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Sleep in the 21st Century: Impacts of Sleep Dysregulation on Health and Well-Being

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Sleep in the 21st Century:

Impacts of Sleep Dysregulation on Health and Well-Being

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Majors: Physiology and Psychology

Honors Senior Project

Sleep has always been a topic of great interest. Scientists have just begun to uncover the mysteries of this essential aspect of our lives; but much remains unknown. As humans have evolved, so have our sleep patterns. Throughout history, there has been a progressive decrease in prioritizing sleep in our lives, coupled with an increased emphasis on efficiency and productivity in society. This study pursues various aspects of sleep patterns, including circadian and homeostatic rhythms, the measurement and importance of sleep, and the impacts of poor sleep schedules. Recent changes and ideas concerning sleep habits, as well as their effects, are addressed, in an effort to examine the overall purposes and benefits of a good night's rest.

Normal Monophasic Sleep Cycles

Humans spend about 1/3 of their life asleep; a typical sleep pattern consists of one rest period lasting on average 6-8 hours each night. Louise M. Paterson discusses this and various other aspects of a normal sleep cycle in *The Science of Sleep*¹. A typical monophasic sleep episode involves a single rest period, and goes through a specific pattern of phases, which makes up an individual's "sleep architecture" (Figure 1). This pattern begins in the Non-Rapid Eye Movement (NREM) phase, which includes Stages 1, 2, 3, and 4. During Stage 1 (S1) an individual will begin to feel drowsy, and fall into a light state of sleep that often involves brief moments of wakefulness, muscle jerks and twitches, and slow rolling of the eyes. After passing into Stage 2 (S2), breathing and heart rate begin to slow, and muscle tone in the body decreases. The deepest stages of sleep occur during Stages 3/4 (S3/S4), also referred to as delta sleep or slow wave sleep (SWS). During S3/S4 it is much harder to wake an individual. The amount of time spent in SWS depends in part on the amount of time a person has spent awake

before falling asleep. Slow wave sleep is important in cognitive maturation², physical restoration of the brain³, and proper immune functioning⁴.

Following NREM, the individual will move into Rapid Eye Movement (REM) sleep. The first round of REM sleep usually happens around 60-90 minutes after sleep onset. The individual will very suddenly enter this stage of sleep, which is also referred to as paradoxical sleep due to the similarities between brain waves of individuals who are awake and those who are in REM sleep. During REM the body has a loss of skeletal muscle tone, and rapid eye movements can be observed. This is the stage where the most vivid and intricate dreams happen, although dreams are also able to occur during other stages throughout rest. REM sleep is thought to be important for growth and development of the nervous system², as well as memory consolidation and mood regulation⁵.

A complete sleep cycle is referred to as passing through NREM stages followed by REM sleep. A typical cycle lasts about 90 minutes and an individual will usually go through 3-6 cycles per night. Throughout the course of a night, the components of these cycles begin to change. At the beginning of the night an individual will systematically go through each stage of NREM (S1-S4) until they reach SWS, then will ascend back up to a lighter sleep, followed by short episodes of REM sleep. But as the cycles continue, the period of time spent in NREM gradually decreases, with the cycles containing less deep sleep and more time spent in REM sleep. In the last half of a sleep episode NREM periods usually only descend to stages 1 and 2 before switching back to REM activity. Because the brain is so active during REM, this stage is seen as a transition into consciousness. This explains why more REM sleep can be observed as an individual gets closer to waking up (Figure 1).

Many factors impact sleep, including age and gender (Table 1). Young infants typically sleep more than older individuals do, and their patterns are initially polyphasic, meaning they have multiple sleep episodes throughout the day and night.⁶ Eventually they begin to grow accustomed to a 24-hour schedule during childhood. Children, adolescents, and young adults require more sleep than older adults. It is not unusual for a young individual to still feel very tired while getting 8 hours of sleep per night.⁷ There is also a phase delay experienced during adolescence, which is observed in the “night-owl” behavior of this age group, causing them to stay up and sleep-in later. The phase shift moves back to an earlier schedule once adolescence has ended in the early 20s.⁸ Elderly adults over the age of 65 tend to experience a phase advance, causing them to go to bed and get up earlier, and typically sleep less overall during the nighttime.⁹ These older individuals often experience many arousals during the night, but make up for some lost sleep with naps throughout the day (Figure 2).¹

Examination of gender in sleep show many differences in the sleep architecture between men and women. Women often experience changes in their pattern during puberty, menstruation, and later in life during menopause due to the impacts of various hormones.¹⁰ During these periods, women are at an increased risk to develop a number of clinical sleep disorders, including Obstructive Sleep Apnea, Restless Leg Syndrome, and insomnia.¹¹ Women also tend to experience deeper sleep than men, and spend significantly more time in SWS.¹² In agreement with this, a meta-analysis of sleep parameters over the life course showed that amount of time spent in REM sleep decreases with age, and men spend more time in REM sleep than women.¹³

Measuring Sleep

Sleep is measured and examined through a number of different procedures, including polysomnography (PSG), multiple sleep latency tests (MSLT), maintenance of wakefulness tests (MWT), and actigraphy.¹⁴ Actigraphy records gross motor movements in order to display circadian rhythm (Figure 3). It is useful in observing sleep patterns in individuals who struggle with insomnia, circadian rhythm disorders, and excessive fatigue, without the need for a time-consuming and expensive PSG. In MSLT, participants are asked to nap multiple times over several hours, with two hours of wakefulness between each napping session. The time between lights out and falling asleep is measured each time in order to calculate the mean sleep latency. MSLT is often used when testing for narcolepsy, which is diagnosed when multiple episodes of sleep onset REM (REM occurring within 15 minutes of falling asleep) are observed during the napping sessions. MWT is utilized when measuring how long an individual is able to stay awake while sitting in a comfortable position in a dark room, and consists of five 20-minute sessions. Most normal sleepers are able to stay awake for at least 8 minutes before falling asleep. Falling asleep more quickly may indicate abnormal sleep patterns. PSG involves a number of measurements, including those captured by an electroencephalogram (EEG), electrooculogram (EOG), electromyogram (EMG), and electrocardiogram (ECG), as well as several respiratory measurements such as blood oxygen saturation, airflow, and respiratory effort.¹⁴ These methods capture a wide variety of information about an individual over the course of one night's sleep: the EEG measures brain activity, EOG monitors eye movement, EMG captures muscle activity, and ECG monitors the electrical activity produced in the heart.^{1,15} The respiratory measurements are used mainly in the diagnosis of sleep disorders that impact

breathing, such as Obstructive Sleep Apnea.¹⁵ The various measurements involved in polysomnography are utilized in clinical settings when attempting to diagnose and treat a multitude of sleep disorders and disturbances.

One of the more important tests included in PSG is the EEG. This test is explained in great depth in *Recording and Quantifying Sleep*¹⁴, and the important topics are discussed here. An EEG is a physical record of brain activity that can be recorded and graphed. It is able to pick up microscopic electrical potentials created by neurons in the brain. The graph produced by these measurements is used to identify the various stages of sleep (Figure 4). When an individual is awake, the patterns recorded on an EEG are called an alpha and beta rhythms. Alpha rhythms can be seen best when an individual is awake and active but also relaxed, and are measured by electrodes placed on the posterior part of the head. The frequency of an alpha rhythm ranges from 8-13 Hz and reaching amplitudes up to 200 microvolts. The rhythm is destroyed when a person begins to concentrate on any visual stimuli or think about more difficult topics. Once alertness and logic are activated, beta activity is observed. This is seen throughout the brain, and occurs at a higher frequency and lower amplitude than alpha bands, with beta frequency ranging from 13-30Hz (Figure 5).

