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ORIGINAL ARTICLE

Association between obesity and periodontal risk indicators in adolescents

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Abstract

Objective. In a cross-sectional study design we test the hypothesis of whether obesity in adolescence is associated with periodontal risk indicators or disease. *Study design.* Obese adolescents (n=52) and normal weight subjects (n=52) with a mean age of 14.5 years were clinically examined with respect to dental plaque, gingival inflammation, periodontal pockets and incipient alveolar bone loss. The subjects answered a questionnaire concerning medical conditions, oral hygiene habits, smoking habits and sociodemographic background. Body mass index (BMI) was calculated and adjusted for age and gender (BMI-SDS). Samples of gingival crevicular fluid (GCF) were analyzed for the levels of adiponectin, plasminogen activator inhibitor-1 (PAI-1), interleukin-1β (IL-β), interleukin-8 (IL-8) and tumor necrosis factor α (TNF-α). *Results.* Obese subjects exhibited more gingival inflammation ($P<0.001$) and more pathological periodontal pockets (>4 mm) ($P<0.001$) but not incipient alveolar bone loss compared with the normal weight subjects. Higher levels of IL-1β ($P<0.001$) and IL-8 ($P=0.002$) were measured in GCF from obese subjects compared with the controls. In a multivariate logistic regression analysis, adjusted BMI-SDS ($P=0.03$; Odds Ratio [OR]=1.87) was significantly associated with the occurrence of pathological periodontal pockets. *Conclusion.* The study demonstrates an association between obesity and periodontal risk indicators in adolescents that in the long term may lead to oral morbidity. This result further strengthens obesity's negative effect on teenagers' periodontal health and highlights the importance of a close collaboration betweens dentists and pediatricians in the prevention and treatment of obesity.

Key words: *Obesity, adolescents, periodontal disease, IL-8, IL-1β*

Introduction

An increasing prevalence of obesity is well documented in all ages and ethnicities worldwide (1–3). Obesity has become a major global health concern as it is associated with increased risk of cardiovascular disease (4) and reduced life expectancy (5,6). From an oral perspective it has been reported that obese adolescents exhibited more proximal caries lesions (7), although a systematic review revealed contradictory results (8). In addition, a relationship between obesity and periodontal disease was first demonstrated by Saito et al. (9). Since then, several cross-sectional studies have reported that obesity is associated with enhanced prevalence of chronic periodontitis in adults (10–16). The association between obesity and chronic periodontitis has also been shown in young adults (15), whereas no association was demonstrated between obesity and periodontal disease among children (12).

The mechanism(s) whereby obesity may affect periodontal health is so far unclear. In an animal model; however, obesity interferes with the ability of the immune system to appropriately respond to the periodontal pathogen *Phorphyromonas gingivalis* infection in terms of TNF-α, IL-6 and serum amyloid response, suggesting that this immune dysregulation participates in the increased alveolar bone loss after bacterial infection (17).

Adipose tissue, a dynamic endocrine organ, secretes a number of pro- and anti-inflammatory cytokines, so called adipokines, including TNF-α, IL-6, IL-8, leptin, plasminogen activator inhibitor-1,

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resistin, adiponectin, and angiotensinogen (18–20), and the levels of these adipokines in plasma are influenced by the obese condition. Moreover, obesity is accompanied by generalized inflammation characterized by increased plasma level of CRP, as well as dysregulated cytokine production and the presence of endothelial and vascular dysfunction (21,22).

In adolescents aged 18 years old with severe obesity, we previously reported that the level of TNF-α in gingival crevicular fluid (GCF) is positively correlated with BMI, indicating that the level of TNF-α in GCF was affected by the obese condition (23). Although there are data indicating an association between obesity and periodontal disease, it is uncertain whether the association between obesity and periodontal disease can be already observed during early adolescence. Therefore, here we test the hypothesis of whether obese adolescence is associated with periodontal risk indicators or disease compared with age- and gender-matched controls.

