Alcohol and Sleep

"It has long been known that alcohol, when consumed by healthy individuals before going to sleep, shortens the amount of time needed to fall asleep, reduces the amount of REM sleep, and increases the amount of non-REM sleep time (Gresham et al., 1963; Knowles et al., 1968; MacLean and Cairns, 1982; Rundell et al., 1972; Williams et al., 1983; Yules et al., 1966).

As alcohol is metabolized relatively quickly (Madsen and Rossi, 1980), these changes in sleep architecture typically are limited to the first half of the night, while the effects of alcohol withdrawal may disturb sleep during the second half of the night (Knowles et al., 1968; Williams et al., 1983). In particular, late sleep following alcohol ingestion before going to bed can be ''shallow'' and interrupted by frequent periods of wakefulness; the amount of REM sleep is increased and dreams and nightmares frequently occur (Rundell et al., 1972)."

Feige (2006), p. 1527

Alcohol and sleep in young adults

Orvis H. Rundell, Boyd K. Lester, William J. Griffiths & Harold L. Williams

Psychopharmacologia volume 26, pages 201–218 (1972)

Abstract

In two separate experiments, sleep patterns of 17 young male adults were examined following single and repeated doses (0.9 g/kg body weight) of alcohol. A third study of 10 additional Ss measured the rate of elimination of alcohol from blood during sleep and waking. With a single dose of alcohol, onset of sleep was brisk, the latency of SW sleep (stages 3+4) was reduced and the first episode of stage REM was shortened. These transient alterations were accompanied by loss of high-frequency beta rhythms in the EEG and a gain in abundance and synchrony of activity in the alpha-rhythm range.

Experiment 3 found no significant difference between sleep and waking for rate of elimination of alcohol from blood. Starting with peak blood alcohol concentrations (BACs) of about 75 mg percent, average BACs after 4 h of bedtime had decreased to about 30 mg percent. The effects noted above, associated with higher BACs, were confined to the first half of the night. Thus, these results are consistent with conclusions of previous investigators that the depressant effects on EEG sleep patterns of a moderate dose of alcohol are due to its direct action on the brain.

There are, however, longer range compensation and adaptation effects associated with single and repeated doses of alcohol which cannot be directly related to its presence in the brain. For example, in the single-dose study reported here, and in most previous studies, rebound of stage REM occurred during the second half of the alcohol night. Further, in the repeated-dose study, most of the effects of alcohol on sleep stages and EEG frequencies observed during the first dose session, disappeared on the second and third alcohol sessions. Finally, heart and respiration rates increased whereas eye movements during stage REM, sigma spindles in stage 2 and non-specific GSR responses in SW sleep tended to be suppressed throughout the night in each alcohol session. Several mechanisms are discussed which might account for these more persistent alterations.

Dose-response effects of ethanol on the sleep of young women.

Williams DL, Maclean AW, Cairns J.

J Stud Alcohol. 1983; 44(3):515–523. doi:10.15288/jsa.1983.44.515.

Abstract

In healthy young women, rapid eye movement sleep decreased, slow-wave sleep increased, sleep-onset latency decreased and late-night disturbance of sleep increased with increasing doses of alcohol.

Nocturnal and next-day effects of ethanol and basal level of sleepiness.

Roehrs, T., et al.

Human Psychopharmacology: Clinical and Experimental, Vol 6(4), Dec, 1991. pp. 307-311.

Abstract:

Five healthy men consumed ethanol (0.8 g/kg) and placebo at 2200–2230 hrs after 4 and 8 hrs in bed (TIB) the previous night. Standard sleep recordings were collected from 2300 to 0700 hrs. Next-day latency to sleep onset was tested at 1000, 1200, 1400, and 1600 hrs, and divided attention performance and hangover symptoms were assessed 30 min after each latency test. The 4-hr TIB increased sleep efficiency but did not interact with effects of ethanol. Ethanol altered sleep staging: It increased percentage Stage 3/4 sleep in the 1st half of the night and increased percentage Stage 1 sleep in the 2nd. The following day, mean daily sleep latency was reduced and mean divided attention tracking errors were increased with ethanol.

Sleep, sleepiness, and alcohol use.