Once an individual begins to fall asleep, the bands on the EEG change dramatically. During the first stage of NREM sleep, when an individual is beginning to fall into a light sleep, there is a gradual shift from alpha to theta activity. The frequency of theta bands run from 4-8Hz and are often seen on the EEG along with vertex sharp waves in S1 stage. A vertex sharp wave is observed when there is a sharply pointed wave of higher amplitude on the EEG. After moving into stage 2, K-complexes, sleep spindles, and positive occipital sharp transients of

sleep (POSTS) are found on the EEG. A K-complex occurs when a vertex sharp wave is followed by high amplitude but low frequency delta wave, which forms the shape of a K on the graph. Sleep spindles often occur after K-complexes and can be identified by short bursts of 12-14Hz activity that occurs intermittently throughout stage 2. POSTS are low amplitude triangular shaped waves that appear on the EEG, originating from signals in the occipital region of the brain. As the sleep cycle moves on to slow wave sleep, the frequency of the bands decline, leading to the appearance of delta activity at frequencies lower than 4Hz.

After the various stages of NREM, the appearance of REM sleep on an EEG is distinctively different. While in REM sleep, a person's brain activity is very similar to being awake or in S1 of NREM. The amplitude is much lower, with mixed levels of frequencies interspersed throughout the graph. Rapid eye movement is identified based on regions of high amplitude activity coming from anterior brain electrodes. It is easy to see how an individual could move from this REM state into being awake with relative ease based on the similarity in EEG graphs during these distinct phases.

In addition to PSG, other clinical parameters are measured when attempting to quantify sleep quality.¹⁶ Percentages of time spent in each sleep stage is calculated based on PSG data, and is used to examine the depth of sleep and whether sleep architecture is imbalanced in any way. Sleep efficiency measures the amount of time actually asleep while in bed, and sleep onset latency shows how long it takes for an individual to fall asleep after lights out. Arousals and wake after sleep onset are utilized to show how much disturbance an individual experiences while sleeping. The number of arousals during a sleep episode are totaled, while the amount of wake after sleep onset is expressed as a percentage. Poor sleep efficiency, long

sleep onset latency, and high amounts of arousals and time awake after sleep onset are all indicators of low quality of sleep, and may be used in a clinical diagnosis.

Regulation of Sleep

Although the main regulation of sleep is determined by areas of the brainstem and hypothalamus, there are several other contributing biological processes that help the body to regulate both when we sleep and for how long.¹⁷ The main model that has been used for the past three decades to understand the regulation of our sleep-wake cycle is called the two-process model.^{18,19} This original model is divided into two independent categories: a circadian process (Process C), and a homeostatic process (Process S) (Figure 6). The main genes associated with creating circadian rhythmicity are the transcription factors CLOCK and BMAL1 genes, which form a dimer and are able to bind to the promoter elements PER1, PER2, PER3 and CRY genes. The PER and CRY genes demonstrate negative feedback on the transcription factors, allowing for a specific circadian oscillation.²⁰ The main genetic mechanism involved in the homeostatic process involves adenosine receptors. Adenosine concentrations in the brain accumulate throughout the day, which decrease wakefulness and cause increased propensity for sleep.²¹ A common antagonist of adenosine receptors is caffeine, which is used as a stimulant of the central nervous system to decrease adenosine uptake, thereby increasing arousal. The interaction between these two processes explains our propensity to sleep at specific times while being awake at others.

The circadian process regulates our bodily rhythms to a one-day (approximately 24-hour) routine. It is generated in our brains by a group of cells in the suprachiasmatic nucleus (SCN) of the hypothalamus, which is the main timekeeper for our entire body; all other systems

synchronize their rhythm to that of the SCN. The SCN is able to adjust to a 24-hour rhythm due to light/dark cues received by the retinohypothalamic tract.¹⁹ Environmental cues that influence our circadian rhythm are referred to as zeitgebers, and include temperature, light, exercise, food, and social influences. An individual's chronotype is their propensity to sleep and feel alert at certain times of the day and night. Different chronotypes also impact circadian rhythmicity by causing specific genetic predisposition toward being a morning-type vs. an evening-type individual.²² Although an individual is predisposed to a particular sleeping pattern, circadian rhythms are constantly shifting due to physical and social cues. In the absence of zeitgebers, the SCN is able to independently 'free-run' and keep the body in a daily cycle; however, the biological cycle of our body is actually slightly longer than 24 hours, so eventually this free-running pattern would deviate from that of society.²³ When the circadian process is displayed visually (Figure 6), it creates an oscillation graph, where the higher numerical values on the y-axis correspond to the time when we most strongly need sleep and the lower values represent the periods when we are biologically awake.²⁴

The homeostatic process attempts to maintain a constant stable condition in the body. Throughout the day, homeostatic pressure gradually increases until we go to sleep, at which point the homeostatic pressure quickly declines back to a baseline level. This process is highly dependent on the amount of time we spend awake before attempting to sleep.^{18,19} Sleep deprivation results in a sleep debt, and we may have to rest longer than usual. However, if we sleep in very late one morning, we may have difficulty falling asleep at our usual bedtime because we have not spent as much time awake as normal.

The two-process model states that Process C and S work independently of one another, but together help determine our sleep-wake cycle. The two processes are able to compensate for one another, which allow humans to be able to function even when sleep conditions are inadequate.^{18,19} For example, if someone stays up late studying for an exam they will experience a sleep debt the next day, along with high homeostatic pressure. However, because the circadian rhythm is still intact, the individual is able to cope. Another example is night shift workers. Although their schedules deviate from the circadian rhythm, homeostatic pressure allows them to remain in this cycle, leading them to sleep during the day. This flexibility in the process allows humans to deviate from a “typical” schedule, and to be influenced by their external environment.

Since the original proposal of the two-process model, it has been widely used to explain a number of phenomena that occur in mammalian sleep patterns. However, recent research provides evidence that the circadian and homeostatic processes are not wholly independent. Initially, it was proposed that sleep occurs when the homeostatic pressure reaches the upper limit of the circadian process, and vice versa for waking up. The overlap of these two systems resulted in a propensity toward sleep or wakefulness, but the processes themselves did not affect each other. Recently it has been shown that the maximum amplitude of the circadian process actually declines according to increasing homeostatic pressure.²⁵ A study on sleep-deprived individuals showed that a higher homeostatic pressure due to low amounts of sleep led to an increase of the sleep-promoting signal at night, as well as a decrease in the wake-promoting signal in the morning.²⁴ This indicates that the circadian process deviates from its normal levels due to changes in the homeostatic process, demonstrating that the two processes

are actually continuously interacting with one another, rather than being independent as originally believed.

Localization of brain regions must also be taken into account with regard to the homeostatic process of the sleep-wake cycle. Studies show that brain regions relied on more heavily throughout the day have increased homeostatic drive throughout the day, independent of other brain regions. This eventually leads to a greater amount of SWS observed in these fatigued brain regions during the next sleep episode.²⁶ This means that the homeostatic drive toward sleep may not be a global brain phenomenon, but may rely on the various homeostatic pressures throughout the brain.

Scientists have begun to understand the importance of monoaminergic (MA) brainstem nuclei and their connections with the ventrolateral preoptic area (VLPO) of the hypothalamus, and the influence that this interaction has on sleep-wake signals.²⁷ Monoaminergic neurons are part of the arousal system and promote wakefulness when active. These neurons inhibit the VLPO neurons, which are responsible for promoting sleep (Figure 7). The interaction between these areas of the brain is mutually inhibitory, and leads to the sleep-wake switch called a “flip-flop” behavior.²⁸ An aroused and awake brain state is caused by number of interacting brain systems that make up the Ascending Arousal System.²⁹ The two categories of brainstem nuclei that make up this system are monoaminergic (MA) and acetylcholinergic (ACh).¹⁷ The ACh nuclei follow a pathway that runs to the thalamus, which allows for information transmission into the cerebral cortex. The second pathway involves the MA nuclei, activating the lateral hypothalamus, basal forebrain, and the cerebral cortex.³⁰ The MA nuclei are mainly associated with the Ascending Arousal System, while the ACh nuclei play a smaller role in the arousal

state.¹⁷ The firing of the MA nuclei serves to inhibit the VLPO throughout the day, and allows an individual to stay awake and vigilant.

