

THE ASSOCIATION BETWEEN OBESITY AND PERIODONTAL DISEASE: NHANES 2009-2012 ANALYSIS

Ahmed Ali Alhassani* 

ABSTRACT

This study investigated the association between body fatness and periodontitis. The study population comprised 6,811 individuals who participated in the United States National Health and Nutrition Examination Survey (NHANES) 2009-2012 cycles. Body adiposity was evaluated using body mass index (BMI), and visceral adiposity was evaluated using waist circumference (WC). Periodontitis was defined using two methods: the Center for Disease Control and Prevention (CDC) and the American Academy of Periodontology (AAP) periodontitis case definition (CDC/AAP) and the Arbes *et al.* 2001 case definition (at least one site with ≥ 3 mm clinical attachment loss and ≥ 4 mm probing depth). The results showed that the prevalence of periodontitis was 41.8% according to the Arbes *et al.* 2001 definition and 45.7% according to the CDC/AAP definition. Based on Arbes *et al.* definition, those with BMI of 27-29.9 had a 34% increase in the odds of periodontitis (odds ratio (OR): 1.34, 95% confidence interval (CI): 1.08-1.66). Those who were obese (BMI ≥ 30) had a 47% increase in the odds of periodontitis (OR: 1.47, 95% CI: 1.22-1.76). High WC was associated with 63% higher odds of periodontal disease (OR: 1.63, 95% CI: 1.79-2.06). Based on the CDC/AAP case definition, neither high BMI nor WC were associated with periodontitis.

KEYWORDS: Periodontitis; periodontal disease; epidemiology; obesity.

INTRODUCTION

Gingivitis is a common condition defined as inflammation of the superficial periodontal tissue that does not lead to periodontal attachment loss. Periodontitis is an inflammatory disease that leads to breakdown of the tooth-supporting apparatus through destruction of the periodontal ligament, alveolar bone resorption, and apical migration of the

junctional epithelium. This results in the development of periodontal pockets and/or gingival recession. While gingivitis lesions are reversible and do not cause loss of periodontal support once the etiologic factor(s) is/are removed, the vast majority of periodontitis lesions are irreversible.¹ If remains untreated, periodontitis can progress and cause increasing tooth mobility and eventual tooth loss.²

* Assistant Professor, Department of Periodontics, Faculty of Dentistry, King Abdulaziz University, Saudi Arabia

Periodontitis is one of the most common chronic diseases in human adults. Eke *et al.* used data from two cycles of the National Health and Nutrition Examination Survey (NHANES) 2009-2012 data to estimate the prevalence of periodontitis in the United States.³ Data from that study estimated that 46% of the US population aged 30 years or older had some degree of periodontal disease with 8.9% of all the subjects having severe forms of periodontitis.³ Those two cycles of NHANES, 2009-2010 and 2011-2012, represent the first cycles with full-mouth periodontal examination, versus random partial-mouth examination in the previous NHANES.⁴ Several reports in the past have concluded that partial mouth exam in NHANES would lead to underestimation of the prevalence of periodontal disease.⁴⁻⁶

The obesity epidemic continues to increase worldwide in both industrialized and developing countries. In some parts of the world, overweight and obesity even coexist with undernutrition.⁷ About 68.5% of the US population are either overweight or obese, and of these, 6.4% are morbidly obese i.e. body mass index (BMI) ≥ 40 .⁸

Most reports that investigated the association between increased body adiposity and periodontal disease found direct associations. For example, Saito *et al.* published the first reports about a positive association between periodontist and obesity.^{9, 10} Al-Zahrani *et al.* investigated this association in NHANES III, and reported a significant positive association between obesity and periodontal diseases in young adults only.¹¹ Three meta-analyses that included cross sectional and longitudinal studies concluded a positive association between periodontal disease and body adiposity.¹²⁻¹⁴ A large study investigated the causality of the association between obesity and periodontal disease using Mendelian randomization (MR) analysis. The authors concluded that there was a null association.¹⁵

The association between obesity and periodontitis is biologically plausible through a number of

mechanisms including systemic inflammation, impaired insulin sensitivity, and glucose metabolism.¹⁶

The clinical measurements most commonly used to assess periodontal conditions include probing depth (PD), clinical attachment level (CAL), and bleeding on probing (BoP). It is widely accepted that the 'gold standard' for periodontal examination is a full dentition exam excluding the 3rd molars with 6 measurements per tooth.^{17, 18} Unfortunately, there is no consensus on the case definition of periodontitis in epidemiological studies, which in turn leads to wide heterogeneity among studies and could complicate comparing results.¹⁷

Most of the reports vis-à-vis the relationship between obesity and periodontal disease concluded a positive association. However, there is a wide heterogeneity in the results, which can be partially attributed to the differences in case definition used in each study.

