BMC Psychiatry

RESEARCH Open Access

Association between body roundness index and sleep disorder: the mediating role of depression

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Abstract

Background Several studies have indicated a potential association between obesity, depression, and sleep disorders. However, the role of depression in mediating the relationship between obesity and sleep disorders remains unclear. The Body Roundness Index (BRI), a more precise anthropometric measure of obesity than the traditional body mass index (BMI), is particularly effective in assessing body and visceral fat levels. This study examines the relationship between BRI and sleep disorders, with a focus on whether depression influences this association.

Methods This study included data from 32,504 participants in the National Health and Nutrition Examination Survey (NHANES) 2005–2018 cycle. The association between BRI and sleep disorders was examined through subgroup analysis, restricted cubic spline (RCS) modeling, threshold effect analysis, and multivariable logistic regression. Furthermore, the predictive capabilities of various anthropometric indices—including BRI, weight-adjusted waist index (WWI), BMI, and weight—on sleep disorder incidence were assessed using Receiver Operating Characteristic (ROC) curve analysis. Finally, a Mediation analysis was also performed to explore the potential role of depression in this relationship.

Results This study included 32,504 participants, of whom 4,568 reported sleep disorders. After adjusting for all covariates using multivariable logistic regression, each one-unit increase in BRI was associated with a 13% higher prevalence of sleep disorders (OR = 1.13, 95% CI: 1.09, 1.16) and an 8% higher prevalence of depression (OR = 1.08, 95% CI: 1.05, 1.11). Similar results were obtained when BRI was divided into tertiles, with a significant trend (P for trend < 0.05). RCS and threshold effect analyses revealed a nonlinear relationship between BRI and sleep disorder prevalence, with a breakpoint of 3.508. The ROC curve analysis revealed that BRI had a superior predictive capability compared to traditional obesity indices, with an area under the curve (AUC) of 0.637 (95% CI, 0.628–0.645, all $P < 0.001$). Mediation analysis further indicated that 14% of the association between BRI and sleep disorders was mediated by depression ($P < 0.001$).

Conclusion Elevated BRI levels were linked to a higher prevalence of sleep disorders, with depression acting as a partial mediator in this relationship. These findings emphasize the potential connection between obesity, depression, and sleep disorders, highlighting the importance of managing visceral fat to mitigate the risk of sleep disorders.

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Clinical trial number Not applicable.

Keywords Body roundness index, Sleep disorder, Depression, NHANES, Mediation analysis

Introduction

Sleep disorders are a widespread global health concern, severely affecting individuals' quality of life and overall well-being. Common types include insomnia, obstructive sleep apnea (OSA), and restless legs syndrome (RLS) [1]. In the United States, an estimated 50 to 70 million adults suffer from chronic sleep disorders [2], imposing a significant burden on the public health system.

In addition, sleep disorders are strongly linked to various metabolic, cardiovascular, and psychological conditions, including obesity, type 2 diabetes, heart disease, hypertension, stroke, and depression [3–6]. These comorbidities not only complicate disease management but also intensify the overall burden on patients. Most existing studies have concentrated on the relationship between sleep disorders and obesity, with body mass index (BMI) commonly used as the measure of choice. However, BMI fails to accurately capture fat distribution, particularly visceral fat [7, 8], which may be more closely associated with the onset of sleep disorders [9, 10]. Compared with BMI, the Body Roundness Index (BRI), a more accurate indicator of fat distribution [11], thus the use of BRI as an anthropometric indicator of fat distribution to study the association with sleep disorders deserves further exploration.

In recent years, the relationship between obesity and sleep disorders has garnered significant attention, with numerous studies demonstrating that obesity not only increases the risk of sleep disorders but also negatively impacts sleep structure and quality [12]. Concurrently, depression, a common mental health issue, has been closely linked to sleep disorders, potentially affecting sleep through mechanisms such as emotional dysregulation, circadian rhythm disturbances, and neuroendocrine imbalances [13, 14]. Moreover, a bidirectional relationship exists between depression and obesity: depression can lead to abnormal eating behaviors and weight gain, while obesity may exacerbate depressive symptoms through mechanisms like inflammation and hormonal dysregulation [15, 16]. Based on this evidence, this study hypothesizes that depression may serve as a mediator, partially explaining the association between obesity and sleep disorders. By exploring this potential mediation mechanism, we aim to deepen the understanding of the complex interplay among obesity, depression, and sleep disorders, providing valuable insights for developing targeted intervention strategies.

Hence, there is a critical need to investigate the relationship between BRI and sleep disorders, with a particular focus on the moderating role of depression. Examining the association between depression-mediated BRI and sleep disorders could offer new insights for enhancing the prevention and treatment of sleep disorders, especially when leveraging large-scale datasets like NHANES for multifactorial analysis. The current cross-sectional study holds significant clinical and public health implications.

Methodology

Study design and population

This investigation utilized data from NHANES collected between 2005 and 2018 to explore the relationship between BRI, depression, and sleep disorders. NHANES offers a nationally representative sample of the U.S. population, providing comprehensive data on health, nutrition, and demographics. The survey employs a complex, multistage probability cluster sampling design, with further details available at www.cdc.gov/nchs/Nhanes/. All participants provided informed consent, and the study was approved by the National Center for Health Statistics Research Ethics Review Board. Of the 70,190 participants across seven NHANES cycles (2005–2018), 39,041 were aged ≥ 20 years and were not pregnant. After excluding those with incomplete BRI data ($n = 3,857$), depression data ($n = 2,648$), and sleep disorder data ($n = 32$), 32,504 participants remained in the final analysis (Figure S1).

