

Relation of the body mass index to salivary cortisol and periodontal indices in children: A comparative analysis

Aida Mehdipour^{1,A–F}, Amirhossein Khorrami^{2,B,D–F}, Mohammad Aghaali^{3,C–F}, Ali Saleh^{4,D–F}

¹ Cellular and Molecular Research Center, Qom University of Medical Sciences, Iran

² Department of Pediatric Dentistry, Qom University of Medical Sciences, Iran

³ Department of Community Medicine, School of Medicine, Qom University of Medical Sciences, Iran

⁴ Student Research Committee, Qom University of Medical Sciences, Iran

A – research concept and design; B – collection and/or assembly of data; C – data analysis and interpretation;

D – writing the article; E – critical revision of the article; F – final approval of the article

Dental and Medical Problems, ISSN 1644-387X (print), ISSN 2300-9020 (online)

Dent Med Probl. 2026;63(4):857–868

Address for correspondence

Aida Mehdipour

E-mail: mehdipour_aida@yahoo.com

Funding sources

None declared

Conflict of interest

None declared

Acknowledgements

None declared

Received on February 22, 2023

Reviewed on April 12, 2023

Accepted on May 1, 2023

Published online on August 11, 2026

Abstract

Background. Obesity is a chronic disease resulting from the interaction of genetic, environmental, socio-economic, and behavioral factors, and is associated with the development of numerous comorbidities. Its prevalence is increasing worldwide, making obesity one of the most significant public health challenges.

Objectives. The aim of the present study was to investigate the relationship between obesity and the cortisol levels and periodontal status in children.

Material and methods. Ninety eligible children aged 8–12 years were enrolled in the study and allocated into 3 groups: normal-weight; overweight; and obesity. After the parents completed a demographic questionnaire, the children underwent periodontal examinations and saliva collection. The salivary cortisol levels were measured using the ELISA method. Data were analyzed using ANOVA, Pearson's correlation test, the χ^2 test, linear regression, and the Kruskal–Wallis test.

Results. The children's body mass index (BMI) was significantly associated with the salivary cortisol levels and the gingival index (GI), bleeding on probing (BoP) and periodontal probing depth (PPD) ($p = 0.010$, $p < 0.001$, $p < 0.001$, and $p = 0.005$, respectively). However, BMI was not significantly associated with father's education, mother's education, the parental smoking addiction status, total family income, the frequency of mouthwash use per week, toothbrushing frequency per day, or daily study duration ($p = 0.599$, $p = 0.143$, $p = 0.480$, $p = 0.875$, $p = 0.884$, $p = 0.203$, and $p = 0.338$, respectively). The salivary cortisol levels were also significantly associated with age and GI, BOP and PPD ($p = 0.010$, $p = 0.001$, $p = 0.018$, and $p = 0.016$, respectively). In the multivariable linear regression analysis, the salivary cortisol levels remained significantly associated with age ($\beta = 0.276$; $p = 0.004$), obesity ($\beta = 0.269$; $p = 0.006$), father's education level ($\beta = 0.393$; $p < 0.001$), and the parental smoking addiction status ($\beta = 0.238$; $p = 0.026$).

Conclusions. The salivary cortisol levels and periodontal indices were higher in overweight and obese children. Therefore, encouraging children and adolescents to adopt a healthier lifestyle and achieve weight reduction may help reduce the salivary cortisol levels and improve periodontal health.

Keywords: body mass index, periodontal pocket, cortisol, gingival index, periodontal indices

Cite as

Mehdipour A, Khorrami A, Aghaali M, Saleh A. Relation of the body mass index to salivary cortisol and periodontal indices in children: A comparative analysis. *Dent Med Probl.* 2026;63(4):857–868. doi:10.17219/dmp/165842

DOI

10.17219/dmp/165842

Copyright

Copyright by Author(s)

This is an article distributed under the terms of the Creative Commons Attribution 3.0 Unported (CC BY 3.0) (<https://creativecommons.org/licenses/by/3.0/>)

Highlights

- Childhood obesity was associated with higher salivary cortisol levels and a poorer periodontal status.
- Higher salivary cortisol levels were significantly associated with increased periodontal inflammation.
- Weight management may contribute to improved periodontal health and help reduce physiological stress in children.

Introduction

Obesity refers to the abnormal or excessive accumulation of adipose tissue in the body.¹ It is considered not only an esthetic concern, but also a chronic disease resulting from the complex interaction of genetic, environmental, socioeconomic, and behavioral factors.² It has been suggested that the genes associated with sweet and bitter taste perception may influence both obesity and oral health.³ In recent years, obesity has replaced smoking as one of the most important lifestyle-related risk factors for premature death because of its association with numerous health problems and chronic diseases.^{1,4}

According to the World Health Organization (WHO), more than 340 million children and adolescents aged 5–19 years were overweight or obese in 2016.⁴ Akbari and Mohammadi reported an overall obesity prevalence of 11.4% among Iranian children.⁵ The diagnosis of obesity in children is usually based on the calculation of the body mass index (BMI). Although BMI has limitations as a direct measure of body fat, it remains the most widely used and practical method for diagnosing obesity in children.⁶

Cortisol is commonly known as the body's stress hormone.⁷ Studies have demonstrated alterations in cortisol secretion, function and metabolism in individuals with obesity.⁸ Subclinical hypercortisolism increases the risk of osteoporotic fractures, hyperglycemia, hypertension, dyslipidemia, and obesity.^{9,10} In addition, hypercortisolism may promote insulin resistance, thereby perpetuating a vicious cycle.¹¹

However, the available evidence is conflicting. Several studies have reported that the mean salivary and serum cortisol levels are lower during the daytime in children with obesity.^{12–14} In contrast, other studies have demonstrated a positive association between the cortisol levels and BMI in children.^{15–17} Similarly, conflicting findings have been reported regarding the relationship between the serum cortisol levels and BMI in adults.^{18–20}

Severe periodontal disease is the 11th most prevalent disease worldwide.²¹ It often begins as gingivitis, which is characterized by gingival bleeding, swelling and pain. If left untreated, gingivitis may progress to periodontitis, resulting in the loss of periodontal attachment, alveolar bone, and ultimately teeth, thereby impairing mastication, esthetics, self-confidence, and quality of life.²²

Concern about periodontal disease lies not only in its high prevalence, but also in its well-established association

with several systemic conditions, including cardiovascular disease, diabetes mellitus, and adverse pregnancy outcomes, such as low birth weight.^{23–25} Perinatal exposures and pregnancy outcomes have also been associated with an increased predisposition to obesity.²⁶

The association between obesity and inflammatory processes has long been recognized.²⁷ Obesity may be considered a risk factor for periodontal disease, as it alters immune function and promotes a chronic low-grade inflammatory state associated with excess adipose tissue.²⁸ Although numerous studies have investigated periodontal health in adults with obesity,^{29,30} only limited studies have been conducted in children and adolescents, and these suggest a possible association between obesity and periodontal health.^{2,28,31} Given the increasing prevalence of overweight and obesity among children, understanding the early periodontal changes associated with obesity is of considerable importance.^{32,33}

Considering the insufficient number of studies and the conflicting findings regarding the relationship between obesity and periodontal health in children and adolescents, further research in this field is warranted. Therefore, the aim of the present study was to investigate the relationship between overweight and obesity, the salivary cortisol levels, and the periodontal status in children.

Material and methods

Study design and participants

This cross-sectional comparative descriptive study was conducted from 2021 to 2022 in 3 downtown schools in Qom, Iran. The study protocol was developed in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement.

According to a study by Putignano et al., considering the average salivary cortisol levels in 2 groups of individuals with obesity and normal weight, with an alpha level of 5% and a beta level of 10%, the required sample size was calculated as 30 participants per group.³⁴ Therefore, a total of 90 participants were included in this study, using the non-random convenience sampling method.

