Lipids in Health and Disease

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Association between lipid accumulation product (LAP) index and self-reported oral health outcomes: a cross-sectional study

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Abstract

Background Lipid Accumulation Product (LAP) is a biomarker associated with excessive lipid accumulation and various metabolic diseases. Despite the well-established link between oral and systemic health, the association between LAP and oral health remains largely unexplored.

Methods Data from 7,124 participants aged over 18 years from the National Health and Nutrition Examination Survey (NHANES) between 2009 and 2014 were analyzed. Multivariate logistic regression models were applied to evaluate the independent association between LAP and self-reported oral health outcomes, adjusting for potential confounders.

Results There was a significant correlation between increasing LAP levels and deteriorating oral health status ($\beta = 0.26$, 95% CI: 0.20 to 0.32, $P < 0.0001$). Subgroup analyses revealed that the negative correlation between LAP and self-reported oral health outcomes was more pronounced in younger individuals and those with higher education levels. A non-linear relationship was identified, with oral health deteriorating significantly as LAP increased up to a threshold of 83, after which the relationship became non-significant.

Conclusions This study demonstrates a robust association between LAP and self-reported oral health outcomes, suggesting that LAP could be a reliable indicator for assessing oral health status. The findings highlight the importance of targeted health interventions for populations with higher LAP indices to prevent oral health deterioration and potential systemic health issues.

Keywords NHANES, LAP, Oral health, Cross-sectional study

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Introduction

Waist circumference(WC) and blood triglycerides(TG) are essential indicators for evaluating the risk of excessive lipid accumulation in adults, with the LAP serving as a biomarker for this assessment. The formula for LAP is LAP = (WC − 65) ×TG for men and LAP = (WC − 58) × TG for women, where WC represents waist circumference (cm) and TG denotes serum triglycerides (mmol/L) [1]. Research indicates that LAP is linked to several illnesses, including polycystic ovarian syndrome (PCOS) [2], metabolic syndrome [3], diabetes [4, 5], cardiovascular diseases [6, 7], mild cognitive impairment [8], hepatic steatosis [9], and sarcopenia [10]. This highlights the importance of LAP in identifying the risk of diseases associated with excessive lipid accumulation.

Oral health is not merely a local tissue concern but also intricately linked to systemic disease progression. Emerging evidence demonstrates that oral inflammation triggers chronic low-grade systemic inflammation through microorganism-host interactions, thereby contributing to metabolic disorders. A bidirectional vicious cycle exists between diabetes [11] and periodontitis, exemplifying this interconnection. Notably, significant associations have been established between oral inflammatory conditions and various systemic pathologies, including metabolic syndrome [12], rheumatoid arthritis [13], osteoporosis [14], and certain malignancies [15, 16]. The underlying mechanisms involve: (1) IL-6 and TNF-α-mediated exacerbation of insulin resistance [17]; (2) oxidative stress-induced vascular endothelial dysfunction [18]; and (3) systemic immune activation through bacteremia caused by oral pathogens such as Porphyromonas gingivalis [19]. These pathways collectively underscore the pivotal role of oral health as a critical modulator of systemic health.

LAP has demonstrated strong diagnostic capabilities in metabolic diseases [3, 20, 21] and is closely associated with systemic inflammation, obesity, and other systemic disorders. These factors, along with the oral microbiome, are well-established risk factors for poor oral health. Despite the clear overlap between LAP-related diseases and those associated with oral health, the role of LAP in oral health outcomes remains unexplored. This gap in the literature underscores the need for further investigation. Therefore, this study aims to explore the relationship between LAP and oral health, enhancing our understanding and potentially establishing LAP as a diagnostic tool for assessing oral health risks.

Methods

Data sources and study population

NHANES collected data on exposures and their outcomes. This data was collected throughout three consecutive cycles from 2009 to 2014. The data were collected through health interviews conducted at participants' residences, health checks carried out at mobile testing centres (MECs), and laboratory samples. There was no need for a supplementary ethical review for this manuscript, as NHANES underwent an ethical review by the National Ethical Review Board for Research in Health Statistics [22]. The missing data were imputed using multiple imputation.

The study initially comprised 30,468 participants from NHANES data collected between 2009 and 2014, incorporating comprehensive self-assessed oral health data of U.S. adults. Participant eligibility was determined based on the following criteria: [1] complete waist circumference data; [2] complete triglyceride data; [3] complete self-assessed oral health information; and [4] age over 18 years. In the end, 7,124 people were involved in of the research. Figure 1 illustrates the data filtration process.

Outcome variable

The Oral Health Questionnaire (OHQ) was employed to evaluate the oral health outcomes of individuals, with a five-point scale extending from Excellent to Poor. Oral health status shows progressive deterioration with increasing scores on self-reported oral health outcomes. These metrics have been increasingly employed in population-based research to evaluate both systemic diseases and oral health conditions in recent years. Previous study has established the strong validity of self-reported oral health outcomes and evaluations of periodontal disease in extensive populations [23–25]. The outcome of professional dental examinations is comparable to the reliability and validity of OHQ [26, 27]. Collectively, self-reported oral health outcomes demonstrate significant predictive value for oral health status, highlighting their efficacy in large-scale epidemiological investigations.

