Sex differences in the association between dual-energy x-ray absorptiometry-measured body composition and periodontitis

Zhu, Peijun; Li, An; Cai, Qingqing; Chen, Yuntao; Liu, Yang; Jager-Wittenaar, Harriët; E. Tjakkes, Geerten Has; Xu, Shulan

Published in:
Journal of Periodontology

DOI:
10.1002/JPER.23-0162

Received: 12 March 2023 | Revised: 26 June 2023 | Accepted: 24 July 2023

DOI: 10.1002/JPER.23-0162

JOURNAL OF Periodontology

ORIGINAL ARTICLE

Sex differences in the association between dual-energy x-ray absorptiometry-measured body composition and periodontitis

Peijun Zhu[1] | **An Li**[2,3] | **Qingqing Cai**[4] | **Yuntao Chen**[5] | **Yang Liu**[6] | **Harriët Jager-Wittenaar**[7,8,9] | **Geerten-Has E. Tjakkes**[3] | **Shulan Xu**[1]

[1]Center of Oral Implantology, Stomatological Hospital, School of Stomatology, Southern Medical University, Guangzhou, China

[2]Department of Periodontology, Stomatological Hospital, School of Stomatology, Southern Medical University, Guangzhou, China

[3]Department of Periodontology, Center for Dentistry and Oral Hygiene, University Medical Center Groningen, University of Groningen, Groningen, The Netherlands

[4]Division of Nephrology, Nanfang Hospital, Southern Medical University, National Clinical Research Center of Kidney Disease, Guangzhou, China

[5]Department of Epidemiology and Public Health, University College London, London, UK

[6]Department of Oral Medicine, Stomatological Hospital, School of Stomatology, Southern Medical University, Guangzhou, China

[7]Research Group Healthy Ageing, Allied Health Care and Nursing, Hanze University of Applied Sciences, Groningen, The Netherlands

[8]Department of Oral and Maxillofacial Surgery, University of Groningen, University Medical Center Groningen, Groningen, The Netherlands

[9]Research Unit Experimental Anatomy, Faculty of Physical Education and Human Anatomy, Vrije Universiteit Brussel, Brussels, Belgium

Correspondence
Shulan Xu, Center of Oral Implantology, Stomatological Hospital, School of Stomatology, Southern Medical University, 366 Jiangnan S Ave, 510280, Guangzhou, China.
Email: xushulan_672588@smu.edu.cn

Funding information
Science Research Cultivation Program of Stomatological Hospital, Southern Medical University, China, Grant/Award Number: PY2021007; Clinical Research Initiation Plan of Stomatological Hospital, Southern Medical University, China, Grant/Award Number: KQIIT2021001; Guangdong Basic and Applied Basic Research Foundation, Grant/Award Number: 2022A1515110379

Abstract

Background: A positive association between obesity based on body mass index (BMI) and periodontitis has been reported. Fat tissue-related systemic inflammation acts as the link to periodontal comorbidities of obesity. However, the BMI is unable to distinguish fat and fat-free tissues. More precise measures are required to evaluate body composition, including fat and fat-free tissues. This study aimed to determine the sex differences in the association between dual-energy x-ray absorptiometry (DXA)-measured body composition (i.e., fat mass and muscle mass) and phenotypes with periodontitis.

Methods: Cross-sectional data of 3892 participants from the National Health and Nutrition Examination Survey (NHANES) study 2011–2014 were analyzed. Adiposity indices (fat mass index [FMI] and percentage body fat [%BF]) and muscle mass index (MMI) were calculated. The participants were categorized by the quintiles of FMI, MMI, and %BF. Body composition phenotypes were categorized as: low adiposity-low muscle (LA-LM), low adiposity-high muscle (LA-HM), high adiposity-low muscle (HA-LM), or high adiposity-high muscle (HA-HM), respectively. Periodontitis was defined by the CDC/AAP (Centers for Disease Control and Prevention/American Academy of Periodontology) criteria.

Peijun Zhu and An Li share the first authorship.

Multivariable logistic regression analysis was conducted, stratified by sex. We further adjusted for white blood cell (WBC) counts in the sensitivity analysis.

Results: Restricted cubic splines revealed non-linear associations between body composition indices and periodontitis risk. Women with a higher FMI (odds ratio for Q5 vs. Q1 [OR_{Q5vs1}] = 1.787, 95% confidence interval: 1.209–2.640) or %BF (OR_{Q5vs1} = 2.221, 1.509–3.268) had increased odds of periodontitis. In addition, women with HA-LM phenotype were more likely to develop periodontitis (OR = 1.528, 1.037–2.252). Interestingly, the WBC count, a systemic inflammatory biomarker, attenuated these associations. No statistically significant associations were found in men.

Conclusions: The association between DXA-measured body composition and phenotypes with periodontitis differs per sex. Only in women higher adiposity indices and HA-LM phenotype were associated with an increased risk of periodontitis.

KEYWORDS
body composition, cross-sectional study, dual-energy x-ray absorptiometry, periodontitis, sex difference

1 | INTRODUCTION

Periodontitis is a chronic inflammatory disease that destroys the tooth-supporting apparatus, due to an immune response to dysbiotic plaque biofilms.[1] With over 50% of the worldwide population having periodontitis, it is the sixth most prevalent global condition.[2] Notably, the role of obesity in developing periodontitis has been highlighted by the 2017 classification of periodontal diseases and conditions.[1] Obesity, a chronic inflammatory disease, shares common socioeconomic determinants and systemic risk factors with periodontal diseases.[3] Excessive adipose tissue functions as an active endocrine gland that releases chemokines and elicits systemic inflammation, worsening periodontal inflammation and increasing periodontitis risk.[4] A recent review indicated a positive dose-response association between obesity and periodontal disease, in which obesity was mainly measured by the body mass index (BMI).[5]

Although BMI is commonly used as a marker of obesity, it cannot distinguish between fat and fat-free components.[6] Specifically, BMI involves both fat and muscle mass; however, obesity solely refers to fat accumulation. Therefore, BMI-based obesity would lead to a measurement error. It has been reported that this definition of obesity may misclassify health risks among moderately overweight individuals.[6,7] A recent population-based study indicated a positive association between periodontitis and metabolically unhealthy phenotype among adults with overweight/obesity.[8] Undoubtedly, excessive fat accumulation plays a critical role in obesity comorbidities. Dual-energy x-ray absorptiometry (DXA)-measured body composition is a more accurate indicator of excess body fat, and is appropriate for confirming the risks associated with obesity.[6,7] Nevertheless, inadequate data on the association between DXA-measured body composition and periodontitis are available.

In prevalence data regarding both periodontal disease and obesity, sex differences have been reported. Man-to-woman prevalence ratios were 1.4 and 1.7 for ≥4 mm and ≥5 mm probing depths, respectively.[9] In addition, the overall prevalence of obesity in the US population was higher in women than in men.[10] However, sex differences in the associations between the two disorders are inconsistent.[11] Specifically, sex plays a critical role in obesity outcomes, with women having higher fat accumulation but fewer metabolic complications than men.[12] In contrast, the metabolic syndrome might have a stronger association with periodontitis in women than in men.[9] In addition, obese women exhibit more elevated systemic inflammatory markers, indicating a molecular interrelationship between obesity and periodontal diseases.[4,13] Therefore, whether the association between obesity and periodontitis differs in terms of sex is still obscure, and further studies are required.

DXA-measured body composition effectively distinguishes body composition components, including adipose and muscle tissues. DXA is widely used in epidemiological research to measure adiposity due to its very low radiation dose and accuracy.[14] A recent review on periodontitis

and obesity suggested that a DXA scan is a more accurate method for measuring adiposity.[5] To the best of our knowledge, few studies have explored the role of sex in the association of DXA-measured adiposity and muscle with periodontitis. Therefore, we investigated the sex-specific association between body composition and periodontitis, using DXA-measured body composition as a measuring tool for fat mass and muscle mass.

