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ORIGINAL RESEARCH

Association Between Body Roundness Index and Chronic Obstructive Pulmonary Disease in US Adults: Data from NHANES 2013–2018

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Background: The association between the body roundness index (BRI) and the prevalence of chronic obstructive pulmonary disease (COPD) in US adults remains unclear. This study aims to investigate the association between BRI and the likelihood of developing COPD.

Methods: This study was conducted based on data from the 2013–2018 National Health and Nutrition Examination Survey. Participants were classified as having COPD if they met any of the following criteria: (i) self-reported physician diagnosis of COPD; (ii) physician-confirmed diagnosis of emphysema; or (iii) physician-confirmed diagnosis of chronic bronchitis. Those who responded "no" to all of the above were categorized as non-COPD. To assess the association between BRI and COPD, weighted logistic regression models, subgroup analyses, and interaction tests were employed. The dose-response relationship was investigated using a restricted cubic spline (RCS) model.

Results: A total of 14,254 individuals were included. The overall weighted prevalence of COPD was 8.3%. After adjusting for multiple confounders, continuous BRI was found to be positively associated with COPD (odds ratio [OR] = 1.140, 95% confidence interval [CI]: 1.033–1.259, P = 0.012). The RCS analysis confirmed a linear dose-response relationship between BRI and COPD. Subgroup analyses demonstrated substantial heterogeneity across sex, hypertension, and cardiovascular disease subgroups, indicating that the association between BRI and COPD may be impacted by these factors.

Conclusion: Higher BRI levels were positively associated with an increased likelihood of developing COPD among US adults. Our study suggests that BRI holds promise as a tool for assessing the odds of having COPD.

Keywords: chronic obstructive pulmonary disease, body roundness index, BRI, abdominal fat, obesity, cross-sectional study, NHANES

Introduction

Chronic obstructive pulmonary disease (COPD) is a group of progressive respiratory disorders caused by chronic airway inflammation, and it is characterized by airway obstruction or persistent airflow limitation.[1] Major symptoms of this disease include chronic cough, sputum production, and dyspnea. Clinically, COPD often exhibits recurrent exacerbations and is difficult to cure, and the common diseases of COPD include asthma, emphysema, and chronic bronchitis.[2] Progressive decline in lung function leads to reduced physical activity (PA) in individuals with COPD and multiple complications, including congestive heart failure (CHF) and diabetes, as well as psychological issues (such as severe

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anxiety and depression). These factors severely impair patients' quality of life and can be life-threatening. The World Health Organization reports that nearly 299 million individuals worldwide suffer from COPD. It stands as the fourth major cause of death worldwide,[3] with incidence rates rising every year. For example, in 2017, the prevalence of COPD in the United States was 15.2% among current cigarette smokers, 7.6% among former smokers, and 2.8% among adults who had never smoked.[4] COPD typically develops insidiously and is often overlooked during the early stages. Patients usually receive medical attention only after symptoms become pronounced, thereby resulting in a poor prognosis.[5,6] COPD also requires substantial healthcare resources, imposing a considerable burden on global medical and public health systems. Therefore, identifying factors influencing COPD and developing effective prevention strategies to reduce its incidence is of great importance in alleviating this global health and socioeconomic burden.

Obesity, a prevalent metabolic disorder, is linked to a range of systemic complications. It can cause functional impairments in organs and tissues, ultimately resulting in organic lesions. The relationship between obesity and COPD has received growing attention.[7] In particular, the accumulation of visceral fat (VF) in individuals with obesity may elevate the risk of COPD.[8] Obesity may trigger pulmonary disease by inducing oxidative stress (OS) and systemic inflammation.[7,9] Thus, a reliable indicator for assessing VF is essential for effective chronic disease control and enhancing quality of life.

The Body Roundness Index (BRI), a novel anthropometric metric based on waist circumference (WC) and height, can comprehensively reflect visceral adiposity and body fat percentage.[10] Increasing evidence demonstrates that BRI holds great potential for disease risk stratification. Elevated BRI levels are associated with many chronic diseases, such as chronic kidney disease (CKD), diabetes, and cardiovascular diseases (CVD), including atherosclerosis and hypertension (HP).[11,12] Nonetheless, the relationship between BRI and COPD remains unclear. Using data from the National Health and Nutrition Examination Survey (NHANES) 2013–2018, this study aims to examine the association between BRI and COPD.

