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Obesity and periodontitis in Australian adults: A population-based cross-sectional study

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Objectives: Obesity and periodontitis are public health issues in Australia. This study aimed to determine the association between overweight/obesity and periodontitis in Australian adults. **Materials and methods:** The cross-sectional National Survey of Adult Oral Health 2004–2006 data were analysed. Body mass index was calculated, and a self-reported questionnaire was used to measure the estimated daily intake of added sugar. The mean number of sites with probing depth (PD) ≥ 4 mm and clinical attachment loss (CAL) ≥ 4 mm and presence of periodontitis were used as outcome measures. CDC/AAP periodontitis case definition was adopted. Bivariate analyses and multiple variable regression models were constructed. **Results:** The study sample was 4,170 participants. The proportion of people that were overweight/obese was 51.9% [95% confidence interval (CI): 48.1%, 54.1%]. Overall 21.3% (95% CI: 19.3%, 23.5%) people experienced periodontitis. The mean number of sites with PD ≥ 4 mm and CAL 4 mm were recorded as 0.7 (95% CI: 0.5, 0.9) and 2.4 (95% CI: 2.1, 2.6), respectively. Multiple variable analysis suggested that periodontal parameters [sites with PD ≥ 4 mm (0.13, 95% CI: −0.86, 0.35) and sites with CAL ≥ 4 mm (0.11, 95% CI: −0.58, 0.35) and presence of periodontitis (1.23, 95% CI: 0.96, 1.57)] were not associated with overweight/obesity when controlled for putative confounders. **Conclusion:** A positive association was found between overweight/obesity and periodontitis (PD and CAL). However, the statistical significance disappeared in the multiple variable regression analysis, where age, sex, smoking and dental visiting behaviour were found to be key determinants of periodontitis.

Key words: *Periodontal disease, obesity, oral health, public health, chronic disease, body mass index*

INTRODUCTION

Obesity is a global epidemic. The prevalence of being overweight or obese in Australian adults is 63% and 28%, respectively[1]. Overweight/obesity is a result of a complex system that includes multiple influences (contextual, cultural, psychological, metabolic and genetic risk factors), often referred to as the socio-ecological framework of obesity[2]. Obesity is defined as "*imbalanced levels of energy intake and energy expenditure*", that is impacted, compounded and driven by these multiple influences[3]. Chronic co-morbidities, including type 2 diabetes and cardiovascular diseases, are significant health outcomes associated with obesity[4].

Periodontitis occurs as a pro-inflammatory state that affects the supporting periodontal tissues of the teeth. The aetiopathogenesis of periodontitis occurs as a result of the ecological interaction of dental plaque biofilm[5] and the host immune system response leading to metabolic changes in the bone and localisation of pro-inflammatory cytokines[6], under the influence of lifestyle factors[7] and chronic co-morbidities[8]. The prevalence of periodontitis in Australia is reported to be 24.5%[9]. Socio-economic factors, for example low income, low education and unemployment; lifestyle habits including dental visiting behaviour and oral hygiene behaviour; and risk behaviours such as smoking and alcohol consumption are factors significantly associated with periodontitis in Australian adults[9].

The relationships between obesity and periodontitis have been reported in previous studies[8,10]. Obesity contributes to increased complexity of periodontal pathogens[11] and raised levels of pro-inflammatory cytokines[12]. Lifestyle changes, including higher added sugar intake, increase the susceptibility to energy

imbalance often leading to weight gain[13] and other long-term health consequences including periodontitis[8].

Obesity and periodontitis are significant health problems in Australia, with common contributing factors including socio-economic, diet and chronic comorbidities. This leads to the hypothesis that obesity or being overweight is associated with periodontitis in Australian adults, and the research question "Is obesity or being overweight a risk factor for periodontitis in Australian adults?" Hence, this study aimed to determine the association of obesity and periodontitis in Australian adults.

MATERIALS AND METHODS

Study design

This study was a secondary analysis design using data from the cross-sectional survey, National Survey of Adult Oral Health 2004-06 (NSAOH 2004-06). This survey was conducted by the Australian Research Centre for Population Oral Health (ARCPOH), and recruited the representative sample of dentate Australians aged 15 years and older using a clustered randomised sampling technique. The NSAOH had three stages that included computer-aided telephone interview (CATI), oral examination and mailed questionnaire. The details of the NSAOH 2004–2006 data are described elsewhere[14]. STROBE statement guidelines, essential for good reporting of cross-sectional studies, were followed.

Computer-aided telephone interview

The CATI was undertaken using Windows-based WinCati 4.2 software. Approximately 10 days prior to the CATI, a primary approach letter was sent to the participants outlining the purpose of the survey. A toll-free telephone number was provided to participants, in the event of any queries raised regarding the survey. The telephone number of participants who made contact to withdraw from the survey were removed from the interview sample list. The CATI consisted of 79 questions regarding social demographics (age, gender, rural location, indigenous identity, remoteness, country of birth, and language spoken at home), perceived need for treatment, subjective periodontal outcome measures, questions about gum treatment, medical history (self-reported diabetes), dental visit behaviour, oral hygiene behaviour, risk behaviour, income and education, height and weight.

