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# Evaluation of leptin levels in gingival crevicular fluid and serum in periodontitis individuals and its correlation with serum lipid, anthropometric and periodontal parameters: A clinico-biochemical study

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**Abstract**
**Background:** Leptin, an adipocytokine with pro-inflammatory properties has shown a fundamental role in the association of obesity and periodontitis with cardiovascular disease. However, little is known regarding the potential association between leptin and periodontitis with obesity parameters like Body Mass Index (BMI), Body Fat Mass (BFM) and lipid profile (LP). Hence, the aim of this study is to evaluate GCF and serum levels of leptin and compare with obesity parameters in periodontal health and disease to get a clear picture of periodontal-systemic health relationship in terms of leptin.
**Methods:** Two hundred and eight subjects with both gender in the age range of 30-39 years were divided into Periodontally Healthy (PH) and Moderate to severe Periodontitis (MP) groups based on clinical parameters. The groups were subcategorised based on BMI, BFM and LP into Normal Weight (NW), Over Weight (OW), Obese Weight (Ob), Average Fat Mass (AFM), Obese Fat Mass (OFM), Normolipidemic (NL) and Hyperlipidemic (HL). GCF and serum levels of leptin were evaluated using Enzyme Linked Immuno Sorbent Assay (ELISA).
**Results:** In serum, leptin levels were higher in the MP compared to PH and GCF leptin levels were higher in PH and lower in the MP group ($p<0.05$). The GCF leptin in PH group had a positive correlation with BMI (r=0.3), BFM (r=0.4) and LP (r=0.1) and no significant difference was observed in the MP group whereas the serum leptin showed a positive correlation only with BFM (r=0.6) and LP (r=0.2) in both MP and PH groups, respectively.
**Conclusion:** In obese and non-obese individuals, there was an inverse relationship in GCF leptin levels in MP and PH groups. GCF leptin was correlated with BFM & LP in periodontal health whereas serum leptin was correlated with BFM & LP in both periodontal health and disease.

**Keywords:** Leptin, gingival crevicular fluid, hyperlipidemia, body mass index, cardiovascular disease, periodontitis

## Introduction

Leptin is a peptide hormone which has a molecular weight of 16-kDa released into circulation after its primary production by adipocytes [1, 2]. It's regulatory functions ranges from regulation of bone metabolism, body weight and various endocrine axes [3]. It enhances the macrophage, natural killer cells, monocyte phagocytosis and increase the pro-inflammatory cytokine production as it possess immuno-modulatory functions stimulating the immune system [4, 5].

Several reports have indicated an association between leptin and generalized moderate periodontitis. (MP).1-6 Rise in leptin concentrations is seen highest in healthy gingiva1 and its decline with progressing periodontal disease reflecting increase in serum leptin concentrations [3]. This rise in leptin concentration in healthy gingiva suggests its protective role in periodontal disease and the rise in serum leptin concentration is considered a risk factor for the development of cardiovascular disease [4, 7].

Obesity affects leptin levels in MP subjects. Zimmermann *et al*. [8] have shown higher serum levels of leptin in obese than those in non-obese individuals suggesting a role of both periodontitis and obesity in enhancing systemic inflammatory burden predisposing to systemic complications. However, little is known regarding the potential association between leptin and

MP in obese individuals when compared to healthy individuals in terms of obesity parameters such as BMI values, different body fat mass and serum lipid profile which may provide more detail and clear association of leptin on various parameters of obesity [9].

Hence, the present study is undertaken, to correlate various obesity markers such as serum lipid, anthropometric and periodontal parameters in periodontal health and disease. Secondly, to estimate GCF and serum leptin levels to explore periodontal systemic health relationship in terms of leptin.