Sleep disorder assessment

This study assessed sleep disorder using the NHANES question, "Have you ever been told by a doctor or other health professional that you have a sleep disorder?" Participants who responded "Yes" were categorized as having a sleep disorder, while those who answered "No" were categorized as not having a sleep disorder. Responses of "Refused," "Don't know," or missing data were classified as missing and excluded from the analysis [17]. These questions were asked, in the home, by trained interviewers using the Computer-Assisted Personal Interviewing (CAPI) system. The CAPI system is programmed with built-in consistency checks to reduce data entry errors. CAPI also uses online help screens to assist interviewers in defining key terms used in the questionnaire (https://wwwn.cdc.gov/Nchs/Data/Nhanes/Public/2009/DataFiles/slq_f.htm). This sleep disorder questionnaire has been extensively applied and validated in various studies [9, 17].

Assessment of BRI

The Body Roundness Index (BRI) is a novel body shape assessment metric that

evaluates and calculates participants' measurements based on their height (cm) and waist circumference (cm):

$$BRI = 364.2 - 365.5 \times \sqrt{1 - \frac{\left(\frac{waist\ circumference}{2\pi}\right)^2}{(0.5 \times height)^2}}$$ [11]

Depression assessment

Depression was assessed using the 9-item Patient Health Questionnaire (PHQ-9), which is widely utilized as a screening tool in clinical settings. The PHQ-9 evaluates depressive symptoms over the past two weeks through face-to-face interviews. The total score ranges from 0 to 27, with a score of 10 or higher typically indicating the presence of depression [18, 19]. This threshold demonstrates 88% sensitivity and specificity in diagnosing depression [20].

Covariables

Based on previous research and clinical considerations, this study included several potential covariates that may influence the correlation between BRI and sleep disorders [21–23]. These covariates include age, sex, race, marital status, education level, poverty income ratio (PIR), obesity, smoking, alcohol consumption, hypertension, diabetes, and high cholesterol. For detailed information regarding these covariates, please refer to Table S1.

Statistical analysis

Statistical analyses were conducted using R (version 4.3.1), with all analyses employing sampling weights to ensure the national representativeness of the estimated data. In this study, the weight variable "WTMEC2YR" was utilized, and the new weights for the years 2005–2018 were calculated as 1/7 × WTMEC2YR [24]. Continuous variables are presented as mean ± standard deviation, with p-values derived from t-tests. The percentages of categorical variables (weighted N, %) and their associated p-values were computed using weighted chi-square tests.

Multivariable logistic regression models were employed to analyze the relationships between BRI and sleep disorders, as well as between BRI and depression. Three models were constructed: (1) a crude model without covariate adjustment; (2) a model adjusted for age, sex, education level, marital status, PIR, and race; and (3) a model adjusted for age, sex, education level, marital status, PIR, race, obesity, smoking, alcohol consumption, hypertension, diabetes, and high cholesterol. The BRI was converted into tertile for regression analysis to re-verify the robustness of the results. Restricted cubic spline (RCS) and threshold effect models were utilized to investigate the linear or nonlinear relationships and threshold effects of BRI on sleep disorders. Subgroup analyses were performed to explore the association between BRI and sleep disorders. Additionally, the discriminative ability of BRI, WWI, BMI, and weight for predicting sleep disorders was assessed using Receiver Operating Characteristic (ROC) curves and calculating the area under the curve (AUC).

Mediation analysis was conducted to evaluate the indirect, direct, and overall effects of depression as a mediator between BRI and sleep disorders, with the mediation proportion calculated as (indirect effect) / (indirect effect + direct effect) × 100%. The mediation effect was computed using the "mediation" package in R [24]. A two-tailed p-value of less than 0.05 was considered statistically significant.

Result

Baseline characteristics

This study included 32,504 participants aged 20 and older, representing approximately 196 million adults in the United States. The prevalence of sleep disorders was 15%, affecting around 30.35 million individuals. Participants with sleep disorders exhibited statistically significant differences in age, sex, race, education level, obesity, smoking, alcohol consumption, hypertension, diabetes, and high cholesterol ($p < 0.05$). Additionally, the BRI levels and depressive scores in the sleep disorder group were higher than those in the non-sleep disorder group. More detailed information can be found in Table 1. In addition, the unweighted baseline is shown in Table S2.

Association between BRI, depression, and sleep disorder

Table 2 presents the findings from three distinct models assessing the association between BRI and sleep disorders, all of which indicate a positive correlation (all $p < 0.001$). In Model 3, after adjusting for various covariates, each 1-unit increase in BRI was associated with a 13% increase in the prevalence of sleep disorders [odds ratio (OR): 1.13 (95% confidence interval: 1.09, 1.16)]. Additionally, when BRI was categorized into tertiles, participants in the highest tertile (T3) exhibited a 50% higher prevalence of sleep disorders compared to those in the lowest tertile (T1) [OR: 1.50 (95% confidence interval: 1.20, 1.87)]. However, the T2 group was not statistically significant.

Furthermore, an evaluation of the relationship between BRI and depression revealed a positive correlation across all three models (all $p < 0.05$); higher BRI levels were associated with increased prevalence of depression, with statistically significant results ($p < 0.05$). The results from the restricted cubic spline analysis (Fig. 1A) illustrated a J-shaped nonlinear association between BRI and sleep disorders, with an inflection point at 3.508 (nonlinear $P < 0.001$). Threshold effect analysis indicated a negative correlation between BRI and sleep disorders at BRI levels below 3.508 [OR = 0.859, 95% CI: 0.753, 0.983]. Conversely, BRI levels exceeding 3.508 were associated with a significant positive correlation with sleep disorders

Table 1 Baseline characteristics of all participants were stratified by sleep disorder, weighted

