International Journal of Oral Health Dentistry 2022;8(2):128–134

Content available at: https://www.ipinnovative.com/open-access-journals

International Journal of Oral Health Dentistry

Journal homepage: www.ijohd.org

Original Research Article

Effect of non-surgical periodontal therapy on salivary resistin and chemerin levels in type 2 diabetes mellitus with chronic periodontitis

Himanshu Aeran[1,*], Amrinder Singh Tuli[2], Rebecca Chowdhry[2], Anissa Atif Mirza[3], Raman Kumar[3], Basant Kaur Aulakh[2]

[1]*Dept. of Prosthodontics, Seema Dental College & Hospital, Rishikesh, Uttarakhand, India*
[2]*Dept. of Periodontology, Seema Dental College & Hospital, Rishikesh, Uttarakhand, India*
[3]*Dept. of Biochemistry, All India Institute of Medical Sciences, Rishikesh, Uttarakhand, India*

ARTICLE INFO

Article history:
Received 22-05-2022
Accepted 02-06-2022
Available online 16-06-2022

Keywords:
Combined oral contraception
Hormonal contraceptives
Progestin-only
Hypothalamic-pituitary-ovarian

ABSTRACT

Background: A bidirectional relationship is seen between periodontal disease and diabetes mellitus. Adipokines like resistin and chemerin act as a proinflammatory molecule. Their action is mediated by the generation of pro-inflammatory cytokines and the stimulation of inflammatory cell chemotaxis to the site of inflammation.

Aim and Objectives: This study aimed to compare the levels of resistin and chemerin in saliva in diabetes mellitus type 2 and chronic periodontitis after non-surgical periodontal therapy (NSPT).

Materials and Methods: Forty-five subjects were recruited and divided into three groups – healthy controls (Group A), chronic periodontitis (Group B) and diabetes mellitus type 2 with chronic periodontitis (Group C). Group B and C received NSPT. The parameters included were gingival index, plaque index, clinical attachment loss and probing pocket depth which were evaluated at baseline and end of three months. Levels of resistin and chemerin in saliva were evaluated using resistin and chemerin enzyme linked immunosorbent assay (ELISA) kit.

Result: At 3 months, PI, GI, PPD and CAL was reduced statistically significant in B and C groups as compared PI and GI of group A. Salivary resistin and chemerin levels were also reduced significantly in all three groups except chemerin in group A. Mean salivary resistin and chemerin levels at baseline were highest in group C followed by group B and least in group A.

Conclusion: Non-surgical periodontal therapy reduces all clinical variables, resistin and chemerin levels. Salivary resistin and chemerin levels can be used as a likely biomarker to assess the outcome following NSPT.

1. Introduction

Periodontal Disease (PD) is a chronic inflammatory disease that has been identified as the leading cause of tooth loss. Because of the related systemic inflammatory response, the effect of PD may not be restricted to the mouth cavity, but may have systemic implications.[1]

Diabetes Mellitus (DM) is demonstrated by unusually high glucose levels in the blood due to impaired secretion or action of insulin or both. Diabetes is a contributing factor in deteriorating of PD. Similarly, periodontitis is seen more in diabetics. PD is considered to be the sixth complication of DM. The failure of the body's immune system to completely destroy the source of inflammation, such as microorganisms, which causes a persistent inflammatory reaction due to systemic overexpression of

* *Corresponding author.*
E-mail address: drhimanu4@gmail.com (H. Aeran).

https://doi.org/10.18231/j.ijohd.2022.026

pro-inflammatory cytokines such interleukins (IL-1, 4, 6, 8, and 10), tumour necrosis factor (TNF-), and prostanoids (PGE2), is attributed to the coexistence of periodontitis in diabetes individuals. This persistent inflammatory response is the main cause of A two-way relationship exists between PD and DM.[2]

Adipose tissue is a metabolically active and complicated endocrine organ that secretes bioactive chemicals known as adipokines. As with periodontitis and diabetes, the interaction of pro-inflammatory and anti-inflammatory adipokines can result in a low-grade inflammatory disease.[3]

