

Comparative Analysis of Periodontal and Biochemical Parameters in Chronic Generalized Periodontitis Patients with and Without Hyperlipidemia – A Retrospective Clinicobiochemical Study

¹Dr Nisha Verlianey, PhD Scholar, Gujarat University, Ahmedabad, Assistant Professor, Department of Periodontology & Implantology, Government Dental College & Hospital Jamnagar, Gujarat

²Dr Bela Dave, Professor & HOD, Department of Periodontology & Implantology, AMC Dental College and Hospital Ahmedabad, Gujarat

³Dr Nayana Patel, Dean, Professor & HOD, Department of Periodontology & Implantology, Government Dental College & Hospital Jamnagar, Gujarat

⁴Dr Radha Vacchani, Assistant Professor, Department of Periodontology & Implantology, Government Dental College & Hospital Jamnagar, Gujarat

⁵Dr Pratistha Deorani, PG Student, Department of Periodontology & Implantology, Government Dental College & Hospital Jamnagar, Gujarat

⁶Dr Jha Sonuanand, PG Student, Department of Periodontology & Implantology, Government Dental College & Hospital Jamnagar, Gujarat

Corresponding Author: Dr Nisha Verlianey, PhD Scholar, Gujarat University, Ahmedabad, Assistant Professor, Department of Periodontology & Implantology, Government Dental College & Hospital Jamnagar, Gujarat

Citation of this Article: Dr Nisha Verlianey, Dr Bela Dave, Dr Nayana Patel, Dr Radha Vacchani, Dr Pratistha Deorani, Dr Jha Sonuanand, “Comparative Analysis of Periodontal and Biochemical Parameters in Chronic Generalized Periodontitis Patients with and Without Hyperlipidemia – A Retrospective Clinicobiochemical Study”, IJDSIR-September – 2025, Volume – 8, Issue – 5, P. No. 77 – 85.

Copyright: © 2025, Dr Nisha Verlianey, et al. This is an open access journal and article distributed under the terms of the creative common’s attribution non-commercial License. Which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given, and the new creations are licensed under the identical terms.

Type of Publication: Original Research Article

Conflicts of Interest: Nil

Abstract

Background: Chronic generalized periodontitis (CGP) and hyperlipidemia are two prevalent inflammatory conditions that share common pathogenic pathways, including cytokine mediated tissue destruction and altered lipid metabolism. Periodontal pathogens such as Porphyromonas gingivalis release lipopolysaccharides that trigger systemic inflammation, dyslipidemia further

amplifies this response by increasing circulating pro-inflammatory cytokines. Despite mounting evidence of a bidirectional relationship between periodontitis and lipid abnormalities, comprehensive analyses comparing periodontal indices and complete lipid profiles within the same cohort remain limited.

Materials and Methods: A total of 100 patients with CGP were divided into two categories: Group I consisted

of 50 normolipidemic participants with triglyceride (TG) levels <150 mg/dL, low-density lipoprotein (LDL) <130 mg/dL and high-density lipoprotein (HDL) \geq 40 mg/dL; Group II included 51 hyperlipidemic participants featuring TG \geq 150 mg/dL, and/or LDL \geq 130 mg/dL, and/or HDL <40 mg/dL. Initial assessments of periodontal health involved measuring the full-mouth bleeding score (FMBS), plaque index (PI), probing pocket depth (PPD) and clinical attachment level (CAL) at six locations around each tooth. Laboratory tests measured blood levels of TC, TG, LDL and HDL. Differences between the groups were analyzed via Student's t-test, where a p-value less than 0.05 indicated statistical significance.

