BMC Pulmonary Medicine

RESEARCH

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Association between lipid accumulation product and chronic obstructive pulmonary disease: a cross-sectional analysis

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Abstract

Background Research indicates that obesity can worsen the clinical manifestations of chronic obstructive pulmonary disease (COPD). Timely detection of COPD has the potential to enhance treatment results. This study seeks to investigate the association between a new metabolic indicator, the lipid accumulation product (LAP), and the risk of developing COPD.

Methods The observational analysis employs data from the National Health and Nutrition Examination Survey (NHANES) conducted from 2007 to 2016. Multivariate logistic regression was used to explore the association between LAP levels and COPD. Further analysis methods included subgroup analysis, smooth curve modeling, and threshold effect evaluation.

Results Within the sample of 12,089 individuals, 1,072 were diagnosed with COPD. A positive correlation between LAP levels and COPD risk was identified through logistic regression analysis, even after controlling for potential confounders. The analyses by subgroup showed an enhanced association in participants without hypertension. The smooth curve fitting analysis highlighted particular saturation effects of LAP, with significant inflection points identified at 65.5278 and a *P*-value of 0.013.

Conclusion This study's findings suggest that elevated LAP levels are associated with an increased risk of COPD. It is suggested that dietary modifications and exercise routines be implemented to mitigate COPD risk in those with higher LAP levels.

Keywords Chronic obstructive pulmonary disease, Lipid accumulation product, The National Health and Nutrition Examination Survey

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Introduction

Chronic obstructive pulmonary disease (COPD) is a slowly progressive respiratory condition characterized by an obstructive ventilatory pattern that is often irreversible. It is frequently associated with tobacco smoking and can lead to chronic respiratory failure [1], which is marked by structural changes in the lungs, including chronic inflammation, narrowing of small airways, and destruction of alveolar walls. These changes contribute to air trapping and impaired gas exchange, resulting in progressive airflow limitation and hyperinflation [2, 3]. Globally, COPD ranks as the fourth leading cause of death and is projected to become the third leading cause by 2030 [4]. The disease has emerged as a significant public health issue, with rising prevalence, mortality rates, and economic burden, particularly in developing countries [5]. While cigarette smoking is the primary cause of COPD, nearly half of all cases worldwide are attributed to non-tobacco-related risk factors [6]. Research has indicated that the impacts of COPD extend beyond the lungs, with many patients experiencing systemic symptoms such as anemia, osteoporosis, and cardiovascular issues [7]. Furthermore, COPD is often accompanied by various comorbidities, including metabolic syndrome, cardiovascular disease, anxiety, depression, osteoporosis, anemia, diabetes mellitus, and obesity, which commonly occur alongside COPD [8, 9].

Recent studies on the prevalence of metabolic syndrome in patients with COPD have shown that a substantial number of individuals exhibit various metabolic abnormalities, including central obesity, diabetes mellitus, hypertension, and dyslipidemia [10]. Obesity, which is increasingly recognized as a global health concern, is considered a significant comorbidity [11]. Obese patients often experience numerous pulmonary complications [12, 13]. Several investigations have found that obese individuals with COPD report more respiratory symptoms, greater restrictions in daily activities, diminished health-related quality of life, and increased healthcare utilization [14–16]. Obesity, marked by the accumulation of visceral fat, traditional metrics such as BMI provide only a general assessment and do not distinguish between subcutaneous and visceral fat. The lipid accumulation product (LAP), introduced by Kahn in 2005, serves as a novel index for evaluating central lipid accumulation and is linked to metabolic and cardiovascular diseases [17]. LAP is a reliable and practical measure of excess central fat in adults, derived from two simple parameters: waist circumference (WC) and fasting triglyceride (TG) levels [18]. Furthermore, LAP is strongly associated with insulin resistance and correlates with a range of metabolic and cardiovascular risk factors [19].

The National Health and Nutrition Examination Survey (NHANES) is a well-organized, national clinical registry that provides continuous follow-up, leading it a valuable resource to examine the relationship between LAP and COPD. This study is the first clinical investigation to evaluate the correlation of LAP with COPD and the first to utilize data from the NHANES database. This study aims to explore the association between LAP and COPD, positing that LAP will have significant predictive value for the disease. By effectively leveraging the comprehensive NHANES database, this research intends to conduct an in-depth analysis of COPD through the framework of LAP, with the expectation that the findings will offer meaningful insights for clinical practice.