Material and methods

The present cross-sectional study was conducted on 104 subjects (Table I) with a mean age of 14.5 years, 52 obese (range 11.0–17.9 years) and 52 normal weight adolescents (controls) (range 10.9–17.1 years). The obese subjects were treated at the National Childhood Obesity Center, Karolinska University Hospital, Huddinge and consecutively referred to the Division of Pediatric Dentistry in order to study their oral co-morbidity. All subjects had a BMI within the obesity range for age (BMI >30) (24). The normal weight control patients (BMI <25) were recruited randomly from the Division of Pediatric Dentistry, Department of Dental Medicine, Karolinska Institutet and consisted of individuals receiving their regular dental check-up at the department. The controls were age and gender matched. The following exclusion criteria were used for both groups; any antibiotic treatment during the last 3 months and/or ongoing orthodontic treatment. Subjects that admitted to smoking daily were also excluded. Body weight (kg) and height (m) of the subjects were determined and their body mass was expressed as BMI (kg/m^2), as well as by BMI adjusted for age and sex (BMI-SDS) (25).

Questionnaire

All the subjects answered a questionnaire that covered topics of the subject's medical condition, medication, dietary habits and oral hygiene habits, as well as their parent's educational level and country of birth. In case subjects did not understand the Swedish language an interpreter assisted. Parent's country of birth was categorized into "born in Sweden" or "born abroad". The educational level of the parents was stratified according to years of schooling as: 1) (≤9 years); 2) (10–12 years); and 3) ≥12 years.

Table I. Characteristics of the subjects.

Variables	Obesity (n=52) Mean (SD)	Control (n=52) Mean (SD)	P-value
Male/female	29/23	29/23	1.000[3]
Age	14.5 (1.6)	14.5 (1.7)	0.837[2]
Weight (kg)	107.0 (23.9)	54.3 (11.7)	<0.001[2]
Height (m)	1.69 (0.10)	1.65 (0.12)	0.037[2]
BMI (kg/m^2)	37.0 (5.7)	19.7 (2.3)	<0.001[2]
BMI-SDS[1]	5.7 (1.1)	0.3 (0.9)	<0.001[2]
Chronic disease			
Asthma	4/52	3/52	
Diabetes type 1	0/52	1/52	
Diabetes type 2	3/52	0/52	
Thyroid dysfunctions	4/52	0/52	
Epilepsy	0/52	1/52	
Neuropsychological	1/52	0/52	
Polycystic ovaries	1/52	0/52	
Country of birth			
Mother			
Sweden	26/52	21/52	
Not Sweden	25/52	31/52	
Unknown	1/52	0/52	0.280[3]
Father			
Sweden	25/52	27/52	
Not Sweden	26/52	24/52	
Unknown	1/52	1/52	0.540[3]
Education, mother			
≤9 years	12/52	9/52	
10–12 years	26/52	24/52	
>12 years	13/52	14/52	
Unknown	1/52	5/52	0.583[3]
Education, father			
≤9 years	14/52	11/52	
10–12 years	23/52	24/52	
>12 years	13/52	11/52	
Unknown	2/52	6/52	0.133[3]

[1]Roland-Cacherra.
[2]ANOVA as statistical method.
[3]Chi-square as statistical method.
SD: Standard deviation.

Clinical examination

Gingival inflammation. The presence of dental plaque on tooth surfaces were recorded when clearly visible on tooth surfaces and expressed using the Visible Plaque Index (VPI%) (26). Gingival inflammation was based on Bleeding on Probing (BOP%) of the gingival sulcus of all teeth (wisdom teeth excluded) at six points. The proportion of surfaces (%) with visible dental plaque and gingival inflammation, respectively, was calculated for each individual.

Pathological periodontal pocket. Pocket depth (mm) was recorded by using a graded periodontal probe

(LM-instruments OY, Finland) and measured to the nearest mm. The occurrence of pathological periodontal pocket was classified when the subject exhibited one or more sites with a pocket depth of >4 mm.

