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ORIGINAL RESEARCH

# Perceived Stress and Clinical Insomnia in Primary Care: Associations with Lifestyle and Medication Use

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**Purpose:** Insomnia and perceived stress are among the most prevalent health concerns in primary care, yet their complex relationship remains underexplored. This study examines the association between perceived stress and insomnia severity, while considering the potential moderating or mediating effects of lifestyle factors and medication use.

**Methods:** A cross-sectional study was conducted with 300 consecutive primary care patients who completed validated questionnaires assessing insomnia severity (Insomnia Severity Index, ISI) and perceived stress (Perceived Stress Scale, PSS). Logistic regression models were employed to identify predictors of clinical insomnia and explore key relationships.

**Results:** A moderate but statistically significant correlation ($r = 0.460$, $p < 0.001$) was observed between perceived stress and insomnia severity. In logistic regression analysis, higher perceived stress scores (OR = 1.134, $p < 0.001$) and hypnotic medication use (OR = 3.220, $p < 0.001$) were significant predictors of clinical insomnia. Interestingly, alcohol consumption was unexpectedly associated with insomnia (OR = 0.551, $p = 0.048$), warranting further exploration. No significant associations were found for caffeine intake, tobacco use, or demographic variables. The model explained 33% of the variance in insomnia (Nagelkerke $R^2 = 0.328$).

**Conclusion:** These findings highlight the significant role of perceived stress in insomnia severity and underscore the complex interplay between pharmacological interventions and sleep disturbances. While hypnotic medications are commonly used, their long-term impact and potential dependence require careful consideration. Integrating stress-reduction strategies into primary care may improve insomnia management while reducing reliance on pharmacological treatments. Future research should employ longitudinal designs to clarify causality and further explore additional factors influencing sleep disturbances.

**Why was this study conducted?:** Stress and insomnia are common in primary care, but their relationship remains unclear. This study explores how perceived stress influences insomnia severity and whether factors like medication use, alcohol, or caffeine affect this link.

**What did the study find?:** Higher stress levels significantly increased the risk of clinical insomnia; sleep medication use tripled the likelihood of insomnia, suggesting possible dependence; alcohol consumption was unexpectedly linked to lower insomnia risk, though this requires further investigation; and, caffeine and tobacco had no significant impact on insomnia.

**What do these results mean?:** Managing stress is key to improving sleep. While sleep medications are widely used, they may not always be the best solution. Healthcare providers should prioritize stress-reduction strategies and non-pharmacological insomnia treatments to improve sleep quality in primary care patients.

**Keywords:** insomnia, perceived stress, primary care, sleep medication, lifestyle factors, mental health

## Introduction

Insomnia and perceived stress are two of the most common health challenges affecting individuals globally, significantly impacting both mental and physical well-being.[1–4] Insomnia, characterized by persistent difficulty in initiating or maintaining sleep, affects approximately 10–30% of the general population, with prevalence rates being higher among



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older adults and individuals with chronic health conditions.[5] Beyond its effects on sleep quality, chronic insomnia is a significant risk factor for depression, anxiety disorders, cardiovascular diseases, and metabolic disorders.[6–9]

Similarly, perceived stress, defined as the subjective appraisal of life events as overwhelming, has been associated with adverse health outcomes, including hypertension, immune system dysfunction, and psychiatric conditions such as anxiety and post-traumatic stress disorder.[10,11] Despite the individual impact of insomnia and stress, their bidirectional relationship suggests a cyclical pattern where stress disrupts sleep patterns through physiological hyperarousal, dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, and increased cortisol levels.[12] Conversely, insomnia exacerbates stress by impairing emotional resilience and cognitive functioning.[13]

## The Primary Care Context

Primary care settings play a pivotal role in managing both stress and insomnia, as they are often the first point of contact for patients experiencing these conditions. Epidemiological data suggest that up to 50% of primary care patients report sleep disturbances, and stress-related complaints are among the most frequently cited reasons for medical consultations.[14] These conditions are frequently under-recognized in clinical settings, with time constraints and somatic symptom focus contributing to their underdiagnosis. However, insomnia and stress are often underdiagnosed and undertreated in primary care, as they may present with non-specific symptoms or be overshadowed by coexisting medical conditions.[15] Moreover, patients may receive treatments that target only one of these conditions, neglecting the complex interplay between stress and sleep disturbances. Understanding the associations between perceived stress and insomnia in a primary care population is crucial for developing integrated, early intervention strategies.

