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A primer on sleeping, dreaming, and psychoactive agents

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ABSTRACT

As a necessary part of physiological and emotional well-being, sleep is often overlooked or undervalued in Western society, in spite of the role restorative sleep plays in growth, healing and recovery, immune response, and emotional regulation. The effect of psychoactive drugs on sleep, including pharmacological substances meant to assist in sleep, is notable and profound, especially in their disruption of REM sleep, the stage of sleep associated with dreaming. This article provides an overview of sleep stages, the brain and sleep, sleep disorders, and the effect of various psychoactive drugs on sleep architecture and hygiene. Future research that treats substance use and sleep as bi-directional in nature and longitudinally explores sleep-related interventions in treatment and longer-term recovery that are sustainable and person-centered is merited.

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Dreaming; nREM sleep; psychoactive drugs; REM sleep; sleep disorders; sleep hygiene

The importance of sleep

Sleeping is an integral part of our lives, and yet the scope of its importance is typically not fully appreciated. While most realize sleep is necessary for physical health, what is not as readily acknowledged is the importance of dreaming for mental health. The need for sleep is so essential that if we miss one night of sleep, our body tries to recover what was lost in subsequent nights. Sleep appears to be universal in that virtually every species has some kind of sleep. There are various theories behind why we must sleep, with physical rest being only a partial explanation. There is no argument that sleep allows our bodies to save and restore energy, and that while we sleep, our metabolism is much slower than when we are awake but there are also periods of sleep when our brain is actually more active than during wakefulness. While we are asleep, our brains also reorganize and store information, something for which dreaming is crucial; the rapid eye movement (REM) stage of sleep plays a role in memory retention and consolidation, for even one night without REM sleep decreases the ability to retain newly learned information. The retention of complex information is greatly reduced when a person is deprived of the REM stage of sleep. It has also been hypothesized that REM sleep is designed to remove useless information from memory in a selective pruning process that balances the number of new synapses the brain generates during development and learning. Thus, dreaming is as important for removing unwanted information as it is for storing important data (Diekelmann & Born, 2010; Li et al., 2017).

Psychoactive drugs have a direct and profound impact on all activities of the brain associated with the sleep cycle, especially dreaming. As well, many drugs that promote sedation actually limit and disrupt the sleep cycle, limiting the amount of time spent in

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restorative sleep while also having significant effects on REM sleep. Likewise, stimulants minimize the need for sleep, creating additional physical and mental health issues along with the risk of physical and psychological dependency (Chan et al., 2013). Sleep deprivation has a host of negative physical outcomes, as well as contributing to and exacerbating mental health issues and emotional dysregulation (Drapeau & Nodorff, 2017; Sisman et al., 2021).

The brain and sleep

The brain is composed of three core components: the forebrain, the midbrain, and the hindbrain (Figure 1). The forebrain consists of the cerebrum, where the majority of information processing occurs; the thalamus, whose function includes relaying sensory and motor signals to the thin layer of cells covering the brain; the cerebral cortex, where the regulation of consciousness, sleep, and alertness occurs; and the limbic system. The hindbrain extends from the spinal cord and is critical for maintaining balance and equilibrium, movement coordination, and the conduction of sensory information. The reticular activating system (RAS), a set of nuclei critical in regulating autonomic nervous system functions,

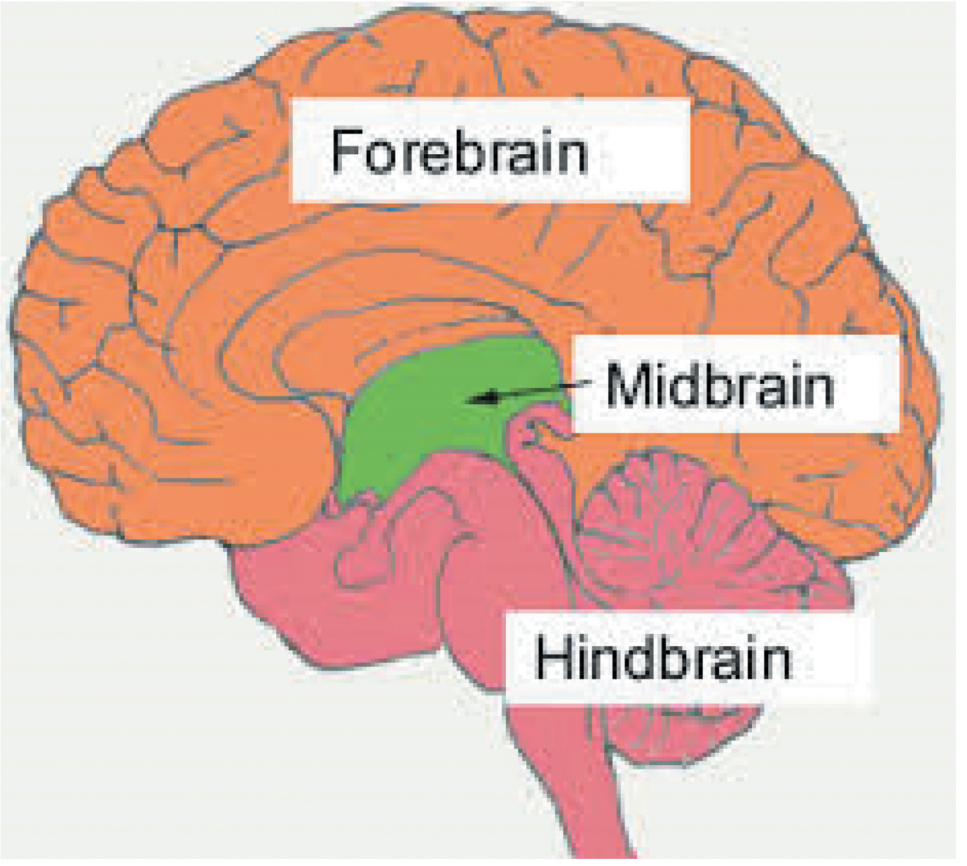

Figure 1. Cross Section of the Human Brain. Source: https://cerebralcomponents.weebly.com/forebrain-midbrain-and-hindbrain.html

