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Association Between Caffeine Intake and Depressive Symptoms: A Cross-sectional Analysis Using the NHANES 2017-2018 Database

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Abstract

Background: *Depression is a leading global cause of disability, and identifying modifiable lifestyle factors such as caffeine intake may help mitigate its burden. Although caffeine consumption has been proposed as protective against depressive symptoms, existing evidence remains inconsistent and often insufficiently adjusted for key confounders.*

Objective: *To examine the association between caffeine intake and depressive symptoms among U.S. adults using data from the 2017–2018 National Health and Nutrition Examination Survey (NHANES).*

Methods: *This cross-sectional study was conducted and reported according to STROBE guidelines. Adult NHANES participants were included. Daily caffeine intake (mg/day), assessed via 24-hour dietary recall, was categorized as low, moderate, or high. Depressive symptoms were measured using the Patient Health Questionnaire-9 (PHQ-9) and classified into five clinically meaningful severity levels. Weighted ordered logistic regression models were used to evaluate associations between caffeine intake and depressive symptom severity, adjusting for demographic, socioeconomic, lifestyle, and clinical covariates, including age, sex, education, body mass index (BMI), marital status, family income, ethnicity, smoking status, sedentary behavior, thyroid disease, diabetes, and cardiovascular disease.*

Results: *The analytic sample included 4,503 adults (mean age 47.3 years; 51.8% female). Caffeine intake was low in 47%, moderate in 27%, and high in 26% of participants. In weighted univariable analyses, both moderate (OR 0.73; 95% CI 0.60–0.90; p=0.006) and high caffeine intake (OR 0.65; 95% CI 0.51–0.82; p=0.002) were associated with lower odds of more severe depressive symptoms compared with low intake. After multivariable adjustment, only high caffeine intake remained independently associated with lower depressive symptom severity (OR 0.62; 95% CI 0.48–0.80; p=0.001). In adjusted models, female sex, obesity, unmarried status, current smoking, thyroid disease, and cardiovascular disease were independently associated with higher odds of depressive symptoms, whereas higher educational attainment and higher family income were independently associated with lower odds of depressive symptoms.*

Conclusions: *High caffeine intake was independently associated with lower depressive symptom severity in U.S. adults, even after accounting for a broad range of confounders. However, several demographic, socioeconomic, and health-related factors exerted stronger and opposing influences on depressive symptoms. These findings suggest that while caffeine consumption may play a modest protective role, addressing broader social and health determinants is likely more impactful for depression prevention and management. Longitudinal studies are needed to clarify causality and temporal relationships.*

Introduction

Depression is a global public health concern, affecting more than 300 million people worldwide and contributing substantially to disability, reduced quality of life, and increased healthcare costs (2021 Global Burden of Disease (GBD) [Online Database]. Seattle: Institute for Health Metrics and Evaluation; 2024, n.d.). Identifying modifiable lifestyle factors that influence the risk or severity of depression is therefore essential for prevention and treatment efforts.

Caffeine, the most widely consumed psychoactive substance globally, has attracted attention because of its potential effects on mood, alertness, and cognition (Temple et al., 2017). It is naturally present in coffee, tea, and chocolate and is commonly added to sodas, energy drinks, and dietary supplements. The average caffeine intake for adults is approximately 2.4 mg/kg/day, and daily doses up to 400 mg are considered safe. Typical beverages contain between 70 and 100 mg per serving. In contrast, toxic effects such as tachycardia, arrhythmia, seizures, and altered mental status are associated with acute intakes around 1.2 g, with severe intoxication reported at blood levels of 80 mg/L. The average blood level in fatal caffeine toxicity cases is 180 mg/L, and the estimated potentially fatal dose ranges from 10 to 14 g (Cappelletti et al., 2018; Neves & Caldas, 2017; Poole et al., 2017). Given its widespread and psychostimulant properties, understanding how caffeine intake may influence depressive symptoms has important implications for mental health prevention and promotion.

Previous research suggests that moderate caffeine intake may be linked to a lower risk of depressive symptoms, potentially improving mood, concentration, and motivation while reducing fatigue and apathy (Meamar et al., 2024). Caffeine, naturally present in beverages such as coffee and tea, is known for its stimulant effects on the central nervous system through adenosine receptor antagonism, influencing alertness and mood regulation (Temple et al., 2017). Understanding how caffeine intake can influence depressive symptoms is crucial for developing effective prevention and treatment strategies worldwide. The relationship between caffeine and mental health is therefore essential for identifying behavioral factors that may contribute to improved well-being and for generating evidence-based public health interventions.