The inclusion criteria were children aged 8–12 years with total dmft and DMFT (decayed, missing and filled teeth in primary and permanent dentition, respectively) scores <3.

The exclusion criteria comprised a history of any systemic disease (such as epilepsy, diabetes, hypothyroidism, cancer, psychological disorders, precocious puberty, or short stature), severe periodontitis, the use of any medication, a history of surgery, trauma, or other stressful events within the previous 3 months, participation in professional sports activities, living with only one parent (due to parental divorce or death), saliva sampling during the menstrual period in female participants, and resistance or the lack of cooperation during saliva collection and clinical examinations.

After obtaining informed consent from the children's parents, eligible children were enrolled in the study and equally divided into 3 groups: 30 children with normal weight; 30 children with overweight; and 30 children with obesity. The groups were matched based on age and sex. The participants were also divided into 2 groups according to sex, including 45 males and 45 females. Each sex group consisted of 15 children with obesity, 15 children with overweight and 15 children with normal weight.

Measurements and the questionnaire

The children's weight was measured while wearing light clothing (such as a T-shirt and pants) and without shoes or belts, using a digital scale (SBS 4414; Sinbo Precision Industry, Shenzhen, China) with an accuracy of 100 g. Height was measured using a wall-mounted stadiometer with an accuracy of 0.5 cm. The body mass index was then calculated by dividing weight in kilograms by the square of height in meters.

Based on the BMI values and the standard BMI charts developed by WHO,^{6,35} the 90 eligible children were included in the study and equally divided into 3 groups – normal weight, overweight, and obesity – with 30 participants in each group. According to the WHO standards for children and adolescents aged 2–20 years, a BMI percentile of 85–97% was considered overweight, while a BMI above the 97th percentile was considered obesity.^{6,35}

The children and their parents were instructed on proper toothbrushing and flossing techniques 1 month before data collection. The parents were also encouraged to carefully supervise their children's oral hygiene practices during this period.

Then, the designed questionnaire was completed by the children's parents, and the children's demographic characteristics, parental smoking status, daily activity status, medical and dental history, frequency of mouthwash use per week, toothbrushing frequency per day, and daily study duration in hours were recorded.

Collection of saliva samples

Before saliva collection, the participants were instructed to refrain from toothbrushing, eating, drinking, and vigorous physical activities for 1 h. During this period, they were allowed to relax through activities

such as reading, writing and drawing.¹⁵ Due to circadian variations in cortisol concentrations, saliva samples were collected at noon (12:00). Saliva samples were obtained using the spitting method.³⁶ After rinsing the mouth with water and swallowing saliva once, each participant was instructed to allow saliva to accumulate in the floor of the mouth, and then transfer it into a Falcon tube 1–2 times per minute for 5 min while sitting with the head and neck positioned at a 45-degree angle.³⁷

Oral examinations

All oral examinations were performed by a trained dentist. To avoid bias related to the incomplete eruption of some permanent teeth, periodontal indices were assessed only on first permanent molars and maxillary and mandibular central and lateral incisors. Primary teeth were not included in the periodontal evaluation.²

Gingival index

The gingival index (GI) was scored from 0 to 3 according to the Löe and Silness criteria as follows³⁸:

- score 0: no bleeding on probing (BoP); normal gingiva without signs of inflammation;
- score 1: presence of non-bleeding gingival changes; mild inflammation of the marginal gingiva detected during a clinical examination (the marginal gingiva is slightly redder than normal, with slight edema, and colorless gingival fluid may be observed at the entrance of the sulcus);
- score 2: bleeding of the marginal gingiva after stimulation with a dental probe or air spray; moderate gingival inflammation (the gingiva appears red or bluish-red and shiny, with the edema of the marginal gingiva);
- score 3: spontaneous gingival bleeding; severe gingival inflammation (the gingiva is markedly red or bluish-red, enlarged, and swollen).

Bleeding on probing

To evaluate BoP, a Williams probe (Smart Instrument, model MS5201; Hu-Friedy Manufacturing, Chicago, USA) was gently inserted into the sulcus of all teeth up to the base of the pocket or sulcus without applying additional pressure. After 10–15 s, the number of teeth exhibiting gingival bleeding was recorded.³⁹

Periodontal probing depth

To assess periodontal probing depth (PPD), a Williams probe (Smart Instrument, model MS5201) was inserted into the sulcus or periodontal pocket with gentle force while maintaining contact with the tooth surface and keeping the probe parallel to the root surface (the long axis of the tooth). The probe was advanced until the tip

reached the base of the pocket and encountered increased resistance, indicating the limit of further penetration. The distance between the base of the pocket (the point where the probe stopped) and the gingival margin was measured and recorded.⁴⁰

Measurements were performed at 6 sites for each tooth (mesiobuccal, mid-buccal, distobuccal, distolingual, midlingual, and mesiolingual). A pocket depth greater than 3 mm was considered indicative of an inflammatory condition.² The mean PPD values for each tooth were then calculated and recorded.⁴¹

Salivary cortisol levels

The cortisol levels can be measured using 3 types of samples: blood; saliva; and urine. The salivary cortisol levels have a direct and strong correlation with the serum cortisol levels and follow the circadian pattern of serum cortisol secretion. Due to the simplicity and non-invasive nature of saliva collection, as well as the ease of saliva transport and storage, the salivary cortisol measurement is widely used in pediatric research. In addition, blood sampling can be stressful for children and may result in elevated serum cortisol levels.^{42,43}

The salivary cortisol levels were measured using a salivary cortisol kit (DKO020; DiaMetra, Spello, Italy) based on the enzyme-linked immunosorbent assay (ELISA) method.⁴⁴

Bias

The risk of bias was minimized by excluding children with total dmft and DMFT scores >3 from the study, providing oral hygiene instructions to the participants and their parents, and assessing the periodontal status using reliable methods (GI, BoP and PPD). The salivary cortisol levels were analyzed in a specialized laboratory in Qom, Iran.

Statistical analysis

Data were analyzed using IBM SPSS Statistics for Windows, v. 26 (IBM Corp., Armonk, USA). Descriptive and analytical statistics were applied. The Kolmogorov–Smirnov test and the Q–Q plot were used to assess the normality of data distribution. Differences between the groups were analyzed using the one-way analysis of variance (ANOVA) and the Kruskal–Wallis test according to the distribution of data (normal and not normal, respectively). The χ^2 test was used for categorical variables, and Pearson's correlation test was used to assess correlations between variables. The multivariable linear regression analysis was performed to assess the associations between the salivary cortisol levels (the quantitative dependent variable) and the independent variables while controlling for potential confounding factors.

Results

Characteristics of the participants

In this study, 90 eligible children, including 45 males and 45 females, were enrolled. The mean age was 10.00 ± 1.48 years in the obese and normal-weight groups, and 10.16 ± 1.39 years in the overweight group. There were no significant differences among the 3 groups regarding age and sex ($p = 1.000$).

Correlation between BMI and the demographic status

The BMI status was not significantly associated with the paternal or maternal education level (χ^2 test; $p = 0.599$ and $p = 0.143$, respectively). The relationship between the children's BMI status and total family income was also non-significant (χ^2 test; $p = 0.875$).

In addition, 50% of parents of normal-weight children reported smoking addiction, compared with 36.7% of parents of children from the overweight and obesity groups. Therefore, parental smoking was more prevalent among the parents of normal-weight children than among those of overweight and obese children. However, no significant association was found between the children's BMI status and the parental smoking addiction status (χ^2 test; $p = 0.480$).

