"ESTIMATION OF SALIVARY RESISTIN LEVELS IN INDIVIDUALS WITH HEALTHY PERIODONTIUM AND PERIODONTITIS"

By

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LIST OF ABBREVIATIONS

GCF	Gingival Crevicular Fluid
RTN	Resistin
MMP-9	Matrixmetalloproteinase-9
MMP-8	Matrixmetalloproteinase-8
IL-1β	Interleukin 1β
IL-6	Interleukin 6
LPS	Lipopolysaccharides
TNF-α	Tumour necrosis factor-α
TZD	Thiazolidinediones
FIZZ	Found in Inflammatory Zones
RELMs	Resistin-like molecules
kDa	Kilodalton
PBMC	Peripheral blood mononuclear cells
mRNA	Messenger RNA
PD	Pocket depth
CAL	Clinical attachment level

RBL %	Radiographic Bone loss %
OHI-S	Oral hygiene index simplified
BMI	Body Mass Index
T2DM	Type 2 Diabetes Mellitus
ELISA	Enzyme-linked immunosorbent assay
OD	Optical Density
G-AgP	Generalized aggressive periodontitis
CP	chronic periodontitis

ABSTRACT

INTRODUCTION

Research is currently underway to explore saliva as a noninvasive method for detecting biomarkers in the early monitoring of both oral and systemic diseases, with a specific emphasis on identifying the initial stages of periodontal tissue damage. Clinicians have long sought to establish connections between periodontal diseases and various systemic conditions by exploring different biological markers. Resistin has emerged as a significant marker which plays a vital role in various biological processes, especially inflammation. Initially recognized as an adipose-secreted hormone associated with obesity and insulin resistance in rodents, human resistin has been implicated in inflammation-related conditions, including periodontitis. Given its involvement in periodontal inflammation, resistin holds promise as a biomarker for identifying individuals at risk of developing periodontitis. Studies on resistin levels in periodontal health and disease have yielded inconclusive results.

AIM

Estimation of salivary Resistin levels in individuals with healthy periodontium and Periodontitis.

MATERIALS AND METHODS

A cross-sectional study was conducted on a total of 78 systemically healthy subjects who were divided into three groups - Group A: periodontally healthy subjects, Group B: subjects with moderate periodontitis and Group C: subjects with severe periodontitis, as per the proposed criteria for classifying periodontitis in the

2017 World Workshop. The saliva samples were collected from all the subjects and resistin values were evaluated using ELISA. Statistical analysis was done using One-way ANOVA, post hoc test and Karl Pearson's correlation coefficient ($p<0.05$).

RESULTS

The mean Resistin levels (ng/ml) in periodontal health, moderate periodontitis and severe periodontitis were 2.43 ± 1.92, 8.18 ± 1.02, and 11.47 ± 2.19, respectively. The correlation between resistin levels and PD, CAL, RBL%, and OHI-S scores, as determined by Karl Pearson's correlation coefficient, showed a statistically significant association, with a p-value of 0.0001.

CONCLUSION

Resistin was detected in the saliva samples of all the groups, with levels increasing progressively from periodontal health to moderate and severe periodontitis. An association was observed between resistin and the periodontal parameters like PD, CAL, RBL%, and OHI-S, suggesting that the levels of resistin increased with the severity of the periodontal disease. Hence, it may serve as a valuable diagnostic biomarker for detecting periodontal disease.

KEYWORDS: Biomarkers, Resistin, periodontitis, saliva, salivary biomarkers

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INTRODUCTION

Periodontitis encompasses a set of inflammatory conditions impacting the attachment of surrounding tissue and the alveolar bone around teeth. While periodontopathogenic microorganisms initiate periodontitis, the host's reaction to this infection has a key role in the disease's advancement. If untreated, the disease progresses, causing gradual bone loss, tooth mobility, and eventual tooth loss.[1]

Hence, early detection of such infections is beneficial in order to maintain the balance of a dynamic oral environment. The objective of periodontal diagnosis and its related methods is to furnish the periodontitist with valuable insights into the severity of the condition. Treatment planning is based on the findings obtained during this process. Conventionally, for periodontal diagnosis we perform clinical examination to assess several periodontal parameters. While these conventional techniques offer simplicity and cost-effectiveness, their inherent limitation lies in assessing only disease history rather than current disease status. Progress in clinical diagnostic research is shifting towards procedures that can recognize and quantify periodontal risk through objective measures.[2,3]

Currently, a range of naturally occurring chemicals, recognized as biomarkers, are being utilized for early diagnosis of periodontitis, evaluating the current extent of periodontal disease, or predicting future risks associated with the disease. A biomarker is present in the host as an indicator of normal biological processes, pathological processes, and a therapeutic interventions. Many biomarkers have been discovered to date which are commonly found inside bodily fluids like “gingival crevicular fluid (GCF)”, saliva and serum for studying “periodontal disease”.[3]

The biomarkers commonly associated with periodontal disease include interleukins (IL1β and IL6), "tumour necrosis factor-α (TNF-α)".[4,5] Some of these biomarkers are implicated in collagen breakdown, while others are associated with bone remodelling processes.[6,7,8,9] Selecting saliva as a point of care has many benefits such as a convenient collection of samples, time efficiency, relatively inexpensive, presence of factors and molecules from GCF, and usage in screening large populations. Salivary biomarkers provide an early, non-invasive means to detect host defense, inflammatory events that typically occur before clinical or radiographic signs of periodontal tissue damage emerge. Resistin, a recently acknowledged marker, exemplifies all the characteristics of an ideal biomarker in this context.[10]

Resistin & its role in inflammation and inflammation relation diseases.

Resistin, a recently discovered hormone secreted by adipocytes, belongs to the proteins characterized by their abundance of cysteine amino acids, which contain sulphur. Mainly located in rodents, this substance has also been identified in various human tissues. Initially, resistin was linked with insulin resistance and type 2 diabetes. Nowadays, its association with inflammatory conditions such as atherosclerosis has been recognized. Resistin was initially identified in 2001 while searching for genes involved in adipocyte differentiation. The value was seen to be decreased in mature adipocytes when exposed to the drug "thiazolidinediones (TZD)", leading to the discovery of a protein named Resistin. (derived from resistance to insulin).[10,11]

Resistin belongs to the found in inflammatory zones family, which is now also referred to as "RELMs", which represent a group of tissue-specific signalling molecules. It was noted that the family has a distinct feature consisting of a rich in cysteine C terminus with invariant cysteine spacing. Rodents have four sects in the RELMs family: resistin, "RELMα, RELMβ, and RELMγ" where as in human population there are two categories : resistin and RELMβ.[11]

RELMα (FIZZ1) is primarily found in adipose tissue, while the other strain that is found in humans (RELMβ, FIZZ2) is seen to be in high numbers in the gastrointestinal tract, specifically secreted by goblet and epithelial cells. In mice, the hematopoietic system releases the RELMβ strain, specifically in the bone marrow. A small amount is also released in the WBCs. Both types of resistin genes share 46.7% DNA, 64.4% mRNA and 59% amino acid similarity. The resistin gene in humans produces a protein that is structured into three primary domains.[11,12]

The configuration includes a disulfide-rich β-sandwich at the carboxy-terminal, coupled with an α-helical segment at the amino-terminal. These elements contribute to the development of a parallel coil. Subsequently, hexamers are formed by two trimers and every unit is linked from one trimer to a protomer and establishing a compact six-helix bundle.[12]

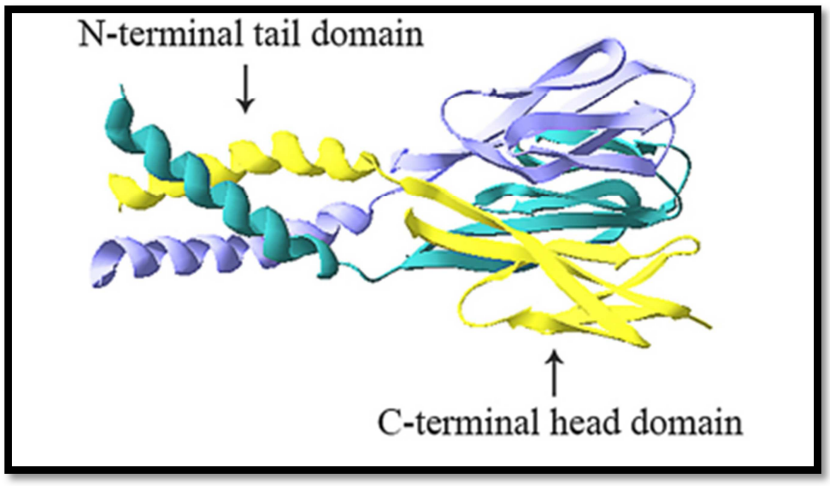


Figure 1: Schematic presentation of the structure of resistin

While initially associated with insulin resistance, resistin is increasingly recognized for its involvement in inflammatory processes. There is growing evidence pointing to its role in inflammatory pathways. Agents such as "tumour necrosis factor-alpha (TNF-α)" modulate the secretion of the gene of the resistin. Particularly, it has been noted in peripheral blood mononuclear cells (PBMC) where TNF alpha increases resistin mRNA value in human beings.[13] IL-6 has been observed to upregulate resistin expression in PBMCs, and LPS has been documented to increase resistin mRNA levels in both white adipose tissue and WBCs in rats.[14]

Further evidence connecting resistin to inflammation includes the observation of plasma resistin levels being correlated with numerous inflammatory markers in certain pathophysiological conditions like obstructive sleep apnoea syndrome, atherosclerosis and chronic kidney disease.[15,16,17] Emerging data show an association between resistin and atherosclerosis, as it is implicated in initiating or perpetuating the atherosclerotic state through the activation of vascular endothelial cells.[18] Moreover, a connection exists between resistin and arthritis, exhibiting a positive correlation with

both synovial leukocyte count and IL-6 levels, suggesting its potential contribution to the inflammatory processes associated with arthritis.[19]

Patients suffering from periodontal inflammation typically exhibit heightened inflammatory response and burden which is due to increased levels of local proinflammatory cytokines. In light of this observation, it is plausible to suggest that salivary resistin levels in individuals with periodontitis may be elevated when compared to those in periodontally healthy patients. The literature regarding values on salivary resistin seen in subjects with healthy periodontium and periodontitis remains limited and controversial. Hence, the investigation was designed to assess salivary Resistin levels in subjects with healthy periodontium and periodontitis.

AIM AND OBJECTIVES

AIM OF THE STUDY:

Estimation of salivary Resistin levels in individuals with healthy periodontium and periodontitis.

OBJECTIVES:

1. To estimate salivary Resistin levels in individuals with healthy periodontium, moderate and severe periodontitis.
2. To compare salivary Resistin levels in individuals with healthy periodontium, moderate and severe periodontitis.
3. To correlate salivary Resistin levels in individuals with healthy periodontium and periodontitis

REVIEW OF LITERATURE

Afacan et al. (2019) [20] examined salivary resistin and TNF-α levels in various periodontal conditions. They collected salivary specimen and recorded clinical parameters such as "probing pocket depth (PD) and clinical attachment levels (CAL)". Salivary Resistin and TNF-α levels measured using "enzyme-linked immunosorbent assay (ELISA)". Although salivary resistin and TNF-α were more in periodontal disease categories compared to healthy, these differences were not statistically significant. However, both periodontitis categories had significantly greater PD and CAL than the gingivitis group. The study suggests that salivary "resistin" and TNF-α levels may not distinguish effectively between periodontal disease and health, highlighting the need for larger investigations on the relationship between salivary cytokine levels and periodontal inflammation.

Zeinab Rezaei Esfahrood et al. (2018) [21] conducted a study investigating resistin levels present in subjects with chronic periodontitis compared to those with healthy periodontium. The research involved 34 male and female participants, categorized into two groups: one with healthy periodontium and the other with chronic periodontitis. The results revealed significantly elevated presence of resistin in the saliva of patients diagnosed with "chronic periodontitis" when compared to individuals with periodontally healthy conditions. Based on these findings, the study suggested that resistin, acting as a pro-inflammatory molecule, may have the potential to stimulate the release of other inflammatory mediators such as TNF-α and IL-12. This indicated importance resistin in the pathogenesis of chronic periodontitis, highlighting its involvement in the inflammatory processes associated with this condition.

Akram Z et al (2017) [22] aimed to explore changes in the periodontium after nonsurgical treatment (NSPT) and investigate the relationship in alterations in resistin values in obese Malaysians with chronic periodontitis. Results showed that plaque score significantly decreased in the test compared to controls at both 6 and 12 weeks. The NSPT group also saw reductions in shallow and moderate pocket percentages, with no changes in deeper pockets. Salivary resistin levels decreased significantly post-NSPT, but no significant link was found between resistin changes and periodontal outcomes. Notably, salivary resistin levels did not correspond with improvements in any periodontal parameter.

Mittal M et al. (2015) [23] categorized them into four groups: healthy individuals, chronic periodontitis patients, rheumatoid arthritis patients, and patients with both conditions. They assessed periodontal parameters, panoramic radiographs, and GCF Resistin. All patients revealed the presence of GCF resistin, with the highest levels in patients with both conditions and the lowest in first group. The study identified positive correlations between GCF resistin and periodontal parameters and rheumatoid factor, indicating the potential of GCF resistin as an inflammatory marker for both chronic periodontitis and rheumatoid arthritis.

Ahmed MA et al (2015) [24] aimed to assess salivary levels of resistin and leptin measured along with other parameters in “chronic periodontitis” patients with well or poor control of “type 2 diabetes mellitus (T2DM)”. The greatest value of salivary resistin were found in “chronic periodontitis” patients with good-control of T2DM. Poorly controlled T2DM patients showed more periodontal tissue destruction and lower salivary flow rates. Salivary resistin and leptin were proposed as potential markers for assessing periodontal tissue damage and managing periodontal diseases and T2DM.

Neeraja H. Gokhale et al. (2014) [25] conducted a clinico-biochemical comparing resistin levels among healthy individuals, those having "chronic periodontitis (CP)", and "type 2 diabetes mellitus (T2DM)". Sixty subjects aged over 35 were categorized into four groups: healthy individuals, CP patients, T2DM patients, and those with both T2DM and CP. Various parameters were assessed, including "plaque index, gingival index". GCF resistin concentrations showed notable differences between groups, and positive correlations were found with gingival index, probing depth, plaque index, RBS and glycated haemoglobin. The study suggested that GCF resistin levels could serve as a potential inflammatory marker for periodontitis in individuals with T2DM.