2 | MATERIALS AND METHODS

2.1 | Study population

Cross-sectional data were obtained from the 2011 to 2014 National Health and Nutrition Examination Survey (NHANES) with full-mouth periodontal measurements and DXA examinations. NHANES collected nationally representative health-related data on the US non-institutionalized civilians using a stratified, multistage probability sampling design.[15] Participants with missing periodontal exams and incomplete DXA body composition data were excluded. All survey protocols were approved by the National Center for Health Statistics Ethics Review Board. All the participants provided written informed consent before participation. We reported this study following the reporting of observational studies in epidemiology (STROBE) criteria.[16]

2.2 | Body composition based on whole-body DXA data

Using a fan-beam densitometer,[*] whole-body DXA scans defined the body composition based on their specific x-ray attenuation properties. Subsequent analyses were conducted using software.[†] The NHANES body composition procedures manual has been detailed on the NHANES website. Each subject's whole-body DXA measurements of fat mass (representing lipid tissue) and muscle mass (representing lipid-free/muscle tissue) were included in the NHANES datasets. In addition, each subject's height was measured with a precision stadiometer after deep inspiration. The whole-body DXA measurements were used to calculate the fat mass index (FMI, fat mass (kg)/height $(m)^2$), muscle mass index (MMI, muscle mass (kg)/height $(m)^2$), and percentage body fat (%BF, fat mass/total mass). Moreover, the four-body composition phenotype model was defined based on the sex-specific medians of FMI and MMI.[17] The participants were categorized into:

1. Low adiposity and low muscle (LA-LM, FMI < median and MMI < median),
2. Low adiposity and high muscle (LA-HM, FMI < median and MMI ≥ median),
3. High adiposity and low muscle (HA-LM, FMI ≥ median and MMI < median),
4. High adiposity and high muscle (HA-HM, FMI ≥ median and MMI ≥ median).

We set LA-LM as a reference group.

2.3 | Periodontal examination

Trained examiners conducted a full-mouth periodontal assessment according to a standardized protocol. Periodontal assessments were carried out at six sites (mesiobuccal, mid-buccal, distobuccal, mesiolingual, mid-lingual, and distolingual), consisting of probing pocket depths (PPD) and attachment loss (AL) for each tooth in four quadrants. The inter-class correlation coefficients of PPD and AL, which varied from 0.80 to 0.90 and 0.79 to 0.86, respectively, reflect their excellent dependability.[18] According to the CDC/AAP case definition (Centers for Disease Control & Prevention/American Academy of Periodontology),[19] periodontal status was defined as:

1. Moderate periodontitis: at least two inter-proximal sites (not on the same tooth) with an AL of at least 4 mm or with a PPD of at least 5 mm,
2. Severe periodontitis: at least two inter-proximal sites (not on the same tooth) with an AL of at least 6 mm and at least one inter-proximal site with a PPD of at least 5 mm,
3. No/mild periodontitis: no evidence of periodontitis mentioned above.

All the participants were divided into no/mild periodontitis and moderate/severe periodontitis categories, as previously reported.[20]

2.4 | Covariate assessments

Specific covariates were analyzed for descriptive and inferential statistics in this study. In particular, age, race/ethnicity (non-Hispanic white, non-Hispanic black, and others), educational level (≤high school, college, and >college), and annual household income (< 20,000$, 20,000-75,000$, and >75,000$) were included as sociodemographic variables. Behavioral variables included alcohol intake (≥12 drinks/year and <12 drinks/year), smoking status (never smoked, former smoker, and current

[*] Hologic, Inc., Bedford, Massachusetts, USA.
[†] Hologic APEX version 4.0 software

smoker), and physical activity (inactive, somewhat active, and active). Self-reported physical activity was classified by calculating metabolic equivalent (MET)-min/week for household, transportation, and recreational physical activity. We categorized participants as inactive (no reported physical activity), somewhat active (>0 to <500 MET-min/week), and active (≥500 MET-min/week), as previously described.[21] Additional health conditions of interest included diabetes, hypertension, dyslipidemia, cardiovascular disease, and cancer. The detailed definitions of health conditions are shown in Table S1 in the online *Journal of Periodontology*, as previously described.[20] Serum lipids (total and high-density lipoprotein cholesterol, triglycerides) and white blood cell (WBC) counts were also considered. We pre-defined confounders to make adjustments in multivariable regression models consisting of known or suspected risk factors for obesity and periodontitis.[4] Potential confounders were also selected using a directed acyclic graph.[22]

2.5 | Statistical analysis

The population characteristics in terms of body composition phenotypes were analyzed using the variance analysis for continuous variables (reported as mean ± standard deviation) and the chi-squared test for categorical variables (reported as percentages). All statistical analyses were conducted by considering the survey design and weighting. The primary aim of the present study was to determine the association between DXA-derived body composition and periodontitis risk, with periodontal status as the outcome variable. The exposure variables were body composition indices (FMI, MMI, and %BF) and body composition phenotypes (LA-LM, LA-HM, HA-LM, and HA-HM). Notably, the interactive effect of sex-body composition on periodontitis status was statistically significant in preliminary statistics (Table S2 in online *Journal of Periodontology*). Therefore, all analyses in this work were stratified in terms of sex. A restricted cubic spline identified the dose-response associations of FMI, MMI, and %BF with moderate/severe periodontitis. Multivariable logistic models were used to estimate the association of FMI, MMI, %BF quintiles, and body composition phenotypes with moderate/severe periodontitis. We adjusted for age and race/ethnicity in Model 2. In the fully adjusted model, we adjusted for educational level, annual household income, alcohol intake, smoking status, physical activity, number of teeth, diabetes, and dyslipidemia. In addition, we further adjusted for WBC counts in the multivariable regression analyses to examine whether the systemic inflammation could explain the observed association. In the sensitivity analysis, we further adjusted the female hormone therapy in the women population, and the associations between body composition indices and phenotypes with mean PPD and AL were further analyzed, as well as the associations between BMI and waist-height ratio with periodontitis risk. Due to the low proportion of missing data (<5%), we conducted a complete case analysis in this study. All statistical analyses were performed using software.[‡] We considered a two-sided p-value of <0.05 as statistically significant.

3 | RESULTS

3.1 | Characteristics and body composition phenotypes

Participants without complete periodontal and DXA data were excluded, leaving 3892 participants (1959 men and 1933 women) eligible for inclusion in the analysis (Figure 1). Participant characteristics by body composition were presented for men and women, as shown in Table 1, and characteristics of the study population by sex are shown in Table S3 in the online *Journal of Periodontology*. Of 3892 participants, the average age was 44.0 ± 8.6, and the overall percentages of participants with LA-LM, LA-HM, HA-LM, and HA-HM phenotypes were 37.2%, 12.8%, 12.8%, and 37.2%, respectively. The percentages of participants with LA-HM and HA-LM phenotypes were significantly higher in men than in women. Compared with the other phenotypes, participants with the LA-HM phenotype tended to be younger, non-Hispanic black, and physically active; they also exhibited a lower prevalence of cancer. On the other hand, participants with HA-LM and HA-HM phenotypes were more likely to report prevalent cardiovascular disease (CVD), hypertension, diabetes, and dyslipidemia and were less likely to be physically active.

3.2 | Body composition indices and periodontitis

In men, restricted cubic spline analyses revealed statistically significant U-shaped associations for FMI, MMI, and %BF with moderate/severe periodontitis ($p_{\text{non-linearity}} < 0.05$, Figure 2A,C,E). In women, the FMI ($p_{\text{non-linearity}} = 0.015$) and %BF ($p_{\text{non-linearity}} = 0.042$) also had a non-linear association with moderate/severe periodontitis (Figure 2B,F). However, the non-linear association between MMI and periodontitis risk was insignificant in women ($p_{\text{non-linearity}} = 0.076$, Figure 2D). Based on these findings, we grouped FMI, MMI, and %BF into quintiles.