Despite being in an aroused state, there is a progressive drive toward VLPO activation throughout the day due to the circadian and homeostatic processes.²⁷ Drive toward VLPO activation is partially time dependent, relying on the circadian process. The homeostatic process is also activated due to the buildup of somnogens throughout the day, which include adenosine, prostaglandin D₂, and immunomodulatory cytokines.^{27,31} These buildups create a homeostatic pressure leading to tiredness and eventually sleep. During sleep, clearance of somnogens increases, and the homeostatic pressure quickly declines. This new model expands on the original two-process model by including the impacts of the MA and VLPO neurons on sleep/wake cycles.¹⁷ This creates a more accurate model by taking into account the effects of inhibition of one brain region on another as well as the impacts of external cues on these processes (Figure 8).

Mistimed and Inadequate Sleep Patterns

Receiving more or less than 6-8 hours of sleep can be detrimental to overall health and brain function, and is associated with an increased mortality rate.³² Abnormal sleep schedules, such as overnight or split-shift hours, also lead to harmful effects. Night shift workers are at a higher risk for obesity, type-2 diabetes, and metabolic syndrome.^{33,34} In addition to this, working overnight shifts for extended periods is also correlated with an increased risk for breast cancer in women.³⁵ This association occurs because circadian misalignment alters cell cycles within the body, which can lead to complications in cell maintenance, eventually leading to the production of tumors.³⁶ Along with these physical ailments, cognitive impairment is also caused

by alterations in sleep cycles.³⁷ Neurocognitive deficits correlated with inadequate sleep include poor working memory, decision-making, and attentiveness.³⁸ Studies on mice found that when the animals were sleep deprived for a 3-month period they produced higher levels of amyloid- β and senile plaque development in the cortex and hippocampal areas, which are associated with Alzheimer's Disease, as well as impaired learning and memory.³⁹ The same study also found that all biological harm done to the brain during sleep deprivation was stable, and could not be reversed, implicating serious long-term impacts of poor sleep habits. This correlation is also found in humans, where high levels of daytime sleepiness have been found to be associated with increased loss of cognitive function, and may be an early indicator of eventual diagnosis of Alzheimer's Disease/Dementia.⁴⁰

Animal studies performed by Rechtschaffen and colleagues induced complete sleep deprivation on rats, which led to eventually death of the animals within 2-3 weeks.⁴¹ A follow up study to determine the specific physiological cause of death for these animals predicted that cell death caused by lack of sleep eventually killed the rats. However, after examining the brains of the deceased animals, no such brain abnormalities were found.⁴² The specific cause of death by complete sleep deprivation continues to be one of the great mysteries of sleep science. These findings demonstrate the importance of healthy sleep habits, as well as the risks that are involved with alternative schedule types. Due to the high-speed, high-demand, and high-stress nature of our country, different types of work and sleep schedules are becoming more common every day. Hence, research on the impact of a "sleepless" society, and discovery of optimal sleep schedules, is vital.

Many medical and psychological illnesses contribute to poor sleep patterns.⁴³ Medical conditions that cause pain in patients can be particularly disrupting to sleep patterns, and include arthritis, fibromyalgia, cardiovascular disease, cancer, as well as other chronic diseases. Certain illnesses have symptoms that are exacerbated during sleep, such as respiratory and gastrointestinal illnesses. Another illness that has extremely disruptive effects on circadian rhythms is Alzheimer's disease. Not only is disrupted circadian rhythm a contributing cause for Alzheimer's, but the disease itself leads to disruption of circadian rhythms and behavioral abnormalities that are often correlated to the time of day.⁴⁴ Psychological illnesses such as depression and anxiety can disrupt sleep, due to tension and insomnia associated with anxiety, and frequent nightly and early-morning arousals experienced with depression.⁴³ In addition to the negative physical and psychological impacts of dysregulated sleep patterns, the impacts of fatigue on decision making and accuracy can be detrimental. Large-scale disasters such as the Chernobyl catastrophe and the Exxon Valdez oil spill were both the result of errors of the staff that were made after working extreme hours, leading to fatigue.⁴⁵ The rate of error that people make increases drastically with sleep deprivation, and in some cases can lead to devastating results.

Given society's requirement for 24-hour productivity, attempts have been made at adjusting sleep patterns in order to accommodate today's expectations. Modern society now includes a diverse array of work and sleep schedules due to more demanding jobs as well as leisure activities and hobbies. This review will now look at the effects of a variety of sleep schedules, in an attempt to assess whether alternative sleep patterns are feasible and sustainable. A number of studies have been performed on the impacts of split shift vs. single

shift schedules on the quality of sleep. In a study performed by Jackson and colleagues, sleep quality between monophasic nighttime sleep, monophasic daytime sleep, and a split sleep schedule were compared.⁴⁶ The results indicate that nighttime monophasic sleep has the best quality, based on more time spent in REM, longer total sleep time, and lower rating of sleepiness while awake. The split shift schedule also had higher total sleep time than the daytime sleep group, with the latter expressing higher levels of sleepiness while awake. This data indicates the importance not just of the opportunity to sleep, but when during the circadian rhythm this sleep occurs, showing that split shift opportunities may be less harmful than nightshift work.

A similar study was performed by Roach and colleagues, but in addition to examining the differences between single and multiple sleep episodes, this study included a forced desynchrony protocol which allowed sleep to be examined in both groups over various circadian rhythms.⁴⁷ Polysomnography data collected on both groups indicated that in some measures sleep quality was better for the consolidated sleep group, while in other cases the split sleep group showed greater quality. The split condition participants had a longer sleep onset, and had more arousals during the night, demonstrating lower quality sleep. However, the split condition also showed less amount of time spent awake in bed after initially falling asleep, as well as a higher percentage of slow wave sleep. Both of these factors indicate sleep of a higher quality. Because the levels of SWS are higher during the first two hours of a sleep episode, it can be argued that this is an advantage to a split sleep schedule because it allows for a higher percentage of deep recovery SWS. Another advantage comes from the decreased

homeostatic pressure in a split sleep schedule due to being awake for shorter periods of time, thus decreasing sleepiness while awake.

Certain groups in society have begun experimentation with various kinds of polyphasic sleep patterns, as a way to “cheat sleep” and spend a higher percentage of life awake and productive, calling themselves “Polyphasic Society”.⁴⁸ As discussed previously, monophasic sleep patterns are typical in western culture, with the biphasic sleep pattern of split-shift workers or individuals who nap during the day also being common. Popular polyphasic patterns break up sleep to a greater extent, and in the more extreme examples, significantly cut down on total amount of sleep. Each of the sleep schedules are all explained on the “Polyphasic Society” website. The least extreme version is triphasic sleep, which allows for three periods of rest during the 24-hour day. Each rest period lasts about 1.5 hours, and occurs during the circadian low-points of the day (after dusk, before dawn, and in the afternoon), which makes it one of the easier patterns to adjust to. The other patterns are much more difficult, and include an array of napping times throughout the day, the most common of these being Everyman, Uberman, and Dymaxion schedules. The Everyman schedule includes one core sleep during the nighttime, usually between 4-6 hours long, and anywhere from 2-4 twenty minute naps dispersed throughout the day. The Uberman schedule involves 6-8 twenty minute naps over the 24-hour period for a total of 2-3 hours of sleep per day. The Dymaxion pattern is the most difficult, and includes 4 thirty minute naps evenly dispersed throughout the day, leading to 2 hours of sleep per 24-hour period. The effectiveness of these schedules are based on subjective accounts of individuals on the website, and have not been evaluated scientifically.