Resistin is a 12-kDa adipokine with the function of resisting insulin. Resistin expression appears to be higher in diabetes and inflammatory disorders. Resistin, a proinflammatory molecule, increases the production and secretion of pro-inflammatory cytokines (TNF-, IL-6, IL-12) and monocyte chemoattractant protein (MCP-1). Because diabetes and periodontitis are both considered inflammatory disorders, resistin may have a pathogenic function in both diseases.[4]

Chemerin is a pro-inflammatory adipocytokine generated by adipose tissue, liver, epithelial cells, endothelium, fibroblasts, and keratinocyte. Chemerin's inflammatory effect is mediated through the generation of pro-inflammatory cytokines as well as the promotion of inflammatory cell chemotaxis to the site of inflammation. It also recruits innate immune cells and functions as an antibacterial peptide against infections. Chemerin levels in saliva were found to be greater in periodontitis patients; as a result, chemerin has been recommended as a biomarker for periodontitis.[5]

Therefore, this study aims to estimate and compare salivary resistin and chemerin level in healthy, chronic periodontitis (CP) and type 2 DM with CP.

2. Materials and Methods

The ethics committee approved this study, which was conducted at the Department of Periodontology at Seema Dental College and Hospital in Rishikesh, Uttarakhand. With the consent to treatment agreement, the patients were given a full verbal and written summary of the therapy's risk and benefit.

2.1. Inclusion criteria

1. Patients of age 30-66 years including both genders.
2. Patients having probing depth of ≥4 mm in at least four sites.
3. Patients with well-controlled Type 2 diabetes (fasting plasma glucose of 126 mg/dl or 7 mmol/L and HbA1c of 6.5 percent, or 48 mmol/mol) and no additional diabetic complications.

2.2. Exclusion criteria

1. Any systemic condition that might have an impact on periodontal therapy's outcome.
2. Smokers, alcoholics & patients with other adverse habits
3. Pregnant or lactating women.
4. Patients having allergic reaction or hypersensitivity to any product used in the study.
5. Patients on antibiotics and anti-inflammatory
6. Patient who underwent any periodontal treatment within the past 6 months.

2.3. Study design

A total of 45 study participants were divided into three groups: Group A (15) were healthy people, Group B (15) were people with CP, and Group C (15) were people with type 2 diabetes with CP.

2.4. Periodontal examination

Gingival index (GI), plaque index (PI), clinical attachment loss (CAL) and probing pocket depth (PPD) were evaluated at baseline and after 3 months. A dental explorer was used to assess PI at four locations on each tooth surface. A calibrated periodontal probe was used to measure the remaining measurements.

2.5. Collection of saliva

The saliva samples were taken using the unstimulated saliva collection method. To avoid diurnal differences in saliva collection, all of the samples were obtained between 9 a.m. and 12 p.m. Participants were instructed to sit comfortably with their heads inclined forward and swallow their saliva before allowing it to flow passively for nearly 20 minutes over the lower lip into a sterile container. A total of 5 mL of saliva was collected.

2.6. Assay kit

Resistin and Chemerin (Boster Biological Technology, USA) levels were determined using a separate enzyme-linked immunosorbent assay (ELISA) kit for each marker.

2.7. Statistical analysis

The data was imported into spreadsheets and analysed with the SPSS 23.0 edition of the Social Package for Statistical System (IBM; Chicago). The difference between baseline and 3 months for groups A, B, and C was determined using a paired t test. To find intergroup differences between all of the groups, the ANOVA test was performed. The data was analysed using a p value of less than 0.05.

