Phylogeny refers to the sequence of events involved in the evolutionary development of an organism or trait.

Restoration Theory

Preservation & Protection Theory

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Stages of Sleep

Awake with eyes open

Awake with eyes closed

Non-REM sleep
Stage 1

Stages 2/3 (sleep spindle)

Stage 4 (slow-wave sleep)

REM sleep

50 μV 1 sec
<table>
<thead>
<tr>
<th>Stage</th>
<th>Waveform</th>
<th>Characteristic wave</th>
<th>% total sleep time</th>
<th>Associated with</th>
</tr>
</thead>
<tbody>
<tr>
<td>Waking</td>
<td><img src="image.png" alt="Waking brainwaves" /></td>
<td>Beta waves, Alpha waves</td>
<td>-</td>
<td>Awake, vigilant, eyes open, Relaxed, eyes closed</td>
</tr>
<tr>
<td>Stage 1</td>
<td><img src="image.png" alt="Stage 1 brainwaves" /></td>
<td>Alpha waves, Vertex Spike, Theta waves</td>
<td>5-10%</td>
<td>Drowsiness, decrease in heart rate, decreased muscle tension</td>
</tr>
<tr>
<td>Stage 2</td>
<td><img src="image.png" alt="Stage 2 brainwaves" /></td>
<td>Theta waves, Sleep spindles, K-complex</td>
<td>45-50%</td>
<td>Preconscious state upon waking and just before falling asleep, Dreaming characterised by 'thinking'</td>
</tr>
<tr>
<td>Stage 3</td>
<td><img src="image.png" alt="Stage 3 brainwaves" /></td>
<td>Delta waves</td>
<td>(8%)</td>
<td>Profound relaxation &amp; deep sleep</td>
</tr>
<tr>
<td>Stage 4</td>
<td><img src="image.png" alt="Stage 4 brainwaves" /></td>
<td>Delta waves</td>
<td>(12%)</td>
<td>Profound relaxation &amp; deep sleep</td>
</tr>
<tr>
<td>REM</td>
<td><img src="image.png" alt="REM brainwaves" /></td>
<td></td>
<td>5-60 minutes (20%)</td>
<td>Dreaming characterised by imagery</td>
</tr>
</tbody>
</table>
Healthy Sleep Cycle

Cycle 1  Cycle 2  Cycle 3  Cycle 4  Cycle 5

AWAKE: 10:00 PM  11:30 PM  1:00 AM  2:30 AM  4:00 AM  5:30 AM

1. Early stage
2. Deep Sleep
3. DEEP SLEEP
4. Most of the deep sleep we require happens earlier in the night
Sleep involves both neural and chemical mechanisms. Circadian rhythms are regular changes in mental and physical characteristics that occur over a 24-hour period. Most circadian rhythms are controlled by the suprachiasmatic nucleus (SNC), a tiny set of brain structures in the hypothalamus the size of a pinhead and containing just 20,000 neurons.
Signals from the SCN travel to several brain regions, including the pineal gland, which responds to light-induced signals by switching off production of the hormone melatonin. The SCN also affects functions which are synchronised with the sleep/wake cycle, including body temperature, hormone secretion and changes in blood pressure.
Forebrain areas key to the neuropsychology of dreaming

Prefrontal cortex:
- Ventromedial
- Dorsolateral

Anterior limbic structures:
- Amygdala, anterior cingulate, ventral striatum

Posterior cortices:
- Inferior parietal
- Visual association

Thalamocortical control of NREM sleep rhythms; EEG activation and deactivation

Hippocampal-cortical control of memory consolidation

Origin and expression of circadian rhythms
Hypothalamic nuclei:
- Suprachiasmatic
- Subparaventricular
- Dorsomedial

Diencephalic control of sleep onset
Hypothalamic nuclei:
- Ventrolateral preoptic
- Lateral
- Tuberal mammary
- Basal forebrain

Pontine control of the REM-NREM cycle
Mesopontine nuclei:
- Laterodorsal tegmental
- Pedunculopontine
- Dorsal raphe
- Locus coeruleus
<table>
<thead>
<tr>
<th>Brain Structure</th>
<th>Brain Area</th>
<th>Neurotransmitter</th>
<th>Behavioural Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basal forebrain</td>
<td>Cerebral cortex</td>
<td>2 transmitters: ACh, GABA</td>
<td>Excites the thalamus and cortex, switches sleep from NREM to REM; Inhibits thalamus and cortex</td>
</tr>
<tr>
<td>Pontomesencephalon</td>
<td>Reticular system, brain stem</td>
<td>ACH, glutamate</td>
<td>Increases cortical arousal</td>
</tr>
<tr>
<td>Dorsal raphe and pons</td>
<td>Brain stem</td>
<td>5-HT</td>
<td>Interrupts REM sleep</td>
</tr>
<tr>
<td>Locus coeruleus</td>
<td>Brain stem</td>
<td>NA</td>
<td>Suppresses REM sleep; increases information storage during wakefulness</td>
</tr>
<tr>
<td>Hypothalamus</td>
<td>Midbrain</td>
<td>Histamine</td>
<td>Increases arousal</td>
</tr>
</tbody>
</table>

1) A basal forebrain system in the ventral frontal lobe can induce SWS by releasing GABA into the tuberomammillary nucleus, producing synaptic and behavioural inhibition. Damage to the basal forebrain can result in a decrease in arousal and attention, as well as impairment of learning and memory.
2) A brainstem reticular system which activates the forebrain to wakefulness and dreaming. Neurons in the pontomesencephalon generate impulses of their own, sending their axons to the thalamus and the basal forebrain, where ACh and glutamate are released to produce widespread cortical arousal.
3) A pontine system which triggers REM sleep and paralysis.

