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CAFFEINE USE AS A MODULATOR OF SLEEP QUALITY AND DURATION: IMPLICATIONS FOR HEALTH AND BEHAVIOUR

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ABSTRACT

Caffeine is the most commonly consumed psychoactive substance worldwide, and it is a key factor modulating wakefulness. This makes its impact on sleep an important clinical and public health issue. The objective of this study was to provide a synthetic overview of current data on the effects of caffeine consumption on objective sleep parameters, including sleep latency, total sleep time, sleep efficiency, sleep architecture, and circadian rhythm regulation. These effects were taken to be contingent on dose, time of consumption, and population characteristics. A comprehensive literature review was conducted using the PubMed database, encompassing the period from 2000 to 2025. The focus of the review was on studies from the last decade or so, including randomized, observational, experimental, systematic review, and meta-analysis studies. The extant evidence unequivocally suggests that caffeine has a clear impact on the duration of sleep latency, the total duration of sleep, and the efficiency of sleep. It has been demonstrated that caffeine results in a reduction of slow-wave sleep (N3/SWS). These effects are dose- and time-dependent, and may persist even when caffeine is consumed up to six hours before bedtime. Polysomnography and electroencephalogram (EEG) studies have confirmed the presence of NREM sleep pressure disturbances and alterations in sleep organisation. The influence of individual variability, to a certain extent determined by polymorphisms of the ADORA2A and CYP1A2 genes, is of significance. Despite the extensive evidence base, there is still a lack of long-term prospective studies and projects that take genetic stratification into account. The results of the review indicate that caffeine should be considered an important modifiable risk factor for sleep disorders, and its rational use is a potentially simple but underutilized intervention to improve sleep health in the general population.

KEYWORDS

Caffeine, Caffeine Consumption, Adolescent Caffeine Use, Sleep Quality, Sleep Duration, Sleep Latency, Sleep Disturbances and Caffeine Metabolism

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1. Introduction

Sleep is considered a cornerstone of human health, playing a pivotal role in preserving neurocognitive performance, emotional regulation, and metabolic equilibrium. Chronic sleep insufficiency or fragmentation has been linked to memory impairments, mood disorders, insulin resistance, and increased cardiometabolic risk (1). In view of these critical functions, any factor that disrupts sleep architecture or sleep continuity warrants careful consideration.

Caffeine, the world's most widely consumed psychoactive substance, is one of the most pervasive modulators of wakefulness. The habitual intake of caffeine is characterised by its pervasiveness across a wide range of delivery forms, including coffee, tea, soft drinks, energy beverages, and even dietary supplements. This phenomenon is deeply entrenched in the daily routines of a significant proportion of the global population (2). A substantial body of research suggests that a considerable proportion of adults consume caffeine on a daily basis. However, the mean intakes and patterns of caffeine consumption vary considerably between countries and age-groups (3). This extensive exposure emphasises the public health significance of comprehending caffeine's interactions with sleep.

From a pharmacological perspective, the stimulant effects of caffeine are primarily attributable to its non-selective antagonism of adenosine A1 and A2A receptors within the central nervous system. The inhibition of these receptors by caffeine has been demonstrated to attenuate the endogenous sleep-promoting effects of adenosine, in addition to altering both homeostatic sleep pressure and circadian regulation (4). These molecular interactions can translate into measurable changes in sleep structure. For example, randomised crossover trials demonstrate that caffeine ingestion, even up to six hours before bedtime, prolongs sleep onset latency and reduces total sleep time and sleep efficiency (5).

Furthermore, controlled human studies employing polysomnography and actigraphy have indicated dose- and timing-dependent effects: the administration of larger caffeine doses (300–600 mg) and consumption in closer proximity to bedtime have been shown to yield more pronounced reductions in deep sleep (N3) and increases in light sleep (N1/N2) (6). Inter-individual variability is considerable: genetic polymorphisms related to caffeine metabolism and adenosine receptor signalling may modulate sensitivity to caffeine's sleep-disruptive effects (7). Paradoxically, habitual consumers may report no subjective sleep disruption despite objective changes, pointing to a discrepancy between physiological effects and perceived sleep quality (8).