## Gaps in Existing Research

Previous studies examining the role of alcohol, caffeine, and tobacco in insomnia have reported inconsistent findings. While these substances are often linked to disrupted sleep, some studies have suggested no significant or even paradoxical effects, highlighting the need for more nuanced analyses. Despite the well-documented link between stress and insomnia, several knowledge gaps remain.[16] Many studies have relied on cross-sectional designs, limiting the ability to establish causality or temporal precedence in these associations. Furthermore, while biological mechanisms such as HPA-axis dysregulation have been proposed, the influence of lifestyle and behavioral factors—including caffeine and alcohol consumption—remains underexplored. Additionally, previous research has often overlooked how demographic characteristics such as age, gender, and medication use may moderate the relationship between perceived stress and insomnia. Notably, insomnia has also been identified as an independent risk factor for suicidal ideation and behavior, further underscoring its public health relevance.[17] Addressing these gaps is essential for tailoring effective interventions in clinical practice.

## Study Objectives and Potential Contributions

This study aims to explore the associations between perceived stress and insomnia in primary care patients, with a focus on identifying key mediators and moderators that might explain or alter the strength of these relationships. Specifically, we will:

1. Describe patients' sleep habits and perceived stress levels to establish a comprehensive clinical profile.
2. Evaluate the mediating role of substance use (eg, caffeine and alcohol) in the relationship between stress and sleep disturbances.
3. Investigate whether age, gender, or medication use moderate the association between perceived stress and insomnia severity.

This study is among the first to simultaneously model perceived stress, lifestyle behaviors, and medication use as joint predictors of clinical insomnia in a primary care sample. By addressing these questions, our findings are expected to inform more effective, integrative therapeutic strategies, combining stress-reduction interventions (eg, cognitive-

behavioral therapy, mindfulness-based approaches) with sleep-focused treatments. Ultimately, this research aims to enhance early identification and management of stress-related sleep disturbances within primary care settings.

# Methods

## Study Design and Participants

This cross-sectional study was conducted in a primary care center, recruiting 300 consecutive adult patients attending routine consultations. Participants were ≥18 years old, fluent in Spanish, and capable of completing the assessments independently.

Exclusion criteria included: a history of severe psychiatric disorders (eg, schizophrenia, bipolar disorder, or major depressive disorder requiring hospitalization); neurological conditions affecting cognitive function (eg, epilepsy, Parkinson's disease); medical conditions requiring urgent hospitalization at the time of recruitment. All participants provided informed consent before study participation, and ethical approval was obtained from the institutional review board (Canary Islands Health Service Ethics and Research Committee protocol code TFG 2024/25). The study was conducted from September 2024 to January 2025, encompassing patient recruitment, data collection, and preliminary analysis.

## Measures

### Insomnia Severity Index (ISI)

The ISI is a 7-item self-report questionnaire assessing the severity of insomnia symptoms over the past two weeks.[18,19] Each item is scored on a 5-point Likert scale (0 = no difficulty to 4 = very severe difficulty), with total scores ranging from 0 to 28. The ISI classifies insomnia severity as follows: 0–7: No clinically significant insomnia; 8–14: Subthreshold (mild) insomnia; 15–21: Moderate insomnia; 22–28: Severe insomnia. The ISI is widely validated and has demonstrated high internal consistency (Cronbach's $\alpha = 0.91$) and strong correlations with objective and subjective sleep measures in primary care and clinical settings.

### Perceived Stress Scale (PSS-14)

The PSS-14 is a 14-item scale that measures perceived stress levels over the past month, focusing on how unpredictable, uncontrollable, and overwhelming respondents find their lives.[20,21] Items are rated on a 5-point scale (0 = never to 4 = very often), with total scores ranging from 0 to 56. Higher scores indicate greater perceived stress, with typical classifications as: 0–18: Low stress: 19–37: Moderate stress; 38–56: High stress The PSS-14 is one of the most widely used stress assessment tools, demonstrating strong psychometric properties across diverse populations (Cronbach's $\alpha = 0.84–0.86$).

