Comparison of levels of Oncostatin M cytokine in saliva and serum in periodontitis – a clinico-biochemical study

RUNNING TITLE: Salivary and serum levels of Oncostatin M in periodontitis

Shweta Sonkusle*, MDS; Varsha Singh*, MDS

*Department of Periodontics, PMNM Dental College and Hospital, Karnataka, India

Corresponding author: Dr. Shweta Sonkusle

Department of Periodontics

PMNM Dental College and Hospital

Navanagar, Bagalkot—587103

Karnataka

INDIA

shwetasonkusle@gmail.com

ABSTRACT

Background: Oncostatin M (OSM), a 28 kDa gp130 pleiotropic cytokine belonging to the Interleukin-6 family is mainly produced by neutrophils, activated T cells, monocytes, and macrophages. In response to tissue injury, it may stimulate the production of IL-6 alone or act synergistically with IL-6 or TNF-α contributing to the inflammatory cycle. Periodontitis is an inflammatory disease resulting from a dysbiosis bacterial community which has a strong association with increased OSM production. **Objectives**: To investigate and compare the levels of saliva and serum OSM in periodontally healthy and periodontitis subjects. **Materials and methods:** 88 individuals between the ages of 18 and 60 years were divided into two groups of 44: a periodontally healthy group (Group A) and a periodontitis group (Group B) based on clinical examination and radiographic analysis. Evaluation of salivary and serum Oncostatin M (OSM) by Enzyme Linked Immunosorbent Assay (ELISA) was performed in both groups. **Results**: There was a significant difference between Groups A and B ($p<0.01$) OSM levels, with higher values in Group-B. There was a positive correlation between the clinical parameters and OSM levels indicating that OSM has a significant role in modulating the inflammatory response of periodontal tissues. **Conclusion:** The expression of cytokine OSM may have a potential role in the immunopathogenesis of periodontitis suggesting a role as an inflammatory diagnostic marker.

KEYWORDS: Oncostatin M, cytokine, biomarker, saliva, periodontitis, interleukin-6, serum

CDHA RESEARCH AGENDA CATEGORY: risk assessment and management

INTRODUCTION

Periodontal diseases are chronic inflammatory diseases activated in response to a dysbiosis of the oral microbiome resulting in significantly higher levels of periodontopathogens. The dysbiotic oral microbiome in dental plaque triggers the local host response by stimulating the inflammatory cells including neutrophils, lymphocytes, and macrophages.[1]

Oncostatin M (OSM), a 28 kDa glycoprotein primarily secreted by activated T-lymphocytes, neutrophils, dendritic cells, and macrophages, is a member of the IL-6 family which comprises IL-6, IL-11, OSM, leukaemia inhibitory factor (LIF), ciliary neurotrophic factor (CNTF), and cardiotrophin-1 (CT-1)[2]. Similar to IL-6, OSM is pleiotropic and participates in diversified physiological processes such as wound healing, inflammatory response and cellular proliferation and differentiation[2,3] in chronic inflammatory conditions including rheumatoid arthritis, chronic obstructive sialadenitis, atherosclerosis, and cardiovascular diseases.[4-10] It was biochemically demonstrated on the basis of its antiproliferative activity on the A375 human melanoma cell line *in vitro* and a potential function in the regulation of cancer was discovered.[11]

In the event of inflammation, OSM is considered double-edged, acting both as anti-inflammatory and proinflammatory cytokine.[12] It illustrates its pro-inflammatory effects by stimulating chemokine production by endothelial cells and inducing chemotaxis and adhesion of neutrophils.[13] It causes periodontal tissue destruction by augmenting RANKL production by upregulating or acting synergistically with IL-6 or TNF-α, activating osteoclastic alveolar bone resorption.[14]

Thorat et al[15] and Lin et al[16] reported a significant increase in the OSM levels in serum and GCF with increased destruction of periodontal tissues and a significant decrease following non-

surgical periodontal therapy (NSPT) in chronic periodontitis patients. This suggests that OSM plays an enigmatic role in periodontal health and disease.