Confounding variables

This study included the following covariates: gender, age, race, education level, ratio of family income to poverty (RIP), diabetes, hypertension, coronary heart disease, hypercholesterolemia, gout, stroke, cancer or malignancy, alcohol use, and cigarette use. Based on tertiles, age was divided into three categories. The participant's diabetes status was categorized into three categories: yes, no, or borderline, depending on whether a physician had diagnosed the condition. Cigarette usage was classified into three categories: never smokers, defined as individuals who have smoked fewer than 100 cigarettes in their lifetime; former smokers, who have smoked more than 100 cigarettes but are currently abstinent; and current smokers, who have smoked more than 100 cigarettes and continue to smoke [28]. Alcohol consumption was similarly categorized into three groups: non-drinkers, who do not consume alcohol; moderate drinkers, characterized

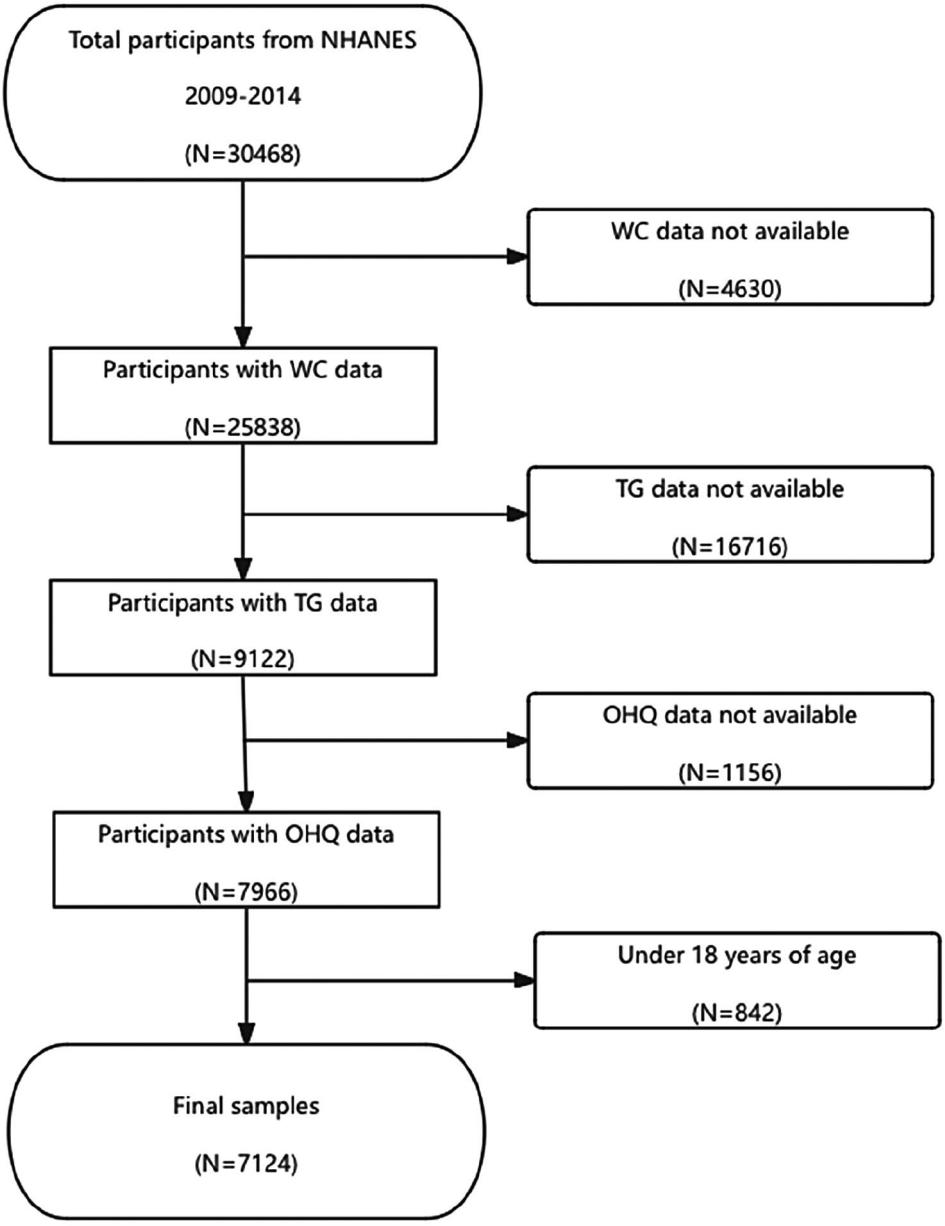

Fig. 1 Flowchart of participant selection. NHANES, National Health and Nutrition Examination Survey; WC, waist circumference; TG, triglycerides; OHQ, Oral Health Questionnaire

as men consuming 14 or fewer drinks per week or 5 or fewer drinks on any single day, and women consuming 7 or fewer drinks per week or 5 or fewer drinks on any single day; and heavy drinkers, defined as men consuming more than 14 drinks per week or 5 or more drinks on any single day at least once in the past year, and women consuming more than 7 drinks per week or 5 or more drinks on any single day at least once in the past year [29].