3. Results

3.1. Clinical variables

On intergroup comparison, there was a statistically significant difference in mean PI, GI, CAL, PPD and HbA1c score at 3 months between Group A, Group B and Group C. ($p<0.001$) (Table 1) On intragroup comparison, Group B and C showed statistically significant reduction for PI, GI. ($p<0.001$) For PPD and CAL, all groups showed statistically significant reduction. ($p<0.05$) The mean HbA1c increased after 3 months in group A. ($p<0.05$) In other two groups, the reduction in mean HbA1c was not statistically significant. ($p>0.05$) (Tables 2, 3 and 4)

3.2. Biochemical variables

Comparison of the baseline biochemical variables between the groups shows that baseline resistin and chemerin is higher in Group C and Group A respectively. Comparison of the 3-month biochemical variables between the groups shows that 3-month resistin and chemerin is higher in Group C and Group A respectively. There was non-significant difference in mean resistin and chemerin score at 3 months between Group A, Group B and Group C. (Table 1) On intragroup comparison, group B and C showed statistically significant reduction for salivary resistin and chemerin ($p<0.05$) Group A showed statistically significant reduction for salivary resistin ($p<0.05$) (Tables 2, 3 and 4)

Correlation analysis showed correlation between PPD and CAL with resistin for group A. For group B, gingival index and chemerin showed statistically significant correlation. HbA1c with chemerin showed a strong correlation for group C. (Tables 5, 6 and 7)

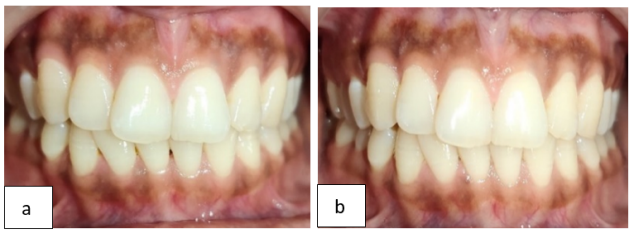

Fig. 1: Healthy patients **a:** Pre-operative (baseline); **b:** Post-operative (3 months)

4. Discussion

Periodontitis is characterised by gingival bleeding, alveolar bone loss, and attachment loss. Proinflammatory cytokines released locally in response to the bacterial plaque are likely to alter the levels of proinflammatory biomarkers. As a result, periodontitis may have a higher impact on diabetic's systemic inflammatory condition. Diabetes, on the other hand, is a well-known risk factor for PD, which causes greater periodontal tissue degradation.[6]

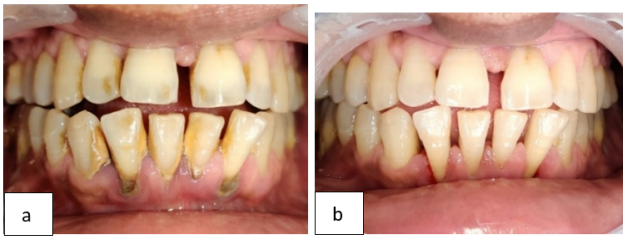

Fig. 2: CP **a:** Pre-operative (baseline); **b:** Post-operative (3 months)

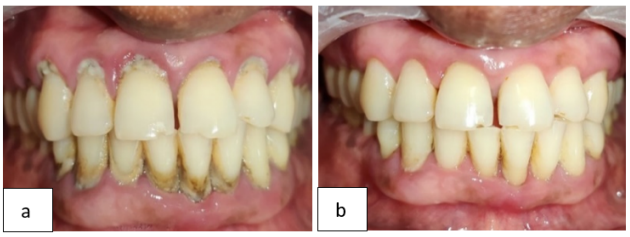

Fig. 3: CP + DM **a:** Pre-operative (baseline); **b:** Post-operative (3 months)

Adipose tissue secretes adipokines which are a collection of bioactive peptides or proteins.[7] Sgolastra et al. found that adipokines play an important role in pathogenesis of periodontitis in diabetic patients and reinforced that periodontal therapy can improve diabetes control.[8]

In the present study, mean difference in PI, GI, PPD and CAL on intragroup comparison from baseline to 3 months were highly significant for group C and B with $p<0.05$. Shehab RSA et al. showed statistical reduction in CAL after NSPT in all three groups.[9]

