

The Prevalence of the Periodontal Pathogen *Selenomonas noxia* and Salivary Visfatin Level in Overweight and Obese Individuals with Periodontitis

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Background: Periodontal diseases are prevalent and can be worsened by conditions such as obesity. Understanding obesity impact on periodontal health is essential for developing effective management strategies. Hence, the aim of the present study was to assess the prevalence of *Selenomonas noxia* and pro-inflammatory visfatin marker for periodontitis in overweight/obese and non-obese population and correlate them with periodontal status.

Materials and Methods: Eighty-five participants aged from (30–50) years were divided into three groups: healthy controls (n=25), normal-weight periodontitis patients (n=30), and overweight/obese periodontitis patients (n=30). Unstimulated saliva was analyzed for visfatin level using ELISA, and subgingival plaque was examined for *S. noxia* prevalence using qPCR. Data were analyzed using Kruskal–Wallis, Spearman correlation, and ROC curves.

Results: Overweight/obese periodontitis patients had significantly higher salivary visfatin level (111.1 ± 277.4 ng/mL) compared with normal-weight periodontitis (28.2 ± 20.6 ng/mL) and controls (19.3 ± 4.6 ng/mL) ($P < 0.001$). Similarly, *S. noxia* was more prevalent in overweight/obese individuals with periodontitis ($155052.5 \pm 647,130.3$) compared to persons with periodontitis and periodontal health ($P < 0.001$). Strong positive correlation encountered between bacteria and visfatin level in both patient groups ($P < 0.001$) and with BMI in the overweight/ obese group ($P = 0.001$). ROC analysis showed excellent diagnostic accuracy for *S. noxia* in overweight/obese patients with periodontitis (AUC=0.86) and strong performance for visfatin (AUC=0.83).

Conclusion: Obesity might play a devastating role in the pathogenesis of periodontitis through the increased salivary visfatin level and *S. noxia* prevalence. This indicates that they may serve as potential markers for periodontal inflammation in the obese population.

Keywords: periodontitis, obesity, salivary biomarkers, visfatin, *Selenomonas noxia*, qPCR, ELISA

Introduction

Periodontal disease, is an extremely prevalent disease with a significant global frequency, affecting half of all adults worldwide.^{1,2} It is a nonspecific inflammatory response to oral biofilm, affecting periodontal attachment.^{3,4} In recent years, increasing attention has been directed toward systemic conditions that may exacerbate this inflammatory process, particularly obesity. Obesity, in the same arrow, is a chronic condition defined by excessive fatty tissue building, which has many consequences to the human body. Obese people are in a condition of low-grade inflammation owing to the constant release of inflammatory mediators into the circulation and mouth fluids, which may link it to periodontitis.⁵ Excess body weight is currently the sixth most significant risk factor contributing to the entire burden of disease globally.⁶

The bidirectional link between obesity and periodontitis is complicated, Adipose tissue works as an endocrine organ, producing bioactive molecules known as adipokines. These adipokines continuously enter the systemic circulation and oral environment, creating a pro-inflammatory milieu that may increase susceptibility to periodontal breakdown. Among the adipokines, visfatin cytokines that contribute to systemic inflammation and oxidative stress, both of which share comparable

pathophysiological pathways,⁷ which was identified by Fukuhara et al in 2005.⁸ It is a pleotropic mediator that promotes inflammation, visfatin is detected in a variety of white blood cells, including tissue-bound macrophages, indicating that it is important for controlling immunological and defensive processes.⁹ In addition, the elevated level of pro-inflammatory cytokines such IL-1, IL-6, and TNF- α may significantly enhance visfatin release.¹⁰ Periodontal ligament cells produce visfatin, which is regulated by microbial, inflammatory, and biomechanical signals.¹¹ In patients with advanced periodontitis, dysbiotic subgingival biofilms, and inflamed gingiva contain a broad population of important pathogenic microorganisms including *Selenomonas noxia* (*S. noxia*).^{12,13} It is a gram-negative, obligatorily anaerobic, non-spore-forming, curved, crescent-shaped, motile rod that may serve as a periodontal pathogen in periodontal pockets.^{14,15} Saliva, gingival crevicular fluid (GCF), and supra/subgingival tooth biofilms are the main places where *S. noxia* detected.^{15,16} However, it may also be found on other oral surfaces¹⁷ and in some people's gastrointestinal tracts.¹⁷ According to the recent research findings, *S. noxia* may be linked to detrimental health problems, such as obesity.¹⁸ Given that this organism can ferment "indigestible" carbohydrates and extract extra calories from foods, routine saliva screening for this organism may offer important clinical insights into the risks of oral and systemic health among patients receiving routine dental care.¹⁹ Despite the growing interest in understanding the interplay between obesity-related inflammation, adipokine dysregulation, and periodontal microbiology, the combined role of visfatin and *S. noxia* in obesity-associated periodontitis has not been adequately explored. Although previous studies have examined each component independently, Li et al (2024) reported elevated visfatin levels in individuals with periodontal inflammation,²⁰ whereas another study carried by Qadir and Abdulrahman (2019) identified a higher prevalence of *S. noxia* among obese subjects.²¹ To the best of our knowledge, this is the first study to investigate the potential association of salivary visfatin with the prevalence of *Selenomonas noxia* in overweight and obese patients with periodontitis. We hypothesized that the chronic inflammatory response in obesity and their associated disturbances may impact periodontal health by modulation of adipokine (visfatin) expression and altered microbial prevalence in the context of periodontitis, leading to marked and significantly altered response in overweight/obese patients relative to non-obese periodontitis patients and healthy controls.

