INTERNATIONAL JOURNAL OF PHARMACEUTICAL SCIENCES
[ISSN: 0975-4725; CODEN(USA): IJPS00]
Journal Homepage: https://www.ijpsjournal.com

OPEN ACCESS

Review Article

Personalized Periodontics: Review

Nowfiya M*, Deepa S, Jenifer Cynthia R A, Shanmuga priya R, Arunmozhi U

Sri Venkateswara Dental College and Hospital, Chennai, Tamil Nadu, India

ARTICLE INFO

Published: 18 Sept 2026
Keywords:
personalized periodontics; precision medicine; P4 periodontics; periodontal biomarkers; risk factors; periodontitis
DOI:
10.5281/zenodo.22842324

ABSTRACT

Periodontitis is a multifactorial, host microbial interaction whose onset and progression are governed by the interaction of environmental, genetic/epigenetic, microbial, and lifestyle factors.9,10 Traditional periodontal therapy follows a uniform protocol aimed at eliminating deep pockets, reducing bleeding, and achieving plaque control, yet nearly one-fifth of patients respond inadequately to this conventional therapy.2,7 This has driven a paradigm shift toward personalized (precision) periodontics, an approach in which patients are grouped into distinct groups so that diagnosis, prevention, and treatment can be tailored to the particular individual.3,4 This review covers the P4 framework of personalized periodontics (Personalization, Prediction, Prevention, Participation), its precision-medicine ecosystem, its diagnostic toolkit — salivary, GCF, plaque-based point-of-care tests, spectroscopic methods, and bone-loss imaging.5,15,17 The role of the periodontal microbiome (keystone-pathogen hypothesis), host genetics and epigenetic regulation, and major systemic and behavioral risk factors — smoking, diabetes mellitus, cardiovascular disease, obesity, psychological stress, and rheumatoid arthritis — in shaping individualized risk and treatment planning is discussed in detail, along with practical management protocols for each.18,20,24,32,35 This narrative review going to deal with Personalized periodontics.

INTRODUCTION

Periodontal disease (gingivitis and periodontitis) begins with subclinical inflammation within 2 to 4 days of its early stages.[9] This stage typically produces no perceptible symptoms, so a large proportion of affected individuals remain unaware of the disease until significant damage has occurred.[8] Left untreated, it progresses to gingival bleeding, tooth mobility, bone loss and tooth loss, which increases the systemic morbidity, and considerable financial burden on both the patient and the healthcare system.[9,10] Periodontitis is now recognized as a complex disease in which multiple causal risk factors act simultaneously to drive its onset and progression.[1,9] Environmental exposures, genetic and epigenetic predisposition, lifestyle behaviors, and coexisting systemic

***Corresponding Author:** Nowfiya M
Address: *Sri Venkateswara Dental College and Hospital, Chennai, Tamil Nadu, India*
Email : nowfiyam661@gmail.com
Relevant conflicts of interest/financial disclosures: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

diseases interact continuously, together shaping an individual's susceptibility to, and trajectory of, periodontal breakdown[1,8,9,10]