Materials and methods

Study population

This study utilized data from the NHANES database funded by the Centers for Disease Control and Prevention (CDC) and conducted every two years since 1999. Annually, approximately 5,000 individuals are surveyed in their homes, with physical examinations and biological sample collections performed in mobile units. NHANES encompasses participants from 15 urban areas across the USA, representing various ethnic backgrounds, including African, Asian, and Hispanic descent [20]. Details about the NHANES database can be accessed at http://www.cdc.gov/nhanes.

In this study, data from the NHANES database collected during the 2007-2016 survey cycle, comprising a total of 50,588 participants, were analyzed. After applying rigorous inclusion and exclusion criteria, a sample of 12,089 U.S. adults was ultimately selected. Specifically, 20,922 participants were excluded due to missing COPD data, 17,495 due to missing LAP data, and 82 individuals under the age of 20 (Fig. 1).

The protocol for NHANES was approved by the Institutional Review Board of the National Center for Health Statistics, with informed consent obtained from each participant and legal guardian consent provided for those under 18 years old [21].

Assessment of COPD

Participants with COPD was defined as those with FEV1 (forced expiratory volume in one second) / FVC (forced vital capacity) < 0.7 [22] after inhaling bronchodilators or those who answered "yes" to any of the following questions in their self-reports [23]: "Have doctors diagnosed you with chronic bronchitis?" "Have doctors diagnosed you with emphysema?" and "Have doctors or other health professionals diagnosed you with chronic obstructive pulmonary disease?" [24].

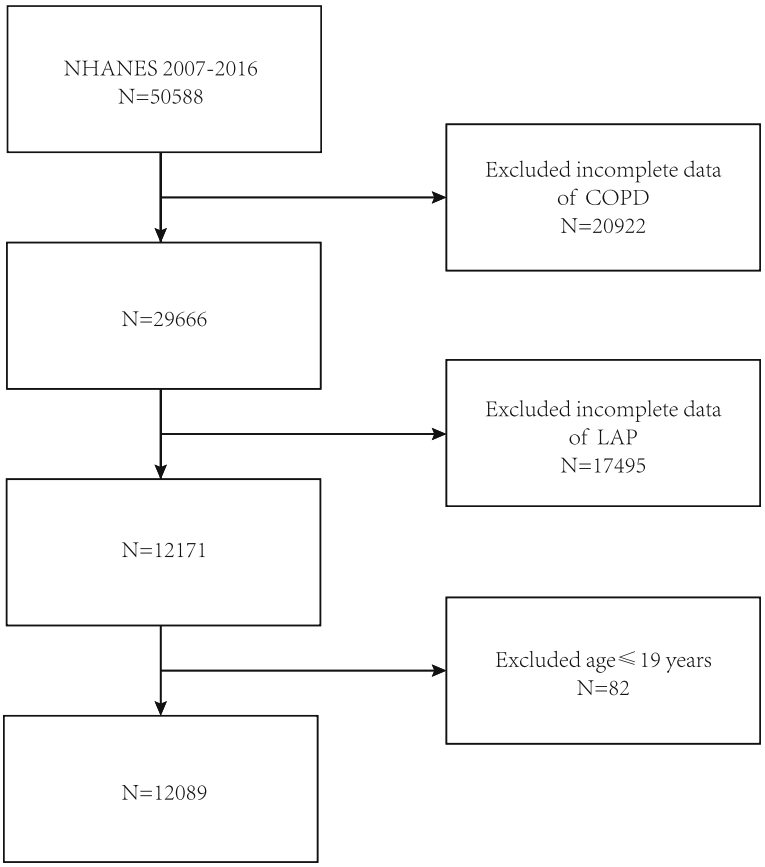

Fig. 1 Flow chart of patient screening. Abbreviations: NHANES, National Health and Nutrition Examination Survey: LAP: Lipid accumulation product; COPD, Chronic obstructive pulmonary disease

Assessment of LAP

LAP was calculated using gender-specific formulas developed by Kahn [18]. For males, the formula is: LAP = (WC - 65) * TG. For females, the formula is: LAP = (WC - 58) * TG, where WC represents waist circumference in centimeters and TG denotes triglycerides measured in mmol/L.

