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Sleep Disturbance in Substance Use Disorders

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Introduction

Drugs of abuse and alcohol have disruptive effects on sleep, in particular, interfering with the ease of falling asleep, increasing the difficulty in maintaining sleep, and altering the cycling of sleep stages from non-rapid eye movement (NREM) sleep to rapid eye movement (REM) sleep. These sleep effects then have a consequent impact on next-day function, including increasing daytime sleepiness and impairing alertness. The sleep and daytime sleepiness/alertness disturbances can be seen both during active substance use and during discontinuation of use. The specific characteristics of the sleep/alertness disturbances for various substances of abuse have been reviewed elsewhere and the reader is directed to these reviews for substance-specific information (1, 2). The purpose of this chapter is to discuss the modulatory role these sleep disturbances may play in substance use disorders (SUD).

It has been suggested that these sleep and alertness alterations, although not the primary reinforcing mechanism, function as contributing/modulatory factors in initiating and maintaining drug and alcohol abuse and as factors that increase the risk for relapse. After briefly discussing diagnostic and measurement issues, this chapter will discuss the role these sleep and alertness alterations play in initiating and maintaining alcohol and substance abuse and in relapse. We also discuss the extent to which treatment of sleep/alertness disturbances impact the risk of relapse. Finally, we briefly make note of the common hypothesized neurobiological pathophysiology underlying insomnia and SUD.

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Diagnostic Issues

Sleep Disturbances

The two diagnostic systems for sleep disturbances are the *International Classification of Sleep Disorders, third edition* (ICSD3) of the American Academy of Sleep Medicine and the *Diagnostic and Statistical Manual of Mental Disorders, fifth edition* (DSM-5) of the American Psychiatric Association (3, 4). The most common sleep diagnosis associated with SUD is insomnia. Both ICSD3 and DSM-5 define insomnia as difficulty initiating, maintaining sleep, and/or awakening early in the morning that occurs on three or more nights per week, endures for three months or more and causes significant impairment in daytime functioning. Importantly, the sleep disturbance is present despite an adequate opportunity and circumstance to sleep. The diagnostic systems diverge slightly in classifying sleep disturbance in substance use disorders. In DSM-5, insomnia is classified as co-morbid with the specific SUD if it occurs beyond the immediate substance use (i.e., prior to and after the discontinuation of substance use), while in ICSD3 one of the listed subtypes of insomnia is “insomnia due to drugs or substances”. Note that the ICSD3 makes a somewhat stronger causal inference regarding the relation of sleep/alertness disturbance to substance use.

Substance Use Disorders

The generally accepted diagnostic classification system for substance-related disorders is the DSM-5. DSM-5 reflects a major departure from DSM-IV and DSM-IVR with regard to the diagnosis of SUD (5). Substance-related disorders are divided into two major classes; 1) Substance Use Disorders and 2) Substance-Induced Disorders. Substance Use Disorders (SUD), formerly termed substance abuse and substance dependence (DSM-IVR), are characterized by behaviors and consequences (a total of 11 criteria are listed) in groupings that show *impaired control, social impairment, risky use, and pharmacological consequences* (i.e. tolerance, withdrawal) associated with the use of 10 classes of substances. Rather than distinguishing abuse and dependence as in DSM-IVR, in DSM-5 the severity of the disorder is rated by the number of symptoms present: mild 2-3, moderate 4-5, and severe 6. Substance-Induced Disorders (SID) are characterized by symptoms reflecting the presence of intoxication, withdrawal, or a mental disorder. As this category name implies, the disorder has to be associated with current or very recent use of the substance.

While most of the drugs of abuse are disruptive of sleep and/or daytime alertness, such disturbances are not major criteria for SUD in DSM-5. They are mentioned as possible symptoms in a withdrawal syndrome, which is one of the eleven criteria for a SUD. DSM-5 emphasizes that symptoms of tolerance development and withdrawal, occurring only in the context of medical treatment (i.e., prescribed medications), should not be diagnosed as SUD (distinguishing drug-seeking from therapy-seeking in clinical practice is discussed later). Also as discussed later in this chapter, the role of disruptions of sleep and daytime alertness in SUD is not elaborated in DSM-5. One of the 11 major diagnostic criteria is “repeated attempts to quit/control use” (i.e., relapse). Some of the strongest evidence regarding the role of sleep disturbance in substance abuse relates to the associated increased risk of relapse (6)