The aim of this study was to investigate the association between overall adiposity using BMI and central obesity using waist circumference (WC) with periodontal disease according to two widely accepted periodontitis case definitions. First, the association was examined via the case definition used by Arbes *et al.*¹⁹. The association was examined using the CDC/AAP periodontitis case definition. The use of these two definitions on the same data allowed us to directly compare the impact of different case definitions on the strength of association.

MATERIALS AND METHODS

This study used data from two cycles of NHANES: 2009-2010 and 2011-2012. NHANES is a cross sectional stratified multistage survey of civilian non-institutionalized individuals in the United States and the District of Columbia. Trained interviewers evaluated the participants in their homes using Computer-Assisted Personal Interviewing (CAPI). The questions were followed by

a comprehensive medical and dental examination. The appended two NHANES cycles included 20,293 participants.

In NHANES, calibrated examiners performed the periodontal examination in a mobile examination center (MEC). For participants who were ≥ 30 years old, the PD, recession, and CAL were recorded on six sites per tooth (mid-facial, mid-lingual, mesio-facial, mesio-lingual, disto-facial and disto-lingual) for all the teeth excluding the third molars. Individuals with contributory medical history findings were excluded from the examination.

Periodontitis was defined in this study using two case definitions. The first was the definition by Arbes *et al.* 2001. Participants were considered to be a case if they had least one site with both PD of ≥ 4 mm and CAL of ≥ 3 mm—this definition has been used in previous NHANES studies, and it has the advantage of including what are most likely active periodontal lesions.^{11, 19} The second was the CDC/AAP case definition. Severe periodontitis was defined as the presence of two or more interproximal sites with ≥ 6 mm CAL (not on the same tooth) and at least one interproximal site with ≥ 5 mm PD. Moderate periodontitis was defined as the presence of two or more interproximal sites with ≥ 4 mm CAL (not on the same tooth) or at least two interproximal sites with PD ≥ 5 mm. Slight periodontitis was defined as the presence of at least two interproximal sites with ≥ 3 mm CAL and at least two interproximal sites with ≥ 4 mm PD (not on the same tooth) or one site with ≥ 5 mm. Total periodontitis was used in this study, which is the sum of severe, moderate, and slight periodontitis.

Overall adiposity was evaluated using BMI, and visceral adiposity was evaluated using WC. BMI was first categorized using the WHO 4 categories: underweight ($<18.5\text{kg/m}^2$), healthy weight ($18.5\text{--}24.9\text{kg/m}^2$), overweight ($25\text{--}29.9\text{ kg/m}^2$), and obese ($\geq 30\text{kg/m}^2$). The healthy and overweight groups were subcategorized due to the wide range of those

categories. Hence, the final analysis was done using six BMI categories: $<18.5\text{kg/m}^2$, $18.5\text{--}22.9\text{kg/m}^2$, $23\text{--}24.9\text{ kg/m}^2$, $25\text{--}26.9\text{ kg/m}^2$, $27\text{--}29.9\text{ kg/m}^2$, and $\geq 30\text{ kg/m}^2$. WC was categorized using both clinical guidelines and previous publications into four categories using the following cutoff points: For males, the cutoff points were <84 cm, between $84\text{--}93.9$ cm, between $94\text{--}102$ cm, and $>102\text{cm}$. For females, the cutoff points were <71 cm, between $71\text{--}79.9$ cm, between $80\text{--}88$ cm, and $>88\text{cm}$.²³ The lowest WC category was called category 1 and the highest was called category 4.

Several variables were used to adjust for confounding in the current analysis: age, gender, race/ethnicity, smoking status, socioeconomic status, and education. Age was categorized into three categories: 30-34, 35-59, and ≥ 60 . Race/ethnicity was categorized into four groups: Hispanic (which included Mexican-American and other Hispanic), non-Hispanic Black, non-Hispanic White, and other Race including multi-Racial people. Cigarette smoking was grouped into current smoker, never smoker, and former smoker. Socioeconomic status was classified based on the ratio of family income to poverty: Participants were categorized into < 1 , $1\text{--}1.99$, $2\text{--}3.99$, and more than 4. The education level was categorized as less than 12 years of schooling, 12 years of schooling, and more than 12 years of schooling. All statistical analysis was done using SAS (Version 9.4, SAS Institute, Cary, NC).

