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Mini Review

# The Relationship Between Hypertension and Periodontitis: A Minireview

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## Abstract

The Perio medicine relationship between Hypertension and periodontitis have been widely addressed in many studies. Hypertension and periodontitis belong to a group of illnesses called non-communicable diseases (NCDs); they are rising in prevalence due to an increasingly aging population. NCDs comprise 71% of global deaths. The host microbial interaction, oxidative stress, the levels of nitrous oxide, endothelial dysfunction and increased levels of pro-inflammatory cytokines are possible links. Periodontitis patients often manifest higher arterial blood pressure and higher prevalence rates of hypertension. After performing non-surgical periodontal therapy (NSPT) hypertension patients showed lower levels of blood pressure. The purpose of this minireview is to highlight recent studies that show strong associations between both diseases and demonstrate novel approaches in diagnosing, predicting and preventing the occurrence of both chronic inflammatory conditions thus reducing the global burden and improving lifestyle.

**Keywords:** Hypertension; Periodontitis; NSPT

## Abbreviations

NSPT: Non-Surgical Periodontal Therapy; ELISA: Enzyme Linked Immunosorbent Assay; HT: Hypertension, PD: Periodontitis; NCDs: Non-Communicable Diseases

## Introduction

In a world with an aging population with an expected rise in the mean age of the population from 30 in 2021 to 36 in 2050 [1]. The prevalence of chronic conditions like Hypertension (HT) and Periodontitis (PD) rise dramatically, becoming 60-70% of people above 65 years old and 50-70 % of people above 65 years old, respectively [2,3]. This makes it crucial for our attention to shift towards controlling those diseases to improve the lifestyle of the aging population and reduce their death rates. Periodontal disease has a strong inflammatory basis induced by complex bacterial species that elicit host response and result in consequent damage

of the teeth' supporting tissues, including the gingiva, cementum, alveolar bone, and periodontal ligaments [4]. Many modifiable risk factors like smoking, poor oral hygiene, diabetes, age, and stress are linked to periodontitis. It's equally important to mention that Periodontitis is a modifiable risk factor for many systemic conditions like cardiovascular diseases (CVDS), diabetes, and adverse pregnancy outcomes like preterm/ low birth weight [5].

Hypertension is a condition in which there is a sustained elevation in arterial blood pressure [6]. It is a multifactorial disease that can be caused by endothelial dysfunction (ED), oxidative stress, and inflammation. The host-bacterial interaction is the link between periodontitis and hypertension, as well as other chronic illnesses like Diabetes Mellitus and Alzheimer's [7].

Among studies, a Case-control study based the link between both diseases on the fact that periodontitis patients' show higher arterial BP values and a 30% to 70% higher chance of also being diagnosed with hypertension, specifically in the presence of gingivitis [8].

Periodontitis patients express elevated levels of biomarkers of inflammation, like C-reactive protein (CRP), TNF-α, neutrophilic enzymes, WBCs, disparity in T-cell subtypes, neutrophil dysfunction, it's important to note that all of which play a role in changes in vasculature as well as endothelial dysfunction (ED). Pathogens related to periodontitis showed a relationship to hypertension in epidemiological studies. Periodontal pathogens result in an elongated time of T-cell activation and elevated concentrations of CRP, TNF-α, and IL (interleukin)-1β, inducing elevated BP values [8].

The first study to suggest a correlation between periodontal pathogens and hypertension was INVEST (infections and vascular disease epidemiology study) in 2012. Its results concluded that showed a positive correlation between the number of subgingival periodontal pathogens and both systolic blood pressure (SBP) and diastolic blood pressure (DBP) as well as hypertension prevalence [9].

Thus, it's of utmost importance to highlight the possible links between those two diseases and to present different modalities in diagnosis to halt these diseases and reduce their global burden.

## Etiopathogenesis of periodontitis

If an optimum environment for bacteria exists it would be the oral cavity. It provides a niche rich in nutrients essential for the sustainable living of microorganisms in a complex biofilm. It's estimated that 700-1000 species of bacteria reside in the dental biofilm [10]. These bacterial species cause Periodontitis, not in their planktonic free form, but as they live in a complex tenaciously adherent biofilm [5].