Statistical analysis

The statistical analysis of the results was performed using R software (R 4.2.1) in combination with Empower Stats 4.1. The study utilized multivariate logistic regression to investigate the independent association between LAP and self-reported oral health outcomes. Three models were employed: an unadjusted model; a partial adjustment model adjusted for gender, age, race, education level, and the RIP; and fully adjusted model adjusted for gender, age, race, education level, RIP, diabetes, hypertension, coronary heart disease, hypercholesterolemia, gout, stroke, cancer or malignancy, alcohol use and cigarette use. Interaction tests, subgroup analyses, threshold effect experiments, and smoothed curve fitting were conducted

to explore the multidimensional relationship between self-reported oral health outcomes and LAP.

Results

Baseline characteristics of the population

The Table 1 displays the fundamental features of the research population. This study conducted a comparative analysis of 7124 participants stratified into three groups based on the LAP index (LAPT1, LAPT2, and LAPT3). There were significant age differences among the three groups, with the LAPT1 group having an average age of 35.18 years, while the LAPT3 group had an average age of 51.56 years ($P < 0.001$). The LAPT3 group exhibited a considerably higher percentage of individuals with lesser education levels in terms of educational attainment ($P < 0.001$).

The prevalence of chronic diseases also differed significantly, with rates of diabetes, hypertension, hypercholesterolemia, and gout rising with an increasing LAP index. Specifically, the incidence of diabetes increased from 2.60% in the LAPT1 group to 18.49% in the LAPT3 group ($P < 0.001$).

In terms of lifestyle behaviors, the percentage of current smokers increased substantially from 13.22% in LAPT1 to 20.33% in LAPT3 ($P < 0.001$), while the percentage of heavy drinkers did not see a statistically significant increase ($P = 0.010$). In all three groups, oral health deteriorated progressively with increasing LAP levels ($P < 0.001$).

In summary, participants with different LAP indices exhibited significant variations in health status and lifestyle factors, highlighting the importance of targeted health interventions for populations with higher LAP indices.

Univariate analysis

This study investigated the impact of various factors on self-reported oral health outcomes using univariate analysis, with the results summarized in Table 2. The base-10 logarithm was employed to transform LAP in order to guarantee the accuracy of the results and the feasibility of data analysis. The analysis demonstrated a substantial correlation between deteriorating oral health status and elevated levels of LAP ($P < 0.0001$). Additionally, females exhibited poorer oral health compared to males ($P = 0.0069$). Moreover, higher age, specific racial backgrounds, and lower educational levels were significantly correlated with deteriorating oral health ($P < 0.0001$). Oral health was also significantly impacted by chronic conditions, including hypertension, coronary heart disease, diabetes, and hypercholesterolemia ($P < 0.0001$). In terms of smoking behavior, current smokers showed significantly worse oral health status ($P < 0.0001$). Conversely, alcohol use, as well as cancer and malignancy, did not show a significant correlation with oral health (P were 0.9458 and 0.0530, respectively).

Relationship between LAP and self-reported oral health outcomes

In order to assess the correlation between self-reported oral health outcomes and LAP, we implemented a multivariable logistic regression analysis (Table 3). The unadjusted model demonstrated a substantial correlation between self-reported oral health outcomes and the log-transformed LAP, with a coefficient of $\beta = 0.42$ (95% CI: 0.37 to 0.48, $P < 0.0001$). The result in the partially adjusted model indicated a modest change ($\beta = 0.31$, 95% CI: 0.25 to 0.37, $P < 0.0001$), whereas in the fully adjusted model, $\beta = 0.26$ (95% CI: 0.20 to 0.32, $P < 0.0001$). The log-transformed LAP was classified into tertiles for sensitivity analysis, and a comparable trend was observed($P < 0.0001$). With an increase in LAP, the oral health status significantly declines.

The non-linear relationship between LAP and self-reported oral health outcomes

A nonlinear relationship between LAP and self-reported oral health outcomes was identified after adjusting for various confounding factors (Fig. 2). The folding point was identified as 83 through threshold effect analysis (Table 4). When LAP are below 83, there is a significant deterioration in oral health as LAP increases ($P < 0.0001$); conversely, when LAP exceed 83, the relationship between LAP and oral health becomes non-significant ($P = 0.1648$).

