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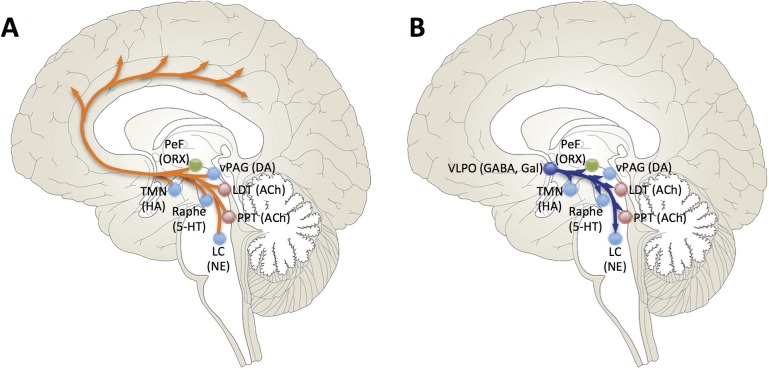
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ASSIGNMENT

Question 1; Discuss the Physiology of sleep.

As depicted in Figure A, the cortical activation necessary to maintain wakefulness is supported by an extensive network of subcortical structures and pathways. Major neurochemicals of this “ascending arousal system” include excitatory norepinephrine arising from the locus ceruleus (LC), serotonin from the midline raphe nuclei, histamine from the tuberomammillary nucleus, dopamine from the ventral periacqueductal grey matter, acetylcholine from the pedunculopontine tegmentum, and the laterodorsal tegmentum of the pons and orexin from the perifornical area. Despite their apparent redundancy, normal behavioural functioning may require all of these arousing systems. For example, it is now clear that narcolepsy results from a selective loss of orexin-releasing neurons in the forebrain, accounting for the excessive daytime sleepiness, fragmented sleep, and cataplexy (sudden muscle weakness without loss of consciousness) associated with this disorder.



FIGURE; Brain networks regulating sleep and wakefulness. Panel A depicts key elements of the ascending arousal systems, with diffuse excitatory projections to the cortex. Panel B shows pathways arising from the hypothalamus that inactivate the ascending arousal system during sleep. ACh, acetylcholine; DA, dopamine; GABA, gamma amino-butyric acid; Gal, galanin; HA, histamine; LDT, laterodorsal tegmentum; NE, norepinephrine; ORX, orexin; PeF, perifornical region; PPT, pedunculopontine tegmentum; TMN, tuberomammillary nucleus; vPAG, ventral periaqueductal gray matter; 5-HT, 5-hydroxytryptamine.

Initiation and maintenance of sleep require suppression of activity in the ascending arousal systems. This is accomplished by inhibitory neurons of the ventrolateral pre-optic area (VLPO; Figure B), which remain active throughout sleep. The molecular “triggers” that activate the VLPO and initiate sleep onset have not been fully defined, but a substantial body of evidence points to extracellular adenosine as a candidate. Adenosine accumulates in basal forebrain during wakefulness and diminishes with ongoing sleep. Adenosine receptors are expressed in the VLPO and adenosine activates VLPO neurons in vivo, making it a reasonable candidate for the “sleep switch.” Caffeine and theophylline are potent adenosine receptor antagonists, which may form the basis for their well-known alerting effects. Despite this evidence, it is almost certain that other molecules also play important signalling roles controlling the initiation and maintenance of sleep. The monoaminergic arousal centres project to the VLPO and may serve to inhibit its activity. This creates the concept of “flip-flop” control of behavioural state, in which, at any given time, activity of either arousal-producing or sleep-producing neurons dominates and suppresses the other. In addition, the VLPO receives important circadian modulation from the suprachiasmatic nucleus—the central circadian clock.

