

Original Article

Correlation of serum adipocytokine levels with glycolipid metabolism and inflammatory factors in obese patients with periodontal disease

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Abstract: Objective: To analyze the correlation of serum levels of visfatin, leptin, resistin, and adiponectin (APN) with glycolipid metabolism and inflammatory factors in obese patients with periodontal disease. Methods: 116 obese adults (OB), of whom 78 participants were diagnosed with different degrees of chronic periodontitis (CP), and 50 healthy adults were recruited into the study. Fasting peripheral venous blood was extracted to determine serum levels of adipocytokines (e.g., visfatin, leptin, resistin, and APN), glycolipid metabolism (e.g., fasting blood glucose (FBG), fasting insulin (FINS), C-peptide (C-P), cortisol (Cor), homeostasis model of assessment for insulin resistance index (HOMA-IR), glycosylated hemoglobin (HbA1c), triglyceride (TG), total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and non-high-density lipoprotein cholesterol (non-HDL)), and micro-inflammation-related indexes like C-reactive protein (CRP), interleukin (IL)-1 β , IL-6, IL-10, and tumor necrosis factor (TNF)- α . Correlation between levels of adipocytokines and levels of glycolipid metabolism and inflammatory factors was further analyzed. Results: Assays for plasma levels of adipocytokines showed that both the OB group and the OB with CP group had significantly higher serum levels of visfatin, leptin, and resistin than the normal control group and significantly lower serum levels of APN than the normal control group ($P < 0.05$). Detection of serum glycolipid metabolism levels showed that FBG, FINS, C-P, Cor, HOMA-IR, TG, TC, LDL-C, HDL-C, Non-HDL-C of OB group, and OB with CP patients were significantly higher than those of normal patients ($P < 0.05$). Assay for plasma levels of inflammatory factors showed that both the OB group and the OB with CP group had significantly higher serum levels of CRP, IL-1 β , IL-6, and TNF- α than the normal control group and significantly lower serum levels of IL-10 than the normal control group. Spearman's correlation analysis revealed that serum levels of visfatin, leptin, resistin, and APN were significantly correlated with concentrations of FBG, FINS, C-P, Cor, TG, TC, LDL-C, HDL-C, Non-HDL-C, CRP, IL-1 β , IL-6, IL-10, and TNF- α . Conclusions: There were high expression levels of inflammatory factors and glycolipid metabolism disorder in obese patients with periodontal disease and excessively expressed adipocytokines may be important factors of persistent and worsened obesity and of periodontitis.

Keywords: Obese, chronic periodontitis, adipocytokines, glycolipid metabolism, inflammatory factors

Introduction

Obesity is due to excessive body fat accumulation or abnormal distribution that leads to weight gain. It not only attributes to the occurrence of diabetes, cardiovascular disease, and cancer but is also a risk factor for periodontitis [1-3]. In recent years, experimental and clinical studies have indicated that there is a correlation between obesity and periodontitis [4, 5]. Secretion of adipokines and oxidative stress are involved in occurrence and development of periodontitis [6, 7]. In addition, periodontal

pathogens and tooth loss may alter diet and lead to obesity [8].

Adipose tissue is closely associated with obesity. Adipose tissue is not only the energy storage organ but also can secrete many bioactive molecules such as visfatin [9], leptin [10], resistin [11], and adiponectin (APN) [12]. These adipocytokines are involved in inflammation while regulating insulin sensitivity and energy consumption at the same time [13]. Expression levels of visfatin leptin, and resistin which play a part in pro-inflammatory, are higher in obesity

patients while the expression level of APN which acts as an inflammation suppression factor is low. The imbalance of pro-inflammatory and anti-inflammatory cytokines leads to inflammation in an obese patient's body [14].

Periodontitis is a kind of disease caused by bacterial infection and results in chronic inflammation of periodontal tissue. Periodontitis is not only simple oral inflammation but also relates to whole body health. Periodontal disease can affect the body health or disease. In turn, systemic diseases can also influence the health of periodontal tissue [15-17]. However, whether periodontitis affects adipocytokines, glucolipid metabolism, and inflammatory factors in obese patients has remained unknown. Thus, our study aimed to analyze the correlation of serum levels of visfatin, leptin, resistin, and APN with glycolipid metabolism and inflammatory factors in obese patients with periodontal disease.

Materials and methods

Participants and assessment of obese status and periodontitis

The study was approved by the Human Research Ethics Committee of the First Hospital of Jinan University. Written informed consent was obtained from each subject.