Understandably, these patterns are only used by a small sub-group of the human population, and many individuals are unable to sustain such extreme sleep patterns. However, it is not difficult to understand the appeal of decreasing overall sleep time, as well as being immune to the ill-effects of sleep deprivation. Matthew Wolf-Meyer discusses various aspects of completely forgoing sleep in *Fantasies of Extremes: Sports, War, and the Science of Sleep*⁴⁹, which includes the desire to create armies that will never become fatigued, or super-human athletes who can perform at exceptional levels for extended amounts of time. To date, there are no direct experiments testing the effectiveness or safety of specific polyphasic patterns; however, studies of solo-sailing racers and their sleep patterns during competition have provided the closest examination of polyphasic sleep so far. These races are multiple days long, and require nearly round the clock focus, leading to a very disrupted sleep pattern in the athletes. A study by Hurdie and colleagues observed the sleep patterns of 16 sailors on a two-leg transatlantic race, and found that on average the participants took multiple naps per day that summed to an average of approximately 4 hours of sleep per day. A number of functional impairments were reported by the sailors during both legs of the race, including psychomotor impairments leading to errors, difficulties with fatigue, hallucinations, and mood disturbances.⁵⁰ One interesting aspect of the data was the difference between the number of impairments between the two legs of the race. Significantly more disturbances were experienced and recorded during the first leg of the race, implying that after initially adjusting to this difficult sleep pattern the participants were able to function fairly well. When comparing this data to other sailing studies, it appears that the length of a race impacts the level of sleep deprivation sustained: with 3-day race participants averaging 2.8 hours of sleep per day in the

Tiberge study, 1-3 week long race averaging 4 hours in the study by Hurdiel and colleagues, and a month-long race at 6.3 hours per day in the Stampi study.⁵⁰ This supports the data from modern sleep models indicating that consistent sleep deprivation is not sustainable over long periods of time.⁵¹

Although this research on sailing competitions gives us a glimpse into polyphasic sleep and its effects, more research is required before we can truly understand the long-term impacts of this type of lifestyle. The idea behind the success of these extreme sleep schedules is that with time and adherence to the schedule, your body will learn to adapt by shortening the time spent in each of the sleep stages in order to complete full sleep cycles in a condensed period of time.⁴⁸ If an individual who normally has one monophasic sleep episode per night takes a nap, they will most likely stay in S1 and S2 throughout the duration of the episode, without ever entering SWS or REM. The goal of polyphasic schedules is to eventually be able to have normal sleep architecture that includes all stages during these short sleep intervals, so that you are still getting the required elements of sleep, just in smaller, more efficient doses. This idea and a large amount other information is presented on the Polyphasic Society website, however there is no evidence to support that this is what actually occurs, as there are only a handful of references to back-up these claims, with no specific experiments performed. Although there are a number of testimonials of individual success, large scale controlled experiments must be performed to determine the validity of these claims, and to rule out the possibility of placebo effects. It is an intriguing idea that could benefit a host of people if proven to be effective, and should be further pursued for more information.

Feasibility of Polyphasic Sleep Patterns

Although no studies have been performed on specific polyphasic sleep patterns, sailing studies as well as research on various aspects of sleep schedules gives us insight into the benefits and drawbacks of such patterns. During a normal night of sleep, an individual will cycle through the various sleep stages multiple times; but when an individual is sleep deprived, their sleep architecture become significantly altered. When participants are sleep deprived for several days in a row, the levels of S1 and S2 during sleep diminish or completely disappear, while S3, S4, and REM sleep levels dramatically increase.⁵² This pattern demonstrates the importance of SWS and REM in sleep architecture, and also demonstrates that as fatigue increases the quality of sleep also increases, based on shorter sleep latency, and higher levels of SWS, which promotes deep recover sleep. This data mirrors that found in rebound sleep, except for the main difference in the prolonged duration of most typical rebound sleep episodes. It appears that if someone is consistently getting only a few hours of sleep a day, their short sleep episodes will be deeper and more efficient¹, leading some to disagree with mathematical models demonstrating that such patterns are not sustainable.

An advantage could be predicted from the alteration of the two-process sleep model, given the decrease of homeostatic pressure buildup due to the various sleep episodes throughout the day. The continuous clearing of somnogens throughout the day could decrease feelings of fatigue between sleep episodes, and has been found to be one positive effect of split-sleep schedules.⁵³ However, findings addressing the interconnection of processes C and S may indicate that any benefit from decreased homeostatic pressure may be cancelled out or decreased by misaligned circadian rhythm.³⁰ This drawback is likely to impact the majority of

people who attempt polyphasic sleep schedules; however, there is one subgroup of individuals who can greatly benefit from the effects of sleep deprivation. Studies on depressed patients have found that sleep deprivation, along with other interventions including therapy and medication, can be very effective at decreasing depressive moods.⁵³ These therapeutic impacts can also be seen in Parkinson's patients, as one night of sleep deprivation often leads to decreased symptoms for several days.^{52,54} Future studies on the effects of polyphasic sleep patterns on various psychological and physiological illness could lead to innovative new treatments and discoveries.

Another interesting concept that could impact quality of life on a polyphasic schedule involves experiences of sleep inertia, which is the feeling of disorientation, confusion, fatigue, and lack of mental acuity following a sleep episode.⁵⁵ Many people experience this problem when they struggle to get their brain and body moving in the morning after waking up. The phenomenon appears to be linked to both circadian factors as well as sleep architecture. Sleep inertia is experienced more commonly when an individual is awakened out of SWS or REM sleep⁴⁹ as well as when they wake up at the low point of circadian body temperature rhythm.⁵⁶ These are both very likely possibilities given the sleep deprivation and disregard for circadian rhythms in polyphasic patterns, leading to a question of whether the extra time awake will be negated by the negative effects of sleep inertia.

Conclusion:

It is abundantly apparent that sleep is an essential aspect of our lives. Being completely sleep deprived leads to eventual death in animals, and even partial sleep deprivation causes an array of physical and mental-health problems. Important information about this fundamental

life process continues to elude scientists, and the quest for answers is ongoing. Loopholes to avoid sleep in an attempt to increase productivity are constantly being sought-out, in many cases with disastrous consequences. On the horizon of sleep science is research into polyphasic sleep, in an attempt to understand its long-term effects and sustainability. Currently, there are numerous well-known disadvantages to poor sleep hygiene, and many benefits appear to be outweighed by these consequences. However, future research may bring us closer toward being the truly “sleepless” society that we try so hard to be.

Age	Sleep Needed average per day
Infants	Up to 18 hours
1-12 months	14-18 hours
1-3 years	12-15 hours
3-5 years	11-13 hours
5-12 years	9-11 hours
Teens	9-10 hours
Adults	7-8 hours
Pregnant women	Varies

Table 1⁵⁷: Describes the sleep requirements for individuals at different developmental stages.