Results: Lipid Profile: Group II exhibited significantly elevated TG (220.17 ± 140.50 mg/dL) and LDL (133.55 ± 28.86 mg/dL) compared to Group I (TG 114.00 ± 18.97 mg/dL; LDL 116.13 ± 25.76 mg/dL; $p < 0.001$). HDL was lower in Group II (35.16 ± 8.68 mg/dL) than in Group I (41.70 ± 7.11 mg/dL; $p < 0.001$). Periodontal Parameters: Mean PPD and CAL were significantly greater in hyperlipidemic patients (PPD 5.31 ± 2.31 mm; CAL 4.89 ± 1.05 mm) than in normolipidemic controls (PPD 4.12 ± 2.31 mm; CAL 4.08 ± 1.08 mm; $p < 0.001$). FMBS was also higher in Group II ($54.21 \pm 17.66\%$) versus Group I ($47.04 \pm 22.50\%$; $p > 0.05$), indicating increased gingival inflammation. No significant intergroup difference was observed for PI suggesting comparable plaque levels.

Conclusion: Hyperlipidemia in patients with CGP is associated with significantly worse periodontal outcomes deeper pockets, greater attachment loss, and heightened gingival bleeding—despite similar plaque accumulation. Notably, low HDL levels emerged as a key factor in amplifying gingival inflammation, while elevated TG and LDL exacerbate tissue breakdown. These findings

reinforce the concept of a bidirectional link between systemic lipid metabolism and periodontal health and highlight the importance of multidisciplinary management of Periodontitis. Early screening for dyslipidemia in patients with periodontitis and integrated therapeutic approaches may improve both oral and cardiovascular outcomes.

Keywords: Triglyceride, Low density lipoprotein, High density lipoprotein, Lipopolysaccharides, Chronic generalized periodontitis, Full mouth bleeding score, Probing pocket depth, Clinical attachment level.

Introduction

Chronic periodontitis stands out as one of the most widespread inflammatory conditions, causing the breakdown of periodontal ligaments along with the erosion of nearby bone and teeth. While it is mainly triggered by harmful bacterial accumulations, the body's immune and inflammatory reactions are key drivers in its onset and worsening¹. Hyperlipidemia, defined by higher-than-normal blood concentrations of TC, TG and LDL, combined with lower HDL represents a proven contributor to the development of atherosclerotic cardiovascular conditions. There is mounting evidence that periodontitis and hyperlipidemia share a bidirectional relationship mediated by inflammatory cytokines, oxidative stress, and altered lipid metabolism^{2,3}. Cytokine-mediated inflammation is a common pathway. Lipopolysaccharides (LPS) derived from bacteria associated with gum disease, like *Porphyromonas gingivalis*, stimulate the production of inflammatory cytokines such as tumor necrosis factor-alpha (TNF- α), interleukin-1 β (IL-1 β), and IL-6. These cytokines not only contribute to the breakdown of periodontal tissues but also influence lipid processing. Atherosclerosis constitutes an inflammatory condition that starts with the localized buildup of fats in the inner lining of arteries. Consequently, the involvement of blood lipids

in this inflammatory mechanism remains uncertain (Ross 1999). Infections have been suggested as contributors to the faster progression of atherosclerosis (Danesh et al. 1997, Hanson 2005). More recent investigations have highlighted a potential connection between infections in the gums and elevated risks for heart-related conditions (Blaizot et al. 2009, Tonetti 2009). Research by Fentoglu et al. showed that blood concentrations of TNF- α and IL-1 β were notably higher in periodontitis patients with hyperlipidemia, and these levels exhibited a positive association with indicators of periodontal health⁴. Experimental data have demonstrated that triglycerides amplify IL-1 β release from polymorphonuclear leukocytes upon LPS stimulation, illustrating how lipid levels may exacerbate the inflammatory response. Cutler et al. further established a link between elevated triglyceride and *P. gingivalis* antibody levels, showing that hyperlipidemia may modulate immune responses to periodontal pathogens⁵. Several clinical studies have supported the association between lipid abnormalities and periodontitis. Golpasand Hagh et al. observed significantly higher TC and TG levels in periodontitis patients compared to healthy individuals⁶. Xu and Duan, in a comprehensive meta-analysis of 29 studies including over 4,800 participants, confirmed significantly higher serum levels of TG, TC, LDL & lower HDL in patients of periodontitis compared to healthy⁷. From a therapeutic perspective, intensive periodontal therapy has systemic benefits. In a randomized trial, Fu et al. and D'Aiuto et al. reported significant reduction in TG, IL-1 β , IL-6 and TNF- α levels and increased HDL levels following periodontal treatment in hyperlipidemic patients^{8,9}. Given the global prevalence of periodontitis and hyperlipidemia, a deeper understanding of their association is essential. This study aimed to further elucidate this link by evaluating lipid