The BMI status was not significantly associated with the frequency of mouthwash use per week, toothbrushing frequency per day or daily study duration in hours (Kruskal–Wallis test; $p = 0.884$, $p = 0.203$ and $p = 0.338$, respectively) (Table 1).

Correlation between BMI and the salivary cortisol levels and periodontal indices

The mean salivary cortisol levels in the normal-weight, overweight and obesity groups were 2.42 ± 0.74 , 2.93 ± 1.32 and 3.68 ± 2.26 ng/mL, respectively. Thus, the mean salivary cortisol levels were highest in children with obesity, followed by overweight children and normal-weight children. There was a significant association between the BMI status and the salivary cortisol levels (one-way ANOVA; $p = 0.010$) (Table 2).

The mean GI values in the normal-weight, overweight and obesity groups were 0.42 ± 0.13 , 0.51 ± 0.16 and 0.61 ± 0.15 , respectively. Thus, the mean GI was highest in children with obesity, followed by overweight children and normal-weight children.

The mean BoP values in the normal-weight, overweight and obesity groups were $17.83 \pm 7.20\%$, $21.50 \pm 7.61\%$ and $26.00 \pm 6.82\%$, respectively. Accordingly, the mean BoP was highest in children with obesity, followed by overweight children and normal-weight children.

The mean PPD values in the normal-weight, overweight and obesity groups were 1.52 ± 0.22 , 1.60 ± 0.24 and 1.72 ± 0.23 mm, respectively. Similarly, the mean PPD was

highest in children with obesity, followed by overweight children and normal-weight children.

There was a significant association between the children's BMI status and GI, BoP and PPD (one-way ANOVA; $p < 0.001$, $p = 0.001$ and $p < 0.005$, respectively) (Table 2).

The pairwise comparisons of the salivary cortisol levels among the study groups showed a significant difference only between the obesity and normal-weight groups (post hoc test; $p = 0.003$). No significant differences were observed between the obesity and overweight groups, or between the overweight and normal-weight groups (post hoc test; $p = 0.069$ and $p = 0.209$, respectively) (Table 3).

The pairwise comparisons of GI showed significant differences between the obesity and overweight groups, the obesity and normal-weight groups, and the overweight and normal-weight groups (post hoc test; $p = 0.011$, $p < 0.001$ and $p = 0.017$, respectively) (Table 3).

For BoP, significant differences were observed between the obesity group and both the overweight and normal-weight groups (post hoc test; $p = 0.018$ and $p < 0.001$, respectively). However, no significant difference was found

between the overweight and normal-weight groups (post hoc test; $p = 0.052$) (Table 3).

For PPD, a significant difference was observed only between the obesity and normal-weight groups (post hoc test; $p = 0.001$). No significant differences were found between the obesity and overweight groups, or between the overweight and normal-weight groups (post hoc test; $p = 0.054$ and $p = 0.166$, respectively) (Table 3).

According to Pearson's correlation analysis, there were significant positive correlations between GI and BoP ($r = 0.948$; $p < 0.001$), and between GI and PPD ($r = 0.680$; $p < 0.001$). A significant positive correlation was also observed between BoP and PPD ($r = 0.674$; $p < 0.001$). In addition, the salivary cortisol levels were positively correlated with GI ($r = 0.340$; $p = 0.001$), BoP ($r = 0.250$; $p = 0.018$) and PPD ($r = 0.253$; $p = 0.016$) (Table 4).

Based on the results of the univariable linear regression analysis, the salivary cortisol levels were significantly and positively associated with BMI ($\beta = 0.116$; $p = 0.001$), obesity ($\beta = 1.221$; $p = 0.005$), GI ($\beta = 3.325$; $p = 0.001$), and age ($\beta = 0.311$; $p = 0.010$). However, no significant association

Table 1. Relationship between the body mass index (BMI) status and the frequency of mouthwash use per week, toothbrushing frequency per day and daily study duration (Kruskal–Wallis test)

Variable	N	M $\pm$ SD	min	max	Percentile			p-value
					25 th	50 th (Me)	75 th	
Frequency of mouthwash use per week	90	0.1556 $\pm$ 0.4719	0.00	3.00	0.0000	0.0000	0.0000	0.884
Toothbrushing frequency per day	90	0.9333 $\pm$ 0.6670	0.00	3.00	1.0000	1.0000	1.0000	0.203
Daily study duration [h]	90	1.22 $\pm$ 1.24	0	6	0.00	1.00	2.00	0.338
BMI status [kg/m ²]	90	21.34 $\pm$ 4.91	13.15	33.07	17.20	21.54	24.69	–

M – mean; SD – standard deviation; min – minimum; max – maximum; Me – median.

Table 2. Relationship between the body mass index (BMI) status and the salivary cortisol levels and periodontal indices (one-way ANOVA)

Variable	BMI status	N	M $\pm$ SD	SE	95% CI		min	max	p-value
					lower bound	upper bound			
Salivary cortisol level [ng/mL]	normal weight	30	2.4167 $\pm$ 0.7438	0.1358	2.1389	2.6944	0.70	3.60	0.010*
	overweight	30	2.9300 $\pm$ 1.3231	0.2416	2.4360	3.4240	0.90	5.80	
	obesity	30	3.6767 $\pm$ 2.2583	0.4123	2.8334	4.5199	0.40	9.50	
	total	90	3.0078 $\pm$ 1.6380	0.1727	2.6647	3.3508	0.40	9.50	
GI	normal weight	30	0.4193 $\pm$ 0.1309	0.0239	0.3705	0.4682	0.22	0.82	<0.001*
	overweight	30	0.5130 $\pm$ 0.1606	0.0293	0.4530	0.5730	0.25	0.95	
	obesity	30	0.6133 $\pm$ 0.1533	0.0280	0.5561	0.6706	0.25	0.84	
	total	90	0.5152 $\pm$ 0.1673	0.0176	0.4802	0.5503	0.22	0.95	
BoP [%]	normal weight	30	17.8333 $\pm$ 7.1959	1.3138	15.1463	20.5203	8.00	40.00	<0.001*
	overweight	30	21.5000 $\pm$ 7.6090	1.3892	18.6588	24.3412	8.00	45.00	
	obesity	30	25.9833 $\pm$ 6.8172	1.2446	23.4378	28.5289	12.50	41.00	
	total	90	21.7722 $\pm$ 7.8812	0.8308	20.1215	23.4229	8.00	45.00	
PPD [mm]	normal weight	30	1.5203 $\pm$ 0.2163	0.0395	1.4396	1.6011	1.20	1.98	0.005*
	overweight	30	1.6020 $\pm$ 0.2369	0.0433	1.5135	1.6905	1.18	2.15	
	obesity	30	1.7163 $\pm$ 0.2257	0.0412	1.6320	1.8006	1.33	2.21	
	total	90	1.6129 $\pm$ 0.2381	0.0251	1.5630	1.6627	1.18	2.21	

SE – standard error; CI – confidence interval; GI – gingival index; BoP – bleeding on probing; PPD – periodontal probing depth; * statistically significant ($p < 0.05$).