Despite the biological plausibility of a protective role, evidence on caffeine's relationship with depression remains inconsistent. Some population-based studies have reported an inverse association, such as analyses from NHANES 2017–2018 (Bao et al., 2022) and the NHANES 2005-2006, which showed a protective effect of caffeine against depressive symptoms (Iranpour & Sabour, 2019). Conversely, other studies have shown no significant relationship between caffeine consumption and depression in male and female students (El Hashem et al., 2025; Makki et al., 2023), while excessive caffeine intake has been associated with greater depressive symptom severity (Aleem et al., 2024). These discrepancies may reflect differences in study design, population characteristics, and inadequate control for confounding factors such as socioeconomic status, obesity, smoking, and comorbid diseases. Therefore, a more comprehensive, population-based analysis is warranted to clarify the relationship between caffeine intake and depressive symptoms.

Several variables are known to influence both caffeine consumption and depression. For instance, depression prevalence and caffeine metabolism vary with age and gender (Bao et al., 2022; Dotson et al., 2020; Rehm et al., 2020; Riera-Sampol et al., 2022; Salk et al., 2017). Socioeconomic factors such as income and education also shape dietary habits and mental health outcomes (Iranpour & Sabour, 2019; Jespersen et al., 2025), while marital status and body mass index (BMI) have been linked to differences in depression risk (Badillo et al., 2022; Lone et al., 2023; Zhai et al., 2024). Smoking, another lifestyle factor strongly associated with caffeine use, is also a well-established risk factor for depression (Fluharty et al., 2017; Wu et al., 2023). Other comorbidities have impact on depressive symptoms and caffeine intake, such as thyroid disease (Kim et al., 2015; Zheng et al., 2023), diabetes (Rotella & Mannucci, 2013), and major cardiovascular events, including coronary heart disease, myocardial infarction, heart failure, and stroke (Wu & Kling, 2016), with caffeine intake associated with a protective effect against several of these conditions (Turnbull et al., 2017). Racial and ethnic disparities in depression have been consistently documented, with prevalence and severity varying across groups, influencing caffeine consumption as well (Lim et al., 2021; Vyas et al., 2020). Sedentary activity has been studied, showing an independent association with both greater depressive symptomatology and higher caffeine consumption (Guest et al., 2021; Pearce et al., 2022). Sleep duration and quality have complex, bidirectional and have been described as mediators

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or colliders in their relationship between caffeine and depression (Clark & Landolt, 2017; Drake et al., 2013; Van Der Linden et al., 2023). Together, these variables represent key potential confounders in the relationship between caffeine intake and depressive symptoms, as supported by previous literature and a directed acyclic graph (DAG) developed a priori.

The objective of this study was to investigate the association between caffeine intake and depressive symptoms in a cross-sectional analysis using data from the 2017–2018 National Health and Nutrition Examination Survey (NHANES). We also sought to evaluate potential effect modification by demographic and health factors. We hypothesized that higher caffeine intake would be associated with lower odds of being in a higher category of depressive symptom severity, as assessed by the PHQ-9 scale, among adults living in the United States. These included demographic, socioeconomic, behavioral, and health-related factors such as age, gender, body mass index (BMI), marital status, educational level, smoking status, diabetes, thyroid disease, cardiovascular diseases, monthly family income, and sedentary activity, which were included as confounders in the statistical analysis.

Materials and Methods

Study design

This study is based on data from the 2017-2018 wave of the National Health and Nutrition Examination Survey (NHANES), a complex, multistage probability sampling design survey, conducted by the National Center for Health Statistics (NCHS). NHANES follows a cross-sectional structure across repeated waves. Data were collected under standardized protocols, including household interviews, physical examinations, laboratory testing, and dietary assessments conducted in Mobile Examination Centers (MECs) (CDC/NCHS, 2017–2018). This study was designed and reported in accordance with the STROBE (Strengthening the Reporting of Observational Studies in Epidemiology) guidelines for cross-sectional studies.