Assessing Sleep and Daytime Alertness Disturbances

It should be noted that in both diagnostic systems, insomnia is a symptom based diagnosis. Objective markers such as polysomnographic (PSG) measures of sleep and sleep stages are not part of the diagnostic criteria. PSG is the term used to refer to the continuous recording of multiple physiological parameters during sleep that differentiate sleep and wake and the two distinct sleep states NREM and REM sleep. Among insomnia patients, PSG defined sleep often does not reflect insomnia symptoms (7). On the other hand, as noted above, in insomnia co-morbid with SUD, PSG-defined disturbances of sleep, or more specifically alteration of sleep stages, have been shown for most all of the substances with abuse liability and have been shown to predict relapse (i.e., slow wave sleep or REM sleep disturbance) (6).

An important diagnostic criterion for insomnia is that the sleep disturbance “causes significant impairment in daytime functioning”. One of the most consistent findings of daytime impairment associated with insomnia is the presence of elevated latencies on the Multiple Sleep Latency Test (MSLT) (7). The MSLT uses standard PSG technology and scoring to assess the level of daytime sleepiness/alertness, calculated as the average time to fall asleep on 4-5 sleep opportunities conducted at two-hour intervals across the day. People with insomnia have unusually high MSLT latencies compared to healthy volunteers, particularly given their disrupted and reduced nocturnal sleep times (7). In healthy volunteers, reducing sleep time and disrupting sleep continuity reduces MSLT latencies (7). MSLT-elevation (i.e., hypervigilance) is consistent with neuroimaging, sympathetic nervous system, and hypothalamic-pituitary-adrenocortical (HPA) axis monitoring findings that all show the presence of a state of “hyperarousal” associated with insomnia that is present across the night and day (7). Later we discuss the role “hyperarousal” may play in initiating and maintaining SUD.

An important qualification in diagnosing insomnia is that there be adequate opportunity and circumstances for sleep. That implies that there be adequate bedtime to express the full duration and complement of sleep stages. NREM and REM sleep cycle across the night in 90-120 min cycles. However, the relative presence of NREM and REM are not proportionally distributed over those cycles. The majority of slow wave sleep occurs during the first two cycles and the majority of REM sleep occurs during the last two cycles. Reducing sleep opportunity experimentally or by allowing patient/subject determination of the end of the sleep period may artificially reduce the amount of REM sleep. This is particularly important given that one prominent sleep disturbance in SUD is early awakening with difficulty returning to sleep. Notably, a prominent effect of alcohol and many abused substances involves the suppression of REM sleep followed by a rebound and fragmentation of REM sleep during discontinuation (6).

Distinguishing Therapy-seeking and Drug-seeking

The DSM-5 criteria for SUD involve an important caution that tolerance development and withdrawal symptoms occurring only in the context of medically indicated use of a prescribed drug should not lead to a SUD diagnosis. The sleep-specific signs during

discontinuation may include REM sleep rebound or rebound insomnia, as many of the substances of abuse and alcohol alter these sleep parameters (6). This rebound may lead to therapy seeking, making it difficult to differentiate drug-seeking from therapy-seeking.

In drug-seeking, the drug and its effects, typically its “euphorogenic” effects, are the focus of the drug use, while in therapy-seeking, the alleviation of the disease-related symptoms is the focus of the drug use. However, in the clinic, drug-seeking and therapy-seeking can become closely intertwined and what was once therapy-seeking can shift to drug-seeking. The challenge is to differentiate the two behaviors in making diagnoses and appropriately treating patients. The defining characteristic of drug seeking is evidence that the drug is taken in excessive amounts, in non-therapeutic contexts, and is preferred over other commodities (e.g. money) and various social and occupational activities. The degree to which that the drug is chosen over other commodities or social activities provides evidence supporting its risk for abuse. The scientific literature for most drugs of abuse indicates that the drug is readily discriminated from placebo by behavioral and subjective assessments, which include ratings of the drug for its “euphorogenic” and drug-liking effects (8). That is, the drug's mood effects are the focus of its use.

In contrast, therapy seeking is evident if the drug has demonstrated efficacy for the disorder or condition being treated. Therapy seeking is also supported if the patient has the signs and symptoms of the appropriate diagnosis for the indicated use of the drug. The pattern of drug taking, including its dose, timing, and duration of use, should be consistent with its therapeutic effects. If the drug is no longer effective, its use is discontinued. Evidence supporting therapy seeking behavior also includes that the patient believes that the drug is effective and readily experiences its therapeutic benefits.

