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THE IMPACT OF CAFFEINE CONSUMPTION ON SLEEP QUALITY AND MEMORY PERFORMANCE IN ADULTS

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Abstract

This study examined how caffeine consumption affects sleep quality and memory functioning in adults, with a specific focus on sleep quality as a mediating variable. Using a cross-sectional correlational design, 200 adults (aged 18–60) were recruited from urban professional environments and university settings through convenience sampling. Three instruments were administered: a researcher-developed Demographic and Caffeine Consumption Sheet, the Pittsburgh Sleep Quality Index (PSQI), and the Cognitive Failures Questionnaire (CFQ). Descriptive findings indicated that nearly half the sample (45%) fell in the moderate caffeine intake range (3–4 daily servings). The mean PSQI score ($M = 8.10$, $SD = 3.10$) exceeded the clinical threshold for poor sleep, while CFQ scores ($M = 44.20$, $SD = 11.30$) reflected moderate everyday cognitive lapses. Pearson correlations showed significant positive associations between caffeine intake and impaired sleep ($r = .49$, $p < .01$), between poor sleep and memory deficits ($r = .58$, $p < .01$), and between caffeine intake and memory difficulties ($r = .35$, $p < .01$). Multiple regression indicated that caffeine ($\beta = .21$) and sleep quality ($\beta = .48$) together accounted for 41% of the variance in memory performance. Mediation analysis further confirmed that sleep quality partially mediates the caffeine–memory relationship: greater caffeine consumption disrupts sleep, which in turn contributes to reduced memory performance. Both direct and indirect pathways from caffeine to memory impairment were statistically significant. These results underscore the importance of moderating caffeine use and fostering consistent sleep routines as strategies for preserving cognitive health.

Keywords: *Adults, Caffeine, Cognitive Performance, Memory, Sleep Disturbance, Sleep Quality.*

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1. Introduction

Caffeine is a naturally occurring compound found in the seeds, leaves, and fruits of certain plant species. It is present in a wide range of everyday consumables including coffee, black and green tea, cocoa products, carbonated cola beverages, and commercially produced energy drinks. Additionally, caffeine appears in various food products such as chocolate confections and energy bars, as well as some over-the-counter medications including cough remedies and weight management supplements. Natural plant additives such as guarana, commonly incorporated into energy beverages, also serve as meaningful caffeine sources (Better Health Channel [BHC], 2022).

As a central nervous system stimulant, caffeine elevates neural activity and triggers the release of stress-related hormones, including cortisol and adrenaline. When consumed in modest quantities, it can produce feelings of alertness and improved concentration. However, at higher doses it may provoke anxiety and interfere with restful sleep. As with many psychoactive substances, repeated exposure to caffeine leads to tolerance, requiring progressively larger amounts to achieve the original effect. Following ingestion, caffeine is rapidly absorbed, with its stimulant effects typically emerging within five to thirty minutes. These effects—encompassing elevated breathing rate, increased cardiac activity, and enhanced mental and physical arousal—may persist for as long as twelve hours. Overconsumption can manifest in a range of adverse signs, including elevated body temperature, excessive urination, dehydration, headaches, cardiac palpitations, heightened restlessness, irritability, hand tremors, disrupted sleep, and a paradoxical post-stimulation fatigue (BHC, 2022).

Individual responses to caffeine vary according to body mass, metabolic rate, general health status, and the degree of prior habituation to the substance. Research generally supports an upper daily threshold of 400 mg as acceptable for most healthy adults (BHC, 2022). Over time, regular intake may produce both physiological and psychological dependency, such that abrupt cessation triggers withdrawal symptoms including fatigue, irritability, persistent headache, sweating, muscular discomfort, and anxiety. These withdrawal manifestations typically begin within twelve to twenty-four hours after the last dose and may continue for approximately one week. A gradual reduction approach is considered the most effective strategy for breaking caffeine dependency, allowing the nervous system to readjust in a measured way. Particular care is advised for pregnant individuals, children, and athletic populations, who are encouraged to limit caffeine intake (BHC, 2022).