Devanoorkar et al (2014) [11] emphasized biomarkers as precise disease activity indicators, highlighting resistin's role as a potent adipocytokine implicated in various chronic inflammatory diseases. Initial beliefs about its exclusive production by adipocytes shifted with emerging evidence indicating its production by immunoinflammatory cells. Resistin's ability to induce insulin resistance in mice led to its name derivation. A three-way association was identified among obesity, insulin resistance, and diabetes. The authors' previous research showed a rise in resistin levels with periodontal disease activity, decreasing post-periodontal therapy. Thus, resistin emerged as a molecular link connecting periodontal disease with other systemic diseases.

Patel SP et al. (2013) [26] conducted a study involving 96 subjects who were divided into healthy, periodontitis associated with uncontrolled diabetes, controlled diabetes associated periodontitis, and chronic periodontitis without T2DM. Using "enzyme-linked immunosorbent assay", the study measured resistin levels in both sulcular fluid and serum, comparing them across the groups. The findings showed no noteworthy

correlation between resistin value and PD/CAL. However, resistin levels were elevated in periodontitis or DM with periodontitis compared to healthy subjects and were positively linked with GI. Additionally, individuals with a specific type of genotype were more susceptible to developing periodontal disease as confirmed by more scores of GI, PD, and CAL compared to those with the other genotypes, indicating a potential association between resistin levels, genetic factors, and periodontal parameters.

Zimmermann GS et al. (2013) [27] investigated local and systemic concentrations of adipocytokines such as resistin in fluid samples from periodontal pocket sites and evaluated them utilizing "enzyme-linked immunosorbent assay (ELISA)" in individuals with "chronic periodontitis (CP)" who were either obese or of normal weight (NW). The findings showed that CP primarily influenced circulating levels of resistin and adiponectin, while both obesity and CP together affected circulating levels of leptin, creating a proinflammatory environment. Additionally, obesity was linked to increased local expression of TNF-α.

R. Furugen et al. (2012) [28] investigated how resistin release from neutrophils changes when exposed to "lipopolysaccharide (LPS) from *Porphyromonas gingivalis*" and "*Escherichia coli"*. They added different density of "*P. gingivalis*-LPS and *E. coli*-LPS" into neutrophils and measured resistin levels in the supernatant using ELISA. Findings showed that both types of LPS induced resistin release, with E. coli-LPS causing stronger release at lower concentrations. Blocking CD14, CD18, and TLR4 reduced resistin release. These observation indicated that "*P. gingivalis"*-LPS and "*E. coli"*-LPS through specific cell receptors and intracellular pathways, influence the release of Resistin

Hiroshima et al (2012) [29] examined human gingival crevicular fluid for the presence of Resistin across individuals with chronic periodontitis, "diabetes mellitus" suffering from periodontitis, and controls. They used "western blot analysis" and ELISA to measure resistin levels. Specimens collected from the first two groups showed significantly higher resistin levels compared to healthy subjects. Resistin levels associated with the gingival index score but not with blood HbA1c values. Porphyromonas gingivalis lipopolysaccharide (P-LPS) was found to raise the amount of resistin secreted from human neutrophils, an effect that was reduced by actin polymerization inhibitors. This study marked the first identification of resistin in "gingival crevicular fluid", suggesting possible association between elevated resistin levels in periodontitis and induced neutrophils.

Saito T et al. (2008) [30] explored the functions of resistin and adiponectin, two newly identified adipokines produced by fat cells and known for their contrasting roles in insulin resistance and inflammation. Particularly, resistin, abundant in macrophages, has a role in inflammation. The research aimed to understand the levels of these adipokines in the serum of women with periodontitis. Serum adipokine levels were compared between the groups, considering variables like obesity, smoking habits, and age. The results demonstrated a significant link between periodontitis and increased resistin levels, as shown in both single-factor and multi-factor analyses. However, the connection between periodontitis and reduced adiponectin levels did not show statistical significance. These findings suggested elevated serum resistin levels in Japanese women who are middle aged suffering from periodontitis might have systemic health implications, hinting at a possible association between periodontitis and systemic inflammation mediated by resistin.

Silswal et al. (2005) [31] conducted a review focusing on resistin, a protein linked to insulin resistance, "type 2 DM". Raised serum resistin levels in such a case correlate with inflammation. The study showed that adding "recombinant human resistin protein (hResistin)" to macrophages increased pro-inflammatory cytokine secretion, akin to lipopolysaccharide (LPS). Both oligomeric and dimeric hResistin forms activated cytokines independently. Heat-denatured hResistin did not induce cytokine production, ruling out endotoxin involvement. hResistin also induced NF-κB translocation, and its TNF-α induction was reduced by inhibitors, highlighting its dual role.

Patel L et al (2003) [32] examined how Peroxisome Proliferator-Activated Receptor Gamma (PPARγ) regulates human primary macrophages for the release of resistin. The procedure was done using "fluorescent real-time PCR (Taqman) analysis", assessing resistin across different human tissues, showing higher levels in marrow of bone than in other tissues. Both "Taqman analysis and Western blotting" demonstrated that a PPARγ agonist, reduced release of resistin in vitro. Exposure to 100 nM rosiglitazone for 96 hours led to an 80% decrease in resistin expression. Furthermore, an analysis of the genomic sequence upstream of resistin using bioinformatics tools identified potential PPAR response elements. Among these elements, one was validated to bind with PPARγ based on electrophoretic mobility shift assays. These findings indicate PPARγ's direct involvement in regulating resistin expression.

Kaser S et al. (2003) [13] investigated resistin, a polypeptide known to induce resistance of insulin in rodents. It was seen that the Resistin present in rodent adipocyte and peripheral blood mononuclear cells (PBMC) in humans which appear to be a major origin of resistin. The study aimed to elucidate the regulation of resistin

mRNA expression in human PBMCs. Their findings, determined by "fluorescence-based real-time polymerase chain reaction (PCR)", revealed that proinflammatory cytokines such as "interleukin (IL)-1, IL-6" significantly increased resistin mRNA expression in human PBMCs. Conversely, no significant effect was observed with "interferon-γ (IFN-γ) or leptin." These observations suggested that in humans, resistin may serve as a link in the well-established association between inflammation and insulin resistance. This indicated a potential role for resistin in mediating the effects of proinflammatory cytokines and LPS on insulin sensitivity, highlighting its relevance in the context of metabolic disorders characterized by inflammation and insulin resistance.

MATERIALS AND METHODS

SOURCE OF DATA:

It was a descriptive cross-sectional study, conducted in the "Outpatient Department of Periodontics, KAHER's KLE V.K. Institute of Dental Sciences, Belagavi." The laboratory procedures were carried out in the "Hi-Tech Laboratory, KLE's Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi". An ethical clearance was obtained from the "Ethical Committee, KAHER's KLE V.K. Institute of Dental Sciences, Belagavi" before conducting the study.

CRITERIA FOR GROUP SELECTION:

The study population was divided into three groups:

Group A : Periodontal health

Group B : Moderate Periodontitis

Group C : Severe Periodontitis

A thorough clinical examination was done. Diagnosis of periodontal disease was given based on proposal elements for inclusion in the classification of periodontitis 2017 World Workshop.[33] Before starting, the study purpose and format were described to the patients in a language they understood and a written consent was acquired from them. All subjects were examined for their periodontal condition. After recording the demographic and clinical data, collection of saliva sample was carried out.

INCLUSION CRITERIA:

Group A : Periodontal health

1. Patients above 30 years of age.
2. No Clinical attachment loss
3. Absence of periodontal pockets & Radiographic bone loss

Group B : Moderate Periodontitis

1. Patients above 30 years of age.
2. Clinical attachment loss (CAL) ≤ 4mm
3. Probing Depth (PD) ≤ 5mm
4. Radiographic Bone Loss (RBL) : coronal third (15%-33%)

Group C : Severe Periodontitis

1. Patients above 30 years of age.
2. "Clinical attachment loss (CAL) ≥ 5 mm"
3. Probing Depth (PD) ≥ 6mm
4. "Radiographic Bone Loss (RBL) : extending to mid-third of root and beyond (≥ 33%)"

EXCLUSION CRITERIA:

1. Patients with history of smoking and tobacco chewing
2. Pregnant or lactating women.
3. Patients diagnosed with Salivary gland disorders or Xerostomia.
4. Patients on any kind of medications