Measurement of covariates

Drawing from previous studies [25–27], several possible confounding factors affecting LAP and COPD were considered in the final analysis. These factors encompassed demographic variables such as age, gender, race, the income-to-poverty ratio (PIR), and educational status. Race was categorized into the following groups: Mexican American, Non-Hispanic Black, Non-Hispanic White, and other race. Educational attainment was classified into three levels: high school, above high school, and less than high school. The questionnaire data also collected information on diabetes, smoking status, hypertension, and alcohol consumption. Participants reported their medical history to indicate diagnoses of specific health conditions made by a doctor or other health professional. Smoking status was defined by whether participants had smoked 100 or more cigarettes in their lifetime. Alcohol consumption was assessed based on whether participants had consumed at least 12 alcoholic drinks in one year.

Statistical analysis

The LAP values were organized into quartiles (Q1 to Q4), progressing from the lowest to the highest range. The analysis expressed categorical variables in terms of proportions, while continuous variables were conveyed as means accompanied by standard errors (SE). Continuous variables are expressed as means ± standard deviations (SDs), and categorical variables are expressed as frequencies or percentages. For continuous variables, the *P* value was calculated by the linear regression model; for categorical variables, the *P* value was calculated by the chi-square test. Multiple logistic regression models were employed to calculate the odds ratios (ORs) and 95% confidence intervals (CIs) to assess the association between COPD and LAP. Three models were used in the

analysis: Model 1 (unadjusted), Model 2 (adjusted for age, race and gender), and Model 3 (fully adjusted for age, gender, race, income-to-poverty ratio, educational level, alcohol use, smoking status, hypertension, and diabetes). Subgroup analyses were conducted to identify potential modifying factors, stratifying by age, gender, education level, diabetes status, hypertension, smoking status, and alcohol consumption.

Furthermore, Generalized additive models (GAM) were applied for smooth curve fitting to explore the nonlinear relationship between LAP and COPD. A significance level of $P < 0.05$ was established to determine statistical significance. R (version 4.2.0) and EmpowerStats (version 4.2) were utilized for all statistical analyses. It is important to note that to minimize the impact of extreme values, the LAP value excluded extremes below the 1st percentile and above the 99th percentile during the multiple logistic regression models, subgroup analyses, threshold effect analysis and smooth curve fitting.

Results and analysis

Baseline profile of the study participants

In this study, 12,089 participants were enrolled. Table 1 provides a summary of the baseline characteristics of the participants. The individuals were divided into four quartiles based on average LAP values. Higher LAP quartiles exhibited a greater proportion of males and higher odds of COPD prevalence compared to the lower quartiles (quartile 1: 5.7247%; quartile 2: 5.7247%; quartile 3: 9.6262%; quartile 4: 12.6034%, $P < 0.001$). Significant differences were observed in LAP strata concerning age, race, education level, gender, smoking status, PIR, diabetes and hypertension (all $P < 0.05$). Participants in the higher LAP group were more likely to be male, Non-Hispanic White, and smokers, as well as to have lower income levels, lower educational attainment, and a higher likelihood of hypertension and diabetes compared to those in the lower LAP subgroup. No statistically significant differences were identified in alcohol consumption ($P > 0.05$).

Association between LAP and COPD

To further investigate the relationship between LAP and COPD, an evaluation of three multiple regression models was performed (Table 2). In the unmodified Model 2, logistic regression revealed a significant positive correlation between COPD and LAP. This association remained significant in the fully adjusted Model 3 (OR = 1.0024, 95% CI: 1.0009, 1.0040, $P = 0.002209$), implying that every one-unit increase in LAP levels leads to a 0.24% higher risk of COPD. For sensitivity analysis, LAP was categorized into quartiles. In Model 3, those in the highest LAP quartile showed a significantly higher risk of COPD than individuals in the lowest quartile, with an increase of 45.72% (OR = 1.4572, 95% CI: 1.1728, 1.8107, $P = 0.000679$).