Sleep itself is not a homogenous process. There exist two fundamentally distinct types of sleep: rapid eye movement (REM) sleep, which is associated with active dreaming, and non–rapid eye movement (NREM) sleep. Switches between NREM and REM sleep appear to be controlled by reciprocal inhibition between monoaminergic neurons and a specific subset of cholinergic neurons within the brainstem. These “REM-on” cholinergic neurons exhibit reciprocal inhibitory connections to noradrenergic (LC) and serotonergic (raphe) neurons. When REM sleep is triggered, REM-on cholinergic neurons become maximally active, while noradrenergic and serotonergic neurons become virtually silent. The switching between activity and inhibition of these neurons results in a characteristic cycling between NREM and REM during the sleep period.

TIMING

Circadian clock

Sleep timing depends greatly on [hormonal](https://en.wikipedia.org/wiki/Hormone" \o "Hormone) signals from the circadian clock, or Process C, a complex neurochemical system which uses signals from an organism's environment to recreate an internal day–night rhythm. Process C counteracts the homeostatic drive for sleep during the day (in [diurnal](https://en.wikipedia.org/wiki/Diurnality" \o "Diurnality) animals) and augments it at night. The [suprachiasmatic nucleus](https://en.wikipedia.org/wiki/Suprachiasmatic_nucleus" \o "Suprachiasmatic nucleus) (SCN), a brain area directly above the [optic chiasm](https://en.wikipedia.org/wiki/Optic_chiasm" \o "Optic chiasm), is presently considered the most important nexus for this process; however, secondary clock systems have been found throughout the body.

An organism whose circadian clock exhibits a regular rhythm corresponding to outside signals is said to be entrained; an entrained rhythm persists even if the outside signals suddenly disappear. If an entrained human is isolated in a bunker with constant light or darkness, he or she will continue to experience rhythmic increases and decreases of body temperature and melatonin, on a period which slightly exceeds 24 hours. Scientists refer to such conditions as [free-running](https://en.wikipedia.org/wiki/Free-running_sleep" \o "Free-running sleep) of the circadian rhythm. Under natural conditions, light signals regularly adjust this period downward, so that it corresponds better with the exact 24 hours of an Earth day.

The circadian clock exerts constant influence on the body, effecting [sinusoidal](https://en.wikipedia.org/wiki/Sine_wave" \o "Sine wave) oscillation of [body temperature](https://en.wikipedia.org/wiki/Thermoregulation" \o "Thermoregulation) between roughly 36.2 °C and 37.2 °C. The suprachiasmatic nucleus itself shows conspicuous oscillation activity, which intensifies during subjective day (i.e., the part of the rhythm corresponding with daytime, whether accurately or not) and drops to almost nothing during subjective night. The circadian pacemaker in the suprachiasmatic nucleus has a direct neural connection to the [pineal gland](https://en.wikipedia.org/wiki/Pineal_gland" \o "Pineal gland), which releases the hormone [melatonin](https://en.wikipedia.org/wiki/Melatonin" \o "Melatonin) at night. [Cortisol](https://en.wikipedia.org/wiki/Cortisol" \o "Cortisol) levels typically rise throughout the night, [peak in the awakening hours](https://en.wikipedia.org/wiki/Cortisol_awakening_response" \o "Cortisol awakening response), and diminish during the day. Circadian prolactin secretion begins in the late afternoon, especially in women, and is subsequently augmented by sleep-induced secretion, to peak in the middle of the night. Circadian rhythm exerts some influence on the night-time secretion of growth hormone.

The circadian rhythm influences the ideal timing of a restorative sleep episode. Sleepiness increases during the night. REM sleep occurs more during body temperature minimum within the circadian cycle, whereas [slow-wave sleep](https://en.wikipedia.org/wiki/Slow-wave_sleep" \o "Slow-wave sleep) can occur more independently of circadian time.

The internal circadian clock is profoundly influenced by changes in light, since these are its main clues about what time it is. Exposure to even small amounts of light during the night can suppress melatonin secretion, and increase body temperature and wakefulness. Short pulses of light, at [the right moment](https://en.wikipedia.org/wiki/Phase_response_curve" \o "Phase response curve) in the circadian cycle, can significantly 'reset' the internal clock. Blue light, in particular, exerts the strongest effect, leading to concerns that [electronic media use](https://en.wikipedia.org/wiki/Electronic_media_and_sleep" \o "Electronic media and sleep) before bed may interfere with sleep.