With the aim of scrutinizing the local host response to a bacterial challenge, investigation of cytokine production levels has been demonstrated. Saliva, being a biological fluid, reflects various systemic changes in the body by mirroring the general health of the individual. This makes it an important diagnostic tool.[17] Increased serum concentration of OSM in chronic periodontitis subjects may promote metalloproteinase and chemokine production increasing the risk of systemic inflammatory conditions like rheumatoid arthritis.[15]

Further studies are required to add to the existing literature linking Oncostatin M production in serum and saliva to periodontitis. Therefore, in our study we investigated and evaluated the expression of salivary and serum OSM in patients with periodontitis and in healthy individuals.

The null hypothesis was that there would be no significant difference in the mean OSM levels in different subjects with periodontitis and healthy subjects.

MATERIAL AND METHODS

A total of 135 patients aged 25 to 60 years were screened at the Outpatient Department of Periodontology, P. M. N. M. Dental College and Hospital, Navanagar, Bagalkot, Karnataka, India. Eighty-eight patients were enrolled and divided into healthy and periodontitis groups based on their periodontal exam results.

Sample size was determined using the mean and standard deviation values from literature using the formula

$$n = \frac{2 (Z_\alpha + Z_\beta)^2 [s]^2}{d^2}$$

where Z_α is the z variate of alpha error i.e. a constant with value 1.96, Z_β is the z variate of beta error i.e. a constant with value 0.84.

Approximate estimates:

1. 80% power
2. Type I error to be 5%
3. Type II error to be 20%
4. True difference of at least 50 units between the groups
5. Pooled standard deviation of 59

Substituting the values,

$$n = \frac{2\,(2.8)^2\,[\,59\,]^2}{(\,50\,)^2}$$

n = 21.83

A minimum of 22 subjects / patients per group should complete the trial.

The sample size was increased to 44 subjects per group to decrease the margin of error.

All potential participants received clear explanations regarding the need and design of the study. A written duly signed informed consent was obtained from all the subjects and the study was approved by Institutional Ethical Committee of PMNM Dental College and Hospital, Bagalkot **(PMNMDCH/1586/2019-20)** with the Helsinki Declaration of 1975(revised in 2000)**.** Saliva and serum samples were collected from all subjects. Inclusion criteria encompassed systemically healthy individuals in the age range of 25-60 years having at least 20 natural teeth. Individuals with systemic diseases that could influence periodontal conditions; those who had undergone periodontal therapy in 6 months prior to the investigation; those on any systemic antibiotics, anti-inflammatory or hormonal/corticosteroid therapy within the

previous 3 months; those consuming tobacco; pregnant and lactating mothers; or those having other infections or pathology in the oral cavity other than periodontitis were excluded from the study.

Based on the clinical parameters including Gingival Index (GI) (Loe & Silness)[18], Plaque Index (PI) (Silness & Loe)[19], Bleeding on Probing (BOP) (Muhlemann & Son[20], Pocket Probing Depth (PPD), Clinical Attachment Loss (CAL); and radiographic analysis, the subjects were divided into 2 groups in accordance with the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions.[21,22]

Healthy Group (A) (n=44): Subjects with absence of bleeding on probing (BOP<10%), probing depth (PD) ≤ 3mm, no clinical attachment loss (CAL), no radiographic bone loss, no sign of other inflammatory lesions in the oral mucosa.[21]

Periodontitis Group (B) (n=44): Subjects with presence of interdental CAL ≥ 5mm, PD ≥ 6mm and radiographic bone loss extending to 2/3 of the root or beyond. Subjects had lost no more than 4 teeth due to periodontitis. Evaluation of radiographic bone loss/age was done using the tooth showing the most severe bone loss as a percentage of root length. Since all the subjects had the % bone loss/age in the range of 0.25-1. Therefore, all of them were considered to be in Stage III Grade B Periodontitis.[22]

A proforma was designed to record all systematic and methodical information by clinical observations. All the patients were scrutinized clinically and biochemically.