connects the lower brain to the upper brain, with the descending portion being connected to the nerves essential to the sleep cycle. While brain activity is lessened during a normal night of sleep, various parts of the brain remain quite active. The hypothalamus, just above the brain stem, regulates the chemicals that promote sleep and then arousal. The thalamus, located in the forebrain, blocks input from the senses during sleep, allowing the brain to focus on processing sensory inputs perceived during the preceding day. The hippocampus, part of the limbic system, replays memories that will be stored by the brain. It is also hypothesized that the cerebral cortex is active during dreaming and that it is the cerebral cortex that attempts to interpret and make sense of all the information gathered during waking hours. During stage three of the sleep cycle(Figure 3), growth hormone is secreted from the pineal gland, which encourages bone and muscle growth in children and aids in tissue repair in adults (Dorland, 2019; McNamara, 2019; Reinoso-Suarez et al., 1999).

In order to gain an understanding of how psychoactive drugs affect the central nervous system (CNS), one must have a basic understanding of the process that underlies the functioning of the brain. Of the billions of cells of which the brain is composed, it is only the neuron, or nerve cell, that processes information. Messages travel within each cell as electrical transmissions, but as one neuron has no direct physical contact with another, electrical transmission between cells cannot occur. Thus, information between nerve cells must be communicated chemically. A neuron consists of the cell body, or soma, where metabolic activity occurs featuring the nucleus and dendrites. Dendrites are the extension of the soma that receive messages from the axons of adjoining cells. The axon is the part of the neuron along which signals are transmitted to adjoining cells that terminate in axon terminals. It is in the axon terminals where the various neurotransmitters including Gamma-Aminobutyric Acid (GABA), dopamine, norepinephrine, serotonin, endorphins, and endocannabinoids are found (Figure 2). The gap across which the neurotransmitters must travel is referred to as the synaptic cleft. The synaptic cleft is typically 10—20 nanometers across. This is such a tiny space that it takes only 0.1 ms for a neurotransmitter to drift, or defuse, across the gap to the next axon. Neurotransmitters are chemicals found in the brain that are used to relay, amplify, and modulate signals between neurons that produce physical actions, feelings, and behaviors and also affect

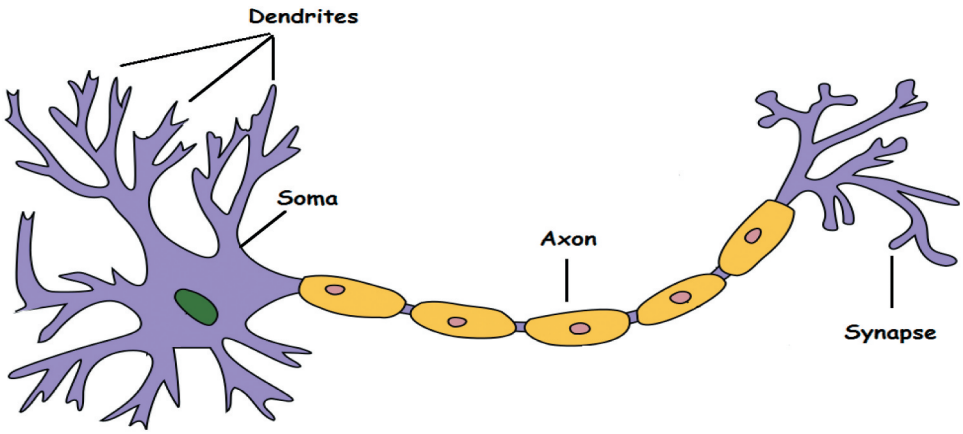

Figure 2. Neuron. Source: Tanuj Shrivastava

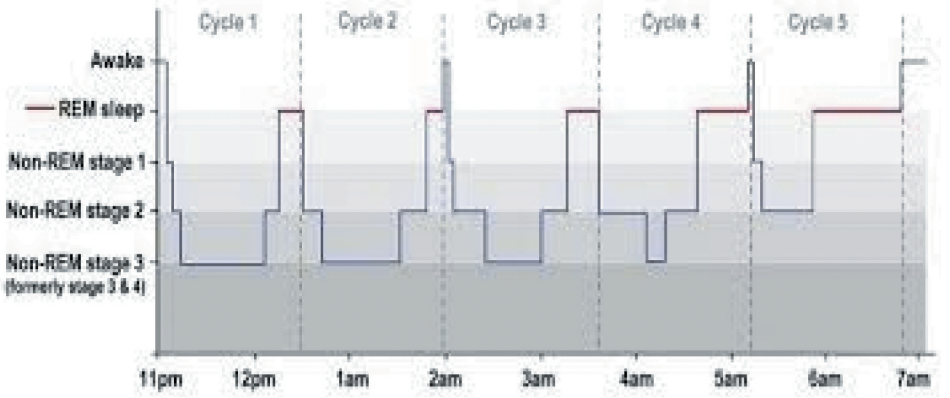

Figure 3. Sleep Cycle. Source: Dumay and Gaskell (2005).

sleeping and dreaming (Brick & Erickson, 2013; Dorland, 2019; Goldstein & Cacciamani, 2021).

Retrieved from: https://medium.com/analytics-vidhya/mp-neuron-model-f4feb53c21a2

Key neurotransmitters that play a role in the sleep cycle

Dopamine stimulates nerve receptors in the brain, regulating mood and playing a prominent role in motivation and reward through its ability to create sensations of power, energy, and, most importantly, euphoria. The dopaminergic system also plays an important part in human wakefulness and cognition. The role of dopamine follows a U-shape pattern, which means either too little or too much signaling impairs cognitive performance (Nakajima et al., 2013). Excessive amounts of dopamine can not only lead to increasing alertness but in even larger amounts of psychotic-like behavior including schizophrenia, while having too-low levels produces hyperirritability, anxiety, and sleepiness (Kocherlakota, 2014; Monti & Monti, 2007).