Oral epidemiological examination

The oral epidemiological examination of participants was carried out following a standardised dental examination procedure. Clinical appointments were scheduled at the nearest public dental clinic within or near the postcode of the participant. Informed consent and a medical history were obtained prior to the dental examination. Participants were asked to complete a medical history form about conditions which, if present, would preclude a periodontal examination as follows: (i) a physician or dentist told that the participant must take antibiotics before getting a dental check-up or care; (ii) a physician told the participant they had a congenital heart murmur, heart valve problem, congenital heart disease or bacterial endocarditis; (iii) rheumatic fever; (iv) kidney disease requiring dialysis; (v) haemophilia; (vi) a pacemaker or automatic defibrillator; other artificial material in the participant's heart, veins or arteries (this was a contraindication in 2004–2006 when the NSAOH 2004–2006 was conducted, hence it has been retained as a contraindication for periodontal examination in this paper); (vii) a hipbone or joint replacement that has been inserted in the last 3 months; (viii) transplanted organs. People who answered yes to at least one of these questions were excluded from the periodontal examination. Individuals with no contraindication underwent full-mouth periodontal charting (three sites per tooth – mesio-buccal, mid-buccal and disto-buccal) of probing depths (PD) and clinical attachment loss (CAL) using a periodontal condition probe (PCP2, Hu-Friedy, Chicago, USA). Third molars were excluded from the oral examination.

Mailed questionnaire

The questionnaire included 13 food frequency questions on dietary sugar intake. These questions asked about the intake of fruits, sweetened fruit drinks and soft drinks, plain and flavoured milk, sweetened dairy products, breakfast cereals, biscuits and cakes, table sugar, chocolate and confectionery, syrups and jams, and muesli bars. For each food item, the total number of servings consumed on a usual day and in the last hour before bed was requested. The AUSNUT 2011–2013 food composition database[15], that enables food, dietary supplement and nutrient intake estimates to be made from dietary data, was used to generate grams per day intake of added sugar in FoodWorks version 8 dietary analysis package (a nutritional analysis software)[16]. A qualified nutritionist (KK) carried out the dietary analysis. Questionnaire and database foods were matched using the most similar generic food item. Serving sizes were initially generated by consulting the NSAOH questionnaire; where these were unclear, a serving size for the selected food or beverage was obtained through the standard portion sizes, as outlined in the Australian Guide to Healthy Eating (AGHE). Where foods were not available from the

AGHE (e.g. discretionary foods), a value of ~600 kJ was attributed.

Data analysis

The STATA version 15 was used for statistical analysis (StataCorp. 2017)[17]. The complex survey design of the NSAOH was accounted for; data were stratified by metropolitan and rural regions, clustered by post code and weighted for the probability of participants being selected for inclusion in the questionnaire. Categorisations used were: age (15–44 years; 45–59 years; and ≥ 60 years); sex (male; female); education (degree/teaching and nursing; trade/diploma/certificate; and high school or less); total household income in Australian dollars (< $30 thousand; $30 thousand to < $60 thousand; and ≥ $60 thousand); alcohol intake (2 drinks per day; ≥ 2 drinks per day); smoking (current; former; and never); self-reported diabetes (yes or no); toothbrushing (< 2 times per day; and ≥ 2 times per day); flossing (yes; and no); mouth rinsing (yes; and no); usual reason for dental visit (problem-based dental visits or regular checkup); and body mass index (BMI; underweight/normal weight '< 25 kg/m^2'; and overweight/obese '≥ 25 kg/m^2'). Periodontitis was defined according to the Center for Diseases Control and the American Academy of Periodontology case definition of periodontitis (no periodontitis or periodontitis)[18], where no or mild periodontitis were grouped as 'no periodontitis', and moderate or severe periodontitis were grouped as 'periodontitis'. National guidelines for alcohol consumption were used as a recommendation for daily alcohol intake, which defined a standard alcohol beverage contains 10 g of alcohol is an established metric for estimating alcohol consumption across drink types[19]. The American Heart Association recommendation that added sugar intake should be limited to 36 g/day for men and 25 g/day for women[20] was adopted for the analysis.

The variables were analysed using frequency distribution for categorical variables and mean (standard deviations) for the continuous variables. Bivariate analysis (using Rao-Scott chi-square test and independent sample *t*-test) were conducted for obesity and its putative confounders; and for periodontitis and its putative confounders. The independent sample *t*-test was used to analyse periodontal parameters (sites with PD ≥ 4 mm and sites with CAL ≥ 4 mm) in relation to putative confounders. The American Academy of Periodontology and Center for Disease Control criteria were used to allow comparisons with previous studies. Multiple variable models (linear and logistic regression models) were generated to analyse the association of obesity and periodontitis when controlled for the putative confounders.