4) A hypothalamic system which affects all three systems to determine sleep/wakefulness, via inhibitory/excitatory effects. At least two hypothalamic pathways stimulate attention by releasing the neurotransmitter histamine. One of the side effects of antihistamine drugs is drowsiness as they interfere with histamine neurotransmitter systems.
Why Do We Dream?

• With the exception of the dolphin and echidna (spiny anteater) all mammals display REM sleep, as do birds. Hence it is thought that REM sleep evolved in a common ancestor of both mammals and birds.

• Stage 2 sleep is also associated with dreaming, however dreams during this stage are more concerned with thinking through problems than the more realistic, visual dreams associated with REM sleep.
Why Do We Dream?

• REM sleep causes key neurotransmitters affected – NA, 5-HT, HA (monoamines) – active during waking, to stop discharging, allowing receptor systems to regain sensitivity

• Monoamines also play a role in neural rewiring in response to new experiences. Turning them off may prevent changes in brain connections during REM
Dreams are a by-product of physiological processes that occur during REM sleep. They are the result of essential activity of groups of neurons in the brain. Some cognitive processes appear to function whilst we sleep, although related regulatory circuits which usually limit thought abstraction and illogical thought progression are not active. Dreaming appears to be the neural sum of sensory and motor neurons, and the flow of thought unhindered by mechanisms such as those above.
Dominant sleep since the late 1970s, this postulates that the characteristic formal aspects of dreams correspond to characteristic aspects of physiological activation during REM sleep. There is cortical arousal during REM sleep, however there is no external sensory input and little frontal lobe activity. Dreams seem illogical because of this lack of input from the planning, reasoning, thinking frontal lobe. There is no meaning to dreams - they are simply the brain's reaction to random and chaotic neurophysiological and neurochemical events which occur during sleep.
Many researchers have proposed that REM sleep plays an important role in memory consolidation. Results from animal studies are inconsistent and methodologically confounded; human studies of psychopharmacologically and neuropathologically-induced REM impairments are not associated with memory deficits. The 3 major classes of antidepressant drugs (MAOIs, TCAs and SSRIs) all significantly suppress REM sleep. Despite this, there is no apparent disruption of learning or memory.
Vertes & Eastman (2000) have proposed that the primary function of REM sleep is to provide periodic endogenous stimulation to the brain. This serves to maintain requisite levels of CNS activity throughout sleep. REM is thus the mechanism used by the brain to promote recovery from sleep rather than a consolidation mechanism for learning and the storage of memories.
There is a high correlation between the REM stage of sleep and dreaming. Do brainstem mechanisms which generate REM also generate dreaming?

REM sleep and dreaming may be **doubly dissociable states**. REM can occur without dreaming and dreaming can occur without REM. REM may be controlled by cholinergic brainstem mechanisms and dreaming by dopaminergic forebrain mechanisms.
DA agonists increase the frequency, vivacity and duration of dreaming without similarly affecting the frequency, intensity and duration of REM sleep. This observation, together with the equally important fact that damage to ventromesial frontal fibres obliterates dreaming but spares the REM cycle, suggests a specific dopaminergic dream-on mechanism which is dissociable from the cholinergic REM-on mechanism.
Sleep disorders include problems with falling or staying asleep, falling asleep at inappropriate times, problems with adhering to a regular sleep schedule and sleep disruptive behaviours.
Over 100 sleep disorders exist:

• insomnia, an inability to sleep or remain asleep through the night

• obstructive sleep apnoea, when breathing stops intermittently during sleep

• narcolepsy, characterised by daytime sleep attacks in spite of adequate sleep at night

• night terrors, abrupt awakening from sleep with fear and fairly common in children

• sleepwalking, one of the most common sleep disorders in children
Narcolepsy

Narcolepsy - an uncontrollable, overwhelming desire to sleep during the day and may be described as an intrusion of REM sleep into the waking state.

The 4 classic symptoms of the disorder are:

• excessive daytime sleepiness
• cataplexy
• sleep paralysis
• hypnagogic hallucinations
Narcolepsy

Dobermans and Labrador retrievers with narcolepsy have a defective HO gene which impairs HO activity by inactivating development of an HO-regulating protein. Narcoleptic mice have been bred which lack the HO gene. What is the behavioural consequence of this lack of HO? The mice suffer narcoleptic attacks. Exactly how
During wakefulness, hypocretin activity excites noradrenergic, histaminergic and serotonergic neurons, which give rise to enhanced cortical activity and arousal. Slow-wave sleep (SWS) is characterised by synchronous intrinsic cortical activity, and most subcortical afferents show reduced activity. During rapid eye movement (REM) sleep, low hypocretin activity results in the disinhibition of REM-on cholinergic neurons.

DRN, dorsal raphe nucleus; LC, locus coeruleus; PPT, pedunculopontine tegmental nucleus; PRF, pontine reticular formation; TMN, tuberomammillary nucleus.
The HO system interacts with other areas of the brain during waking and sleeping is one current area of interest, as is the relationship of HO to human narcolepsy. HO genes in narcoleptic humans appear mostly normal, and rather than a defective gene being the cause, it may be that disease or some form of induced brain damage might impair HO function. HO-replacing therapies may produce future treatments for the disorder.