On intergroup comparison, mean PI for baseline and 3 months was statistically highly significant among group B and C when compared to group A ($p<0.001$) but was non-significant when comparing group B and C. On intergroup comparison, mean GI, PPD and CAL for baseline and 3 months was statistically highly significant among group B and C when compared to group A. ($p<0.001$). When diabetics with periodontitis and non-diabetics with periodontitis were compared to healthy control groups, Lahariya SN et al found a significant difference in PI, GI, PPD, and CAL.[10] Similar results were seen by Furugen R et al for PPD and CAL when they compared healthy group with periodontitis and reported a statistically significant result.[11]

The decrease in PI, GD, PPD, CAL could be due to NSPT which reduces local inflammation.[12]

In the present study, mean difference in HbA1c on intragroup comparison from baseline to 3 months significantly increased for group A with $p<0.05$. Mean difference in HbA1c on intragroup comparison from baseline to 3 months was not statistically significant for

Table 1: Intergroup comparison between clinical variables

Variable	Group	Mean±SD	ANOVA	p value
Plaque Index	Group A	0.34±0.45	20.782	0.000
	Group B	1.13±0.33		
	Group C	1.08±0.32		
Gingival Index	Group A	0.05±0.12	76.373	0.000
	Group B	1.15±0.41		
	Group C	1.12±0.54		
Probing Pocket Depth	Group A	0.66±0.31	55.605	0.000
	Group B	2.21±0.57		
	Group C	2.20±0.45		
Clinical Attachment Level	Group A	0.12±0.13	44.441	0.000
	Group B	2.30±0.79		
	Group C	2.29±1.27		
HbA1c	Group A	5.16±0.24	86.063	0.000
	Group B	5.22±0.31		
	Group C	6.98±0.63		
Salivary Resistin	Group A	4742.16±1943.51	0.472	0.627
	Group B	4141.13±1873.45		
	Group C	4169.30±1916.93		
Salivary Chemerin	Group A	82.10±40.10	2.201	0.123
	Group B	59.83±23.32		
	Group C	74.47±21.53		

Table 2: Intragroup comparison for group A

Variable	Time interval	Mean±SD	Mean difference	t value	p value
Plaque Index	Baseline	0.46±0.45	0.11±0.25	1.76	0.099
	3 Months	0.34±0.45			
Gingival Index	Baseline	0.06±0.11	0.05±0.10	0.21	0.832
	3 Months	0.05±0.12			
Probing Pocket Depth	Baseline	0.78±0.36	0.11±0.10	4.23	0.001
	3 Months	0.66±0.31			
Clinical Attachment Loss	Baseline	0.19±0.20	0.67±0.84	3.08	0.008
	3 Months	0.12±0.13			
HbA1c	Baseline	5.02±0.28	0.14±0.15	3.60	0.001
	3 Months	5.16±0.24			
Salivary Resistin (pg/nl)	Baseline	6013.45±1880.38	1271.28±946.35	5.20	<0.001
	3 Months	4742.16±1943.51			
Salivary Chemerin (pg/ml)	Baseline	122.92±114.09	40.82±10.50	1.50	0.155
	3 Months	82.10±40.10			

Table 3: Intragroup comparison for group B

Variable	Time interval	Mean±SD	Mean difference	t value	p value
Plaque Index	Baseline	1.71±0.30	0.57±0.25	8.83	0.000
	3 Months	1.13±0.33			
Gingival Index	Baseline	1.88±0.51	0.72±0.44	6.29	0.000
	3 Months	1.15±0.41			
Probing Pocket Depth	Baseline	2.91±0.54	0.69±0.41	6.51	0.000
	3 Months	2.21±0.57			
Clinical Attachment Loss	Baseline	3.12±0.91	0.81±0.43	7.22	0.000
	3 Months	2.30±0.79			
HbA1c	Baseline	5.27±0.38	0.46±0.22	0.81	0.432
	3 Months	5.22±0.31			
Salivary Resistin (pg/nl)	Baseline	6291.36±2569.24	2150.23±1724.07	4.83	0.000
	3 Months	4141.13±1873.57			
Salivary Chemerin (pg/ml)	Baseline	109.74±51.61	49.90±56.76	3.40	0.004
	3 Months	59.83±23.32			