Materials and Methods

Study Design and Population

Conducted by the National Center for Health Statistics (NCHS), the NHANES employs a stratified, multistage sampling design and various data collection methods to analyze the nutritional and health status of American children and adults. All NHANES protocols were approved by the Ethics Review Board of NCHS. Written informed consent was provided by all enrolled individuals. To ensure participant confidentiality, all data were de-identified. According to the Ethical Review Methods for Life Science and Medical Research Involving Human Participants, Article 32 exempts certain research from requiring ethical approval under specific conditions. Research utilizing legally obtained public data or data derived from non-intrusive observation of public behavior does not require ethical approval.

Data from three consecutive NHANES cycles (2013–2018) were collected. A total of 29,400 individuals were initially included. Individuals were excluded if they were under 20 years of age (n = 12,343), had incomplete BRI data (n = 1766), or lacked information on key covariates, including smoking status (n = 9), alcohol consumption (n = 1011), educational background (n = 9), marital status (n = 3), and PA (n = 5). Ultimately, 14,254 individuals were enrolled (Figure 1).

COPD Outcomes

COPD was defined according to affirmative responses to any of the following self-reported questions: "Have you been diagnosed with emphysema?", "Have you been diagnosed with chronic bronchitis?", or "Has a doctor ever informed you that you have COPD?" Individuals who answered "no" to all three questions were considered non-COPD.

Calculation of Anthropometric Indices

BRI was treated as the independent variable. According to the BRI calculation formula proposed by Tomas et al,[10] data on WC and height were extracted from the anthropometric measurements in NHANES for the calculation.

$$BRI = 364.2 - 365.5 \times \sqrt{1 - \left(\frac{WC}{2\pi}\right)^2 / \left(\frac{Height}{2}\right)^2}$$

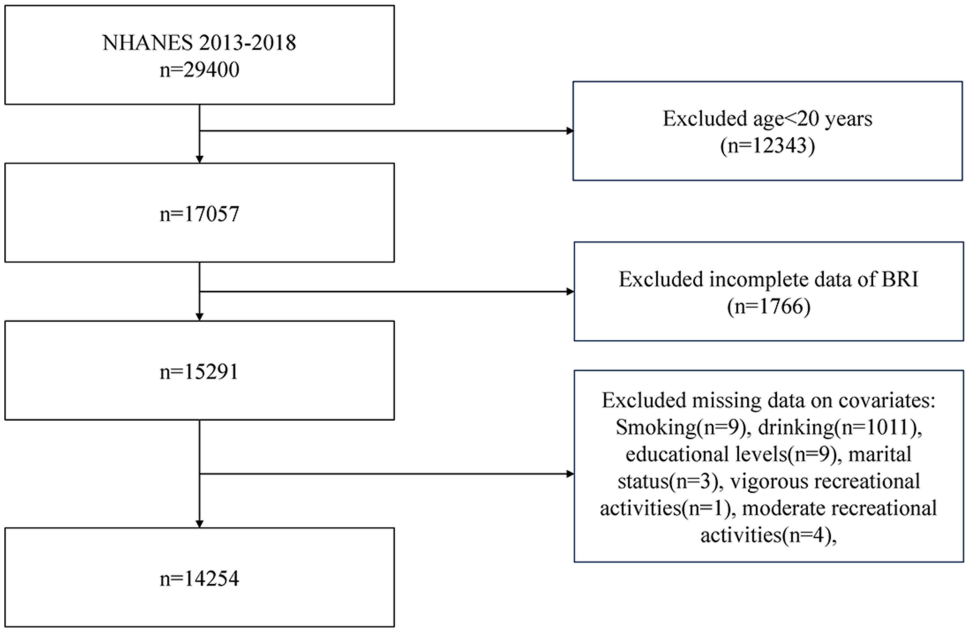

Figure 1 Flowchart of participant selection.

Assessment of Covariates

A wide range of lifestyle, demographic, and health-related variables were collected: alcohol consumption, poverty income ratio (PIR), sex, total cholesterol, educational background, total energy intake (TEI), race/ethnicity, high-density lipoprotein cholesterol (HDL), protein, total sugar, age, HP, marital status, height, carbohydrate, dietary fiber (DF), CVD, total fat, smoking status, vigorous and moderate recreational activities, weight, eosinophil count, diabetes, white blood cell count, monocyte count, WC, and BMI.