The primary host-response is inflammation. The inflammatory process starts with vasodilation, increased capillary permeability, and recruitment of innate immune cells like Neutrophils and Macrophages, these cells not only kill the pathogen they also release pro-inflammatory cytokines like Interleukin-1 (IL-1), Interleukin-6 (IL-6), Tumor necrosis factor-α (TNF-α) that attract other cells as well as further degrade the pathogen and facilitate its engulfment and presentation by Antigen presenting cells (APCs) to be recognized and counteracted by adaptive immune [11].

In case of a prolonged and exaggerated bacterial stimulus, excessive super oxygen ions, along with pro-inflammatory cytokines induce destruction of the connective tissue matrix and enhance bone resorption, which leads to the bystander damage of the periodontium and consequent tooth loss if left untreated [12].

Risk factors for PD are inclusive of poor oral hygiene, smoking, diabetes mellitus, osteoporosis, rheumatoid arthritis (RA), obesity and stress. Additionally, many polymorphisms in genes have been inconsistently related to an elevated chance of periodontitis and atherosclerotic cardiovascular disease (ASVD) [13].

## Diagnosis and Therapy of periodontitis

Bleeding on probing (BOP), probing depth (PD), clinical attachment level (CAL), plaque index (PI) and radiographic bone loss, are the basis of the traditional diagnosis of periodontitis [14].

Oral fluid-based point-of-care diagnostic procedures (Ex: GCF samples) have been documented as potentially better tests for diagnosing periodontal diseases. A mutual secretion of pro-inflammatory cytokines, including interleukin (IL)-1β, IL-6, and tumor necrosis factor (TNF)-α is present in both diseases and is believed to play a significant role in their pathophysiology [15-19].

The investigation of cytokines and their role in the pathogenesis of periodontitis have significantly contributed to a more reliable assessment of the current disease status (health vs disease) especially when correlated to clinical findings like BOP, PD and CAL thus facilitating early diagnosis. Also, assessing the progression rate along with highlighting potential therapeutic strategies targeting cytokines [20-24]. Although Non-surgical periodontal therapy (NSPT) is the gold standard when it comes to the therapy of periodontitis as well as studies show its effect in reducing cardiovascular risk markers as well as reducing blood pressure [8,25]. Adjunctive periodontal therapy aiming at host modulation can be pivotal in improving efficacy of NSPT. The use of monoclonal antibodies against IL1-β (e.g., canakinumab) or TNF-α (e.g., infliximab, etanercept), are currently being used for treating RA patients', which is also a result of a dysregulated immune system, this paves the road to potential control over periodontal disease [26].

## Systemic effect of periodontitis

The local inflammatory mediators associated with periodontitis don't remain confined to the oral cavity but rather spread through the systemic circulation and share in the occurrence as well as development of major illnesses by causing systemic low-grade inflammation (LGI) [27].

The main pro-inflammatory mediators in periodontitis that affect the general health are IL-1$\beta$, IL-6, TNF-$\alpha$, and IL-17 [28]. There's so much evidence supporting the link between PD and Diabetes mellitus. This is manifested in poor control over blood glucose levels through increasing insulin resistance by increasing concentrations of IL-1$\beta$, IL-6, and TNF-$\alpha$. Hyper glycemia in terms increases the levels of proinflammatory cytokines and creates an infinite loop of inflammation [29-31].

Periodontitis and cardiovascular diseases have a possible link that is well established through available evidence. TNF-α also regulates Angiogenesis, which is the process of new capillary formation, through stimulating Vascular Endothelial Growth Factor 2 (VGEF-2), which plays a destructive role on the periodontium. There are findings given that TNF-α upregulates secretion of Endocan, a marker of endothelial dysfunction which is one of the early signs of CVDs [16,32,33]. Pro-inflammatory cytokines, like IL-1β and IL-6, increase vascular inflammation and promote prothrombotic events, whereas IL-17 has been associated with the process of new blood vessels formation through its influence on the endothelium function as well as smooth vascular muscle cell growth and increase in number. The cumulative systemic inflammation generated by these mediators may provide a biological foundation for the observed epidemiological correlation between compromised periodontal health and an elevated risk of cardiovascular disease [34].