The results of subgroup analyses

The test for interactions was significant for age, education level, and gout (*P for interaction* < 0.05), as illustrated in Fig. 3, while it was not significant for gender, race, RIP, diabetes, hypertension, coronary heart disease, hypercholesterolemia, stroke, cancer or malignancy, alcohol use, and cigarette use (*P for interaction* = 0.1995, 0.6675, 0.3864, 0.3327, 0.6608, 0.4353, 0.57, 0.101, 0.3073, 0.1135, and 0.0605, respectively). Younger individuals and those with higher levels of education exhibited a more pronounced negative correlation between self-reported oral health outcomes and LAP. Additionally, this negative association was significant in participants without gout, whereas it was not significant in those with gout. The visualized results of the subgroup analysis are presented in the Supplementary Material 1.

Discussion

No studies have yet been conducted to establish an association between oral health and LAP. Both oral health issues and several metabolic disorders share inflammation as a common etiology. This study involved 7,124

Table 1 Characterization of the study population

Variables	LAPT1	LAPT2	LAPT3	*P*-value
N	2655	2655	2656	
Age	35.18 ± 19.73	49.78 ± 18.50	51.56 ± 16.58	< 0.001
RIP	2.41 ± 1.63	2.52 ± 1.59	2.26 ± 1.53	< 0.001
Gender				0.351
Male	1268 (47.76%)	1318 (49.64%)	1308 (49.25%)	
Female	1387 (52.24%)	1337 (50.36%)	1348 (50.75%)	
Race				< 0.001
Mexican American	315 (11.86%)	376 (14.16%)	485 (18.26%)	
Other Hispanic	232 (8.74%)	281 (10.58%)	304 (11.45%)	
Non-Hispanic White	937 (35.29%)	1102 (41.51%)	1253 (47.18%)	
Non-Hispanic Black	680 (25.61%)	562 (21.17%)	405 (15.25%)	
Other Race	491 (18.49%)	334 (12.58%)	209 (7.87%)	
Education Level				< 0.001
Less Than 9th Grade	121 (4.56%)	258 (9.72%)	315 (11.86%)	
9-11th Grade	214 (8.06%)	342 (12.88%)	443 (16.68%)	
High School Grad/GED or Equivalent	660 (24.86%)	606 (22.82%)	644 (24.25%)	
Some College or AA degree	894 (33.67%)	786 (29.60%)	787 (29.63%)	
College Graduate or above	766 (28.85%)	663 (24.97%)	467 (17.58%)	
Diabetes				< 0.001
YES	69 (2.60%)	279 (10.51%)	491 (18.49%)	
NO	2562 (96.50%)	2311 (87.04%)	2074 (78.09%)	
Borderline	24 (0.90%)	65 (2.45%)	91 (3.43%)	
Hypertension				< 0.001
YES	390 (14.69%)	920 (34.65%)	1299 (48.91%)	
NO	2265 (85.31%)	1735 (65.35%)	1357 (51.09%)	
Coronary Heart Disease				< 0.001
YES	37 (1.39%)	110 (4.14%)	123 (4.63%)	
NO	2618 (98.61%)	2545 (95.86%)	2533 (95.37%)	
Hypercholesterolemia				< 0.001
YES	342 (12.88%)	913 (34.39%)	1205 (45.37%)	
NO	2313 (87.12%)	1742 (65.61%)	1451 (54.63%)	
Gout				< 0.001
YES	303 (11.41%)	673 (25.35%)	872 (32.83%)	
NO	2352 (88.59%)	1982 (74.65%)	1784 (67.17%)	
Stroke				< 0.001
YES	51 (1.92%)	91 (3.43%)	99 (3.73%)	
NO	2604 (98.08%)	2564 (96.57%)	2557 (96.27%)	
Cancer Or Malignancy				< 0.001
YES	141 (5.31%)	241 (9.08%)	265 (9.98%)	
NO	2514 (94.69%)	2414 (90.92%)	2391 (90.02%)	
Alcohol Use				0.01
Never drinking	291 (10.96%)	338 (12.73%)	369 (13.89%)	
Moderate drinking	2289 (86.21%)	2245 (84.56%)	2198 (82.76%)	
Heavy drinking	75 (2.82%)	72 (2.71%)	89 (3.35%)	
Cigarette Use				< 0.001
Never smoker	1435 (54.05%)	1447 (54.50%)	1343 (50.56%)	
Former smoker	869 (32.73%)	737 (27.76%)	773 (29.10%)	
Current smoker	351 (13.22%)	471 (17.74%)	540 (20.33%)	
Self-reported oral health outcomes				< 0.001
Excellent	409 (15.40%)	277 (10.43%)	235 (8.85%)	
Very good	678 (25.54%)	560 (21.09%)	465 (17.51%)	
Good	928 (34.95%)	1005 (37.85%)	992 (37.35%)	

Table 1 (continued)

Variables	LAPT1	LAPT2	LAPT3	*P*-value
Fair	476 (17.93%)	558 (21.02%)	624 (23.49%)	
Poor	164 (6.18%)	255 (9.60%)	340 (12.80%)	

Results in table: Mean ± SD / N(%)

participants. The results indicate a robust and significant correlation between LAP and the deterioration of oral health status after adjusting for various confounding factors (β = 0.26, 95% CI: 0.20 to 0.32, $P < 0.0001$). Subgroup analyses also demonstrated consistent patterns. The study indicates that LAP serves as a reliable indicator for assessing oral health, demonstrating exceptional diagnostic accuracy.

