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ORIGINAL RESEARCH

Weight-Adjusted Waist Index as a Novel Predictor of Chronic Obstructive Pulmonary Disease: Evidence from NHANES 2013-2018

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Background: Chronic obstructive pulmonary disease (COPD) is a major global health burden. The weight-adjusted waist index (WWI), a novel adiposity metric, may improve COPD risk prediction, but its association remains underexplored.

Methods: Using the National Health and Nutrition Examination Survey (NHANES) 2013–2018 data (n=15,278), we assessed the WWI-COPD relationship via multivariable logistic regression, ROC analysis, and subgroup evaluations.

Results: Higher WWI tertiles correlated with elevated COPD incidence. After full adjustment, each WWI unit increase linked to 70% higher COPD risk (OR=1.70, 95% CI: 1.48–1.95). Participants in the highest quartile of WWI faced a 290% increased risk compared to the lowest quartile (OR=3.90, 95% CI: 2.60–5.86). WWI (AUC=0.707) outperformed BMI (AUC=0.525) and waist circumference (AUC=0.609) in COPD prediction. A nonlinear threshold effect emerged at WWI=12.54. Subgroup analyses confirmed robustness across demographics.

Conclusion: WWI is a simple, cost-effective tool for early COPD detection, outperforming BMI and waist circumference, especially in resource-limited settings, enabling timely intervention and reducing disease burden.

Keywords: weight-adjusted-waist index, chronic obstructive pulmonary disease, obesity, NHANES, cross-sectional study

Introduction

Chronic obstructive pulmonary disease (COPD) is one of the leading causes of death and disability worldwide and became the third leading cause of death globally in 2019.[1] COPD is a lung disease characterized by persistent airflow limitation, which is usually progressive and not fully reversible, and its etiology may involve interactions between genetic and environmental factors.[2] Smoking is traditionally an environmental risk factor. However, research has suggested that non-tobacco-related factors are responsible for approximately half of COPD cases worldwide. Recent studies have identified the following factors: air pollution,[3] occupational exposure,[4] asthma, lung function, low socio-economic status, and impaired lung development. Early onset of COPD may lead to a worse prognosis, and its worsening course is inevitable; identifying more possible risk factors and earlier diagnosis can help prevent and slow its progression.[5,6]

In recent years, the impact of obesity on COPD has received increasing attention from researchers. Some studies have shown that overweight or obese patients with chronic diseases have a better prognosis, while obesity may be protective for patients with severe COPD, and weight loss may increase the risk of death in patients with COPD.[7] However, this may reflect an "obesity paradox" rooted in the limitations of conventional metrics like body mass index (BMI) and waist circumference (WC). Critically, BMI and WC fail to differentiate between fat and muscle mass or account for fat distribution patterns, leading to inconsistent associations with COPD outcomes.[8] For instance, while high BMI may

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correlate with survival benefits in COPD patients, this paradox likely arises from BMI's inability to distinguish protective muscle mass from harmful visceral adiposity.[9] Similarly, WC neglects intramuscular and subcutaneous fat variations, potentially misclassifying individuals with similar waist measurements but divergent metabolic risks.[8] These shortcomings underscore the need for obesity indices that better reflect body composition dynamics.

Currently, the weight-adjusted waist circumference index (WWI) has emerged as a new tool for assessing obesity. By calculating the ratio of body weight to WC, the WWI provides a more accurate assessment.[10] The advantage of the WWI is that it provides a more comprehensive assessment of an individual's adiposity and lean body mass beyond the limitations of BMI and WC. As such, it has demonstrated potential predictive value in a variety of disease areas, including periodontitis, peripheral artery disease and erectile dysfunction.[11–13] Although previous studies have demonstrated associations between WWI and a variety of adverse health outcomes, the association between WWI and COPD has not been well studied.

Therefore, our study analysed the correlation between the WWI and COPD incidence using National Health and Nutrition Examination Survey (NHANES) data from 2013 to 2018, aiming to provide new perspectives and evidence for COPD risk prediction and early intervention.

Methods

Study Population

This study used data collected from the NHANES, a cross-sectional survey conducted by the National Center for Health Statistics (NCHS), to evaluate the health and nutritional status of the US population. The NHANES gathers information through interviews and physical examinations. The NHANES study design employed a stratified multistage probability sampling method, contributing to the high representativeness of the included samples. All data used in the analysis are publicly available at https://www.cdc.gov/nchs/nhanes/.

The survey was conducted over three cycles over six years (2013–2014, 2015–2016, and 2017–2018). Among 29,400 participants, 12,363 with no COPD data and 1759 with missing waist circumference or weight data were excluded. Ultimately, the study included 15,278 participants (Figure 1).

The NCHS Ethical Review Board approved the conduct and study protocol of the NHANES in human subjects. Written informed consent was obtained from all participants.

Weight-Adjusted Waist Index

Physical waist circumference and weight measurements were performed under controlled conditions at a mobile testing center (MEC) by trained health technicians. The decimal rounding of the WWI results for each participant was kept to two decimal places. In our analysis, the WWI was considered a continuous variable, and participants were categorized into groups based on their WWI data for additional study. The following method was used to calculate the WWI:

$$\mathrm{WWI}(\mathrm{cm}/\sqrt{\mathrm{kg}}) = \mathrm{WC}/\sqrt{\mathrm{Weight}}$$

The Definition of COPD

Whether a participant had COPD was included in this study as the exposure variable. The values of this variable were extracted from NHANES questionnaire data. In the Medical Conditions section (MCQ160o), investigators asked participants whether a doctor had informed them that they had COPD. Participants who answered "yes" were included in the COPD group, while those who answered "no" were included in the control group. Participants who had missing data for this question were excluded from this study.