The ethics approval for NSAOH 2004–2006 was obtained from University of Adelaide Human Research Ethics Committee, and the study was conducted according to the World Medical Association Declaration of Helsinki (version, 2008). All examined subjects provided a signed, informed consent to participate in the study. For participants under the age of 18 years, a written informed consent was obtained from the parents/guardians, approved by the University of Adelaide Human Research Ethics Committee.

RESULTS

A total of 4,170 participants completed the mailed questionnaire and were included in the data analysis. The prevalence of overweight and obesity in the NSAOH was found as 32.7% and 16.6%, respectively. Characteristics of participants were described in *Table 1*. More than half of the participants were overweight/obese, aged 15–44 years, had high school or lower education level, were non-smokers, consumed less than two drinks of alcohol per day, rinsed their mouth daily, brushed their teeth twice daily, and visited their dentist for regular check-ups (*Table 1*). About 48% of the participants flossed daily. Nearly 47% of participants had an annual income more than $60,000 per annum. Males and females were equally distributed in the sample. Less than 5% of participants had self-reported diabetes. The proportion of participants with periodontitis was 22.7%. Approximately 17% of the participants had added sugar intake above the American Heart Association recommended level of added sugar intake. Individuals aged 15–19 years and 20–30 years were distributed as 3.5% and 8.4%, respectively, in the sample population. Only one participant in the 15–19 years had periodontitis, and 21 participants aged 20–30 years had periodontitis. This sample size was not considered large enough for further analysis.

Bivariate analysis found that the factors significantly associated with obesity included age (45–59 years; $P < 0.001$); male ($P < 0.001$); no university level of education ($P < 0.05$); former smoking habit ($P < 0.001$); and problem-based dental visits ($P < 0.001$; *Table 2*). No association was observed between added sugar intake and obesity in the bivariate analysis. It was found that the factors that were significantly associated with periodontitis (*Table 3*) included being age ≥ 60 years ($P < 0.05$); male ($P < 0.05$); having an annual income less than $30,000 ($P < 0.05$); less than secondary education ($P < 0.05$); a current smoking habit ($P < 0.05$); problem-based dental visiting behaviour ($P < 0.05$); and being overweight/obese ($P < 0.05$). Added sugar intake was inversely associated with periodontitis ($P < 0.05$).

Table 1 Characteristics of participants

Characteristic	Level	*n*	Weighted percentage %	95% CI (lower)	95% CI (upper)
Age	15–44	1,591	56.7	54.2	59.3
	45–59	1,333	25.2	23.3	27.1
	60+	1,246	18.1	16.4	19.7
Sex	Male	1,604	50.0	47.4	52.6
	Female	2,566	50.0	47.4	52.6
Income	<30k	1,253	24.1	21.7	26.6
	30k–< 60k	567	29.4	26.9	31.9
	60k+	2,093	46.5	43.3	49.8
Education	High school or less	1,349	56.4	52.7	60.0
	Trade/Dip/cert	1,351	38.6	35.2	42.0
	Degree/teaching/nursing	1,372	5.0	3.9	6.3
Diabetes	Yes	213	4.3	3.2	5.5
	No	3,956	95.7	94.5	96.8
Smoking	Current	578	15.2	13.2	17.1
	Previous	1,315	27.3	25.1	29.5
	Never	2,277	57.5	55.0	60.1
Alcohol	≤ 2 drinks	2,191	57.7	54.5	60.8
	> 2 drinks	1,152	42.3	39.2	45.5
Usual reason for dental visit	Check-up	1,712	58.5	52.6	58.8
	Problem	1,602	41.5	41.2	47.4
Mouth rinsing	No	2,821	41.9	39.3	44.6
	Yes	1,347	58.1	55.4	60.7
Tooth brushing	< 2 day	1,612	44.5	41.8	47.2
	≥ 2 day	2,535	55.5	52.8	58.2
Flossing	No	1,952	51.5	48.6	54.3
	Yes	2,217	48.5	45.7	51.4
BMI	Underweight/normal	1,685	48.1	45.9	51.9
	Overweight/obese	2,060	51.9	48.1	54.1
		Mean	SE		
Added sugar	Grams/day	19.1	0.5	18.1	20.1

BMI, body mass index; CI, confidence interval.

In bivariate analysis, periodontal parameters of sites with PD ≥ 4 mm ($P < 0.05$) and sites with CAL ≥ 4 mm ($P < 0.001$) were significantly associated with obesity. The putative confounders that were significantly associated with mean number of sites with PD ≥ 4 mm included sex ($P < 0.01$), smoking ($P < 0.001$), alcohol ($P < 0.05$), usual reason for dental visit ($P < 0.001$), BMI ($P < 0.05$), mouth rinsing ($P < 0.001$), toothbrushing ($P < 0.01$) and flossing ($P < 0.03$; *Table 4*). The putative confounders that were significantly associated with mean number of sites with CAL ≥ 4 mm included age ($P < 0.001$), sex ($P < 0.001$), education ($P < 0.001$), income ($P < 0.001$), self-reported diabetes ($P < 0.05$), smoking ($P < 0.001$), usual reason for dental visit ($P < 0.001$), BMI ($P < 0.001$) and added sugar intake ($P < 0.001$).