Characteristic	Overall, *N* = 196,564,414 (100%)	Non-sleep disorder, *N* = 166,204,826 (85%)	Sleep disorder, *N* = 30,359,588 (15%)	*P* Value
No. of participants in the sample	32,504	27,936	4,568	-
Age (%)				**< 0.001**
20–40	74,034,819 (38%)	66,607,606 (40%)	7,427,213 (24%)	
41–60	74,592,164 (38%)	60,892,057 (37%)	13,700,107 (45%)	
> 60	47,937,430 (24%)	38,705,162 (23%)	9,232,268 (30%)	
Gender (%)				**0.012**
Male	97,263,639 (49%)	82,954,618 (50%)	14,309,021 (47%)	
Female	99,300,775 (51%)	83,250,208 (50%)	16,050,567 (53%)	
Race (%)				**< 0.001**
Non-Hispanic White	133,934,058 (68%)	111,686,840 (67%)	22,247,218 (73%)	
Non-Hispanic Black	21,389,862 (11%)	18,430,226 (11%)	2,959,636 (9.7%)	
Other	24,896,910 (13%)	21,312,743 (13%)	3,584,167 (12%)	
Mexican American	16,343,584 (8.3%)	14,775,017 (8.9%)	1,568,567 (5.2%)	
Married/live with partner (%)				0.181
No	71,345,693 (36%)	59,966,570 (36%)	11,379,123 (37%)	
Yes	125,218,720 (64%)	106,238,255 (64%)	18,980,465 (63%)	
Education level (%)				**< 0.001**
Below high school	30,232,965 (15%)	26,575,677 (16%)	3,657,288 (12%)	
High School or above	166,331,449 (85%)	139,629,149 (84%)	26,702,300 (88%)	
PIR (%)				0.460
Not Poor	145,882,122 (79%)	123,452,211 (80%)	22,429,911 (79%)	
poor	37,711,542 (21%)	31,718,585 (20%)	5,992,957 (21%)	
Obesity (%)				**< 0.001**
No	123,211,303 (63%)	108,848,913 (66%)	14,362,390 (47%)	
Yes	73,156,357 (37%)	57,177,115 (34%)	15,979,242 (53%)	
Smoking (%)				**< 0.001**
Never	107,246,847 (55%)	93,522,796 (56%)	13,724,050 (45%)	
Former	49,218,380 (25%)	39,527,432 (24%)	9,690,949 (32%)	
Current	40,099,186 (20%)	33,154,598 (20%)	6,944,589 (23%)	
Drinking (%)				**< 0.001**
former	25,136,363 (13%)	20,853,080 (13%)	4,283,283 (15%)	
heavy	41,578,312 (22%)	35,972,808 (22%)	5,605,504 (20%)	
mild	70,407,890 (37%)	59,658,002 (37%)	10,749,888 (38%)	
moderate	33,755,420 (18%)	28,236,757 (17%)	5,518,663 (19%)	
never	20,266,329 (11%)	18,097,362 (11%)	2,168,968 (7.7%)	
Hypertension (%)				**< 0.001**
No	121,935,742 (62%)	107,834,952 (65%)	14,100,791 (46%)	
Yes	74,621,188 (38%)	58,362,391 (35%)	16,258,797 (54%)	
Diabetes (%)				**< 0.001**
No	169,023,763 (86%)	145,632,256 (88%)	23,391,507 (77%)	
Yes	27,537,696 (14%)	20,569,615 (12%)	6,968,080 (23%)	
High cholesterol (%)				**< 0.001**
No	110,102,517 (63%)	94,267,700 (65%)	15,834,817 (54%)	
Yes	64,506,595 (37%)	50,814,882 (35%)	13,691,714 (46%)	
BRI (mean (SD))	5.37 (2.32)	5.20 (2.20)	6.34 (2.69)	**< 0.001**
BRI (%)				**< 0.001**
T1	65,519,602 (33%)	59,084,902 (36%)	6,434,700 (21%)	
T2	65,518,290 (33%)	56,822,184 (34%)	8,696,106 (29%)	
T3	65,526,522 (33%)	50,297,740 (30%)	15,228,782 (50%)	
Depressive score (mean (SD))	3.05 (4.22)	2.64 (3.83)	5.27 (5.42)	**< 0.001**
Depression (%)				**< 0.001**

Table 1 (continued)

Characteristic	Overall, *N* = 196,564,414 (100%)	Non-sleep disorder, *N* = 166,204,826 (85%)	Sleep disorder, *N* = 30,359,588 (15%)	*P* Value
No	181,203,729 (92%)	156,641,065 (94%)	24,562,664 (81%)	
Yes	15,360,684 (7.8%)	9,563,761 (5.8%)	5,796,924 (19%)	

Mean (SD) for continuous variables: the P value was calculated by the weighted Students T-test

Percentages (weighted N, %) for categorical variables: the P value was calculated by the weighted chi-square test

Abbreviation: BRI, body roundness index; PIR, Ratio of family income to poverty

Table 2 Associations between the BRI and sleep disorder and depression, NHANES 2005–2018

Characteristics	Model 1 [OR (95% CI)]	*p*-value	Model 2 [OR (95% CI)]	*p*-value	Model 3 [OR (95% CI)]	*p*-value
BRI - sleep disorder						
Continuous	1.21 (1.18, 1.23)	< 0.001	1.20 (1.17, 1.22)	< 0.001	1.13 (1.09, 1.16)	< 0.001
Tertile						
T1	1 (ref.)		1 (ref.)		1 (ref.)	
T2	1.41 (1.25, 1.58)	< 0.001	1.25 (1.09, 1.43)	0.001	1.01 (0.87, 1.18)	0.900
T3	2.78 (2.43, 3.18)	< 0.001	2.51 (2.17, 2.90)	< 0.001	1.50 (1.20, 1.87)	< 0.001
P for trend	< 0.001		< 0.001		< 0.001	
BRI - depression						
Continuous	1.12 (1.10, 1.15)	< 0.001	1.10 (1.08, 1.13)	< 0.001	1.08 (1.05, 1.11)	< 0.001
Tertile						
T1	1 (ref.)		1 (ref.)		1 (ref.)	
T2	1.08 (0.96, 1.22)	0.200	1.19 (1.04, 1.36)	0.012	1.11 (0.94, 1.32)	0.200
T3	1.77 (1.55, 2.04)	< 0.001	1.75 (1.50, 2.04)	< 0.001	1.38 (1.10, 1.73)	0.005
P for trend	< 0.001		< 0.001		0.002	

Model 1: no covariates were adjusted

Model 2: age, gender, education level, marital, PIR, and race were adjusted

Model 3: age, gender, education level, marital, PIR, race, obesity, smoking, drinking, hypertension, diabetes, and high cholesterol.