RESULTS

There were 20,293 participants in the sample, and 7,753 had complete periodontal data; 692 participants were excluded from the examination due to medical reasons. After exclusion of participants with missing BMI or WC values, the final sample size was 6,811.

The major characteristics of the study population and prevalence of periodontitis in each subpopulation are summarized in Table 1. The reported prevalence

and percentages are based on the weighted sample. There were slightly more females than males. Most of the participants (62.2%) were in the 35-59 age category. The least represented group was 30-34. Here, 69% were non-Hispanic white; Hispanics and non-Hispanic Blacks represented 13.4% and 10.5% respectively. More than half of the study participants (56%) had never smoked, 26.4% were former smokers, and 17.6% were current smokers. More than 62.9% of the subjects completed more than 12 years of schooling. According to BMI cutoffs, most of the participants were either overweight or obese, 36% and 36.5% respectively. Only 26.4% of the participants were in the normal weight range, and about 1% were underweight. Based on WC, 42.2% of the sample were in the high category.

The prevalence of periodontitis in the study sample was 41.8% according to the Arbes *et al.* 2001 definition and 45.7% according to the CDC/AAP definition: 6.2% had slight periodontitis, 30.7% had moderate disease, and 8.8% had severe periodontitis.

The prevalence of periodontitis was higher in males. Older age was associated with an increased risk of periodontal disease. The prevalence of periodontitis was highest among current smokers and lowest among never smokers. Participants who identified as either Hispanic or non-Hispanic Black had the highest occurrence of periodontitis while those who identified as non-Hispanic White had the lowest. Lower income and education levels were associated with a higher prevalence of periodontal disease. Participants in the 18.5-22.9 BMI category had the lowest prevalence of periodontitis. According to the Arbes *et al.* case definition, the risk of periodontal disease was highest among those with BMI \geq 30. According to the CDC/AAP cases definition, the highest periodontal disease prevalence was among the underweight group.

Based on Arbes *et al.* definition, those at category-3 WC had a slightly lower prevalence of periodontitis. Based on the CDC/AAP definition,

individuals at category-2 of WC had a slightly lower risk of periodontal disease. Both case definitions agreed that the lowest WC category and had the highest prevalence of periodontal disease. Table 2 shows the agreement between the two case definitions. Based on the weighted sample, 77.6% of periodontitis cases were categorized as cases by both case definitions; 88.3% of non-periodontitis cases were classified as non-cases according to both definitions.

Table 3 presents the results of the fitted logistic regression models using categories of BMI as the main exposure. The BMI category of 18.5-22.9 was used as the reference. For Arbes *et al.*, in the adjusted model, being in the 27-29.9 category was associated with a 34% increase in the odds of periodontitis (odds ratio (OR): 1.34, 95% confidence interval (CI): 1.08-1.66). Obesity (BMI \geq 30) was associated with a 47% increase in the odds of periodontitis (OR: 1.47, 95% CI: 1.22-1.76). The 23-24.9 and the 25-26.9 BMI categories both had a non-significant increase in the odds of periodontitis. Underweight individuals were at a non-statistically significant elevated 27% odds of having periodontitis. For the CDC/AAP definition, underweight individuals had a 89% elevated odds of having periodontitis (OR: 1.89, 95% CI: 1.11-3.23). All other BMI categories were non-statically and significantly associated with the risk of periodontitis.

Table 4 shows the results of the fitted logistic regression models using categories of WC as the main exposure. For Arbes *et al.*, the fully adjusted model shows that the odds of periodontitis in the highest WC categories were 63% higher versus the lowest WC category (OR: 1.63, 95% CI: 1.79-2.06). There was a non-statistically significant increase in odds of having periodontitis in category-2 and category-3 (19% and 25%, respectively). For the CDC/AAP definition, results from the fully adjusted model showed that none of the WC categories was statistically significantly associated with the risk of periodontitis.

TABLE (1) Characteristics of the study population and prevalence of periodontitis.