Specimen collection

Saliva and blood samples were collected from the same subjects for biochemical analysis. Subjects were refrained from drinking or eating anything for two hours prior to sample

collection. Non-stimulated whole expectorated saliva(~3ml) was collected from each subject into sterile tubes.[23] Saliva samples were cleared by centrifugation at 9300x g for 5 minutes and the supernatant was kept at -70° C and transferred to laboratory for evaluation of saliva Oncostatin M by enzyme linked immunosorbent assay (ELISA).[24]

Two (2) mLs of blood were aseptically collected from the antecubital vein of each subject and placed into plain tubes[25], rested at room temperature for 30 minutes, and then centrifuged at 3000x g for 5 minutes. The extracted serum was transferred into clean eppendorf tubes and stored at -70° C until the time of the assay.[25]

OSM assay

The saliva and serum samples were analysed using Enzyme-Linked Immunosorbent Assay (ELISA) obtained from Everon Life Sciences, New Delhi, India (**Catalogue No. E1663Hu).** The samples were analysed at Maratha Mandal's Nathajirao G. Halgekar's Institute of Dental Science & Research Center, Belagavi.

All reagents, standard solutions and samples were prepared as per the manufacturer's instructions. All reagents were brought to room temperature before use. The assay was performed at room temperature. 50µl of standard solution was added to the standard well, 40µl of the sample was added to each sample wells along with 10µl anti-OSM antibody. Then 50µl streptavidin-HRP was added to both sample and standard wells and thoroughly mixed. The plate was covered with a sealer and then it was incubated for 60 minutes at 37°C. The sealer was removed, and the plate was washed 5 times with wash buffer. 50µl substrate solution A was added to each well followed by 50µl substrate solution B. The plate was covered with a new sealer and incubated for 10 minutes at 37°C in the dark. 50µl Stop Solution was added to each well, the blue color changed into yellow immediately. The optical density (OD value) of

each well was determined immediately using a microplate reader set to 450 nm within 10 minutes after adding the stop solution.

The readings for each standard, control, and sample were duplicated and the average zero standard optical density (O.D.) was subtracted. A standard curve was used to test the concentration of OSM in the samples.[15,16]

Statistical analysis

Data were subjected to statistical analysis using Statistical package for social sciences (SPSS v 26.0, IBM). Normality of numerical data was checked using Shapiro-Wilk test. Parametric tests have been used where data followed a normal curve. A paired t-test was used for intergroup comparison of age whereas chi-square test was used for intergroup comparison of gender. Non-parametric tests have been used for comparisons where the data did not follow a normal curve; hence pairwise comparison for clinical parameters and serum and salivary concentration of OSM was done using Mann Whitney U test. Spearman's correlation test was done to evaluate correlation of clinical parameters and salivary and serum concentrations of OSM.

RESULTS

The study involved 88 participants divided into 2 groups of 44; healthy (Group A) and Periodontitis (Group B). The mean age of patients for Group A was 28.20±3.57years and Group B was 42.16±7.45 years.

There was a statistically significant difference found for the values between the groups ($p<0.01$) for GI, PI, BOP, PPD, CAL, saliva, and serum OSM levels with higher values in the Periodontitis group. The mean salivary levels of OSM in Group A and B were 1337.87±972.06 ng/L and 5146.01±2681.04 ng/L, respectively. The mean serum Oncostatin M levels were

729.97±505.21 ng/L in Group A and 5049.82±2152.89 ng/L in Group B. (TABLE 1) (Graph 1,2,3)

Spearman's correlation coefficient test was carried out to check the bivariate correlation between the clinical parameters, and salivary and serum Oncostatin M levels in both groups.

There was a statistically positive correlation between saliva OSM levels & serum OSM levels and the clinical parameters in the periodontitis group (Group B) ($r=0.375$, $p<0.05$) whereas a statistically non-significant correlation was observed in the Healthy group (Group A) (TABLE 2 & 3).