Covariables

The covariates included in the analysis were selected based on their established or plausible biological and epidemiological relevance to both obesity and COPD. Demographic variables (age, sex, race) were adjusted due to their known associations with body composition and COPD risk.[8] Socioeconomic factors (education level, marital status, PIR) were included to account for health disparities and access to care.[14] Behavioral factors (smoking status, alcohol consumption)

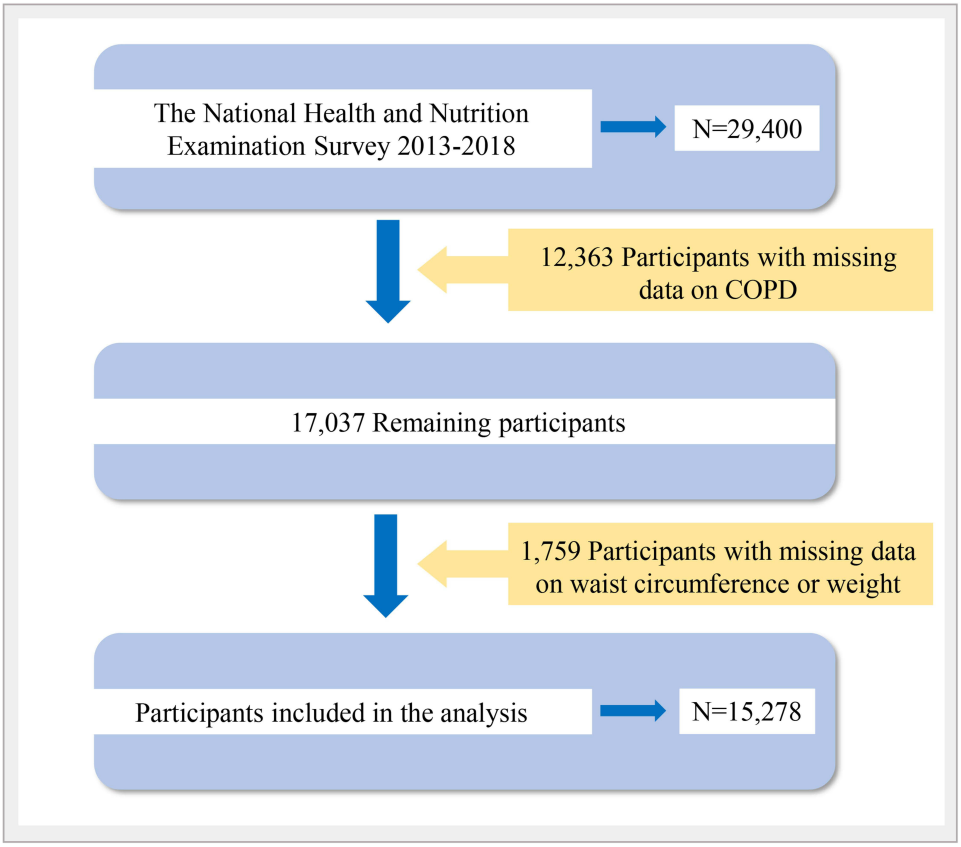

Figure 1 Flow chart of participant selection.
Abbreviations: COPD, chronic obstructive pulmonary disease; Blue background, participant included; yellow background, participant excluded.

were adjusted as smoking is a primary COPD risk factor, and alcohol intake may influence metabolic health.[15] Comorbidities (hypertension, diabetes, CVD) were included due to their shared inflammatory pathways with COPD.[16,17] Laboratory variables (TG, HDL-C, total cholesterol) reflect metabolic profiles that may mediate obesity-related pulmonary dysfunction.[18] This comprehensive adjustment minimizes confounding and isolates the independent effect of WWI on COPD.

Demographic Variables

The NHANES demographic files contain information on individuals' age, sex, race, family income-to-poverty ratio, education level, and marital status at the interview.

Body Mass Index

Data on the BMI of the study participants were gathered from NHANES body measurements collected by trained health technicians.

Smoking Status

Smoking status was constructed from responses to the questionnaire section: "Have you smoked at least 100 cigarettes in your entire life?". Smokers were defined as those who had smoked 100 or more cigarettes, whereas never smokers were defined as those who had never smoked 100 cigarettes.

Alcohol Consumption

Alcohol consumption was constructed from responses to the questionnaire section: "Have you drunk at least 12 drinks in your entire life?". Drinkers were those who consumed more than 12 drinks in their lifetime, and never drinkers were those who consumed fewer than 12 drinks in their lifetime.

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Hypertension

Hypertension in the NHANES was defined as self-reported hypertension (answer "yes" to the question "Have you ever been told by a doctor or other health professional that you had hypertension, also called high blood pressure?") or elevated blood pressure during physical examination (mean systolic blood pressure ≥140 mm Hg or mean diastolic blood pressure ≥90 mm Hg).

Diabetes

A subject meeting any of the following criteria will be diagnosed with diabetes: 1) Doctor told you that you have diabetes; 2) glycohemoglobin HbA1c (%) >6.5; 3) fasting glucose (mmol/L) ≥7.0; 4) random blood glucose (mmol/L) ≥11.1; 5) 2-hour oral glucose tolerance test (OGTT) blood glucose (mmol/L) ≥11.1; and 6) use of antidiabetic agents.

Cardiovascular Disease

Participants who had previously been diagnosed with congestive heart failure, coronary heart disease, angina, or heart attack were referred to the CVD group.

Laboratory Variables

Triglyceride (TG), high-density lipoprotein (HDL-C), and total cholesterol levels were obtained from the NHANES laboratory data files.

Statistical Analysis

Given the complex sampling design of the NHANES data, all experiments were adjusted for weighted variables and survey design.[19] This included the integration of sample weights, clustering, and stratification to guarantee the precision and dependability of the findings. Analysis was conducted using complex weighting, with sample weights was obtained by multiplying the WTSAF2YR by 3.