Gamma-Aminobutyric Acid (GABA) is an amino acid that acts as a depressant neurotransmitter that mitigates the human anxiety response. It works by occupying receptor sites and preventing their stimulation. The message that GABA transmits is an inhibitory one: it tells the neurons that it contacts to slowdown or stop firing. As approximately 40% of the billions of neurons throughout the brain respond to GABA, this means that GABA has a general quieting influence on the brain, thus, it serves as the body's natural tranquilizer. GABA has a particularly powerful effect upon the reticular activating system (RAS), which, as previously discussed, is vital in regulating wakefulness and attention as well as sleep and dreaming (Golan, Golan et al., 2016; Scammell & Lipton, 2017).

Endorphin, or endogenous morphine, is mimicked in nature and through synthesis, by the opioid family of psychoactive drugs. In addition to blocking the perception of pain, endorphins modulate dopamine transmission in the brain creating a sense of euphoria, but also sedation, respiratory depression, nausea, mental clouding, plus the production of physical dependency (Contet et al., 2004; Grella et al., 2014; Lalanne et al., 2014; Pellissier et al., 2016). Endorphin administration also modifies the sleep-wakefulness cycle and

research has found that an imbalance of endorphins can worsen chronic sleep disorders' effects on the body (Pilozzi et al., 2020).

The endocannabinoid system is a complicated biological system involved in regulating movement, mood, memory, appetite, fertility, pain, and physiological homeostasis. It consists of cannabinoid receptors and cannabinoid receptor proteins that are active throughout both the central and peripheral nervous systems. Two endogenous molecules that activate the endocannabinoid system have been found. The first, 2-arachidonoyl glycerol (2-AG), occurs in peripheral tissues, while anandamide (Sanskrit for 'supreme joy') is a neurotransmitter. The psychoactive component of cannabis, Δ9-tetrahydrocannabinol (THC), mimics the actions of anandamide, whereas the main therapeutic component of cannabis, cannabidiol (CBD), mimics 2-AG (Mechoulan & Parker, 2013). Data from the past 20 years indicate that the endocannabinoid system plays a role in modulating the human sleep-wake cycle and can play a part in the decrease of sleep disturbance or insomnia through the restoration of sleeping and dreaming though the regulation of how this occurs is not yet fully understood (Kesner & Lovinger, 2020; Prospéro-García et al., 2016).

Norepinephrine, which is synthesized from dopamine, is released by the adrenal gland and acts not only as a neurotransmitter but is also a hormone. It is essential in the transmission of information in the sympathetic nervous system. In its hormonal form, it works in conjunction with adrenaline and epinephrine to boost the body under stressful situations, underlying the human fight-flight response: increasing heart rate, increasing blood flow to the muscles, and leading to the release of glucose from the body's energy stores. As a neurotransmitter, norepinephrine is crucial in maintaining general arousal and in maintaining normal sleep states (Katzung, 2020; Mitchell & Weinshenker, 2010).

Finally, there is serotonin. It has been referred to by some as the happiness transmitter. This neurotransmitter plays a role in reducing feelings of depression, alleviating anxiety, elevating mood, and increasing feelings of self-worth. It also impacts mood, appetite, and sleep as it is the chemical precursor to melatonin, the main hormone involved in inducing sleep. Increasing serotonin levels through the use of drugs as varied as Selective Serotonin Reuptake and 3,4-Methylenedioxymethamphetamine (MDMA) decreases REM sleep negatively impacting mental well-being (National Institute on Drug Abuse, 2008; Rao & Tripathi, 2022).

The sleep cycle

Figure 3 illustrates a typical 8 hr sleep cycle, though the reality is that few adult North Americans actually sleep eight consecutive hours on a regular basis. Sleep is divided into two segments: rapid eye movement sleep, which is when dreaming occurs, and non-REM (nREM) sleep, which is furthered separated into three distinct stages. The initial stages of the sleep cycle, stage one and two, are those from which we are most easily roused. During stage one, a person is in a twilight stage, half asleep and half awake. It is the stage when a person is relaxed, breathing is regular and gradually deepens, and one feels groggy. Stage one constitutes approximately 5% of the total duration of sleep on any given night. During stage two, which constitutes 40% to 50% of the sleep cycle, sleep deepens, and one is slowly removed from perceiving stimuli in the outside world. However, it is stage three, which was previously divided into stage three and four, which is the most important for our physical

recovery. Stage three is the deepest stage of sleep, where our brain activity is at its lowest (Sharma et al., 2018; Steiger, 2010). This stage is essential in physical recovery for humans as it is during this stage of the 24 hr cycle of waking and sleep when the greatest amount of physical restoration occurs in the human body. What is important to recognize is that even for people who have insomnia or who have short sleep durations, given when most of stage three occurs, their bodies are still able to engage in physical self repair. Reflect on the last time you had a cut and how long it took to heal. Other than putting a bandage on the cut and perhaps some antiseptic, the majority of the healing process is not the result of medication or treatment; rather, the vast majority of physical recovery is driven by the body itself. The greatest amount of healing and replenishment of body functioning occurs during stage three sleep. Upon entering stage three of the sleep cycle, brain activity becomes more synchronized, cells are firing far more in unison than when a person is awake. If awakened at this point in the sleep cycle, a person responds slowly and is usually momentarily confused and uncertain of their surroundings (McNamara, 2019; Steiger, 2010).

