International Journal of Community Medicine and Public Health
Albalbisi AM et al. Int J Community Med Public Health. 2025 Oct;12(10):4782-4789
http://www.ijcmph.com
pISSN 2394-6032 | eISSN 2394-6040

DOI: https://dx.doi.org/10.18203/2394-6040.ijcmph20253285

Review Article

Oxidative stress and its contribution to chronic periodontal inflammation

Ayman M. Albalbisi[1]*, Moahmmed S. Asiri[2], Noura F. Alshahrani[2], Saleh A. Alqhtani[2], Saleh S. Alqahtani[3], Shahd A. Alqahtani[4], Abdulkarim A. Alnasrallah[5], Aliya S. Alqhatani[6], Hassan G. Alasmari[2], Hadeel A. Jiffry[7], Mohammed S. Alqahtani[8]

[1]Department of Periodontics, North Jeddah Specialist Dental Center, Jeddah, Saudi Arabia
[2]College of Dentistry, King Khalid University, Abha, Saudi Arabia
[3]College of Dentistry, Aljouf University, Riyadh, Saudi Arabia
[4]College of Dentistry, Princess Noura University, Riyadh, Saudi Arabia
[5]College of Dentistry, University of Hail, Hail, Saudi Arabia
[6]Department of Dentistry, Private Clinic, Khamis Mushait, Saudi Arabia
[7]Department of General Dentistry, Bros Dental Clinic, Riyadh, Saudi Arabia
[8]Department of General Dentistry, Private Clinic, Jeddah, Saudi Arabia

Received: 01 September 2025
Revised: 15 September 2025
Accepted: 17 September 2025

***Correspondence:**
Dr. Ayman M. Albalbisi,
E-mail: Aalbalbisi@moh.gov.sa

ABSTRACT

Periodontal disease is a group of diseases that affect the tissues that support the teeth. It is among the most prevalent oral diseases worldwide. Various mechanisms contribute to the pathogenesis of chronic periodontal inflammation, including oxidative stress. Oxidative stress leads to the production of reactive oxygen species (ROS), both exacerbating inflammatory reactions and worsening the health of gums and surrounding tissues, thus increasing disease severity. In addition, oxidative stress is a key factor mediating the influence of periodontitis on systemic diseases. However, the role of oxidative stress in the pathogenesis of chronic periodontal inflammation is unclear. This review aims to discuss the mechanisms through which oxidative stress contributes to chronic periodontal inflammation. Chronic periodontitis leads to continuous activation of polymorphonuclear neutrophils, which may be hyperactivated, resulting in overproduction of ROS and exacerbation of oxidative stress. Oxidative stress results in intracellular damage of proteins, lipids, and DNA, along with connective tissue destruction and bone resorption. Oxidative stress biomarkers, such as malondialdehyde and hydrogen peroxide, are closely linked to the development and severity of periodontitis. Antioxidants, such as vitamin C, vitamin E, and glutathione, can play a key role in the prevention and treatment of chronic inflammatory diseases, including periodontitis. Periodontitis also contributes to systemic diseases, such as cardiovascular diseases, diabetes mellitus, chronic kidney diseases, and liver diseases, via mechanisms, including systemic inflammation and oxidative stress. Future research should focus on clarifying oxidative stress molecular pathways in chronic periodontal disease, improving antioxidant-based therapies, and integrating personalized approaches through genomics and biomarkers.

Keywords: Chronic periodontal inflammation, Periodontitis, Oxidative stress, Reactive oxygen species, ROS

INTRODUCTION

Periodontitis is a chronic inflammatory disease that affects the tissues that support the teeth, including the gums, alveolar bone, and periodontal ligament. It is one of the most prevalent oral diseases, significantly influencing individual oral health and quality of life.[1]

The prevalence of periodontitis globally is 61.6%, while the prevalence of moderate to severe periodontitis is 53.2%.[2] Thus, periodontitis is considered a prevalent and serious global health issue impacting oral health.

Chronic periodontal inflammation induced by chronic periodontitis is associated with increased oxidative stress and reduced antioxidant capacity in the oral cavity.[3] Oxidative stress is a phenomenon occurring when the production of oxygen free radicals and other oxidants in the cellular and extracellular environment exceeds the antioxidants produced by the body, leading to cellular functional damage.[4,5] Oxidative stress is considered a key pathophysiological process that contributes to chronic periodontal inflammation. It may exacerbate inflammatory reactions and worsen the health of gums and surrounding tissues, thus increasing disease severity.[4,5] Furthermore, proteolytic enzymes and reactive oxygen species (ROS), generated through interactions between polymorphonuclear neutrophils and microorganisms, contribute to tissue destruction in the gingival connective tissue and alveolar bone.[6]

Periodontal disease has been linked to various chronic diseases, such as diabetes mellitus, cardiovascular disease (CVD), chronic kidney disease, and liver diseases.[7] Oxidative stress is a key factor mediating the influence of periodontitis on systemic diseases.[8] However, the mechanisms of how oxidative stress contributes to chronic periodontal inflammation and how this periodontitis-induced oxidative stress contributes to different chronic diseases are still unclear. The aim of this review is to explore current evidence for data on the contribution of oxidative stress to chronic periodontal disease, highlighting the association between periodontitis-induced oxidative stress and different chronic diseases.