**Materials and Method**

It was a cross sectional human clinical study consisting of 208 patients conducted during the study period from September 2014 to March 2015 visiting the outpatient section of the Department of Periodontology, Krishnadevaraya College of Dental Sciences and Hospital, Bangalore. Before participation, all participants were informed about the study goals and procedures, and all participants gave written informed consent in accordance with the Declaration of Helsinki. Ethical clearance for the study was obtained pertaining to the Helsinki guidelines for Research Studies by the institutional review board affiliated to Rajiv Gandhi University for Health Sciences. The following were Inclusion criteria: 1) Age group of patients within 30 to 39 years. 2). Obese and non-obese subjects with and without moderate periodontitis. 3). Patients who are co-operative and able to attend follow up. 4). Patients who had not received any periodontal treatment in the last six months. 5). Dentition with at least 15 functioning teeth.

The following were the exclusion criteria: 1) any systemic diseases, such as diabetes mellitus or thyroid disease ( confirmed by lab test); 2) any bone disease; 3) any bacterial infection; 4) immunologic disorders; 5) hepatitis; 6) any other bacterial oral infections; 7) pregnant and lactating females;8) former and current smokers.9). None of the participants received antibiotics (that would affect serum Leptin levels) within the previous 3 months or treatment of periodontal disease (that would affect GCF Leptin levels) within the 6 months before the study. The dentition of each volunteer was examined clinically and radiographically to assess the suitability of the participants for the study. The participants of the study had to have ≥ 16 teeth who were co-operative and able to attend follow up were only included.

**Study Groups**

The study population was classified into two groups based on their periodontal condition according to criteria proposed by the 1999 International World Workshop for classification of periodontal disease and conditions and its 2017 modification of the same [10, 35]. When all the 6 sites of all the teeth were examined in the mouth 1). Periodontally Healthy (PH) group had no sites with PD >3mm and CAL >0 mm and no alveolar bone loss. 2). Generalized moderate to severe periodontitis (MP) group showed moderate to severe alveolar bone loss and CAL ≥ 5 mm and PD 6mm in multiple sites of all four quadrants of the mouth but no evidence of rapid progression.

**Clinical periodontal and anthropometric parameters**

The PD and clinical attachment level (CAL) using manual periodontal probe††were determined at six sites per tooth and the plaque index (PI) and gingival index (GI) were determined at four sites per tooth in the whole mouth, excluding third molars. All measurements were performed by a calibrated examiner (AJO).

BMI was calculated by dividing the weight in kilograms by square of height in meters. (Kg/m2). Based on BMI the subjects were sub-categorised based on Misra Classification for Asian Population8 into Normal Weight- 18-22.9kg/m2, over weight - 23-24.9kg/m2, Obese Weight > 25kg/m2.

Body Fat Mass (BFM) was analyzed using DEXA (Dual Energy X Ray Absorptiometry) scan using a total-body scanner. ‡‡ BFM were sub-categorised for female and male into Average Fat Mass (AFM) and Obese Fat Mass (OFM) categories.11 BFM in AFM category for male was 18-24% and female was 25-31% and BFM in OFM for male was ≥25percent and for female was ≥32percent.

Lipid profile analysis was performed on serum obtained from patient's blood to analyze lipid profile such as Total Cholesterol (TC), Triglyceride (TG), High Density Lipoproteim (HDL) using the enzymatic calorimetric method§§ and Low density lipoprotein (LDL) were measured using Friedewald formula:

LDL = TC- (HDL+ TG/5).

The patients were categorised into Normolipidemic (NL) patients12 when TC ≤ 200 mg/dl, TG between 50-200mg/dl, LDL between 80-130 mg/dl and HDL within 35-75mg/dl whereas Hyperlipidemic (HL) 10 when patient's TC is ≥ 200 mg/dl, TG ≥ 200mg/dl, LDL ≥130mg/dl and HDL < 35mg/dl.

†† William's periodontal probe, Hu-Friedy, Chicago, IL.

