

*ALCOHOLISM IN A SHOT GLASS*

**A POSSIBLE BRIDGE BETWEEN THE  
NEUROCHEMISTRY OF  
DREAMING & BLACKOUTS**



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# **Appendix:**

## **A Possible Bridge Between the Neurochemistry of Dreaming and Blackouts**

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## **Appendix:** **A Possible Bridge Between the Neurochemistry of Dreaming and Blackouts**

In this appendix I would like to explore and suggest a possible link between the neurochemistry of dreaming, the pharmacology of alcohol, the disruptive effect of alcohol on REM sleep, and palimpsests (blackouts). The theory of dreaming I am drawing on is Jonathan Winson's (1990), summarized in his article "The Meaning of Dreams." (See also Winson, 1985.)

There are a number of competing theories about the function and mechanisms of dreams. The best known of these are Freud's theory of the psychological meaningfulness of dreams as compromise formations, which give disguised expression to repressed,

forbidden wishes and drives; the Hobson-McCarley activation-synthesis hypothesis that dreams are the cortex's best fit interpretation of random stimulation from the brain stem and are inherently psychologically meaningless; and the Crick-Mitcheson reverse learning theory that holds that information overload from the day's experiences prevents orderly storage of relevant information into memory and that REM sleep with its concomitant dreaming erases spurious associations allowing the coding and storage of information to proceed. The later theory has been summarized as "We dream to forget." The trouble with this theory is that the dreamer has to "know" what to forget and what to retain for processing, and the theory provides no criterion for, nor mechanism of, such a discrimination. Hobson has abandoned the position that dreams are

psychologically meaningless and now holds that the order imposed on the random spikes from the brain stem is a function of the personal view of the world and of remote (childhood) memories of the dreamer.

Winson believes that dreams are meaningful and that they play a vital role in the processing of memory. He bases his theory partly on evolutionary evidence and partly on studies of the hippocampus, REM sleep, and theta rhythms. The hippocampus is a subcortical brain structure importantly involved in learning and in memory; REM sleep is the sleep stage during which dreaming occurs; and theta rhythms are brain waves that occur during survival behavior and during dreaming in most mammals. According to this theory, dreams are a nightly record basic to the mammalian

memory process, and a means by which animals form strategies for survival by evaluating current experience.

Sleep in healthy human adults normally follows a pattern of stages from hyponogogic to slow wave to rapid eye movement (REM) to slow wave to REM to slow wave to REM to slow wave to REM. A similar pattern prevails in all marsupial and placental mammals. REM sleep is under the control of the brain stem. Pontine-geniculate-occipital (PGO) electrical spikes go from the pons in the brain stem to the visual processing center in the occipital lobe of the cortex demonstrating electrochemical transmission along this path. At the same time, theta wave activity can be measured.

Theta waves are 6 cycles per second (CPS) brain waves found in the hippocampus of animals during survival behavior: predation in the cat, apprehension of danger in the rabbit, and exploration in the rat. Each of these behaviors is not rigidly genetically controlled and requires response to changing environmental conditions. Theta waves accompany the REM sleep of all of these species. They can be measured in the hippocampus where they probably play a vital role in memory processing. Egg laying mammals (monotremes) do not have REM sleep, nor do they dream. This evolutionary evidence suggests that REM sleep has survival value for higher mammals. Winson argues that theta waves reflect neural processes whereby information essential for survival (Freud's "day residues") are reprocessed into memory during REM



sleep.

