

The Complex Relationship Between Urinary Albumin-to-Creatinine Ratio and Depression

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Abstract: *Background:* One of the main causes of disability in the world is depression. A recent study has found substantial links between metabolic indicators, including serum albumin and uric acid, and depressive symptoms, pointing to the possibility that metabolic-inflammatory pathways are involved in the pathophysiology of depression. The Urinary Albumin-to-Creatinine Ratio (UACR) is an important indicator for evaluating kidney damage and cardiovascular risk; however, its link to depression is not well understood. This research aims to explore the potential association between UACR and depression. *Methods:* Data from NHANES from 2005 to 2018 were used in this study, focusing on individuals 20 years and older with available urine albumin-to-creatinine ratio (UACR) information. The relationship between UACR levels and depression was assessed using weighted multivariate logistic regression, with adjustments for important factors. To determine the strength of this association, subgroup analyses were performed considering various demographic and health-related variables. *Results:* According to the unadjusted analysis, there was a significant relationship between increased UACR and a greater risk of depression. Each unit rise in UACR, after applying a natural logarithmic transformation, was linked to an 18% higher risk of depression (OR: 1.18, 95% CI: 1.14–1.23). Furthermore, individuals in the highest tertile of UACR exhibited a 112% elevated risk of depression relative to those in the lowest UACR tertile (OR: 2.12, 95% CI: 1.57–2.86). This association between UACR and the risk of depression was consistently observed across diverse demographic subgroups. *Conclusions:* This study's preliminary unadjusted analyses suggested a correlation between elevated UACR, higher proteinuria grades, and an increased risk of depression. However, after thorough adjustment for covariates, these associations were no longer statistically significant. These results imply that the connection between UACR, proteinuria levels, and depression might be intricate and possibly affected by different socioeconomic and lifestyle factors. Further investigation is essential to uncover the basic mechanisms and clinical impacts of this association.

Keywords: American adults; Cross-sectional study; Depression; National health and nutrition examination survey; Urinary albumin-to-creatinine ratio

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1. Introduction

Depression is a prevalent mental disorder, and its global incidence has been on a consistent rise over recent decades ^[1]. Projections indicate that depression will become the foremost cause of disability by 2030 ^[2]. The pathogenesis of depression is multifactorial, encompassing genetic, psychological, biological, and environmental risk factors. Recent research indicates a potential association between depression and impaired renal function, but the underlying mechanisms remain unclear ^[3]. Therefore, it is crucial to investigate the correlation between depression and renal function.

Increased urine albumin levels, known as proteinuria, are viewed as a precursor to early kidney problems ^[4]. In the beginning stages of kidney damage, urinary albumin reflects reduced glomerular filtration and could influence tubular function through multiple mechanisms ^[5]. Abnormalities in UACR have been linked to the onset of cardiovascular disease, hypertension, obesity and other diseases ^[6–10]. For early detection of CKD, UACR is a significant indicator. Depression is a prevalent condition among CKD patients, with a reported prevalence of 39.3% among individuals with end-stage renal disease ^[11]. An investigation within a Chinese demographic indicated a significant positive correlation between UACR and depressive symptoms ^[12]. However, research investigating this link within the American population is scarce. To fill this gap, we conducted a large-scale cross-sectional study utilizing data from the National Health and Nutrition Examination Survey (NHANES) spanning the years 2005 to 2018. The goal of this study was to investigate and confirm the relationship between UACR and depression in adults.

2. Methods

2.1. Study population

NHANES is a perpetual program focused on evaluating the health and nutrition status of the American populace. Roughly 5,000 U.S. residents participate each year, delivering detailed data on their demographics, socioeconomic status, dietary habits, and health conditions. Gathering data includes in-person interviews and comprehensive physical evaluations, including physiological measurements and lab tests. The Institutional Review Board at the National Center for Health Statistics approved the study ethically and provided informed consent. Data spanning from 2005 to 2018 from the NHANES database were utilized in this observational research. Initially, 70,190 potential participants were evaluated according to predefined inclusion and exclusion criteria. Exclusion criteria included incomplete or missing PHQ-9 data (33,743), incomplete UACR data (527), or missing data on important covariates (6,694). After these exclusion criteria were implemented, 29,226 individuals were included in the final dataset for analysis. For further details, please refer to **Figure 1**.