Subjects Materials and Methods

This case-control observational research conducted in Baghdad between December 2024 and May 2025. Participants included in this study were individuals seeking care from a nutrition expert center and the periodontal department. Participants were given a detailed information regarding the study's goals; this study conforms with the Declaration of Helsinki. All participants filling out a consent form approved by the ethical committee in College of Dentistry/ Mustansiriyah University under the reference number of (REC185) and study number of (MUOSU-202127).

To determine the appropriate sample size for the study, salivary visfatin was employed as a primary outcome measure. By using G power (authored by Franz-Faull, University of Kiel, Germany) with a power of 0.90 for the research and a two-sided alpha error of probability of 0.05, an effect size of F is 0.4 (large effect size),^{22,23} under these conditions the required sample size is 51 subjects; we expand the sample size to 85 subjects.

Therefore, the total sample size in this study will be 85 subjects their age range from 30 to 50 years divided into three distinct groups as follows: group I, the control group, comprising 25 healthy normal weight volunteers with healthy intact periodontium: had no clinical attachment loss (CAL), and probing pocket depth (PPD) ≤ 3 mm, with bleeding on probing (BOP) less than 10%.²⁴ Group II, consisting of 30 normal weight individuals diagnosed with periodontitis (stage II and III) and group III comprised of subjects with overweight and obesity and had periodontitis (stage II and III). The study's inclusion criteria for patients were encompassed who had detectable interdental clinical attachment loss (CAL) at ≥ 2 non-adjacent teeth, or the presence of ≥ 3 mm CAL on the buccal (facial) or lingual/palatal surfaces associated with pocketing >3 mm at $\geq$ two teeth.²⁵ In addition, all periodontitis cases were generalized and unstable (PPD ≥ 4 mm with BOP or PPD >5 mm with or without BOP), with no risk factors, eg, diabetes mellitus (DM) and/or smoking.²⁶ Body mass index (BMI) and Each patient's waist circumference (WC) was measured and estimated as an indication of obesity based on the World Health Organization's obesity categorization guidelines (BMI calculated as kg/m^2 , BMI from 18.5 to 24.9 was considered normal, $\geq 25 \text{ kg/m}^2$ was considered overweight, and BMI $\geq 30 \text{ kg/m}^2$ was considered obese).²⁷ Regarding WC (Normal weight: WC <80 cm in women and <90 cm in men, Overweight: if WC ≥ 90 cm in women and in men ≥ 100 cm and Obese if WC ≥ 105 cm in women and ≥ 110 cm in men). All participants should demonstrate excellent general health, lack of any past systemic conditions, and have a minimum of 20 teeth present.

Conversely, the exclusion criteria were the presence of any inflammatory oral illness (other than periodontitis) that may affect the levels of biomarker being studied, Individuals suffering from systemic illnesses, a history of alcohol use or smoking, pregnant, and nursing women, individuals who had taken antibiotics or anti-inflammatory drugs within the previous three months, or who have had extensive periodontal therapy and using vitamins, antioxidants, or contraceptives were also excluded in this study.

The clinical parameters used to assess periodontal health of participants were Plaque Index (PLI), Gingival Index (GI), Bleeding on Probing (BOP%), Probing Pocket Depth (PPD), and Clinical Attachment Loss (CAL), measurements were recorded using William periodontal probe. Teeth's surfaces were examined at six different points: mesio-lingual, mid-lingual, disto-lingual, disto-buccal, and mid-buccal. The third molars were excluded.