The drug seeking versus therapy seeking distinction becomes difficult in situations where therapy seeking shifts to drug seeking behavior. As noted earlier, while pre-sleep alcohol use may initially be effective in improving sleep onset for someone with insomnia, rapid tolerance development is likely, which may lead to dose escalation. Further, other of alcohol's reinforcing effects (i.e., its “euphorogenic” effects) may be discovered by the person, especially as dose is escalated. At that point, its use may extend beyond the therapeutic context (i.e., solely before sleep as a sleep inducer). A similar shifting pattern can be described for stimulant or opiate use. On the other hand, drug seeking may be maintained because the drug, in addition to its mood altering and “euphorogenic” effects, also has therapeutic effects (i.e., the stimulant effects of cocaine or amphetamine do in fact reverse the excessive sleepiness that is experienced during drug discontinuation). Thus, the dependence is maintained by a combination of its mood altering effects and its therapeutic effects, a circumstance that has been referred to as “self-medication”.

Alertness/Sleep Disturbance and Initiation of Substance Abuse

Tetrahydrocannabinol (THC), sedative-hypnotics, and alcohol may become reinforcers and lead to substance abuse through their capacity to induce sleep in persons with insomnia or to reverse a waking “hyperaroused” state. Volunteers with DSM-IVR diagnosed insomnia and PSG-determined sleep disturbance, but no history of alcoholism or drug abuse, when given

an opportunity to choose between previously experienced color-coded alcohol and placebo beverages at night before sleep, chose alcohol while healthy volunteers with a similar level of self-reported social drinking chose placebo (9). At baseline, the insomniacs had less slow wave sleep than the age- matched volunteers and the alcohol normalized their slow wave sleep. After six consecutive nights of the same alcohol dose, tolerance developed to the sleep effects. When given an opportunity to self-administer multiple doses before sleep after six nights of prior alcohol exposure, the insomniacs increased the dose compared to insomniacs randomized to take placebo for six nights (10). A number of questions arise from these studies including:

1. would the insomniacs continue dose escalation in the face of the loss of hypnotic efficacy?
2. would the hypnotic use lead to increased social drinking?
3. would other of alcohol's effects such as its “euphorogenic” effects be discovered with increased dose or daytime use?

Studies have also assessed characteristics of sleep and responses to sleep challenges that reflect sleep regulatory processes in people with alcoholism or at risk for alcoholism. In children with a positive parental history of alcoholism compared to those that are family history negative, spectral analyses of the sleep electroencephalogram (EEG) revealed less power in the slow wave sleep frequency bands and lower spindle frequency ranges (11). In light of the fact that sleep is regulated by homeostatic processes, one approach to assessing the sleep homeostat is to assess slow wave activity (SWA) in response to sleep challenges such as sleep restriction or sleep deprivation (12). A study compared SWA in alcohol-dependent adults to age-matched healthy volunteers at baseline and after sleep restriction. The alcohol-dependent group showed a blunted SWA response (i.e., decreased homeostatic sleep drive) to the sleep restriction (12). These studies suggest that a slow wave sleep deficiency may be associated with the development of alcoholism. One of the known effects of alcohol on sleep is an enhancement of slow wave sleep with acute use.

Benzodiazepine receptor agonists (BzRA) are known to have a moderate abuse liability. In a series of studies of the abuse liability of BzRAs, subjects with DSM-IVR insomnia and PSG-determined sleep disturbance were randomized to 12 months of nightly zolpidem (10 mg) or placebo (13). During one-week probes in months 1, 4, and 12 they were given an opportunity to self-administer up to 15 mg of zolpidem (three 5 mg capsules) before sleep. In the subjects randomized to zolpidem the dose was not escalated over the 12 months of nightly use. However, case reports of hypnotic abuse continue to appear in the literature and an analysis of the reports has identified two characteristic patterns in the cases: 1) the dose is escalated within two weeks of initiating use and 2) the nighttime hypnotic use extends to daytime use (14). But one caution is to be made, stable daytime use does not always imply abuse. An earlier study not only showed nighttime self-administration of triazolam versus placebo by people with insomnia, but during separate daytime self-administration assessments in the same individuals, triazolam was also self-administered relative to placebo during the daytime. But, only those who showed evidence of daytime physiological “hyperarousal” (i.e., elevated latencies on the MSLT) self-administered during the day and triazolam normalized their MSLTs (15). Whether these drug self-administration patterns

reflect drug-seeking or therapy-seeking needs to be considered in the context of issues discussed above