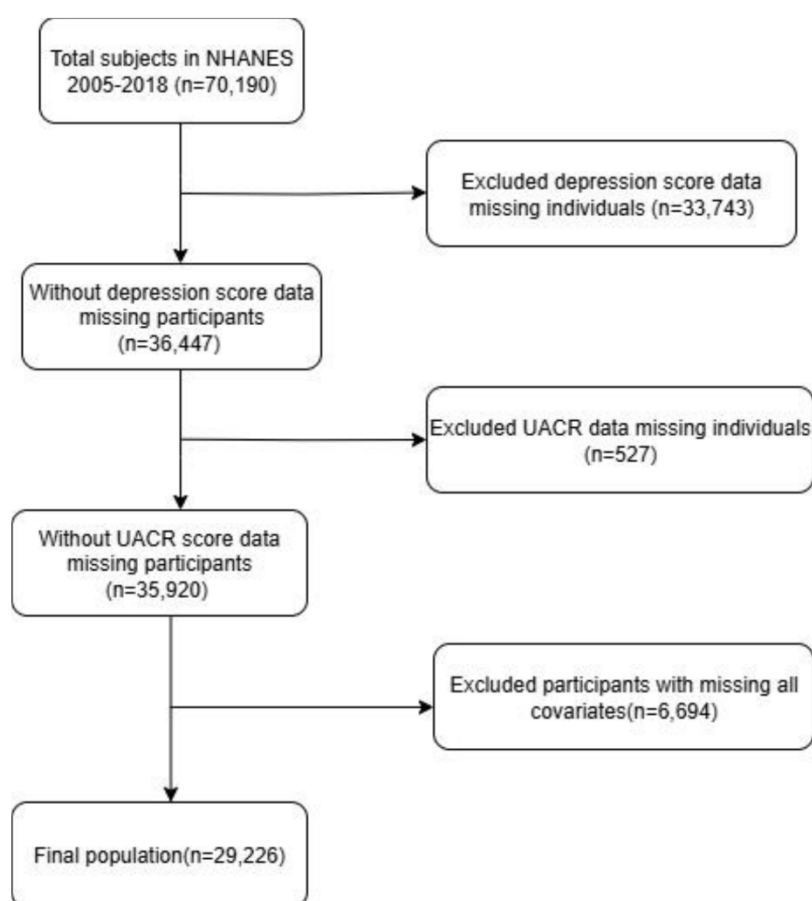


Figure 1. Study's flow diagram.

2.2. Measurement of UACR

To ensure accurate analysis, urine samples were prepared with care and maintained at a temperature of -30°C . A solid-phase fluoroimmunoassay was used to detect urinary albumin, whereas urinary creatinine was measured using an enzymatic quantitative technique. A comprehensive overview of the laboratory methodologies is accessible on the NHANES website^[13]. Before 2007, the kinetic Jaffe rate reaction method was used to measure urine creatinine levels; after 2007, the enzymatic method was used. Therefore, we adjusted urine creatinine levels from the 2005–2006 cycles using the NHANES-recommended equation. UACR was expressed in milligrams per gram (mg/g), and urinary albumin and creatinine levels were measured and normalized using established gold-standard techniques. In the context of clinical practice, a UACR of 30 mg/g or higher is regarded as proteinuria, with microalbuminuria being defined as a UACR between 30 mg/g and 300 mg/g. A UACR greater than 300 mg/g is considered macroalbuminuria^[14].

2.3. Diagnosis of depression

The NHANES used the validated nine-question PHQ-9 to evaluate symptoms of depression. This widely recognized instrument demonstrates a sensitivity and specificity of 88%^[15]. The PHQ-9 serves as a highly effective screening tool for depressive symptoms, evaluating symptoms experienced over the preceding two weeks. Technical abbreviations are clarified upon their initial introduction. The questionnaire measures

depressive symptoms in participants over the past two weeks, encompassing issues such as disturbed sleep, loss of appetite, and feelings of loneliness. Every item is evaluated using a 4-point ordinal scale that indicates how often symptoms occur (0, not at all; 1, several days; 2, more than half the days; 3, nearly every day)^[16]. The total score for the PHQ-9 is the result of adding the scores of its nine components. Consequently, participants' PHQ-9 scores were categorized into two groups: < 10 , indicating no depression, and ≥ 10 , indicating the presence of depression.