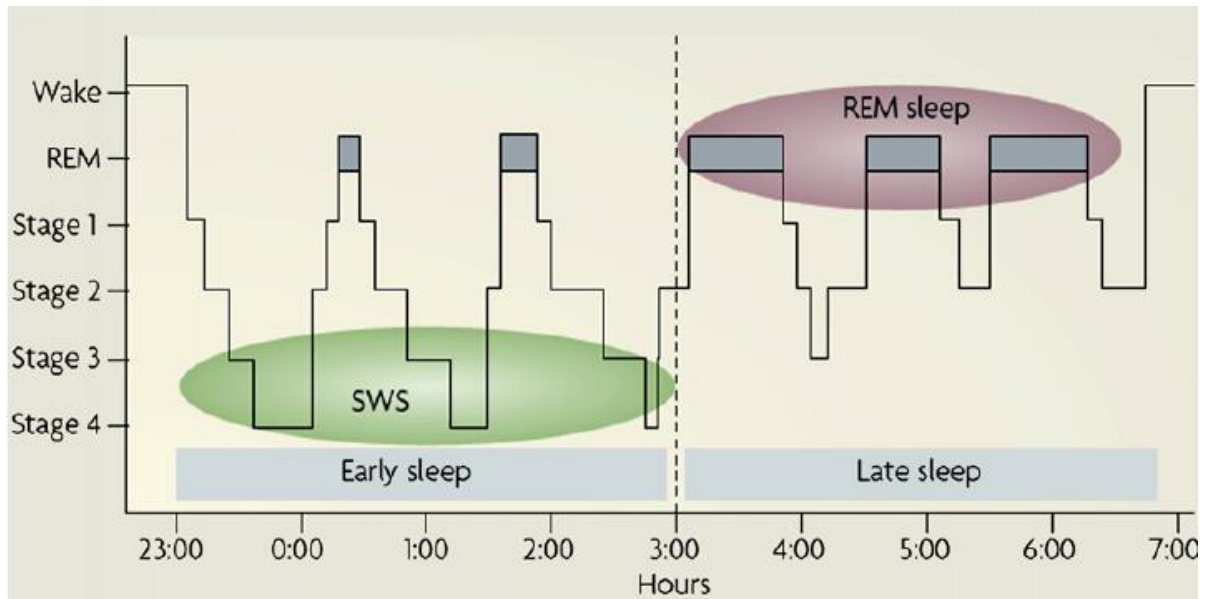


Figure 1⁵⁸: A hypnogram showing the typical progression of sleep stages throughout the night.

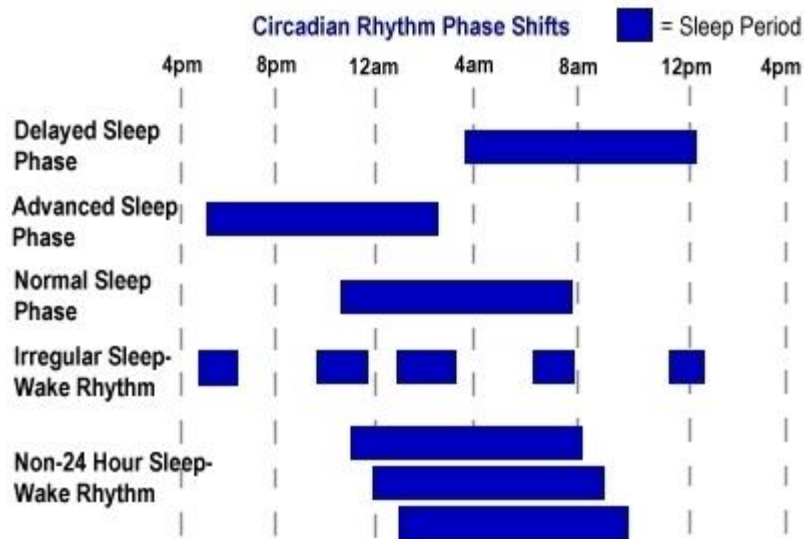


Figure 2⁵⁹: A graph demonstrating the effect of circadian rhythm phase shift on the timing of sleep periods.

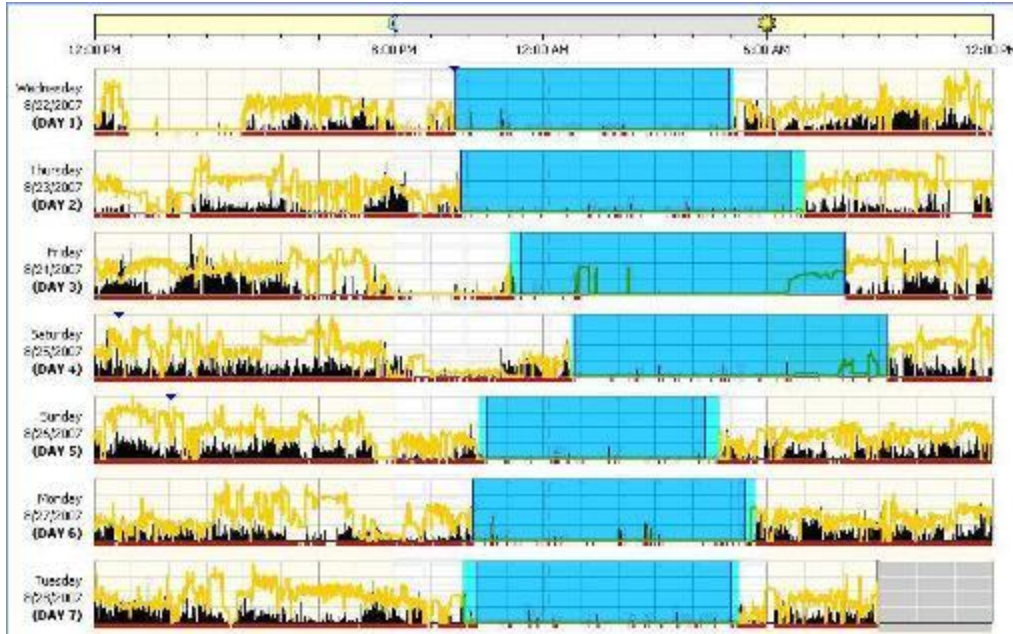
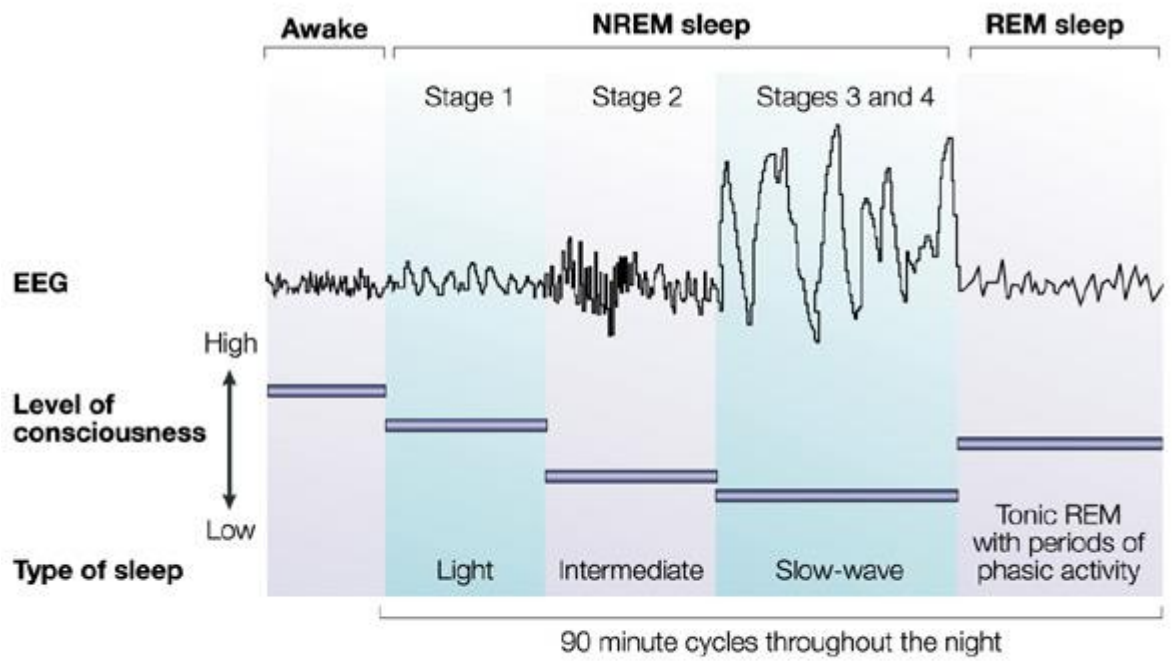


Figure 3⁶⁰: Actigraph recording motor movements in order to visually display circadian rhythm.



