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The effect of initial periodontal treatment on gingival crevicular fluid galectin-3 levels in participants with periodontal disease

M Karsiyaka Hendek,* E Olgun,* U Kisa†

*Department of Periodontology, Faculty of Dentistry, Kirikkale University, Kirikkale, Turkey.
†Department of Biochemistry, Faculty of Medicine, Kirikkale University, Kirikkale, Turkey.

ABSTRACT

Background: The aim of study was to evaluate galectin-3 levels in gingival crevicular fluid (GCF) from periodontally healthy (H) patients and those with periodontitis (P), gingivitis (G) and the effect of initial periodontal treatment on GCF galectin-3 level.
Methods: A total of 75 participants, 25 patients with periodontitis, 25 with gingivitis and 25 periodontally healthy subjects were included into the study. Patients with periodontal disease received initial periodontal treatment. GCF galectin-3 level was assessed at baseline and at the 6th-8th weeks after completion of periodontal treatment. GCF galectin-3 level was evaluated by enzyme-linked immunosorbent assay.
Results: GCF galectin-3 level was the lowest in the H group (102.31[63.07] μg/30 s), followed by the G group (241.45 [145.89] μg/30 s) and the highest in the P group (338.27[219.37] μg/30 s). These differences were statistically significant between H and the other groups ($P < 0.001$). After initial periodontal treatment, GCF galectin-3 level significantly decreased in the G and P groups compared to baseline values ($P < 0.01$).
Conclusion: The results of this study suggest that GCF galectin-3 level is a potential biomarker for the evaluation of gingival inflammation and initial periodontal treatment is effective in decreasing GCF galectin-3 level.

Keywords: Galectin-3, initial periodontal treatment, periodontal disease.

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INTRODUCTION

Periodontitis is an inflammatory disease that affects the supporting tissues of the tooth. The interaction between bacterial plaque and immune response plays an important role in the pathogenesis of periodontitis. Disruption of the balance between host destruction and defense mechanism results in bone destruction and tooth loss.[1] Galectins are a family of proteins with small molecular weight that are involved in cell growth and activation. Galectins have been proposed to be regulators of immune cell homeostasis regarding adaptive immune responses.[2] In this context, B-cell maturation and differentiation can be modulated by interactions compromised by galectins both at the central and peripheral immune compartments.[3] Similarly, galectins also exert regulatory functions in T-cell homeostasis.[4] Given the regulatory roles of galectins on cells that mediate both innate and adaptive immune responses, their effects can be both beneficial or detrimental to pathological conditions associated with an exacerbated or depressed immune function, such as inflammatory, allergic and autoimmune disorders and cancer.[5] Galectin-3, a member of this family, is a galactose-specific protein with a molecular weight of about 30-kD. Galectin-3, a member of the galectin family, is a proinflammatory protein and is involved in T cell-mediated inflammation.[6] Galectin-3 is widely expressed in all human tissues, including all immune cell types (macrophages, monocytes, dendritic cells, eosinophils, mast cells, natural killer cells and activated T and B cells), epithelial cells, endothelial cells, and sensory neurons.[7,8] Galectin-3 is predominantly located in the cytoplasm and moves towards the nucleus. It is also released to the cell surface and biological fluids.[7] In recent studies, galectins have been shown to play a key role in the homeostasis of immune cells and inflammation.[9,10] In the nucleus, galectin-3 supports pre-mRNA splicing and regulates gene transcription; extracellular galectin-3 modulates cell-cell interactions between

epithelial cells and the extracellular matrix. Thus, it takes part in cell differentiation, inflammation, fibrogenesis and host defense.[7,11] Therefore, galectin-3 plays a role in many biological activities, including cell growth, apoptosis, differentiation, transformation, angiogenesis, inflammation, fibrosis, and host defense. Previous evidence has shown that galectin-3 is involved in the pathogenesis of various autoimmune and inflammatory processes as well as cardiovascular diseases.[8,12–20] In bone-related tissues, galectin-3 is expressed by chondrocytes, osteoblasts and osteoclasts,[21–23] where it may serve adjunctive functions in bone remodelling. In rats, galectin-3 reportedly inhibited ex vivo osteoclastogenesis and decreased osteoclast numbers in a rat model of adjuvant-induced arthritis.[24] In another study, extracellular recombinant galectin-3 inhibited terminal differentiation of a human pre-osteoblast cell line.[25] Simon et al. (2017) suggested the secretion of galectin-3 as a novel mechanism for osteoblasts to control osteoclastogenesis and to maintain trabecular bone homeostasis independently of the RANKL/OPG-axis.[26] To the best of our knowledge, there is no study to evaluate the galectin-3 level in gingival crevicular fluid (GCF) in patients with periodontal disease. Based on this, the study aimed to compare galectin-3 level in GCF in periodontitis, gingivitis and periodontal healthy individuals and to evaluate the effect of initial periodontal treatment on GCF galectin-3 level. The hypothesis of the study was that 'GCF galectin-3 level decreases after initial periodontal treatment of periodontal diseases.