At the top of the sleep cycle is REM sleep, the opposite in brain activity to stage three. REM constitutes 20% to 25% of our time asleep. REM is the dreaming state where our mind is extremely active, replaying recent events, reliving past events, both pleasant and traumatic, and also recalling nonevents that, while they never occurred, arise in the sleep patterns of people universally. The first REM cycle begins about 90 min after a person has entered stage one of sleep. During REM sleep, the synchronized pattern that emerges in stage three is interrupted and brain wave patterns become desynchronized, looking much more like that of a waking state (Rué-Queralt et al., 2021; Xu et al., 2022). At this stage, muscles are very relaxed and for all intents and purposes you are paralyzed, though your eyes remain active. Although your muscles are not able to work, you still experience the sensation of moving while dreaming, which means that you feel the dream as though it were real. It is thought that eye movements during REM sleep are related to the scanning of the often vivid visual scene of a dream. In other words, we are looking at the surroundings in the dream, the same way we observe our surroundings when we are awake. During REM sleep the brain is more active than in waking and you can live entire days, weeks, and months during one dream cycle. However, to this time, the complexities of this sleep stage have led to little consensus surrounding dreams' functions and there remains a dearth of research regarding dream construction. Within cognitive and neurosciences, the dreaming that occurs during REM sleep is a virtual reality generator that attempts to predict its waking environment (McNamara, 2019). Other hypotheses posit that our brains are engaged in complex, dynamic dialogue in which integration and segregation of episodic and remote memory are negotiated (Llewellyn, 2013). Conversely, alternative assertions highlight REM sleep as an arguably necessary physiological and neurological function for procedural and emotional memory consolidation (Walker & Stickgold, 2004). While dreaming's precise role(s) has not been conclusively determined, our dreams are not meaningless or trivial. Rather, dreams and the REM sleep, which enables them, are potentially essential elements that help us integrate and process memories and make sense of events, including the traumatic episodes in our lives.

While at this phase of the sleep cycle one remains mostly disconnected from the outside world, a person is very easily roused if their name is called or if a loud sound is heard. During the course of 8 hr of sleep, a person can experience four or even five REM cycles, with each successive REM cycle lasting longer than the one before. When REM and nREM

sleep are disrupted, especially over a sustained duration, emotional regulation, energy stores, and immune response are diminished. Recovery/restorative sleep is the body's attempt to compensate for sleep deprivation. During this process of recovery sleep, however, NREM sleep is made up first, followed by REM (Sawai et al., 2021; Sharma et al., 2018). A healthy human being can go upwards of 60 days without food, whereas 10 days without sleeping or dreaming can be fatal. Likewise, disrupted sleep leads to a range of physical health issues, and if a person is allowed to sleep but not enter REM sleep, their mental health will rapidly deteriorate (Everson & Szabo, 2009; Everson, 1993; McNamara, 2019).

A critical take away is to understand that the resting state, the state of homeostasis for all humans, is one of anxiety. Our dreams provide us a protected opportunity to deal with real and perceived threats for the most commonly reported dreams can be categorized anxiety-related. Dreams fall into one of four categories:

(i) Recent history events; repeating or engaging in an event over and over;
(ii) Past history; historic events, people from our past, substantive events in our lives including trauma events;
(iii) Universal dreams; falling, flying, being chased, sexually explicit activity; and,
(iv) Unique dreams – distinct dreams involving anxiety issues not falling into the other categories (C. Yu, 2014, 2015).

Thus, while the perceived role of dreams and specificity of certain dream content is culturally determined, influenced by gender identity, and differs across the lifespan (McNamara, 2019), major themes or 'typical dreams' which contain similar content and are reported by high rates of dreamers have been noted in multiple studies (C. K. Yu, 2015, 2016; Mathes et al., 2014).

Sleep disorders

Despite the knowledge that deep, restful sleep is essential to physical, psychological, and immunological wellbeing, sleep disorders are often inadequately assessed and treated, especially as they relate to mental health. The secondary nature and comorbidity of many sleep disorders, such as insomnia, also present a challenge of diagnosis and causation. While sleep disorders can be classified into two broad categories of dyssomnias and parasomnias, the International Classification of Sleep Disorders-3 (ICDS-3) lists seven Major Diagnostic Sections (Sateia, 2014).

Dyssomnias are disorders wherein a person experiences changes to sleep duration and sleeps too much or too little. Of the numerous sleep disorders, which fall under the dyssomnia category, insomnia, the difficulty of initiating or staying asleep, is the most common and has been classified as a North American public health crisis. Over 95% of the studied population have claimed to have experienced at least one period of insomnia during their lifetime (Sateia, 2014). With the exception of Fatal Familial Insomnia,[1] there exists no consensus on any other intrinsic or primary insomnia. Secondary insomnia, often referred to as chronic insomnia disorder, does not involve intrinsic sleep-wake neurological systems that regulate transitions into and out of sleep, rather, it is typically linked to medical

[1]A rare genetic condition that causes progressively worsening insomnia.

conditions, psychological issues, or everyday anxiety and work-related stress. Parasomnias are disruptions of behavior and consciousness during sleep, typically occurring between states, including sleep to waking, or REM to nREM sleep. Table 1 provides a summary of the ICDS-3 Major Diagnostic Sections and the affiliated sleep disorders.

How psychoactive drugs disrupt sleeping and dreaming

Overview

Psychoactive drugs decrease, increase, or disrupt activity in the central (CNS) and peripheral, including the autonomic, nervous systems. Depressants produce a reduction of arousal and activity in the CNS. These drugs have long been used therapeutically as anesthetics and aids for sleeping as well as anti-anxiety agents, and sedatives. Depressants produce their effect on the brain and thus on the person by enhancing the existing GABA in the brain. Drug groups that fall into this family are barbiturates, benzodiazepines including Z-drugs (zaleplon, zolpidem, zopiclone, and eszopiclone), inhalants and solvents, alcohol, and antihistamines though this latter group alters histamine rather than GABA. Opioids are a specific subgroup of CNS depressants. The distinct attribute that differentiates these psychoactive agents from other within this category is their ability to mask pain and also to suppress cough but as a prominent side effect also produce sedation by mimicking endorphins. In contrast, stimulants including cocaine, amphetamines, and methamphetamine produce a general increase in the activity of the cerebral cortex, creating mood elevation, increased vigilance, and the postponement of fatigue. Some stimulants are also used as appetite suppressants and decongestants, and to treat attention-deficit/hyperactivity disorder (ADHD). These drugs produce changes through their effect primarily upon dopamine but also serotonin and norepinephrine. Two of the world's most commonly used psychoactive drugs fall within the stimulant family of drugs: nicotine and caffeine. Hallucinogens affect the CNS in a much different manner than do depressants and stimulants. Hallucinogens produce a generalized disruption in the brain, especially of perception, cognition, and mood. Several, such as ecstasy, have secondary CNS stimulant effects. Dissociative anesthetics including ketamine and PCP (phencyclidine) have an associated pharmacological effect more closely associated with CNS depressants. Cannabis, which some group with hallucinogens, though pharmacologically it is a truly unique psychoactive agent, also has associated CNS depressant effects. The withdrawal effects of psychoactive drugs produce equal and opposite effects to their use. Thus, if a person were using a psychoactive substance to relax or sleep, the expected withdrawal reaction would be agitation and insomnia. Likewise, if a person were using an energy drink to stay awake, withdrawal would be extended and even excessive tiredness.