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Study population

The analytic sample included adults aged 18 years and older. Eligibility required complete data for the primary exposure (caffeine intake) and the main outcome (depressive symptoms), assessed using the Patient Health Questionnaire-9 (PHQ-9). Patients were excluded if they were younger than 18 years old, had missing data on either the exposure or the outcomes. Caffeine intake of 400 mg per day was considered the maximum safe intake. The sample size was not determined a priori, as data were obtained from the existing NHANES 2017-2018 cycle. However, given the large and nationally representative nature of the sampling, this study provides enough statistical power to detect moderate associations between our exposure and our outcome.

Variables

Exposure: Caffeine intake

Dietary caffeine intake was derived from the dietary questionnaire of the 24-hour caffeine intake in the previous day, which was prioritized over other variables due to their higher response rate and quality (CDC/NCHS, 2017–2018). Intake was expressed in milligrams per day (mg/day) and categorized into three levels according to international thresholds and clinical interpretability: low intake (0–100 mg/day), moderate intake (101–200 mg/day), and high intake (201–400 mg/day). These cutoffs are supported by guidance from the U.S. Food and Drug Administration and the European Food Safety Authority, which consider daily intakes up to 400 mg safe and correspond to typical beverage consumption (about 100 mg per cup of brewed coffee).

Outcome: Depressive symptoms

The dependent variable was the depression symptoms, using a validated self-reported instrument, the Patient Health Questionnaire (PHQ-9) extracted from the NHANES mental health questionnaire, with 9 items, each scored from 0 (“not at all”) to 3 (“nearly every day”), yielding a total score ranging from 0 to 27. For this analysis, PHQ-9 was categorized into five standard levels of depressive symptoms severity: minimal (0–4), mild (5–9), moderate (10–14), moderately severe (15–19), and severe (20–27). These categories

are widely used in clinical and epidemiological research, facilitating the interpretability and comparability of results across studies (Kroenke et al., 2001).

Covariates

Potential confounders were identified a priori using a directed acyclic graph (DAG) and supporting evidence from previous literature. The final adjustment set included age, gender, marital status, educational level, body mass index (BMI), family income-to-poverty index, ethnicity, smoking status, daily minutes of sedentary activity, thyroid disease, diabetes, and cardiovascular disease. Sleep duration and quality were excluded from the primary adjustment model because they likely act as mediators or colliders in the relationship between caffeine and depression. All variables were obtained from the 2017–2018 NHANES public-use datasets using the demographic, dietary, examination, and questionnaire data. Diagnostic conditions (thyroid disease, diabetes, and cardiovascular events) were based on physician-confirmed self-reports.

Statistical Analysis

All analyses incorporated the NHANES complex, multistage probability sampling design by specifying the primary sampling units (SDMVPSU), strata (SDMVSTRA), and the day-1 dietary weights (WTDRD1) as recommended by the National Center for Health Statistics.

Continuous variables were summarized as weighted means and 95% confidence intervals (CI), and categorical variables as weighted proportions with 95% CI. All estimates accounted for the complex NHANES survey design, incorporating sampling weights, strata, and primary sampling units (PSUs) to ensure nationally representative results. To evaluate the association between caffeine intake and depressive symptoms, we used a survey-weighted ordered logistic regression model. Caffeine intake was analyzed in three categories: low intake (0–100 mg/day), moderate intake (101–200 mg/day), and high intake (201–400 mg/day), with the low-intake group as the reference. Both unadjusted and adjusted models were estimated. The adjusted model included age, gender, BMI, marital status, educational level, family income-to-poverty index, smoking, sedentary activity, thyroid disease, diabetes, cardiovascular disease, and ethnicity, identified a priori by the DAG framework.

Effect modification was explored by including interaction terms between caffeine intake and age, gender, BMI, marital status, educational level, family income-to-poverty index, smoking, sedentary activity, thyroid disease, diabetes, cardiovascular disease, and ethnicity. Interactions were assessed using the Variance Inflation Factor (VIF) test. Sensitivity analyses modeled caffeine intake as a continuous variable (between 0 and 400 mg/day) to assess the stability of the results.