Smooth curve fitting based on Model 3 was conducted to provide additional insight into the relationship between LAP and COPD. As shown in Fig. 2, the results indicate a nonlinear connection between LAP and COPD. Following this, an analysis of threshold effects was undertaken to clarify the nature of their association (Table 3). At a LAP inflection point of 65.5278 (log-likelihood ratio 0.013), each unit increase in LAP below this threshold was associated with a 0.76% higher COPD risk. However, once LAP levels exceeded 65.5278, the correlation with COPD risk weakened, demonstrating that additional LAP increases did not significantly influence COPD risk, underscoring a threshold-dependent impact of LAP on COPD risk.

Analysis by subgroups

Multiple stratified analyses and interaction tests were performed to verify the stability of the association between COPD and LAP, as well as to identify potential differences among populations (Table 4). Subgroup results showed a reliable association between LAP and COPD across various groups. Interestingly, a notable interaction was found between LAP and hypertension (interaction $P < 0.05$). In those without hypertension, a strong positive correlation was found (OR = 1.0048, 95% CI: 1.0025, 1.0071, $P < 0.0001$), while this relationship was not significant for individuals with diabetes. This suggests that individuals without hypertension may face a higher risk of developing COPD.

Strengths and limitations

This study is the first to evaluate the relationship between the LAP and the odds of COPD prevalence. The strengths of this study lie in several aspects. First, it explores the relationship between LAP and COPD. As a novel metabolic marker, LAP is more closely associated with insulin resistance and metabolic diseases compared to traditional indicators. Second, the study uses a large dataset from the 10-year NHANES survey, which enhances the representativeness and robustness of the findings. Third, by controlling for multiple confounders and conducting subgroup, threshold effect, and smooth curve fitting analyses, the study deepens and strengthens the credibility of the results. Lastly, LAP is easy to measure and has potential as a screening tool for COPD, particularly in resource-limited clinical settings, helping to identify high-risk individuals for early detection. These strengths give the study a certain degree of innovation and practical application value. However, there are notable limitations. The diagnosis of COPD in some participants

Table 1 Baseline characteristics of participants in the NHANES 2007-2016

Characteristics	Lipid accumulation product				*P*-value
	Q1 (*N*=3022)	Q2 (*N*=3021)	Q3 (*N*=3023)	Q4 (*N*=3023)	
LAP	14.61 ± 5.61	32.67 ± 5.37	55.72 ± 8.81	127.41± 91.59	<0.001
COPD, n(%)					<0.001
No	2849 (94.28%)	2794 (92.49%)	2732 (90.37%)	2642 (87.40%)	
Yes	173 (5.72%)	227 (7.51%)	291 (9.63%)	381 (12.60%)	
Gender, n(%)					0.047
Male	1419 (46.96%)	1446 (47.86%)	1482 (49.02%)	1523 (50.38%)	
Female	1603 (53.04%)	1575 (52.14%)	1541 (50.98%)	1500 (49.62%)	
Age, n(%)					<0.001
≦60	2435 (80.58%)	2048 (67.79%)	1923 (63.61%)	1990 (65.83%)	
>60	587 (19.42%)	973 (32.21%)	1100 (36.39%)	1033 (34.17%)	
Race, n(%)					<0.001
Mexican American	304 (10.06%)	432 (14.30%)	566 (18.72%)	582 (19.25%)	
Non-Hispanic White	1171 (38.75%)	1255 (41.54%)	1225 (40.52%)	1517 (50.18%)	
Non-Hispanic Black	745 (24.65%)	648 (21.45%)	589 (19.48%)	359 (11.88%)	
Other race	802 (26.54%)	686 (22.71%)	643 (21.27%)	565 (18.69%)	
PIR, n(%)					<0.001
≦1.0	621 (22.44%)	588 (21.32%)	608 (21.99%)	666 (24.11%)	
>1.0, ≦3.0	1080 (39.02%)	1095 (39.70%)	1216 (43.98%)	1233 (44.64%)	
>3.0	1067 (38.55%)	1075 (38.98%)	941 (34.03%)	863 (31.25%)	
Education level, n(%)					<0.001
<High school	610 (20.19%)	733 (24.26%)	876 (28.98%)	913 (30.20%)	
High school	622 (20.58%)	669 (22.15%)	668 (22.10%)	733 (24.25%)	
>High school	1790 (59.23%)	1619 (53.59%)	1479 (48.92%)	1377 (45.55%)	
Diabetes, n(%)					<0.001
Yes	122 (4.04%)	286 (9.47%)	468 (15.48%)	633 (20.94%)	
No	2868 (94.90%)	2671 (88.41%)	2478 (81.97%)	2287 (75.65%)	
borderline	32 (1.06%)	64 (2.12%)	77 (2.55%)	103 (3.41%)	
Smoking status, n(%)					<0.001
Yes	1173 (38.82%)	1301 (43.07%)	1384 (45.78%)	1539 (50.91%)	
No	1849 (61.18%)	1720 (56.93%)	1639 (54.22%)	1484 (49.09%)	
Hypertension, n(%)					<0.001
Yes	577 (19.09%)	1000 (33.10%)	1273 (42.11%)	1535 (50.78%)	
No	2445 (80.91%)	2021 (66.90%)	1750 (57.89%)	1488 (49.22%)	
Alcohol consumption, n(%)					0.069
Yes	2015 (73.62%)	2022 (72.73%)	2011 (71.26%)	2014 (70.79%)	
No	722 (26.38%)	758 (27.27%)	811 (28.74%)	831 (29.21%)	