## Lifestyle and Substance Use Factors

Participants self-reported their consumption of stimulants and substances affecting sleep, including: daily caffeine intake (frequency of coffee, tea, cola, or energy drink consumption) daily tobacco use (current smoker vs non-smoker), and daily alcohol consumption (frequency of drinking over the past month).

## Medication Use

Participants were asked to indicate their frequency of hypnotic medication use, categorized as: never, less than once a week, 1–2 times per week, and 3 or more times per week.

# Statistical Analysis

All analyses were conducted using SPSS Statistics (version 29).[22] Descriptive statistics (means, standard deviations, and percentages) were used to summarize participant characteristics. Pearson's correlation analysis examined the association between perceived stress (PSS-14) and insomnia severity (ISI total score). Binary logistic regression was performed to identify predictors of clinical insomnia (ISI ≥ 15). The independent variables included perceived stress scores, hypnotic medication use, and lifestyle factors (caffeine, alcohol, and tobacco use). Odds ratios (OR) with 95% confidence intervals (CI) were reported. A significance threshold of $p < 0.05$ was applied for all statistical tests.

## Ethical Considerations

The study adheres to the General Data Protection Regulation (GDPR) and the principles of the Declaration of Helsinki. Participation is voluntary, with participants able to withdraw at any stage without repercussions. All data will remain anonymized, and the study protocol has been approved by the local ethics committee.

## Results

### Demographic and Anthropometric Characteristics

The study included 300 primary care patients (mean age 49.7 ± 17.6 years), with 61.3% females (Table 1). The average weight was 74 ± 17 kg, and the mean height was 168 ± 10 cm. In terms of BMI, 43.7% were overweight and 18.3% were classified as obese, while only 2.3% were underweight.

### Sleep Patterns and Insomnia Severity

Participants reported a median bedtime of 23:00 and a wake-up time of 07:00, with an actual sleep duration of 6.4 ±1.4 hours per night, notably lower than the perceived optimal sleep duration of 8.1±0.8 hours. Sleep onset latency varied, with 42.3% requiring more than 30 minutes to fall asleep.

The mean Insomnia Severity Index (ISI) score was 11.1±6.1, with 33% reporting no significant insomnia, 38% mild insomnia, 24% moderate insomnia, and 5% severe insomnia. Clinical insomnia was defined as ISI ≥ 15, as recommended in prior literature.

**Table 1** Demographic, Clinical, and Sleep Characteristics of the Primary Care Study Sample

| Variable | Mean ± SD (or %) |
|---|---|
| ***Demographic Data*** | |
| *Age* (years) | 49.7±17.6 |
| *Sex* | |
| Male | 38.7% (116/300) |
| Female | 61.3% (184/300) |
| *Weight* (kg) | 74±17 |
| *Height* (cm) | 168±10 |
| *Body Mass Index* | |
| Underweight (< 18.5) | 2.3% (7/300) |
| Normal Weight (18.5–24.9) | 37.7% (113/300) |
| Overweight (25.0–29.9) | 43.7% (131/300) |
| Obese class I (30.0–34.9) | 10.0% (30/300) |
| Obese class II (35.0–39.9) | 3.7% (11/300) |
| Obese class III (≥ 40.0) | 2.3% (7/300) |
| ***Sleep Patterns*** | |
| *Bedtime* (median time) | 23.00 |
| *Sleep onset latency* | |
| <15 min | 27.0% (81/300) |
| 16–30 min | 35.0% (105/300) |
| 31–60 min | 22.7% (68/300) |
| >60 min) | 15.3% (46/300) |
| *Wake-up time* (median time) | 7.00 |
| *Actual sleep hours per night* | 6.4±1.4 |
| *Perceived optimal sleep hours* | 8.1±0.8 |

(*Continued*)