Figure 2: Periodontal health

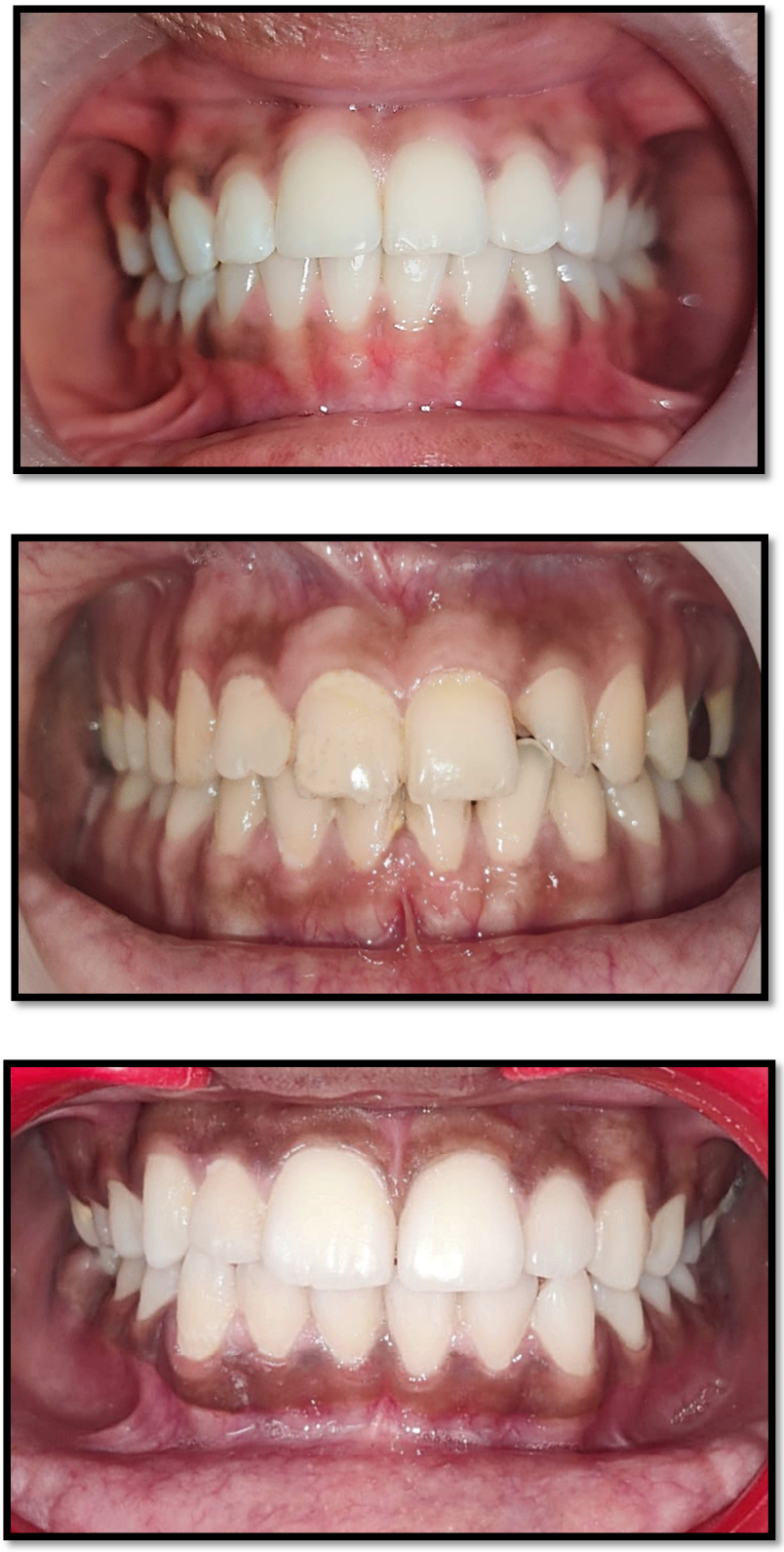


Figure 3 : Moderate Periodontitis

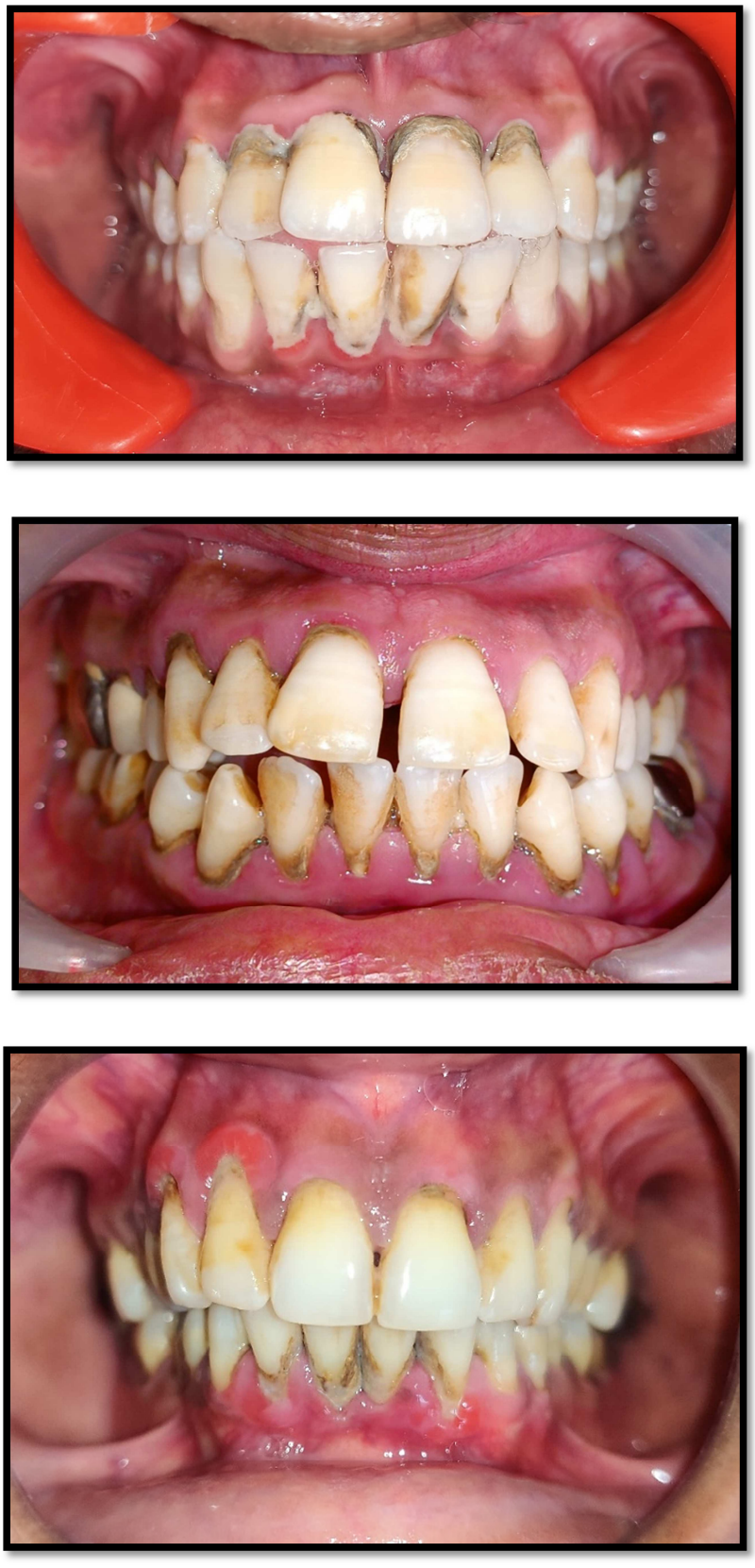
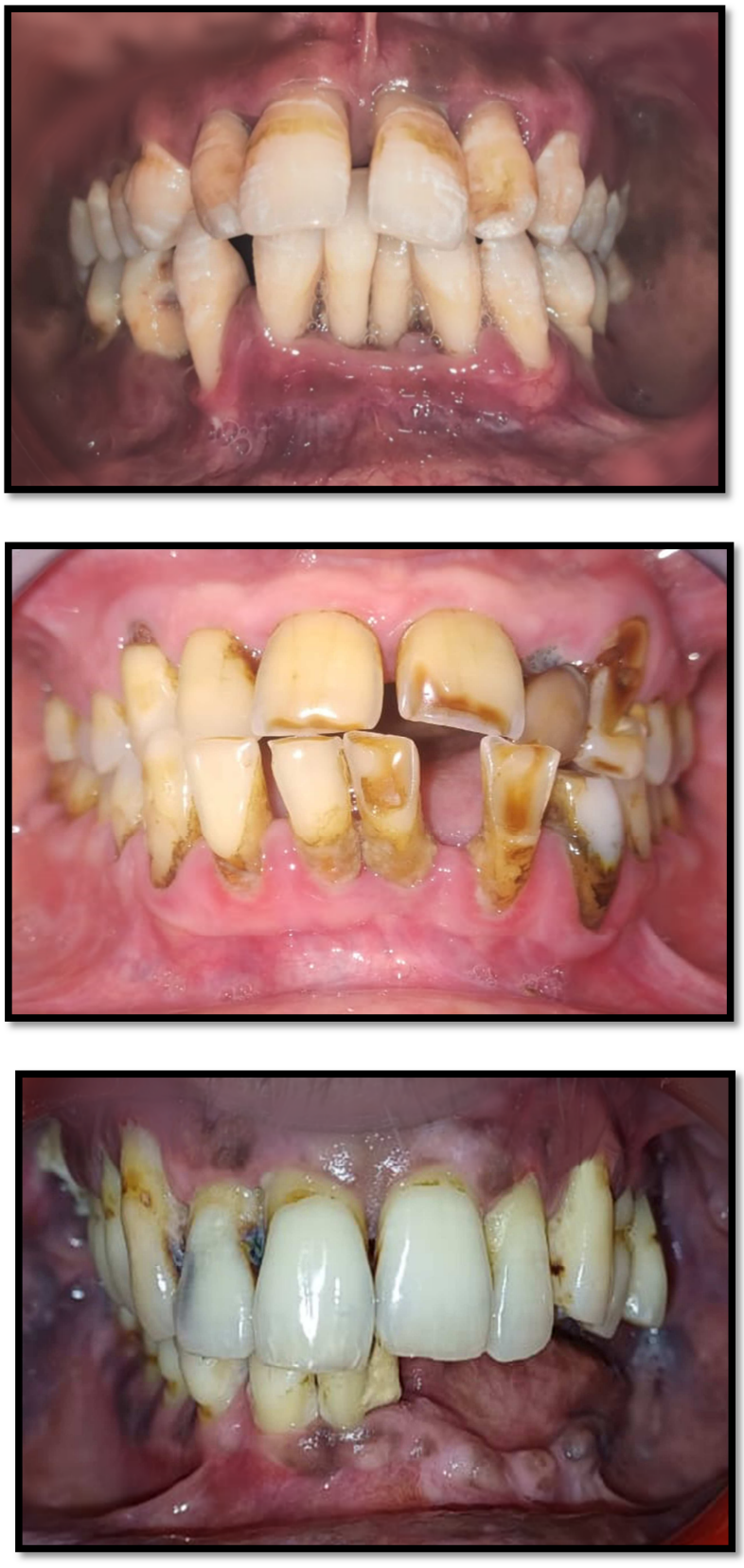


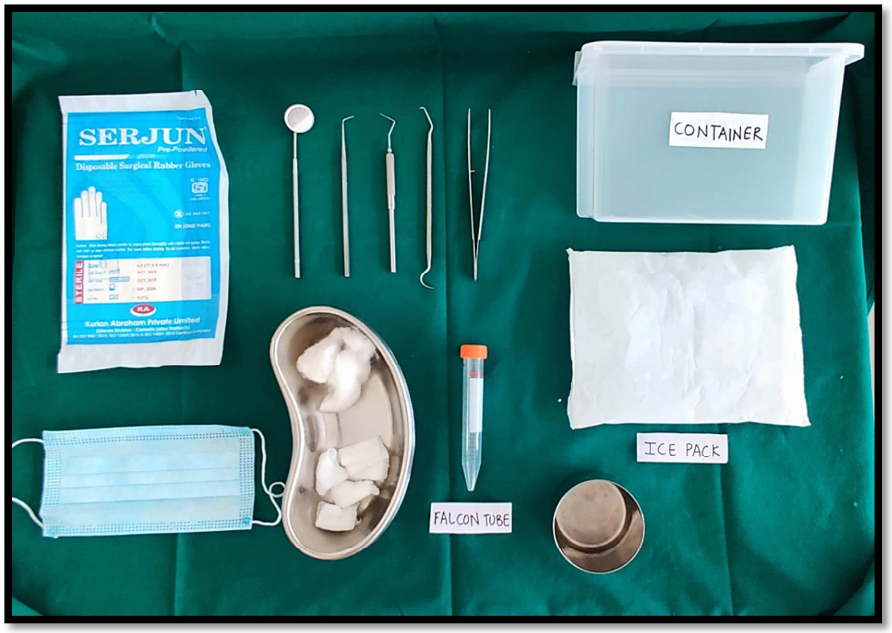
Figure 4 : Severe Periodontitis



CLINICAL ARMAMENTARIUM:

1. Mouth mirror
2. Explorer
3. Straight probes
4. Tweezers
5. Kidney tray
6. Cotton roll and gauze
7. William's graduated periodontal probe
8. Mouth mask
9. Disposable latex gloves
10. Distilled water for rinsing
11. 15 ml falcon tube
12. Ice for transportation to the laboratory

Figure 5 : Clinical Armamentarium



PARAMTERES RECORDED:

Following parameters were recorded:

1. Probing depth
2. Clinical attachment level
3. Radiographic bone loss (%)
4. OHI-S index
5. Body Mass Index (BMI)

Pocket Probing Depth [34] :

A "Williams graduated periodontal probe" was employed to measure PPD, measuring from the free margin of gingiva to the base of the pocket. The probe was carefully maneuvered along the circumference of the tooth within the gingival sulcus during the measurement process. Three measurements were done the buccal aspect and on the lingual aspect of each tooth.

Clinical Attachment Level [34] :

The clinical attachment levels were determined by measuring from the cementoenamel junction (CEJ) to depth of the pocket base using a Williams graduated periodontal probe.

A. If the gingival margin was coronally placed (above) to the CEJ, the attachment level was calculated by subtracting the distance between the free gingival margin and the CEJ from the pocket probing depth.

B. If it was at the level of the CEJ, the attachment level was considered the same as the pocket probing depth.

C. If the gingival margin was apical (below) to the CEJ, the attachment level was determined by adding the distance between the free gingival margin and the CEJ to the pocket probing depth.

Radiographic bone loss (%) [35] :

Intra-oral radiographs were taken. Measurements were done as follows:

1) Normal bone level = "The distance from the cementoenamel junction (CEJ) to the apex of the root. Subtract 2mm from the CEJ to apex measurement to determine where the bone levels should be in health."
2) Current bone level at the site = Height of bone to the root apex.
3) Bone loss = Normal bone level – Current bone level

   Hence Radiographic bone loss = $\frac{\text{Bone loss}}{\text{Normal bone level}} \times 100$

OHI-S index [36] :

OHI-S index:

"Debris index- simplified (DI-S)

16	11	26
46	31	36

Calculus index-simplified (CI-S)"

16	11	26
46	31	36

OHI-S = DI-S + CI-S =

Body Mass Index (BMI) [37] :

The BMI calculation divides an adult's weight in kilograms (kg) by their height in metres (m) squared.

$$\text{Body Mass Index (BMI)} = \frac{\text{Weight (kg)}}{\text{Height (m}^{2)}}$$

ESTIMATION OF SALIVARY RESISTIN USING "ELISA KIT (SHANGHAI COON KOON BIOTECH CO., LTD)" [38]

LABORATORY ARMAMENTARIUM:

1. -20°C Refrigerator for storage of samples
2. ELISA microplate reader (absorbance at 450 nm)
3. Micropipette
4. Centrifuge machine
5. Laminar air flow
6. Eppendorf tubes of 2 ml
7. 37 °C incubator
8. Precision pipettes to deliver 2 ml to 1 ml volumes.
9. 100 ml and 1 litre graduated cylinders.
10. Absorbent paper
11. Precision pipettes and disposable tip
12. Log graph paper standard or sample dilutions

ELISA KIT CONTENTS:

1. Pre-coated 96-well Strip Microplate: 12 strips of 8 wells each- 1 unit.
2. Standard A : 0ng/mL
3. Standard B : 1.25ng/mL
4. Standard C : 2.5ng/mL
5. Standard D : 5ng/mL
6. Standard E : 10ng/mL
7. Standard F : 20ng/mL
8. Sample Diluent : 6.0ml
9. HRP-Conjugate reagent : 10.0ml
10. 20X Wash solution : 25ml
11. Chromogen Solution A : 6.0ml
12. Chromogen Solution B : 6.0ml
13. Stop Solution A : 6.0ml
14. Closure plate membrane : 2
15. User manual : 1
16. Sealed bags : 1

Figure 6 : -20° C Refrigerator

Figure 7 : Laminar Air Flow

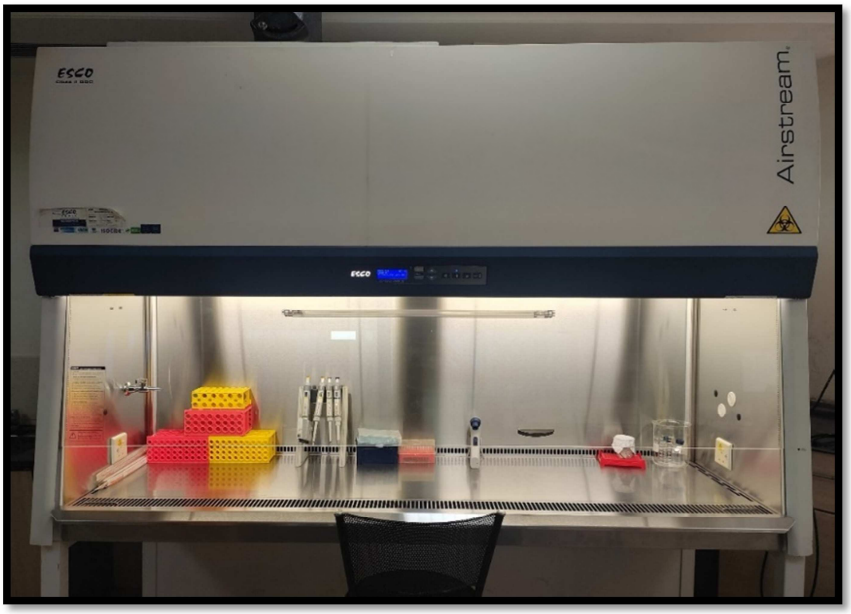


Figure 8 : Micropipette

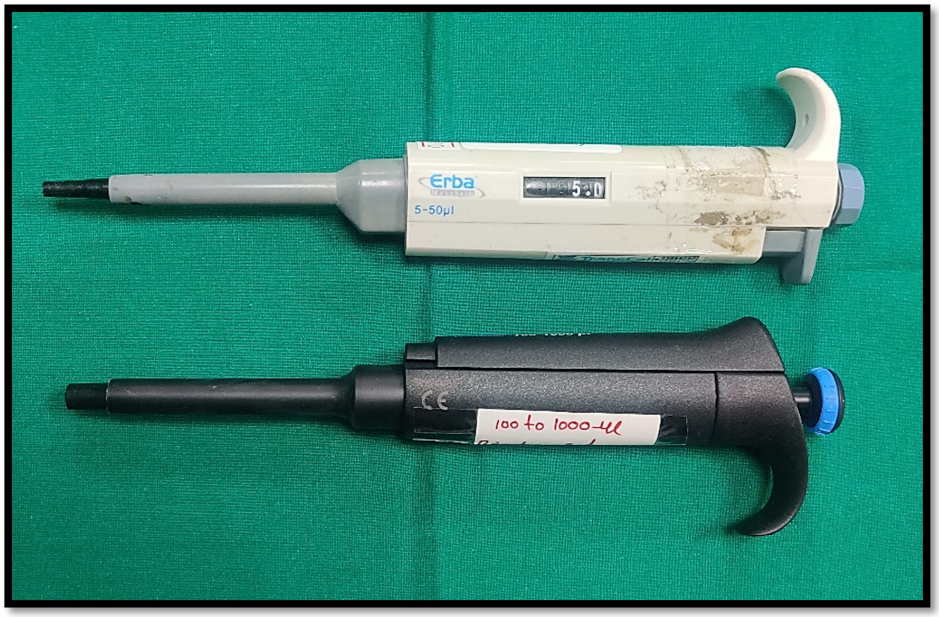


Figure 9 : Centrifuge machine

Figure 10 : Vortex machine

Figure 11 : ELISA microplate reader

Figure 12 : ELISA kit contents

PROCEDURE

Collection of Saliva Sample [39] :