[‡] R Project (version 4.2.1).

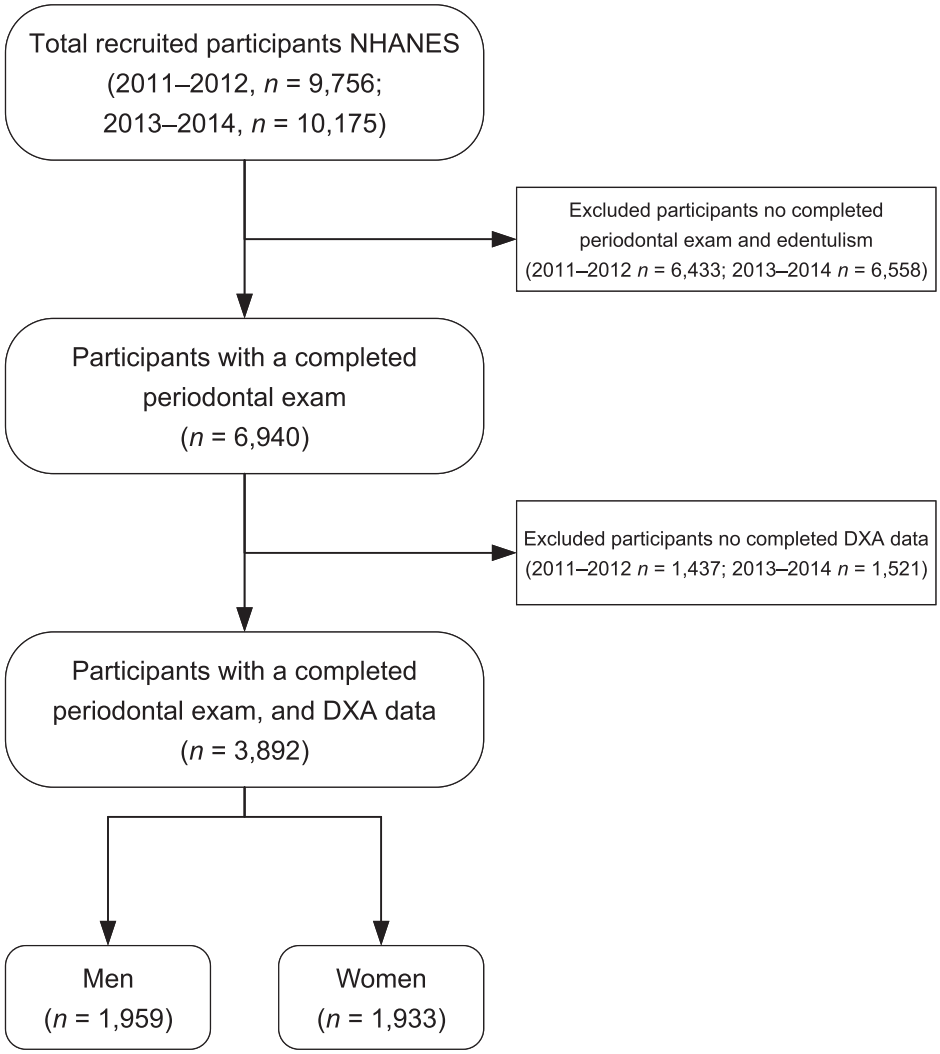

FIGURE 1 Flow diagram of final sample from the NHANES 2011-2014. DXA, dual-energy x-ray absorptiometry. NHANES, National Health and Nutrition Examination Survey.

The risk of moderate/severe periodontitis in quintiles of FMI, MMI, and %BF was estimated and stratified in terms of sex (Table 2). In men, periodontitis risk did not significantly differ with higher quintiles of FMI, MMI, and %BF. In women, FMI was associated with greater odds of periodontitis in the unadjusted and age- and race/ethnicity-adjusted models. After full adjustment, woman participants in the highest quintile of FMI exhibited a 78.7% higher risk of periodontitis than those in the lowest quintile (odds ratio for Q5 vs. Q1 [OR_{Q5vs1}] = 1.787, 95% CI: 1.209–2.640). Similarly, women in the higher quintile of %BF had a significantly higher periodontitis risk than women in the lowest quintile (OR_{Q5vs1} = 2.221, 95% CI: 1.509–3.268). Moreover, the observed associations were attenuated after adjustment for WBC counts (OR_{Q5vs1} for FMI = 1.641 [1.097–2.457]; OR_{Q5vs1} for %BF = 2.105 [1.419–3.121]) (Table S4 in online *Journal of Periodontology*). No significant association was found between MMI quintiles and periodontitis in women. In the sensitivity analysis, taking account of female hormone therapy, the consistent results on the association of FMI and %BF with periodontitis were found (OR_{Q5vs1} for FMI = 1.873 [1.265–2.775]; OR_{Q5vs1} for %BF = 2.320 [1.571-3.424]) (Table S5 in online *Journal of Periodontology*). In the sensitivity analysis, we analyzed the associations of BMI and waist-height ratio (WHI) with periodontitis. BMI was significantly associated with periodontitis in women instead of men, consistent with the results of DXA-derived measures (Table S6 in online *Journal of Periodontology*).

3.3 | Body composition phenotypes and periodontitis

Figure 3A shows the associations between moderate/severe periodontitis and four body composition

TABLE 1 Characteristics of the study population by sex and body composition.