**Table 1** (Continued).

| Variable | Mean ± SD (or %) |
|---|---|
| ***Insomnia*** | |
| *Insomnia Severity Index Score* | 11.1±6.1 |
| No clinically significant insomnia (scores 0–7) | 33% (99/300) |
| Subthreshold (mild) insomnia (scores 8–14) | 38% (114/300) |
| Moderate insomnia (scores 15–21) | 24% (72/300) |
| Severe insomnia (scores 22–28) | 5% (15/300) |
| *Medication use* | |
| Never | 73.7% (221/300) |
| Less than once a week | 5.3% (16/300) |
| Once or twice a week | 5.7% (17/300) |
| Three or more times a week | 15.3% (46/300) |
| ***Perceived Stress*** | |
| *Perceived Stress Scale Score* | 26.2±7.9 |
| Low Stress (scores 0–18) | 18.0% (54/300) |
| Moderate Stress (scores 19–37) | 75% (225/300) |
| High Stress (scores 38–56) | 7.0% (21/300) |
| ***Lifestyle Habits*** | |
| *Coffee* (Yes) | 73.3% (220/300) |
| *Tea* (Yes) | 19% (57/300) |
| *Cola drinks* (Yes) | 21.3% (64/300) |
| *Energy drinks* (Yes) | 5.3% (16/300) |
| *Cigarettes* (Yes) | 25.3% (76/300) |
| *Alcohol* (Yes) | 46.7% (140/300) |

## Use of Sleep Medication

Despite 29% of the sample experiencing moderate-to-severe insomnia, only 26.3% reported using sleep medication, with most (73.7%) never using it.

## Perceived Stress Levels

The mean Perceived Stress Scale (PSS) score was 26.2±7.9, with 75% experiencing moderate stress and 7% high stress, suggesting a substantial burden of psychological distress in this population.

## Lifestyle Habits and Substance Use

The average daily caffeine intake among participants was 145.38 mg/day (SD = 111.74), with a wide range from 0 to 605 mg/day, reflecting substantial variability. The majority (68.0%) had low caffeine intake, while 30.0% reported moderate intake, and only 2.0% fell into the high intake category. Regarding sources of caffeine, 73.3% of participants consumed coffee, followed by 21.3% who drank cola beverages and 5.3% who consumed energy drinks. Additionally, 25.3% of participants smoked cigarettes, and 46.7% reported alcohol consumption, highlighting common lifestyle behaviors within the sample.

## Correlation Between Perceived Stress and Insomnia Severity

A Pearson correlation analysis was conducted to examine the relationship between perceived stress (PSS Total Score) and insomnia severity (ISI Total Score). Results revealed a moderate positive correlation between the two variables ($r = 0.460$, $p < 0.001$), indicating that higher levels of perceived stress were significantly associated with greater insomnia severity.

The 95% confidence interval (CI) for the correlation coefficient ranged from 0.365 to 0.544, confirming the robustness of this association. Given the statistical significance ($p < 0.001$), the observed relationship is unlikely to be due to chance.

These findings suggest that individuals experiencing higher stress levels are more likely to report more severe insomnia symptoms, reinforcing the potential interplay between psychological distress and sleep disturbances in primary care patients. Further analyses may explore potential mediators or moderators of this relationship, such as sleep medication use or lifestyle factors.

## Interpretation of Logistic Regression Analysis

### Overview of the Model

A binary logistic regression analysis was conducted to examine the association between clinical insomnia (dependent variable: 0 = No/Mild Insomnia, 1 = Clinical Insomnia) and multiple independent variables, including gender, age, body mass index (BMI), hypnotic use, daily alcohol consumption, perceived stress score (PSS Total), daily caffeine intake, and tobacco use. The stepwise backward Wald method was applied, allowing for the iterative removal of non-significant variables to refine the final model.

### Baseline Model (Block 0)

The initial baseline model (Block 0) included only the intercept and classified all cases as belonging to the No/Mild Insomnia category. This yielded an overall classification accuracy of 71.0%, demonstrating that, in the absence of predictors, the model effectively identified the majority class but failed to distinguish cases of clinical insomnia.