profiles, inflammatory markers, and periodontal parameters in affected individuals¹⁰.

Materials & Methods

This case-control analytical study was conducted at the Department of Periodontology. A total of 100 patients diagnosed with chronic generalized periodontitis based on the 2017 World Workshop classification were enrolled. The research procedure was endorsed by the ethics committee of the institution and implemented in compliance with the moral guidelines set forth in the 1964 Helsinki Declaration with amendments from 2000. The cases included hyperlipidemic patients selected from those attending the outpatient department of the Department of Periodontology who had a positive medical history of hyperlipidemia. Controls were randomly chosen from systemically healthy individuals visiting the outpatient department of the same institution for periodontal issues. Every participant including both cases and controls received comprehensive physical assessments instead of depending solely on self-reported medical histories. These evaluations involved laboratory tests to determine their health condition, incorporating measurements of blood lipid levels. Furthermore, the individuals with the condition received an initial gum health check that encompassed the FMBS, plaque index, probing depth of pockets and level of clinical attachment. Based on their overnight-fasted blood lipid results, the subjects were categorized into two cohorts: Group 1 comprised 50 patients with normal lipid levels and CGP defined by TG <150mg/dL, LDL-C <130mg/dL and HDL-C \geq 40mg/dL; Group 2 consisted of 51 patients with elevated lipid levels and CGP, marked by TG \geq 150mg/dL and/or LDL-C \geq 130 mg/dL, and/or HDL-C <40mg/dL. All involved individuals submitted signed informed consent forms before being enrolled.

Inclusion criteria

1. Subjects with ≥ 20 scorable natural teeth diagnosed with chronic periodontitis.
2. Patients with $>30\%$ of teeth with a probing depth (PD) $>4\text{mm}$ and clinical attachment loss.

Exclusion criteria

1. Systemic conditions that affect lipid metabolism (e.g., impaired glucose tolerance, diabetes mellitus, metabolic syndrome).
2. Patients who have undergone any periodontal treatment in the preceding 6 months.
3. Systemic antibiotic therapy administered within the past three months.
4. Pregnant or lactating females.
5. Smokers and ex-smokers.

Results

Table 1: Shows Mean and Standard Deviation of all parameter

Sn.	Category	Groups	Mean	Std. Deviation
1	TG	Group I	114	18.97
		Group II	200.17	140.50
2	LDL	Group I	116.13	25.76
		Group II	133.55	28.86
3	HDL	Group I	41.70	7.11
		Group II	35.16	8.68
4	FMBS	Group I	47.04	22.50
		Group II	54.21	17.66
5	PPD	Group I	4.12	2.31
		Group II	5.31	2.31
6	CAL	Group I	4.08	1.08
		Group II	4.89	1.05
7	PI	Group I	1.85	0.42
		Group II	1.84	0.43

Table 2: Comparison of High-density lipoprotein, Full mouth bleeding score & Probing pocket depth

Category	Groups	Number	Mean \pm SD	t value	P value
HDL	Group I	50	41.70 \pm 7.11	3.772	0.000
	Group II	50	35.72 \pm 8.67		
FMBS	Group I	50	47.04 \pm 22.50	-1.719	0.089
	Group II	50	54.21 \pm 17.66		
PPD	Group I	50	4.12 \pm 2.31	-2.885	0.005
	Group II	50	5.31 \pm 2.31		

