize acknowledged Koch's criteria for identifying the causal agent of an infectious disease. These criteria remain useful and the data reviewed below show that the cholinergic contributions to REM sleep control are confirmed by Koch's postulates.
[HOBSON ET AL.]

We congratulate **HOBSON ET AL.** for stimulating synthesis of cognitive science and sleep neurobiology. Their article demonstrates the unifying power of the localization-of-function concept so successfully advanced by nineteenth century German neurology. This commentary focuses on their proposal that the cholinergic hypothesis of REM sleep generation has been confirmed. We also draw from the nineteenth century, showing how data concerning the cholinergic modulation of REM sleep satisfy Koch's postulates (Brock 1999). Available data support Koch's postulates when one evaluates medial pontine reticular formation (mPRF) levels of acetylcholine (ACh) as a causal agent modulating the state of REM sleep.

> Postulate 1: The state must be reproduced when the agent is administered.

Microinjection of cholinergic agonists (Baghdoyan et al. 1984b) and ACh-esterase inhibitors (Baghdoyan et al. 1984a) into the mPRF causes REM sleep enhancement. REM sleep is inhibited by mPRF injection of the muscarinic cholinergic antagonist atropine (Baghdoyan et al. 1989; Lee et al. 1995) and by drugs that block the vesimacol receptor regulating the vesicular packaging of ACh (Capece et al. 1997). Normally, the mPRF is never exposed to cholinomimetics. How does cholinergically-induced REM sleep affect mPRF levels of the endogenous ligand ACh?

> Postulate 2: The agent is recovered during the experimentally-induced state.

Microdialysis data demonstrate significant enhancement of mPRF ACh release during the REM sleep-like state caused by contralateral mPRF administration of carbachol (Lydic et al. 1991b). Additional data satisfying this postulate include the finding that REM sleep is enhanced by electrical stimulation (Thakkar et al. 1996) of laterodorsal and pedunculopontine (LDT/PPT) neurons shown to regulate ACh release within the mPRF (Lydic &Baghdoyan 1993). A limitation of postulates one and two is that they are based on a REM sleep-like state produced by exogenous stimulation of the pons. The relationship between mPRF ACh levels and natural REM sleep is addressed by a third postulate.

> Postulate 3: The putatively causal agent should be present during every naturally occurring case.

Microdialysis data show that mPRF ACh release is significantly greater during spontaneous REM sleep than during waking or non-REM sleep (Leonard & Lydic 1995; 1997). Thus, levels of the putatively causal agent (ACh) are greatest in the mPRF during natural REM sleep.

> Postulate 4: Requires the isolation of the putatively causal agent from the host.

A ligand such as ACh is irrelevant without a functionally significant binding site. Therefore, in addition to ACh, cholinergic receptors also may be considered as agents to be isolated in the host (mPRF). Receptor mapping studies have identified M2 muscarinic cholinergic receptors (mAChRs) in the mPRF (Baghdoyan 1997; Baghdoyan et al. 1994). Functional data show that M2 muscarinic autoreceptors modulate ACh release in the mPRF (Baghdoyan et al. 1998) while mPRF M2 heteroreceptors contribute to REM sleep generation (Baghdoyan & Lydic 1999). All mAChRs are coupled to G proteins and in many brain regions M2/M4 mAChRs are linked to an inhibitory G protein (G_i). Pertussis toxin selectively ADP ribosylates G_i proteins thereby preventing interaction with mAChRs. Cholinergic REM sleep enhancement is blocked by mPRF administration of pertussis toxin (Shuman et al. 1995). These data are consistent with G protein mediation of REM sleep, a conclusion supported by additional signal transduction studies showing cholinergic REM sleep modulation by mPRF adenylate cyclase, cAMP, and protein kinase A (Capece & Lydic 1997). Postulate four is supported by direct measurement of mPRF G protein activation by carbachol and inactivation by atropine (Capece et al. 1998).

We conclude that Koch's postulates have been satisfied for the cholinergic hypothesis of REM sleep generation. These advances concerning cholinergic neurotransmission in a defined LDT/PPT-to-mPRF network provide a solid basis for continued progress in sleep neurobiology.

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"Spandrels of the night?"

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Abstract: Vertes & Eastman argue against the popular idea that dreams promote memory consolidation and suggest instead that REM provides periodic endogenous stimulation during sleep. Although we suspect that much of the debate on the function of dreams reflects a too eager acceptance of the "adaptationist program," we nonetheless support the position of the authors and propose a specific advantage of periodic REM activity.
[VERTES & EASTMAN]

In the essay, "The spandrels of San Marco and the Panglossian paradigm: A critique of the adaptationist programme," Gould and Lewontin (1979) describe the remarkable aesthetic beauty of the spandrels of St. Mark's Cathedral in Venice. Their design is so "elaborate, harmonious, and purposeful" that one forgets that the spandrels are merely a necessary result of the original architectural purpose: mounting a dome on rounded arches. Similarly, the vivid intensity of dreams tempts investigators to believe that they evolved to serve a high-level cognitive purpose, such as memory consolidation (see article) or protection from repressed, "unconscious wishes" (Freud 1900). Although Freud's psychoanalytic theory of dreams has been abandoned for the most part, the memory consolidation hypothesis has survived, despite the lack of a convincing body of evidence to support it. Is it possible that the elaborate sensations of dreaming are analogous to the visual beauty of the spandrels of St. Mark's? Blinded by their beauty, we forget that dreams may not have directly evolved to serve a higher purpose but instead may be a necessary by-product of the basic structure of sleep.

REM sleep appears first phylogenetically in birds and is also present in almost all mammalian species. This has led researchers to conclude that REM sleep evolved to serve a high order cognitive function, namely memory consolidation. However, dolphins do not exhibit REM sleep (Mukhametov 1984), although dolphins are certainly capable of learning.

Bob Vertes's extensive contributions to the understanding of

hippocampal theta rhythm production and possible function allow him certain insights into its role in REM sleep. We support his position that the theta rhythm of waking states does not have the same function as the theta rhythm of REM sleep. In fact we, along with others, suspect that each type of oscillation has a specific role in information processing. **VERTES** has previously proposed "that the theta rhythm may serve to gate or facilitate the transfer of information to the hippocampus, a process that may be involved in the long-term storage of that information" (Vertes & Kocsis 1997). Traub and colleagues have proposed that gamma oscillations in the cortex signify the details of a percept, while beta oscillations may reflect the occurrence of a stimulus with particular significance (Traub et al. 1999). The absence of this interplay of the different field potential oscillations during REM sleep may suggest that mnemonic functioning is absent, and theta is not functioning as it does during consciousness.

VERTES & EASTMAN are making wider claims than indicated by their title. Not only do they assert that there is no memory consolidation in REM sleep, they maintain that the function of REM sleep is to provide periodic stimulation to the brain to offset the depressed brain activity of slow wave sleep. This position is plausible, considering that REM sleep never occurs prior to episodes of slow-wave sleep, except in the case of narcolepsy.

A possible example of the importance of periodic stimulation involves the link between sleep and depression. A characteristic of depression is reduced latency of entry into REM sleep following sleep onset (Kupfer & Thase 1983). Sleep deprivation has an antidepressant effect (Wu & Bunney 1990, for a review), and the three major classes of antidepressants suppress REM sleep, as discussed by the authors in detail. In normal awake and slow wave sleep states, the raphe nucleus is releasing serotonin. At the onset of REM, raphe nucleus activity ceases, and serotonin release is suppressed. Serotonergic neurons have 5-HT1A autoreceptors, which regulate their function. Following REM sleep deprivation, these autoreceptors become less responsive to the effects of serotonin reuptake blockers, probably due to a desensitization of the autoreceptors, resulting in enhanced serotonergic transmission (Maudhuit et al. 1996). We would like to propose that REM activation serves to prevent the desensitization of autoreceptors that would occur if serotonin continued to be released. Similar needs for periodic activation via REM could involve endocrine functioning, as evidenced by body temperature increases (Wehr 1992) and hormonal changes (Obal & Krueger 1999) during REM sleep.

It may be premature to assert definitively that dreaming serves no higher cognitive function. Spandrels become an important artistic grammar in their own right. As Nietzsche wrote, "the cause of the origin of a thing and its eventual utility, its actual employment and place in a system of purposes, lie worlds apart" (Nietzsche 1992).

Dream production is not chaotic

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Abstract: The AIM model proposed by Hobson et al. is interesting: We know the neurophysiological aspects of the activation process (A) and the external input (I), but very little about the internal input and neurocognitive process (M). Internal input could be an expression of unconscious experiences memorised by the subject containing his emotional and cognitive history. Therefore internal input could not be chaotic but might have an emotional and affective sense bound to the unconscious. The fact that dreams are present in the absence of REM sleep means that they may originate from other structures besides the pons. These structures may represent the archives of dreamer's affective history.
[HOBSON ET AL.; SOLMS]

My comment on the work of **HOBSON ET AL.** is essentially epistemological. My criticism concerns the concept of an isomorphism between the phenomenology of dreams and the physiology of the various phases of sleep. I agree that dreaming can be approached using a principle of ontological monism, (i.e., that every kind of mental activity, and hence also dreaming, is the result of processes taking place in the brain). Because we do not know the complex chain of events linking physiological and mental events, it is epistemologically incorrect to talk of mind/brain isomorphism as if mental events could be entirely identified with physiological events. I accordingly do not agree with Hobson (1988b) that dreaming is a physiological event. Dreaming is too complex a mental process to be directly explained using a model, albeit an interesting and sophisticated one, such as the AIM model proposed by **HOBSON ET AL.**

If we look at this model, we see that while we have neurophysiological evidence regarding the activation process (A), and we know the external input (I), we know little about what **HOBSON ET AL.** call internal input and even less about what they define as the cognitive neuromodulator process (M). Regarding the internal input, why not think of it as unconscious dynamics and experiences memorised by the subject, containing his emotional and cognitive history, rather than think in terms of chaotic input?

HOBSON ET AL.'s argument in favour of a double dream generator, organised in a profoundly different way on qualitative and quantitative levels during the phases of non-REM and REM sleep, comes up against equally convincing arguments proposed by other authors (Antrobus 1983; Bosinelli 1995; Foulkes 1997; 1999) in favour of a single dream generator relatively independent of the various biological phases of sleep itself. But even if the double organization of sleep were experimentally confirmed during the REM and non-REM phases, we would still need to explain the presence of dreams even in subjects with pontine lesions and no REM sleep (Solms 1995). **SOLMS**'s observation seems to have been greatly minimised by HOBSON ET AL. whereas they have stressed the importance of the pons in the process of neocortex and limbic system activation. In fact, it is reasonable to think that the visual cortex is activated in REM sleep to produce the visual hallucinations of dreams and that the amygdala and other limbic structures are activated to produce their emotions and anxieties. What is more difficult to accept, also owing to the absence of clear evidence, is the idea that this activation comes only from the pons and is chaotic, and that it activates the associative neocortices and the limbic system in a disorderly and unfocussed manner.

Since dreaming occurs even in the absence of pontine structures (Solms 1995), it might be thought that the source of the process of cortical and limbic dream activation is not only pontine but may also be found in other cortical and subcortical structures. Furthermore, the process may not be chaotic at all, but may instead retrieve from the memory archive emotional and cognitive experiences organised in the internal world of the dreamer, thus activating a neurocognitive system that takes into account possibly traumatic processes deposited in and removed from the unconscious. This retrieval may occur in a way which is not necessarily that linear, but distorted, so as to create manifest dream contents which, owing to condensation, symbolisation, oddities and absurdities, are different from the latent ones.

This neurocognitive hypothesis would bring the process of dreaming closer to that studied by psychoanalysis. In this line of thought, I believe the contribution of Eric Kandel (1998) is very important: rather than proposing a critical and destructive approach towards psychoanalysis, he proposes the constructive hypothesis that the speech and learning on which the psychoanalytical process is based may modify genic expression and therefore protein synthesis and consequently even the long-term functionality of certain synaptic structures.

In their review **HOBSON ET AL.** limit themselves to suggesting neurocognitive models with neurophysiological bases, and explain dreams using mental categories (hallucinations, thoughts, affection, emotions), without taking into consideration the contribution of psychoanalysis in the study of dreams and their significance. These authors apparently fail to recognise the simple historical

fact that in the person of Freud psychoanalysis was dealing with dreams at least 50 years before neuroscientists and cognitivists were. I believe it is useful, as Kandel suggests, to bring neuroscience closer to psychoanalysis but for this to happen, it is not enough for psychoanalysts to confidently embrace neuroscience; it is also necessary for neuroscientists to know about psychoanalysis and the transformations that have taken place in the psychoanalytical method over the last 100 years, and to accept the extremely significant contributions that psychoanalysis has brought to the study and significance of dreams. I believe that every good scientist must know the limits of his method and must accept the possibility of integration from other disciplines, even those operating with different methods from his own, without falling victim to epistemological confusion.

Novel concepts of sleep-wakefullness and neuronal information coding

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Abstract: A new working hypothesis of sleep-wake cycle mechanisms is proposed, based on ontogeny and functional/anatomic compression of two stochastic neuronal models of information coding that complement each other in a key/lock fashion: the axonal arbor patterns (AAP – "hardware") and the neuronal spike interval inequality patterns (SIIP – "software").
[HOBSON ET AL.; NIELSEN; REVONSUO; SOLMS; VERTES & EASTMAN]

Impressive analyses of clinical, behavioral, EEG and neuronal firing associated with sleep-wake cycles have been provided by **HOBSON ET AL., SOLMS, NIELSEN, VERTES & EASTMAN,** and **REVONSUO**. All five reviews ably described interactions between neuronal systems, but only in global terms of activation and inhibition. This conventional approach leaves out subtle important modes of neuronal interactions: the word-like intraburst timing of emitted action potentials, defined as spike interval inequality patterns (SIIP) that naturally, via axon arbor filters, seek their axonal arbor patterns (AAP). It is postulated that this communication system resulted from ontogenic compression of two stochastic processes: one linked to emission of SIIP and the other linked to shaping AAP, both aimed at efficient real-time information storage and retrieval.

In our studies of SIIP, the computer measured sequential spike intervals with 0.1 msec resolution and stored the data in sequential computer memory bins. Partially inspired by Norbert Wiener's (1921) criticism of mindless "infinitesimal" clockwork measures used in exploring brain cognitive functions and his favorable comments on the Weber's law that introduces measures derived from relative responses to sensory stimuli, we have explored the concept of SIIP as potential carriers of information emitted by brain neurons (Brudno & Marczynski 1977; Marczynski & Sherry 1971). A computer "window" for comparing sequential pairs of spike intervals moved one spike interval at a time; if the second interval in a pair was longer or shorter than the first interval, a (+) and (−) was entered respectively into another series of sequential computer memory bins. Excessively long intervals (>200 msec) were treated as "punctuation" gaps after which the inequality testing was resumed. Subsequently, the sequences of inequality signs were arranged into transition frequency matrices of various complexity. If the matri columns and the rows are labeled (+) and (−) respectively, the matrix cells tell how many times a (+) was followed by a (−) or by (+) and (−). In this manner, higher order matrices have been constructed that counted the occurrences of "words" composed of 3 through 6 inequality signs (trigrams through hexagrams). Based on the novel stochastic model (Brudno & Marczynski 1977), the probabilities were assigned to each SIIP permutation. The departures of SIIP occurrence from the model, that is excessive emissions or deficits, were quantified using the chi square statistics.

Figure 1 summarizes the physiologic rationale of the SIIP concept. The arrow between SIIP-A and SIIP-B shows the direction of SIIP propagation in the main axon with seven collaterals. To keep the essential details simple, it was assumed that the geometric ratios at each axonal branching and the presence of the nodes of Ranvier (not shown) permit uninterrupted propagation of action potentials, although in reality most arbors work as electric filters that discriminate between SIIP (Deschenes & Landry 1980; Manor et al. 1991). In Figure 1, the inequalities between sequential pairs of spike intervals (moving one spike at a time) are expressed by signs (+) and (−). The mean spike rate and the mean spike interval are identical in SIIP-A and SIIP-B, yet their relative timing and therefore theoretical probabilities based on the stochastic model (Brudno & Marczynski 1977) are different. Thus, the timing of SIIP propagation into 7 axon collaterals must be different for SIIP-A and SIIP-B. Thus, these two SIIP must have different effects on functional dynamic "binding" in neuronal assemblies to which they project, despite that their SIIP Gaussian statistics are identical. There is a key/lock relationship and functional compression between each SIIP conceptualized as "software," and the corresponding axonal arbor patterns (AAP) conceptualized as "hardware." The term meta-organizing system (MOS) stands for the Hebb-like (1949) heteromodal association systems assumed to have "knowledge" of most sensory information and primary drives of the organism (MacKay 1965; Marczynski 1993), a system that operates mainly via dynamic interactions – "binding" among neurons and in real time (cf. von der Malsburg 1999).

In Figure 1 the ideas conveyed by SIIP-A and SIIP-B are presented as momentary "snapshots" disregarding the intermediate time frames. The SIIP-A is depicted at its most influential time frame. In contrast, SIIP-B is shown in the least influential time frame, and, due to its temporal structure, it could never achieve the effects of SIIP-A. If a condition represented by SIIP-B spike train would prevail for a longer time period and involve many neurons, a functional differentiation of cognitive systems would result, leading to a loss of consciousness and slow wave sleep (SWS).

Origins of the SIIP information code. One can argue that the SIIP code, even though it ignores scalar data, should be acceptable as "hard" science. The SIIP code is most likely the product of unicellular organisms, because of its simplicity and reliability for selecting, in real time, adaptive cognitive/motor behavior. The SIIP code probably stems from the cell's ability to sense gradients, that is, inequalities of environmental stimuli, such as temperature and/or concentrations of attractant/repellent chemicals (Koshland 1974; Stock & Surette 1996). The brain ontogeny is one of the most complex processes malleable to environmental influences (cf. Aigneret al. 1995; Barinaga 1999; Benowitz & Routten-

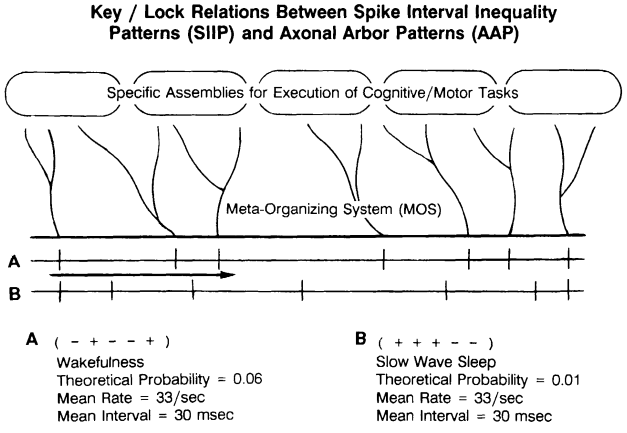

Figure 1 (Marczynski). Key/Lock relations between Spike Interval Inequality Patterns (SIIP) and Axonal Arbor Patterns (AAP).

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berg 1997; Maurer et al. 1999; Numgung et al. 1997; Shatz 1990; 1992 Shea 1994; Smith & Skene 1997; Stirling & Dunlop 1995; Tessier-Levigne & Goodman 1996). Despite considerable computational capacities of cell proteins (Bray 1995), the ontological processes most likely disregard the physics-inspired infinitesimal clock-work formalism, the scalar data and the Gaussian statistics, and use instead biologically inspired sensing of gradients, that is, inequality judgments (cf. Korovkin 1975).

Even in adult humans, cognitive processes are largely based on inequality judgments, as revealed by experiments in which a subject is asked to compare two digit numbers. The reaction time, measured in milliseconds, referred to as symbolic distance effects, is significantly longer for numbers that are close together and require increased cognitive scrutiny, as compared to judging numbers that are far apart. This indicates that cognitive processes use abstract inequality concepts, even if they are in some way attached to sensory scalar values (Link 1990; Moyer & Landauer 1967; cf. Anderson 1995).

The simplicity of inequality judgments found the way to the commercially ubiquitous bar codes that label supermarket products. If scanned by a hand-held laser beam, the bar code retrieves, in real time, all pertinent information about the purchased product encoded in a few bar interval inequalities. The theoretically attractive-to-neurobiologists concept of brain look-up information tables had been tarnished by its use with convoluted Gaussian statistics and the radial basis function (Poggio 1990), which might increase the latency of real time brain responses, with disastrous consequences.

The self-organizing properties of SIIP matrices. The autoassociative memory is defined as a memory system in which every component or signal is encoded and is retrievable from other signal characteristics that have occurred simultaneously during storage (Kohonen 1984). Thus, in the model of auto-association SIIP matrices, a randomly selected subset of SIIP can be used to recover details of distributed memory comprising most of the remaining SIIP (Brudno & Marczynski 1977). This type of storage/retrieval of memory is failure-tolerant in the holographic system (Westlake 1970), which is deterministic and therefore useless for studying the mammalian brain in which the storage-retrieval of memories and other functions must be probabilistic to "protect" the living organism from mindless robot-like behavior. This conclusion is supported by the fact that the statistical distribution of SIIP in our model is probabilistic (cf. Brudno & Marczynski 1977). This is shown by the fact that in each SIIP matrix, the SIIP can be divided into two groups: (1) the essential, without which a matrix completion process in case "damage" would not be possible; and (2) the "redundant" SIIP whose statistical distribution can be deduced from distribution of the essential SIIP. As an example, in a matrix of 8 possible permutations of trigrams, that is, SIIP composed of three inequalities, 5 SIIP are essential and 3 are redundant. In a total of 64 possible hexagrams, 31 are essential and 33 SIIP are "redundant." When making cognitive/behavioral decisions, the mammalian brain "chooses" patterns from both pools (Brudno & Marczynski 1977; for in depth discussion of these topics, see Marczynski 1983).

Our biologically explored autoassociations in SIIP matrices seem to be powerful mechanisms for storing and retrieving memories, considering the availability of large numbers of neurons that may be recruited into cognitive functions of a healthy mammalian brain. These functions can be amplified by "training" the autoassociation network to handle heteroassociations, that is, input from the heteromodal sensory systems (cf. Churchland & Sejnowski 1992) which, in theory, have "knowledge" of virtually all cognitive sensory-behavioral transactions that occur in the brain (cf. MacKay 1966; cf. Marczynski 1993).

The autoassociative memory represented in statistical SIIP distribution differs from that of Anderson (1972) which is based on the mean neuronal firing rate. The SIIP autoassociative memory also differs from that of the celebrated Hopfield's network model (1982) and from the Boltzmann machine thermodynamic model (cf. Hinton & Sejnowski 1986). These models seek the lowest energy level, a "motivation" which is hardly compatible with biological systems. Moreover, all operations are based on the mean neuronal firing rates. Our SIIP associative model also differs from that of Kohonen (1984) who uses learning rules based on physical deterministic laws of neuronal mean firing rates, a criterion that we rejected as misleading. Our biologically inspired SIIP model, by definition, uses inequalities of neuronal firing and therefore emphasizes nonlinear neuronal behavior as the carrier of information. On the other hand, the Hopfield model and the Boltzmann model regard the non-linear neuronal behavior as a "nuisance" to be ignored by "squashing" the non-linear data through the sigmoid function where the discrete temporal properties of spike trains are ignored and converted into a "static form of nonlinearity" (Hertz et al. 1991). Using the metaphor of a spoken language, this procedure is equivalent to trying to understand the meaning of a spoken word by averaging the pronunciation of its vowels and consonants!

Spike interval inequality patterns (SIIP) correlate with subject's cognitive-motor functions. For instance, in cat's transitions from an aroused state to a relaxed wakefulness, slow wave sleep (SWS) and REM sleep, are often not correlated with changes in the mean neuronal firing rate. However, the most interesting are the inversions in statistical distribution of patterns with reference to the stochastic model (Marczynski et al. 1984; 1992). This phenomenon is observed in about 6% of neurons monitored in the centrum medianum nucleus and in the nucleus reticularis of thalamus. The example from the latter region is shown in Figure 2 in the middle part of Figure 2, all 64 permutations of hexagram patterns are printed vertically and numbered from left to right. Each pattern should be "read" vertically from the bottom to the top sign. The chi square ordinates for each behavior measure pattern departures from the stochastic model. The black circle columns and the open circle columns represent respectively excessive emissions and deficits of patterns occurrences with reference to the stochastic model. The spike trains were monitored during cat's four behavioral states: vigilant, attentive, relaxed (REL), and slow wave sleep (SWS). The attentive state was caused by introduction to the experimental chamber of a transparent box containing a live mouse.

The overall impression from Figure 2 is that many patterns were emitted (filled circle columns) and others were suppressed (open circle columns) with reference to the stochastic model. The REL and SWS episodes show inversions in distribution of patterns, particularly obvious by comparing the attentive state with SWS, where the emitted and suppressed patterns changed to suppressions and emissions respectively. On the left of Figure 2, the four vertical scales of chi square values measure departures of pattern occurrences from the stochastic model. The legends on the right for each behavioral episode show: N = the number of spike intervals in a sample; MR = means neuronal firing rate; the chi square values without subscripts measure the sum total discordance of pattern distribution from the stochastic model. In the REL and SWS sample, the chi square values with subscripts i(28) and i(57) respectively represent values only for patterns that inverted their direction in deviating from the stochastic model. However, the most important message of Figure 2 is that the pattern inversion magnitudes are not random, but correlated, that is the larger emissions in an attentive behavioral state tend to be followed by proportionally large suppression of the same pattern in SWS, and vice versa. These relationships were quantified at the bottom of the figure by plotting the roots of chi square statistics. Plotting the "attentive" sample 18 emissions (Att)e versus SWS the same pattern suppressions (SWSs), a high degree of correlation was found ($p < 0.005$). An even more significant correlation was found by plotting (Att)e versus (REL + SWS)s which resulted in a correlation coefficient $r = 0.75$; $p < 0.005$. However, no significant correlations were found for (Att)s going to (SWS)e, nor (Att)s going to (REL+SWS)e.

The inversion phenomenon indicates that the occurrences of select SIIP are homeostatically controlled, most likely by the use-

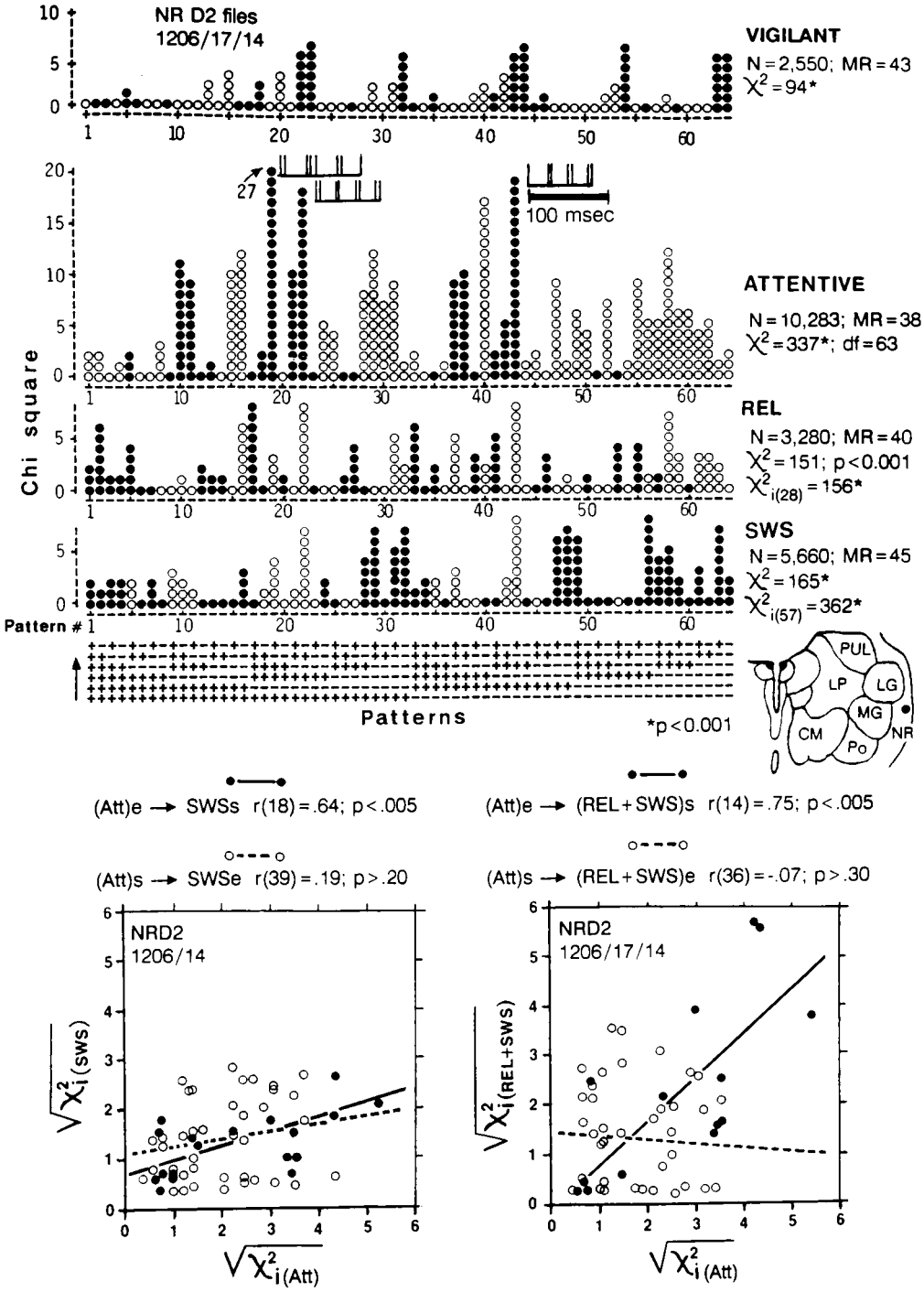

Figure 2 (Marczynski). Single neuronal firing patterns during four behavioral states.

dependent desensitization of receptors to select transmitters/modulators which are specifically distributed on neuronal somata and dendrites, the latter, however, having most powerful influences on neuronal firing patterns (Mainen & Sejnowski 1966), as if modulating neuronal information coding by controlling SIIP "vocabulary." In many instances, the inversions in statistical distribution of patterns occurred without significant alterations in the mean neuronal firing rates (MR), indicating that SIIP seeking their AAP are more important than MR.

Another feature of the stochastic SIIP model is that it seems to be physiologically natural, because statistically significant and behaviorally correlated SIIP have been monitored in behaving felines from the CA1 region of the dorsal hippocampus, the pulvinar nucleus, the thalamic reticular nucleus, the centrum medianum, the visual cortex, and the feline nucleus abducens. The important finding was that during SWS there are always episodes during which single neurons generate virtually perfect SIIP stochastic distribution that has been conceived on the basis of theoretical assumptions of SIIP statistical distribution (Brudno & Marczynski 1977; Marczynski 1983; Marczynski et al. 1984).

As argued by Farley (1966), the main problems in constructing biologically inspired connectionist models of cognitive functions is to provide the system with the capacity to generalize and interpret newly encountered environments on the basis of previous experience. The Hopfield model (1982) and its extension, the Boltzmann machine of Hinton and Sejnowski (1986) have little or no a

priori knowledge of what might be the consequences of particular "behavior." These models are "mindless" thermodynamic machines, which if turned on, begin to function from the "tabula rasa" baseline. A question arises of whether the stochastic SIIP distribution has memory of its own which could be utilized by neurons for encoding and transmitting information. The answer to this question is surprisingly positive and it is exemplified by the fact that the transition probability of a pentagram (+-++-) going to a hexagram (+-++--) equals 0.022024 which is greater than the probability of a pentagram (--++-) going to a hexagram (--++--) which equals 0.014987, despite the fact that the "history" of both pentagrams, going back four steps, is identical and differs only in the first event (Marczynski 1983; Marczynski et al. 1982). Less distant spike events have proportionally stronger influences on SIIP probabilities. Thus, the SIIP stochastic model of neuronal firing is sensitive to the history of events that can be formally defined as memory. Undoubtedly, this memory is generated by sequential inequality testing of spike intervals, because the process of comparing sequential intervals is advancing in a nonsaltatory manner, that is, one spike interval at a time. Whether or not brain neurons use this memory, remains to be investigated.

Sleep can be related to memory, even if REM sleep is not

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Abstract: As reported by Vertes & Eastman, convincing evidence rules out any role for REM sleep in memory consolidation. However, they do not provide convincing evidence for their claim that sleep in general – as opposed to REM sleep per se – has no influence on memory consolidation. Recent correlational data suggest that the number of NREM/REM cycles is associated with performance on a verbal recall task.
[VERTES & EASTMAN]

The target article by **VERTES & EASTMAN** reviews an impressive amount of evidence that convincingly rules out any role of REM sleep on memory consolidation. It is correct, for example, to consider the results of deprivation studies in animals as biased by strong artifacts. In post-learning deprivation studies, animals deprived of REM sleep are overly stressed and their poor performance after REM deprivation might reflect a problem due to performance decrements, rather than to poor memory consolidation. Studies on REM deprivation in humans have the same flaw. Furthermore, compelling evidence about the role of REM sleep on memory consolidation cannot be derived from studies showing an increase in REM activity following a significant and enriched learning situation during waking. In this case the design does not provide a good test of the hypothesis that memory is consolidated during REM sleep, not only for the reasons put forward by VERTES & EASTMAN, but also because the method of testing the hypothesis is logically flawed. These are confirmatory tests; they do not consider potential alternative explanations of the data, some of which are accurately summarized by VERTES & EASTMAN.

Thus, overall, the article makes a good and important point, that is, there is no convincing evidence on the role of REM sleep in memory consolidation. But while the arguments concerning sleep are convincing, those concerning memory are much less so. It is arbitrary and wrong to assert that "memory requires consciousness." This is clearly not correct, since there is at least one type of memory (i.e., implicit memory) that does not require consciousness (for a review, Schacter et al. 1993). As for particular memory processes, studies on subliminal processing indicate that to a certain extent even encoding can occur successfully outside of consciousness (Draine & Greenwald 1998; Merikle et al. 1995). Retrieval typically requires consciousness, but the example of implicit memory shows that this is not always the case. Consolidation does not require consciousness. The idea that memory consolidates only through conscious rehearsal has long been abandoned. It is now accepted that consolidation can occur out of consciousness.

Let us distinguish then the concept of consciousness from the concept of a waking state. The claim of the authors should be rephrased as follows: Consolidation in memory cannot occur outside of the waking state, or, as they also claim, Sleep has no role in memory consolidation. But here they overstate their claim. Whereas the authors provide strong evidence against the role of REM sleep, they do not provide enough evidence on the lack of role of sleep in general on memory consolidation. The fact that REM sleep does not play a role in memory consolidation does not imply that sleep, as a whole, cannot play a role in memory consolidation.

Sleep cannot be studied as a juxtaposition of single stages, independent one of another. Sleep is a highly interconnected structure, or organization, where a modification in one stage can strongly influence the others. This has at least two consequences. First, it is difficult to conceive that by disrupting REM sleep the rest of the sleep activity in an individual remains unaltered, and this represents an additional criticism of the REM deprivation studies reviewed by **VERTES & EASTMAN.**

Second, and more important, the organization of the structure of sleep – rather than individual sleep stages – might affect the degree to which materials are consolidated in memory. Sleep cycles (NREM/REM cycles) can be an operational definition of sleep structure or sleep organization. An initial demonstration that the integrity of the structure of sleep as a whole plays a role in memory consolidation comes from a recent correlational study that still needs to be replicated in different populations (Mazzoni et al. 1999.) In this study, it was found that while REM sleep had no significant bearing on memory performance of a list of words learned just before sleep, two indices of sleep organization did. One was the total number of NREM-REM cycles, the other was the proportion of sleep spent in NREM/REM cycles over total sleep time (TST). A NREM/REM cycle was defined as a portion of sleep that contains Stage 1, 2, 3, and 4, followed by a period of REM sleep, without any sizable intervening spontaneous awakening. These data suggest that memory consolidation may not be a function of a single stage of sleep, but rather can be a function of the degree of sleep organization.

The illusory function of dreams: Another example of cognitive bias

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Abstract: Patterns of dream content indicating a predominance of themes relating to threat are likely to reflect biases in dream recall and dream scoring techniques. Even if this pattern is not artifactual, it is yet reflective of threat-related biases in our conscious and nonconscious waking cognition, and is not special to dreams.
[REVONSUO]

REVONSUO presents an elegant argument for a functional model of dreaming consistent with the reasoning that form suggests function. However, when applying this type of reasoning to infer dream function, two problems exist. First, because of biases in dream recall and dream scoring techniques, we do not really know the true "form" of dreams, and without knowing their true form, we cannot usefully apply the argument of design. Second, even if we were to accept the premise that dream contents (and not just dream recall or dream scoring systems) emphasize threat-detection, we have no null model to test the hypothesis that threat-detection biases in dreaming cognition do not simply reflect threat-detection biases of our (conscious and nonconscious) waking cognition.

As **REVONSUO** and other evolution-minded theorists suggest, humans, like other animals, should have evolved a plethora of special threat-detection devices. The results of empirical research leave no doubt that this is, in fact, the case. Psychologists have documented a variety of threat-related cognitive biases at a variety of conscious and subconscious levels (e.g., Cosmides & Tooby 1992; Davey 1995; Hansen & Hansen 1988; 1994; Mathews & MacLeod 1985; McNally 1987; Mealey et al. 1996; Occhipinti & Siegal 1994; Ohman 1993; Shoemaker 1996; Spinks & Mealey, submitted). Survival-related threats are selectively perceived, attended to, remembered, and discussed.

This fact confounds our efforts to interpret dream content. Because dreams are elusive and dream recall is far from complete, dreams that are remembered are bound to be those that are particularly salient (Cohen 1974). Which dreams are most salient? Like waking stimuli, those that contain powerful emotions and elements of threat. Comparison of dream reports obtained under conditions of (1) immediate post-REM awakenings, (2) daily dream diaries, and (3) free recall after long delay, suggests that the least salient (least interesting and least emotional) dreams are forgotten first and progressively, until only the most salient dreams remain in memory. These particularly salient dreams are the ones most likely to be reported in the bulk of research studies, providing a highly selective and non-representative sample. If the function of dreams is to be deduced from the form and content of dreams, then a much less biased method of dream reporting must be used (see e.g., Foulkes 1999; Merrit et al. 1994).

Besides our selective recall of dreams, our existing cognitive biases for attending to threat also result in biases in categorizing dream content. Schredl and Doll (1998) showed that external raters impute relatively more negative emotion to dreams than do dreamers themselves, as a result of paying selective attention to the negative and ignoring the positive elements of dream reports. Furthermore, the most commonly used dream content scoring system (Hall & Van de Castle 1966) is itself biased for picking up on negative rather than positive emotions. The five emotion categories in this scoring system are anger, apprehension, happiness, sadness, and confusion, three of which are clearly negative and only one of which is clearly positive; the remaining "emotion" (confusion) is arguably more likely to be perceived by most people as negative than positive. It is hard to avoid the conclusion that dream content is full of threatening images and emotions when a majority of available scoring categories have negative valence.

Now, it may be that the Hall and Van de Castle system simply reflects the content of dreams rather than constructing it. Alternatively, perhaps the system reflects an extant bias in the English language. Spinks and Mealey (submitted) have shown that English (and, it seems, other languages) is biased to facilitate the labeling of threats: categorizing trait adjectives on dimensions of dominant-subordinate and prosocial-antisocial, they found far more adjectives describing dominant, antisocial people than people in any of the three other quadrants. Furthermore, it is quite possible that this bias of descriptive language in turn, reflects an actual bias of our emotions and our brain. There is strong consensus that the "primary" (cross-cultural and instinctive) human emotions are anger, fear, sadness, happiness, and disgust (e.g., Ekman 1971; Izard 1991; Panksepp 1982; 1999; Plutchick 1980). These are very close to the categories in the Hall & Van de Castle system and, of these, only one has positive valence while the other four are clearly negative. Indeed, anger, fear, and disgust seem to be phenomenological experiences designed specifically for threat-detection.

What this means in the context of a search for the function of dreams is that even if dreams are biased toward threat-related content, and this bias is significant in comparison to a baseline of everyday experience, it still may not reflect a special attribute of dreams. If that is the case, then the argument from design no longer holds.

Tooby (1999) asks "How do you test whether something is an adaptation?" His answer? "To establish something as an adaptation, all one needs to do is to collect evidence that justifies the rejection of the hypothesis that the structure arose by chance (with respect to function.)" With dreams, we cannot do this. Even if there is a bias in dream content, as **REVONSUO** argues, we cannot reject the hypothesis that this bias is a by-product of other adaptive biases in our cognition, and is not specific to dreams.

Indeed, as Tooby continues "hypothesis testing is based on statistical inference, and the probability of obtaining the observations that support the hypothesis if the hypothesis were true, as compared to the probability of obtaining the same observations if the hypothesis were not true." With respect to testing a hypothesized function of an organ or process, "(t)his method involves comparing the problem-solving quality of a hypothesized adaptation with the problem-solving properties of other possible alternatives" (p. 3). I have not been convinced that the probability of observing biases in dream content is any different whether **REVONSUO**'s model is true or whether REVONSUO's model is not true; we do not have an appropriate null model that includes the effects of known (non-dream related) cognitive biases. Furthermore, the problem-solving abilities of dreams (if they exist) are clearly inferior to the problem-solving abilities of other conceivable alternatives (Blagrove 1992a), suggesting that dreams are not a product of design after all (see also Flanagan 1995).

I suggest that dream recall is the end product of the serial treatment of REM-sleep neural processes through successive stages of cognitive processing, and as such, that it reflects all the biases of each of those sequential steps. This view is clearly consistent with HOBSON's activation-synthesis model as presented in this issue and elsewhere. I also suggest that our relentless desire to attribute function to dreams is simply one more manifestation of the same evolved cognitive processes that, as a byproduct of their otherwise effective heuristic functions (Gigerenzer & Todd 1999; Kahneman et al. 1982), attribute meaning to other meaningless patterns and create dreams in the first place.

A more general evolutionary hypothesis about dream function

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Abstract: Revonsuo's evolutionary theory of dream function is extremely interesting. However, although threat avoidance theory is well grounded in experimental data, it does not take other significant dream research data into account. The theory can be integrated into a more general hypothesis which takes these data into consideration.
[REVONSUO]

REVONSUO provides us with an original example of psychological dream analysis in terms of dream contents and behavioural adaption. His target article addresses a fundamental question related to dreaming – the function of our nocturnal representations – and proposes a very interesting theory, at once plausible and well grounded within theoretical arguments and experimental data from dream psychology and neurobiology. REVONSUO develops his arguments so skillfully that at first glance they seem convincing. Unfortunately, however, some of his assertions are questionable and his threat simulation theory remains conjectural and cannot explain the existence of the majority of our dreams. I accordingly suggest some complementary hypotheses.

REVONSUO's theory is based on two general ideas that I would be ready to accept. First, it is certain that if dreaming has an adaptive function, it should have enhanced biological fitness in the environment of our ancestors. Second, as Jung (1933) demonstrated with his idea of archetype, it is possible to establish a correspondence between the dream content of contemporary people and the needs and fear of humankind in the remote past. Unfortunately, however, the threat simulation theory of dreaming is based

on other ideas that are not convincing. Let us consider the six propositions which summarise the theory.

Two of them are undoubtedly correct. First, the recall of our own dreams as well as the cognitive study of large samples of dreams show that they consist of an organised and selective simulation of the perceptual world (Proposition 1). Second, the threat simulations produced in dreams are indeed perceptually and behaviorally realistic rehearsals of threatening events (Proposition 4).

REVONSUO's other propositions are clearly overgeneralizations. It is true that dream consciousness is very adept at simulating threatening events. However, this does not mean that simulating threatening events is *the* specialised function of the dreaming process, as seems to be suggested in Proposition 2. If the frequency of topics dealt with in dreams is taken into account, it can also be hypothesised that dream consciousness is specialised for simulating human relationships, novel situations, highly desirable future events, and so forth. Another overgeneralization concerns the predominance of negative emotions in dreams. In fact, the majority of dreams are not accompanied by negative emotions. In the sample of 500 dreams studied by Strauch and Meier (1996), less than 30% of the dreams contained negative emotions. A similar overgeneralization can be observed in Proposition 6. It exaggerates the importance of threat representations activated by the experience of dangerous events, such as those that were frequent in primitive environment. Actually we know nothing about the frequency of nightmares experienced by our ancestors. As far as the dream content of Mehinaku Indians mentioned in the article is concerned, I suppose they were spontaneously remembered dreams. If this is the case, they constitute a biased sample of dreams. Everyone can remember a nightmare (at least for some time), while most people are unaware of an enormous quantity of more peaceful and mundane dreams that they could report if they were awakened during the night. For the same reason REVONSUO's numerous references to Hall and Van de Castle's studies (1996, etc.) are not pertinent.