Roehrs, Timothy and Roth, Thomas.

Alcohol Research & Health, Vol 25(2), 2001. pp. 101-109.

Abstract:

Provides an overview of alcohol's effects on normal sleep, sleep physiology, and daytime alertness in nonalcoholic people. Normal sleep physiology is reviewed and alcohol's effects on the various sleep states and sleep stages are described. The relationship of nocturnal sleep to daytime alertness and how alcohol affects this relationship is also summarized. The article ends with a discussion of alcohol's effects on sleep in people with primary insomnia.

"*The study of alcohol's effects on sleep dates back to the late 1930s. Since then, an extensive literature has described alcohol's effects on the sleep of healthy, nonalcoholic people. For example, studies found that in nonalcoholics who occasionally use alcohol, both high and low doses of alcohol initially improve sleep, although high alcohol doses can result in sleep disturbances during the second half of the nocturnal sleep period. Furthermore, people can rapidly develop tolerance to the sedative effects of alcohol. Researchers have investigated the interactive effects of alcohol with other determinants of daytime sleepiness. Such studies indicate that alcohol interacts with sleep deprivation and sleep restriction to exacerbate daytime sleepiness and alcohol-induced performance impairments. Alcohol's effects on other physiological functions during sleep have yet to be documented thoroughly and unequivocally*." Roehrs (2001), p. 101

Effects of alcohol on polysomnographically recorded sleep in healthy subjects.

Feige B, Gann H, Brueck R, et al.

Alcoholism Clin Exp Res. 2006;30(9):1527–1537. doi:10.1111/j.1530-0277.2006. 00184.x.

Abstract:

Background: After studying the sleep of alcohol-dependent patients at the beginning and over the course of abstinence in earlier studies, our interest in the current study focused on the direct effect of 2 doses of alcohol [0.03 and 0.1% blood alcohol level (BAL)] on healthy sleep. This is the first poly-somnographic study testing the impact of 2 doses of alcohol

ingestion (thus reflecting ‘‘normal’’ social drinking and alcohol abuse) in a single-blind randomized design in healthy volunteers. The study evaluated a short-term acute drinking period for 3 and 2 days of withdrawal from alcohol not only for polysomnographic variables but also for subjective estimates of sleep quality.

Methods: In a crossover design with a 1-week interval, healthy subjects received alcohol to raise their blood alcohol to either 0.03 or 0.1% BAL at bedtime for 3 consecutive nights after an alcohol- free baseline night. Objective (polysomnography) and subjective sleep (questionnaires) was recorded each night. During the following 2 days, alcohol was discontinued with simultaneous measurements of sleep to gauge withdrawal effects.

Results: At a dose of alcohol leading to BAL of 0.03%, no clear effects could be detected. Fol- lowing an evening BAL of 0.1%, a hypnotic-like effect (shortened sleep latency, reduced number of wake periods, decreased stage 1 sleep) occurred primarily during the first half of the night with signs of rebound effects being already present during the second half of the night (increased stage 1 sleep). At this dose, alcohol significantly increased slow-wave sleep (SWS) in the first half of the night and reduced REM density in the beginning of the night. After discontinuation of the higher alcohol dose, REM sleep amount increased. No significant withdrawal or rebound effects could be observed for parameters of sleep continuity during the 2 nights after discontinuation from alcohol at a BAL of 0.1%.

Conclusions: Owing to the small sample size, the results of this study need to be interpreted with caution. Short-term moderate alcohol consumption (BAL 0.03%) did not significantly alter objective or subjective parameters of sleep. Higher doses of alcohol resulting in a BAL level of 0.10% immediately before going to bed mainly influenced sleep in the first half of the night, resembling the effects of a short-acting hypnotic drug, including a suppression of phasic aspects of REM sleep (REM density). Interestingly, analysis of the latter part of these nights indicated the immediate presence of withdrawal effects (increased light sleep). No statistically significant effects on sleep parameters were observable during the 2 nights of withdrawal from alcohol at the higher BAL. Interpreted carefully, our data indicate that negative effects on sleep occur already with short-term use of alcohol at doses of BAL of 0.10%, despite hypnotic-like effects during the first hours of sleep, especially during the latter part of the night.