	Overall ($n = 3892$)	Men ($n = 1959$)				Women ($n = 1933$)			
		LA-LM ($n = 673$)	LA-HM ($n = 306$)	HA-LM ($n = 307$)	HA-HM ($n = 673$)	LA-LM ($n = 775$)	LA-HM ($n = 191$)	HA-LM ($n = 191$)	HA-HM ($n = 776$)
Continuous variables, mean (SD)									
Age, years	44.03 (8.57)	44.08 (8.85)	41.88 (8.30)	45.81 (8.66)	43.56 (8.53)	43.96 (8.45)	41.80 (7.61)	46.28 (8.68)	44.59 (8.43)
BMI, kg/m^2	29.04 (6.50)	23.60 (2.28)	27.32 (1.96)	28.31 (1.99)	34.08 (5.08)	23.21 (2.64)	27.52 (2.14)	29.23 (1.97)	36.53 (5.76)
Waist-height ratio, cm/cm	0.59 (0.09)	0.51 (0.04)	0.53 (0.04)	0.59 (0.04)	0.65 (0.07)	0.52 (0.05)	0.57 (0.05)	0.61 (0.04)	0.69 (0.08)
SBP, mmHg	120.16 (15.37)	119.64 (15.22)	122.85 (13.43)	121.03 (13.54)	125.92 (14.96)	114.35 (15.15)	116.45 (13.42)	116.49 (15.27)	121.90 (15.54)
DBP, mmHg	73.16 (11.14)	72.52 (10.33)	74.61 (10.40)	75.37 (10.75)	77.07 (11.95)	70.28 (10.11)	70.42 (10.51)	71.03 (11.15)	72.98 (11.47)
Glucose, mg/dL	102.60 (42.00)	99.79 (34.30)	97.84 (34.93)	105.24 (39.37)	111.29 (50.69)	95.18 (38.39)	99.79 (41.14)	100.11 (36.63)	106.97 (46.02)
HbA1c, %	5.72 (1.15)	5.59 (0.97)	5.58 (0.91)	5.69 (1.06)	6.02 (1.38)	5.45 (0.83)	5.68 (1.38)	5.65 (1.14)	5.94 (1.32)
Total cholesterol, mg/dL	197.72 (42.36)	195.82 (41.12)	192.31 (36.89)	203.21 (41.02)	199.56 (43.00)	196.91 (39.90)	191.61 (41.19)	203.36 (40.33)	198.56 (47.76)
HDL cholesterol, mg/dL	51.96 (15.57)	51.99 (15.48)	49.23 (12.69)	45.33 (10.86)	42.38 (9.95)	62.80 (17.81)	55.18 (14.92)	55.72 (13.93)	51.35 (13.00)
Triglycerides, mg/dL[a]	123.0 (116.0)	118.5 (113.0)	119.0 (112.0)	159.5 (135.0)	169.0 (163.0)	94.0 (80.0)	107.0 (102.0)	117.0 (95.5)	125.0 (105.0)
WBC, 1000 cells/μL[b]	7.15 (2.09)	6.93 (2.06)	6.62 (2.04)	7.24 (1.92)	7.31 (1.93)	6.71 (1.97)	7.19 (2.00)	7.28 (1.98)	7.79 (2.30)
No. of teeth[a]	26.00 (5.00)	26.00 (6.00)	27.00 (5.00)	27.00 (4.00)	26.00 (4.00)	27.00 (4.00)	26.00 (4.00)	26.00 (5.00)	26.00 (6.00)
FMI	10.02 (4.26)	5.70 (1.26)	6.26 (1.07)	9.05 (1.25)	10.77 (3.01)	8.31 (1.81)	9.79 (1.39)	13.00 (1.26)	16.03 (3.55)
MMI	7.98 (1.68)	7.70 (0.69)	9.51 (0.69)	8.04 (0.53)	10.12 (1.14)	5.87 (0.61)	7.44 (0.60)	6.30 (0.44)	8.39 (1.21)
%BF	33.35 (8.25)	23.76 (3.84)	22.64 (3.01)	31.74 (2.78)	31.06 (4.16)	35.22 (4.69)	35.27 (3.50)	44.25 (2.44)	43.51 (3.55)
Categorical, *n* (%)									
Race/ethnicity									
Non-Hispanic White	1535 (39.4)	263 (39.1)	101 (33.0)	147 (47.9)	270 (40.1)	333 (43.0)	61 (31.9)	94 (49.2)	266 (34.3)
Non-Hispanic Black	809 (20.8)	99 (14,7)	110 (35.9)	20 (6.5)	168 (25.0)	67 (8.6)	73 (38.2)	16 (8.4)	256 (33.0)
Other	1548 (39.8)	311 (46.2)	95 (31.0)	140 (45.6)	235 (34.9)	375 (48.4)	57 (29.8)	81 (42.4)	254 (32.7)
Education level									
≤High school	1560 (40.1)	309 (45.9)	129 (42.2)	115 (37.5)	292 (43.4)	250 (32.3)	62 (32.5)	74 (38.7)	329 (42.4)
College	1161 (29.8)	150 (22.3)	83 (27.1)	92 (30.0)	222 (33.0)	202 (26.1)	67 (35.1)	70 (36.6)	275 (35.4)
>College	1171 (30.1)	214 (31.8)	94 (30.7)	100 (32.6)	159 (23.6)	323 (41.7)	62 (32.5)	47 (24.6)	172 (22.2)
Annual household income [b]									
<20,000$	639 (16.4)	124 (19.3)	38 (13.0)	44 (14.9)	100 (15.4)	106 (14.3)	27 (14.9)	39 (21.4)	161 (21.4)
20,000−75,000$	1818 (46.7)	293 (45.6)	162 (55.3)	128 (43.4)	325 (50.1)	315 (42.6)	99 (54.7)	87 (47.8)	409 (54.3)
>75,000$	1278 (32.8)	225 (35.0)	93 (31.7)	123 (41.7)	224 (34.5)	319 (43.1)	55 (30.4)	56 (30.8)	183 (24.3)
Alcohol intake ≥12 drinks/year	2799 (71.9)	543 (85.8)	251 (87.5)	254 (86.4)	563 (86.2)	479 (67.8)	125 (71.4)	121 (66.9)	463 (63.2)

(Continues)

TABLE 1 (Continued)

	Overall ($n = 3892$)	Men ($n = 1959$)				Women ($n = 1933$)			
		LA-LM ($n = 673$)	LA-HM ($n = 306$)	HA-LM ($n = 307$)	HA-HM ($n = 673$)	LA-LM ($n = 775$)	LA-HM ($n = 191$)	HA-LM ($n = 191$)	HA-HM ($n = 776$)
Smoking status[b]									
Never smoker	2216 (56.9)	302 (44.9)	154 (50.3)	154 (50.2)	360 (53.6)	506 (65.3)	118 (61.8)	119 (62.3)	503 (64.8)
Former smoker	767 (19.7)	131(19.5)	72 (23.5)	84 (27.4)	162 (24.1)	127 (16.4)	37 (19.4)	28 (14.7)	126 (16.2)
Current smoker	907 (23.3)	239 (35.6)	80 (26.1)	69 (22.5)	150 (22.3)	142 (18.3)	36 (18.8)	44 (23.0)	147 (18.9)
Physical activity (MET min/week)									
Inactive	791 (20.3)	103 (15.3)	31 (10.1)	63 (20.5)	103 (15.3)	176 (22.7)	28 (14.7)	64 (33.5)	223 (28.7)
Somewhat active	518 (13.3)	77 (11.4)	18 (5.9)	41 (13.4)	84 (12.5)	123 (15.9)	23 (12.0)	25 (13.1)	127 (16.4)
Active	2583 (66.4)	493 (73.3)	257 (84.0)	203 (66.1)	486 (72.2)	476 (61.4)	140 (73.3)	102 (53.5)	426 (54.9)
Time since the last dental visit [b]									
Less than 1 year	2208 (56.7)	341 (50.9)	176 (57.5)	182 (59.3)	354 (52.7)	502 (64.8)	112 (58.6)	103 (53.9)	438 (56.4)
1–3 years	824 (21.2)	122 (18.2)	54 (17.6)	63 (20.5)	148 (22.0)	159 (20.5)	49 (25.7)	53 (27.7)	176 (22.7)
More than 3 years	856 (22.0)	207 (30.9)	76 (24.8)	62 (20.2)	170 (25.3)	114 (14.7)	30 (15.7)	35 (18.3)	162 (20.9)
Diabetes[b]	335 (8.6)	44 (6.6)	15 (5.0)	27 (9.0)	81 (12.5)	27 (3.5)	17 (9.1)	18 (9.7)	106 (14.2)
Hypertension[b]	1094 (28.1)	128 (19.0)	64 (20.9)	98 (31.9)	254 (37.7)	126 (16.3)	41 (21.5)	56 (29.5)	327 (42.1)
Dyslipidemia[b]	1203 (30.9)	181 (26.9)	91 (29.9)	121 (39.7)	257 (38.5)	184 (23.7)	42 (22.0)	75 (39.7)	252 (32.6)
CVD [b]	91 (2.3)	11 (1.6)	4 (1.3)	8 (2.6)	26 (3.9)	8 (1.0)	2 (1.0)	5 (2.6)	27 (3.5)
Cancer	175 (4.5)	26 (3.9)	6 (2.0)	10 (3.3)	20 (3.0)	45 (5.8)	4 (2.1)	12 (6.3)	52 (6.7)

Abbreviations: %BF, percentage body fat; BMI, body mass index; CVD, cardiovascular disease; DBP, diastolic blood pressure; FMI, fat mass index; HA-HM, high adiposity and high muscle; HA-LM, high adiposity and low muscle; HbA1c, glycohaemoglobin A1c; HDL, high-density lipoprotein; LA-HM, low adiposity and high muscle; LA-LM, low adiposity and low muscle; MET, metabolic equivalent; MMI, muscle mass index; SBP, systolic blood pressure; SD, standard deviation; WBC, white blood cell count.

[a]Non-normal distribution continuous variable, median (interquartile range).