This study was comprised of a series of cross-sectional and interventional studies of 116 obese patients and 50 healthy adults from the general population and from outpatients of the Stomatology Department at the First Affiliated Hospital, Jinan University, China. The trial was conducted from January 2015 to December 2016.

OB group: Height and weight were measured and BMI was calculated as the weight divided by the square of height (kg/m^2). Body mass index (BMI) was used to define non-obese and obese status. The cut-off point was $25 \text{ kg}/\text{m}^2$. This BMI threshold is based on the definition advocated by Western Pacific Regional Office of WHO (WPRO) for obesity in adult Asians [18, 19]. Meanwhile, these obese patients were ruled out with type 2 diabetes mellitus.

OB with CP group: Full-mouth periodontal examinations, except third molars, were performed

by one examiner with a standardized method using a manual periodontal probe. Bleeding on probing (BOP), probing depth (PD), and clinical attachment loss (AL) were measured at six sites (distobuccal, midbuccal, mesiobuccal, distolingual, midlingual, and mesiolingual) around each tooth to accurately diagnose periodontal disease. PD was measured from the gingival margin to the base of the clinical pocket. Clinical AL was recorded as the distance from the cement-enamel junction to the base of the clinical pocket, with the probe tip parallel to the long axis of the tooth. Chronic periodontitis (CP) was diagnosed when BOP, $\text{PD} \geq 3 \text{ mm}$, and AL were presented in at least 1 site. Severity was categorized on the basis of the amount of AL. The clinical criteria was where maximum $\text{AL} > 4 \text{ mm}$ is classified as severe CP, maximum $1 \leq \text{AL} \leq 4 \text{ mm}$ is diagnosed as moderate CP, and maximum $1 \leq \text{AL} \leq 4 \text{ mm}$ is diagnosed as mild CP [20, 21]. CP was included but aggressive periodontitis was excluded from the study. Localized or generalized extent of CP was not separated due to the limited number of participants [22]. After full-mouth periodontal examinations, 78 of the recruited 116 participants were diagnosed with periodontitis, 33 participants were diagnosed as mild CP, 42 participants were diagnosed as moderate CP, and 13 participants were diagnosed as severe CP.

Control group: 50 adults with regular menstrual period were enrolled as the control group.

Assays for plasma levels of adipocytokines

Plasma levels of visfatin, leptin, resistin, and APN were measured using ELISA kit (CUSABIO Life Science, Inc.), according to the manufacturer's instructions.

Detection of serum glucolipid metabolism levels

Fasting blood glucose (FBG) and glycated hemoglobin A1c (HbA1c) were measured by the glucose oxidase method and anion exchange high-performance liquid chromatography (HPLC), respectively. Triglyceride (TG), total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C) were analyzed enzymatically using an autoanalyzer (Hitachi). Non-HDL cholesterol (Non-HDL-C) concentration was measured by

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Table 1. Comparison of serum levels of adipocytokines in obese patients with periodontal disease ($\bar{x} \pm SD$)

Indices ($\mu\text{g/L}$)	OB (n=38)	OB with CP (n=78)			Normal Control (n=50)
		Mild (n=33)	Moderate (n=42)	Severe (n=13)	
Visfatin	33.27 \pm 4.21 ^b	35.52 \pm 4.19 ^b	40.84 \pm 5.66 ^{a,b}	49.81 \pm 5.57 ^{a,b,c}	18.78 \pm 2.72
Leptin	12.37 \pm 2.06 ^b	12.89 \pm 2.14 ^b	14.66 \pm 2.37 ^{a,b}	17.39 \pm 3.84 ^{a,b,c}	6.90 \pm 1.58
Resistin	21.48 \pm 3.70 ^b	21.69 \pm 3.43 ^b	29.64 \pm 3.87 ^{a,b}	32.33 \pm 4.06 ^{a,b,c}	11.42 \pm 2.41
APN	8.83 \pm 3.25 ^b	7.64 \pm 2.86 ^b	9.33 \pm 3.24 ^{a,b}	9.89 \pm 3.96 ^{a,b,c}	23.67 \pm 3.94

Abbreviations: OB = obese; CP = chronic periodontitis; APN = adiponectin. ^aP-values were assessed by independent samples t-test between OB group and each group of OB with CP, and $P < 0.05$; ^bP-values were assessed by independent samples t-test between Normal Control group and each of the other groups, and $P < 0.05$; ^cP-values were assessed by One-Way ANOVA analysis among OB with mild CP group, OB with moderate CP group and OB with Severe CP group, and $P < 0.05$.