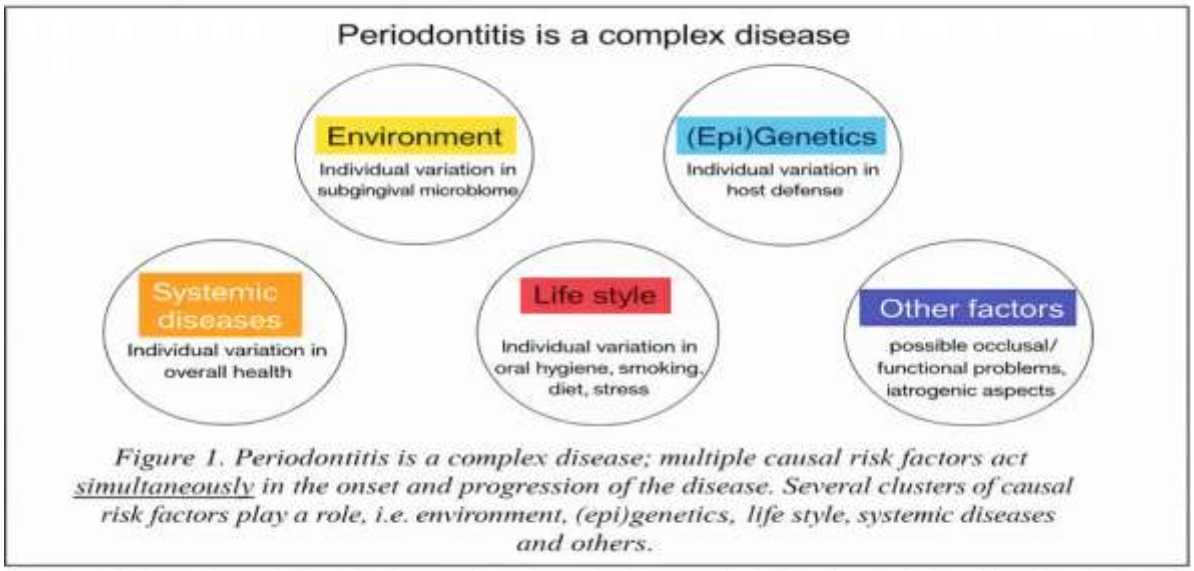


Figure 1. Periodontitis is a complex disease; multiple causal risk factors act simultaneously in the onset and progression of the disease. Several clusters of causal risk factors play a role, i.e. envir-

Adapted from, Loos BG, Van Dyke TE. The role of inflammation and genetics in periodontal disease. Periodontology 2000. 2020;83(1).

2. From Traditional to Contemporary Periodontal Therapy

2.1 Goals of Traditional Periodontal Therapy

The goals of traditional periodontal therapy are the reduction of gingival bleeding, elimination of deep pockets and achievement of adequate plaque control.[2] This is conventionally achieved by treating periodontal disease as an opportunistic infection modified by the host's inflammatory response, controlling infection and inflammation, reducing predisposing and modifying factors, and providing continuous assessment through supportive periodontal care.[7] However, clinical evidence indicates that nearly 20% of patients do not respond adequately to this standardized regimen, exposing the limitations of a uniform treatment.[2,7]

2.2 The Shift Toward Contemporary, Individualized Care

Modern periodontics recognizes that disease progression varies according to genetic background, host immune response, and environmental influence.[3] Traditional therapy follows a one-size-fits-all model, but because of this individual variation a subset of patients remain non-responders.[4] Personalized treatment has therefore become a clinical necessity rather than an option.[3,4]

3. Defining Personalized Periodontics

Personalized periodontics has been defined, in Periodontology 2000 (2018), as the separation of patients into different groups, with clinical decisions, practices, and treatments customized according to the individual patient.[2] The overarching purposes of personalized periodontal therapy are to select the optimal therapy for a given patient, improve treatment outcomes, and predict

disease susceptibility before overt clinical presentation.[2,6]

4. The P4 Model of Periodontics

P4 periodontics represents an integrative framework for periodontal care built around four interlinked components: **P**ersonalization, **P**rediction, **P**revention, and **P**articipation. [13]

Personalization involves identifying the specific disease pathway operating in a given patient, understanding their individual pattern of disease progression, and tailoring treatment accordingly.[3] It draws on the patient's genetic profile, microbial composition, inflammatory response, and systemic health status to move away from the traditional "one-size-fits-all" protocol toward a stratified model of care, in which patients with similar disease phenotypes and risk profiles are grouped together and managed with regimens suited to that group.[4] In practice, this translates into individualized decisions on the choice of non-surgical versus adjunctive antimicrobial or regenerative therapy, the frequency of supportive periodontal maintenance, and the selection of implant versus conventional prosthetic rehabilitation based on a patient's specific healing capacity and risk profile. [3,4]

Prediction emphasizes assessing risk before symptoms appear, using genetic markers and biomarkers to detect susceptible individuals at an early, pre-clinical stage.[13] This is achieved through host-genetic testing (for example, Interleukin-1 [IL-1] gene polymorphisms), salivary and gingival crevicular fluid (GCF) biomarker panels, subgingival microbial profiling, and, increasingly, Artificial Intelligence (AI)-assisted risk-calculators that combine clinical, microbial, and genetic data into a single susceptibility score.[4,37] By identifying at-risk individuals before clinically detectable attachment loss occurs, prediction allows clinicians to intervene during the subclinical phase of disease, when treatment is simplest and most cost-effective. [4,13]

Prevention focuses on early diagnosis, effective plaque control, and lifestyle modification to reduce disease occurrence altogether.[13] Rather than a single generic recall schedule, prevention under the P4 model is risk-stratified: patients identified as high-risk through the prediction pillar receive more frequent supportive periodontal therapy, targeted oral-hygiene instruction, and closer monitoring, while low-risk patients follow a standard maintenance interval.[3,4] Prevention also extends to modifying shared systemic and behavioral risk factors — such as smoking cessation counselling and glycaemic control in patients with diabetes mellitus — thereby addressing periodontal risk and general health simultaneously. [3,13,20,27]