Smoking status was categorized as never smoked, current smoker, or former smoker based on whether the individuals had smoked more than 100 cigarettes over a lifetime and their duration since quitting. Individuals were defined as having diabetes according to the following criteria: self-reported physician diagnosis of diabetes, fasting blood glucose >126 mg/dL, administration of glucose-lowering medication or insulin, or glycated hemoglobin (HbA1c) ≥6.5%.[13] HP was defined according to the following criteria: self-reported HP, current use of antihypertensive medication, average systolic blood pressure ≥130 mmHg or average diastolic blood pressure ≥80 mmHg across three measurements.[14] CVD was defined if individuals reported the following physician diagnoses: coronary heart disease, CHF, heart attack, or stroke.[13]

Statistical Analysis

Following NHANES methodological guidelines, this study applied the recommended multistage probability sampling weights to account for the complex survey design during statistical analyses. BRI was divided into four quartiles based on its distribution: Q1 (1.167–3.965), Q2 (3.965–5.282), Q3 (5.282–6.940), and Q4 (6.940–23.482). The Kolmogorov–Smirnov test was used to assess the normality of continuous variables. Given that these continuous variables were non-normally distributed, they were expressed as medians and interquartile ranges, and between-group comparisons were conducted using the Kruskal–Wallis test. Categorical variables were expressed as frequencies and percentages, and between-group comparisons were made using weighted Chi-square tests. Multicollinearity among covariates was examined, and variables with a variance inflation factor (VIF) ≥ 5 were excluded. Weighted logistic regression (WLR) models were employed to investigate the association between BRI and COPD prevalence. Three models were constructed: Model 1 was unadjusted; Model 2 was adjusted for race, sex, and age; Model 3 was further adjusted for educational background, marital status, PIR, TEI, DF, smoking status, alcohol consumption, moderate PA, diabetes, HP, CVD,[15] direct HDL cholesterol, and eosinophil percentage. Restricted cubic spline (RCS) analysis with five knots was employed to investigate the dose-response relationship between BRI and COPD prevalence. Subgroup analyses stratified

by sex, smoking status (never, current, former), alcohol consumption, diabetes, HP, and CVD were conducted to test the robustness of the findings. Statistical significance was defined as two-sided p-values less than 0.05. R version 4.4.2 was used to conduct statistical analysis.

Result

Baseline Characteristics of Study Population

Table 1 illustrates the basic characteristics of the included 14,254 individuals (stratified by BRI quartiles). The overall prevalence of COPD was 8.3%, corresponding to a weighted estimate of 210,015,248 individuals. The median age of individuals was 48 years, with 49% men and 51% women. BRI was divided into four quartiles: Q1 (1.167–3.965), Q2 (3.965–5.282), Q3 (5.282–6.940), and Q4 (6.940–23.482).

Table 1 Baseline Characteristics of Participants

Characteristic	N[a]	Overall	Q1	Q2	Q3	Q4	p-value[c]
		N = 210,015,248[b]	N = 56,552,863[b]	N = 53,641,568[b]	N = 49,902,520[b]	N = 49,918,298[b]	
Age	14,254	48(33,61)	36(26,52)	48(36,61)	53(39,65)	53(38,64)	<0.001
Gender	14,254						<0.001
Male		6980(49%)	1918(51%)	2011(56%)	1816(52%)	1235(36%)	
Female		7274(51%)	1646(49%)	1552(44%)	1747(48%)	2329(64%)	
Races	14,254						<0.001
Mexican American		2114(8.8%)	261(5.2%)	498(8.4%)	691(11%)	664(11%)	
Other Hispanic		1493(6.2%)	279(5.4%)	383(6.6%)	444(7.1%)	387(5.6%)	
Non-Hispanic White		5399(65%)	1338(66%)	1327(65%)	1312(64%)	1422(66%)	
Non-Hispanic Black		3047(11%)	843(12%)	665(9.0%)	690(9.8%)	849(13%)	
Other Race		2201(8.9%)	843(11%)	690(11%)	426(7.7%)	242(5.2%)	
Educational levels	14,254						<0.001
Less than 9th grade		1225(4.2%)	131(2.0%)	311(4.5%)	404(5.7%)	379(4.9%)	
9–11th grade		1722(8.6%)	398(7.7%)	419(8.5%)	451(8.7%)	454(9.6%)	
High school graduate		3264(23%)	748(20%)	783(22%)	837(25%)	896(27%)	
Some college or AA degree		4478(32%)	1071(29%)	1037(31%)	1125(32%)	1245(37%)	
College graduate or above		3565(31%)	1216(41%)	1013(34%)	746(28%)	590(21%)	
Marital status	14,254						<0.001
Married		7284(55%)	1583(48%)	2000(60%)	1949(59%)	1752(53%)	
Widowed		989(5.4%)	120(2.4%)	223(4.8%)	281(6.1%)	365(8.7%)	
Divorced		1601(10%)	318(7.6%)	379(11%)	437(11%)	467(12%)	
Separated		479(2.4%)	96(1.9%)	116(2.4%)	139(2.5%)	128(3.0%)	
Never married		2657(18%)	1076(30%)	523(14%)	479(13%)	579(16%)	
Living with a partner		1244(8.6%)	371(10%)	322(8.6%)	278(7.9%)	273(7.7%)	

(*Continued*)

Table 1 (Continued).