Saliva Sampling

Before saliva collection, the patients under examination were asked to avoid eating or drinking anything other than water for at least sixty minutes. To prevent contamination, the salivary samples collected before any oral clinical examinations; 5 mL of whole unstimulated saliva was collected between 9:00 a.m. and 12:00 p.m. using the passive saliva drooling method. The subject was instructed not to swallow during the collection process and passively drool the saliva over the lower lip into the plastic cup.²⁸ To extract the clear supernatant, the samples were centrifuged for 10 minutes at 3000 rpm. In order to prevent bacterial growth and minimize the loss of biomarkers due to protein denaturation, the samples were stored at -20°C within 4 hours of collection until ELISA analysis. All samples were thawed to room temperature before being analyzed.

Dental Plaque Sampling

Samples of subgingival plaque were obtained from the deepest periodontal pocket in the patient groups and the gingival sulcus in the control group. To reduce the chance of contamination by saliva or supragingival plaque, the sample region was thoroughly scaled supragingivally using a sterile curette, separated by cotton rolls, and cleaned with sterile cotton pellets.^{29,30} After that, a fine, sterile Gracey curette was used to collect plaque samples, which was inserted into the pocket as deeply as possible without exerting any pressure on the tooth surface to avoid the curette from dislocating the subgingival plaque into the depth of the pocket. When the curette contacted tissue resistance in the apical region of the pocket, subgingival sampling was accomplished with a single vertical stroke. The instrument was then quickly put into an Eppendorf tube with 0.5 mL of TE buffer (10 mM Tris-HCl, 1 mM EDTA, pH 7.6) and the tip of the instrument was vigorously stirred in the solution.³⁰ After that, the sample was kept at -40°C until the DNA extraction procedure.

ELISA Detection of Human Visfatin

Concentrations of salivary Visfatin were determined according to the manufacturer's instructions using ELISA kit (Shanghai YL Biont/China) catalog No.: YLA0808HU. A wavelength of 450 nm was used for spectrometric measurements, then the concentration (ng/mL) was calculated using a standard curve.

DNA Extraction

The DNA extracted from samples of dental plaque according to the manufacturer protocol of ABIO pure Extraction. The DNA quantity in each sample was measured by a spectrophotometer and afterwards kept at a temperature of -20°C until qPCR.

Quantitative PCR (qPCR) Screening

For precise detection and quantification of *S. noxia* bacteria, primers were used depending on previous study³¹ for *S. noxia* species-specific region on the 16S rRNA. Primer-BLAST at NCBI was used to validate primers sequence and check their specificity, Table 1.

A standard curve was established using known quantities of bacterial-specific DNA. In the qPCR procedure, the standard curve approach uses a dilution series with a known template copy number. For further quantifying the target DNA from the clinical samples, a standard curve was created using linear regression analysis using serial dilutions against CT (CT is the cycle number at which the fluorescence generated inside a reaction passes the threshold line), Figures 1 and 2.

Table 1 Primers Used for *S. noxia* Detection

Primers	Sequence '5-3'	Annealing Temp. (°C)
<i>S. noxia</i> FI	TCTGGGCTACACACGTACTACAATG	60
<i>S. noxia</i> RI	GCCTGCAATCCGAACTGAGA	
<i>S. noxia</i> P	FAM CAGAGGGCAGCGAGAGAGTGATCTTAAGCTAMRA	

Statistical Analysis

The computerized analysis statistics package for the social sciences (SPSS) software was used for all statistical analyses of the research data (version 28, IBM, USA). Descriptive data were expressed as minimum (Min), maximum (Max), mean, standard deviation (SD), and standard error (SE). The data distribution was examined using the Shapiro–Wilk test, which revealed a non-parametric distribution; thus, the Kruskal–Wallis test was used for multi-group comparisons and Dunnes post hoc test for further intergroup comparisons. For the correlation, Spearman's rank test was used. Receiver operating characteristic (ROC) and area under the curve (AUC) were used to assess the measured biomarker's diagnostic accuracy. A P-value of ≤ 0.05 was considered to be statistically significant.