Depressants

Synthesized depressants including barbiturates and benzodiazepines were created in part with the goal of aiding sleep. These drugs, that with chronic use produce both a physical and psychological dependency, are also able to relieve anxiety, tension, and convulsions by producing calmness and muscular relaxation through their inhibition of GABA. However, individuals not requiring these drugs for therapeutic use experience a significant sense of

Table 1. Summary of Major Diagnostic Sections and Related Sleep Disorders (Sateia, 2014).

Major Diagnostic Sections of ICDS-3	Related Sleep Disorders
Insomnia	• Chronic insomnia disorder • Short-term insomnia disorder • Other insomnia disorder
Sleep-related breathing disorders	**OSA disorders** • OSA, adult • OSA, pediatric **Central sleep apnea syndromes** • Central sleep apnea with Cheyne-Stokes breathing • Central sleep apnea due to a medical disorder without • Cheyne-Stokes breathing • Central sleep apnea due to high altitude periodic • breathing • Central sleep apnea due to a medication or substance • Primary central sleep apnea • Primary central sleep apnea of infancy • Primary central sleep apnea of prematurity • Treatment-emergent central sleep apnea **Sleep-related hypoventilation disorders** • Obesity hypoventilation syndrome • Congenital central alveolar hypoventilation syndrome • Late-onset central hypoventilation with hypothalamic dysfunction • Idiopathic central alveolar hypoventilation • Sleep-related hypoventilation due to a medication or substance • Sleep-related hypoventilation due to a medical disorder **Sleep-related hypoxemia disorder**
Central disorders of hypersomnolence	• Narcolepsy type 1 • Narcolepsy type 2 • Idiopathic hypersomnia • Kleine-Levin syndrome • Hypersomnia due to a medical disorder • Hypersomnia due to a medication or substance • Hypersomnia associated with a psychiatric disorder • Insufficient sleep syndrome
Circadian rhythm sleep-wake disorders	• Delayed sleep-wake phase disorder • Advanced sleep-wake phase disorder • Irregular sleep-wake rhythm disorder • Non-24-h sleep-wake rhythm disorder • Shift work disorder • Jet lag disorder • Circadian sleep-wake disorder not otherwise specified
Parasomnias	**nREM-related parasomnias** • Confusional arousals • Sleepwalking • Sleep terrors • Sleep-related eating disorder **REM-related parasomnias** • REM sleep behavior disorder • Recurrent isolated sleep paralysis • Nightmare disorder **Other parasomnias** • Exploding head syndrome • Sleep-related hallucinations • Sleep enuresis • Parasomnia due to a medical disorder • Parasomnia due to a medication or substance • Parasomnia, unspecified

(*Continued*)

Table 1. (Continued).

Major Diagnostic Sections of ICDS-3	Related Sleep Disorders
Sleep-related movement disorders	• Restless legs syndrome • Periodic limb movement disorder • Sleep-related leg cramps • Sleep-related bruxism (teeth grinding) • Sleep-related rhythmic movement disorder • Benign sleep myoclonus of infancy • Propriospinal myoclonus at sleep onset • Sleep-related movement disorder due to a medical disorder • Sleep-related movement disorder due to a medication or substance • Sleep-related movement disorder, unspecified
Other sleep disorders	Any sleep disorder that does not meet the criteria of ICDS-3's existing list of disorders

euphoria when self-administering. Barbiturates, which are no longer prominently dispensed or used illicitly, disrupt both the sleep and dream cycles; thus, while people often took these drugs to aid in sleeping, it is now known that they do not produce normal sleep patterns, with users feeling tired and irritable upon waking, regardless of what early advertising of the drugs promoted. Barbiturates are among the few psychoactive agents that are not readily flushed from the human body, as they accumulate in body fat. With regular use, tolerance develops to their effects; it develops more slowly to the harmful effects than to the sleep-inducing or intoxicating effects. At high doses or when mixed with other CNS depressants, particularly alcohol, respiratory depression and/or cardiovascular depression leading to death are possible. Temporary sleep disturbances may lead a user to incorrectly decide that more of the drug is required, also leading to risk of overdose. There is a high cross-tolerance between barbiturates and other depressants, particularly alcohol (Hancock & McKim, 2018).

As with barbiturates, chronic use of benzodiazepines leads to both physical and psychological dependency. Withdrawal symptoms from benzodiazepines are similar to other sedative-hypnotics: tremors, sweating, hypersensitivity to sensory stimuli, blurred vision, tingling sensations, tinnitus, headache, difficulties in concentration, anorexia, increased lethargy, indifference to one's surroundings, memory, cognitive, and psychomotor impairment, irritability and emotional flatness, disorientation and confusion, sleep disturbances, gastrointestinal upsets, sexual dysfunction, and menstrual irregularities. However, among the most important withdrawal effects, given that these drugs are often used to aid in sleeping, is insomnia. In fact, during withdrawal insomnia is intensified and prolonged as the body attempts to return to a normal homeostatic balance. Benzodiazepines impact all activities of the brain associated with the sleep cycle. In general, while they induce stages one and two sleep in the sleep cycle, benzodiazepines reduce the amount of time a person spends in stage three. Their major negative effect, however, is suppressing REM sleep (Cascade & Kalali, 2008; Pagel & Parnes, 2001). For example, Xanax has been demonstrated to drastically reduce the amount of time spent in stage three sleep and produces delays in moving into REM. In one study, during the first three nights, use of Xanax led to rapid sedation and prolonged sleep throughout the night. However, by the end of the first week of regular use, the drug had lost about 40% of its efficacy, and within 3 days of drug termination, there was a significant degree of rebound insomnia (Kales et al., 1987). Lorazepam has been shown to

decrease stage one sleep and increase stage two sleep while having no impact on stage three; however, it significantly decreases the amount of time spent in REM sleep, affecting the mind's ability to process memory (Roth et al., 1980).