The multiple variable analysis indicated that periodontal parameters: sites with PD ≥ 4 mm [odds ratio (OR) 0.13; 95% confidence interval (CI): −0.86, 0.35]; sites with CAL ≥ 4 mm (OR: −0.11; 95% CI: −0.56, 0.35; *Table 5*); and presence of periodontitis (OR: 1.23; 95% CI: 0.96, 1.57; *Table 6*) were not associated with overweight/obesity when controlled for putative confounders that were significant in the respective bivariate analysis. As mean estimates can be skewed, the bivariate and multiple variable analyses were recalculated using proportions for CAL ≥ 4 mm and PD ≥ 4 mm, and the results were very similar to those found using means (Appendices S1 and S2).

DISCUSSION

After adjusting for the putative confounders in three multiple variable regression models, no association was found with overweight/obesity and periodontitis. This could be a result of complexity in the relationship between overweight/obesity, periodontitis and putative confounders, including smoking, diabetes and dental visiting behaviour.

The finding is supported by de Castilhos *et al.*[21]; their study suggested there was no association between obesity and periodontitis in young adults. Similar results were found in the fourth Korean National Health and Nutrition Examination Survey (KNHANES) and Health 2000 Health Examination Survey of Finland, where obesity was not associated with periodontitis in adults after adjusting for putative confounders[22,23]. Contrary to this finding, numerous systematic reviews report a significant association between obesity and periodontitis[8,10].

Table 2 Bivariate analysis of BMI (underweight/normal BMI ≤ 25 kg/m^2, overweight/obese BMI as > 25 kg/m^2) and putative confounders

Characteristic	Level	Underweight/ normal %	95% CI (lower)	95% CI (upper)	Overweight/ obese %	95% CI (lower)	95% CI (upper)	*P*-value
Age	15–44	57.5	52.7	62.3	42.5	37.7	47.3	
	45–59	37.8	33.9	41.6	62.2	58.4	66.1	
	60+	37.1	33.3	40.9	62.9	59.1	**66.7**	**< 0.001**
Sex	Male	43.2	38.1	48.2	56.8	51.8	61.9	
	Female	54.9	51.7	58.1	45.1	41.9	**48.3**	**< 0.001**
Income	< 30k	44.0	37.9	50.2	56.0	49.8	62.1	
	30k–< 60k	47.1	42.7	51.5	52.9	48.5	57.3	
	60k+	49.4	44.3	54.5	50.6	45.5	55.7	0.366
Education	High school or less	50.7	42.2	52.7	49.3	47.3	57.8	
	Trade	41.1	36.4	47.3	58.9	52.7	63.6	
	Degree	39.6	46.0	56.8	60.4	43.2	**54.0**	**0.027**
Diabetes	Yes	47.4	42.2	52.7	52.6	46.0	56.8	
	No	41.9	36.4	47.3	58.1	47.3	57.8	0.1222
Smoking	Current	50.2	43.0	57.5	49.8	42.5	57.0	
	Previous	37.1	32.3	41.8	62.9	58.2	67.7	
	Never	54.1	50.2	57.9	45.9	42.1	**49.8**	**< 0.001**
Alcohol	≤ 2 drinks	45.0	42.0	49.8	55.0	50.2	58.0	
	> 2 drinks	47.8	42.7	54.3	52.2	45.7	57.3	0.401
Usual reason for dental visit	Check-up	54.3	50.1	58.5	45.7	41.5	49.9	
	Problem	40.9	36.4	45.4	59.1	54.6	**63.6**	**< 0.001**
Mouth rinsing	No	47.6	42.9	52.2	52.4	47.8	57.1	
	Yes	49.9	46.1	53.6	50.1	46.4	53.9	0.498
Tooth brushing	< 2 times/day	45.0	39.7	50.4	55.0	49.6	60.3	0.08
	≥ 2 times/day	50.6	47.1	54.1	49.4	45.9	52.9	
Flossing	No	46.5	41.8	51.3	53.5	48.7	58.2	0.24
	Yes	49.9	46.3	53.5	50.1	48.7	54.9	

		Underweight/normal mean	95% CI (lower)	95% CI (upper)	Overweight/ obese mean	95% CI (lower)	95% CI (upper)	*t*-test *P*-value
Added sugar (mean)	Grams/day	19.7	17.7	21.7	18.8	17.5	19.8	0.455

Chi-squared test for significance used for categorical predictors, *t*-test for continuous predictors.
Bold values indicate significance at $P < 0.05$.
CI, confidence interval.