Table 3. Pairwise comparisons of the salivary cortisol levels and periodontal indices in the study groups (post hoc statistical analysis – the least significant difference (LSD) test)

Dependent variable	Obesity status (I)	Obesity status (J)	Mean difference (I–J)	SE	95% CI		p-value
					lower bound	upper bound	
Salivary cortisol level [ng/mL]	normal weight	overweight	–0.5133	0.4056	–1.3195	0.2929	0.209
		obesity	–1.2600	0.4056	–2.0662	–0.4538	0.003*
	overweight	normal weight	0.5133	0.4056	–0.2929	1.3195	0.209
		obesity	–0.7467	0.4056	–1.5529	0.0595	0.069
	obesity	normal weight	1.2600	0.4056	0.4538	2.0662	0.003*
		overweight	0.7467	0.4056	–0.0595	1.5529	0.069
GI	normal weight	overweight	–0.0937	0.0384	–0.1700	–0.0173	0.017*
		obesity	–0.1940	0.0384	–0.2704	–0.1176	<0.001*
	overweight	normal weight	0.0937	0.0384	0.0173	0.1700	0.017*
		obesity	–0.1003	0.0384	–0.1767	–0.0240	0.011*
	obesity	normal weight	0.1940	0.0384	0.1176	0.2704	<0.001*
		overweight	0.1003	0.0384	0.0240	0.1767	0.011*
BoP [%]	normal weight	overweight	–3.6667	1.8628	–7.3692	0.0359	0.052
		obesity	–8.1500	1.8628	–11.8525	–4.4475	<0.001*
	overweight	normal weight	3.6667	1.8628	–0.0359	7.3692	0.052
		obesity	–4.4833	1.8628	–8.1859	–0.7808	0.018*
	obesity	normal weight	8.1500	1.8628	4.4475	11.8525	<0.001*
		overweight	4.4833	1.8628	0.7808	8.1859	0.018*
PPD [mm]	normal weight	overweight	–0.0817	0.0585	–0.1979	0.0346	0.166
		obesity	–0.1960	0.0585	–0.3122	–0.0798	0.001*
	overweight	normal weight	0.0817	0.0585	–0.0346	0.1979	0.166
		obesity	–0.1143	0.0585	–0.2306	0.0019	0.054
	obesity	normal weight	0.1960	0.05847	0.0798	0.3122	0.001*
		overweight	0.1143	0.05847	–0.0019	0.2306	0.054

* statistically significant ($p < 0.05$).**Table 4.** Correlation between the salivary cortisol levels and periodontal indices (Pearson's correlation analysis)

Variable	Pearson's correlation analysis	GI	BoP	PPD	Cortisol test result
Cortisol test result	r	0.340	0.250	0.253	1
	p -value (two-tailed)	0.001**	0.018*	0.016*	–
	N	90	90	90	90
GI	r	1	0.948	0.680	0.340
	p -value (two-tailed)	–	<0.001**	<0.001**	0.001**
	N	90	90	90	90
BoP	r	0.948	1	0.674	0.250
	p -value (two-tailed)	<0.001**	–	<0.001**	0.018*
	N	90	90	90	90
PPD	r	0.680	0.674	1	0.253
	p -value (two-tailed)	<0.001**	<0.001**	–	0.016*
	N	90	90	90	90

 r – Pearson's correlation coefficient; * significant correlation at $p < 0.05$ (two-tailed); ** significant correlation at $p < 0.01$ (two-tailed).

was observed between the salivary cortisol levels and the overweight status ($\beta = 0.474$; $p = 0.263$) (Table 5).

Based on the results of the multivariable linear regression analysis, the salivary cortisol levels were significantly and positively associated with age ($\beta = 0.276$; $p = 0.004$), obesity ($\beta = 0.269$; $p = 0.006$), father's education level

($\beta = 0.393$; $p < 0.001$), and the parental smoking addiction status ($\beta = 0.238$; $p = 0.026$) (Table 6). These findings indicate that obesity remained independently associated with the salivary cortisol levels after adjusting for age, father's education level and the parental smoking addiction status.

Table 5. Univariable linear regression analysis

Variable	β	Standardized β	p -value	R^2
BMI	0.116	0.348	0.001*	0.121
Obesity status	normal-weight	reference		
	overweight	0.138	0.263	0.093
	obese	0.354	0.005*	
Age	0.311	0.269	0.010*	0.072
GI	3.325	0.340	0.001*	0.115

* statistically significant ($p < 0.05$).**Table 6.** Multivariable linear regression analysis for the salivary cortisol level variable

Model	Unstandardized coefficients		Standardized coefficient	t	p -value	R^2
	B	SE	β			
1	(constant)	−3.301	1.925	−	−1.715	0.090
	GI	1.485	1.102	0.149	1.347	0.182
	age	0.281	0.116	0.239	2.410	0.018
	overweight	0.298	0.396	0.087	0.752	0.454
	obesity	0.875	0.444	0.254	1.971	0.052
	father's education level	0.672	0.216	0.381	3.110	0.003
	total family income	0.029	0.386	0.009	0.074	0.941
	parental smoking addiction	0.806	0.353	0.242	2.282	0.025
2	(constant)	−3.197	1.312	−	−2.437	0.017
	GI	1.475	1.088	0.148	1.356	0.179
	age	0.280	0.115	0.238	2.427	0.017
	overweight	0.301	0.392	0.087	0.768	0.445
	obesity	0.880	0.435	0.255	2.024	0.046
	father's education level	0.664	0.185	0.376	3.590	0.001
	parental smoking addiction	0.809	0.348	0.243	2.321	0.023
3	(constant)	−3.066	1.298	−	−2.363	0.021
	GI	1.665	1.056	0.168	1.577	0.119
	age	0.273	0.115	0.232	2.377	0.020
	obesity	0.690	0.357	0.200	1.935	0.056
	father's education level	0.672	0.184	0.381	3.648	0.000
	parental smoking addiction	0.785	0.346	0.236	2.268	0.026
4	(constant)	−2.863	1.303	−	−2.197	0.031
	age	0.324	0.111	0.276	2.922	0.004*
	obesity	0.927	0.326	0.269	2.842	0.006*
	father's education level	0.693	0.185	0.393	3.738	<0.001*
	parental smoking addiction	0.793	0.349	0.238	2.271	0.026*

* statistically significant ($p < 0.05$).

Discussion

Correlation between the salivary cortisol levels and age

In this study, the salivary cortisol levels were measured to investigate physiological changes in cortisol metabolism during childhood and adolescence.

The present study found that the salivary cortisol levels increased significantly with increasing age in children. According to Kiess et al., the cortisol levels are age-dependent, such that after the age of 6 years, they are significantly correlated with maturation stages.¹⁶ Törnåge⁴⁵ and Larsson et al.⁴⁶ also reported higher salivary cortisol levels in older children. This positive association between age and increased cortisol levels has been reported in other

studies as well.⁴⁷ However, these findings are inconsistent with those of Kjölhede et al.¹² and Knutsson et al.⁴⁸ The discrepancies between the present findings and those of the aforementioned studies may be attributed to differences in the sampling location and time, the methods used to measure salivary cortisol, and the age range of the study populations.

Correlation between the salivary cortisol levels and obesity

The present study found that the salivary cortisol levels were positively and significantly associated with increasing BMI and obesity. Chu et al. also reported that obese children had higher morning salivary and urinary cortisol levels.¹⁵ Several other studies have likewise demonstrated a positive relationship between the salivary, serum and urinary cortisol levels and obesity.^{16,17,43,49} These studies suggest that cortisol concentrations increase long before a child becomes obese.¹⁵ Yu et al. also found that the salivary nocturnal cortisol levels were positively correlated with weight gain, abdominal fat distribution (the waist-to-height ratio (WHtR)), and body fat percentage in all participants,⁴³ consistent with the findings of the present study. However, the present findings are inconsistent with those reported by Kjölhede et al., who found lower morning and evening salivary cortisol levels in overweight and obese children aged 6–12 years,¹² as well as with the studies by Hillman et al.¹³ and Chalew et al.,¹⁴ which reported lower plasma cortisol levels in obese children. Hillman et al. further showed a negative association between the daytime cortisol levels and BMI z-score and central obesity, but a positive association during the night.¹³ The discrepancies between the present findings and those of these studies may be attributed to differences in the type of cortisol measured (e.g., plasma rather than salivary cortisol), the cortisol measurement methods and the timing of saliva sample collection.