Calculus. Supragingival calculus was recorded on all teeth as present or absent when clearly visible. Subgingival calculus was recorded as present or absent on proximal surfaces of first molar and premolars on the radiographs taken, as well as clinically after probing the gingival sulcus.

Incipient alveolar bone loss. In order to determine marginal alveolar bone loss, two bitewing radiographs were taken using standardized technique. The distance between cemento-enamel junction (CEJ) and alveolar bone crest (AC) on the radiographs was measured on the mesial and distal surfaces of premolars and first molar by using a Peak scale loupe (Carton Optic Tokyo Japan; 7-fold magnification). Incipient marginal alveolar bone loss was classified as positive when the distance from CEJ to AC on the radiographs was ≥2 mm (27). The clinical recording was performed by one of the authors (CB).

Crevicular fluid samples. Gingival crevicular fluid (GCF) was collected at two sites 16 and 41 from each subject using a paper strip (Periopaper, Proflow, Inc Amityville, NY, USA). The strip was inserted into the gingival crevice and left for 30 seconds. The strip was then analyzed using Periotron 8000 sensor and the volume was calculated by interpolation from a standard curve and expressed as μL GCF. The periopaper was placed in 120 μL of assay buffer containing 0.9% Na Cl, 0.01 M EDTA, 0.3% bovine-globulin, 0.005 % Triton-X-100, 0.05% sodium azide, 0.0255 M NaH_2PO_4, 0.0245 M Na_2HPO_4, pH 6.8 and kept frozen at −70°C (23).

The GCF samples were analyzed with respect to the levels of adiponectin, IL-1β, IL-6, IL-8 and PAI-1 by using commercially available kits (Linco Research, Inc., Missouri, USA) in accordance with the manufacturer's instruction. TNF-α levels were measured using the Bio-Plex cytokine assay (Bio-Rad laboratories CA, USA).

Statistics

Data analysis was carried out using the statistical software package SPSS, version 17.0. For analyzing the data, frequency tables, cross tables, ANOVA, Chi-square and logistic regression were used. In the logistic regression analysis the Wald test was also performed. Bivariate analyses association was carried out between the dependent variable "pathological periodontal pocket" and the independent variables by applying logistic regression binary model. In the multiple logistic regression analysis with pathological periodontal pocket as dependent variable, the independent variable BMI-SDS was adjusted for potential confounders. The odds ratio (OR) and confidence interval (95% CI) were calculated and the level of significance was accepted at P-values less than 0.05.

Statement of ethics

We certify that all applicable institutional and governmental regulations concerning the ethical use of human volunteers were followed during the research. The Ethics Committee at the Karolinska University Hospital, Huddinge approved the study and all subjects gave oral consent before participating in the study.

Results

The characteristics of the subjects with respect to age, gender, chronic diseases, weight, height, BMI-SDS, as well as the educational level and country of birth of their parents are shown in Table I. Except for the variables height, "BMI" and BMI-SDS according to Rolland-Cacherra (25), there was no significant difference between obesity and control group.

The oral hygiene habits are presented in Table II. The obese subjects demonstrated significant lower frequency of tooth brushing (P=0.006), use of dental floss (P=0.040) and less use of electric toothbrush (P=0.041) compared with the control patients. The clinical variables in terms of VPI%, BOP%, pathological periodontal pockets (>4 mm) as well as supragingival and subgingival calculus are also demonstrated in Table II. Regarding gingival inflammation, there was a significantly higher frequency of BOP (>25%) in obese subjects compared with controls (P<0.001). The occurrence of one or more pathological periodontal pocket (>4 mm) was also more frequent among the obese subjects compared with the controls (P=0.001). There was no significant difference regarding supragingival or subgingival calculus between the two groups. Incipient alveolar bone loss was diagnosed in two cases of the obese subjects and one of the controls (Table II).

The level of cytokines in GCF is demonstrated in Table III. The levels of IL-1β (P<0.001) and IL-8 (P=0.002) were significantly higher in obese subjects compared with the controls. By contrast, the levels of PAI-1, TNF-α and adiponectin in GCF did not differ significantly between the groups.