The mean ± standard deviation (SD) was used for continuous variables, and categorical variables are expressed as proportions. Continuous variables were analysed using t tests, and categorical variables were analysed using chi-square tests to determine significant differences. Linear associations between the WWI, waist circumference, BMI, and COPD incidence were analysed using weighted multiple linear regression and logistic regression. A trend test was conducted to assess the nonlinear association trend between the WWI and COPD incidence by transforming the WWI from a continuous variable into quartiles. Additionally, smoothed curve fitting and threshold effects were employed to evaluate the potential nonlinear associations between the WWI, WC, BMI, and COPD incidence. The area under the receiver operating characteristic curve (ROC) was adopted to measure the ability of the WWI, WC, and BMI to predict COPD. In this study, three models were employed for analysis. Model 1 did not include any adjustment variables. Model 2 was adjusted for gender, age, and race. Finally, Model 3 was adjusted for gender, age, race, education level, marital status, PIR, alcohol consumption, hypertension, CVD, triglycerides, HDL-C, diabetes status, smoking status, and total cholesterol.

Subgroup analyses were conducted to explore the associations between the WWI and COPD among individuals of different genders, ages, races, smoking statuses, diabetes statuses, and hypertension statuses. Gender accounts for sex-specific differences in fat distribution and COPD susceptibility.[8] Age group reflect metabolic shifts (eg, sarcopenia) across life stages.[20] Smoking status was included due to its dual role as a primary COPD risk factor and potential interaction with obesity-related inflammation.[15] Statistical analyses were carried out using R (version 4.2) and Empower software (version 4.2), which are statistical computing and plotting software. A two-sided p value of less than 0.05 was considered to indicate statistical significance in this study.

Results

Participant Characteristics

This study identified 15,278 individuals in the NHANES 2013–2018, of which 567 individuals suffered from COPD. Compared to non-COPD participants, COPD participants were more prone to be older, male, non-Hispanic White, less than high school, and living alone and were more likely to smoke and have hypertension, CVD, and diabetes.

Individuals with COPD had significantly greater BMIs and TG levels than individuals without COPD. In addition, significant differences in PIR, alcohol consumption, HDL-C, total cholesterol, and WWI were detected between the two groups ($P < 0.05$). The detailed characteristics of the included participants with COPD are shown in Table 1.

Table 1 Characteristics of the Study Participants: NHANES 2013–2018

	Overall n = 15278	Non-COPD n = 14711	COPD n = 567	P value
Gender				0.014
Male	7390 (48.37%)	7087 (48.17%)	303 (53.44%)	
Female	7888 (51.63%)	7624 (51.83%)	264 (46.56%)	
Race				<0.001
Mexican American	2248 (14.71%)	2223 (15.11%)	25 (4.41%)	
Other Hispanic	1619 (10.60%)	1594 (10.84%)	25 (4.41%)	
Non-Hispanic White	5633 (36.87%)	5261 (35.76%)	372 (65.61%)	
Non-Hispanic black	3287 (21.51%)	3193 (21.70%)	94 (16.58%)	
Other race	2491 (16.30%)	2440 (16.59%)	51 (8.99%)	
Education level				<0.001
Less than high school	3246 (21.25%)	3082 (20.95%)	164 (28.92%)	
High school or GED	3460 (22.65%)	3292 (22.38%)	168 (29.63%)	
Above high school	8561 (56.03%)	8326 (56.60%)	235 (41.45%)	
Unclear	11 (0.07%)	11 (0.07%)	0 (0.00%)	
Marital status				<0.001
Married or living with partner	9175 (60.05%)	8904 (60.53%)	271 (47.80%)	
Living alone	6096 (39.90%)	5801 (39.43%)	295 (52.03%)	
Unclear	7 (0.05%)	6 (0.04%)	1 (0.18%)	
Smoking status				<0.001
Yes	2954 (19.33%)	2694 (18.31%)	260 (45.86%)	
No	12324 (80.67%)	12,017 (81.69%)	307 (54.14%)	
Alcohol consumption				0.025
>12	6834 (44.73%)	6577 (44.71%)	257 (45.33%)	
<12	7429 (48.63%)	7141 (48.54%)	288 (50.79%)	
Unclear	1015 (6.64%)	993 (6.75%)	22 (3.88%)	
Hypertension				<0.001
Yes	6611 (43.27%)	6212 (42.23%)	399 (70.37%)	
No	8667 (56.73%)	8499 (57.77%)	168 (29.63%)	
CVD				<0.001
Yes	1588 (10.39%)	1340 (9.11%)	248 (43.74%)	
No	13688 (89.59%)	13,369 (90.88%)	319 (56.26%)	
Unclear	2 (0.01%)	2 (0.01%)	0 (0.00%)	
Diabetes				<0.001
Yes	3064 (20.05%)	2860 (19.44%)	204 (35.98%)	
No	12214 (79.95%)	11,851 (80.56%)	363 (64.02%)	
Age (years)	49.52 ± 17.44	48.96 ± 17.38	64.12 ± 11.78	<0.001
PIR	2.51 ± 1.54	2.54 ± 1.54	1.85 ± 1.29	<0.001
BMI (kg/m2)	29.38 ± 7.01	29.35 ± 6.95	30.30 ± 8.38	0.045
TG (mg/dL)	114.12 ± 74.44	113.90 ± 74.96	119.92 ± 59.02	0.002
HDL-C (mg/dL)	53.40 ± 16.10	53.45 ± 16.00	52.28 ± 18.50	0.006
Total cholesterol (mg/dL)	189.53 ± 40.67	189.84 ± 40.63	181.38 ± 40.89	<0.001
WWI (cm/√kg)	11.12 ± 0.86	11.10 ± 0.85	11.71 ± 0.77	<0.001

Notes: Mean ± SD for continuous variables: the P value was calculated by the weighted linear regression model; (%) for categorical variables: the P value was calculated by the weighted chi-square test.