Another strongly related systemic condition to periodontitis is RA whereas Both diseases show a dysregulated immune response associated with bystanding tissue destruction with similar increase in cytokines levels including IL-1$\beta$, TNF-$\alpha$, and IL-17. Among the key pathogenic micro-organisms in periodontal disease is Porphyromonas gingivalis (PG). It plays a role in RA by inducing deamination of host proteins, possibly inducing an autoimmune response in patients with high genetic susceptibility. This further proves the common pathway between both diseases and how periodontitis induced LGI can worsen symptoms of RA like joint pain and edema [27].

## Hypertension

Hypertension is considered the most common of all cardiovascular diseases; thus, it is the number one reason for global death worldwide. Its prevalence is more than a billion people worldwide. It's often referred to as the "silent killer" as it's asymptomatic in most cases [35].

The ISH 2020 guidelines define hypertension as repeated office blood pressure values equal to or more than 140 mm/hg systolic blood pressure and equal to or more than 90 mm/hg diastolic blood pressure [36].

Hypertension can be classified as essential (or primary) hypertension and secondary hypertension. Essential hypertension, which accounts for 90-95% of all hypertension cases, is characterized by the absence of a specific underlying cause. In contrast, secondary hypertension, which is attributed to an identifiable cause, affects approximately 5-10% of grown-ups in the United States of America with a positive diagnosis of hypertension [37].

According to the WHO 2023, there are many risk factors for hypertension, the major ones being genetics, older age, obesity, high-salt diet, and drinking too much alcohol. Chronic

periodontitis has been associated with increased atherogenesis and thromboembolic events thus contributing to the incidence of CVDs including hypertension [38].

## Etiopathogenesis of hypertension

It is a complex disorder caused by the interplay between genetics and environmental factors. Increase in salt intake is associated with impaired Renin angiotensin aldosterone system (RAAS) this leads to increasing blood volume this increases sympathetic nervous system activation and increases peripheral resistance thus the blood pressure increases. Obesity as part of the metabolic syndrome causes insulin resistance which further increases sodium retention [39-41].

Another possible mechanism for hypertension is Oxidative stress; it can be explained as the lack of Nitrous oxide (NO)/ Reactive Oxygen species (ROS) balance in favor of ROS. ROS release is increased in response to many factors including chronic inflammation, smoking, high fat diet, high coffee and alcohol consumption. These factors result in an increase in production of ROS which is associated with damage to DNA and RNA leading to damage on the level of cells and tissues. NO on the other hand is a potent vasodilator released by endothelial cells, in the presence of high amounts of ROS the NO availability is reduced leading to endothelial dysfunction, stiffer blood vessels, more vasoconstriction and increase in blood pressure [42].

## Possible links between periodontitis and hypertension

### Periodontal pathogen

Many epidemiological studies link Hypertension to periodontal related micro-organisms. Periodontal pathogens cause an increased time of T-cell activation and elicited increased levels of CRP, TNF-α, and IL (interleukin)-1β, leading to an elevated BP value [8].

### Common inflammatory pathway

Periodontitis patients express elevated levels of local and systemic inflammatory biomarkers, such as C-reactive protein (CRP), TNF-α, neutrophilic enzymes, WBCs, disparity in T-cell subtypes, neutrophil dysfunction, which are all mechanisms resulting in vascular changes and endothelial dysfunction (ED) [8].

### Macro and Micro vasculature damage and ischemia

The atherosclerotic events associated with hypertension cause ischemia and result in microvascular damage in the periodontium thus increasing the severity of periodontitis [35]. On the other hand, the chronic inflammatory state that exists in chronic periodontitis is associated with further destruction of the vasculature along with endothelial dysfunction which in terms increases the risk for hypertension [13].

### Endothelial dysfunction

Endothelial dysfunction is a common pathway between the two diseases and represents one of the earliest steps in the pathogenesis of hypertension. It is characterized by impaired vasodilation, increased arterial stiffness, and a pro-inflammatory vascular state. Patients with periodontitis demonstrate measurable endothelial dysfunction, assessed by flow-mediated dilation and carotid intima-media thickness, which parallels findings in hypertensive populations [43]. In a study conducted by Kumar in 2020, there was a statistically significant decrease in Endocan ($p < 0.01$) among all the groups after NSPT [44].