Periodontitis occurs as a result of interaction between dental plaque biofilm and the host immune response from modifiable factors including obesity. People with obesity have a pro-inflammatory state, due to the deposition of visceral adipose tissues that secrete immune-modulatory factors such as leptin, adiponectin, complement components, plasminogen activator inhibitor-1, proteins of the renin-angiotensin system, and resistin[24]. These bioactive molecules modulate angiogenesis, hormonal and metabolic changes, that lead to increased systemic inflammation and acute phase reactions[24]. These events appear to be related to periodontal disease and an enhanced host response in the periodontal tissues.

This study found that obesity was significantly associated with the periodontal parameters of sites with PD ≥ 4 mm in the bivariate analysis. This result was in accord with the Hisayama study that found that obesity was significantly associated with deep pockets (sites with PD ≥ 4 mm)[25]. Similar results were found in Japanese adults where, with each 1 kg/m^2 increase in BMI, there was a 16% increased risk of periodontitis (sites with PD ≥ 4 mm)[26]. Deep pocket is a measure of active or existing periodontal disease, therefore it could be proposed that obesity was associated with the primary stage of periodontal disease.

In this study, no association was found between obesity and sites with CAL ≥ 4 mm. The results of our study are similar to those reported by Saito *et al.*[25], where it was found that obesity was not associated with CAL. CAL is a measure of alveolar bone loss, and is an indicator of history of periodontal disease. It occurs as a result of inflammation that leads to periodontal tissue destruction[25].

Periodontal disease experience among the NSAOH 2004–2006 sample was significantly associated with increasing age, being male, a current or former smoking habit, and problem-based dental visiting behaviours. These findings are in line with previous studies[27–33].

Periodontitis is an age-dependent disease with increasing prevalence and severity of periodontitis among adults 30 years and older[27]. The results of this study reflect a similar population-based study amongst 30–39-year-old and 50–59-year-old Thai adults, suggesting a significant increase in periodontal outcome measures (PD and CAL) among the older age group as compared with the younger counterpart[28]. The

Table 3 Bivariate analysis of periodontitis and putative confounders

Characteristic	No periodontitis	95% CI (lower)	95% CI (upper)	Periodontitis	95% CI (lower)	95% CI (upper)
Age						
15–44	89.1	86.7	91.1	10.9	8.9	13.3
45–59	68.4	64.5	72.1	31.6	27.9	35.5
60+	52.4	48.3	56.4	**47.6***	43.6	51.7
Sex						
Male	72.4	69	75.6	**27.6***	24.4	31.0
Female	81.9	79.9	83.7	18.1	16.3	20.1
Income						
≤ 30k	63.4	59	67.6	36.6*	32.4	41.0
>30k–< 60k	75.3	71.4	78.8	24.7	21.2	28.6
60k+	83	79.9	85.7	17	14.3	20.1
Education						
University/teaching/nursing	79	75.9	81.8	21	18.2	24.1
Trade certificate/Dip/Cert	75.9	72.3	79.3	24.1	20.7	27.7
No post-secondary education	64.1	52.2	74.6	**35.9***	25.4	47.8
Diabetes – self-reported						
Yes	62.2	51.9	71.6	37.8	28.4	48.1
No	77.9	75.9	79.8	22.1	20.2	24.1
Smoking						
Current	70.5	64.8	75.6	29.5*	24.4	35.2
Former	71.1	67.3	74.7	28.9	25.3	32.7
Never	82.1	79.7	84.3	17.9	15.7	20.3
Alcohol						
≤ 2 drinks	76.3	73.5	78.8	23.7	21.2	26.5
> 2 drinks	77.1	73.3	80.4	22.9	19.6	26.7
Mouth rinsing						
No	79.2	76.5	81.7	20.8	18.3	23.5
Yes	75.9	73.2	78.5	24.1	21.5	26.8
Tooth brushing						
< 2 times/day	79.2	75.8	82.2	20.8	17.7	24.2
≥ 2 times/day	78.1	75.4	80.5	21.9	19.4	24.6
Flossing						
No	77.1	73.9	80.0	22.9	20.0	26.1
Yes	80.2	77.6	82.6	19.8	17.4	22.4
Usual reason for dental visit						
Check-up	81.3	79.1	83.4	18.7	16.6	20.9
Problem	71.6	68.5	74.5	**28.4***	25.5	31.5
BMI						
Underweight/normal	83.9	80.4	85.3	16.1	14.7	19.6
Overweight/obesity	74.7	70	76.2	**25.3***	23.8	30.0
Variables						
Added sugar	19.2	18.0	20.2	**17.2***	16.2	18.1

Chi-squared test for significance used for categorical predictors, *t*-test for continuous predictors.
Bold values indicate significance at $P < 0.05$.
BMI, body mass index; CI, confidence interval.

concept of ageing as a potential marker for periodontitis has evolved with research, from being an inevitable consequence of ageing to the current concept of periodontitis as a cumulative effect of long-term exposure to true risk factors[29].