‡‡ Hologic Imaging, Discovery Series, Bedford, Massachusetts §§ Accuchem 240T analyser, Diriu Technology, China

**Examiner Calibration for Periodontal Examination**

Reproducibility of the examiner (AJO) was assessed by carrying out clinical periodontal data collection on five patients. Each subject was assessed twice (BVK) in one visit, over a 1-h interval. The second set of recordings were carried out ''blinded'' to the first assessment. Reproducibility of the data collection was determined by calculation of the per-centage of the sites examined where the scores should be repeated exactly or to an accuracy of 1 mm for each site. Assessment of the mean difference in the scores (with 85% accuracy) between visits indicates that there was no systematic bias in measurement. The kappa value for intra-examiner agreement for AJO and BVK, between the two measurements was recorded to be 0.91 and 0.94 respectively. The inter-examiner calibration was recorded to be 0.

**Sample Size calculation**

The ideal sample size was calculated to provide an adequate power considering difference of 1300 ± 300pg/ml of GCF leptin and 3500 ± 1700pg/ml of serum between MP and PH groups. 14 patients in each group was considered necessary with 95 % confidence intervals to provide a 85% power with a $\alpha$ of 0.05.

Further, based on the serum lipid profile, BMI values and body fat mass patients were sub-divided into various subcategories as shown in Table 1.

**Collection of GCF and serum for determination of leptin levels**

All participants before GCF sampling, gentle removal of supra gingival plaque was completed using dry gauze and perio paper strip*** was gently inserted into the entrance of the gingival sulcus of the deepest probing site buccal sites from until the first sign of

*** Oraflow, Amityville, USA resistance was felt and held in place for 30 seconds and unstimulated GCF sample was

collected. The volume of GCF was determined in Periotron‡ where a digital readout was converted to microliters using software (MLCONVERT.EXE software version 2.52, OraFlow). GCF samples were placed in plastic vials containing 200 µL of PBS, the GCF from paper strips were eluted at 2500 rpm (2750g) for 20 minutes using micro centrifuge.

**Leptin Analysis**

The GCF and serum samples stored at -700 C were assayed for leptin using sandwich Enzyme Linked Immuno Sorbent Assay (ELISA)13††† with an inter assay variation of 12%. The percentage of recovery of leptin was 94.49% for serum and 93.85% for GCF. Samples were run in duplicates and mean was taken into consideration.

**Statistical Analysis**

The data for GCF and serum leptin levels was subjected to the tests of normality (Kolmogorov-Smirnov & Shapiro-Wilk test).All results were expressed as the mean ± SD for each group. Non- parametric tests were used for statistical analysis Kruskall-Wallis analysis was carried out for inter sub-group comparison and Mann- Whitney U test was done for pairwise comparison between subgroups in the PH and MP categories. Spearman's Rank correlation analysis was done for correlation analysis between clinical parameters with GCF and serum leptin levels. A P value <0.05 was considered to be statistically significant.

**Results**

**Demographic and Clinical Parameters**

All 208 patients (145 males and 63 females) (ref Table 1) showed statistically significant difference in the gender and no significant differences in age, BFM and BMI in PH and MP categories. The levels of leptin in GCF and serum and their correlation with clinical parameters such as MGI,

*** Raybiotech, California, USA PI, BOP, RAL and PD showed statistically significant differences in both PH and MP groups as shown in Table 1.

Correlation of GCF leptin and serum leptin with clinical parameters

Correlation between the GCF and serum leptin levels and the clinical parameters using Spearman's rank correlation test demonstrated that the mean GCF leptin levels showed negative correlation and serum leptin showed positive correlation with MGI, PI, PD, BOP and RAL in both periodontal health and disease as shown in Table 2.

When inter group pairwise comparison was done among sub categories in PH and MP using Mann Whitney U test, the GCF leptin showed no correlation among the sub categories in PH and MP. The serum leptin shows highest correlation with the OFM and NL categories which is statistically significant as shown in graph 1&2.