The third proposition of the theory – according to which nothing but exposure to real threatening events can fully activate the threat simulation system – is false. Quite a number of people have frequent nightmares even though they have never been exposed to any particular danger. Depressed people, for example, are known to experience frequent nightmares which are due to inner psychological conditions rather than external causes. It must also be noted that many people drive too fast, cross streets outside pedestrian crossings, or practice dangerous sports, knowing that they may lose their lives during a moment of inattention. To my knowledge, these individuals do not have more frequent nightmares than cautious persons who avoid all danger. It is highly likely that those of our ancestors whose personality characteristics correspond to those of today's fast car drivers and dangerous sports lovers liked to go near wild animals, to swim through dangerous rivers, and attack enemies. They probably had no more nightmares than their remote descendants, however, and yet were adapted to these dangerous activities.

The most controversial proposition of threat avoidance theory, in my opinion, is No. 5, which states that the realistic rehearsal of threat avoidance skills in dreams can lead to enhanced performance. I quite agree with **REVONSUO** that motor actions represented in dreams might facilitate subsequent actual actions in the waking through implicit learning. However, no experimental data have shown that people's threat avoidance skills are improved after having nightmares. Second, mental images of motor activity can facilitate the subsequent performance of highly complex and non-instinctual movements like those involved in figure skating or golf. But threat avoidance "skills" represented in dreams, such as fleeing or hiding, are so elementary and instinctual that it is difficult to imagine how their representation could help to improve them. Nobody needs numerous rehearsals in order to know how to run away, to hide behind a rock, or to lie flat in the grass in presence of a danger.

In summary, the rehearsal function of dreaming threat avoidance theory is interesting because it draws our attention to the relatively high frequency of archaic content and threatening situations in dreams. However it can be criticised on the following grounds:

1. Most dreams deal with non-threatening situations. **REVONSUO**'s theory therefore cannot explain the functional significance of the majority of dreams.
2. Spontaneously remembered dreams constitute a biased sample of dream content.
3. Negative dream contents are not necessarily linked to actual dangers in real life.
4. The "skills" of threat avoidance in nightmares are so limited and instinctual that they hardly need any rehearsal.

I have suggested a more general hypothesis about the function of dreaming (Montangero 1999) which I would like to state here in slightly different terms. Dreaming is necessary in order to provide the mind with material to process during sleep. There are two reasons for this necessity to keep the mind active during the night. First, conscious reflection is so developed in the human species that if it were not busy with dream content, it might turn to external stimuli or to the current concerns of the sleeping person. This would tend to disrupt sleep. Dreaming thereby has the "guardian of sleep" function noted by Freud (1900/1955), but for different reasons. In this perspective, dreaming serves a biological function by permitting a full night's sleep, which in the long term favors the fittest physical condition in the daytime.

A second important benefit of dreaming is to maintain cognitive capacities such as encoding perceptions, making decisions, and planning actions. Specifically, if these capacities were not used for eight hours every twenty-four hours, they could be impaired in the long run and surely upon awakening. The threat avoidance function suggested by **REVONSUO** could therefore be included in this more general function of fundamental cognitive capacity preservations and mental vigilance.

Apart from this function of providing the mind with material to process, dreaming may have positive effects such as favoring the emotional balance by mastering or avoiding stress (Koulack 1991), or facilitating the discovery of novel solutions to problems upon awakening. However it must be admitted, as **REVONSUO** notes, that there are no conclusive experimental findings concerning these effects.

In conclusion, once it had endowed the human species with a high level of conscious reflection, nature had no choice. Cognitive processes involved in consciousness needed to produce evocations or simulations of reality when there was no need to encode perceptions or to plan actions (see, e.g., Foulkes & Fleisher 1975). The result was daydreaming, anticipation, and reminiscence in the daytime and dreaming at night.

Sorting out additions to the understanding of cognition during sleep

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Abstract: The target articles by Hobson et al., by Solms, and by Nielsen can be combined to further our understanding of the neurological basis of dreaming during REM and, notably, NREM sleep. Revonsuo adds to our understanding of the function of dreams from the perspective of behavioral biology but overstates its importance. Vertes & Eastman fail in their effort to discount memory enhancement as a function of REM sleep.
[HOBSON ET AL.; NIELSEN; REVONSUO; SOLMS; VERTES & EASTMAN]

HOBSON ET AL. do a thorough review of the disparate views of dreaming to show how new data from new technologies and approaches, including that of **SOLMS** using neuropsychological methods, is helping to resolve old questions, expand understand-

ing, and show where more research is needed. **NIELSEN** offers a very reasonable explanation of the occasional presence of dreams during NREM sleep. In the past, too few scientists have been willing and able to work concurrently in the areas of the phenomenology and neural mechanisms of dreams. These target articles do a commendable job of attending to these areas.

HOBSON ET AL.'s overall goal of exploring how dreaming can be explained in terms of brain physiology is presented as three subgoals. Although in such a long paper it is easy to find details that can be questioned or data that should have been included, tantamount is whether there are points of disagreement that might prove fatal to the theory or show a need for serious revision. Deficiencies that do not severely wound the author's intent are not as important, although some may be important in their own right. From this standpoint, I would like to comment on how well the authors succeed in achieving their stated sub-goals as relevant to their overall goal.

Sub-goal 1: That REM and NREM mentation differ from one another and, to lesser extent, from waking. The analysis of published data by **HOBSON ET AL.** rightly shows that it is at least premature at this time to consider REM dreaming and all NREM mentation identical. (**NIELSEN** concurs in this.) Likewise, the data are not sufficient to conclude that they are the same as waking mentation. While there may be some overlap in the characteristics of the mentation in the three stages, there are sufficient differences to at least allow the probability that they are fundamentally different. There is a real danger in proceeding as if REM and NREM mentation are the same, for which **SOLMS** seems to argue, if indeed they are not because of the difficulty of trying to discern the fundamental nature and purpose of a heterogeneous mix. Continuing to treat them as mostly unique would enable greater clarity in discovering more about their sources and purposes in the future. Should it turn out that they are indeed the same then it would be a relatively simple and unambiguous matter to combine the information gathered about them.

Sub-goal 2: That REM and NREM substantially differ physiologically (regionally, cellularly, and molecularly) from each other as well as from waking. This section of the paper also succeeds by marshaling evidence and drawing implications from it showing that REM, NREM, and waking are separable states.

Consistent with **HOBSON ET AL.**'s sub-goals 1 and 2 **NIELSEN** shows convincingly that dreaming sometimes occurs during NREM sleep concomitant with the occasional presence of certain physiological aspects of REM sleep. This (with acknowledgment of hints by others) he calls "covert REM sleep processes." This conclusion is given credence by HOBSON ET AL. and is compatible with **SOLMS**'s activation of forebrain circuits as the final common path for dreaming. It is more comprehensive, comprehensible, and sensible than any other explanation yet put forward to explain the occasional occurrence of dreams during NREM sleep. This explanation implies that non-dreamlike mentation of NREM must emanate from brain mechanisms different from those that produce dreaming.

Sub-goal 3: That the phenomenological and physiological data about REM can be comfortably and usefully integrated (in this case as a three-dimensional model) leading to greater understanding of how dreams are generated. **HOBSON ET AL.** succeed here as well. In doing so they make a contribution toward the understanding of a "cognitive neuroscience of brain-mind state." It should be noted that along the way they show how activation of areas of the brain important in emotion play a sighificant role in the shaping of dream content.

Insofar as **HOBSON ET AL.** achieved their three sub-goals, it appears that they have achieved their overall goal. Specifically, this paper culminates with an updating of the activation-synthesis model, especially to the synthesis portion, by incorporating new findings. These revisions incorporate fresh data and the theoretical implications derived from them (including some important new insights from **SOLMS** based on his neuropsychological study of dreaming in brain damaged patients). These successful revisions show that the activation-synthesis model is still the main contender among models seeking to show how dreams are generated. However, HOBSON ET AL. must make a better case of explaining SOLMS's data from patients who completely or nearly completely ceased dreaming following brain damage localized to two distinct forebrain regions. In over 99% of these cases the REM generators in pontine brainstem were spared and REM sleep was unaffected. Other patients show the apparent absence of any change in dreaming following pontine damage. SOLMS's data show REM sleep is not necessary for dreaming. However, as SOLMS points out, they do not eliminate the possibility that REM may be the sufficient and most favorable state for dreaming in the intact brain. Yet SOLMS still needs to explain how the forebrain areas become activated in order to instigate dreaming. It is possible that while these forebrain areas are preferentially activated by pontine influences during REM they may also be activated by non-pontine sources.

These models do what good models should do – summarize known relevant facts (including those most recently discovered), make some informed speculation, and point the way to future research. Additionally, the newer AIM model complements the activation-synthesis model by offering a testable explanation of the neurological basis of states of the brain/mind. Furthermore, the AIM model has broader implications for the understanding of human cognition beyond that of its focus on dreaming in the area of the phenomenology and neuronal basis of dreaming.

Nevertheless, I offer the following comments on two details in **HOBSON ET AL.,** while not crucial to the main thrust of the paper, are important in their own right. In section 2. 3. 4, point 6 they confuse access to stored memories with utilization of them when they reason that because dreaming during REM sleep is so infrequently affected by manipulation of pre-sleep experience it must have extremely poor access to recent waking memories. However, because the dream content does not frequently show use of such stored memories does not mean that there is no access to them. In fact, studies of "dream incubation" (cf. Cartwright & Lamberg 1992; Delaney 1998), HOBSON ET AL.'s dismissal of them notwithstanding, suggest that there is indeed access to stored memories during the dream state. An alternative to the explanation of HOBSON ET AL. of why so little of pre-sleep experience finds its way into dreams may be that the agenda of dreams focusing on recent, waking emotional concerns ignores most experimenter-imposed pre-sleep experiences. As both **SOLMS** and **REVONSUO** point out, preferential activation of the limbic system during REM reactivates the neural networks containing these emotional memories that then play a major role in determining the content of the dream. It may be difficult to manipulate these emotional memories by artificial pre-sleep experience compared to naturally occurring, emotionally relevant experiences.

In section 4. 1. 1, paragraph 2, **HOBSON ET AL.** state that brain activation is defined as the "mean firing frequency of brain stem neurons." Cortical EEG intensity also serves to measure brain activation. By making their exclusive assertion they too easily eliminate any explanations of dreaming having a cortical origin. Allowing for cortical measures of arousal opens the possibility of finding non-brainstem origins for brain arousal, such as **SOLMS** proposes.

REVONSUO'S paper about dreaming adds to our understanding of the functions of dreaming by showing its evolutionary roots. However it goes too far in claiming the "threat stimulation theory" is the sole explanation for dreaming. The argument presented in this paper hinges on its definition of function that emanates from the relatively new field of behavioral biology. The basis of this field is the belief that evolutionary success ("inclusive fitness") is the only primary function of any characteristic, including behavior, of any living organism. While this approach has yielded good insights into some of the ultimate reasons for the behavior of animals, it is not yet possible to fully accept the exclusiveness of its explanations.

Simply put, **REVONSUO**'s thesis – that all dreaming stems from perceived threats to bodily welfare as were experienced by ancestral humans – is too narrowly wrought. In contrast, the hypothe-

sis explains the same data if it is broadened to encompass the current emotional concerns of the individual as the focus of dream formulation. In ancestral humans such immediate, emotional concerns would indeed be bodily welfare and safety. So too, as this paper shows, for children and present day primitive people. However, in the contemporary Western world the emotional concerns of humans are more frequently psychological as shown by many studies (see REVONSUO's citations of Kramer 1993; Hartmann 1998). The attempts in this paper to discount such research are weak at best. For example, brushing off the research of Cartwright as simply correlation without causation does not hold because in some of her studies, Cartwright established causation when she actively trained some of her patients to change their dreams which resulted in significantly improved waking mood compared to the untrained subjects.

Portions of **REVONSUO**'s paper also reflect a misunderstanding of the application of some of the neurocognitive theories of dreaming such as those presented in **HOBSON ET AL.** That the source of dream generation is a random stimulation of brain structures involved in cognition does not mean that the resulting dreams are meaningless ("disorganized sensations and isolated precepts"). As stated in a later section of this paper, it is the brain areas that contain the individual's own thoughts, memories, emotions, sensations, motor movements, and patterns-of-cognitive-integration that are activated during dreaming. The initial activation may be random but the output has meaning for the individual. (The attempt to dismiss this possibility based solely on Penfield's memory research [Penfield 1975] does not work because Penfield's interpretation of his data as revealing true memories is no longer held to be valid.) Furthermore, as REVONSUO states, recent research has shown that the areas of the brain involved in emotions are preferentially activated during dreaming thus suggesting a primary role for emotions when dreaming. If we add the assumption, as stated in another section of the paper, that those brain networks most recently activated when awake are the ones that are most likely to be activated even by random inputs, then dreams are more likely to contain recent emotional concerns and "day residue" of relevance to the dreamer.

On a different note, **VERTES & EASTMAN** unconvincingly endeavor to directly dismiss the research that shows memory is enhanced during REM sleep by (1) pointing to some studies that fail to show this effect and (2) by showing how some of the earliest findings may have been owing to the stress of the procedures. Apparently the authors were not convinced by their efforts because they then devote most of its pages to attempting to show theoretically why memory cannot be enhanced by REM sleep. This later portion of the paper is akin to proving, using engineering principles, that hummingbirds cannot fly, in the face of reports that they sometimes do!

Since the most critical portion of **VERTES & EASTMAN**'s paper, then, is the first main section, I will focus most of my comments on it. This section fails to make its case that there is no valid research showing that an enhancement of memory can occur during REM. First, the listing of reviews is decidedly one sided, ignoring several reviews of the literature that conclude there is such an effect (for example: Cohen 1980; Dujardin et al. 1990; Smith 1993; Tilley et al. 1992). It also dismisses the positive findings by stating that there is roughly an equal number of negative findings. But negative findings easily result when looking in the wrong place – such as in the wrong REM window – not always because there is nothing to find.

Second, **VERTES & EASTMAN** focus their criticism on the methods used in some older studies rightly showing that they may be confounded by stress. Other, more recent research showing positive results using better methods (for example, Smith's REM window studies), is dismissed by stating that no one has endeavored to replicate these findings and that the explanation for part of the findings (in this example, the shifting nature of the REM window) is unknown. However, the history of science is replete with examples of how well established knowledge originated from a single source; examples that, for a time, were not fully explainable. (It should also be noted that stress cannot explain the results of REM window studies because there was no detriment of tested behaviors resulting from REM deprivation in general; memory deficits only occurred when the deprivation was during the REM window.)

Third, some recent research is ignored. For example, the study of people in intensive language learning situations (DeKoninck et al. 1990a) in which the more successful students had an increase in REM percent but no increase in total sleep time. These subjects also dreamt in the language more and had more verbal communication in their dreams. In other human research, positive results tended to be obtained for tasks with affective importance to the subject and tasks that required the learner to structure the material and use divergent thinking. Negative results occurred when the material was unimportant to the subject, or already structured, or required convergent thinking. An example of this kind of research is a study by Pirolli and Smith (1989). In this experiment, subjects learned a difficult logic task and a simpler paired word task. Subsequently, one group of subjects slept through the night, a second group was totally sleep deprived, a third REM deprived, and a fourth NREM deprived. One week later they were tested on both tasks. All groups performed equally well on the simple paired word task but only those subjects without REM (REM deprived and total sleep deprived) did worse on the difficult logic task.

There is another line of research not recognized by **VERTES & EASTMAN.** During the retention interval following new learning, some subjects are allowed to sleep (usually nap) when they would get much of one kind of sleep (REM or NREM) but little of the other kind. Other subjects remain awake. A problem for such research has been controlling successfully for time-of-day (e.g., circadian rhythm) confounds of when the sleep occurs. Nevertheless, some of this research has supported the notion that REM sleep is beneficial for memory consolidation, but a few studies have concluded that NREM is more beneficial. For example, Scrima (1984) administered a complex associative memory task to narcoleptics. They were then allowed a 20 minute nap or were to remain awake for 20 minutes. Since narcoleptics have a high amount of REM napping, many of the naps were mostly REM. Recall was best after REM and worse after remaining awake, with recall after NREM intermediate between the two.

In addition, **VERTES & EASTMAN** dismiss statistically significant but small gains in memory during REM because they are inconsequential. Yet a 10% enhancement in memory can be far from trivial especially if accumulated night after night. Consider, for example, what a 10% enhancement of exam scores would mean for a university student.

A problem for "proposed function for REM sleep" of **VERTES & EASTMAN** is the contradiction posed by the lengthening of successive REM periods. The shortest REM period (the first one of the night) follows the longest period of SWS. If this theory is correct then it would seem that the REM period at this time would instead be of considerable length if its function were to keep the brain aroused. On a related note, while it is possible that the REM periods get longer during the sleep period because the need for alertness becomes more likely with the increasing probability of waking as the end of the normal sleep period approaches, it should be noted that REM sleep shows a strong circadian propensity (peaking in the early morning hours) regardless of the timing of the sleep period (cf. Lavie & Segal 1989). Also REM sometimes occurs during naps following little NREM sleep. Finally, while it is possible that a function of REM is to maintain CNS arousal, this does not, necessarily, eliminate additional functions of REM such as memory consolidation. Indeed, most animal systems have multiple functions.

In the end, **VERTES & EASTMAN** show the need for continuing research on, rather than outright dismissal of, memory enhancement during REM sleep while **HOBSON ET AL., SOLMS, NIELSEN,** and **REVONSUO** make significant contributions to the understanding of the sources of dreaming.

Dreams and sleep: Are new schemas revealing?

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Abstract: In this series of articles, several new hypotheses on sleep and dreaming are presented. In each case, we feel the data do not adequately support the hypothesis. In their lengthy discourse, Hobson et al. represent to us the familiar reciprocal interaction model dressed in new clothes, but expanded beyond reasonable testability. Vertes & Eastman have proposed that REM sleep is not involved in memory consolidation. However, we do not find their arguments persuasive in that limited differences in activity in REM and waking do not lend credence to the idea that memory consolidation occurs in one state and not the other. Solms makes an argument that dreams are generated from the dopaminergic forebrain based largely on pathological lesion studies in humans. We recognize that this argument has some intuitive appeal and agree with some of the tenets but we do not feel that the arguments are completely convincing due to the lack of anatomical controls, including symmetry and laterality. On the whole, there are interesting arguments put forward in these target articles but the evidence does not convince us that new vistas are opened. No Holy Grail of sleep here!

[HOBSON ET AL.; SOLMS; VERTES & EASTMAN]

The new reciprocal interaction model-reverie or revelation? A Hobson's choice situation and déjà vu all over again: Hobson et al. In **HOBSON ET AL.**'s lengthy discourse attempting to move toward a cognitive neuroscience of conscious states, one comes away with a remorphed reciprocal interaction model dressed in new clothes but is, in reality, old wine in new bottles. The basic REM circuitry is draped in a multiple neurotransmitter type organization, including a dab of autoreceptor neurobiology. This "updated" reciprocal interaction scheme has now gone the way of most studies on regulatory systems, that is, the newer models of regulatory systems are widespread (distributed), involve multiple neurotransmitter pathways (not just, for example, the old "classic" aminergic/cholinergic "simpler" systems), and special newer families of receptors, including autoreceptors. Generally these complex, multilevel regulatory systems (brain substrates) are not reasonably testable and **HOBSON ET AL.** do not suggest experiments to clarify outstanding issues.

The "new" Hobson model presented has, in our opinion, only nebulous connection to reality so that the real versus virtual REM sleep/dreaming complex does not emerge. Why do **HOBSON ET AL.** not develop the dopamine aspects of dream sleep regulation as broached in his long discourse? This is especially relevant given **SOLMS**'s views on a separate dopamine dream system entirely divorced from the REM brainstem mechanisms. And whatever happened to the hippocampal formation in this theorizing, given conclusive evidence of vigilance state-dependent gating of information flow through the hippocampal formation? How can this be totally ignored in the new "distributed model"? We should also remind Hobson that Hernandez-Peon et al. (1963) chemically mapped a cholinergic system that closely followed the limbic-forebrain pathways from the limbic midbrain. Since this issue is noted in the text (i.e., that Jouvet postulated such a pathway) we emphasize that Hernandez-Peon et al. actually mapped such a trajectory.

Let us give credit where is due. The original Hobson cellular neurophysiology of sleep cycle control was sound ground work for future studies. But that was only the scaffolding of the extended (distributed) system that has been coming into view over the past 20 or so years. It certainly has had its day, and invaluably so, but we cannot see much new in this re-review. Why codify the reciprocal interaction model so that the parts (pontine "generator," raphe, locus coeruleus) have become greater than the whole'? The overall claim here is that the "essential tenets" of the reciprocal interaction model has been strongly confirmed which, to us, appears self-serving given that this component is such a limited part of the extended REM sleep/dreaming complex.

To sleep, perchance to learn – aye, there's the rub! Vertes & Eastman. Two of the principal premises of the **VERTES & EASTMAN** article are, first, that the primary function of REM is the endogenous stimulation of the brain to maintain requisite levels of CNS activities throughout sleep. Secondly, that sleep involves basic biological functions whereas memory requires consciousness. Relative to the latter, we might well ask whether dreams are actually a form of consciousness. We don't see sound arguments to the contrary!

We do not find strong reasoning behind the view that the brain needs "requisite" levels of activity throughout sleep. **VERTES & EASTMAN** also postulate that theta serves memory function in waking but not in REM. Why so? We can imagine that without a great deal of extraneous "noise" seen in waking that such is tuned out in REM so that in this state processing could occur unencumbered. Might this help consolidation since theta is associated with selective diminished inhibition in the hippocampal formation in both waking and REM? Hence, why not assume theta involvement in memory functions in both waking and REM? The authors categorically state that the theta of REM is a "byproduct" of intense activity of the pontine region in REM sleep and thus may have no functional significance in REM. Why isn't waking theta a byproduct of similar activation? The authors also state, without proper documentation, that there is no mechanism in REM for selection and transfer of information to the hippocampal formation from other sources. Further, they state that since information in REM is chaotic, it can have no functional value. No clear distinctions are made as to theta quality so that it would serve memory function in waking but not in REM. Finally, the authors serve up, without adequate background reasoning and references, that REM is a mechanism to insure and promote recovery from sleep. This idea does not strike us as viable. Further, it is not intuitively appealing! The whole complex realm of REM components could not likely serve such a basic primary function as waking up the sleeping brain.

Dopamine in the dream machine? Solms. Are we really ready for a major paradigm shift, that is, that REM is controlled by pontine brainstem mechanisms whereas dreaming seems to be controlled by dopamine forebrain mechanisms? Is this a form of blasphemy against long accepted views that REM sleep is the physiological equivalent of dreaming'? How can dreaming be put forward as not an intrinsic function of REM? Only by separating it in space?

Evidence that dreaming is generated by dopamine circuits (particularly mesocortical and mesolimbic components), however, is somewhat soft. The author gives little or no proof of exactness of clinical lesions. Were these presumed to be bilateral (unlikely)? If unilateral, do they alter sleep only on the lesion side? Surely we cannot prove one-sided dreaming? **SOLMS** reviews work showing brainstem lesions leaving dreaming intact, whereas forebrain focal lesions result in cessation or near cessation of dreaming. The link between forebrain seizures and recurrent nightmares also does not constitute strong evidence that dopamine systems play causal roles in generation of dreams. It is suggestive and intriguing but certainly not causal!

SOLMS postulates that so-called "motivational mechanisms" (volition and adynamia) are essential for the generation of dreams. What reasoning is this based on? To us "motivation" is still the "phlogiston of psychology." **SOLMS** claims that the activation state "engages" the dopamine circuits of the ventromesial forebrain. What does this actually mean and how and where does such "engagement" occur? How do specific aspects of the REM state (NE and 5-HT demodulation) facilitate primary dopamine effects'? Is the assumption that stoppage of NE and 5-HT activity activate the dopamine system(s) in the ventral tegmental area and, possibly, the substantia nigra? There is no direct neurophysiological evidence of this that these commentators are aware of.

The relationship of the putative dopamine "dream-on" mechanism and the cholinergic "REM-on" mechanism of the reciprocal interaction model is not developed to any extent, thus leaving us without any viable link. Dopamine may well be part of the dream machine but relations with physiological REM processes remain elusive.

Critical brain characteristics to consider in developing dream and memory theories

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Abstract: Dreaming in sleep must depend on the activity of the brain as does cognition and memory in wakefulness. Yet our understanding of the physiological subtleties of state differences may still be too primitive to guide theories adequately in these areas. One can state nonetheless unequivocally that the brain in REM is poorly equipped to practice for eventualities of wakefulness through dreaming, or for consolidating into memory the complex experiences of that state.
[HOBSON ET AL., NIELSEN, SOLMS, VERTES & EASTMAN, REVONSUO]

Dreams. To discuss views on dreaming – its nature; whether dreams occur in both REM and NREM; and, if so, their degree of similarity – first requires discussion of the characteristics of the brain in sleep. We are of the mind, of course, that mental functioning requires brain functioning. We hasten to add, though, that the current view of how the brain functions may not match a future reality. The "computer" brain of today may be an analogy no closer to the brain's actual mode of operation than was the "telephone-line" brain of the past. Nevertheless, we believe that dreams will always be grounded in the physical workings of the brain.

The foregoing suggests an answer to the continuing debate on the nature of dreams or mental activity in non-REM and REM reviewed by **HOBSON ET AL., NIELSEN,** and **SOLMS.** NIELSEN suggests a way to resolve the conflict between the one-generator model of dreaming advanced by Foulkes and others and the two-generator model championed by HOBSON and colleagues: Covert REM processes can intrude into NREM to color mental activity like that in REM. Indeed, identifiable REM or "REM" events can appear in NREM as NIELSEN has reviewed. Foulkes (1997), however, has essentially rejected this idea, warning of a tautology if REM is defined to be present whenever dreaming occurs.

Returning to our introductory idea, may we suggest that both our traditional, "digital" staging of sleep/wake states and our understanding of just what neurophysiological processes equate with elements of cognitive processes are too primitive to resolve the debate one way or the other? Otherwise, it is impossible for us to believe that total absence of the pervasive aminergic activity seen in wakefulness and NREM, which is a hallmark of REM, as **HOBSON ET AL.** have reviewed, should not be reflected in a measurable difference in mental activity in the two sleep states. In our opinion, the AIM model developed by HOBSON and colleagues has considerable merit as a way to order one's thinking on the complexities of the state concept although we doubt that it will lead to an early truce.

A physiological difference between the two states that may ultimately bear on the problem, but ignored in the articles, is the profound alteration in hypothalamic regulation that is a feature of REM (Parmeggiani & Morrison 1990). One would think that such a dramatic change in regulation would, in some way, feed back into mental activity.

Readiness to move, although greatly suppressed, appears to be a feature of REM, while NREM is a quiescent state. **HOBSON ET AL.** noted that motor areas are highly active in REM although the background of atonia limits peripheral expressions to the intermittent brief muscle contractions that one sees in various striated muscles that result in limb and rapid eye movements. Pontine tegmental lesions in cats that eliminate the usual muscle atonia of REM reveal an impetus to move in REM (Henley & Morrison 1974; Jouvet & Delorme 1965). The movements are expressed as well organized behavior. However, no organized behavior emerges in NREM, another clear difference in the two states.

Yet, the elaborate behaviors observed in REM without atonia (REM-A) may not be regarded as a true expression of normal brain activity unencumbered by muscle paralysis, for the brain has been damaged. Furthermore, behaviors observed depend on the sites of the lesions in a very predictable way (Hendricks et al. 1982). Also, the animals in wakefulness have distinct abnormalities. In a study of activity during wakefulness of cats exhibiting REM-A in sleep (Morrison et al. 1981) found that all of them had a significant increase in exploratory locomotion.

Further, aggression during REM-A only appears in those with particular rostral lesions (Hendricks et al. 1982) and has the characteristics of predatory attack (Leyhausen 1979). In our study of 28 cats, eight expressed predatory attack behavior, not affective defense during REM-A; but the six that were also aggressive in wakefulness demonstrated their release of aggressive tendencies as affective defense (Morrison 1979). Unilateral lesions of the central nucleus of the amygdala also released aggressive behavior that differed in a similar way in wakefulness and REM-A (Zagrodska et al. 1998). Although these cats exhibited predatory attack during REM-A, they showed no increase in predation when tested during wakefulness but were very aggressive toward conspecifics. The different expressions of aggression most probably reflect the great reduction in sympathetic tone and hypothalamic control during REM in cats (Parmeggiani & Morrison 1990).

The characteristics of REM-A we have described, confounded as they are by brain damage, lead us to doubt that the behaviors observed serve as evidence that REM is a period when waking behaviors are being practiced. However, the behaviors certainly are consistent with the idea that the brain in REM is most like the brain in very alert wakefulness when an animal orients (Morrison 1979; Sanford et al. 1993). But practice for the realities of a stressful existence during REM, whether by cats, whatever their capacity for mentation, or by humans during dreams as **REVONSUO** proposes, would seem to be severely hampered by the absence or alteration of critical regulating systems of the brain during REM.

Both **HOBSON ET AL.** and **SOLMS** provide diagrams that suggest circuitry in the forebrain underlying dream elaboration. These are based on both lesion and imaging data. It is well to keep in mind that activity in the forebrain very likely plays a key role in maintaining, and most certainly in initiating, REM. Morrison and Reiner (1985) first emphasized that the important decerebrate experiments of Jouvet (1962) focused excessive attention on the caudal brain as the site of initiation of REM. Most certainly much has been learned as a consequence of this focus, but at the same time the forebrain was forgotten as a site also important for REM in *intact* individuals. Decerebrate cats are inordinately predisposed to enter a REM-like state following all sorts of strange stimuli: insertion of rectal thermometers, passing of stomach tubes, and pinches (Jouvet 1964). Morrison and Reiner (1985) reasoned that decerebration substituted for the processes in NREM that led to the suppression of hypothalamic control we have mentioned earlier. Now, much needed attention is being paid to the forebrain with regard to initiation and maintenance of REM (Morrison et al. 1999), which should feed in to further elaborations of dream theories.

Memory. The case for a role for REM in learning and memory consolidation appears, for the most part, to be built on somewhat tenuous correlational relationships between REM occurrence and indicators of performance. **VERTES & EASTMAN** present, in our minds, compelling arguments questioning the temporal relationship between the occurrence of REM and memory consolidation. While we make no claim to expertise in learning and memory, we have recently become interested in a learning paradigm, fear conditioning, and its effect on REM. In essence, this is a classical conditioning procedure training an animal to make an association between a neutral stimulus (cue) or situation (context) and an aversive stimulus (usually shock). Explicitly cued fear conditioning produces long-term potentiation-like changes in the lateral amygdala (Rogan et al. 1997), and contextual fear conditioning involves the hippocampus (Desmedt et al. 1998). Given arguments that learning is associated with increases in REM (Ambrosini et al. 1993; Smith 1985; 1995), one would have expected increased REM following fear conditioning. Far from resulting in enhanced REM,

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

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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 attachment 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

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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

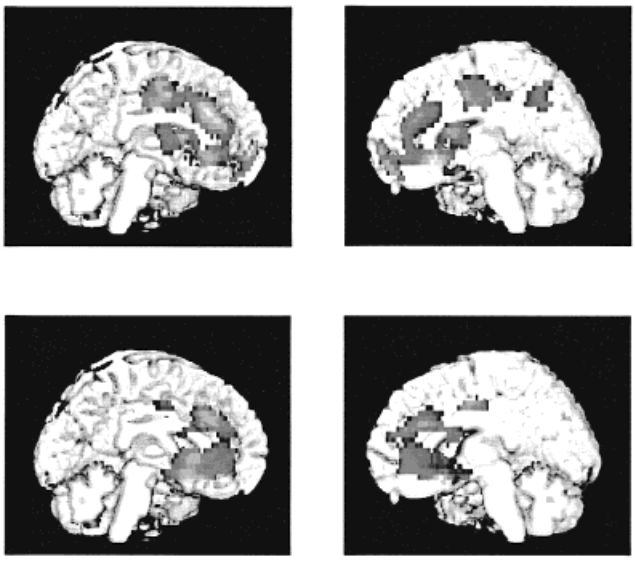

Figure 1 (Nofzinger). Bilateral mid-sagittal sections showing REM sleep minus wake activations. Two figures on top demonstrate regions activated in four healthy controls at each of two time-points separated by 12 weeks. Two figures on bottom demonstrate regions activated in six independent healthy subjects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

functional brain imaging study was repeated on two occasions separated by 12 weeks. The figures on the previous page show that the pattern of activation present in the original subjects is also present in an independent group of subjects at each of two time-points. The areas of activation are remarkably similar to the areas of interest diagrammed by the **HOBSON ET AL.** in their Figure 7: "Forebrain processes in normal dreaming – integrated model." They include activation of their anterior paralimbic group, Zone 3, beginning in the ventral striatum and continuing into sub-, pre-, and supragenual anterior cingulate cortex with some medial prefrontal cortex; bilateral activation of the basal ganglia, Zone 5; bilateral activation of inferior parietal cortex (not shown), Zone 9; and perhaps difficult to see, activation of basal forebrain, Zone 2. These additional findings support the original findings on which the HOBSON ET AL. integrated model are built.

Even in this replication study, however, we did not see clear evidence for a change from waking to REM sleep in the pontine reticular formation, an important structure in a bottom-up approach to REM sleep. Perhaps the absence of a change simply reflects that this region is similarly active between waking and REM sleep, both states of cortical activation. Perhaps this is a limitation of the method in which there are spatial resolution constraints of scanners and in which there is significant smoothing of images that is performed across subjects to control for inter-subject regional variations in brain morphology. These factors would preclude our ability to see small pontine nuclei in the PET images. In an attempt to address some of these issues, we then drew regions-of-interest (ROIs) in the pontine reticular formation across several axial planes, then compared the activity in these regions across behavioral states. As expected, a clear drop in functional activity in the pontine reticular formation was noted from waking to NREM sleep, then a return of the waking level of activity was noted following entry into REM sleep, although not to levels exceeding that of wakefulness. This pattern of change paralleled changes in global metabolism across the entire forebrain, supporting at a metabolic level distinctions in global forebrain function and pontine reticular formation function across these unique behavioral states.

Still, we remain puzzled by some preliminary observations of our REM sleep imaging data when we explored the relationships between the pontine ROIs and other brain structures thought to play a role in an integrated model of forebrain processes in dreaming. We were reassured to find a positive correlation between relative metabolism in the pontine reticular formation and that of the thalamus (G). We also found a positive correlation between relative metabolism in the pontine reticular formation (P) and that in (0) primary visual cortex (pons × left occipital cortex correlation = .928). Do these relationships represent a metabolic correlate of PGO wave generation? Also of interest is that functional activity in the amygdala (A) paralleled activity in the pons, thalamus, and occipital cortex. This would be supportive of more recent efforts to more directly link amygdala function, and presumably its role in emotional behavior, with REM sleep.

In contrast, our conceptual model of forebrain function during REM sleep began to break down when we explored the relationships between this PGO-A system and that of the anterior paralimbic REM activation axis that is becoming a signature of forebrain function during REM sleep in human functional brain imaging studies. Relative activity in the anterior paralimbic system was negatively correlated with that in the PGO-A REM system (pons × left pregenual anterior cingulate correlation = −.77; pons × right pregenual anterior cingulate cortex correlation = −.945). How can this be? Shouldn't functional activity in the anterior paralimbic system parallel that in the pontine reticular formation if REM sleep is generated by the brainstem with consequent forebrain manifestations? Similar preliminary studies in depressed subjects help clarify this to some degree. When we additionally looked at the relationship between basal forebrain and hypothalamus function in relation to the pontine reticular formation and anterior paralimbic system in depressed patients, we found that functional deficits in basal forebrain and hypothalamus paralleled those deficits in the anterior paralimbic system. In contrast, in healthy subjects, there was more of a direct relationship between functional activity in the basal forebrain and hypothalamus and the reticular formation consistent with the notion that these structures may be rostral extensions of an ascending activation system. The findings in depressed patients suggests that there may be unique functional roles served by a more generalized posterior ascending activation system from the pontine reticular formation, through thalamus and on to cortex and a more specific anterior paralimbic activation system from the basal forebrain and perhaps hypothalamus in the service of mediating adaptive or motivational behavior. Given the more selective activation of anterior paralimbic structures over occipital cortex during REM sleep in human imaging studies, it may be that the more selective anterior activating system is preferentially activated during normal, healthy REM sleep.

Does the pontine reticular formation play a role in triggering function in this anterior ascending system? If the primary forebrain function during REM sleep is the maintenance of anterior paralimbic forebrain activity, it may be that the inverse relationship between pontine reticular formation activity and the anterior paralimbic system represents the efficiency of the system. The easier it is for the ascending system to engage anterior paralimbic activity, the less work it has to do. In support of this, with increasing severity of depression, depressed patients show increasing difficulty in activating the anterior paralimbic system from waking to REM sleep. Concurrently, they also demonstrate increasing relative activity in the pontine reticular formation during REM sleep with increasing depression severity. This raises the possibility that the increased REM sleep production in depressed patients reflects a compensatory brainstem drive of more posteriorly located ascending activation in response to behavioral deficiencies in anteriorly located ascending activation mediated through the basal forebrain and into anterior paralimbic structures. These preliminary observations, however, await confirmation in larger sample sizes as well as replication as we have now done for the findings in healthy subjects.

In closing, we agree that the recent sleep imaging work in humans is important and we are glad that the findings have helped shape models regarding forebrain processes in dreaming. We also recognize the inherent limitations of these methods, primarily in terms of spatial and temporal resolution in more clearly identifying the temporal sequencing of regional brain activity in proximity to the time that the functional activity is occurring at the electrophysiological level. We feel that future developments in this area will only come via collaborative interchanges between the preclinical research labs and the labs performing the human studies as each can amplify, extend, and provide meaningful interpretation of the others' data. Future refinements in imaging technology providing increased spatial and temporal resolution will undoubtedly make these early human sleep imaging studies obsolete and leave us with richer datasets on which to further refine these models of human brain function in relation to dreaming.

Toward a new neuropsychological isomorphism

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Abstract: The deactivation of the dorsolateral prefrontal cortex is likely to be essential for generating some characteristics of the dream. The heterogeneous nature of NREM sleep makes it difficult to assume that there are different NREM dream triggers. Different cortical and subcortical neurophysiological conditions modulate mentation both in waking and in sleeping without any specific direct triggering factor.
[SOLMS]

SOLMS's is a target article of unquestionable elegance. The conclusions he draws from the analysis carried out on manifold sources allow us to free oneiric activity from the neurophysiological processes of REM sleep. Besides "rehabilitating" the cortex in the production of sleep mentation, SOLMS's model gives dreams back the dignity of thought. Dreaming becomes the active product of cognitive operations taking place through the intervention of areas that are normally delegated to control complex functions. The dream is no longer an epiphenomenon derived from the assignment of meaning by the forebrain to casual stimuli coming from the mechanisms that regulate the REM/NREM cycle alternation. What is puzzling is that SOLMS is still looking for specific anatomic-functional mutual relationships, even in dealing with mental processes as complex as oneiric thought.

A second point on which we do not agree is SOLMS's conclusion about the deactivation of the dorsolateral prefrontal cortex. This cerebral area carries out the very highest cognitive processes, regulating self-monitoring and strategic control over functions. Neuroimaging studies have shown that this area is deactivated during sleep and neuropsychological observations have shown that a lesion here does not affect oneiric activity. SOLMS concludes from this that "the dorsolateral prefrontal cortex is inessential for dreaming sleep." Some peculiar characteristics of dreaming (e.g., inefficiency in reality testing, the lack of strategic control over the course of thought, the frequent presence of temporal-spatial distortion) might be the consequence of weak and ineffective control by the executive functions of the dorsolateral prefrontal cortex. The hypoactivation of this area may subserve the specific cognitive organization of oneiric activity: this is hardly "inessential."

Dream production may accordingly be the result of complex patterns of cerebral reorganization depending on CNS's specific functional equilibrium (activation-hypoactivation) and to specific dream content. A point that requires further clarification from SOLMS is "the necessary presence of triggers" for dream production. If the pontine cholinergic mechanism (REM-on) triggers a sudden modification of the electro-encephalographic pattern, the NREM trigger is not well specified. NREM sleep in fact consists of heterogeneous stages with no possible precise boundary between them; changes are progressive rather than abrupt.

SOLMS should clarify what he means by NREM trigger. At present, one frequent question answerable only approximately, concerns the continuity/non continuity of oneiric activity during sleep. If separate, stage-specific NREM triggers were documented, one would expect from SOLMS's model, that the oneric activity in NREM depends on them. This would support the hypothesis that sleep mentation is not continuous. In contrast, the absence of specific state-dependent triggers would free dream like activity from specific neurophysiological layers, favoring the hypothesis that it may occur in any sleep stage, even in a continuous way.

SOLMS does a broad review of neuroanatomical, neurochemical, and neuropsychological data; however, he disregards psychological data somewhat. He uses only a small portion of REM-like dreams in NREM to be able to support his hypotheses. To identify a neurophysiological layer that acts as NREM trigger, SOLMS emphasizes the high percentage of dream recall in SO-stage 1 and in the morning awakenings. These involve some physiological activation that is close to wakefulness in both conditions. He further adds that there should be a negative correlation between depth of sleep (as measured by the acoustic threshold for awakening) and the presence of dream-like activity. However, the author he cites, Zimmermann (1970), only did the awakenings in stage 2. SOLMS does not take into account documented dream-like activity (>60%) in SWS, in which the threshold for awakening is notoriously high. (Cavallero et al.1992; Occhionero et al. 1998).

From a psychophysiological point of view, interpreting activity SO-Stage 1 as dream-like is not very convincing. Sleep onset is a condition in which a gradual transition from waking to sleep takes place. Its electrographic boundaries stretch from relaxed waking to Stage 2. The mental activity present in this period has special characteristics. SO-Stage 1 is not strictly oneric; rather it is a gradual disorganization of voluntary thought. Stage 2 (I cycle) does unquestionably exhibit more dream-like features than the preceding stage. This suggests that there is a gradual modification of cognitive organization in a dream-like direction, while the signals indicating an ongoing state of sleep are present in the recording (Bosinelli 1991). Furthermore in a study in which reports obtained in sleep onset (Stage 2) and in morning spontaneous awakening were compared, the morning stage 2 reports were more dream-like than those obtained in SO Stage 2 (Cicogna et al. 1998).

In our opinion, it is not necessary to look for triggers of dreaming; different cortical and subcortical neurophysiological constraints modulate mentation in waking and in sleep; however are not its direct and triggering cause waking and sleeping thought are functionally autonomous. The relationship with the physiological background interactive, not one of the cause and effect.

Expanding Nielsen's covert REM model, questioning Solms's approach to dreaming and REM sleep, and reinterpreting the Vertes & Eastman view of REM sleep and memory

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Abstract: Nielsen's covert REM process model explains much of the mentation found in REM and NREM sleep, but stops short of postulating an interaction of waking cognitive processes with the dream mechanisms of REM sleep. It ranks with the Hobson et al. paper as a major theoretical advance. The Solms article does not surmount the ever-present problem of defining dreams in a manner conducive to advancing dream theory. Vertes & Eastman review the REM sleep and learning literature, but make questionable assumptions in doing so.
[HOBSON ET AL.; NIELSEN; SOLMS; VERTES & EASTMAN]

NIELSEN mentation in REM and NREM sleep: A review and possible reconciliation of two models HOBSON ET AL. Dreaming and the brain. The target articles by **HOBSON ET AL.** and by **NIELSEN** provide very well-constructed arguments. Both point out current problems in the methodological and theoretical aspects of mind-brain relationships in a logical and fair manner based on enormous evidence including many new findings using neuroimaging techniques.

The AIM model of **HOBSON ET AL.** is noteworthy because it tries to capture the entire concept of our consciousness, using three dimensions that are supported by neurophysiological findings. It is unique to postulate "the state of the brain-mind at any given instant of time can be described as a point in this space." Under this proposition, we could predict the dynamics among different manifestations in our consciousness such as normal, altered, and abnormal states. We believe that their AIM model incorporating the "evolved" Activation-Synthesis model will inspire dream researchers to drive forward, further challenging concepts of consciousness in the same manner that their original A-S model has influenced current dream research.

The covert REM model by **NIELSEN** gives dream researchers positive perspectives on the controversy regarding whether mentations from REM and NREM sleep originate from the same process. His review represents the most creative theoretical formulation of sleep mentation since Foulkes's (1962) discovery that dreams occur outside REM sleep. NIELSEN cites compelling evidence for REM sleep intrusions or precursors at both physiological and psychological levels. Covert REM sleep processes may account for a considerable amount of sleep mentation variance and will generate testable hypotheses including several outlined in his review. It is particularly useful in terms of his recognizing the fluidity of the REM/NREM boundaries. He shows that setting aside the convenience of

the conventional R&K system appears quite productive when studying dreams and other states associated with sleep behavior. Thus NIELSEN's model of covert REM sleep processes has succeeded in merging previously incompatible and controversial findings represented by two models "the 1-gen model and the 2-gen models" into a more flexible and accountable model of sleep mentation.

