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BEYOND THE NIGHTCAP: A NARRATIVE REVIEW OF ALCOHOL'S IMPACT ON SLEEP ARCHITECTURE, CIRCADIAN RHYTHMS, AND RECOVERY

Martyna Susek *(Corresponding Author, Email: martyna.susek788@gmail.com)*
Beskidzkie Centrum Onkologii Szpital Miejski im. Jana Pawła II w Bielsku-Białej, Bielsko-Biała, Silesia, Poland
ORCID ID: 0009-0006-7383-8121

Amadeusz Furmanek
Szpital Wojewódzki w Bielsku-Białej, Bielsko-Biała, Silesia, Poland
ORCID ID: 0009-0003-6839-2207

ABSTRACT

Background: Alcohol is one of the most commonly used substances to facilitate sleep, yet its impact on sleep architecture and long-term health is profoundly detrimental. This narrative review explores the complex relationship between alcohol consumption and sleep disturbances, with a particular focus on the biological mechanisms, epidemiological trends, and clinical implications for recovery.
Methods: A comprehensive literature search was conducted across PubMed, ScienceDirect, and Google Scholar databases, covering research from 2001 to 2026. The review synthesized 30 key sources, including meta-analyses, clinical trials, and epidemiological reports from international (WHO, Eurostat) and national (PAN, PARPA) organizations. Inclusion criteria focused on studies detailing alcohol's impact on sleep architecture, circadian rhythms, and pharmacological interventions in Alcohol Use Disorder (AUD).
Results: The evidence confirms that alcohol follows a biphasic effect: initial GABAergic sedation and shortened sleep latency are followed by a "glutamatergic rebound" in the second half of the night. This results in significant REM sleep suppression, increased autonomic stress, and fragmented sleep architecture. Chronobiological data indicate that ethanol disrupts molecular clock gene expression and melatonin secretion, leading to systemic inflammation and cognitive deficits. Furthermore, epidemiological data from Poland and the UK Biobank suggest a causal link between even moderate consumption and chronic insomnia.
Conclusions: The "nightcap" myth is biologically refuted; alcohol is a potent chronodisruptor that degrades sleep quality and hinders neurological recovery. In clinical practice, especially within AUD treatment, addressing sleep health through CBT-I and non-addictive pharmacotherapy is a mandatory pillar of relapse prevention. Public health strategies must shift toward a "no safe level" approach to protect the population's circadian and sleep health.

KEYWORDS

Alcohol Consumption, Sleep Architecture, Circadian Rhythm, Insomnia, REM Sleep Suppression, Alcohol Use Disorder (AUD)

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1. Introduction

Alcohol consumption and its impact on public health remains one of the most significant challenges in modern medicine. According to the latest guidelines from the World Health Organization (2023), there is no level of alcohol consumption that is entirely safe for human health. European statistics indicate a persistently high level of alcohol intake, as evidenced by data from Eurostat (2024). On a local scale, reports from the Polish Academy of Sciences (2023) and statistics from PARPA (2023) highlight specific consumption patterns in Poland that directly translate into a deterioration of the population's health status, particularly concerning the prevalence of sleep disorders.

The relationship between alcohol and sleep is multidimensional and paradoxical. Although alcohol is frequently used as a sleep aid (a "nightcap"), its metabolism—detailed by Cederbaum (2012)—leads to drastic changes in sleep architecture during the second half of the night. As noted by Ebrahim et al. (2013) and a recent meta-analysis by Gardiner et al. (2025), this substance significantly disrupts REM sleep and sleep homeostasis, a finding further supported by neurobiological research on addiction conducted by Koob and Volkow (2016). Alcohol acts directly on the "sleeping brain" (Colrain et al., 2014), disrupting natural circadian rhythms (Bailey, 2026; Sharma & Nelson, 2024) and leading to chronic inflammation and a decrease in both subjective and objective rest quality (Irwin et al., 2016; Park et al., 2015).