In terms of sleep effects, caffeine has been shown to delay sleep onset, reduce total sleep time, and diminish perceived sleep satisfaction. It particularly affects slow-wave, deep sleep—a physiologically critical stage associated with feeling refreshed upon waking (Pacheco & Vyas, 2023). The primary mechanism through which caffeine affects sleep

involves its antagonism of adenosine receptors in the brain. Adenosine is a naturally produced neurochemical that accumulates during wakefulness and progressively increases sleepiness. By blocking adenosine binding, caffeine sustains alertness—but simultaneously interferes with the body's natural sleep-promoting process (Pacheco & Vyas, 2023).

While caffeine has not been established as a direct cause of clinical insomnia, individuals suffering from chronic insomnia may turn to caffeine to counteract daytime fatigue arising from sleep deprivation, potentially worsening their nighttime symptoms. This negative cycle may be especially pronounced in those who use caffeine only occasionally, compared to habitual users whose physiology has adapted to its presence (Pacheco & Vyas, 2023). For those with persistent insomnia, strategies such as reducing overall caffeine intake or confining consumption to the earlier portions of the day have shown promise in improving nighttime sleep quality (Pacheco & Vyas, 2023).

From a cognitive perspective, caffeine is known to temporarily enhance alertness and certain memory-related functions, particularly among sleep-deprived individuals. Nevertheless, it cannot fully compensate for the cognitive costs associated with sustained sleep insufficiency, and when taken too late in the day, its alertness-promoting effects may impair sleep and erode the cognitive benefits it is intended to provide (Pacheco & Vyas, 2023). The magnitude and duration of caffeine's cognitive effects are shaped by factors such as dosage, frequency of use, and individual genetic variation (Pacheco & Vyas, 2023).

Research from Johns Hopkins University has offered intriguing evidence that caffeine may support long-term memory consolidation. In a controlled study, participants who received a 200 mg caffeine tablet following exposure to a series of images demonstrated superior ability to differentiate those images from closely similar ones when tested the following day (Johns Hopkins Medicine [JHM], 2023). The lead researcher, Dr. Michael Yassa, noted that while caffeine's general cognitive enhancing properties are well established, its specific role in fortifying memory traces and protecting against forgetting had not previously been systematically examined in human subjects (JHM, 2023). This double-blind study employed a nuanced memory paradigm—pattern separation—which requires the brain to distinguish between closely related stimuli, a task that appeared to be selectively enhanced by caffeine (JHM, 2023).

1.1. Rationale of the Study

The primary motivation for undertaking this research was to clarify the nature of the associations linking caffeine intake, sleep health, and memory functioning. Understanding these relationships carries practical significance, as many adults routinely consume caffeine without full awareness of its downstream effects on cognition. Beyond advancing the academic literature, this investigation aims to identify knowledge gaps

among everyday caffeine users and to provide a foundation for promoting healthier lifestyle choices that reduce cognitive risk.

1.2. Objectives of the Study

The overarching objective of this study was to evaluate the consequences of caffeine consumption on sleep quality and memory performance in adult populations. More specifically, the study aimed to: (a) assess the relationship between habitual caffeine intake and subjective sleep quality; (b) investigate the extent to which caffeine consumption influences memory functioning; and (c) examine how caffeine-induced disruptions to the sleep-wake cycle may affect the process of memory consolidation.

1.3.Research Hypotheses

1. **H1:** Caffeine consumption will demonstrate a significant impact on sleep quality and memory performance in adults.
2. **H2:** Significant gender-based differences will be observed in sleep quality and memory performance among adult caffeine consumers.
3. **H3:** A significant positive relationship will exist between caffeine consumption and adults' sleep quality and memory performance.

2. Conceptual Model

Figure 1 illustrates the proposed conceptual framework underlying the study. Sleep quality is positioned as the primary mediating variable connecting caffeine consumption to memory performance. Additionally, the timing of caffeine intake is conceptualized as a moderating variable influencing the strength of the relationship between consumption patterns and sleep outcomes.