Abbreviations: CVD, cardiovascular disease; PIR, the ratio of income to poverty; BMI, body mass index; TG, triglyceride; HDL-C, high-density lipoprotein; WWI, weight-adjusted waist index; COPD, chronic obstructive pulmonary disease.

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Association Between Weight-Adjusted Waist Index and COPD Incidence

In this study, the unadjusted (model 1) linear association analysis demonstrated a positive correlation between the WWI and COPD incidence, yielding an odds ratio (OR) of 2.34 (95% CI: 2.11, 2.59). This association was also consistently observed in models 2 and 3. According to the fully adjusted model (Model 3), which accounted for all covariates, the association between the WWI and COPD incidence remained consistent, with an OR of 1.70 (95% CI: 1.48, 1.95). This suggests that each unit increase in the WWI is associated with a 70% increase in the likelihood of developing COPD (Table 2). The WWI was more strongly associated with COPD than BMI (OR=1.03, 95% CI: 1.02–1.05) and waist circumference (OR=1.02, 95% CI: 1.01–1.03) (Table 3). In addition, we obtained reliable findings even after dividing the continuous variables into quartiles. The WWI was 290% greater in Q4 than in Q1 (OR=3.90, 95% CI: 2.60, 5.86) (Table 2). Furthermore, exploration through the GAM and smoothing curves revealed a nonlinear positive association between the WWI or WC and COPD (Figure 2A–C). In addition, a nonlinear association was found between BMI and COPD (Figure 2D). Further exploration of the association between the WWI and COPD revealed that the cut-off point was 12.5. A significant correlation was found before the turning point (OR=1.88, 95% CI: 1.60–2.22) (Table 4). The AUCs of the WWI, WC, and BMI for discriminating COPD patients are shown in Figure 3. The WWI had the highest AUC of 0.707, followed by WC (AUC=0.609) and BMI (AUC=0.525).

Table 2 Association of the WWI with COPD

Exposure	OR (95% CI)		
	Model 1 (n = 15278)	**Model 2 (n = 15278)**	**Model 3 (n = 15,278)**
WWI (cm/√kg)	2.34 (2.11, 2.59)	1.93 (1.70, 2.18)	1.70 (1.48, 1.95)
WWI quartile			
Quartile 1	Reference	Reference	Reference
Quartile 2	2.62 (1.78, 3.86)	1.91 (1.29, 2.84)	1.94 (1.29, 2.92)
Quartile 3	3.88 (2.68, 5.62)	2.33 (1.58, 3.44)	2.06 (1.38, 3.10)
Quartile 4	9.02 (6.37, 12.79)	4.67 (3.20, 6.83)	3.90 (2.60, 5.86)
P for trend	< 0.001	< 0.001	< 0.001

Notes: Model 1: No covariates were adjusted. Model 2: Gender, age, and race were adjusted. Model 3: Gender, age, race, education level, marital status, PIR, hypertension, alcohol consumption, CVD, TG, HDL-C, diabetes, smoking status, and total cholesterol were adjusted.
Abbreviations: CVD, cardiovascular disease; PIR, the ratio of income to poverty; TG, triglyceride; HDL-C, high-density lipoprotein; WWI, weight-adjusted waist index; COPD, chronic obstructive pulmonary disease.

Table 3 Associations of BMI and WC with COPD

Exposure	OR (95% CI)		
	Model 1 (n = 15278)	**Model 2 (n = 15278)**	**Model 3 (n = 15,278)**
BMI (kg/m2)	1.02 (1.01, 1.03)	1.03 (1.02, 1.04)	1.03 (1.02, 1.05)
WC (cm)	1.02 (1.02, 1.03)	1.02 (1.01, 1.02)	1.02 (1.01, 1.03)

Notes: Model 1: No covariates were adjusted. Model 2: Gender, age, and race were adjusted. Model 3: Gender, age, race, education level, marital status, PIR, hypertension, alcohol consumption, CVD, TG, HDL-C, diabetes, smoking status, and total cholesterol were adjusted.
Abbreviations: CVD, cardiovascular disease; PIR, the ratio of income to poverty; TG, triglyceride; HDL-C, high-density lipoprotein; BMI, body mass index; WC, waist circumference; COPD, chronic obstructive pulmonary disease.