The study was described to all patients in the language they understood. Unstimulated whole saliva was collected by means of spitting method as described by Navazesh. Patients were advised not to consume any liquid or solid food substances 1 hour prior to saliva collection. At first, patients rinsed their mouth with water. This was followed by expectoration of whole saliva into 15 ml Falcon tube. A final volume of 3 to 5 ml whole saliva was obtained for each patient. Each saliva sample was immediately placed on icepacks for transportation to the laboratory. Samples were stocked at -20° C till the time of assay. At the time of assay, samples were first centrifuged at 1100g at 4°C for 10 minutes. The estimation of salivary resistin was done using "Enzyme- linked Immunosorbent assay (ELISA, Shanghai coon koon biotech co., ltd)"

Figure 13 : Collection of saliva

ASSAY PROCEDURE [38] :

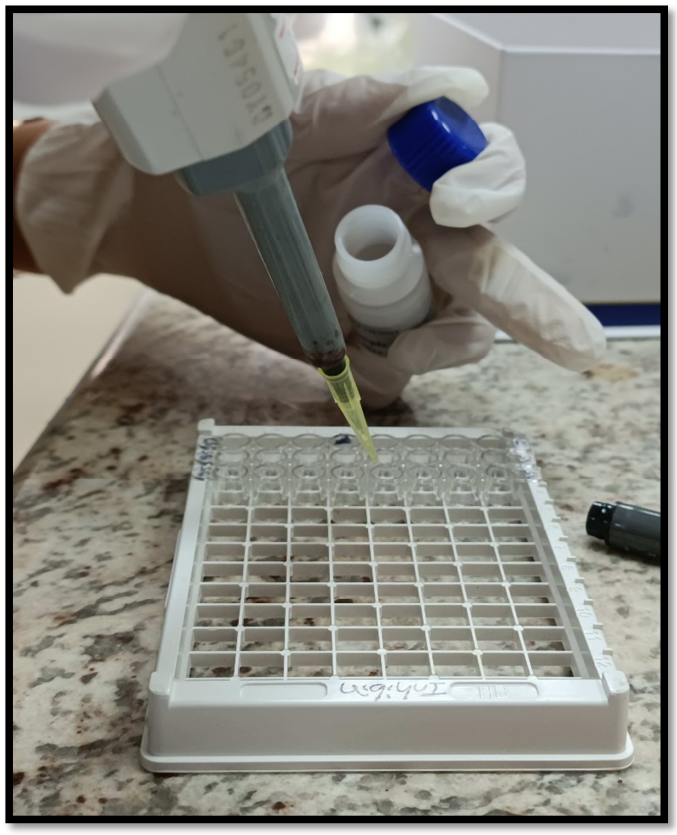
Estimation of Salivary Resistin was done using "ELISA kit (Shanghai Coon Koon Biotech Co., Ltd)".

1. The standard was introduced into the designated Standard wells and sample wells were tested accordingly. Specifically, 50 microliters of standard was added to the well, while the blank well was kept empty.
2. 10 microlitre sample was deposited into the testing sample well, and subsequently, 40µl of sample diluent was introduced into the same well.
3. A volume of 100µl of HRP-conjugate reagent was applied to both the Standard and testing sample wells, followed by sealing the plate with a membrane. The plate was then gently shaken and mixed for 60 minutes during incubation at 37°C.
4. The washing solution was made by diluting the concentrated washing solution (20X) with distilled or deionized water for future usage.
5. In the manual washing method, the sealing film was delicately removed, the liquid was drained, and each well was dried. Next, the wells were filled with washing solution, left for 1 minute, drained again, and this process was repeated 5 times before pat drying the plate. Alternatively, for automatic washing, 350µL of wash solution was injected into each well, soaked for 1 minute, and the plate was washed 5 times.
6. For colour development, 50 microlitre chromogen sol-A and 50 microlitre of chromogen sol-B were sequentially added. The plate was gently shaken, incubated for 15 minutes at 37°C while shielded from light.
7. To terminate the reaction, 50µl of Stop Solution was introduced to each well, leading to an immediate color change from blue to yellow. If any wells

showed a green color or if the color change was uneven, gentle tapping ensured thorough mixing.

8. During the assay, the blank well served as the zero point, and the absorbance (OD) of each well was measured individually at a wavelength of 450nm. This measurement was performed within 15 minutes after adding the stop solution.
9. The linear regression equation for the standard curve was established using the concentrations of the standards and their respective OD values.

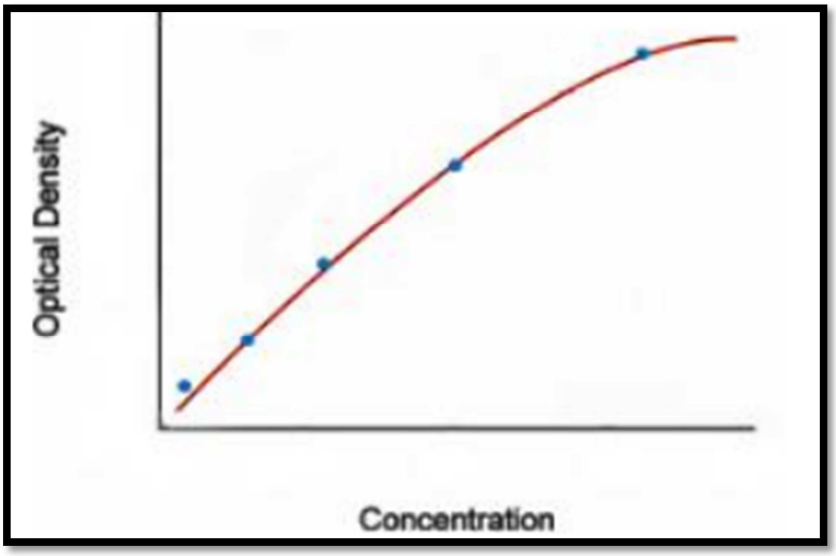
Figure 14 : Assay procedure



Calculation of results

1. To quantify the quantity present in an unknown sample, a standard curve was employed
2. This curve was established by plotting the average Optical Density (O.D.) values obtained at 450 nm for each of the six standard concentrations on the Y-axis against their respective concentrations on the X-axis.
3. Subsequently, O.D was adjusted through subtracting the mean value of the zero standard. Generation of standard curve done using graph paper
4. The O.D. value was located on Y axis to check the quantity in each sample , and a horizontal line intersected the standard curve. The corresponding concentration was then read by drawing a vertical line from the intersection point to the X-axis.

Figure 15 : OD value vs Concentration



SAMPLE SIZE ESTIMATION:

At

95 % confidence interval

95 % power,

$$n = \frac{(Z_{1-\alpha/2} + Z_{1-\beta})^2 (SD_1^2 + SD_2^2)}{(x_1 - x_2)^2}$$

n = 26 per group

The total estimated sample size is 78

SD_1: 7.0

SD_2: 15.0

$Z_{1-\alpha/2}$: 1.96

$Z_{1-\beta}$: 1.64

x_1: 8.1

x_2: 19.7

n: sample size number

α error = 5%

β error = 20%

STATISTICAL ANALYSIS:

- The data was put into Excel sheet and analysed using SPSS version 20.0 for statistical analyses. Mean and SD were estimated for each parameter within each of the three groups.
- Distribution of age and gender was assessed in all three groups. Percentage distribution of gender was done for all groups.
- Correlation between gender distribution and Resistin values was assessed using Chi square test.
- The mean of PPD, CAL, RBL%, OHI-S and BMI and levels of salivary Resistin for every group were assessed by "Analysis of variance (ANOVA) and Tukey's multiple post hoc test."
- Association between the salivary Resistin values with PPD, CAL, RBL%, OHI-S and BMI were done using Karl Pearson's Correlation ratio.
- Level of significance was fixed at p= 0.05. Any value less than or equal to 0.05 was considered as statistically significant.

RESULTS AND OBSERVATIONS

Table 1: Groupwise comparison based on gender

Gender	Group A	%	Group B	%	Group C	%	Total
Male	13	50.00	20	76.92	16	61.54	49
Female	13	50.00	6	23.08	10	38.46	29
Total	26	100.00	26	100.00	26	100.00	78
Chi-square=4.0620, p=0.1310							

Observations:

1. Out of the 78 subjects, 49 were males and 29 were females.
2. In Group A, the number of Males and females were 13 each with a frequency percentage of 50 %.
3. In Group B, the number of Males and females was 20 and 6 with a frequency percentage of 76.92 % and 23.08 % respectively.
4. In Group C, the number of Males and Females was 16 and 10 with a frequency percentage of 61.54 % and 38.46 %.

Table 2: Groupwise comparison based on mean age by one way ANOVA

Groups	Minimum	Maximum	Mean	Std.Dev.
Group A	22.00	43.00	33.38	5.56
Group B	22.00	45.00	33.08	6.52
Group C	22.00	41.00	31.46	4.76
F-value	0.8668			
p-value	0.4245			

Observations:

The mean age in Group A, Group B and Group C was 33.38 ± 5.56 , 33.08 ± 6.52 , 31.46 ± 4.76, respectively.

Table 3: Comparison of three groups with Resistin levels by one way ANOVA

Groups	n	Means	Std.Dev.	Std.Err.	95% CI for mean	
					Lower bound	Upper bound
Group A	26	2.43	1.92	0.38	1.65	3.21
Group B	26	8.18	1.02	0.20	7.77	8.60
Group C	26	11.47	2.19	0.43	10.58	12.35
F-value	171.3339					
p-value	0.0001*					

*p<0.05

Observations:

1. The mean Resistin levels (ng/ml) in all groups were 2.43 ± 1.92, 8.18 ± 1.02, 11.47 ± 2.19 respectively, indicating that resistin increased with more severity of the periodontal disease, with a statistically notable distinction between the mean values of all the three groups (p-value = 0.0001)

Table 4: Pair wise comparison of three groups with Resistin levels by Tukeys multiple posthoc procedures

Group (I)	Group (J)	Mean Difference (I-J)	SE	p-value	95% CI	
					Lower Bound	Upper Bound
Group A	Group B	-5.7539	0.4941	0.0001*	-6.9353	-4.5724
	Group C	-9.0346	0.4941	0.0001*	-10.2161	-7.8531
Group B	Group A	5.7539	0.4941	0.0001*	4.5724	6.9353
	Group C	-3.2808	0.4941	0.0001*	-4.4623	-2.0993
Group C	Group A	9.0346	0.4941	0.0001*	7.8531	10.2161
	Group B	3.2808	0.4941	0.0001*	2.0993	4.4623

*$p<0.05$

Observations:

On comparison between the groups, there was a significant distinction between the mean of all the groups

Table 5: Comparison of three groups with Pocket Depth (PD) scores by one way ANOVA

Groups	n	Mean	SD	SE	95% CI	
					Lower bound	Upper bound
Group A	26	1.99	0.36	0.07	1.84	2.14
Group B	26	4.90	0.52	0.10	4.69	5.11
Group C	26	7.23	1.12	0.22	6.78	7.68
F-value	324.8533					
p-value	0.0001*					

*$p<0.05$

Observations:

1. The mean PD (mm) were 1.99 ± 0.36, 4.90 ± 0.52, 7.23 ± 1.12 respectively.
2. Significant association between the mean values of all the three groups (p-value = 0.0001)

Table 6: Pair wise comparison of three groups with PD scores by Tukeys multiple posthoc procedures

		Mean Difference	SE	p-value	95% CI	
					Lower Bound	Upper Bound
"Group A	Group B	-2.9077	0.2059	0.0001*	-3.4001	-2.4153
	Group C	-5.2385	0.2059	0.0001*	-5.7309	-4.7461
Group B	Group A	2.9077	0.2059	0.0001*	2.4153	3.4001
	Group C	-2.3308	0.2059	0.0001*	-2.8232	-1.8384
Group C	Group A	5.2385	0.2059	0.0001*	4.7461	5.7309
	Group B"	2.3308	0.2059	0.0001*	1.8384	2.8232

*$p<0.05$

Observations:

The Pair wise comparison of the three groups with PD revealed that there was a significant distinction in all the groups when compared between the groups.

Table 7: Comparison of three groups with Clinical Attachment Loss (CAL) scores by one way ANOVA

Groups	n	Means	SD	SE	95% CI	
					Lower bound	Upper bound
Group A	26	0.77	0.20	0.04	0.69	0.85
Group B	26	4.07	0.54	0.11	3.85	4.29
Group C	26	7.55	1.06	0.21	7.12	7.97
F-value	619.4530					
p-value	0.0001*					

*p<0.05

Observations:

1. The mean CAL (mm) in Group A, Group B, Group C was 0.77 ± 0.20, 4.07 ± 0.54, 7.55 ± 1.06 respectively (p-value = 0.0001)

Table 8: Pair wise comparison of three groups with CAL scores by Tukeys multiple posthoc procedures

Group (I)	Group (J)	Mean Difference (I-J)	SE	p-value	95% CI	
					Lower Bound	Upper Bound
Group A	Group B	-3.2962	0.1925	0.0001*	-3.7563	-2.8360
	Group C	-6.7731	0.1925	0.0001*	-7.2332	-6.3129
Group B	Group A	3.2962	0.1925	0.0001*	2.8360	3.7563
	Group C	-3.4769	0.1925	0.0001*	-3.9371	-3.0168
Group C	Group A	6.7731	0.1925	0.0001*	6.3129	7.2332
	Group B	3.4769	0.1925	0.0001*	3.0168	3.9371

*$p<0.05$

Observations:

The Pair wise comparison of the three groups with CAL revealed that there was a statistically significant distinction among the mean of all the groups

Table 9: Comparison of three groups with Radiographic Bone loss (RBL %) scores by one way ANOVA

Groups	n	Means	SD	SE	95% CI	
					Lower bound	Upper bound
Group A	26	0.65	2.17	0.43	-0.22	1.53
Group B	26	26.83	6.86	1.34	24.06	29.60
Group C	26	48.69	7.15	1.40	45.80	51.58
F-value	438.8495					
p-value	0.0001*					

*p<0.05

Observations:

1. The mean RBL (%) in Group A, Group B, Group C was 0.65 ± 2.17, 26.83 ± 6.86, 48.69 ± 7.15 respectively. (p-value = 0.0001)

Table 10: Pair wise comparison of three groups with RBL % scores by Tukeys multiple posthoc procedures

Group	Group	Mean Difference	Std. Error	p-value	95% Confidence Interval	
					Lower Bound	Upper Bound
Group A	Group B	-26.1731	1.6237	0.0001*	-30.0555	-22.2907
	Group C	-48.0385	1.6237	0.0001*	-51.9208	-44.1561
Group B	Group A	26.1731	1.6237	0.0001*	22.2907	30.0555
	Group C	-21.8654	1.6237	0.0001*	-25.7478	-17.9830
Group C	Group A	48.0385	1.6237	0.0001*	44.1561	51.9208
	Group B	21.8654	1.6237	0.0001*	17.9830	25.7478

*$p<0.05$

Observations:

The Pair wise comparison of the three groups with RBL% revealed that there was an association between the mean of all the groups when compared between the groups.