Taken together, these observations reflect a growing public health and clinical imperative: understanding how caffeine consumption affects both objective and subjective sleep metrics. The objective of this review is to synthesise contemporary evidence on the impact of caffeine intake on sleep quality. In order to achieve this, epidemiological data, mechanistic insights, and polysomnographic findings will be integrated. The objective is to derive pragmatic recommendations for the safe consumption of caffeine in relation to sleep, with consideration for factors such as dosage, timing, and particular demographics.

2. Methodology

The present work constitutes a review of the existing literature on the subject. A systematic search of publications was conducted using PubMed database, supplemented by an analysis of references cited in key articles. The search strategy included terms related to caffeine consumption and sleep, namely: caffeine, caffeine consumption, adolescent caffeine use, sleep quality, sleep duration, sleep latency, sleep disturbances and caffeine metabolism. The analysis included articles published primarily between 2000 and 2025, with a particular emphasis on studies from the last 10–15 years. Original research studies (randomised, observational and experimental), as well as systematic reviews and meta-analyses focusing mainly on adult populations, were included. The applied inclusion criteria were as follows: publications in English, availability of full-text articles and a direct focus on the effect of caffeine on sleep quality, duration, architecture, as well as on related health and behavioural consequences. The literature review included a critical appraisal of study methodology, characteristics of the investigated populations, methods used to assess caffeine intake and sleep parameters and reported limitations. Conclusions were formulated through a synthetic integration of research findings on the mechanisms of caffeine action, individual variability (including metabolic determinants), sleep disturbances, and their implications for health and behaviour.

3. Results

3.1. Pharmacological and Neurophysiological Effects of Caffeine

Caffeine exerts its primary influence on sleep through its central neurophysiological action, as evidenced by objective changes in the electroencephalogram (EEG) recording during sleep. A number of experimental studies have demonstrated that the ingestion of caffeine results in a reduction of markers associated with the pressure of NREM sleep, in addition to modifying the parameters of neuronal activity during sleep, particularly within the NREM phases (9). The aforementioned alterations include a reduction in slow wave activity and alterations in EEG signal complexity measures, which are interpreted as sleep shallowing (7). Significant individual variability in response to caffeine has been demonstrated, partly dependent on variants of genes associated with adenosine signaling (ADORA2A). Individuals exhibiting specific polymorphisms appear to demonstrate an increased propensity for sleep disturbances and electroencephalogram (EEG) alterations following caffeine consumption, thereby substantiating the biological underpinnings that underpin individual variations (10).

3.2. Impact on Objective Sleep Parameters

Polysomnographic analyses demonstrate that caffeine modifies the architecture of sleep, leading to a shift towards more superficial sleep. Research has demonstrated that the ingestion of caffeine is associated with a reduction in slow-wave sleep (N3/SWS) by an average of 10–20 minutes and an increase in the proportion of N1/N2 stages, while simultaneously increasing sleep latency and reducing sleep efficiency (9, 11). SWS reduction has been identified as one of the most consistent effects of caffeine observed in PSG recordings. A recent meta-analysis of crossover studies confirmed a significant reduction in the proportion of slow-wave sleep (−0.8 to −1.2%), with no consistent effect on the total percentage of REM sleep, indicating greater variability in the results for this sleep phase (12). The findings from individual polysomnographic studies indicate that the impact of caffeine on REM sleep primarily pertains to its temporal organisation. In a randomised study, regular caffeine consumption led to an increase in REM latency from approximately 54 to

79 minutes, without significant changes in total REM time (13). Conversely, studies examining the relationship between caffeine dosage and timing of consumption have demonstrated that a dose of 400 mg of caffeine consumed ≤12 hours prior to sleep onset results in substantial alterations to the architecture of NREM and REM sleep. The magnitude of these effects is known to increase in proportion to the reduction in the time interval preceding sleep (4). Table 1 shows changes in sleep parameters caused by the effects of caffeine.

Table 1. Summary of objective and neurophysiological data on the effects of caffeine on sleep architecture and parameters.