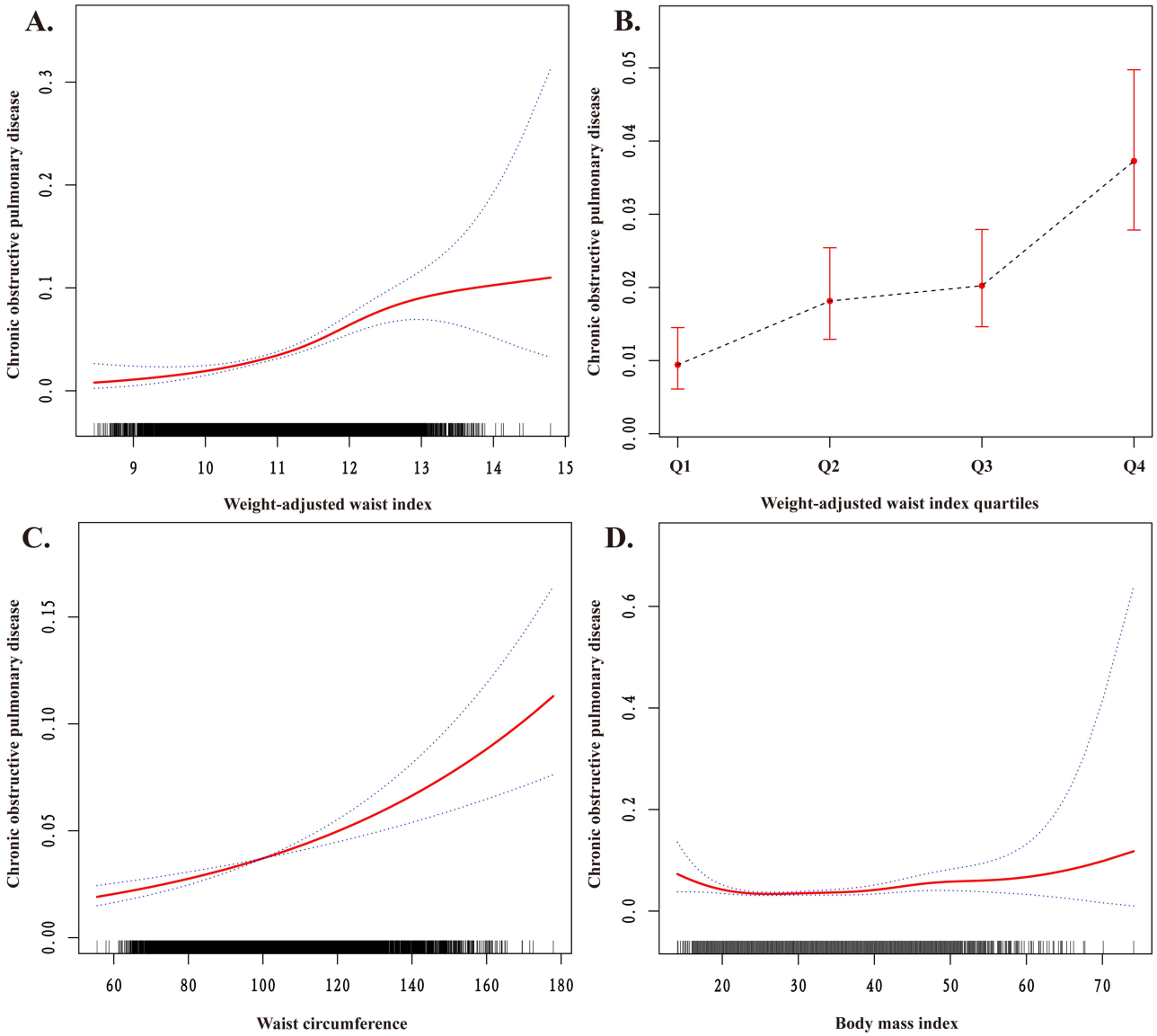

Figure 2 (**A**) Association between the weight-adjusted waist index and COPD incidence. (**B**) Association between weight-adjusted waist index quartiles and COPD incidence. (**C**) Association between waist circumference and COPD incidence. (**D**) Association between body mass index and COPD incidence.
Abbreviation: COPD, chronic obstructive pulmonary disease.

Subgroup Analysis

To assess the reliability of the association between the WWI and COPD incidence, we conducted subgroup analyses based on gender, age, race, hypertension status, diabetes status, and smoking status. The results showed that all subgroups exhibited a positive association between the WWI and the risk of COPD (Figure 4). WWI was more positively associated with COPD in males (OR =2.29, 95% CI: 1.85–2.84, P <0.001), those aged 20–39 years (OR =3.07, 95% CI: 1.47–6.42, P = 0.003), Mexican Americans (OR =3.24, 95% CI: 1.73–6.07, P <0.001), those in the non-hypertensive group (OR =1.70, 95% CI: 1.34–2.17, P <0.001), those in the diabetic group (OR =1.81, 95% CI: 1.41–2.31, P <0.001), and those in the smoking group (OR =1.76, 95% CI: 1.42–2.19, P <0.001).

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Table 4 Threshold Effect Analysis of the WWI on COPD Using a Two-Piecewise Linear Regression Model

WWI	COPD
	Adjusted β (95% CI) P value
Fitting by the standard linear model	1.70 (1.48, 1.95) <0.0001
Fitting by the two-piecewise linear model	
Inflection point	12.54
<K segment effect	1.88 (1.60, 2.22) <0.0001
>K segment effect	0.83 (0.44, 1.56) 0.5608
Log likelihood ratio	0.017

Notes: Gender, age, race, education level, marital status, PIR, hypertension, alcohol consumption, CVD, TG, HDL-C, diabetes, smoking status, and total cholesterol were adjusted.
Abbreviations: CVD, cardiovascular disease; PIR, the ratio of income to poverty; TG, triglyceride; HDL-C, high-density lipoprotein; WWI, weight-adjusted waist index; COPD, chronic obstructive pulmonary disease.

Sensitivity Analysis

In sensitivity analysis, the relationship between WWI and COPD was consistently observed after the exclusion of individuals with asthma, chronic bronchitis or emphysema, respectively. After adjusting for potential confounders and excluding individuals with asthma, chronic bronchitis or emphysema, the ORs were 1.98 (95% CI, 1.64–2.38; P < 0.001), 1.74 (95% CI, 1.45–2.09; P < 0.001), and 1.67 (95% CI, 1.42–1.96; P < 0.001), respectively (Table 5).