Mean ± SD for continuous variables: the *P* value was calculated by the linear regression model; (%) for categorical variables: the *P* value was calculated by the chi-square test. *Abbreviations*: *COPD* Chronic obstructive pulmonary disease, *LAP* Lipid accumulation product, *NHANES* National health and nutrition examination survey, *PIR* Poverty Income Ratio, *Q* Quartile

was based on self-reported symptoms, which may lack objective confirmation and could result in misclassification or recall bias. Additionally, the cross-sectional design restricts the ability to establish causal relationships between LAP and COPD. Moreover, the study did not explore the relationship between varying severities of COPD, as COPD severity was not assessed. Additionally, this study only includes data from Americans, and the selected sample may not fully represent a diverse range of regions and populations, which could limit the generalizability and accuracy of the findings. And this study did not quantify smoking exposure, smoking plays a crucial role in the development of COPD, and the lack of smoking quantification data in some of the study populations

Table 2 Multivariable logistic regression models for the association between LAP and COPD in adults in the NHANES 2007–2016

COPD	OR (95% CI), *P*-value		
	Model 1	**Model 2**	**Model 3**
Continuous	1.0059 (1.0047, 1.0072) <0.000001	1.0053 (1.0040, 1.0066) <0.000001	1.0024 (1.0009, 1.0040) 0.002209
Categories			
Q1	Reference	Reference	Reference
Q2	1.3380 (1.0904, 1.6417) 0.005280	1.2078 (0.9814, 1.4866) 0.074684	1.0866 (0.8664, 1.3627) 0.472366
Q3	1.7541 (1.4428, 2.1325) <0.000001	1.5823 (1.2960, 1.9319) 0.000007	1.2692 (1.0183, 1.5818) 0.033852
Q4	2.3749 (1.9692, 2.8641) <0.000001	2.0814 (1.7174, 2.5226) <0.000001	1.4572 (1.1728, 1.8107) 0.000679

Abbreviations: *COPD* Chronic obstructive pulmonary disease, *LAP* Lipid accumulation product, *NHANES* National Health and Nutrition Examination Survey, *OR* Odds ratio, *CI* Confidence interval