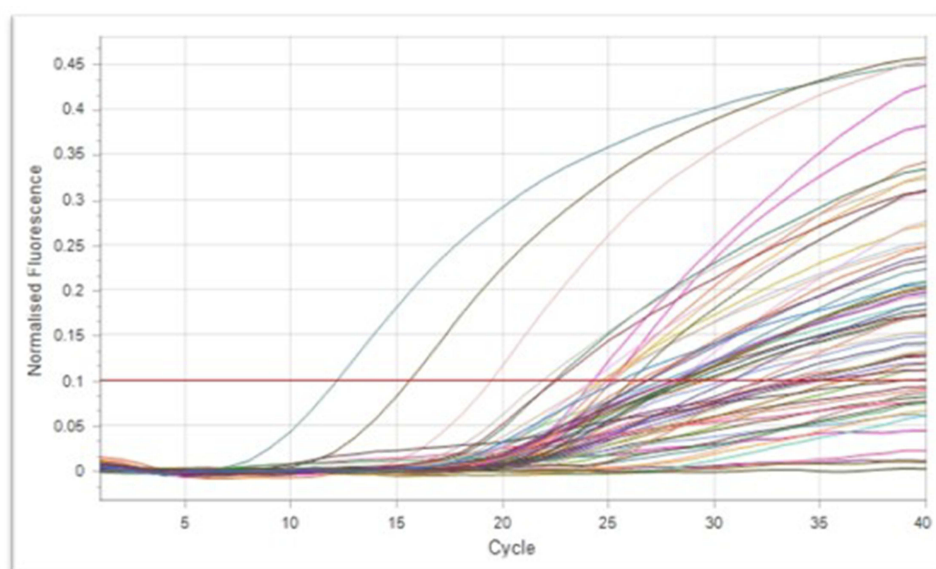
Results

Demographic Data of Participants

The demographic variables of the subjects enrolled in this study are illustrated in Table 2. The differences in age range among the study groups were statistically non-significant ($P > 0.05$). Regarding gender distribution, the normal weight periodontitis group showed male predominance, on other hand females were higher in patients group with overweight/obesity, the differences in gender distribution were statistically significant ($P < 0.001$).

Analysis of Clinical Periodontal and Body Parameters

Intergroup comparisons of the clinical periodontal and body parameters are summarized in Table 3. Statistically, highly significant differences were observed upon the comparison of PLI, GI, BOP%, BMI and WC scores among the study groups. Contrarily, the PPD scores showed a significant difference between patients (Group II and III). While CAL demonstrated a non-significant difference ($P > 0.05$).

**Figure 1** Amplification plot of *S. noxia* genomic DNA serial dilutions.

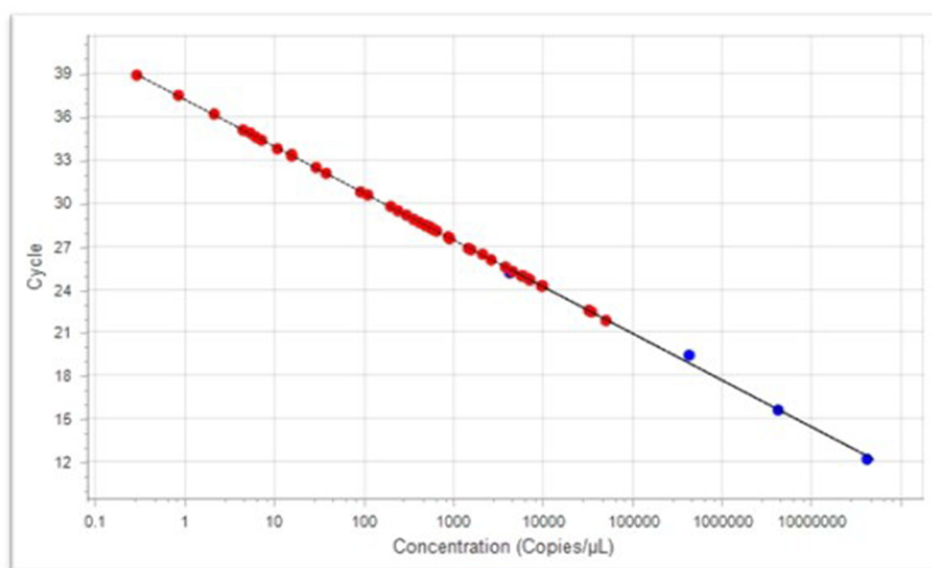


Figure 2 *S. noxia* standard curve.