Table 4: Intragroup comparison for group C

Variable	Time interval	Mean±SD	Mean difference	t value	p value
Plaque Index	**Baseline**	1.53±0.32	0.45±0.29	5.85	0.000
	3 Months	1.08±0.32			
Gingival Index	**Baseline**	1.61±0.70	0.49±0.34	5.48	0.000
	3 Months	1.12±0.54			
Probing Pocket Depth	**Baseline**	2.83±0.53	0.62±0.44	5.49	0.000
	3 Months	2.20±0.45			
Clinical Attachment Loss	**Baseline**	3.60±1.18	0.67±0.84	7.43	0.000
	3 Months	2.29±1.27			
HbA1c	**Baseline**	7.12± 0.73	1.40±0.34	1.57	0.137
	3 Months	6.98±0.63			
Salivary Resistin (pg/nl)	**Baseline**	6906.94±1473.91	2737.64±1854.64	5.71	0.000
	3 Months	4169.30±1916.93			
Salivary Chemerin (pg/ml)	**Baseline**	274.12±330.25	199.64±324.88	2.38	0.032
	3 Months	74.47±21.53			

Table 5: Correlation between clinical variables and salivary resistin and chemerin for group A

	Resistin		**Chemerin**	
Variables	**Correlation coefficient**	**p value**	**Correlation coefficient**	**p value**
PI	0.353	0.196	-0.386	0.156
GI	0.275	0.321	-0.335	0.223
PPD	0.629	0.012	-0.440	0.101
CAL	0.564	0.029	-0.177	0.529
HbA1c	-0.499	0.058	-0.168	0.550

Table 6: Correlation between clinical variables and salivary resistin and chemerin for group B

	Resistin		**Chemerin**	
Variables	**Correlation coefficient**	**p value**	**Correlation coefficient**	**p value**
PI	0.046	0.869	0.318	0.248
GI	0.243	0.383	0.632	**0.011**
PPD	0.257	0.355	0.082	0.771
CAL	0.075	0.791	0.154	0.585
HbA1c	0.167	0.553	-0.319	0.247

Table 7: Correlation between clinical variables and salivary resistin and chemerin for group C

	Resistin		**Chemerin**	
Variables	**Correlation coefficient**	**p value**	**Correlation coefficient**	**p value**
PI	-0.381	0.162	0.014	0.960
GI	-0.377	0.166	0.011	0.970
PPD	0.093	0.742	0.136	0.630
CAL	-0.209	0.454	0.046	0.869
HbA1c	0.513	0.050	-0.638	0.011

group B and C. Ghalwash DMfound a reduction in HbA1c in diabetic periodontitis group post-treatment which was statistically significant.[13]

On intergroup comparison, mean HbA1c for baseline and 3 months was statistically highly significant among group A and group B when compared to group C. ($p<0.001$). Similar findings were reported by Weigert J et al, they found a statistically significant difference between T2DM and healthy normal weight subjects at baseline.[14]

In the present study, mean difference in resistin level on intragroup comparison from baseline to 3 months were highly statistically significant for all the groups with $p<0.001$. Similar results were seen by Furugen R et al., they compared healthy group with periodontitis at baseline and reported a statistically significant result.[11]

On intergroup comparison, mean resistin level for baseline and 3 months was not statistically significant among group A, B and C. Hiroshima Y et al found elevated levels of GCF resistin in CP and diabetic periodontitis

patients as compared to healthy individuals.[15] On the contrary, a statistically significant difference in resistin level was seen between healthy and periodontitis group in a study by Saito et al.[16] Similar results were seen in a study conducted by Weigert J et al. when comparing T2DM and healthy normal weight.[14]

In the present study, mean difference in chemerin level on intragroup comparison from baseline to 3 months were highly statistically significant for all the groups with $p<0.001$. In a study by Ziaei et al, similar reduction in salivary chemerin levels was seen for diabetic periodontitis group.[6]

On intergroup comparison, mean chemerin level for baseline and 3 months was not statistically significant among group A, B and C. Similar results were published by Godlewska U et al., they compared healthy group with periodontitis and a non-significant result was obtained.[17] Contrary to our findings, El-Mesallamy et al found difference between control and type 2 DM at baseline for serum chemerin level to be statistically significant.[18]