Missing data were evaluated across all study variables. The proportion of missing values ranged from 0% to 20.5%. The main outcome (PHQ-9 categories) had 13.0% missing data (763 of 5856 participants), while the primary exposure variable (Caffeine intake) had 20.0% missing data (1169 of 2856). These proportions are consistent with the planned inclusion and exclusion criteria. Missing data among covariates included BMI (422), Thyroid disease (302), heart disease (363), diabetes (4), education level (300), family income (1,203, accounting for 20.5%), and sedentary activity (45). The largest proportion of missingness occurred in the family income variable, likely reflecting the sensitive nature of this question within NHANES.

All tests were two-sided, with statistical significance set at $p < 0.05$. Analyses were conducted using Stata/BE 19.5 (StataCorp LLC, College Station, TX, USA).

Results

Study population characteristics

The analysis was conducted using data from the NHANES 2017-2018, a complex multistage cross-sectional study with a large population-based survey. All estimates were derived using sampling weights, strata, and primary sampling units (PSUs), ensuring adequate representation. All values obtained are presented as weighted means and percentages with 95% CI. The majority of adults (47%) reported low caffeine consumption (0–100 mg/day), followed by 27% consuming moderate caffeine (101–200 mg/day) and 26% of participants having higher caffeine intake (201-400 mg/day). Mean age increased with caffeine intake. Gender distribution was balanced across caffeine intake groups. Regarding gender, the proportion of females was 55% in the lower intake group, 47% with moderate intake, and 47% with high caffeine consumption. Table 1 summarizes the weighted demographic and baseline characteristics of participants, stratified by levels of caffeine intake.

Univariate analysis

Univariable analysis was performed for the weighted data. In the unadjusted ordered logistic regression model, moderate caffeine intake was

Caffeine intake			
	Low (0-100 mg/day)	**Moderate** (101-200 mg/day)	**High** (201-400 mg/day)
Participants	47% (44-49%)	27% (26-29%)	26% (23-26%)
Age in years	43.7 (42.0-45.4)	47.9 (45.5-50.3)	51.6 (49.5-53.7)
Gender			
Male	45% (43-48%)	46% (41-52%)	53% (46-59%)
Female	55% (52-57%)	54% (48-59%)	47% (41-54%)
Educational level			
< 9th grade	5% (4-7%)	3% (2- 4%)	2% (1-3%)
9-11th grade	9% (7-10%)	6% (4- 8%)	5% (4- 8%)
High School	30% (26-34%)	27% (22-33%)	26% (22-31%)
Some college	30% (27-32%)	33% (27-39%)	29% (23-36%)
College Graduate or above	27% (22-32%)	32% (25-38%)	37% (28-46%)
BMI (kg/m^2)			
Obese	44% (39-49%)	40% (33-48%)	40% (35-45%)
Non obese	56% (51-61%)	60% (52-67%)	60% (55-65%)
Marital status			
Married*	47% (44-50%)	36% (32-42%)	35% (30-40%)
Unmarried**	53% (50-56%)	64% (59-68%)	65% (60-70%)
Family Monthly Income			
Low	21% (17-26%)	14% (11-17%)	11% (7-16%)
High	79% (74-83%)	86% (83-89%)	89% (84-93%)
Ethnicity			
Mexican American	13% (9-20%)	8% (6-12%)	5% (3-8%)
Other Hispanic	8% (6-11%)	8% (6-11%)	5% (3-7%)
Non-Hispanic White	46% (39-53%)	67% (61-72%)	76% (70-82%)
Non-Hispanic Black	19% (14-25%)	8% (6-11%)	4% (3-7%)
Non-Hispanic Asian	9% (6-12%)	5% (4-8%)	3% (2-4%)
Other Race Including Multi-Racial	5% (3-6%)	3% (2-5%)	7% (4-10%)
Smoking status			
Never smoked	69% (66-73%)	57% (51-63%)	52% (46-59%)
Former smoker	17% (15-20%)	28% (22-34%)	32% (27-37%)
Current smoker	13% (11-16%)	15% (12-20%)	16% (12-21%)
Sedentary activity (min/day)	336.5 (320.8-352.3)	353.0 (338.0-368.1)	352.0 (328.5-375.6)
Thyroid disease			
Yes	11% (10-13%)	12% (10-16%)	13% (10-17%)
No	89% (87-90%)	88% (84-90%)	87% (83-90%)
Diabetes			
Yes	11% (9-13%)	10% (8-12%)	11% (8-14%)
No	89% (87-91%)	90% (88-92%)	89% (86-92%)
Cardiovascular disease			
Yes	8% (6-10%)	8% (6-11%)	10% (8-14%)
No	92% (90-94%)	92% (89-94%)	90% (86-92%)
PHQ-9			
None/Minimal	72% (70-75%)	78% (74-82%)	80% (76-84%)
Mild	18% (16-21%)	5% (12-17%)	13% (11-16%)
Moderate	6% (5-8%)	5% (3-8%)	4% (2-6%)
Moderately Severe	2% (2-4%)	2% (1-3%)	3% (1-5%)
Severe	0.7% (0.4-1.2%)	0.6% (0.2-1.5%)	0.4% (0.2-0.7%)