Modern research, including large-scale analyses from the UK Biobank (Zheng et al., 2024), proves that even moderate alcohol consumption correlates with the occurrence of insomnia and other sleep-related issues (Zheng et al., 2021; Britton et al., 2020). Consuming alcohol over several consecutive nights is particularly dangerous, as it permanently modifies sleep structure (McCullar et al., 2024). These effects are not limited to the night itself; the consequences in the form of impaired cognitive functions—commonly referred to as a hangover—significantly affect daily activity (Stephens et al., 2008; Gunn et al., 2018). Furthermore, in adolescents and young adults, these disturbances may serve as predictors of future alcohol-related problems (Hasler et al., 2024).

This article aims to analyze the mechanisms through which alcohol degrades sleep quality and to evaluate potential pharmacological treatment options for individuals struggling with insomnia in the context of alcohol use disorder recovery (Roehrs et al., 2020).

2. Methodology

The present review was conducted to synthesize current knowledge regarding the impact of alcohol consumption on sleep architecture, circadian rhythms, and overall sleep quality. The literature search was performed across major electronic databases, including PubMed/MEDLINE, ScienceDirect, and Google Scholar, covering publications from 2001 to early 2026.

Search Strategy

The search strategy employed a combination of MeSH terms and keywords, including: *"alcohol consumption"*, *"sleep architecture"*, *"circadian rhythm"*, *"insomnia"*, *"REM sleep"*, and *"alcohol use disorder"*. To ensure a comprehensive perspective, the search was supplemented by gray literature and official health reports from international and national organizations, such as the World Health Organization (2023), Eurostat (2024), and Polish agencies including PARPA (2023) and the Polish Academy of Sciences (2023).

Inclusion and Exclusion Criteria

Studies were included if they met the following criteria:

1. Peer-reviewed primary research, systematic reviews, or meta-analyses (e.g., Gardiner et al., 2025; Gunn et al., 2018).
2. Studies involving human subjects across different age groups, from adolescents (Hasler et al., 2024) to older adults (Britton et al., 2020).
3. Research focusing on both acute effects of alcohol (Ebrahim et al., 2013) and chronic exposure (McCullar et al., 2024).

Articles were excluded if they were not available in English or Polish, or if they lacked a clear focus on the physiological or psychological intersection of alcohol and sleep.

Data Synthesis

A total of 30 key sources were selected for final inclusion. These were categorized into three main thematic areas:

1. Epidemiological Trends: Utilizing global and local data to establish the scale of the problem.
2. Biological Mechanisms: Analyzing alcohol metabolism (Cederbaum, 2012), circadian disruption (Bailey, 2026; Sharma & Nelson, 2024), and neurobiology (Koob & Volkow, 2016).
3. Clinical Outcomes: Reviewing quality of sleep (Park et al., 2015), cognitive impacts (Stephens et al., 2008), and pharmacological interventions (Roehrs et al., 2020).

3. Results

3.1. Epidemiological Landscape: From Global Trends to Polish Statistics

The global prevalence of alcohol consumption remains a primary driver of non-communicable diseases and sleep-related morbidity. The World Health Organization (2023) has recently reinforced its stance, stating that there is no "safe" threshold for alcohol consumption, as even low-level intake can trigger oncogenic and neurobiological changes. This paradigm shift is crucial, considering that European nations continue to report some of the highest per capita consumption rates in the world (Eurostat, 2024). Within this European context, the relationship between alcohol availability and the erosion of sleep health has become an urgent public health priority.

In Poland, the epidemiological situation presents unique challenges. Reports from the Polish Academy of Sciences (2023) indicate a troubling trend: while the frequency of drinking episodes remains high, the volume of spirits and high-percentage beverages consumed per occasion is increasing. Statistics from PARPA (2023) show that this pattern is directly linked to a surge in reported sleep disturbances among the adult population. The misuse of alcohol as a "self-medication" tool for stress-induced insomnia is particularly prevalent in the Polish workforce, leading to a cycle of nocturnal disruption and diminished daytime productivity.