Characteristic	N[a]	Overall	Q1	Q2	Q3	Q4	p-value[c]
		N = 210,015,248[b]	N = 56,552,863[b]	N = 53,641,568[b]	N = 49,902,520[b]	N = 49,918,298[b]	
PIR	14,254	2.95(1.48,5.00)	3.23(1.56,5.00)	3.27(1.59,5.00)	2.90(1.48,5.00)	2.43(1.28,4.32)	<0.001
Energy	14,254	2014(1510,2672)	2071(1566,2780)	2050(1528,2652)	2006(1496,2653)	1933(1447,2583)	<0.001
Protein	14,254	76(54,104)	80(56,109)	77(55,105)	75(54,103)	73(53,99)	<0.001
Carbohydrate	14,254	231(167,312)	240(176,325)	229(166,311)	229(164,311)	224(161,300)	<0.001
Total sugars	14,254	93(57,140)	96(61,145)	94(56,142)	92(58,138)	91(55,135)	0.004
Dietary fiber	14,254	15(10,22)	16(11,24)	15(10,22)	15(10,22)	14(9,20)	<0.001
Total fat	14,254	78(54,110)	79(55,111)	78(55,107)	79(54,110)	78(54,110)	0.796
Smoking	14,254						<0.001
Never		8114(57%)	2131(61%)	2013(56%)	1980(54%)	1990(53%)	
Current		2763(18%)	862(21%)	695(19%)	603(16%)	603(16%)	
Former		3377(25%)	571(18%)	855(24%)	980(29%)	971(30%)	
Alcohol consumption	14,254						<0.001
No		3285(18%)	716(15%)	761(16%)	833(18%)	975(22%)	
Yes		10,969(82%)	2848(85%)	2802(84%)	2730(82%)	2589(78%)	
Vigorous recreational activities	14,254						<0.001
No		10,830(72%)	2154(56%)	2636(70%)	2911(80%)	3129(87%)	
Yes		3424(28%)	1410(44%)	927(30%)	652(20%)	435(13%)	
Moderate recreational activities	14,254						<0.001
No		8294(53%)	1781(44%)	2020(50%)	2135(56%)	2358(63%)	
Yes		5960(47%)	1783(56%)	1543(50%)	1428(44%)	1206(37%)	
Diabetes	14,254						<0.001
No		11,551(86%)	3379(97%)	3069(91%)	2775(82%)	2328(70%)	
Yes		2703(14%)	185(3.2%)	494(8.9%)	788(18%)	1236(30%)	
Hypertension	14,254						<0.001
No		6699(52%)	2448(73%)	1750(54%)	1407(44%)	1094(33%)	
Yes		7555(48%)	1116(27%)	1813(46%)	2156(56%)	2470(67%)	
CVD	14,254						<0.001
No		12,853(92%)	3398(97%)	3252(93%)	3159(91%)	3044(87%)	
Yes		1401(7.9%)	166(3.3%)	311(7.0%)	404(9.3%)	520(13%)	
HDL	14,254	51(42,63)	60(48,72)	52(43,64)	48(40,59)	47(40,56)	<0.001
Total Cholesterol	14,254	187(162,215)	178(156,204)	193(167,221)	192(166,219)	187(162,214)	<0.001
White blood cell count	14,254	7.10(5.80,8.50)	6.40(5.30,7.80)	6.90(5.70,8.30)	7.30(6.00,8.60)	7.90(6.60,9.40)	<0.001
Eosinophils percent	14,254	2.30(1.50,3.50)	2.10(1.30,3.40)	2.30(1.50,3.50)	2.30(1.50,3.50)	2.40(1.60,3.60)	<0.001

(*Continued*)

Table 1 (Continued).

Characteristic	N[a]	Overall	Q1	Q2	Q3	Q4	p-value[c]
		N = 210,015,248[b]	N = 56,552,863[b]	N = 53,641,568[b]	N = 49,902,520[b]	N = 49,918,298[b]	
Lymphocyte percent	14,254	30(25,35)	31(26,37)	30(25,36)	30(25,35)	29(23,34)	<0.001
Monocyte percent	14,254	8.00(6.80,9.40)	8.10(6.90,9.60)	8.10(6.80,9.40)	8.10(6.90,9.40)	7.50(6.40,9.00)	<0.001
Weight	14,254	80(68,96)	65(57,73)	77(68,87)	87(76,98)	104(90,120)	<0.001
Height	14,254	168(161,176)	170(163,177)	169(162,177)	168(161,176)	165(158,172)	<0.001
BMI	14,254	28(24,33)	23(21,24)	27(25,29)	31(29,33)	37(34,42)	<0.001
WC	14,254	99(88,111)	82(77,87)	95(91,100)	105(100,110)	120(113,129)	<0.001
COPD	14,254						<0.001
No		13,022(92%)	3,376(95%)	3,316(93%)	3,271(92%)	3,059(86%)	
Yes		1,232(8.3%)	188(5.0%)	247(7.1%)	292(8.1%)	505(14%)	
BRI	14,254	5.14(3.85,6.81)	3.12(2.61,3.56)	4.61(4.29,4.94)	6.00(5.64,6.43)	8.38(7.54,9.84)	<0.001