Nevertheless, the commentators wondered if more phenomena could be explained by introducing *waking cognitive behavior* including micro-arousals into the covert REM sleep process as a second primary determinant of sleep mentation. Thus, this different type of 2-gen model – one in which one generator subserves waking cognitive activity with another serving REM dream processes – might allow one to predict mentation throughout waking and sleeping. Such an attempt might provide a means of broadening NIELSEN's very useful model to include some mentation that could not be fully explained by covert REM mechanisms: It would allow his model to explain NREM and REM instances of non-dream-like activity as well.

Waking cognitive behaviors might influence sleep mentation more than we think when one recalls how often micro-arousals appear during sleep (Mathur & Douglas 1995). Subjects are often unaware of these arousals even though their EEG clearly shows arousal during sleep (Ogilvie et al. 1989). During such arousals, some "wake-type" cognition or mentation may occur. When people wake up, they might recall or amend these mentations as if they had been experienced during sleep. For example, our data obtained from sleep-onset NREM and REM periods in normal subjects showed an interaction of brief arousals on REM and NREM dream recall rate (Takeuchi et al. 1999a; 1999b). This enables us to postulate different dream production processes for REM and NREM sleep, that is, the arousal process promotes NREM dreaming but blocks REM dreaming. Adapting waking cognition to the covert REM model would enable one to explain more fully the variety of mentations that seem to appear outside covert REM windows, such as mentations at the initial sleep onset in the first NREM-REM cycle and slow wave sleep as follows.

1. *Sleep onset mentation:* **NIELSEN** tries to explain sleep-onset mentation by his covert REM model. However, it is difficult for us to imagine covert REM mechanisms underlying sleep onset mentation in the first NREM-REM cycle in healthy nocturnal sleep considering the circadian nature of sleep-onset REM periods (Sasaki et al. 2000). Sleep-onset REM periods appear specifically when the sleep cycle is disrupted (Carskadon & Dement 1980; Fukuda et al. 1987) but do *not* appear during initial sleep onset in the first NREM-REM cycle in healthy nocturnal sleep. Hence, it seems unlikely that REM mechanisms function during the initial sleep onset in normal individuals. Thus, it seems more parsimonious to postulate that the initial sleep onset mentation may be explained by waking cognition rather than covert REM processes.

2. *Slow wave sleep mentation:* **NIELSEN** finds it difficult to explain mechanisms underlying slow wave sleep mentation by his model. There are a number of NREM phenomena (including parasomnia and mentation related to pre-awakening stimuli) which are difficult for the covert REM model to explain.

We feel that expanding **NIELSEN**'s probabilistic model to include waking cognition might explain virtually all sleep mentation. Further, his covert REM model and **HOBSON ET AL.**'s AIM model would complement each other in terms of their direction. These models – both bottom-up and top-down approaches – will provide theoretical direction for future dream studies.

Solms. Dreaming and REM sleep are controlled by different brain mechanisms. A useful basis is provided by **SOLMS** for continuing the debate about REM sleep and dreams. However, much of this debate may be semantic in nature. Dreaming is a somewhat ambiguous concept and his distinctions may add to the ambiguity. Differing definitions of "dreaming" create different results. The increase in NREM dream reports over time is owing to a changing definition of dreaming (**NIELSEN**). SOLMS argues that dreams are generated by the forebrain. However, some of the evidence for this proposition comes from people with seizures, brain damage, or drug use. These *dreams* are described by the author as "nightmares," "unusually vivid," and so on; in other words, atypical. If we limit ourselves to a more typical, intrinsic definition of "dreaming" then REM sleep and brain stem activity become crucial to dreaming.

SOLMS claims that the increasing frequency of dream recall during the late NREM stages in the rising morning phase of the diurnal rhythm suggests that these REM-like dreams are generated by specific NREM mechanisms. However, these longer NREM mentations are typically obtained within 15 minutes of prior REM sleep (Stickgold et al. 1994a) and could stem from covert REM windows (**NIELSEN**). Considering the circadian influence on REM pressure, it is logical that more mentation would be obtained from wider covert-REM windows in parallel with higher REM pressure in the morning. SOLMS claims that "cessation of dreaming has not been demonstrated in cases with elimination of REM sleep due to brain stem lesions." However, the elimination of observable REM sleep is not proof that no REM mechanisms are active. He also claims that the brain stem is not solely responsible for producing dreams by using the indirect argument that frontal lobe damage does eliminate dreaming. This can be debated on at least two levels.

First, forebrain damage may affect dream recall processes. The frontal lobes are involved in working memory (Smith & Jonides 1999). Working memory is required to keep thoughts active for an extended period of time such as during the arousal process. Thus, dream cessation after forebrain damage does not mean that the forebrain "produces" dreams. These people may have dreams, but do not recall them. **SOLMS** claims that 70–90% of people with prefrontal leukotomy experience a "complete or nearly complete loss of dreaming." Hence, 10–30% still dream. If he accepts that "25% of NREM dreams are indistinguishable from REM dreams" as proof that REM and dreaming are not the same, then having 10–30% of patients with frontal leukotomies still dreaming is equally compelling evidence that the forebrain is not the generator of dreaming. The hypothesis that dreams are generated by the forebrain after cerebral activation is worthy of further study. However, at this time we remain unconvinced that this explanation can negate the data showing such a strong relationship between REM sleep and dreaming. This is especially true if atypical dreams associated with various abnormal brain states (seizures, medications, etc.) are not considered.

Vertes & Eastman. The case against memory consolidation in REM sleep. Last, we would like to comment on the **VERTES & EASTMAN** article. In particular, we would like to raise several questions about the assumptions and logic used in their critique of the REM sleep and memory literature:

1. *Competing theories and assumptions:* We agree with **VERTES & EASTMAN** that an important function of REM sleep is to provide CNS activation periodically through the night. Their formulation is an interesting blend of the Roffwarg et al. (1966) Ontogenetic Hypothesis and Snyder's (1966) Sentinel Hypothesis. But the authors seem to imply that if REM serves to activate the brain periodically during sleep, it cannot also be involved in other activities – particularly memory consolidation, which they feel must take place during wakefulness. "Sleep involves basic biological functions and *memory requires consciousness*" (sect. 1, para. 5). This is a *huge* unsubstantiated assumption, following which, they begin their review of the literature. To assume that waking and sleeping processes do not interact is inconsistent with the evidence, particularly in light of the papers of **HOBSON ET AL.** and **NIELSEN.**

Another difficulty is that by simply counting "for" and "against" studies, they weigh studies which fail to reject the null hypothesis equally with those which *do* reject it, overlooking the powerful difference in the logical strength of these two positions, that is, one can never prove the existence of "no differences" – in this case that memory consolidation does not take place in REM.

2. *Animal REMD studies:* **VERTES & EASTMAN** of course, are right in saying that the flower pot method of REM deprivation in-

duces stress, but it is also a very effective means of almost totally eliminating REM (Smith & Gisquet-Verrier 1996). They are right in recommending the use of multiple platforms (pots) or Rechtschaffen's rotating disc-over-water/yoked control apparatus for REM deprivation studies. Both provide better controls for stress and are preferable to the flowerpot technique, but are not used routinely in REMD studies because they are much more costly and tedious to use.

But here an important judgment call must be made: Does one categorically reject all studies using the flower pot method, as **VERTES & EASTMAN** do, or should one interpret such studies with caution, being cognizant of the stress effect, but looking for convergent validation across labs and approaches? The latter appraisal leads to very different conclusions from those reached by VERTES & EASTMAN. As they note, the REM window studies are less susceptible to the stress criticism because the animals are deprived for shorter periods.

VERTES & EASTMAN are concerned that "windows" appear to move as a function of different tasks and species. However this may be a rather elegant demonstration of the specificity of memory processing. To our knowledge, this is the first behavioral evidence that parallels the superb neuroimaging work, which shows how dramatically individually patterned is the neural activity accompanying a number of behavioral tasks *and* a variety of different types of memory. That being so, is it so strange that the consolidation of these demonstrably different memory processes should take place at different rates?

Recent studies using the post-REMD design have almost unanimously found that REMD following learning produces deficits in the days or weeks to follow. **VERTES & EASTMAN** have incompletely reviewed this literature. The series of papers by Smith and colleagues is apparently dismissed because of their use of the pedestal technique. We would prefer to consider these experiments, noting the stress element.

3. *Human studies:* In the reviewed recent human studies of REM sleep and memory, there is no mention of Smith's work, though he and his coworkers are one of the most active groups working in this area. This exclusion seems unwarranted. After all, Smith does not place his *human* participants on flower pots.

In conclusion, we agree that there is insufficient evidence to accept that REM is solely for memory consolidation or that memory consolidation occurs only during REM sleep. However, there does appear to be sufficient evidence to confirm the existence of a link between REM sleep and memory consolidation.

Nielsen's concept of covert REM sleep is a path toward a more realistic view of sleep psychophysiology

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Abstract: Nielsen's concept of "covert REM sleep" accounts for more of the complexity in sleep psychophysiology than its conceptual predecessors such as the tonic-phasic model. With new neuroimaging findings, such concepts lead to more precise sleep psychophysiology including both traditional polysomnographic signs and neuronal activity in greater proximity to the actual point sources and distributed networks which generate dreaming.

[HOBSON ET AL.; NIELSEN]

NIELSEN's current reviews comparing REM and NREM sleep mentation (see also Nielsen 1999) provide a much-needed comprehensive review and incisive analysis of the extant psychophysiological data bearing on this controversy. His attention to the detailed physiology of sleep, and his concept of "covert REM sleep" offers an empirical path around the conceptual impasse imposed by over-reification of the cardinal sleep states as unitary, nondissociable physiological "black boxes" whose subjective manifestations must be compared dichotomously. NIELSEN's consideration of conceptually and experimentally diverse comparisons between REM and NREM associated cognitive processes (e.g., state-dependent differences in the effects of sleep manipulation on memory consolidation, ERP responses to stimulation, and post awakening performance) is essential in that it moves this debate away from the conceptually flawed (see the **HOBSON ET AL.** and NIELSEN discussions of Hunt et al. 1993) and inherently limited method of normalization for report length which has dominated the REM/NREM controversy to date. Equally important is his appreciation for and analyses of the difficulties inherent in achieving construct validity for sleep mentation variables and obtaining unambiguous psychological physiological correlations in any state.

In one sense, **NIELSEN**'s idea of covert REM sleep allows a reconsideration of the tonic-phasic model (Molinari & Foulkes 1969; Ogilvie et al. 1980; Pivik 1991) originally proposed to explain the lack of an exclusive association between dreaming and REM sleep. The tonic-phasic model itself encountered difficulties due to the very weak (although consistently positive) associations between the phasic events of REM and NREM sleep and the details of associated mentation reports (see Pivik 1991 for a comprehensive review). However, as we have argued elsewhere (Kahn et al. 1997), the polysomnographically measurable phasic events of REM and NREM sleep are most realistically viewed as integrated, attenuated "surface readouts" of complex and varied brain events and, therefore, their lack of a tight temporal relationship to the reported features of mentation is fully predictable. NIELSEN has now taken a fresh and more in-depth look at the psychological and physiological manifestations of these CNS events.

This new consideration comes at a time when the underlying brain events producing the psychophysiologically measured phasic and tonic events of sleep are also beginning to be revealed by functional neuroimaging studies of both sleep (e.g., Andersson et al. 1998; Braun et al. 1997; 1998; Bootzin et al. 1998; Hofle et al. 1997; Hong et al. 1995; Kajimura et al. 1999; Lovblad et al. 1999; Maquet et al. 1996; 1997; Nofzinger et al. 1997) and dream-like intrusions on waking (e.g., ffytche et al. 1998; Rabinowicz et al. 1997; Silbersweig et al. 1995). The neuroimaging data leads to an updated concept of the neural circuitry underlying dreaming in REM sleep which emphasizes activity in limbic circuits under conditions of decreased activity in executive cortical regions (e.g., Braun et al. 1998; Hobson et al. 1998a; 2000, and this volume; Maquet & Franck 1997; Nofzinger et al. 1997).

These new findings provide a first look at how **NIELSEN**'s hypothesized generators might be physically instantiated. For example, the comparative activation patterns of REM and NREM sleep suggest that one significant difference between the REM sleep and NREM sleep generators lies in the much greater activity of limbic structures during REM (for details, see **HOBSON ET AL.** 2000 and this volume). PET findings and their interpretation by Braun et al. 1998 suggest that an additional difference between the two generators may involve the neural sources of visual hallucinosis. NREM imagery may be initiated or generated further upstream in visual processing networks such as in the striate cortex (see also Hofle et al. 1997) whereas REM imagery may arise more from activity of visual association cortex (Braun et al. 1998) and thus perhaps be more similar to waking hallucinosis (see Ffytche et al. 1998).

When viewed in the light of abundant new neuroimaging data on a multitude of waking cognitive functions and subjective experiences (see Cabeza & Nyberg 1997; 2000; Gazzaniga 2000), the question of one versus two generators of sleep mentation seems inadequate to encompass the diversity of distributed and point sources of neural activity which must occur in multiple CNS networks in order to generate the complex phenomena of sleep mentation. For example, in addition to the distributed sensory and associative cortical modules hypothesized or implied by "one-generator" theories (e.g., Antrobus 1990; Foulkes 1993b), dream phenomenology indicates that subcortical-cortical networks sub-

serving emotion, instinct, and motivation (see Cummings 1993; Kalivas & Barnes 1993) must contribute at least as powerfully to the neural basis of sleep mentation. In addition, the wide difference in affective tone between individual dreams must reflect differential activation of limbic circuits subserving different emotional states. For example, nightmares compared to euphoric dreams may respectively reflect greater relative activation of amygdalar fear circuits versus mesolimbic reward circuits.

Toward the goal of dissecting the neural mechanisms that comprise sleep mentation generators, several of the ways in which **NIELSEN** compares cognitive processes in REM and NREM point toward specific neural networks and suggest hypotheses that might allow us to examine sleep mentation at a level of specificity comparable to current waking state paradigms. For example:

1. Comparison of memory sources between REM and NREM suggests the differential cortico-hippocampal information transfer in different sleep states that has been demonstrated in animal models (Buzsaki 1996). Such state-dependent differences in information processing have recently been hypothesized to underlie some of the state-dependent differences in memory sources available for the elaboration of sleep mentation (Stickgold 1998; Stickgold et al. 1999b)

2. Normal and abnormal variations in the extent or stability of lateral prefrontal cortex deactivation during sleep may contribute to subject differences in dream recall from REM and NREM sleep. The lateral prefrontal cortices have been shown to be essential to both encoding and retrieval of episodic memory (Fletcher et al. 1997). Therefore, for example, the greater NREM mentation recall in light sleepers (see **NIELSEN**'s discussion of Zimmerman 1970) might result from lesser NREM associated deactivation of prefrontal structures subserving episodic memory encoding, retrieval or both. Similarly, Braun (1999) has suggested that sleep-related prefrontal deactivation may degrade working memory resulting in encoded dream memory traces which then become relatively inaccessible to retrieval processes owing to a paucity of simultaneously encoded contextual cues.

Relative regional prefrontal activation also becomes a useful dependent variable for neuropsychological hypothesis testing of mechanisms for dream lucidity (i.e., relative engagement of executive functions) or for the variations in the degree of interrelationship between mentation content from different reports of the same night, which **NIELSEN** discusses (reflecting, perhaps again, hypothetical individual and temporal variations of encoding and retrieval). The great advantage of such specific hypotheses over the vaguer theories of "sleep mentation generation systems" proposed in the past is that they are eminently testable with current and developing technologies in cognitive neuroscience such as functional MRI or transcranial magnetic stimulation. Moreover, hypothesis formation in dream research can now be rapidly informed by new wake-state findings as they are reported.

3. The interrelationships between psychopathology and dreaming can be tested at both the psychological and physiological level using validated psychological instruments (such as the MMPI scales **NIELSEN** discusses) in combination with the ability to visualize activity in brain regions which respond to sleep-based treatments such as the anterior cingulate (Smith et al. 1999; Wu et al. 1999). Pioneering work in this area investigating dream anxiety among normals has already been reported by Gottschalk et al. 1991a; 1991b.

NIELSEN's model of covert REM sleep and his enumeration of nine factors by which it might be evoked suggests new and powerful ways of identifying and differentiating neural mechanisms underlying the known features of sleep mentation. For example:

1. The notion of a stage of "intermediate sleep" in the transition of REM to and from NREM has been well described in animals by Gottesmann and colleagues (see Gottesmann 1996, for a review) but has been under-investigated by students of the psychophysiology of sleep mentation (e.g., the only specific investigations cited by **NIELSEN** or Gottesmann appear to be early work by Lairy and colleagues; see also Larson & Foulkes 1969). In seeking specific and relatively isolated physiological correlates of sleep mentation, this polysomographically identifiable period should be a prime target for combined electrophysiology and neuroimaging studies especially given the apparent predictability in the non-simultaneous appearance of the cardinal PSG signs of REM (Sato et al. 1997).

2. **NIELSEN** notes that sleep onset similarly represents a prime candidate for a natural transitional state in which the dissociated psychophysiological correlates of REM may appear non-synchronously. As with REM onset, there may exist a degree of predictability as to the order in which different types of dream-like mentation may arise (Rowley et al. 1998).

3. Pathological and/or experimentally manipulated transitional or dissociated states suggested by **NIELSEN** (e.g., cataplexy; see Asenbaum et al. 1995, REM-deprivation induced sleep onset REM, pharmacologically altered sleep, auditorily evoked NREM mentation) provide an abundance of potential paradigms for combining electrophysiology and neuroimaging.

Studies such as the above might not only better define the neural substrate of physiological signs which are known to correlate with REM and/or dreaming (e.g., gamma-frequency oscillations: Gross & Gotman 1999; Llinas & Ribary 1993), but may identify previously unmeasured depth events with an even higher degree of correlation to psychological events such as the elusive (in humans) PGO wave. The current development of event-related fMRI techniques (e.g., Jessen et al. 1999) is particularly promising in this regard. Moreover, **NIELSEN**'s empirically tested model of probability for encountering covert REM sleep processes in NREM based upon temporal proximity to REM periods illustrates the straightforward methodology which could be employed in such studies (e.g., temporal correlation of regional activations with the incidence of mental events).

In summary, **NIELSEN**'s paper represents an important theoretical advance in the psychophysiological study of dreaming. In the future, such ideas may be viewed as important steps in a transition from viewing dreaming as a manifestation of a defined behavioral state to directly linking its components to the ebb and flow of neuronal processes which themselves define the behavioral states. In this sense, what we now refer to as "REM sleep" may come to be defined as that configuration of possible states in varied neuronal networks during sleep which results in the concurrent distinct experience of emotion, hallucination, and movement in synchrony with maxima of diverse physiological processes. In that sense, there is no covert REM sleep, only various degrees of dissociation or synchrony in the functioning of these modules. In turn, an overriding ultradian oscillatory mechanism may normally cause these interacting networks to reach maximal activity in phase with one another, thus producing the synchronous rise and fall of physiological processes and subjective experiences which we now characterize as the REM-NREM cycle.

Dreaming is *not* a non-conscious electrophysiologic state

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Abstract: There has been no generally accepted cognitive definition of dreaming. An electrophysiologic correlate (REM sleep) has become its defining characteristic. Dreaming and REM sleep are complex states for which the Dreaming = REMs model is over-simplified and limited. The target articles in this *BBS* special issue present strong evidence for a dissociation between dreaming and REM sleep.

[**HOBSON ET AL.; NIELSEN, REVONSUO; SOLMS; VERTES & EASTMAN**]

> *The vision of dreams is this against that, the likeness of a face confronting a face.*
>
> *Sira, Dead Sea Scrolls, 180 BC*
> (Sira 180/1973)

Dreaming is a cognitive state. If no one experienced the recall of sleep mentation on awakening, dreaming would not exist as a phenomenon for discussion. Instead we would be discussing the perceptual isolation of sleep, and the non-conscious significance of RFM sleep, NRFM sleep, PGO spikes, hippocampal theta and sawtooth waves. In the last thirty years these electrophysiologic (particularly REMS = Dreaming) have become the defining characteristics of dreaming. That period may be ending. The target articles in this *BBS* special issue present strong evidence for a dissociation between dreaming and REM sleep. The scientific study of dreaming is in the midst of a millennial year's change.

What is a dream? Aristotle, with characteristic conceptual clarity, defined dreaming as the mental activity of the sleeper insofar as he is asleep (Aristotle 235/1952). For Aristotle, dreaming was an idealized state with a reality independent of waking experience. This definition has survived more than two thousand years, to be found almost verbatim in current dictionary definitions: a dream is a series of thoughts, images, or emotions occurring (passing through the mind) during sleep (Webster's Dictionary 1993; Random House Dictionary 1983). In this definition, dreaming is a state occurring during sleep, not accessible to study during the wakefulness. It suggests that dreaming may be best defined by its nonconscious correlates. The cognitive dream, in other words, the recall waking of a dream, is not part of the Aristotelian definition.

Scientific methodology was developed as a formal approach in order to analyze external reality and hence to differentiate it from dreams (Descartes 1637). The modern age of the scientific study of sleep can be dated back to the staging of sleep based on FEG, EMG, and EOG criteria from the mid 1950s and 1960s (Aserinsky & Kleitman 1953; Rechtschaffen & Kales 1968). Although sleep has well defined electrophysiologic correlates, sleep onset is still generally defined behaviorally rather than electrophysiologically. Sleep is defined as a reversible behavioral state of perceptual disengagement from and unresponsiveness to the environment (Kryger et al. 1994).

Archeological research has shown that human interest in dreaming dates back over at least 4,000 years. Codices recovered from the Sumerian city of Ladak (2500 B.C.) describe King Gudea's orientation of his new temple based on insight obtained from a dream experience. Despite such a prolonged interest, limited progress has been made towards understanding dreaming. Freud wrote on page 1 of *The interpretation of dreams*,"In spite of many thousands of years of effort, the scientific understanding of dreams has made very little advance . . . little or nothing that touches upon the essential nature of dreams." Freud's contribution was to identify a role for dreaming in the diagnosis and therapy of psychiatric illness (Freud 1900). Psychoanalysts, psychologists, and psychiatrists continue to use dreaming in diagnosis and therapy. However, modern researchers and therapists often allude to dreams without definition. The tomb of psychiatry (the Diagnostic and Statistical Manual of Mental Health, DSM) avoids definition of a dream, mentioning only the nightmare – a frightening dream (DSM-IV 1994). The meaning, content, allusions, and associations of dream (undefined) are regularly the focus of discussion and publication. A topic may be REM sleep, personal motivation, neuroanatomy, film, hallucinations, literature, neuropharmacological effects of medication, real estate, the unconscious mind, or anthropology; and dreaming (undefined) is often the title. Check any library catalog system for its listing on dreams.

The issue is compounded by the personal nature of dreaming. Between two and four years of age most children develop a conceptual definition of a dream (Olson et al. 1988). This private dream reality remains outside study and definition, being made available only as the individual dreamer desires. In the words of Shakespeare, "The eye of man hath not heard, the ear of man hath not seen, man's hand is not able to taste, his tongue to conceive, nor his heart to report, what my dream was" (Shakespeare 1595/1986). When we gather to study dreams, we each bring to the table our personal definitions. Dreaming is not the only aspect of mental activity that has such loose attributes. Imagination, creativity, intelligence, and even consciousness are poorly defined cognitive concepts that suffer the same diffuse social attributions. Dreaming has an advantage in that it has a long history of studies that have described the dream personally, physiologically, and psychologically. There is, however, a generalized tendency in the field to avoid defining dreaming (Pagel 1999a).

Different fields have widely varying definitions for dreaming. Psychoanalytic definitions of dreaming date from Freud – unconscious wish fulfillment (Freud 1900). One current psychiatric definition of dreaming can be considered: bizarre or hallucinatory mental activity occurring during a continuum that extends through stages of sleep and waking . The newer field of sleep medicine often defines dreaming as mental activity (images, feelings or emotions) occurring during sleep (Mahowald et al. 1998). Psychology has generally avoided a definition of dreaming, concentrating on defining methodology and study populations. A wide spectrum of fields from anthropology to literature characterize dreaming by its associations and attributes. In popular culture, the most generally accepted definition is loosely Freudian: dream marriages, dream homes – the projected image of conscious wish fulfillment.

The topic of one study in which dreaming is mentation occurring during sleep may be very different from hallucinatory or delusional thought content. Results are likely to be comparable only when comparable definitions of the topic of study are used. This has produced conceptual confusion that has contributed to a lack of structural rigor in the field of study. Single structural definition has not been possible owing to the diverse spectrum of fields and an historic multiplicity of definitions applied to the study of dreaming. A classification schema for definitions of dreaming has been described for incorporation into scientific studies of dreaming so the results of studies across epistemologically diverse fields can be compared (Table 1) (Pagel 1999b).

As Descartes points out, "Now the principal and most frequent error that can be found in judgments consists in the fact that I judge that the ideas, which are in me, are similar to, or in conformity with certain things outside me." (Descartes 1641/1980). The present target articles reflect the disarray in the fields of dream study that have occurred because of the fields' inability or disinclination to cognitively define the topic of study – dreaming. It is the electro-

Table 1 (Pagel). *AASM dream definition paradigm*

(a)	Sleep	Sleep Onset	Dreamlike States	Routine Waking		Alert Wake
(b)	No Recall	Recall	Content	Associative Content	Written Report	Behavioral Effect
(c)	Awareness of Dreaming	Day Reflective	Imagery	Narrative Story	Illogical Thought	Bizarre Hallucinatory

physiologic correlates of dreaming, especially REM sleep, that have been used to define its presence or absence. This Aristotelian conceptual approach (Dreaming = REMS) has achieved almost mythological status despite extensive criticism. A cognitive definition of dreaming should incorporate the awake recall of dreaming. Sleep, a state with clearer electrophysiologic correlates than dreaming, is still defined by its behavioral and cognitive correlates. The reproducibility of results is based on clear limits of one's object of measure. In part, that is why the view that Dreaming = REMS has been so attractive. It would make this loosely defined cognitive state much easier to study and understand.

SOLMS's paper is the shortest but perhaps most important in this series owing to the clarity with which research and clinical findings have been applied to the question of whether dreaming is "an epiphenomenon of REM sleep." Since not all dreaming is correlated with REM sleep, SOLMS asks whether what has been defined as the REM sleep type of dreaming occurs outside of REM sleep. He presents evidence that the REM sleep can occur without dreaming and dreaming without REM sleep. A consistent body of literature suggests that dreaming correlates better with cerebral activation during sleep than with the occurrence of REM sleep. SOLMS's argument is that REM sleep and dreaming are doubly dissociable states with different physiological mechanisms in all likelihood serving different functional purposes. He suggests that the REMS = Dreaming premise upon which most prevailing neuroscientific theories of dreaming are based, is invalid.

VERTES & EASTMAN consider the lack of evidence for memory processing and consolidation in REM sleep. This too reflects some of the difficulties rising from prevailing REMS = Dreaming theories. As the authors state, "The mental/cognitive content of REM sleep is dreams . . . Dreams are the sole window to cognitive processes of REM sleep." Their reviews of imaging brain studies of brain activity during REM sleep are accordingly extrapolated to dreaming: "in summary, the pattern of brain activity in REM sleep is consistent with dreams, but inconsistent with the orderly evaluation, organization and storage of information which is the domain of attentive, waking consciousness." It may very well be, as they suggest, that REM sleep can be understood within the context of sleep without invoking mental phenomena or quasi-conscious processes – the cognitive process of dreaming.

REVONSUO presents an eloquent and well researched argument for the role of dreaming as a cognitive process in the evolution of threat avoidance. He defines dreaming as conscious experiences during sleep: "We may define a dream as a subjective experience during sleep, consisting of complex and organized images that show temporal progression." He argues that consciousness can be reconceptualized as the phenomenal level of organization of the brain with dreaming a subjective experience realized at the phenomenal level. Based on prevailing REMS = Dreaming theories he concludes, "the phenomenal level of organization of the brain is realized in its characteristic ways during REM sleep." He incorporates research from both REM sleep studies and cognitive dream research, considering that both fully reflect dreaming. It is suggested that dreaming and REM sleep may have complementary yet different evolutionary roles.

NIELSEN acts as an apologist for the Dreaming = REMS Paradigm, redefining REM sleep as a state in which REMS type dreaming occurs: hallucinoid imagery, narrative structure, cognitive bizarreness, hyperemotionality, delusional acceptance, and deficient memory of previous mental content. He distinguishes "dreaming" (REMS type mental processes as described above) from other cognitive activity occurring during sleep which is not considered to be "dreaming." REM sleep is characterized by a general motor atony, bursts of sciatic eye movements, cardiac and respiratory irregularity, PGO spikes, hippocampal theta rhythms, genital tumescence, and low frequency "awake-like" EEG activity which may include sawtooth waves (Kryger et al. 1994; Rechtschaffen & Kales 1968). NIELSEN redefines REM sleep as any of these REM sleep associated events occurring in association with "dreaming" as defined above. It is suggested that REM sleep may occur even in states which are not clearly sleep (the disorganized EEG drug effects induced by katamine and LSD) because "dreaming" is associated with these states. This paper concludes that if both dreaming and REM sleep are radically redefined the REMS = Dreaming model can be preserved.

HOBSON ET AL.'s important paper comes from the group that first advanced the REM = Dreaming hypothesis. Although they persist in correlating dream features with distinctive REM sleep physiology, the authors suggest that the correlation should be loosened, with cognitive states better viewed as inter-related psychophysiological continua manifested at the levels of both the brain and the mind.

Dreaming is initially defined as a series of images, ideas, emotions, and sensations occurring involuntarily in the mind during certain stages of sleep and REM sleep as well as during the numerous forms of wake-state and sleep-state mentation. Later in the paper, dreaming is redefined by the narrower constraints **HOBSON ET AL.** prefer: "mental activity occurring in sleep characterized by vivid sensorimotor imagery that is experienced as waking reality despite such distinctive cognitive features as impossibility or improbability of time, place, persons, and actions; emotions, especially fear, elation and anger predominate over sadness, shame and guilt and sometimes reach sufficient strength to cause awakening; memory for even very vivid dreams is evanescent and tends to fade quickly upon awakening unless special steps are taken to retain it." The focal argument, however, is that dreaming and other states of consciousness can be best defined by a model that includes: (1) the information processing capacity of the system (activation); (2) the degree to which the information processed comes from the outside world and is or is not reflected in behavior (information flow); and (3) the way in which the information in the system is processed (mode). This model corresponds to the various axes of the AASM dream definition protocol (Table 1) in which the activation axes can be equated with the awake/sleep axis, the information axis with the recall axis, and the mode axis with the content axes (Table 1).

This model is applied to the description of a series of cognitive states viewed as brain-body-mind isomorphisms varying in multidimensional combinations. With this model, **HOBSON ET AL.** Dreaming = REMS controversy, utilizing the dream axis definition criteria to model "dreaming" and other cognitive states of both waking and sleep. It is the authors' contention that their model is both necessary and sufficient to distinguish in a preliminary way among the basic wake-sleep states: the resulting state space model, while still necessarily overly simplistic, is nonetheless a powerful tool for studies of consciousness."

Comparing definitions of dreaming. In these papers, the REMS = Dreaming model – until now so pervasive in the fields of sleep and dream study – is useful for comparing studies that are based on irreconcilable cognitive definitions of dreaming. These papers use a spectrum of definitions of dreaming. **NEILSEN** uses a narrow content based definition for dream in his presentation: mentation characterized by hallucinoid imagery, narrative structure, cognitive bizarreness, hyperemotionality, delusional acceptance, and deficient memory of previous mental content. His definition excludes other cognitive activity occurring during sleep which is not considered to be "dreaming." The Awake/Sleep or Activation axis of his definition is broad and undefined with dreaming considered to occur in sleep, wake, and drug induced states (Table 2). The dreaming that **REVONSUO** considers is very different, including a broad spectrum of content (subjective experience realized at the phenomenal level), specific recall (evolutionary effects), and specific Awake/sleep axis (occurring only during sleep). **VERTES & EASTMAN** (the mental/cognitive content of REM sleep is dreams) are also considering an unlimited range of content occurring out of an even narrower segment of the Awake/Sleep axis – REM sleep. VERTES & EASTMAN and REVONSUO are studying dreaming that NIELSEN defines as sleep cognitive activity (non dreaming). **SOLMS** and **HOBSON ET AL.** define dreaming as an epiphenomenom of REM sleep in order to argue for and against the REMS =

Table 2 (Pagel). *Dream definition paradigm comparing definitions used in these papers*

(a)	Sleep	Sleep Onset	Dreamlike States	Routine Waking	Alert Wake	
(b)	No Recall	Recall	Content	Associative Content	Written Report	Behavioral Effect
(c)	Awareness of Dreaming	Day Reflective	Imagery	Narrative Story	Illogical Thought	Bizarre Hallucinatory

NEILSEN: mentation characterized by hallucinoid imagery, narrative structure, cognitive bizarreness, hyperemotionality, delusional acceptance, and deficient memory of previous mental content.

(a)	Sleep	Sleep Onset	Dreamlike States	Routine Waking	Alert Wake	
(b)	No Recall	Recall	Content	Associative Content	Written Report	Behavioral Effect
(c)	Awareness of Dreaming	Day Reflective	Imagery	Narrative Story	Illogical Thought	Bizarre Hallucinatory

REVONSUO: subjective sleep experience realized at the phenomenal level affecting evolutionary process.

(a)	Sleep	Sleep Onset	Dreamlike States	Routine Waking	Alert Wake	
(b)	No Recall	Recall	Content	Associative Content	Written Report	Behavioral Effect
(c)	Awareness of Dreaming	Day Reflective	Imagery	Narrative Story	Illogical Thought	Bizarre Hallucinatory

VERTES & EASTMAN: the mental/cognitive content of REM sleep is dreams
SOLMS: dreaming is an epiphenomenon of REM sleep.

(a)	Sleep	Sleep Onset	Dreamlike States	Routine Waking	Alert Wake	
(b)	No Recall	Recall	Content	Associative Content	Written Report	Behavioral Effect
(c)	Awareness of Dreaming	Day Reflective	Imagery	Narrative Story	Illogical Thought	Bizarre Hallucinatory

HOBSON ET AL.: mental activity occurring in sleep characterized by vivid sensorimotor imagery that is experienced as waking reality despite such distinctive cognitive features as impossibility or improbability of time, place, person, and actions; emotions, especially fear, elation, and anger predominate over sadness, shame, and guilt and sometimes reach sufficient strength to cause awakening; memory for even very vivid dreams is evanescent and tends to fade quickly upon awakening unless special steps are taken to retain it.

Dreaming paradigm. In creating their cognitive state models, HOBSON ET AL. use a definition of dreaming that specifically defines all three axes (Table 2).

Both **SOLMS** and **HOBSON ET AL.** address the diversity of definitions for the dream state, applying specific definitions to compare data while maintaining an awareness that adaptive models are required to address the different phenomenon that researchers call dreaming. If interdisciplinary studies are to be compared, such a specific yet adaptive multi-axis approach to dreaming state definition, as HOBSON ET AL. suggest, provides us with a powerful tool that can be applied to cognitive study of other conscious states.

Dreaming is not a non-conscious electrophysiologic state. These are exciting times in the field. The papers in this special issue of *BBS* describe a major paradigm shift in our understanding of the association between sleep and dreaming. **SOLMS** argues co-

gently that REM sleep and dreaming are doubly dissociable states with different physiological mechanisms in all likelihood subserving different function. Support comes from **VERTES & EASTMAN** who argue that REM sleep can be understood within the context of sleep without invoking mental phenomena or quasi-conscious processes – the cognitive process of dreaming.

As often seems to be the case, the greatest support for this paradigm shift away from REMS = Dreaming comes from its defenders. **NIELSEN** shows how the paradigm could be saved by radically defining both REM sleep and dreaming. In order to preserve the model, cognitive activity occurring during sleep must be excluded from the definition of dreaming, and REM sleep must be re-defined to occur throughout the Awake/Sleep axis. In order to preserve REMS Dreaming, we are required to restructure the entire sleep and dream study. Perhaps the greatest support for this paradigm shift comes from **HOBSON ET AL.,** the developers of the REMS = dreaming model, who have chosen to side-step defense of their paradigm and move to a multi-dimensional cognitive state model for dreaming in which REM sleep is but one point on an Activation = Awake/Sleep continuum.

Suggestions that the REMS = Dreaming premise upon which most prevailing neuroscientific theories of dreaming are based is no longer valid, **SOLMS** proposes new neuroanatomical and clinical approaches to the study of REM sleep and dreaming. **VERTES & EASTMAN** have set the framework for the analysis of dreaming using memory paradigms independent of the states' association with REM sleep. **REVONSUO** demonstrates that an evolutionary model is applicable to both REM sleep and dreaming as independent yet complementary phenomenon. **HOBSON ET AL.** apply a multi-axis paradigm for dreaming that can be used to describe other cognitive states, and perhaps even model non-biologic systems.

Descartes asserted that scientific method could be applied to reality and not to dreams (Descartes 1637/1980). But science can use multi-axis definitions to study the specific cognitive and electophysiologic correlates of dreaming that are reality. Dreaming and REM sleep are complex states for which the Dreaming = REMS model has become excessively simple and limited. Progress in a scientific field occurs when structural models change. As this collection of papers suggests, the paradigm has shifted.

"The dream of reason creates monsters" . . . especially when we neglect the role of emotions in REM-states

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Abstract: As highlighted by Solms, and to a lesser extent by Hobson et al. and Nielsen, dreaming and REM sleep can be dissociated. Meanwhile Vertes & Eastman and Revonsuo provide distinct views on the functions of REM sleep and dreaming. A resolution of such divergent views may clarify the fundamental nature of these processes. As dream commentators have long noted, with Revonsuo taking the lead among the present authors, emotionality is a central and consistent aspect of REM dreams. A deeper consideration of emotions in REM dreams may serve as the conceptual salve to help heal the emerging rifts in this field of inquiry.
[HOBSON ET AL.; NIELSEN; REVONSUO; SOLMS; VERTES & EASTMAN]

I use Goya's epigram for one of his final engravings in my title to highlight how our widespread failure to deal with basic emotional processes of the brain may have led to many conundrums in our search for the adaptive functions of dreaming and REM sleep. The same can be said about our aspirations to understand many other mind-brain relations, from consciousness to intrinsic organismic values.

SOLMS has done a great service in highlighting, more dramatically than ever before, the increasingly evident fact that REM mechanisms and dreaming are not strictly isomorphic. Clearly, REM sleep reflects very ancient aspects of neural organization while the richness of human dreams is a comparatively recent brain development. As **SOLMS** discusses, distinct types of arousal during sleep may activate dreams in the absence of REM, and fairly restricted damage to neocortex can lead to the disappearance of dreams while REM persists. As a result of his challenge, we must no longer equate REM sleep and dreaming as casually as in the past. How these distinct but interactive brain-mind processes can be understood without doing injustice to either, is the central theme of the present discussion.

HOBSON ET AL. provide us with a masterful update of neural and psychological aspects of REM sleep and dreaming, and a new synthetic model of mind to boot. Even though they mention emotions repeatedly and at times foreshadow **REVONSUO**'s intriguing proposal emphasizing anxiety over other emotions as the prime "shaper" of dream plots, **HOBSON ET AL.** fail to cultivate emotional concepts in any comprehensive way. Since their AIM model of mental space "strained to account for differences between various emotional substates," it was good to have **REVONSUO** develop an emotional theme elegantly as an exemplar of where we need to go. The analyses of **NIELSEN** and **VERTES & EASTMAN** also remained regrettably devoid of emotional concerns. Despite their rich tapestries of evidence and argumentation, a judicious consideration of brain emotional systems remains a most promising path to understand the role of REM in dreams as well as brain-mind evolution. Thus, I would encourage all participants to consider brain emotional issues further in their final comments, and also provide additional predictions (especially disconfirmatory ones) for their various viewpoints.

NIELSEN attempts mightily to bind up the wound that **SOLMS** has opened up. The one- and two-generator models of dreaming can be reconciled in various ways, but how, without circularity, might the "covert REM" hypothesis deal with the fact that elimination of overt REM sleep does not necessarily eliminate dreaming? If we accept the traditional idea that REM is a uniquely powerful, though not an obligatory trigger for dreaming (and most studies of the intact brain-mind do affirm that conclusion), I do not see how we can escape some variant of a two generator point of view. Perhaps the truth is still rather close to what most have been led to believe – REM dreams are fundamentally emotional while NREM dreams are more strictly cognitive. In other words, the two major types of dreaming may emerge from measurably distinct albeit interactive neuromental spaces.

Despite the dissociations between REM mechanisms and dreaming in broken brains, many of us – including perhaps **HOBSON ET AL., NIELSEN,** and **REVONSUO** – will continue to believe that the relationships between the two (especially the most vivid emotional dreams) in normal physiology are as dynamically interwoven as those of conductor and orchestra (or horse and carriage for the more blatantly behavioristic). However, as implied by both **SOLMS** and **HOBSON ET AL.**, we should remain open to the possibility that several functionally distinct conductors can guide the dream orchestra – each with different tempos, neuropsychological consequences, and state spaces in the brain-mind. In this view, the distinction between the emotional dreams of REM and the non-emotional ones of NREM, as well as their varied functions, may still prove to be physiologically and psychologically meaningful. However, **SOLMS**'s point would still hold – the cognitive contents of dreams are not choreographed in any detail by REM processes; rather, REM is simply the most emotionally minded conductor of one dream symphony.

The classic Freudian division between latent and manifest contents may also help us parse these issues in reasonable ways. For instance, while REM sleep arises substantially from lower brainstem processes, providing latent energies for many dream possibilities, the manifest contents require the participation of higher, heteromodal cortical processes. **SOLMS'**s neuropsychological work jibes remarkably well with modern brain imaging data on REM

dreams as detailed comprehensively by **HOBSON ET AL.** – the dorsolateral prefrontal working-memory systems remain asleep in REM while basomedial appetitive-emotional systems and most other limbic areas become remarkably aroused. In contrast, those emotional brain areas remain fairly quiescent during NREM sleep. Whether REM characteristic brain patterns accompany NREM dreams remains unknown, but I hope SOLMS will clarify whether he would predict massive neurophysiological congruences between the two? If not, would he accept, in principle, the likelihood of at least two distinct types of dream life?

Although **SOLMS** and **VERTES & EASTMAN** seem to imply that we may have overvalued REM-dreams, I would not discard the possibility that REM dreams are of substantial functional importance in normal brain-mind homeostasis. The REM process appears quite privileged in breathing primary-process affective life into the cognitive flow of dreams within the intact brain. REMs may sequentially arouse various basic emotional tendencies during paradoxical sleep, providing latent neurodynamic support for reprocessing affective experiences encountered during waking within the manifest dreams percolating in higher brain areas. Indeed, it may be that only the less emotional dreams survive damage to REM circuits. Considering this, the emotional analysis of dreams of individuals whose REM mechanisms are impaired may be informative, and perhaps SOLMS can enlighten us on such issues.

Despite the striking dissociations highlighted by **SOLMS,** I suspect that the most emotion laden dreams remain so only because of the sustaining influences of REM arousal. Indeed, since the co-occurrence of REM and emotional dreams may be the only way we shall ever decode the biological, neuroadaptive nature of our most vivid dreams, we should continue to pursue the strategy that a detailed study of REM related processes provides our best opportunity to understand the nature and functions of our most memorable (perhaps archetypal) dreams. Despite **VERTES & EASTMAN**'s well-reasoned skepticism about any global information-processing hypothesis, REM dreams may still have specific functions in the integration of affective information, perhaps along the lines outlined by **REVONSUO.**

REVONSUO shared his ideas concerning dreams at a Tucson III Plenary panel on the "Neural Correlates of Consciousness" (May 2, 1998). I inquired from the floor whether he or other panel members had an explanation for the perplexing neurological fact that REM arousal mechanisms, and hence presumably the rudiments of dreaming, are more ancient in brain evolution than the waking circuits of the Extended Reticulo Thalamic Activating System (ERTAS). All seemed perplexed by this prickly question. It has also received inadequate attention from the sleep-research community. My puzzlement was prompted by the recognition that the epicenter for REM sleep is slightly more caudal in brainstem tissues than the epicenter for waking, at least as affirmed by the classic pre-trigeminal preparation.