	N	Weighted % (SE)	Prevalence of Periodontitis				Arbes <i>et al.</i> 2001 (SE)
			CDC/AAP Slight (SE)	CDC/AAP Moderate (SE)	CDC/AAP Severe (SE)	CDC/AAP Total (SE)	
Age:							
30-34	815	12.18(0.53)	6.93(1.06)	15.74(1.69)	2.03(0.55)	24.71(1.96)	31.92(2.27)
35-59	3815	62.16(0.65)	6.96(0.71)	26.72(1.37)	9.31(0.75)	42.99(1.63)	41.43(1.57)
≥60	2181	25.66(0.76)	4.17(0.58)	47.34(2.28)	10.73(1.33)	62.24(2.39)	47.34(2.05)
Gender:							
Male	3397	49.13(0.64)	7.33(0.71)	34.10(1.48)	13.23(0.83)	54.66(1.67)	50.25(1.27)
Female	3414	51.87(0.64)	5.19(0.58)	27.36(1.55)	4.50(0.50)	37.05(1.81)	33.61(1.51)
Race/ethnicity:							
Hispanic	1729	13.36(1.81)	9.24(1.14)	36.76(1.40)	12.66(1.22)	58.66(1.47)	55.77(1.68)
Non-Hisp Black	1435	10.49(1.17)	7.74 (0.90)	35.94(1.60)	15.77(1.41)	59.45(2.23)	56.87(1.72)
Non-Hisp White	2924	69.09(2.41)	5.54(0.61)	28.46(1.78)	6.77(0.72)	40.49(2.06)	36.54(1.58)
Other	723	7.05(0.71)	3.83(0.60)	36.05(3.49)	11.84(2.06)	48.27(3.65)	44.28(2.91)
Non-Hisp Asian [#]	450	1.19(0.19)	2.95(0.90)	34.32(3.14)	11.72(1.98)	48.99(3.75)	44.06(3.13)
Smoking:							
Never	3796	56.02(1.00)	6.99(0.64)	25.60(1.08)	5.38(0.57)	37.97(1.53)	34.57(1.59)
Former	1707	26.41(1.13)	4.80(0.69)	33.90(2.46)	9.60 (1.04)	48.30(2.48)	43.14(2.06)
Current	1305	17.58(0.62)	6.01(0.75)	41.95(2.14)	18.41(1.69)	66.37(1.78)	62.71(1.69)
N missing=3							
Education:							
<12	1709	16.32(1.05)	5.56(0.71)	44.12(1.76)	17.07(1.42)	66.74(1.56)	59.21(1.40)
12	1450	20.74(0.96)	7.84(0.82)	35.47(2.05)	11.65(0.94)	54.96(2.22)	49.42(2.18)
>12 N missing=10	3642	62.94(1.53)	5.90(0.65)	25.53(1.41)	5.71(0.56)	37.14(1.70)	34.74(1.47)
Income:							
<100% FPL	1774	17.34(0.95)	8.10(1.07)	36.96(1.78)	13.97(1.24)	59.04(1.85)	55.63(1.43)
100% to 199% FPL	1618	17.60(0.87)	6.75(0.84)	36.46(2.01)	13.43(1.33)	56.63(2.02)	52.49(1.69)
200% to 399% FPL	1621	26.94(1.49)	6.33(0.71)	33.71(2.27)	8.12(0.97)	48.16(2.77)	42.49(2.28)
≥400% FPL	1798	38.12(1.56)	5.09(0.83)	23.00(1.30)	4.77(0.67)	32.85(1.76)	30.06(1.66)
BMI:							
<18.5	74	1.11(0.17)	1.27(0.95)	39.30(8.84)	14.42(6.29)	54.99(8.15)	42.54(6.14)
18.5-22.9	1746	26.41(0.86)	3.58(0.87)	27.37(2.20)	9.57(0.93)	40.52(2.41)	34.00(2.10)
23-24.9	852	12.71(0.59)	4.23(0.95)	32.94(2.33)	7.63(1.20)	44.80(2.63)	40.71(2.38)
25-26.9	9943	15.38(0.61)	6.76 (0.78)	26.53(1.92)	9.32(1.29)	42.61(2.32)	38.15(2.34)
27-29.9	1428	20.64(0.64)	6.86 (0.85)	33.10(2.14)	8.93(0.90)	48.89(2.41)	44.28(2.54)
≥30	2590	36.46(0.99)	7.52 (0.75)	31.24(1.81)	8.42(0.93)	47.18(2.00)	45.20(1.76)
WC*:							
Category-1(low)	383	5.03(0.35)	4.99(1.32)	28.71(3.17)	17.66(2.78)	51.35(3.91)	44.03(3.49)
Category-2	1035	15.11(0.78)	5.92(1.02)	29.04(2.38)	7.66(0.96)	42.61(2.72)	39.41(2.20)
Category-3	1450	22.09(0.72)	5.81(0.76)	27.62(1.31)	10.12(0.91)	43.56(1.86)	38.71(1.80)
Category-4(high)	3943	42.23(1.21)	6.59(0.69)	32.44(1.74)	7.80(0.67)	46.84(1.88)	43.39(1.63)
Total	6811		6.24(0.52)	30.67(1.36)	8.79 (0.58)	45.70(1.60)	41.79(1.31)