Notes: [a]N not Missing (unweighted); [b]Median (Q1, Q3); n (unweighted)(%); [c]Design-based KruskalWallis test; Pearson's X^2: Rao & Scott adjustment.
Abbrevations: BMI, body mass index; PIR, poverty income ratio; TC, total cholesterol; HDL, high-density lipoprotein cholesterol; CVD, cardiovascular disease; WC, waist circumference; COPD, chronic obstructive pulmonary disease; BRI, body roundness index.

The highest BRI quartile had a greater proportion of women and Mexican Americans in comparison to the lowest BRI quartile. Moreover, higher BRI levels were associated with lower engagement in vigorous PA, older age, and being widowed. Moreover, the highest BRI group exhibited a greater prevalence of CVD and diabetes.

Associations Between BRI and the Likelihood of COPD

The association between BRI and COPD prevalence was examined using WLR models (Table 2). For continuous BRI, the results indicated that BRI was positively associated with COPD prevalence. In Model 1 (unadjusted), each unit increase in BRI was associated with a 16.8% increase in the likelihood of COPD ($P < 0.001$). In model 2 (adjusted for race/ethnicity, sex, and age), this association remained stable (OR = 1.141, 95% CI: 1.106–1.176, $P < 0.001$). In model 3 (further adjusted for comorbidities, dietary intake, and laboratory indicators), the association persisted, although slightly attenuated (OR = 1.085; 95% CI: 1.036–1.136; P = 0.002).

Table 2 WLR Analysis of BRI and COPD

	Model 1			Model 2			Model 3		
	OR	95% CI	P	OR	95% CI	P	OR	95% CI	P
BRI (continuous)	1.168	1.136,1.202	<0.001	1.141	1.106,1.176	<0.001	1.085	1.036,1.136	0.002
BRI (quartiles)									
1.167–3.965	Ref			Ref			Ref		
3.965–5.282	1.442	1.082,1.921	0.014	1.155	0.867,1.540	0.315	1.036	0.758,1.414	0.814
5.282–6.940	1.671	1.290,2.164	<0.001	1.251	0.959,1.631	0.096	1.02	0.756,1.376	0.890
6.940–23.482	2.972	2.384,3.705	<0.001	2.162	1.728,2.706	<0.001	1.466	1.091,1.969	0.015
P for trend			<0.001			<0.001			<0.001

Note: Model 1: uncontrolled; Model 2: controlled for race/ethnicity, sex, age; Model 3: further controlled for educational background, marital status, PIR, TEI, DF, SS, AC, moderate PA, diabetes, HP, CVD, HDL cholesterol, and eosinophil percentage.