**GCF Leptin levels and Serum Leptin levels**

Leptin being detected in all serum and GCF samples, GCF leptin was highest in PH category than in MP category. In the PH category, the highest mean GCF leptin concentration was seen in the ObW sub-category (1880±339 pg/mL) which was statistically significant and in MP category of patients, the highest mean GCF leptin was seen among the OFM sub-category (422±113 pg/mL) as shown in Table 3.

The mean serum leptin concentration is least in PH category group when compared to highest mean serum concentration in MP category which was statistically significant. In the PH category the highest mean serum leptin concentration can be seen in the ObW category (3585±277 pg/mL) which was non-significant. In the MP category, the highest mean serum concentration was seen with the OFM patient (7157±389 pg/mL) whereas the lowest concentration is seen in NW category (3095±564 pg/mL) which was statistically significant as shown in Table 4.

**Discussion**

Obesity is associated with oral diseases particularly periodontitis and recent studies have strongly suggested that leptin could be one of the potential mechanism that links obesity to periodontitis since higher serum levels of leptin are seen in these individuals [8, 14].

Of late, there is increasing evidence15 that BMI cut-off values are not valid for all populations and differs between different population groups.16 In addition it has been stated that the amount of body fat and lipid profile rather than the amount of excess weight determines the health risk of obesity (state of adipose tissue) and gives a better index of adiposity and gives a more accurate information on the state of obesity [16].

McNeely *et al*. drafted that leptin might be one of the links between obesity and periodontitis on observing the fluctuation in the level of leptin and decrease and increase in obesity along with the change in level of leptin in relation to periodontal inflammation.36 In the present study, we could find there is a relationship leading to a association of leptin with periodontitis in normal, overweight and obese patients. There is a drop in leptin levels in periodontitis and the raise in serum leptin can be because periodontitis causes increase in systemic inflammatory markers including CRP, IL 1, IL 6, IL 8, and TNF α.

Hence, we speculate that obesity and periodontal disease studied in detail in terms of BMI, BFM and lipid profile may provide more detail and clear information about the periodontal systemic health relationship in terms of leptin. Therefore, the present study is undertaken, which is first of its kind to evaluate the alteration in the GCF and serum leptin levels on the various parameters of obesity like anthropometric (BMI), Body Fat mass (BFM) and serum marker of obesity lipid profile (LP) in patients in periodontal health and disease to explore periodontal systemic health relationship in terms of leptin.

The present investigation demonstrated that GCF leptin levels was highest in PH category (878±20 pg/ml) compared to MP category (299±46 pg/ml) which was statistically significant. Conversely, mean serum leptin concentration was highest mean in MP category (4358±44pg/ml) and least in PH category (2787±315 pg/ml) which was statistically significant showing a negative correlation with GCF and serum leptin levels in periodontal health and disease, which further fortifies reports of our previous studies2,6 that in periodontal health high leptin concentration might protect the gingiva from periodontal disease this protective effect is lost owing to decline in leptin concentration [1, 2, 6, 17].

Although the decline of leptin in periodontitis is not entirely understood, a few recent studies have addressed this conjecture. The reduced GCF leptin levels found in periodontitis could be because of (1) Expansion of the vascular network caused by vascular endothelial growth factor during gingival inflammation might increase the net rate of leptin depletion from the gingival tissues [18]. (2) Inflammation may cause cytopathic changes on endothelial cells with increasing expression of leptin receptors, which might allow more leptin–leptin receptor complex formation in the gingival tissue, resulting in decreased detectable GCF

leptin levels [17]. (3) Transition of T lymphocytes to B lymphocytes, as it is known that one source of OB is T lymphocytes [19]. (4) the high biomechanical forces in reduced periodontium due to periodontitis could inhibit and impact local leptin synthesis by PDL cells.16 (5) Periodontopathic bacteria and inflammation can cause a significant down regulation of leptin and its receptors in PDL cells [20].