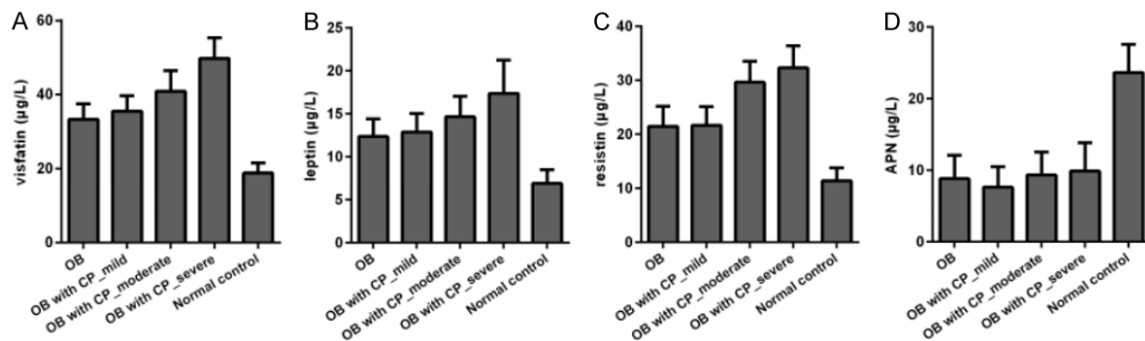


Figure 1. Comparison of serum levels of adipocytokines in obese patients with periodontal disease. A: Comparison of serum levels of visfatin; B: Comparison of serum levels of leptin; C: Comparison of serum levels of resistin; D: Comparison of serum levels of APN.

subtracting HDL-C concentration (mmol/L) from TC concentration (mmol/L) [23]. Fasting insulin (FINS), C-peptide(C-P), and cortisol (Cor) were tested by using automatic chemiluminescence immunoassay analyzer (Siemens ADVIA Centaur XP, Tarrytown, USA). In order to assess the insulin sensitivity, the homeostasis model of assessment for insulin resistance index (HOMA-IR) was evaluated by using FBG (mmol/L) and FINS (mU/L), $\text{HOMA-IR} = \text{FBG} \times \text{FINS} / 22.5$ [24].

Assay for plasma levels of CRP, IL-1 β , IL-6, IL-10, and TNF- α

Plasma was thawed at room temperature for protein measurement by enzyme-linked immunosorbent assay (ELISA). The human C-reactive protein (CRP), interleukin (IL)-1 β , IL-6, IL-10, and tumor necrosis factor (TNF)- α commercial ELISA kit (CUSABIO Life Science, Inc.) was used for CRP, IL-1 β , IL-6, IL-10, and TNF- α measurement, respectively.

Statistical analysis

The data were analyzed using version 16.0 of Statistical Package of Social Sciences software (SPSS Inc., Chicago, IL, USA). Data are presented as mean values \pm standard deviation and analyzed using independent samples t-test. Pearson's correlation coefficient(r) was used to calculate the correlation between serum levels of visfatin, leptin, resistin, and APN with glycolipid metabolism and inflammatory factors. Two-tailed $P < 0.05$ was considered statistically significant.

Results

Serum adipocytokines levels in OB group, OB with CP group, and normal control group

As shown in **Table 1**, both the OB group and the OB with CP group had significantly higher serum levels of visfatin, leptin, and resistin than the normal control group and significantly lower serum levels of ANP than the normal control

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Table 2. Comparison of serum levels of glucose and lipid metabolism in obese patients with periodontal disease