The limitations of this study include the following: (i) the self-reported body height and weight measures, that introduced a reporting bias. Obesity is a widespread societal issue. Individuals with overweight/obesity tend to underestimate their weight status as compared with people of normal weight[30]; (ii) the cross-sectional study design is another limitation of this study, which makes it difficult to determine the temporal relationship between obesity and periodontitis. It is advised that in future studies should incorporate a prospective cohort study design, to investigate the causality between obesity and periodontitis; (iii) self-reported 13 dietary questions, that resulted in inability to calculate the absolute intake of sugar and total energy intake, and other macronutrients in the diet. The ecology of nutrition is based on a combination of dietary intake factors, rather than a single factor or caloric intake; (iv) type 2 diabetes was self-reported, and no objective measure of type 2 diabetes was used. Hence, it hindered the ability to determine the severity of type 2 diabetes in relation to risk and progression of periodontitis; (v) the use of conventional regression analysis is another limitation of this study. In regression analysis, the confounders and mediators of exposure (obesity) and outcome (periodontitis) are grouped together as putative confounders/mediators, which limits the ability to truly understand the effect of exposure on outcome; (vi) NSAOH did not

Table 4 Bivariate analysis of periodontal parameters (Chi-squared test for significance used for categorical predictors, *t*-test for continuous predictors)

Characteristic	PD ≥ 4 mm (mean)	95% CI (lower)	95% CI (upper)	*P*-value	CAL ≥ 4 mm	95% CI (lower)	95% CI (upper)	*P*-value
Age								
15–44	**0.6**	**0.4**	**0.9**	**0.33**	**0.9**	**0.7**	**1.1**	**< 0.001**
45–59	0.9	0.6	1.1		3.4	3.0	3.9	
60+	0.7	0.6	0.9		6.1	5.4	6.9	
Sex								
Male	0.9	0.6	1.2	**0.01**	2.8	2.4	3.2	**< 0.001**
Female	0.5	0.4	0.6		1.9	1.7	2.1	
Income								
< 30k	1.1	0.6	1.6	0.06	4.3	3.6	5.0	**< 0.001**
> 30k–< 60k	0.7	0.5	0.9		2.6	2.1	3.1	
60k+	0.6	0.4	0.8		1.6	1.3	1.9	
Education								
University/teaching/nursing	0.5	0.6	0.7	0.15	2.1	1.8	2.5	**< 0.001**
Trade certificate/Dip/Cert	0.8	0.6	1.0		2.7	2.3	3.2	
No post-secondary education	2.0	−0.8	4.8		4.8	2.8	6.9	
Diabetes – self-reported								
Yes	0.8	0.4	1.3	0.64	3.8	2.2	5.3	**0.03**
No	0.7	0.5	0.9		2.3	2.1	2.6	
Smoking								
Current	1.9	1.0	2.7	**< 0.001**	3.6	2.8	4.5	**< 0.001**
Former	0.6	0.5	0.8		3.1	2.7	3.6	
Never	0.4	0.3	0.5		1.7	1.4	1.9	
Alcohol								
≤ 2 drinks	0.6	0.4	0.7	**0.02**	2.4	2.1	2.7	0.669
> 2 drinks	1.0	0.6	1.4		2.5	2.0	2.9	
Usual reason for dental visit								
Check-up	0.4	0.3	0.5	**< 0.001**	1.9	1.7	2.2	**< 0.001**
Problem	1.1	0.8	1.5		3.0	2.6	3.4	
BMI								
Underweight/normal weight	0.5	0.4	0.7	**0.05**	1.9	1.5	2.2	**< 0.001**
Overweight/obese	0.8	0.6	0.9		2.8	2.4	3.2	
Mouth rinsing								
No	0.5	0.4	0.6	**< 0.001**	2.3	1.9	2.6	0.52
Yes	0.9	0.6	1.1		2.4	2.1	2.7	
Tooth brushing								
< 2 times/day	0.9	0.6	1.3	**< 0.01**	2.2	1.8	2.6	0.15
≥ 2 times/day	0.5	0.4	0.6		2.5	2.2	2.8	
Flossing								
No	0.9	0.6	1.2	**0.03**	2.5	2.1	2.9	0.20
Yes	0.5	0.4	0.7		2.2	2.0	2.5	
Added sugar								
Grams/day	19.2	18.2	20.3	0.66	19.2	18.2	20.3	0.009

Bold values indicate significance at $P < 0.05$.
BMI, body mass index; CAL, clinical attachment loss; CI, confidence interval; PD, probing depth.