An initial test for predictor significance (Step 0) indicated that hypnotic use ($p < 0.001$), alcohol consumption ($p = 0.014$), and perceived stress ($p < 0.001$) were significantly associated with clinical insomnia. Other variables, including gender, age, BMI, caffeine intake, and tobacco use, did not show significant associations at this stage.

### Final Model (Block 1 – Stepwise Selection)

Through stepwise elimination, age, tobacco use, caffeine intake, and gender were sequentially removed, as they did not significantly contribute to the model ($p > 0.10$). The final model retained BMI, hypnotic use, alcohol consumption, and perceived stress score as significant predictors of clinical insomnia.

### Model Fit and Performance

The final model demonstrated a significant improvement over the null model, as indicated by the Omnibus Test of Model Coefficients ($\chi^2 = 78.272$, $df = 4$, $p < 0.001$), confirming that the included predictors meaningfully contributed to explaining the outcome. The Nagelkerke $R^2$ value of 0.328 suggested that the model accounted for approximately 33% of the variance in clinical insomnia.

In terms of classification accuracy, the final model improved to 79.0%, correctly identifying 93.4% of No/Mild Insomnia cases. However, it showed limited sensitivity for detecting Clinical Insomnia cases, with an accuracy of only 43.7%. This indicates that while the model effectively classifies non-clinical cases, its ability to detect insomnia remains constrained.

### Key Predictors and Their Effects

- Hypnotic use emerged as the strongest predictor of clinical insomnia (OR = 3.22, $p < 0.001$), indicating that individuals using hypnotics were more than three times as likely to experience clinical insomnia compared to non-users.
- Perceived stress (PSS Total Score) was also a significant predictor (OR = 1.134, $p < 0.001$), with each one-unit increase in stress score associated with a 13.4% increase in the likelihood of clinical insomnia.
- Alcohol consumption showed a negative association with clinical insomnia (OR = 0.551, $p = 0.048$), suggesting that individuals consuming alcohol were approximately 45% less likely to report clinical insomnia. However, given the small effect size, this finding warrants further investigation.
- BMI exhibited a marginal effect ($p = 0.061$), indicating a potential association with clinical insomnia, though it did not reach statistical significance.

**Table 2** Key Predictors of Clinical Insomnia in Primary Care Patients: Results From the Final Logistic Regression Model

| Variable | B | Wald | p-value | Exp(B) (OR) | 95% CI for OR |
|---|---|---|---|---|---|
| BMI | 0.055 | 3.518 | 0.061 | 1.056 | 0.998–1.119 |
| Hypnotic Use (Yes) | 1.169 | 13.928 | <0.001 | 3.220 | 1.742–5.951 |
| Alcohol Use (Yes) | −0.596 | 3.905 | 0.048 | 0.551 | 0.305–0.995 |
| Perceived Stress | 0.126 | 31.708 | <0.001 | 1.134 | 1.086–1.185 |
| Constant | −5.941 | 31.100 | <0.001 | 0.003 | — |

**Abbreviations**: B, Regression coefficient; OR, Odds Ratio; CI, Confidence Interval.

The final logistic regression model included BMI, hypnotic use, alcohol consumption, and perceived stress as significant predictors of clinical insomnia (Table 2). A forest plot visualizing the adjusted odds ratios and 95% confidence intervals from the final logistic regression model is shown in Figure 1.

### Variables Removed from the Model

Gender, age, caffeine intake, and tobacco use were excluded in successive steps due to their lack of statistical significance ($p > 0.10$). This suggests that these factors did not independently contribute to predicting clinical insomnia within this sample.