Modern epidemiological research has moved beyond simple correlation to investigate causality. Utilizing data from the UK Biobank, Zheng et al. (2024) employed Mendelian randomization to demonstrate that genetic predispositions for higher alcohol consumption are directly associated with poorer sleep traits, including shorter sleep duration and increased daytime sleepiness. This is supported by earlier community-based studies (Zheng et al., 2021), which found that the risk of developing clinically significant insomnia is markedly higher in individuals who exceed moderate drinking guidelines. Furthermore, the demographic impact is shifting; while older populations show a high correlation between alcohol use and chronic sleep disorders (Britton et al., 2020), recent data suggests that the "sleep-alcohol" nexus is increasingly affecting younger cohorts. As noted by Hasler et al. (2024), sleep disturbances in late adolescence act as a potent predictor for the development of future Alcohol Use Disorders (AUD).

3.2. Pathophysiology: Alcohol Metabolism and the "Sleep Rebound" Mechanism

The disruption of human sleep by alcohol is a multi-stage, time-dependent process governed by the pharmacokinetics of ethanol and its subsequent dynamic effect on the central nervous system. As established by Cederbaum (2012), ethanol acts as a powerful modulator of neurotransmission, primarily through the allosteric enhancement of gamma-aminobutyric acid (GABA-A) receptors and the simultaneous non-competitive inhibition of N-methyl-D-aspartate (NMDA) glutamate receptors. This acute neurochemical shift creates a profound sedative effect during the initial phase of sleep, leading to the significantly shortened sleep onset latency documented by Ebrahim et al. (2013). However, the brain rapidly initiates compensatory homeostatic mechanisms to counteract this forced sedation, leading to a state of profound neurochemical instability as ethanol is metabolized by alcohol dehydrogenase (ADH).

According to the comprehensive synthesis by Gardiner et al. (2025), the physiological transition as blood alcohol concentration (BAC) declines — typically occurring 4 to 6 hours after consumption — is characterized by several critical disruptions that define the "rebound" phenomenon:

- Biphasic Sleep Architecture and Glutamatergic Rebound: Alcohol induces a distinct, pathological division of the nocturnal period. In the first half, there is a significant increase in slow-wave sleep (SWS) and a reduction in wakefulness. However, as the sedative effects of GABA-A enhancement wane, the previously inhibited NMDA receptors undergo a "rebound" hyperexcitability. McCullar et al. (2024) observed that this transition is marked by a surge in glutamate and norepinephrine, leading to increased sympathetic nervous system activity, frequent micro-arousals, and a persistent shift toward lighter Stage 1 sleep. This fragmented second half of the night is biologically characterized by a state of sub-acute withdrawal, which prevents the transition into deep, restorative NREM stages.
- REM Sleep Suppression and Cholinergic Interference: Ethanol interferes with the brainstem's cholinergic systems — specifically the pedunculopontine and laterodorsal tegmental nuclei — which are responsible for the initiation of Rapid Eye Movement (REM) sleep. During the first half of the night, REM sleep is dose-dependently suppressed. As ethanol concentration drops, the brain attempts to compensate for this deficit through a "REM rebound." As noted by Gardiner et al. (2024) and Ebrahim et al. (2013), this results in a disproportionate concentration of REM cycles in the pre-dawn hours. These cycles are often interrupted

by autonomic surges, leading to vivid dreams, high-intensity nightmares, and a subjective feeling of mental exhaustion upon waking.