Abbreviation: BRI, body roundness index; PIR, Ratio of family income to poverty; OR, odds ratio; CI, confidence interval

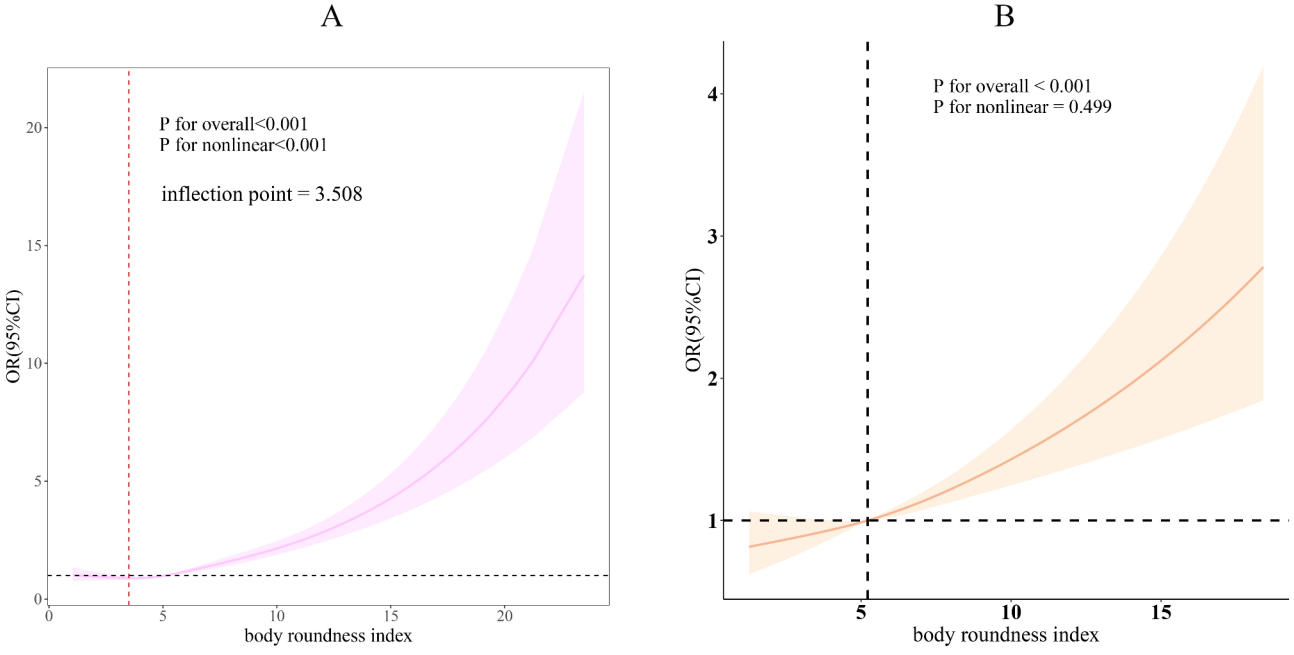


Fig. 1 Dose-response relationships between BRI and sleep disorder and depression. **A**, BRI - sleep disorder; **B**, BRI - depression. OR (solid lines) and 95% confidence levels (shaded areas) were adjusted for age, gender, education level, marital, PIR, race, obesity, smoking, drinking, hypertension, diabetes, and high cholesterol

[OR = 1.157 (1.132, 1.182)] (Table S3). Additionally, there was a linear positive correlation between BRI and depression (Fig. 1B).

Subgroup analyses stratified by age, sex, race, marital status, education level, PIR, obesity, smoking, alcohol consumption, hypertension, diabetes, and high cholesterol (Fig. 2) consistently demonstrated a significant positive correlation between BRI, depression, and sleep disorder across all subgroups.

BRI as a predictor for sleep disorder

We compared the predictive ability of BRI and various body measurement indicators for sleep disorder likelihood by calculating the area under the curve (AUC) (Fig. 3). In this analysis, BRI demonstrated a strong advantage over the other five indicators (WWI, BMI, Weight) with an AUC of 0.637 (95% CI, 0.628–0.645), all P-values < 0.001.

Mediation effect

The mediation model depicted in Fig. 4 illustrates BRI as the independent variable, sleep disorders as the dependent variable, and depression as the mediator variable. As shown in Table 3, after adjusting for other covariates, a significant correlation between depression and sleep disorders was observed (OR = 3.67, 95% confidence interval: 3.17, 4.25). Following the adjustment for all covariates, the mediating effect of depression was evident, with an indirect effect of 0.0014 ($P < 0.001$) and a direct effect of 0.0086 ($P < 0.001$). The mediation proportion was calculated at 14% ($P < 0.001$).

Discussion

The current cross-sectional study utilized a representative sample of 32,504 U.S. citizens aged 20 and older from the NHANES dataset. Multivariate logistic regression determined that higher BRI was significantly associated with a higher prevalence of sleep disorders. This robust correlation remained significant after adjusting for various covariates, indicating that BRI may serve as a reliable tool for assessing sleep disorder risk. Importantly, the study identified a nonlinear relationship, revealing a critical threshold of 3.508 for BRI, beyond which the risk of sleep disorders increased. These findings emphasize that individuals with a BRI higher than 3.508 are at higher risk. Additionally, BRI demonstrated superior predictive