“It has long been known that alcohol, when consumed by healthy individuals before going to sleep, shortens the amount of time needed to fall asleep, reduces the amount of REM sleep, and increases the amount of non-REM sleep time (Gresham et al., 1963; Knowles et al., 1968; MacLean and Cairns, 1982; Rundell et al., 1972; Williams et al., 1983; Yules et al., 1966).

As alcohol is metabolized relatively quickly (Madsen and Rossi, 1980), these changes in sleep architecture typically are limited to the first half of the night, while the effects of alcohol withdrawal may disturb sleep during the second half of the night (Knowles et al., 1968; Williams et al., 1983). In particular, late sleep following alcohol ingestion before going to bed can be ‘‘shallow’’ and interrupted by frequent periods of wakefulness; the amount of REM sleep is increased and dreams and nightmares frequently occur (Rundell et al., 1972).”

Feige (2006), p. 1527

"*In summary, our findings in healthy subjects underscore the hypnotic-like effects of acute alcohol intake at higher doses before sleep and possible sleep-disturbing sequelae during the second part of the night. Accordingly, it can be concluded that when consumed in greater quantities and for a period of several consecutive days, alcohol is far from being suitable as a hypnotic, because of immediate rebound phenomena during the later part to the night that run parallel to the pharmacokinetics of alcohol. The present study probably failed to detect more pronounced rebound phenomena because of the rather small sample size, young age of the sample, and the rather short drink- ing periods observed (compared with the situation in alcohol-dependent subjects or insomniacs chronically using alcohol as a ''sleep aid'').*" Feige (2006), p. 1536

Alcohol and sleep I: effects on normal sleep.

Ebrahim IO, Shapiro CM, Williams AJ, Fenwick PB.
Alcohol Clin Exp Res. 2013; 37(4):539–549. doi:10.1111/acer.12006.

Abstract

This review provides a qualitative assessment of all known scientific studies on the impact of alcohol ingestion on nocturnal sleep in healthy volunteer's. At all dosages, alcohol causes a reduction in sleep onset latency, a more consolidated first half sleep and an increase in sleep disruption in the second half of sleep. The effects on rapid eye movement (REM) sleep in the first half of sleep appear to be dose related with low and moderate doses showing no clear trend on REM sleep in the first half of the night whereas at high doses, REM sleep reduction in the first part of sleep is significant. Total night REM sleep percentage is decreased in the majority of studies at moderate and high doses with no clear trend apparent at low doses. The onset of the first REM sleep period is significantly delayed at all doses and appears to be the most recognizable effect of alcohol on REM sleep followed by the reduction in total night REM sleep. The majority of studies, across dose, age and gender, confirm an increase in slow wave sleep (SWS) in the first half of the night relative to baseline values. The impact of alcohol on SWS in the first half of night appears to be more robust than the effect on REM sleep and does not appear to be an epiphenomenon REM sleep reduction. Total night SWS is increased at high alcohol doses across gender and age groups.

The acute effects of alcohol on sleep architecture in late adolescence.

Chan JK, Trinder J, Andrewes HE, Colrain IM, Nicholas CL.
Alcohol Clin Exp Res. 2013;37(10).1720–8. doi:10.1111/ acer.12141.

Abstract

Background

Alcohol consumption is prevalent in late adolescence; however, little is known about its effect on sleep in this group. In mature adults, alcohol decreases sleep onset latency (SOL) and sleep efficiency (SE) and increases wake after sleep onset (WASO). It also increases slow wave sleep (SWS) and decreases rapid eye movement (REM) sleep in the first half of the night, with the inverse occurring in the second half. Alcohol's effect on sleep during late adolescence is of interest given that this age group shows both dramatic increases in alcohol consumption and significant developmental changes in the central nervous system. This study examined the effect of alcohol on sleep architecture in women and men aged 18 to 21 years and whether previously reported sleep architecture effects may have been as an artificial result of changes to sleep cycle length.

Methods

Twenty-four (12 women) healthy 18- to 21-year-old light social drinkers (19.1 ± 1.0 years) underwent 2 conditions: presleep alcohol (target breath alcohol concentration [BAC] 0.10%)

and placebo-administered under controlled conditions, followed by standard polysomnography.