Table 11: Comparison of three groups with OHI-S scores by one way ANOVA

Groups	n	Means	SD	SE	95% CI for mean	
					Lower bound	Upper bound
Group A	26	0.48	0.25	0.05	0.38	0.58
Group B	26	1.21	0.54	0.11	0.99	1.43
Group C	26	2.07	0.65	0.13	1.80	2.33
F-value	63.7323					
p-value	0.0001*					

*p<0.05

Observations:

1. The mean OHI-S index score in the three groups was 0.48 ± 0.25, 1.21 ± 0.54, 2.07 ± 0.65 respectively. (p-value = 0.0001)

Table 12: Pair wise comparison of three groups with OHI-S scores by Tukeys multiple posthoc procedures

		Mean Difference	SE	p-value	95% CI	
					Lower Bound	Upper Bound
Group A	Group B	-0.7308	0.1405	0.0001*	-1.0667	-0.3948
	Group C	-1.5846	0.1405	0.0001*	-1.9206	-1.2487
Group B	Group A	0.7308	0.1405	0.0001*	0.3948	1.0667
	Group C	-0.8539	0.1405	0.0001*	-1.1898	-0.5179
Group C	Group A	1.5846	0.1405	0.0001*	1.2487	1.9206
	Group B	0.8539	0.1405	0.0001*	0.5179	1.1898

*$p<0.05$

Observations:

The Pair wise comparison of the three groups with OHI-S index revealed that there was a statistically significant difference between the mean of all the groups when compared between the groups.

Table 13: Comparison of three groups with Body Mass Index (BMI) scores by one way ANOVA

Groups	n	Mean	SD	SE	95% CI	
					Lower bound	Upper bound
Group A	26	22.12	3.55	0.70	20.68	23.55
Group B	26	22.38	2.25	0.44	21.48	23.29
Group C	26	23.27	2.78	0.55	22.15	24.39
F-value	1.1209					
p-value	0.3314					

Observations:

1. The mean BMI score was 22.12 ± 3.55, 22.38 ± 2.25, 23.27 ± 2.78 respectively.
2. No statistical significance (p-value = 0.3314)

Table 14: Pair wise comparison of three groups with BMI scores by Tukeys multiple posthoc procedures

Group (I)	Group (J)	Mean Diff	SE	p-value	95% CI	
					Lower Bound	Upper Bound
"Group A	Group B	-0.2692	0.8063	0.9400	-2.1973	1.6588
	Group C	-1.1539	0.8063	0.3300	-3.0819	0.7742
Group B	Group A	0.2692	0.8063	0.9400	-1.6588	2.1973
	Group C	-0.8846	0.8063	0.5190	-2.8127	1.0434
Group C	Group A	1.1539	0.8063	0.3300	-0.7742	3.0819
	Group B"	0.8846	0.8063	0.5190	-1.0434	2.8127

Observations:

No significance was observed across the groups.

Table 15: Correlation between Resistin levels with PD, CAL, RBL%, OHI-S scores by Karl Pearson's correlation coefficient

Clinical parameters	Summery	Correlation between Resistin levels with			
		Total (n=78)	Group A (n=26)	Group B (n=26)	Group C (n=26)
PD	"r-value	0.9224	0.2139	0.3183	0.6851
	p-value	0.0001*	0.2940	0.1130	0.0001*
CAL	r-value	0.9097	0.1264	0.0420	0.6525
	p-value	0.0001*	0.5380	0.8380	0.0001*
RBL %	r-value	0.9065	-0.2773	0.3730	0.6124
	p-value	0.0001*	0.1700	0.0610	0.0010*
OHI-S	r-value	0.7762	0.3262	0.4020	0.2664
	p-value	0.0001*	0.1040	0.0420*	0.1880"

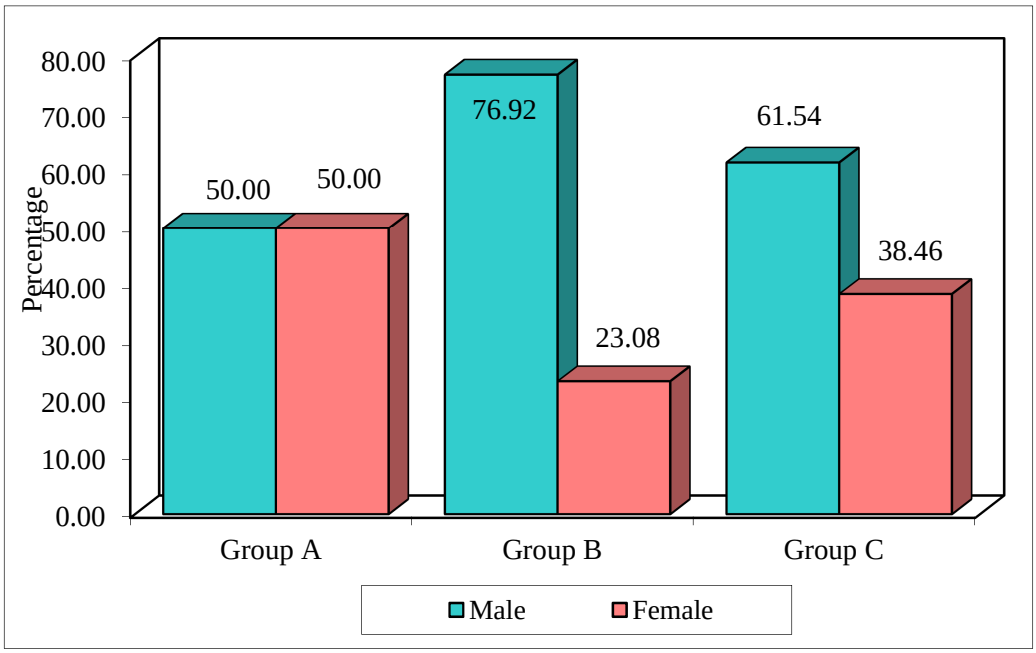
*$p<0.05$ indicates significant correlation

Observations:

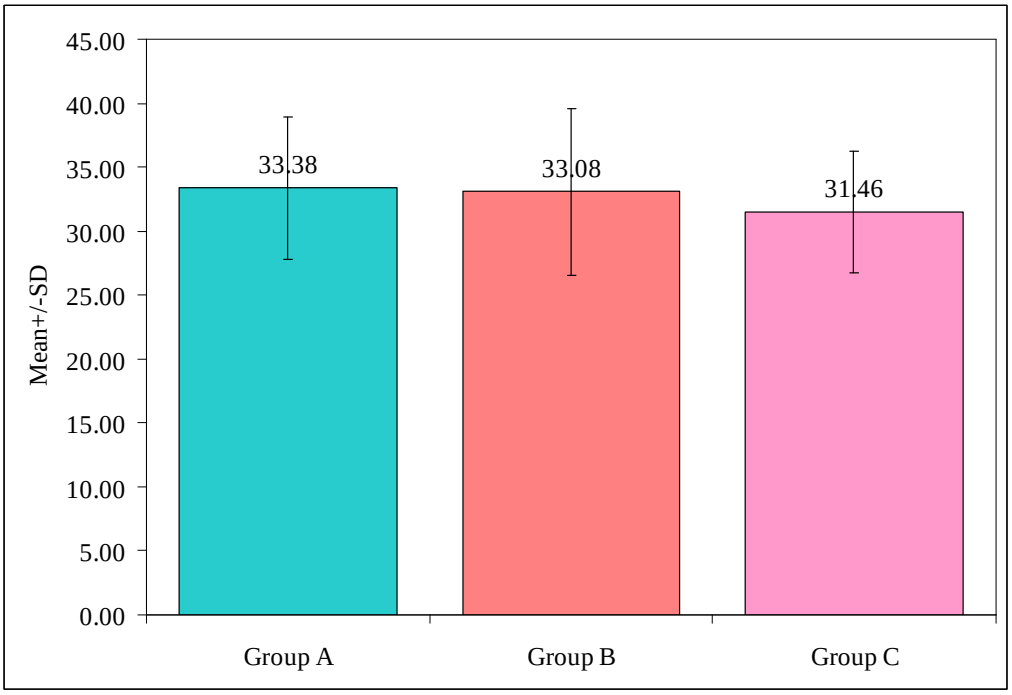
1. Significant correlation between the overall mean value of resistin with the overall mean PD, CAL, RBL% and OHI-S scores.
2. Amongst the three groups, Group C revealed a statistically significant correlation between the mean resistin value and PD, CAL and RBL%.
3. This indicates that there is a positive correlation present between Resistin with the parameters of periodontal disease.

GRAPHS

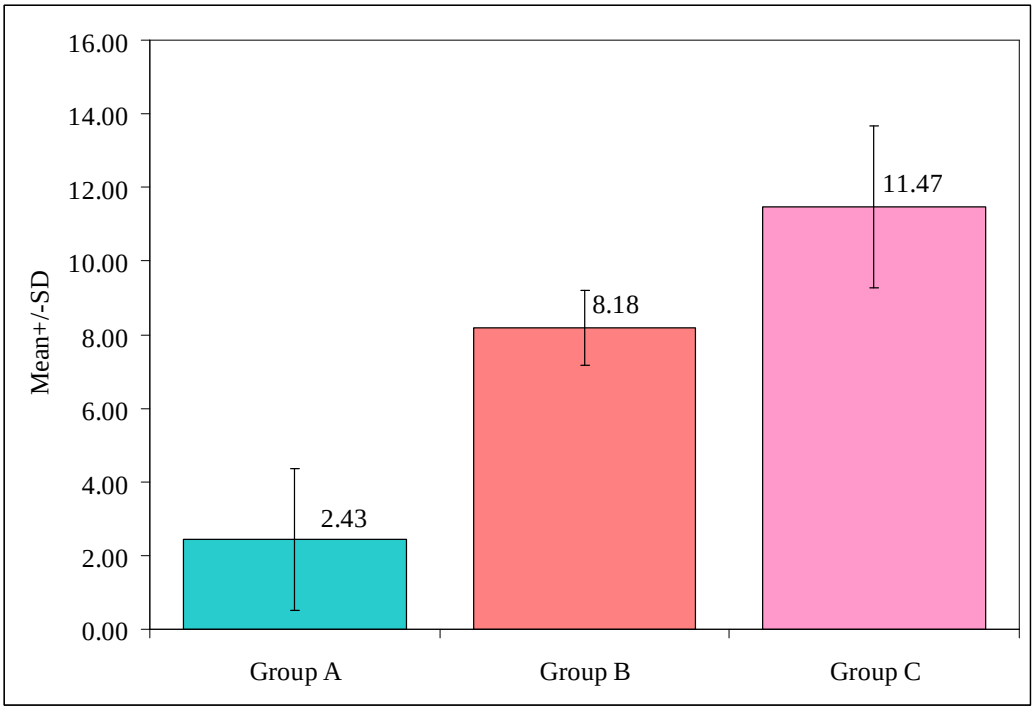
Graph-1: Groupwise comparison based on gender



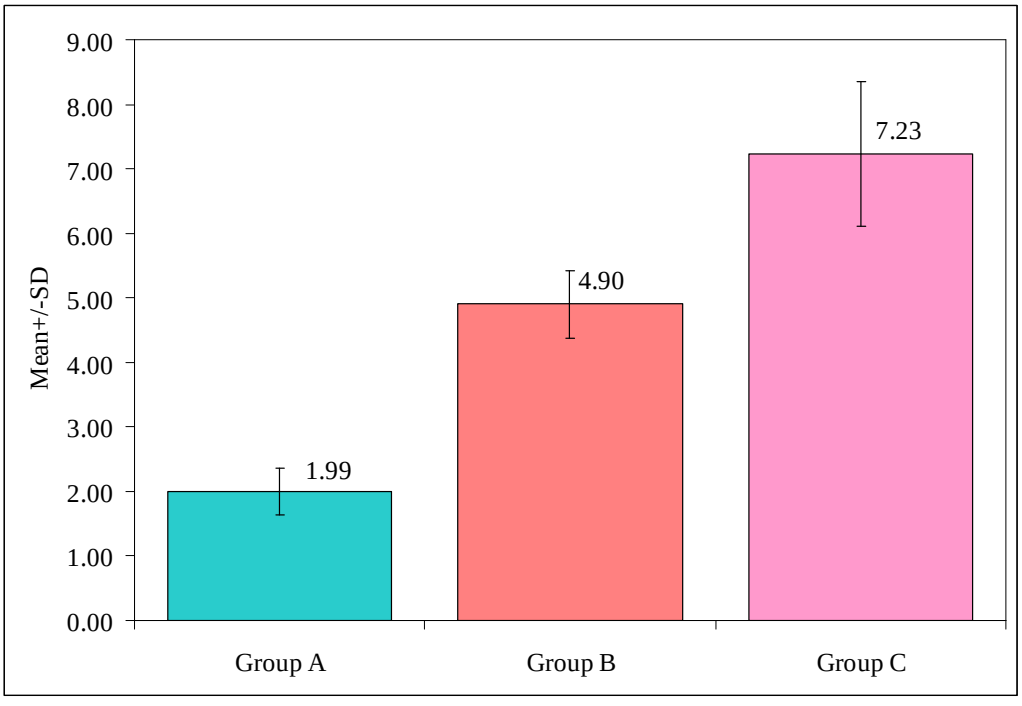
Graph-2: Groupwise comparison based on mean age