[#] Only oversampled in the 2011-2012 cycle. In this study, non-Hispanic Asian are included within the 'Other' category. That is, the 450 non-Hispanic Asian individuals are a part of the 723 categorized as 'Other'.

*For males, the cutoff pointes were <84, between 84-93.9, between 94-102 and >102 cm
For females, the cutoff pointes were <71, between 71-79.9, between 80-88 and >88cm

TABLE (2) Agreement between the periodontitis definitions used in this study.

		CDC/AAP		Weighted % of agreement (SE)
		Yes	No	
Arbes <i>et al.</i>	Yes	2937	239	77.55(1.30)
	No	792	2693	88.31(0.97)

TABLE (3). Logistic regression models estimating OR (95% CI) relating BMI categories to periodontitis, using the Arbes *et al.* 2001 and the CDC/AAP case definitions.

	Model-1	Model-2	Model-3
Arbes <i>et al.</i> 2001			
BMI Categories:			
<18.5	1.44 (0.87-2.38)	1.47 (0.89-2.43)	1.27 (0.79-2.04)
18.5-22.9	Reference	Reference	Reference
23-24.9	1.32 (1.01-1.73)*	1.32 (1.01-1.73)*	1.24 (0.96-1.61)
25-26.9	1.20 (0.91-1.58)	1.19 (0.90-1.58)	1.12 (0.85-1.47)
27-29.9	1.54 (1.24-1.91)*	1.52 (1.24-1.87)*	1.34 (1.08-1.66)*
≥30	1.60 (1.33-1.92)*	1.59 (1.32-1.91)*	1.47 (1.22-1.76)*
CDC/AAP			
BMI Categories:			
<18.5	1.80 (0.944-3.44)	1.95 (1.04-3.65)*	1.89 (1.11-3.23)*
18.5-22.9	Reference	Reference	Reference
23-24.9	1.19 (0.90-1.57)	1.18 (0.91-1.54)	1.07 (0.81-1.41)
25-26.9	1.09 (0.84-1.42)	1.09 (0.83-1.43)	0.99 (0.74-1.31)
27-29.9	1.41 (1.12-1.77)*	1.38 (1.12-1.71)*	1.16 (0.90-1.49)
≥30	1.32 (1.08-1.60)*	1.30 (1.08-1.57)*	1.15 (0.94-1.42)

*Model 1: crude.**Model 3: adjusted for age.**Model 3: adjusted for age, gender, race/ethnicity, education, smoking and income.**Model 4: adjusted for age, gender, race/ethnicity, education, smoking and income, excluding current smokers.***p-value < 0.05*Table 4. Logistic regression models estimating OR (95% CI) relating WC categories to periodontitis, using the Arbes *et al.* 2001 and the CDC/AAP case definitions.

	Model-1	Model-2	Model-3
Arbes <i>et al.</i> 2001			
WC Categories:			
Category-1	Reference	Reference	Reference
Category-2	0.83 (0.57-1.20)	0.81 (0.56-1.17)	1.19 (0.86-1.66)
Category-3	0.81 (0.58-1.11)	0.77 (0.56-1.06)	1.25 (0.90-1.72)
Category-4	0.98 (0.75-1.27)	0.90 (0.69-1.18)	1.63 (1.79-2.06)*
CDC/AAP			
WC Categories:			
Category-1	Reference	Reference	Reference
Category-2	0.70 (0.47-1.04)	0.65 (0.44-0.96)	0.98 (0.64-1.48)
Category-3	0.73 (0.50-1.07)	0.64 (0.44-0.94)	1.06 (0.70-1.62)
Category-4	0.84 (0.61-1.14)	0.68 (0.50-0.92)	1.25 (0.89-1.48)

*Model 1: crude.**Model 3: adjusted for age.**Model 3: adjusted for age, gender, race/ethnicity, education, smoking and income.***p-value < 0.05*

DISCUSSION

This study examined the association between body adiposity and periodontal disease using data from two NHANES cycles: 2009-2012. A key finding in this analysis was that Arbes *et al.* definition of periodontitis was significantly more powerful in detecting the association between increased body fatness and periodontal disease. This may be partially explained by the idea that Arbes *et al.* definition has more specificity towards active periodontal lesions than the CDC/AAP definition. Another explanation is that due to the higher prevalence of periodontitis when using the CDC/AAP definition, the effect of adiposity may have been attenuated by other stronger risk factors in the adjusted model.