Sleep domain	Effect of caffeine	Direction of change	Source
Sleep onset latency (SOL), Total sleep time (TST), Sleep efficiency (SE), N1/N2, N3/SWS, REM	Sleep shallowing, reduced continuity	↑ SOL; ↓ TST, ↓ SE; ↓ N3; ↓ N3/SWS; ↑ N1/N2; inconsistent effects on REM	(14)
SOL, N1, N2, SWS, TST, SE, REM (recovery sleep)	Sleep onset delay; increased light sleep; reduced deeper stages and sleep efficiency; reduced REM in recovery sleep	↑ SOL; ↑ N1; ↓ N2; ↓ SWS; ↓ TST & SE; ↓ REM (only in daytime recovery sleep)	(15)
NREM sleep (EEG spectral power)	Neurophysiological changes	↓ delta power; gentype-dependent sigma changes	(16)
REM latency, REM temporal distribution	Delayed REM initiation	↑ REM latency; no increase in REM	(13)
Sleep onset latency, Slow-wave sleep (chronic exposure)	Insomnia-like pattern	↑ SOL; ↓ SWS	(17)

3.3. Relationship between Caffeine Dose, Time of Consumption and Population Characteristics in Modulating Sleep Outcomes

The impact of caffeine on sleep is dose- and timing-dependent. In controlled studies, the ingestion of 400 mg of caffeine 0, 3, or 6 hours before bedtime resulted in a reduction in total sleep time of approximately 41–60 minutes and a decrease in sleep efficiency, with significant disruption persisting even when caffeine was consumed 6 hours prior to sleep (18). Polysomnographic data further indicate that regular caffeine intake (3 × 150 mg/day) prolongs REM sleep latency by approximately 25 minutes without increasing total REM duration, suggesting altered temporal organisation of sleep rather than changes in REM quantity (13).

It has been demonstrated that the presence of caffeine in the diet is associated with a reduction in the total duration of sleep (average difference of approximately –35 minutes), a decrease in sleep efficiency (approximately −4–5%), increased sleep latency (approximately +8 minutes), and a reduction in slow-wave sleep (SWS/N3) of approximately −1%, with no significant consistent effect on the total percentage of REM sleep in the pooled analyses (12).

The observed variations in these relationships are indicative of significant disparities among different population groups. Observational studies in adolescents and young adults indicate that higher caffeine intake, particularly in the form of energy drinks, is associated with shorter sleep duration, delayed sleep onset, and a higher frequency of subjective sleep problems (19, 20). Conversely, studies undertaken in the context of everyday life among older adults, utilising accelerometry as a method of data collection, have indicated comparatively weaker or non-existent associations between moderate caffeine intake (up to 200 mg per day) and sleep parameters, including sleep efficiency and duration (21).

Furthermore, cohort analyses employing polysomnography demonstrate that elevated habitual caffeine intake is associated with diminished objective sleep parameters, even in the absence of laboratory conditions, thereby substantiating the clinical significance of the observed effects within the general population (6).

4. Research Gaps and Future Directions.

Despite the extensive body of literature on this topic, there remains a notable paucity of long-term prospective studies examining the relationship between chronic caffeine consumption, objectively measured sleep parameters, and long-term health outcomes. Most existing evidence is derived from short-term experimental or cross-sectional studies, which substantially limits the ability to draw causal inferences. Interindividual variability in caffeine sensitivity, partly attributable to genetic polymorphisms in genes such as *ADORA2A* and *CYP1A2*, remains insufficiently characterised and is rarely incorporated into large-scale population-based research. An additional methodological limitation is the lack of standardisation in the assessment of both caffeine intake and sleep parameters, which hinders meaningful comparison across studies and complicates the conduct of robust meta-analyses. Future research should prioritise well-designed intervention studies, including caffeine restriction protocols, modification of consumption timing, and substitution with decaffeinated beverages. Furthermore, the integration of contemporary sleep-monitoring technologies—such as wearable devices and home-based electroencephalography (EEG)—is essential to enable long-term assessment of sleep in real-world settings.

5. Conclusions

The findings of this study demonstrate that caffeine exerts a substantial influence on sleep parameters, manifesting in the prolongation of sleep latency, the reduction of total sleep time, the diminution of sleep efficiency, and the decrease in slow-wave sleep. The observed effects are contingent on the administered dose, the time of consumption, and the individual characteristics of the subject. The findings of this study provide a scientific rationale for recommending the moderation of caffeine intake, particularly during the afternoon and evening hours, with a recommended minimum interval of 6–8 hours before bedtime. The significance of these observations extends beyond the individual level, thus holding important implications for clinical practice and public health. Further progress in this area is contingent on the execution of long-term, genetically stratified, and interventional studies founded upon objective methodologies of sleep measurement. The implementation of such studies will facilitate the development of precise, evidence-based recommendations.

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