Indices	OB	OB with CP			Normal control
		Mild	Moderate	Severe	
FBG (mmol/L)	6.73±0.50 ^a	5.38±0.42 ^a	6.69±0.57 ^a	6.84±0.53 ^a	2.93±0.86
FINS (μU/mL)	22.73±13.44 ^a	22.54±12.67 ^a	21.51±12.29 ^a	21.26±10.83 ^a	5.31±1.28
C-P (ng/ml)	3.67±1.45 ^a	3.55±1.28	3.61±1.52	3.66±1.40 ^a	0.64±0.08
Cor (nmol/L)	284.14±34.11 ^a	269.14±31.57 ^a	283.22±33.96 ^a	275.14±32.69 ^a	157.37±24.74
HOMA-IR	6.52±3.82 ^a	4.52±2.31 ^a	5.88±2.67 ^a	6.33±3.29 ^a	1.02±1.01
HbA1C (%)	6.14±1.66	6.19±1.63	6.07±1.88	6.14±1.77	6.02±1.54
TG (mmol/L)	2.18±2.03 ^a	2.09±1.84 ^a	2.24±2.06 ^a	2.13±1.96 ^a	0.82±0.09
TC (mmol/L)	5.09±0.81 ^a	5.17±0.94 ^a	5.29±1.05 ^a	5.13±0.87 ^a	2.56±0.33
LDL-C (mmol/L)	3.19±0.71 ^a	3.35±1.01 ^a	3.24±0.82 ^a	3.22±0.81 ^a	1.84±0.26
HDL-C (mmol/L)	1.83±0.25 ^a	1.86±0.22 ^a	1.89±0.27 ^a	1.95±0.21 ^a	1.07±0.23
Non-HDL-C (mmol/L)	3.26±0.81 ^a	3.31±0.72 ^a	3.40±0.83 ^a	3.08±0.86 ^a	1.49±0.24

Abbreviations: OB = obese; CP = chronic periodontitis; FBG = fasting blood glucose; FINS = fasting insulin; C-P = C-peptide; Cor = cortisol; HOMA-IR = homeostasis model of assessment for insulin resistance index; HbA1C = glycosylated hemoglobin; TG = triglyceride; TC = total cholesterol; LDL-C = low-density lipoprotein cholesterol; HDL-C = high-density lipoprotein cholesterol; Non-HDL-C = Non-HDL cholesterol; ^aP-values were assessed by independent samples t-test between Normal Control group and each of the other groups, and P<0.05.

Table 3. Comparison of serum inflammatory factors in obese patients with periodontal disease

Indices	OB	OB with CP			Normal control
		Mild	Moderate	Severe	
CRP (mg/dL)	6.71±2.75 ^b	6.98±2.43 ^{a,b}	7.46±3.01 ^{a,b}	8.81±3.27 ^{a,b,c}	4.35±2.14
IL-1β (ng/mL)	1.17±0.14 ^b	1.25±0.12 ^{a,b}	1.37±0.15 ^{a,b}	1.56±0.24 ^{a,b,c}	0.74±0.02
IL-6 (ng/mL)	2.10±0.34 ^b	2.21±0.25 ^{a,b}	2.69±0.48 ^{a,b}	2.94±0.61 ^{a,b,c}	0.56±0.08
IL-10 (ng/mL)	1.03±0.11 ^b	0.87±0.06 ^{a,b}	0.74±0.09 ^{a,b}	0.69±0.04 ^{a,b,c}	1.76±0.53
TNF-α (ng/mL)	1.62±0.21 ^b	2.39±0.47 ^{a,b}	4.83±1.24 ^{a,b}	7.66±2.53 ^{a,b,c}	0.93±0.09

Abbreviations: OB = obese; CP = chronic periodontitis; CRP = C-reactive protein; IL-1β = interleukin-1β; IL-6 = interleukin-6; IL-10 = interleukin-10 and TNF-α = tumor necrosis factor-α; ^aP-values were assessed by independent samples t-test between OB group and each group of OB with CP, and P<0.05; ^bP-values were assessed by independent samples t-test between Normal Control group and each of the other groups, and P<0.05; ^cP-values were assessed by One-Way ANOVA analysis among OB with mild CP group, OB with moderate CP group and OB with Severe CP group, and P<0.05.

group. When compared with the OB group, the serum levels of visfatin, leptin, resistin, and ANP of the OB with moderate and severe CP group were all significantly higher. Among the three degrees of OB with CP groups, the serum levels of visfatin, leptin, resistin, and ANP of the severe group were significantly higher than that of mild and moderate groups (**Figure 1**).

Biochemical analysis of serum glucose and lipid metabolism levels

As shown in **Table 2**, measurements of serum glucose metabolism levels showed that FBG, FINS, C-P, Cor, and HOMA-IR of OB patients and OB with CP patients were significantly higher than those of normal patients. At the same

time, measurements of serum lipid metabolism levels showed that TG, TC, LDL-C, HDL-C, and Non-HDL-C of OB group and OB with CP patients were significantly higher than those of normal patients, as expected.