METHODS

A comprehensive literature search was conducted in Medline (via PubMed), Scopus, and Web of Science databases up to 05 May 2025. Medical Subject Headings (MeSH) and relevant free-text keywords were used to identify synonyms. Boolean operators (AND, OR) were applied to combine search terms in alignment with guidance from the Cochrane Handbook for Systematic Reviews of Interventions. Key search terms included: “chronic periodontal inflammation” OR “periodontitis” OR “periodontal disease” AND “oxidative stress” OR “reactive oxygen species”. Summaries and duplicates of the found studies were exported and removed by EndNote X8. Any study that discusses the role of oxidative stress in the pathogenesis of chronic periodontal inflammation and is published in peer-reviewed journals was included. All languages are included. Full-text articles, case series, and abstracts with related topics are included. Case reports, comments, and letters were excluded.

DISCUSSION

Oxidative stress in periodontal disease

Multiple studies have explored the contribution of oxidative stress in the pathogenesis of chronic periodontal inflammation. The role of oxidative stress in the pathogenesis of periodontitis is shown in Figure 1. The presence of dental plaque and its migration to periodontal tissues induces inflammation and leads to the accumulation of leukocytes in the site of infection, especially polymorphonuclear neutrophils.[9] These polymorphonuclear neutrophils defend against the bacterial pathogens contained within dental plaque through various mechanisms, including phagocytosis, degranulation, NETosis, chemotaxis, and the release of ROS.[10] However, these polymorphonuclear neutrophils may be hyperactivated, leading to overproduction of ROS. Despite the antimicrobial and signaling roles of polymorphonuclear neutrophils and ROS, their overproduction has been linked to various inflammatory diseases, such as type 2 diabetes, atherosclerosis, rheumatoid arthritis, cancer, lung diseases, renal diseases, and periodontitis.[11-15]

This can be attributed to the increase in oxidative stress caused by ROS. Oxidative stress results in intracellular damage to proteins, lipids, and DNA, connective tissue destruction, and bone resorption.[15-17] Erythroid 2-related factor 2 (Nrf2) is a key antioxidant regulator down-regulated by oxidative stress, and the suppression of Nrf2 can lead to the progression of inflammatory diseases such as periodontitis and rheumatoid arthritis.[18] ROS-mediated damage also affects extracellular connective tissues, leading to clinical attachment loss.[19]

Furthermore, the overproduction of ROS and increased respiratory burst activity from hyperactive polymorphonuclear neutrophils result in the expression of proinflammatory cytokines, neutrophil priming, and prolonged neutrophil lifespan in periodontitis.[20,21] These cytokines play a role in tissue and bone destruction through both direct and indirect mechanisms. A disruption in the RANKL/osteoprotegerin axis may also occur, resulting in increased osteoclastogenesis, further increasing bone resorption.[22] This axis is also essential in other inflammatory bone diseases such as osteoporosis and rheumatoid arthritis.[17] Proinflammatory cytokines such as IL-1, IL-6, and TNF-α further contribute to uncoupled bone remodeling and osteoclast activation.[23] Additionally, ROS can reduce collagen synthesis, impair fibroblast function, and induce the overexpression of matrix metalloproteinases (MMPs), which degrade bone and

connective matrices.[24-26] An imbalance between MMPs and their inhibitors exacerbates tissue degradation.[27]

Oxidative stress markers in periodontal disease

Oxidative stress biomarkers are closely linked to the development and severity of periodontitis. Elevated levels of markers like malondialdehyde (MDA), hydrogen peroxide, and oxidative DNA damage, along with reduced activity of antioxidants such as superoxide dismutase (SOD) and catalase (CAT), have been consistently observed in periodontitis patients. These biomarkers correlate with disease progression and severity, making them valuable tools for diagnosing periodontitis and monitoring treatment outcomes.[28]