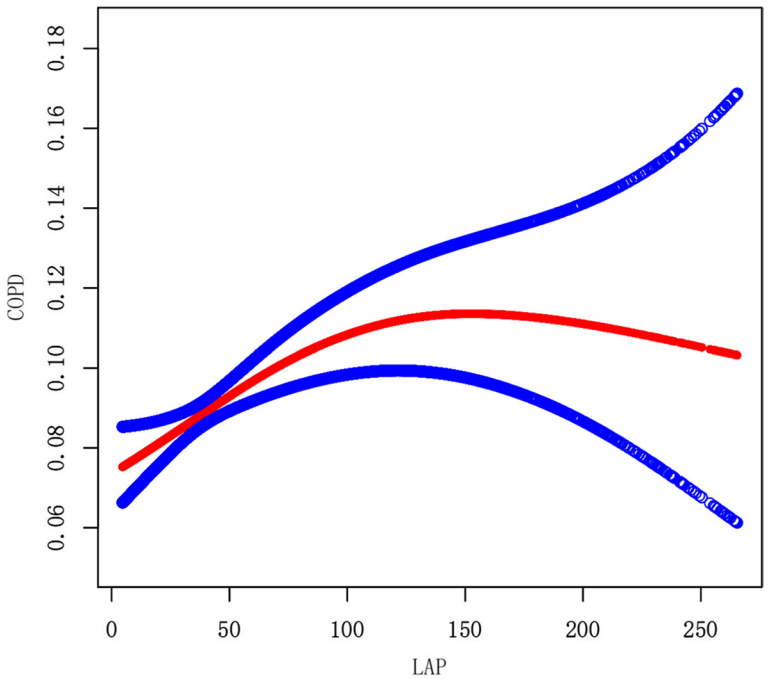

Fig. 2 The nonlinear associations between the LAP and COPD. The solid red line represents the smooth curve fit between variables. Blue bands represent the 95% confidence interval from the fit

Table 3 Threshold effect analysis of LAP on COPD using a two-piecewise logistic regression model in adults in the NHANES 2007–2016

Threshold effect analysis	COPD
	OR (95%CI) *P*-value
Inflection point of LAP (K)	65.5278
<K slope	1.0076 (1.0032, 1.0121) 0.0007
>K slope	1.0001 (0.9977, 1.0026) 0.9132
Log-likelihood ratio test	0.013

Abbreviations: *COPD* Chronic obstructive pulmonary disease, *LAP* Lipid accumulation product, *NHANES* National Health and Nutrition Examination Survey, Notes: Age, race, gender, education level, poverty income ratio, alcohol consumption, smoking status, hypertension and diabetes were adjusted.

restricts further analysis of its impact. These limitations should be taken into account when interpreting the study's conclusions. To improve generalizability, future research should include diverse populations from economically disadvantaged countries and various ethnic groups. Multi-center studies across regions will help confirm the global applicability of LAP as a COPD risk predictor. In terms of research methodology, future research should adopt longitudinal designs to track LAP changes and COPD development, rely on objective diagnostic methods like lung tests and imaging instead of self-reports, and better control for confounders using advanced statistical techniques. Given the current lack

Table 4 Stratified analysis of the correlation between LAP and COPD in adults in the NHANES 2007–2016

Subgroup	OR (95% CI), *P*-value	
	LAP	*P* interaction
Gender		0.1363
Male	1.0012 (0.9990, 1.0035) 0.2669	
Female	1.0035 (1.0014, 1.0057) 0.0011	
Age		0.5304
≦60	1.0026 (1.0005, 1.0046) 0.0129	
>60	1.0016 (0.9991, 1.0040) 0.2095	
Education level		0.0565
Less than high school	1.0001 (0.9973, 1.0029) 0.9397	
High school	1.0016 (0.9984, 1.0049) 0.3302	
Above high school	1.0044 (1.0021, 1.0067) 0.0001	
Diabetes		0.1562
Yes	1.0039 (1.0007, 1.0070) 0.0155	
No	1.0021 (1.0002, 1.0039) 0.0287	
borderline	0.9956 (0.9873, 1.0039) 0.2986	
Hypertension		0.0070
Yes	1.0005 (0.9984, 1.0026) 0.6414	
No	1.0048 (1.0025, 1.0071) <0.0001	
Smoking status		0.2387
Yes	1.0019 (1.0001, 1.0037) 0.0436	
No	1.0039 (1.0010, 1.0067) 0.0073	
Alcohol consumption		0.1293
Yes	1.0031 (1.0013, 1.0048) 0.0006	
No	1.0003 (0.9970, 1.0035) 0.8693	