2.4. Explanation of covariates

Various factors related to UACR levels and the risk of depression were included in the study, organized into demographic, lifestyle, and health-related categories. The research accounted for demographic elements like age, sex, race, marital status, education, and the family income poverty ratio (PIR). Included in lifestyle factors were alcohol drinking habits (yes/no), smoking history (defined by having smoked over 100 cigarettes, categorized as never, former, or current), and Physical activity. [Physical activity was quantified using MET values, weekly frequency, and exercise duration. The formula used was $\text{MET-min/week} = \text{MET} \times \text{weekly frequency} \times \text{activity duration}$. According to this, participants were grouped into three categories: no PA (0 MET-min/week), low-level PA (1 to 599 MET-min/week), and high-level PA (≥ 600 MET-min/week)]^[17]. Body mass index (BMI), estimated glomerular filtration rate (eGFR), and diseases including diabetes, hypertension, and cardiovascular disease (CVD) were among the health indicators. The data collection process included standardized surveys and medical assessments, and eGFR was calculated via the CKD Epidemiology Collaboration equation^[18].

2.5. Analysis of statistics

To guarantee the sample's representativeness, the NHANES dataset's 'Full sample 2-year MEC exam weight' was utilized in accordance with the sampling and weighting guidelines. To guarantee that the results are nationally representative and to account for the bias brought on by the intricate sample design and oversampling of the population. For combining data from multiple survey cycles, we adjusted the weights in accordance with official guidelines, and in this study, data from seven cycles were combined, and the weights were divided by seven.

The Kolmogorov-Smirnov test was employed to verify the normality of continuous variables. Due to the right-skewed distribution of UACR levels, a natural logarithmic transformation was applied to achieve normality. Continuous variables conforming to a normal distribution were reported as mean $\pm$ standard deviation (SD), while non-normally distributed continuous variables were summarized using medians and interquartile ranges (IQR). Categorical variables were presented as frequencies and percentages. Comparative analyses of continuous variables between groups were conducted using t-tests, whereas chi-square tests were utilized for categorical variables, as appropriate. Multivariable logistic regression was employed to compute odds ratios (ORs) and 95% confidence intervals (CIs) for the purpose of evaluating the association between UACR and depressive symptoms. All statistical analyses were executed using the R software (<http://www.R-project.org>, The R Foundation) and Free Statistics software, version 2.2. A two-sided *P*-value of less than 0.05 was considered indicative of statistical significance.

3. Results

3.1. Baseline characteristics of participants

This study analyzed data from 29,226 individuals across seven NHANES cycles spanning 2005 to 2018. The weighted analysis allows sample results to be extrapolated more precisely to the entire target US non-institutionalized civilian population. The demographic summary in **Table 1** shows an average age of 47.27 $\pm$ 16.73 years and a gender distribution that is fairly balanced, with 51.2% of the population being female. Depressed individuals are characterized by: The depression group was more likely to be female (63.9% vs 50.1%), black or minority ethnic, less educated (8.0% vs 4.4%), living alone (51.4% vs 34.3%), and low-income (41.0% vs 18.9%) than the non-depression group. In terms of health, the prevalence of smoking (38.8%), obesity (47.6%), lack of physical activity (33.4%), hypertension (37.9%), and cardiovascular disease (15.8%) was significantly higher in the depression group. Importantly, there were no notable differences in age and kidney function metrics between the two groups. These data demonstrate strong associations between depressive status and socioeconomic disadvantage, poor health behaviors, and chronic diseases.

