Alcohol and Sleep

The average adult sleeps 7.5 to 8 hours every night. Although the function of sleep is unknown, abundant evidence demonstrates that lack of sleep can have serious consequences, including increased risk of depressive disorders, impaired breathing, and heart disease. In addition, excessive daytime sleepiness resulting from sleep disturbance is associated with memory deficits, impaired social and occupational function, and car crashes (1,2). Alcohol consumption can induce sleep disorders by disrupting the sequence and duration of sleep states and by altering total sleep time as well as the time required to fall asleep (i.e., sleep latency). This Alcohol Alert explores the effects of alcohol consumption on sleep patterns, the potential health consequences of alcohol consumption combined with disturbed sleep, and the risk for relapse in those with alcoholism who fail to recover normal sleep patterns.

Sleep Structure, Onset, and Arousal

Before discussing alcohol's effects on sleep, it is helpful to summarize some basic features of normal sleep. A person goes through two alternating states of sleep, characterized in part by different types of brain electrical activity (i.e., brain waves). These states are called slow wave sleep (SWS), because in this type of sleep the brain waves are very slow, and rapid eye movement (REM) sleep, in which the eyes undergo rapid movements although the person remains asleep.

Most sleep is the deep, restful SWS. REM sleep occurs periodically, occupying about 25 percent of sleep time in the young adult. Episodes of REM normally recur about every 90 minutes and last 5 to 30 minutes. REM sleep is less restful than SWS and is usually associated with dreaming. Although its function is unknown, REM appears to be essential to health. In rats, deprivation of REM sleep can lead to death within a few weeks (3). In addition, a transitional stage of light sleep occurs at intervals throughout the sleep period (4).

Sleep was formerly attributed to decreased activity of brain systems that maintain wakefulness. More recent data indicate that sleep, like consciousness, is an active process. Sleep is controlled largely by nerve centers in the lower brain stem, where the base of the brain joins the spinal cord. Some of these nerve cells produce serotonin, a chemical messenger associated with sleep onset (5) and with the regulation of SWS. Certain other nerve cells produce norepinephrine, which helps regulate REM sleep and facilitates arousal (6). The exact roles and interactions of these and other chemical messengers in orchestrating sleep patterns are not known (6). Significantly, however, alcohol consumption affects the function of these and other chemical messengers that appear to influence sleep.

Alcohol and Sleep in Those Without Alcoholism

Alcohol consumed at bedtime, after an initial stimulating effect, may decrease the time required to fall asleep. Because of alcohol's sedating effect, many people with insomnia consume alcohol to promote sleep. However, alcohol consumed within an hour of bedtime appears to disrupt the second half of the sleep period (7). The subject may sleep fitfully during the second half of sleep, awakening from dreams and returning to sleep with difficulty. With continued
consumption just before bedtime, alcohol's sleep-inducing effect may decrease, while its disruptive effects continue or increase (8). This sleep disruption may lead to daytime fatigue and sleepiness. The elderly are at particular risk, because they achieve higher levels of alcohol in the blood and brain than do younger persons after consuming an equivalent dose. Bedtime alcohol consumption among older persons may lead to unsteadiness if walking is attempted during the night, with increased risk of falls and injuries (3).

Alcoholic beverages are often consumed in the late afternoon (e.g., at "happy hour" or with dinner) without further consumption before bedtime. Studies show that a moderate dose \(^1\) of alcohol consumed as much as 6 hours before bedtime can increase wakefulness during the second half of sleep. By the time this effect occurs, the dose of alcohol consumed earlier has already been eliminated from the body, suggesting a relatively long-lasting change in the body's mechanisms of sleep regulation (7,8).

The adverse effects of sleep deprivation are increased following alcohol consumption. Subjects administered low doses of alcohol following a night of reduced sleep perform poorly in a driving simulator, even with no alcohol left in the body (9,10). Reduced alertness may potentially increase alcohol's sedating effect in situations such as rotating sleep-wake schedules (e.g., shift work) and rapid travel across multiple time zones (i.e., jet lag) (9). A person may not recognize the extent of sleep disturbance that occurs under these circumstances, increasing the danger that sleepiness and alcohol consumption will co-occur.