Participation underscores the importance of active patient involvement — maintaining good oral hygiene, adopting healthier lifestyle behaviors, and attending regular periodontal recall visits.[13] It reframes the patient from a passive recipient of care into an informed partner in decision-making, achieved through patient education about their individual risk profile, shared goal-setting, motivational interviewing to support behaviour change, and the use of digital tools such as mobile reminders and tele-monitoring to reinforce compliance between visits.[13] Sustained participation is essential for the long-term success of the other three P's, since even an accurately predicted and personalized treatment plan will fail without consistent patient adherence. [13]

Collectively, the P4 model works by modifying modifiable risk factors and embedding a comprehensive, patient-centered strategy into everyday periodontal care.[3]

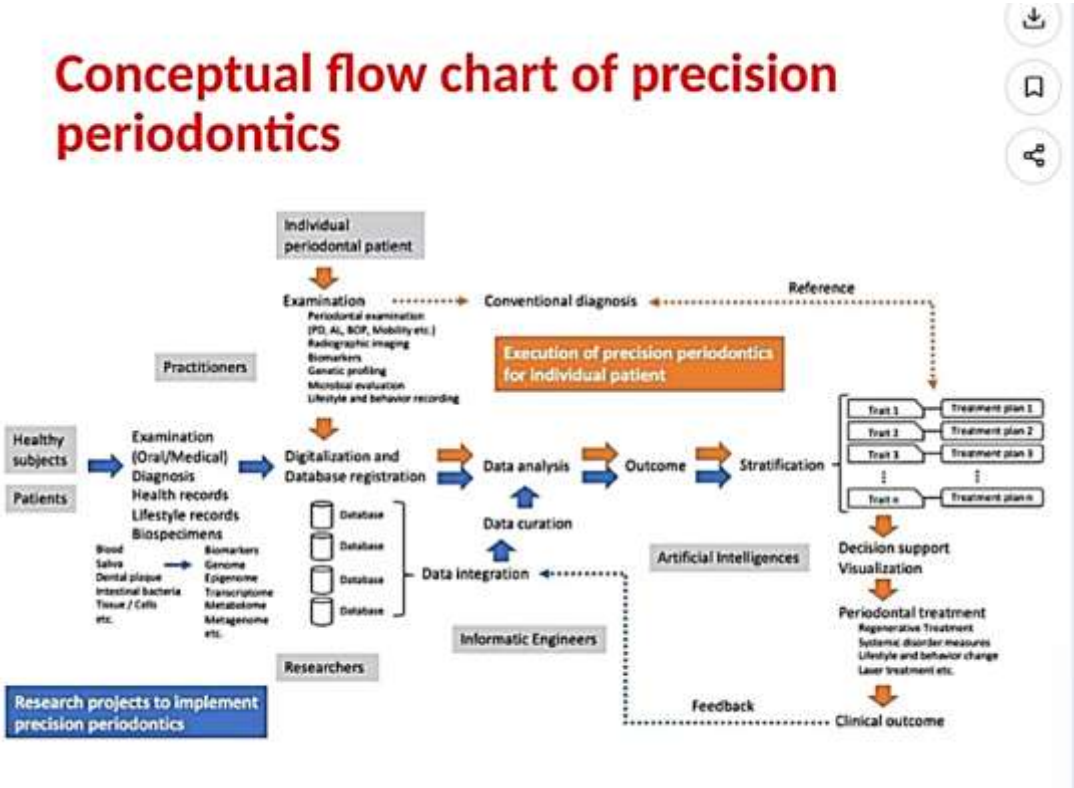
5. Conceptual Framework of Precision Periodontics

5.1 The Diagnostic-to-Treatment Flow

The process begins with comprehensive patient examination — clinical parameters (probing depth, attachment loss, bleeding on probing, mobility), radiographs, biomarkers, genetics, microbial profiling, and lifestyle factors — combined with medical history and biospecimens (blood, saliva, plaque, tissue). This data is digitized into structured databases.[5]

Multi-omics layers (genomics, epigenomics, transcriptomics, metabolomics, metagenomics) are integrated and analyzed using Artificial Intelligence (AI)-driven computational tools, guided by informatics specialists. This enables patient stratification and generates individualized treatment plans.[5]

Decision-support and visualization tools then help clinicians select therapy — regenerative procedures, systemic disease management, lifestyle modification, or laser treatment. Outcomes are monitored continuously, with feedback looped back into the system — closing the loop toward truly personalized, precise periodontal care.[5]



Adapted from - Kikuchi T, Hayashi J, Mitani A. Next-Generation Examination, Diagnosis, and Personalized Medicine in Periodontal Disease. *J Pers Med.* 2022 (5)