Age, race, gender, education level, poverty income ratio, alcohol consumption, smoking status, hypertension and diabetes were adjusted. The strata variable was not included in the model when stratifying by itself

Abbreviations: *COPD* Chronic obstructive pulmonary disease, *LAP* Lipid accumulation product, *NHANES* National Health and Nutrition Examination Survey, *OR* Odds ratio, *CI* Confidence interval

of mechanistic research, we believe that as basic studies advance, the biological mechanisms linking LAP and COPD will become clearer. Future research, particularly using animal models and cell experiments, will be crucial in advancing this field and supporting early COPD diagnosis and treatment. Furthermore, future studies should incorporate more clinical data, reduce missing data, and quantify key covariates to enhance research reliability and clinical significance.

Discussion

The association between LAP and the odds of COPD prevalence was examined by analyzing data from 12,089 participants in the 2007–2016 NHANES through a cross-sectional analysis. Results reveal a significant positive association between LAP levels and the odds of COPD prevalence, suggesting that higher LAP values are linked to increased odds of developing COPD. Metabolic factors, such as abdominal obesity and dyslipidemia, could be essential in the development of COPD. An inflection point was identified at LAP = 65.5278, where the relationship between LAP and COPD showed a significant positive association for LAP values below this threshold ($P = 0.013$). These results underscore the potential of LAP as a target for early intervention in COPD patients. Additionally, hypertension was identified as a significant moderator of this association, highlighting the importance of considering blood pressure in the assessment and management of COPD. Overall, these findings stress the relevance of LAP levels in the evaluation and management of COPD.

The relationship between obesity and COPD has attracted increasing attention in recent years. Research indicates that obesity not only exacerbates respiratory symptoms but also significantly elevates the risk of developing COPD [28–31]. Excess fat mass, particularly abdominal obesity, contributes to respiratory difficulties by limiting lung expansion and increasing the effort required for breathing. Additionally, obesity disrupts adipose tissue function, leading to heightened systemic inflammation, which further aggravates COPD pathology [32]. In nonsmoking populations, a strong dose-response relationship has been observed between levels of obesity and the prevalence of COPD, indicating that greater obesity correlates with an increased risk of the disease. Therefore, regular COPD screenings are advised for obese individuals without a smoking history, especially those over 50, to enable early intervention [29]. The intricate relationship between BMI and COPD also merits attention. While obesity may worsen symptoms such as dyspnea and negatively impact health-related quality of life, a phenomenon known as the "obesity paradox" has been documented, where mild to moderate obesity is linked to better survival rates among COPD patients. This paradox may be influenced by factors such as smoking status, health behaviors, and lifestyle choices, complicating the understanding and intervention strategies regarding the connection between obesity and COPD [2]. Furthermore, adipokines such as adiponectin play crucial roles in inflammatory responses and organ communication in obese individuals. As an anti-inflammatory adipokine, adiponectin interacts with specific receptors to regulate lung function, potentially offering protective effects for COPD patients [28]. Investigating these molecular mechanisms may reveal new therapeutic pathways and aid in developing more tailored, individualized treatments for COPD. For COPD patients, decreased physical activity levels further increase the risk of obesity, particularly among those undergoing long-term glucocorticoid therapy, who are more prone to central obesity and fat redistribution [33, 34]. These

changes in weight and fat distribution not only exacerbate COPD progression but also complicate overall health management for these patients. Thus, COPD prevention and treatment strategies should take into account BMI, fat distribution, medication use, and lifestyle factors to formulate a comprehensive, personalized approach.