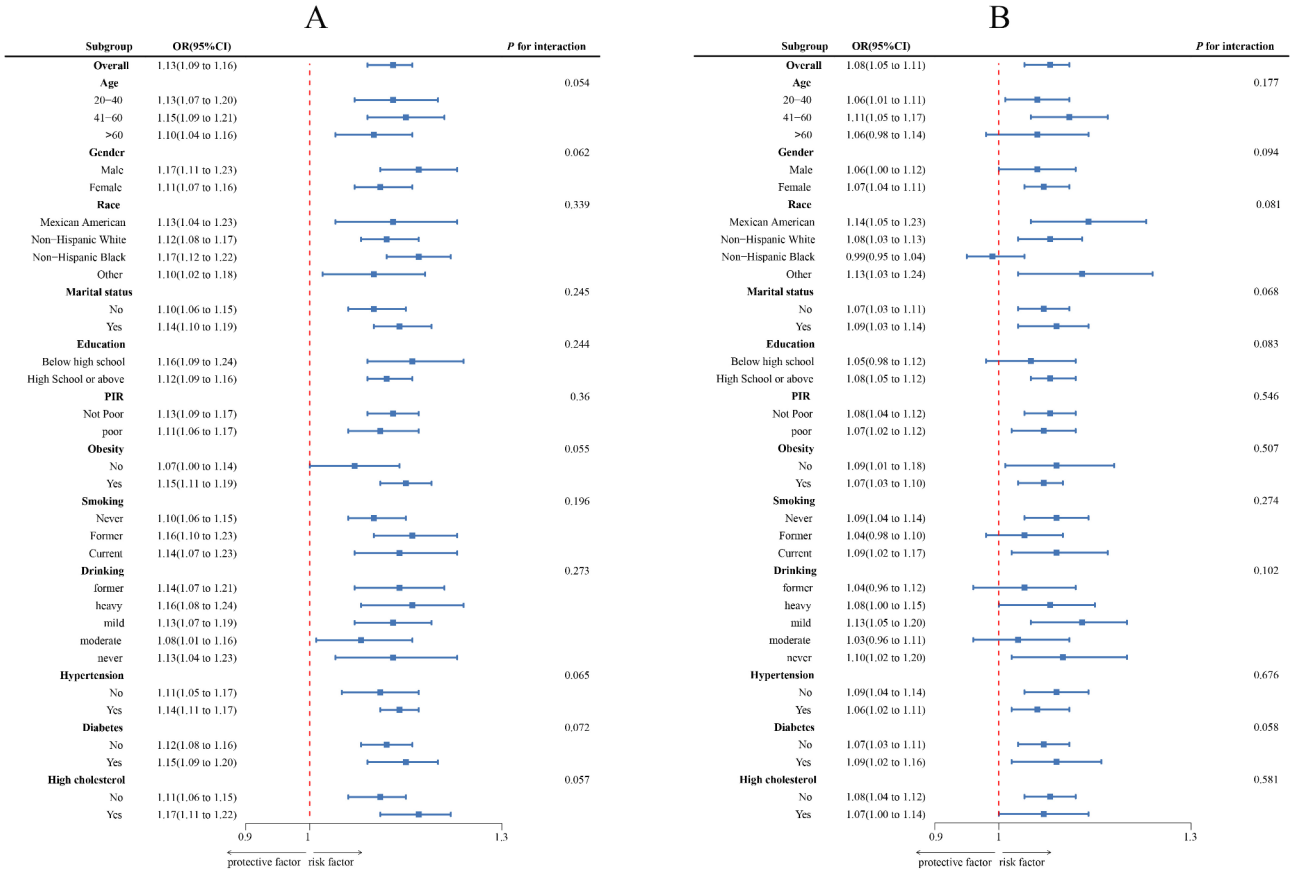
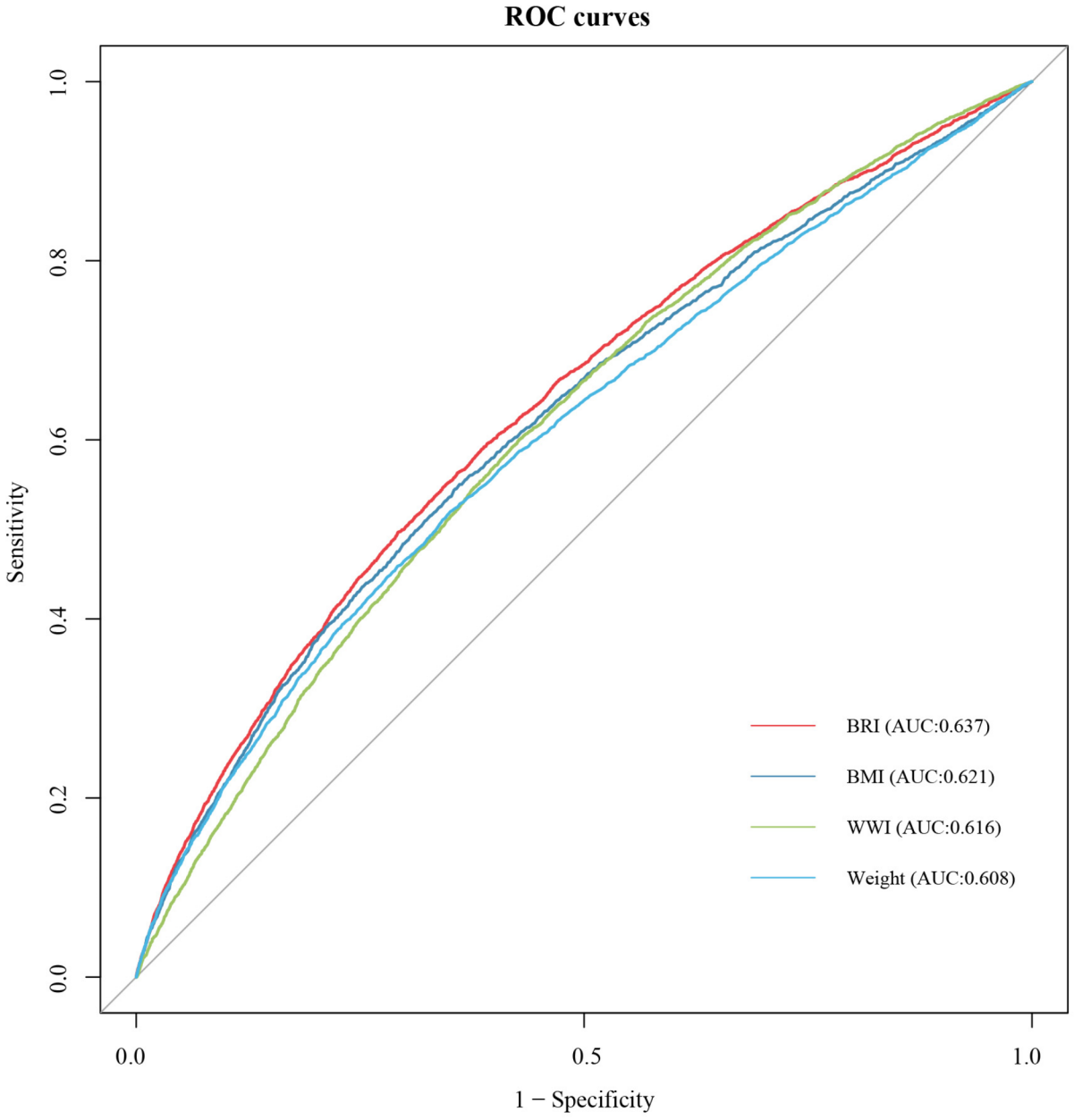