In a study conducted by Akram Z et al., none of the clinical periodontal factors were found to be linked with changes in salivary resistin levels.[12] Ghalwash DM et al. discovered a direct link between serum visfatin and chemerin levels and the percent change in serum glycosylated haemoglobin A1c.[13] Devanoorkar A et al. discovered a positive link between serum resistin levels and PI, GI, BI, PPD, but a negative correlation with CAL, which contradicted the findings of the current investigation.[19]

Changes in periodontal variables did not correspond with changes in salivary resistin levels following NSPT, according to our findings. It's possible that this result is due to the fact that only shallow and moderately deep periodontal pockets were chosen. Salivary resistin levels have been studied in several research, although only in individuals with widespread and progressing illness.[12]

Because periodontitis is a chronic inflammatory disease that requires therapy to resolve, as well as a systemic inflammatory state caused by hyperglycemia, the levels for resistin and chemerin have decreased following NSPT.[9]The fact that resistin is primarily produced by polymorphonuclear leukocytes and macrophages in inflammatory circumstances, including periodontitis, might explain the rise in these adipokines in patients with periodontitis. Human resistin enhances the synthesis and secretion of TNF-α and IL-12, resulting in a positive feedback cycle that induces its own creation. Second, potential pathogens such as Porphyromonas gingivalis produce resistin from neutrophils in response to a lipopolysaccharide stimulation.[20] However, because the concentration of this adipokine in saliva is lower than in serum, the alterations are minor.[16] The reduction in parameters and salivary adipokine levels implies that periodontal inflammation and insulin resistance may be linked.[10]

5. Conclusion

The study results concluded that, salivary resistin and chemerin levels are higher in diabetic periodontitis subjects followed by periodontitis subjects and least in healthy subjects. Non-surgical periodontal therapy reduces all variables, resistin and chemerin levels. Salivary resistin and chemerin levels can be used as probable biomarker to assess the outcome following NSPT.

6. Source of Funding

None.

7. Conflict of Interest

The authors declare no conflict of interest.

References

1. Suhail AL, Esther NH, Kumar AP, Devi RR. Salivary biomarkers of periodontal disease - the ultimate diagnostic tool. *Int J Recent Sci Res*. 2018;9(4):25927–32.
2. Amr E, Mostafa R, Shaker O. Possible role of gingival crevicular fluid levels of chemerin and fibroblast growth factor 21 as biomarkers of periodontal disease in diabetic and non-diabetic patients. A diagnostic accuracy study. *Adv Dent J*. 2019;1(2):52–63.
3. Conde J, Scotece CM, Gomez R, Lopez V, Gomez-Reino JJ, Lago F, et al. Adipokines: Biofactors from White adipose tissue. A complex hub array inflammation, metabolism and immunity. *Biofactors*. 2011;37(6):413–20.
4. Steppan CM, Bailey ST, Bhat S, Brown EJ, Banerjee RR, Wright CM, et al. The hormone resistin links obesity to diabetes. *Nature*. 2001;409(6818):307–12.
5. Roman AA, Parlee SD, Sinal CJ. Chemerin: A potential endocrine link between obesity and Type II Diabetes Mellitus. *Endocrine*. 2012;42:243–51.
6. Ziaei N, Golmohammadi S, Ataee M, Ardalani F, Abbasi MM. Effect of non-surgical treatment on three salivary adipokines in diabetic patients with periodontitis. *J Dent Res Dent Clin Dent Prospects*. 2020;14(3):199–205.
7. Qasim QA, Fareed YY, Jabir MA, Sahib HB. Evaluation of chemerin and apelin adipokines in obese and non-obese type 2 diabetes mellitus. *Int J Pharm Sci Rev Res*. 2016;40(1):228–33.
8. Sgolastra F, Severino M, Pietropaoli D, Gatto R, Monaco A. Effectiveness of periodontal treatment to improve metabolic control in patients with chronic periodontitis and type 2 diabetes: A meta-analysis of randomized clinical trials. *J Periodontol*. 2013;84(7):958–73.
9. Shehab RSA, Shoriebah EA, Mansour MS. Evaluation of resistin levels in gingival crevicular fluids of patients with periodontitis and type 2 diabetes mellitus after non-surgical periodontal therapy and subantimicrobial dosage of doxycycline. *Al-Azhar Dent J Girls*. 2020;7(1):49–55.
10. Lahariya SN, Sarkar PD, Dwivedi S. Role of adipokines in periodontal disease with diabetes mellitus and without diabetes mellitus. *Int J Biol Med Res*. 2015;6(3):5078–81.
11. Furugen R, Hayashida H, Yamaguchi N, Yoshihara A, Ogawa H, Miyazaki H, et al. The relationship between periodontal condition and serum levels of resistin and adiponectin in elderly Japanese. *J Periodontal Res*. 2008;43(5):556–62.
12. Akram Z, Baharuddin NA, Vaithilingam RD, Rahim ZHA, Chinna K, Krishna VG, et al. Effect of nonsurgical periodontal treatment on clinical periodontal variables and salivary resistin levels in obese Asians. *J Oral Sci*. 2017;59(1):93–102.