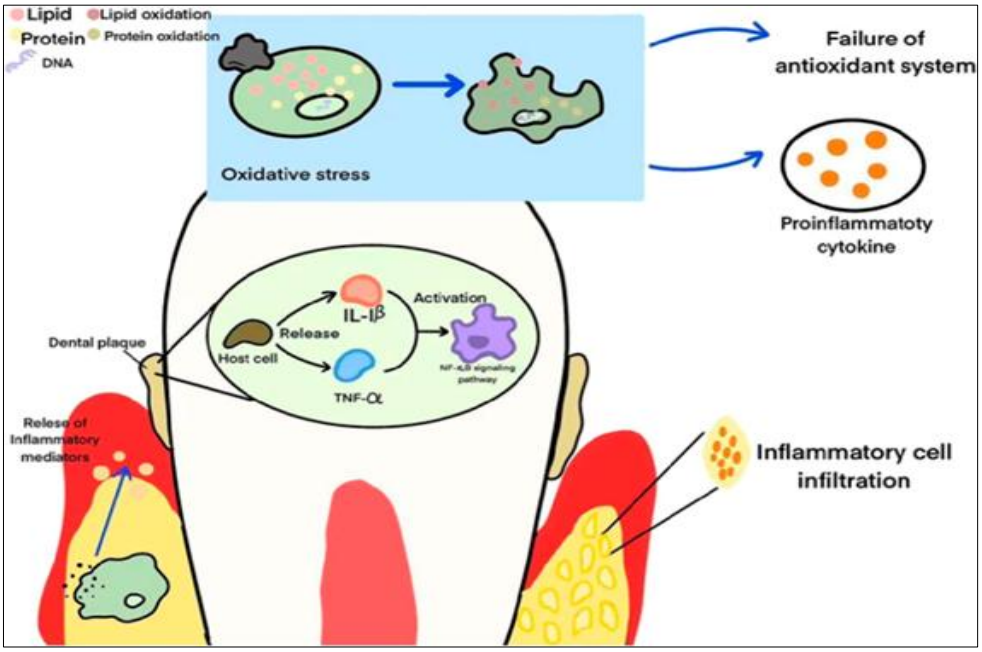

Figure 1: The role of oxidative stress in the pathogenesis of periodontitis.

Oxidative stress leads to the production of biomarkers, such as MDA, 8-hydroxy-2-deoxyguanosine (8-OHdG), and protein carbonyls, which were found elevated in the saliva, gingival crevicular fluid, and plasma of periodontitis patients.[16,28,29] These oxidative markers correlate with disease severity and tissue attachment loss.

Notably, oxidative stress biomarkers tend to decrease following non-surgical periodontal therapy, such as scaling and root planning, and may respond further to adjunctive antioxidant therapies, highlighting their utility in monitoring treatment response. However, their levels may vary depending on the sampling site and methodology used, raising concerns regarding standardization and reliability. The imbalance in antioxidant defense, marked by diminished SOD and glutathione peroxidase activity, is closely associated with increased oxidative burden and tissue destruction.[30] Emerging markers such as isoprostanes and advanced oxidation protein products (AOPP) are being explored for improved sensitivity and specificity.[31] Individual factors such as smoking, diabetes, and genetic variations affecting oxidative pathways can significantly influence the levels of biomarkers in the body. These factors can lead to increased oxidative stress and inflammation, which are reflected in changes to various biomarkers, suggesting a need for personalized interpretation in clinical settings.

Role of oxidative stress in linking periodontal disease and systemic diseases

Besides damaging periodontal tissues, periodontitis contributes to systemic diseases, such as cardiovascular diseases, diabetes mellitus, chronic kidney diseases, and liver diseases, via mechanisms, including systemic inflammation and oxidative stress.[7]

Cardiovascular diseases

There is a link between periodontal inflammation and CVD through oxidative stress. As mentioned, periodontal inflammation induces the production of ROS, which enter the bloodstream, inducing vascular endothelial dysfunction and enhancing atherosclerotic processes.[4] ROS also activate redox-sensitive transcription factors like NF-κB, which upregulate proinflammatory cytokines.[4] It has been reported that patients with both periodontitis and acute myocardial infarction exhibit high levels of lipid peroxides and increased oxidative stress markers.[32] Coenzyme Q10, an antioxidant crucial for mitochondrial function, has also been reported to be lower in these patients.[33]

Additionally, animal studies reported that periodontitis can stimulate inflammatory and oxidative responses in cardiac

tissue, which can be mitigated by antioxidants such as crocin and caffeic acid phenethyl ester.[34,35] In diabetic patients, where mitochondrial ROS production is already elevated, periodontitis may further increase cardiovascular risk.[36]