Salivary Visfatin Level and Its Correlation with Periodontal and Body Parameters

The current study revealed a markedly elevated level of salivary Visfatin in overweight/obese patients with periodontitis (Group III) with a mean of (111.1 ng/mL) followed by normal weight patients with periodontitis (Group II) (28.2 ng/mL) and the lowest values recorded by controls (Group I) (19.3 ng/mL). The differences among the three study groups were highly significant as presented in Table 4. To reveal the exact statistical differences in Vasfatin levels between each pair of the study groups, Post hoc analysis was applied, which showed a non-significant difference between group I and group II ($P = 1.0$), while a highly significant differences were encountered between group I and III; group II and III, respectively ($P < 0.001$) as depicted in Table 5.

Regarding Spearman's rank correlation, the results demonstrated a significant positive correlation between increased salivary visfatin level and the presence of bleeding sites and BMI, WC in overweight/obese patients with periodontitis, whereas a non-significant correlation noticed regarding the rest of parameters in other study groups ($P > 0.05$), Table 6.

Quantification of Selenomonas noxia Bacteria Using Quantitative Real Time PCR (qPCR)

S. noxia showing the highest prevalence in the overweight/obese patient group (155052.5) than that of normal weight periodontitis and control groups with highly significant difference ($P < 0.01$), Table 7. Post hoc test also was used to

Table 2 The Demographic Characteristic of the Study Population

Age	Gr I No. 25		Gr II No. 30		Gr III No. 30		P-value**
	Mean ± SD	SE	Mean ± SD	SE	Mean ± SD	SE	
	41.2 ± 2.1	0.4	42.4 ± 7.5	1.3	41.1 ± 6.4	1.1	0.6
Gender							
Male	14 (56.0%)		25 (83.3%)		10 (33.3%)		<0.001 ^{HS}
Female	11 (44.0%)		5 (16.7%)		20 (66.7%)		

Notes: Statistical test used: **Kruskal–Wallis test.

Abbreviations: No., Number; Gr, Group; SD, Standard Deviation; SE, Standard Error; %, Percentage; HS, Highly Significant.

Table 3 Clinical Periodontal and Body Parameters

Parameters	Gr I No. 25		Gr II No. 30		Gr III No. 30		P-value**
	Mean $\pm$ SD	Median	Mean $\pm$ SD	Median	Mean $\pm$ SD	Median	
PLI	0.3 $\pm$ 0.1	0.2	1.6 $\pm$ 0.6	1.4	2.2 $\pm$ 0.5	0.09	<0.001 ^{HS}
GI	0.2 $\pm$ 0.1	0.2	1.6 $\pm$ 0.7	1.1	2.3 $\pm$ 0.5	2.6	<0.001 ^{HS}
BOP (% of score I)	2.4%		89.3%		89.1%		<0.001 ^{HS}
PPD	-	-	5.7	5.9	5.4	5.5	0.009 ^S
CAL	-	-	6.0	6.1	5.9	6.0	0.447
BMI	21.9 $\pm$ 0.7	21.9	22.0 $\pm$ 0.7	22.0	35.8 $\pm$ 1.1	36.9	<0.001 ^{HS}
WC	75.8 $\pm$ 4.5	74.0	76.5 $\pm$ 0.7	77.0	114.2 $\pm$ 1.7	116.5	<0.001 ^{HS}

Notes: Statistical test used: **Kruskal–Wallis test, Chi-square test for (BOP%).

Abbreviations: No., Number; Gr, Group; SD, Standard Deviation; %, Percentage; S, Significant; HS, Highly Significant; PLI, Plaque Index; GI, Gingival Index; BOP, Bleeding on Probing; PPD, Probing Pocket Depth; CAL, Clinical Attachment Loss; BMI, Body Mass Index; WC, Waist Circumference.

Table 4 Descriptive and Inferential Statistics of Salivary Visfatin

Parameters	Gr I No. 25		Gr II No. 30		Gr III No. 30		P-value**
	Mean $\pm$ SD	Median	Mean $\pm$ SD	Median	Mean $\pm$ SD	Median	
Visfatin ng/mL	19.3 $\pm$ 4.6	20	28.2 $\pm$ 20.6	17.5	111.1 $\pm$ 277.4	63.7	<0.001 ^{HS}

Notes: Statistical test used: **Kruskal–Wallis test.

Abbreviations: No., Number; Gr, Group; SD, Standard Deviation; HS, Highly Significant; ng/mL, nanogram per millilitre.

Table 5 Pairwise Comparison Among the Study Groups

Parameter	Groups	MD	SE	P value**
Visfatin ng/mL	Gr I – Gr II	–4.7	6.6	1.0
	Gr I – Gr III	–28.8	6.6	<0.001 ^{HS}
	Gr II – Gr III	–24.1	6.3	<0.001 ^{HS}

Note: Statistical test used: **Dunnes post hoc test.