Table 1. Population characteristics by depression category

Variables	Participants*			<i>p</i> value
	Overall	Non-depression	Depression	
Age (mean $\pm$ SD)	47.27 $\pm$ 16.73	47.30 $\pm$ 16.81	46.89 $\pm$ 15.76	0.359
Sex, n (%)				< 0.001
Male	87981226.5 (48.8)	82918762.1 (49.9)	5062464.4 (36.1)	
Female	92299197.8 (51.2)	83336148.3 (50.1)	8963049.5 (63.9)	
Race, n (%)				< 0.001
Non-Hispanic White	125378377.2 (69.5)	116260296.1 (69.9)	9118081.0 (65.0)	
Non-Hispanic Black	18428061.8 (10.2)	16618550.1 (10.0)	1809511.7 (12.9)	
Mexican American	14561956.2 (8.1)	13486627.7 (8.1)	1075328.5 (7.7)	
Other	21912029.0 (12.2)	19889436.4 (12.0)	2022592.6 (14.4)	
Education, n (%)				< 0.001
Below high school	8486523.3 (4.7)	7371254.5 (4.4)	1115268.9 (8.0)	
High school	59942753.2 (33.2)	53814707.6 (32.4)	6128045.6 (43.7)	
Above high school	111851147.7 (62.0)	105068948.4 (63.2)	6782199.3 (48.4)	
Marry, n (%)				< 0.001
Married or living with partners	115962842.1 (64.3)	109146959.3 (65.7)	6815882.9 (48.6)	
Living alone	64317582.1 (35.7)	57107951.2 (34.3)	7209630.9 (51.4)	
Alcohol, n (%)				0.788
No	36250993.4 (20.1)	33466790.3 (20.1)	2784203.0 (19.9)	
Yes	143927354.4 (79.9)	132725643.4 (79.9)	11201711.0 (80.1)	
PIR, n (%)				< 0.001
Low income	37148809.0 (20.6)	31395759.5 (18.9)	5753049.5 (41.0)	
Medium income	64420718.8 (35.7)	59285523.5 (35.7)	5135195.3 (36.6)	
high income	78710896.4 (43.7)	75573627.3 (45.5)	3137269.0 (22.4)	
Smoke status, n (%)				< 0.001
Never	98523541.2 (54.7)	93211745.9 (56.1)	5311795.3 (37.9)	

Former	45414502.6 (25.2)	42149670.4 (25.4)	3264832.1 (23.3)	
Current	36342380.5 (20.2)	30893494.1 (18.6)	5448886.4 (38.8)	
BMI, n (%)				< 0.001
< 25	52856043.2 (29.3)	49145708.3 (29.6)	3710334.9 (26.5)	
≥ 25 < 30	59551606.0 (33.0)	55913845.9 (33.6)	3637760.1 (25.9)	
≥ 30	67872775.0 (37.6)	61195356.2 (36.8)	6677418.8 (47.6)	
Hypertension, n (%)				< 0.001
No	133647652.1 (74.1)	124942278.3 (75.2)	8705373.8 (62.1)	
Yes	46632772.1 (25.9)	41312632.1 (24.8)	5320140.0 (37.9)	
CVD, n (%)				< 0.001
No	165377770.4 (91.7)	153572512.7 (92.4)	11805257.7 (84.2)	
Yes	14902653.9 (8.3)	12682397.7 (7.6)	2220256.1 (15.8)	
PA (MET-min / week), n (%)				< 0.001
0	37251025.2 (20.7)	32563411.7 (19.6)	4687613.4 (33.4)	
1–599	30469464.2 (16.9)	28149361.4 (16.9)	2320102.9 (16.5)	
≥ 600	112559934.8 (62.4)	105542137.3 (63.5)	7017797.5 (50.0)	
Diabetes, n (%)				< 0.001
No	163405894.6 (90.7)	151492347.1 (91.2)	11913547.5 (85.0)	
Yes	16804945.5 (9.3)	14705570.1 (8.8)	2099375.5 (15.0)	
eGFR (mean ± SD)	93.81 ± 21.88	93.75 ± 21.81	94.58 ± 22.59	0.126
UACR (median [IQR])	6.55 (4.34, 11.72)	6.49 (4.30, 11.54)	7.29 (4.78, 14.66)	< 0.001

Notes: eGFR, estimated glomerular filtration; UACR, Urinary Albumin-to-Creatinine Ratio; SD, standard deviation; IQR, interquartile range. *,Data for categorical variables are presented as weighted numbers (weighted percentages).

3.2. Relationship between UACR and depression

To evaluate the link between the urinary albumin/creatinine ratio (UACR) and depression, we developed four stepwise adjusted models by weighted multivariate logistic regression analysis.

In unadjusted crude models, each natural logarithmic unit increase in the urine albumin-to-creatinine ratio (UACR) was associated with an 18% increase in the risk of depression [95% confidence interval (CI): 1.14–1.23; $p < 0.001$]. With the progressive adjustment for confounding variables, the effect size diminished: the association remained statistically significant in Model 3 (odds ratio [OR] = 1.06, 95% CI: 1.01–1.10), but was no longer significant in the fully adjusted Model 4 (OR = 1.01, 95% CI: 0.97–1.06, $p = 0.63$). The significance of the association in the microalbuminuria group (30–300 mg/g) was lost in Model 3 (OR = 1.09, 95% CI: 0.94–1.26). In the macroalbuminuria group (≥ 300 mg/g), In Model 4, the OR dropped to 1.19(95% CI: 0.86–1.66) as shown in **Table 2**.