Fig. 2 Subgroup analysis between BRI and sleep disorder and depression. **A**, BRI - sleep disorder; **B**, BRI - depression. ORs were calculated as each unit increased in BRI. Analyses were adjusted for age, gender, education level, marital, PIR, race, obesity, smoking, drinking, hypertension, diabetes, and high cholesterol



Anthropometric Measures	Best thresholds	Specificity	Sensitivity	AUC (95%CI)	P for difference in AUC
BRI	6.200	0.708	0.497	0.637 (0.628–0.645)	Reference
BMI	29.895	0.639	0.550	0.621 (0.612–0.630)	<0.001
WWI	11.159	0.573	0.600	0.616 (0.607–0.624)	<0.001
Weight (Kg)	85.350	0.649	0.520	0.608 (0.598–0.617)	<0.001

Fig. 3 Receiver operating characteristic (ROC) curve analysis for sleep disorder

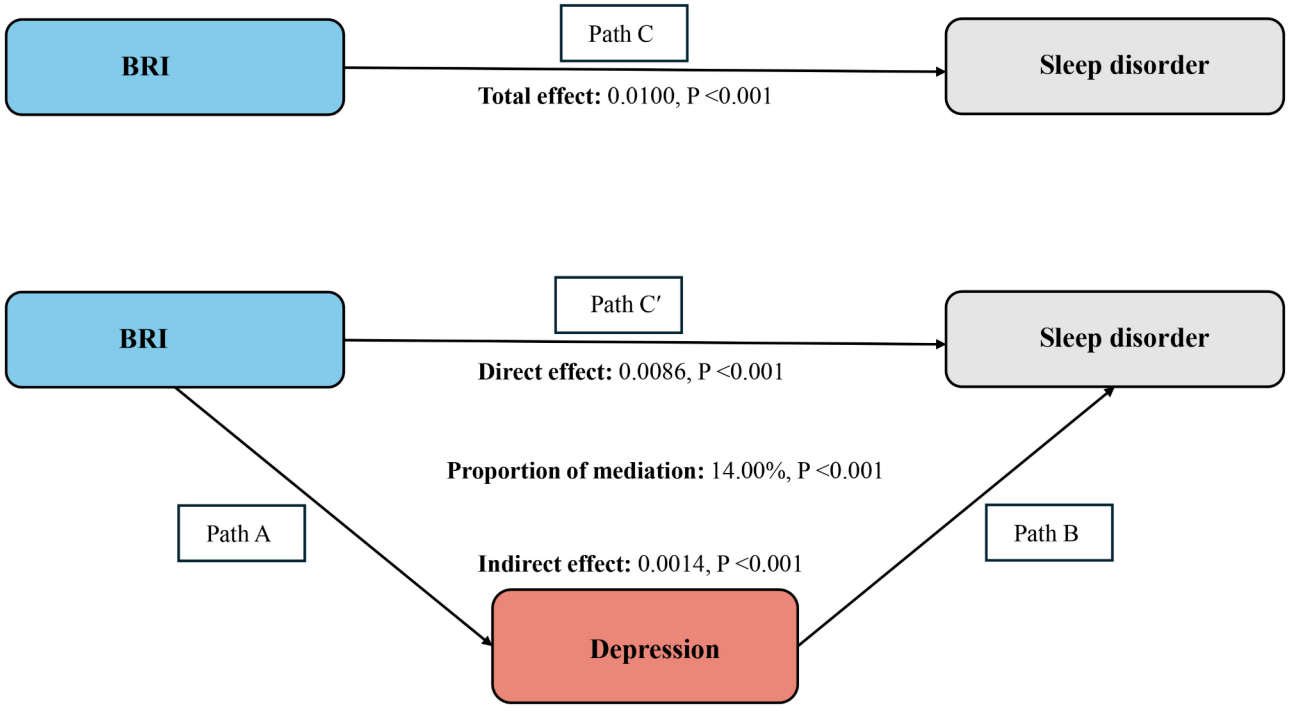


Fig. 4 Schematic diagram of the mediation effect analysis. Path C indicates the total effect; path C′ indicates the direct effect. The indirect effect is estimated as the multiplication of paths **A** and **B** (path **A***B). The mediated proportion is calculated as indirect effect/ (indirect effect + direct effect) × 100%. Abbreviation: BRI, body roundness index. Analyses were adjusted for age, education level, marital, PIR, race, smoking, drinking, Physical activity, hypertension, diabetes, and high cholesterol

Table 3 Associations between depression and sleep disorder

Depression	Model 1 [OR (95% CI)]	*p*-value	Model 2 [OR (95% CI)]	*p*-value	Model 3 [OR (95% CI)]	*p*-value
No	1 (ref.)		1 (ref.)		1 (ref.)	
Yes	3.87 (3.46, 4.32)	< 0.001	4.19 (3.70, 4.75)	< 0.001	3.67 (3.17, 4.25)	< 0.001

Model 1: no covariates were adjusted

Model 2: age, gender, education level, marital, PIR, and race were adjusted

Model 3: age, gender, education level, marital, PIR, race, obesity, smoking, drinking, hypertension, diabetes, and high cholesterol.