Alcohol and Breathing Disorders

Approximately 2 to 4 percent of Americans suffer from obstructive sleep apnea (OSA), a disorder in which the upper air passage (i.e., the pharynx, located at the back of the mouth) narrows or closes during sleep (11). The resulting episode of interrupted breathing (i.e., apnea) wakens the person, who then resumes breathing and returns to sleep. Recurring episodes of apnea followed by arousal can occur hundreds of times each night, significantly reducing sleep time and resulting in daytime sleepiness. Those with alcoholism appear to be at increased risk for sleep apnea, especially if they snore (12). In addition, moderate to high doses of alcohol consumed in the evening can lead to narrowing of the air passage (13,14), causing episodes of apnea even in persons who do not otherwise exhibit symptoms of OSA. Alcohol's general depressant effects can increase the duration of periods of apnea, worsening any preexisting OSA (14).

OSA is associated with impaired performance on a driving simulator as well as with an increased rate of motor vehicle crashes in the absence of alcohol consumption (9,10). Among patients with severe OSA, alcohol consumption at a rate of two or more drinks per day is associated with a fivefold increased risk for fatigue-related traffic crashes compared with OSA patients who consume little or no alcohol (15). In addition, the combination of alcohol, OSA, and snoring increases a person's risk for heart attack, arrhythmia, stroke, and sudden death (16).

Age-Related Effects and the Impact of Drinking

Little research has been conducted on the specific effects of alcohol on sleep states among different age groups. Scher (17) investigated the effects of prenatal alcohol exposure on sleep patterns in infants. Measurements of brain electrical activity demonstrated that infants of mothers who consumed at least one drink per day during the first trimester of pregnancy exhibited sleep disruptions and increased arousal compared with infants of nondrinking women. Additional studies revealed that infants exposed to alcohol in mothers' milk fell asleep sooner but slept less overall than those who were not exposed to alcohol (18). The exact significance of these findings is unclear.

Normal aging is accompanied by a gradual decrease in SWS and an increase in nighttime wakefulness. People over 65 often awaken 20 times or more during the night, leading to sleep that is less restful and restorative (3). Age-related sleep deficiencies may encourage the use of alcohol to promote sleep, while increasing an older person's susceptibility to alcohol-related sleep disturbances (3,19). Potential sources of inconsistency among study results include different doses of alcohol employed and failure to screen out subjects with preexisting sleep disorders (3).

Effects of Alcohol on Sleep in Those With Alcoholism

Active Drinking and Withdrawal. Sleep disturbances associated with alcoholism include increased time required to fall asleep, frequent awakenings, and a decrease in subjective sleep quality associated with daytime fatigue (3). Abrupt reduction of heavy drinking can trigger alcohol withdrawal syndrome, accompanied by pronounced insomnia with marked sleep fragmentation. Decreased SWS during withdrawal may reduce the amount of restful sleep. It has been suggested that increased REM may be related to the hallucinations that sometimes occur during withdrawal. In patients with severe withdrawal, sleep may consist almost entirely of brief periods of REM interrupted by numerous awakenings (3,20).

Recovery and Relapse. Despite some improvement after withdrawal subsides, sleep patterns may never return to


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normal in those with alcoholism, even after years of abstinence (3,21). Absentile alcoholics tend to sleep poorly, with decreased amounts of SWS and increased nighttime wakefulness that could make sleep less restorative and contribute to daytime fatigue (22). Resumption of heavy drinking leads to increased SWS and decreased wakefulness. This apparent improvement in sleep continuity may promote relapse by contributing to the mistaken impression that alcohol consumption improves sleep (23-25). Nevertheless, as drinking continues, sleep patterns again become disrupted (3).

Researchers have attempted to predict relapse potential using measures of sleep disruption. Gillin and colleagues (26) measured REM sleep in patients admitted to a 1-month alcoholism treatment program. Higher levels of REM predicted those who relapsed within 3 months after hospital discharge in 80 percent of the patients. A review of additional research (3) concluded that those who eventually relapsed exhibited a higher proportion of REM and a lower proportion of SWS at the beginning of treatment, compared with those who remained abstinent. Although additional research is needed, these findings may facilitate early identification of patients at risk for relapse and allow clinicians to tailor their treatment programs accordingly.

References


1 A standard drink is generally considered to be 12 ounces of beer, 5 ounces of wine, or 1.5 ounces of distilled spirits, each drink containing approximately 0.5 ounce of alcohol. In addition, terms such as light, moderate, or heavy drinking are not used consistently by alcoholism researchers. Therefore, in each case, the terms used in this text are those of the author or authors cited.