Diabetes mellitus

Diabetes mellitus and periodontitis have a bidirectional relationship, mainly driven by chronic inflammation and oxidative stress.[37] Oxidative injury and reduced antioxidant capacity are common in both diseases. Studies addressing the diabetes-periodontitis relationship demonstrated increased levels of oxidative biomarkers such as 8-hydroxydeoxyguanosine, MDA, and 3-nitrotyrosine. They also reported a downregulation of Nrf2 expression, a key antioxidant regulator.[38] This dual disease state worsens oxidative damage both locally and systemically, leading to more severe periodontal destruction. Notably, rutin and curcumin have been shown to improve antioxidant status and reduce oxidative stress in diabetic periodontitis.[39]

Chronic kidney disease

Studies have shown that oxidative stress caused by periodontal inflammation may affect renal tissues negatively, resulting in disruption of renal tubules and histomorphological alterations.[40] Resveratrol, an antioxidant, can restore gingival redox balance and decrease systemic oxidative stress, potentially protecting against kidney damage.[41]

Liver injury

Oxidative stress also contributes to liver injury by inducing lipid peroxidation, DNA damage, and mitochondrial dysfunction.[42] Studies indicate that periodontitis can reduce hepatic glutathione levels and increase oxidative markers such as HEL and 8-OHdG, particularly when combined with other stressors like alcohol.[43] This suggests that periodontitis-related oxidative stress may exacerbate hepatic damage.

Oxidative stress precipitating factors

Smoking

Smoking can significantly increase oxidative stress in periodontal disease by accelerating the production of ROS and suppressing antioxidant defense mechanisms. Smoking also induces a hyperactive neutrophil response, resulting in excessive ROS production and cytokine release, which contributes to tissue destruction via oxidative stress.[44] Multiple studies reported that smoking reduces antioxidant levels, including glutathione peroxidase, superoxide dismutase, and catalase. Smoking also increases markers of oxidative damage, such as 8-hydroxy-2′-deoxyguanosine and C-reactive protein.[45,46] It also disrupts bone homeostasis by promoting osteoclastogenesis and bone resorption and altering the RANKL/osteoprotegerin ratio.[47] Furthermore, smoking has been associated with reduced treatment response and a dose-dependent decline in antioxidant capacity.[48] These changes collectively worsen periodontal outcomes and suggest that antioxidant therapies could play a critical role in counteracting smoking-induced oxidative stress in periodontal disease.

Obesity

Obesity contributes significantly to oxidative stress in periodontal disease through chronic systemic inflammation and excessive production of ROS. Adipose tissue in obesity secretes proinflammatory cytokines like TNF-α and IL-6, which disrupt immune regulation and promote oxidative imbalance.[49] Elevated oxidative markers such as myeloperoxidase, protein carbonyls, and 8-hydroxy-2′-deoxyguanosine have been detected both in serum and gingival crevicular fluid of obese individuals, indicating systemic and local oxidative stress.50 Simultaneously, total antioxidant capacity is diminished, impairing the body's ability to counteract ROS. This oxidative environment amplifies gingival inflammation and periodontal tissue destruction, even in the absence of active infection.[50]

The bidirectional link between obesity and periodontitis suggests that oxidative stress not only worsens periodontal damage but may also perpetuate systemic inflammation, reinforcing obesity-related metabolic disturbances.[49]

Future directions

Future research should focus more on the mechanisms of antioxidant therapy in reducing oxidative stress in periodontal disease. It should explore how antioxidant therapy improves endogenous antioxidant defenses, reduces ROS production, and regulates inflammatory and immune responses to induce periodontal tissue healing. Understanding these molecular interactions will clarify antioxidant efficacy. Antioxidant therapy can also play a role in personalized therapy. The integration of biomarker and genomics data can result in individualized treatment based on a patient's genetic background, environment, and health status. Tools like artificial intelligence and big data can support personalized analysis and treatment optimization.

Furthermore, it is critical to improve clinical trial design by prioritizing multicenter, randomized, and double-blind designs with comprehensive assessment metrics, including inflammatory markers and tissue regeneration outcomes. Meta-analyses should also be performed to help synthesize findings and support evidence-based clinical application. More studies should focus on lifestyle factors, such as diet and smoking, as they remarkably impact oxidative stress and periodontitis progression.

Diets low in antioxidants and high in sugars increase oxidative stress, while foods rich in antioxidants (e.g., vitamins C, E, and carotenoids) can help decrease inflammation. Additionally, smoking induces the generation of free radicals and impairs antioxidant defenses, worsening periodontal damage. Addressing such factors is crucial in comprehensive treatment strategies.