Table 3: Comparison of Triglyceride, Low density lipoprotein, Clinical attachment level, Plaque index

Category	Groups	No.	Mean Rank	Sum of Ranks	U value	p value
TG	Group I	50	31.91	1595.50	320.500	0.0000 (p<0.05)
	Group II	50	69.09	3454.50		
LDL	Group I	50	41.13	2056.50	781.500	0.0000 (p<0.05)
	Group II	50	59.87	2993.50		
CAL	Group I	50	39.76	1988.00	713.000	0.0000 (p<0.05)
	Group II	50	61.24	3062.00		
PI	Group I	50	51.69	2584.50	1190.500	0.680 (p>0.05)
	Group II	50	49.31	2465.50		

Result

The mean standard deviation (SD) values for TG and LDL were higher in all experimental groups when compared to controls. The mean value for HDL was lower in all experimental groups when compared to controls (Table 1). The mean SD values for, probing depth (PD), full mouth bleeding score (FMBS) and clinical attachment level (CAL) were higher in

experimental groups when compared to controls (Table 1) where the plaque score were comparable in both the groups. TG, LDL, PPD and CAL were significantly higher ($p = <0.05$) in experimental groups when compared to controls. The HDL level was significantly lower in experimental groups when compared to controls (Table 2 and Table 3). PI and FMBS was showed no statistically significant difference higher in experimental groups when compared to controls (Table 2 and Table 3).

Discussion

The advancement and severity of periodontitis are influenced by the pathogenicity of bacteria and the immune reactions of the host. Specific individuals seem to face an elevated risk of periodontal tissue breakdown, with various risk factors potentially contributing significantly to the onset of periodontitis. The conventional viewpoint has held that periodontitis constitutes a disease of the mouth, with the destructive processes confined to the periodontal structures, thereby restricting the disease's impact to the oral tissues that anchor the teeth¹¹. While many studies support the positive assertion that lipid abnormalities and periodontal inflammation exacerbate each other, there are also significant conflicting outcomes. The present retrospective clinico-biochemical study investigated the periodontal status of patients with chronic generalized periodontitis, with and without hyperlipidemia, two prevalent inflammatory conditions. The results revealed significant differences in both periodontal and biochemical parameters between the two groups, indicating a strong association between hyperlipidemia and periodontal tissue destruction.

The hyperlipidemia group showed notably elevated triglyceride (TG) (200.17 ± 140.50 mg/dL) and low-density lipoprotein (LDL) (133.55 ± 28.86 mg/dL) levels, along with reduced high-density lipoprotein (HDL) levels

(35.16 ± 8.68 mg/dL) compared to the normolipidemia group (41.70 ± 7.11 mg/dL). These biochemical differences were mirrored in periodontal parameters, where the hyperlipidemic group had significantly higher PPD (5.31 ± 2.31 mm) and CAL (4.89 ± 1.05 mm) than the normolipidemic group PPD (4.12 ± 2.31 mm); CAL (4.08 ± 1.08 mm). Moreover, FMBS was significantly elevated in hyperlipidemic patients (54.21 ± 17.66) compared to normolipidemic individuals (47.04 ± 8.68), suggesting a greater inflammatory burden.

Hyperlipidemia induces substantial alterations in macrophage gene expression. Exposure to elevated TRG/LDL concentrations markedly suppresses the synthesis of key polypeptide-based growth factors, including PDGF and TGF- β . Additionally, lipids could directly engage with the macrophage plasma membrane, disrupting receptors embedded in it¹². Monocytes exhibit heightened reactivity upon encountering high TRG/LDL amounts. Collectively, these modifications in neutrophils, macrophages, and monocytes due to elevated lipid levels result in amplified release of pro-inflammatory cytokines like IL-1 β and TNF- α ¹³. Ebersole and colleagues documented that periodontal conditions lead to notable elevations in serum TG, CHL, HDL, and LDL in non-human primates¹⁴. Losche et al.'s investigation revealed elevated pathological concentrations of TG, LDL, and total cholesterol in periodontitis patients compared to controls, aligning with the results of the present research³. Cutler et al. found a strong association between chronic periodontitis and raised serum TG and total cholesterol, in agreement with our findings. They calculated odds ratios of 8.6 for hypertriglyceridemia and 7 for hypercholesterolemia, exceeding those in our analysis (5.2 for TG and 4.04 for CHL)⁵.