Assay for plasma levels of CRP, IL-1β, IL-6, IL-10, and TNF-α

As shown in **Table 3**, both the OB group and the OB with CP group had significantly higher serum levels of CRP, IL-1β, IL-6, and TNF-α than the normal control group and significantly lower serum levels than the normal control group. When compared with the OB group, the serum levels of CRP, IL-1β, IL-6, and TNF-α of the three degrees of OB with CP groups were all signifi-

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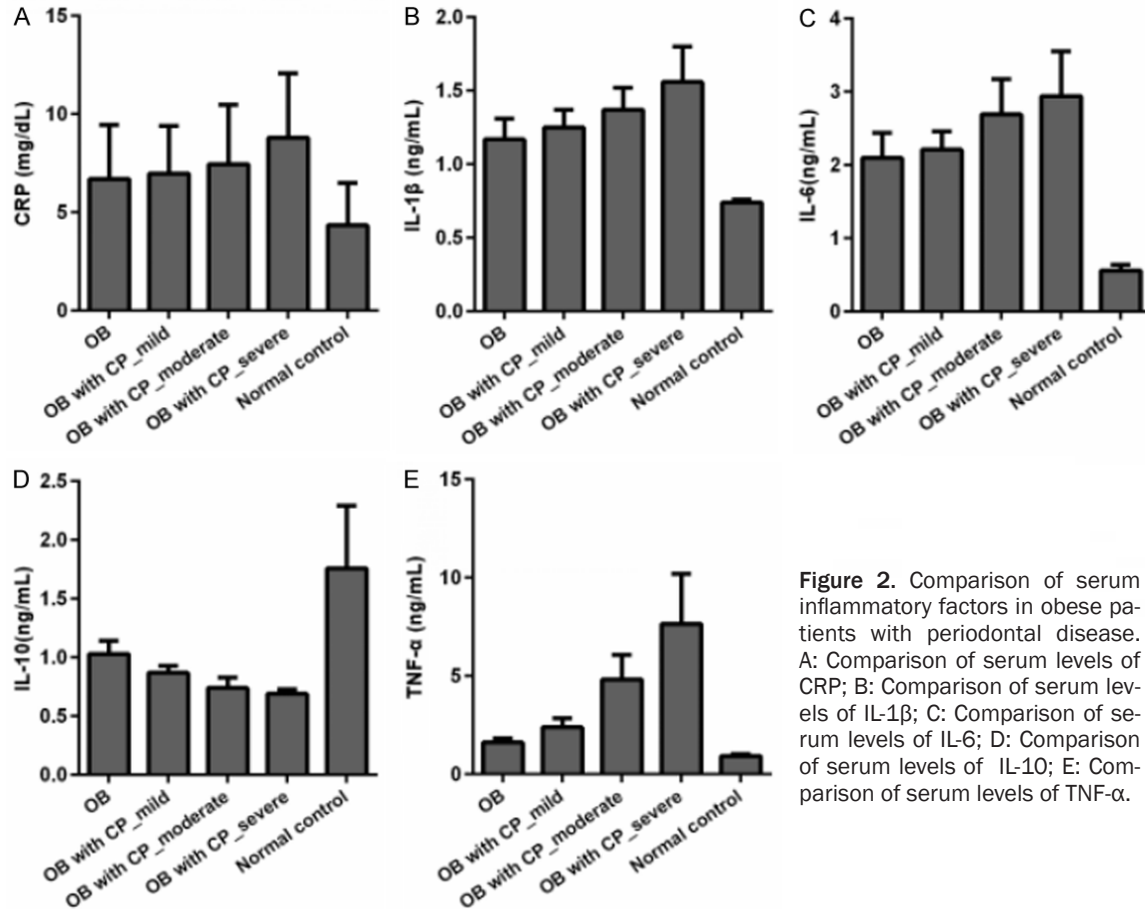


Figure 2. Comparison of serum inflammatory factors in obese patients with periodontal disease. A: Comparison of serum levels of CRP; B: Comparison of serum levels of IL-1 β ; C: Comparison of serum levels of IL-6; D: Comparison of serum levels of IL-10; E: Comparison of serum levels of TNF- α .

cantly higher while the serum level of IL-10 was significantly lower. Among the three degrees of OB with CP groups, the serum levels of CRP, IL-1 β , IL-6, and TNF- α of the severe group were significantly higher than that of mild and moderate groups while the serum level of IL-10 was significantly lower (**Figure 2**).