Cyclopyrrolones, otherwise known as Z-drugs, while pharmacologically distinct from benzodiazepines, still bind to GABA receptors. The most prominent feature of Z-drugs is that they decrease sleep latency and increase the duration of sleep. The duration of stage one sleep is shortened, while the time spent in stage two sleep is typically increased. In most studies, stage three sleep tended to be increased, but no change and actual decreases was observed in clinical trials. The effect of zopiclone on stage three sleep differed from that of traditional benzodiazepines, which suppress slow-wave sleep. While the onset of REM sleep was delayed this drug group does not appear to reduce the total duration of REM sleep according to pharmaceutical agency sponsored studies (Aventis, 2018; Pharm, 2009). However, in 2014 Health Canada placed a recommended dosage limitation on the use of zopiclone due to the adverse behaviors its users were experiencing, including impairment lasting for up to 2 days. Amnesia can result from using this drug, as highlighted by reports of people getting out of bed, while not fully awake after taking cyclopyrrolones and unknowingly engaging in activities, with no memory of the action the next day (Aventis, 2018).

Antihistamines, first used therapeutically in the 1940s, constitute a diverse group of drugs that can inhibit histaminic actions in the body. They are structurally similar to histamine, a chemical found throughout the human body, and act to prevent histamine–receptor interaction through competition with histamine for histamine receptors. The primary function of antihistamines is to aid with allergies, excessive stomach acid, and motion sickness. Antihistamines can be used as sleep-inducing agents as they can produce sedation; however, they do not produce high-quality sleep. This leads to more time spent in stages one and two sleep and less in stage three. Older generation antihistamines also reduce REM sleep, impair learning, and reduce work efficiency (Church et al., 2010; Raman-Wilms, 2014).

Another distinct CNS depressant subgroup is inhalants, which include volatile gases, substances that exist in a gaseous form at body temperature, refrigerants, solvents, general anesthetics, and propellants. Except for nitrous oxide, more commonly known as laughing gas, and related aliphatic nitrates, all inhalants are hydrocarbons. In the case of anesthesia, brain arousal is deactivated significantly and cortical and thalamic neurons become activated, just as in slow-wave sleep. Both integration capacities and information processing are diminished through the use of inhalants as they block the interactions between specific brain regions (Alkire et al., 2008). In two Algerian-based studies, it was observed that individuals with high levels of occupational exposure to solvents reported poorer sleep quality than non-exposed workers, as well as higher rates of reported irritation, depression, and memory loss (Hurley & Taber, 2015; Sekkal et al., 2016). Further, increasing solvent exposure through the lifespan is correlated with sleep-related respiratory issues and apneic episodes (Doran and Aschengrau, 2022;Viaene and Godderis, 2008).

The most commonly used CNS depressant, alcohol, when consumed in adequate amounts creates drowsiness and sleep; however, it is not restful sleep as alcohol disturbs the natural sleep cycle of users. There is a bidirectional association between alcohol use and sleep, as alcohol creates sleep disturbances, but likewise sleep disturbances lead to a greater use of alcohol with difficulty in sleeping cited as a risk factor for relapse to substance use.

Alcohol affects sleep by disrupting the rhythm of both alpha and delta waves, leading to inadequate stage three sleep that can persist for upwards of 2 years for those who had been physically dependent for extended periods of time. As well, when an alcohol-dependent person stops drinking, the withdrawal symptoms that they typically experience include restlessness and sleeplessness (Dziegielewski, 2005; Hasler et al., 2012).

As stated previously, the common effects of opioid administration beyond pain masking are drowsiness, sedation, dizziness, light-headedness, mood swings from euphoria to dysphoria, depressed reflexes, altered sensory perception, stupor, and the potential for a drug-induced coma. Opioids, despite producing sedation among users, actually produce interrupted sleep patterns. Many opioid receptors are located in the same part of the brain that is responsible for sleep regulation. Natural endorphins are believed to be involved in the induction and maintenance of one's sleep state. The ongoing activation of endorphin responses in the brain through regular opioid use increases the number of shifts in sleep-waking states while decreasing total sleep time, sleep efficiency, delta sleep, and time spent dreaming. Even a single dose of a potent opioid can reduce stage three sleep, contributing to opioid-produced fatigue. Chronic opioid use also decreases the total time spent in REM sleep (D. Wang & Teichtahl, 2007; Dimsdale et al., 2007). Many heroin users experience hypersomnia, the state of being excessively sleepy during the day even after a prolonged night's sleep. This occurs because heroin use increases stage one sleep while diminishing stage two sleep and decreasing the time spent in REM sleep (Liao et al., 2011). Similarly, morphine increases light sleep (stage two) but decreases deep sleep (stage three) and dreaming (Q. Wang et al., 2013). Methadone also reduces the length and amount of REM sleep cycles and delays the initial REM episode, each of which detrimentally affects the ability to dream. Additionally, by increasing awakenings throughout sleep, methadone causes more light sleep, disrupting a person's ability to achieve slow-wave, restorative stage three sleep. With sustained use and steady dosing, methadone users develop a tolerance to these sleep patterns. If a person is involved in a methadone treatment protocol rather than methadone maintenance and methadone use is safely ended, REM and sleep-quality abilities are restored to normal after an extended period (Hsu et al., 2012).