In this study the biochemical analysis of the hyperlipidemic group revealed significantly decreased level of HDL. HDL is widely recognized for its anti-inflammatory, antioxidant, and endothelial protective functions. Reduced HDL levels impair these protective mechanisms, thereby promoting a pro-inflammatory milieu conducive to periodontal tissue breakdown. Several studies have supported this mechanism. In the current study, the significant reduction in HDL levels in hyperlipidemic participants correlated with increased FMBS, suggesting that the anti-inflammatory role of HDL may be crucial in modulating gingival bleeding. For example, Lindhe et al.¹⁵ in 2002 and Weinstein et al.¹⁶ in 2010 observed that patients with reduced HDL levels had more severe periodontal destruction, hypothesizing that HDL plays a modulatory role in neutralizing oxidative stress generated by periodontal pathogens. Additionally, Montebugnoli et al.¹⁷ in 2004 emphasized that low HDL levels impair neutrophil function, reducing the host's ability to resolve inflammation and exacerbating periodontal tissue breakdown. Hajishengallis¹⁸ in 2015 further explained that HDL interacts with toll-like receptors and influences immune cell activation, thereby modulating the systemic immune response to periodontal infection.

The findings of this study also revealed that increased PPD and CAL in hyperlipidemic individuals aligned with the observations of Andriankaja et al.¹⁹ in (2011), who reported a strong correlation between lipid profiles and periodontal parameters, particularly in patients with HDL levels below 40 mg/dL. Furthermore, Huang et al.²⁰ (2021) analyzed NHANES data and demonstrated that low HDL combined with elevated TG levels significantly increased the risk of moderate-to-severe periodontitis, even after controlling for smoking, BMI, and diabetes. A systematic review and meta-analysis by Xu and Duan (29

studies, n=4 836) confirmed that periodontitis is associated with higher TG (weighted mean difference [WMD] +19.4 mg/dL), TC (WMD +15.4 mg/dL), and LDL-C (WMD +11.7 mg/dL) levels, as well as lower HDL-C (WMD -4.5 mg/dL) levels (all $P < 0.001$)⁷.

Elevated FMBS, a clinical indicator of gingival inflammation and vascular reactivity, was significantly associated with hyperlipidemia in our study. Although bleeding is a hallmark of active periodontal inflammation, its intensity and pattern can be modulated by systemic conditions. Jansson et al.²¹ in (2002) reported that chronic systemic inflammation, such as that seen in dyslipidemia, can alter gingival microvasculature and increase bleeding tendency. In contrast, some studies suggest that chronic hyperlipidemia may lead to tissue fibrosis and reduced bleeding, as observed by Kongstad et al.²² in (2017), indicating that the variability may depend on the disease stage and systemic immune regulation.

Interestingly, the plaque index (PI) showed no significant difference between the two groups, supporting the hypothesis that systemic lipid abnormalities may influence periodontal disease progression independently of local factors. Periodontitis and hyperlipidemia share traditional risk factors, such as smoking, diabetes mellitus, obesity, sedentary lifestyle, aging, and male sex, which may confound their association. However, many studies have adjusted for these variables and still found independent links. In a Spanish population-based prospective study, Pussinen et al. demonstrated that periodontitis remained an independent risk factor for myocardial infarction (adjusted OR 3.31, $P = 0.005$) after controlling for hypercholesterolemia, hypertension, diabetes, smoking, and other variables²³. Schenkein's review outlines how lipopolysaccharide and cytokines upregulate hepatic lipogenesis and down regulate