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Figure 4⁶¹: EEG wave patterns in various stages of sleep.

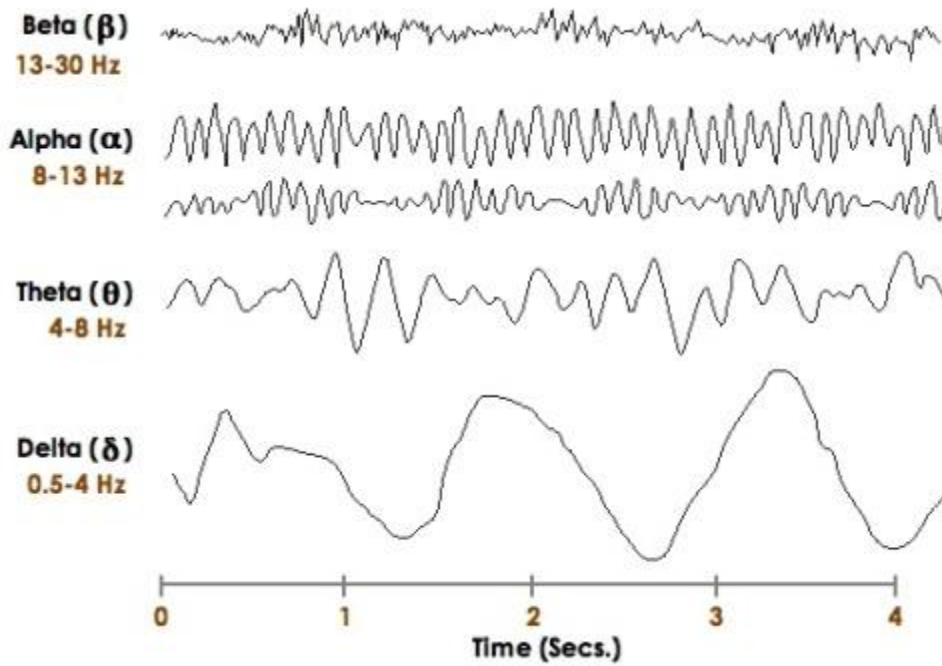


Figure 5⁶²: Four different types of waveforms that can be identified in EEG.

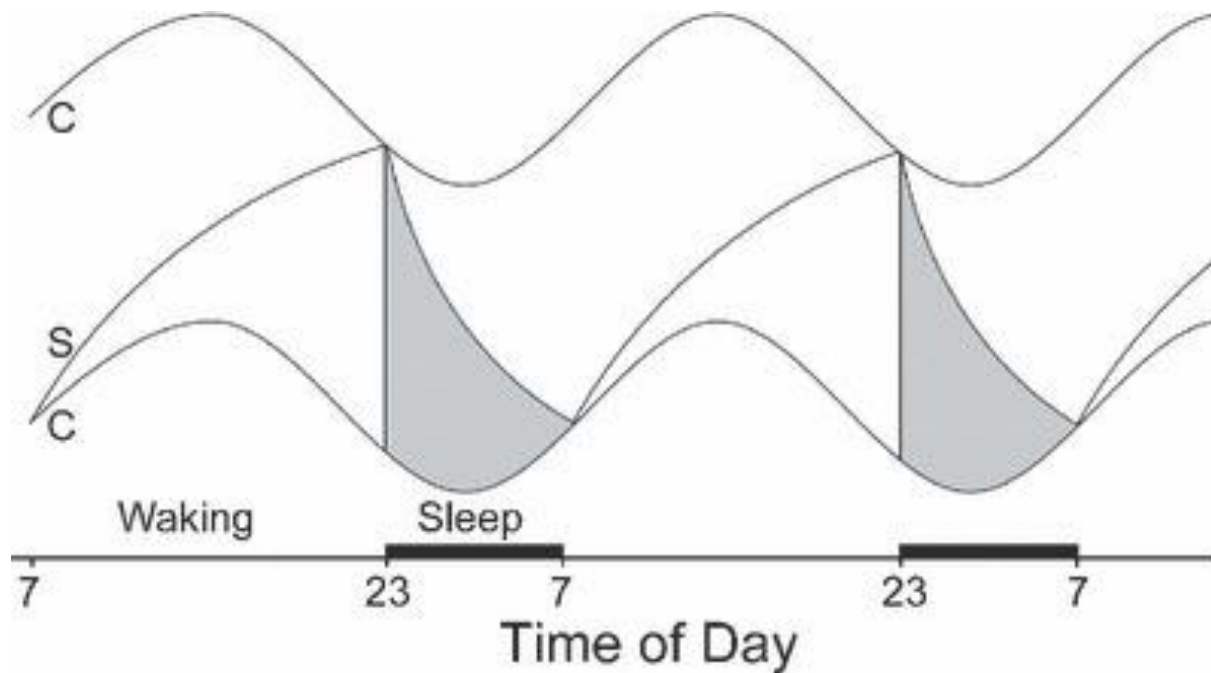


Figure 6⁶³: Circadian and Homeostatic rhythms plotted throughout the day in order to examine their combined effect on sleep propensity.

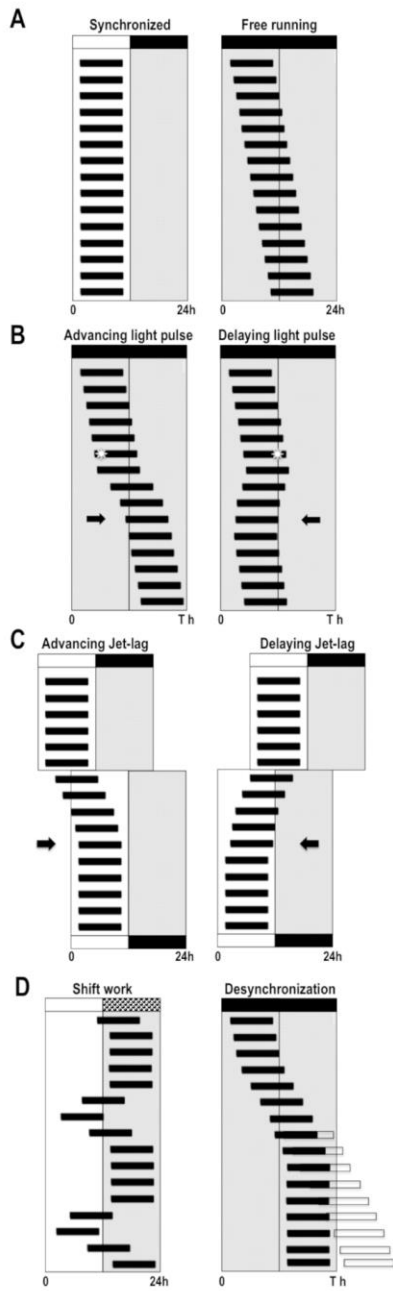


Figure 7⁶⁴: Actigraphy demonstrating the impacts of external variables on circadian rhythm.