- Homeostatic and Melatonin Disruption: Alcohol fundamentally disrupts the adenosinergic system, the primary mediator of homeostatic sleep pressure. Thakkar et al. (2015) and Colrain et al. (2014) have demonstrated that while alcohol initially inflates extracellular adenosine levels (promoting sleep), this is followed by a premature collapse of adenosine signaling, leading to the characteristic "early morning awakening." Furthermore, ethanol suppresses the nocturnal secretion of melatonin and prevents the body from reaching its physiological thermoregulatory nadir. Chaput (2026) emphasizes that the sustained metabolic work of ethanol processing keeps the core body temperature elevated, which inhibits the glymphatic system's ability to clear metabolic waste, such as beta-amyloid, from the brain.
- Autonomic Overdrive and Cardiovascular Stress: The influence of alcohol extends to heart rate variability (HRV), a critical marker of autonomic health. Park et al. (2015) argue that the sustained elevation in heart rate and the marked reduction in HRV throughout the night reflect a state of sustained physiological stress. This autonomic imbalance signifies that even when the individual remains unconscious, the "sleeping brain" is operating under high metabolic and cardiovascular demand, rather than the low-energy restorative state required for cognitive and physical recovery.

The complex interaction between ethanol metabolism, neurotransmitter fluctuations, and their corresponding effects on sleep architecture is summarized in the table below.

Table 1. Pathophysiological Mechanisms of Alcohol-Induced Sleep Disruption

Mechanism	Phase of Sleep	Biological Action	Clinical Outcome
GABAergic Modulation	First Half	Enhancement of GABA-A receptors	Shortened sleep onset latency
Glutamergic Rebound	Second Half	NMDA receptor hyperexcitability; NE surge	Sleep fragmentation, micro-arousals
REM Suppression	Throughout night	Cholinergic interference in the brainstem	Reduced restorative sleep, vivid dreams
Adenosine Disruption	Early Morning	Premature collapse of adenosine drive	Early morning insomnia (early waking)
Chronodisruption	Circadian	Inhibition of *PER2* and *BMAL1*genes	Chronic desynchrony, inflammation
Thermoregulation	Metabolism phase	Failure to reach core temperature nadir	Impaired glymphatic clearance

Note: NE = Norepinephrine; NMDA = N-methyl-D-aspartate; PER2/BMAL1 = core circadian clock genes. The table synthesizes the biphasic effect of ethanol based on Ebrahim et al. (2013) and Gardiner et al. (2025).

3.3. Chronobiological Impact: Circadian Rhythm and Homeostatic Disruption

The human sleep-wake cycle is governed by a sophisticated interplay between homeostatic sleep pressure and the endogenous circadian timing system. Alcohol serves as a potent "chronodisruptor," capable of desynchronizing the master biological clock located in the suprachiasmatic nucleus (SCN) of the hypothalamus, as well as peripheral oscillators throughout the body.

- **Molecular Clock Interference:** According to Bailey (2026) and Sharma & Nelson (2024), ethanol exposure interferes with the expression of core "clock genes" (such as *PER2*, *BMAL1*, and *CLOCK*). This molecular interference blunts the amplitude of circadian rhythms, meaning the signals that tell the body when to be awake and when to sleep become weakened and "noisy." This state of internal desynchrony is a primary driver of the persistent fatigue and insomnia observed in regular drinkers.

- **Melatonin and Thermoregulation:** Alcohol significantly suppresses the nocturnal secretion of melatonin, the hormone responsible for signaling biological darkness. Research synthesized by Chaput (2026) and O'Keefe & St-Onge (2022) indicates that even low doses of alcohol consumed in the evening can delay the onset of melatonin production. Furthermore, alcohol induces peripheral vasodilation which initially lowers core temperature, but the subsequent metabolic heat production prevents the body from reaching its thermal nadir. This failure to cool the core temperature efficiently prevents the brain from entering and maintaining deep NREM sleep stages.
- **Adenosinergic Homeostasis:** The homeostatic drive for sleep — often referred to as "Process S" — is mediated by the accumulation of adenosine in the brain. Thakkar et al. (2015) and Colrain et al. (2014) have demonstrated that alcohol artificially inflates adenosine levels, which explains the initial heavy sleepiness. However, this artificial surge is followed by a rapid collapse of adenosine signaling, leading to a state of "physiological wakefulness" in the early morning hours, long before the individual is actually rested.
- **Long-term Neurobiological Shifts:** For individuals with repeated exposure, the disruption of these rhythms becomes a chronic condition. Koob and Volkow (2016) highlight that the neurocircuitry of the "sleeping brain" undergoes neuroadaptive changes. The system becomes "locked" in a state of hyperarousal (allostasis), where the natural circadian signals are ignored by the brain's stress-response systems. This explains why, as noted by Vandekerckhove & Wang (2017), sleep disturbances are so closely linked to emotional dysregulation and the severity of depression in alcohol-dependent populations (Wescott et al., 2023).