5.2 The Precision Medicine Ecosystem

The precision medicine ecosystem begins with the patient, whose information — family history, environmental exposures, genotype, phenotype, and clinical outcomes — is recorded within electronic health records.[14] In parallel, biospecimens such as blood, saliva, or tissue are collected and analyzed by researchers to generate research data.[16] Clinicians interact with these electronic records to guide diagnosis and treatment, while clinical laboratories contribute

additional diagnostic information.[14] All of this data is organized into a curated, continuously refined database that supports both clinicians and researchers, enabling data-driven decision-making.[16] A dynamic feedback loop results: research findings improve clinical practice, and clinical outcomes, in turn, enrich the research dataset, together advancing personalized and precision-based patient care.[14,16]

6. Diagnostic Approaches in Precision Periodontics

Examination and diagnosis in precision periodontics increasingly rely on biomarkers for both the diagnosis and prognosis of periodontal disease.[15] Identifying biomarkers capable of accurately predicting disease progression is essential for early detection and effective disease control.[17] Traditionally, proteomic analysis has used biological samples such as saliva, gingival crevicular fluid, pocket-associated tissue, and serum to comprehensively evaluate disease-related biomarkers.[12] The diagnostic validity of any such biomarker rests on two key parameters — sensitivity, the ability to correctly identify individuals with disease, and specificity, the ability to correctly identify those without disease — which together determine its clinical usefulness in precision periodontal care.[15,17]

6.1 Periodontal Biomarkers

Biomarkers assist across the full continuum of care, from prediction and prevention to diagnosis, treatment, and maintenance of periodontal disease.[12] They are broadly classified into two types: static biomarkers, which are genetic and do not change over time, reflecting inherited susceptibility; and dynamic biomarkers, which are biochemical or microbiological in nature and reflect current disease activity and progression.[11,12,15]

6.2 Point-of-Care Diagnostic Tests

6.2.1 Saliva-Based Tests

Test Kit	Principle	What It Detects	Output
Oral Fluid Nanosensor Test	Nanotechnology-based biosensor detecting biomarkers in saliva	Proteins, enzymes, inflammatory markers, bacterial products	Electronic/digital signal (usually no visible color change)
Electronic Taste Chip	Microelectromechanical sensor analyzing salivary composition	Oral pathogens, metabolites, volatile sulfur compounds (VSCs)	Digital/electronic readout
OraQuick	Immunochromatographic lateral flow assay	Human Immunodeficiency Virus (HIV) antibodies in oral fluid	Colored test line (similar to a pregnancy strip)
Integrated Microfluidic Platform	Lab-on-chip microfluidic analysis	Deoxyribonucleic acid (DNA), Ribonucleic acid (RNA), proteins, inflammatory biomarkers in saliva	Usually fluorescent/ electronic signal
My PerioPath	Polymerase Chain Reaction (PCR)-based microbiological assay	Major periodontal pathogens in saliva	Lab-generated report (no direct color change)
Omnigene	DNA probe / molecular diagnostic test	Specific periodontal bacteria	No color change; molecular report
IAI Pado Test	RNA/DNA probe technology	Periodontal pathogens such as Porphyromonas gingivalis (P. gingivalis), Aggregati bacter actinomycetem comitans (A. actinomycetem comitans)	No visible color change

MyPerioID	Genetic susceptibility test	Interleukin-1 (IL-1) gene polymorphism associated with periodontitis risk	Genetic report; no color change

6.2.2 Gingival Crevicular Fluid (GCF)-Based Tests

Test Kit	Principle	What It Detects	Color Change / Output
Periogard	Enzyme-based assay (AST activity)	Aspartate aminotransferase (AST) released from damaged cells, indicating tissue destruction	Colorless → Purple/Violet
Pocket Watch	Enzymatic detection of proteolytic activity	Neutral proteases from periodontal pathogens	Colorless → Blue
Periocheck	Hydrolysis of a synthetic substrate by enzymes	Neutral proteases, especially from Treponema denticola (T. denticola) and P. gingivalis	Colorless → Blue
Prognostik	Enzyme–substrate reaction	Elastase enzyme from neutrophils, a marker of inflammation	Colorless → Red/Pink
MMP Dipstick Test	Immunochromatographic assay	Matrix metalloproteinase-8 (MMP-8), a collagen breakdown marker	Visible test line (similar to a pregnancy strip)