Cortisol concentrations are not elevated in obese adults with peripheral fat distribution, whereas individuals with central obesity exhibit increased cortisol levels.⁵⁰ Unfortunately, due to the limitations of the present study, fat distribution was not assessed using waist circumference or other anthropometric measures. Therefore, based on the present findings, no definitive conclusions can be drawn regarding fat distribution and its relationship with the cortisol levels in children. Further studies are needed to investigate this association.

Correlation between obesity and the periodontal status

According to several studies, a high BMI may be a risk factor for periodontal disease in adolescents and young adults, as assessed by the plaque index (PI), BoP, PPD, clinical attachment loss (CAL), and alveolar bone loss.⁵¹

Several hypotheses have been proposed to explain biological interactions between obesity and periodontal disease, including alterations in inflammatory and immune responses, impaired glucose tolerance, dyslipidemia, changes in host immunity, increased macrophage activation, impaired microvascular function, physiological responses, psychosocial stress, and the secretion of pro-inflammatory cytokines (tumor necrosis factor- α (TNF- α), interleukin-1 (IL-1), IL-6, and IL-8), adipokines (leptin, adiponectin, resistin, and plasminogen activator inhibitor-1 (PAI-1)), and other bioactive substances, such as reactive oxygen species (ROS) from adipose tissue and C-reactive protein (CRP).^{52,53} However, the precise molecular and cellular mechanisms underlying these interactions remain unclear, and further studies are needed to elucidate these mechanisms, which may contribute to the development of preventive and therapeutic strategies.

According to some reports, periodontal disease in obese individuals is associated with alterations in the composition of the oral microbiota.^{54,55} However, most studies investigating the relationship between obesity and periodontitis have primarily considered immunological and metabolic alterations as the main predisposing factors for periodontal disease.

Several mechanisms have been proposed to explain this relationship. Adipocytes may contribute to a systemic pro-inflammatory state through the release of various inflammatory mediators.^{56,57} Some studies have shown that obesity is associated with adverse postoperative outcomes, including infectious complications and impaired wound healing.^{58,59} Therefore, the altered immune responses and impaired wound healing associated with obesity may influence the clinical response to periodontal treatment. However, Suvan et al.³⁰ and Chaffee and Weston⁶⁰ report that the evidence regarding the association between BMI, overweight or obesity and the clinical response to periodontal treatment remains limited.

In the present study, the GI, BoP and PPD indices were used to assess the periodontal status. The findings showed that obesity was positively and significantly associated with all 3 indices.

Although the association between obesity and periodontal disease has been widely investigated in adults, it has been less extensively studied in the pediatric population.

De Castilhos et al. reported a statistically significant association in young adults between obesity and GI and calculus, both of which are indicators associated with periodontal disease; however, no significant association was observed between obesity and periodontitis.⁶¹ On the other hand, Suvan et al. confirmed the association between increasing BMI and the severity and extent of periodontitis,⁶² consistent with the findings of the present study.

Other researchers have shown that obesity in children is associated with increased PI, gingival inflammation and

BoP, and that BMI is significantly correlated with periodontal indices, including PI, BoP, PPD, CAL, the oral hygiene index (OHI), and the community periodontal index (CPI).^{2,41,51,63–65} Martens et al. likewise demonstrated a positive association between overweight and obesity and the prevalence of periodontal disease.⁵³ They further reported that obesity was associated with less frequent daily toothbrushing and flossing, a higher prevalence of dental caries, a lower parental education level, a higher socioeconomic status, and less healthy dietary habits.⁵³

The increased BoP observed in obese participants may be attributed to elevated levels of pro-inflammatory cytokines in gingival crevicular fluid (GCF).³¹

Vaziri et al. reported that the mean PPD was higher in obese than in non-obese participants; although this difference was statistically significant, it was not considered clinically significant.⁴¹ Similarly, Mod  r et al.³¹ and Scorzetti et al.⁶³ found that participants in the obese groups had a significantly higher prevalence of PPD ≥ 4 mm.

In a study by Vallogini et al., the findings were inconsistent with those of the present study and the aforementioned studies.⁶⁶ The obese group showed lower visible PI values and gingival bleeding index (GBI) scores, as well as a better periodontal status in terms of PPD. In their study, obese pediatric participants were recruited from a children's hospital, whereas non-obese participants were recruited from a school.⁶⁶ Therefore, the discrepancies between the findings of that study and the present study may be attributed to differences in the sampling location, as participants recruited from a hospital setting may have received specific dietary and oral hygiene counseling, potentially resulting in improved periodontal parameters.

Kim et al. showed that individuals with a BMI higher than 25 (classified as overweight or obese) had a lower risk of periodontitis as compared to individuals of normal weight, whereas those with abdominal obesity had a significantly higher risk of periodontal disease.⁶⁷ These findings suggest that metabolic syndrome may play an important role in periodontal inflammation.^{68–70}

Reeves et al. concluded that the onset of periodontitis may be associated with weight gain and increased waist circumference.⁷¹ They found that among individuals aged 17–21 years, each 1-kilogram increase in body weight was associated with a 6% increase in the risk of periodontal disease, while each 1-inch increase in waist circumference was associated with a 5% increase in the risk of periodontitis. In contrast, no significant association was observed between these parameters and periodontal disease among younger children aged 13–16 years.⁷¹

The findings of most of the aforementioned studies indicate an adverse effect of obesity on periodontal health in young individuals, which is likely due to a combination of factors, including metabolic and inflammatory changes, inadequate attention to oral and dental hygiene practices and preventive measures, an unbalanced diet, and irregular dental examinations.^{72,73}

Correlation between the salivary cortisol levels and the periodontal status

The findings of this study showed that the salivary cortisol levels were positively and significantly associated with the GI, BoP and PPD indices. Various studies have investigated the role of chronic stress as an underlying factor in the development of oral and dental health problems. Based on substantial scientific evidence, stress and psychological disorders may cause individuals to neglect oral and dental hygiene, thereby accelerating plaque accumulation and the progression of periodontal disease.⁷⁴ Academic stress has also been reported as a risk factor for gingival inflammation, through increased levels of IL-1 β in GCF, as well as reduced oral hygiene.^{75–77}

It has been hypothesized that stress induces behavioral changes, such as overeating and an increased preference for high-fat diets, which may contribute to immunosuppression through increased cortisol production.⁷⁴ Furthermore, chronic stress can alter the salivary flow rate, pH and chemical composition.^{74,78} Rai et al. demonstrated that stress-related biomarkers, including the salivary cortisol concentration, were increased in children with dental caries.⁷⁹ In the present study, children with dmft/DMFT scores above 3 were excluded due to the potential association between the cortisol levels and dental caries.^{80–82} Therefore, the influence of this confounding variable was reduced in the current study, which represents one of its strengths.

It appears that periodontal disease and stress have a bi-directional relationship. Several studies have reported that the chronic elevation of the cortisol levels may be a potential risk factor for the onset or severity of periodontal disease in adults.^{74,83,84}

Consistent with the findings of the present study, Schmidt et al. showed that the salivary cortisol levels were significantly higher in adolescents with periodontitis than in those without periodontitis.⁸⁵ Individuals with elevated cortisol levels appear to be at a higher risk of developing periodontitis. In contrast, Panagiotou et al. found no association between stress and dental caries or periodontal disease.⁸⁶ One possible explanation for this discrepancy may be the lack of adherence by parents and participants to the scheduled saliva collection times at home.

Considering the complex nature of periodontal disease and numerous confounding factors that influence its development, comprehensive longitudinal studies are recommended in the future to further clarify and confirm the potential relationship between periodontal disease, BMI and the cortisol levels.

Limitations

The difficulty in obtaining participants' cooperation during oral examinations or saliva collection under calm conditions resulted in the need for repeated sampling.