LAP is a straightforward and efficient metric of visceral adiposity, closely associated with overall health condition [30]. Higher LAP levels significantly correlate with increased risks of hypertension [31], type 2 diabetes [4, 5], cardiovascular diseases [32], and liver conditions [9]. In populations with PCOS [2], LAP is associated with metabolic disturbances, reflecting impaired glucose tolerance. Moreover, LAP has demonstrated significant predictive value for cardiovascular [33]. It is also significantly associated with subclinical cardiovascular organ damage in elderly populations and is effective in identifying metabolic syndrome in adults with growth hormone deficiency [34]. LAP is an effective tool for diagnosing metabolic diseases and influences changes in systemic inflammatory markers [35, 36], aiding in the identification and management of high-risk populations. This metric is particularly convenient for clinical application, as it can be easily calculated using routine clinical measurements (WC and TG). Therefore, LAP serves not only as an important instrument for assessing individual health risks but also provides a foundation for effective health management and clinical interventions.

Oral diseases are closely linked to various systemic conditions. Firstly, oral health may impact respiratory health by transferring oral microbes to the lungs, thereby increasing the risk of pneumonia [37]. Secondly, periodontal disease has been associated with certain cancers [38, 39]. Most importantly, oral health is related to an increased risk of cardiovascular disease [40] and may exacerbate type 2 diabetes [41] by worsening insulin resistance. As a result, there is a growing awareness of the relationship between oral health and metabolic diseases.

The LAP significantly impacts oral health through several interconnected mechanisms. Firstly, LAP is associated with systemic inflammatory responses, which may exacerbate the progression of periodontal disease by elevating levels of cytokines and chemokines that contribute to periodontal tissue damage [42–45]. Studies have confirmed a significant correlation between inflammatory markers and oral health [46]. Additionally, LAP-induced lipid peroxidation increases oxidative stress, further harming periodontal tissues [47]. Secondly, abnormal lipid profiles are strongly linked to periodontal inflammation [48, 49], with LAP playing a crucial role in these lipid abnormalities. Furthermore, the link between obesity-related systemic inflammation and a higher prevalence of periodontal disease underscores the influence of LAP [50]. Lastly, periodontal treatment not only improves oral health but may also alleviate systemic inflammation and improve lipid profiles, thereby enhancing overall health [51, 52]. The prevention as well as treatment of oral diseases and related systemic health issues are contingent upon the effective monitoring and management of the LAP.

Recent studies highlight a correlation between oral health and lipids [53, 54], with the relationship between the LAP and oral health being particularly noteworthy. Although research in this area is currently limited, findings suggest that oral health significantly deteriorates with increasing LAP. Notably, factors such as age, education level, and gout significantly influence this correlation. Patients without gout often exhibit superior oral health, and the correlation between LAP and dental health is more significant in this demographic. This may be due to the association of gout with aberrant lipid metabolism [55, 56], resulting in generally poorer oral health among gout patients, which may obscure the impact of rising LAP.

The study reveals that oral health deteriorates rapidly at lower LAP levels but stabilizes once LAP exceeds 83, suggesting a plateau effect where further increases have minimal impact. This threshold effect may stem from both biological and statistical factors. Biologically, the stabilization at LAP > 83 could reflect the body's adaptation to excessive lipid accumulation [57, 58]. Given the strong link between obesity and chronic inflammation, systemic inflammation may plateau beyond this threshold, reducing its impact on oral health. Statistically, the small sample size in the LAP > 83 group may limit statistical power, while adjustments for multiple covariates could attenuate LAP's direct effect. Additionally, in individuals with high LAP, metabolic comorbidities such as diabetes, hypertension, and cardiovascular disease, along with genetic and lifestyle factors [59, 60], may exert a stronger influence, diminishing LAP's independent role. Further study is essential to elucidate the precise role and processes of LAP in oral health.