* Married or living with a partner

** Widowed, separated, divorced, never married.

Mean and 95% CI for continuous variables; Percentages and 95% CI for categorical variables.

Table 1: *Demographic and baseline characteristics of the study population (NHANES 2017–2018).*

	Unadjusted analysis			Multivariable model		
	OR	(95% CI)	p-value	OR	(95% CI)	p-value
Caffeine intake						
Low (0-100mg/ day)						
Moderate (101–200 mg/day)	0.73	(0.60–0.90)	0.006	0.79	(0.60-1.06)	0.106
High (201–400 mg/day)	0.65	(0.51–0.82)	0.002	0.62	(0.48–0.80)	0.001

*Model adjusted for age, gender, educational level, marital status, smoking status, BMI, ethnicity, family monthly income, sedentary activity (min/day), thyroid problems, diabetes, and Cardiovascular Disease.

Table 2: *Crude and adjusted ordered logistic regression for caffeine intake and PHQ-9 categories.*

associated with 27% lower odds of being in a higher PHQ-9 category compared with those with low caffeine intake (OR 0.73, 95% CI 0.60 to 0.90; p=0.006). Higher caffeine consumption also showed a significant 35% decrease in the odds of being in a higher category of PHQ-9 compared to low caffeine intake (OR 0.65, 95% CI 0.51 to 0.82; p-value =0.002) (Table 2).

Multivariable analysis

After adjusting for age, gender, BMI, marital status, education level, smoking status, ethnicity, family monthly income, sedentary activity (min/day), thyroid disease, diabetes, and cardiovascular disease, we observed that high caffeine consumption was associated with a 38% decrease in the odds of being in a higher PHQ-9 category compared with low caffeine consumption (OR 0.62, 95% CI 0.48-0.80, p-value: 0.001). The statistically significant results observed in the univariable analysis with the moderate caffeine intake were lost after the multivariable analysis (OR 0.79, 95% CI 0.60-1.06, p-value: 0.106; Figure 1). We also observed that with our multivariable analysis, the adjusted R2 increased from 0.0007 in the univariable analysis to 0.09, which indicates that the proportion of variance in the PHQ-9 variable is explained by 9% of the caffeine intake.

Multicollinearity

We tested for multicollinearity to assess if the predictor variables are highly correlated with each other, and to determine the individual effect of each variable. Multicollinearity was assessed using variance inflation factors (VIFs). Elevated VIFs (>5) suggest strong correlations among predictors, increasing variance, and possibly rendering coefficient estimates unstable. The variables used in our model had VIF between 1.08 and 5.03, so it is concluded that no significant collinearity was detected, and the independent variables are not excessively influencing each other. This is positive as it allows for a clearer interpretation of the model's odds ratios.

Proportionality of odds

Due to the nature of our outcome variable with 5 clinically significant categories, we tested for proportionality of the odds to assess if the effect of an independent variable on the odds of being in a higher PHQ-9 category is consistent across all categories of the dependent variable. The results of this analysis gave a Chi2 of 36.5 and a p-value of 0.86. Therefore, there is evidence that the ordinal logistic regression model is fit for our data.

Sensitivity analysis

Caffeine was evaluated as a continuous variable as part of the statistical analysis to assess the robustness of our model. Using caffeine as a continuous variable, the results were similar to the primary analysis, showing that the model is robust.