A resolution of this apparent paradox may yield a coherent understanding of REM mechanisms and the related affective dream contents. My personal solution was offered in passing several years ago (Panksepp 1993) and, as **REVONSUO** notes, also (Panksepp 1998a, pp. 134–35): The REM process may be the functional residue of an ancient form of waking – a simple-minded form of emotional arousal that was "reined in" through the evolution of REM-atonia as higher "pos-trigeminal" ERTAS systems prevailed over primordial pre-propositional forms of waking. This would help explain why REM still arouses basic emotional processes and infuses affect into cognitively manifested dream deliberations. Might it be that people with damage to cortical areas that abolish manifest dreams still experience affective states but no longer have memorial residues of those experiences as is the case in amnesic temporal lobe patients (Damasio 1999)?

From an evolutionary perspective, it should be obvious that basic waking mechanisms have a long history, and certain simple-minded solutions may have gradually been superseded by more sophisticated ones. Perhaps older forms could not be eliminated but, because of adaptive constraints, had to be integrated, often in perplexing ways, with subsequent layers of neural control. According to this view, REM arousal may reflect an ancient form of waking arousal that was devoted largely to activating genetically ingrained emotional subroutines, which guided behavioral actions in ancestral species long before the behavioral flexibility provided by higher cerebral evolution. Those ancient, value-coding processes may still provide background operations that help higher brain mechanisms sift and integrate fundamental survival concerns from the Niagara of cognitive information flowing in from newly evolved forebrain regions.

Although we cannot be privy to the cognitive contents of animal dreams, we do have some behavioral access to their emotional contents. Following destruction of their atonia mechanisms, cats exhibit at least four "archetypal" REM-dreams – predatory intent, fearful withdrawal, angry assertiveness, and licking/grooming (Sastre & Jouvet 1979). These categories reflect primal emotional concerns of all mammals, elaborated by subcortical systems which may have had a more important role in the emergence of consciousness than is commonly recognized (Damasio 1999; Panksepp 1998b; 2000). In unpublished work, I have repeatedly found these four primal themes to prevail in human dream reports.

If REM merely reflected some type of homogeneous, nonspecific cerebral arousal as **VERTES & EASTMAN** suggest, these oneiric behaviors would remain puzzling. However, these emotional expressions provide excellent clues for unraveling the nature of the neural "conductors" that may guide REM-dreams in all species. Perhaps a search for the psychological functions of REM sleep should be premised on an understanding of these "archetypal" psycho-behavioral themes. Would **NIELSEN** and VERTES & EASTMAN consider including such affective themes in their analysis of REM functions?

Any comparable analysis of NREM dreams will remain more problematic until someone learns to extract the cognitive contents of sleeping animals, a project that is becoming conceivable with neurophysiological measures of brain representational activities: It has been surprising to many that spatial data processed during waking are better represented in hippocampal circuitries during slow-wave sleep than in REM sleep (Shen et al. 1998; Wilson & McNaughton 1994). However, perhaps this simply affirms that NREM dreams are more laden with past cognitive experiences. I trust future investigators will eventually find predominating neuronal footprints of past emotional experiences in REM sleep

In accepting the connectedness of REM dreams and the ancient emotional force of fear, **REVONSUO** clarifies why threat perception and harm avoidance lie at the heart of so many of our dreams. However, one empirical difficulty for REVONSUO's fear-constrained analysis may be the tendency of predatory mammals to exhibit more REM sleep than their prey. Surely the latter should exercise their defensive capacities more than the former. One could, of course, suggest that predators have a continual fear of starvation, but that would be taking the analysis in circular directions that have traditionally yielded more confusion than clarity in our attempt to scientifically understand the nature of psychological processes.

In any event, fear has been a most compelling way to bring basic emotional issues to the attention of the scientific community. It will be especially interesting to know whether humans have fear dreams more in anticipation of harm than its aftermath. Perhaps a study of well-motivated students or sick people before difficult mental and bodily examinations, as well as dream analyses of winning and losing teams around closely contested championships, could shed light on such issues.

Of course, the other basic emotional systems also deserve study in such an "experience expectant" dream framework. Since Freud focused on wish fulfillment as one common theme of dreams, let me briefly follow the lead of **SOLMS** on the possibility that dopamine arousal helps to mediate such urges in all animals (Ikemoto & Panksepp 1999). There are remarkable similarities between the emotional energies of REM and dopaminergic lateral hypothalamic urges reflected in self-stimulation behaviors (Panksepp

1998a, pp. 142, 163); it is also established that dopamine neurons do not shut down during REM as do norepinephrine and serotonin ones (Steinfels et al. 1983; Trulson & Preussler 1984). Unfortunately, as **HOBSON ET AL.** emphasize, we don't really know whether dopamine is vigorously released during activated sleep. Indeed, with all the many other neurochemical participants in REM sleep, dopamine may only be one conductor of the dream symphony.

In any event, if dopamine is a major player in certain dreams, we might anticipate dopamine neurons to begin exhibiting "bursting" activity during REM sleep. Such changes, have not yet been empirically evaluated to my knowledge, although the DA supersensitivity following REM deprivation, as noted by **HOBSON ET AL.,** support that view. From simple ethnological observations we do know that rats exhibit lots of sniffing – an excellent indicator of dopamine arousal and appetitive engagement – during REM. In **REVONSUO'S** perspective, we might also note that avoidance of danger may operate through dopaminergic "wish-fulfillment" type processes since "approach to safety" rather than mere "escape from danger" may be the self-centered dynamic around which many avoidance behaviors are constructed in the brain (Ikemoto & Panksepp 1999). With such ideas, the views of **SOLMS** and REVONSUO could be integrated nicely.

In any event, **REVONSUO** has provided an impressive evolutionary scenario that can help us make sense of the chaotic evidence for the information-integration views of REM sleep that **VERTES & EASTMAN** criticized. If one restricted such information-integration inspired analyses of REM functions to difficult emotional realms, rather than including all cognitive problems animals need to solve, the existing evidence for REM-promoted memory consolidation may be less chaotic than VERTES & EASTMAN portrayed.

For example, the largest and most consistent effects of post-training REM deprivation ever described in animal studies are those that have employed devilishly complex tasks such as the two-way shuttle avoidance (Smith 1985). No animal has been prepared by evolution to continually run between fluctuating danger and safe zones, and it takes a great deal of training for animals to master such emotionally horrendous tasks (Greenberg & Pearlman 1974). The severe deficiency of REM-deprived animals (using the stressful island method) in the acquisition of shuttle avoidance supports **REVONSUO**'s hypothesis that complex harm avoidance strategies may be strengthened by REM sleep. As Smith (1985) has highlighted, such effects can be obtained with short periods of REM deprivation and many studies have employed rather good controls to help rule out generalized stress effects – one of the most, compelling being that of Leconte et al. (1974). The failure of van Hulzen and Coenen (1982) to obtain such effects with their kinder form of REM deprivation (rocking), may have been owing to their use of a pre-learning deprivation paradigm on learning of rather mild shock avoidance, which may have aroused less fear than is present in many other studies. Clearly, more work is needed on the issue before we have definitive conclusions. Still, **VERTES & EASTMAN**'s assertion that REM-deprivation technique typically produces a great deal of stress having distinct behavioral effects is certainly correct (e.g., Kovalzon & Tsibulsky 1984).

With regard to the information-processing dilemma that **VERTES & EASTMAN** highlight, from the present vantage, REM deprivation should selectively impair emotionally loaded tasks that truly challenge animals' coping resources toward the breaking point. REM dreams may operate effectively on the statistical contingencies present in such difficult situations, providing opportunities for new emotion-relevant "insights," or at least coping adjustments, to emerge from REM inspired information juggling in higher regions of the brain. However, rather than REM merely consolidating information in a cognitive realm (which may be much more of a NREM function as highlighted by Buzsaki [1998] as well as the aforementioned work from the McNaughton group), REM may help create novel psycho-behavioral connections within the subconscious emotional habit-structures of animals. As **REVONSUO** and others have emphasized, dreams may allow organisms to deal better with emotionally charged situations in novel ways. Despite VERTES & EASTMAN's compelling challenge, this alternative view has yet to attract adequate empirical investigations.

To digress, the fact that antidepressants which eliminate REM have few cognitive effects may not be pertinent to the present argument: First, these agents allow animals to cope better with emotionally stressful situations independently of their REM suppressing effects. Second, since there is good reason to believe that REM sleep normally sustains the synaptic efficacy of transmitters such as serotonin and norepinephrine, the utilization of antidepressive agents may ameliorate some of the neurochemical deficits normally produced by REM deprivation (Panksepp 1998a, p. 140). Regrettably, the emotional tendencies of patients with brainstem damage disrupting REM sleep remain to be adequately evaluated, but the present view would predict problems in long-term emotion/cognition integrations. Also, **VERTES & EASTMAN** fail to note that the absence of apparent waking-up deficits in such individuals might be a negation of their suggestion that REM helps sleeping brains to recover from the psychological torpor of deep somnolent states. Likewise, I anticipate the antidepressant and many other psychological effects of REM-deprivation might be difficult to explain with the minimalist view espoused by VERTES & EASTMAN.

If most REM-dreams reflect forward directed, experience-expectant emotional processes, then they may not be the epiphenomenal or psychologically irrelevant flotsam (as **REVONSUO** puts it "random noise generated by the sleeping brain as it fulfills various neurophysiological functions") that many investigators are coming to believe. If REM-dreams are truly laden with self-referential configurations and permutations of emotional problems to be solved, while NREM dreams are laden with less affective contents, then we still have a relatively straightforward conceptual solution to several dilemmas highlighted by this excellent series of papers: A careful consideration of our fundamental emotional nature. which like REM itself emerged in brain evolution long before sophisticated cognitive abilities, may be essential to make sense of the most activated phases of mammalian sleep and the dreams they energize. Slow wave sleep, and its duller dreams, may be more important for dealing with the less passionate cognitive deliberations and adjustments of the brain and body.

Neurotransmitter mechanisms of dreaming: Implication of modulatory systems based on dream intensity

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Abstract: Based on increasing dream intensity and alterations in neurophysiological activity from waking, through NREM to REM sleep, dreaming appears to correlate with sustained midbrain dopaminergic and basal forebrain cholinergic, in conjunction with decreasing brainstem 5-HT and noradrenergic neuronal activities. This, model, with features in common with the modulatory transmitter models of Hobson et al. and Solms, is consistent with some clinical observations on drug induced alterations in dreaming and transmitter correlates of delusions.
[HOBSON ET AL.; SOLMS]

The growing realization that dreaming occurs, to a greater or lesser extent throughout sleep, and essentially independent of REM, requires – as **SOLMS** suggests – a paradigm shift regarding neurobiological mechanisms. Linking dreaming to the function of individual transmitter systems is no doubt simplistic, given the multiplicity of transmitters that – as in waking consciousness or cognition – are likely to be involved. Nevertheless a hypothetical framework of selective neuromodulation provides the focus for investigations ranging from disease to drug effects. The original

evidence that brainstem cholinergic neuronal activation underpins REM sleep has for example been linked to REM sleep abnormalities in Lewy body disease in which pedunculopontine neuropathology occurs and REM behavioural disorders may be presymptomatic (Boeve et al. 1999). Objective measures of REM sleep, not yet established for dreaming, have no doubt provided much of the attraction of the original hypothesis that REM sleep and dreaming are equivalent in terms of brain mechanisms.

Because dreaming occurs throughout the sleep cycle, hypotheses regarding neurobiological mechanisms need to be based on patterns of transmitter activity that, rather than distinguish REM from NREM sleep, distinguish sleep from wakefulness. Moreover, if as evidence suggests, dreaming intensity is greater in REM than NREM sleep, transmitter correlates of dreaming should vary accordingly.

Based on activation or deactivation during REM and NREM (reviewed, Gottesman 1999, **HOBSON ET AL., SOLMS**) the following neuromodulatory systems have been implicated in dreaming: brainstem and basal forebrain cholinergic, brainstem 5-HT and noradrenaline, and midbrain dopaminergic. As dreaming normally ranges from none to moderate to more intense in parallel with changes for wakefulness to NREM to REM sleep, candidate transmitter correlates would be those demonstrating a similar gradation in activation or deactivation. Activity of dopaminergic neurons in the ventral tegmental area, implicated by SOLMS, is no doubt essential but not alone sufficient to account for dreaming, because these neurons are active throughout the entire sleep-wake cycle (Miller et al. 1983). Similarly basal forebrain cholinergic neurons are, to judge by cortical release of acetylcholine, active to a greater or lesser extent throughout (Marrosu et al. 1995). Brainstem cholinergic neuronal activation, originally highlighted by Hobson, cannot, as SOLMS concludes be essential since this does not generally occur during NREM. The only modulatory systems so far identified with the required variation in firing patterns are apparently the 5-UT and NE brainstem neurons which both demonstrate a gradation between wakefulness, NREM, and REM from active through less active to inactive (Hobson et al. 1975; McGinty & Harper 1976). Histaminergic neurons in the hypothalamus are incidentally equally silent during both NREM and REM sleep (Vanni-Mercier et al. 1984). As Gottesman (1999) has concluded, disinhibition in target projection areas including cerebral cortex, associated with declining 5-UT and noradrenergic activities, may underpin the generation of dreaming and explain the delusional, altered affective and other characteristics associated with information processing that is repressed during waking. It is thus suggested that dreaming is essentially associated with decreased 5-HT and noradrenergic activity in conjunction with the maintenance of VTA dopaminergic and basal forebrain cholinergic activities – a model that includes aspects of both models of transmitter modulation proposed by HOBSON ET AL. and SOLMS.

Continued activation during NREM and REM of midbrain dopaminergic and basal forebrain cholinergic neurons may provide the basis of, respectively, motivation or drive (albeit nonvolitional), and selective attention or conscious awareness (variably reduced) during dreaming. Variations in cholinergic function between REM and non-REM, which include brainstem neuronal activation in REM, and increased forebrain activation during REM compared to NREM, may contribute to the increased frequency and/or intensity of dreaming reported during REM. Since pontine cholinergic neurons activate VTA dopaminergic neurons (Gronier & Rasmussen 1998), enhanced dopaminergic function may also contribute to dream intensification during REM. However variations in reported intensity could reflect reduced levels of conscious awareness during NREM compared to REM. Reduced release of acetylcholine, which has been specifically implicated in conscious awareness (Perry et al. 1999), occuring during NREM compared to REM and alert wakefulness could contribute to decreased registration of dreams during NREM.

The proposed association between dreaming, on the one hand, and a distinct activation/deactivation pattern in specific modulatory transmitter pathways may not have the merit of simplicity, but is consistent with some aspects of dream physiology, pharmacology, and pathology. It is for example not easy to identify a single system which alone can account for the broad range of brain areas activated during dreaming, to judge from the lesion and in vivo neuroimaging data reviewed by **HOBSON ET AL.** and **SOLMS.** Innervation by dopaminergic axons, suggested by SOLMS, may correlate less well than innervation by dopamine and acetylcholine together and/or by 5-HT and noradrenaline. Reports regarding the dopaminergic innervation of primate cortex are variable, depending on the transmitter indices and, based on tyrosine hydroxylase, indicate that the motor cortex is particularly densely innervated (Berger et al. 1991; Parnavelas 1990), and yet this area is not reported to be activated during dreaming. Activated cortical areas, including the amygdala, limbic cortex, and also hypothalamus receive dense cholinergic in addition to dopaminergic innervations. Areas where activity is decreased during dreaming, such as primary visual cortex contains the highest density of 5-HT innervation and 5HT2 receptors (Parnavelas 1990; Pazos et a1. 1987).

Other aspects of dreaming which can be examined in the context of the multitransmitter hypothesis proposed in this commentary in response to the models of **HOBSON ET AL.** and **SOLMS,** are alterations in dreaming induced by drugs or related to disease. Alterations in dreaming are potentially relevant to clinical practise. Thus for example in Parkinson's disease, dreaming abnormalities (vivid dreams, nightmares, night terrors) have been reported to precede the occurrence of hallucinations, delusions and delirium induced by levo-dopa (Factor et al. 1995; Pal et al. 1999), and REM abnormalities have been linked to drug induced hallucinations in Parkinson's disease (Comella et al.1993). In relation to increased dreaming intensity associated with anti-Parkinsonian medication, it is interesting that pramipexole (a dopamine agonist with preference for the D3 and no affinity for the Dl receptor) is more likely to induce this as a side effect than l-dopa (Pal et al. 1999), and the distribution of the D3 receptor may provide information on the role of dopamine in dreaming. In Alzheimer's disease, treatment with cholinesterase inhibitors can induce dreaming abnormalities including nightmares (Ross & Shua-Haim 1998) that may restrict the use of this type of drug; similar increased dreaming has been reported in accidental cases of organophosphate toxicity (Warburton 1979).

There have been few reports so far on abnormalities in dreaming, independent of drug treatment, associated with diseases of the brain which involve degeneration of specific neuronal nuclei. Alzheimer and Lewy body types of diseases affect, to varying degrees all of the modulatory systems discussed above and alterations in dreaming are likely to provide new insights in disease mechanisms and prognosis. There is one report of increased dream intensity in patients with Parkinson's disease which was not correlated to medication (Van Hilten et al. 1993) and more recently of one patient with cessation of dream recall independent of medication (Sandyk 1997). It might be predicted that basal forebrain cholinergic and/or VTA dopaminergic pathology would be associated with decreased dreaming, locus coeruleus, and/or dorsal raphe pathology with increased dreaming, and various degrees of combined pathology with intermediate effects. More attention has been focused on the parallel between dreaming and delusions associated with various cerebral disorders especially schizophrenia. Dopamine receptor antagonists reduce delusional symptomatology in a variety of disorders. Delusions or psychosis in Alzheimer's disease have been related to reductions in 5-HT and 5-HIAA in the subiculum (Zubenko et al. 1991). In dementia with Lewy bodies delusions have been linked to elevated muscarinic Ml receptor binding in temporal cortex (Ballard et al., submitted). Delusions can be induced by organophosphate cholinesterase inhibitors in normal individuals (Warburton 1979), although in Alzheimer's disease delusions are reduced by such drugs as metrifonate. Pathology in the locus coeruleus and raphe nuclei may in this instance contribute to a differential drug response in the disease condition. If delusions during waking are closely re-

lated to a dream-like state, then these observations implicating dopamine, 5-HT, and acetylcholine are generally consistent with the transmitter model of dreaming suggested in this commentary.

In conclusion, original contributions to understanding neurobiological mechanisms of dreaming, such as those of **HOBSON ET AL.** and **SOLMS,** will no doubt continue to generate not only new models but also new directions for research in neuropsychiatric disease.

Metaphoric threat is more real than real threat

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Abstract: Dreams represent threat, but appear to do so metaphorically more often than realistically. The metaphoric representation of threat allows it to be conceptualized in a manner that is constant across situations (as what is common to all threats begins to be understood and portrayed). This also means that response to threat can come to be represented in some way that works across situations. Conscious access to dream imagery, and subsequent social communication of that imagery, can facilitate this generalized adaptive process, by allowing the communicative dreamer access to the problem solving resources of the community.
[REVONSUO; SOLMS]

REVONSUO believes that dreaming enhanced adaptive fitness in the ancestral environment and that dreams provide "perceptually and behaviorally realistic rehearsals of threatening events." His notion of "realistic," however, appears shaped by the implicit presumption that environmental selection mechanisms can best be considered as the array of things and situations that "leap out at us" and "cry out to be named" (Brown 1965, p. 478), and not as something more generally conceptualized. In REVONSUO's scheme, these are the particular and namable elements of the Pleistocene savannah – the specific dangers that lurked there (predators, enemies, disasters). It is not clear, however, that the ancestral and current human environments are fundamentally different, or that threat can best be mastered as a consequence of its "basic-level" representation.

On the surface, this appears contentious. How can the environment of the prototypical African human progenitor be considered reasonably equivalent to that of the modern individual? The problems we face while sitting at our computers seem very much unlike those of the more "natural" world. What constitutes threat, however, or even "environment," depends on level of abstraction, and there are levels that allow for representations of danger that are isomorphic across all conceivable frames of reference (Peterson 1999).

Let us first determine just what "threat" means, in the broadest possible sense. Humans are goal-directed (Adler, in Ansbacher & Ansbacher 1956; Gray 1982; Oatley 1999). Emotions, including anxiety, signal the interruption of specific goal-directed schemes of conceptualization and patterns of action. Anxiety signals threat, to be sure (Gray 1982) – but more generally indicates the emergence of the unknown or the anomalous (which is initially nothing but undifferentiated evidence for the insufficiency of current plans) (Peterson 1999). This means that the concrete dangers of the natural world may be most usefully considered specific exemplars of a more general category. This more general category – the anomalous – lurks everywhere; it is a universal constituent element of experience. This is because we dramatically simplify the world (Miller 1956), while engaging in our goal-directed processes, and because these simplifications may constantly be revealed as insufficient, in the real environment. It is such revelation that constitutes the most basic and universal threat (Binswanger 1963).

This implies that the dream may represent threat most usefully at the highest level of abstraction – that level allowing for most cross-situational generalization. Once this is understood, the relationship between the dream, consciousness, and the adaptive activity of cultural construction can be explicitly comprehended. Consider an actual dream, as exemplar – the production of a highly verbal five year old boy, about to leave his family and join the novel world of kindergarten. He was happy during the day, although deeply immersed in a pretend world: He spent much of his time dressed as a knight, with a plastic helmet and sword. He was not sleeping well, however, and frequently screamed for his mother late in the evening. One morning he described a nightmare. Armless, greasy, dwarf-like beaked creatures had been jumping on and biting him. Each creature had a cross shaved on the top of its hairy head. In the background loomed a fire-breathing dragon. The dragon exhaled smoke and fire, which promptly transformed itself into more biting beaked dwarves. Everyone who heard his dream report was fascinated and shocked.

It was clear that this boy had never really encountered biting dwarves or dragons. What possible purpose could such representation therefore serve? Well, after the boy had recited his tale, he was asked a question: "What could you do about this dragon?" This seems something simple, but it is not. It is instead the sort of utterance that allows a lawyer to "lead" a witness. It is a question full of "triggers" (Bruner 1986) or implicit information. The question says as much as it asks. It says, "something can be done about dragons," for example, and "small boys like you can do that something." This leading question therefore puts forth in exceedingly compressed form the plot and character elements necessary to successfully complete the narrative of the dream – that is, to solve the problem it poses.

The boy said, excitedly: "I would take my dad, and go after the dragon. I would jump on its head and poke its eyes out with my sword. I would go down its throat to the fire. I would cut out the box the fire came from, and make a shield from it." This is a complete and spontaneous recreation of a traditional hero myth – and hero myths detail successful encounters with the unknown (Peterson 1999). It is not necessary, however, to posit the derivation of this tale from the "collective unconscious" (Jung 1959). This boy had seen many movies, heard many stories, and had observed patterns of successful (and unsuccessful) real-world behavior. So the pattern for the "hero" was something thoroughly embedded in his social world. But he had never conceptualized himself as heroic. One leading question, however, provided sufficient motivation for that. His dream represented him as threatened by "archetypal" dangers – not so much by particular threats (in the form of the dwarves), but by threat itself (in the form of the dragon). When he reconceptualized himself, therefore – as a consequence of social prompting – he came to understand that he was more than someone who could face particular threats: He was someone who could overcome the class of threatening things itself. This is a far more useful conceptualization because of its cross-situational generalizability (and one that did in fact eliminate his nightmares).

The fact that it was social prompting that led to such reconceptualization also sheds light on an additional mystery. Why communicate dream content? There is a simple answer to this question: Two heads – or two thousand – are better than one. Traumatized individuals experience intrusive thoughts about the threatening occurrence (Tait & Silver 1989) and need to talk about their experience (Ersland et al. 1989; Rime 1995). Those denied opportunities to engage in social-mediation of such experiences tend to suffer more, in the aftermath (reviewed in Petrie et al. 1998). Why? What good does talking do? Well, ability to report on internal states in a communicable manner also means capacity to draw on the problem-solving resources of the community to deal with threat. This capacity to communicate dreams could have been selected for after the rise of language. So – threatening dreams become memorable and compel communication (become nightmares) precisely when they represent a threat so profound that it exceeds the current adaptive capacities of the dreamer. Such dreams are then reported, in a dramatic and intrinsically fascinating fashion. Then the community helps solve the problems they pose.

The global significance of this process should not be underestimated. Dreams are part of the lengthy, historically-elaborated process by which threats, as a class, come to be metaphorically represented – as something reptilian, for example, unpredictable, chaotic, devouring (Eliade 1978) – and then, as a class, come to be mastered (Peterson 1999). The construction of protective culture itself can reasonably be regarded as a consequence of this motivated process – not so much to escape from the specific dangers of the Pleistocene environment, but to alleviate the total consequences of human vulnerability, across all conceivable contexts.

SOLMS's observations on the potential dopaminergic mediation of dreams are interesting in this regard and help tie the threat representation capacity of the dream to its evident facility for bizarre conceptual portrayal. We know, for example, that dopaminergic activation is associated with exploration (Gray 1982) and with increased categorical flexibility (Ashby et al. 1999; Lubow 1989). These two phenomena are logically related: Categorical flexibility should increase during exploration, so that current schemes of apprehension may be modified as a consequence of learning. Adaptation to threat means either reconceptualization of self and the acquisition of new and relevant skills, or reconceptualization and recategorization of the feared object (Foa & Kozak 1986; Williams et al. 1989; 1997). Finally, we have the fact that general mood states in dreams tend to be positive (**REVONSUO**) – something in keeping with the first two phenomena, as dopaminergic activation is associated with positive emotion (Gray 1982; Ashby et al., 1999). This all implies that dreams may be positive, exploratory, creative play, when they are not dealing specifically with an anomaly intense enough to be traumatizing. So it seems reasonable to posit that dreams may be considered more broadly part of the process of adjustment to novelty, and that their facility for dealing with threat might be considered as something subsidiary to that broader function.

One machinery, multiple cognitive states: The value of the AIM model

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Abstract: The AIM model represents an original and comprehensive example of how changes in conscious states can be reconciled with specific neurophysiological factors. However, further elucidation of the biological parameters necessary to define a specific space-state relationship should be considered.

[HOBSON ET AL.; SOLMS]

HOBSON ET AL. and **SOLMS** review an impressive amount of phenomenological and physiological data in relation to waking, NREM, and REM sleep. The major objective of these authors is to find an integrative model of conscious experience in which distinct cognitive states may be quantified and tracked down to specific neurobiological events. The importance of the activation information processing modulation (AIM) model formulated by this group is owing not to this integrative approach only but especially to the understanding of consciousness as a multidimensional and dynamic process. Hence, normal and abnormal cognitive states find their own definition on the basis of the neurobiological parameters taken in account (only three in the AIM model but arguably many more). Thus the AIM-type unitary approach may be applied when studying cognitive states very different in appearance (e.g., hallucination, anesthesia, coma, etc.). In this regard the value of the AIM model goes far beyond the confines of sleep physiology.

Despite my overall appreciation of the AIM model, there are in my view several points of oversimplification that should be further discussed.

Activation. In the activation domain, it appears that the sleep-wake related brain activity shown by functional imaging studies has been overstressed and only one of the many possible interpretations of the data is offered to the reader. This commentary does not provide the space to address detailed criticisms of the functional imaging studies, however, some elements of misinterpretation should be considered. I will limit my comments to a couple of examples.

1. It has been suggested that the higher activation of the brainstem during REM sleep is dependent on intense neuronal activity of the REM sleep generator system. This is a likely possibility and one that I happen to favor, as do the authors, our shared opinion being that the brainstem generates REM sleep's phasic and tonic phenomena. However, I am interested in why brainstem activity should be higher in REM sleep than in wakefulness. It is known that the neuronal populations responsible for arousal are located in the brainstem. For instance, the locus coeruleus noradrenergic neurons, the raphe dorsalis serotonergic neurons, the reticular activating system neurons, and so on, all lie in the proximity of the REM sleep generator system (McCormick & Bal 1997). Also it is known that in the lateral dorsal tegmentum (LDT) and ponto pedunculum tegmentum (PPT), the number of neurons selectively active during REM sleep are outweighed by the number of neurons active during both REM sleep and waking or waking alone (Kayama et al. 1992). From this observation I would expect to see similar patterns of brainstem activation in both wakefulness and REM sleep. This point remains unresolved.

2. Another example is given by the higher activation of the anterior cingulate cortex during REM sleep compared to NREM sleep or waking. This effect has been discussed by the authors in the following way: "As in waking, anterior cingulate activation contributes additional emotional features to dreaming such as valence biases, the assessment of motivational salience, and the integration of dream emotion with fictive actions" (from Subcortical and Cortical limbic and perilimbic structures in the Activation Synthesis, sect. 3.4.4, **HOBSON ET AL.**). This interpretation is one possibility amongst several others. However, I question how we can reconcile this view with the large amount of data showing that the anterior cingulate cortex is a crucial part of the executive attentional and executive system and activates in tasks requiring performance monitoring and error detection (e.g., Awh & Gehring 1999; Carter et al. 1998). Such complex cognitive features do not easily fit with the authors' proposition that the dreaming brain lacks of self-awareness, judgment capability, volitional control, and so on. Finally, the higher activation of the anterior cingulate cortex contrasts with the lesion studies reviewed by **SOLMS** which show a correlation between anterior cingulate lesions and increased frequency and vivacity of dreaming (Solms 1997a). In conclusion, I suggest that neither the functional imaging nor the lesion studies results should be overemphasized at this stage. These results are not conclusive. In addition, several discrepancies (more than are usually considered) are present among different functional imaging studies (see Table 2 of **HOBSON ET AL.** article).

Information processing. The information processing domain of the AIM model implies a blockade of information flow during NREM and REM sleep in particular. Despite the fact that threshold for awakening is higher in NREM and REM sleep, there is evidence that sensory inputs are processed at the thalamo-cortical level during sleep (Mariotti & Formenti 1990; Pare & Llinas 1995) and a recent study has shown differential processing of relevant and irrelevant auditory stimuli during sleep (Portas et al. 1999). In addition, paradoxical phenomena like sleepwalking imply a certain degree of sensory processing (being sensory processing necessary for ambulation). Thus, the possibility of residual sensory processing and therefore cognitive functionality during sleep should be acknowledged and the concept of sensory blockade should be drastically reviewed in the AIM model.

Modulation. Another point worth discussing is the necessity of experiments that may address more directly the modulator mechanisms of the AIM model. The long, tedious (and necessary!) list of single cell recording, microdialysis, receptor binding, and so on, experiments used by the authors to support the validity of the Rec-

iprocal Interaction model, loses its strength when applied to explain the highly dynamic aspects of the AIM model. In fact, it is my belief that the dynamic functional models call for more dynamic experimental evidence. In other words it would be necessary to test on-line neuromodulatory interactions which are able to manipulate the space-state relationship of the AIM model (and consequently the conscious state). Ungerstedt and collaborators have made attempts in this direction. The Swedish groups, using microdialysis, systematically monitor the level of several neurotransmitters and metabolites in distinct areas of the human brain after traumatic or ischemic injuries (e.g., in humans: Hillered & Persson 1992; Nillson 1999; Persson & Hillered 1992; in animal models: Nillson et al. 1990). The aim of their study is to correlate patterns of decreased/increased level of neurochemicals with cognitive outcomes. More experiments of this type are required to test the neuromodulatory requirements of the conscious states.

Neural constraints on cognition in sleep

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Abstract: Certain features of Stage NREM sleep – for example, rhythmic voltage oscillation in thalamic neurons – are physiologically inhospitable to "REM sleep processes." In Stage 2, the sleep spindle and its refractory period must limit the incursion of "covert REM," and thus the extent of REM-like cognition. If these hyperpolarization-dependent events also inform Stage NREM cognition, does a "1-gen" model suffice to account for REM-NREM differences?
[NIELSEN]

To a debate that persists (despite the field's outgrowing it), **NIELSEN**'s target article proposes a singularly constructive and clearly stated resolution. This commentary examines the concept of covert REM sleep, which NIELSEN defines as "any episode of NREM sleep for which some REM sleep processes are present but for which REM sleep cannot be scored with standard criteria." Of the questions this definition raises, I shall address three: In which NREM episodes does "covert REM" *not* reside? Which "profiles of NREM Sleep physiology" *do* admit "REM sleep processes" and may thus generate "intermittent REM-like mentation"? Does that intermittence account robustly, as NIELSEN claims, for differences between Stage REM reports and Stage NREM reports?

1. In which NREM episodes does "covert REM" not reside? Omitting the K complex, Stage 2 sleep (on which I shall focus) is epitomized by the *sleep spindle.* In a neural network that topographically interconnects (GABAergic) reticular thalamic neurons and (glutamatergic) thalamocortical neurons, spindling indexes a repeating cycle of (1) slow membrane depolarization, mediated by the low voltage-dependent calcium current I_T, and (2) at action potential threshold, rhythmic burst discharge (for a review, see McCormick & Bal 1997). This cycle waxes and wanes at variable intervals; between spindles, thalamocortical neurons are depolarized. Do "REM sleep processes" take advantage of this intermittent depolarization to insinuate themselves – if only here and there – into NREM sleep?

Not necessarily. In my view, the spindle wave refractory period – the portion of the interspindle interval mediated by the hyperpolarization-dependent action current I_H – must prevent a ubiquitous invasion of covert REM into the interspindle interval. To be sure, the refractory period does inactivate I_T, replacing the rhythmic burst with the single spike as the unit of thalamic network neural discharge (McCormick & Bal 1997). But does the refractory period (1) excite the reticular activating system, both tonically and phasically; (2) phasically inhibit reticular thalamic neurons; and (3) depress the release of serotonin, norepinephrine, and histamine? I suggest that insofar as any NREM episode merely concatenates sleep spindles, that episode will exclude these REM sleep processes.

2. Which "profiles of NREM sleep physiology" do admit Stage REM processes? The interspindle interval in Stage 2 NREM does not always exclude covert REM. For one thing, standard sleep stage scoring rules, acknowledging both the necessity of spindling and its intermittence, allow an interspindle interval of up to 3 minutes in Stage 2. For another, the duration and constitution of the interspindle interval will change across the sleep period.

As **NIELSEN** rightly suggests, sleep's circadian architecture must influence the variability (and viability) of covert REM sleep. Indeed, in accord with the principle of "delta homeostasis," neural network state can be instantaneously quantified as the sum of (1) globally diminishing "delta power" (Feinberg & March 1995) and (2) periodically alternating hyperpolarization and depolarization in the thalamic network (the NREM-REM cycle; cf. Kahn et al. 1997). Therefore in the "missed REM period" – or in the first REM period, where Stage REM is visible but often mixes with spindling – network hyperpolarization may dominate emergent Stage REM at the surface electrode, but cannot obliterate it altogether in the thalamic neural network.

Thus question No. 2 may be answered as follows: "Covert REM" may reside in the interspindle interval in Stage 2 NREM, but only inside the 3 minute limit for scoring Stage 2, and only outside the spindle wave refractory period. On one hand, "REM sleep processes" may become increasingly salient in the interspindle interval as (cortically instigated) network hyperpolarization diminishes across the sleep cycle. On the other hand, the effect of diminishing delta power on the duration of the I_h-mediated spindle refractory period remains to be elucidated. Could waning delta power in fact aggrandize the refractory period?

3. Does the "intermittence" of REM-like mentation account robustly, as **NIELSEN** claims, for differences between Stage REM reports and Stage NREM reports? Clearly, the structure of the Stage 2 NREM episode can accommodate "intermittent REM-like mentation." Nonetheless, a "1-gen" model does not account for all of the differences between Stage REM reports and Stage 2 NREM reports. In my view the sleep spindle alone can support a robust "2 gen" model. It is well known that spindling opposes thalamic neurons' ability to respond articulately to stimuli in their receptive fields. In this and other ways, spindling is like its bigger, $GABA_B$ receptor-mediated cousin, the "spike and wave" network discharge that characterizes absence epilepsy. The symptoms of absence seizure run a gamut from "mental confusion" to "complete blackout" joined to a blank, staring facial expression (Penfield & Erikson 1941). Might "spindling mentation" display similar symptoms? Support exists for this comparison. For example, a set of Stage 2 mentation reports previously rejected as uninformative (Antrobus 1991) in fact contains many absence-like reports: "I blanked out," or – consonant with subjects' typical inability to recall absence – " I can't remember." In the same sample, significantly fewer Stage REM reports (in fact, virtually none) qualify as absence-like (Porte 2000).

If the sleep spindle does not produce dreamlike mentation, and if the spindle wave refractory period does not accept "REM sleep processes" but does (theoretically) produce mentation, a "second generator" of cognition in sleep – possibly, even a third – cannot be rejected out of hand.

In conclusion, I would like to ask whether *any* NREM episode displays the phasic, structured, endogenous network excitation that we measure at the surface electrode as (for example) eye movement, or by virtue of the system's adaptive response to strong phasic somatic motor excitation – as muscle atonia. Certainly NREM Stage 1, as **NIELSEN** would agree, is a candidate. But doesn't Stage 1 (minus the embryonic spindling that heralds Stage 2) occupy a physiologic and cognitive continuum with Stage REM? Take eye movement, for example. Might higher levels of serotonin, norepinephrine, and histamine – and thus greater reduction of "leak" potassium currents (McCormick & Bal 1997) – contribute to slower eye movement velocities in Stage 1 than in stage REM?

Likewise, do Stage 1 levels of those neuromodulators permit some somatic motor excitation, but not enough to warrant adaptive paralysis? In this light, I suggest that "REM-like" processes are not covert at all in Stage 1 NREM. Rather – owing to an old accident of stage classification, they are called by the wrong name.

By raising issues such as these, **NIELSEN**'s target article does much to move us beyond an old debate, clearing the ground for questions of greater current importance: What is the relevance of cognition in sleep to the structure of cognition in waking? What are the adaptive uses of spontaneous neural activity in sleep?

The contents of consciousness during sleep: Some theoretical problems

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Abstract: The approach of Hobson et al. is limited to the description of global states of consciousness, although more detailed analyses of the specific contents of consciousness would also be required. Furthermore, their account of the mind-brain relationship remains obscure. Nielsen's discussion suffers from conceptual and definitional unclarity. Mentation during sleep could be clarified by reconceptualizing it as an issue about the contents of consciousness. Vertes & Eastman do not consider the types of memory (emotional) and learning (implicit) that are relevant during REM sleep, and therefore dismiss on inadequate grounds the possibility of memory functions associated with REM sleep.
[HOBSON ET AL.; NIELSEN; VERTES & EASTMAN]

Dreaming provides us with a unique window to the way the phenomenal level (consciousness) is generated and organized in the brain. The study of dreaming and its neural correlates is an important source of empirical data for consciousness research: The dreaming brain can be regarded as a model system that isolates consciousness from the normal sensory and motor interactions and reveals consciousness in a very basic form (Revonsuo 1995). Progress in understanding dreaming and consciousness requires conceptual clarity and tight interaction between theory and empirical research. In this commentary I point out some conceptual and theoretical issues that have been inadequately dealt with in the target articles by **HOBSON ET AL., NIELSEN,** and **VERTES & EASTMAN.** The resolution of these issues would clarify considerably the positions advocated by these theorists.

HOBSON ET AL. present a detailed model of the states involved in waking and sleep. Such cross-disciplinary work is a step in the right direction, for it integrates the study of dreaming with mainstream cognitive neuroscience. However, some difficulties with the approach of HOBSON ET AL. remain. They wish to make their own position crystal clear on the more general mind-brain problem (sect. 3), but their view remains rather obscure philosophically. They say that distinctions at either level (mind or brain) imply the existence of isomorphic distinctions at the other: changes in brain function virtually guarantee concomitant changes in mental function, and, conversely, for each phenomenological difference it is possible to identify a specific physiological counterpart. While the latter claim (that there can be no mental difference without a corresponding physical difference) is commonly accepted by philosophers as the mind-brain supervenience relation (e.g., Kim 1998), the former claim is more problematic. Even if we accept supervenience between the mental and the physiological, it does not follow that all distinctions at the physiological level are necessarily accompanied by phenomenological ones: There can be physiological distinctions, realized at completely nonconscious levels, that have no direct counterparts at the level of conscious experience. HOBSON ET AL. seem to deny this. It remains unclear how exactly they construe the mind-brain relation: do they adopt the standard supervenience relation or not?

At the empirical level, **HOBSON ET AL.**'s model concentrates on global states instead of specific contents of consciousness. There are compelling methodological reasons for this: it is much easier to measure and model the physiological and phenomenological changes at a coarse-grained level, where only global states need to be distinguished from each other, than at a fine-grained level, where specific contents of consciousness and their neural correlates should be defined. The psychological and neurophysiological methods currently available may be inadequate for the latter task. However, studies at a fine-grained phenomenological level would seem to be the most revealing ones when we try to understand the mechanisms of consciousness. Consider the detailed analysis of dream bizarreness (to which Hobson's group has made important contributions, e.g., Rittenhouse et al. 1994).

The bizarreness of dreams is of particular interest for the study of consciousness, because the concept of "bizarreness" in dream research is closely related to the concept of "binding" in cognitive neuroscience and consciousness research (Revonsuo 1999b). Indeed, what dream researchers have conceptualized as different forms of bizarreness can in many cases be reconceptualized as referring to different forms of binding, or, to be precise, to different types of deviations or aberrations in the binding of dream images coherently together (Kahn et al. 1997; Revonsuo 1995; Revonsuo & Salmivalli 1995). For example, one specific form of bizarreness, the incongruity of dream images, can be characterized in the following way: Incongruous dream elements are ones that either have features that do not belong to corresponding elements in waking reality or that appear in contexts in which the corresponding elements would not appear in waking reality. Thus, seeing a blue banana, finding a banana growing in an apple-tree, or seeing the President of the United States in one's home, would all be examples of incongruous elements in dreams. These bizarre elements can be characterized in more detail with the help of the concept of binding. A blue banana is a good example of erroneous feature binding: the representation of "banana" in our semantic memory should primarily associate with bananas the color yellow and to a lesser degree the color green, but not the color blue. And a banana growing in an apple-tree, or the President having a cup of coffee in my kitchen, are cases of erroneous contextual binding: even though the individual elements of such dream images are internally coherent, the images do not fit together in the light of our semantic knowledge of the world. Another variety of dream bizarreness is the discontinuity of dream elements: for example, a banana may suddenly appear, disappear, or be transformed into an apple in a manner not possible in the waking world.

Discontinuity seems to be a case of defective binding across time: Successive dream images do not always retain or update individual phenomenal representations in a consistent manner, which results in sudden and inexplicable appearance, disappearance or transformation of objects, persons, and places in dreams. Explaining the different types of bizarreness in dream images thus turns out to be the task of explaining how the mechanisms of binding and the unity of consciousness operate during sleep.

Hence in addition to a general model about the global states of waking and dreaming, we need detailed phenomenological descriptions of the specific contents of dreams and neurocognitive theories to explain how the different kinds of dream images are generated in the brain. This implies further methodological improvement both in dream content analysis and brain imaging. Even more important, there should be fruitful conceptual integration between dream research and cognitive neuroscience. Phenomena described in traditional dream research by a set of more or less folk-psychological concepts (e.g., "bizarreness," "anxiety dream") should be reconceptualized in such a manner that their detailed description can be given in a cognitive neuroscience framework (e.g., "deviations of visual binding caused by the spreading of activation in a neural network," "threat simulation response"). This kind of cross-disciplinary conceptual integration will be necessary to reach **HOBSON ET AL.**'s ultimate goal, the reunification of the psychological and neuroscientific approaches in the study of consciousness.

NIELSEN attempts to answer the questions: What generates "mentation" during sleep? What are the causal mechanisms involved? He divides "sleep mentation" into two categories, "dreaming" and "cognitive activity." It seems to me that these basic concepts could be defined much more clearly and precisely, by emphasizing the fact that we are actually talking about varieties of subjective conscious experience during sleep. "Sleep mentation" includes all kinds of subjective (conscious) experiences during sleep. The crucial division is between contents of consciousness that fulfill the criteria of dreaming and those that do not. Reconceptualizing the issues with concepts that directly refer to the content of consciousness would give us the following, much clearer distinctions: (1) Complex, temporally progressing contents of consciousness during sleep (dreaming); (2) Other contents of consciousness during sleep (non dreaming; NIELSEN's "cognitive activity"); (3) Nonconscious information processing during sleep (NIELSEN's "cognitive processes").

Because **NIELSEN** does not make such clarifications, various conceptual and definitional difficulties abound in the target article. According to him, dreaming is likely to be defined as imagery that consists of sensory hallucinations, emotions, story-like or dramatic progressions, and bizarreness. NIELSEN's definition of "dreaming" thus comes dangerously close to the definitions typically given by the proponents of 2-gen models and criticised by the proponents of 1-gen models.