Table 2 The results of univariate analysis

Exposure	Mean ± SD / *N*(%)	β (95%CI)	*P*-value
LAP log10 transform	1.54 ± 0.43	0.42 (0.37, 0.48)	< 0.0001
LAP log10 transformed three groups			
Low	2638 (33.32%)	ref	
Medium	2640 (33.34%)	0.24 (0.18, 0.30)	< 0.0001
High	2640 (33.34%)	0.40 (0.34, 0.46)	< 0.0001
Age	45.51 ± 19.73	0.01 (0.00, 0.01)	< 0.0001
Age Tertile			
AGET1	2646 (33.22%)	ref	
AGET2	2615 (32.83%)	0.28 (0.22, 0.34)	< 0.0001
AGET3	2705 (33.96%)	0.22 (0.16, 0.28)	< 0.0001
RIP	2.40 ± 1.59	-0.19 (-0.20, -0.17)	< 0.0001
RIP Tertile			
RIPT1	2655 (33.33%)	ref	
RIPT2	2646 (33.22%)	-0.31 (-0.37, -0.26)	< 0.0001
RIPT3	2665 (33.45%)	-0.71 (-0.76, -0.65)	< 0.0001
Gender			
Male	3894 (48.88%)	ref	
Female	4072 (51.12%)	-0.07 (-0.12, -0.02)	0.0069
Race			
Mexican American	1176 (14.76%)	ref	
Other Hispanic	817 (10.26%)	-0.21 (-0.31, -0.11)	< 0.0001
Non-Hispanic White	3292 (41.33%)	-0.45 (-0.53, -0.38)	< 0.0001
Non-Hispanic Black	1647 (20.68%)	-0.27 (-0.35, -0.19)	< 0.0001
Other Race	1034 (12.98%)	-0.47 (-0.56, -0.38)	< 0.0001
Education Level			
Less Than 9th Grade	694 (8.71%)	ref	
9-11th Grade	999 (12.54%)	-0.06 (-0.16, 0.04)	0.2518
High School Grad/GED or Equivalent	1910 (23.98%)	-0.33 (-0.43, -0.24)	< 0.0001
Some College or AA degree	2467 (30.97%)	-0.63 (-0.72, -0.54)	< 0.0001
College Graduate or above	1896 (23.80%)	-1.00 (-1.09, -0.91)	< 0.0001
Diabetes			
YES	839 (10.53%)	ref	
NO	6947 (87.21%)	-0.28 (-0.36, -0.19)	< 0.0001
Borderline	180 (2.26%)	-0.13 (-0.31, 0.05)	0.158
Hypertension			
YES	2609 (32.75%)	ref	
NO	5357 (67.25%)	-0.22 (-0.27, -0.17)	< 0.0001
Coronary Heart Disease			
YES	270 (3.39%)	ref	
NO	7696 (96.61%)	-0.27 (-0.41, -0.13)	0.0001
Hypercholesterolemia			
YES	2460 (30.88%)	ref	
NO	5506 (69.12%)	-0.10 (-0.16, -0.05)	0.0002
Gout			
YES	1848 (23.20%)	ref	
NO	6118 (76.80%)	-0.19 (-0.24, -0.13)	< 0.0001
Stroke			
YES	241 (3.03%)	ref	
NO	7725 (96.97%)	-0.21 (-0.36, -0.07)	0.0035
Cancer Or Malignancy			
YES	647 (8.12%)	ref	
NO	7319 (91.88%)	0.09 (-0.00, 0.18)	0.053
Alcohol Use			

Table 2 (continued)

Exposure	Mean ± SD / *N*(%)	β (95%CI)	*P*-value
Never drinking	998 (12.53%)	ref	
Moderate drinking	6732 (84.51%)	-0.00 (-0.08, 0.07)	0.9458
Heavy drinking	236 (2.96%)	0.03 (-0.13, 0.19)	0.6907
Cigarette Use			
Never smoker	4225 (53.04%)	ref	
Former smoker	2379 (29.86%)	0.10 (0.05, 0.16)	0.0002
Current smoker	1362 (17.10%)	0.65 (0.58, 0.71)	< 0.0001

Table 3 Relationship between LAP and self-reported oral health outcomes in different models

Exposure	Unadjusted model	Model 1	Model 2
LAP log10 transform	0.42 (0.37, 0.48) < 0.0001	0.31 (0.25, 0.37) < 0.0001	0.26 (0.20, 0.32) < 0.0001
LAP log10 transformed three groups			
Low	ref	ref	ref
Medium	0.24 (0.18, 0.30) < 0.0001	0.16 (0.10, 0.22) < 0.0001	0.14 (0.08, 0.20) < 0.0001
High	0.40 (0.34, 0.46) < 0.0001	0.24 (0.18, 0.31) < 0.0001	0.21 (0.14, 0.27) < 0.0001

Results in table: β (95%CI) P-value

Outcome variable: self-reported oral health outcomes

Exposure variable: LAP

unadjusted model adjusts for: None

model 1 adjusts for gender, age, race, education level, RIP

model 2 adjusts for gender, age, race, education level, RIP, diabetes, hypertension, coronary heart disease, hypercholesterolemia, gout, stroke, cancer or malignancy, alcohol use and cigarette use