Alertness/Sleep Disturbance and Maintenance of Substance Abuse

The alerting effects of stimulants are reinforcing for individuals who experience sleepiness, fatigue, or have difficulty functioning to a “normal” level. Healthy volunteers will self-administer a stimulant when they are sleepy, but not when alert (16). Thus, self-administration of stimulants does not necessarily imply abuse. But, in substance abuse, sleepiness may be present as part of a withdrawal syndrome (i.e., abstinence) following chronic non-medical use of a stimulant. An early laboratory study assessed the disruptive effects of cocaine administration and discontinuation on sleep and daytime alertness (17). Cocaine (600 mg/day) insufflated between 6-9 pm on five consecutive nights delayed sleep onset by 3-4 hrs. During the first two discontinuation days, average daily sleep latency on the MSLT was less than 5 min (i.e. a pathological level of sleepiness), at least in part due to the severely shortened sleep over the prior five days of cocaine use. The MSLT also showed multiple sleep onset REM periods (SOREMPs), probably due to a REM rebound secondary to the prior REM sleep suppression during the cocaine administration nights. After 14 days of abstinence, a nocturnal sleep disturbance and REM sleep rebound remained, although the MSLTs were free of SOREMPs and the latencies returned to essentially normal levels.

It has been hypothesized that continued substance use, difficulty reducing use, and relapse may reflect “self-medication” to reverse the excessive sleepiness of the abstinence. In chronic caffeine or nicotine dependence, the 7-8-hr sleep period is functionally an enforced abstinence and given the pharmacokinetics of these drugs, the 8 hr abstinence during the sleep period is followed by enhanced sleepiness in the morning, and in extreme cases, smoking during the night. Caffeine or nicotine taken immediately when arising reverses the sleepy state. In amphetamine or cocaine abuse, excessive sleepiness during the initial drug abstinence has been consistently reported. Again, use of these stimulants will reverse the sleepiness.

During a period of chronic drug use, daytime sleepiness may also result from a drug-induced disturbance of nocturnal sleep as illustrated above. All the stimulants disrupt nocturnal sleep, with severity depending on dose and proximity of use to the sleep period. Disrupted and fragmented sleep produces daytime sleepiness. One could hypothesize that a drug-induced sleep disturbance at night leads to daytime sleepiness, which then enhances the likelihood of the self-administration of a stimulant during the day. This is a complex balance that always has to be dealt with in patients with excessive daytime sleeping being prescribed stimulants. Also, this is the common vicious circle seen in heavy coffee drinkers. The vicious cycle raises questions as to how much dose escalation of stimulants, including coffee, is due to tolerance versus an exacerbation of the daytime sleepiness associated with accumulated sleep loss.

The state of sleepiness may not necessarily be drug induced. It may also occur due to chronic insufficient sleep in healthy people or due to disturbed sleep efficiency and circadian dysrhythmia seen in persons with altered work and sleep-wake schedules, such as seen

among shift workers or with “jet-lag” (see Chapter 12). As noted above, healthy volunteers will self-administer a stimulant when experiencing sleepiness. Night workers and rotating shift workers have shortened and disturbed sleep when sleeping during the day, as well as increased sleepiness when awake at night. Rotating shift workers and night workers report a disproportionate use of sedating drugs, especially alcohol, to improve sleep and stimulants especially caffeine to improve alertness (18, 19). This substance use may increase risks of substance abuse.

Studies have shown that acute REM deprivation by awakening enhances pain sensitivity (20). Thus, whether the known REM suppression of opiates is reducing their analgesic effect is a critical question. Additionally, whether the hypothesized reduced analgesic effect then leads to the need for higher opiate doses and to the development of physical dependence is also a critical issue.

Alertness/Sleep Disturbance and Relapse of Substance Abuse

The majority of research on sleep disturbance as predictive of relapse in the substance abuse literature is reported for alcoholism (6). Abnormal sleep patterns can persist for up to 3 years in alcoholism. Sleep remains shortened and REM sleep pressure elevated as reflected in elevated REM percentages, shortened latencies to REM sleep and higher REM densities (21). While it is tempting to attribute these sleep abnormalities to the excessive alcohol drinking of the patients, the sleep problems could have preceded the development of the alcoholism (see section above) or they could be secondary to the development of other medical and psychiatric disorders that have developed during the excessive alcohol drinking.