Table II. Clinical variables and oral hygiene habits of the obese subjects and age- and gender-matched controls.

Variables	Obesity (n=52)	Controls (n=52)	P-value[1]
VPI (%)			
0–25	38	48	
>25	14	4	0.010
BOP (%)			
0–25	33	48	
>25	19	4	<0.001
Pocket depth (>4 mm)			
Yes	10	0	
No	42	52	0.001
CEJ >2 mm			
Yes	2	1	
No	50	51	0.540
Subgingival calculus			
Yes	7	3	
No	45	49	0.183
Supragingival calculus			
Yes	24	25	
No	28	27	0.844
Tooth brushing			
In the morning			
Daily	27	42	
Not daily	25	10	0.006
In the evening			
Daily	29	44	
Not daily	23	8	0.002
Electric toothbrush			
Daily	3	37	
Not daily	49	15	0.040
Flossing			
Daily	3	0	
Not daily	49	52	0.040
Self-perceivedgingival bleeding			
Daily	7	0	
Not daily	45	52	0.002

[1]Chi-Square test as statistical method.

In a bivariate logistic regression analysis with the occurrence of one or more pathological periodontal pockets (>4 mm) as dependent variable, the following variables were significantly associated; BMI-SDS (P=0.01), BOP (>25%) (P=0.001) and subgingival calculus (P=0.003) (Table IV).

In a multivariate logistic regression model we tested the association between BMI-SDS and pathological periodontal pocket (>4 mm), as dependent variable, when adjusting for the significant variables from the bivariate analysis. As demonstrated in Table V the variable BMI-SDS was significantly (P=0.030) associated with the occurrence of pathological periodontal pockets (>4 mm) even after adjusting for the variables BOP (>25%), and subgingival calculus. The OR of adjusted BMI-SDS was 1.87. As demonstrated in Table V, BOP (>25%) did not become significantly associated with pathological periodontal pockets when the variable BOP (>25%) was adjusted for both BMI-SDS and subgingival calculus in the model. By contrast, the variable "subgingival calculus" was significantly associated with pathological periodontal pockets in the model even after adjusting for BMI-SDS and BOP (>25%) (Table V).

Table III. The level of cytokines and adiponectin in gingival crevicular fluid of obese subjects (n=52) and controls (n=52).

Variables	Obesity Mean (SD)	Controls Mean (SD)	P-value[1]
GCF (µl)	0.4 (0.2)	0.4 (0.2)	0.500
Adiponectin (pg/ml)	2 540 (2815)	1 481 (2972)	0.072
PAI-1 (pg/ml)	102 (68)	101 (113)	0.966
IL-1B (pg/ml)	23.8 (34.0)	5.3 (9.3)	<0.001
IL-8 (pg/ml)	173 (91)	77 (44)	0.002
TNFα (pg/ml)	1.0 (0.2)	0.8 (0.3)	0.060

[1]ANOVA as statistical method.
SD: Standard deviation.

Discussion

We demonstrate the novel finding that an association exists between obesity and pathological periodontal pockets in adolescents. In addition, the level of the

Table IV. Bivariate logistic regression analysis with the occurrence of pathological periodontal pocket (>4 mm) as dependent variable.