Although some dreams are emotional, dramatic and bizarre, most are not, and it would be a mistake to uncritically include these features in the definition of a dream. In order for us to ever resolve the empirical issues about the neural correlates of dreaming, it would be crucially important to first find a theoretically more neutral definition for "dreaming," for example, "dream" = subjective conscious experience during sleep that involves complex organized mental images which show temporal progression or change (Farthing 1992). Another conceptual confusion can be found in Figure 1 where **NIELSEN** says that "cognitive activity" is included in the class of "cognitive processes," which seems odd since the latter category consists of nonconscious or preconscious processes and the former of the subjective contents of consciousness. How could the contents of consciousness be a part of nonconscious/preconscious processes? It would seem more logical to treat the categories "dreaming," "cognitive activity," and "cognitive processes" as forming different levels of description. Phenomena at the lower nonconscious levels are probably necessary for the processes at higher levels of description, but the higher-level phenomena (e.g., dreaming) cannot be coherently depicted as "a part" of the nonconscious levels.

After the reconceptualization suggested above, the central question of the target paper could be formulated much more clearly: "Is there a qualitative difference between contents of consciousness during REM sleep and NREM sleep, and must this possible difference be explained by referring to two distinct causal mechanisms or just one?" It is difficult to see how any evidence that does not directly reflect the contents of consciousness could resolve this issue one way or the other. In spite of this fact, only three of the nine lines of evidence that **NIELSEN** considers are like that. However, it does not suffice to show that REM and NREM differ with respect to physiology or nonconscious information processing. Such differences are not at the correct level of description and reveal little if anything about possible qualitative differences in the contents of consciousness.

NIELSEN introduces the concept of "covert REM sleep": an episode of NREM sleep for which some REM sleep processes are present, but for which REM sleep cannot be scored with standard criteria. These REM sleep processes are responsible for bringing about the "imaginal experiences," that is, contents of consciousness (dreams and non-dreams) during NREM as well as during REM sleep. It remains unclear how exactly NIELSEN's model disagrees with the paradigmatic 1-gen model by Foulkes who says that "we have one dream production system, rather than two (or many). It explains non-REM dreaming as involving some measure of degradation in the operations of the same dreaming system that is generally working at full steam during REM sleep" (Foulkes 1985, p. 61). This position seems to be entirely consistent with NIELSEN's model; the only difference is what Foulkes calls "degradations in the operations of the dream production system," NIELSEN calls "covert REM processes." This raises the suspicion that the novelty of postulating "covert REM processes" may be more verbal than substantial.

VERTES & EASTMAN claim that brain activity in REM sleep is "inconsistent with the orderly evaluation, organization and storage of information." Such a conclusion is, however, not quite implied by the evidence. VERTES & EASTMAN fail to consider the possibility that memory systems other than working memory and explicit episodic memory might be at work during REM sleep. The neurophysiological evidence appears to be consistent with the hypothesis that memory systems specialized in the processing of emotionally charged memories are very active during REM sleep. Limbic areas (especially the amygdala) are very active in REM sleep. Recent evidence by Hamann et al. (1999) shows that the human amygdala modulates the strength of conscious episodic memories according to their emotional importance. Other researchers (e.g., Metcalfe & Jacobs 1998) have recently proposed that there is an amygdala-centered "hot" emotional memory system, separate from the "cool" hippocampal system. Thus, areas very central to the evaluation and processing of episodic memory traces are highly active in REM sleep, which shows that, contrary to VERTES & EASTMAN's claims, REM sleep is consistent with the orderly evaluation, organization, and storage of emotionally charged information. Although this processing does not typically lead to the consolidation of explicit episodic memories, this does not exclude the possibility that other types of memory traces might be formed or strengthened. There are in fact good reasons to believe that REM sleep and dreaming implicitly strengthen memory patterns related to threat perception and avoidance (see my target article in this issue). VERTES & EASTMAN dismiss the possibility of any memory processing or consolidation in REM, although they fail to consider all the different kinds of memory systems that exist in the brain.

Furthermore, if the principal function of REM sleep is to decrease the harmful side effects of prolonged low brain activation associated with slow wave sleep, as **VERTES & EASTMAN** claim, then shouldn't it follow from this that total sleep deprivation (at least in small doses) – that is, avoiding the harmful low-level activation altogether so that REM is not needed to fight its side-effects – is in fact less harmful than pure REM-deprivation? Should we not be better off totally without sleep than wakened after a couple of hours of SWS but with little if any REM? Such an implausible prediction would seem to flow from their hypothesis.

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Search activity: A key to resolving contradictions in sleep/dream investigation

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Abstract: The target articles on sleep and dreaming are discussed in terms of the concept of search activity integrating different types of behavior, body resistance, REM sleep/dream functions, and the brain catecholamine system. REM sleep may be functionally sufficient or insufficient, depending on the dream scenario, the latter being more important than the physiological manifestation of REM sleep. REM sleep contributes to memory consolidation in the indirect way.
[NIELSEN; REVONSUO; SOLMS; VERTES & EASTMAN]

General introduction. My approach to all problems discussed in this *BBS* special issue is based on the concept of search de-

signed to change the situation (or the subjects attitude to it) in the absence of certainty about outcome but with constant monitoring of outcomes at all stages of the activity. Search activity, manifesting itself in flight, fighting, creativity, orienting behavior, and so on, increases body resistance, especially in a stressful situation (Rotenberg & Arshavsky 1979; Rotenberg 1984). Search activity is absent in stereotyped behavior.

Renouncing search (giving up) manifests itself in freezing, panicky behavior, helplessness, depression, neurotic anxiety; this is maladaptive, regressive, and decreases body resistance. It is important to emphasize that the positive effects of search activity on survival depend on the process of searching per se, not its pragmatic results. Search activity may diminish not only after continuous failures but also after very meaningful achievements, causing the diseases attending achievement. In functionally sufficient REM sleep dreams, represent a special form of search activity which compensates for the renouncing search in prior wakefulness and restores search activity in the subsequent wakefulness (for details see Rotenberg 1993a). With this general introduction, let us now turn to comments on particular papers.

1. The differentiation of functionally sufficient versus insufficient REM sleep [Solms]. A very clear and convincing differentiation is presented by **SOLMS** between brainstem mechanisms responsible for REM sleep as a physiological condition for dream mentation, and forebrain mechanisms responsible for dream mentation, as a psychological phenomenon. Although previous investigators understood that forebrain mechanisms are obligatory for the subjective dream experience, two contradictory and restrictive approaches to this topic have dominated the scientific literature. According to the first approach, REM sleep performs purely physiological functions not requiring any subjective experiences, while the dream is a side effect of general brain activation in REM sleep. As a result, the dream represents some occasional combinations of images free from any special psychological function. The second approach, although more common, is less definitively expressed in the literature. This approach acknowledges the importance of psychological functions in dreams, yet implicitly connects dream experience to REM sleep as its natural, obligatory, and inseparable component. According to this point of view, which has caused many paradoxes and misunderstandings, forebrain mechanisms responsible for dream production are considered to be a part of the REM sleep system. If REM sleep is considered a physiological mechanism isomorphic to dream mentation, it is difficult to reconcile with the fact that in mental and psychosomatic diseases, physiological REM sleep mechanisms are often strained but idle, being "empty" of dream content. Thus, sleep structure is not in itself sufficient to indicate whether REM sleep is efficient. It is also necessary to go on and investigate dream reports.

A few years ago, I suggested distinguishing functionally sufficient and functionally insufficient REM sleep (Rotenberg 1988). The latter is characterized by impoverished dream content or by the absence of dream reports after awakenings during REM sleep. I suggested that dream experience is responsible for the adaptive capacity of REM sleep. Based on **SOLMS**'s concept, functionally insufficient REM sleep is characterized by dissociation between the preserved brainstem mechanisms controlling REM sleep and the disturbed forebrain mechanisms responsible for dream experience. SOLMS presents strong arguments for the role of forebrain mechanisms dream generation. I would add only that a correlation between forebrain focal epileptic seizures and recurring anxious dreams does not confirm that dreams are produced by forebrain mechanisms. It shows only that dream content can be influenced by forebrain activity.

Although animals do not report dreams, they probably do have dreams based on their behavior in REM sleep without muscle atonia. If so, they may also demonstrate the dissociation between REM sleep and dreams, and presumably in such cases REM sleep will likewise be functionally insufficient. This may be pertinent to the contradictions in the data on REM sleep alterations under stress or sleep deprivation. I would conjecture that the predominance of hippocampal theta rhythm in REM sleep indicates an integration of REM sleep and dreams in animals.

I highly appreciate **SOLMS**'s hypothesis that dreaming is generated by the dopamine circuit which instigates goal-seeking behavior. This hypothesis accords concepts of search activity (Rotenberg 1984; 1993a), which starts in the presence of a certain critical level of brain monoamines utilized in the course of search activity. However, in parallel with utilization, brain monoamines are continuously restored in the process of search activity, providing the circuit with positive feedback (Rotenberg, 1994a). In the state of renouncing search, this positive feedback is blocked. This agrees with SOLMS's suggestion that dreaming is generated by the same dopamine circuit that stimulates goal-seeking behavior. However, I expect that other monoamines such as norepinephrine may also participate in this mechanism.

It is important to stress that, although in pathology both systems – brainstem system for REM sleep, and forebrain system for dream generation – may be separated from one another; in healthy subjects both systems function in a highly integrated way. REM sleep provides the best physiological condition for dream generation: on the one hand, it causes a massive general activation of the forebrain; on the other hand, contact with the outside world is substantially reduced. At the same time, the relationship between the two systems is reciprocal: Dreams may have a secondary influence on REM sleep physiology. For instance, there is evidence for a directional correspondence between dream imagery and rapid eye movements in REM sleep (Herman et al. 1984). It has also been shown that in healthy subjects eye movement density correlates with the active participation of the dreamer in dream events (Rotenberg 1988). In his attempts to show the independence of two systems, **SOLMS** underestimates some of these facts. Nonetheless functional interrelationship between the systems does not diminish the centrality or importance of his conclusion that these are two separate systems which can be disintegrated in some special conditions.

2. Behavioral attitude in dreams: The main variable. [Revonsuo]. The topic of **REVONSUO**'s target article is especially interesting and intriguing if we consider the evolutionary context and biological function of dreaming. In most investigations, biological functions are ascribed to REM sleep, while dream mentation is considered to belong to the domain of human psychology. However, the behavior of animals in REM sleep without muscle atonia (the model of Jouvet and Morrison) allows speculation that animals may also have dream experience (see Jouvet & Delorme 1965; Morrison 1982). Thus, an integrative theory of dream function must be relevant to humans as well as animals, and to psychology as well as biology. There are many inner contradictions in REVONSUO's paper, however, which are not sufficiently discussed, even though the author seems aware of some of them.

2.1. Revonsuo's main idea is that the biological function of dreaming is to simulate threatening events in order to rehearse threat perception and the appropriate threat avoidance skills. He stresses that in order to perform this function, dreams must contain recurring realistic threat simulations (perceptual realism) and must activate realistic avoidance skills relevant to these threats. Rehearsing threat avoidance skills in the simulated environment in dreams is likely to lead to improved performance in real threat avoidance situation in wakefulness, in the same way that mental training for specific movement skills improves these skills in real behavior.

REVONSUO supports his hypothesis with data from numerous investigations of dream content containing threatening situations critical for physical survival, for example, confrontations with wild animals, snakes, aggressive strangers, and so on. One can agree with the author's conjecture that such dream contents mirror the threatening environment of our ancestors. However, in modern society the real threat in most cases is psychological rather than physical. Precise behavioral strategies that are relevant for the ancestral environment are not relevant for modern life. They are not applicable to modern threatening events such as failures, injuries, and conflicts in different social relationships. Such failures threaten

not physical survival, but survival of the self. The special skills trained by fighting or escaping aggressive animals in dreams are not useful in coping with modern social threats and frustrations. In this context, the ancestral dream content has no sense for the modern life and can be considered a psychobiological atavism.

The concept of dream scenario preparing the subject for waking behavior does make sense, however, in the context of the search activity concept discriminating two opposite types of behavior in a stressful situation. Surprisingly, **REVONSUO** has missed this concept in his very substantial review of psychological and biological functions of dreams, although it is very relevant and avoids the fundamental contradiction noted earlier. If the task of the dream is not to prepare the subject for a special threatening situation, but to restore search activity as a general adaptive mechanism, then dream events have no special meaning. They may reflect recent modern problems or problems more typical of the ancestral environment. *The only important feature of the dream content is the dreamer's behavior in the dream scenario: search activity versus renunciation of search.* I suppose that even in ancient times, dreams performed the same function because the restoration of search activity after giving up is more important for adaptive waking behavior and survival than even the training of special skills.

2.2. In considering the dream as a simulation of the threatening system, **REVONSUO** does not acknowledge that such a simulation may evoke different forms of behavior – not only successful coping strategies but also giving up (helplessness). As a result, he comes against another very serious inner contradiction. In his view, nightmares and recurring threatening dreams are typical and efficient threat stimulation dreams. Thus, they must have high adaptive value and increase adaptive skills in wakefulness. However, for clinicians, nightmares (a typical symptom of post-traumatic disorder, PTSD) are a pathological sign. Sleep disturbance associated with nightmares is difficult to consider as a side effect of the positive outcome of anxiety dreams because there are no positive effects on waking behavior. In nightmares, the subject is usually a helpless victim of threatening events, which means that the dream is functionally insufficient and unable to restore search activity.

Space limitation does not permit me to discuss other contradictions, such as those related to the nature of images of wild animals in the dreams of children. The author ignores a well known fact that for children events in fairy-tales are as real as objective reality, and this may be the source of such images, especially since they are absent in dreams of Bedouin children who have no experience with such tales.

3. Covert REM state: Possible physiological and psychological manifestations [Nielsen]. A covert REM sleep process in NREM sleep would explain dream-like reports after awakenings in NREM. Many studies on humans as well as animals confirm this. This idea is also free of internal contradictions compromising the 1-gen and 2-gen models. Aside from all the contradictions cited by **NIELSEN**, I wish to stress that the qualitative similarity between REM and NREM mentation implied by the 1-gen model does not correspond to different effects of REM and NREM deprivation on mental state and behavior in humans. On the other hand, as has been shown, the 2-gen model has problems in explaining the similarity between REM and NREM reports.

Among data confirming REM state intrusion in NREM there is the forgotten investigation of Toth (1971). Using special subtle electrodes connected directly to eyelids, Toth detected, in different stages of NREM, short periods of electrical activity very similar to that accompanying small eye movements. When awakened in these NREM periods, including SWS, healthy subjects reported dream-like states. This has not been replicated so far as I know. However, if it is valid, this method can be used in psychophysiological investigations of NREM mentation, allowing quantitative measurement of covert REM states.

In our work on galvanic skin reaction (GSR) distribution in night sleep 25 years ago (Rotenberg et al. 1975), we detected a phenomenon presumably related to covert REM states in NREM. It is well known that in healthy subjects, GSR is usually less prominent in the first cycle than in the second (first cycle just after the first REM sleep episode). It is usually very visible, as if REM sleep were changing some physiological conditions allowing GSR to express itself. At the same time, in the first cycle, GSR is greater in stage 2 following SWS than in stage 2 before SWS. In the second cycle there is no such difference. Moreover, in the first cycle in SWS alone, GSR often does not increase for some period and then increases suddenly. Sometimes it looks like an explosion of GSR. It looks as if A is a critical point in the first cycle (not necessary in SWS) after which GSR begins to increase. In view of the regular increase of GSR in the second cycle after the first REM sleep period, one can conjecture that such a critical point in the first cycle corresponds to the covert REM state.

The idea of a covert REM state in NREM sleep may be helpful in explaining paradoxes of total sleep and REM sleep deprivation in animals. There is a discussion in the literature about whether NREM sleep and pre-REM state can partly repay REM sleep debt (see *Sleep,* 1999, n. 8). If NREM sleep includes covert REM-like states, such a compensation may be possible. It is also possible that the shift of REM sleep towards sleep onset (decrease of REM sleep latency) in depressed patients reflects the unmasking of covert REM sleep episodes. Although covert REM sleep in NREM sleep can explain a substantial portion of the data on NREM mentation, it does not explain it all. Not all mental experiences in NREM sleep are caused by covert REM sleep episodes in NREM sleep, and **NIELSEN** realizes this. Thus, his model does not reduce the importance of distinguishing mentation during "pure" NREM and during REM sleep incorporation in NREM. As **NIELSEN** stresses in his review, self-reflectiveness and self-involvement are especially typical of REM reports. Self-representation maybe a singular feature of dream mentation. In humans, the dream serves to undo repression (Grieser et al. 1972) and is an important part of psychological defense mechanisms protecting the self from disintegration. Self-representation in dreams may be related to this dream function and perhaps can be used as a sign of true dream experience. On the other hand, active self-participation in a dream scenario is crucial for the restoration of search activity as a primary biological function of dreaming (Rotenberg 1993a).

I propose that there is also another feature of true dream mentation separating it from any other form of sleep mentation. This is related to the right-hemispheric nature of dream experience. In contrast to the left-hemispheric monosemantic way of thinking, which is dominant in wakefulness and in NREM, typical dreams are polysemantic (see Rotenberg 1994b). This is evident in subjects' reflections on their own dream reports after awakenings. While remembering all details of dream images and events, subjects feel that their report is missing something very important, the thing that made the dream experience so meaningful and affective. Subjects feel that something very substantial and crucial has been lost during the presentation of the dream content, and that the dream story they are reporting differs significantly from their actual experience. There are numerous relationships between dream events and images forming the polysemantic context of the dream; these are lost during a verbal monosemantic presentation.

4. REM sleep and memory consolidation [Vertes & Eastman]. I agree with **VERTES & EASTMAN**'s main conclusion that REM sleep does not in itself play a crucial role in memory consolidation. However, my approach differs enough to make it worthwhile to discuss this point in detail. The studies have been performed on animals, and the authors present a vast amount of experimental data. They emphasize that the pedestal REM deprivation technique includes stress, which may cause the negative outcome of REM deprivation on memory and behavior. This is a reasonable assumption. However, one must go on to discuss the particular nature of this stress. Stress on the pedestal includes the relative restriction of activity, isolation, deprivation of search activity, regular frustration of the biological need for REM sleep, and punishments for any attempt to achieve REM sleep. Such regular frustration and punishment usually causes helplessness. Stress

evoked by the combination of these factors increases the requirement for REM sleep in order to compensate for the lack of search activity (Rotenberg 1993a). However, REM sleep is prevented. As a result, helplessness is not abolished, and this state itself may cause failures in testing memory function (Rotenberg 1992). Thus, I agree that REM sleep alone does not have a special function in memory consolidation. However, REM sleep may have indirect positive influence on memory as well as on many other functions in compensating for a state of helplessness (renouncing search) which is harmful for any mental and biological functions and even for survival (see General Introduction). Performance deficits are only a part of this general impairment of all functions caused by REM deprivation on pedestal.

In order to induce this state of search renuciation, a stressful situation combined with REM sleep deprivation has to last long enough. This can explain the data of Fishbein (1971), that the marked deficits in performance appeared only after REM deprivation for longer than one day. The state of helplessness caused by REM deprivation prior to training can also explain later impaired learning. Thus, I agree with **VERTES & EASTMAN** that performance deficits caused by REM deprivation can explain the data memory consolidation in REM sleep. However, the performance deficit in itself and the disturbance of mental functions is an outcome of renouncing search (helplessness). This is not caused by stress alone, but by the combination of stress and ensuing helplessness with the parallel suppression of REM sleep. If REM sleep is not prevented and if it is functionally sufficient, it compensates for the state of helplessness. If stress is less prominent, such as in the rotating water tank, the development of this state will take more time.

The same line of reasoning can explain the results of the work on humans. First, not every learning situation is stressful enough to produce a state of helplessness. In the human studies, experimental paradigms which can evoke this state are rarely used. It is obvious that complex tasks are more available to produce this state than simple tasks, and it is exactly these complex tasks as opposed to simple tasks that are affected by REM deprivation

VERTES & EASTMAN present an interesting discussion of the function of hippocampal theta rhythm in wakefulness and sleep. I agree that theta rhythm is involved in the mnemonic functions of waking and not those of REM sleep. However, I cannot agree that theta rhythm in REM sleep is a byproduct of the intense activation of the pontine region and has no function. Moreover, I assume that in wakefulness and in REM sleep this rhythm displays the same state-search activity which can manifest itself in different forms of overt behavior and in dreams. This is based on a balance between theta rhythm in wakefulness and REM sleep: The more pronounced it is in wakefulness, the less it is in subsequent REM sleep, and even REM sleep itself is diminished (Oniani & Lortkipanidze 1985). In wakefulness, mnemonic functions (selection and encoding of information) require search activity and correlate with the theta rhythm. In REM sleep, search activity does not relate to mnemonic functions but compensates the lack of search activity in the preceding wakefulness.

I agree that the fact that REM sleep reduction with antidepressants is not accompanied by memory disturbances is good evidence against memory consolidation in REM sleep. I have presented some additional supporting arguments (Rotenberg 1992): (1) Activating drugs such as amphetamine have a beneficial effect on memory, although they suppress REM sleep; (2) REM sleep has a tendency to increase with neuroleptic treatment and with reserpine, although this does not have a beneficial effect on memory.

My final comment is related to **VERTES & EASTMAN**'s hypothesis that REM sleep is a mechanism used by the brain to ensure and promote recovery from sleep. I disagree, and not only because I ascribe a function to REM sleep in restoring search activity. This hypothesis is also inconsistent with certain facts: (1) In humans, REM sleep is concentrated in the last third of the night when NREM sleep is superficial; (2) On the first night of rebound effect after sleep deprivation, sleep in psychologically stable subjects is deeper than usual and contains increased SWS but not increased REM sleep. According to **VERTES & EASTMAN** it would be reasonable to expect a compensatory increase of REM sleep. In addition, there are special mechanisms protecting the brain from unlimited increases in SWS – spontaneous shifts to the more superficial sleep stages, and shifts after body movements. Both types of stage shifts, and especially shifts after body movements, cause decreases in SWS.

General conclusion. By taking into consideration (1) the fundamental difference between two types of behavior in wakefulness and dreaming, and (2) the role of the functionally sufficient REM sleep in the restoration of search activity, one can explain many contradictions in the data related to REM sleep functions in dream experience, resistance to stress, and memory consolidation.

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Some myths are slow to die

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Abstract: Solms and the other authors in this series of *BBS* target articles accept the findings that the executive control of the REM/NREM cycle is still localized within a narrow region of the pontine brainstem. However, recent findings challenge this notion. We will review the recent data and suggest instead that the hypothalamus is the primary regulator of states of consciousness. If the hypothalamus indeed controls all the fun stuff, such as sex, eating, drinking, sleeping, and so on, then one can more easily accept Solms's argument that dreams are also generated from the forebrain. **[SOLMS]**

Nauta (1946) found that the hypothalamus regulates sleep and wakefulness. However, his findings were set aside because Moruzzi and Magoun's work (1949) began to emphasize that an ascending reticular activating system emanating from the brainstem activated the cortex. The idea of an ascending brainstem arousal system took firm hold, once it was established in (Dahlstrom & Fuxe 1964) that noradrenergic neurons were localized to specific regions of the brainstem. During that period, Jouvet also demonstrated changes in the electroencephalogram following transections and lesions of the brainstem, which prompted him to propose the monoaminergic theory of sleep-wake regulation (Jouvet 1969). Galvanized by the neuroanatomical and physiological data, investigators began to monitor the firing patterns of neurons within the brainstem to determine whether neuronal firing could be associated with specific sleep-wake states. For half a century, the transection, lesion, and electrophysiology studies have been the driving force that has shaped theories regarding sleep-wake regulation.

Although much of the sleep research community focused on the brainstem, a group of investigators at UCLA, headed by Carmine Clemente and Barry Sterman, who continued to investigate the hypothalamus. Studies from that group supported Nauta's findings that the preoptic area was important for sleep (reviewed in Szymusiak 1995). For instance, lesion of the preoptic area produces long-lasting insomnia while electrical or pharmacological stimulation and warming induces sleep. Electrophysiology studies have identified sleep active neurons in this region (Szymusiak et al. 1998). Recent studies (Sherin et al. 1996) have identified a specific neuronal group within the preoptic area that projects monosynaptically to wake-active neurons in the tuberomammillary nucleus (TMN) in the posterior hypothalamus. These neurons are located in the ventral lateral preoptic (VLPO) area and are sleep-active based on electrophysiology and c-Fos studies. Neurons in the VLPO are GABAergic (and also contain the inhibitory peptide galanin) (Sherin et al. 1998), and their activation would release in-

hibitory agents onto wake-active posterior hypothalamic wake-active cells and sleep would ensue (Shiromani 1998). Lesions of the VLPO produces long-lasting insomnia (Lu et al. 2000).

Recently, another group of neurons were identified in the posterior hypothalamus as being important for the regulation of wakefulness (De Lecca et al. 1998; Sakurai et al. 1998). These neurons contain the neuropeptide hypocretin, also named orexin. These neurons project to virtually the entire brain and spinal cord, providing especially heavy innervation to regions implicated in the regulation of wakefulness such as the TMN and the locus coeruleus (Nambu et al. 1999; Peyron et al. 1998). This peptide exerts an excitatory influence on target neurons (Hagan et al. 1999; Horvath et al. 1999) and some believe that this peptide is important for wakefulness and given the innervation of neurons implicated in wakefulness (Peyron et al. 1998). Consistent with this possibility it has been found that canine narcolepsy is associated with a mutation in the hypocretin-2 receptor (Lin et al. 1999), and another study found that orexin knockout mice sleep more at night, have more REM sleep, and also experience cataplexy (Chemelli et al. 1999).

The sleep disorder narcolepsy has provided insight into the neurobiology of sleep-wakefulness. That the disorder is associated with a neuronal population located in the hypothalamus is one more reason to stop looking to the pons and the brainstem for all the answers. The hypothalamus also contains neurons regulating other vital bodily functions, such as feeding, drinking, sexual behavior, and temperature. Moreover, the suprachiasmatic nucleus, which represents the master clock, also resides in the hypothalamus. Thus, it is not at all surprising that neurons regulating another fundamental behavior, sleep, are also present here.

Time course of dreaming and sleep organization

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Abstract: The complexity and mysteriousness of mental processes during sleep rule out thinking only in term of generators. How could we know exactly what mental sleep experience (MSE) is produced and when? To refer to REM versus NREM as separate time windows for MSE seems insufficient. We propose that in each cycle NREM and REM interact to allow mentation to reach a certain degree of complexity and consolidation in memory. Each successive cycle within a sleep episode should contribute to these processes with a different weight according to the time of night and distance from sleep onset. This view would avoid assuming too great a separation between REM and NREM functions and attributing psychological functions only to a single state.
[**NIELSEN**]

The experimental approach to dreams (or to mental sleep experience (MSE; Cipolli et al. 1981; Salzarulo & Cipolli 1979; Salzarulo et al. 1973) underwent enormous development in the sixties, mainly because of the heuristic value of the discovery of REM sleep by Aserinski and Kleitman (1953). For the next two decades (see Schulz & Salzarulo 1993), however, much less work was devoted to dreams. The theoretical work of **NIELSEN** in this issue is accordingly welcome and much appreciated. What follows are a few remarks and a proposal concerning problems raised by the complex **NIELSEN** paper.

One of the main arguments developed by **NIELSEN** concerns whether sleep mentation is generated by a single or a double source. Sleep mentation is assessed from reports obtained after REM and NREM sleep. This raises some questions. The term "generator," referring here to mental activity, reminds us of physiological terminology and concepts (e.g., the use of the term generator for PGO activity, Siegel 1989). This use of physiological paradigms for the study of mentation has led to equating dreaming with REM sleep, which has been criticized (Lairy & Salzarulo 1975). Now, NIELSEN is speculating about 1 versus 2 generators, that is one versus two forms of sleep mentation. Why look for two generators? First, there are two "containers," that is two sleep states, REM and NREM. Second, in a well known experimental work, Foulkes (1962) showed that there could be a substantial recall of sleep mental activity not only in REM sleep (Dement & Kleitman 1957b) but also in NREM sleep. Once it was established that mental activity could also occur in NREM, it was question of comparing NREM and REM mental activity. Psychological measures were used by some researches, while others (including the NIELSEN paper) used mainly physiological measures (see for discussion Salzarulo et al. 1973).

The complexity and the mysteriousness of mental processes during sleep rule out thinking solely in term of generators. How can we know precisely what MSE is produced and when? Are we sure that the indicators of physiological events, with their specific time-windows, are useful for "localizing" mental processes in time? What kinds of events should be included? To refer constantly to REM versus NREM is in our opinion insufficient. There is another possibility partially connected with the last part of the NIELSEN paper.

Retrospective evaluation of mental functions from post-awakening recall has revealed prominent differences in memory processes between NREM and REM (Cipolli 1995). Analyzing memory to understand sleep mentation is one of the methods used by some researchers in recent years. These studies not only explained differences between REM and NREM reports (see Antrobus 1983; Salzarulo & Cipolli 1979) but also found various degrees of consolidation of MSE using psycholinguistic indicators (Salzarulo & Cipolli 1974; 1979). Cipolli et al. (1998) further showed that memory consolidation improves across the night sleep cycles thanks to an iterative process. Indeed, a role for sleep cycles in memory processes, rather than sleep states per se, has been demonstrated recently (Ficca et al., 2000; Mazzoni et al. 1999; Salzarulo et al. 1997).

We proposed that in each cycle NREM and REM interact to allow mentation to attain a certain degree of complexity and consolidation in memory (Mazzoni et al. 1999; Salzarulo 1995). Each successive cycle within a sleep episode should contribute to these processes with a different "role" (weight) according to the time of night and distance from sleep onset. This avoids assuming too great a separation between REM and NREM functions and attributing psychological functions to a single state; it also emphasizes the temporal dimension and sleep organization.

To emphasize the usefulness of taking into account the temporal dimension in the comparison between physiological and psychological processes, we show in Figure 1 the time course of EEG activity (Dijk et al. 1991) and memory processes involved in dream recall (Cipolli et al. 1998). The increasing number of units consolidated in memory parallels the decreasing amount of slow wave activity. Hence, the physiological S process declines in parallel with increasing of memory consolidation.

In conclusion, I prefer not to speak about (or to seek) dream generators; instead I see physiological sleep activities as conditions (frames) within which sleep mentation can be elaborated and consolidated in memory. Consolidation can be achieved by iterative access to contents, possibly related to a single production system. Beyond this, how the production system functions, and why it starts to function during sleep, still remains a mystery.

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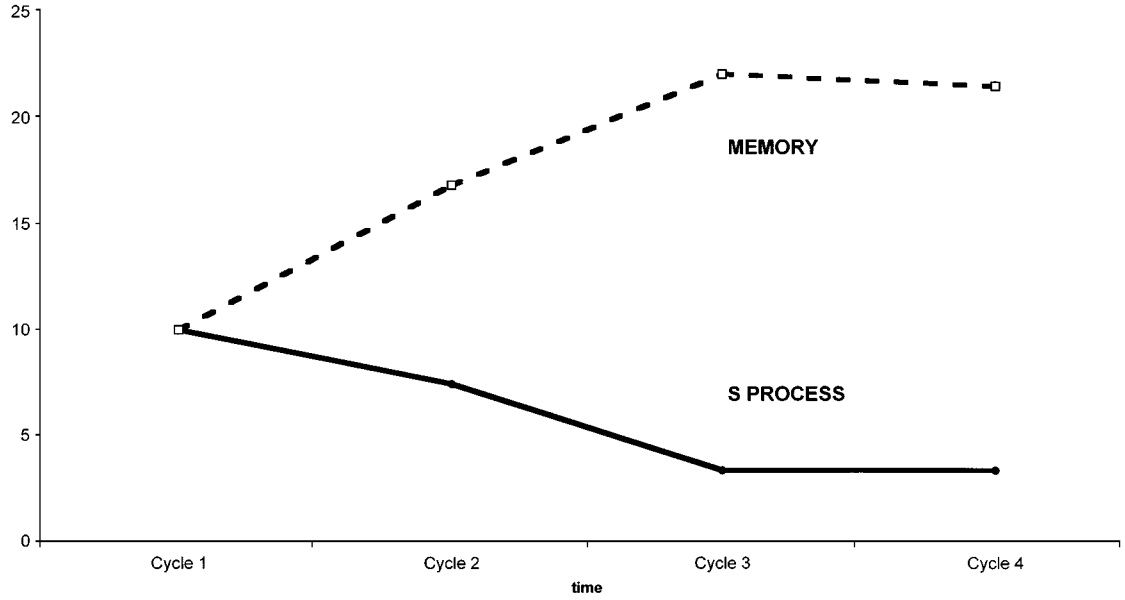

Figure 1 (Salzarulo). Trends of night-time memory and the S (sleep) process. Abscissa: rank of NREM-REM cycles Ordinate: Relative changes of memory process (scores) (Cipolli et al. 1998) and S process (SWA: total power density in 0.5–4 HZ frequency bin) (Dijk et al. 1991) across cycles. First cycle value is conventionally assigned 10.

Dream research: Integration of physiological and psychological models

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Abstract: All five target articles are of high quality and very stimulating for the field. Several factors such as dream report length and NREM/REM differences, may be affected by the waking process (transition from sleep to wakefulness) and the recall process. It is helpful to distinguish between a model for REM sleep regulation and a physiological model for dreaming. A third model accounting for cognitive activity (thought-like dreaming) can also be of value. The postulated adaptive function of dreaming in avoidance learning does not seem very plausible because the two major basic assumptions (specificity of dream content and benefit of negative dreams) are not clearly supported by modern dream research: The critique of studies investigating memory consolidation in REM sleep is justified. Future studies integrating the knowledge of memory processes and sleep research will shed more light on the role of sleep, especially REM sleep in memory consolidation.
[HOBSON ET AL.; NIELSEN; REVONSUO; SOLMS; VERTES & EASTMAN]

The five target articles discuss important aspects of dreaming and REM sleep, that is, physiological mechanisms of dreaming, the function of dreaming, and the role of REM sleep in memory consolidation. In view of the vast range of topics addressed in the articles, these following comments are restricted to a few selected points. First, some basic issues of contemporary dream research will be discussed.

Basics of dream research. First of all, it seems important to look at the definition of dreaming. Although it is not always explicitly mentioned, it is obvious that only the dream report, that is, the part of the mental activity during sleep that can be reported upon awakening, is available to the researcher. This process involves the transition of at least two thresholds: (1) recall of previous experiences and (2) sleep/wake transition. Both factors may be important considering the issue of dream length and the REM/NREM differences in dream content. Rosenlicht et al. (1994) found a relationship (r = .23) between dream report length and the report length of a re-narration of a previously shown video film. This finding indicates that reporting style may well affect dream length.

Because dreams are not perceived as films, it will be interesting to use standardized situations which are recounted afterwards and to test whether the length of these reports correlates substantially with dream report length. From experience with recording dreams and listening to dream reports, it seems obvious that a variety of factors in addition to report style can affect dream report length. Dreams (at least REM dreams) are experiences similar to those of waking life, that is, a dream scene can be described in a few words if one focuses on the major dream action. On the other hand, the report of the same scene will be much longer if all the small visual details are recounted (if they were remembered).

The effect of motivation and in-depth inquiries about specific dream details on dream length was seldom investigated in a systematic way, but other studies, for example, investigating the experience of tactile sensations (Strauch & Meier 1996) or emotions (Schredl & Doll 1998) have shown that the dream report comprises only parts of dreamed experiences. A study of dreaming among the elderly (Schredl et al. 1996) revealed a marked relationship between dream report length and verbal short-term memory, whereas visual memory and overall cognitive performance were not correlated. This finding points to the fact that specific abilities are necessary in the recall process after awakening to record or report the dream experience in a temporal sequence. These investigations make clear that the assumption of **HOBSON ET AL.** that dream report length is largely determined by the quantity of dream experiences should be regarded with caution since at least the above mentioned factors must also be taken into account.

Second, the sleep/wake transition may also affect the generation of the dream report. A comprehensive model was proposed by Koukkou and Lehmann (1983). The basic assumptions of their model are the following:

Cognitive activity can be assigned to different functional states which are associated with differences in processing modes, memory stores, and EEG pattern. They assume that information of lower levels of activation (NREM sleep, REM sleep) can only be sparsely recalled by functional states of high activation (wakefulness) and that the closer the two functional states are, the better is the transference of information. On the other hand, information

from higher memory stores is available for lower states. Although the findings of EEG studies are inconsistent (cf. Schredl & Montasser 1996; 1997), the model offers a plausible interpretation of the differences in recall rates of NREM and REM awakenings, since NREM sleep is associated with a lower functional state than REM sleep. This may be comparable to the activation component of the AIM model (**HOBSON ET AL.**).

The functional state-shift model stresses the fact that both functional states, that is, prior and after awakening, are important in the process of recalling the dream experience. Spitzer et al. (1993), for example, have proven so-called carry-over effects, that is, after REM awakenings semantic priming in a word association task was significantly more pronounced than after NREM awakenings and in the waking state. The authors interpreted their finding as inhibition of the semantic associative network.

Similarly, **HOBSON ET AL.** pointed out that the cholinergic-aminergic modulation of the brain did not change abruptly. In view of these findings regarding the carryover effect, the effect of the awaking process and the differences between the functional states prior to and after awakening on dream report length and other dream characteristics should be investigated in detail.

In the future it may be possible to utilize brain imaging techniques. It may be that the NREM/REM differences in dream content (**NIELSEN**) are partly owing to the different states of the brain after the awakening. Another aspect of dream recall is the large inter-individual variability of dream recall frequency and the fact that dream recall frequency can easily be enhanced by training (cf. Schredl 1999). It seems implausible that a purely physiological model (cholinergic-aminergic modulation of the brain) can explain why some persons recall a dream almost every morning whereas others have not remembered a dream for years. Similar patterns were detected for low dream recallers in the sleep laboratory (e. g., Meier Faber 1988). Recall after REM awakenings differed considerably between subjects in this study. In this context, it would be very interesting to conduct studies testing whether dream recall frequency after NREM awakenings could be enhanced by training.

Another important issue is the definition of dreaming, which was attempted by **NIELSEN** (Fig. 1). From this it seems clear that only a part of the brain activity can be recalled. Dreaming is defined in contrast to cognitive activity as a mixture of sensory hallucinations, story-like or dramatic progression, and bizarreness (similar to the definition of **HOBSON ET AL.**). This definition, however, is based on dream reports with all the above-mentioned limitations.

The formal characteristics of dream reports also vary considerably from person to person, for example, the "thin versus thick boundaries" personality dimension is related to dream bizarreness (Schredl et al. 1999). Since a strict differentiation between the types of mental activity during sleep (apex dreaming, dreaming, cognitive activity, cognitive processes) based on dream reports is difficult, it will be necessary to carry out studies that elaborate on a clear definition and link these types to physiological processes. The investigation of inter- and intra-individual differences will be very promising because, all types of mental sleep experiences (except cognitive processes) can be obtained after REM awakenings as well as after NREM awakenings.

The authors themselves have pointed it out, but it nevertheless seems important to stress that the AIM model provides an explanation for the formal characteristics of the dream, for example, bizarreness. The model does not, however, provide anything about dream content. For this reason, researchers focus on the so-called continuity hypothesis (e.g., Domhoff 1996; Hartmann 1998), which states that emotional concerns and preoccupations of the waking life are reflected in dreams. However, the findings regarding the dream-lag effect, the temporal references of dream elements, and the effects of experimental manipulation on dream content are not consistent in a way to formulate a more specific model explaining dream content.

The AIM model describes the states of consciousness using three dimensions. **HOBSON ET AL.** pointed out that this is an oversimplification, and this is evident when the authors apply the model to other states of consciousness, e.g., lucid dreaming, and REM behavior disorder, since they have to introduce other dimensions. It seems not clear whether the model is useful beyond the descriptive aspect. On the other hand, the model contains a modulation component, as an explanatory aspect, because the preponderance of a specific neuro-transmitter affects the mode of information processing.

In this context, studies as carried out by Hartmann et al. (1980) with L-dopa investigating the effect of drugs such as muscarinic cholinergic agonists and acetylcholinesterase inhibitors on formal dream characteristics, such as bizarreness, will allow a specific test of the M component of the AIM model. Preliminary data of a pilot study (Schredl et al. 2000) carried out in our laboratory indicate that donepezil, an acetylcholinesterase inhibitor, intensifies dreaming but has only a small effect on REM sleep percent although REM latency is shortened and REM density is heightened under medication. It will be interesting to test whether such drugs affect dream content in the predicted way. In addition, the investigation of interindividual differences in dream content in relation to parameters of neurotransmission will also be of value.

The findings of **SOLMS** suggest that it may be helpful to develop two models, one to explain the physiology of REM sleep regulation and the second for the physiology of dreaming which explains the formal characteristics of dreams. In order to do this, it will be necessary to have a clear and exact definition of dreaming to correlate mental activity with brain physiology beyond sleep stages. The next step could be a search for a link between the two models (cf. **NIELSEN**). This differentiation may add to the explanation of other states of consciousness (sensory deprivation, daydreaming, narcosis, near-death experiences).

Another problem which was discussed at the Third International Congress of the World Federation of Sleep Societies (held 1999) is the definition of sleep. What does it mean for a single cell or groups of cells whether the brain as a whole is sleeping? This argumentation is also evident in the article of **NIELSEN** who has shown that distinct REM features which are present during NREM sleep are related to dream recall. Therefore, a differentiation between REM sleep physiology and physiology of dreaming seems promising. It may be that a third model is necessary to explain the occurrence of cognitive activity (thought-like dreaming) if one does not agree with Hartmann's hypothesis (1998) in which a continuum ranging from bizarre dreams to focused waking thought is conceptualized.

Functions of dreaming. Quite a few hypotheses about the function of dreams were formulated over the years. Some of them are reviewed by **REVONSUO**. First, it is important to draw attention to the fact that the function of dreaming cannot be studied empirically in a direct way, because dream content can not be elicited without the involvement of the waking mind, for example, dreams have to be recalled in order to test the hypothesis whether they are helpful in problem solving. It may also be possible that thinking about the dream causes subsequent changes in waking life and not the dream per se.

A few arguments which should lead to a critical evaluation of **REVONSUO**'s theory will be briefly enumerated. First, it seems implausible to assume that dreams are specialized in replaying threatening experiences. The widely acknowledged continuity hypothesis states that emotional concerns and preoccupations are reflected in dreams (e.g., Domhoff 1996; Hartmann 1998). A component which REVONSUO has not considered is the fact that not only real experiences of the waking-life can be found in dreams but also thoughts, emotions, things seen on TV, movies, and so forth. If one examines this input, for example, TV news, it may be possible that one finds a preponderance of negative themes which affect dream content. In order to compare waking life and dreaming, it will be necessary to elicit the inner world of the person as completely as possible.

The second point is related to the assumed preponderance of

negative dream emotions. Schredl and Doll (1998) have pointed out that several methodological issues (recalling dreams which occurred a long time ago, only focusing on explicitly mentioned emotions within the dream report, the using of raters instead of self-ratings) limit the generalizability of many studies in this field. These arguments are also valid for the study of Mehinaku dreams which was cited by **REVONSUO,** that is, very vivid and negatively toned dreams which could be remembered easily were reported to the researcher. Dreams recorded immediately upon awakening show a balanced ratio of negative and positive emotions (Schredl & Doll 1998).

Another example to support the continuity hypothesis and not the specificity for threatening contents is the occurrence of the male stranger as major dream aggressor; almost all murderers and soldiers are male, that is, the pattern of male aggressiveness is reflected in dreams (e.g., Schredl & Pallmer 1998). There is evidence from studies in the field of learning that some learning occurs abruptly, for example, classical conditioning in avoidance tasks (avoiding an aversive stimulus), acquiring an aversion that is very resistant to extinction to specific food (e.g., Margraf 1996). This kind of learning makes a repetition of negative experiences unnecessary. In addition, the adaptive function of PTSD or anxiety disorders seems not to be very reasonable. **REVONSUO** pointed out that these negative effects may only be present in war-related PTSD, but investigations of rape victims and persons who experienced natural disasters did not support the hypothesis that war-PTSD is specific (cf. Barrett 1996). The marked relationship between psychopathology and negative dream emotions (e.g., Schredl & Engelhardt 2000) did not support the idea that threatening dreams serve an adaptive function; on the contrary, depressive patients or patients with anxiety disorders suffer from their dreams. Similarly, the correlation between low life satisfaction and negative dream emotions in the elderly (Schredl et al. 1996) can be interpreted in a way that negative dreams are associated with poor psychological adjustment. Hartmann (1991) has investigated persons with thin boundaries who often suffer from frequent nightmares. He described these persons as not sensitive to possible danger in foreign cities; an observation which does not fit within REVONSUO' s framework.

The question of utmost importance is whether threat simulation during dreaming increases the probability of coping successfully with comparable real threats. As mentioned above, this hypothesis could not be tested empirically in a direct way since the remembering of nightmares or post-traumatic re-enactments may stimulate waking thoughts which affect subsequent behavior and not the nightmares themselves. It will be very interesting to explore whether or not animals experience nightmares (strongly negative dreams which end by an awakening). It may be possible to traumatize animals and measure the physiological anxiety responses during REM sleep. Animals with nightmares should learn an avoidance task more easily. To my knowledge, such studies have yet to be carried out. If – as shown for example by Hublin et al. (1999) – a genetic factor plays an important role in the etiology of nightmare, one might argue that the group of frequent nightmare sufferers should be increased by natural selection. Since this group is small (about 5 percent, e.g., Bixler et al. 1979), this seems not to be very plausible.