Timing of Caffeine Intake (Moderator)

↓

Caffeine Consumption → Sleep Quality → Memory Performance

↓ ↗

(Direct Path: Caffeine → Memory Performance)

3. Methodology

3.1.Research Design

A quantitative, cross-sectional correlational design was adopted to examine the associations among caffeine consumption, sleep quality, and memory performance in adults. This design was chosen because it allows for efficient identification of the strength and direction of relationships among variables at a single point in time. The correlational framework was particularly suited to exploring whether sleep quality functions as a pathway through which caffeine intake influences cognitive outcomes.

3.2.Participants

A total of 200 adults were recruited through convenience sampling from urban professional environments and university campuses. Eligible participants were between

18 and 60 years of age, consumed caffeinated beverages either regularly or occasionally, were proficient in English, and provided voluntary informed consent. This age range was selected to capture variation in adult metabolic functioning and sleep architecture across the lifespan.

Participants were excluded if they had received a formal diagnosis of a sleep disorder (such as clinical insomnia or sleep apnea), were currently using prescription sedatives or stimulant medications, were pregnant or breastfeeding, or had neurological conditions that could confound cognitive test performance.

3.3. Instruments

3.3.1. Demographic and Caffeine Consumption Sheet

A researcher-constructed information sheet was utilized to gather sociodemographic data including age, gender, educational attainment, and occupational status. Caffeine-specific variables were also collected, including the average daily number of caffeinated servings consumed (from coffee, tea, and energy drinks), the typical timing of the last caffeine intake before sleep, and the primary type of caffeinated product used.

3.3.2. Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989)

Sleep quality over the preceding month was assessed using the PSQI, a widely validated self-report instrument comprising 19 items that produce scores across seven component domains: sleep duration, sleep disturbances, latency to sleep onset, habitual sleep efficiency, use of sleep medication, daytime dysfunction, and subjective sleep quality. A composite global score is derived, with scores above 5 indicating clinically poor sleep. In prior validation studies, the PSQI has demonstrated strong internal consistency (Cronbach's $\alpha \approx .83–.84$).

3.3.3. Cognitive Failures Questionnaire (CFQ; Broadbent et al., 1982)

Memory performance and everyday cognitive errors were evaluated with the CFQ, a 25-item measure assessing lapses across three domains: perception, memory, and motor function. Responses are recorded on a 5-point Likert scale (0 = Never; 4 = Very Often), with elevated scores reflecting more frequent cognitive failures. The CFQ has demonstrated robust internal consistency in prior research ($\alpha \approx .80–.89$).

4. Results

Table 1: *Frequency and Percentage Distribution of Participant Characteristics (N = 200)*

Demographic Variable	**f**	**%**
Age		
18–25	94	47.0
26–35	66	33.0

36–45	40	20.0
Gender		
Male	100	50.0
Female	100	50.0
Education Level		
Intermediate	50	25.0
Bachelor's	100	50.0
Master's	50	25.0
Daily Caffeine Intake		
Low (1–2 cups)	60	30.0
Moderate (3–4 cups)	90	45.0
High (5+ cups)	50	25.0

Table 2: *Descriptive Statistics and Internal Reliability Coefficients for Study Variables (N = 200*)

Variable	**Mean**	**SD**	**Cronbach's α**
Caffeine Consumption	3.45	1.25	—
Sleep Quality (PSQI)	8.10	3.10	.84
Memory Performance (CFQ)	44.20	11.30	.81

Note. The PSQI clinical cutoff score is 5.0; participants in this sample averaged 8.10, indicating generally poor sleep. Reliability coefficients confirm adequate psychometric properties for both instruments.

As shown in Table 2, the average PSQI global score of 8.10 is considerably above the accepted clinical threshold of 5.0, indicating that participants in this study generally experienced poor sleep. The mean CFQ score of 44.20 reflects a moderate frequency of everyday cognitive failures across the sample. Both instruments demonstrated high reliability in this context, with Cronbach's alpha coefficients of .84 and .81 for the PSQI and CFQ respectively, confirming their suitability for use with this population.