MATERIAL AND METHODS

A total of 75 participants (14 females and 11 males, aged 36–64 years [mean ± SD = 45.44 ± 5.88 years] with P; 11 females and 14 males, aged 26–39 years [mean ± SD = 32.12 ± 3.21 years] with G, 15 females and 10 males, aged 22–38 years [mean ± SD = 30.56 ± 3.94 years] with H) screened from July 2018 to June 2019 were selected. After the participants were informed about the aim and methods of the study, written informed consent was obtained from each one. The study was approved by the local ethics committee. (Date: 12.06.2018 Number: 14/02).

The periodontitis group included 25 patients with Stage II-III Grade B periodontitis. These individuals had a minimum of two non-adjacent teeth with sites with 3–4 mm or ≥5 mm interdental clinical attachment level (CAL) and 15% to 33% or extending to middle third of root and beyond radiographic bone loss.[27] A patient with an intact periodontium was diagnosed as a gingivitis case (defined as a patient presenting with a BOP score ≥10% without attachment loss and radiographic bone loss) or a patient with a reduced periodontium but without a history of periodontitis (e.g. gingival recession), CAL <3 mm and a BOP score ≥10% was included into the gingivitis group (n = 25).[28] The periodontally healthy group (n = 25) consisted of individuals with no attachment loss (due to periodontal disease), PD ≤3 mm, and whole-mouth bleeding scores <10%. Individuals who had any systemic diseases, smokers and alcohol consumer, had received any periodontal treatment in the last 6 months, took any antibiotics, anti-inflammatory drugs, regularly and pregnant or lactating were excluded.

Periodontal clinical parameters and periodontal treatment protocol

Periodontal status of each individual included into the study was determined by measuring plaque index (PI),[29] gingival index (GI),[30] PD and CAL. PD and CAL were measured on six sites of the teeth with a periodontal probe. Panoramic radiographs were used to determine the alveolar bone loss. The total number of teeth for each participant was ≥20. All clinical measurements were performed by a single investigator (EO). Within 14 days from the screening visit, scaling and root planing under local anaesthesia using manual and ultrasonic devices in a single appointment were performed by a different operator (MKH) and oral hygiene instructions were given to all participants with periodontal disease.

All periodontal clinical measurements and samples were recorded at baseline and the 6th-8th week after the periodontal treatment in patients with P and G and at one time point (baseline) in H group.

GCF collection and preparation of samples

In the periodontitis group, the GCF samples were collected from single-rooted teeth with ≥4 and <7 mm PD. In G group, they were collected from teeth with GI ≥2. In periodontally healthy individuals, first incisors and/or canine teeth providing a total of two sites were determined for sampling. Two GCF samples were collected from each patient. After the sample sites were isolated with cotton rolls and slightly air-dried to avoid contamination, the standardized strips kept in the sulcus for 30 s to collect GCF and volume was measured on a precalibrated device. The level of GCF galectin-3 was measured by Enzyme-Linked ImmunoSorbent Assay (ELISA) using commercial kits (Cloud-Clone, Katy, USA). The minimum detectable dose of galectin-3 is typically less than 0.060 ng/mL and the assay range of test was 0.156–10 ng/mL.

Statistical analysis

To achieve 95% power (effect size, $f = 0.25$) and detect differences among groups, 22 participants in each group were required. To protect from possible dropouts, the sample size was increased to 25 participants per group. Number and percentage (n, %) were used in the representation of categorical variables. The female-male ratio in the groups was compared with the chi-square test. The Shapiro–Wilk test was used to examine the normality of the data distribution. The differences among three groups were investigated with the Kruskal–Wallis test. Post-hoc binary comparisons were made with the Bonferroni-corrected Mann–Whitney test to identify the differences. The variables before and after treatment in the groups were compared using the Wilcoxon signed-rank test. Multiple logistic regression was employed to identify explanatory variables for GCF galectin-3. SPSS IBM Statistics version 22.0 (Armonk, NY, USA) was used for statistical analysis. Statistical significance level was considered at $P < 0.05$.

RESULTS

With regard to demographic data, sex distribution of participants was similar among groups ($P = 0.428$), and patients with P were significantly older than the other groups ($P < 0.001$) (Table 1).