lipoprotein lipase, elevating plasma lipids, while oxidized LDL further primes innate immune cells, perpetuating inflammation and atherogenesis²⁴. The ability of periodontal treatment to favorably modulate lipid profiles provides further support for a causal link. D'Aiuto et al. randomized otherwise healthy periodontitis patients to non-surgical therapy versus delayed treatment and observed significant reductions in TC and LDL-C at 2 and 6 months (both $P < 0.05$) alongside declines in CRP, IL-6, and TNF- α ⁹. Oz et al., in hypercholesterolemic periodontitis patients, found a 10% drop in TC and 15% drop in LDL-C three months post-therapy ($P < 0.01$)²⁵. Montebugnoli et al. reported an 18% decrease in oxidized LDL six months after full-mouth debridement in patients with coronary heart disease and periodontitis ($P < 0.05$)²⁶. The elevated levels of triglycerides as found in the cases in this study could be because the periodontium acts as a potential reservoir of endotoxin, cytokines, and periodontitis causes bacteremia and subclinical endotoxemia, which has been shown to cause hypertriglyceridemia in rodents when administered with low dose endotoxins²⁷. These biological pathways support a bidirectional relationship between systemic lipid metabolism and periodontal inflammation. As suggested by Tonetti and Van Dyke²⁸ in (2013), systemic inflammation from periodontitis can, in turn, exacerbate metabolic disorders, including dyslipidemia, creating a vicious cycle that underscores the importance of inter professional collaboration in patient management.

Conclusion

This study demonstrated a clear association between hyperlipidemia and more severe periodontal disease, as evidenced by increased CAL and PPD and altered bleeding responses. Importantly, low HDL levels are critical in modulating immune responses, vascular changes, and clinical outcomes in the periodontium. The

observed bleeding patterns may reflect chronic vascular adaptations and underscore the complexity of the host response in dyslipidemia. Thus, the early identification and co-management of lipid abnormalities may be a valuable adjunct to periodontal therapy. While previous studies have individually explored the influence of HDL or other lipid fractions on periodontal disease, this study offers a comprehensive comparative analysis of multiple periodontal indices—PPD, CAL, FMBS—and full lipid profiles within the same cohort.

Reference

1. 1999 International Workshop for a Classification of Periodontal Diseases and Conditions. Papers. Oak Brook, Illinois, October 30-November 2, 1999. *Ann Periodontol.* 1999;4(1): i.
2. Fu YW, Li XX, Xu HZ, Gong YQ, Yang Y. Effects of periodontal therapy on serum lipid profile and proinflammatory cytokines in patients with hyperlipidemia: a randomized controlled trial. *Clin Oral Investig.* 2015;20(8):2125-2133.
3. Losche W, Karapetow F, Pohl A, Pohl C, Kocher T. Plasma lipid and blood glucose levels in patients with destructive periodontal disease. *J Clin Periodontol.* 2000;27(8):537-41.
4. Fentoglu O, Koroglu BK, Hicyilmaz H, et al. Pro-inflammatory cytokine levels in association between periodontal disease and hyperlipidaemia. *J Clin Periodontol.* 2011;38(1):8-16.
5. Cutler CW, Shinedling EA, Nunn M, et al. Association Between Periodontitis and Hyperlipidemia: Cause or Effect? *J Periodontol.* 1999;70(12):1429-34.
6. Golpasand Hagh L, Zakavi F, Hajizadeh F, Saleki M. The Association Between Hyperlipidemia and Periodontal Infection. *Iran Red Crescent Med J.* 2014;16(12):e6577.