### Oxidative stress

The ROS increase in hypertension is considered a mediator of vasoconstriction and vascular inflammation [45]. A cross-sectional study among the French population in 2010 suggests that periodontitis increases the risk of metabolic syndromes (MS), characterized by oxidative stress and diabetes. On the basis of a systematic review and meta-analysis, there was strong evidence for an association between MS and PD. Thus, insulin resistance and oxidative stress are cornerstones in MS, periodontitis, CVD and hypertension [46-48].

### Evidence across studies

Among studies, a Case-control study based the link between both diseases on the fact that periodontitis patients' show higher arterial BP values and a 30% to 70% higher chance of also being diagnosed with hypertension, specifically in the presence of gingivitis [8].

A study showed that untreated periodontitis was positively correlated to increased levels of both systolic and diastolic blood pressure in adults, while another study showed that NSPT reduced systemic inflammatory biomarkers and improved vascular health among hypertensive patients [47,49].

The effect of NSPT on blood pressure values is a point of debate among studies. Many recent studies prove that NSPT lowers

systolic and diastolic blood pressure [25,49-51]. On the contrary, a systematic review of randomized controlled trials, for example, did not find consistent reductions in systolic or diastolic blood pressure after NSPT [52]. This can be explained that the direct hemodynamic effects of NSPT may be limited in the short term and require longer follow-ups and larger samples [50,51].

## Conclusion

The relationship between periodontitis and hypertension is complex and deeply rooted in chronic inflammation. There is a rich pool of evidence available that supports their interplay. Within the available evidence there are some limitations including the need for larger sample sizes, longer follow-up durations, measurement of both systemic and local biomarkers for a more precise assessment of both conditions. The employment of biomarkers quantification in the process of diagnosis results in high precision and opens doors to novel cytokines targeted therapy.

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Doesn't apply.

## Conflict of Interest

There's no conflict of interest.

## Disclosure

A related original research manuscript by the authors is currently under review in another journal. The present submission is a narrative mini review and does not include overlapping data.

## Bibliography

1. United Nations, Department of Economic and Social Affairs, Population Division. World Population Prospects 2022: Summary of Results. United Nations (2022).

2. Mills Katherine T., *et al*. "Global Disparities of Hypertension Prevalence and Control: A Systematic Analysis of Population-Based Studies from 90 Countries". *Circulation* 134.6 (2016): 441-450.

3. Kassebaum Nicholas J., *et al*. "Global Burden of Severe Periodontitis in 1990-2010: A Systematic Review and Meta-Regression". *The Lancet* 381.9879 (2013): 1495-1504.

4. Könönen Eija., *et al*. "Periodontitis: A Multifaceted Disease of Tooth-Supporting Tissues". J*ournal of Clinical Medicine* 8.8 (2019): 1135.

5. Nazir Muhammad Ashraf. "Prevalence of Periodontal Disease, Its Association with Systemic Diseases and Prevention". *International Journal of Health Sciences* 11.2 (2017): 72-80.

6. Whelton Paul K., *et al*. "2017 Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults". *Journal of the American College of Cardiology* 71.19 (2018): e127-e248.

7. Muñoz Aguilera Eduardo, *et al*. "Periodontitis Is Associated with Hypertension: A Systematic Review and Meta-Analysis". *Cardiovascular Research* 116.1 (2020): 28-39.

8. Muñoz Aguilera Eduardo, *et al*. "Association Between Periodontitis and Blood Pressure Highlighted in Systemically Healthy Individuals". *Hypertension* 77.5 (2021): 1765-1774.

9. Desvarieux Moïse., *et al*. "Periodontal Bacteria and Hypertension: The Oral Infections and Vascular Disease Epidemiology Study (INVEST)". *Journal of Hypertension* 28.7 (2010): 1413-1421.

10. Lazar V., *et al*. "Impact of Dental Plaque Biofilms in Periodontal Disease: Management and Future Therapy". *Periodontitis - A Useful Reference* (2017).

11. Cekici Ayca., *et al*. "Inflammatory and Immune Pathways in the Pathogenesis of Periodontal Disease". *Periodontology* 2000 64.1 (2014): 57-80.

12. Segal Anthony W. "How Neutrophils Kill Microbes". *Annual Review of Immunology* 23 (2005): 197-223.

13. Schaefer Andreas S., *et al*. "Genetic Susceptibility to Periodontal Disease and Its Association with Cardiovascular Disease". *Journal of Clinical Periodontology* 36.s10 (2009): 65-79.