Conclusions

The findings of the present study showed that:

- children's BMI was positively associated with the salivary cortisol levels and the GI, BOP and PPD indices, indicating that overweight and obese children have a poorer periodontal status compared with children of normal weight;
- the salivary cortisol levels, as an indicator of the serum cortisol levels, were higher in overweight and obese children than in children of normal weight.

These findings highlight the importance of encouraging healthy lifestyle modifications and weight management from early childhood and adolescence to prevent the long-term adverse effects of elevated cortisol levels on the body, and to improve periodontal health.

Ethics approval and consent to participate

The study was conducted in accordance with the Declaration of Helsinki after obtaining approval from the Ethics Committee of Qom University of Medical Sciences, Iran (No. IR.MUQ.REC.1400.231). All methods were carried out in accordance with the relevant guidelines and regulations. Written informed consent was obtained from the parents or legal guardians of all participating children.

Data availability

The datasets supporting the findings of the current study are available from the corresponding author on reasonable request.

Consent for publication

Not applicable.

Use of AI and AI-assisted technologies

Not applicable.

ORCID iDs

Aida Mehdi pour  <https://orcid.org/0000-0002-1456-4267>
 Amirhossein Khorrami  <https://orcid.org/0000-0001-6063-410X>
 Mohammad Aghaali  <https://orcid.org/0000-0003-3417-1580>
 Ali Saleh  <https://orcid.org/0000-0002-1137-1259>

References

- Panuganti KK, Nguyen M, Kshirsagar RK, Doerr C. *Obesity (Nursing)*. StatPearls [Internet]. Treasure Island, FL: StatPearls Publishing; 2021. Updated 2023. <https://www.ncbi.nlm.nih.gov/books/NBK568702>.
- Sfasciotti GL, Marini R, Pacifici A, Ierardo G, Pacifici L, Polimeni A. Childhood overweight–obesity and periodontal diseases: Is there a real correlation? *Ann Stomatol (Roma)*. 2016;7(3):65–72. doi:10.11138/ads/2016.7.3.065
- Kiliç M, Gurbuz T, Kahraman CY, Cayir A, Bilgiç A, Kurt Y. Relationship between the *TAS2R38* and *TAS1R2* polymorphisms and the dental status in obese children. *Dent Med Probl*. 2022;59(2):233–240. doi:10.17219/dmp/143252
- McPhee PG, Singh S, Morrison KM. Childhood obesity and cardiovascular disease risk: Working toward solutions. *Can J Cardiol*. 2020;36(9):1352–1361. doi:10.1016/j.cjca.2020.06.020
- Akbari H, Mohammadi M. The Prevalence of obesity in Iranian children: A systematic review and meta-analysis. *J Pediatr Rev*. 2022;10(2):93–102. doi:10.32598/jpr.10.2.875.2
- Tyson N, Frank M. Childhood and adolescent obesity definitions as related to BMI, evaluation and management options. *Best Pract Res Clin Obstet Gynaecol*. 2018;48:158–164. doi:10.1016/j.bpobgyn.2017.06.003
- Kaur J, Gandhi J, Sharma S. *Physiology, Cortisol*. StatPearls [Internet]. Treasure Island, FL: StatPearls Publishing; 2021. Updated 2025. <https://www.ncbi.nlm.nih.gov/books/NBK538239>.
- Silva Morais JB, Severo JS, Beserra JB, et al. Association between cortisol, insulin resistance and zinc in obesity: A mini-review. *Biol Trace Elem Res*. 2019;191(2):323–330. doi:10.1007/s12011-018-1629-y
- Di Dalmazi G, Pasquali R, Beuschlein F, Reincke M. Subclinical hypercortisolism: A state, a syndrome, or a disease? *Eur J Endocrinol*. 2015;173(4):M61–M71. doi:10.1530/EJE-15-0272
- Whitworth JA, Williamson PM, Mangos G, Kelly JJ. Cardiovascular consequences of cortisol excess. *Vasc Health Risk Manag*. 2005;1(4):291–299. doi:10.2147/vhrm.2005.1.4.291
- Van Rossum EF. Obesity and cortisol: New perspectives on an old theme. *Obesity (Silver Spring)*. 2017;25(3):500–501. doi:10.1002/oby.21774
- Kjölhede EA, Gustafsson PE, Gustafsson P, Nelson N. Overweight and obese children have lower cortisol levels than normal weight children. *Acta Paediatr*. 2014;103(3):295–299. doi:10.1111/apa.12499
- Hillman JB, Dorn LD, Loucks TL, Berga SL. Obesity and the hypothalamic–pituitary–adrenal axis in adolescent girls. *Metabolism*. 2012;61(3):341–348. doi:10.1016/j.metabol.2011.07.009
- Chalew SA, Lozano RA, Armour KM, Zadik Z, Kowarski AA. Reduction of plasma cortisol levels in childhood obesity. *J Pediatr*. 1991;119(5):778–780. doi:10.1016/s0022-3476(05)80302-6
- Chu L, Sheng K, Liu P, et al. Increased cortisol and cortisone levels in overweight children. *Med Sci Monit Basic Res*. 2017;23:25–30. doi:10.12659/msmbr.902707
- Kiess W, Meidert A, Dressendörfer RA, et al. Salivary cortisol levels throughout childhood and adolescence: Relation with age, pubertal stage, and weight. *Pediatr Res*. 1995;37(4 Pt 1):502–506. doi:10.1203/00006450-199504000-00020
- Rosmalen JG, Oldehinkel AJ, Ormel J, De Winter AF, Buitelaar JK, Verhulst FC. Determinants of salivary cortisol levels in 10–12 year old children; a population-based study of individual differences. *Psychoneuroendocrinology*. 2005;30(5):483–495. doi:10.1016/j.psyneuen.2004.12.007
- Filipovský J, Ducimetière P, Eschwege E, Richard JL, Rosselin G, Claude JR. The relationship of blood pressure with glucose, insulin, heart rate, free fatty acids and plasma cortisol levels according to degree of obesity in middle-aged men. *J Hypertens*. 1996;14(2):229–235. doi:10.1097/00004872-199602000-00012
- Walker BR, Soderberg S, Lindahl B, Olsson T. Independent effects of obesity and cortisol in predicting cardiovascular risk factors in men and women. *J Intern Med*. 2000;247(2):198–204. doi:10.1046/j.1365-2796.2000.00609.x
- Ward AM, Fall CH, Stein CE, et al. Cortisol and the metabolic syndrome in South Asians. *Clin Endocrinol (Oxf)*. 2003;58(4):500–505. doi:10.1046/j.1365-2265.2003.01750.x
- GBD 2016 Disease and Injury Incidence and Prevalence Collaborators. Global, regional, and national incidence, prevalence, and years lived with disability for 328 diseases and injuries for 195 countries, 1990–2016: A systematic analysis for the Global Burden of Disease Study 2016. *Lancet*. 2017;390(10100):1211–1259. doi:10.1016/S0140-6736(18)31891-9
- Nazir M, Al-Ansari A, Al-Khalifa K, Alhareky M, Gaffar B, Almas K. Global prevalence of periodontal disease and lack of its surveillance. *ScientificWorldJournal*. 2020;2020:2146160. doi:10.1155/2020/2146160
- Martins Chávayry NG, Vettore MV, Sansone C, Sheiham A. The relationship between diabetes mellitus and destructive periodontal disease: A meta-analysis. *Oral Health Prev Dent*. 2009;7(2):107–127. PMID:19583037.