Table 2. Association between UACR and depression

Variable	Crude		Model 1		Model 2		Model 3		Model 4	
	OR (95% CI)	<i>p</i> value	OR (95% CI)	<i>p</i> value	OR (95% CI)	<i>p</i> value	OR (95% CI)	<i>p</i> value	OR (95% CI)	<i>p</i> value
UACR(ln)	1.18 (1.14–1.23)	< 0.001	1.17 (1.13–1.22)	< 0.001	1.08 (1.03–1.13)	0.001	1.06 (1.01–1.10)	0.016	1.01 (0.97–1.06)	0.63

0 < UACR < 30 mg/g	1(Ref)		1(Ref)		1(Ref)		1(Ref)		1(Ref)	
30 ≤ UACR < 300 mg/g	1.44 (1.25–1.67)	< 0.001	1.45 (1.27–1.67)	< 0.001	1.17 (1.01–1.36)	0.036	1.09 (0.94–1.26)	0.265	0.98 (0.84–1.16)	0.85
UACR ≥ 300 mg/g	2.12 (1.57–2.86)	< 0.001	2.23 (1.64–3.03)	< 0.001	1.41 (1.03–1.92)	0.031	1.41 (1.03–1.95)	0.035	1.19 (0.86–1.66)	0.31
Trend test		< 0.001		< 0.001		0.002		0.0018		0.57

Notes: Crude: unadjusted model. Model 1: Adjusted for age and gender. Model 2: Adjusted for age, gender, hypertension, diabetes, cardiovascular disease. Model 3: Adjusted for age, gender, hypertension, diabetes, cardiovascular disease, estimated glomerular filtration, race, education level, marry, alcohol consumption, body mass index. Model 4: Adjusted for age and gender, hypertension, diabetes, cardiovascular disease, estimated glomerular filtration, race, education level, marital status, alcohol consumption, body mass index, poverty status, smoking, and physical activity. OR, odds ratio; CI, confidence interval.

3.3. Subgroup analyses

In this study, we performed stratified and interaction analyses to determine whether the association between UACR and the risk of depression was consistent across various subgroups (**Figure 2**). The results remained consistent when stratified by age (< 65 years or ≥ 65 years), sex (male or female), race/ethnicity (non-Hispanic white, non-Hispanic black, Mexican American, or other), educational attainment (below high school, high school, or above high school), marital status (married or living with partners, or living alone), body mass index (BMI) (< 25, ≥ 25 < 30, or ≥ 30), hypertension (no or yes), cardiovascular disease (CVD) (no or yes), and diabetes (no or yes). Despite conducting stratified analyses to assess potential effect modifications, our research found no statistically significant interactions in the link between UACR and depression within these subgroups ($p > 0.05$). In other words, the p -values for all interactions exceeded 0.05, indicating the absence of significant effect modification in our study.