Results
In the alcohol condition, mean BAC at lights out was 0.084 ± 0.016%. Time in bed, total sleep time, and SOL (all $p > 0.05$) did not differ between conditions. However, there was less REM ($p = 0.011$) and more stage-2 sleep ($p = 0.035$) in the alcohol condition. Further, alcohol increased SWS ($p = 0.02$) and decreased REM sleep ($p < 0.001$) in the first half of the night and disrupted sleep in the second half, with increased WASO (interaction: $p = 0.034$), and decreased SE ($p = 0.04$) and SWS ($p = 0.01$) and no REM sleep rebound in the second half of the night ($p = 0.262$). Additionally, alcohol had no effect on sleep cycle length ($p = 0.598$).

Conclusions
The results were broadly consistent with the adult literature with the novel extension that half night sleep architecture effects could not be attributed to changes in sleep cycle length. However, alcohol did not reduce SOL, or result in a REM rebound following reduced REM in the first half of the night. The results suggest that the effects of alcohol on sleep are modified by sleep's prevailing developmental stage.

The effect of acute alcohol ingestion on systemic hemodynamics and sleep architecture in young, healthy men.
Payseur, Daniel K., et al.
Journal of American College Health, Vol 70(2), Feb-Mar, 2022. pp. 509-516.
Abstract:

Objective: Heightened nocturnal blood pressure (BP) may be attributed to the disruption of sleep, a condition worsened by alcohol ingestion. This study investigated the effects of acute alcohol ingestion on hemodynamics and sleep architecture in a young, healthy cohort of male.

METHODS: Subjects (n = 17) underwent acute alcohol ingestion reaching a breath alcohol content of 0.08. Each subject endured a battery of hemodynamic tests and had their sleep architecture and nocturnal blood pressure monitored pre- and post-ingestion.

Results: Systolic blood pressure (SBP) increased both 30 minutes and 12 hours after alcohol. Ambulatory nocturnal SBP significantly increased after alcohol compared to baseline measures. Minutes of total, rapid eye movement, and light sleep all increased after alcohol ingestion, while a decrease was observed for sleep latency.

Conclusions: An acute bout of heavy alcohol consumption may attenuate nocturnal BP dipping that, in turn, may hasten the progression of hypertension-related cardiovascular disease.

"Total sleep time after alcohol was significantly higher than baseline (394.9min ± 18.6 vs. 333.9min ± 14.7; $p<0.05$; Figure 4). A significant decrease was observed for time to sleep (16.1 min ± 3.2 vs. 32.5 min ± 6.8); $p < 0.05$; Figure 5), while time to wake and number of awakenings showed no significant differences from baseline. Time in rapid eye movement (REM) sleep significantly increased after alcohol (108.4 min ± 0.1 vs. 85.1 min ± 0.1; $p < 0.05$; Figure 6), as did light sleep (219.5 min ± 0.1 vs. 186.9 min ± 1.0; $p < 0.05$; Figure 6). There were no significant differences observed for deep sleep." Payseur, p. 513

"While an increase was observed for total sleep time after alcohol, the changes in stage sleep are important to consider. Increases in light and REM stages indicate an altered sleep cycle and a nonsignificant decrease in deep sleep further points to a diminished sleep quality. The literature on alcohol-related sleep disturbances largely includes older adults and less is known on younger cohorts.[46] It is well-accepted that nocturnal effects of alcohol vary between the first and second halves of sleep. During the first half of sleep when alcohol is at peak concentration in the body, more time is spent in deep sleep and less in light and REM stages. In the second half of sleep during alcohol metabolism, a "REM rebound" occurs resulting in lengthened REM and less slow wave sleep.[47,48] The current study observed a small increase in REM stage, while other studies conducted in young adults have not shown this change.[49,50] The disturbances in sleep stages after alcohol are present, although subtle, in the present study and further investigation of these mechanisms is warranted in young adults. The present study is specifically limited by employment of a relatively small sample of healthy college-age male who did not endorse concerning patterns of alcohol consumption." Payseur, p. 514