## Discussion

### Summary of Key Findings

The results of the logistic regression analysis provide valuable insights into the factors associated with clinical insomnia. Among the examined predictors, hypnotic use, perceived stress, and alcohol consumption emerged as statistically significant, while gender, age, caffeine intake, and tobacco use were not independently associated with clinical insomnia. The model explained approximately 33% of the variance in clinical insomnia (Nagelkerke $R^2 = 0.328$) and had a high

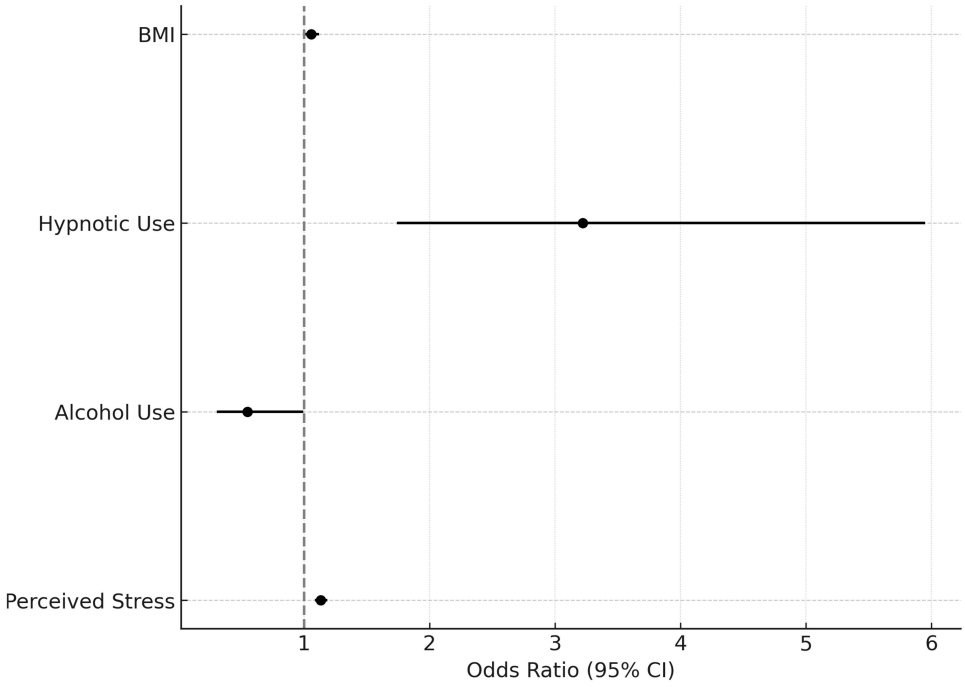


**Figure 1** Forest plot showing adjusted odds ratios (ORs) and 95% confidence intervals for significant predictors of clinical insomnia in the final logistic regression model.


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specificity (93.4%) but relatively low sensitivity (43.7%), meaning that while it effectively identified individuals without clinical insomnia, it was less reliable in detecting those suffering from it.

## Interpretation of Significant Predictors

### Hypnotic Use and Clinical Insomnia

The strongest predictor of clinical insomnia was hypnotic use (OR = 3.22, $p < 0.001$), indicating that individuals taking hypnotic medications were more than three times more likely to have clinical insomnia compared to non-users. This finding aligns with existing literature suggesting a complex relationship between hypnotic use and sleep disorders. While hypnotics are prescribed to manage sleep disturbances, long-term dependence, tolerance, and withdrawal effects may contribute to worsening sleep quality over time.[23] Additionally, rebound insomnia upon discontinuation and the risk of developing maladaptive coping mechanisms for sleep difficulties may explain this association.

Furthermore, it is important to consider whether hypnotics are being used as a consequence of severe insomnia rather than being a direct cause. That is, patients who already experience chronic sleep problems may be more likely to seek pharmacological treatment, creating a reverse causality issue. Future research should explore longitudinal data to better understand whether hypnotic use precedes or follows insomnia onset.

### Perceived Stress as a Major Contributor to Insomnia

The Perceived Stress Scale (PSS) score was another strong predictor (OR = 1.134, $p < 0.001$), showing that for each one-unit increase in stress, the odds of clinical insomnia increased by 13.4%. This result reinforces well-established research linking psychological stress to sleep disturbances.[24] Stress is known to activate the hypothalamic-pituitary-adrenal (HPA) axis, leading to increased cortisol levels, hyperarousal, and difficulties in sleep initiation and maintenance.[25]

Chronic stress has also been associated with dysregulated sleep architecture, particularly a reduction in slow-wave sleep (SWS) and rapid eye movement (REM) sleep, both of which are crucial for restorative rest.[26] Cognitive factors, such as rumination and heightened worry, may also contribute to stress-induced insomnia by preventing sleep onset and increasing nighttime awakenings. These findings highlight the importance of stress management interventions, such as cognitive-behavioral therapy for insomnia (CBT-I), mindfulness-based techniques, and relaxation therapies, as key non-pharmacological strategies for improving sleep.