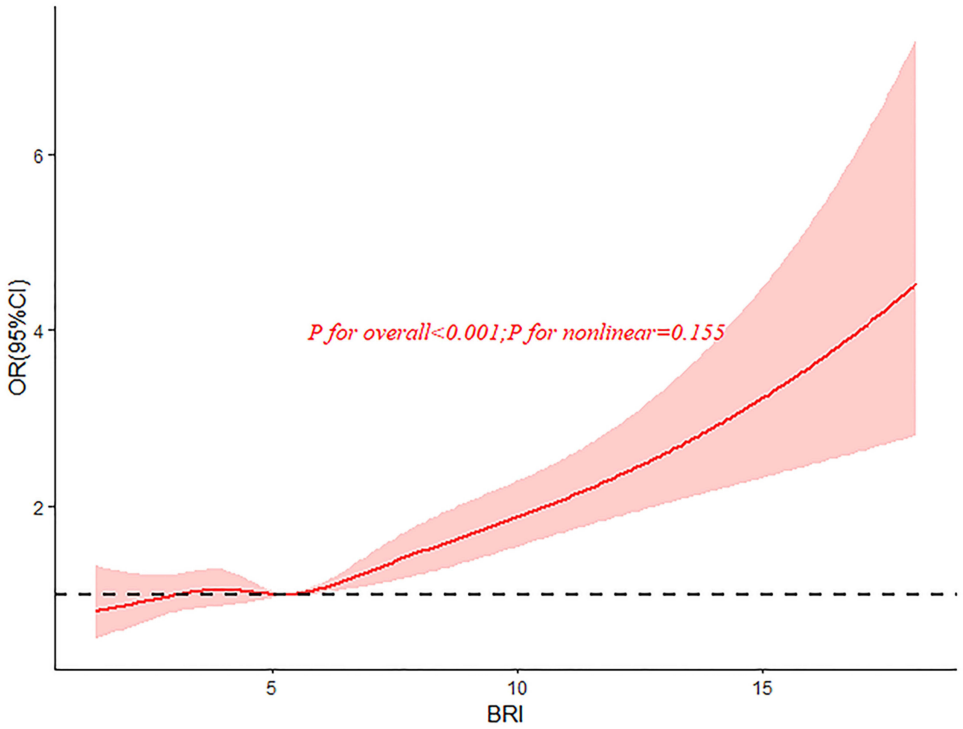

Figure 2 The association between BRI and the likelihood of developing COPD through RCS analysis. The RCS curve was adjusted for race/ethnicity, sex, PIR, educational background, marital status, age, TEI, DF, smoking status, alcohol consumption, diabetes, HP, moderate PA, direct HDL cholesterol, and eosinophil percentage.

For categorical BRI, Model 1 demonstrated that individuals in the highest BRI quartile had a 2.972-fold higher likelihood of COPD in comparison to those in the lowest quartile ($P < 0.001$). This association remained significant in Model 2 (OR = 2.162; 95% CI: 1.728–2.706; $P < 0.001$) and Model 3 (OR = 1.466; 95% CI: 1.091–1.969; P = 0.015).

RCS Analysis

RCS analysis examined the dose-response relationship between BRI and the likelihood of developing COPD. The results revealed an approximately linear positive association between BRI and the likelihood of developing COPD (P for nonlinearity = 0.155) (Figure 2).

Subgroup Analysis

The robustness of the association between BRI and COPD prevalence was assessed by subgroup analyses based on sex, smoking status, alcohol consumption, age, diabetes, HP, and CVD. The results demonstrated a significant association between BRI and the likelihood of developing COPD in all subgroups except for former smokers and individuals with CVD. In addition, interaction tests revealed substantial differences in COPD prevalence across subgroups stratified by sex, HP, and CVD (Figure 3).

Discussion

This cross-sectional study, which involved 14,254 individuals, suggested that higher BRI levels were positively associated with COPD prevalence in the US adult population. This association was confirmed to be linear based on RCS analysis. Although no threshold effect was detected in the RCS analysis, it was observed that when BRI exceeded 5.332, COPD prevalence increased significantly with rising BRI. Subgroup analyses further supported the robustness of these findings. Interaction tests indicated that sex, HP, and CVD significantly modified this association. These results suggest that maintaining an appropriate BRI level is particularly important for COPD prevention. Moreover, BRI, due to its cost-effectiveness and efficiency, may hold substantial value in large-scale COPD screening and risk stratification.