Abbreviations: Gr, Group; SE, Standard Error; MD, Mean Difference; HS, Highly significant; ng/mL, nanogram per millilitre.

compare between each pair of study groups and found a highly significant difference ascertained between controls and obese patients with periodontitis and a significant difference between controls and non-obese patients with periodontitis; while between the two patient groups there was a non-significant difference as declared in Table 8.

Correlation of *S. noxia* Counts with Measured Parameters & Salivary Visfatin

The results of this investigation showed no significant relationship between the clinical periodontal parameters and *S. noxia* copy numbers ($P > 0.05$). While a positive significant correlation demonstrated with body parameter, BMI ($P = 0.001$) in patients with overweight/obesity. Contrariwise, a highly significant very strong positive correlation was observed with increased salivary visfatin level in both patient groups, as shown in Table 9.

Table 6 Correlation of Salivary Visfatin with Periodontal and Body Parameters Among the Study Groups

Parameters	Visfatin					
	Gr I No. 25		Gr II No. 30		Gr III No. 30	
	r	P-value**	r	P-value**	r	P-value**
PLI	0.2	0.1	0.3	0.07	0.2	0.1
GI	0.2	0.1	0.3	0.09	0.1	0.3
BOP I	0.1	0.6	-0.05	0.7	0.4	0.01 ^S
PPD	-	-	-0.2	0.2	-0.1	0.3
CAL	-	-	-0.1	0.4	0.05	0.7
BMI	0.293	0.155	-0.107	0.570	0.573	0.009 ^S
WC	-0.016	0.932	-0.107	0.570	0.370	0.043 ^S

Notes: Statistical test used: **r: Spearman's rank correlation.

Abbreviations: No., Number; Gr, Group; S, Significant; PLI, Plaque Index; GI, Gingival Index; BOP, Bleeding on Probing; PPD, Probing Pocket Depth; CAL, Clinical Attachment Loss; BMI, Body Mass Index; WC, Waist Circumference.

Table 7 Descriptive and Inferential Statistics of *S. noxia* Bacteria in Dental Plaque

Parameters	Gr I No. 25		Gr II No. 30		Gr III No. 30		P-value**
	Mean $\pm$ SD	Median	Mean $\pm$ SD	Median	Mean $\pm$ SD	Median	
<i>S. noxia</i>	125.9 $\pm$ 188.9	46	6709.8 $\pm$ 7562.06	4778	155,052.5 $\pm$ 647,130.3	12,700	<0.001 ^{HS}

Note: Statistical test used: **Kruskal-Wallis test.

Abbreviations: No., Number; Gr, Group; SD, Standard Deviation; HS, Highly Significant.

Table 8 Pairwise Comparison of *S. noxia* Levels Among the Study Groups

Parameter	Group	MD	SE	P-value**
<i>S. noxia</i>	Gr I – Gr II	-20.7	6.5	0.005 ^S
	Gr I – Gr III	-33.7	6.5	<0.001 ^{HS}
	Gr II – Gr III	-12.9	6.2	0.1

Note: Statistical test used: **Dunnet's post hoc test.

Abbreviations: Gr, Group; SE, Standard Error; MD, Mean Difference; S, significant; HS, Highly significant.

Table 10 provides crucial data on the diagnostic accuracy of *S. noxia* bacteria and salivary visfatin in discriminating between periodontitis (normal weight, overweight/obese) and periodontal health. In comparison of controls with normal weight periodontitis; *S. noxia* demonstrated good diagnostic accuracy with an AUC of 0.77 ($P=0.001$). At an optimal cutoff point of 64.5, the sensitivity and specificity were 0.66 and 0.88, respectively. While salivary Visfatin had a poor discriminatory ability, with an AUC of 0.56 ($P=0.4$), indicating no significant diagnostic value in this comparison.

Interestingly, in comparison of controls to overweight/obese patients with periodontitis; *S. noxia* again demonstrated high diagnostic accuracy (AUC=0.86, $P<0.001$), sensitivity of 0.80, and specificity of 0.88. Salivary Visfatin showed strong performance with an AUC of 0.83 ($P<0.001$). At the cutoff point of 22.5, the sensitivity was 0.76 and specificity was 0.67.