Discussion

The present study examined the association between caffeine intake and depressive symptoms among U.S. adults using nationally representative data from the 2017–2018 NHANES cycle. In unadjusted analyses, both moderate and high caffeine intake were associated with lower odds of depressive symptoms; however, after comprehensive adjustment for sociodemographic, lifestyle, and clinical confounders, only high caffeine intake remained independently associated with lower depressive symptom severity. In addition, several demographic and health-related variables—including sex, educational attainment, obesity, marital status, income, smoking, and cardiometabolic and thyroid conditions—emerged as independent predictors of depressive symptoms.

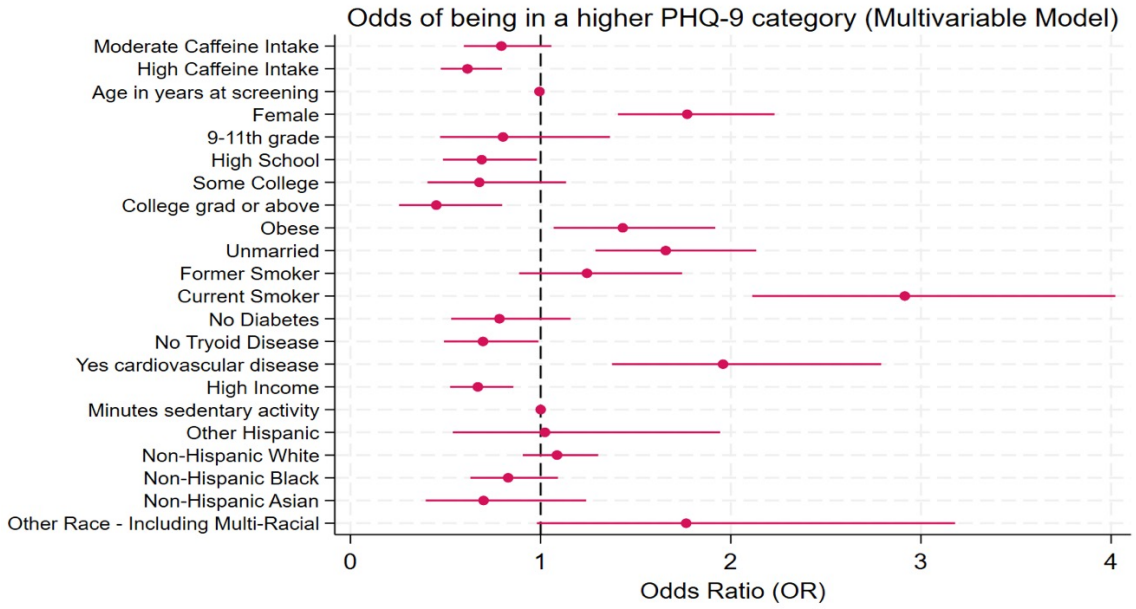

Figure 1: *Complementary results of the multivariable analysis.*

Notably, caffeine intake accounted for only a modest proportion of the variance in PHQ-9 scores (9%) in adjusted models, suggesting that its contribution to depressive symptomatology is relatively small compared with broader social and health determinants.

These findings underscore the complex and multifactorial nature of depression and suggest that the relationship between caffeine intake and depressive symptoms is not straightforward. While caffeine may exert neurobiological effects relevant to mood regulation—such as adenosine receptor antagonism, dopaminergic modulation, and enhancement of alertness and cognitive performance—these effects likely interact with individual characteristics, comorbidities, and lifestyle factors. The attenuation of the association for moderate caffeine intake after adjustment suggests that observed protective effects in univariable analyses may partially reflect confounding by socioeconomic status, health behaviors, or underlying health conditions rather than a direct causal effect.

Our results align with prior NHANES-based analyses reporting inverse associations between caffeine intake and depressive symptoms that weaken or disappear after controlling for confounders (Bao et al., 2022; Iranpour & Sabour, 2019). Such consistency across large population-based studies strengthens the interpretation that caffeine's apparent protective association may be modest and context-dependent. Differences across studies may be attributable to variations in analytic strategies, categorization of caffeine exposure, population characteristics, and adjustment sets. Importantly, subgroup analyses in the literature suggest effect modification by sex, smoking status, and educational level, factors that were also independently associated with depressive symptoms in our study (Li et al., 2023; Riera-Sampol et al., 2022). These interactions may partially explain why the association between caffeine intake and depressive symptoms diminished in fully adjusted models.