[b]Missing values for total study ($n = 3892$): WBC ($n = 103$; 2.6%), annual household income ($n = 157$; 4.0%), smoking status ($n = 2$; <0.1%), time since the last dental visit ($n = 4$; 0.1%), diabetes ($n = 92$; 2.4%), hypertension ($n = 2$; <0.1%), dyslipidemia ($n = 15$; 0.4%), and CVD ($n = 4$; 0.1%).

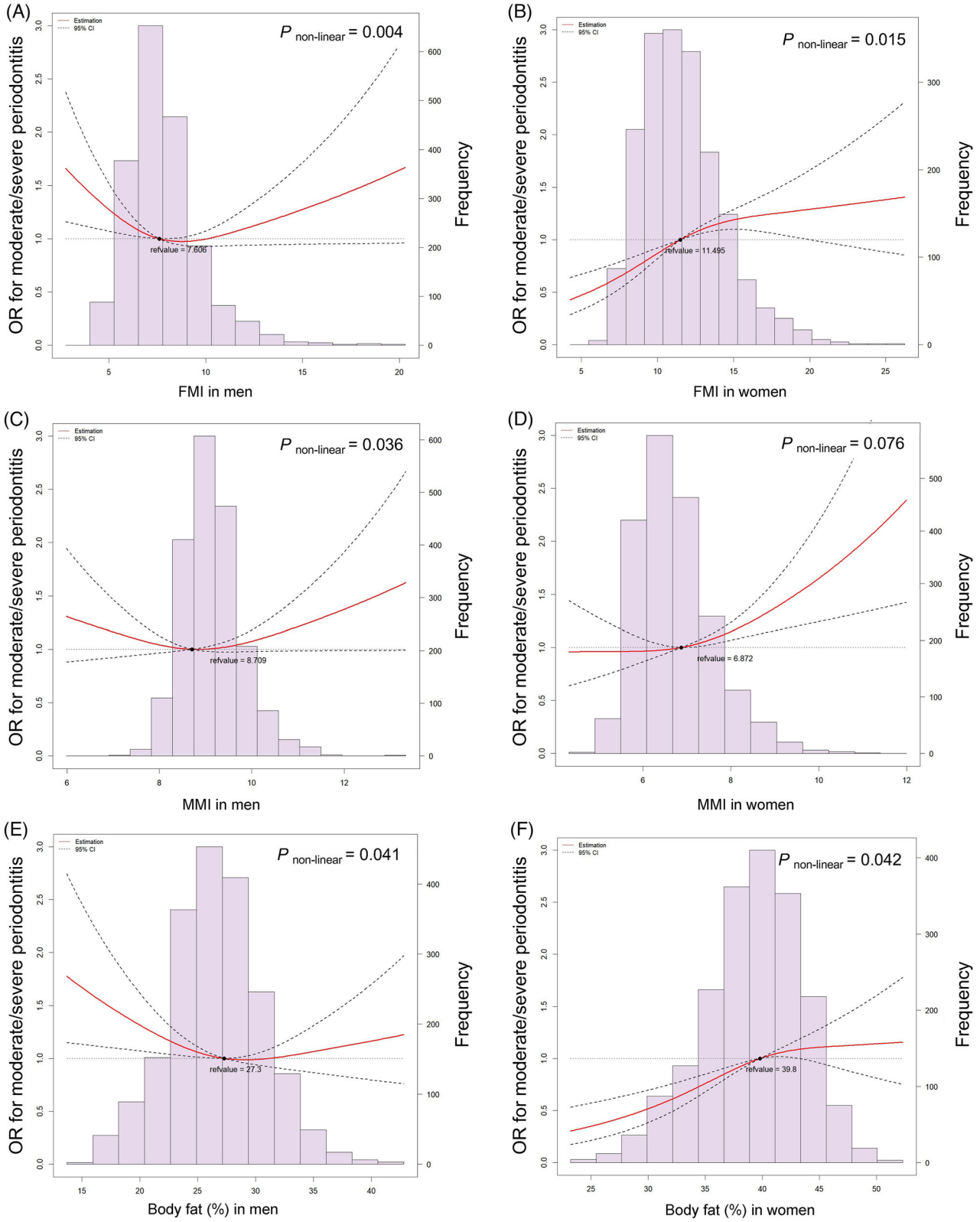

FIGURE 2 Restricted cubic spline of the association between (A)–(B) fat mass index, (C)–(D) muscle mass index, and (E)–(F) percentage body fat with moderate/severe periodontitis in men and women from the NHANES 2011–2014. The solid red line indicates the odds ratio values for moderate/severe periodontitis. Dotted lines represent point-wise 95% confidence intervals. Spline analyses were adjusted for age and race/ethnicity. %BF, percentage body fat; FMI, fat mass index; MMI, muscle mass index; NHANES, National Health and Nutrition Examination Survey.

TABLE 2 Weighted associations between the quintiles of the fat mass index, muscle mass index, and percentage body fat with moderate/severe periodontitis in male and female populations, National Health and Nutrition Examination Survey 2011–2014.

	Men				Women			
	Case/participants	Model 1[a] OR (95% CI)	Model 2[a] OR (95% CI)	Model 3[a] OR (95% CI)	Case/participants	Model 1[a] OR (95% CI)	Model 2[a] OR (95% CI)	Model 3[a] OR (95% CI)
FMI quintiles								
1st	204/391	1 Reference	1 Reference	1 Reference	86/386	1 Reference	1 Reference	1 Reference
2nd	178/392	0.762 (0.576–1.010)	**0.733 (0.537–0.999)**	0.837 (0.594–1.179)	118/387	**1.530 (1.108–2.114)**	**1.405 (1.010–1.955)**	1.411 (0.956–2.083)
3rd	194/393	0.894 (0.675–1.183)	0.842 (0.619–1.145)	0.914 (0.653–1.279)	145/387	**2.090 (1.524–2.867)**	**1.858 (1.342–2.571)**	**1.740 (1.184–2.556)**
4th	191/392	0.871 (0.658–1.153)	0.846 (0.622–1.150)	0.990 (0.705–1.390)	149/387	**2.184 (1.593–2.993)**	**1.950 (1.410–2.698)**	**1.534 (1.042–2.260)**
5th	194/391	0.903 (0.682–1.195)	0.926 (0.680–1.261)	1.042 (0.738–1.473)	164/386	**2.577 (1.884–3.525)**	**2.275 (1.643–3.151)**	**1.787 (1.209–2.640)**
MMI quintiles								
1st	202/391	1 Reference	1 Reference	1 Reference	120/386	1 Reference	1 Reference	1 Reference
2nd	192/392	0.898 (0.679–1.189)	0.872 (0.642–1.183)	0.809 (0.579–1.130)	117/387	0.961 (0.707–1.304)	1.045 (0.741–1.475)	1.017 (0.704–1.468)
3rd	180/392	0.794 (0.600–1.052)	0.828 (0.609–1.125)	0.847 (0.607–1.182)	128/387	1.095 (0.810–1.482)	1.176 (0.837–1.652)	1.048 (0.728–1.507)
4th	190/392	0.880 (0.665–1.165)	0.900 (0.663–1.221)	0.853 (0.611–1.192)	140/387	1.256 (0.932–1.694)	1.279 (0.906–1.804)	0.973 (0.672–1.408)
5th	197/392	0.945 (0.714–1.251)	0.955 (0.697–1.308)	1.015 (0.718–1.436)	157/386	**1.520 (1.130–2.044)**	**1.477 (1.041–2.095)**	1.184 (0.811–1.728)
%BF quintiles								
1st	207/400	1 Reference	1 Reference	1 Reference	81/388	1 Reference	1 Reference	1 Reference
2nd	194/399	0.882 (0.669–1.165)	0.865 (0.636–1.177)	0.934 (0.667–1.306)	136/391	**2.021 (1.466–2.787)**	**1.893 (1.319–2.717)**	**1.480 (1.005–2.179)**
3rd	194/392	0.914 (0.691–1.207)	0.878 (0.648–1.190)	0.946 (0.678–1.321)	148/396	**2.262 (1.645–3.110)**	**2.258 (1.585–3.217)**	**1.930 (1.316–2.829)**
4th	175/382	0.788 (0.595–1.044)	**0.733 (0.538–0.998)**	0.920 (0.656–1.290)	126/372	**1.941 (1.402–2.689)**	**1.750 (1.216–2.519)**	1.348 (0.909–2.001)
5th	191/386	0.913 (0.690–1.208)	0.976 (0.716–1.331)	1.147 (0.813–1.620)	171/386	**3.014 (2.196-4.137)**	**2.701 (1.891–3.857)**	**2.221 (1.509–3.268)**

Note: Bold indicates p-value < 0.05.