The relationship and correlation of GCF and Serum leptin levels with different indicators and markers of obesity (BMI, BFM, lipid profile) were further explored in the present study. When GCF leptin levels was compared to BMI, BFM, lipid profile there was a statistically significant and positive correlation in the PH category and inversely in the MP category, the GCF leptin levels did not show statistical significance. This suggests that high serum leptin levels in obesity could partially contribute to the source of leptin in GCF in periodontally healthy individuals (transport from circulation as GCF is considered to be serum exudate) and drop in GCF leptin levels in MP individuals may be influenced by periodontal inflammatory state.

In addition, when serum leptin levels in periodontal health and disease was compared to BMI, BFM and LP there was a positive and strong correlation with BFM and no association and correlation was found with BMI despite it being considered an important indicator of obesity. This study confirms and extends similar observation on leptin reported in previous studies [21, 22]. the reason for this finding is unclear. BMI alone is considered to have limitation in predicting an individual's BF% and it often under predicts obesity [23].

The relation between serum leptin and lipid profile showed that there is a significant and positive correlation of leptin with lipid profile in periodontal health and disease. It is reported that there is a key role of leptin in control of lipid metabolism by directly stimulating the enzyme involved with fatty acid oxidation [24]. The possible mechanism through which lipid and serum leptin are inter related were not identified as it is not within the aim of the study. It is suggested that an underlying mechanism responsible for the elevation of lipid may be associated with influence of leptin. However, controversial results are also reported25 that the serum leptin and lipid profile did not show any correlation and this discrepancy may be due to methodological differences, complexity in lipid metabolism and variety in the metabolic lipid parameters.

When the serum leptin levels were related to BFM in MP and PH, we found a similar but more strong and positive correlation compared to lipid profile indicating BFM was closely related with serum leptin than BMI and LP. Although Kavazarakis *et al*. [34] showed that triglyceride values were positively correlated with the leptin levels, whereas HDL levels were inversely associated with leptin indicating that triglycerides are independently associated with leptin. The key to this relation may be that BFM is considered a better predictor of obesity as compared to BMI and LP as it takes into account the amount of body fat (true weight of adipose tissue) which is a real indicator of obesity. Previous studies in normal weight subjects revealed a similar positive correlation between leptin levels and total body fat [26, 27].

Recent meta-analysis [28] has shown that obesity is associated with MP. The exact mechanism through which periodontal disease influences the CVD has not been clearly elucidated to explain the impact of periodontitis on obesity. Preliminary investigations have reported that in periodontal patients with obesity there is elevated levels of leptin. Similarly, this raise in serum leptin is also reported in our present study and could be due to (1) Endothelial dysfunction due to inflammation. (2) As a part of immune response and host defence mechanism. (3) Spillage of leptin from periodontal tissues owing to expansion of vascular network. (4) Obesity leads to expansion of adipose tissue and leptin secretion. (5) High systemic inflammatory load due to periodontitis enhances production of pro-inflammatory cytokines and release of dysregulated secretion of adipokines such as leptin from adipose tissue.8. This raise in leptin could act and might be a critical pathomechanistic and bidirectional link in the association between MP and CVD [29, 30-33].

There are some limitations in the current study in determining the role of leptin in periodontal systemic relationship and caution must be exercised while extrapolating our results. (1) Cross sectional in nature. (2) There was no equal distribution of sex and number of patients leading to disparity among the study group. (3) Relatively small number of patients in the subcategories of the study parameters. (4) Obese patients were mostly moderately obese and more severe cases of obesity were not included. (5) Leptin regulation are still to be examined especially with respect to association between obesity and periodontitis. (6) ELISA estimates only unbound leptin (majority of leptin circulates as a bound form).

Thus, there is a clear need for future studies to overcome the above potential limitations to establish the periodontal systemic relationship in terms of leptin (1) A larger cohort study population with a well-controlled group would be needed due to high biological variants among the participants. (2) Simultaneously, compare leptin with all markers of obesity (adipokines) in concert with periodontitis (inflammatory mediators) to get a true holistic picture. (3) Evaluate, the impact of periodontal treatment on leptin levels and obesity parameters.