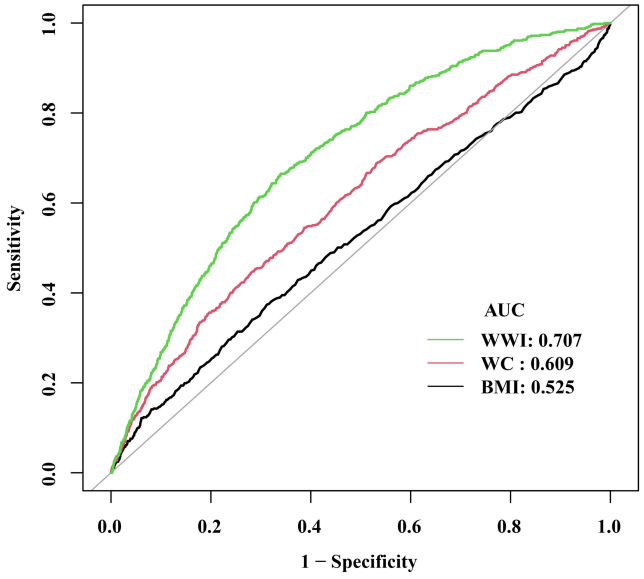

Figure 3 ROC curves for the ability of the WWI, BMI, and WC to predict COPD. AUC values range from 0.5 (no discrimination) to 1.0 (perfect discrimination). WWI's AUC of 0.707 indicates moderate-to-strong predictive ability, outperforming BMI (AUC=0.525) and WC (AUC=0.609).
Abbreviations: WWI, weight-adjusted waist index; BMI, body mass index; WC, waist circumference; COPD, chronic obstructive pulmonary disease; AUC: area Under the Curve.

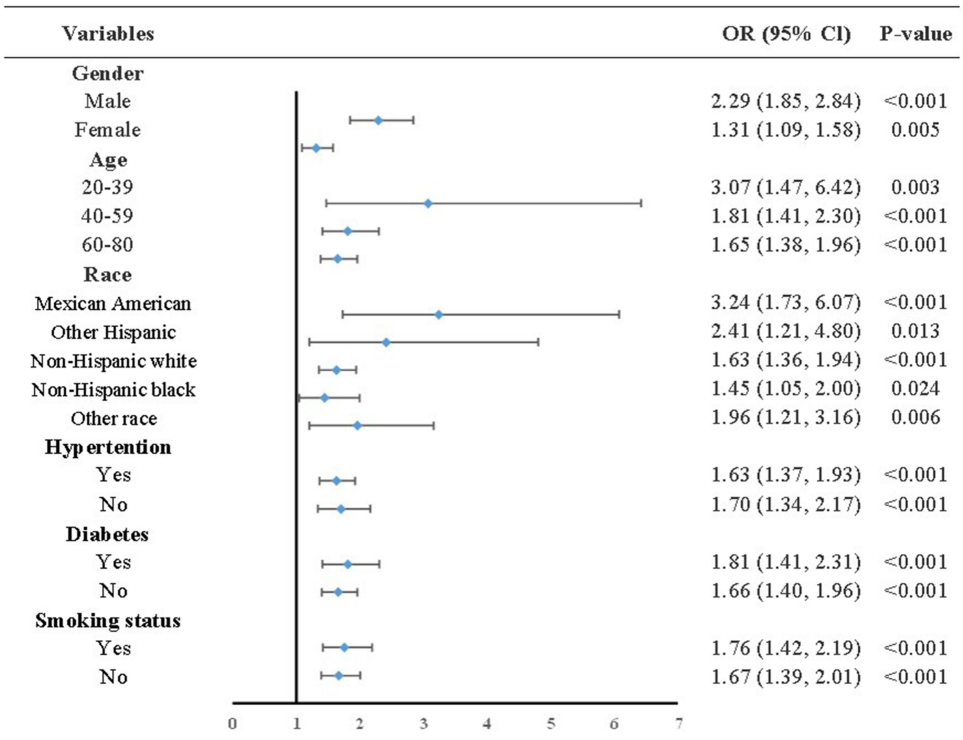

Figure 4 Subgroup analyses. In the subgroup analysis stratified by gender, age, race, hypertension status, diabetes status, and smoking status, the model was not adjusted for gender, age, race, hypertension status, diabetes status, or smoking status.

Discussion

This cross-sectional study utilized NHANES data from 2013 to 2018 involving 15,278 participants to explore the potential association between the WWI and COPD incidence. To our knowledge, this is the first investigation to assess the association between the WWI and COPD. The findings revealed a positive association between the WWI and COPD

Table 5 Sensitivity Analyses

Exposure	OR (95% CI)		
	Model 1	Model 2	Model 3
Excluding individuals with chronic bronchitis			
WWI (cm/√kg)	2.39 (2.09, 2.73)	1.98 (1.68, 2.33)	1.74 (1.45, 2.09)
WWI quartile			
Quartile 1	Reference	Reference	Reference
Quartile 2	2.85 (1.64, 4.96)	1.97 (1.12, 3.48)	2.09 (1.17, 3.74)
Quartile 3	5.04 (2.98, 8.50)	2.82 (1.63, 4.87)	2.51 (1.43, 4.41)
Quartile 4	10.71 (6.49, 17.66)	5.13 (2.99, 8.79)	4.40 (2.49, 7.76)
P for trend	< 0.001	< 0.001	< 0.001
Excluding individuals with emphysema			
WWI (cm/√kg)	2.35 (2.08, 2.65)	1.95 (1.68, 2.26)	1.67 (1.42, 1.96)
WWI quartile			
Quartile 1	Reference	Reference	Reference
Quartile 2	2.44 (1.51, 3.93)	1.87 (1.15, 3.03)	1.92 (1.17, 3.15)
Quartile 3	3.86 (2.46, 6.07)	2.46 (1.53, 3.94)	2.09 (1.28, 3.41)
Quartile 4	9.07 (5.93, 13.87)	4.96 (3.13, 7.86)	3.86 (2.37, 6.28)
P for trend	< 0.001	< 0.001	< 0.001

(*Continued*)

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Table 5 (Continued).