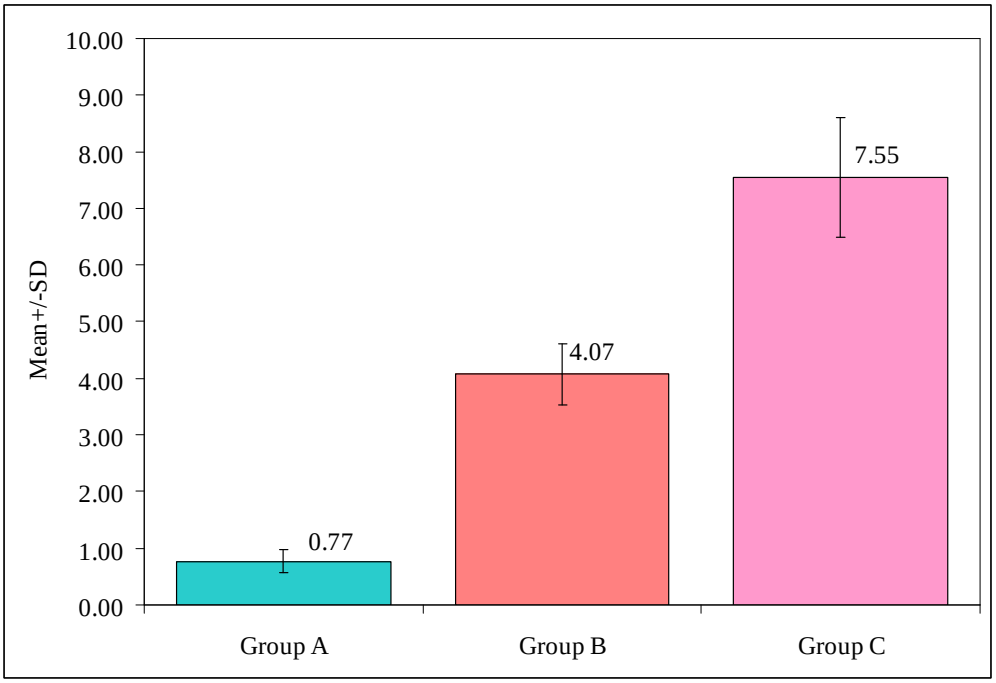
Graph-3: Pair wise comparison of three groups with Resistin levels