Correlation analysis of adipocytokines with glycolipid metabolism and inflammatory factors

As shown in **Table 4**, positive associations of serum levels of visfatin, leptin, and resistin with FBG, FINS, C-P, Cor, TG, TC, LDL-C, HDL-C, Non-HDL-C, CRP, IL-1 β , IL-6, IL-10, and TNF- α were found, while there was negative correlation of serum levels of APN with FBG, FINS, C-P, Cor, TG, TC, LDL-C, HDL-C, Non-HDL-C, CRP, IL-1 β , IL-6, IL-10, and TNF- α . Spearman's correlation analysis revealed that serum levels of visfatin, leptin, resistin, and APN were significantly correlated with the concentrations of FBG, FINS, C-P, Cor, TG, TC, LDL-C, HDL-C, Non-HDL-C, CRP, IL-1 β , IL-6, IL-10, and TNF- α in obese patients

with periodontal disease. No correlation was observed between visfatin, leptin, resistin, and APN with HOMA-IR and HbA1C.

Discussion

Obesity is the second highest risk factor, following smoking, of the occurrence and development of periodontitis. Perlstein [25], for the first time, reported an association between obesity and periodontitis. The experimental periodontitis model was established in normal rats and Zucker obese rats by the ligation method. The test found that, compared with the periodontitis group, the periodontal tissue of Zucker obese rats with periodontitis were more obviously inflamed and the extent of absorption of the alveolar bone was more serious. The degree of inflammation suggested that obesity might exacerbate periodontitis, which might make obesity a risk factor for periodontal disease. Epidemiological investigation and meta-analysis also supports the hypothesis that obesity is a risk factor of periodontal disease. Meta-

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Table 4. Associations of levels of serum level of adipocytokines with glycolipid metabolism and inflammatory factors in obese patients with periodontal disease

Indices	visfatin		leptin		resistin		APN	
	γ	<i>P</i>	γ	<i>P</i>	γ	<i>P</i>	γ	<i>P</i>
FBG	0.639	0.018*	0.773	0.014*	0.621	0.011*	-0.639	0.005*
FINS	0.565	0.022*	0.679	0.015*	0.662	0.033*	-0.737	0.001*
C-P	0.752	0.006*	0.723	0.005*	0.824	0.000*	-0.837	0.000*
Cor	0.714	0.014*	0.703	0.017*	0.726	0.009*	-0.692	0.013*
HOMA-IR	0.254	0.098	0.273	0.064	0.195	0.344	-0.229	0.256
HbA1C	0.166	0.312	0.209	0.438	0.103	0.545	-0.284	0.194
TG	0.753	0.019*	0.762	0.023*	0.715	0.034*	-0.788	0.014*
TC	0.886	0.001*	0.857	0.001*	0.824	0.001*	-0.892	0.006*
LDL-C	0.743	0.017*	0.726	0.015*	0.711	0.024*	-0.696	0.029*
HDL-C	-0.773	0.001*	-0.782	0.001*	-0.759	0.001*	-0.812	0.000*
Non-HDL-C	0.725	0.001*	0.756	0.001*	0.781	0.001*	-0.739	0.000*
CRP	0.816	0.001*	0.811	0.001*	0.818	0.001*	-0.813	0.000*
IL-1 β	0.822	0.001*	0.829	0.001*	0.792	0.003*	-0.826	0.000*
IL-6	0.784	0.002*	0.827	0.001*	0.761	0.010*	-0.843	0.000*
IL-10	0.799	0.001*	0.809	0.001*	0.804	0.001*	-0.827	0.000*
TNF- α	0.829	0.001*	0.831	0.001*	0.817	0.001*	-0.839	0.000*

Abbreviations: APN = adiponectin; FBG = fasting blood glucose; FINS = fasting insulin; C-P = C-peptide; Cor = cortisol; HOMA-IR = homeostasis model of assessment for insulin resistance index; HbA1C = glycosylated hemoglobin; TG = triglyceride; TC = total cholesterol; LDL-C = low-density lipoprotein cholesterol; HDL-C = high-density lipoprotein cholesterol; Non-HDL-C = Non-HDL cholesterol; CRP = C-reactive protein; IL-1 β = interleukin-1 β ; IL-6 = interleukin-6; IL-10 = interleukin-10 and TNF- α = tumor necrosis factor- α ; *Indicates $P < 0.05$.

analysis of 57 independent experiments found that the prevalence of periodontitis in obesity groups had increased 1/3 more than in non-obesity groups [26]. It was also found that the loss of attachment for obese patients with periodontitis was more severe than in non-obesity patients [27]. Many studies have found that incidence of periodontitis was increased with the increase of body mass index, showing a significant positive correlation [28]. The above experimental and clinical data indicate that there is a correlation between periodontitis and obesity, which can interact with each other.