Table 5 Multiple variable linear regression models for periodontal parameters (mean number of sites CAL ≥ 4 mm and mean number of sites with PD ≥ 4 mm) in relation to potential confounding variables

Characteristic	Level	Coefficient (CAL ≥ 4 mm)	95% CI (lower)	95% CI (upper)	*P*-values	Coefficient (PD ≥ 4 mm)	95% CI (lower)	95% CI (upper)	*P*-values
BMI	Overweight/obese	−0.1	−0.6	0.4	0.61	0.1	−0.9	0.3	0.23
Age	60+	2.8	2.4	3.3	< 0.001				
Sex	Female	−0.6	−1.1	−0.1	0.01	−0.1	−0.3	0.1	0.17
Education	Degree/teaching/nursing	0.3	−0.3	0.8	0.29				
Smoking	Never	−0.6	−1.00	−0.2	0.002	−0.4	−0.6	0.2	**< 0.001**
Usual reason for dental visit	Check-up	0.7	0.2	1.2	0.007	0.4	0.2	0.6	**< 0.001**

Bold values significance at $P < 0.05$.
BMI, body mass index; CAL, clinical attachment loss; CI, confidence interval; PD, probing depth.

provide information to enable the measurement of the severity and extent of periodontitis. A major strength of this study was that NSAOH is only the second nationwide survey on oral health in Australia; it had a large sample size; and a small degree of non-participation bias[34].

Table 6 Multiple variate binary logistic regression analysis – adjusted model for periodontitis, obesity and confounding factors (age, sex, smoking and usual reason for dental visit)

Characteristic	Levels	OR	95% CI (upper)	95% CI (lower)	*P*-value
BMI	Normal weight	1 (Reference)			
	Overweight/obese	1.2	0.9	1.5	0.09
Age	15–44	1 (Reference)			
	4–59	1.2	0.9	1.4	< 0.001
	60+	2.5	1.7	3.6	< 0.001
Sex	Female	1 (Reference)			
	Male	1.7	1.3	2.1	< 0.01
Smoking	Never	1 (Reference)			
	Current	1.8	1.2	2.6	< 0.001
	Former	1.3	1.0	1.7	< 0.05
Usual reason for dental visit	Check-up	1 (Reference)	–	–	–
	Problem	1.6	1.2	2.0	< 0.05

BMI, body mass index; CI, confidence interval; OR, odds ratio.

Future research needs to be undertaken with recording of complete 24-hour food-recall diaries of participants, and with utilisation of robust statistical methods such as mediation analysis or marginal structural modelling to truly understand the causal relationship between obesity and periodontitis. In addition, future studies should not combine 'overweight' and 'obesity', and that four rather than two BMI categories be used in the analysis.

CONCLUSION

Within the limitations of this study, no association was observed between overweight/obesity and periodontitis. Future studies should adopt objective measures of obesity and diet. The risk factors of age, sex, smoking and dental visiting behaviour were found to be associated with periodontitis.

PUBLIC HEALTH IMPLICATIONS

Risk behaviours such as poor dental visiting behaviour, smoking, and age are significantly associated with periodontitis as compared with obesity or being overweight. Actions are required towards promotion of healthy behaviours, including regular visits to a dentist, maintenance of oral hygiene and avoiding smoking habit to reduce the periodontitis burden in Australians.

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Conflict of interests

The authors declare that they have no competing interests.

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AUTHOR CONTRIBUTIONS

SK, SB, TB and LC conceived and designed the study. SK, SB and LC acquired the data. SK, KK, SB and LC analysed and interpreted the data. SK drafted the report. SK, SB, KK, TB, MP and LC critically revised the report for important intellectual content. SK performed the statistical analysis.

SUPPORTING INFORMATION

Additional supporting information may be found in the online version of this article:

Appendix S1. Bivariate analysis of proportion of sites with PD $\geq$ 4 mm and proportion of sites with CAL $\geq$ 4 mm in relation to putative confounders. **Appendix S2**. Multiple variable logistic regression model of proportion of sites with CAL $\geq$ 4 mm adjusted for BMI, age, sex, smoking and usual reason for dental visit.

REFERENCES

1 ABS. *National Health Survey: First Results, 2014–15*. Canberra: Australian Bureau of Statistics; 2015.

2 Rutter H, Bes-Rastrollo M, De Henauw S *et al.* Balancing upstream and downstream measures to tackle the obesity

epidemic: a position statement from the European Association for the Study of Obesity. *Obes Facts* 2017 10: 61–63.

3 Swinburn BA, Sacks G, Hall KD *et al*. The global obesity pandemic: shaped by global drivers and local environments. *Lancet* 2011 378: 804–814.

4 AIHW, editor. *Impact of overweight and obesity as a risk factor for chronic conditions: Australian Burden of Disease Study*. Canberra: AIHW; 2017.

5 Löe H, Theilade E, Jensen SB. Experimental gingivitis in man. *J Periodontol* 1965 36: 177–187.

6 Van Dyke TE, Van Winkelhoff AJ. Infection and inflammatory mechanisms. *J Clin Periodontol* 2013 40: S1–S7.

7 Khan S, Khalid T, Awan KH. Chronic periodontitis and smoking prevalence and dose-response relationship. *Saudi Med J* 2016 37: 889.