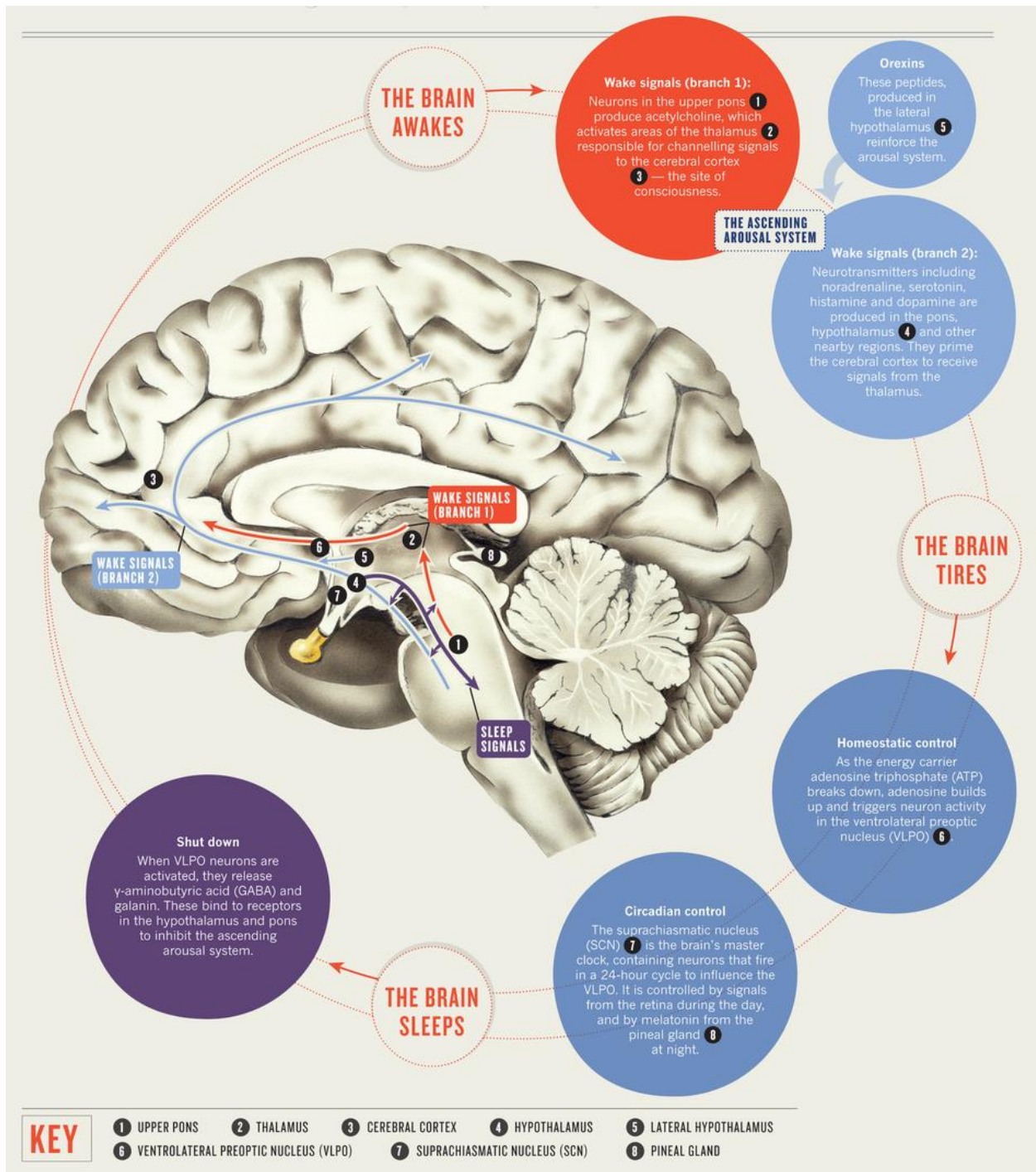


Figure 8⁶⁵: Demonstrates the impacts of neurotransmitter signals on sleep propensity. Production of Ach activates the thalamus, which allows signals to be sent to the cerebral cortex, enabling consciousness. VLPO is activated and releases GABA and galanin to inhibit the Ascending Arousal System and promote sleep.

References:

1. Paterson LM. The Science of Sleep. In: Sleep: Multi-professional Perspectives. London, Philadelphia, PA: Green A, Westcombe A, Varma V. 2012: 18-40.
2. McCarley RW. Neurobiology of REM and NREM Sleep. *Sleep Medicine*. 2007; 8(4): 302–330.
3. Schulze G. Sleep protects excitatory cortical circuits against oxidative damage. *Medical Hypotheses*. 2004; 63(2): 203–207.
4. Besedovsky L, Lange T, Born J. Sleep and immune function. *European Journal of Physiology*. 2012; 463(1): 121-137.
5. Wagner U. The Brain Never Sleeps: How Sleep Improves our Memory. In: *Sleeping and Dreaming*. Black Dog Publishing. Monem N. 2007: 63-71.
6. Skeldon AC, Derks G, Dijk D. Modelling changes in sleep timing and duration across the lifespan: Changes in circadian rhythmicity or sleep homeostasis? *Sleep Medicine Reviews*. 2015; 28(2016): 96-107.
7. Dijk D, Winsky-Sommerer R. Sleep. *New Scientist*. 2012; 213(2850): i-8.
8. Roenneberg T, Kuehnle T, Pramstaller PP, et al. A marker for the end of adolescence. *Current Biology*. 2004; 14(24): R1038-R1039.
9. Campbell SS, Dawson, D. Aging young sleep: a test of the phase advance hypothesis of sleep disturbance in the elderly. *Journal of Sleep Research*. 1992; 1(3): 205-210.
10. Mallampalli MP, Carter CL. Exploring Sex and Gender Differences in Sleep Health: A Society for Women’s Health Research Report. *Journal of Women’s Health*. 2014; 23(7): 553-562.

11. Lee KA, Kryger MH. Women and Sleep. *Journal of Women's Health*. 2008; 17: 1189–1190.
12. Redline S, Kirchner HL, Quan SF, et al. The Effects of Age, Sex, Ethnicity, and Sleep-Disordered Breathing on Sleep Architecture. *Archives of Internal Medicine*. 2004; 164(4): 406-418.
13. Ohayon MM, Carskadon MA, Guilleminault C, et al. Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals: developing normative sleep values across the human lifespan. *Sleep*. 2004;27(7):1255-1273.
14. Tripathi M. Technical notes for digital polysomnography recording in sleep medicine practice. *Annals of Indian Academy of Neurology*. 2008; 11(2): 129–138
15. Hudson N. Recording and Quantifying Sleep. In: *Sleep: Multi-professional Perspectives*. London, Philadelphia, PA: Green A, Westcombe A, Varma V. 2012: 149-166.
16. Roach GD, Zhou X, Darwent D, et al. Are two halves better than one whole? A comparison of the amount and quality of sleep obtained by healthy adult males living on split and consolidated sleep–wake schedules. *Accident Analysis and Prevention*. 2015.
17. Phillips AJ, Robinson PA. A quantitative model of sleep-wake dynamics based on the physiology of the brainstem ascending arousal system. *Journal of Biological Rhythms*. 2007; 22(2): 167-179.
18. Borbély AA. A two process model of sleep regulation. *Human Neurobiology*. 1982; 1(3): 195-204.
19. Daan S, Beersma DG, Borbély AA. Timing of human sleep: recovery process gated by a circadian pacemaker. *American Journal of Physiology*. 1984; 246(2 Pt 2): R161-183.