Biotechnological innovations like nanomedicine and CRISPR show promise for targeted therapy and tissue regeneration. Nanoparticles can deliver antioxidants directly to inflamed sites, while CRISPR may enable gene editing to reduce inflammation and restore tissue function. Though still in development, these technologies could revolutionize periodontal treatment. Finally, biomarker advancements in genomics and molecular biology can enable more precise treatment approaches. Identifying susceptibility genes and microbial profiles could guide personalized interventions and improve treatment efficacy, supported by multicenter trials and precision medicine frameworks.

Antioxidants in periodontal disease

Antioxidants are compounds produced to neutralize and clear oxygen-free radicals and other oxidants within cells. They play a key role in the prevention and treatment of chronic inflammatory diseases. One of the key antioxidants is vitamin C (ascorbic acid). It is a water-soluble antioxidant that neutralizes hydroxyl radicals (•OH) and singlet oxygen ($^1O_2$), thus suppressing cellular damage caused by oxidative stress.[51,52] Vitamin C is critical in diseases such as neurodegenerative conditions, CVDs, and cancer.[53] Vitamin E (α-tocopherol) is a lipid-soluble antioxidant that is present predominantly in cell membrane phospholipid bilayers. It is capable of suppressing lipid peroxidation by neutralizing lipid peroxides and stabilizing membrane structure.[54]

Glutathione (GSH) is another antioxidant with a tripeptide form (glycine, cysteine, and glutamic acid) crucial for intracellular redox homeostasis. It neutralizes ROS and regenerates its reduced form (GSH) from its oxidized form (GSSG) via NADPH-dependent glutathione reductase, supporting the activity of vitamins C and E.[55] Reduction of GSH is associated with increased oxidative stress, leading to increased risk of diseases such as periodontitis.[56]

Carotenoids, such as β-carotene, lycopene, and lutein, are fat-soluble antioxidants that neutralize singlet oxygen and other ROS through their conjugated double bond structure.[57] They protect cell membranes and organelles under oxidative stress. However, under certain environmental conditions, such as high oxygen tension, carotenoids may shift to pro-oxidant behavior.

Oxidative stress can be assessed by antioxidant enzymes, including glutathione peroxidase, superoxide dismutase, and catalase. Antioxidant enzymes are released in response to oxidative stress to restore redox balance.[58] Non enzymatic antioxidants such as albumin and uric acid are also released to support oxidative stability in the oral cavity.[59] Multiple studies showed that antioxidant levels increase following periodontal treatment, supporting the association between oxidative stress and periodontitis progression.[6,58,60]

A previous study found compromised DNA integrity and reduced antioxidant status in periodontitis patients.[60] Another study found that levels of uric acid, albumin, total antioxidant capacity, and glutathione peroxidase were restored after nonsurgical treatment.[58]

However, findings on systemic antioxidant status (e.g., in plasma) remain debatable. Notably, total antioxidant capacity showed no differences between healthy and diseased individuals in some studies, while others reported that total antioxidant capacity decreases because of inflammation rather than being a cause.[61,62]

CONCLUSION

Oxidative stress plays a central role in the pathogenesis and progression of periodontitis, contributing to local tissue destruction and systemic inflammatory effects. The overproduction of reactive oxygen species, particularly by hyperactive polymorphonuclear neutrophils, leads to cellular and extracellular damage, promoting periodontal tissue breakdown and alveolar bone resorption. Moreover, oxidative stress serves as a key mechanistic link between periodontitis and systemic diseases such as CVD, diabetes, chronic kidney disease, and liver injury. Future research should focus on clarifying molecular pathways, improving antioxidant-based therapies, and integrating personalized approaches through genomics and biomarkers. Targeting oxidative stress represents a vital strategy for preventing periodontal progression and its systemic consequences.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Könönen E, Gursoy M, Gursoy UK. Periodontitis: A Multifaceted Disease of Tooth-Supporting Tissues. J Clin Med. 2019;8(8).
2. Trindade D, Carvalho R, Machado V, Chambrone L, Mendes JJ, Botelho J. Prevalence of periodontitis in dentate people between 2011 and 2020: A systematic review and meta-analysis of epidemiological studies. J Clin Periodontol. 2023;50(5):604-26.
3. Liu W, Guo D. Oxidative stress in periodontitis and the application of antioxidants in treatment: a narrative review. Front Physiol. 2025;16:1485367.
4. Liu C, Mo L, Niu Y, Li X, Zhou X, Xu X. The Role of Reactive Oxygen Species and Autophagy in Periodontitis and Their Potential Linkage. Front Physiol. 2017;8:439.