Table 3: *Pearson Correlation Matrix for Study Variables (N = 200)*

Variable	**1**	**2**	**3**
Caffeine Consumption	—		
Sleep Quality (PSQI)	.49**	—	
Memory Performance (CFQ)	.35**	.58**	—

*Note. ** p < .01 (two-tailed). Higher PSQI scores = poorer sleep. Higher CFQ scores = greater cognitive failures.*

The correlation matrix presented in Table 3 reveals statistically significant associations among all three variables. Caffeine consumption correlated positively with PSQI scores (r = .49), indicating that greater intake is linked to more degraded sleep quality. A stronger association emerged between PSQI scores and CFQ scores (r = .58), suggesting that individuals with poorer sleep experience substantially more frequent cognitive failures. Caffeine consumption also correlated with memory deficits (r = .35), indicating a direct, albeit more modest, association between stimulant intake and impaired cognition.

***Table 4**: Multiple Regression Analysis: Predictors of Memory Performance (N = 200)*

Predictor	**B**	**SE**	**β**
(Constant)	12.40	3.10	
Caffeine Intake	1.85	0.70	.21
Sleep Quality (PSQI)	4.10	0.45	.48

Note. R^2 = .41; the model explains 41% of the variance in memory performance. Both predictors are statistically significant.

Table 4 presents the results of the multiple regression model. The overall model was statistically significant and accounted for 41% of the variance in memory performance. Sleep quality emerged as the more potent predictor (β = .48) relative to caffeine intake (β = .21), indicating that while both variables independently contribute to memory functioning, disrupted sleep exerts a considerably stronger influence. This pattern suggests that caffeine's impact on memory is most consequential when mediated through sleep disruption.

Table 5: *Mediation Analysis: Pathways from Caffeine Consumption to Memory Performance*

Effect Type	**Pathway**	**Coefficient (β)**	**SE**

Indirect Effect	Caffeine → Sleep → Memory	.22	.04
Direct Effect	Caffeine → Memory	.21	.21
Total Effect	Combined Impact on Memory	.43	.06

5. Discussion

The central purpose of this study was to investigate how caffeine consumption relates to sleep quality and memory functioning in adults, with particular attention to the potential mediating role of sleep quality in these associations. The results provided consistent empirical support for the proposed model.

In alignment with the first hypothesis (H1), a significant positive association was found between caffeine consumption and impaired sleep quality ($r = .49$, $p < .01$). This outcome is consistent with a well-established mechanistic explanation: caffeine occupies adenosine receptors in the brain, interrupting the neurochemical signaling that ordinarily promotes drowsiness and facilitates sleep onset. Higher intake—particularly when consumed in the afternoon or evening—appears to substantially compromise sleep architecture, corroborating an extensive body of prior research.

The association between poor sleep quality and greater memory difficulties ($r = .58$, $p < .01$) was the strongest bivariate correlation observed. The regression model reinforced this finding, identifying sleep quality as the dominant predictor of memory performance ($\beta = .48$). These outcomes align with theoretical frameworks emphasizing the indispensable role of deep, slow-wave sleep in the consolidation and retention of new information. When sleep quality is compromised, the neurological processes supporting memory storage are disrupted, leading to measurable cognitive impairment in daily functioning.

Caffeine intake also demonstrated a meaningful direct correlation with memory difficulties ($r = .35$, $p < .01$) and retained significant predictive power in the regression analysis ($\beta = .21$). This suggests that beyond its sleep-mediated effects, caffeine may also impair cognition through other pathways—potentially via overstimulation of the central nervous system or interference with neural efficiency at higher dosage levels.