Both objective and subjective measures of sleep after acute abstinence predict the likelihood of relapse during long-term abstinence. Early laboratory studies suggested that low levels of slow wave sleep are predictive of alcohol drinking relapse (22). Other more recent studies have identified REM sleep disturbances, either elevated REM sleep percent or shortened REM sleep latency as predictive of relapse (23). Sleep-related relapse risk was greater than that associated with other variables such as age, marital status, employment, duration and severity of alcoholism, hepatic enzymes, and depression ratings.

A first of its kind study in cocaine-dependent individuals undergoing 12 days of in-patient abuse treatment and 6 weeks of out-patient behavioral treatment collected PSGs on weeks 1, 3, and 6 post discontinuation of cocaine (24). Total sleep time was positively related to days of abstinence over the six-week study. Other than alcohol, this is one of the first studies in substance abusers that we are aware of relating PSG-defined sleep measures to relapse. These data raise the question whether insomnia-focused treatment would have a beneficial effect on substance use treatment.

Treatment of Sleep/Alertness in Substance Use Disorders

The drug class of choice for insomnia treatment in patients without co-morbid alcoholism is the BzRAs. However, while these drugs have a relatively moderate abuse liability in those without alcoholism or a history of sedative abuse, their risk in out-patients with alcoholism after acute in-patient withdrawal is unknown (25). These drugs are effective for the

immediate in-patient withdrawal syndrome, because they share the same mechanism of action as alcohol itself, promotion of gamma-aminobutyric acid (GABA) inhibition (26). A further caution to their out-patient use is that they have a high potential for toxicity and overdose when combined with alcohol (26), thereby being dangerous for the people with the potential for alcoholism relapse.

Sedating antidepressant medications such as trazodone or doxepin are often used in the United States to treat insomnia and insomnia co morbid with depression. Trazodone (50-300 mg) has been used to treat sleep disturbance in patients with alcoholism in open label, non-controlled studies and has shown improved self-report measures of insomnia (26). Drinking outcomes in trials with sleep promoting agents are mixed. A recently completed large placebo-controlled trial of the anticonvulsant, gabapentin (900 mg or 1800 mg), showed improvement in both sleep and drinking outcomes for both doses of gabapentin (27). Currently, the three FDA approved alcoholism treatments are disulfiram, naltrexone, and acamprostate. A placebo-controlled trial of acamprostate 666 mg for 15 days improved wake after sleep onset and stage 3 sleep on the two PSG nights, night 2 and night 15 (26). Unfortunately, drinking outcomes and their relation to sleep were not reported in this study.

Cognitive-behavioral treatment for insomnia (CBT-I) is an alternative to medications and several trials of CBT-I in patients with alcoholism associated with sleep disturbance have been conducted. A randomized controlled trial of a brief cognitive behavioral treatment for the insomnia associated with alcoholism was conducted in 60 insomnia patients without co-morbid depression (28). The CBT-I treatment compared to wait-list controls improved sleep diary measures of sleep quality, sleep efficiency, awakenings, and time to fall asleep. But CBT-I had no impact on drinking relapse rates over the 6-month follow-up period. An open trial of CBT-I in patients with alcoholism similarly found improved sleep, but not improved drinking outcomes (29).

The literature on treating sleep disturbance in drug abuse is even more limited than that for alcoholism, but results reported for the few studies that have been carried out to date are provocative. A trial of CBT-I for insomnia and daytime sleepiness in adolescents reported improved sleep for those completing more than 4 sessions (30). The improved sleep showed a trend toward reducing substance abuse problems at the 1 year follow-up. A treatment trial involving nicotine dependent adults compared a 16-hr versus 24-hr nicotine patch during smoking abstinence (31). The nicotine patch reduced smoking urges, with the 24 hr patch having a greater effect than the 16-hr patch. Interestingly, the 24-hr patch improved sleep, specifically the amount of slow wave sleep. This result in nicotine dependent adults is in contrast to the sleep-disruptive effects of a nicotine patch on the sleep of non-smokers. In the drug abuse treatment literature, sleep is rarely included as an outcome measure or considered a mediator of other outcome measures. A placebo-controlled trial of modafinil 400 mg/day in cocaine-dependent individuals improved the latency and sleep staging of sleep and reduced daytime sleepiness, but relapse outcomes were not measured (32). The need for clinical trials that focus on treatment of sleep complaints in substance abuse is clearly evident. An inherent assumption in this discussion is that sleep disturbance is causally related to alcoholism or drug abuse, either as the precipitant or consequence. It should be noted that the sleep disturbance may be co-morbid and independent or related to a

third common factor. A recent review of the treatment literature concluded that the disorders are comorbid and treatment must be directed at both disorders (33).