The basic mechanism of memory is believed to involve *Long-Term-Potentialiation* (LTP), a process in which relatively enduring changes in nerve cells are induced by intense electrical stimulation. After tetanic (high frequency electrical) stimulation of the pathway from the cortex to the granule cells of the hippocampus, a single electrical pulse causes rapid firing, far above baseline, of the granule cell neurons. The potentiation of these neurons is considered a model for memory, a mechanism by which neural structure and function is altered. LTP is induced by activity of the N-methyl-D-aspartate (NMDA) receptor. This receptor molecule is embedded in the dendrites of the neurons of all of the above mentioned structures

and circuits. The NMDA receptor is activated by a neurotransmitter, the amino acid, glutamate. Glutamate opens a non-NMDA channel that permits the entry of sodium ions from the intracellular space depolarizing the neuron. NMDA is unique among receptor molecules because it can be further activated by glutamate opening a second channel that allows the influx of calcium ions ( $\text{Ca}^{2+}$ ).  $\text{Ca}^{2+}$  serves as a “second messenger”; its presence in the cell provides additional electric stimulation eventuating in LTP. LTP is caused naturally by theta waves activating NMDA receptors. Thus, NMDA in the presence of theta waves provides the neurochemical basis for long-term storage of biologically significant information.

It has been demonstrated that hippocampal

neurons that fire during neural mapping of survival information also fire during REM sleep, which suggests that re-mapping or re-processing is occurring. Non-REM mammals (metromes) have a large amount of neo-cortex; REM mammals have proportionately less; they do not need it to interpret, integrate, and reprocess survival information. The information being re-processed is both motor and visual, and inhibition of motor, but not eye, activity is required if REM is not to be interrupted. And, indeed, motor inhibition is characteristic of REM states.

So each species processes information vital to its survival: location of food, means of predation, or escape strategies. In REM sleep this information is re-accessed and integrated with past experience to form a

survival strategy for the future. Since animals don't have language their dreams are visual, so are those of humans. This suggests that human dreams reflect individual strategies for survival. Experiments have demonstrated that people dream about current crises and try to work out strategies for dealing with them in their dreams. Dreams are hard to interpret because the associations the neocortex brings to them are complex. These associations tend to reflect childhood experiences. Dreams give us access to and themselves constitute an infrastructure by which new experience is compared to past and integrated.

This brings us to alcohol. The pharmacology of ethanol is still poorly understood but two types of mechanisms have been demonstrated (see chapter 1).

The classical theory holds that alcohol asserts its effect by disorganizing the fatty material in the cell membrane of the neuron, which in turn disorganizes and disrupts the functioning of the receptor protein molecules embedded in those fatty layers. The trouble with this theory is that effects of alcohol on these phenomena, particularly in low doses, is too small to account for the subjective and behavioral consequences of drinking. More recent theories have demonstrated and highlighted ethanol's direct effects on nerve cell receptors, particularly on the NMDA receptor (the recognition site for glutamate that activates the second messenger system by opening a channel for  $\text{Ca}^{2+}$ ). The response of the neuron to glutamate is greatly reduced by alcohol. (The effects of alcohol on the GABA receptor also plays an important

role in intoxication.) But, as we saw above, the glutamate-NMDA-Ca<sup>2+</sup> second messenger system is the basis of LTP which in turn is the basis of memory. Further, it is well known that alcohol disrupts and disturbs REM sleep, in fact, reducing it; and that REM rebound occurs when drinking ceases. NMDA plays a vital role in the hippocampal-neocortex re-processing that occurs during REM. If alcohol blocks NMDA functioning, preventing LTP during REM, this may be the mechanism of blackouts. The kind of blackouts where there is no recall at all may be due to the lack of REM re-processing that results from alcohol's blockage of LTP by the NMDA-glutamate-Ca<sup>2+</sup> mechanism; while the kind of blackout that the drinker comes out of while still drinking may be due to blockage of primary memory processing by the same mechanism in the

hippocampus during awakeness.

Further, if Winson's dream theory is correct, we have another instance of an attempt at self-cure through the use of alcohol not only failing, but making things worse. If some people drink because they have difficulty coping, and dreaming is an important coping mechanism through which current information is fixed in memory and integrated with past experience, excessive drinking disrupts and diminishes this process. The heavy drinker is depriving himself of an important coping response and a vicious cycle is set up. A fundamental resource for understanding self and world is lost, and the anxiety engenders by that loss may well set up the next drinking episode.

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