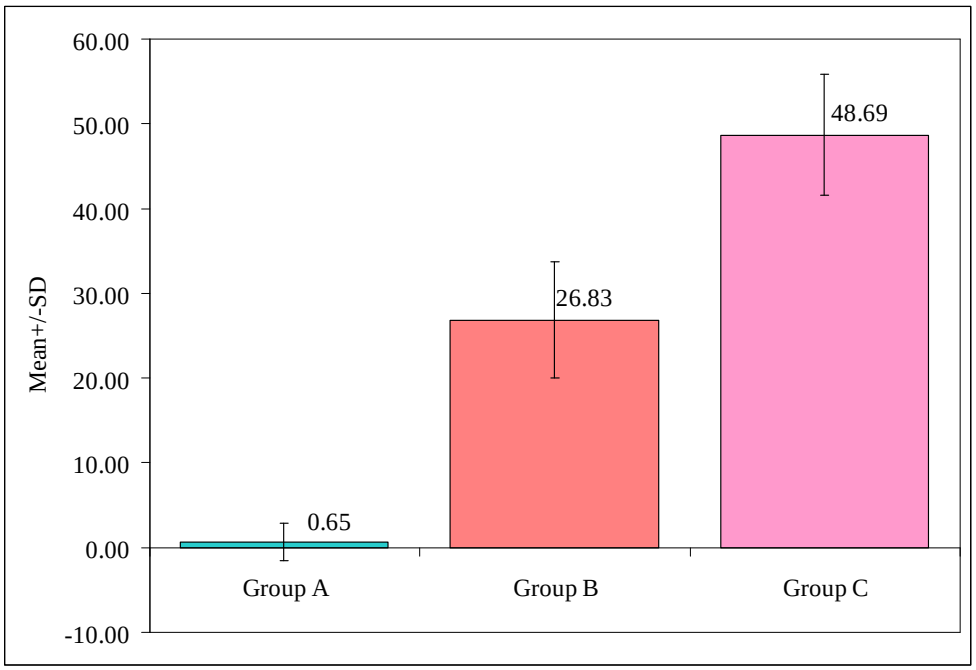
Graph-4: PD scores



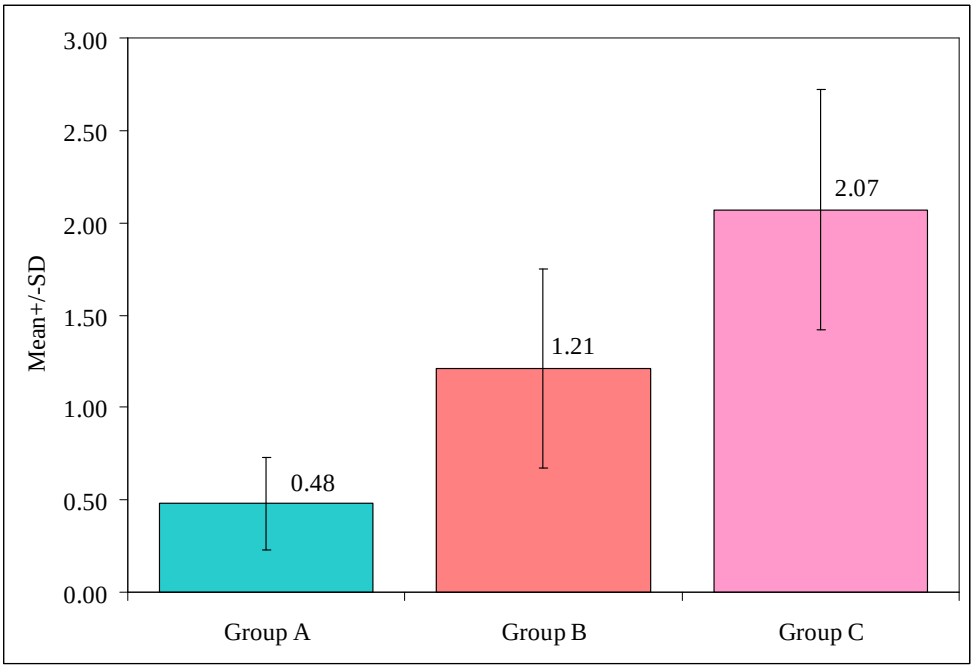
Graph-5: CAL scores



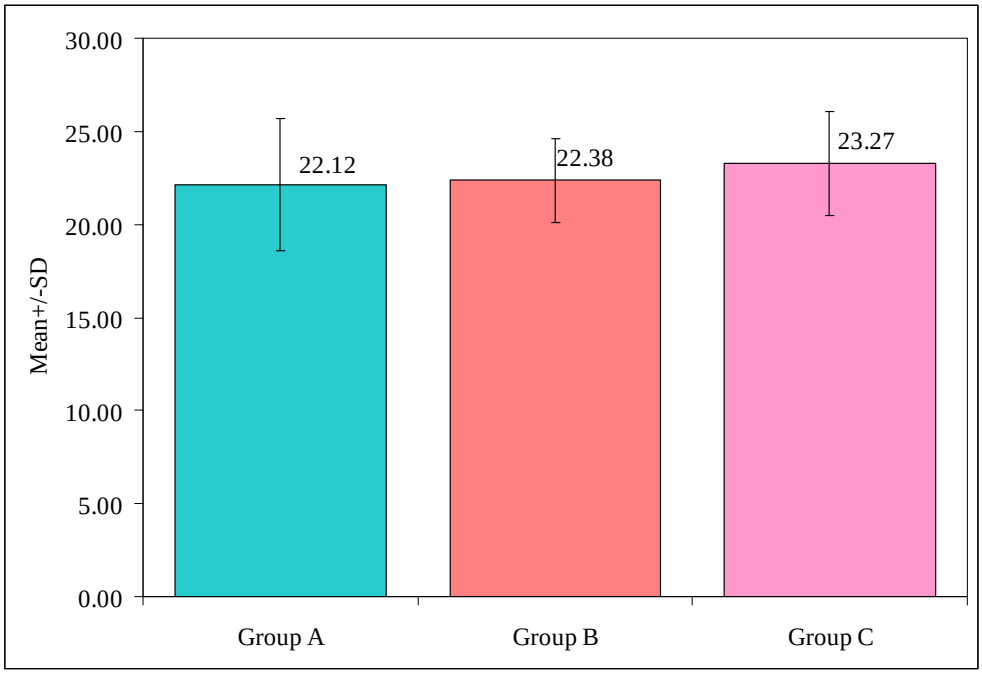
Graph-6 : RBL % scores



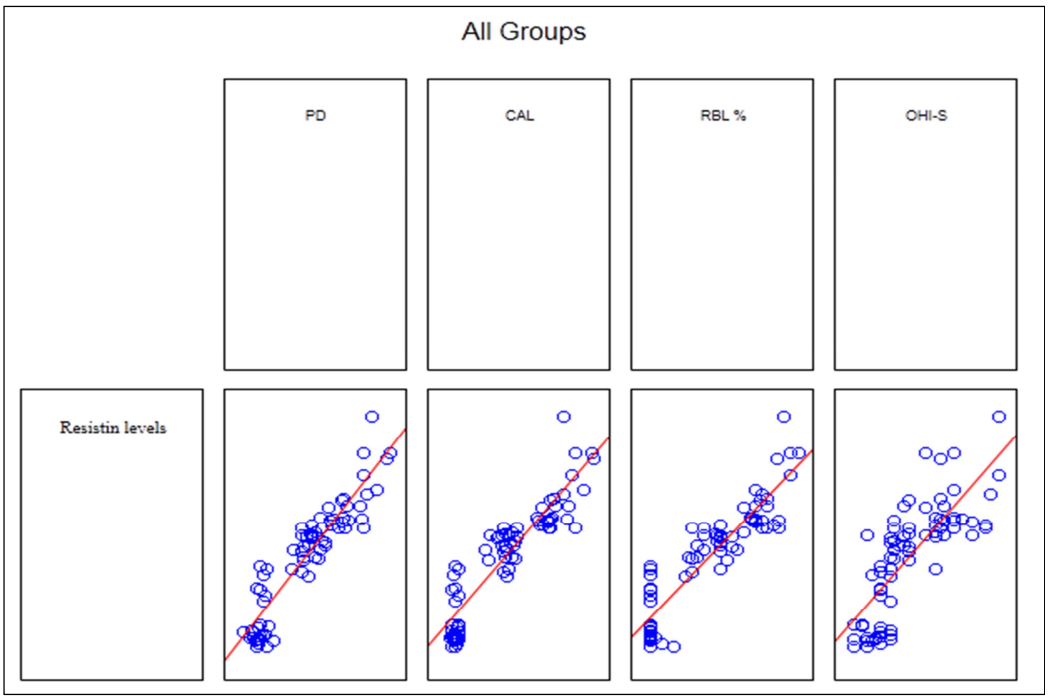
Graph-7: OHI-S scores

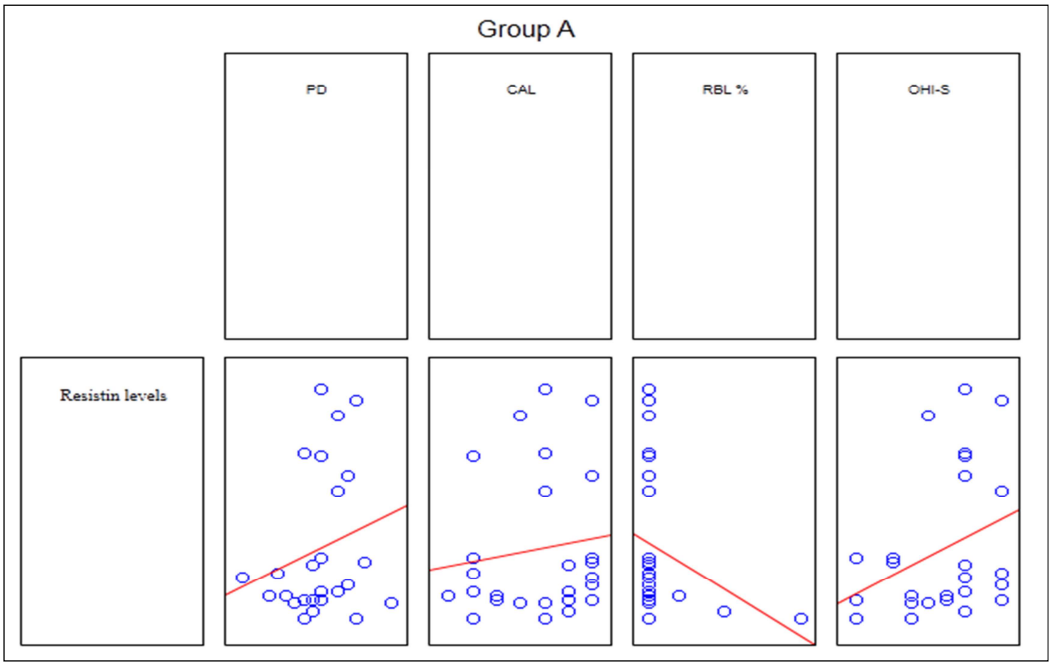


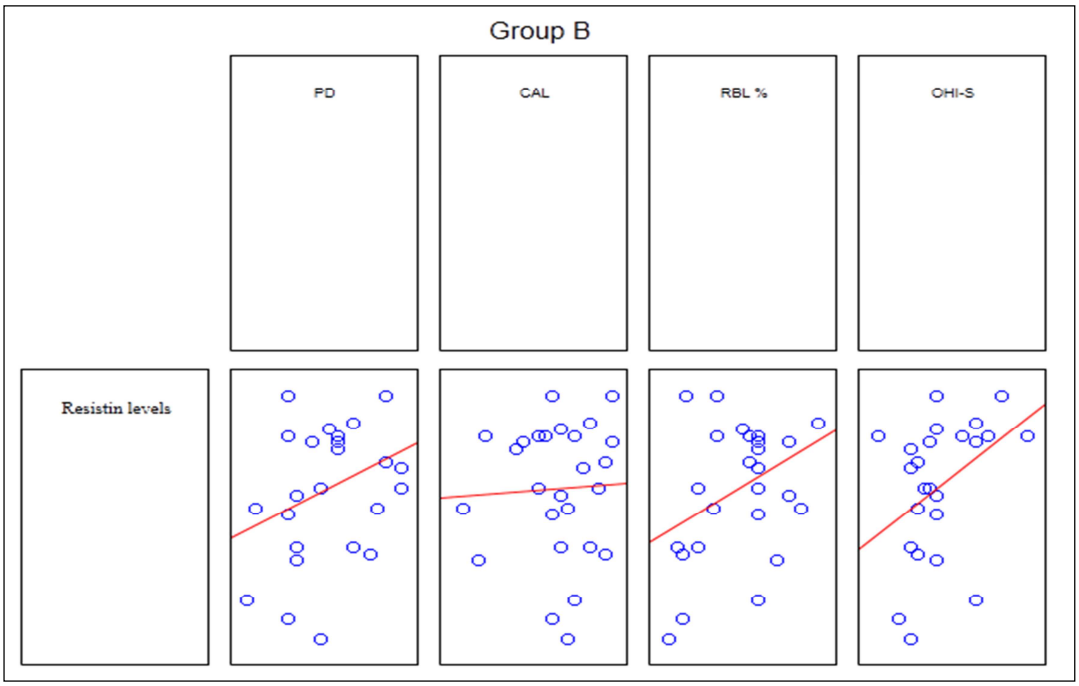
Graph-8 : BMI scores

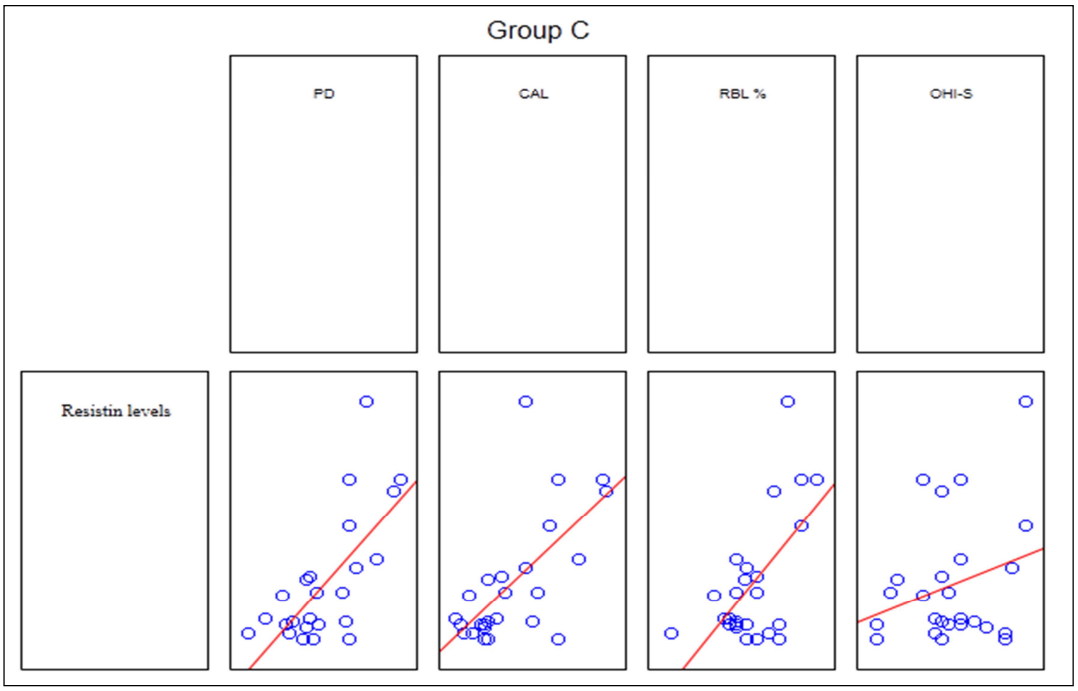


Graph-9: Correlation between Resistin levels with PD, CAL, RBL%, OHI-S and BMI scores






Group B
PD
CAL
RBL %
OHI-S
Resistin levels


Group C
PD
CAL
RBL %
OHI-S
Resistin levels

DISCUSSION

The present study evaluated the Resistin levels in individuals with periodontal health, moderate periodontitis and severe periodontitis. Resistin, a recently discovered biomarker, has shown to be associated with inflammation and inflammation related diseases. It is a secretory protein rich in cysteines that plays crucial regulatory roles in a number of biological processes, including inflammation. Human Resistin is produced in periodontal cells, macrophages, neutrophils and lymphocytes. It is strongly triggered in reaction to different proinflammatory triggers like “lipopolysaccharide (LPS)” and “TNF-α”, which are typical of periodontal disease. Several cross-sectional studies have demonstrated an elevation in the expressed levels of Resistin in serum & “Gingival crevicular fluid” among periodontitis patients as compared to healthy controls. In contrast, some studies have shown comparable Resistin levels among periodontally compromised and healthy subjects. The results regarding Resistin values present in periodontal health and disease are contradictory. There is a scarcity of literature on values of salivary Resistin in individuals with periodontal health and disease.

The study enrolled 78 participants who were systemically healthy and categorized into three categories according to certain basis ,i.e, periodontal health (Group A), moderate periodontitis (Group B), and severe periodontitis (Group C), following the 2017 world workshop classification. Demographic analysis showed no notable difference in gender distribution among the three groups in terms of male and female participants(Table 1 , Graph 1). Similarly, the mean age in the three groups was 33.38 ± 5.56, 33.08 ± 6.52, 31.46 ± 4.76 respectively, with no association between age and Resistin values in the three groups (Table 2 , Graph 2).

The findings from this study demonstrate a significant elevation in salivary Resistin levels within severe periodontitis in comparison to both the moderate periodontitis and periodontally healthy groups. This suggests a clear association between Resistin levels and the severity of periodontal disease. This relationship was further confirmed by pairwise comparisons conducted among the three groups, which showed statistically significant variations in Resistin levels. (Table 3 & 4, Graph 3)

These results align with the research conducted by Esfahrood et al (2018) [21], who conducted a case-control study aiming to assess resistin values in subjects having healthy periodontium and those with chronic periodontitis. The study revealed markedly elevated salivary resistin levels in subjects diagnosed with chronic periodontitis than in periodontally healthy individuals. The rise in salivary resistin levels can be linked to increased infiltration of inflammatory cells during periodontitis, which induces the expression of proinflammatory cytokines like TNF-α and IL-6 in response to lipopolysaccharide. This series of events contributes to the elevated salivary resistin levels observed in individuals with periodontal disease.[21]

A positive correlation was observed between resistin levels and periodontitis. This aligns with Gokhale NH et al (2014) [25], where a positive association was identified between gingival crevicular fluid (GCF) resistin levels and pocket depth (PD). This correlation suggests that the increased expression of resistin is linked to the inflammatory state of the periodontal tissue and the consequential destruction of the periodontium. The study's results reinforce the notion that resistin may play a significant role in reflecting and contributing to the inflammatory processes associated with periodontitis.[25]

A thorough examination and comparison of various periodontal parameters among three groups was done. The mean values for pocket depth (PD) in these groups were recorded as 1.99 ± 0.36, 4.90 ± 0.52, and 7.23 ± 1.12, respectively (Table 5 & 6, Graph 4). Additionally, the mean "clinical attachment level (CAL)" values, the mean RBL % values were also recorded (Tables 7, 8, 9, & 10, Graphs 5 & 6). The study demonstrated notable variations in the average values of all these periodontal parameters across the three groups. These findings emphasize the distinctive and progressive nature of periodontal conditions, with increasing severity corresponding to notable variations in "pocket depth, clinical attachment level, radiographic bone loss, and oral hygiene index scores". The observed findings are in accordance with the diagnostic criteria and multidimensional approach proposed by Tonetti MS et al (2017). [33]

The potential association and correlation of Resistin levels with periodontal health, moderate periodontitis and severe periodontitis was made, utilizing parameters such as "pocket depth (PD), clinical attachment level (CAL)" and "radiographic bone loss % (RBL)". Significant association was found between the average resistin level and the mean values of PD, CAL, and RBL% (refer to Table 15 and Graph 9). An important discovery emerged, showing a significant link between the mean resistin value and PD, CAL, and RBL% specifically in the severe periodontitis group. The same was not found in the other two groups. This indicates a positive relationship between resistin levels and clinical parameters that intensify with the severity of periodontal disease.

This is in agreement with Mittal M et al. (2015) [23] they explored the relationship between periodontitis and rheumatoid arthritis by using resistin as an inflammatory marker. Their analysis revealed a positive association between GCF resistin levels and "plaque index (PI), modified gingival index (GI), probing depth (PD), and rheumatoid factor (RF)". Similar findings were reported by Gokhale NH et al. (2014) [25], where a positive association was established between resistin and pocket depth (PD). However a study by Patel SP et al. (2013) [26] reported serum and GCF resistin concentrations did not exhibit any notable link with probing depth (PD) and clinical attachment level (CAL). Similarly, Afacan et al. (2019)[20] investigated salivary resistin and TNF-α levels in patients with aggressive periodontitis (G-AgP), chronic periodontitis (CP), and gingivitis where no association was found regarding the same.

The mean OHI-S index score, reflecting oral hygiene, was 0.48 ± 0.25, 1.21 ± 0.54, and 2.07 ± 0.65 in the three respective groups (Table 11 & 12, Graph 7). The OHI-S score did not exhibit an association with severe periodontitis. However, an overall significant correlation was identified between the total OHI-S score and the mean Resistin values. The OHI-S index assesses a patient's oral hygiene and indicates the presence of plaque deposits on the tooth surfaces. The presence of these deposits serves as an indirect indicator of disease progression.[33,36]

Across the three groups, the mean Body Mass Index (BMI) values showed no statistically significant differences (Table 13 & 14, Graph 8). This aligns with the findings by Gokhale NH et al. (2014)[25], who found no substantial variances among the groups concerning BMI. Additionally, when analyzing all samples collectively, resistin exhibited a slight negative correlation with BMI. One potential explanation for this finding lies in the fact that the present study specifically focused on individuals who were not only systemically healthy but also within the normal weight

range. This could be attributed to the relatively uniform and healthy weight status of the participants, limiting the variability in BMI values and, consequently, any potential associations with Resistin levels. [25]

LIMITATIONS

Since this study was cross-sectional, it gathered data at a singular moment in time and did not monitor the participants throughout a period of time. As a result, it could only offer an overview of the relationship between variables at that particular time. Hence, it's challenging to determine whether the Resistin levels influenced the development of periodontal disease or if periodontal disease altered the levels of Resistin. Further investigations are necessary to thoroughly assess how resistin levels may change before and after undergoing periodontal therapy, which could provide valuable insights into the potential impact of periodontal treatment on resistin levels.

Therefore, to establish causality among the levels of Resistin and periodontal status, in the future, longitudinal studies need to be conducted, which track individuals over an extended period.

SUMMARY AND CONCLUSION

The study enrolled a total of 78 participants who were then categorized into periodontal health, moderate periodontitis and severe periodontitis. Clinical examination was carried out and salivary specimens were obtained from all the subjects, followed by estimation of resistin using an "ELISA kit (Shanghai Coon Koon Biotech Co., Ltd) at department of Clinical Biochemistry (High-Tech Laboratory), KLE's Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi."

Upon evaluation, resistin exhibited an upward trend from periodontal health to severe periodontitis, suggesting an increase with the severity and progression of the disease. There was an association observed between resistin values and the clinical parameters like PD, CAL and RBL %. This correlation indicates that resistin could potentially serve as a valuable surrogate diagnostic measure for assessing periodontal health and disease progression.

This could pave the way for early detection of individuals at risk for periodontitis, facilitating prompt intervention and preventive measures. The identification of resistin as a potential biomarker opens up avenues for the development of chair-side diagnostic tools and expands future research opportunities.

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ANNEXURES- I: ETHICAL CLEARANCE CERTIFICATE

Research and Ethics Committee
KLE VK INSTITUTE OF DENTAL SCIENCES
A Constituent Unit of KLE Academy of Higher Education & Research
Accredited 'A' Grade by NAAC Placed in Category 'A' by MHRD (GoI)
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Sl. No. : 1570

CERTIFICATE

EC/NEW/INST/2021/2435
Research & Ethics Committee

This is to Certify that the synopsis titled

Estimation of Salivary Resistin level in Individuals with Healthy Periodontium and Periodontitis

Submitted by

Dr. _____ *P. G. Student /*

Staff, Guided by _____ *from Department of*

PERIODONTICS *has been critically evaluated by committee members and granted ethical clearance to conduct the above mentioned study*

Date : 11/3/24

Member Secretary
Research and Ethical Committee
KLEVK Institute of Dental Sciences
Belagavi

Research & Ethical Committee
KLEVK Institute of Dental Sciences
BELAGAVI.

Chairman
Research and Ethical Committee
KLEVK Institute of Dental Sciences
Belagavi

Chairman
Research and Ethical Committee
KLE VK Institute of Dental Sciences
Belgaum

ANNEXURE- II: BIOSTATISTICIAN CERTIFICATE

K L E VISHWANATH KATTI
INSTITUTE OF DENTAL SCIENCES
(A Constituent unit of KLE Academy of Higher Education & Research
(Formerly known as KLE University) Deemed-to-be-University u/s 3 of the UGC Act, 1956)
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Biostatistics Clearance Certificate

This is to certify that Biostatics aspect of the Dissertation/Research work of **Post Graduate Student,** under the guidance of **Professor, Department of Periodontics,** entitled **"Estimation of salivary resistin levels in individuals with healthy periodontium and periodontitis"** has been done under my guidance and completed satisfactorily.

Place: Belagavi
Date : 15/2/24

Name & Signature of Biostatistician

DR. S. B. JAVALI Ph.D.
Sr. Associate Professor in Statistics
Department of Community Medicine
USM KLE International Medical Programme
BELAGAVI-590010.

ANNEXURE- III- PLAGIARISM REPORT

Scientific Correspondence and Review Committee

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Date : 2. 04. 2024 **Serial No. :** 165

PLAGIARISM CHECK REPORT

Name of the Applicant :
UG / PG / Ph.D / Staff : POST GRADUATE
Batch & Year : 2021 - 2024
Department : PERIODONTICS

The soft copy of Research Work / Manuscript by ..... entitled

"....ESTIMATION...OF....SALIVARY...RESISTIN...LEVELS...IN.....
..INDIVIDUALS...WITH...HEALTHY...PERIODONTIUM...AND...PERIODONTITIS..."

under the guidance of ....has been submitted for Anti-Plagiarism check to the Scientific Correspondence & Review Committee of KLE VK Institute of Dental Sciences using "Turn-it-in" software.

The scan has been carried out and the scanned output reveals a Similarity Index of ....5.....%, which is **within / not within** the acceptable limits of 10% as per the UGC guidelines.

2/04/2024

Member Secretary
Scientific Correspondence and Review Committee
KLEVK Institute of Dental Sciences
KAHER-Belagavi

Chairman
Scientific Correspondence and Review Committee
KLEVK Institute of Dental Sciences
KAHER - Belagavi

ANNEXURE-IV- CONSENT FORM

CONSENT FORM

DEPARTMENT OF PERIODONTICS

KLE V.K. INSTITUTE OF DENTAL SCIENCES

BELAGAVI.

ESTIMATION OF SALIVARY RESISTIN LEVELS IN INDIVIDUALS WITH HEALTHY PERIODONTIUM AND PERIODONTITIS

Principal Investigator:

I _____, aged _____ years have been informed about my involvement in the study.
I agree to give my personal details like Name, Age, Gender, Residential Address, past and Present dental history and any other details if required for the study to the best of my knowledge.
I will co-operate with the dentist.
I will follow the instructions given by the dentist during study.
I permit the dentist to utilize the information given by me and the results obtained from this study for presentation and publication without disclosing my identity.
I have been informed that saliva sample will be taken from me and I will be exposed to radiation as radiographs will be taken, which will be used for the study. I permit the dentist to perform the same.
If by chance any complications arise during the above said procedure, I permit the dentist to take necessary actions to prevent the same.
In my full consciousness and presence of mind, after understanding all the procedures and related complications if any, in my vernacular language, I am willing and give my consent to participate in this study.