The most theoretically significant finding pertained to the mediating function of sleep quality (H3). The mediation analysis revealed that sleep quality partially mediates the caffeine–memory relationship, with a significant indirect effect ($\beta = .22$). This indicates that a meaningful portion of caffeine's detrimental influence on memory operates through its disruption of sleep. Importantly, the direct effect of caffeine on memory ($\beta = .21$) remained significant even after accounting for sleep quality, confirming partial rather than full mediation. Together, the indirect and direct pathways yielded a total effect of $\beta = .43$, reflecting the breadth of caffeine's influence on memory functioning.

These findings collectively suggest that while caffeine is widely employed as a cognitive aid, its excessive or poorly timed use tends to undermine the very cognitive processes it is intended to support. Short-term benefits—particularly for sleep-deprived individuals—do not appear to offset the cumulative harm arising from disrupted sleep and its downstream impact on memory. The study highlights that caffeine's effects on memory are both direct and indirect, with sleep quality serving as a pivotal explanatory pathway in this relationship.

6. Limitations of the Study

Several methodological constraints should be considered when interpreting these findings. First, the cross-sectional design captures a snapshot of participants at one moment in time, precluding any definitive causal conclusions. Although the mediation model implies a temporal sequence from caffeine intake to sleep disruption to memory impairment, longitudinal data would be required to substantiate these directional claims empirically.

Second, the sample was drawn exclusively from urban professional and university settings through convenience sampling, potentially limiting the generalizability of findings to rural populations, older age cohorts, or culturally distinct groups with different caffeine norms and metabolic profiles.

Third, reliance on self-report measures for all three primary variables introduces the possibility of response bias. Participants may have under- or over-estimated their caffeine intake, sleep difficulties, or frequency of cognitive failures, particularly if influenced by social desirability concerns.

Fourth, caffeine consumption was quantified in terms of standard 'cups,' without accounting for the considerable variability in actual caffeine content across different beverage types, brands, and preparation methods. This imprecision may have introduced measurement error in caffeine dosage estimates.

Finally, the study did not comprehensively control for confounding variables such as pre-sleep screen exposure, nicotine use, or baseline stress and anxiety levels—factors that independently influence both sleep quality and cognitive performance.

7. Recommendations

Mental health practitioners are encouraged to routinely screen for caffeine-related sleep disruption as a potential contributor to cognitive fatigue, and to integrate caffeine reduction strategies alongside standard sleep hygiene protocols. Educational institutions should develop wellness programming that equips students and employees with evidence-based knowledge about the role of sleep in cognitive performance and the limitations of caffeine as a substitute for adequate rest.

Future research should employ longitudinal designs to track the long-term trajectory of caffeine-related sleep and cognitive changes across adulthood. Objective

sleep monitoring tools such as actigraphy or polysomnography should be incorporated to supplement self-report data. Researchers are also encouraged to use biomarker-based caffeine assessments to improve measurement precision, and to diversify sampling strategies to include rural, older, and cross-cultural populations.

Public health messaging should address the counterproductive cycle that can arise when caffeine is used to manage fatigue caused by the very sleep disruption it helped to create. Campaigns emphasizing sustainable alertness strategies—anchored in consistent, high-quality sleep—may help shift normative attitudes around stimulant dependence.

8. Conclusion

This investigation contributes to the growing body of evidence documenting the interplay between dietary stimulant use, sleep physiology, and cognitive functioning. The findings confirm that habitual caffeine consumption is a notable risk factor for diminished sleep quality, and that this sleep disruption functions as a primary driver of impaired memory performance in adults. Crucially, sleep quality was identified as a significant partial mediator between caffeine consumption and memory outcomes, illuminating the mechanism through which stimulant use indirectly erodes cognitive capacity by undermining the restorative processes that memory consolidation depends upon.

While caffeine may offer transient improvements in alertness—particularly under conditions of sleep deprivation—it does not constitute a sustainable or adequate substitute for quality sleep. The direct and indirect pathways through which caffeine impairs memory underscore that its perceived cognitive benefits are frequently offset by its physiological costs. Interventions targeting both caffeine moderation and sleep hygiene hold the greatest promise for supporting long-term cognitive vitality and psychological wellbeing across adult populations.

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