### Alcohol Consumption and Insomnia

The observed negative association between alcohol consumption and clinical insomnia (OR = 0.551, $p = 0.048$) is somewhat counterintuitive, as alcohol is generally recognized as a sleep disruptor rather than a protective factor. This finding warrants careful consideration, as it may reflect unmeasured confounding factors or specific characteristics of the study population rather than a true protective effect of alcohol on sleep.

One possible explanation is that alcohol consumption serves as a proxy for other lifestyle factors associated with lower insomnia risk, such as social engagement, relaxation routines, or physical activity. Individuals who consume moderate amounts of alcohol may also have lower stress levels or engage in social activities that indirectly contribute to better sleep patterns.[27] However, this hypothesis requires further investigation, as it remains unclear whether the association is causal or merely correlational.

Potential methodological biases should also be considered. Underreporting of alcohol intake is a well-known limitation in self-reported data, particularly in health-related studies where participants may minimize their consumption due to social desirability bias.[28,29] Additionally, selection bias could influence the results if individuals with severe insomnia avoid alcohol due to its known disruptive effects on sleep, leading to an apparent but misleading inverse relationship.[30,31]

While some studies suggest alcohol can promote sleep onset, its rebound effects often disrupt sleep architecture.[30] Further research is needed to clarify whether these effects vary by consumption patterns. Future research should explore the dose-response relationship between alcohol and sleep, considering factors such as frequency, quantity, and timing of alcohol intake. Longitudinal studies with objective sleep measures (eg, actigraphy or polysomnography) could help

clarify whether alcohol has differential effects on sleep depending on individual consumption patterns and underlying sleep disorders.

## Non-Significant Predictors and Their Implications

### Lack of Association Between Gender and Insomnia

Despite previous studies suggesting a higher prevalence of insomnia in females,[32] gender was not a significant predictor in this analysis ($p = 0.387$ after stepwise elimination). One explanation could be that gender differences in insomnia may be mediated by other factors, such as hormonal fluctuations, caregiving roles, and psychological distress, which were not explicitly controlled in this model. Additionally, the sample characteristics and potential differences in self-reported vs clinically diagnosed insomnia may have influenced this result.

### Caffeine Intake and Insomnia

Caffeine is a well-known stimulant that disrupts sleep by delaying sleep onset, reducing total sleep duration, and impairing sleep quality.[33] It acts by blocking adenosine receptors, increasing wakefulness, and interfering with sleep regulation.[34] While stress was a significant predictor of insomnia in this study, caffeine intake was not significantly associated with clinical insomnia ($p = 0.621$). This finding may be explained by several factors.

First, individual differences in caffeine metabolism may play a role. Genetic variations in CYP1A2 enzyme activity influence caffeine clearance, with fast metabolizers experiencing reduced physiological effects compared to slow metabolizers.[35,36] Second, the timing of caffeine consumption is crucial—if most participants consume caffeine in the morning rather than the evening, its effects on sleep may be minimal. Third, habitual caffeine consumers may develop tolerance, leading to physiological adaptations that reduce its disruptive impact on sleep.[37] These factors may explain why some individuals experience marked sleep disturbances from caffeine, while others show little to no impairment.[38,39]

Chronic caffeine use, particularly in individuals with high stress levels, may create a cycle of poor sleep, increased fatigue, and greater caffeine reliance, exacerbating insomnia.[40] Given the complex interplay between caffeine, stress, and sleep, future research should explore caffeine as a potential moderator in the stress-insomnia relationship. Additionally, primary care interventions should incorporate caffeine screening and reduction strategies, particularly for high-risk individuals.