Common Neurobiological Disturbances

Insomnia and SUD may share a common neurobiological disturbance. A convergence of data from nighttime and daytime electrophysiology, event-related brain potential recordings, neuroimaging studies, sympathetic nervous system, and hypothalamic-pituitary-adrenal (HPA) axis monitoring all suggest that insomnia is a disorder of “hyperarousal” (6). The pathology underlying this hyperarousal is in part a HPA axis dysfunction involving corticotropin releasing hormone (CRF) and norepinephrine (NE) (7). Many theories of addiction hypothesize that stress increases vulnerability to drug abuse (34). Animal literature and human neuroimaging studies have identified brain circuits involved in stress that include release of CRF from the paraventricular nucleus and NE activation initiated in locus coeruleus (34). This CRF/NE activation also activates dopaminergic brain motivational pathways known to be engaged by drugs of abuse including the ventral tegmental area and nucleus accumbens (34). (see Chapter 2) Thus, stress co-activates brain stress and reward circuits simultaneously. Therefore, at a behavioral level, stress enhances the positive rewarding properties of drugs with abuse liability.

Conclusions

The role of sleep/alertness disturbance in SUD is not fully understood. As the above discussion indicates, sleep disturbances are at least contributory to SUD. From the epidemiological literature it is known that insomnia is a risk factor for substance abuse (17, 18). Yet, the extent to which insomnia or daytime sleepiness leads to new cases of alcoholism or drug abuse is not known. Furthermore, the degree to which treatment of insomnia or daytime sleepiness in abstinent alcoholics and drug abusers reduces risk of relapse has yet to be determined. To date, the few alcoholism treatment trials have failed to clearly demonstrate that improved sleep reduces relapse, and the only available drug abuse treatment trial, while encouraging, is not conclusive.

Another important remaining question relates to the role of specific sleep stages on SUD and their treatment. In reviews of the sleep effects of alcohol and drugs of abuse, it is noted that most of the drugs of abuse suppress REM sleep, with tolerance to the REM-suppression developing rapidly (1,2). The significance of these effects is not clear. Most antidepressants (tricyclics, monoamine oxidase inhibitors and selective serotonin reuptake inhibitors) are highly potent REM suppressants, typically driving REM sleep to below 10% of the night and tolerance does not develop to the REM suppression produced by these antidepressant drugs, even with chronic use (35). The degree of REM suppression in depressed patients is associated with improvement in mood (35). Further, total sleep deprivation and REM sleep deprivation by awakening depressed patients on entry to REM sleep has anti-depressant effects in patients with depression (35).

At the very least, the REM effects may reflect the development of physical dependence and an altered central nervous system neurobiology. Chronic alcohol and drug use likely alters

the neurobiology of sleep and the control of REM sleep, a predominately pontine cholinergic phenomenon. As noted above, in abstinent alcoholics, REM sleep disturbance remaining after acute discontinuation is predictive of relapse. We are unaware of studies regarding the presence of a REM disturbance following abstinence for other drugs of abuse and the predictive value of such a disturbance, if present, to relapse.

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List of Abbreviations

NREM	Non-rapid eye movement
REM	Rapid eye movement
SUD	Substance use disorders
ICSD3	International Classification of Sleep Disorders, third edition
DSM 5	Diagnostic and Statistical Manual of Mental Disorders, fifth edition
PSG	Polysomnography

MSLT	Multiple Sleep Latency Test
THC	Tetrahydrocannabinol
EEG	Electroencephalogram
SWA	Slow wave activity
BzRA	Benzodiazepine receptor agonists
SOREMPs	Sleep onset REM periods
GABA	Gamma-aminobutyric acid
CBT-I	Cognitive behavioral therapy for insomnia
HPA	Hypothalamic-pituitary-adrenal
CRF	Corticotropin releasing hormone
NE	Norepinephrine
OSA	Obstructive sleep apnea
PLMD	Periodic Limb Movements Disorder
RLS	Restless legs syndrome

Key Points

1. All drugs of abuse and alcohol have disruptive effects on sleep, sleep stages, and consequent next-day alertness.
2. These sleep and alertness disturbances may have a contributory role in the initiation, maintenance and relapse in substance use disorders.
3. Whether treatment of the sleep and alertness disturbances alters the risks is unclear.

Synopsis

This chapter discusses the role sleep and alertness disturbance plays in the initiation, maintenance and relapse of substance use disorders.

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