Table 9 Correlation Between *S. noxia* and Different Parameters Among the Study Groups

Parameters	<i>S. noxia</i>					
	Gr I		Gr II		Gr III	
	r	P value**	r	P value**	r	P value**
PLI	-0.1	0.6	0.3	0.07	-0.03	0.8
GI	0.2	0.2	0.2	0.2	-0.01	0.9
BOP%	0.04	0.8	-0.1	0.4	0.07	0.6
PPD	–	–	0.04	0.8	-0.01	0.9
CAL	–	–	0.08	0.6	-0.1	0.5
WC	0.3	0.09	0.1	0.3	0.2	0.1
BMI	0.3	0.1	0.1	0.5	0.5	0.001 ^S
Visfatin	0.1	0.3	0.7	<0.001 ^{HS}	0.7	<0.001 ^{HS}

Notes: Statistical test used:** r:Spearman's rank correlation.

Abbreviations: No., Number; Gr, Group; S, Significant; HS, Highly Significant; PLI, Plaque Index; GI, Gingival Index; BOP, Bleeding on Probing; PPD, Probing Pocket Depth; CAL, Clinical Attachment Loss; BMI, Body Mass Index; WC, Waist Circumference.

Table 10 Diagnostic Performance of *S. noxia* & Salivary Visfatin

Parameters	Gr	AUC	P-value	Optimal Cutoff Point	Sensitivity	Specificity
<i>S. noxia</i>	Gr I – Gr II	0.77	0.001 ^{HS}	64.5	0.66	0.88
Visfatin		0.56	0.4			
<i>S. noxia</i>	Gr I – Gr III	0.86	0.000 ^{HS}	64.0	0.80	0.88
Visfatin		0.83	0.000 ^{HS}	22.5	0.76	0.67

Abbreviations: Gr, Group; HS, Highly Significant; AUC, Area Under the Curve.

Discussion

Periodontists should recognize obesity as a multifactorial risk factor affecting both systemic and oral health. Emerging evidence links obesity with periodontitis and other chronic inflammatory diseases. Adipokines like visfatin may mediate this relationship, though their role in periodontal inflammation which remains unclear. Regular assessment of BMI and WC is recommended to evaluate periodontal risk in obese individuals.³²

In general, age is one of the challenging factors for periodontitis.³³ In this study, non-significant difference was observed between patients and controls regarding to age; this result was in agreement with other Iraqi studies.^{34,35} Additionally, there was a male predominance in the periodontitis patient group. This may be attributed to the fact that males have been perceived as being more susceptible to PD than females as a result of inadequate personal oral hygiene practices and insufficient health prevention behaviors.^{36,37} Furthermore, it has been suggested that the immune response plays an integral role that may be more intense in males and highly regulated in females.³⁸ There was a female predominance in patient group with obesity; the possible explanation is the considering factors such as hormones, specific resources for physical activity and exercise programs.³⁹ Socioeconomic factors contribute to obesity, as well as cumulative stress at the community level.⁴⁰

In terms of periodontal parameters, PLI, GI, and BOP% demonstrated highly significant differences among the study groups that's consistent with the known association between higher plaque accumulation and periodontal disease as the primary etiological factor for periodontitis.⁴¹ Plaque accumulation is consistently and substantially associated with GI in

all analysis.⁴² Bleeding on probing is an important objective sign of clinical inflammation, the presence of BOP may be considered as a predictor for the progression of periodontal disease.⁴³ This demonstrates how the clinical parameters of the gingival tissues reflect the state of the deeper portion of the periodontal soft tissues. In term of PPD, a significant difference between patient groups was found, this agreed with studies which found a positive correlation between obesity and periodontal parameters.^{44–46} Regarding CAL this study did not find any significant differences between the two patient groups. This result consistent with findings of previous studies which found no effect of obesity on CAL.^{47,48}

Chronic inflammation of periodontal tissue can promote the complex system of cytokines and mediators associated with obesity. The inflammatory signal in the gingival crevice produces advanced periodontal damage, which is exacerbated by markers released from adipose tissue.⁴⁹ Adipose tissue produces a vast number of cytokines and hormones, collectively called adipokines, including visfatin.⁵⁰ This study detected that the overweight/obese persons with periodontitis experienced significantly higher levels of salivary visfatin, followed by normal weight patients with periodontitis and controls, this came in accordance with findings of previous study.⁵⁰ Also, other studies^{51–54} found that salivary visfatin level was increased in obese people with and without chronic periodontitis. This increase in adipokines level is due to increased fat volume, as visfatin is positively correlated with human visceral fat.⁵⁵ Moreover, a study by Cetiner et al, (2019) reported that visfatin levels in GCF were higher in obese periodontitis patients than periodontitis and healthy controls.⁵⁶