The Arbes *et al.* 2001 cases definition has been used in previous NHANES studies. It seems to have a high specificity for true active periodontal lesions.^{11, 19} The periodontitis case definition by the CDC/AAP was developed to standardize clinical case definitions in population-based studies.²⁰ There was some discrepancy, but overall, there was good agreement between the two periodontitis definitions. The inconsistency can be attributed to the higher sensitivity of the CDC/AAP classification.

The positive association between obesity and periodontal disease seen here agrees with most of the published literature. Chaffee and Weston conducted a meta-analysis of cross-sectional studies and found that the OR of developing periodontitis in obese individuals was 1.35 (95% CI: 1.23-1.47).¹² They also concluded that the association was stronger in younger age, women, and non-smokers.¹² A large longitudinal study was published by Jimenez *et al.*²¹ The authors followed 36,910 healthy male participants of the Health Professionals Follow-Up Study (HPFS) for about 20 years. Self-reported body measures and self-reported periodontal disease were used for this analysis. The authors concluded that obesity was associated with a higher risk of periodontal disease with a hazard

ratio (HR) of 1.30 (95% CI: 1.17–1.45). They also found that high WC and high waist-to-hip ratio (WHR) were significantly associated with a higher risk of periodontal disease.²¹ Another prospective study was published by Gorman *et al.*²² That study followed 1,038 healthy men as a part of the VA Dental Longitudinal Study between 1969 and 1996. The study concluded that obese men (BMI of ≥ 30) had a higher risk of periodontal disease progression (higher adjusted HR by 41–72%).²² A meta-analysis of the prospective studies that assessed the effects of weight gain on the incidence of periodontitis in adults was conducted by Nascimento *et al.*¹³ The study included five prospective studies (Jimenez *et al.* and Gorman *et al.* were included in the pooled analysis). The authors concluded that the incidence of periodontitis was higher in subjects who became overweight or obese through the follow-up period: risk ratio (RR) of 1.13 (95% CI: 1.06–1.20) and RR of 1.33 (95% CI: 1.21–1.47), respectively.¹³

The causality of obesity on periodontal disease was questioned by Shungin *et al.* using MR approach.¹⁵ The study included 13 US and European cohorts. In the MR analysis, the genotypes at FTO (rs1121980), MC4R (rs17782313), and TMEM18 (rs6548238) were combined into a genetic risk score (GRS) and used as the instrument for BMI. The authors found a null association between BMI and periodontal disease (OR of 1.05, 95% CI: 0.80, 1.38). Some of the limitations of the analysis have been addressed. First, it is possible that some aspects of adiposity were not captured by the genetic instruments used. Second, there is a possibility of biological confounding. Third, there is a possibility of population stratification bias. Finally, there is a possibility of selection bias due to the exclusion of edentulous participants.¹⁶ There was a wide heterogeneity in defining periodontitis in the Shungin *et al.* study; definitions included the use of the CDC/AAP, CPITN, PD, and self-reported periodontitis. This study illustrates that the association between obesity and periodontal disease is sensitive to the periodontitis case definition used

in a study. Another recent MR study found a direct association between BMI and periodontitis.²³

The association between periodontal disease and body fatness is biologically plausible via several pathways. Adipose tissue can act as an active endocrine organ, and obesity is associated with increased production of inflammatory mediators such as interleukin-1 β (IL-1 β), tumor necrosis factor- α (TNF- α), and IL-6.^{24,25} Pro-inflammatory mediators are key players in the pathogenesis of periodontal disease.²⁶ This low-grade systemic inflammation is associated with increased body fatness and is thought to be causally related to a variety of conditions linked to obesity including periodontal disease.²⁷ Oxidative stress and mitochondrial dysfunction are other mechanisms that may explain the link between obesity and periodontal disease.²⁸ Obesity is also associated with the dyslipidemia related to impaired glucose metabolism and reduced insulin sensitivity.^{29,30} Obesity through elevated TNF- α may also cause insulin resistance.³¹ The increased free fatty acid in obesity may be associated with β -cell apoptosis, which increases the risk of developing diabetes.³²

Diabetes significantly alters the host response and is strongly associated with periodontal tissue breakdown.^{5,33} Several studies that investigated the association between obesity and periodontal disease adjusted for diabetes. Current understanding is that diabetes is a mediator in the proposed causal pathway. Hence, this analysis did not adjust for diabetes.