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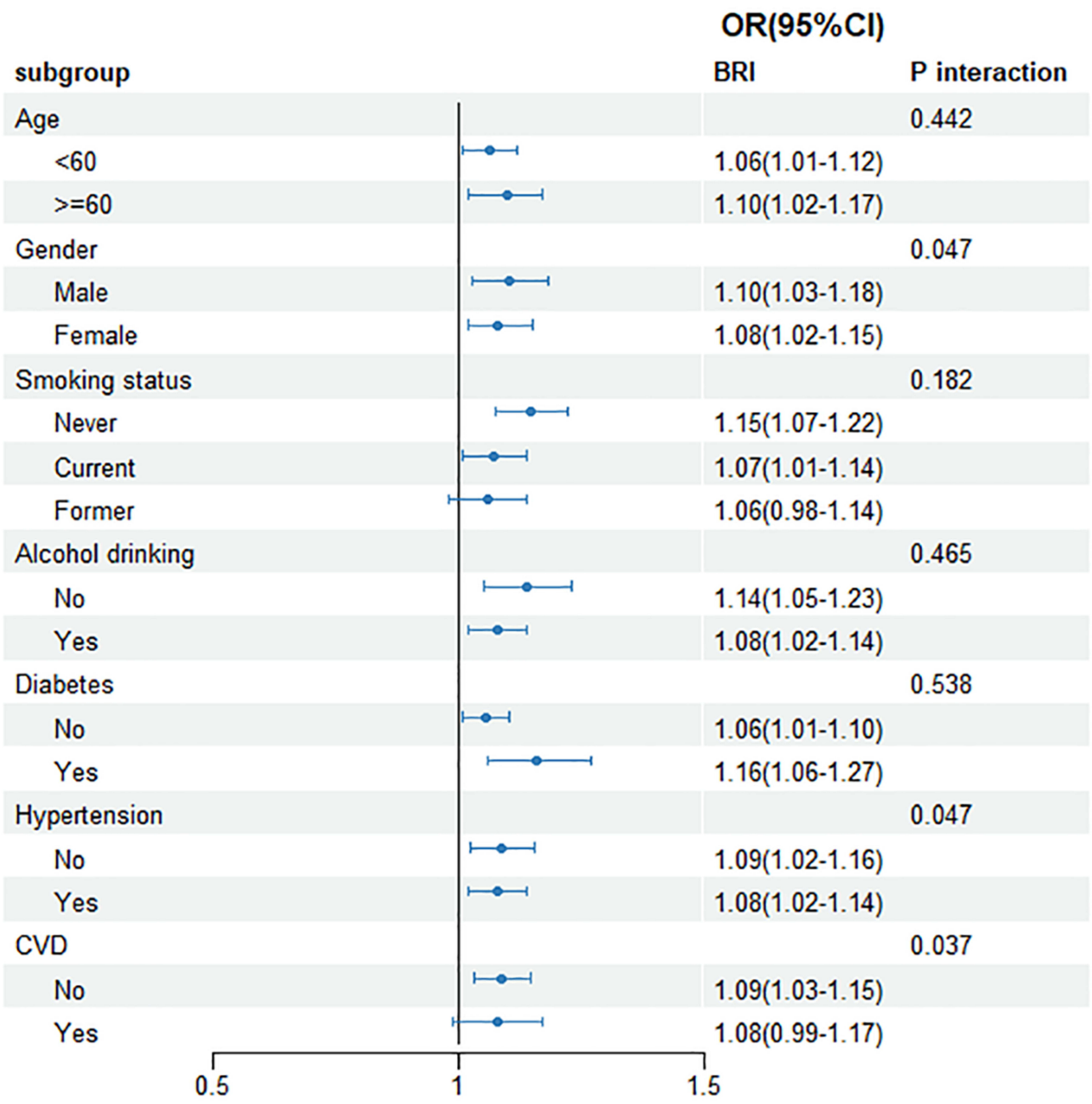

Figure 3 The association between BRI and COPD prevalence through subgroup analyses.

Obesity, as a major contributor to the risk of COPD, has garnered increasing research interest. Prior studies mainly employed BMI to quantify obesity, but BMI cannot differentiate between fat mass (FM) and fat-free mass (FFM). Since FFM and FM exert distinct effects on pulmonary physiology, Zhang et al highlighted the necessity of investigating the association between fat distribution and COPD risk.[16] Although WC reflects abdominal fat accumulation (AFA), it does not distinguish between subcutaneous and visceral fat. The A Body Shape Index may outperform BMI and WC in predicting ACM, but is less reliable for predicting chronic diseases.[17–19] In contrast, BRI, which integrates WC and height into a cylindrical model, more accurately estimates abdominal obesity.[20–22] Current studies have demonstrated relationships between BRI and obstructive respiratory diseases. BRI is a superior predictor of obstructive sleep apnea.[23] Xu et al demonstrated that both weight-adjusted waist index (WWI) and BRI are independent factors influencing asthma risk, with BRI exhibiting better predictive performance than WWI.[24] These conclusions support the utility of BRI in

early COPD detection and risk stratification. Therefore, timely identification and intervention in individuals with elevated BRI may help slow disease progression and improve their quality of life.

Several mechanisms may underlie the association between BRI and COPD. Individuals with high BRI tend to have significant AFA, which increases the COPD risk. This finding has been supported by previous research.[25,26] Lam et al reported that central obesity is associated with both obstructive and restrictive ventilatory impairments in COPD.[26] This may be attributed to obesity-induced systemic inflammation, which can impair the normal structure and function of lung tissue.[27] Abnormal accumulation of visceral adipose tissue (VAT) can cause adipocyte hypoxia by impairing ventilation and reducing tissue oxygenation,[7,15] ultimately triggering inflammatory responses. The activation of immune cells (such as neutrophils, macrophages, and eosinophils) contributes to pulmonary inflammation.[28,29] Furthermore, VAT is recognized as an active endocrine organ.[28] In individuals with obesity, VAT homeostasis is disrupted, thereby leading to abnormal secretion of adipokines. This includes elevated levels of pro-inflammatory mediators such as leptin, IL-6, and TNF-α, and decreased levels of anti-inflammatory factors such as adiponectin. These imbalances can further stimulate the NOD-like receptor family pyrin domain-containing 3 inflammasome and IL-1β signaling,[28,30,31] potentially accelerating airway remodeling, impairing lung function, and ultimately contributing to COPD progression.