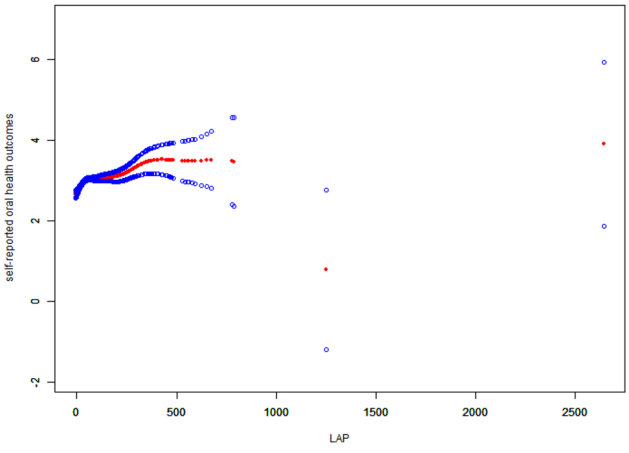

Fig. 2 The non-linear relationship between LAP and self-reported oral health outcomes. The solid red line illustrates the smooth curve fit between variables, with blue bands indicating the 95% confidence interval, while adjusting for confounding factors including gender, age, race, education level, the ratio of family income to poverty (RIP), diabetes, hypertension, coronary heart disease, hypercholesterolemia, gout, stroke, cancer or malignancy, alcohol use, and cigarette use

Table 4 Analysis of the threshold effect of LAP and self-reported oral health outcomes

Outcome:	Self-reported oral health outcomes
A straight-line effect	0.00 (0.00, 0.00) < 0.0001
Folding point (K)	83
< K Segment Effect 1	0.00 (0.00, 0.00) < 0.0001
> K Segment effect 2	0.00 (-0.00, 0.00) 0.1648
Difference in effect between 2 and 1	-0.00 (-0.00, -0.00) < 0.0001
Predicted value of the equation at the fold point	3.20 (3.15, 3.25)
Log-likelihood ratio test (p)	< 0.001

Results in table: β (95%CI) P-value

Outcome variable: self-reported oral health outcomes

Exposure variable: LAP

Adjusted variable: gender, age, race, education level, RIP, diabetes, hypertension, coronary heart disease, hypercholesterolemia, gout, stroke, cancer or malignancy, alcohol use and cigarette use

Addressed within this research is the inaugural demonstration of a correlation between the LAP and oral health. Furthermore, the sample size is substantial and dependable. This analysis was conducted retrospectively and cross-sectionally, limiting our ability to establish a causal relationship between LAP and oral health. Efforts to address numerous confounding variables have been made, yet unaccounted confounders may still affect the connection between LAP and oral health.

Conclusion

In conclusion, a significant association was observed between self-reported oral health outcomes and LAP within the American population, even after controlling for various confounding factors. This study proposes that future research should explore both the potential of LAP as a predictive biomarker for oral health outcomes and its applicability in clinical practice to guide personalized early interventions and prevention strategies.