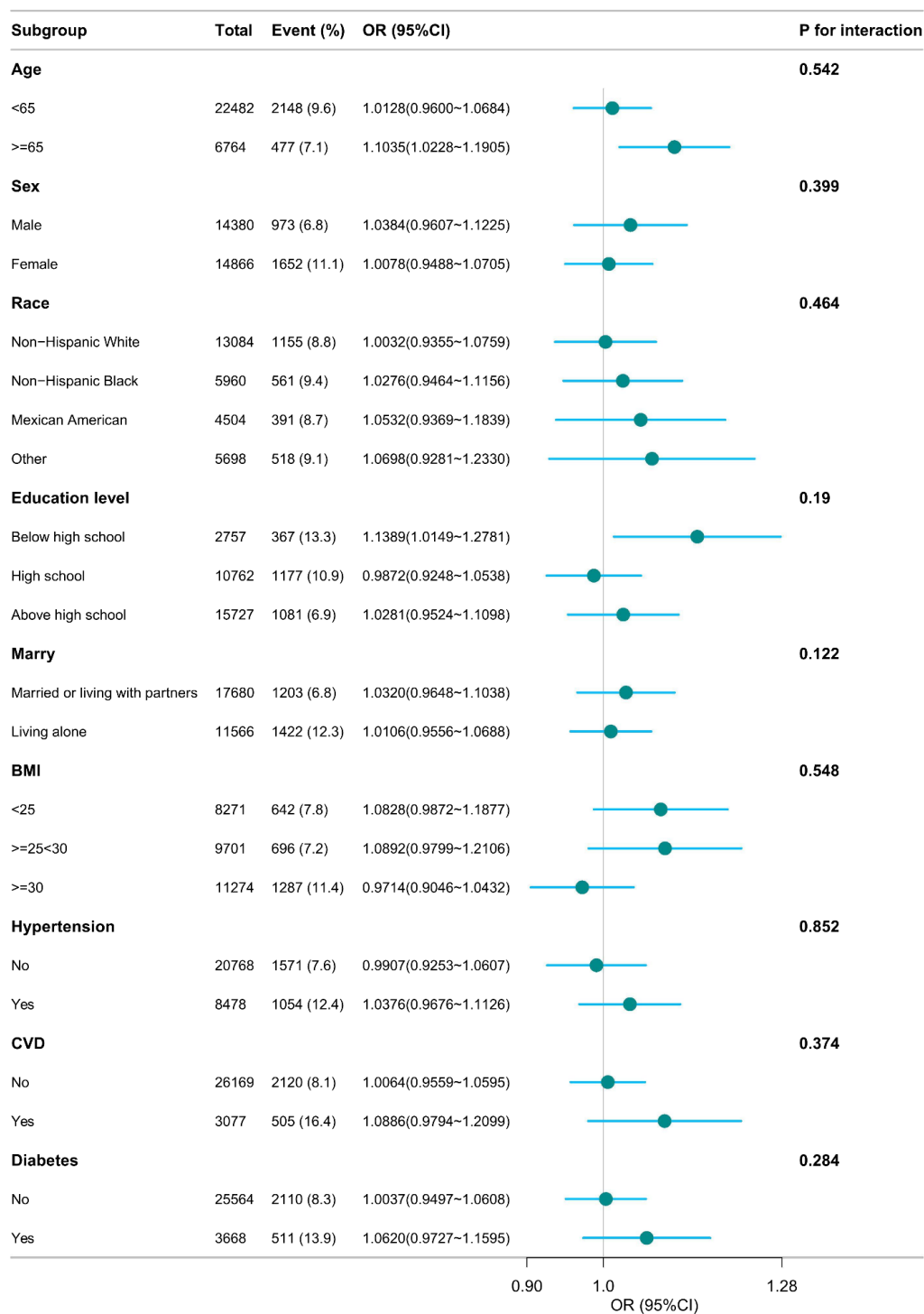


Figure 2. Association of urinary albumin-to-creatinine ratio (UACR) with depression.

Notes: Each stratification factor was adjusted for all other variables except the stratification component itself (age and gender, hypertension, diabetes, cardiovascular disease, estimated glomerular filtration, race, education level, marry, alcohol consumption, body mass index, poverty status, smoking, and physical activity). The green circle represents the odds ratio (OR) and the horizontal line represents the 95% confidence interval (CI).

4. Discussion

To our knowledge, this research marks the first extensive cross-sectional evaluation of the association between UACR and depression, utilizing weighted analyses of data from NHANES for American adults spanning 2005 to 2018. Our findings indicate that UACR independently serves as a risk factor for depression. In the absence of adjustments for covariates, the natural log-transformed UACR demonstrated a significant association with depression. However, this association diminished after comprehensive adjustment for covariates (model 4), with the significance level exceeding 0.05. Further subgroup analyses consistently revealed a robust association between UACR and depression.

UACR is a critical marker for the early diagnosis of chronic kidney disease (CKD), and individuals with CKD frequently exhibit reduced physical activity levels, which are closely linked to inflammation, oxidative stress, and metabolic disorders^[19]. These factors not only impair renal function but may also contribute to the manifestation of depressive symptoms.