Abbreviations: %BF, percentage body fat; CI, confidence interval; FMI, fat mass index; MMI, muscle mass index; NHANES, National Health and Nutrition Examination Survey; OR, odds ratio.

[a]Model 1 was unadjusted; Model 2 was adjusted for age and race/ethnicity; Model 3 was further adjusted for education level, annual household income, alcohol intake, smoking status, physical activity, number of teeth, diabetes, and dyslipidemia.

phenotypes: LA-LM, LA-HM, HA-LM, and HA-HM. The DXA-measured body composition phenotypes in men were not associated with periodontitis risk. Women with HA-HM phenotype were more likely to have moderate/severe periodontitis than those with LA-LM in the age- and race/ethnicity-adjusted model (odds ratio [OR] = 1.478, 95% confidence interval [CI]: 1.158–1.885). However, this significant association was not demonstrated in the fully adjusted model (OR = 1.170, 95% CI: 0.897–1.526) (Figure 3B). Compared to women with LA-LM phenotype, those with HA-LM phenotype exhibited a higher risk of periodontitis, with age- and race/ethnicity-adjusted odds ratios of 1.705 (95% CI: 1.186–2.452) and fully adjusted odds ratios of 1.528 (95% CI: 1.037–2.252) (Figure 3B). After further adjusting for WBC counts, the association between HA-LM and periodontitis was attenuated (OR = 1. 493, 1.008–2.212) (Table S3 in online *Journal of Periodontology*), indicating that systemic

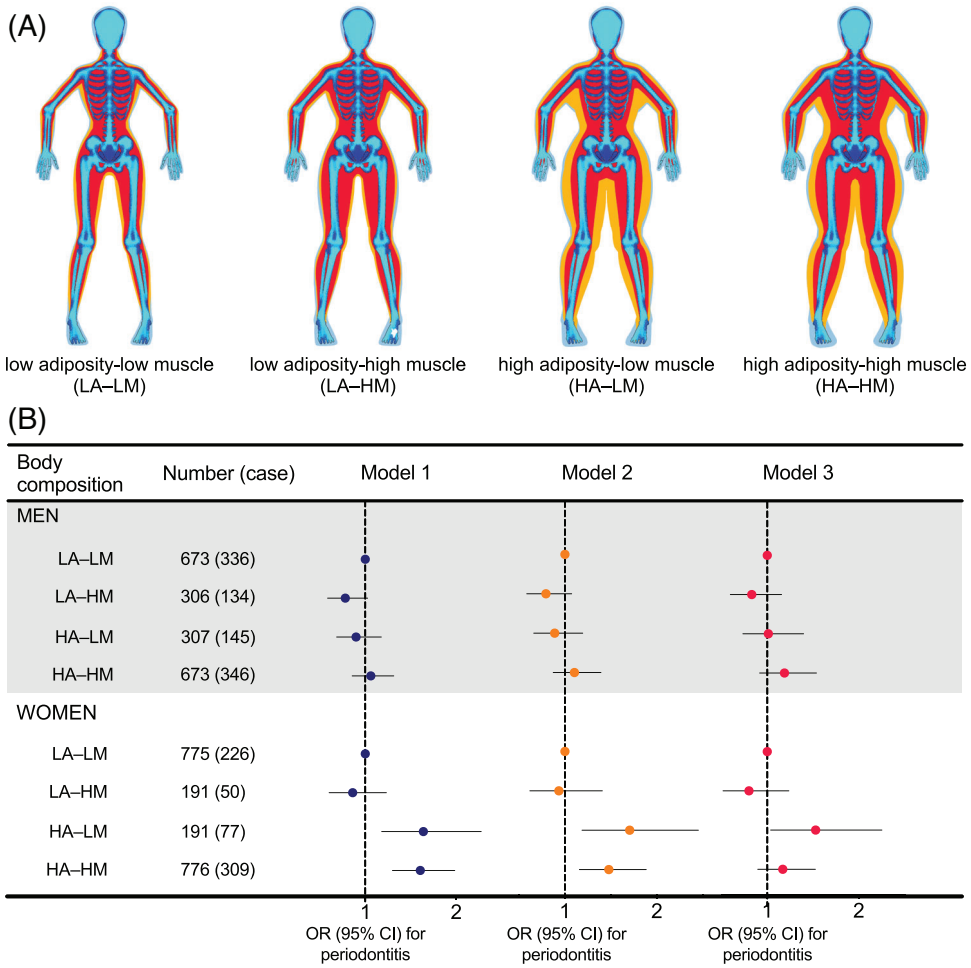

FIGURE 3 The association between body composition phenotypes and periodontitis. (A) The schematic diagram for body composition phenotypes (B) OR of body composition phenotypes for moderate/severe periodontitis in man and woman populations. The 95% CI, not including zero, was considered for statistical significance. Potential confounders in the adjusted model are detailed in the legend of Table 2. CI, confidence interval; OR, odds ratio.

inflammation could explain the observed association. Similar results were found when female hormone therapy was adjusted in women (OR = 1.518, 1.027–2.242) (Table S4 in online *Journal of Periodontology*). We further analyzed the multiplicative and additive interaction. No significant interaction effects of adiposity and muscle on periodontitis risk were found either in women or men (Table S7 in online *Journal of Periodontology*). The sensitivity analyses investigated the relationships of body components with mean AL and mean PPD and provided similar results (Tables S8–S9 in online *Journal of Periodontology*).

4 | DISCUSSION

The present study shows that the association between DXA-measured body composition indices and phenotypes with periodontitis differs per sex. Higher FMI and %BF in women were associated with greater odds of moderate/severe periodontitis after adjustment for potential confounders. Regarding the association between body composition phenotypes and periodontitis risk, women with the HA-LM phenotype were more likely to have periodontitis than those with the LA-LM phenotype. These associations were attenuated after adjustment for WBC counts. In contrast, no significant associations between body composition indices and phenotypes with periodontitis were observed in men.

In the present study, the sex differences in the association between DXA-measured adiposity and periodontitis brought more focus on the comparison with previous literature. Our findings are consistent with a 5-year longitudinal study demonstrating an association between obesity and periodontitis progression in women (relative risk [RR] = 1.64, 95% CI: 1.11–2.43) but not in men (RR = 1.13, 95% CI: 0.75–1.69).[23] Conversely, some other

studies provided a conflicting perspective that men are more likely to develop obesity-associated hyperinflammation and comorbidities.[24,25] Some prior studies suggested no significant sex differences regarding obesity and periodontitis.[26,27] The effect of sex on the association between obesity and periodontitis has been controversial. The DXA technique used to distinguish fat from muscle tissues and determine body fat percentage has been recognized as a more accurate method for adiposity measurement,[5] with the DXA body composition reported to be associated with mortality.[28] However, no study had previously examined the association between DXA-measured body composition and periodontitis risk. Our study helps to bridge this knowledge gap. According to the results, higher fat mass and proportion were associated with increased periodontitis risk in women but not men. These findings provide new insights into the sex paradox concerning the association between obesity and periodontitis.