Abbreviation: PIR, Ratio of family income to poverty; OR, odds ratio; CI, confidence interval

performance, as evidenced by the highest AUC. Finally, mediation analysis showed that depression mediated the association between BRI and sleep disorders. These findings emphasize the clinical significance of maintaining optimal BRI levels as a potential protective factor against sleep disorders.

In this study, we conducted subgroup analyses by categorizing participants into three age groups: 20–40 years, 41–60 years, and > 60 years to explore the potential impact of age on the association between BRI and sleep disorders. Although no significant interaction between age and the association was observed ($P > 0.05$), we found that participants in the > 60 years group exhibited a lower odds ratio (OR) for the association. This attenuated association in older adults may reflect the influence of other factors, such as physiological aging, comorbid chronic conditions, and lifestyle changes, which could play a more prominent role in sleep disorders in this population. Additionally, the unique fat distribution patterns and metabolic adaptations in older adults might weaken the explanatory power of BRI as a body shape index for sleep disorders. These findings highlight the need for future studies to consider potential confounding factors in older populations, such as comorbidity burden, mental health status, and physical activity levels, to better elucidate the complex relationship between BRI and sleep disorders. Although no significance was observed in the T2 group, it is notable that the T3 group still showed significant results, and the P-value for the trend test across T1-T3 was less than 0.05. This suggests a trend in the relationship between BRI and sleep disorders, as well as between BRI and depression. Therefore, we believe this result still supports a positive correlation between BRI and both sleep disorders and depression.

The Body Roundness Index (BRI), as an indicator of body shape and fat distribution, reflects the concentration of body fat, particularly abdominal fat. Abdominal fat, especially visceral fat, is closely associated with lipid metabolism [25]. Leptin and adiponectin are two important hormones secreted by adipose tissue that play critical roles in regulating energy metabolism, appetite, and inflammatory responses [26]. Leptin is a satiety hormone that typically exhibits elevated levels in obese individuals; however, these individuals often develop leptin resistance [26]. Leptin resistance refers to a condition in which, despite increased leptin levels, the body's sensitivity to its signals diminishes, leading to ineffective appetite suppression and impaired energy expenditure. This dysregulation can disrupt the functioning of neural circuits in the hypothalamus, thereby affecting normal sleep cycles [27]. Adiponectin is an anti-inflammatory hormone that regulates glucose metabolism and fatty acid oxidation [28]. In obese individuals, adiponectin levels are generally lower [29]. Reduced levels of adiponectin can exacerbate chronic inflammatory states in the body [30], and inflammatory factors such as TNF-α and IL-6 can cross the blood-brain barrier to affect the central nervous system, thereby disrupting sleep. Studies have shown that decreased adiponectin levels are closely linked to reductions in deep sleep and sleep disturbances [31].

Elevated BRI is associated with increased adipose tissue, which triggers immune responses [32]. These immune factors can disrupt the immune regulation of the central nervous system, potentially leading to sleep disorders. Adipose tissue is not merely a storage organ for energy; it also plays significant endocrine and immune roles [33]. Particularly in the context of obesity, adipose tissue becomes highly active, secreting various pro-inflammatory and anti-inflammatory cytokines collectively known as adipokines [34]. Among these, leptin, adiponectin, resistin, various interleukins (such as IL-6 and IL-1β), and tumor necrosis factor-alpha (TNF-α) are produced in excess in adipose tissue and participate in systemic inflammation and metabolic regulation [33]. Some adipokines, especially leptin, resistin, and TNF-α, have been shown to be closely linked to sleep quality. For instance, leptin not only regulates appetite and energy balance but also directly affects the hypothalamic centers responsible for sleep regulation [35]. Obese individuals often exhibit leptin resistance [36], meaning that even with elevated leptin levels, physiological functions are impaired, potentially disrupting the normal sleep-wake cycle. Additionally, resistin [37] and TNF-α [38] are potent pro-inflammatory factors that can activate inflammatory pathways, such as the NF-κB signaling pathway, leading to both systemic and local inflammatory responses. This inflammation not only impacts overall metabolism but can also cross the blood-brain barrier, affecting sleep regulatory networks in the brain [39, 40]. Studies have found that high levels of TNF-α associated with obesity are closely related to sleep disorders, including insomnia and sleep apnea [39, 41].

Interleukin-6 (IL-6) [42] is a crucial pro-inflammatory cytokine involved in various physiological and pathological processes, including immune responses, inflammation, and metabolic regulation. Studies have shown a close association between elevated IL-6 levels, obesity, chronic inflammation, and sleep disorders [43]. As the Body Roundness Index (BRI) increases, the accumulation of adipose tissue can lead to the infiltration of immune cells such as macrophages, resulting in heightened secretion of pro-inflammatory cytokines like IL-6 [44]. IL-6 can cross the blood-brain barrier [45] and directly affect sleep-regulating regions in the brain, including the hypothalamus and brainstem. High levels of IL-6 [46] are associated with decreased sleep quality, reduced sleep duration, and disrupted sleep architecture. Furthermore, IL-6 influences the synthesis and release of neurotransmitters [47], such as serotonin, which plays a key role in regulating the sleep-wake cycle [48]. Additionally, IL-6 can stimulate the activation of the hypothalamic-pituitary-adrenal (HPA) axis, leading to increased cortisol levels [49]. Elevated cortisol levels are closely linked to sleep disorders, including difficulties in falling asleep and maintaining sleep [50]. The rise in IL-6 levels can promote oxidative stress and neuroinflammation [51], impairing neuronal function and disrupting the normal operation of sleep regulatory networks.