Stimulants

The stimulants amphetamines and methamphetamine act upon the body similarly to adrenaline. They are chemically related to norepinephrine and dopamine and are used to raise energy levels and reduce appetite and the need for sleep, while providing feelings of clearheadedness and power. Amphetamines work by increasing synaptic levels of primarily dopamine, but also serotonin (5-HT) and norepinephrine. Amphetamine and methamphetamine are white, odorless, bitter-tasting crystalline powders whose use can lead to memory loss, aggressive behavior, violence, and paranoid and psychotic behavior when misused. Part of this behavior is due to the fact that the use of these psychoactive agents allows individuals to remain awake for extended periods of time, thus not needing to sleep. The lack of REM sleep, and thus the lack of dreaming, is associated with paranoid and psychotic behavior. As the drug is metabolized, euphoria is replaced with dysphoria, restlessness, agitation, nervousness, paranoia, violence, aggression, lack of coordination, pseudo-hallucinations, delusions, psychosis, and drug craving, while physically, as fatigue brings sleepiness with sudden starts, itching, picking, and/or scratching can occur. Even a single

morning oral administration of methamphetamine can produce robust disruptions in nighttime sleep, with larger amounts creating greater issues (Herrmann et al., 2017). Unfortunately, the use of even minor amphetamines such as methylphenidate (Ritalin and Concerta) typically given to children and youth diagnosed with ADHD can delay and reduce REM sleep periods (Sangal et al., 2006).

Cocaine, while acting in a distinct pharmacological manner than amphetamine and methamphetamine, still alters the same neurotransmitters in the brain, only by blocking reuptake rather than pushing more dopamine, serotonin, and norepinephrine into the synaptic cleft. Thus, cocaine use and withdrawal both affect sleep and the human circadian clock (Stowie et al., 2015). A single administration of cocaine will suppress REM sleep, though rebound with increased REM occurs if the use is acute rather than chronic. However, for those who had become dependent upon cocaine a substantive decrease in short-wave stage three sleep has been observed. Regular cocaine users' behavior has been equated to individuals with chronic insomnia, with both groups having difficulty falling asleep, both at night and during the day. Loss of sleep depth produces poor cognitive and motor performance and further increases the risk of cardiovascular disease, hypertension, and infectious disease, as sleep depth contributes to homeostatic regulation of the autonomic, neuroendocrine, and immune systems. Time spent sleeping is lessened with cocaine use but increases post-initial withdrawal to compensate for sleep deprivation that occurred while the person was using the drug (Irwin et al., 2016; Morgan & Malison, 2007; Schierenbeck et al., 2008).

All CNS stimulants negatively affect sleep, but nicotine is particularly deleterious to sleep quality. Given nicotine's short half-life, smokers wake earlier on average than non-smokers to address withdrawal. This may be in part responsible for the association between nicotine dependency severity and poor sleep outcomes, shorter sleep duration, and excessive daytime sleepiness among tobacco smokers. Both tobacco smokers and smokeless tobacco users have twice the odds of insufficient sleep compared to nonsmokers, while individuals exposed to secondhand smoke also obtain less sleep on average and have greater sleep disruption than do nonsmokers not exposed to secondhand smoke (Branstetter et al., 2016; Caviness & Anderson, 2018; Sabanaygam & Shankar, 2011).

When taken even in moderate amounts, such as one or two cups of brewed or percolated coffee, caffeine can produce stimulant effects upon the central nervous system similar to those of small doses of amphetamines. These can include mild mood elevation, feelings of enhanced energy, an increased alertness and reduced performance deficit due to boredom or fatigue, postponement of feelings of fatigue and the need for sleep. Small doses of caffeine alter sleep patterns including delaying the onset of sleep, diminish sleep time, and reduce the depth of sleep, including altering REM patterns, while also increasing respiration, blood pressure, and metabolism. Given caffeine's half-life, those experiencing any sleep disturbances should not consume caffeine within 10 hr of going to bed (Drake et al., 2013).

Hallucinogens

Hallucinogens can be placed into three distinct pharmacological families: indolylalkylamines (DMT, LSD, and psilocybin), phenylethylamines (Ibogaine, MDA, and MDMA), and arylcycloalkylamines (ketamine, PCP, and salvia divinorum). Drugs in this family produce sensations of separation from self and reality, as well as unusual changes in thoughts,

feelings, and perceptions, including delusions and illusions but not typically delirium. Illusions and delusions may include a loss or confusion of body image, altered perceptions of colors, distance, and shape, and an apparent distortion, blending, or synthesis of senses whereby one sees sounds and smells colors (Hancock & McKim, 2018). Low doses of LSD administered 1 hr prior to going to sleep increase initial REM periods but then lead to shortened subsequent periods, the opposite pattern to normal REM sleep. Test subjects also had less eye movements during REM sleep when using LSD compared to non-drug-using sleep periods, though overall time spent in REM sleep was longer when given LSD (Passie et al., 2008). Psilocybin can place a person into a similar state as during REM sleep that has been called a waking dream state while DMT does not induce any deterioration of sleep quality, initiation, or maintenance, though it does inhibit REM sleep, decreasing its duration both in absolute values and as a percentage of total sleep time (Barbanoj et al., 2008).

Unlike the indolylalkylamines which only produce hallucinogenic effects, the phenylethylamines also have secondary stimulant effects not only altering serotonin but also norepinephrine and dopamine, though to a lesser degree than drugs that amphetamines. DOM (2,5-Dimethoxy-4-methylamphetamine), also known as 'STP' (serenity, tranquility, and peace), can include sleeplessness, dry mouth, nausea, blurred vision, sweating, flushed skin, and shaking, along with exhaustion, confusion, excitement, delirium, and convulsions, which also impact sleep. MDMA can cause the acute depletion of presynaptic serotonin which play a direct role in regulating aggression, mood, sexual activity, sensitivity to pain, and sleep. MDMA users typically experience restless, disturbed sleep for up to 48 hr following drug use. Total sleep time has been found to be reduced, with increased time spent in transition, stage one sleep and less time in stage two. While there was no reported change in REM sleep, there was an increased risk of sleep apnea with continued use of MDMA (McCann et al., 2009; Schierenbeck et al., 2008).