5. Wang Y, Andrukhov O, Rausch-Fan X. Oxidative Stress and Antioxidant System in Periodontitis. Front Physiol. 2017;8:910.
6. Chapple IL, Matthews JB. The role of reactive oxygen and antioxidant species in periodontal tissue destruction. Periodontol 2000. 2007;43:160-232.
7. Shang J, Liu H, Zheng Y, Zhang Z. Role of oxidative stress in the relationship between periodontitis and systemic diseases. Front Physiol. 2023;14:1210449.
8. Ye D, Li FY, Lam KS, Li H, Jia W, Wang Y, et al. Toll-like receptor-4 mediates obesity-induced non-alcoholic steatohepatitis through activation of X-box binding protein-1 in mice. Gut. 2012;61(7):1058-67.
9. Sczepanik FSC, Grossi ML, Casati M, Goldberg M, Glogauer M, Fine N, et al. Periodontitis is an inflammatory disease of oxidative stress: We should treat it that way. Periodontol 2000. 2020;84(1):45-68.
10. Landzberg M, Doering H, Aboodi GM, Tenenbaum HC, Glogauer M. Quantifying oral inflammatory load: oral neutrophil counts in periodontal health and disease. J Periodont Res. 2015;50(3):330-6.
11. Sari A, Davutoglu V, Bozkurt E, Taner IL, Erciyas K. Effect of periodontal disease on oxidative stress markers in patients with atherosclerosis. Clin Oral Investig. 2022;26(2):1713-24.
12. Martínez Nieto M, De Leon Rodríguez ML, Anaya Macias RDC, Lomelí Martínez SM. Periodontitis and chronic kidney disease: A bidirectional relationship based on inflammation and oxidative stress. World J Clin Cases. 2024;12(35):6775-81.
13. Mirnic J, Djuric M, Brkic S, Gusic I, Stojilkovic M, Tadic A, et al. Pathogenic Mechanisms That May Link Periodontal Disease and Type 2 Diabetes Mellitus-The Role of Oxidative Stress. Int J Mol Sci. 2024;25(18).
14. Cheng Z, Meade J, Mankia K, Emery P, Devine DA. Periodontal disease and periodontal bacteria as triggers for rheumatoid arthritis. Best Practice Res Clin Rheumatol. 2017;31(1):19-30.
15. Buczko P, Zalewska A, Szarmach I. Saliva and oxidative stress in oral cavity and in some systemic disorders. J Physio Pharmacol. 2015;66(1):3-9.
16. Su H, Gornitsky M, Velly AM, Yu H, Benarroch M, Schipper HM. Salivary DNA, lipid, and protein oxidation in nonsmokers with periodontal disease. Free Radical Bio Med. 2009;46(7):914-21.
17. Bartold PM, Marshall RI, Haynes DR. Periodontitis and rheumatoid arthritis: a review. J Periodontol. 2005;76(11):2066-74.
18. Chiu AV, Saigh MA, McCulloch CA, Glogauer M. The Role of NrF2 in the Regulation of Periodontal Health and Disease. J Dental Res. 2017;96(9):975-83.
19. Johnstone AM, Koh A, Goldberg MB, Glogauer M. A hyperactive neutrophil phenotype in patients with refractory periodontitis. J Periodontol. 2007;78(9):1788-94.
20. Dias IH, Matthews JB, Chapple IL, Wright HJ, Dunston CR, Griffiths HR. Activation of the neutrophil respiratory burst by plasma from periodontitis patients is mediated by pro-inflammatory cytokines. J Clin Periodontol. 2011;38(1):1-7.
21. Kinney JS, Morelli T, Braun T, Ramseier CA, Herr AE, Sugai JV, et al. Saliva/pathogen biomarker signatures and periodontal disease progression. J Dental Res. 2011;90(6):752-8.
22. Lee NK, Choi YG, Baik JY, Han SY, Jeong DW, Bae YS, et al. A crucial role for reactive oxygen species in RANKL-induced osteoclast differentiation. Blood. 2005;106(3):852-9.
23. Graves D. Cytokines that promote periodontal tissue destruction. J Periodontol. 2008;79(8):1585-91.
24. Reynolds JJ. Collagenases and tissue inhibitors of metalloproteinases: a functional balance in tissue degradation. Oral Dis. 1996;2(1):70-6.
25. Guan SM, Shu L, Fu SM, Liu B, Xu XL, Wu JZ. Prevotella intermedia upregulates MMP-1 and MMP-8 expression in human periodontal ligament cells. FEMS Microb Letters. 2009;299(2):214-22.
26. Kaur S, White S, Bartold PM. Periodontal disease and rheumatoid arthritis: a systematic review. J Dental Res. 2013;92(5):399-408.
27. Hienz SA, Paliwal S, Ivanovski S. Mechanisms of Bone Resorption in Periodontitis. J Immunol Res. 2015;2015:615486.
28. Almerich-Silla JM, Montiel-Company JM, Pastor S, Serrano F, Puig-Silla M, Dasí F. Oxidative Stress Parameters in Saliva and Its Association with Periodontal Disease and Types of Bacteria. Dis Markers. 2015;2015:653537.
29. Konopka T, Król K, Kopeć W, Gerber H. Total antioxidant status and 8-hydroxy-2'-deoxyguanosine levels in gingival and peripheral blood of periodontitis patients. Arch Immunol Ther Exp. 2007;55(6):417-22.
30. Afzal S, Abdul Manap AS, Attiq A, Albokhadaim I, Kandeel M, Alhojaily SM. From imbalance to impairment: the central role of reactive oxygen species in oxidative stress-induced disorders and therapeutic exploration. Front Pharmacol. 2023;14:1269581.
31. Witko-Sarsat V, Friedlander M, Capeillère-Blandin C, et al. Advanced oxidation protein products as a novel marker of oxidative stress in uremia. Kidney Intern. 1996;49(5):1304-13.
32. Díaz CM, Bullon B, Ruiz-Salmerón RJ, ernández-Riejos P, Fernández-Palacín A, et al. Molecular inflammation and oxidative stress are shared mechanisms involved in both myocardial infarction and periodontitis. J Periodontol Res. 2020;55(4):519-28.
33. Ferlazzo N, Currò M, Isola G, Maggio S, Bertuccio MP, Trovato-Salinaro A, et al. Changes in the Biomarkers of Oxidative/Nitrosative Stress and Endothelial Dysfunction Are Associated with Cardiovascular Risk in Periodontitis Patients. Curr Issues Mol Biol. 2021;43(2):704-15.
34. Kocaman G, Altinoz E, Erdemli ME, Gul M, Erdemli Z, Zayman E, et al. Crocin attenuates oxidative and