Regarding the correlation of visfatin and periodontal parameters, this study revealed a positive significant correlation with BOP % in patient group with overweight/obesity. Elevated Visfatin levels contribute to amplified inflammatory response in periodontal tissue, upregulation of pro-inflammatory cytokines such as IL-6 and TNF- α and vascular changes in gingival tissues, leading to increased bleeding tendency.⁵⁴ This results consistent also with the findings of other studies.^{50,57–59} Likewise, salivary visfatin record a positive significant correlation with BMI and WC in overweight/obese patients which came in line with the findings of Ceylan Şen et al, study who suggested that increased visfatin levels in obesity may affect tissue destruction.⁶⁰ In the current investigation, the RT-PCR approach was effectively employed to identify and count *S. noxia* in subgingival microbial samples. The results showed that *S. noxia* was detected in just ten subject in healthy controls, with a much greater detection rate in patient's groups ($P < 0.01$). Several investigations^{61–63} confirmed that due to the use of bacterial quantitative data rather of dichotomous data (presence/absence or positive/negative), this improves the statistical power of discovering the predictive link with disease severity. These results were agree with recent study of Rams et al, (2025) that found significant detection of *S. noxia* in subgingival plaque of severe periodontitis patients (2.6-fold higher proportions) than controls.⁶⁴ The relationship between *S. noxia* and periodontal disease suggests that this organism is also associated with overweight and obese people without periodontal disease, which may imply that more complicated and interconnected pathways were involved.^{18,65–67} All relationships between *S. noxia* levels and clinical periodontal parameters were non-significant among study groups, the possible explanations for this outcome that *S. noxia* may contribute to disease onset or progression, but once the disease is established, its count alone might not explain the differences between patients who are all already diseased, also *S. noxia* in this study is measured at specific sites, but clinical parameters are assessed at the full-mouth level. These findings were in accordance with results of up-to-date studies^{68,69} who demonstrated that subgingival microbial profiles in obese periodontitis patients differ from non-obese ones, but these microbial alterations were not associated with greater PPD or CAL. This finding conflicting with other research who revealed a significant difference in bacterial levels based on CAL and PPD.¹⁵

In term of BMI, a strong positive association between increased BMI and *S. noxia* prevalence and this is coincided with Goodson et al, 2009 and Williams et al, 2024 observations. Goodson et al concluded that *S. noxia* could serve as a biomarker for overweight people.¹⁸ In Duhok, Iraq, Qadir *et al* in 2019 founded that *S. noxia* present in 86.8% of obese, 85.4% of overweight, and 77% of normal-weight individuals - confirming a strong association between elevated BMI and *S. noxia*.²¹

In terms of diagnostic accuracy between normal weight periodontitis and control groups, *S. noxia* demonstrated good diagnostic accuracy with an AUC of 0.77 ($P = 0.001$) and the sensitivity and specificity were 0.66 and 0.88, respectively, and visfatin had a poor discriminatory ability, with an AUC of 0.56 ($P = 0.4$), indicating no significant diagnostic value.

Again *S. noxia*, showed a strong diagnostic performance (AUC=0.86, $P < 0.001$), (sensitivity of 0.80, specificity of 0.88). Moreover, Visfatin was very good indicator for the difference between periodontal health and overweight/obese periodontitis patients with an AUC of 0.83 ($P < 0.001$), (sensitivity 0.76 and specificity 0.67) these were consistent with previous research findings.^{70,71}

Within the context of these findings, certain limitations should be acknowledged, as an observational case–control study, it identifies associations rather than causality between obesity, visfatin, *S. noxia*, and periodontal disease. In addition, a gender imbalance and several unassessed confounders such as diet, oral hygiene, hormonal status, and lifestyle behaviors may have influenced the results. Future longitudinal or interventional studies are needed to strengthen the evidence.

Conclusion

To summarize, this study demonstrated that the obesity is associated with elevated salivary visfatin and high prevalence of *S. noxia* bacteria which highlighting the link between metabolic dysregulation and periodontal inflammation affirming that the obesity may exacerbate the inflammatory and microbial burden linked to periodontitis. More studies are required to better understand the underlying processes and create appropriate treatment strategies. Collaboration between periodontal health specialists and obesity experts is critical for moving toward more integrated and individualized ways to addressing these interconnected disorders.

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Disclosure

The authors declare that they have no conflicts of interest in this work.

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