DISCUSSION

Oncostatin M (OSM), a member of the interleukin-6 (IL-6) cytokine family, signals cancer cells predominantly through STAT3 which has effects on tumour cell proliferation, invasion, attachment to substratum, epithelial-to-mesenchymal transition (EMT) and migration promoting a metastatic phenotype proliferation. In addition to this, OSM has been reported to drive other pathological conditions including arthritis, insulin resistance, Alzheimer's disease and atherosclerosis.[26,27]

In periodontitis, Oncostatin M is secreted by the local inflammatory cells that stimulate IL-6. The presence of pro-inflammatory, inflammatory and bone resorption markers in the GCF and serum of the patients has been proposed to be a diagnostic and prognostic marker.[15,27] Neutrophil recruitment at sites with low OSM concentration have been observed after periodontal therapy which increases the risk for relapse.[28]

In this present study, significantly higher levels of mean salivary Oncostatin M levels were found in the chronic periodontitis group when compared to the healthy group, which are

similar to results obtained by **Lin SJ et al [16], Thorat MS et al [15], Jones et al [29] and Aydin T et al[30],** who found that patients with periodontitis had a higher prevalence of GCF and salivary OSM levels than patients without periodontitis. This may be attributed to autocrine and paracrine RANKL regulation by IL-6 activated by OSM which acts on both osteoblastic and osteoclastic cells ultimately causing periodontal tissue destruction. It was reported by **Hosokawa et al[31]** that OSM may induce CXCL10 and ICAM-1 expression in human gingival fibroblasts which promote Th1 cell infiltration and retention in diseased tissues. This is in contrast with a study conducted by **Becerik et al[32]** where they investigated the levels of OSM in GCF and plasma in patients with gingivitis, CP and aggressive periodontitis and found no correlation between the levels of OSM in GCF and plasma in all these periodontal diseases.

Mean values of serum Oncostatin M levels in CP were statistically significantly higher when compared to the healthy group in this present study which is similar to the results obtained by **Pradeep AR et al [25]**, **Thorat MS et al [15] and Khan F et al[33]** who found that the levels of OSM in GCF and serum increased significantly with severity of periodontal disease which could be attributed to the possible spillover from the diseased periodontal tissues to peripheral tissues or a systemic inflammatory response to progressive disease in the periodontal pocket.[20]

The positive correlation between the clinical parameters and Oncostatin M levels indicates a significant role of OSM in modulating the inflammatory response of periodontal tissues. This is in accordance with the study done by **Lin SJ et al [16], Thorat MS et al [15] and Pradeep AR et al [25]** where they found that the increased levels of OSM in GCF and serum were positively correlated to the severity of chronic periodontitis. However, this is in contrast with study findings from **Becerik S et al [32]** who reported no significant correlation between Oncostatin M levels and clinical parameters.

Interestingly, a two-way relationship has been observed in other studies where increased OSM levels in periodontal tissues may lead to the development or exaggeration of rheumatoid arthritis by modulation of chemokine and metalloproteinase production by synovial cells of the joints. Increased serum concentration of OSM may instigate neutrophil chemotaxis leading to exaggerated periodontal tissue destruction.[25,34,35] This suggests a potential role of OSM in local tissue response to inflammation and subsequent systemic complications.

However, in a recent study, **Kolluri et al [36]** established that the concentration of OSM rises during inflammatory changes seen in the local tissue environment, irrespective of diabetic status. This may be attributed to the periodontal pathogens in the subgingival environment which stimulate OSM release from neutrophils and macrophages by degranulation and de novo synthesis.[36]

A finite number of studies demonstrate the significance of OSM in the periodontal environment.[9,15,16,29-33,36] The present research elucidated the elevated level of OSM in saliva and serum in periodontitis, which adds to the existing data justifying the local as well as systemic effects of OSM as an immune modulator.

Limitations and future perspective

One limitation of this present study is the cross-sectional design which prevented the establishment of a temporal association and lack of measurement of other differences such as race and population variations, which does not allow generalization of these findings to other population. Therefore, further studies focusing on OSM levels in different stages of periodontal and peri-implant diseases and a multi-center level study including populations of various races are required to consolidate the findings of the present study. Futuristic studies on the correlation

of Oncostatin M with osteo-arthritis and other inflammatory diseases that might be determined in saliva, could contribute to the importance of diagnosing periodontal diseases.