Modern humans often find themselves desynchronized from their internal circadian clock, due to the requirements of work (especially [night shifts](https://en.wikipedia.org/wiki/Shift_work" \o "Shift work)), long-distance travel, and the influence of universal indoor lighting. Even if they have sleep debt, or feel sleepy, people can have difficulty staying asleep at the peak of their circadian cycle. Conversely they can have difficulty waking up in the trough of the cycle. A healthy young adult entrained to the sun will (during most of the year) fall asleep a few hours after sunset, experience body temperature minimum at 6 a.m., and wake up a few hours after sunrise.

QUESTION 2; Discuss the role of the basal ganglia in coordinating movement

The **basal ganglia**, or basal nuclei, are a group of [subcortical structures](https://www.kenhub.com/en/library/anatomy/subcortical-structures-anatomy) found deep within the white matter of the [brain](https://www.kenhub.com/en/library/anatomy/cerebral-cortex). They form a part of the [extrapyramidal motor system](https://www.kenhub.com/en/library/anatomy/extrapyramidal-system) and work in tandem with the pyramidal and [limbic systems](https://www.kenhub.com/en/library/anatomy/limbic-system).

The basal ganglia consist of five pairs of nuclei: caudate nucleus, putamen, globus pallidus, subthalamic nucleus, and substantia nigra.

The function of the basal ganglia is to fine-tune the voluntary [movements](https://www.kenhub.com/en/library/anatomy/types-of-movements-in-the-human-body). They do so by receiving the impulses for the upcoming movement from the [cerebral cortex](https://www.kenhub.com/en/library/anatomy/cortical-cytoarchitecture), which they process and adjust. They convey their instructions to the [thalamus](https://www.kenhub.com/en/library/anatomy/thalamus), which then relays this information back to the cortex. Ultimately, the fine-tuned movement instruction is sent to the [skeletal muscles](https://www.kenhub.com/en/library/anatomy/histology-of-skeletal-muscle) through the tracts of the pyramidal motor system. Basal ganglia mediate some and other higher cortical functions as well, such as planning and modulation of movement, memory, eye movements, reward processing, and motivation.

The basal nuclei modulate motor function through various [pathways](https://www.kenhub.com/en/library/anatomy/direct-and-indirect-pathways-of-the-basal-ganglia) in order to initiate, terminate, or modulate the extent of the movement.

These are the following:

* Direct pathway: which is responsible for the initiation of the movement. In order to make this happen, the direct pathway funnels the information from the striatum to GPi/SNr via GABAergic inhibitory projections. This inhibition releases the firing from the thalamocortical neurons to initiate the movement.
* Indirect pathway, which has a net excitatory effect on the same structures. The neurons from the external part of globus pallidus send inhibitory fibers to the subthalamic nucleus instead of sending directly to the thalamus (hence its name “indirect”). From the subthalamic nucleus, neurons send their axons to the internal part of the globus pallidus and reticular part of the substantia nigra and then continue as the direct pathway with GABAergic inhibitory neurons to the thalamus and glutamate excitatory efferents to the cortex. So, functionally, the striatum inhibits the external globus pallidus, and that causes disinhibition of the subthalamus.
* Hyperdirect pathway, via which the internal part of globus pallidus and reticular part of the substantia nigra receive strong excitatory signals from the cortex directly through STN and has a shorter conduction time compared to the direct and indirect pathways. The hyperdirect pathway consists of neurons projecting from the cortex directly to the subthalamic nucleus (STN), skipping the striatum. Therefore, the glutamatergic excitatory neurons of the STN can then excite the GPi/SNr thus suppressing thalamic activity on the cerebral cortex and increasing inhibitory influences on the upper motor neurons.

Considering the conduction path and time, we can say that the hyperdirect and indirect pathways make clear initiation and termination of the selected motor program, while at the same time canceling other competing motor programs.