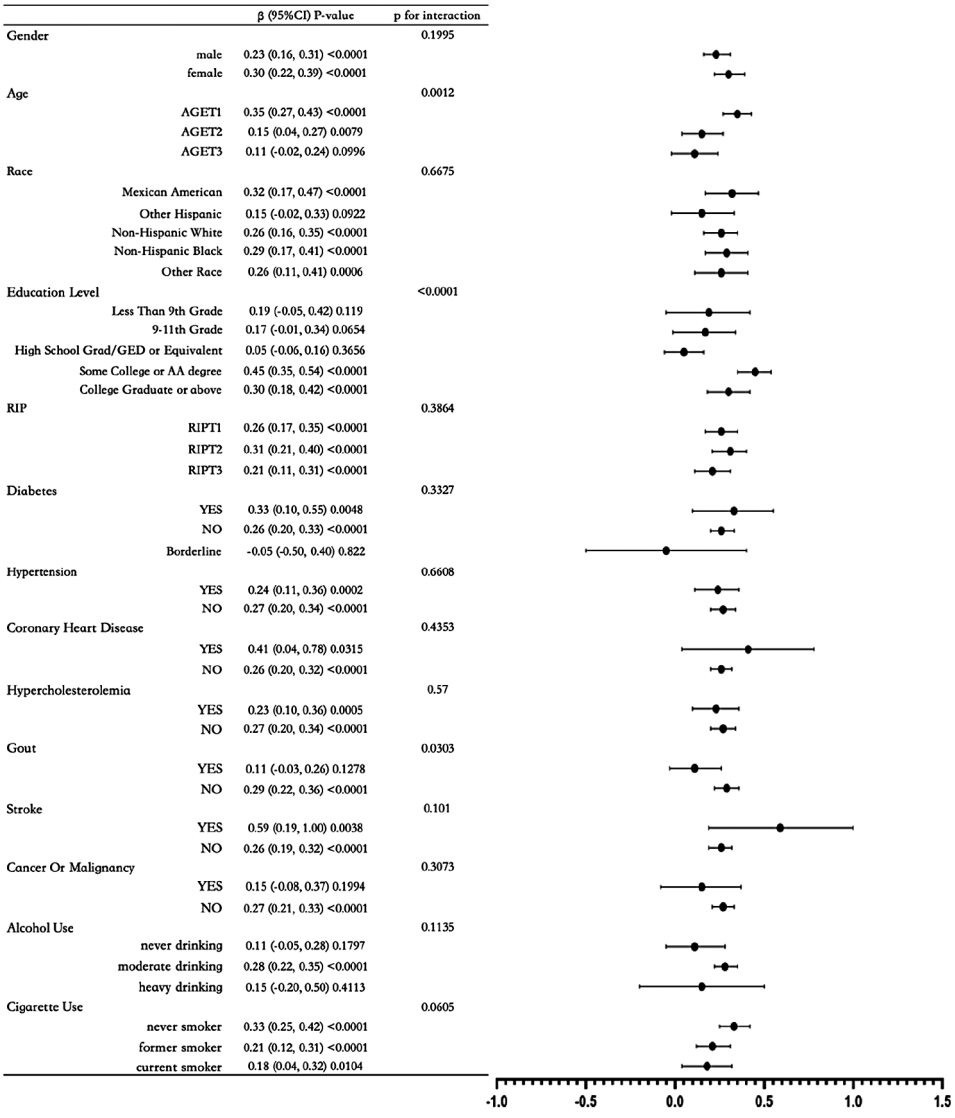

Fig. 3 The results of subgroup analyses

Abbreviations

LAP	Lipid Accumulation Product
NHANES	National Health and Nutrition Examination Survey
WC	Waist Circumference
TG	Triglycerides
PCOS	Polycystic Ovarian Syndrome
CI	Confidence Interval
MECs	Mobile Examination Centers
RIP	Ratio of Family Income to Poverty
OHQ	Oral Health Questionnaire

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12944-025-02543-4.

Supplementary Material 1: Attached Fig. 1 The association between LAP and self-reported oral health outcomes stratified by gender. In the diagram, red represents males, and blue represents females. Attached Fig. 2 The association between LAP and self-reported oral health outcomes stratified by age. In the diagram, red represents the low age group (AGET1), green represents the middle age group (AGET2), and blue represents the high age group (AGET3). Attached Fig. 3 The association between LAP and self-reported oral health outcomes stratified by Education Level. In the diagram, red represents "Less Than 9th Grade," yellow represents "9-11th Grade," green represents "High School Grad/GED or Equivalent," blue represents "Some College or AA degree," and purple represents "College Graduate or above.". Attached Fig. 4 The association between LAP and self-reported oral health outcomes stratified by Race. In the diagram, red represents "Mexican American," yellow represents "Other Hispanic," green represents "Non-Hispanic White," blue represents "Non-Hispanic Black," and purple represents "Other Race.". Attached Fig. 5 The association between LAP and self-reported oral health outcomes stratified by RIP. In the diagram, red represents the low RIP group (RIPT1), green represents the middle RIP group (RIPT2), and blue represents the high RIP group (RIPT3). Attached Fig. 6 The association between LAP and self-reported oral health outcomes stratified by Diabetes. In the diagram, red represents "YES," green represents "NO," and blue represents "Borderline.". Attached Fig. 7 The association between LAP and self-reported oral health outcomes stratified by Hypertension. In the diagram, red represents "YES," and blue represents "NO.". The association between LAP and self-reported oral health outcomes stratified by Coronary Heart Disease. In the diagram, red represents "YES," and blue represents "NO.". Attached Fig. 9 The association between LAP and self-reported oral health outcomes stratified by Hypercholesterolemia. In the diagram, red represents "YES," and blue repre-

sents "NO.". Attached Fig. 10 The association between LAP and self-reported oral health outcomes stratified by Gout. In the diagram, red represents "YES," and blue represents "NO.". Attached Fig. 11 The association between LAP and self-reported oral health outcomes stratified by Stroke. In the diagram, red represents "YES," and blue represents "NO.". Attached Fig. 12 The association between LAP and self-reported oral health outcomes stratified by Cancer Or Malignancy. In the diagram, red represents "YES," and blue represents "NO.". Attached Fig. 13 The association between LAP and self-reported oral health outcomes stratified by Alcohol Use. In the diagram, red represents "never drinking," green represents "moderate drinking," and blue represents "heavy drinking.". Attached Fig. 14 The association between LAP and self-reported oral health outcomes stratified by Cigarette Use. In the diagram, red represents " never smoker," green represents " former smoker," and blue represents " current smoker."

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Author contributions
(1) LSN was involved in the study design, literature review, data analysis, and manuscript preparation. (2) YW was involved in the development of the concept and was responsible for the critical review of the manuscript. (3) FXH carried out the data collection. (4) DJF and LJL were involved in the development of the concept and were responsible for the critical review of the manuscript.

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Data availability
Publicly available datasets were analyzed in this study. This data can be found here: www.cdc.gov/nchs/nhanes/.

Declarations

Ethics approval and consent to participate
Not applicable.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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