Furthermore, elevated levels of chronic low-grade inflammatory mediators related to obesity, such as TNF-α, leptin, and IL-6 secreted by adipose tissue, are associated with neutrophil-mediated OS responses.[9] Neutrophil activation contributes to the depletion of systemic antioxidants by releasing reactive oxygen species (ROS),[32] thereby reducing pulmonary defense capacity. Moreover, it can exacerbate damage to the alveolar wall by releasing proteases. In addition, ROS can activate inflammation-related signaling pathways,[32,33] alter the extracellular matrix, and stimulate goblet cell activation, thereby resulting in mucus hypersecretion and subsequent airway obstruction. Current evidence demonstrates that neutrophil elastases damage elastic fibers and mucociliary structures in the lungs, ultimately impairing mucus clearance and promoting goblet cell metaplasia and mucin production.[9] These effects further reduce pulmonary compliance and increase airway resistance, thereby elevating COPD risk.

Moreover, inadequate PA also contributes to COPD progression among individuals with obesity. In individuals with COPD, obesity may further impair ventilation through such mechanical constraints as limited diaphragmatic movement and increased chest wall resistance.[7,34,35] Additionally, these constraints can elevate the risk of comorbid conditions, including metabolic syndrome and CVD, thus indirectly accelerating disease deterioration.[15,30]

According to subgroup analyses, no significant association between BRI and COPD was observed in participants with CVD or in former smokers, suggesting that the generalizability of BRI may vary across populations. The lack of a significant association in the CVD subgroup may be attributable to residual confounding from unmeasured factors, such as medication interventions or complications, or to potential reverse causality.[36] After smoking cessation, systemic inflammation persisted in patients with COPD, whereas it declined in healthy former smokers.[37] This difference may affect the BRI–COPD relationship through pathways involving fat and lung function. Existing research reveals that although smoking cessation is frequently accompanied by weight gain, it does not lead to an elevated risk of COPD.[38] These findings warrant further investigation in prospective cohort studies. In addition, sex, HP, and CVD significantly modified the association between BRI and the odds of having COPD. The association between obesity and CVD may be mediated by shared intermediate metabolic risk factors, including impaired glucose tolerance, insulin resistance, HP, and hypertriglyceridemia, all of which contribute to adverse cardiovascular events.[39,40] In the CVD subgroup, several plasma markers are linked to obesity.[41] For example, abnormally low natriuretic peptide levels are associated with an increase in total white adipose tissue mass.[42] These factors may partly disrupt the BRI–COPD relationship, distinguishing this subgroup from individuals without CVD. A U.S.-based study suggests that men are more prone to exhibit AFA.[43] Abdominal fat, particularly VAT, increases COPD risk through metabolic and inflammatory mechanisms.[44]

This study has several strengths. First, the generalizability of our results was enhanced by incorporating a nationally representative sample and applying a complex multistage probability sampling design. Second, this study systematically analyzed the association between BRI and COPD and adjusted for multiple covariates to minimize confounding. Moreover, regression and subgroup analyses further reinforced the reliability of the observed associations.

Nevertheless, some limitations should be acknowledged. First, a causal relationship between BRI and COPD cannot be established because of the cross-sectional design. Second, since the data were derived from a US population, the

generalizability of the findings is uncertain and warrants further validation. Third, compared with standardized diagnoses based on lung function tests, self-reported diagnoses may substantially underestimate the true prevalence of COPD, resulting in underdiagnosis. Consequently, self-report diagnosis is an imprecise tool for identifying COPD patients,[45] potentially affecting the interpretation of the findings. Hence, these findings should be confirmed by prospective cohort studies.

Conclusion

This study observed a positive linear association between BRI and the likelihood of developing COPD. This result suggests that BRI could serve as a potential marker for assessing the likelihood of COPD and may help reduce the cost of early COPD screening. In addition, BRI has been shown to be more accurate and sensitive than BMI and WC. Therefore, these findings hold significant implications for clinical practice and epidemiological research. Future studies should further investigate whether BRI-based interventions can improve clinical outcomes in individuals with COPD, as well as explore the potential threshold value of BRI.

Data Sharing Statement

The datasets analysed during the current study are available in the National Center for Health Statistics (NCHS), https://www.cdc.gov/nchs/nhanes/about/index.html.

Ethics Approval and Informed Consent

All NHANES protocols obtained approval from the Ethics Review Board of NCHS. Written informed consent was offered by enrolled individuals.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.\.

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Disclosure

The authors declare that they have no competing interests.

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