Variable	Wald	df	P	OR	95% CI
BMI-SDS	7.32	1	0.010	1.63	1.14–2.32
BOP (>25%)	10.7	1	0.001	11.4	2.65–48.8
VPI (>25%)	3.57	1	0.060	3.81	0.95–15.2
CEJ (>2 mm)	1.60	1	0.210	5.00	0.41–60.7
Calculus					
- supragingival	2.17	1	0.140	2.89	0.70–11.9
- subgingival	8.74	1	0.003	9.78	2.16–44.3
Tooth brushing					
- evening	0.06	1	0.800	0.87	0.30–2.53
- morning	0.27	1	0.600	0.77	0.29–2.07
Dental floss	0.08	1	0.790	0.81	0.91–3.45
Self perceivedgingival bleeding	0.01	1	0.920	0.95	0.31–2.92
Country of birth					
- mother	0.14	1	0.710	1.29	0.34–4.87
- father	0.53	1	0.470	1.64	0.43–6.19
Educational level					
- mother					
≤9 years	0.60	2	0.740		
10–12 years	0.27	1	0.600	0.67	0.14–3.09
>12 years	0.58	1	0.450	0.48	0.07–3.17
- father					
≤9 years	1.35	2	0.510		
10–12 years	0.37	1	0.540	1.68	0.31–9.03
>12 years	0.30	1	0.580	0.50	0.04–5.91
GCF (µl)	0.97	1	0.330	0.14	0.00–6.92
Adiponectin (pg/ml)	0.22	1	0.640	1.00	1.00–1.00
PAI (pg/ml)	0.16	1	0.690	1.00	1.00–1.01
IL-β (pg/ml)	0.89	1	0.350	1.01	0.99–1.03
IL-8 (pg/ml)	0.89	1	0.340	1.00	1.00–1.01
TNF-α (pg/ml)	0.30	1	0.590	2.03	0.16–25.9

OR=Odds ratio.
CI=Confidence interval.

Table V. Multiple logistic regression analysis with the occurrence of pathological periodontal pocket (>4 mm) as dependent variable.

Variable	Wald	df	P	OR	95% CI
BMI-SDS					
Not adjusted	7.32	1	0.010	1.63	1.14–2.32
Adjusted for BOP (>25%)	4.47	1	0.040	1.53	1.03–2.28
Adjusted for subgingival Calculus	7.02	1	0.010	2.02	1.20–3.40
Adjusted for BOP (>25%) and subgingival calculus	4.96	1	0.030	1.87	1.08–3.26
BOP (>25%)					
Not adjusted	10.7	1	0.001	11.4	2.65–48.8
Adjusted for BMI-SDS	5.62	1	0.020	6.26	1.37–28.6
Adjusted for subgingival calculus	8.13	1	0.004	9.07	1.21–35.4
Adjusted for BMI and subgingival calculus	2.85	1	0.090	4.01	0.80–20.6
Subgingival calculus					
Not adjusted	8.74	1	0.003	9.78	2.16–44.3
Adjusted for BMI-SDS	9.08	1	0.003	20.5	2.88–146.4
Adjusted for BOP (>25%)	4.77	1	0.029	6.55	1.99–41.3
Adjusted for BMI and BOP (>25%)	6.30	1	0.012	13.6	1.77–104.1

OR=Odds ratio.
CI=Confidence interval.

periodontal risk indicators IL-1β and IL-8 in GCF were significantly higher in the obese subjects compared with controls.

The end point of periodontal disease includes alveolar bone loss, so called periodontitis, and subsequently tooth loss. In two of the obese subjects incipient alveolar bone loss was diagnosed compared with one among the controls. One has to consider that our subjects are young and the effect of chronic inflammation on periodontal tissue needs longer exposure time until alveolar bone loss is possible to detect. In the statistical analysis we therefore used pathological periodontal pocket as outcome, a surrogate variable related to inflammation of periodontal tissue, which can be used as a periodontal risk indicator.

The BMI-SDS is used in the statistical analysis instead of the regular BMI to compensate for the age-dependent variation of BMI. In addition we also adjusted for a number of other potential confounders including gender, age, gingival inflammation, subgingival calculus and oral hygiene variables related to dental health behavior when analyzing the association between obesity and pathological periodontal pockets. None of the adolescents admitted to being regular smokers, eliminating possible modifying factors of host response in the periodontal tissue, although we cannot completely rule out the possibility that some subjects in the study have smoked or used tobacco in some form.