Adipose tissue is loose connective tissue and its functions can be divided into white adipose tissue and brown adipose tissue. White fat stores energy and heat while brown fat produces heat because of the mitochondria that is rich in fat cells. In addition to triglyceride storage, white adipose tissue can produce a series of cytokines and hormones such as leptin, resistin, and inflammatory factors like TNF- α , IL-1 β , IL-6, and IL-10.

Leptin is the first discovered fat hormone, a molecular weight of 16 kDa non-glycosylated peptide hormone. Leptin is produced mainly by the fat cells, placenta, T-cells, osteoblasts, and gastric epithelial tissue. In addition to regulating food intake, energy expenditure, and lipid and bone metabolism, leptin can also regulate immune inflammatory process (mainly pro-inflammatory effects) [29]. Prior study has found that expression of leptin could be detected in normal or inflammatory gingival but in gingivitis the expression level was decreased and the serum expression level was increased [30]. Clinical studies have shown that,

compared with periodontal health patients, the serum leptin concentration in patients with periodontitis was significantly increased, periodontal parameters (probing depth and attachment loss) were positively related, and periodontal treatment could make the level of serum leptin decreased [31]. These results suggest that leptin plays an important role in the development of periodontitis.

APN, secreted by fat cells, was decreased in obese individuals. APN can not only inhibit the generation of TNF- α and IL-6 but also induce IL-1 receptor antagonist. Thus, its effects are anti-inflammatory, promote vascular protection, and are anti-diabetic. APN can maintain periodontal health, improve periodontal disease, and promote periodontal tissue regeneration and recovery [32]. However, some researchers have believed that there was no effect of APN in periodontitis due to the fact that its reduced receptor change will cause aggravated periodontitis [33]. So, the role of APN in periodontitis remains to be confirmed.

Resistin does not come directly from fat cells but is produced by inflammatory cells infiltrated inside the adipose tissue. Inflammatory stimulation, LPS, and IL-6 fat can promote the release of resistin and resistin, in return, can promote the secretion of TNF- α and IL-6 and weaken the anti-inflammatory effects of APN in activated inflammatory cells [34]. The function of resistin in fat cells can lead to insulin resistance. Resistin level also can increase with the increased degree of obesity. These are the main causes of obesity, susceptibility to type 2 diabetes, and periodontitis. Studies have found that the severity of periodontitis and gingival crevicular fluid in resistin level was positively related [35].

Mild inflammation caused by excessive accumulation of adipose tissue may play an important role in changes of the oral micro environment and this kind of inflammation mainly results in the increased secretion of pro-inflammatory cytokines in fat cells and macrophages in white adipose tissue (such as IL-1 β , IL-6, IL-10, and TNF- α) [36]. These inflammatory factors can change the host immune response and cause increased susceptibility to bacterial infection, resulting in periodontitis development [37]. Studies have found that high concentrations of TNF- α can stimulate fibroblast cells and then promote the synthesis of degrading enzymes and activation of osteoclasts bone resorption, exacerbating the development of periodontitis [38].

The interaction mechanism between obesity and periodontitis is still not clear. Many studies have shown that visfatin and leptin could promote the synthesis of pro-inflammatory cytokines and proteolytic enzymes in periodontal cells while APN could down regulate expression of these molecules [39]. In addition, visfatin and leptin could inhibit enamel matrix proteins which play a role in periodontal cells to enhance the role of APN and promote tissue regeneration. Serum levels of visfatin and leptin in obese patients were high while APN was low [40]. Obesity and obesity-related systemic disease increase the risk of patients with periodontal disease and periodontal disease, at the same time, also contributes to the development of obesity-related diseases [41].

Conclusion

In conclusion, there are high expression levels of inflammatory factors and glucolipid metabo-

lism disorder in obese patients with periodontal disease. This study suggests a role of periodontitis in the systemic inflammatory response in obese patients and that excessively expressed adipocytokines may be the important factors of persisting and worsening obesity and periodontitis.

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Disclosure of conflict of interest

None.

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