14. Tonetti Maurizio S., *et al*. "Staging and Grading of Periodontitis: Framework and Proposal of a New Classification". *Journal of Clinical Periodontology* 45.S20 (2018): S149-S161.

15. Rosa Rafael AC., *et al*. "The Relationship between Hypertension and Periodontitis: A Cross-Sectional Study". *Journal of Clinical Medicine* (2023).

16. Singh Siddharth, *et al*. "Obesity and Response to Anti-Tumor Necrosis Factor-α Agents in Patients with Select Immune-Mediated Inflammatory Diseases: A Systematic Review and Meta-Analysis". *PLOS ONE* 13.10 (2018): e0195123.

17. Dinarello Charles A. "Proinflammatory Cytokines". *Chest* 118.2 (2000): 503-508.

18. Yilmaz Mahmut I., *et al*. "Plasma Endocan Levels Associate with Inflammation, Vascular Abnormalities, Cardiovascular Events, and Survival in Chronic Kidney Disease". *Kidney International* 86.6 (2014): 1213-1220.

19. Hirooka Yoshitaka. "How Can Endocan Be Used as a Specific Biomarker of Endothelial Dysfunction in Hypertension?" *Hypertension Research* 47.3 (2024): 794-795.

20. Tan A., *et al*. "Increase in Serum and Salivary Neutrophil Gelatinase-Associated Lipocalin Levels with Increased Periodontal Inflammation". *Journal of Applied Oral Science* 28 (2020): e20200276.

21. Holmström S B., *et al*. "MMP-12 and S100s in Saliva Reflect Different Aspects of Periodontal Inflammation". *Cytokine* 113 (2019): 155-161.

22. Lorenz K., *et al*. "Evaluation of a Novel Point-of-Care Test for Active Matrix Metalloproteinase-8: Agreement between Qualitative and Quantitative Measurements and Relation to Periodontal Inflammation". *Journal of Periodontal Research* 52 (2017): 277-284.

23. Dolińska E., *et al*. "Periodontal Molecular Diagnostics: State of Knowledge and Future Prospects for Clinical Application". *International Journal of Molecular Sciences* 25 (2024): 12624.

24. Moore G. "The Effect of Interproximal Home Regimens on Inflammatory Biomarkers in Periodontal Maintenance Patients". Master's thesis, University of Nebraska Medical Center, Omaha, NE, USA (2022).

25. Meng Rijing., *et al*. "Effect of Non-Surgical Periodontal Therapy on Risk Markers of Cardiovascular Disease: A Systematic Review and Meta-Analysis". *BMC Oral Health* 24.1 (2024): 1.

26. Church Lucy D and M F McDermott. "Canakinumab, a Fully-Human mAb against IL-1beta for the Potential Treatment of Inflammatory Disorders". *Current Opinion in Molecular Therapeutics* 11 (2009): 81-89.

27. Martínez-García M and E Hernández-Lemus. "Periodontal Inflammation and Systemic Diseases: An Overview". *Frontiers in Physiology* 12 (2021): article 709438.

28. Mazurek-Mochol Małgorzata., *et al*. "The Role of Interleukin 6 in Periodontitis and Its Complications". *International Journal of Molecular Sciences* 25.4 (2024): article 2146.

29. Cecoro Giulia., *et al*. "Periodontitis, Low-Grade Inflammation and Systemic Health: A Scoping Review". *Medicina* 56.6 (2020): article 272.

30. Baeza M., *et al*. "Effect of Periodontal Treatment in Patients with Periodontitis and Diabetes: Systematic Review and Meta-Analysis". *Journal of Applied Oral Science* 28 (2020): e20190248.

31. Cardoso E M., *et al*. "Chronic Periodontitis, Inflammatory Cytokines, and Interrelationship with Other Chronic Diseases". *Postgraduate Medicine* 130.1 (2018): 98-104.

32. Whayne Thomas F. "Endocan in Hypertension and Cardiovascular Disease". *Angiology* 65.9 (2014): 757-759.

33. Roudnicky Florian., *et al*. "Endocan Is Upregulated on Tumor Vessels in Invasive Bladder Cancer Where It Mediates VEGF-A-Induced Angiogenesis". *Cancer Research* 73.4 (2013): 1097-1106.