### Tobacco Use and Insomnia

Tobacco use did not show a significant association with clinical insomnia in this study ($p = 0.719$), which contrasts with prior research indicating that nicotine acts as a central nervous system stimulant, reducing sleep efficiency, increasing nighttime awakenings, and shortening total sleep duration.[41] One possible explanation for this discrepancy is that the impact of nicotine on sleep may be highly dependent on individual smoking patterns and physiological adaptations.

Several factors may influence the relationship between tobacco use and insomnia. First, nicotine dependence and withdrawal cycles could moderate sleep outcomes. While acute nicotine intake has stimulating effects, habitual smokers may experience nighttime withdrawal symptoms, leading to sleep fragmentation and early morning awakenings.[42] Conversely, some smokers may use cigarettes as a coping mechanism for stress and anxiety, which might contribute to subjective improvements in relaxation before bedtime, despite nicotine's physiological arousal effects.

Another consideration is the timing and frequency of tobacco use. Studies suggest that smoking closer to bedtime is more disruptive to sleep than smoking earlier in the day, likely due to nicotine's half-life of approximately 1–2 hours, which can prolong its stimulating effects into the night.[43] Additionally, polypharmacy and substance use interactions—such as concurrent caffeine or alcohol intake—may confound the effects of nicotine on sleep.

Although our findings did not show a significant association, future studies should account for nicotine dependence levels, withdrawal symptoms, and smoking timing to better understand how tobacco use influences sleep disturbances. Clinically, primary care interventions for insomnia may benefit from assessing smoking habits in greater detail, particularly among individuals with coexisting stress, anxiety, or other risk factors for poor sleep.

## Limitations

While this study provides valuable insights, several limitations should be considered. The *cross-sectional design* prevents establishing causal relationships between stress, hypnotic use, and insomnia, making it unclear whether hypnotics exacerbate or result from severe insomnia, or whether the stress-insomnia relationship is bidirectional. The reliance on *self-reported measures introduces recall bias and social desirability effects*, particularly for lifestyle factors such as caffeine, alcohol, and tobacco use, and insomnia classification was based on subjective symptoms rather than objective sleep measures (eg, polysomnography), which may result in misclassification errors. Additionally, several *unmeasured confounders* were not assessed, including psychiatric comorbidities (eg, anxiety, depression, PTSD), other medications, sleep hygiene behaviors, and genetic factors, which could further explain insomnia risk. The *study's generalizability is limited*, as the sample size (n=300) may not fully represent broader populations, with demographic and socioeconomic factors left unexplored. The logistic regression model explained 33% of the variance in clinical insomnia, suggesting moderate explanatory power but also the possibility of *overfitting*, limiting predictive accuracy in other datasets. Lastly, *selection bias* may have occurred if participants were self-selected due to sleep concerns, potentially inflating insomnia-related risk factors. Future longitudinal studies incorporating objective sleep measures are essential to confirm these associations and guide targeted interventions in primary care.

### Clinical and Research Implications

- Targeted interventions: Stress management should be a primary target in insomnia treatment, alongside cognitive-behavioral therapy and non-pharmacological approaches.
- Reevaluation of hypnotic prescriptions: Given the strong association between hypnotics and insomnia, clinicians should consider alternative therapies and limit long-term hypnotic use to prevent dependence.
- Future research: Studies exploring genetic factors, neurobiological markers, and sleep architecture could provide deeper insights into individual variability in insomnia risk.

## Public Health and Policy Implications

Given the high prevalence of stress and insomnia symptoms in primary care patients, these findings underscore the importance of implementing routine stress screening during general medical visits. Integrating non-pharmacological interventions—such as cognitive-behavioral therapy for insomnia (CBT-I), mindfulness-based stress reduction, and psychoeducation—into primary care workflows could significantly enhance sleep outcomes and reduce reliance on hypnotic medications. Policymakers should consider these data when developing mental health support strategies tailored for primary care environments.

## Conclusion

This study highlights the complex interplay between hypnotic use, stress, alcohol consumption, and insomnia. While stress emerged as a key modifiable risk factor, the association between alcohol and insomnia warrants further investigation. The findings support the need for comprehensive, multidimensional treatment approaches that address both psychological and behavioral contributors to sleep disturbances.

## Disclosure

The authors report no conflicts of interest in this work.

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