24. Xiong X, Buekens P, Fraser WD, Beck J, Offenbacher S. Periodontal disease and adverse pregnancy outcomes: A systematic review. *BJOG*. 2006;113(2):135–143. doi:10.1111/j.1471-0528.2005.00827.x
25. Humphrey LL, Fu R, Buckley DJ, Freeman M, Helfand M. Periodontal disease and coronary heart disease incidence: A systematic review and meta-analysis. *J Gen Intern Med*. 2008;23(12):2079–2086. doi:10.1007/s11606-008-0787-6
26. Nardi GM, Grassi R, Ndokaj A, et al. Maternal and neonatal oral microbiome developmental patterns and correlated factors: A systematic review – does the apple fall close to the tree? *Int J Environ Res Public Health*. 2021;18(11):5569. doi:10.3390/ijerph18115569
27. Zermeno-Ibarra JA, Delgado-Pastrana S, Patiño-Marín N, Loyola-Rodríguez JP. Relationship between overweight–obesity and periodontal disease in Mexico. *Acta Odontol Latinoam*. 2010;23(3):204–209. PMID:21638960.
28. Perez MM, Pessoa JS, Ciamponi AL, et al. Correlation of salivary immunoglobulin A with body mass index and fat percentage in overweight/obese children. *J Appl Oral Sci*. 2018;27:e20180088. doi:10.1590/1678-7757-2018-0088
29. De Moura-Grec PG, Marsicano JA, Paz de Carvalho CA, De Carvalho Sales-Peres SH. Obesity and periodontitis: Systematic review and meta-analysis. *Cien Saude Colet*. 2014;19(6):1763–1772. doi:10.1590/1413-81232014196.13482013
30. Suvan J, D'Aiuto F, Moles DR, Petrie A, Donos N. Association between overweight/obesity and periodontitis in adults. A systematic review. *Obes Rev*. 2011;12(5):e381–e404. doi:10.1111/j.1467-789X.2010.00808.x
31. Modéer T, Blomberg C, Wondimu B, Lindberg TY, Marcus C. Association between obesity and periodontal risk indicators in adolescents. *Int J Pediatr Obes*. 2011;6(2–2):e264–e270. doi:10.3109/17477166.2010.495779
32. López R, Fernández O, Baelum V. Social gradients in periodontal diseases among adolescents. *Community Dent Oral Epidemiol*. 2006;34(3):184–196. doi:10.1111/j.1600-0528.2006.00271.x
33. Pari A, Ilango P, Subbareddy V, Katamreddy V, Parthasarthy H. Gingival diseases in childhood – a review. *J Clin Diagn Res*. 2014;8(10):ZE01–ZE04. doi:10.7860/JCDDR/2014/9004.4957
34. Putignano P, Dubini A, Toja P, et al. Salivary cortisol measurement in normal-weight, obese and anorexic women: Comparison with plasma cortisol. *Eur J Endocrinol*. 2001;145(2):165–171. doi:10.1530/eje.0.1450165
35. Kumar S, Kelly AS. Review of childhood obesity: From epidemiology, etiology, and comorbidities to clinical assessment and treatment. *Mayo Clin Proc*. 2017;92(2):251–265. doi:10.1016/j.mayocp.2016.09.017
36. Navazesh M. Methods for collecting saliva. *Ann N Y Acad Sci*. 1993;694:72–77. doi:10.1111/j.1749-6632.1993.tb18343.x
37. Arhakis A, Karagiannis V, Kalfas S. Salivary alpha-amylase activity and salivary flow rate in young adults. *Open Dent J*. 2013;7:7–15. doi:10.2174/1874210601307010007
38. Löe H. The gingival index, the plaque index and the retention index systems. *J Periodontol*. 1967;38(Suppl 6):610–616. doi:10.1902/jop.1967.38.6.610
39. Joss A, Adler R, Lang NP. Bleeding on probing. A parameter for monitoring periodontal conditions in clinical practice. *J Clin Periodontol*. 1994;21(6):402–408. doi:10.1111/j.1600-051x.1994.tb00737.x
40. Hefti AF. Periodontal probing. *Crit Rev Oral Biol Med*. 1997;8(3):336–356. doi:10.1177/10454411970080030601
41. Vaziri F, Bahrololoomi Z, Savabieh Z, Sezavar K. The relationship between children's body mass index and periodontal status. *J Indian Soc Periodontol*. 2022;26(1):64–68. doi:10.4103/jisp.jisp_899_20
42. Blair J, Adaway J, Keevil B, Ross R. Salivary cortisol and cortisone in the clinical setting. *Curr Opin Endocrinol Diabetes Obes*. 2017;24(3):161–168. doi:10.1097/MED.0000000000000328
43. Yu T, Zhou W, Wu S, Liu Q, Li X. Evidence for disruption of diurnal salivary cortisol rhythm in childhood obesity: Relationships with anthropometry, puberty and physical activity. *BMC Pediatr*. 2020;20(1):381. doi:10.1186/s12887-020-02274-8
44. Shimada M, Takahashi K, Ohkawa T, Segawa M, Higurashi M. Determination of salivary cortisol by ELISA and its application to the assessment of the circadian rhythm in children. *Horm Res*. 1995;44(5):213–217. doi:10.1159/000184628
45. Törnhaage CJ. Reference values for morning salivary cortisol concentrations in healthy school-aged children. *J Pediatr Endocrinol Metab*. 2002;15(2):197–204. doi:10.1515/jpem.2002.15.2.197
46. Larsson CA, Gullberg B, Råstam L, Lindblad U. Salivary cortisol differs with age and sex and shows inverse associations with WHR in Swedish women: A cross-sectional study. *BMC Endocr Disord*. 2009;9:16. doi:10.1186/1472-6823-9-16
47. Kudielka BM, Buske-Kirschbaum A, Hellhammer DH, Kirschbaum C. HPA axis responses to laboratory psychosocial stress in healthy elderly adults, younger adults, and children: Impact of age and gender. *Psychoneuroendocrinology*. 2004;29(1):83–98. doi:10.1016/s0306-4530(02)00146-4
48. Knutsson U, Dahlgren J, Marcus C, et al. Circadian cortisol rhythms in healthy boys and girls: Relationship with age, growth, body composition, and pubertal development. *J Clin Endocrinol Metab*. 1997;82(2):536–540. doi:10.1210/jcem.82.2.3769
49. Reinehr T, Kulle A, Wolters B, et al. Steroid hormone profiles in prepubertal obese children before and after weight loss. *J Clin Endocrinol Metab*. 2013;98(6):E1022–E1030. doi:10.1210/jc.2013-1173
50. Ljung T, Andersson B, Bengtsson BA, Björntorp P, Mårin P. Inhibition of cortisol secretion by dexamethasone in relation to body fat distribution: A dose–response study. *Obes Res*. 1996;4(3):277–282. doi:10.1002/j.1550-8528.1996.tb00546.x
51. Khan S, Barrington G, Bettiol S, Barnett T, Crocombe L. Is overweight/obesity a risk factor for periodontitis in young adults and adolescents? A systematic review. *Obes Rev*. 2018;19(6):852–883. doi:10.1111/obr.12668
52. Genco RJ, Borgnakke WS. Risk factors for periodontal disease. *Periodontol 2000*. 2013;62(1):59–94. doi:10.1111/j.1600-0757.2012.00457.x
53. Martens L, De Smet S, Yusof MY, Rajasekharan S. Association between overweight/obesity and periodontal disease in children and adolescents: A systematic review and meta-analysis. *Eur Arch Paediatr Dent*. 2017;18(2):69–82. doi:10.1007/s40368-017-0272-1
54. Goodson JM, Groppo D, Halem S, Carpino E. Is obesity an oral bacterial disease? *J Dent Res*. 2009;88(6):519–523. doi:10.1177/0022034509338353
55. DiBaise JK, Zhang H, Crowell MD, Krajmalnik-Brown R, Decker GA, Rittmann BE. Gut microbiota and its possible relationship with obesity. *Mayo Clin Proc*. 2008;83(4):460–469. doi:10.4065/83.4.460
56. Maury E, Brichard SM. Adipokine dysregulation, adipose tissue inflammation and metabolic syndrome. *Mol Cell Endocrinol*. 2010;314(1):1–16. doi:10.1016/j.mce.2009.07.031
57. Ouchi N, Parker JL, Lugus JJ, Walsh K. Adipokines in inflammation and metabolic disease. *Nat Rev Immunol*. 2011;11(2):85–97. doi:10.1038/nri2921
58. Wilson JA, Clark JJ. Obesity: Impediment to wound healing. *Crit Care Nurs Q*. 2003;26(2):119–132. doi:10.1097/00002727-200304000-00006
59. Doyle SL, Lysaght J, Reynolds JV. Obesity and post-operative complications in patients undergoing non-bariatric surgery. *Obes Rev*. 2010;11(12):875–886. doi:10.1111/j.1467-789X.2009.00700.x
60. Chaffee BW, Weston SJ. Association between chronic periodontal disease and obesity: A systematic review and meta-analysis. *J Periodontol*. 2010;81(12):1708–1724. doi:10.1902/jop.2010.100321
61. De Castilhos ED, Horta BL, Gigante DP, Demarco FF, Peres KG, Peres MA. Association between obesity and periodontal disease in young adults: A population-based birth cohort. *J Clin Periodontol*. 2012;39(8):717–724. doi:10.1111/j.1600-051X.2012.01906.x
62. Suvan J, Petrie A, Moles DR, et al. Body mass index as a predictive factor of periodontal therapy outcomes. *J Dent Res*. 2014;93(1):49–54. doi:10.1177/0022034513511084
63. Scorzett L, Marcattili D, Pasini M, Mattei A, Marchetti E, Marzo G. Association between obesity and periodontal disease in children. *Eur J Paediatr Dent*. 2013;14(3):181–184. PMID:24295000.
64. Franchini R, Petri A, Migliario M, Rimondini L. Poor oral hygiene and gingivitis are associated with obesity and overweight status in paediatric subjects. *J Clin Periodontol*. 2011;38(11):1021–1028. doi:10.1111/j.1600-051X.2011.01770.x
65. Zuza EP, Machado Nascimento LA, Caetano SL, et al. Periodontal disease and body weight assessment in children. *J Dent Child (Chic)*. 2017;84(1):3–8. PMID:28387183.
66. Vallogini G, Nobili V, Rongo R, et al. Evaluation of the relationship between obesity, dental caries and periodontal disease in adolescents. *Eur J Paediatr Dent*. 2017;18(4):268–272. doi:10.23804/ejpd.2017.18.04.02
67. 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(4):533–542. doi:10.1902/jop.2010.100274