inflammatory stress-related periodontitis in cardiac tissues in rats. Adv Clin Exp Med. 2021;30(5):517-24.
35. Otan Özden F, Lütfioğlu M, Demir E, Bilgici B. Antioxidant effect of caffeic acid phenethyl ester in experimentally induced periodontitis. Clinl Oral Investig. 2021;25(8):4959-66.
36. Masi S, Orlandi M, Parkar M, Bhowruth D, Kingston I, O'Rourke C, et al. Mitochondrial oxidative stress, endothelial function and metabolic control in patients with type II diabetes and periodontitis: A randomised controlled clinical trial. Int J Cardiol. 2018;271:263-8.
37. Luong A, Tawfik AN, Islamoglu H, Gobriel HS, Ali N, Ansari P, et al. Periodontitis and diabetes mellitus co-morbidity: A molecular dialogue. J Oral Biosci. 2021;63(4):360-9.
38. Li X, Sun X, Zhang X, Mao Y, Ji Y, Shi L, et al. Enhanced Oxidative Damage and Nrf2 Downregulation Contribute to the Aggravation of Periodontitis by Diabetes Mellitus. Oxid Med Cell Longev. 2018;2018:9421019.
39. Iova GM, Calniceanu H, Popa A, Szuhanek CA, Marcu O, Ciavoi G, et al. The Antioxidant Effect of Curcumin and Rutin on Oxidative Stress Biomarkers in Experimentally Induced Periodontitis in Hyperglycemic Wistar Rats. Mol (Basel, Switzerland). 2021;26(5):1332.
40. França LFC, Vasconcelos A, da Silva FRP, Alves EHP, Carvalho JS, Lenardo DD, et al. Periodontitis changes renal structures by oxidative stress and lipid peroxidation. J Clin Periodontol. 2017;44(6):568-76.
41. Li X, Liu XC, Ding X, Liu XM, Cao NB, Deng Y, et al. Resveratrol protects renal damages induced by periodontitis via preventing mitochondrial dysfunction in rats. Oral Dis. 2023;29(4):1812-25.
42. Huo X, Liu C, Gao L, Xu X, Zhu N, Cao L. Hepatoprotective Effect of Aqueous Extract from the Seeds of Orychophragmus violaceus against Liver Injury in Mice and HepG2 Cells. Int J Mol Sci. 2017;18(6).
43. Pessoa LS, Pereira-da Silva FR, Alves EH, França LF, di Lenardo D, Carvalho JS, et al. One or two ligatures inducing periodontitis are sufficient to cause fatty liver. Med Oral Patol Oral Cir Bucal. 2018;23(3):e269-76.
44. Johannsen A, Susin C, Gustafsson A. Smoking and inflammation: evidence for a synergistic role in chronic disease. Periodontol 2000. 2014;64(1):111-26.
45. Aziz AS, Kalekar MG, Suryakar AN, Benjamin T, Prakashan MJ, Ahmed BM, et al. Assessment of some biochemical oxidative stress markers in male smokers with chronic periodontitis. Indian J Clin Biochem. 2013;28(4):374-80.
46. Tonguç M, Öztürk O, Sütçü R, Ceyhan BM, Kılınç G, Sönmez Y, et al. The impact of smoking status on antioxidant enzyme activity and malondialdehyde levels in chronic periodontitis. J Periodontol. 2011;82(9):1320-8.
47. César-Neto JB, Duarte PM, de Oliveira MC, Tambeli CH, Sallum EA, Nociti FH, Jr. Smoking modulates interleukin-6:interleukin-10 and RANKL: osteoprotegerin ratios in the periodontal tissues. J Periodont Res. 2007;42(2):184-91.