Currently, there is no direct study investigating the biological mechanisms behind the relationship between LAP and COPD, but existing evidence suggests that LAP, as an index of fat accumulation, is closely related to insulin resistance, metabolic abnormalities, and chronic inflammation, all of which may influence the pathogenesis of COPD through various mechanisms. An increase in LAP typically reflects the accumulation of visceral fat, which is considered a key driver of insulin resistance [35]. In COPD patients, the accumulation of visceral fat not only causes metabolic abnormalities but also promotes systemic and localized chronic inflammation through the secretion of pro-inflammatory cytokines such as Tumor Necrosis Factor-alpha (TNF-α) and Interleukin-6 (IL-6) [36]. These inflammatory mediators play a crucial role in airway remodeling and lung tissue damage, and may further exacerbate chronic airway inflammation by altering the function of macrophages and T cells [37]. Furthermore, the strong association between LAP, metabolic syndrome (MetS), and insulin resistance suggests that LAP may play an important role in the metabolic dysregulation and immune response seen in COPD. Studies have shown that increased LAP levels are closely linked to metabolic disturbances such as hyperglycemia, dyslipidemia, and insulin resistance in COPD patients [38]. These metabolic abnormalities not only exacerbate oxidative stress levels and fatty acid accumulation in the lungs, promoting tissue damage, but may also enhance systemic inflammation, worsening the pathological changes associated with COPD [39]. Therefore, LAP may serve as a potential biomarker for COPD by influencing metabolic and immune regulation, offering new insights into the disease's underlying mechanisms.

LAP provides an innovative, safe, and effective method for assessing central lipid accumulation in adults. In contrast to conventional techniques like magnetic resonance imaging and computed tomography, LAP is straightforward and noninvasive. There is a significant link between it and insulin resistance, as well as various metabolic and cardiovascular risks [19, 40]. Prior research has shown that LAP demonstrates superior predictive capability, providing better results than other measures of adiposity such as body mass index (BMI), waist-to-hip ratio (WHR), waist circumference (WC), visceral adiposity index (VAI) and waist-to-height ratio (WHtR) [17]. This study establishes a positive correlation between LAP and COPD, consistent with findings from Park et al. Consequently, LAP could be included in the clinical approach to managing COPD [11].

Through subgroup analyses, it was observed that increased LAP levels correlate with a higher risk of developing COPD in individuals without hypertension, whereas this association is diminished in the hypertensive population. It is hypothesized that this may be due to patients with hypertension being more health-conscious and actively managing their COPD risk factors. In contrast, individuals without hypertension may lack such discipline and could already meet the diagnostic criteria for COPD by the time they seek medical care. Furthermore, hypertension in COPD patients may influence metabolic markers, which could further weaken the association between LAP and COPD.

Conclusion

Our results reveal a positive association between the LAP levels and COPD. By practicing dietary and exercise interventions, and monitoring the LAP, we can effectively prevent the development of COPD.

Abbreviations

BMI	Body mass index
CDC	Centers for disease control and prevention
CI	Confdence interval
COPD	Chronic obstructive pulmonary disease
FEV1	Forced expiratory volume in one second
FVC	Forced vital capacity
GAM	Generalized additive models
IL-6	Interleukin-6
LAP	Lipid accumulation product
NHANES	The national health and nutrition examination survey
OR	Odds ratio
PIR	Income-to-poverty ratio
SD	Standard deviation
SE	Standard errors
TG	Triglyceride
TNF-α	Tumor necrosis factor-alpha
VAI	Visceral adiposity index
WC	Waist circumference
WHR	Waist-to-hip ratio
WHtR	Waist-to-height ratio

Acknowledgements

We thank the National Center for Health Statistics of the Centers for Disease Control staff for designing, collecting, and collating the NHANES data and creating the public database.

Authors' contributions

Qiang Xiao and Shaofeng Zhang conceptualized and designed the study. Jia Jiang, Yaling Zeng, Jiayi Liu and Wei Lei extracted and organized the clinical data. Qiang Xiao, Xin Chen, Zhongli Li, Siqin Chen and Shaofeng Zhang contributed to statistical analysis, literature review and drafted the manuscript. Qiang Xiao, Jia Li and Jia Jiang interpreted the data. Zhongli Li, Xin Chen, and Yaling Zeng have made revisions to address the deficiencies in the article and have refined the language. All authors gave final approval for the version to be published.

Funding

The research received funding from the Natural Science Foundation of Guangdong Province (Grant No. 2024A1515012935).

Data availability
The datasets analysed during the current study are available at (https://www.cdc.gov/nchs/nhanes/index.htm).

Declarations

Ethics approval and consent to participate
The datasets were obtained from the NHANES database, and all data were under ethics approval before recorded in the database.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

Received: 9 November 2024 Accepted: 27 December 2024
Published online: 06 January 2025

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