20. Dijk DJ, Archer SN. PERIOD3, circadian phenotypes, and sleep homeostasis. *Sleep Medicine Reviews*. 2010; 14(3): 151-160.
21. Jones BE. Glia, Adenosine, and Sleep. *Neuron*. 2009; 61(2): 156–157.
22. Lin C, Gamble J, Yang Y, et al. Estimating the Influence of Chronotype and Social Zeitgebers on Circadian Rhythms using an Accelerometer-based Sensor Network. Paper presented at: International Conference on Biomedical and Health Informatics; Jan 2012; Hong Kong and Shenzhen, China.
23. Wever RA. Properties of human sleep-wake cycles: parameters of internally synchronized free-running rhythms. *Sleep*. 1984; 7(1): 27-51.
24. Borbély AA, Daan S, Wirz-Justice A, et al. The two-process model of sleep regulation: a reappraisal. *Journal of Sleep Research*. 2016; 25(2): 131-143.
25. Dijk DJ, Czeisler CA. Contribution of the Circadian Pacemaker and the Sleep Homeostat to Sleep Propensity, Sleep Structure, Electroencephalographic Slow Waves, and Sleep Spindle Activity in Humans. *The Journal of Neuroscience*. 1995; 15(5): 3526-3538.
26. Kattler H, Dijk DJ, Borbély AA. Effect of unilateral somatosensory stimulation prior to sleep on the sleep EEG in humans. *Journal of Sleep Research*. 1994; 3(3): 159-164.
27. Phillips AJK, Robinson PA, Kedziora DJ, et al. Mammalian Sleep Dynamics: How Diverse Features Arise from a Common Physiological Framework. *PLoS Computational Biology*. 2010; 6(6): e1000826.
28. Saper CB, Chou TC, Scammell TE. The sleep switch: hypothalamic control of sleep and wakefulness. *Trends in Neuroscience*. 2001; 24(12): 726-31.

29. Hobson JA, Pace-Schott EF. The cognitive neuroscience of sleep: neuronal systems, consciousness and learning. *Nature Reviews Neuroscience*. 2002; 3(9): 679-693.
30. Saper CB, Scammell TE, Lu J. Hypothalamic regulation of sleep and circadian rhythms. *Nature*. 2005; 437(7063): 1257-1263.
31. Nagata N, Urade Y. Sleep-wake regulation by prostaglandin D2 and adenosine. *Brain Nerve*. 2012; 64(6): 621-628.
32. Grandner MA, Hale L, Moorea M, et al. Mortality associated with short sleep duration: The evidence, the possible mechanisms, and the future. *Sleep Medicine Reviews*. 2010; 14 (3): 191-203.
33. Pan A, Schernhammer ES, Sun Q, et al. Rotating Night Shift Work and Risk of Type 2 Diabetes: Two Prospective Cohort Studies in Women. *PLoS Med*. 2011; 8(12): e1001141.
34. Tucker P, Marquié J, Folkard S, et al. Shiftwork and Metabolic Dysfunction. *Chronobiology International*. 2012; 29(5): 549–555.
35. Hansen J, Lassen CF. Nested case-control study of night shift work and breast cancer risk among women in the Danish military. *Occupational and Environmental Medicine*. 2012; 69(8): 551.
36. Bieler J, Cannavo R, Gustafson K, et al. Robust synchronization of coupled circadian and cell cycle oscillators in single mammalian cells. *Molecular Systems Biology*. 2014; 10: 739.
37. Marquié J, Tucker P, Folkard S, et al. Chronic effects of shift work on cognition: findings from the VISAT longitudinal study. *Occupational and Environmental Medicine*. 2015; 72(4): 258.

38. Durmer JS, Dinges DF. Neurocognitive consequences of sleep deprivation. *Seminars in Neurology*. 2005; 25(1): 117-129.
39. Qiu H, Zhong R, Liu H, et al. Chronic Sleep Deprivation Exacerbates Learning-Memory Disability and Alzheimer's Disease-Like Pathologies in A β PP(swe)/PS1(Δ E9) Mice. *Journal of Alzheimer's Disease*. 2015; 50(3): 669-685.
40. Jausent I, Bouyer J, Ancelin M, et al. Excessive Sleepiness is Predictive of Cognitive Decline in the Elderly. *Sleep*. 2012; 35(9): 1201–1207.
41. Rechtschaffen A, Gilliland MA, Bergmann BM, Winter JB. Physiological correlates of prolonged sleep deprivation in rats. *Science*. 1983; 221(4606): 182-184.
42. Cirelli C, Shaw PJ, Rechtschaffen A, et al. No evidence of brain cell degeneration after long-term sleep deprivation in rats. *Brain Res*. 1999; 840(1-2): 184-193.
43. Rusak B., Ruyak P. Sleep. In: *Encyclopedia of Aging*. Vol 4. New York, NY: Ekerdt D.J.; 2002: 1277-1281.
44. Musiek ES, Xiong DD, Holtzman DM. Sleep, circadian rhythms, and the pathogenesis of Alzheimer Disease. *Experimental & Molecular Medicine*. 2015; 47(3): e148.
45. Zully J. Without Sleeping, there is No Waking: An Introduction to Sleep in Modern Society. In: *Sleeping and Dreaming*. Black Dog Publishing. Monem N. 2007: 27-46.
46. Jackson ML, Banks S, Belenky G. Investigation of the effectiveness of a split sleep schedule in sustaining sleep and maintaining performance. *Chronobiology International*. 2014; 31(10): 1218–1230.

47. Roach GD, Zhou X, Darwent D, et al. Are two halves better than one whole? A comparison of the amount and quality of sleep obtained by healthy adult males living on split and consolidated sleep–wake schedules. *Accident Analysis and prevention*. 2015.
48. Polyphasic Society. www.polyphasicsociety.com. Accessed November 23, 2016.
49. Wolf-Meyer M. Fantasies of Extremes: Sports, War and the Science of Sleep. *BioSocieties*. 2009; 4(2-3): 257-271.
50. Hurdziel R, Monaca C, Mauviex B, et al. Field study of sleep and functional impairments in solo sailing races. *Sleep and Biological Rhythms*. 2012; 10: 270–277
51. McCauley P, Kalachev LV, Smith AD, et al. A new mathematical model for the homeostatic effects of sleep loss on neurobehavioral performance. *Journal of Theoretical Biology*, 2009; 256(2): 227-239.
52. Orzel-Gryglewska J. Consequences of Sleep Deprivation. *International Journal of Occupational Medicine and Environmental Health*. 2010; 23(1): 95-114.
53. Wirz-Justice A, Van den Hoofdakker RH. Sleep deprivation in depression: what do we know, where do we go? *Biological Psychiatry*. 1999; 46: 445-453.
54. Demet E, Chicz-Demet A, Fallon JH, et al. Sleep deprivation therapy in depressive illness and Parkinson’s Disease. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*. 1999; 23: 753-784.
55. Tassi P, Muzet A. Sleep inertia. *Sleep Medicine Reviews*. 2000; 4(4): 341-353.
56. Dinges DF, Orne MT, Orne EC. Assessing performance upon abrupt awakening from naps during quasi-continuous operations. *Behavior Research Methods, Instruments, & Computers*. 1985; 17: 37–45.

57. Sleep Centers of Middle Tennessee. How much sleep does one need?
<http://sleepcenterinfo.com/index.php/disorders/>.
58. Payne JD. Learning, Memory, and Sleep in Humans. *Sleep Medicine Clinics* 2011; 6(1): 15-30.
59. Mastin L. Sleep Disorders: Circadian Rhythm Sleep Disorders. Sleep: what it is – how it works – why we do it – what can go wrong.
http://www.howsleepworks.com/disorders_crsd.html
60. Kaiser Permanente. Actigraphy. 2016. <https://thrive.kaiserpermanente.org/care-near-you/northern-california/sanjose/departments/sleep-medicine-services/patient-resources/actigraphy/>.
61. Bryant PA, Trinder J, Curtis N. *Nature Reviews Immunology*. 2004; 4: 457-467.
62. Ricker J. Stages of Sleep. PSY 101-Introduction to Psychology.
https://sccpsy101.com/home/chapter-2/section-8/brain_waves-3/
63. Antle MC, Silver R. Neural basis of timing and anticipatory behaviors. *European Journal of Neuroscience* 2009; 30(9): 1643-1649.
64. Elvira A, Ivette C, Dalia DI, et al. The Circadian Timing System: A Recent Addition in the Physiological Mechanisms Underlying Pathological and Aging Processes. *Aging and Disease*. 2014; 5(6): 406-418.
65. Peplow M. Structure: The anatomy of sleep. *Nature* 2013; 497: S2–S3.