Our work is in line with findings from a previously conducted Mendelian randomization with respect to the potential causal association between obesity and periodontitis,[29] suggesting that obesity is a risk factor exacerbating periodontal status. The possible mechanisms behind this interrelationship include hyperinflammation, immune dysfunction, microbiome, insulin resistance, genetics, body homeostasis, nutrition, and diet.[5] In the present study, the obesity–periodontitis association can be explained by systemic inflammation. Adiposity-related hyperinflammation is the center of the various pathways underlying the connections between obesity and periodontitis.[4,6] Specifically, the increased inflammation causes immune cell dysfunction, changes the microbiome composition and modifies the host's immune response to bacteria.[30,31]

Another important finding of the current study is that women with high adiposity-low muscle phenotype were more likely to have periodontitis, indicating that high-adiposity mass and low-muscle mass may be synergistically associated with higher periodontitis risk. To the best of our knowledge, no previous study has examined the association between adiposity-muscle phenotypes and periodontal diseases. Cross-sectional studies on the association between grip strength/sarcopenia and periodontitis have indicated that sarcopenia/low grip strength may be associated with a higher periodontitis risk.[32,33] Specifically, low muscle mass was reportedly associated with elevated levels of C-reactive protein (CRP) and pro-inflammation cytokines as biomarkers of chronic systemic inflammation.[34] Furthermore, the skeletal muscle is the most important organ to absorb and metabolize glucose by regulating insulin activity.[35] Targeted disruption of muscle-specific glucose transporter contributes to severe insulin resistance.[36] Systemic inflammation and insulin resistance are crucial mechanisms underlying the interrelationship between adiposity and periodontitis. Therefore, it is reasonable to suspect that low muscle mass may be a risk factor in adiposity-related periodontitis. Thus, periodontal health care and disease prevention are necessary for women with high adiposity-low muscle synergistic phenotype. It is necessary to reduce fat mass and increase muscle mass to reduce periodontitis risks in obese women.

It is well-established that sex differences exist in obesity-related comorbidities. Sex-specific immune responses and subclinical inflammation are critical determinants of these sex differences.[12,13] Several hypotheses may explain these sex differences. First, there is a sex difference in body composition, with women having a higher proportion of adiposity and men having a higher muscle mass.[37] High adiposity mass and low muscle mass contribute to systemic inflammation and are especially relevant in women. Second, adiposity-related systemic inflammation has been introduced as a factor for periodontal complications of obesity.[6] A stronger association was proposed between adiposity and systemic inflammation stronger in women than in men, especially for CRP.[38] CRP is associated with advanced periodontitis.[39] Most strikingly, estrogen plus progestin therapy for postmenopausal women was associated with an increased risk of CRP-related systemic inflammation.[40] Third, it was recently reported that adiposity-mediated inflammatory dysregulation mechanisms differed in terms of sex. Obese women exhibited a decreased level of anti-inflammatory adiponectin, with defective anti-inflammatory mechanisms.[41] Furthermore, adiponectin level was inversely associated with worsening periodontitis, with adiponectin playing a protective role in the periodontal structure.[42] Periodontal tissues could be susceptible to microbial invasion in obese women with dysregulated adiponectin. Further studies are warranted to unravel mechanisms underlying the sex-specific associations between body composition and periodontitis and elucidate intricate interplays between systemic inflammation, immune dysfunction, and sex hormones.

The present study has several strengths. First, we used the DXA method to measure body composition, which provides a more accurate assessment of body fat and muscle mass in obese patients. Second, due to the sex differences in body composition and periodontitis risk, we stratified analyses by sex and suggested that the prevention strategy of losing fat and gaining muscle should be considered for women more. Third, the adjusted model included multiple potential confounders, including sociodemographic characteristics, behavioral factors, and health conditions.

The current study also has several limitations. First, because of the exclusion of participants with incomplete DXA data from our analyses, a certain degree of selection

bias may have been introduced. It is important to note that participants undergoing DXA examinations tended to be younger and metabolically healthier than those without DXA. Second, muscle mass was estimated by the DXA method using two-dimensional images without distinguishing lipid infiltration and determining muscle composition.[37] Third, no data were available on gingival bleeding in the NHANES 2011-2014. Gingival bleeding is an essential sign of periodontal inflammation with systemic impact.[43] We did not include this inflammatory parameter in our analyses to reveal their potential effect on the sex-specific association between body composition and periodontitis. Fourth, since the analyses used cross-sectional data, they cannot provide evidence of temporal or causal association. Future longitudinal studies are required to examine the association of body composition with periodontitis and confirm the sex difference.

5 | CONCLUSION

The present study shows that the association between DXA-measured body composition indices and phenotypes with periodontitis differs per sex. Only women with higher fat mass and percentage body fat were more likely to exhibit moderate/severe periodontitis. Additionally, high-fat mass and low-muscle mass combined phenotype was associated with higher periodontitis risk in women. These findings suggest that periodontal health care and disease prevention might be more critical in obese women with high adiposity and low muscle phenotype. Strategies to control body fat and increase muscle mass should be adopted to reduce periodontitis risk in obese women. Future research should attempt to consolidate our findings using a DXA-based measure in a longitudinal study.

AUTHOR CONTRIBUTIONS

Peijun Zhu contributed to statistical analyses, data interpretation, and drafting of the manuscript; An Li contributed to study conception and design, data interpretation, and critical revision of the manuscript; Qingqing Cai contributed to the study conception and critical revision of the manuscript; Yuntao Chen, a statistical consultant, contributed to statistical analyses and critical revision of the manuscript; Yang Liu contributed to the study conception and critical revision of the manuscript; Harriët Jager-Wittenaar contributed to data interpretation and critical revision of the manuscript; Geerten-Has E. Tjakkes contributed to data interpretation and final approval of the manuscript; and Shulan Xu contributed to study conception and design, data interpretation, and final approval of the manuscript. All authors gave final approval and agreed to be accountable for all aspects of this work to ensure integrity and accuracy.

ACKNOWLEDGMENTS

The authors thank the NHANES staff and investigators. A special thanks to people involved in the NHANES study for making this research possible through their participation. The present study was supported by the Science Research Cultivation Program of Stomatological Hospital, Southern Medical University, China (grant number: PY2021007), Clinical Research Initiation Plan of Stomatological Hospital, Southern Medical University, China (grant number: KQIIT2021001), and Guangdong Basic and Applied Basic Research Foundation (grant number: 2022A1515110379).

CONFLICT OF INTEREST STATEMENT

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper. Ethical approval was not required.

ORCID

An Li https://orcid.org/0000-0001-5750-526X
Shulan Xu https://orcid.org/0000-0002-5706-3526

REFERENCES

1. Papapanou PN, Sanz M, Buduneli N, et al. Periodontitis: consensus report of workgroup 2 of the 2017 World Workshop on the classification of periodontal and peri-implant diseases and conditions. *J Clin Periodontol*. 2018;45 (suppl 20):S162-S170.
2. Chen MX, Zhong YJ, Dong QQ, Wong HM, Wen YF. Global, regional, and national burden of severe periodontitis, 1990-2019: an analysis of the global burden of disease study 2019. *J Clin Periodontol*. 2021;48:1165-1188.
3. Pirih FQ, Monajemzadeh S, Singh N, et al. Association between metabolic syndrome and periodontitis: the role of lipids, inflammatory cytokines, altered host response, and the microbiome. *Periodontol 2000*. 2021;87:50-75.
4. Jepsen S, Suvan J, Deschner J. The association of periodontal diseases with metabolic syndrome and obesity. *Periodontol 2000*. 2020;83:125-153.
5. Ganesan SM, Vazana S, Stuhr S. Waistline to the gumline: relationship between obesity and periodontal disease-biological and management considerations. *Periodontol 2000*. 2021;87:299-314.
6. Suvan JE, Finer N, D'Aiuto F. Periodontal complications with obesity. *Periodontol 2000*. 2018;78:98-128.
7. Ortega FB, Sui X, Lavie CJ, Blair SN. Body Mass Index, the most widely used but also widely criticized index: would a criterion standard measure of total body fat be a better predictor of cardiovascular disease mortality? *Mayo Clin Proc*. 2016;91:443-455.
8. Li A, Tang Z, Zhu P, et al. Serum antioxidant vitamins mediate the association between periodontitis and metabolically unhealthy overweight/obesity. *Nutrients*. 2022;14:4939.