To summarize, the two basic questions (selectivity for threatening experiences, benefit of replaying these experiences in dreams) are in my view not supported by the presented evidence. On the other hand, one can follow the assumption of Kuiken and Sikora (1993) that dreaming serves multiple purposes.

Function of REM sleep. In contrast to **REVONSUO** who outlined a possible function of dreaming, **VERTES & EASTMAN** discussed the function of REM sleep in memory consolidation. A major problem of previous research, at least in humans, is in my view the lack of integration between memory research and sleep research (Schredl et al. 1998). Modern research has shown that different types of memory processing (explicit vs. implicit, declarative vs. procedural memory) are associated with different brain areas (e. g., Markowitsch 1996). Recent studies in humans (Karni et al. 1994; Plihal & Born 1997) have shown that procedural memory may be consolidated during REM sleep but declarative memory performance is enhanced after undisturbed slow wave sleep. It may be expected that findings will be more consistent if the knowledge from the two disciplines are combined.

On the other hand, it is important to consider the fact that memory consolidation also takes place during the waking state. Mandai et al. (1989), for example, have formulated a two-step model with memory storage and consolidation during waking and additional memory consolidation in REM sleep. The findings cited by **VERTES & EASTMAN** have shown that REM deprivation (i.e., disturbed sleep, stress, etc.) did not affect simple memory tasks and, thus, the hypothesis of memory consolidation during the waking state is supported. Similarly, the findings regarding drugs which suppress REM sleep, can be interpreted in this way. However, these studies did not retest material or skills which have been trained the previous day (or days) for the first time. A pilot study (Schredl et al. 2000) has shown that the amount of REM sleep in nights with donepezil (an acetylcholinesterase inhibitor) is strongly related to the increase of performance in a memory task from the evening training session to the morning test session. Therefore, more sophisticated studies including training of new knowledge or skills and retest after at least one sleep period have to be carried out to evaluate the effect of REM sleep on memory consolidation.

Buzsaki (1998) has suggested a model which indicates that both slow wave sleep as well as REM sleep play an important role in memory consolidation during sleep. A recent study showed that the diurnal cortisol profile (minimum in the first part of the night) affect the consolidation of declarative memory but not the occurrence of specific sleep stages (Plihal et al. 1999). From a methodological viewpoint, it must be said that modern literature reviews use the technique of meta-analysis. With this technique, effects of the deprivation technique (pedestals, multiple platforms, pendulum technique) can be tested statistically (in this case, a sufficient number of studies allowing the computation of effect sizes were published). In addition, it will be necessary to carry out EEG studies with animals to estimate the bias introduced by stressful REM sleep deprivation techniques.

To summarize, the database regarding the role of REM sleep and sleep in memory consolidation is indeed not very solid because of the limitations pointed out by **VERTES & EASTMAN** and the lack of integration between memory research and sleep research. But recent studies are promising and support that sleep plays a role (in addition to processes during waking) in memory consolidation.

Future directions. In the future, imaging techniques (e.g., MRI) will offer the option to investigate the relationship between brain processes in different sleep stages and dream content. In order to do this properly it will be necessary to improve the present imaging techniques (e.g., time resolution) and to develop precise instruments for measuring dream content. It will be helpful to use two or three models explaining (I) REM sleep regulation, (2) dreaming, and (3) cognitive activity (thought-like dreaming). In addition, it will be important to investigate the awaking process in a more detailed way in order to evaluate the relationship between dream report and original dream experience. Last, a theory modeling the relationship between waking life and dream content should be formulated which goes beyond the simple statement of continuity between these two states of consciousness.

Threat simulation, dreams, and domain-specificity

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Abstract: According to Revonsuo, dreams are the output of a evolved "threat simulation mechanism." The author marshals a diverse and comprehensive array of empirical and theoretical support for this hypothesis. We propose that the hypothesized threat simulation mechanism might be more domain-specific in design than the author implies. To illustrate, we discuss the possible sex-differentiated design of the hypothesized threat simulation mechanism.
[REVONSUO]

REVONSUO proposes that dreaming is the output of an evolved "threat simulation mechanism." According to the author's argument, the hypothesized mechanism was selected for because ancestral humans whose psychology included this mechanism experienced dreams in which threats were simulated and thereby more efficiently and effectively dealt with in waking life. Those early humans who had dreams in which threats to survival and reproductive success were simulated were better able to solve similar classes of threats in waking life and, therefore, out-reproduced conspecifics whose psychology did not include the threat simulation mechanism. We appreciate this argument, and believe that REVONSUO has done an excellent job of synthesizing an impressive array of empirical and theoretical support for the argument. We propose, however, that the hypothesized threat simulation mechanism is more domain-specific than the author presents.

The hypothesized threat simulation mechanism may be too domain-general. According to **REVONSUO,** dreams serve as a means for the dreamer to rehearse events that would have threatened survival or reproductive success in ancestral environments. The author proposes two primary threatening events or episodes – one in which the dreamer is being chased or attacked by an unfamiliar adult male, and one in which the dreamer is being chased or attacked by a wild and dangerous animal. REVONSUO provides sound theoretical arguments why unfamiliar adult males and wild animals were key threats to ancestral humans and, therefore, why they are prominent in the self-reported dreams of modern humans. We believe this is an excellent starting point for an evolutionary psychological analysis of dreams. We propose, however, that the threat simulation mechanism that generates dreams may be far more domain-specific. Instead of generating general threat dreams that include the two key events proposed by REVONSUO, we suggest that perhaps the dreams of modern humans might reveal greater domain specificity. Might the threat simulation mechanism generate dreams that are more specific to the adaptive problems faced recurrently by humans over human evolutionary history? For example, might the threat simulation mechanism generate different classes of threatening dreams when it is operating in a child living with one stepparent and one genetic parent than when it is operating in the psychology of a child who lives with two genetic parents (see Daly & Wilson 1996)? As another example, might the threat simulation mechanism generate different classes of threat scenarios when it is operating in male psychology than in female psychology? We discuss the latter example in the remainder of this commentary.

Is the threat simulation mechanism sex-differentiated? An overwhelming collection of theoretical and empirical work suggests that males and females faced different adaptive problems recurrently over human evolutionary history (see Buss 1994, for a review). For this class of adaptive problems, modern evolutionary psychologists expect the evolution of sex-differentiated psychological mechanisms. One such adaptive problem recurrently faced by ancestral humans is a long-term partner's infidelity. Because fertilization occurs internally to females, females always can be certain that they are the genetic parent of any offspring they produce. Males, in contrast, never can be certain that they are the genetic parent of the offspring produced by their partner. Males, but not females, risk cuckoldry – unwittingly investing in offspring to whom they are genetically unrelated. Although both sexes are upset by a partner's infidelity, males are more upset by a partner's sexual infidelity than by a partner's emotional infidelity – infidelity in which resources such as social support and material wealth are channeled to another person. Females, in contrast, are more upset by a partner's emotional infidelity, which places them at risk of losing to another woman the investment their partner would otherwise channel to them and their children (see Buss 2000, for a review of this work).

If the threat simulation mechanism generates dreams that simulate ancestral threats to survival and reproductive success, we propose that sex-differentiated ancestral threats will have selected for a threat simulation mechanism that generates sex-differentiated dreams. The mechanism might be sensitive to and triggered by different classes of infidelity cues when situated in male psychology than when situated in female psychology. Relative to a partner's emotional infidelity, sexual infidelity presented a graver adaptive problem for ancestral males than for ancestral females. We therefore hypothesize that the threat simulation mechanism will generate in males relative to females more dreams about a partner's sexual infidelity. In addition, we hypothesize that dreams about a partner's sexual infidelity will be more upsetting for males than for females. A partner's emotional infidelity presented a graver adaptive problem for ancestral females than for ancestral males. We hypothesize that the threat simulation mechanism will generate in females relative to males more dreams about a partner's emotional infidelity. In addition, we hypothesize that dreams about a partner's emotional infidelity will be more upsetting for females than for males. According to this argument, the threat simulation mechanism is the same in males and in females, but the design features of the mechanism – the class of information that triggers the mechanism, and the output generated by the mechanism, may be sex-differentiated.

In summary, **REVONSUO** has provided us with a wonderful example of the heuristic value of an evolutionary psychological perspective. The target article significantly advances our understanding of dreams by proposing that dreams are generated by an evolved threat simulation mechanism. Although we find the core of REVONSUO's argument compelling and convincing, we suggest that the hypothesized mechanism may be more domain-specific than the author implies. We propose that the dreams of males will more frequently include a partner's sexual infidelity, whereas the dreams of females will more frequently include a partner's emotional infidelity. In addition, we propose that dreams of a partner's sexual infidelity will be more distressing for males than for females, whereas dreams of a partner's emotional infidelity will be more distressing for females than for males. We hope that future work might investigate the domain-specificity of the hypothesized threat simulation mechanism. We suggest as a starting point the investigation of the possible sex-differentiated design features of this mechanism that might be revealed with an analysis of dreams about a partner's infidelity.

Continued vitality of the Freudian theory of dreaming

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Abstract: A minority position is presented in which evidence will be cited from the Hobson, Solms, Revonsuo, and Nielsen target articles and from other sources, supporting major tenets of Freud's theory of dreaming. Support is described for Freud's view of dreams as meaningful, linked to basic motivations, differing qualitatively in mentation, and wish-fulfilling.
[HOBSON ET AL.; NIELSEN; REVONSUO; SOLMS]

In this commentary we will be taking a decidedly minority position. We will point out that there is considerable evidence, some of it provided by the articles themselves, that supports Freud's theory of dreaming. With the exception of **SOLMS**'s paper, the **HOBSON ET AL., REVONSUO,** and **NIELSEN** papers either discard Freud's theory or ignore it. Our commentary will be divided in six sections, each devoted to a proposition from Freud's theory and evidence in its support.

Proposition 1. Despite their bizarre and illogical appearance, dreams are organized on the basis of certain principles and are the outcome of specifiable mental processes. Dreams are not random or epiphenomenal.

The articles by both **HOBSON ET AL.** and **SOLMS** spell out the consistency and complementarity of findings obtained from brain imaging studies of REM sleep and lesion studies of dreaming. From the PET studies HOBSON ET AL. conclude that REM sleep may involve "a specific activation of subcortical and cortical arousal and limbic structures for the adaptive processing of emotional and motivational learning." Similarly, SOLMS's neuropsychological findings indicate that dreams depend upon the concerted activity of a very particular set of mental functions located in various areas of the forebrain. These findings are quite consistent with a view of dreams as specifically organized to carry out particular functions.

Nevertheless, **HOBSON ET AL.** continue to maintain that important features of dreaming are determined by random, chaotic noise from the brainstem impinging on the forebrain. Further, the data HOBSON ET AL. cite concerning the rich bidirectional interactions, reciprocal connections, and feedback loops between brainstem and forebrain structures do not support a view of the brain as functioning in a nonintegrated fashion, with brainstem activity impinging disruptively and randomly on the forebrain. HOBSON characterizes dreams in terms of cognitive deficiencies, and draws an analogy between dreams and epileptic seizures; the possibility is not considered that dreams are organized in a different, rather than deficient, fashion that has its own particular logic.

REVONSUO also views dreams as organized phenomena providing a selective simulation of external threats in a perceptually realistic way so as to evoke rehearsal of skills in the efficient recognition and response to these threats. However, this differs considerably from our view of the specific function of dreams, as discussed in Propositions 3 and 5.

Proposition 2. The occurrence of dreaming is determined by two causes (1) the state of sleep itself, and (2) basic motivations such as sex and aggression whose persistence creates conflict in both the waking and sleep states.

1. It has been clear since the discovery of REM sleep and its association with dreaming that the first part of this proposition requires further specification. Although there is a range of views on the differences between REM and NREM mentation, there is little doubt that REM sleep is particularly facilitative of dreaming. Indeed, Freud as early as 1895 hypothesized that a motor paralysis was a necessary condition for dreaming to occur; such a paralysis is found only in REM sleep. We find persuasive both **HOBSON ET AL.** and **NIELSEN**'s marshalling of evidence that there are important qualitative differences between REM and NREM mentation, and here would differ with **SOLMS,** who views REM as only one of many arousal triggers of dreaming. The very high correlation between REM sleep and dreaming suggests to us a more integral, even if not absolute, connection, as does the very different pattern of brain activation in REM versus NREM sleep reported in the imaging studies and consistent with SOLMS's lesion findings about the neuroanatomy of reported dreams. We note, with HOBSON ET AL., that SOLMS's data have not established that dreaming is preserved with brainstem lesions that eliminate REM sleep. We would characterize the differences between REM and NREM mentation in terms of a greater predominance of, in Freudian terms, primary-process versus secondary-process thinking (see Proposition 4) during REM sleep.

2. Freud's view of instinctual drives and their development implies an ongoing source of conflicted sexual and aggressive wishes that may serve as the motive force for dreaming. We find evidence compatible with this understanding in several of the findings discussed in these papers. **SOLMS** has reported that one of the two major forebrain areas associated with loss of dreaming in lesion studies is the parietaltemporo-occipital junction, which is associated with "appetitive interactions with the world," "the 'SEEKING' or 'wanting' command system of the brain." Similarly, the imaging findings consistently indicate the important participation of areas of the brain involved with emotion, motivation, and reward, "quite in accordance with older, more general views that REM sleep, and specifically dream content, is associated with internally generated, or instinctual behaviors that subserve adaptive mechanisms" (Nofzinger et al. 1997, p. 199). The concept of wishes that are conflicted also finds support. A hallmark of conflict in Freudian theory is that wishes associated with danger arouse anxiety, and the amygdala, which is especially associated with anxiety, is found to be activated in REM sleep.

There is another set of findings that is strikingly consistent with the association of dreams and persistent basic motivations. We refer to studies on the effects of REM sleep deprivation on motivation in the waking state. Dement and colleagues (1967; Dement 1969) carried out a series of prolonged REM deprivation procedures in cats and reported that REM-deprived cats became hypersexual and hyperphagic. The sexual behavior reported involved unusual, persistent efforts on the part of a number of male cats to mount other male cats. These findings were not presented in fully quantified form, and must be regarded as exploratory and suggestive. However, additional credence is lent to them by a number of more formal experimental reports in the literature with similar findings. Some examples: Ferguson and Dement (1969) reported that in REM-deprived rats primed with amphetamine, stereotyped aggressive and sexual behaviors were seen in the absence of the usual releasing stimuli, the aggressive behaviors in virtually all experimental rats and the sexual behaviors in a subset. Peder et al. (1986) found that REM deprivation alone resulted in increased aggressive behavior in rats, and increased genital exploration among female rats. Morden et al. (1967) reported that REM deprivation led to increased shock-induced fighting behavior in rats, which persisted beyond the recovery period. Conversely, electrical stimulation of hypothalamic defence (rage) reactions in cats led to reductions in subsequent REM sleep and, in REM deprived cats, in subsequent REM rebound (Putkonen & Putkonen 1971), a finding suggestive of reciprocal elements in the relation between drive and REM sleep. This sampling of results provides intriguing evidence from animal research of a relationship between REM sleep/dreaming and the expression of sexual and aggressive drives/wishes (see also Vogel 1979).

The dream itself is an attempt to deal with these conflicts with the means available during sleep, just as these conflicts are dealt with in the waking state with the means available in that state

The major disagreement with this proposition derives from **REVONSUO**'s position that dreams function to deal with external threats as experienced by our ancestors. However, a closer examination of REVONSUO's data leads to a different conclusion more in keeping with the Freudian conflict hypothesis.

Proposition 3. First, let us consider the physical threats our ancestors had to guard against which figure so prominently in **REVONSUO**'s theory. These threats were encountered because of the need to venture out in order to hunt and gather food, or seek a mate. Threats would have been considerably diminished by simply staying put in a safe cave. Thus conflict at least between the need for food and the need for a mate and running the risk of injury or death in their pursuit must have existed among our ancestors. If during sleep a desire for food or sex was aroused this would bring with it the threat posed by these desires.

Observations of primate groups reveal that the most frequently experienced threats come from other conspecifics rather than from predators. The constant jockeying for alpha status among males, the need to select an appropriate mate among females, are

not life threatening but rank high as sources of frequent conflict. It is the internal pressure to be dominant among males and to mate by choice among females that results in conflict and threat. Threat cannot be defined solely externally, but is defined by the internal state of the individual and determined by specific motivations. This is entirely consistent with the Freudian theory of dreams.

Proposition 4. Freud's theory is a two generator, or two mentation type theory which Freud called the primary and secondary process. We will cite experimental evidence that REM mentation is organized along primary process lines and NREM along secondary process lines which fits with a two mentation model. The study by Shevrin and Fisher (1967) cited by **HOBSON ET AL.** is to our knowledge the only sleep-dream study in which the effects of a waking subliminal stimulus on REM and NREM mentation has been investigated. The relevance of the study to the current controversy over one versus two mentation theories lies in the fact that it introduces operationally through subliminal stimulation the place of unconscious influences on sleep mentation, an issue of great importance to a Freudian theory of dreaming, and we believe an overlooked factor in sleep-dream research. The subliminal stimulus was designed in accord with Freud's hypothesis that the unconscious mentation underlying dreams was rebus-like in nature, by which he meant that the elements of a dream were juxtaposed and combined not in terms of their customary logical and conventional relationships but in terms of what he referred to as "superficial" associations, borrowing a term from Wundt. In contemporary terms, these would be seen as distant associates. However, in no current linguistic or cognitive theory would the prediction be made that the distant associates would be combined to form a new entity with its own associates. It was to this process that Freud gave the name condensation, one of the primary process mechanisms, in which two quite different unrelated elements are combined to form a new entity.

The stimulus was a picture of a pen and a knee forming the rebus for the word penny. The effects of the stimulus could then be measured along two dimensions: secondary process (logical, conventional) associates of pen (e.g., ink, paper) and knee (e.g., leg, bent) and primary process associates of penny (e.g., cent, money). Scoring was based on association norms collected prior to the experiment. Following awakenings from REM and NREM sleep the subjects' accounts of the immediately prior sleep events were obtained as well as two minutes of free associations. These free associations revealed that the rebus effect (penny associates) was significantly greater following REM awakenings, and the secondary process associates (pen and knee associates) were significantly greater following NREM awakenings. These results support Freud's view that it is necessary not only to know the sleep content but also the unconscious process giving rise to the sleep content which may differ depending on sleep state along primary process (REM) and secondary process (NREM) lines.

In view of the fact that in the Shevrin and Fisher study the results were found in a waking response following sleep awakenings, it is important to note that **NIELSEN** (p. 15) cites eight studies in which cognitive and physiological components of the sleep state carry over and influence waking performance, and one study which failed to find such differences. He concludes that most of the results "support the interpretation that qualitatively different cognitive processes are active following and, by inference, just preceding awakenings from REM and NREM sleep."

In a second study conducted by Castaldo and Shevrin (1970), it is important to note that the pictorial rebus stimulus was not presented subliminally prior to sleep, but was presented as words through earphones during REM or NREM sleep. Measures were based on the waking accounts of sleep mentation rather than on free associations. Despite these considerable differences from the Shevrin and Fisher study, Castaldo and Shevrin found that pen and knee associations were significantly more frequently found in accounts of NREM mentation and significantly less frequently found in accounts of REM mentation.

The rebus effect was not replicated and may depend on such factors as pictorial and subliminal presentation. In his reference to the Shevrin and Fisher study, **HOBSON ET AL.** do not cite the association findings which we believe are of special relevance to understanding the role of unconscious processes in sleep mentation. The Shevrin and Fisher results support **HOBSON ET AL.**'s position on the qualitatively different nature of sleep mentation associated with REM sleep, and fail to support **SOLMS**'s conclusion that there is no qualitatively different mentation related to REM activation.

Proposition 5. The dream will sometimes succeed in providing an hallucinated gratification of sexual and aggressive motivations and thus will be wish-fulfilling; at other times the dream will fail, resulting in anxiety dreams and awakenings. When optimally functioning the dream is thus a protector of sleep

REVONSUO directly challenges the wish-fulfilling aspect of dreaming proposed by Freud, replacing it with an evolutionary explanation based on rehearsal of threat. It is thus of interest that when **REVONSUO** provides some detailed description of dreams from the Mehinaku Indians that the role of threat is cast in a different light. Of the 14 dreams described, 7 can be construed as having manifest wish-fulfilling implications (e.g., desired and approached girl, struck by jealous wife). Our kinship with the Mehinaku Indians resides in the sexual and aggressive desires we share. Dreams from this standpoint are not outmoded rehearsals of prehistoric threats, but serve important current psychological purposes rooted in our evolutionary past.

Proposition 6. Exigent motives are highly activating or, in Freud's terms, cathected with considerable psychic energy and thus can provide the impetus for dreaming. The evidence previously cited that REM deprivation can cause animals to become hypersexual and hyperaggressive suggests that these motives are highly activating. We have also noted that **SOLMS**'s lesion findings indicate that appetitive circuits are essential for the dreaming process, and that imaging studies point to the crucial involvement of motivational and emotional centers in the limbic and paralimbic system during REM sleep. It is intriguing to consider in addition the possible role of the deactivation of prefrontal cortical areas involved in executive functions, such as volitional control and self-monitoring. We would see this as resulting in a shift in the balance between wishes on the one hand and controlling/inhibitory functions on the other in favor of the former, resulting in wishes being relatively stronger and more exigent during REM sleep. This view also seems consistent with the aspect of **HOBSON ET AL.**'s AIM model that stresses a different balance in neuromodulation (his M factor) during REM sleep, with the preference for cholinergic over aminergic modulation favoring structures which mediate emotion over those which mediate directed thought.

Despite the insurance provided by motor paralysis that entertaining powerful motives would not result in acting on them, Freud reasoned that even becoming directly aware of them would disturb sleep and awaken the dreamer. For this reason there was a need for disguise which employed the primary process mechanisms described earlier and supported by the Shevrin and Fisher study. But the Shevrin and Fisher study did not establish the disguising function of the primary process. Further work is needed on this hypothesis, although nothing thus far reported would necessarily be inconsistent or contradict this hypothesis.

Phylogenetic data bearing on the REM sleep learning connection

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Abstract: The phylogenetic data are inconsistent with the hypothesis that REM sleep duration is correlated with learning or learning ability. Humans do not have uniquely high amounts of REM sleep. The platypus, marsupials, and other mammals not generally thought to have extraordinary learning abilities have the largest amounts of REM sleep. The whales and dolphins (cetaceans) have the lowest amounts of REM sleep and may go without REM sleep for extended periods of time, despite their prodigious learning abilities.
[VERTES & EASTMAN]

The idea that REM sleep with its elaborate associated dream mentation has a role in memory consolidation is a very attractive one. It is a pity that the evidence supporting this beautiful idea is so weak. **VERTES & EASTMAN** marshal impressive evidence inconsistent with a major role of REM sleep in learning. To this I add the mammalian phylogenetic data.

Much of the evidence that has been advanced as supporting a positive relationship between REM sleep and learning derives from reported increases in REM sleep after learning and blockade of learning after reduction in REM sleep with deprivation procedures. As **VERTES & EASTMAN** point out, most if not all of this evidence does not withstand careful scrutiny. Nevertheless if we pursue the logic of this approach, one would predict that animals with greater learning capacity would have greater amounts of REM sleep.

Indeed, learning and memory theorists often imply that humans have large amounts of REM sleep. In fact, humans follow the general trends within the animal kingdom. Being large animals they share the inverse relationship between size and total sleep amount (Zepelin 1994). Humans sleep less than most smaller mammals. If REM sleep is calculated as a percentage of total sleep time, humans appear to have a lot of REM sleep, though not a uniquely large amount. However, the animals with the largest amounts of REM sleep are not the primates. The animal with the most REM sleep is the duckbilled platypus, which has, depending on how the calculation is done, approximately 7–8 hours of REM sleep a day (Siegel et al. 1997; 1999). REM sleep in the platypus has some unusual features. Perhaps of most significance is the lack of the low voltage EEG that characterizes REM sleep in other adult mammals. If we put aside the platypus data, the next contenders for the REM sleep championship are the black-footed ferret and the armadillo (Marks & Shaffery 1996; Prudom & Klemm 1973; Van Twyver & Allison 1974). What intellectual attribute do these three animals have in common? Is it intelligence or stupidity'? Without disparaging the beauty and role of these animals in the ecosystem, they are largely instinct driven. Clearly they can learn as can all mammals, but they do not appear to be unique in their mental skills. In general, the marsupials and monotremes have more REM sleep than the placentals (Zepelin 1994).

How about the other end of the spectrum? The mammals with the least REM sleep are the cetaceans (whales and dolphins). Early reports in captive animals did not detect any clear episodes of REM sleep (Mukhametov et al. 1977; Mukhametov 1987; Shurley et al. 1969; Oleksenko et al. 1992; Flanigan 1974). Clearly if dolphins have any REM sleep at all, they can go without it for days or weeks. A more recent study in a captive gray whale demonstrated occasional twitches during sleep (Lyamin et al. 2000). The most generous estimates of the REM sleep total in these animals would be less than 15 minutes a day. How does the learning ability of dolphins and whales, animals with the largest brains ever to exist on earth, compare with that of the platypus, ferret, and armadillo? It would be difficult to defend the notion that the latter are smarter than the cetaceans. Across mammals, REM sleep time is negatively correlated with brain weight (Zepelin 1994).

Work by Jouvet-Mounier (Jouvet-Mounier et al. 1970) and a survey of the literature by Zepelin (1994) led to the conclusion that REM sleep time was correlated with immaturity at birth. Our recent findings in the platypus at the high end of the REM sleep scale and cetaceans at the low strongly support this conclusion. The immaturity of the platypus, hatching from an egg and remaining attached to its mother for an extended period after birth is consistent with its high level of REM sleep. The maturity at birth of the cetaceans, which can swim free of the mother and defend themselves immediately after birth is consistent with their low level of REM sleep. Neither the platypus nor the cetacean data is consistent with a relation to intellectual function or memory.

One way out of this dilemma for the learning-REM supporters is to argue that amount of REM sleep is not an informative variable; that REM sleep in the platypus may be less intense or efficient than that in the cetacean. However, this post hoc reasoning is not persuasive. There is no evidence that the very short REM sleep periods in the cetaceans are more intense or that those in the platypus are less intense. In fact the best evidence in terms of phasic event intensity argues just the reverse. The platypus has more than 6,000 phasic events during sleep/24 hours while the Gray whale has fewer than 10. As to the contention that time in REM sleep is not the important variable; this is the very basis of the claim of a relation between REM sleep and learning. The learning theorists cannot convincingly argue this point both ways.

In conclusion, the phylogenetic data provide additional evidence for the case against a key role for REM sleep in memory consolidation or intellectual function.

Evaluating the relationship between REM and memory consolidation: A need for scholarship and hypothesis testing

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Abstract: The function of REM, or any other stage of sleep, can currently only be conjectured. A rational evaluation of the role of REM in memory processing requires systematic testing of hypotheses that are optimally derived from a complete synthesis of existing knowledge. Our view is that the large number of studies supporting a relationship between REM-related brain activity and memory is not easily explained away.
[VERTES & EASTMAN]

The article by **VERTES & EASTMAN** illustrates nothing so well as the difficulty of unraveling the mechanisms underlying memory consolidation and the current dismaying trend of reducing complex topics to sound bites. In the case of REM sleep, it cannot be said that, without REM, absolutely no memory consolidation of any kind will occur. However, the difference between this statement and the authors' thesis, that REM plays no role in memory processing, is vast. VERTES & EASTMAN's conclusion is possibly the result of their lack of direct experimental work examining the relationship of sleep to learning and memory, or, for that matter, with learning and memory at all. (VERTES is rightly recognized as a foremost expert in the neurophysiology of paradoxical sleep and theta rhythm generation in rodents.) We acknowledge that there is no simple guide through the complex and seemingly contradictory literature that deals with REM and memory consolidation. In the limited space allowed, we will attempt to rescue **VERTES & EASTMAN** from some of the conceptual traps into which they have fallen.

A general comment. Science is an empirical discipline; understanding of a subject is always the consequence of a procession of discoveries.**VERTES & EASTMAN** seem to ignore this fact in assigning equal value to all REM/memory studies, whether they were performed 30 years ago or 5 years ago, and the authors exhibit a

distinct preference for early (1970s) work (Fishbein & Gutwein 1977; McGrath & Cohen 1978) to support their hypothesis. It is germane that more recent studies, utilizing more quantitative methodology, better experimental design (including appropriate control groups), and an appreciation of the existence of multiple memory systems, have clearly and consistently shown that REM deprivation impairs the consolidation of some types of memories, but not others (Plihal & Born 1997; Smith 1985; 1995; 1996). Interesting to note, this work has also revealed that other stages of sleep play a role in consolidating some types of memory for which REM is not involved (Smith 1995; Smith & MacNeill 1994).

Why animal studies using REM deprivation do not always affect learning. First, to reiterate a statement made above, it is clear that REM-based memory consolidation processes are involved with only some, and not all, types of learning. More generally, it must be considered that some tasks are so simple (i.e., that the subject is so genetically and/or experientially prepared to solve the problem) that no consolidation mechanism will be activated to a level that can be detected by experimental intervention (Pearlman 1979; Smith 1985). Second, the existence of REM Windows (Smith 1985; 1995; 1996) can explain why mis-timed periods of REM deprivation would not impair consolidation for a particular task. We are nonplused by VERTES & EASTMAN's lack of appreciation that REM windows could be different in different learning situations (e.g., for different tasks or as the consequence of changing task demands). The idea of having a rigid REM window occurring at the same time after training, no matter what the task or number of training trials, is neither intuitively consistent, based upon how most biological systems are known to function, nor supported by systematic experimental work.

Is stress the mechanism through which REM deprivation affects consolidation? One of the most persistent complaints about the REM-memory literature is that stress associated with techniques related to REM deprivation has not been adequately controlled. It is interesting that the stress argument is repeatedly invoked, despite the general absence of data indicating that stress has a detrimental effect on learning or memory. As has been argued in detail elsewhere, several types of studies appear to counter the argument that stress plays a role in REM-deprivation induced memory impairments: (1) pharmacological blockade of REM is not stressful, yet blocks consolidation (Smith 1995; Smith et al. 1991); (2) REM window experiments expose animals to procedures that produce equivalent stress, but produce memory deficits when brief (4 hour) REM deprivation occurs only at a specific time following training (Smith 1985; 1995; 1996); (3) blockade of all sleep except during REM window periods, a procedure known to be stressful, does not impair learning (Smith & Butler 1982); and (4) under certain conditions, REM deprivation has been shown to enhance memory (Kitahama et al. 1976; Smith & Gisquet-Verrier 1996). Taken together, this work makes it clear that stress has no simple role in REM deprivation studies, and parsimony suggests that it plays none at all.

Studies of REM and learning in humans by Vertes & Eastman. The treatment of this literature is painfully incomplete. Studies in humans to date clearly support the idea that REM is at best a modulator of memory and that it plays a role in the consolidation of certain types of memories, but not others. One of the most salient findings of these studies is that REM is not involved in declarative/explicit type memory tasks, at least of the simple type, exemplified by word list remembering, that are often used (Smith 1995). By contrast, tasks that can be classified as procedural/explicit are sensitive to REM sleep loss, which results in impairments ranging from 20–50%. This knowledge undercuts the author's argument that humans with REM-eliminating lesions or REM-depriving pharmacological treatments are normal, because this cannot be established if the subjects are not tested in tasks in which REM is known to be involved. The REM sleep variables in humans are augmented with learning in the tasks examined so far and can be either the amount of REM sleep (min.) (DeKoninck et al. 1990b; 1989; DeKoninck & Prevost 1991) or the number and density of REMs (Smith 1993; 1999; Smith & Lapp 1991). REM windows have been reported both within a single night (Stickgold 1998) and over several nights (Smith 1993). Although there are fewer human than animal studies, recent results support the REM-memory hypothesis and suggest involvement of a phasic REM component (Karni 1994; Smith 1995; 1999; Stickgold 1998).

In conclusion, we wish to state that while the precise role of REM sleep in memory consolidation is far from being completely defined, VERTES & EASTMAN's conclusion that there is no connection is not supported by a balanced evaluation of the existing data. Advancement of scientific knowledge depends upon scholarship and hypothesis testing, both of which are incomplete in VERTES & EASTMAN's target article.

The mechanism of the REM state is more than a sum of its parts

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Abstract: Nielsen has not demonstrated that NREM dreams are regularly accompanied by fragments of the REM state. However, even if this hypothetical correlation could be demonstrated, its physiological basis would be indeterminate. The REM state is a configuration of physiological variables, the basis of which is a control mechanism that recruits and coordinates multiple sub-mechanisms into a stereotyped pattern. The diverse sub-mechanisms underlying each individual component of the REM state do not have an intrinsic relationship with the REM state itself. **[NIELSEN]**

NIELSEN observes that the frequent occurrence of "almost dreams" during NREM sleep does not invalidate the original observation that most *actual* dreams occur during REM sleep. His attempt to explain by recourse to the notion of "almost REM" (NIELSEN's "covert REM"), the subsequent discovery that some actual dreams (indistinguishable by blind raters from REM dreams) do also occur during NREM sleep, is therefore inconsistent. "Almost REM" is just as problematical a concept as "almost dreams." This would be true even if it were possible to demonstrate that REM-like physiological events routinely accompany NREM dreams (which NIELSEN has not in fact demonstrated, and which is contradicted by some of the available evidence; see Larson & Foulkes 1969; Pivik 1991; Rechtschaffen 1973; Rechtschaffen et al. 1972).

REM sleep is defined as a state in which diverse physiological factors covary in a distinctive pattern (Rechtschaffen & Kales 1968). Research into the neural basis of the REM state has accordingly focused on attempts to isolate "executive control" centres which "recruit and coordinate" the multiple component factors that constitute this distinctive pattern (Hobson 1988b; HOBSON ET AL. 1986; 1998b;). This is because the very existence of a stereotyped pattern of physiological variables suggests the existence of an underlying control mechanism which generates (recruits and coordinates) that pattern. The mechanism in question has no special relationship with each of the individual variables that participate in the pattern. Each of the individual physiological variables has its own mechanism (or, more usually, mechanisms); but these diverse mechanisms are not the issue here. The mechanism by which each of the various factors might be activated (or suppressed) in isolation is therefore of comparatively little consequence for our understanding of the REM state. This is because the state *is* the pattern. What is at issue with respect to the REM state is something else, namely the executive mechanism that recruits and coordinates the various factors into a distinctive configuration. It is *this* mechanism that is said to generate dreams; and it is *this* claim (i.e., the claim that the executive control mechanism of the REM state is also the mechanism whereby dreaming is generated) that is under dispute.

A useful analogy might be drawn with the concept of clinical syndromes. When each of the component elements of a syndrome appears in isolation, the diagnostic implications are indeterminate. It is only when all the elements appear simultaneously in a known, pathognomonic configuration, that they have definite diagnostic implications (i.e., they imply that a known pathophysiological mechanism is operative).

> The essence of a medical syndrome is that a collection of signs or symptoms, *when all present,* indicate the presence of a specific disease. The correlation between elements in a syndrome may be high, low or in-between . . . Indeed, a syndrome is most useful as a diagnostic tool precisely when the elements usually are not found together. When they are found together, this strongly points to some special pathological process. (Strub & Geschwind 1983, pp. 317–18; emphasis added).

A clinician who interprets isolated symptoms or signs as "covert" expressions of a known pathognomonic mechanism would commit serious diagnostic errors (cf. the fallacy of "partial syndromes"; Kinsbourne 1971). By the same reasoning, when individual elements of the REM state appear in isolation, the physiological implications are indeterminate; they have no necessary relationship with the REM state itself, or with its known physiological mechanism. It would be an error to infer that the REM state is "covertly" present, and that its control mechanism has somehow been "partially" activated, when individual features of the state appear in isolation or in bits of the stereotyped configuration.

NIELSEN commits precisely this error when he construes isolated instances or couplings of saccadic eye movement (rapid or slow), or muscle atonia, or EEG desynchronization, and the like, as somehow implying a partial or covert expression of the REM state. The physiological meaning (the underlying mechanism) of these isolated events is indeterminate, and has to be established in its own right in each instance. The events in question might well have nothing to do with activation (partial or otherwise) of the known executive control mechanism that recruits and coordinates the known pattern of events that constitute the REM state.

For example, a burst of EEG desynchronization during a NREM period might reflect a type of forebrain activation derived from a source quite different from and unrelated to the pontine cholinergic/aminergic oscillator that is thought to generate such desynchronization in the REM state. EEG desynchronization comes in many varieties, reflecting a wide range of different states, generated by diverse physiological mechanisms. EEG desynchronization, by itself, therefore, can mean almost anything. One can only "diagnose" the causal presence of the known pontine mechanism of the REM state if the burst of EEG desynchronization in question occurs within the context of the known REM "syndrome." This is true whether the desynchronization co-occurs with dreaming or not. Accordingly, if it can be demonstrated that NREM dreaming is regularly accompanied by phasic EEG desynchronization, then it is not at all justifiable to infer that the dreaming was causally triggered by partial or covert activation of the control mechanism of the REM state. It may well be that it was generated by an entirely different mechanism. This same principle applies to all the hypothetical "covert REM" events that **NIELSEN** refers to.

The pertinent question, therefore, still remains: *What generates those NREM dreams that are "indistinguishable by any criterion" from REM dreams* (Hobson 1988b)? For the reasons outlined in my target article, I do not believe that they are generated by reciprocal interactions between pontine cholinergic and aminergic mechanisms. If **NIELSEN** is suggesting that they are, then his thesis lacks conceptual coherence (and empirical support!). If, on the other hand, he is suggesting merely that the REM state shares scattered variables in common with other physiological states that are also productive of dreams, then he is making a very different claim – and a far weaker one: one which begs the main question that is at stake here. I agree that the REM state and certain NREM states which are productive of REM-like dreams are likely to share some physiological properties in common. The questions then become: (1) What are those shared properties? and (2) *What control mechanism recruits and coordinates them, and thereby generates the dreams?* To my mind, the search for these common properties and this underlying control mechanism should start from the observable fact of the dreams, not from theoretical preconceptions derived from our understanding of *REM* physiology.

One plausible empirical approach to the problem, then, is the classical clinico-anatomical approach; that is, to ascertain what anatomical structures are essential for dreams to occur. (This is the approach that I have taken.) To date, only two such structures have been identified: the parieto-temporo-occipital cortical junction and the ventromesial frontal white matter. (Nobody has ever demonstrated that pontine brainstem structures are essential for dreaming [dreaming in particular, as opposed to consciousness in general]). The task now is to verify whether the control mechanism we are seeking is indeed localizable to one of the identified structures. For the reasons set out in my target article, I believe that the best candidate for this role (in the present state of our knowledge) is a dopaminergic pathway that courses through the ventromesial frontal white matter. Now we need to establish whether and how activation of (and influences upon, and effects of activity in) this pathway (and other pathways in the ventromesial frontal quadrant) correlate with the actual experience of dreaming.

The old master of clinical neurology, Charcot, is reputed to have once said: "*La théorie c'est bon, mais ça n'empêche pas d'exister*" [Theory is good, but it does not prevent facts from existing]. (Freud 1893, p. 13). The psychophysiological theory of REM/dream isomorphism is, I suspect, preventing **NIELSEN** from acknowledging the existence of some unexpected clinico-anatomical facts which are difficult to reconcile with that theory.

Neuronal basis of dreaming and mentation during slow-wave (non-REM) sleep

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Abstract: Although the cerebral cortex is deprived of messages from the external world in REM sleep and because these messages are inhibited in the thalamus, cortical neurons display high rates of spontaneous firing and preserve their synaptic excitability to internally generated signals during this sleep stage. The rich activity of neocortical neurons during NREM sleep consists of prolonged spike-trains that impose rhythmic excitation onto connected cells in the network, eventually leading to a progressive increase in their synaptic responsiveness, as in plasticity processes. Thus, NREM sleep may be implicated in the consolidation of memory traces acquired during wakefulness.
[HOBSON ET AL.; NIELSEN; VERTES & EASTMAN]

Our experimental evidence on reciprocally related activities of neocortical and thalamic neurons supports some concepts in the target articles by **HOBSON ET AL.** and by **NIELSEN**. One of the ideas in the paper by HOBSON and colleagues is that NREM dreaming contains thought-like mentation whose content is much less dissimilar than is commonly believed when compared to that occurring in waking; in other terms, NREM reports are related to waking life. According to NIELSEN, the recall rate of dreaming mentation in NREM sleep is quite high. And, both HOBSON ET AL. and NIELSEN mention the vividness of dreaming near the end of the normal sleep period that may correspond in humans to the period of cat NREM sleep with incipient ponto-geniculo-occipital (PGO) waves, which heralds REM periods.

In this commentary, I would like to present some data that help to understand the neuronal basis of the association between NREM sleep and mentation. This may be surprising in view of previous postulates regarding this sleep state as accompanied by a global inhibition of cortex and subcortical structures (Pavlov 1923),

which would underlie the "abject annihilation of consciousness" (Eccles 1961). The two major claims arising from our recent experiments, using intracellular recordings from cortical and thalamic neurons under anesthesia as well as during the natural waking-sleep cycle of behaving cats, are as follows. (1) Despite the fact that the cerebral cortex is deprived in NREM sleep of signals from the external world because of their blockade within the thalamus, the gateway to neocortex, cortical neurons continue to entertain during this sleep stage a vivid dialogue, which is reflected in the high rates of spontaneous firing and preserved synaptic excitability to internally generated signals. Such aspects were unexpected during a behavioral state which is conventionally qualified as "passive," "resting" or "inactive." (2) The rich activity of neocortical neurons during NREM sleep consists of prolonged spike-trains that impose rhythmic excitation onto connected cells in the network, eventually leading to a progressive increase in their synaptic responsiveness, as in plasticity processes. Thus, we postulate that NREM sleep may be implicated in the consolidation of memory traces acquired during wakefulness. Our data are only indirectly related to the basic assumption in the target paper by **VERTES & EASTMAN**, denying the role of REM sleep in memory consolidation, as we champion the role of NREM sleep in this process.