**Table 1:** Demographic data and grouping of subjects

| | | | |
|---|---|---|---|
| | **1.** | **Periodontal Healthy Normal Weight (NW)** | **(n =16)** |
| Group One | 2. | Periodontal Healthy Over Weight (OW) | (n =14) |
| Periodontal Healthy (PH) group | 3. | Periodontal Healthy Obese(ObW) | (n =20) |
| ( n = 109) | 4. | Periodontal Healthy Normolipidemic (NL) | (n =16) |
| | 5. | Periodontal Healthy Hyperlipidemic (HL) | (n =20) |
| | 6. | Periodontal Healthy Average Fat Mass(AFM) | (n =10) |
| | 7. | Periodontal Healthy Obese Fat Mass (OFM) | (n =13) |

| | | | |
|---|---|---|---|
| | 1. | Moderate Periodontitis Normal Weight(NW) | (n =14) |
| | 2. | Moderate Periodontitis Over Weight(OW) | (n =14) |
| Group Two | 3. | Moderate Periodontitis Obese(ObW) | (n =14) |
| Moderate Periodontitis(MP) group | 4. | Moderate Periodontitis Normolipidemic (NL) | (n =12) |
| ( n = 99) | 5. | Moderate Periodontitis Hyperlipidemic(HL) | (n =14) |
| | 6. | Moderate Periodontitis Average Fat Mass (AFM) | (n =16) |
| | 7. | Moderate Periodontitis Obese Fat Mass (OFM) | (n =15) |

**Table 2:** Clinical Parameters in sampling sites. (Mean ±SD)

| Demographic data | Periodontally Healthy (n=109) | p-value | Moderate Periodontitis (n=99) | p-value |
|---|---|---|---|---|
| MGI | 0.57±0.2 | 0.01* | 2.4±0.2 | 0.013* |
| Plaque Index | 0.48±0.25 | 0.03* | 2.2±0.4 | 0.026* |
| BOP% | 15.5±0.2 | 0.074 | 85±3.4 | 0.08 |
| PD | 1.5±0.5 | 0.023* | 8 ±1.16 | 0.042* |
| RAL | | | 12±0.8 | 0.039* |

MGI-Modified Gingival Index, BOP-Bleeding on Probing, PD-Probing Depth, RAL-Relative Attachment Level. * Statistically significant difference. *P*<0.05

**Table 3:** Results of Spearman's Rank Correlation (r) Test to compare serum and GCF leptin concentration with GI, PI, BOP, PD and RAL within the groups. Correlations (r)

| Group | Leptin | GI | p value | GCF to Serum | PI | P value | BOP | P value | PD | P value | RAL |
|---|---|---|---|---|---|---|---|---|---|---|---|
| PH N=109 | GCF Leptin R | 0.58 | 0.01* | 0.35 | 0.26 | 0.03* | 0.5 | 0.074 | 0.4 | 0.023* | |
| | Serum Leptin R | -0.4 | 0.06 | | 0.36 | 0.089 | 0.39 | 0.063 | -0.26 | 0.071 | |
| MP N=99 | Serum Leptin R | 0.9 | 0.013* | 0.4 | 0.40 | 0.026* | 0.6 | 0.08 | 0.51 | 0.042* | 0.40 (0.039*) |
| | GCF Leptin R | -0.7 | 0.06 | | 0.53 | 0.089 | - 0.44 | 0.063 | -0.45 | 0.071 | -0.26 |

GI- Gingival Index, PI- Plaque Index, BOP- Bleeding on probing, PD- Probing Depth,
RAL- Relative Attachment Level. * Statistically significant difference. *P*<0.05