Exposure	OR (95% CI)		
	Model 1	Model 2	Model 3
Excluding individuals with asthma			
WWI (cm/√kg)	2.59 (2.26, 2.97)	2.23 (1.88, 2.64)	1.98 (1.64, 2.38)
WWI quartile			
Quartile 1	Reference	Reference	Reference
Quartile 2	2.62 (1.44, 4.76)	1.93 (1.05, 3.53)	2.03 (1.09, 3.78)
Quartile 3	5.65 (3.25, 9.82)	3.44 (1.94, 6.10)	3.06 (1.69, 5.52)
Quartile 4	12.49 (7.36, 21.21)	6.51 (3.69, 11.46)	5.49 (3.03, 9.94)
P for trend	< 0.001	< 0.001	< 0.001

Notes: Model 1: No covariates were adjusted. Model 2: Gender, age, and race were adjusted. Model 3: Gender, age, race, education level, marital status, PIR, hypertension, alcohol consumption, CVD, TG, HDL-C, diabetes, smoking status, and total cholesterol were adjusted.

Abbreviations: CVD, cardiovascular disease; PIR, the ratio of income to poverty; TG, triglyceride; HDL-C, high-density lipoprotein; WWI, weight-adjusted waist index; COPD, chronic obstructive pulmonary disease.

among US adults, which remained significant after adjusting for various confounders. Specifically, for every one-unit increase in the WWI, there was a 70% increase in the incidence of COPD. Notably, this association was stronger than those observed for traditional metrics such as BMI and WC. Subgroup analyses demonstrated the stability of the association between the WWI and COPD across different demographic and health-related variables. This association was not affected by age, gender, race, hypertension status, diabetes status, or smoking status. COPD has a low rate of early diagnosis and irreversible pathogenesis, so exploring the risk factors for its onset is essential for effective prevention and treatment strategies.[21] Our findings suggest that the WWI has significant clinical value in identifying COPD and facilitating early disease recognition.

Previous research has highlighted the significant role of obesity in COPD development, with BMI and WC commonly used as indicators. However, the association between BMI and COPD has been the subject of inconsistent studies by various scholars, some of whom have even produced conflicting results. Some researchers have found that low BMI may be a risk factor for the development of COPD and that a decrease in BMI may be strongly associated with worsening COPD and increased mortality.[22,23] Additionally, large-scale prospective studies have indicated that low BMI and weight loss may independently predict mortality in COPD patients, particularly in individuals over 20 years old with substantial weight loss or a BMI <18.5 kg/m2.[24] Poul et al reported that being overweight (BMI >25 kg/m2) may be a predictor of long-term survival in COPD patients.[25] Although many studies suggest a protective effect of high BMI on COPD, this notion may represent an "obesity paradox". Notably, increased WC was positively linked to COPD risk in a decade-long cohort study involving both genders.[26] This result was confirmed in another study, which concluded that abdominal obesity and underweight may be risk factors for COPD in adults.[27] Subgroup analysis revealed that the risk of developing COPD was lowest in women with BMIs ranging from 23–24 kg/m2, and BMI and COPD exhibited a U-shaped relationship, consistent with our findings. It has been reported that under extreme conditions, such as a BMI >40 kg/m2, obesity leading to low mortality no longer occurs, and we can treat weight loss as a deleterious factor rather than obesity as a protective factor.[7,27,28]

However, the focus of most studies on diseases has been limited to BMI, WC, and WHR. These indicators primarily reflect nutritional status, and their utility as obesity indicators is questioned because they overlook the distribution of fat, muscle, and other tissues, thus failing to adequately represent the proportion of muscle to fat. An investigation into obesity and COPD compared the correlation between various measures, including BMI, WC, and WHR, revealing that reliance on a single measure overlooks crucial factors such as body composition distribution, resulting in an underestimation of disease risk.[8]

In recent years, researchers have increasingly focused on parameters related to fat and muscle, such as the fat mass index (FMI), fat-free mass index (FFMI), and other parameters. Both the FMI and FFMI have been implicated in the

severity of emphysema in COPD patients, with the FFMI specifically impacting quality of life and potentially influencing mortality risk.[29,30] In a prospective study, muscle loss and malnutrition during hospitalization in patients with COPD were analysed. During the 6-month follow-up, patients who experienced muscle loss due to malnutrition, along with sarcopenia, had a lower one-year survival rate.[31] According to a related report, patients with COPD may exhibit intramuscular and intermuscular fat infiltration in the abdomen and intercostal space. This fat infiltration can lead to impaired muscle function and atrophy, potentially exacerbating the severity of COPD.[32,33] However, less muscle may also be associated with carbonylation of proteins, which can be caused by ROS and other related oxidative substances, and carbonylation leads to greater structural and functional negative effects.[34] Weight loss and muscle atrophy are prevalent among COPD patients and are linked to systemic inflammatory responses. A study revealed elevated serum levels of growth hormone-releasing peptide and lipocalin in COPD patients with low body weight, while BMI and forced expiratory volume in 1 second (FEV1) were negatively correlated with serum growth hormone-releasing peptide levels.[35] As more studies highlight the role of muscle and weight loss in disease progression, indicators of muscle measurements are becoming increasingly important. However, there have been no major breakthroughs in muscle measurement techniques, and to date, there is no universally recognized method for measuring muscle.