The limit of our hypothesis is that, at the present time, intracellular recordings are performed, by necessity, in animals with a rather limited behavioral repertoire. And, terms such as "memory" are used to describe the occurrence, after rhythmic and prolonged testing volleys, of *spontaneous* neuronal activity displaying exactly the same patterns as those exhibited by stimulus-locked responses evoked during the prior period (see Fig. 6B in Steriade 1999). On the other hand, the advantage of our approach is that, for the first time, dual intracellular recordings from cortical or cortical and thalamic neurons are obtained *in vivo* in animals under an anesthetic that best simulates natural NREM sleep (Steriade et al., 1996) and, furthermore, intracellular recordings are performed during the natural waking-sleep cycle in behaving animals (Fig. 1). There is no need to elaborate on the advantages of intra-

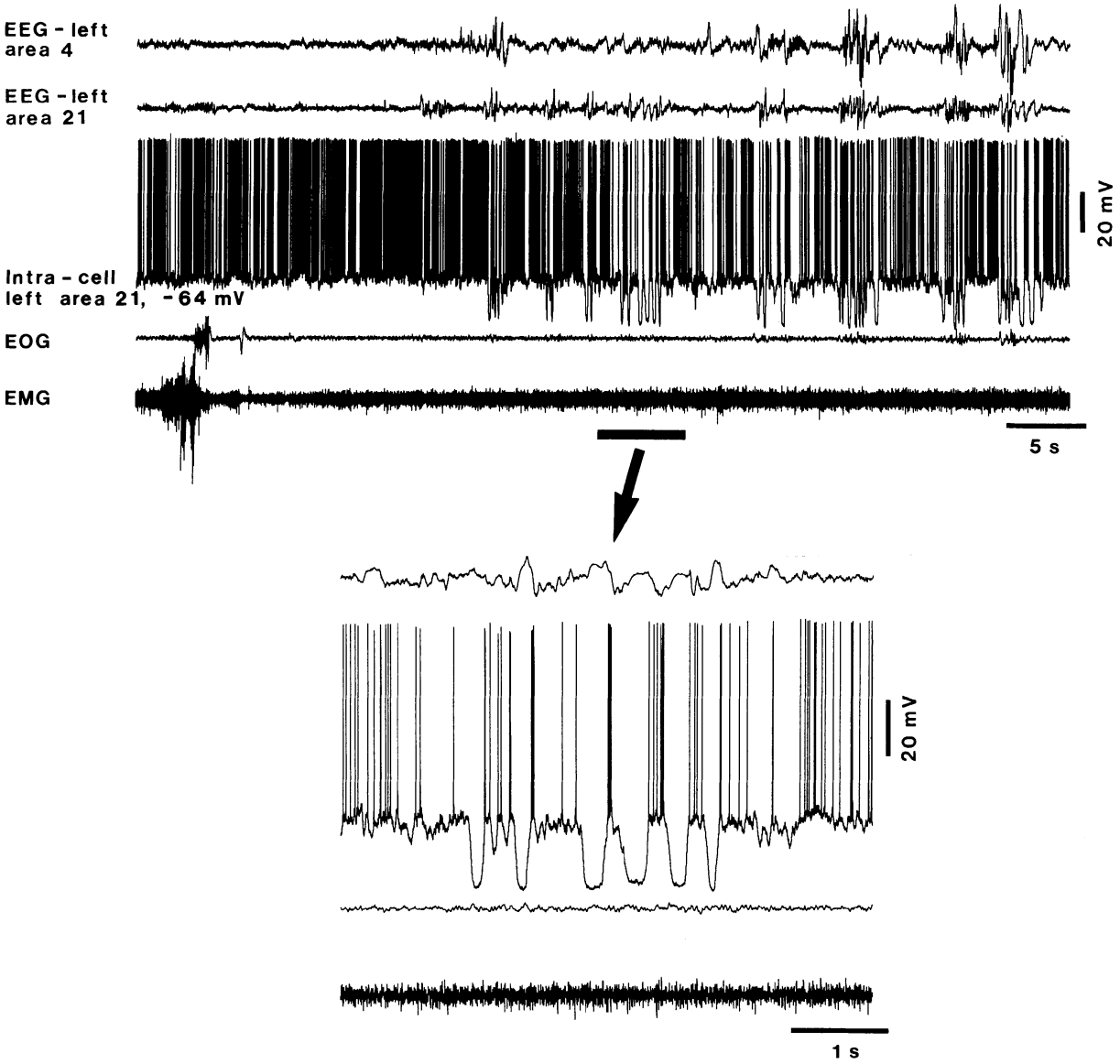

Figure 1 (Steriade). *Intracellular recording of neocortical neuron during natural waking and sleep states in behaving cat.* Upper panel: five traces depict (from top to bottom): EEG from left cortical areas 4 and 21, intracellular activity of neuron from area 21, electro-oculogram (EOG), and electromyogram (EMG). The panel illustrates the transition from wakefulness to NREM sleep (see onset of low-frequency, high-amplitude EEG waves). Part marked by horizontal bar is expanded below (arrow); only EEG from area 21 was illustrated. Note cyclic hyperpolarizations (downward deflections) of neuron during NREM sleep, but brisk firing during the depolarizing periods of the slow sleep oscillation. Unpublished data by M. Steriade, L. Timofeev, and F. Grenier.

cellular recordings from multiple sites, the only method that provides information on subthreshold membrane potential fluctuations during shifts in the state of vigilance and that can reveal the dissociation between exaltation of activity in cortical neurons and simultaneous postsynaptic inhibition in related thalamocortical cells, an inhibition mediated by the activation of GABAergic thalamic reticular neurons driven by corticothalamic projections (Steriade & Contreras 1995).

Let me start with the rich activity of neocortical neurons during NREM sleep, a state when these neurons are deprived of thalamic inputs that originate in pathways from the brainstem and the external world. That the thalamus is the first brain relay station where incoming signals are deeply inhibited from the very onset of drowsiness and even more inhibited during NREM sleep, without any change in the magnitude of the afferent volley that monitors the input reaching the thalamus, was documented by simultaneous recording of pre- and postsynaptic field potentials (Steriade 1991) and intracellular activities (Timofeev et al. 1996). Then, how is it that, without thalamocortical inputs, neocortical neurons still remain spontaneously active and responsive during NREM sleep? The fact is that corticocortical synapses exceed by far those made by thalamocortical axons. The major cortical rhythmic activity that characteristically defines NREM sleep, is a slow oscillation with a frequency between 0.5 and 1 Hz in both cats (Contreras & Steriade 1995) and humans (Achermann & Borbély 1997; Amzica & Steriade 1997). This oscillation is generated intracortically, as it survives an extensive thalamectomy (Steriade et al. 1993) and it disappears in the thalamus following decortication (Timofeev & Steriade 1996).

The distinctive feature of the cortical slow oscillation during NREM sleep is the alternation between prolonged periods of depolarization leading to spike-trains and long lasting periods of hyperpolarizations associated with neuronal silence. This stands in contrast with the tonic depolarization and firing of cortical neurons during both waking and REM sleep (Fig. 1). Although neuronal discharges are absent during the hyperpolarizing periods of the slow sleep oscillation, the overall firing rates of cortical neurons are quite close in NREM sleep and in wakefulness (~12 Hz and ~14 Hz, respectively, in electrophysiologically identified regular-spiking neurons). This is due to the fact that, during the depolarizing phase of the slow oscillation, cortical neurons discharge vigorously, in many instances above the level observed in waking or in REM sleep (Steriade et al. 1999). Not only do cortical neurons fire spontaneously at high rates during NREM sleep, but their synaptic excitability to intracortical signals is enhanced during this stage. In contrast to the synaptic inhibition of thalamic neurons during NREM sleep and particularly during sleep spindles (which explains the thalamic blockade of incoming signals), earlier extracellular recordings in behaving monkeys showed an increased responsiveness of neocortical neurons to callosal volleys in NREM sleep compared to waking (Steriade et al. 1974) and recent intracellular recordings demonstrate that the cortically-evoked excitatory responses of cortical neurons are not diminished during the prolonged hyperpolarization of the slow sleep oscillation (Timofeev et al. 1996).

The result of intracellular recordings in naturally sleeping animals, showing high rates of spontaneous neuronal firing in neocortex during NREM sleep, raised the obvious question: what is the functional role of rhythmic spike-trains fired by neocortical neurons during the slow sleep cortical oscillation (0.5–1 Hz)? With the corollary: What may be the influence of rhythmic spike-bursts fired by thalamocortical neurons during sleep spindles (7–15 Hz) upon cortical neurons? Both these types of sleep rhythms (spindles and slow oscillation) have an impact on neocortical neurons and may change their responsiveness and even induce structural changes in their dendritic arbor that could have important consequences for the consolidation of traces produced by events occurring in other states of vigilance.

We started to work out the above hypotheses by simulating a major sleep oscillation, thalamically-generated spindles, using trains of thalamic stimuli applied at ~10 Hz, while simultaneously recording cortical and thalamic neurons intracellularly (Steriade et al. 1998). The responses to pulse-trains at ~10 Hz grow progressively in size, from the second stimulus in the train and, therefore, are termed *augmenting*. Both the thalamus and the cerebral cortex have the neuronal machinery that is necessary to generate augmenting responses, as shown by the fact that such responses can be recorded in the neocortex of athalamic animals and in the thalamus of decorticated animals. However, the full consequences of the augmenting phenomenon, which include self-sustained oscillations and plastic changes in network activities, require intact-brain preparations. This need for an intact brain, including generalized modulatory systems, is also shown by the state-dependency of augmenting responses which have maximal amplitudes in NREM sleep and lowest amplitudes during full alertness and REM sleep.

Augmenting responses are associated with plasticity processes, that is, decreases in inhibitory responses and persistent and progressive increases in excitatory synaptic responses. Such changes can lead to self-sustained oscillations due to resonant activities in closed loops, as in memory processes (Steriade 1999). The repeated circulation of impulses in reverberating circuits between the cortex and thalamus, may lead to synaptic modifications in target structures, which favor alterations required for memory processes. This hypothesis was also proposed (Buzsáki 1989) and tested experimentally (Wilson & McNaughton 1994) in the hippocampus. The hippocampal "place cells" were found to display higher firing rates and had a tendency to discharge synchronously during sleep, as if neuronal states are played back as part of the memory consolidation process.

Finally, NIELSEN's model, implicating covert REM processes before the full-blown REM sleep, follows experimental data published more than a decade ago in which we attempted to link the dreaming mentation to the appearance of PGO waves well before muscular atonia and EEG activation, during EEG synchronization of the final NREM period. In that paper (Steriade et al. 1989), we proposed that "vivid imagery may appear well before the classical signs of REM sleep, during a period of apparent EEG-synchronized sleep." This idea was based on the fact that visual thalamic neurons fire spike-bursts related to PGO waves (the robust bursts are owing to the fact that thalamic neurons are still hyperpolarized during that final period of NREM sleep); and also because the spontaneous firing rates of thalamic neurons is low, the signal-to-noise ratio during the PGO-related spike-bursts is very high, thus possibly underlying the vivid mental experiences outside REM sleep.

Inclusive versus exclusive approaches to sleep and dream research

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Abstract: By assuming that REM sleep either plays a critical role in all memory consolidation or no role in any, Vertes & Eastman have chosen to reject, rather than explain, robust experimental findings of a role for sleep in memory and learning. In contrast, Nielsen has attempted to integrate conflicting findings in the dispute over REM versus NREM mentation. Researchers must trust the data more and the theories less, and build integrative rather than exclusionary models if they hope to resolve these knotty problems effectively.
[NIELSEN; VERTES & EASTMAN]

Introduction. It is striking that 100 years after Freud (1900), there is absolutely no agreement as to the nature of, function of, or brain mechanism underlying dreaming. There is even disagreement as to what constitutes a legitimate approach to the question. In this environment, it is perhaps not surprising that var-

ious researchers have staked out strikingly different positions that are often presented as incompatible with one another. An alternative interpretation, one that I will argue here, is that we are discovering, but in many cases ignoring, the rich complexity of sleep and dreaming. In what follows, I will argue that the paper of VERTES & EASTMAN has fallen into this trap, choosing to look at only some of the available data to draw a conclusion that rejects, rather than explains, other robust experimental findings. In contrast, I will support NIELSEN's attempt to integrate conflicting findings in the dispute over REM versus NREM mentation. In the end, I will conclude that we should probably trust the data more and the theories less, and build integrative rather than exclusionary models. Only by moving in this direction will we be able to resolve these knotty problems effectively.

Vertes & Eastman: The case against memory consolidation in REM sleep. The argument against REM sleep having a role in memory consolidation by VERTES & EASTMAN is a disappointingly anachronistic and one-sided review of this rich literature. At the end of their introduction, they give the overarching reasons that lead them to conclude that REM sleep can have no possible role in learning and memory: "Sleep involves basic biological functions and memory requires consciousness." What could they possibly mean? Are learning and memory consolidation not basic biological functions? Are these "psychological" phenomena and hence not of any basic importance? Second, assuming for the moment (as they clearly do) that we are unconscious when we dream in REM sleep, are old memories not activated, associated, and integrated to form dream imagery and narrative? And if we remember our dreams on awakening, haven't new memories been laid down during sleep? It is as if they want to say that memory is too complex for sleep and sleep is too important for memory. These attitudes could have been forgiven if they were followed by a good, critical review of the literature. Unfortunately, they were not.

Memory systems. One problem with VERTES & EASTMAN's discussion is that they have a view of memory systems that is at least seven years out of date (Schacter & Tulving 1994). While vaguely acknowledging that there is data supporting the concept that REM sleep is more important for learning complex tasks than simple tasks and making passing reference to suggestions that "information is differentially processed in distinct phases of SWS and/or REM sleep," they never mention procedural or declarative memory systems and only refer to working and episodic memory in a quote from Barbara Jones (1998) about the possible role of sleep-related decreases in frontal lobe activity on subsequent dream recall. Yet for the last decade much of the work on sleep and memory has focused specifically on the differential effects of various sleep stages on procedural versus declarative versus motoric memory systems (Plihal & Born 1997; Smith & MacNeill 1994; Smith and Rose 1997; Smith et al. 1998; Stickgold 1998; Stickgold et al. 2000b). VERTES & EASTMAN seem to take the position that either REM sleep is absolutely critical for all forms of learning and memory consolidation or else it has no role whatsoever in any form of these processes. Obviously, this is an unjustifiable dichotomy.

Biased reviewing. A more worrisome problem is how they choose to cite the literature. Whether it is noting the appearance of Jonathan Winson and Matt Wilson on the Charlie Rose TV program "promoting their shared belief" in a role for sleep in learning, or suggesting that our own findings of sleep-dependent memory consolidation is "very much at odds" with our activation-synthesis model of dreaming, or quoting the frustration of Elizabeth Hennevin and Bill Fishbein with the resistance of the sleep community to their empirical findings (Fishbein 1995; Hennevin et al. 1995a), VERTES & EASTMAN often seem to be spending more time criticizing the researchers than their data. Beyond this, their discussion of the actual data is frustratingly skewed. In their discussion of REM deprivation effects on memory, they pay considerable attention to the legitimate concern over the interpretation of cognitive tests given 30 min or 3 hr after several days of exposure to stressful REM deprivation techniques, but they ignore findings of equally strong effects that are seen a week after a relatively brief 4-hr period of REM deprivation (Smith & MacNeill 1994) or after a single night of sleep deprivation in humans (Aubrey et al. 1999; Smith & MacNeill 1994). Again, they note that both Karni et al. (1994) and our laboratory (Stickgold et al. 2000b) have reported strong relationships between REM sleep and learning on a visual discrimination effect (we reported an *r*-value of 0.89 between post training sleep parameters and subsequent improved performance, a correlation significant at $p < 0.0001$), but then they focus on what we and Karni agree are relatively minor differences between our findings and conclude that "until these discrepancies are resolved, it is difficult to evaluate the reliability of the findings." No such critical lens is held up to the work of those who find no correlation between sleep and learning!

Alternative explanations. Having rejected all findings of effects of REM deprivation on learning under the rubric of "very stressful deprivation procedures," they go on to conclude that there must be no effect at all of REM on memory because "there appears to be no alternative explanation for studies which have failed to show that [REM deprivation] disrupts learning/memory." Yet as all researchers should know, the failure of a test to reach statistical significance only indicates a failure to demonstrate the presence of a difference. It does not demonstrate the absence of such a difference. Beyond the issue of statistics, there is another very simple "alternative explanation." Since all proponents of REM-dependent memory consolidation agree that REM is not involved in consolidation of declarative memories such as those formed in paired associates training, the failure to observe REM-dependent consolidation may simply reflect the testing of a memory system that is not REM-dependent. The failure of the reviewers to address this issue, despite reviewing articles that directly discuss it (Plihal & Born 1997; Smith & MacNeill 1994; Smith & Rose 1997; Smith et al. 1998; Stickgold 1998; Stickgold et al. 2000b), is a frustrating disappointment.

Theta rhythms. A role for theta rhythms is acknowledged by VERTES & EASTMAN in the processing of memories during wake, but they reject any similar role in REM: "If the transfer of information in REM is not orderly, or is essentially chaotic, it would seem that there would be no functional value in consolidating or 'remembering' this information" (sect. 2.6). Fair enough. But there is very often order in nature that we fail to see. Let me suggest an example of how REM sleep might be processing stored information in a less obvious manner. One critical task of the mammalian brain is to search for useful new connections between previously stored memories, most usefully memories stored in neocortical association networks, rather than the hippocampal, declarative system. While not critical on a minute by minute or even day by day basis, these new connections allow organisms to identify classes of related precepts, superclasses of causal relationships, and insights into novel relationships among sets of stored information. These are precisely the types of new associations that people are seeking when they "sleep on a problem." The problems one "sleeps on" are not trying to remember phone numbers. No one believes that sleep will help recall lost episodic or declarative memories. Rather, one sleeps on problems that involve assessing alternative explanations of past events or possible outcomes of future events.

The physiology of REM sleep would seem to support such information processing. Information outflow from the hippocampus to neocortex is shut off (Buzsáki 1989; 1996), cortical association nets are loosened (Stickgold 1998; Stickgold et al. 1999b), and cortical memory formation is enhanced (Hasselmo 1999; Hasselmo & Bower 1993) during REM sleep. Together, these allow the brain to (1) ignore the predictable interpretations driven by the replay of episodic memories (noticeably absent in REM dreaming), (2) seek out and test novel associations within the cortex, and (3) strengthen them as appropriate. The consolidation of procedural memories, also residing in neocortical rather than hippocampal memory systems, could follow a similar strategy. Interestingly, the flow of information back into the hippocampus during REM sleep might then serve to signal the appropriateness of "forgetting"

episodic memories (Crick & Mitchison 1983) after cortical integration. Evidence for such erasure via LTD has been proposed by Poe et al. (1997).

If this is the role of sleep in memory consolidation, or if this even touches on sleep's role, it is not surprising that we find such variable results in published studies. These are not easy forms of consolidation to quantify. Indeed, these processes probably involve memory integration even more than they do simple memory consolidation. So it is not surprising that simple cognitive and psychomotor memory tests fail to show any obvious impairment of performance after administration of drugs that disrupt REM sleep. These tests classically measure working memory and declarative memory systems that we would not expect to be affected by REM deprivation. We know of no cases in which anyone, for example, tested the effects of these drugs on complex perceptual procedural learning.

Magical processes. Near the end of their article, **VERTES & EASTMAN** note that many have been tempted to believe that "magical processes occur during REM sleep." Indeed they do, and not just in REM sleep. We do not understand how dreaming comes about, how memories and concepts are so intriguingly woven to form the narrative of the dream. We do not understand how, in waking, the brain comes to understand language, or color, or beauty. But this does not mean that they do not occur. It merely means we do not know how they are produced. When we find a correlation between sleep and learning that explains 80% of intersubject variance and is significant at the 0.0001 level, something is happening, magical or not.

In the end, I am honestly baffled by **VERTES & EASTMAN**'s decision to reject all of the work that points toward a role for sleep in memory consolidation and learning. It is as if, finding the cup half full (or half empty), they insist that it must be either completely full or completely empty. Surely, the best reading of the literature would say that sleep plays an important, if not necessarily critical, role in some forms of memory consolidation and learning. Drawing such a conclusion is not a compromise. It is scientific fact.

Nielsen: Mentation in REM and NEM sleep: A review and possible reconciliation of two models. The question of how to characterize the role of sleep states in the control of sleep mentation remains a thorny one. In this context, **NIELSEN**'s offering is a welcome breath of fresh air. He presents an impressively complete review of the extant literature and provides a valuable theoretical framework for the critical analysis of this literature. **NIELSEN** has attacked the dichotomous approach to REM and NREM sleep even more effectively than we did in our article (**HOBSON ET AL.**). By offering the concept of "covert REM sleep" to describe the spread of REM sleep physiology into NREM states, he emphasizes one essential aspect of our state space model: the continuity, overlap, and dissociability of state features.

There are two main points I would make about his model. First, while the early part of his paper points strongly toward qualitative differences between REM and NREM mentation, **NIELSEN** seems in the end to suggest that all NREM mentation is due to the covert intrusion of REM state processes. If this is to explain the qualitative differences between REM and NREM, one would have to assume that different REM state processes add different features to the mentation process. While this may, indeed, be true, I still would argue that there is at least a basal tendency toward sleep mentation even when all of these REM state processes are inactive. This could explain the mentation reports of SWS and would further simplify the explanation of qualitative differences; as more and more REM state processes are activated, the mentation shifts more and more from a "pure" NREM phenomenology toward a "pure" REM phenomenology.

My second point is that I believe **NIELSEN** could have taken his model even further. I would propose three basic tenets of such an expanded model:

1. Sleep mentation and dreaming are products of the brain, and are determined at any given time by the levels of activation and interaction of disparate brain systems. These include anatomically defined systems such as the brainstem, amygdala, and frontal cortex, cognitively defined systems such as attention, emotion, and memory, and neuromodulatory systems such as the cholinergic, noradrenergic, and serotonergic systems.

2. All of these systems show fluctuations in their levels of activation and functional connectivity across the sleep cycle.

3. The use of polysomnography to define sleep stages represents a crude division of these rich and complex physiological fluctuations.

Given these assumptions, perhaps Rechtschaffen and Kales's sleep stages (Rechtschaffen & Kales 1968) should be seen as only a first attempt at defining the rich heterogeneity of sleep. For many purposes it is a completely adequate description of the system. For other purposes it will clearly prove only marginally adequate or even totally inadequate. Hobson (1992a) has proposed a three dimensional model of sleep state space based on levels of sensory input, brain activation, and neuromodulation. In our paper in this issue (**HOBSON ET AL.**), we begin to move toward expanding this model so that different brain regions can be in different portions of this state space. Eventually the number of dimensions will necessarily increase as will the number of brain regions that need to be viewed separately. **NIELSEN** is hinting at this complexity, but I suspect that before too long we will have to accept that REM and NREM are useful concepts when looking grossly at sleep phenomena, but inadequate as we refine our investigations of these phenomena.

In conclusion, I suggest that we need to go even beyond **NIELSEN**'s idea of covert REM sleep processes in NREM sleep and, of course, covert NREM sleep processes in REM sleep, and instead accept a rich, complex, and confusing panoply of brain processes that show robust, but nonetheless only statistical, probabilities of co-occurring and of being sustained for periods of minutes or tens of minutes.

Evolutionary psychology can ill afford adaptionist and mentalist credulity

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Abstract: The idea that dreams function as fright-simulations rests on the adaptionist notion that anything that has form has function, and psychological argument relies on the mentalist assumption that dream reports are accurate reports of experienced events. Neither assumption seems adequately supported by the evidence presented.
[REVONSUO]

REVONSUO's core idea – that dreams must be a biological threat simulation system because dream reports are highly structured narratives uniquely sensitive to threatening circumstances – provoked in me both biological and psychological misgivings. On the biological side, the argument fails because it requires the false premise that all the things that have form have function. The form of many recognizable structures (e.g., the human nose, the armpit, the pseudopenis of the female hyena, etc.) is determined by selection on other features of the organism and has no role in their determination (Glickman et al. 1993; Gould & Lewontin 1979). Even the association of "randomness" with "formless" is inappropriate. If you and I were to bump into each other in the street, we might very well speak of a random meeting. We could truthfully characterize the meeting in this way, but not because the behavior that led to it was formless or without direction, nor even because the meeting was not entirely predictable. We would say that the meeting was random because the factors that determined the meeting were not governed by it. To show that the meeting was non-random, we would have to show that our behavior was in some sense designed to produce a meeting.

Similarly, to show that dreams function to improve performance in threatening situations, we must show not only that dreams have this consequence but also that they are designed for it. Even the evidence offered by the author that dreams produce improvements in performance is shaky enough: it seems to be based solely on an analogy with motor imaging experiments. The evidence that dreams are designed to produce these consequences seems even shakier. (Thompson 1981): (1) the comparative method, (2) the engineering method, and (3) the examination of reproductive consequences. Of the three classes of evidence, the only evidence consistent with design is related to the engineering criterion, in this case evidence that dreaming is sensitive to the occurrence of threatening situations in the dreamer's life and that dream content is related to threatening occurrences. No comparative evidence is provided – no evidence that across species, animals dream more that are subject to hazards: nor is there any evidence that non-dreamers live particularly short and unfecund lives in hazardous environments.

On the psychological side, **REVONSUO** seems unaware of the sort of misgivings that might be entertained by behaviorists concerning his research program. The author speaks of dreaming as if it posed no special philosophical or methodological difficulties. According to his account, the dream is a shoe the dreamer puts on for himself because it helps him practice for dire situations. Waking up a dreamer is sort of like interrupting a person who is watching TV drama. Once we get his attention, we can ask him about the nature of the "program" he has been watching, and he can report luridly concerning his experience. Only the dream is problematized. Remembering the dream and reporting on that memory are taken as unproblematic. This stance is unlike any that we would take with reports of emotion-laden events obtained in non-dreaming contexts. Consider, for instance, the stance a therapist would take toward a client who reported an argument he had with his spouse during the previous eight hours. Unlike the dream researchers, the therapist would not presume that the client has exact recall of the argument or that the client would give a faithful report of such intimate and troublesome events.

Such methodological credulity with respect to dream recall and dream report is particularly troubling because dreams are notoriously ephemeral. Dreaming, or being aware of our dreams, or remembering our dreams, or telling coherent accounts of our dreams are not skills that the people of my acquaintance possess equally. Some people seem to expand a lot of effort in rehearsing and relating their dream reports: others to be hesitant to report their dreams, bad at remembering them, or, perhaps, dream rarely if at all. Even the best dreamers around me confess that dreams are so evanescent that they will be lost or altered in memory if they are not written down or related immediately upon awakening.

In fact, why do we assume that there is anything that is dream apart from the subject's reporting of it? Even if I grant that I, too, awake in the morning with the sense that I have had experiences while sleeping, what is the reason for believing that these experiences correspond to any facts of the matter whatsoever? The supporting evidence used to be that the REM sleep syndrome was a necessary concomitant of dream reports, but I gather that even that evidence is no longer credible (see **REVONSUO**, sect. 1.1). Is the ontology of dreaming so firm that we cannot even imagine a skeptical account of dream reports that does not require the existence of dreams? Whatever we conceive sleep to be, forced awakening certainly provokes a massive reorganization of neural activity. Is it such a wonder that such a profound intrusion into ongoing neural activity should not knock off shards of memory and send them hurtling into awareness? Is it such a wonder, storytelling creatures that we are, that these shards should be assembled into fragile narratives? Is it so strange that we should perceive these stories as occurring prior in time, during the previous sleep? Finally, given that these narratives are commonly assembled in the context of an interruption of ongoing activity, is it such a wonder that they should have a threatening tone? Surely, the dream report literature should be subjected to the same sort of skeptical assault that tested the hypnosis literature in the 60s and 70s (Sarbin 1981) Can experimenters reliably distinguish fake from "real" dreamers? Does dream fakery get better if the subject is drowsy when asked to generate a fake dream? What if the drowsy subject is forbidden to tell a dream but allowed only to relate an imaginative story (cf. Fiss et al. 1966)? Or to give an account of something that "happened yesterday"? is a recently awakened subject more likely to relate a negative story than a randomly probed subject? Wouldn't you?

As evolutionary psychology gains attention and respect, the question of what kind of a psychology it is to be – mentalist or behaviorist, adaptionist or selectionist – becomes more important. Is evolutionary psychology to explain feelings, thoughts, beliefs, desires, and other events "within the head" by reference to vaguely conceived benefits? Alternatively, is it to explain patterns in the activities of humans in terms of a history of differential reproduction of individuals enacting those patterns? Insensitivity to these crucial issues will slow the development of the field.

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Critique of current dream theories

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Abstract: Modern lab research has found that contrary to the suggestions of Hobson et al., Nielsen, and Solms, dreams are organized, mundane stories. Hence, their theories to explain the distortions and bizarreness of dreams are misdirected. Hobson et al. propose that REM sleep processes are responsible for dreams. But dreaming occurs in absence of REM sleep and REM sleep is often accompanied by no dreaming. Hence, REM sleep is not necessary or sufficient for dreaming.
[HOBSON ET AL.; NIELSEN; SOLMS]

HOBSON ET AL., SOLMS, and **NIELSEN** propose dream theories to explain distortion, disorganization, and bizarreness in dreams. They assume that these are the salient characteristics of typical dreams, a nineteenth century view also held by Freud. We now know that this view is wrong. It is based mostly upon reports of dreams that are spontaneously recalled upon waking in the morning. These dreams are likely the most dramatic, bizarre dreams and are not representative of dream life in general. The collection of large dream samples from throughout the night from large samples of ordinary people, both children and adults, has shown that most dreams are mundane, organized, everydayish stories (Dorus et al. 1971; Snyder et al. 1968). Though novel, they are not bizarre. Though often vivid, they do not often express inappropriate or extraordinarily intense emotion. Though unreflective, they are not disorganized. HOBSON ET AL., SOLMS, and NIELSEN ignore this massive evidence about the nature of dreams. Thus their dream theories are inappropriately directed and miss the main point – and mystery – about dreams, namely, that even during sleep, the mind/brain produces organized, coherent, understandable mentation.

HOBSON ET AL.'s dream theory, the activation-synthesis hypothesis (ASH) (Hobson & McCarley 1977), is also inconsistent with other twentieth century findings. The hypothesis asserts that during REM sleep, random neuronal discharge of the hindbrain REM sleep generator activates the forebrain to produce the conscious dream. According to this hypothesis, distortion, disorganization, and bizarreness are dream characteristics because the forebrain can only perform "the best of a bad job" in synthesizing its random brain stem stimulation. The hypothesis predicts that dream distortion and bizarreness will increase during more intense "random" stimulation by REM generator (Hobson & McCarley 1977). Tested in several studies, this prediction has been refuted although with some minor exceptions (for review, see Pivik 1991; Rechtschaffen 1973). The hypothesis also predicts that

dreaming will occur only in REM sleep. Many studies found that dreams were reported from more than 50% of awakenings from nonREM sleep during periods far separated from waking and REM sleep and during which the REM generator neurons are silent (for review, see Pivik 1986). And many studies found that dreams were reported from the 70% of awakenings from sleep onset when the REM generator is silent (for review, see Vogel 1991). Most important, two independent studies found that, equated for length, REM and nonREM reports were indistinguishable (Antrobus 1983; Foulkes & Schmidt 1983;). These findings indicate that REM sleep is not necessary for dreaming and in particular for dreaming with the formal characteristics (bizarreness, distortion, disorganization) that Hobson and others claim are the distinctive formal characteristics of REM dreams. HOBSON's response to this critique is that REM-like nonREM mentation is produced by REM sleep processes during nonREM sleep.

HOBSON ET AL. propose that several findings support this requirement of the ASH. Here is a point by point refutation of their proposals.

1. **HOBSON ET AL.** propose that circadian rhythm based increases in late night stage two activation sustain larger and more vivid nonREM dreaming. They then suggest that brain activation during nonREM sleep is a "REM-like phenomenon" (p. 56). In rebuttal, this is about brain activation, which is not a distinctively REM sleep process. Brain activation also occurs when awake. Thus, dreaming during late night Stage 2 activation is not associated with a distinctive sleep process as required by ASH. Furthermore even HOBSON ET AL.'s proposed association between nonREM sleep activation and dreaming is not contradicted by evidence. For example, (a) dreams are reported from nonREM stages 3 and 4, which, based on metabolic studies, are periods of low brain arousal (Pivik 1986; Pivik & Foulkes 1968; Rechtschaffen 1973); and (b) Pivik and Foulkes (1968) found that stage 3 dream recall was similar to stage 2 dream recall.

2. **HOBSON ET AL.** propose that nonREM dreaming occurs during pre REM slow wave sleep in association with preREM sleep PGO waves, which are prominent phenomena during REM sleep. In rebuttal, this proposal is hypothetical. Though pre-REM sleep PGO waves have been observed in the cat, there is no direct evidence of their occurrence in humans. Second, even if they do occur, contrary to HOBSON ET AL.'s proposal, Pivik's review (1991) indicates that nonREM dreaming is not increased during preREM period; and Larson & Foulkes (1969) found fewer, less dream-like reports during the REM EMG drop than during control periods. Although HOBSON has theorized prodigiously about the mental correlates that accompany the initial activation of REM mechanisms, he has conducted absolutely no psychophsysiological research to verify his accounts. Others, however, have studied the phenomenological correlates of immediately pre-REM and early REM physiology. Both Larson and Foulkes (1969) and Foulkes et al. (1980) found that early signs of REM sleep activation (e.g., the sudden loss of muscle tone) were accompanied by reliable decreases in the dreamlike quality of sleep mentation, a remarkable and direct refutation of the heart of HOBSON ET AL.'s theory.

3. **HOBSON ET AL.** propose that during nonREM sleep external stimuli produce PGO spikes which then cause visual imagery, the hallmark of dreams. This is speculation. HOBSON ET AL.'s work indicates that in humans acoustic stimuli during stage 2 enhance visual imagery and they cite other work indicating that in cats sound stimuli stimulates PGO. But they present no evidence of the requirement of the ASH, namely, that in humans external stimuli during nonREM sleep reliably stimulate REM-like activity in association with reported imagery. In fact, in cats, external stimuli produce isolated PGO spikes, not the long trains of PGO spikes that ASH would require of dream substrates.

4. **HOBSON ET AL.** cite a Pivik result that nonREM phasic spinal reflex inhibition was associated with greater recall, auditory imagery, and hostility. In contrast, Rechtschaffen's (1973) analysis of this study concluded that there was no significant relationship of phasic variables studied (including dreamlike fantasy, sexuality, morality, thought, emotion, etc.). only hostility and auditory imagery were significantly related to phasic events. It appears that by selective reporting, HOBSON ET AL. were trying to give a positive spin to what was essentially a negative result.

5. **HOBSON ET AL.** cite a Rechtschaffen result that nonREM PIPs (phasic integrated potentials), were associated "with enhanced recall" of mentation. PIPs are extraocular spikes recorded from surface electrodes in humans. These spikes have morphological and distributional features like the extraocular spikes associated with PGO potentials in cats. Thus, they have been interpreted as surface indicators of PGOs in humans. But HOBSON ET AL. are wrong in their assertions about the relationship between PIPs and recalled nonREM mentation. In Rechtschaffen et al.'s work (1972), 51% of PIP awakenings and 53% of control awakenings showed recalled content, clearly no substantial differences in percentage of nonREM awakenings with recall. As Rechtschaffen (1973) pointed out, "PIPs do not appear responsible for dreaming per se" and "there is more dream activity in nonREM sleep than spike activity." Finally, PIP bursts in nonREM sleep typically last only a few seconds. They are never long enough to account for a long nonREM dream.

The bottom line is that long, detailed dreams – visually indistinguishable from the most elaborate of REM dreams – can be elicited on awakenings from nonREM sleep with little or no evidence of distinctive or near distinctive REM-like phenomena, including (1) low voltage, mixed frequency EEG; (2) rapid eye movements; (3) tonic or phasic EMG suppression; (4) saw-tooth waves; (5) PIPs (possibly representing PGO); and (6) penile erections. Therefore, dreaming is not dependent on REM sleep physiology, and the characteristics of dreaming in terms of REM-like intrusions into nonREM sleep have speculatively relied on relatively subtle indications of such instructions with little evidence that they are related to nonREM dream reports. This evidence indicates that REM sleep phenomena are not necessary for dreaming.

The ASH also predicts that REM sleep is sufficient for the production of bizarre, distorted, disorganized dreams. This is because the hypothesis leads us to expect that excitation of the brainstem REM sleep generators will produce bizarre, distorted dreams. The evidence contradicts this prediction. In a large collection of studies, 17% of awakenings from REM sleep produced no dream reports. Also, dream reports may be sparse or absent on awakenings from REM periods of young children and when present, they are usually mundane and undistorted (Foulkes 1982b). Finally, as mentioned above, short REM reports are like typical nonREM reports – thought-like, mundane, undistorted (Foulkes & Schmidt 1983).

In short, contrary to the ASH, REM sleep is not necessary or sufficient for dreaming. Thus, unique REM sleep processes, such as discharge of brainstem REM sleep generator, cannot be causes of dream production. Thus, the ASH is wrong.

HOBSON ET AL.'s neurophysiogical theory of dream generation rests on what they call a mind/brain isomorphism. The theory is based in the proposition that dream distortion is caused by "random" discharge of neurons in the REM sleep generator. But we have no evidence that "disordered" meaning is caused by a disordered (temporally random) discharge pattern of individual neurons. In more general terms, we do not know the neural correlates of particular sequences of mentation, ordered or disordered. We do not also know that the orderliness of mental sequences is determined by the pattern of hindbrain stimulation of the forebrain. It is possible that mentation is relatively independent of hindbrain stimulation and is primarily determined by the response characteristics of the forebrain. In any case, our ignorance about these issues is very great. In view of such ignorance, the claim that disordered (incoherent) meaning is caused by disordered (random) discharge of individual pontine cells appears to be based on verbal similarity rather than empirical findings (Vogel 1978a). Indeed, the empirical findings listed above do not support this claim.

Aside from the particular dream theories, a major premise of

HOBSON ET AL., SOLMS, and **NIELSEN** is that brain physiology can tell us about the psychology of dream experiences.

> The idea is that the nocturnal activation of previously identified "centers" for emotionality, symbolism, integration, etc., will implicate, or fail to implicate, these physiological processes in dreaming. But the evidence that these brain "centers" actually are substrates of psychological phenomena rests on the correlation of their activity with those phenomena in the first place. Thus, if dreams prove generally to be well integrated sequences of mental activity, while brain scans tell us that alleged integrative brain centers are quiescent, then we have found, not that dreams are disorganized, but rather that contemporary neurophysiology has not yet adequately identified the neural substrates of ideational integrity. This use of brain sciences to "prove" or "disprove" psychological findings or theories is, in principle, erroneous, and since that is the main premise of this account of dreaming, the account is fatally flawed. (Foulkes, personal communication)

Finally, let us apply these methodological comments about mind/brain to Freud's psychological dream hypothesis. This asserts that dreams are instigated by – and express – unconscious, unacceptable wishes. A test of this hypothesis will measure the empirical correlation between dream reports and unconscious, unacceptable wishes disguised in these reports. Neurophysiological findings cannot provide these data. Only reliable psychological data can. The fact that 100 years have passed without such a test suggests that Freud's hypothesis is empirically untestable and hence outside the realm of science.

The pharmacology of threatening dreams

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Abstract: The pharmacological literature on negative dream experiences is reviewed with respect to Revonsuo's threat rehearsal theory of dreaming. Moderate support for the theory is found, although much more work is needed. Significant questions that remain include the precise role of acetylcholine in the generation of negative dream experiences and dissociations between the pharmacology of waking fear and anxiety and threatening dreams.
[REVONSUO]

REVONSUO has generated a provocative and persuasively argued theory for the biological function of dreaming. The author has limited this case (not unreasonably so) to the phenomenal level. What I propose to do in this commentary is to extend the hypothesis to biological and pharmacological levels of analysis and to evaluate whether the author's case is consistent with this literature.

Several predictions reasonably derive from the threat rehearsal hypothesis: (1) biological mechanisms should be found that subserve dreams of a threatening nature – if not in fact, then at least in principle; (2) agents which suppress those biological mechanisms should inhibit such dreams – either yielding dreams of a less threatening nature and/or to eliminate dreaming altogether; (3) the withdrawal of such agents following repeated administration should lead to a reinstatement, perhaps even an exacerbation, of threatening dreams; and (4) there should be significant overlap between drugs that inhibit fear and anxiety in the waking life and those that inhibit fear and anxiety during dreams.

I will now briefly review the pharmacological evidence that is both consistent and inconsistent with **REVONSUO**'s hypothesis. Importantly, I will operate on his assumption that REM sleep is the typical, perhaps even optimal (though not exclusive) phase of sleep for dreams involving threat rehearsal. Because of its association with night terrors (Fisher et al. 1973) deep slow-wave sleep (SWS) will also play a prominent role in my analysis.

First of all, a number of agents with anti-anxiety properties suppress stage 4 slow-wave sleep, rapid-eye movement (REM) sleep, or both. Tricyclic antidepressants (TCAs) (Vogel et al. 1990) MAO inhibitors (Vogel et al. 1990), and selective serotonin reuptake inhibitors (SSRIs) (Nicholson & Pascoe 1988) reduce REM sleep. Barbiturates and benzodiazepines suppress deep SWS and either suppress or delay REM onset (Declerck & Wauquier 1990; Hartmann 1976; Kay et al. 1976). Compared to the other classes of agents listed above, the benzodiazepines are less likely to reduce REM time (Hartmann 1968; Itil 1976).

More directly relevant to this paper, both phenelzine (an MAO inhibitor) and imipramine (a tricyclic antidepressant) have been found to be useful in reducing nightmares in patients with posttraumatic stress disorder (PTSD) (Kosten et al. 1991; Ross et al. 1994). However, the antidepressant nefazodone has no effect on REM sleep, and yet it is useful in reducing nightmares in PTSD sufferers (Gillin et al. 1999).

Benzodiazepines (e.g., diazepam) and TCAs (e.g., imipramine) suppress night terrors (Cooper 1987; Fisher et al. 1973). However, Fisher et al. (1973) found that benzodiazepines could suppress stage 4 sleep in some patients at doses that did not reduce night terrors. Also, Cooper (1987) reports a failure of diazepam in relieving night terrors in two clinical cases that, nevertheless, responded to imipramine.

A large percentage of patients with narcolepsy report vivid, frightening dreams (Lee et al. 1993). Narcolepsy is treated with TCAs and amphetamines, both of which suppress REM (Rechtschaffen & Maron 1964; Vogel et al. 1990). Since this syndrome is characterized by an atypically early onset of REM, the sleep mechanisms responsible for REM onset may also influence bad dreams. Alterations in monoamine function have been suggested in human narcolepsy. A canine model of narcolepsy implicates supersensitivity of muscarinic cholinergic receptors, specifically pontine M2 receptors (Tononi & Pompeiano 1995). TCAs are known for their anticholinergic properties (Baldessarini 1991), whereas the amphetamines have mixed effects at best on cholinergic neurotransmission (Cheney & Costa 1978). The above findings are significant since muscarinic cholinergic mechanisms are implicated in REM sleep generation and presumably dream induction as well (Baghdoyan & Lydic 1999; Hobson 1988b).

On the reverse side are agents that enhance REM and elicit bad dreams. For example, the antihypertensive reserpine enhances REM and induces nightmares in humans (Hartmann 1970). Along the lines of the previous paragraph, reserpine has been found to elevate acetylcholine, at least regionally in some species (see Palfai et al. 1986, for references).

The anxiogenic inverse agonist beta-CCE, which reduces chloride conductance at GABA/benzodiazepine receptors, substantially enhanced REM and SWS duration when administered to cats (Kaijima et al. 1984). In humans, a closely related inverse agonist, FG 7142, produced intense anxiety in two volunteers (Dorow et al. 1983). The authors did not investigate the dreams of subjects the night after taking this agent, so we know nothing about the subjective dream experiences produced by this class of agents.

Theory predicts that withdrawal from agents that suppress REM should lead to REM rebound, accompanied by an increase in the number and/or intensity of bad dreams. Consistent with this theory, Adams and Oswald (1989) found a five-fold increase in bad dreams accompanying REM rebound after withdrawal from the benzodiazepine triazolam (25 nights at 0.5 mg/night). Both Kales and Jacobson (1967) and Oswald and Priest (1965) showed that barbiturate withdrawal is accompanied by negative dream experiences. However, REM rebound following cessation of TCAs and low-potency phenothiazines (which also have anticholinergic properties) is not consistently accompanied by negative dream experiences (Kales & Vgontzas 1995).

A number of neurochemical systems have been implicated in anxiety and fear in animals, and in anxiety disorders in humans. These include (but are not limited to) norepinephrine (NE), serotonin, GABA, and cholecystokinin (CCK) (Charney & Bremner 1999). Any hypothesis attempting to link daytime anxiety with sleep-related anxiety must reconcile significant contradictions,

such as the suppression of locus coeruleus (NE) firing during REM sleep (Aston-Jones & Bloom 1981) with the apparent hyperactivity of NE systems in waking anxiety and fear (Charney & Bremner 1999). Some degree of consistency, on the other hand, appears to exist for GABAergic mechanisms in this context. Blockade of GABA-mediated chloride conductance is linked to anxiety and fear (Charney & Bremner 1999) and there is abundant evidence, albeit indirect, for a GABA-negative mechanism in REM sleep onset, perhaps one involving an endogenous inverse agonist (Wichlinski 1996). This system may occur either upstream or downstream (or both) from the REM generator mediated by acetylcholine, for which there is compelling evidence (Hobson 1992b). This cholinergically mediated REM generator may itself be inhibited by some classes of antidepressants, including the tricyclic antidepressants and the MAO inhibitors.

It is clear that REM, stage 4 SWS, negative dream experiences and daytime anxiety are dissociable, viewed either phenomenologically or pharmacologically. Suppression of REM and/or stage 4 SWS is neither necessary nor sufficient for the elimination of negative dream experiences or daytime anxiety and fear. Moreover, those agents that are successful at eliminating daytime anxiety do not always suppress negative dream experiences. Nevertheless, pharmacological inhibition of stage 4 SWS and REM sleep tends to go hand in hand with inhibition of negative dream experiences and suppression of daytime anxiety.

The mechanisms that underlie threatening dreams most likely overlap those responsible for REM and stage 4 SWS. Moreover, it is plausible that the premature onset of normal REM mechanisms, including REM related threatening dreams, may account for night terrors seen in stage 4 SWS (Arkin 1978). Although the pharmacological data are limited and do not directly address **REVONSUO**'s hypothesis, the preponderance of findings thus far are consistent with it. Nevertheless, significant questions remain:

1. What is the precise role of acetylcholine in generating dreams, especially threatening dreams? Why is it that antimuscarinic agents have hallucinatory properties when theory predicts the exact opposite? Is it due to differential muscarinic subtypes or differing responses to associated GABAergic neurons (Perry & Perry 1995)?

2. Why aren't centrally acting anticholinergic agents useful in treating both daytime anxiety disorders and parasomnias with negative content (e.g., dream anxiety attacks, night terrors)? Or have they simply not been adequately tested for these capacities?

3. How can activation of noradrenergic systems account for both daytime anxiety and nighttime threatening dreams if the locus coeruleus shuts down during REM sleep'?