3.4. Cognitive and Physiological Consequences: The "Morning-After" Deficit

The impact of alcohol-induced sleep loss extends into the post-metabolic phase, manifesting as significantly impaired daytime functioning. The cognitive deficits associated with the alcohol hangover — such as reduced attentional capacity and memory consolidation — cannot be attributed solely to ethanol toxicity; they are fundamentally exacerbated by the preceding night's fragmented sleep architecture.

- **Cognitive and Affective Impairment:** Stephens et al. (2008) and Gunn et al. (2018) provide evidence that the "hangover" state is characterized by a significant drop in executive function, which mirrors the effects of total sleep deprivation. Furthermore, the lack of REM sleep interferes with emotional processing, leading to the morning-after irritability and "affective blunting" described by Wescott et al. (2023).
- **Systemic Inflammation:** Sleep disruption caused by alcohol triggers a pro-inflammatory response. Irwin et al. (2016) found that even acute sleep loss following alcohol consumption increases levels of circulating inflammatory markers (such as IL-6). This systemic inflammation contributes to the overall "malaise" of the hangover and may play a role in the long-term health risks associated with regular drinking, even at moderate levels.

3.5. Clinical Directions: Managing Insomnia and Sleep Hygiene in Recovery

For individuals transitioning into long-term sobriety, insomnia is not merely a transient symptom but a persistent clinical barrier that predicts poor outcomes. Roehrs and Roth (2001) observe that sleep disturbances — characterized by increased sleep onset latency and decreased sleep efficiency — can endure for months or years after the last drink. This is driven by long-lasting neuroadaptive changes in the reward and stress neurocircuitry, particularly within the extended amygdala and the hypothalamic-pituitary-adrenal (HPA) axis, as described by Koob and Volkow (2016). The management of these disturbances requires a sophisticated, nuanced approach that moves beyond traditional sedation:

- **Advanced Pharmacological Interventions:** The selection of sleep-promoting agents for patients with Alcohol Use Disorder (AUD) is complicated by the high risk of cross-tolerance and the potential for secondary addiction. Roehrs et al. (2020) emphasize that benzodiazepines and Z-drugs (e.g., zolpidem) should be avoided due to their potential to induce relapse and their depressant effects on respiratory function during sleep. Current research highlights the efficacy of non-addictive alternatives such as gabapentin, which can stabilize GABAergic signaling, or trazodone and mirtazapine, which target serotonergic pathways. Furthermore, He, Hasler, and Chakravorty (2019) argue that treating sleep disorders and AUD simultaneously is essential, as the bidirectional risk — where poor sleep drives cravings — makes it impossible to treat one successfully without addressing the other.
- **Cognitive Behavioral Therapy for Insomnia (CBT-I): Kurth and Spada (2023)** highlight that many individuals in recovery have used alcohol as a primary "coping tool" for decades, leading to a complete lack of healthy sleep-promoting behaviors. Clinical guidance from American Addiction Centers (2024) advocates for CBT-I as the gold standard non-pharmacological treatment. This includes stimulus control (re-

associating the bed with sleep rather than wakefulness), sleep restriction therapy, and cognitive restructuring to address the anxiety associated with being unable to fall asleep without alcohol.