8 Khan S, Barrington G, Bettiol S *et al*. Is overweight/obesity a risk factor for periodontitis in young adults and adolescents?: A systematic review. *Obes Rev* 2018 19: 852–883.

9 Roberts-Thomson K, Do L. Oral health status. Australia's dental generations: The national survey of adult oral health. Canberra: Australian Institute of Health and Welfare (Dental Statistics and Research Series No. 34): Cat. no. DEN 165; 2007. Contract No.: 2007.

10 Keller A, Rohde JF, Raymond K *et al*. Association between periodontal disease and overweight and obesity: a systematic review. *J Periodontol* 2015 86: 766–776.

11 Maciel SS, Feres M, Gonçalves TED *et al*. Does obesity influence the subgingival microbiota composition in periodontal health and disease? *J Clin Periodontol* 2016 43: 1003–1012.

12 Zimmermann GS, Bastos MF, Dias Goncalves TE *et al*. Local and circulating levels of adipocytokines in obese and normal weight individuals with chronic periodontitis. *J Periodontol* 2013 84: 624–633.

13 Fedewa MV, Das BM, Evans EM *et al*. Change in weight and adiposity in college students: a systematic review and meta-analysis. *Am J Prev Med* 2014 47: 641–652.

14 Slade GDSA, Roberts-Thompson K. *Australia's dental generations: the National Survey of Adult Oral Health 2004–06*. Canberra: Australian Institute of Health and Welfare; 2007.

15 FSANZ. AUSNUT 2011–13 Food Nutrient Database. Canberra: Food Standard Australia New Zealand; 2014.

16 Xyris software. FoodWorks, Dietary Analysis Package version 5, Highgate Hill, QLD, Australia; 2007.

17 StataCorp. Stata Statistical Software: Release 15. Texas: StataCorp LLC; 2017.

18 Page RC, Eke PI. Case definitions for use in population-based surveillance of periodontitis. *J Periodontol* 2007 78: 1387–1399.

19 DOHA. *New National Guidelines for Alcohol Consumption: Standard Drink Guide*. Canberra, Australia: Department of Health and Ageing, Australian Government; 2018.

20 AHA. Added Sugars: American Heart Association; 2017 Available from: http://www.heart.org/HEARTORG/HealthyLiving/HealthyEating/Nutrition/Added-Sugars_UCM_305858_Article.jsp#.WnkSD6iWbct

21 de Castilhos ED, Horta BL, Gigante DP *et al*. Association between obesity and periodontal disease in young adults: a population-based birth cohort. *J Clin Periodontol* 2012 39: 717–724.

22 Kim EJ, Jin BH, Bae KH. Periodontitis and obesity: a study of the Fourth Korean National Health and Nutrition Examination Survey. *J Periodontol* 2011 82: 533–542.

23 Ylöstalo P, Suominen-Taipale L, Reunanen A *et al*. Association between body weight and periodontal infection. *J Clin Periodontol* 2008 35: 297–304.

24 Pischon N, Heng N, Bernimoulin JP *et al*. Obesity, inflammation, and periodontal disease. *J Dent Res* 2007 86: 400–409.

25 Saito T, Shimazaki Y, Kiyohara Y *et al*. Relationship between obesity, glucose tolerance, and periodontal disease in Japanese women: the Hisayama study. *J Periodontal Res* 2005 40: 346–353.

26 Ekuni D, Yamamoto T, Koyama R *et al*. Relationship between body mass index and periodontitis in young Japanese adults. *J Periodontal Res* 2008 43: 417–421.

27 Al-Zahrani MS, Bissada NF, Borawski EA. Obesity and periodontal disease in young, middle-aged, and older adults. *J Periodontol* 2003 74: 610–615.

28 Bælum V, Pisuithanakan S, Teanpaisan R *et al*. Periodontal conditions among adults in Southern Thailand. *J Periodontal Res* 2003 38: 156–163.

29 Lang NP, Schätzle MA, Löe H. Gingivitis as a risk factor in periodontal disease. *J Clin Periodontol* 2009 36: 3–8.

30 Robinson E, Oldham M. Weight status misperceptions among UK adults: the use of self-reported vs. measured BMI. *BMC Obes* 2016 3: 21.

31 Johnson GK, Guthmiller JM. The impact of cigarette smoking on periodontal disease and treatment. *Periodontology 2000* 2007 44: 178–194.

32 Hill K, Chadwick B, Freeman R *et al*. Adult Dental Health Survey 2009: relationships between dental attendance patterns, oral health behaviour and the current barriers to dental care. *Br Dent J*. 2013 214: 25–32.

33 Crocombe LA, Broadbent JM, Thomson WM *et al*. Impact of dental visiting trajectory patterns on clinical oral health and oral health-related quality of life. *J Public Health Dent* 2012 72: 36–44.

34 Do LG, Slade GD, Roberts-Thomson KF *et al*. Smoking-attributable periodontal disease in the Australian adult population. *J Clin Periodontol* 2008 35: 398–404.

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