It is likely that the neural mechanisms underlying threat perception – like most behaviors studied – involve a number of different neurotransmitter systems. Perhaps it is asking too much of the brain to expect it to use the same mechanisms to encode the experience of fear and anxiety in the waking state as in the sleeping state. Finally, it is useful to be reminded that anxiety and fear are not unitary states – either across or within species; therefore, attempts to generate models of brain-behavior relationships which fail to discriminate the various types of fear and types of anxiety in different species under specific conditions are bound to invite confusion (Kagan 1998).

REVONSUO has offered a provocative hypothesis for the biological function of dreaming. Much, though certainly not all, of the pharmacological data on sleep and negative dream experiences are consistent with it. Significant questions remain and further work is needed on the pharmacology of threatening dreams before a comprehensive evaluation can be undertaken on this aspect of the theory. Nevertheless, this hypothesis represents a crucial step toward an enhanced understanding of the basic mechanisms involved in dreaming and in clinical conditions associated with negative dream experiences.

Threat perceptions and avoidance in recurrent dreams

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Abstract: Revonsuo argues that the biological function of dreaming is to simulate threatening events and to rehearse threat avoidance behaviors. He views recurrent dreams as an example of this function. We present data and clinical observations suggesting that (1) many types of recurrent dreams do not include threat perceptions; (2) the nature of the threat perceptions that do occur in recurrent dreams are not always realistic; and (3) successful avoidance responses are absent from most recurrent dreams and possibly nightmares.

[HOBSON ET AL.; REVONSUO]

REVONSUO agrees with Domhoff's (1996) position that to be taken seriously, a theory of dreaming must account for the repetition dimension in dreams and asserts that his evolutionary hypothesis of the function of dreaming explains this dimension as the paradigm case of threat simulation in dreams.

Dreaming is viewed as both an organized and selective simulation of the world. A particularly well-organized form of dream content is the recurrent dream which is distinguished by its complete repetition as a remembered experience (Brown & Donderi 1986). Many kinds of dream theories (e.g., Gestaltist, object-relations, Jungian) converge in their view that recurrent dreams are associated with a lack of progress in recognizing and resolving conflicts in the dreamer's life and that the cessation of a recurring dream indicates that the conflict has been successfully dealt with. Research results support the generic clinical dream theory that recurrent dreams are associated with the presence of unresolved stressors. Studies have shown that in both late teenagers and older adults, recurrent dreams are accompanied by negative dream content in everyday dreams and that they are associated with a relative deficit in psychological well-being (Brown & Donderi 1986; Robbins & Houshi 1983; Zadra et al. 1998). Moreover, the cessation of a previously recurrent dream in adulthood is associated with a positive rebound effect on well-being (Brown & Donderi 1986).

REVONSUO cites a study (Robbins & Houshi 1983) showing that the most frequently reported theme in a student sample of recurrent dreams is one in which the dreamer is being chased (43% of all dreams). However, most of these recurrent dreams had first appeared in childhood. Zadra (1996) conducted content analyses of 110 recurrent dreams from adulthood (i.e., reported as having first occurred after the age of 18) and 53 recurrent dreams from childhood (i.e., ceased to recur before the age of 12). Although chase and pursuit dreams were the most frequently reported theme in both samples, they represented less than 15% of the adult recurrent dreams and 42% of the childhood ones. A broader category encompassing all themes in which the dreamer is in some kind of danger has been found to characterize approximately 40% of recurrent dreams (Cartwright & Romanek 1978; Robbins & Houshi 1983). What is the thematic content of the remaining 50 to 60% of recurrent dreams? The other themes include having difficulties with house maintenance, losing one's teeth, discovering or exploring new rooms in a house, driving a car that is out of control, being unable to find a private toilet, and flying. In addition, 10% of all recurrent dreams contain only pleasant emotions while another 5% are described as containing no affect (Zadra 1996). Our content analyses have also shown that about 25% of negatively-toned recurrent dreams contain emotions other than fear and apprehension (e.g., sadness, anger, confusion, guilt). Finally, the content of over 30% of all recurrent dreams is idiosyncratic and in great part unrelated to any threats. Based on the range of thematic content and affective expression represented in recurrent dreams, it may be misleading to conclude that most recurrent dreams are

dissociated from the dreamer's current concerns. It would appear that a great many recurrent dreams are not realistic rehearsals of a threatening event but rather pictorial metaphors of current concerns.

Nightmares are highly unpleasant dreams which, by definition, awaken the sleeper. Although we have not conducted a systematic content analysis of the several hundred nightmares we collected from non-traumatized adults, it is our clear impression that the overwhelming majority of nightmares contain threat perceptions but not evolutionarily adaptive threat-avoidance programs. In fact, many contain no appropriate behavioral response beyond a relatively straightforward fear reaction: dreamers awaken either while trying (unsuccessfully) to escape or at the moment they are caught or attacked. Furthermore, nightmares have been described where subjects experience the total destruction of their body. These dreams appear to simulate failure rather than any form of adaptive response. As for the nature of the threats themselves, many nightmares do not contain real-life threats based on the human ancestral environment but involve unrealistic and unusual circumstances (e.g., Zadra & Pihl 1997). Nightmares may very well be one type of dream which reveals the prime importance of threat simulation mechanisms. However, **REVONSUO**'s theory does not adequately account for the bizarre and unrealistic content of many nightmares and the fact that most do not contain reasonable and realistic adaptive behaviors. A detailed content analysis of the threatening events in nightmares as well as of the ensuing responses is needed to clarify this issue. Such an analysis is now being conducted by REVONSUO as well as our own research group.

In sum, the data indicate that many recurrent dreams, and possibly nightmares, do not include situations critical for physical survival and reproductive success. Even in those cases where they do, these dreams rarely contain constructive threat avoidance behavior or coping strategies against threats. The simulation of threat recognition during REM sleep may very well fulfill the goal of priming an amygdalocortical network to perform rapid and appropriate emotional evaluation of the potential danger. However, the second stage (i.e., rapid selection of an appropriate behavioral response and its instantiation) appears to be lacking.

That dreams can be realistic is not in doubt. Following her medal-winning performance at the 1988 Winter Olympics, Canadian figure skater Elizabeth Manley explained that she had been having difficulty with her routine, and was worried about it. The night before, she dreamed that she did the entire routine flawlessly, and when it came time to perform the routine before the judges, it was flawless enough for second place. Tholey (1991) provides evidence for the use of lucid dreaming in sports training. Real rehearsal under lucid or semi-lucid control clearly plays a role in dreams. **HOBSON ET AL.** discuss neuromodulation of the limbic-prefrontal axis (sects. 3.2.4, 3.2.7) as an important determinant of dream emotion and direction. They suggest that relative inhibition of the prefrontal cortex and activation of the limbic and paralimbic cortex leads to non-directed, emotionally valent imagery in dreams; lucid dreaming, by contrast, involves relatively less inhibition of the prefrontal cortex, relatively greater control over dream imagery, and self-awareness of dreaming. These are conditions where volition can guide imagery and produce conscious rehearsal. This is the raison-d'être of dreams, according to **REVONSUO**'s theory; we see it as one, and not the most frequent, process that occurs during dreaming sleep.

REVONSUO supposes that preliterate humans dreamed realistic threat dreams that rehearsed their control of a dangerous physical environment. With rare, athletic exceptions, the threat environment we now face is more symbolic than physical. Most threats involve language. Through metaphor and simile, language allows us to both manipulate our own mental images, and to try and control the mental images of others. Freed from goal-directing prefrontal control by the inhibitory processes associated with REM sleep, it may be that language mechanisms act on emotionally valenced memories to create the unpredictable metaphors and similes of dreams: a visual form of metaphoric and symbolic emotional free association. Many recurrent dreams and nightmares are unrealistic because they are imaginally metaphoric and free-associative; just like our own language when freed from goal-directed constraint. If we regard Elizabeth Manley's performance anxiety about figure skating in the winter Olympics as the modern equivalent of an evolutionary threat, then her confirmatory example fits neatly into REVONSUO's theory. It was literally a perceptually and behaviorally realistic rehearsal of a threatening event. REVONSUO's theory explains Elizabeth Manley-type performance dreams, but does not explain the far more numerous anxiety dreams in which the dream images are unrealistic. Perhaps REVONSUO's answer will be that, in this type of dream, the system does not become fully activated. This answer dismisses most dreams as evolutionarily useless epiphenomena that just waste time until a dreamer, under the immediate threat of losing a medal, losing a job, or losing out to a rival, realistically rehearses skating a program, confronting the boss, or carrying out a murder.

Three questions not solved by **REVONSUO**'s theory are: (1) why are realistic threat perceptions absent from many if not most recurrent dreams? (2) Why are efficient or successful avoidance responses absent from most recurrent dreams and possibly nightmares? and (3) Why is the balance of mental activity during dreaming more like metaphoric and symbolic free association, and the balance of mental activity during waking more like goal directed thought? The answer to (3) may be **HOBSON ET AL.**'s distinction between limbic and forebrain-directed dreaming; differences which lie along the neuromodulation (aminergic-cholinergic) dimension of their three-aspect model of physiological changes in dreams. According to HOBSON ET AL. the prefrontal cortex, vitally involved in the goal-directed planning of behavior and therefore in the streaming and directing of mental content towards a goal, is relatively inhibited during normal dream states. Limbic system activation of remembered mental content, unconstrained by goal needs, fills dream content with emotionally loaded but behaviorally non-sequential imagery.

Table NR3. *Models of sleep mentation necessitated by different assumptions about isomorphism and number of mentation generators*

	1-generator true	2-generator true
Isomorphism false	A. One factor mnemonic activation model (Foulkes and others) or equivalent	B. Two-factor psycholinguistic model (Casagrande and others) or equivalent
Isomorphism true	C. Covert REM sleep processes (Nielsen and others) or equivalent	D. Activation-synthesis and AIM models (Hobson, McCarley, and others) or equivalent

quent, and more intensely activated in REM sleep than they will in NREM sleep – or in the waking state for that matter. This fact, the regular association of vivid imagery with REM sleep, still remains as the legacy of last century's neurobiologically driven dream research, regardless of the convincing demonstrations of sleep mentation in NREM sleep. However, a definitive explanation of dreaming awaits a much more detailed understanding of what constitutes REM and NREM sleep, and of precisely how mind and body are inter-related as these states surge, recede, dissociate, and blend together across the sleep/wake cycle.

NOTES

1. I prefer the term "subjective experience" (cf. Helekar 1999) to "conscious experience" and especially to "subjective conscious experience" in the case of sleep mentation because the manner in which dreaming is "conscious" vis-à-vis waking consciousness has not been clearly articulated (although cf. Kahan & Laberge 1996).

2. This kind of explanation is very difficult to evaluate because verbatim mentation reports are only rarely published.

REM sleep is not committed to memory

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Abstract: We believe that this has been a constructive debate on the topic of memory consolidation and REM sleep. It was a lively and spirited exchange – the essence of science. A number of issues were discussed including: the pedestal technique, stress, and early REMD work in animals; REM windows; the processing of declarative versus procedural memory in REM in humans; a mnemonic function for theta rhythm in waking but not in REM sleep; the lack of cognitive deficits in patients on antidepressant drugs that suppress or eliminate REM sleep; the disposition of conscious (dreams) and nonconscious material of REM sleep; and finally our theory of REM sleep. Although our position was strongly challenged, we still hold that REM sleep serves no role in the processing and consolidation of memory.

VR0. Seeds of our target article

Several years ago I (**VERTES**) carried out a series of studies in behaving rats examining the relationship between the activity of cells of the pontine reticular formation (PRF) and the theta rhythm of the hippocampus. I showed that the discharge of a subset of PRF neurons was highly correlated with theta rhythm of waking and REM and subsequently that these PRF cells are directly involved in the generation of the theta rhythm.

Prior to recording, I deprived rats of REM sleep in order to increase the amount of time spent in REM sleep (i.e., REM rebound) during subsequent recording sessions. Rats were deprived of REM for 24–36 hours using the pedestal technique. Although my sole purpose for using REMD was to boost REM during recording periods, I was surprised to observe that even 24 h of REMD produced severe detrimental effects on the rats. The rats were cold and often still wet from having fallen in the water, physically fatigued from balancing on the small diameter surface of the inverted flower pot, tired from a considerable lack of sleep (mostly REM, but both SWS and REM), and generally debilitated (much like we would be without sleep for 1–2 days). Although rats are reportedly hyperactive following REMD, I found that they were essentially immobile for at least 6 h post REMD. This experience led me to question the validity of experiments examining the effects of REMD on learning and memory; that is, if rats were so severely incapacitated following this procedure how could they adequately perform on behavioral tasks following REMD?

In 1995, Peter Shiromani asked me to participate in a forum on sleep and memory for Sleep Research Society (SRS) Bulletin. I agreed and indicated that I would be taking the "con" position: no relationship between REM sleep and memory. Of eight participants in the forum, I was the only one taking this position. Possibly based on my article in SRS Bulletin, Mike Chase invited me to participate in a debate with Carlyle Smith on this same topic at an international workshop on sleep and cognitive function sponsored by the World Health Organization in Cancun, Mexico, in 1999. The debate was fruitful and further fueled my interest in the issue of memory consolidation and REM sleep. The target article by my colleague and me developed from this background.

VR1. Early REMD studies in animals, the pedestal technique, and stress

As we discussed in our target article, there was an intense interest in the role of REM sleep in memory consolidation in the 1960–1970s, interest waned in the 1980s, and has recently resurfaced. This is now a lively topic in the sleep field. As we previously indicated, our coverage of the early REMD work in animals was not meant to serve as a detailed analysis of this area, but rather to convey a general sense of the net contribution of this work to an understanding of the possible involvement of REM sleep in memory consolida-

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tion. We reached two main conclusions: (1) the early studies in animals were quite contradictory with as many reports opposing as favoring a role for REM in memory consolidation; and (2) the "stress" associated with the use of the pedestal technique for REMD was a confounding factor in many studies using this technique.

It appears that, on balance, most commentators agreed with these conclusions; that is, the early work was contradictory and much of it was methodologically flawed (**Born & Gais; Cartwright; Coenen; Feinberg; Mazzoni; Ogilvie et al.; Panksepp; Rotenberg; Siegel**). This was well put by Born and Gais: "There are obvious flaws of REM sleep suppression paradigms which do not allow for any conclusion, either pro or contra the REM sleep-memory hypothesis."

It was generally agreed that the stress associated with the pedestal technique confounded findings obtained with it. Some however, felt that we overplayed the "stress card" (**Fishbein; Greenberg; Moorcroft; Smith & Rose**) or as Greenberg stated, we laid everything at the feet of the "villain" stress.

Although several commentaries addressed this issue, perhaps the most insightful was that of **Coenen**, who has extensively examined the learning abilities of rats using the pedestal technique and has compared its use to other less disruptive forms of REMD. He essentially concludes that the detrimental effects of REMD on learning/memory primarily involve the stress of REMD procedures rather than the loss of REM, per se, pointing to a direct relationship between degree of stress and extent of learning/memory impairments. Coenen made reference to their work (van Hulzen & Coenen 1979) showing that a relatively stressful procedure (platform technique) for REMD, but not a mild one (selective hand awakening), disrupted active avoidance learning in rats. Finally, Coenen questions the use of the pedestal technique in sleep research on ethical grounds; that is, given the controversial findings obtained with this procedure, is it appropriate to continue to expose rats to it?

Fishbein argues that he ruled out stress as a possible confounding factor in pilot REMD studies in mice by the use of a modified version of the pedestal technique that produces little or no stress (Fishbein & Gutwein 1977). With this method, mice are not restricted to the pedestal as with conventional techniques, but are able to latch onto and climb about the underside of a wire mesh lid positioned above the enclosure containing the pedestal. Fishbein concluded that his demonstration that "stress-free" mice still showed learning deficits negates stress as a major contributor to the deficits. While this interpretation is possible, it seems that stress was only assessed by a single measure (open field activity), and to our knowledge, this procedure has not been used with mice outside of Fishbein's laboratory, thereby providing no independent verification of the claim that the mice are stress-free.

Smith & Rose counter the "stress argument" with the following: (1) memory deficits are present following the non-stressful pharmacological suppression of REM sleep; (2) learning impairments are seen only following four hours of REMD (i.e., REM windows); (3) animals deprived of total sleep (excluding windows) which is presumably more stressful than depriving them of four hours of REM sleep (window) show no learning/memory deficits; and (4) in some instances, REMD has been shown to improve memory. With respect to point 1, it seems unlikely that the pharmacological blockade of REM is stress free, and whatever the outcome of these studies, they do not address the question of whether stress was a major contributor to the deficits seen in reports using the pedestal technique. Regarding points 2 and 3, in our target article, we acknowledged that Smith's use of relatively short REMD periods rendered his studies less vulnerable to the stress factor, but not invulnerable. It is quite possible that even short periods of REMD are stressful to rats; that is, sufficiently stressful to disrupt learning. Regarding point 4, the findings that animals show no impairments or even improved performance following REMD would seem to indicate that in some circumstances animals can override the effects of stress.

VR2. REM windows

In our target article, we discussed three issues related to REM windows: (1) several conditions (e.g., species and even strain of animals, type of training tasks, number and distribution of training trials per session and/or per day) can affect the post-learning position of the window (i.e., shifting windows); (2) the phenomenon has only been demonstrated by Smith and colleagues; and (3) REM windows have not been described in humans.

Regarding the first point, several commentators (**Greenberg; Moorcroft; Ogilvie et al.; Smith & Rose**) questioned our "lack of appreciation" of the shifting nature of the REM window. In essence, many expressed surprise that we would expect anything less than fluctuating windows reflecting very different initial learning conditions. According to Smith & Rose, the notion of a rigid window occurring at the same time after training would be intuitively inconsistent. Borrowing from Smith & Rose, we *intuitively* have great difficulty with the notion of REM windows. For argument's sake, if REM were involved in memory consolidation, why would information be selectively consolidated in one shifting four-hour block of REM-dominated time and no other. In separate studies, Smith and co-workers reported post-training REM windows of 53–56 h (Smith & MacNeill 1993) and 48–72 h (Smith & Kelly 1988). What is the utility to animals of consolidating information 2–3 days after it has been learned? Might not the information be needed in the interim? Finally, on a practical level, the shifting nature of the windows allows for the possibility that studies showing no effect of REMD on memory could be dismissed on the grounds that they missed the window.

Regarding the second point, **Greenberg** indicated that Pearlman described REM windows several years before Smith did, but **Smith & Rose** did not dispute our statement that the effect originated with Smith. Finally, with respect to the last point, other than a brief reference by Smith & Rose, no one disputed our claim that REM windows are not present in humans.

VR3. Human studies, declarative and procedural memory

VR3.1. REM sleep is involved in procedural but not in declarative memory

A number of commentators felt that we did not do justice to (or ignored) recent human studies on memory and REM

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sleep (**Born & Gais; Schredl; Smith & Rose; Stickgold**). In our defense, a section was devoted to this, and much of the important work in this area has only recently appeared. For instance, Born & Gais admonished us for giving little attention to this work, but then cited the example that we "briefly mentioned the intriguing work of Stickgold et al. (2000b)." This report was not published at the time we submitted our target article. If our previous coverage was inadequate, we hope to rectify the situation with the present treatment.

Born & Gais point to a very useful approach to the study of memory and sleep in humans, which is to compare rates of learning following either of two equally long periods of sleep; one with high amounts of SWS (SWS-rich) and the other with high amounts of REM sleep (REM-rich). Using this approach, Ekstrand and co-workers (Barret & Ekstrand 1972; Fowler et al. 1973; Yaroush et al. 1971) and subsequently Plihal and Born (1997; 1999a) showed that there was significantly greater improvement in the recall of declarative memories following SWS-rich (first half of night) than REM-rich (second half of night) (see also Born & Gais). In addition, Born and colleagues (Plihal & Born 1997) recently reported significantly improved recall in various procedural (nondeclarative) memory tasks (mirror tracing, word stem priming) following late (REM) but not early (SWS) sleep. These findings indicate that SWS is involved in the processing/consolidation of declarative memories; REM in procedural memories.

In our target article, we described a visual discrimination task developed by Karni and colleagues that involves the identification of the orientation (horizontal or vertical) of three diagonal lines embedded in background of horizontal lines. The task has been described as a procedural learning task (Karni et al. 1994; Stickgold 1998). To date, three laboratories (Karni, Born, and Stickgold) have used this task, and have described relatively consistent findings with it. Karni et al. (1994) reported that subjects showed improved performance on the task following a period of sleep that contained REM sleep (SWS deprived) but not one lacking REM sleep (REM deprived), and concluded that REM was critical for the learning of this procedural task. In like manner, Stickgold et al. (2000b) demonstrated significant increases in amounts of SWS in the first quarter of sleep and REM in the last quarter of sleep in subjects showing improved performance on the task, while Gais et al. (2000) (see **Born & Gais**) reported a three-fold improvement in performance on the task in subjects with both SWS and REM sleep compared to those with only SWS (SWS-rich sleep). All three studies, then, show that REM sleep is directly involved in the acquisition of this procedural learning task, and together with those reviewed above, indicate that REM serves a critical role in procedural learning.

In line with the foregoing, **Smith & Rose** indicate that REM serves to consolidate some types of memories but not others; that is, according to them, "REM is not involved in declarative/explicit type memory tasks"; but serves a clear role in procedural tasks producing "impairment ranging from 20–50% in these tasks." **Stickgold** fully concurs stating: "Since all proponents of REM-dependent memory consolidation agree that REM is not involved in declarative memories such as those formed in paired associates training, the failure to observe REM-dependent consolidation may simply reflect the testing of a memory system that is not REM-dependent."

In summary, leading proponents of the memory consolidation hypothesis seem to have recently come to a very similar conclusion which is that REM sleep is involved in procedural memory but not in declarative memory.

VR3.2. Contesting the position that REM sleep is involved in procedural memory

The position that REM may be uniquely involved in procedural memory is late developing and appears to largely rest on recent work using the perceptual discrimination task of Karni and associates. In our target article, we pointed out differences between the results of Karni and Stickgold using this task. As indicated above, Born and colleagues are also now using this task in studies on sleep and memory in humans. There are, however, inconsistencies among the findings of the three groups. For instance, Born (Gais et al. 2000) described a role for SWS (early sleep) but not REM sleep (late sleep) in the task; Karni et al. (1994) reported the opposite (a role for REM but not SWS) and **Stickgold** contends that both are involved (SWS and REM sleep).

Stickgold suggests that we unfairly highlighted minor differences between his work and that of Karni using this perceptual task. We disagree, particularly with respect to one very important difference between the findings of the two laboratories; that is, the improvement (Karni & Sagi 1993; Karni et al. 1994) or lack of improvement on the task (Stickgold et al. 2000b) in the waking state.

In an initial study devoted entirely to waking, Karni and Sagi (1993) described the important findings that improved performance on the perceptual task simply required the passage of time; no gains were seen immediately after learning but only after a period of 8–10 hrs following learning. In a follow-up report, Karni et al. (1994) replicated their earlier effect (improvement over time in waking) and further demonstrated a comparable improvement with time in REM sleep. Taken together these results indicate that the acquisition of this procedural skill requires consolidation over time but this time need not be in REM sleep. In marked contrast to these findings, Stickgold et al. (2000b) clearly indicated that there was no improvement on the task during waking, commenting: "12 hours of wake behavior was inadequate to produce reliable improvement while as little as 9 hours of sleep reliably produced improved performance." We believe that this is a very important difference in the findings of the two groups (Karni and Stickgold), especially considering that they used an identical perceptual task.

In summary, the results of the three groups using the Karni perceptual task significantly conflict; that is, gains in performance on the task have been variously attributed to waking (Karni & Sagi 1993; Karni et al. 1994), SWS (Gais et al. 2000), REM (Karni et al. 1994), and both SWS and REM (Gais et al. 2000; Stickgold et al. 2000b). We believe that these differences undermine the findings of each of the groups and need to be resolved, especially since this task seems to be evolving as the standard for examining the role of sleep in procedural memory. At the very least, the above results indicate that REM serves no unique role in the acquisition of this procedural learning task.

Jones supports an involvement of REM in procedural memory, arguing that the rehearsal of motor sequences in REM could enhance efficiency of motor performance in waking. For instance, Jones suggests that the high amounts

of REM in the fetus may be used by animals to prepare them for meeting contingencies after birth such as locomotion and flight, and cites the example of the wildebeest who is fully mobile upon birth and immediately begins long treks with its mother in search of water. **Siegel,** however, describes the opposite; that is, an inverse relationship between degree of maturity at birth and amounts of REM sleep: the less mature the species, the more REM sleep; the more mature the species, the less REM sleep. Siegel refers to his recent work with the platypus and cetaceans (whales and dolphins) stating: "The immaturity of the platypus, hatching from an egg and remaining attached to its mother for an extended period after birth is consistent with its high level of REM sleep. The maturity at birth of the cetaceans, which can swim free of the mother and defend themselves immediately after birth is consistent with their low level of REM sleep."

In summary, there appears to be general agreement that REM sleep is not involved in declarative memory. The case for a REM involvement in procedural memory seems to largely rest on the recent work of three groups (**Born,** Karni, and **Stickgold**) using the visual discrimination task of Karni. As indicated, there are marked differences in the findings of these groups, which until resolved, make it difficult to evaluate the reliability of these results.

VR4. A memory processing function for the theta rhythm in waking but not in REM sleep

We surprisingly received more comments than expected on the theta rhythm. Although comments were directed to several issues related to theta, most involved our contention that theta does not serve the same function in waking and REM; that is, a mnemonic function in waking but not in REM sleep. To paraphrase **Jones**: if theta plays a role in memory consolidation, it should do so in REM as well as in waking. Commentators were divided on this issue; some supported our position (**Gottesmann; Lynch et al.; Rotenberg**), others did not (**Conduit et al.; Fishbein; Jones; Morgane & Mokler; Stickgold**). Gottesmann expressed some skepticism that a target structure could function differently in the presence of the same activity (i.e., theta) in two different states, but then described circumstances in which this happens. He cited work of his laboratory with animals with transections at the intercollicular level of the brainstem. This is a unique preparation in that animals are comatose yet show a continuous theta rhythm. Gottesmann argued that it is very unlikely that theta serves a memory processing function in comatose animals.

In further support of our position, **Gottesmann** and **Lynch et al.** argue that the theta rhythm cannot be viewed in isolation from events that occur with it and which undoubtedly affect its functional role in either waking or REM. For instance, Gottesmann points to well documented findings that monoaminergic neurons fire at their highest rate in waking and lowest in REM, indicating that the monoaminergic drive to the hippocampus is very different in waking and REM. This would seem to have obvious functional consequences for the hippocampus as well as for the role of theta in the hippocampus in waking and REM sleep. In like manner, Lynch et al. note that during waking there is an exquisite interplay among various field oscillations (theta, beta, gamma) which signal diverse aspects of the environment, and, also important, that: "the absence of this interplay of the different field potential oscillations during REM sleep may suggest that mnemonic functioning is absent, and theta is not functioning as it does during consciousness." Finally, Gottesmann points out, as did we, that even though the neocortical EEG is the same in waking and REM there are significant differences in the state of the animal in the two states, notably, differences in consciousness.

Jones has difficulty with our position that theta does not serve a mnemonic function in REM sleep. Interestingly, Jones also supports a role for REM in the consolidation of procedural memories (see earlier discussion). Procedural memory, however, is not thought to involve the hippocampus (see **Born & Gais; Stickgold**) and by extension would not likely involve the hippocampal theta rhythm. The developing view that REM is integral to the processing/consolidation of procedural memories would seem to rule out both theta and the hippocampus in these functions in REM sleep. In summary, waking and REM sleep are obviously very different states. It would seem likely that electrophysiological events common to waking and REM would serve different rather than the same functions in these two states.

VR5. Lack of effects on memory of brainstem lesions or antidepressant drugs that profoundly suppress or eliminate REM sleep

As indicated in our target article, perhaps the strongest evidence against the memory consolidation hypothesis involves the demonstration that brainstem lesions or antidepressant drugs significantly suppress or eliminate REM sleep, but do not, on the whole, adversely affect cognition/memory. These findings strongly challenge the view that REM serves a critical role in memory consolidation.

Although we described this work in considerable detail, particularly that dealing with antidepressants, few commentators addressed it. **Born & Gais** remarked, however, that the demonstration that the loss of REM with brainstem lesions does not noticeably impair cognition shows, at minimum, that "phenotypic REM is not a prerequisite for memory consolidation."

While acknowledging the general lack of adverse actions of antidepressants on learning/memory, a number of commentators argued that these compounds may selectively affect certain kinds of memory and not others; that is, they may alter types of memories that were not previously tested for in patients taking antidepressants (**Bednar; Greenberg; Panksepp; Revonsuo; Smith & Rose; Stickgold**). For example, Greenberg described anecdotal evidence from a colleague who treated one of Wyatt's patients on long-term MAOIs. According to the account, while being treated with MAOIs, the patient seemed to have no access to past meaningful emotional experiences and had no dreams (and no REM sleep), but upon removal of the MAOIs the patient became more connected with her emotional past and experienced intense dreams. From this, Greenberg concluded that REM suppression with MAOIs affects emotional but not "cognitive" memories. We question whether "emotional memories" exist separate from their obvious cognitive content.

Smith & Rose and **Stickgold** similarly argue that the apparent lack of learning/memory deficits in patients with brainstem lesions or on antidepressants may have involved

the failure to examine certain types of memory; that is, mainly procedural memory. For instance, Smith & Rose contended that by not distinguishing between different types of memories, we undercut our argument that "humans with REM-eliminating lesions or REM-depriving pharmacological treatments are normal, since this cannot be established if the subjects are not tested in tasks in which REM is known to be involved." In like manner, Stickgold states

> It is not surprising that simple cognitive and psychomotor memory tests fail to show any obvious impairment of performance after administration of drugs that disrupt REM sleep. These tests classically measure working memory and declarative memory systems that we would not expect to be affected by REM deprivation. We know of no cases in which anyone, for example, tested the effects of these drugs on complex perceptual procedural learning.

Although **Smith & Rose** and **Stickgold** seem to acknowledge that antidepressant drugs do not alter declarative memory, we are not willing to concede that they affect procedural memory. As we indicated, the widespread use of antidepressants has prompted a close examination of their possible side-effects, not only cognitive, but motor. With few exceptions, most of the commonly used antidepressants seem to have little or no adverse actions on motor functions – indirectly indicating a lack of an effect on procedural memory. We nonetheless agree with Stickgold that the possible effects of these drugs on specific procedural tasks have not been examined. This should be done.

VR6. Dreams and recent imaging studies of the brain in REM sleep

In our target article, we indicated that the sole window to the cognitive content of REM sleep is dreams. We would be more sympathetic to the position that REM serves a memory consolidating function if dreams more or less faithfully reproduced waking experiences. They obviously do not. Freud (1900) grappled with this issue in *The interpretation of dreams,* considering, but then dismissing, the possibility that dreams merely replicate waking experiences and thus serve to store them. Freud speculates that:

> It might perhaps occur to us that the phenomenon of dreaming could be reduced entirely to that of memory: dreams, it might be supposed, are a manifestation of a reproductive activity which is at work even in the night and which is an end in itself.

And continuing:

> But views of this sort are inherently improbable owing to the manner in which dreams deal with the material to be remembered. Strümpell rightly points out that dreams do not reproduce experiences. They take one step forward, but the next step in the chain is omitted, or appears in altered form, or is replaced by something entirely extraneous. Dreams yield no more than *fragments* of reproductions; and this is so general a rule that theoretical conclusions may be based on it.

Finally, Freud remarked that only in very rare instances do "dreams repeat an experience with as much completeness as is attainable by our waking memory."

Flanagan reached a similar conclusion that dreams are not a mechanism for the storage of information from waking, stating: "since we rarely dream about what we need to remember, the hypothesis that dreams themselves serve any memory enhancing function appears unwarranted."

It would seem that most proponents of the memory consolidation hypothesis would agree that waking experiences are not faithfully reproduced in dreams or committed to memory through dreams. In a twist of logic, however, that we find difficult to understand, it appears that proponents of the consolidation hypothesis seem willing to acknowledge that conscious material of REM (dreams) is not stored in REM sleep, while at the same time holding that material that never reaches dream consciousness (whatever its nature) is somehow magically processed and consolidated in REM sleep.

In our target article, we reviewed recent human imaging studies of the brain in REM sleep. As indicated, the findings show a pattern of activity in REM that is consistent with dreams; that is, a suppression of major sensory inputs and motor outputs, reflecting, as termed by Braun et al. (1997) a "closed system"; a highly activated limbic system reflecting the rich emotional architecture of dreams, and strongly dampened activity within the frontal cortex corresponding to a lack of a higher order processing and integration of information in REM sleep.

As also discussed, activity within memory processing systems of the brain appears to be attenuated in REM as evidenced by the amnesia of that state, or as **Jones** (1998) observed: "an attenuation of processes important in episodic and working memory and perhaps explaining why, unless awakened from a dream, a sleeping person has no memory of the dream." Reviewing the same (imaging) data, Hobson et al. (1998b) similarly concluded that: "some functional process, present and responsible for memory in waking is absent, or at least greatly diminished, in REM sleep." We would argue that the "absent or greatly diminished" mnemonic capacity of the brain in REM affects both conscious (dreams) and nonconscious material of REM sleep.

In line with the foregoing, **Morrison & Sanford** observed that dreaming in REM and cognitive processing in wakefulness represent very different functional states of the brain, and that the "dreaming brain" is ill-equipped to deal with the requirements of wakefulness, including memory. According to them, "one can state unequivocally that the brain in REM is poorly equipped to practice for eventualities of wakefulness through dreaming, or for consolidating into memory the complex experiences of that state."

In summary, the foregoing indicates that the brain is in a non-encoding mode in REM sleep which accounts for the amnesic quality of dreams, and in our view, amnesia for all other cognitive material, conscious or not. We find difficulty with the position that acknowledges, on the one hand, that material reaching awareness in REM (dreams) is lost to memory, while at the same time claiming that material that does not reach dream consciousness is faithfully stored in memory in REM sleep.

VR7. Our theory for the function of REM sleep

Our theory for the function of REM sleep received considerable attention; both positive (**Coenen; Lynch et al.; Ogilvie et al.**) and negative (**Blagrove; Clancey; Conduit et al.; Feinberg; Fishbein; Gottesmann; Hunt; Moorcroft; Morgane & Mokler; Panksepp; Revonsuo; Rotenberg**). We expected that the theory would be challenged, for, among other reasons, few are willing to concede that any current theory can fully account for the intricacies of REM sleep.

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Before considering specific comments on the theory, we believe it is important to address two general issues related to our theory: (1) our intent, as **Hunt** suggests, was not to discredit the memory consolidation hypothesis in order to advance our theory of REM sleep; and (2) the theory did not originate with the target article but as we indicated was an abbreviated summary/restatement of a theory previously published by Vertes (1986b).

To elaborate, our target article was not meant to serve a forum for our theory, but having completed a critical analysis of the memory consolidation hypothesis, we felt it important to present our hypothesis for the function of REM sleep. **Morgan & Mokler** stated that we did not provide necessary background material to our theory, while others indicated that we did not give due credit to earlier theories that were forerunners to ours (e.g., Cohen & Dement 1965; Ephron & Carrington 1966; Roffwarg et al. 1966; Snyder 1965). This was done in the original, complete version of the theory by Vertes (1986b).

A number of comments were directed to our statement/position that the activation of REM serves to offset the inactivation of SWS, thereby, as we proposed, preventing the brain from dwelling too long in SWS and preparing the brain for a return to consciousness. Specifically, commentators questioned our position that REM reverses the effects of SWS, pointing to well documented mismatches between SWS and REM. For instance, (1) REM is present in significantly greater amounts than is SWS in the fetus and in newborns (**Blagrove; Feinberg; Hunt**), and (2) relative amounts of SWS and REM do not precisely co-vary throughout the sleep cycle: amounts of SWS are high and REM relatively low in early sleep; the reverse in late sleep (**Moorcroft; Rotenberg**).

We address the very high amounts of REM sleep in the fetus/newborn in a theoretical treatment on the sudden infant death syndrome (SIDS) (Vertes & Perry 1993). In brief, we discussed evidence showing that the respiratory system is undeveloped in newborns and hence abnormally sensitive to the effects of hypoxia in SWS. We proposed that REM exerts pronounced stimulatory actions not only on the CNS but on the respiratory system in newborns which in part serves to prevent hypoxia-induced respiratory failure during sleep in early infancy. We cited the early work of Baker and McGinty (1977) showing that kittens exposed to hypoxic conditions for several days showed extreme irregularity and slowing of respiration in SWS that led to death unless reversed by the activation of REM sleep. Baker and McGinty (1977) drew parallels between their findings with kittens and SIDS, speculating that active sleep (AS) (REM sleep) may serve to protect human infants from SIDS. They stated: "The predominance and tenacity of the AS state in the newborn period may account for the paradoxical immunity to SIDS in the first month of life. The peak incidence for SIDS coincides with the rapid decrease in AS time between 2 and 3 months of age." In essence, then, the need for more REM in neonates may be related to potentially greater detrimental effects of SWS at this age.

With respect to the relative differences in amounts of SWS/REM throughout sleep, we proposed that REM serves two complementary functions in sleep: it offsets SWS and promotes recovery from sleep. The shorter REM periods in early sleep would seem to be of sufficient length to periodically activate the CNS in sleep, while the increasingly longer REM periods throughout sleep would progressively prepare the brain for a return to consciousness as waking approaches.

In summary, to restate our theory, we propose that the brain/CNS is strongly depressed in SWS, particularly in delta sleep, and the function of REM is to provide periodic endogenous stimulation to the brain which serves to maintain minimum requisite levels of CNS activity throughout sleep. REM is the mechanism used by the brain to ensure and promote recovery from sleep. We further believe that theories of REM should contain two important elements: (1) the function of REM should remain constant throughout the life span and (2) as a state of sleep, the function of REM should be described entirely within the context of sleep. Our theory meets these criteria.

VR8. Other evidence supporting our position

As pointed out by **Morrison & Sanford**, the case for memory consolidation in REM sleep has largely been built on the rather "tenuous correlational relationship between REM occurrence and indicators of performance" and that we presented compelling arguments questioning this temporal relationship. They discussed recent findings of their laboratory that run counter to the memory consolidation hypothesis. They reported a suppression of REM sleep in rats for 1–2 h following training on a conditioned avoidance task (fear conditioning), and concluded that these findings present problems for the position that REM is instrumental in consolidating information following learning.

In an interesting comparison with waking, **Morrison & Sanford** posed the following question: Would the constellation of REM events transferred to waking be conducive for memory consolidation in that state? That is, would a state of wakefulness characterized by a hyperactive brain, irregular respiration, tachycardia, muscle twitches, and the uncontrolled intrusion of extraneous mental images (dreams) be optimal for the consolidation of memory?

Siegel describes phylogenetic data inconsistent with the memory consolidation hypothesis. Siegel argues that if REM were involved in memory consolidation, one might expect that learning ability would be directly correlated with amounts of REM across species, but this is not the case. Siegel reviews evidence showing that: (1) humans do not have uniquely high amounts of REM sleep; (2) animals with the largest amount of REM sleep (7–8 h/day) such as the duckbilled platypus, black-footed ferret, and armadillo are not noted for their intelligence; and (3) species with the lowest amount of REM sleep (less than 15 minutes/day) such as whales and dolphins exhibit "prodigious learning abilities."

Finally, supporting our position, **Rotenberg** reports that some drugs (e.g., amphetamine) have a beneficial effect on memory but suppress REM, while others such as reserpine or neuroleptic agents have no noticeable effects on memory but increase REM sleep.

VR9. Is slow-wave sleep (SWS) involved in memory consolidation?

Several commentators raised the possibility that SWS alone (**Steriade; Morrison & Sanford**) or in combination with REM (**Blagrove; Born & Gais; Cipolli; Mazzoni; Moor-**

croft; Schredl; Smith & Rose; Stickgold) may be involved in memory consolidation. As discussed, Born & Gais described a role for SWS in declarative memory and REM in procedural memory, while Stickgold implicated both SWS and REM in procedural memory.

Commentators drew attention to the recent work of McNaughton and colleagues as well as Buzsáki implicating SWS in memory consolidation. Specifically, McNaughton and co-workers (Wilson & McNaughton 1994; Skaggs & McNaughton 1996) have shown that ensembles of hippocampal place cells tend to repeat patterns of activity of waking in subsequent episodes of SWS, while Buzsáki (1989; 1998) has proposed that hippocampal sharp waves and associated high frequency (200 Hz) bursts (ripple), that are prominent in SWS, serve to transfer information from the hippocampus to the neocortex in SWS.

Steriade pointed out that SWS is not generally viewed as a state of mentation but rather one involving a global inhibition of subcortical and cortical structures. However, recent findings of his laboratory suggest that SWS may not be the "restful" state that it is commonly thought to be. He reports that, despite an absence of external input, neocortical neurons fire at high spontaneous rates and respond to internally generated signals in SWS, indicating that they may be involved in higher order, possible mnemonic, processes in SWS.

As indicated by our title, the focus of our case is memory consolidation in REM sleep. At this time, we take no position on the possible role of SWS in memory consolidation. We do, however, believe that a role for SWS in this process is far from proven, and doing so will involve re-tracing most (or all) of the steps taken by advocates of the memory consolidation in REM hypothesis.

VR10. Conclusion

We believe that this has been a very fruitful debate on the topic of memory consolidation in REM sleep. This is obviously not the final word on this topic. We conclude with an observation of **Born & Gais** that we believe reflects the current state of affairs in this very important area. They stated: "REM sleep facilitates memory consolidation. Currently this is more a belief than a concept with convincing scientific support. Hence, **VERTES & EASTMAN**'s case against memory consolidation in REM sleep is a very timely contribution reflecting the true and persisting darkness in this area of sleep research."

Did ancestral humans dream for their lives?

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Abstract: The most challenging objections to the Threat Simulation Theory (TST) of the function of dreaming include such issues as whether the competing Random Activation Theory can explain dreaming, whether TST can accommodate the apparently dysfunctional nature of post-traumatic nightmares, whether dreams are too bizarre and disorganized to constitute proper simulations, and whether dream recall is too biased to reveal the true nature of dreams. I show how these and many other objections can be accommodated by TST, and how several lines of new supporting evidence are provided by the commentators. Accordingly TST offers a promising new approach to the function of dreaming, covering a wide range of evidence and theoretically integrating psychological and biological levels of explanation.

RR0. Overview

I am grateful for the stimulating and thoughtful commentaries on the Threat-Simulation Theory of dreaming (TST). As expected, the reactions to this reinterpretation of dreams vary greatly. I took note of the following general lines of criticism emerging in a number of commentaries: First, some commentators seem to believe that all the data presented in the target article can in fact be explained without assuming any adaptive biological functions for dreaming, or at least without accepting TST. Second, several commentators suggest that TST implies a restricted view of dreaming and, while TST may contain a partial truth, it ignores many other forms and functions of dreaming. A number of commentators also presented new data or reinterpretations of old data, that lend support to TST and lead to further empirically testable hypotheses and predictions. This, more than anything else, shows that a fruitful and productive research program on dreaming could be established on the theoretical foundations defined by TST. Even if the hypothesis would eventually have to be modified or discarded, it seems that TST will be able to contribute to the progress we are making in the scientific understanding of the dreaming mind-brain.

I will proceed in this response in the following order:

RR1. Random Activation Theory (RAT) does not explain the form and content of dreams
RR2. Alternative explanations of the data
RR3. Negative effects of PTSD and nightmares
RR4. Evidence from typical dreams
RR5. Evidence from recurrent dreams and nightmares
RR6. Other forms and functions of dreaming
RR7. Support for TST from multiple independent sources
RR8. How to test TST properly
RR9. TST and the philosophy of consciousness
RR10. Conclusion: Why do we dream?

In section 1, I discuss the Random Activation Theory and explain why it cannot account for the data in a convincing manner. In section 2, I reply to a variety of counter arguments against TST, and I explain why the alternative explanations of the data are not as convincing as those offered by TST. In section 3, I consider the challenge posed by the negative effects of post-traumatic stress disorder, and I explain why it is unlikely that ancestral humans would have suffered greatly from them. In sections 4 and 5, I discuss the studies about typical and recurrent dreams that were described in the commentaries, and I show that the results lend strong support to TST. In section 6, I reply to those commentators who argued that TST includes too narrow a view on dreaming and its functions. I emphasize that TST does not deny that other forms of dreaming exist; it is just doubtful that any of these forms are biologically functional. In section 7, I summarize the new lines of evidence supporting TST contributed by several commentators. In section 8, I try to describe briefly what kind of empirical tests and findings are most critical for testing TST, and what kind of tests would be ambiguous or irrelevant. In section 9, I briefly comment on the philosophy of consciousness on