48. Chang CH, Han ML, Teng NC, Lee CY, Huang WT, Lin CT, et al. Cigarette Smoking Aggravates the Activity of Periodontal Disease by Disrupting Redox Homeostasis- An Observational Study. Sci Rep. 2018;8(1):11055.
49. Boesing F, Patiño JS, da Silva VR, Moreira EA. The interface between obesity and periodontitis with emphasis on oxidative stress and inflammatory response. Obesity Rev. 2009;10(3):290-7.
50. Atabay VE, Lutfioğlu M, Avci B, Sakallioglu EE, Aydoğdu A. Obesity and oxidative stress in patients with different periodontal status: a case-control study. J Periodont Res. 2017;52(1):51-60.
51. Boo YC. Ascorbic Acid (Vitamin C) as a Cosmeceutical to Increase Dermal Collagen for Skin Antiaging Purposes: Emerging Combination Therapies. Antioxidants (Basel, Switzerland). 2022;11(9).
52. He L, He T, Farrar S, Ji L, Liu T, Ma X. Antioxidants Maintain Cellular Redox Homeostasis by Elimination of Reactive Oxygen Species. Cell Physiol Biochem. 2017;44(2):532-53.
53. Kruk J, Aboul-Enein BH, Duchnik E, Marchlewicz M. Antioxidative properties of phenolic compounds and their effect on oxidative stress induced by severe physical exercise. J Physiolog Sci. 2022;72(1):19.
54. Galli F, Bonomini M, Bartolini D, Zatini L, Reboldi G, Marcantonini G, et al. Vitamin E (Alpha-Tocopherol) Metabolism and Nutrition in Chronic Kidney Disease. Antioxidants (Basel, Switzerland). 2022;11(5):989.
55. Kerksick C, Willoughby D. The antioxidant role of glutathione and N-acetyl-cysteine supplements and exercise-induced oxidative stress. J Intl Soc Sports Nutr. 2005;2(2):38-44.
56. Bagatini M, Assmann C, Blumenberg M. Glutathione system and oxidative stress in health and disease. BoD–Books on Demand. IntechOpen. 2020.
57. Young AJ, Lowe GL. Carotenoids-Antioxidant Properties. Antioxidants (Basel, Switzerland). 2018;7(2):28.
58. Novakovic N, Todorovic T, Rakic M, et al. Salivary antioxidants as periodontal biomarkers in evaluation of tissue status and treatment outcome. J Periodont Res. 2014;49(1):129-36.
59. Waddington RJ, Moseley R, Embery G. Reactive oxygen species: a potential role in the pathogenesis of periodontal diseases. Oral Dis. 2000;6(3):138-51.
60. Baňasová L, Kamodyová N, Janšáková K, Tóthová Ľ, Stanko P, Turňa J, et al. Salivary DNA and markers of oxidative stress in patients with chronic periodontitis. Clin Oral Investig. 2015;19(2):201-7.
61. Chapple IL, Brock GR, Milward MR, Ling N, Matthews JB. Compromised GCF total antioxidant

capacity in periodontitis: cause or effect? J Clin Periodontol. 2007;34(2):103-10.
62. Baser U, Gamsiz-Isik H, Cifcibasi E, Ademoglu E, Yalcin F. Plasma and salivary total antioxidant capacity in healthy controls compared with aggressive and chronic periodontitis patients. Saudi Med J. 2015;36(7):856-61.

Cite this article as: Albalbisi AM, Asiri MS, Alshahrani NF, Alqhtani SA, Alqahtani SS, Alqahtani SA, et al. Oxidative stress and its contribution to chronic periodontal inflammation. Int J Community Med Public Health 2025;12:4782-9.