13. Ghalwash DM, Shaker OG, Mahmoud EA. Local and systemic level of adipokines as markers of inflammation in periodontitis and type 2 diabetes mellitus after periodontal therapy. *Tanta Dent J*. 2019;16(2):104–14.
14. Weigert J, Neumeier M, Wanninger J, Filarsky M, Bauer S, Wiest R, et al. Systemic chemerin is related to inflammation rather than obesity in type 2 diabetes. *Clin Endocrinol (Oxf)*. 2010;72(3):342–8.
15. Hiroshima Y, Bando M, Inagaki Y, Mihara C, Katoka M, Murata H. Resisitn in gingival crevicular fluid and induction of resistin release by Porphyromonas gingivalis lipopolysaccharide in human neutrophils. *J Periodontal Res*. 2012;47(5):554–62.
16. Saito T, Yamaguchi N, Shimazaki Y, Hayashida H, Yonemoto K, Doi Y. Serum levels of resistin and adiponectin in women with periodontitis: the Hisayama study. *J Dent Res*. 2008;87(4):319–22.
17. Godlewska U, Brzoza P, Sroka A, Majewski P, Jentsch H, Eckert M, et al. Antimicrobial and attractant roles for chemerin in the oral cavity during inflammatory gum disease. *Front Immunol*. 2017;8:353. doi:10.1111/j.1365-2362.1990.tb01877.x.
18. El-Mesallamy HO, El-Derany MO, Hamdy NM. Serum omentin-1 and chemerin levels are interrelated in patients with Type 2 diabetes mellitus with or without ischaemic heart disease. *Diabet Med*. 2011;28(10):1194–200.
19. Devanoorkar A, Dwarakanath CD, Gundanavar G, Kathariya R, Patil SR. Evaluation of serum resistin levels in periodontal health and disease and effects of nonsurgical periodontal therapy on its levels. *Dis Markers*. 2012;32(5):289–94.
20. Gokhale NH, Acharya A, Patil VS, Trivedi DJ, Setty S, Thakur SL. Resistin levels in gingival crevicular fluid of patients with chronic periodontitis and type 2 diabetes mellitus. *J Periodontol*. 2014;85(4):610–7.

Author biography

Himanshu Aeran, Director Principal, Professor and Head https://orcid.org/0000-0002-7723-7108

Amrinder Singh Tuli, Professor and Head

Rebecca Chowdhry, Reader

Anissa Atif Mirza, Professor and Head

Raman Kumar, Assistant Professor

Basant Kaur Aulakh, Postgraduate Student

Cite this article: Aeran H, Tuli AS, Chowdhry R, Mirza AA, Kumar R, Aulakh BK. Effect of non-surgical periodontal therapy on salivary resistin and chemerin levels in type 2 diabetes mellitus with chronic periodontitis. *Int J Oral Health Dent* 2022;8(2):128-134.