68. Andriankaja OM, Sreenivasa S, Dunford R, DeNardin E. Association between metabolic syndrome and periodontal disease. *Aust Dent J*. 2010;55(3):252–259. doi:10.1111/j.1834-7819.2010.01231.x
69. Benguigui C, Bongard V, Ruidavets JB, et al. Metabolic syndrome, insulin resistance, and periodontitis: A cross-sectional study in a middle-aged French population. *J Clin Periodontol*. 2010;37(7):601–608. doi:10.1111/j.1600-051X.2010.01571.x
70. Timonen P, Niskanen M, Suominen-Taipale L, Jula A, Knuuttila M, Ylöstalo P. Metabolic syndrome, periodontal infection, and dental caries. *J Dent Res*. 2010;89(10):1068–1073. doi:10.1177/0022034510376542
71. Reeves AF, Rees JM, Schiff M, Hujoel P. Total body weight and waist circumference associated with chronic periodontitis among adolescents in the United States. *Arch Pediatr Adolesc Med*. 2006;160(9):894–899. doi:10.1001/archpedi.160.9.894
72. Trottni M, Bossù M, Corridore D, et al. Assessing risk factors for dental caries: A statistical modeling approach. *Caries Res*. 2015;49(3):226–235. doi:10.1159/000369831
73. Kesim S, Çiçek B, Aral CA, Öztürk A, Mazicioğlu MM, Kurtoğlu S. Oral health, obesity status and nutritional habits in Turkish children and adolescents: An epidemiological study. *Balkan Med J*. 2016;33(2):164–172. doi:10.5152/balkanmedj.2016.16699
74. Goyal S, Gupta G, Thomas B, Bhat KM, Bhat GS. Stress and periodontal disease: The link and logic! *Ind Psychiatry J*. 2013;22(1):4–11. doi:10.4103/0972-6748.123585
75. Deinzer R, Granrath N, Spahl M, Linz S, Waschul B, Herforth A. Stress, oral health behaviour and clinical outcome. *Br J Health Psychol*. 2005;10(Pt 2):269–283. doi:10.1348/135910705X26858
76. Deinzer R, Hilpert D, Bach K, Schawacht M, Herforth A. Effects of academic stress on oral hygiene – a potential link between stress and plaque-associated disease? *J Clin Periodontol*. 2001;28(5):459–464. doi:10.1034/j.1600-051x.2001.028005459.x
77. Hildebrand HC, Epstein J, Larjava H. The influence of psychological stress on periodontal disease. *J West Soc Periodontol Periodontol Abstr*. 2000;48(3):69–77. PMID:11381953.
78. Gupta OP. Psychosomatic factors in periodontal disease. *Dent Clin North Am*. 1966;10(1):11–19. doi:10.1016/S0011-8532(22)03408-5
79. Rai K, Hegde AM, Shetty S, Shetty S. Estimation of salivary cortisol in children with rampant caries. *J Clin Pediatr Dent*. 2010;34(3):249–252. doi:10.17796/jcpd.34.3.l858480k80031jn2
80. Caruso S, Gatto R, Cinque B, Cifone MG, Mattei A. Association between salivary cortisol level and caries in early childhood. *Eur J Paediatr Dent*. 2018;19(1):10–15. doi:10.23804/ejpd.2018.19.01.02
81. Tikhonova S, Booij L, D'Souza V, Crosara KT, Siqueira WL, Emami E. Investigating the association between stress, saliva and dental caries: A scoping review. *BMC Oral Health*. 2018;18(1):41. doi:10.1186/s12903-018-0500-z
82. Martínez M, Montero E, Carasol M, et al. Association between caries and periodontal diseases in a sample of employed adults in Spain: A cross-sectional study. *Clin Oral Investig*. 2021;25(6):3957–3966. doi:10.1007/s00784-020-03726-2
83. Cakmak O, Tasdemir Z, Aral CA, Dundar S, Koca HB. Gingival crevicular fluid and saliva stress hormone levels in patients with chronic and aggressive periodontitis. *J Clin Periodontol*. 2016;43(12):1024–1031. doi:10.1111/jcpe.12614
84. Haririan H, Andrukhov O, Böttcher M, et al. Salivary neuropeptides, stress, and periodontitis. *J Periodontol*. 2018;89(1):9–18. doi:10.1902/jop.2017.170249
85. Schmidt J, Strecker P, Kreuz M, et al. Stress-related hormones in association with periodontal condition in adolescents – results of the epidemiologic LIFE Child study. *Clin Oral Investig*. 2019;23(4):1793–1802. doi:10.1007/s00784-018-2599-3
86. Panagiotou E, Agouropoulos A, Vadiakas G, Pervanidou P, Chouliaras G, Kanaka-Gantenbein C. Oral health of overweight and obese children and adolescents: A comparative study with a multivariate analysis of risk indicators. *Eur Arch Paediatr Dent*. 2021;22(5):861–868. doi:10.1007/s40368-021-00643-0