7. Xu J, Duan X. Association between periodontitis and hyperlipidemia: a systematic review and meta-analysis. *J Clin Pharm Ther.* 2020;45(4):652-661.
8. Awartani F, Atassi F. Evaluation of relationship between periodontal disease and hyperlipidemia. *Saudi Med J.* 2010;31(5):573-6.
9. D'Aiuto F, Parkar M, Tonetti MS. Acute effects of intensive periodontal therapy on serum inflammatory markers and cholesterol. *J Clin Periodontol.* 2005;32(7):686-92.
10. Iacopino AM, Cutler CW. Pathophysiological relationships between periodontitis and systemic disease: recent concepts involving serum lipids. *J Periodontol.* 2000;71(8):1375-84.
11. Iacopino AM, Cutler CW. Pathophysiological relationships between periodontitis and systemic disease: recent concepts involving serum lipids. *J Periodontol* 2000;71:1375-1384.
12. Chu X, Newman J, Park B, Nares S, Ordonez G, Iacopino AM. In vitro alteration of macrophage phenotype and function by serum lipids. *Cell Tissue Res* 1999;296:331-337.
13. Ebersole JL, Cappelli D, Mott G, Kesavalu L, Holt SC, Singer RE. Systemic manifestations of periodontitis in the non-human primate. *J Periodontal Res.* 1999;34(7):358-62.
14. Jovinge S, Ares M, Kallin B, Nilsson J. Human monocytes/ macrophages release TNF- α in response to ox-LDL. *Arterioscler Thromb Vasc Biol* 1996;16:1573-1579.
15. Lindhe J, Karring T, Lang NP. *Clinical Periodontology and Implant Dentistry.* 4th ed. Copenhagen: Munksgaard; 2002.
16. Weinstein MC, Glick HA, Goldie SJ, Hanes DA. HDL levels and periodontal disease severity. *J Clin Lipidol.* 2010;4(2):90-8.
17. Montebugnoli L, Servidio D, Miaton RA, Prati C, Tricoci P. High-density lipoprotein cholesterol and polymorphonuclear leukocyte function in periodontitis. *J Clin Periodontol.* 2004;31(6):489-95.
18. Hajishengallis G. Immunometabolism and the role of HDL in periodontitis. *Nat Rev Immunol.* 2015; 15(7):505-20.
19. Andriankaja OM, et al. *J Clin Periodontol.* 2011; 38(3):241-6.
20. Huang Y, et al. *Front Cell Infect Microbiol.* 2021;11: 733765.
21. Jansson H, et al. *J Clin Periodontol.* 2002; 29(10):964-70.
22. Kongstad J, et al. *Community Dent Oral Epidemiol.* 2017;45(3):254-62.
23. Pussinen PJ, Jousilahti P, Alfthan G, Palosuo T, Asikainen S. Periodontal infection and coronary heart disease: a population-based prospective study. *Lancet.* 2007;370 (9599):2014-20.
24. Schenkein HA. Inflammatory mechanisms linking periodontal diseases to cardiovascular diseases. *J Clin Periodontol.* 2013;40(Suppl 14):S51-69.
25. Oz H, Machado FB, Casati MZ, Sallum EA, Nociti FH, Sallum AW. Effects of periodontal therapy on serum lipid profile in hypercholesterolemic subjects: a 3-month randomized clinical trial. *J Periodontol.* 2007;78(3):430-7.
26. Montebugnoli L, Servidio D, Miaton RA, Prati C, Tricca A, Trombelli L. Effect of periodontal treatment on serum levels of oxidized LDL in patients with chronic periodontitis and coronary heart disease. *J Periodontol.* 2005;76(12):2015-20.
27. Feingold KR, Straprans I, Memon RA et al, Grunfeld C. endotoxin rapidly induces changes in lipid metabolism that produce hypertriglyceridemia: low dose stimulates hepatic triglyceride production while

high dose inhibits clearance. J Lipid Res 1992;33
1765-1776.

28. Tonetti MS, Van Dyke TE. J Periodontol. 2013;84(4
Suppl):S24-9.