Date:

Name of the Patient:

Signature:

Address & Ph. No:

Name of witness/guardian:

Signature:

DEPARTMENT OF PERIODONTICS

KLE V.K. INSTITUTE OF DENTAL SCIENCES

BELAGAVI.

ESTIMATION OF SALIVARY RESISTIN LEVELS IN INDIVIDUALS WITH HEALTHY PERIODONTIUM AND PERIODONTITIS

Principal Investigator:

मी, _____ , वय ____ वर्षे, मला ह्या अभ्यासाबद्दल पूर्ण कल्पना देण्यात आली आहे.

मी माझी वैयक्तिक माहिती जसे की नाव, वय, लिंग, पत्ता, मागील व सध्याची दंत उपचाराची माहिती व अन्य तपशील देण्यास सहमत आहे.

मी दंत चिकित्सकांना त्यांच्या अभ्यासासाठी पूर्ण सहकार्य करेन.

दंतचिकित्सकांचा अभ्यास चालू असताना, मी त्यांनी दिलेल्या सर्व सूचनांचे पालन करेन.

दंत चिकित्सकांच्या अभ्यासदरम्यान त्यांनी प्राप्त केलेली माझी सर्व माहिती व अभ्यासाचे परिणाम माझी ओळख लपवून कुठल्याही प्रकाशनात सादर करायला माझी परवानगी आहे.

मला कळविण्यात आले आहे की माझ्याकडून लाळेचा नमुना घेतला जाईल आणि रेडिओग्राफ्स घेतले जातील म्हणून मला किरणोत्सर्गाचा सामना करावा लागेल, ज्याचा उपयोग अभ्यासासाठी केला जाईल. मी दंतवैद्याला असे करण्याची परवानगी देतो

वर दिलेल्या प्रक्रियेत, जर कधी चुकून काही झालच तर मी दंत चिकित्सकांना योग्य तो उपाय करण्याची परवानगी देत आहे.

मी पूर्ण शुद्धीत व माझ्या मनाच्या जागृत अवस्थेत, सर्व प्रक्रिया व त्यांचे क्वचित होऊ शकणारे दुष्परिणाम समजून, माझ्या मातृभाषेत ह्या अभ्यासात सहभागीहोण्यास संमती देतो/देते.

तारीख :-

पत्ता व दूरध्वनी क्रमांक :-

स्वाक्षरी :-

DEPARTMENT OF PERIODONTICS

KLE V.K. INSTITUTE OF DENTAL SCIENCES

BELAGAVI.

ESTIMATION OF SALIVARY RESISTIN LEVELS IN INDIVIDUALS WITH HEALTHY PERIODONTIUM AND PERIODONTITIS

Principal Investigator:

ನಾನು _____ ವಯಸ್ಸಿನ _____ ವರ್ಷಗಳ ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನ ತೊಡಗಿರುವ ಬಗ್ಗೆ ಮಾಹಿತಿ ಮಾಡಲಾಗಿದೆ.
ನಾನು ಹೆಸರು, ವಯಸ್ಸು, ಲಿಂಗ, ವಸತಿ ವಿಳಾಸ, ಹಿಂದಿನ ಮತ್ತು ಪ್ರಸೆಂಟ್ ಹಲ್ಲಿನ ಇತಿಹಾಸ ಮತ್ತು ನನ್ನ ಜ್ಞಾನದ ಅತ್ಯುತ್ತಮ ಅಧ್ಯಯನಕ್ಕೆ ಬೇಕಾಗುವ ಯಾವುದೇ ಇತರ ವಿವರಗಳು ಹಾಗೆ ನನ್ನ ವೈಯಕ್ತಿಕ ವಿವರಗಳನ್ನು ನೀಡಲು ಒಪ್ಪುತ್ತೀರಿ.
ದಂತವೈದ್ಯ ನಾನು ಕಾಣಿಸುತ್ತದೆ ಸಹಕಾರ.
ನಾನು ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ದಂತವೈದ್ಯ ನೀಡಿದ ಸೂಚನೆಗಳನ್ನು ಅನುಸರಿಸಿ.
ನಾನು ನನ್ನ ಗುರುತನ್ನು ಬಹಿರಂಗಪಡಿಸದೇ ನೀಡಿದ ಮಾಹಿತಿ ಮತ್ತು ಪ್ರಸ್ತುತಿ ಮತ್ತು ಪ್ರಕಟಣೆಗೆ ಈ ಅಧ್ಯಯನದಿಂದ ಪಡೆದ ಫಲಿತಾಂಶಗಳನ್ನು ಬಳಸಿಕೊಳ್ಳಲು ದಂತವೈದ್ಯ ಅನುಮತಿ.
ನನ್ನಿಂದ ಲಾಲಾರಸದ ಮಾದರಿಯನ್ನು ತೆಗೆದುಕೊಳ್ಳಲಾಗುವುದು ಮತ್ತು ರೇಡಿಯೋಗ್ರಾಫ್ ಗಳನ್ನು ತೆಗೆದುಕೊಳ್ಳುವುದರಿಂದ ನಾನು ವಿಕಿರಣಕ್ಕೆ ಒಡ್ಡಿಕೊಳ್ಳುತ್ತೇನೆ ಎಂದು ನನಗೆ ತಿಳಿಸಲಾಗಿದೆ, ಇದನ್ನು ಅಧ್ಯಯನಕ್ಕಾಗಿ ಬಳಸಲಾಗುತ್ತದೆ. ನಾನು ದಂತವೈದ್ಯರಿಗೆ ಅದೇ ರೀತಿ ಮಾಡಲು ಅನುಮತಿಸುತ್ತೇನೆ

ನಾನು ಯಾವುದೇ ಸಂಸ್ಥೆ ಪ್ರಾಯೋಜಿಸುತ್ತಿದೆ ಮಾಡಲಾಗುತ್ತಿದೆ ಸಹ, ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಸಹಕಾರವನ್ನು ಯಾವುದೇ ಆದಾಯ ಮಾತನ್ನಾಡುವುದಿಲ್ಲ. ನನ್ನ ಸ್ವಂತ ಇಚ್ಛೆಯ ಮತ್ತು ಇಚ್ಛೆ ಭಾಗವಹಿಸುವ ನಾನು.
ಯಾವುದೇ ಕಾರಣಕ್ಕೆ ನಾನು ಅಪರಿಚಿತ ಕಾರಣಗಳಿಗಾಗಿ, ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಸಾಧ್ಯವಾಗುವುದಿಲ್ಲ ಏಕೆ ವೇಳೆ, ನಾನು ಸಮಯ ಯಾವುದೇ ಹಂತದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹಿಂದಕ್ಕೆ.
ನನ್ನ ಪೂರ್ಣ ಪ್ರಜ್ಞೆ ಮತ್ತು ಮನಸ್ಸಿನ ಉಪಸ್ಥಿತಿಯಲ್ಲಿ, ನನ್ನ ಸ್ಥಳೀಯ ಭಾಷೆಯಲ್ಲಿ ಎಲ್ಲಾ ಕಾರ್ಯವಿಧಾನಗಳು ಮತ್ತು ಸಂಬಂಧಿತ ತೊಡಕುಗಳು ಇದ್ದಲ್ಲಿ ಅದನ್ನು ಅರ್ಥಮಾಡಿಕೊಂಡ ನಂತರ, ನಾನು ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಸಿದ್ಧನಿದ್ದೇನೆ ಮತ್ತು ನನ್ನ ಸಮ್ಮತಿಯನ್ನು ನೀಡುತ್ತೇನೆ

ದಿನಾಂಕ:
ದಂತವೈದ್ಯ ಹೆಸರು: ಸಹಿ:
ರೋಗಿಯ ಹೆಸರು: ಸಹಿ:
ವಿಳಾಸ ಮತ್ತು ದೂರವಾಣಿ ಸಂಖ್ಯೆ.:
ಸಾಕ್ಷಿ / ಪೋಷಕರ ಹೆಸರು: ಸಹಿ

ANNEXURE-V- PROFORMA

DEPARTMENT OF PERIODONTICS

KAHER'S KLE's V.K. INSTITUTE OF DENTAL SCIENCES

BELAGAVI.

ESTIMATION OF SALIVARY RESISTIN LEVELS IN INDIVIDUALS WITH HEALTHY PERIODONTIUM AND PERIODONTITIS

Case No: OPD No:

Name:

Age: Sex: Occupation:

Address:

Chief Complaint:

Medical History:

Dental history:

Clinical Examination :

Tooth loss (due to periodontitis) :

Probing Depth (PD) :-

8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8

Clinical Attachment Level (CAL) :-

8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8

OHI-S index:

Debris index- simplified (DI-S)

16	11	26
46	31	36

Calculus index-simplified (CI-S)

16	11	26
46	31	36

OHI-S = DI-S + CI-S =

Radiographic bone loss (%) :

Body Mass Index (BMI) = $\frac{\text{Weight (kg)}}{\text{Height } (m^{2})}$

3. <u>Estimation of Resistin levels:</u>

Biochemical Parameter	Reading of Resistin (ng/ml)
Saliva Sample	

ANNEXURE VI- MASTER CHART
Group – A (Periodontal Health)

S.No.	Gender	Age	PD	CAL	RBL %	OHI-S	BMI	Resistin level (ng/ml)
1	M	26	1.6	0.6	2	0.5	23	1.2
2	F	22	1.9	0.9	5	0.6	18	0.8
3	F	37	1.8	0.5	0	0	22	0.6
4	F	31	2.4	0.8	10	0.3	23	0.6
5	M	29	1.9	0.9	0	0.6	16	2
6	M	30	2.3	1	0	0.8	22	1.5
7	F	41	2	1	0	0.2	15	2.2
8	M	39	1.5	0.5	0	0.8	26	1.8
9	M	41	2.2	0.8	0	0.8	23	4
10	F	40	2.5	1	0	0.2	27	2.1
11	F	33	2.8	0.8	0	0.3	27	1
12	M	22	1.9	0.9	0	0	22	1.1
13	M	31	2	0.6	0	0.5	24	1.1
14	M	33	2.2	0.9	0	0.6	26	1.3
15	F	29	1.4	0.4	0	0.3	18	1.2
16	M	41	2	0.5	0	0	22	2.2
17	F	33	1.1	1	0	0.6	22	1.7
18	M	30	1.8	1	0	0.8	19	1.1
19	F	43	1.7	0.7	0	0.4	21	1
20	M	36	2	0.5	0	0.6	22	1.3
21	F	33	2.3	1	0	0.6	28	4.4
22	F	33	2	0.5	0	0.6	25	4.9
23	M	35	1.8	0.8	0	0.6	15	5
24	F	37	2.2	0.7	0	0.4	23	6
25	F	31	2.4	1	0	0.8	22	6.4
26	M	32	2	0.8	0	0.6	24	6.7

Group - B (Moderate periodontitis)

S.No.	Gender	Age	PD	CAL	RBL %	OHI-S	BMI	Resistin level (ng/ml)
1	F	33	4.8	3.8	20	1	23	8.2
2	M	35	5.5	4.2	22.5	0.9	22	7.9
3	F	44	5	3.5	30	0.8	22	8.8
4	M	45	5.8	4.6	30	1.1	22	8.2
5	M	33	5.8	4.4	30	0.8	24	8.5
6	F	32	4.8	4.2	15	0.8	19	5.9
7	M	37	5.4	4.7	17.5	0.9	24	7.2
8	M	30	4.5	4.1	20	0.8	22	7.3
9	M	29	5	3.8	23	0.3	24	9
10	M	31	4.4	4	17.5	0.6	21	6.2
11	M	45	5.2	4.5	16.5	0.8	22	7.3
12	M	34	4	2.8	37	0.9	26	7.9
13	M	25	5	4.8	30	1.1	24	8.9
14	F	32	4.5	4.1	35	1.2	22	8.1
15	M	22	4.4	4	30	1.2	22	7.8
16	M	37	4.4	4	23	1.2	26	9.6
17	M	22	4.5	3	33	1.2	19	7.1
18	F	35	3.9	4.3	30	1.8	22	6.5
19	F	26	4.4	3.1	30	2	26	9
20	M	33	5	4.3	28.5	2.6	18	9
21	M	37	5.6	4.8	18	2.2	18	9.6
22	M	27	5	3.9	30	1.6	25	9
23	M	30	5.6	4.7	28.5	0.9	22	8.6
24	M	43	4.9	4.1	27.5	1.2	24	9.1
25	M	37	4.7	3.6	35	1.8	22	8.9
26	M	26	5.2	4.5	40	1.8	21	9.2

Group – C (Severe periodontitis)

S.No.	Gender	Age	PD	CAL	RBL %	OHI-S	BMI	Resistin level (ng/ml)
1	F	40	7.8	8.2	45	1.1	25	11.2
2	M	31	8	8.7	63.5	1.6	26	15.2
3	M	28	7.9	8.1	45	1.9	26	10.2
4	M	31	8	8.7	55	0.9	21	9.6
5	M	32	6.7	7	47	1.2	20	11.7
6	F	37	7.1	6.9	55	0.9	23	10.1
7	M	31	6.8	7.3	50	1.9	25	11.8
8	M	30	8.8	9.2	45	2.2	25	12.4
9	F	28	6.6	6.9	50	2.9	16	9.6
10	F	34	6.9	7	47.5	1.9	25	9.6
11	F	37	8.5	7.9	57	3.2	22	18
12	F	23	6.7	6.9	45	2.6	22	10
13	F	25	6	6.5	40	1.6	22	11.1
14	M	30	5	6.4	30	1.8	27	9.8
15	M	28	6.1	6.8	45	2.2	27	10.1
16	M	33	7.1	6.3	43.5	2.2	24	10.1
17	M	40	7	7.4	50	2	24	11.2
18	M	32	6.3	7	45	2.4	22	10.2
19	F	22	6.8	7.2	43.5	2.2	22	10.3
20	M	31	5.5	6.2	42.5	1.8	20	10.3
21	M	33	7.1	6.9	47.5	2	21	10.1
22	M	29	6.2	6.6	52.5	2.9	21	9.8
23	F	32	8.2	7.9	45-50	3	22	12.1
24	M	41	8	8.5	60	3.2	22	13.6
25	M	29	9.3	9.9	54	1.9	27	14.8
26	F	31	9.5	9.8	60	2.2	28	15.2