- **Affective Regulation and Sleep Hygiene:** The restoration of sleep is intimately tied to the patient's emotional state. Wescott et al. (2023) demonstrate that depression severity is a major moderator of sleep quality in recovery; patients with higher depressive symptoms experience more severe sleep duration deficits and morning-after affective blunting. Consequently, treatment must include tools for emotional regulation and mindfulness-based stress reduction, as recommended by Vandekerckhove and Wang (2017).
- **Long-term Prognosis and Chronotherapy:** For many in recovery, the circadian rhythm remains permanently shifted. Bailey (2026) suggests that chronotherapeutic approaches, such as morning light exposure and evening melatonin supplementation, can help "reset" the molecular clock that was damaged by chronic ethanol exposure. Addressing these chronobiological predictors early, as noted by Hasler et al. (2024), is not just about improving rest — it is a fundamental pillar of relapse prevention that targets the biological roots of addiction-related distress.

4. Discussion

The synthesis of evidence presented in this review confirms that alcohol-induced sleep disruption is not a transient inconvenience but a systemic pathology affecting neurochemical, chronobiological, and immunological pathways. The shift in the World Health Organization (2023) position toward a "no safe level" policy is a direct reflection of these pervasive effects. When integrating global research with national data from the Polish Academy of Sciences (2023) and PARPA (2023), it becomes evident that the cultural normalization of the "nightcap" in Poland is in direct conflict with physiological requirements for restorative sleep.

4.1 The Self-Medication Trap

A critical takeaway is the paradoxical nature of alcohol as a sleep aid. While Cederbaum (2012) and Ebrahim et al. (2013) confirm that alcohol facilitates rapid sleep onset, the subsequent "rebound effect" and autonomic hyperarousal negate any initial benefit. In the Polish context, where professional stress often correlates with increased alcohol consumption, this creates a dangerous feedback loop. The brain's attempt to achieve homeostasis through ethanol leads to chronic REM suppression, which, as Gardiner et al. (2025) and McCullar et al. (2024) suggest, progressively erodes the brain's natural regulatory capacity, making sobriety even more difficult to achieve due to persistent insomnia.

4.2 From Biology to Public Health

The evidence regarding chronodisruption (Bailey, 2026; Sharma & Nelson, 2024) adds a new dimension to public health concerns. Alcohol-induced desynchrony of the molecular clock is likely a primary driver of the systemic inflammation and metabolic disorders observed in heavy drinkers. When combined with the findings of Irwin et al. (2016) regarding pro-inflammatory cytokines, it appears that poor sleep quality is a key mediator of long-term morbidity. This suggests that public health initiatives in Poland should pivot from focusing solely on liver health to addressing the "invisible" damage to the circadian system.

4.3 Clinical Paradigm Shift

The persistence of sleep disorders in recovery (Roehrs et al., 2020; Koob & Volkow, 2016) underscores that treating sleep as a secondary symptom is a strategic error in addiction medicine. Since persistent sleep deficits significantly reduce inhibitory control, addressing sleep health must be viewed as a mandatory pillar of relapse prevention. The integration of CBT-I (Kurth & Spada, 2023) and non-addictive pharmacotherapy into standard Polish treatment protocols is essential to break the biological cycle of addiction-related distress.

5. Conclusions

Alcohol is a potent chronodisruptor: Even moderate consumption interferes with clock gene expression and melatonin secretion, leading to long-term circadian desynchrony.

The "Sleep Aid" myth is biologically refuted: The initial sedative effect is invariably followed by metabolic hyperarousal, REM suppression, and autonomic stress, resulting in non-restorative sleep.

Public health implications for Poland: National consumption patterns, as reported by PARPA (2023), require urgent educational interventions to correct the widespread misuse of alcohol as a sedative.

Recovery requires sleep stabilization: Managing insomnia through evidence-based methods (CBT-I and non-addictive pharmacotherapy) is critical for sobriety and the long-term cognitive health of individuals with AUD.

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