Periodontal clinical parameters at baseline and after periodontal treatment

The values of the periodontal clinical parameters (full mouth and sampled teeth) are shown in Table 2. At baseline, the PI and GI scores in periodontally healthy individuals were significantly lower than the other groups ($P < 0.001$). PD and CAL values were significantly higher in P groups than the other groups ($P < 0.001$). In P group, all clinical periodontal parameters significantly decreased from baseline to the 6th-8th week ($P < 0.001$). In G group, PI, GI and PD significantly decreased after the treatment compared to baseline ($P < 0.001$) (Table 2).

GCF galectin-3 level at baseline and after treatment

At baseline, the total amount of GCF galectin-3 was significantly lower in periodontal healthy group than the other two groups ($\chi^2 = 39.125$, $P < 0.001$) and there was no significant difference between P and G groups. After periodontal treatment, in both groups, the total amount of GCF galectin-3 significantly decreased from baseline to the 6th–8th week (Table 3). The multivariate regression analysis showed that GI ($P = 0.027$) was significant predictors of GCF galectin-3 (Table 4).

DISCUSSION

The periodontium is an adaptive dynamic tissue that can maintain different microbiological, inflammatory and mechanical states through a series of complex molecular events, including growth factors, transcription factors, and extracellular matrix proteins.[31] In this study, it was found that galectin-3, a proinflammatory protein that plays a role in T-cell inflammation, increased in the presence of periodontal disease and decreased with treatment.

A new revised periodontitis case definition system was introduced in 2018 to define periodontitis more appropriately by its stage (the severity and extent of periodontal tissue destruction and complexity of management) and grade (the future risk of progression). One of its main goals was to provide dentists a framework for individualized identification, treatment and prevention of periodontitis in patients. In addition, it provides a necessary and very much needed framework for the introduction of diagnostic and prognostic biomarkers to extend and improve the information provided by the standard clinical measures. Several fluids have been studied intensively to find suitable biomarkers from oral fluids for the diagnostics of periodontitis.[27]

Table 1. Demographic characteristics of the groups

	Groups			*P*
	Healthy (n = 25)	Gingivitis (n = 25)	Periodontitis (n = 25)	
Age (years)				<0.001
Mean ± SD	30.56 ± 3.94	32.12 ± 3.21	45.44 ± 5.88*	
Min–Max	22–38	26–39	36–64	
Sex (n (%))				0.428
Female	15 (%60.0)	11 (%44.0)	14 (%56.0)	
Male	10 (%40.0)	14 (%56.0)	11 (%44.0)	

*Significant differences compared to the other groups.

Table 2. Comparison of periodontal clinical parameters at baseline and after treatment among groups

	Healthy (n = 25)	Gingivitis (n = 25)	Periodontitis (n = 25)	χ^2	*P*
Plaque Index					
Baseline					
Full mouth	0.39 ± 0.16	1.33 ± 0.22*	1.23 ± 0.33*	48.862	<0.001
Sampled Sites	0.25 ± 0.19	1.13 ± 0.23*	1.50 ± 0.43*	53.431	<0.001
6th–8th week					
Full mouth		0.54 ± 0.32‡	0.70 ± 0.38‡	1.954	0.162
Sampled Sites		0.20 ± 0.15‡	0.56 ± 0.38‡	10.648	0.001
Gingival Index					
Baseline					
Full mouth	0.40 ± 0.11	1.76 ± 0.28*	1.66 ± 0.21*	51.031	<0.001
Sampled Sites	0.27 ± 0.15	1.88 ± 0.21*	1.86 ± 0.20*	52.526	<0.001
6th–8th week					
Full mouth		0.71 ± 0.42‡	1.07 ± 0.29‡	5.376	0.020
Sampled Sites		0.32 ± 0.17‡	0.86 ± 0.25‡	7.281	0.007
Probing depth(mm)					
Baseline					
Full mouth	1.80 ± 0.25	2.79 ± 0.48	5.43 ± 0.82*,†	64.665	<0.001
Sampled Sites	1.48 ± 0.25	2.92 ± 0.50	5.56 ± 0.43*,†	65.765	<0.001
6th–8th week					
Full mouth		2.27 ± 0.21‡	3.18 ± 0.67‡	13.887	<0.001
Sampled Sites		2.41 ± 0.49‡	3.37 ± 0.35‡	24.812	<0.001
Clinical attachment level (mm)					
Baseline					
Full mouth	0	0	5.54 ± 0.30*,†	70.114	<0.001
Sampled Sites	0	0	5.92 ± 0.51*,†	70.137	<0.001
6th–8th week					
Full mouth		0	3.21 ± 0.60‡	32.698	<0.001
Sampled Sites		0	3.61 ± 0.54‡	32.732	<0.001

*Significant differences compared to the periodontally healthy group.
†Significant differences compared to the gingivitis group.
‡Significant differences compared to the baseline values.