Measurement limitations inherent to the NHANES dietary assessment likely also contributed to the observed attenuation. Caffeine intake in NHANES is estimated using the USDA Food and Nutrient Database for Dietary Studies based on a single 24-hour dietary recall. Although this approach is widely accepted, it is susceptible to several sources of error, including day-to-day variability in intake, recall bias, and incomplete capture of usual consumption patterns (Bao et al., 2022; Iranpour & Sabour, 2019). Such nondifferential misclassification generally biases effect estimates toward the null, potentially obscuring true associations. Supporting this interpretation, studies using repeated dietary recalls or food frequency questionnaires—which better approximate habitual caffeine intake—have reported stronger and more consistent associations with depressive outcomes (Jacka et al., 2017; Jespersen et al., 2025; Dotson et al., 2020). Additionally, heterogeneity in how caffeine intake is operationalized across studies (e.g., milligrams per day versus number of cups consumed) further complicates comparisons and contributes to mixed findings in the literature.

The identification of several independent predictors of depressive symptoms in our study reinforces the dominant role of social, behavioral, and medical factors in shaping mental health outcomes. Female

sex, obesity, smoking, unmarried status, and the presence of thyroid or cardiovascular disease were associated with higher odds of depressive symptoms, whereas higher education and income were protective. These findings are consistent with well-established epidemiological patterns and suggest that interventions targeting socioeconomic disparities, cardiometabolic health, and health behaviors may yield substantially greater benefits for depression prevention and management than dietary caffeine modification alone.

From a clinical and public health perspective, our results do not support caffeine intake as a primary or standalone intervention for depression. However, the persistent association observed for high caffeine intake raises the possibility that caffeine may play a modest adjunctive role within a broader lifestyle or behavioral framework. Given caffeine's widespread use, low cost, and general safety at moderate doses, even small effects could be meaningful at the population level. Nevertheless, any recommendation regarding caffeine consumption must be individualized, as excessive intake may exacerbate anxiety, sleep disturbances, or cardiovascular symptoms in susceptible individuals.

To establish causality and clarify mechanisms, future research should move beyond cross-sectional designs. Well-designed longitudinal studies and multicenter randomized controlled trials with standardized caffeine dosing, repeated exposure assessment, and rigorous evaluation of depressive symptoms are needed. Such studies could also explore effect modification by sex, smoking status, metabolic health, and baseline depression severity. If a causal relationship is confirmed, caffeine could potentially be evaluated as a low-cost adjunct to established pharmacological and non-pharmacological treatments for depression. Until then, our findings suggest that caffeine's role in depression is secondary and should be interpreted within the broader context of social, behavioral, and medical determinants of mental health.

Strengths and limitations

Compared to other data analysis studies with the NHANES dataset, our main outcome of depression symptoms was addressed with 5 clinically meaningful categories, derived from the PHQ-9 score, which is in line with how this tool was developed and published. Caffeine intake categorization into low, moderate, and high is also in agreement with guidance from the U.S. Food and Drug Administration and the European Food Safety Authority and corresponds to typical beverage consumption in the population.

Conclusions

This study examined the association between caffeine intake and depressive symptoms in U.S. adults using data from the 2017–2018 NHANES cycle. While initial findings suggested that moderate and high caffeine consumption might be linked to lower odds of depressive symptoms compared to lower caffeine intake, the moderate range of caffeine intake was no longer statistically significant after adjusting for key confounding variables. These results underscore the complexity of the relationship between caffeine and mental health, highlighting the influence of broader sociodemographic and lifestyle factors. Given the limitations of cross-sectional data and self-reported measures, future longitudinal and experimental studies are needed to clarify the causal pathways and potential therapeutic role of caffeine in mental health. Such research could inform more nuanced clinical guidelines and public health strategies aimed at reducing the burden of depression.

Authors Contributions

M.Feddersen, P.Troncoso-Escudero, C.Roa, D.Vicinansa, L.Sandoval, L.Cabreira, P.Bertagnoli, D.Pérez and R.Rosado wrote and edited the manuscript. M.Feddersen, P.Troncoso-Escudero, and C.Roa performed the statistical analysis of this manuscript and prepared the figures. All authors contributed equally to the critical reading of the final manuscript, including figures.

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Conflicts of Interest

The authors declare no conflict of interest.

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