**Table 4:** Comparison of GCF leptin in PH and MP using Kruskal Wallis test.

| Subgroup | N | PH Mean ±SD | Inter group 'p' value | 'p' value | N | MP Mean ±SD | Inter group 'p' value | 'p' value |
|---|---|---|---|---|---|---|---|---|
| NW | 16 | 957.30 ± 255.7 | 0.07* | 0.003 * | 14 | 288.79±21.6 | 0.43 | 0.976 |
| OW | 14 | 1150.81±141.6 | | | 14 | 243.43±10.2 | | |
| ObW | 16 | 1880.08±339.0 | | | 14 | 218.14±13.5 | | |
| NL | 15 | 356.12±34.0 | 0.02* | | 14 | 239.50±17.4 | 0.641 | |
| HL | 20 | 435.40±53.5 | | | 14 | 259.14±13.1 | | |
| AFM | 14 | 101.50±5.7 | | | 14 | 401.38±18.7 | | |
| OFM | 14 | 1341.85± 31.1 | 0.007* | | 15 | 422.13±113.0 | 0.4 | |
| Total | 109 | 878.38±204.8 | | | 99 | 299.00±46.4 | | |

NW-Normal Weight, OW- Over Weight, ObW- Obese Weight, NL- Normolipidemic, HL-Hyperlipidemic, AFM- Average Fat Mass, OFM- Obese Fat Mass. * Statistically significant difference. *P*<0.05

**Table 5:** Comparison of serum leptin in PH and MP using Kruskal Wallis tests.

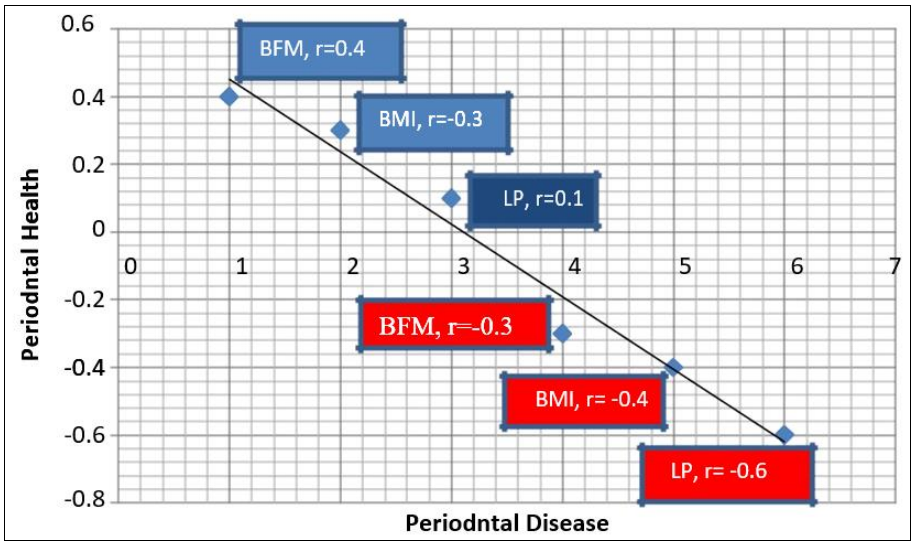
| Subgroup | N | PH Mean ± SD | Inter Group p Value | P value | N | MP Mean ± SD | Inter Grou pp Value | P value |
|---|---|---|---|---|---|---|---|---|
| NW | 16 | 2236.5 ±501.3 | 0.402 | 0.749 | 14 | 3095.01 ± 564.5 | 0.612 | 0.001 |
| OW | 14 | 2293.79 ± 179.3 | | | 14 | 3245.81 ± 252.2 | | |
| ObW | 16 | 3585.57±277.8 | | | 14 | 5119.76 ± 427.2 | | |
| NL | 15 | 1094.75±259.6 | 0.04* | | 14 | 3122.63 ± 519.6 | 0.007 * | |
| HL | 20 | 3774.43 ±395.3 | | | 14 | 4584.55 ± 434.1 | | |
| AFM | 14 | 2989.13±249.6 | 0.035* | | 14 | 4187.6 ± 407.7 | 0.04* | |
| OFM | 14 | 3539.2± 348 | | | 15 | 7157.46 ± 389.4 | | |
| Total | 109 | 2787.62±326.9 | | | 99 | 4358.97 ± 446.4 | | |