Research indicates that individuals with obesity often experience dysbiosis in their gut microbiota [52]. Furthermore, this dysbiosis may be linked to sleep disorders [53]. The gut microbiota can influence the brain through various mechanisms, including the production of metabolites such as serotonin [54], histamine [55], short-chain fatty acids (SCFAs) [56], lipopolysaccharides (LPS) [57], gamma-aminobutyric acid (GABA), melatonin [58], and dopamine. Dysbiosis can lead to increased intestinal permeability, resulting in a condition commonly referred to as "leaky gut," where bacterial metabolites and endotoxins (such as LPS) pass through the intestinal wall into the bloodstream. LPS is a potent inflammatory inducer that activates the immune system, leading to the production of pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α), IL-1β, and interleukin-6 (IL-6), which trigger systemic inflammation [59]. These inflammatory mediators not only affect various bodily systems but can also cross the blood-brain barrier (BBB) and induce neuroinflammation [60]. Neuroinflammation disrupts the balance of neurotransmitters, interfering with the mechanisms that regulate sleep [61]. For instance, TNF-α and IL-1β can affect neurons in the preoptic area and basal forebrain of the hypothalamus, which are

critical regions for sleep regulation [62]. Additionally, TNF-α can inhibit melatonin synthesis via the NF-κB pathway [63]. Melatonin is a key hormone regulating circadian rhythms, and its reduction can lead to sleep disturbances.

The following mechanisms may explain the association between depression-mediated BRI and sleep disorders. An elevated BRI is associated with obesity, which is a known risk factor for depression. Factors such as increased body fat, leading to diminished self-esteem, chronic inflammation [64], and endocrine disorders [65] are closely linked to the onset of depression. In turn, depression negatively impacts sleep quality by causing disruptions in circadian rhythms, imbalances in neurotransmitters, and fluctuations in mood, which can result in insomnia, hypersomnia, or poor sleep quality [66]. Moreover, both obesity and depression are associated with increased pro-inflammatory factors and neuroinflammatory responses [65]. These inflammatory processes not only exacerbate depressive symptoms but also directly interfere with sleep regulation. Behavioral changes, such as reduced physical activity and unhealthy eating habits often seen in individuals with depression, can further contribute to increased body fat [67], worsening sleep problems. Additionally, the side effects of antidepressant medications, such as selective serotonin reuptake inhibitors (SSRIs) [68], can lead to weight gain and may indirectly affect sleep through their impact on BRI.

This study possesses several strengths: (1) It is the first to investigate the correlation between BRI and the prevalence of sleep disorders in the U.S. population, indicating the substantial potential of BRI as a diagnostic and assessment tool for sleep disorders. This offers new recommendations for the diagnosis and evaluation of sleep disorders. (2) BRI, as a novel indicator for assessing visceral fat content, demonstrates superiority over traditional anthropometric methods in screening for sleep disorders, facilitating low-cost identification of sleep disorders in clinical settings. (3) The study incorporates a large population sample, including data from NHANES spanning 2005 to 2018, which accurately represents the demographic characteristics of the entire nation. (4) By constructing various models and conducting subgroup analyses while adjusting for confounding factors, strong positive correlations between BRI and sleep disorders were observed, indicating robust and credible results. (5) To the best of our knowledge, this is the first investigation of the mediating role of depression in the association between BRI and the odds of sleep disorders.

Despite the significant contributions of this study, several limitations must be acknowledged: (1) As a cross-sectional study, it cannot establish a causal relationship between BRI and the prevalence of sleep disorders. Further prospective studies with a larger and more diverse sample are necessary to elucidate the causal relationship between BRI and sleep disorders. (2) The data for this study were derived from the NHANES dataset spanning 2005 to 2018, meaning that the findings are only applicable to U.S. adults, with limited generalizability to populations in other countries. (3) The diagnosis of sleep disorders in this study relied on questionnaire data recorded by NHANES, which may be subject to recall bias. The incorporation of additional objective measures is recommended in future studies to further enhance the comprehensive assessment of sleep disorders. (4) Although many potential confounding factors have been adjusted for, it remains impossible to exclude the influence of unmeasured or unidentified factors on the results. (5) Given that this study is based on a cross-sectional design, we recommend conducting longitudinal studies in the future to clarify the causal relationship between depression, BRI, and sleep disorders. Considering that depression may serve as only a partial mediator, future research could further explore other potential psychological or physiological mediating mechanisms, such as anxiety, inflammatory markers, or endocrine dysfunction, that may influence the relationship between BRI and sleep disorders.

Conclusion

In conclusion, a significant positive correlation between BRI and sleep disorders was identified, with depression acting as a partial mediator in this relationship. This finding underscores the potential link between obesity and sleep disorders, highlighting the importance of managing visceral fat levels in this context. Our research offers new insights into the prevention and management of sleep disorders, emphasizing that a comprehensive approach addressing mental health and obesity management may contribute to reducing the prevalence of sleep disorders.

Abbreviations

BRI	Body roundness index
RCS	Restricted Cubic Spline
OR	Odds Ratio
NHANES	National Health and Nutrition Examination Survey

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12888-025-06664-z .

Supplementary Material 1

Acknowledgements

We sincerely appreciate the NHANES database for all the data.

Author contributions

H.G. contributed to the original draft, Methodology, and Formal analysis. Y.Z. was involved in Writing – review & editing, Supervision, Project administration, and Investigation.

Funding
No specific funding for this research was provided by the government, business, or nonprofit sectors.

Data availability
The corresponding author can provide the datasets used and/or analyzed in this study upon reasonable request.

Declarations

Ethics approval and consent to participate
The National Center for Health Statistics Ethics Review Board approved this study's human subjects components, which followed the Declaration of Helsinki. For every subject, written informed permission was acquired.

Consent for publication
All participants gave informed consent for publication.

Competing interests
The authors declare no competing interests.

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Received: 22 October 2024 / Accepted: 26 February 2025
Published online: 07 March 2025

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