Visible plaque on tooth surface was not significantly associated with occurrence of pathological periodontal pocket, although the variable almost reached significance (P=0.060), indicating other potential contributing factors for the development of periodontal pockets. Interestingly, in the multiple logistic regression analysis, obesity was significantly associated with the occurrence of pathological periodontal pocket even after adjustments for gingival inflammation and subgingival calculus, indicating a direct or indirect link between obesity and periodontal pockets. This assumption is in line with the observation that the periodontal risk indicator BOP (>25%) did not become significantly associated with pathological periodontal pockets when adjusting for both BMI and subgingival calculus, indicating that the strength of BOP % as a predictor for pathological periodontal pockets is relatively weaker than the combination of BMI and subgingival calculus. Altogether the multivariate analysis demonstrates that obese adolescents exhibit an enhanced relative risk for exhibiting pathological periodontal pockets, this is not caused by worse oral hygiene. The enhanced level of cytokines in GCF reported in obese subjects may to some extent be produced by adipose tissue and we hypothesize that obesity either directly or indirectly contributes to an enhanced pro-inflammatory milieu in the periodontal tissue that trigger the inflammatory response to periodontal pathogens residing in the biofilm. However, according to the bivariate logistic regression analysis, there was no significant difference in cytokine concentration in GCF between subjects with or without pathological periodontal pockets. Obesity has also been reported to interfere with the ability of the immune system to appropriately respond to the periodontal pathogen *Phorphyromonas gingivalis* infection in terms of TNF-α, IL-6 and serum amyloid response (17), which might be of importance to explain the link between obesity and periodontal pockets. However, there are also other possible mediators, such as stress due to health related issues among the obese subjects, which has to be taken into consideration (28).

The enhanced level of the pro-inflammatory cytokines IL-1β and IL-8 in GCF among the obese

adolescents are in line with the findings in adults demonstrating an association between obesity and IL-1β, as well as PGE_2 levels in GCF (29). Previously, we reported that the level of TNF-α in GCF was correlated with BMI in the most severe obese subjects (23), and recent data indicates a link between adiposity in children and TNF-α level in GCF that might be mediated by insulin resistance (30). In the present study we did not demonstrate higher GCF level of TNF-α in obese subjects compared with controls, which is probably due to the fact that the subjects were younger and BMI in the present obese subjects was lower, indicating a shorter duration and/or less severe obesity. However, we did not register the duration of obesity of the subjects, which probably in addition to the severity of obesity, is of importance when looking upon the association between obesity and periodontal pockets during adolescence.

Our finding that obesity may have a negative impact on periodontal health and that the link between obesity and periodontal risk indicators can be seen during early adolescence is a novel finding. According to Reeves and co-workers (12), a strong risk for the enhancement of periodontitis by obesity was first seen among young adults, aged 17 to 21 years old. However, one has to take into account that Reeves and co-authors used loss of tissue attachment and a probing depth of 3 mm or more as definition whereas we have used the occurrence of pathological periodontal pocket (>4 mm) as outcome, which reflects an earlier stage of the disease.

In the multivariate analysis, we also demonstrate a significant association between subgingival calculus and pathological periodontal pockets that is well compatible with our previous finding that subgingival calculus is a significant predictor for alveolar bone loss (27). When adjusting for BMI-SDS, the OR, of subgingival calculus increased approximately two fold indicating a strong predictor for pathological periodontal pockets as well.

A decreased level of adiponectin in GCF from the obese subjects is anticipated because the serum level of adiponectin is diminished by obesity (31–33). However, we did not find any significant difference between the groups regarding adiponectin level in GCF. The role of adiponectin in the periodontal tissue; however, is very unclear although a protective role due to its anti-inflammatory properties is just one possibility. This assumption is based on the observation that adiponectin may function as a negative regulator of lipopolysacharide/RANKL-mediated osteoclast formation (34) and thereby suppress bone resorption activity of osteoclasts (35). On the other hand adiponectin may have a negative effect on periodontal health in the view of stimulating IL-6 (36), which induces bone resorption (37).

In conclusion, the study demonstrates an association between obesity and periodontal risk indicators that in the long term may lead to oral morbidity. This result further strengthens obesity's negative effect on teenagers' periodontal health and highlights the importance of a close collaboration betweens dentists and pediatricians in the prevention and treatment of obesity.

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