Ketamine, a glutamate receptor antagonist, is used clinically, primarily in developing nations, as an anesthetic though more recently has become popular as an anti-depressant for treatment-resistant individuals. Thus, it, like PCP, has potent sedative effects that can not only disorientate a user but also induce sleep. When ketamine is used in surgery, recovery tends to be slower than when other anesthetics are employed (Schwenk et al., 2018). Violent dreams and flashbacks have been associated with both clinical and non-medical use of the drug. In larger doses, the 'K-hole' effect occurs, a distinct feeling of mind and body separation that in severe circumstances can lead to stupor or unconsciousness, with a resulting feeling of confusion and loss of short-term memory. Some have equated this to an out-of-body or near-death experience. Ketamine use increases nREM intensity and duration, and while it does not increase or decrease REM sleep, ketamine use does tend to produce more vivid and violent dreaming (Feinberg & Campbell, 1993; Hejja & Galloon, 1975).

Cannabis

While traditionally associated with hallucinogens cannabis is a unique pharmacological psychoactive agent in that unlike hallucinogens it does produce physical dependency. At low to moderate doses, the effects of cannabis products are somewhat similar to those of alcohol: relaxation, disinhibition, a feeling of euphoria, and the tendency to talk and laugh more than usual. THC appears to deepen sleep and allows the user to fall asleep faster,

increasing slow-wave sleep. However, as cannabis dose amounts increase, the amount of time spent in REM sleep decreases, and once nightly cannabis use ends, sleep patterns deteriorate for those who were physically depending upon the drug during the withdrawal period (Angarita et al., 2016). For acute users of cannabis, circadian rhythm is deregulated by cannabinoids but not by THC, which is consistent with clinical observations of the short-term use of therapeutic cannabis to treat insomnia (Lafaye et al., 2018). For physically dependent users, withdrawal symptoms can begin in as little as 4 to 8 hr after the abrupt termination of drug administration and consist of irritability, anxiety, depressed mood, restlessness, anger, sleep disturbances, and insomnia. Symptoms can begin to fade within 48 hr of abstinence as receptors rebound to normal functioning, returning within 1 month of abstinence. Likewise, sleep disturbances can last three to 4 weeks after cessation of drug use before returning to homeostatic levels (Bonnet & Preuss, 2017; Zehral et al., 2018).

Discussion

Sleep remains essential to wellbeing, mental health, growth, and healing (McNamara, 2019). The impact of too little or poor sleep has a profound effect on overall well-being and negatively impacts emotional regulation (Drapeau & Nodorff, 2017; Sisman et al., 2021) and impairs daily functioning (Magnée et al., 2015). Yet, sleep disorders remain difficult to assess, diagnose, and treat due to comorbidity (McNamara, 2019). This article has provided an overview of the interplay between sleep, in particular, REM sleep in which dreaming occurs, and substance use. Specifically, the use of substances largely impairs REM sleep, thereby inhibiting the body's capacity to function optimally (Angarita et al., 2016; Barbanoj et al., 2008; Irwin et al., 2016). This has significant implications for social workers in their ongoing interactions with service users.

Sleep and substance use co-exist in a complicated bi-directional relationship. In general, sleep quality and hygiene are affected by psychoactive substances in a negative fashion, regardless if the substance(s) being used are CNS depressants, stimulants, hallucinogens, or cannabis. Research has shown that sleep disorders can be attributed to the etiology of substance use in that persons experiencing insomnia are at a higher risk for substance misuse. However, sleep disturbances and disorders may also be the result of substance use due to alterations to brain chemistry and sleep architecture (Aja et al., 2016; Angarita et al., 2016) yet treatment for co-occurring disorders are limited. Sleep disorders are interwoven with substance use at all stages: active use, entering treatment, exiting treatment, and recovery. Moreover, sleep disorders and disturbances, especially insomnia, are not only persistent for persons who are actively engaged in substance use but also those engaged in substance use treatment programs (Neale et al., 2019; Schick et al., 2022). While the desire to improve sleep quality and hygiene for persons in substance use treatment programs is common, this objective is challenged by numerous factors that vary from individual to individual. For instance, substance use can alter circadian rhythms, sleep stage physiology, and sleep regulation processes. Sleep quality is also affected by social, emotional, and environmental elements which are nuanced and can be difficult to control (Neale et al., 2017, 2019). Moreover, Colvonen et al.'s, (2019) study demonstrated that sleep indices did not improve over the course of SUD treatment, reinforcing the need for sleep-based interventions. Further, the interaction of drug use and sleep is typically not a primary topic in addiction

treatment protocols. This is especially relevant as research has shown that withdrawal from substances, many of which have been used, at least initially and/or in part, to serve as sleep aids, can be linked to prolonged sleep onset latency, difficulty staying asleep, and decreased total sleep time (Schick et al., 2022). Yet, the ability to fall asleep, stay asleep, and transition between sleep stages are all necessary to overall mental health and SUD management. Thus, the potential for sleep-centered interventions, measured with a standardized set of validated measurements such as the Pittsburgh Sleep Quality Index (PSQI) or Substance Use Sleep Scale (SUSS), as part of inpatient or community-based programs could play an important and as-yet missing part of substance use treatment (Brooks & Wallen, 2014; Neale et al., 2019; Wilkerson et al., 2021). Unfortunately, there still exists relatively little research regarding the impact of sleep and engagement in recovery, management, and early abstinence, especially amongst individuals in early treatment for substance use disorder (Ara et al., 2016; Buckheit et al., 2022; McHugh Hu et al., 2014; Schick et al., 2022; Wilkerson et al., 2021).

The bidirectional nature of substance use and sleep, the impact of sleep on treatment and recovery, as well as the necessity of sleep and dreaming to mental health and maintenance of quality of life, merit further integrated exploration and study. Moreover, each person's lived experiences and relationships to substances is nuanced and may exist alongside numerous comorbidities, all of which may influence sleep architecture. It is this dialectic of sleep disruptions and substance use, compounded by a person's unique relationships to sleep, which highlights the need for further study toward treatment options which incorporate evidence-informed, sustainable, and person-centered interventions which consider the role of sleep in supporting immediate and longer-term recovery.

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No potential conflict of interest was reported by the authors.

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