CONCLUSION

Within the limits of this study, these findings confirmed a positive correlation between periodontal disease and the presence of OSM compared with healthy tissues. Given this finding, OSM could potentially be used as a diagnostic marker. Further studies aimed at examining the role of Oncostatin M in periodontal disease progression and peri-implant diseases should be conducted.

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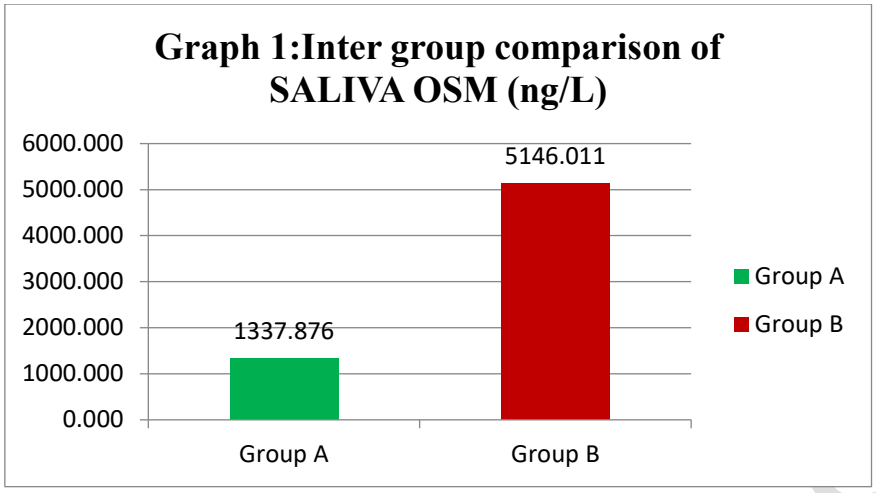
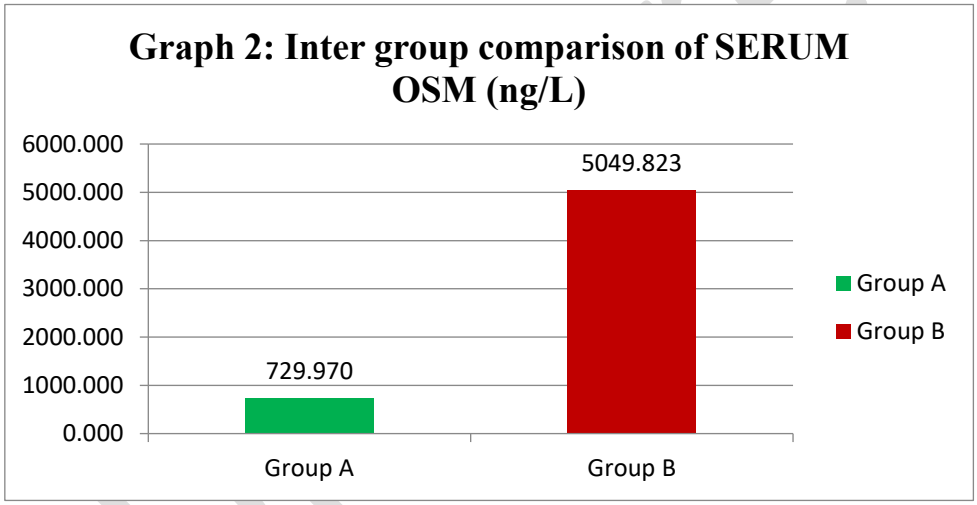
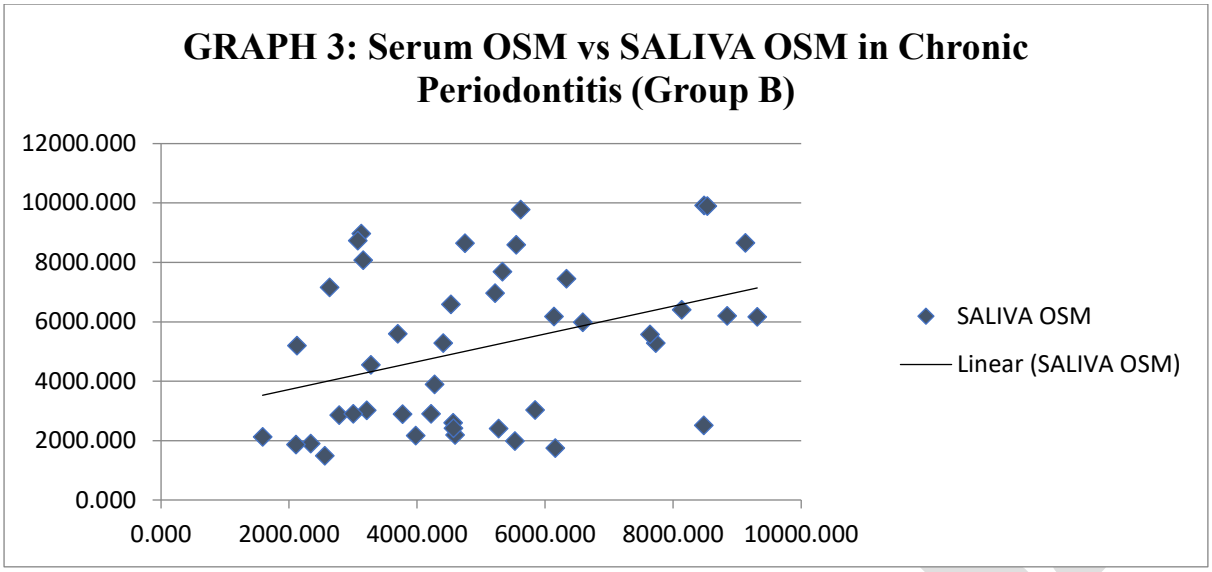