The current study has several limitations. First, it is cross sectional in nature, thus making it impossible to conclude any causal effect. Second, there was no information about gingival inflammation such as gingival bleeding, which could have offered a better understanding of the disease status. However, the use of the Arbes *et al.* case definition may have alleviated this limitation. Finally, the exclusion of participants without teeth may have biased the results.

The study does have several strengths including the large sample size, the high quality of data collection, and the fact that it is representative of the US population.

In conclusion, there was a positive association between periodontal disease and both overall and central obesity. However, this association however was sensitive to the periodontitis case definition used. Future prospective studies that include inflammatory markers analysis are needed to further understand this relationship.

REFERENCES

1. Caton JG, Armitage G, Berglundh T, Chapple ILC, Jepsen S, Kornman KS, L MB, Papapanou PN, Sanz M, Tonetti MS. A new classification scheme for periodontal and peri-implant diseases and conditions - Introduction and key changes from the 1999 classification. *Journal of clinical periodontology* 2018;45 Suppl 20:S1-S8.
2. The pathogenesis of periodontal diseases. *Journal of periodontology* 1999;70:457-470.
3. Eke PI, Dye BA, Wei L, Slade GD, Thornton-Evans GO, Borgnakke WS, Taylor GW, Page RC, Beck JD, Genco RJ. Update on Prevalence of Periodontitis in Adults in the United States: NHANES 2009 to 2012. *Journal of periodontology* 2015;86:611-622.
4. Papapanou PN. The prevalence of periodontitis in the US: forget what you were told. *Journal of dental research* 2012;91:907-908.
5. Albandar JM. Underestimation of periodontitis in NHANES surveys. *Journal of periodontology* 2011;82:337-341.
6. Eke PI, Thornton-Evans GO, Wei L, Borgnakke WS, Dye BA. Accuracy of NHANES periodontal examination protocols. *Journal of dental research* 2010;89:1208-1213.
7. Obesity: preventing and managing the global epidemic. Report of a WHO consultation. *World Health Organ Tech Rep Ser* 2000;894:i-xii, 1-253.
8. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of childhood and adult obesity in the United States, 2011-2012. *Jama* 2014;311:806-814.
9. Saito T, Shimazaki Y, Koga T, Tsuzuki M, Ohshima A. Relationship between upper body obesity and periodontitis. *Journal of dental research* 2001;80:1631-1636.