9. Furuta M, Shimazaki Y, Takeshita T, et al. Gender differences in the association between metabolic syndrome and periodontal disease: the Hisayama Study. *J Clin Periodontol*. 2013;40:743-752.
10. Chen C, Ye Y, Zhang Y, Pan XF, Pan A. Weight change across adulthood in relation to all cause and cause specific mortality: prospective cohort study. *BMJ*. 2019;367:l5584.
11. Keller A, Rohde JF, Raymond K, Heitmann BL. Association between periodontal disease and overweight and obesity: a systematic review. *J Periodontol*. 2015;86:766-776.
12. Varghese M, Griffin C, Singer K. The role of sex and sex hormones in regulating obesity-induced inflammation. *Adv Exp Med Biol*. 2017;1043:65-86.
13. Meisel P, Eremenko M, Holtfreter B, Völzke H, Kocher T. The sex paradox in the interplay between periodontitis, obesity, and serum C-reactive protein: data from a general population. *J Periodontol*. 2019;90:1365-1373.
14. Messina C, Albano D, Gitto S, et al. Body composition with dual energy x-ray absorptiometry: from basics to new tools. *Quant Imaging Med Surg*. 2020;10:1687-1698.
15. Johnson CL, Dohrmann SM, Burt VL, Mohadjer LK. National Health and Nutrition Examination Survey: sample design, 2011-2014. *Vital and Health Statistics Series 2, Data Evaluation and Methods Research*. CDC; 2014:1-33.
16. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. *Lancet*. 2007;370:1453-1457.
17. Kakinami L, Danieles PK, Ajibade K, Santosa S, Murphy J. Adiposity and muscle mass phenotyping is not superior to BMI in detecting cardiometabolic risk in a cross-sectional study. *Obesity*. 2021;29:1279-1284.
18. Dye BA, Afful J, Thornton-Evans G, Iafolla T. Overview and quality assurance for the oral health component of the National Health and Nutrition Examination Survey (NHANES), 2011-2014. *BMC Oral Health*. 2019;19:95.
19. Page RC, Eke PI. Case definitions for use in population-based surveillance of periodontitis. *J Periodontol*. 2007;78:1387-1399.
20. Li A, Chen Y, Schuller AA, van der Sluis LWM, Tjakkes GHE. Dietary inflammatory potential is associated with poor periodontal health: a population-based study. *J Clin Periodontol*. 2021;48:907-918.
21. Kanagasabai T, Thakkar NA, Kuk JL, Churilla JR, Ardern CI. Differences in physical activity domains, guideline adherence, and weight history between metabolically healthy and metabolically abnormal obese adults: a cross-sectional study. *Int J Behav Nutr Phys Act*. 2015;12:64.
22. Schipf S, Knüppel S, Hardt J, Stang A. Directed acyclic graphs (DAGs) – the application of causal diagrams in epidemiology. *Gesundheitswesen*. 2011;73:888-892.
23. Gaio EJ, Haas AN, Rösing CK, Oppermann RV, Albandar JM, Susin C. Effect of obesity on periodontal attachment loss progression: a 5-year population-based prospective study. *J Clin Periodontol*. 2016;43:557-565.
24. Goossens GH, Jocken JWE, Blaak EE. Sexual dimorphism in cardiometabolic health: the role of adipose tissue, muscle and liver. *Nat Rev Endocrinol*. 2021;17:47-66.
25. Delaney KZ, Santosa S. Sex differences in regional adipose tissue depots pose different threats for the development of type 2 diabetes in males and females. *Obes Rev*. 2022;23:e13393.
26. Saxlin T, Ylöstalo P, Suominen-Taipale L, Aromaa A, Knuuttila M. Overweight and obesity weakly predict the development of periodontal infection. *J Clin Periodontol*. 2010;37:1059-1067.
27. Torrungruang K, Tamsailom S, Rojanasomsith K, et al. Risk indicators of periodontal disease in older Thai adults. *J Periodontol*. 2005;76:558-565.
28. Zong G, Zhang Z, Yang Q, Wu H, Hu FB, Sun Q. Total and regional adiposity measured by dual-energy x-ray absorptiometry and mortality in NHANES 1999-2006. *Obesity*. 2016;24:2414-2421.
29. Dong J, Gong Y, Chu T, et al. Mendelian randomization highlights the causal association of obesity with periodontal diseases. *J Clin Periodontol*. 2022;49:662-671.
30. Huang X, Yu T, Ma C, et al. Macrophages play a key role in the obesity-induced periodontal innate immune dysfunction via nucleotide-binding oligomerization domain-like receptor protein 3 pathway. *J Periodontol*. 2016;87:1195-1205.
31. Kim KN, Yao Y, Ju SY. Short chain fatty acids and fecal microbiota abundance in humans with obesity: a systematic review and meta-analysis. *Nutrients*. 2019;11:2512.
32. Aravindakshan V, Hakeem FF, Sabbah W. Periodontal disease and grip strength among older adults. *Geriatrics*. 2020;5:46.
33. Han CH, Chung JH. Association between sarcopenia and tooth loss. *Ann Geriatr Med Res*. 2018;22:145-150.
34. Kim G, Kim JH. Impact of skeletal muscle mass on metabolic health. *Endocrinol Metab*. 2020;35:1-6.
35. Park SJ, Ryu SY, Park J, Choi SW. Association of sarcopenia with metabolic syndrome in Korean population using 2009-2010 Korea National Health and Nutrition Examination Survey. *Metab Syndr Relat Disord*. 2019;17:494-499.
36. Zisman A, Peroni OD, Abel ED, et al. Targeted disruption of the glucose transporter 4 selectively in muscle causes insulin resistance and glucose intolerance. *Nat Med*. 2000;6:924-928.
37. Srikanthan P, Horwich TB, Calfon Press M, Gornbein J, Watson KE. Sex differences in the association of body composition and cardiovascular mortality. *J Am Heart Assoc*. 2021;10:e017511.
38. Meisel P, Wilke P, Biffar R, Holtfreter B, Wallaschofski H, Kocher T. Total tooth loss and systemic correlates of inflammation: role of obesity. *Obesity*. 2012;20:644-650.
39. Duzagac E, Cifcibasi E, Erdem MG, et al. Is obesity associated with healing after non-surgical periodontal therapy? A local vs. systemic evaluation. *J Periodontal Res*. 2016;51:604-612.
40. Jung SY, Yu H, Pellegrini M, Papp JC, Sobel EM, Zhang ZF. Genetically determined elevated C-reactive protein associated with primary colorectal cancer risk: Mendelian randomization with lifestyle interactions. *Am J Cancer Res*. 2021;11:1733-1753.
41. Ter Horst R, van den Munckhof ICL, Schraa K, et al. Sex-Specific regulation of inflammation and metabolic syndrome in obesity. *Arterioscler Thromb Vasc Biol*. 2020;40:1787-1800.
42. Yamaguchi N, Hamachi T, Kamio N, et al. Expression levels of adiponectin receptors and periodontitis. *J Periodontal Res*. 2010;45:296-300.
43. Pietropaoli D, Monaco A, D'Aiuto F, et al. Active gingival inflammation is linked to hypertension. *J Hypertens*. 2020;38:2018-2027.

SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

How to cite this article: Zhu P, Li A, Cai Q, et al. Sex differences in the association between dual-energy x-ray absorptiometry-measured body composition and periodontitis. *J Periodontol*. 2023;1-14. https://doi.org/10.1002/JPER.23-0162