Table 3. Comparison of GCF galectin-3 level (μg/30 s) at baseline and after treatment among groups (mean ± sd)

	Groups			χ^2	*P*
	Healthy (n = 25)	Gingivitis (n = 25)	Periodontitis (n = 25)		
Galectin-3 (μg/30 s)					
Baseline	102.31 ± 63.07	241.45 ± 145.89*	338.27 ± 219.37*	39.125	<0.001
6th–8th week		99.90 ± 93.04†	114.86 ± 85.16†	4.706	0.095

*Significant differences compared to the periodontally healthy group.
†Significant differences compared to the baseline values.

Table 4. Multivariate linear regression analysis for GCF Galectin-3 in all patients

	Variables	Multivariate model	
		β	*P*
GCF galectin-3	Plaque index	−28.935	0.652
	Gingival index	112.564	0.027
	Probing depth	0.269	0.993
	Clinical attachment level	20.356	0.239

In addition to chronic inflammation, galectin-3 has been shown to play a role in acute inflammation, activated T lymphocyte proliferation, and the adhesion of neutrophils on the endothelium. In addition, galectin-3 plays a role in many biological activities such as cell growth, apoptosis, differentiation, transformation, angiogenesis, inflammation and fibrosis.[10,32–34] It has been suggested that plasma galectin-3 concentrations correlate with markers of oxidative stress and inflammation and may be a good biomarker of atherosclerosis-related diseases. It has been shown that galectin-3 can increase endothelial dysfunction by inducing inflammation and serum galectin-3 can be useful as a marker of cardiac function and cardiovascular fibrosis.[35–37] Galectin-3, which acts as a receptor for advanced glycation end products, has been shown to play an important role in the progression of prediabetes to diabetes as a proinflammatory molecule that causes beta cell dysfunction and insulin resistance.[13,38,39] It has been shown that galectin-3 expression is increased in synovial fibroblasts in patients

with rheumatoid arthritis, and serum galectin-3 level is found to be high in Behçet's disease. It has been found that galectin-3 and its glycoligands are strongly increased in dermal capillaries in psoriatic skin samples. It has been pointed out that the increase in the expression of galectin-3 may cause the rearrangement of the capillary network and changes in structure and function.[40–42] It has become that galectins also bind glycans on the surface of potentially pathogenic microbes and parasitic worms, and mediate recognition and effector functions in innate immunity.[43] Recently, galectins have been discovered to bind glycans on the surface of viruses, bacteria, protista and fungi.[44] Thus, the potential role of galectins as pattern recognition receptors has become an area of increased attention. Galectin-3 acts as chemokine, induces monocyte and macrophage migration, contributes to the development of natural and adaptive immune responses, and participates in phagocytosis of macrophages and some microorganisms through intracellular mechanisms, supporting that it may be a positive regulator of the inflammatory response.[45] In an animal model, it was examined a possible effect of galectin-3, as a factor in DM and bone metabolism, on periodontitis with or without DM and shown that osteocyte-derived exosomes carrying miR-124-3p may regulate galectin-3 expression of osteoblasts, especially under high-glucose conditions, suggesting a possible mechanism for DM-related alveolar bone pathologies.[46] In this study, the amount of GCF galectin-3 reached the highest values in periodontitis and the lowest values in periodontal health. In addition, it decreased with initial periodontal treatment, and this is the first clinical study in the literature to show its relationship with periodontal disease. According to these data, galectin-3 may play an active role in the progression of inflammation, as it triggers the expression of proinflammatory mediators in the periodontal inflammatory process. The limitation of this study was that the mean age of the periodontitis group in our study was higher than the other groups. To the best of our knowledge, there is no clear study showing the effect of age on galectin.

GCF galectin-3 is a new biomarker for the presence of periodontal disease and treatment efficacy and may represent as a therapeutic target. Further studies are needed to determine possible specific mechanisms, time-dependent evaluation and its exact role in chronic inflammation, and systemic – periodontal disease relationships.

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CONFLICT OF INTEREST

The authors declare no conflict(s) of interest.

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Address for correspondence:
Meltem Karsiyaka Hendek
Kirikkale University
Faculty of Dentistry
Department of Periodontology
Kirikkale
Turkey
Email: mltmkrsyk@yahoo.com