Group A – Healthy, Group B- Chronic Periodontitis, Mean Oncostatin M levels were 1337.87±972.06 in healthy group, 5146.01±2681.04 in CP group. ($p<0.01$- statistically significant).



Group A – Healthy, Group B- Chronic Periodontitis, Mean Oncostatin M levels were 729.97±505.21 in healthy group and 5049.82±2152.89 in CP group. ($p<0.01$- statistically significant).



Statistically significant and positive corelation was seen between serum OSM and Saliva OSM values in chronic periodontitis.

Table 1: Descriptive statistics of the study population (mean±standard deviation)

Parameters	GROUP A	GROUP B	P
Age (years)	28.20±3.57	42.16±7.45	.000**
GI	0.34±0.20	2.40±0.30	.000**
PI	0.38±0.21	2.71±0.31	.000**
BOP	0.93±0.39	3.70±0.61	.000**
PPD	0	4.72±1.29	.000**
CAL	0	5.24±1.61	.000**
SALIVARY OSM LEVELS	1337.87±972.06	5146.01±2681.04	.000**
SERUM OSM LEVELS	729.97±505.21	5049.82±2152.89	.000**

**Highly statistically significant. GI=Gingival index; Pl=Plaque index; BOP= Bleeding on probing; PPD= probing pocket depth; CAL=Clinical attachment level; OSM=Oncostatin M.

Table 2: Spearman's correlation coefficient test comparing the levels of oncostatin M in saliva and serum to clinical parameters (GI, PI, BOP, PPD, CAL) among both the groups

OSM levels	GROUPS	GI	PI	BOP	PPD	CAL
SALIVA	GROUP A	0.046	-.121	-.031	-	-
	GROUP B	-.037	.036	-.055	.080	.062
SERUM	GROUP A	.136	-.027	.212	-	-
	GROUP B	-.052	.076	.186	.289	.158

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GI=Gingival index; Pl=Plaque index; BOP= Bleeding on probing; PPD= probing pocket depth; CAL=Clinical attachment level; OSM=Oncostatin M.

Table 3: Correlation between Oncostatin M levels in saliva and serum using Spearman's correlation coefficient test

GROUPS	Correlation coefficient	Sig. (2-tailed)
GROUP A	.091	.557
GROUP B	.375*	0.12*

Group A – Healthy, Group B- Chronic Periodontitis (*– statistically significant)