10. Saito T, Shimazaki Y, Sakamoto M. Obesity and periodontitis. *N Engl J Med* 1998;339:482-483.
11. Al-Zahrani MS, Bissada NF, Borawski EA. Obesity and periodontal disease in young, middle-aged, and older adults. *Journal of periodontology* 2003;74:610-615.
12. Chaffee BW, Weston SJ. Association between chronic periodontal disease and obesity: a systematic review and meta-analysis. *Journal of periodontology* 2010;81:1708-1724.
13. Nascimento GG, Leite FR, Do LG, Peres KG, Correa MB, Demarco FF, Peres MA. Is weight gain associated with the incidence of periodontitis? A systematic review and meta-analysis. *Journal of clinical periodontology* 2015;42:495-505.
14. Suvan J, D'Aiuto F, Moles DR, Petrie A, Donos N. Association between overweight/obesity and periodontitis in adults. A systematic review. *Obesity reviews : an official journal of the International Association for the Study of Obesity* 2011;12:e381-404.
15. Shungin D, Cornelis MC, Divaris K, Holtfreter B, Shaffer JR, Yu YH, Barros SP, Beck JD, Biffar R, Boerwinkle EA, Crout RJ, Ganna A, Hallmans G, Hindy G, Hu FB, Kraft P, McNeil DW, Melander O, Moss KL, North KE, Orho-Melander M, Pedersen NL, Ridker PM, Rimm EB, Rose LM, Rukh G, Teumer A, Weyant RJ, Chasman DI, Joshupura K, Kocher T, Magnusson PK, Marazita ML, Nilsson P, Offenbacher S, Davey Smith G, Lundberg P, Palmer TM, Timpson NJ, Johansson I, Franks PW. Using genetics to test the causal relationship of total adiposity and periodontitis: Mendelian randomization analyses in the Gene-Lifestyle Interactions and Dental Endpoints (GLIDE) Consortium. *International journal of epidemiology* 2015;44:638-650.
16. Genco RJ, Grossi SG, Ho A, Nishimura F, Murayama Y. A Proposed Model Linking Inflammation to Obesity, Diabetes, and Periodontal Infections. *Journal of periodontology* 2005;76 Suppl 11S:2075-2084.
17. Holtfreter B, Albandar JM, Dietrich T, Dye BA, Eaton KA, Eke PI, Papapanou PN, Kocher T. Standards for reporting chronic periodontitis prevalence and severity in epidemiologic studies: Proposed standards from the Joint EU/USA Periodontal Epidemiology Working Group. *Journal of clinical periodontology* 2015;42:407-412.
18. Kingman A, Susin C, Albandar JM. Effect of partial recording protocols on severity estimates of periodontal disease. *Journal of clinical periodontology* 2008;35:659-667.
19. Arbes SJ, Jr., Agústsóttir H, Slade GD. Environmental tobacco smoke and periodontal disease in the United States. *Am J Public Health* 2001;91:253-257.
20. Page RC, Eke PI. Case definitions for use in population-based surveillance of periodontitis. *Journal of periodontology* 2007;78:1387-1399.
21. Jimenez M, Hu FB, Marino M, Li Y, Joshupura KJ. Prospective associations between measures of adiposity and periodontal disease. *Obesity (Silver Spring, Md)* 2012;20:1718-1725.
22. Gorman A, Kaye EK, Apovian C, Fung TT, Nunn M, Garcia RI. Overweight and obesity predict time to periodontal disease progression in men. *Journal of clinical periodontology* 2012;39:107-114.
23. Dong J, Gong Y, Chu T, Wu L, Li S, Deng H, Hu R, Wang Y. Mendelian randomization highlights the causal association of obesity with periodontal diseases. *Journal of clinical periodontology* 2022;49:662-671.
24. Exley MA, Hand L, O'Shea D, Lynch L. Interplay between the immune system and adipose tissue in obesity. *J Endocrinol* 2014;223:R41-48.
25. Kinane DF, Preshaw PM, Loos BG. Host-response: understanding the cellular and molecular mechanisms of host-microbial interactions--consensus of the Seventh European Workshop on Periodontology. *Journal of clinical periodontology* 2011;38 Suppl 11:44-48.
26. Pink C, Kocher T, Meisel P, Dorr M, Markus MR, Jablonowski L, Grotevendt A, Nauck M, Holtfreter B. Longitudinal effects of systemic inflammation markers on periodontitis. *Journal of clinical periodontology* 2015;42:988-997.
27. Boesing F, Patiño JS, da Silva VR, Moreira EA. The interface between obesity and periodontitis with emphasis on oxidative stress and inflammatory response. *Obesity reviews : an official journal of the International Association for the Study of Obesity* 2009;10:290-297.
28. Bullon P, Newman HN, Battino M. Obesity, diabetes mellitus, atherosclerosis and chronic periodontitis: a shared pathology via oxidative stress and mitochondrial dysfunction? *Periodontology* 2000 2014;64:139-153.
29. Cutler CW, Shinedling EA, Nunn M, Jotwani R, Kim BO, Nares S, Iacopino AM. Association between periodontitis and hyperlipidemia: cause or effect? *Journal of periodontology* 1999;70:1429-1434.

30. Kolovou GD, Anagnostopoulou KK, Cokkinos DV. Pathophysiology of dyslipidaemia in the metabolic syndrome. *Postgrad Med J* 2005;81:358-366.
31. Hotamisligil GS. The role of TNF α and TNF receptors in obesity and insulin resistance. *J Intern Med* 1999;245:621-625.
32. Shimabukuro M, Zhou YT, Levi M, Unger RH. Fatty acid-induced beta cell apoptosis: a link between obesity and diabetes. *Proc Natl Acad Sci U S A* 1998;95:2498-2502.
33. Kocher T, Konig J, Borgnakke WS, Pink C, Meisel P. Periodontal complications of hyperglycemia/diabetes mellitus: Epidemiologic complexity and clinical challenge. *Periodontology* 2000 2018;78:59-97.