Several mechanisms may elucidate the observed results. An elevation in UACR serves as an indicator of systemic vascular endothelial dysfunction, particularly reflecting damage to the glomerular filtration barrier^[20]. Such injury may concurrently impact cerebral vasculature, resulting in disturbances in cerebral microcirculation that compromise the delivery of oxygen and nutrients to neurons. Chronic insufficiency in cerebral blood flow can precipitate dysfunction in brain regions, including the hippocampus and prefrontal cortex, thereby elevating the risk of depression^[21]. Furthermore, albuminuria is strongly correlated with systemic inflammatory responses, characterized by the release of inflammatory mediators such as tumor necrosis factor- α (TNF- α) and interleukin-6 (IL-6)^[22]. Chronic inflammation can exert direct effects on the central nervous system by activating microglia and disrupting the synthesis and metabolism of neurotransmitters, such as serotonin, thereby potentially inducing depression^[23,24]. Furthermore, an elevated UACR is frequently associated with metabolic disorders such as diabetes and obesity. Empirical evidence indicates that the risk of developing diabetes increases by a factor of 1.8 for every 10 mg/g increment in UACR. Additionally, these metabolic conditions are recognized as independent risk factors for depression^[25]. In a study conducted on a Chinese cohort with diabetic nephropathy, UACR and depression were positively correlated ($R = 0.44$; $p < 0.001$). However, when all variables were accounted for in the model, depression did not demonstrate an independent association with severe proteinuria (UACR > 300 mg/g), a result that aligns with the findings of the present study^[12].

The objective of this study was to analyze the association between UACR and depression. Our analysis revealed that the strong link between UACR and depression weakened after accounting for covariates. This situation frequently arises in statistical analysis and requires thorough attention. First, it's crucial to comprehend how covariates affect the observed relationship. The poverty-income ratio (PIR), smoking, and physical activity are covariates that tend to simultaneously influence both UACR and depression. These factors, if not considered, can interfere with the accurate connection between UACR and depression. By controlling for these covariates, we can identify the independent influence of UACR, resulting in a more accurate evaluation of its association with depression. Secondly, the observed results could have been impacted by the sample size. While our study involved a significant number of participants, larger samples generally produce more precise and dependable estimates of association. Due to the current sample size, detecting subtle or moderate associations might have been challenging, especially after adjusting for several covariates. Studies conducted in the future with bigger sample sizes may deliver more decisive evidence

regarding this topic. Additionally, we must recognize the constraints of our study design. The cross-sectional nature of our study might influence how our findings are interpreted. Cross-sectional research provides a momentary view of associations, but it might not identify temporal connections or cause-and-effect relationships.

The main strengths of our study are the large sample size, coverage, and representation of the national population, which provided adequate power to examine the association between UACR and depression. The strengths of this study also include the use of a well-validated, district-wide electronic healthcare database that records detailed information on all diagnoses, hospitalizations, and medication distribution. Relevant information can therefore be gathered to rule out biases that are common in traditional observational studies. There are a few limitations to our study. To begin with, since it is an observational study, the study sample was mainly drawn from US adults, and the extrapolation of the results may be limited; further validation in other populations is needed. In addition, despite our efforts to adjust for many important confounders, we could not rule out an unmeasured confounder associated with the risk of depression, namely, medical history. Finally, despite the large sample size, the cross-sectional design limits inference of causality and cannot determine the direction of causality between UACR and depression.

The primary strengths of this study include its large sample size, extensive coverage, and representation of the national population, which collectively provided sufficient statistical power to investigate the association between UACR and depression. Additionally, the study benefits from utilizing a well-validated, district-wide electronic healthcare database, which records comprehensive information on diagnoses, hospitalizations, and medication distribution. This enables the collection of relevant data to mitigate biases commonly encountered in traditional observational studies. Nevertheless, the study has several limitations. Firstly, as an observational study predominantly involving American adults, the generalizability of the findings may be restricted, necessitating further validation in diverse populations. Moreover, despite efforts to adjust for numerous significant confounders, the possibility of unmeasured confounders, such as medical history, influencing the risk of depression cannot be entirely excluded. Lastly, although the study's large sample size is a strength, its cross-sectional design limits causal inference, precluding the determination of the directionality of the relationship between UACR and depression.

5. Conclusions

In summary, although our study found that the association of UACR with the prevalence of depression became less significant after adjusting for covariates, this is a common phenomenon in statistical analyses. The influence of covariates, the size of the sample, and limitations of the study design are all potential explanations for this observation. Future studies with larger sample sizes and more robust study designs are needed to further elucidate the complex association between UACR and the prevalence of depression.

Statement of Ethics

The NHANES study protocol received approval from the NCHS Research Ethics Review Board, and participants gave written informed consent when they enrolled (the website is <https://www.cdc.gov/nchs/index.html> for more information). Publicly accessible de-identified data was used in this study, so ethical approval and consent were not required.

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Disclosure statement

The authors declare no conflict of interest.

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