34. Carrizales-Sepúlveda Emmanuel F., *et al*. "Periodontal Disease, Systemic Inflammation and the Risk of Cardiovascular Disease". *Heart, Lung and Circulation* 27.11 (2018): 1327-1334.

35. Macedo Paizan M L and J F Vilela-Martin. "Is There an Association between Periodontitis and Hypertension?" *Current Cardiology Reviews* 10.4 (2014): 355-361.

36. Unger Thomas, *et al*. "Risk Factors for Periodontitis". *Journal of the International Academy of Periodontology* 7.1 (2005): 3-7.

37. Southerland JH., *et al*. "Dental Management in Patients with Hypertension: Challenges and Solutions". *Clinical, Cosmetic and Investigational Dentistry* 8 (2016): 111-120.

38. Joshipura Kaumudi J., *et al*. "Periodontal Disease, Tooth Loss, and Incidence of Ischemic Stroke". *Stroke* 34.1 (2003): 47-52.

39. Iqbal Asma M and Syed F Jamal. "Essential Hypertension". StatPearls, StatPearls Publishing (2023).

40. DeFronzo Ralph A., *et al*. "The Effect of Insulin on Renal Handling of Sodium, Potassium, Calcium, and Phosphate in Man". *Journal of Clinical Investigation* 55 (1975): 845-855.

41. Hammerman Michael R., *et al*. "Insulin Stimulates Pi Transport in Brush Border Vesicles from Proximal Tubular Segments". *American Journal of Physiology-Endocrinology and Metabolism* 247.5 (1984): E616-E624.

42. Takahashi Hakuo., *et al*. "The Central Mechanism Underlying Hypertension: A Review of the Roles of Sodium Ions, Epithelial Sodium Channels, the Renin-Angiotensin-Aldosterone System, Oxidative Stress and Endogenous Digitalis in the Brain". *Hypertension Research* 34.11 (2011): 1147-1160.

43. Sanz Mariano., *et al*. "Periodontitis and Cardiovascular Diseases: Consensus Report". *Journal of Clinical Periodontology* 47.3 (2020): 268-288.

44. Kumar Gayathri., *et al*. "Evaluation of Endocan and Tumor Necrosis Factor-α as Inflammatory Biomarkers in Type 2 Diabetes and Periodontal Disease". *Genetic Testing and Molecular Biomarkers* 24.7 (2020): 431-435.

45. D'Aiuto, Francesco, Marco Orlandi, and John C. Gunsolley. "Evidence That Periodontal Treatment Improves Biomarkers and Cardiovascular Disease Outcomes". *Journal of Periodontology* 84.4-s (2013): S85-S105.

46. Benguigui C., *et al*. "Metabolic Syndrome, Insulin Resistance, and Periodontitis: A Cross-Sectional Study in a Middle-Aged French Population". *Journal of Clinical Periodontology* 37.7 (2010): 601-608.

47. Pietropaoli Daniele., *et al*. "Poor Oral Health and Blood Pressure Control Among US Hypertensive Adults". *Hypertension* 72.6 (2018): 1365-1373.

48. Nibali Luigi., *et al*. "Association between Metabolic Syndrome and Periodontitis: A Systematic Review and Meta-Analysis". *The Journal of Clinical Endocrinology and Metabolism* 98.3 (2013): 913-920.

49. Rodrigues J V S., *et al*. "The Effect of Non-Surgical Periodontal Treatment on Patients with Combined Refractory Arterial Hypertension and Stage III, Grade B Periodontitis: A Preliminary Prospective Clinical Study". *Journal of Clinical Medicine* 12 (2023): 4277.

50. Lanau Núria., *et al*. "Impact of Nonsurgical Periodontal Treatment on Blood Pressure: A Prospective Cohort Study". *European Journal of Dentistry* 18.2 (2023): 517.

51. Gatarayiha Agnes, *et al.* "Oral Hygiene and Periodontal Treatment Interventions for Reducing Blood Pressure in Hypertensive Adults from Low and Middle-Income Countries: A Systematic Review". *PAMJ Clinical Medicine* 15 (2024): article 20.

52. Luo Yuxue., *et al*. "Effect of Periodontal Treatments on Blood Pressure". *Cochrane Database of Systematic Reviews* 12 (2021): article CD009409.