The WWI is an index that evaluates both fat and muscle, and it has been reported that the WWI increases linearly with age and that abdominal fat measurements increase, but muscle measurements decrease.[36,37] As research on WWI has progressed, an increasing body of evidence has demonstrated its reliability as an indicator capable of predicting various diseases. In the field of cardiovascular diseases, the WWI has emerged as a distinctive predictor of hypertension, stroke, and abdominal aortic calcification.[38–40] An elevated WWI may also be a risk factor for the development of renal stones and gallstones.[41,42] Furthermore, research has demonstrated that the WWI is the most accurate predictor of chronic kidney disease and albuminuria among various obesity indicators. Regarding respiratory diseases, ongoing studies have concentrated on the relationship between asthma and the WWI. Cross-sectional investigations have revealed a positive correlation between the WWI and both the prevalence and timing of initial asthma episodes. Additionally, a prolonged duration of asthma may coincide with weight loss and abdominal obesity.[43,44] While research examining the correlation between the WWI and various diseases is expanding, there remains a notable absence of studies addressing COPD, warranting increased attention. First, our findings revealed a robust correlation between elevated WWI and COPD onset, which was particularly notable among individuals aged 20–39 years. Second, in comparison to the cumbersome process of measuring visceral fat, which demands expensive equipment such as MRI scanners and substantial social resources, WWI has emerged as a more accessible and cost-effective obesity index that is especially beneficial for less developed regions. Moreover, Nam's[37] findings suggest that the WWI is positively correlated with obesity and negatively correlated with human muscle mass. Additionally, these correlations are not significantly influenced by factors such as changes in BMI. This implies that the WWI could be a valuable metric in clinical practice for assessing the risk of COPD, as it appears to be less influenced by BMI and other factors and may offer a better reflection of body visceral fat and muscle mass. This could provide clinicians with a more accurate assessment of health risks associated with COPD.[37,45] Third, our findings indicate a threshold effect in the relationship between the WWI and COPD, meaning that this association reaches its peak when the WWI exceeds 12.54 cm/√kg. Beyond this threshold, further increases in the WWI do not lead to a corresponding increase in COPD incidence. This evidence underscores the potential benefit of controlling lower levels of WWI as a preventive measure to mitigate the risk of COPD. Such insights may aid clinicians in devising tailored prevention and treatment protocols for patients as well as in identifying high-risk cohorts for targeted screening initiatives.

Our study has several strengths. First, we pioneered the examination of the correlation between WWI and COPD incidence, utilizing a highly representative sample from the NHANES. This highlights that an elevated WWI may contribute to increased COPD risk, offering insights into potential strategies for COPD prevention and early diagnosis. Second, this study adjusted for covariates that may confound or mediate the WWI-COPD relationship. For instance, smoking status was included to disentangle the direct effects of WWI from tobacco-related lung damage. Similarly, hypertension and diabetes were adjusted to account for systemic inflammation and metabolic dysregulation that may exacerbate COPD progression. Socioeconomic variables (education, PIR) were incorporated to address disparities in healthcare access and lifestyle factors. By rigorously controlling these variables, our findings highlight WWI as an

independent predictor of COPD, robust to confounding from overlapping pathways. Lastly, large-scale cross-sectional studies such as ours employ random sampling methods, rendering their findings potentially representative of the broader population. While NHANES represents US adults, the biological plausibility of the WWI adiposity-muscle balance mechanism suggests potential applicability to other populations. Future multinational cohorts are needed to validate cross-cultural relevance.

Nevertheless, our study is not without limitations. First, cross-sectional survey results can only imply correlation rather than causation. Second, the diagnosis of COPD was based on self-reports, which are susceptible to recall bias and limit the ability to assess disease severity. Conversely, diagnosing COPD using a post-bronchodilation forced expiratory volume in one second (FEV1)/forced vital capacity (FVC) ratio below 70% provides a more objective assessment. Finally, the sparse data in higher WWI quartiles (Q4) may inflate odds ratios due to limited sample size in extreme categories, as noted in similar studies.[46] Therefore, caution should be exercised when interpreting estimates from extreme quartiles.

In conclusion, WWI offers a cost-effective, technically simple metric for COPD risk stratification. Its integration into routine clinical assessments—using waist and weight measurements—can prioritize high-risk individuals (WWI ≥12.54) for targeted spirometry, enhancing early detection. Community-based screening in resource-limited settings could leverage WWI alongside basic metabolic profiling, bypassing costly infrastructure. Public health campaigns promoting WWI self-monitoring via digital tools may empower at-risk populations. Combining WWI with spirometry (eg, FEV1/FVC) or inflammatory markers could refine diagnostics, while policy advocacy for its inclusion in global guidelines would standardize protocols, positioning WWI as a scalable tool to reduce COPD burden through early intervention.

Conclusion

This study demonstrates that the weight-adjusted waist index (WWI) outperforms traditional obesity metrics (BMI, waist circumference) in predicting COPD risk. Clinically, WWI could serve as a cost-effective, first-line screening tool to prioritize high-risk individuals (WWI ≥12.54) for spirometry, particularly in resource-limited settings. Future validation using spirometry-confirmed cases and cross-population studies will strengthen its utility. Integrating WWI into global guidelines may enhance early COPD detection and reduce disease burden through targeted interventions.

Data Sharing Statement

All the data utilized in this study were obtained and analysed through the NHANES database. The codes employed in the analysis are available upon reasonable request by contacting the authors.

Ethics Statement

According to Article 32, items 1 and 2 of the Measures for Ethical Review of Life Sciences and Medical Research Involving Human Subjects (issued by the National Health Commission, the Ministry of Education, the Ministry of Science and Technology, and the State Administration of Traditional Chinese Medicine on February 18, 2023), research using publicly available data obtained legally or data generated through observation without interfering with public behavior, as well as research using anonymized information data, is exempt from ethical review. This study is based on the publicly available database of the NHANES, and all data have been approved by the Ethics Review Committee of the NCHS and have completed the informed consent process for participants. The data involved are fully anonymized and do not contain sensitive personal information or commercial interests, which meets the conditions for exemption under the above regulations. Therefore, this study does not need to be submitted separately to the institutional ethics review committee for approval.

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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 no conflicts of interest in this work.

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