NW-Normal Weight, OW- Over Weight, ObW- Obese Weight, NL- Normolipidemic, HL-Hyperlipidemic, AFM- Average Fat Mass, OFM- Obese Fat Mass. * Statistically significant difference. *P*<0.05

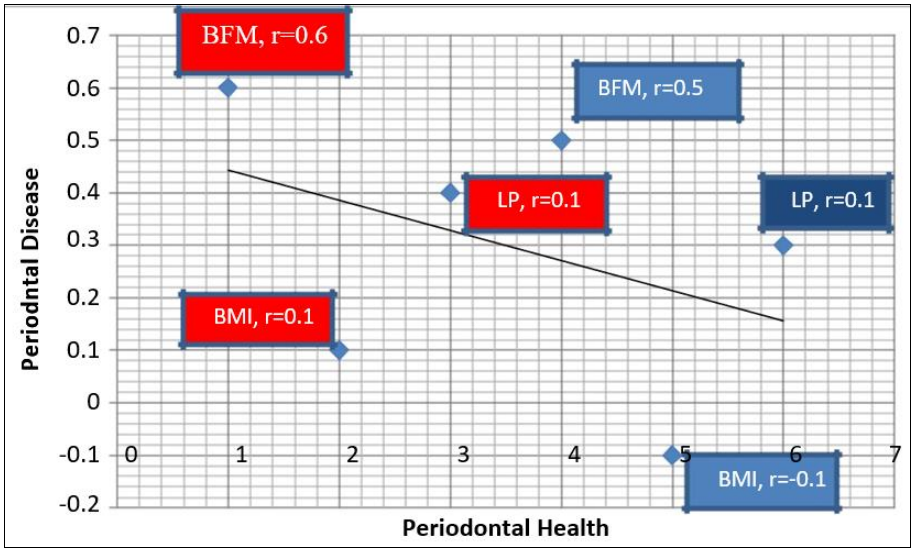
**Table 6:** Group wise comparison of serum leptin in PH and MP categories based on BMI, BFM and LP using Mann Whitney U Test.

| Subgroup | PH Mean± SD | MP Mean± SD | Mann Whitney | 'p' value |
|---|---|---|---|---|
| NW | 2236.8±564.5 | 3095.5±501.3 | 69 | 0.74 |
| OW | 2293.9±252.2 | 3245.7±179.3 | 73.5 | 0.402 |
| ObW | 3585.7±427.2 | 5119.5±277.8 | 116.5 | 0.411 |
| NL | 1094.6±519.6 | 3122.7±259.6 | 70.5 | 0.03* |
| HL | 3774.5±434.1 | 4584.4±395.3 | 125.5 | 0.612 |
| AFM | 2989.6±407.8 | 4187.1±249.6 | 98 | 0.51 |
| OFM | 3539.4±389.4 | 7157.2±343.0 | 67 | 0.004* |

NW-Normal Weight, OW- Over Weight, ObW- Obese Weight, NL- Normolipidemic, HL-Hyperlipidemic, AFM- Average Fat Mass, OFM- Obese Fat Mass. * Statistically significant difference. *P*<0.05




**Graph 1:** Correlation of GCF leptin with BMI, BFM, LP in PH and MP




**Graph 2:** Correlation of serum leptin with BMI, BFM, LP in PH and MP

## Conclusion

There was a positive correlation of GCF leptin levels in periodontal health and a negative correlation of GCF leptin in periodontal disease. Our findings suggest that there is a strong association of serum leptin with periodontitis and parameters of obesity and the possible source of leptin in GCF could be by transport from circulation. This study may be of particular clinical interest because it definitely documents the importance of the association of leptin (an atherosclerotic factor) with periodontitis and obesity parameters.

This piece of information is important that leptin is significantly correlated with LP and BFM but not BMI. Hence future studies on leptin and obesity should consider taking LP and BFM as better indicators of obesity than BMI.

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