6.2.3 Plaque-Based Diagnostic Tests

Test Kit	Principle	What It Detects	Color Change / Output
Perioscan (Benzoyl-DL-arginine-naphthylamide [BANA] test)	Hydrolysis of the BANA substrate by bacterial enzymes	'Red complex' bacteria (P. gingivalis, T. denticola, Tannerella forsythia [T. forsythia])	Colorless → Blue/blue-black
Evalusite	Immunoassay using monoclonal antibodies	Specific pathogens: P. gingivalis, A. actinomycetemcomitans, Prevotella intermedia (P. intermedia)	Appearance of colored dots (pink/purple)
Perio 2000	Enzymatic activity assay	Bacterial proteolytic enzymes in plaque	Colorless → Blue
TOPAS (Toxicity Prescreening Assay)	Measures bacterial toxin activity	Toxic metabolites from periodontal pathogens	Color change (yellow → orange/brown) indicating toxicity level
Genetic test kits	DNA probe / PCR-based detection	Specific periodontal pathogens at the genetic level	No color change (lab-based readout/report)

6.3 Physical and Spectroscopic Methods

Physical methods are increasingly applied in periodontal diagnostics to analyze salivary biomarkers and identify disease activity.[17] Broad-spectrum Fluorescence Resonance Energy Transfer (FRET) measures total protease activity in saliva and helps assess periodontal tissue destruction.[17] Infrared Attenuated Total Reflection (IR-ATR) spectroscopy differentiates healthy individuals from periodontitis patients based on salivary spectral patterns.[17] Secondary Electrospray Ionization (SESI) identifies salivary metabolites produced by periodontal pathogens,

aiding disease detection.[17] Collectively, these physical diagnostic methods offer rapid, non-invasive, and sensitive approaches to the early diagnosis and monitoring of periodontitis.[17]

6.4 Diagnostic Imaging

Diagnostic imaging plays an important role in periodontal assessment, particularly in evaluating alveolar bone loss.[38] Lin et al. developed a radiographic analysis method for localizing alveolar bone-loss areas in periodontitis using threshold segmentation combined with hybrid features of intensity and the H-value of the fractional Brownian motion model. This technique effectively identifies and segments bone-loss areas on periodontal radiographs, helping clinicians accurately assess the severity and extent of periodontal destruction, and may assist in treatment planning and monitoring of disease progression.[38]

6.5 Limitations of Conventional Diagnostic Imaging

- Radiological measurements of attachment loss are not precisely accurate.[4]
- Full-mouth recording is necessary because periodontal disease progresses in a site-specific manner.[7]
- Individual susceptibility to periodontitis varies both genetically and over time.[1]
- Clinical diagnostic techniques mainly reflect past disease activity and cannot accurately diagnose present disease activity.[4,7]

6.6 Benefits of Precision Medicine over Conventional Diagnostic Techniques

- Customization of disease prevention strategies.[3]
- More effective, targeted drug prescription.[4]
- Prediction of disease susceptibility.[13]
- Improved disease detection.[4]
- A shift in clinical emphasis from reaction to prevention.[3]
- Elimination of trial-and-error inefficiencies that raise healthcare costs and undermine patient care.[3,4]

12. Limitations of Personalized Periodontics

Despite its considerable promise, personalized periodontics faces several practical barriers to widespread clinical adoption.[4,7]

- Need for specialized knowledge: application requires understanding of genetics, molecular biology, and pharmacogenomics, and many clinicians are not yet trained to interpret genetic data effectively.[4]
- Complexity of genetic factors: periodontal disease is multifactorial (genetic, environmental, and lifestyle influences combined), and a single genetic marker rarely predicts disease or treatment response with accuracy.[7]
- Limited clinical evidence: there remains a shortage of large-scale, long-term studies validating genetic-based therapies, and standardized clinical guidelines are lacking.[4]
- High cost: genetic testing and analysis can be expensive and are not always cost-effective for routine dental practice.[7]
- Time-consuming process: genetic analysis and interpretation can delay treatment planning.[4]

- Variable patient response: even with genetic data available, treatment outcomes may differ owing to environmental factors such as smoking or oral hygiene.[7]

- Integration challenges: incorporating genetic data into everyday clinical workflows remains logistically difficult.[4,7]

CONCLUSION

A thorough understanding of disease pathways, genomic interactions, and novel biomarkers — established before overt disease occurrence — can meaningfully aid in disease prevention and, to some extent, guide treatment planning.[3] Prompt diagnosis allows patients to benefit from targeted therapies suited to their individual risk profile.[4] However, personalized periodontics remains an evolving field, and further longitudinal studies are needed to validate its diagnostic and therapeutic tools and to better understand its role within routine periodontal practice.[3,4] Despite its promise, personalized periodontics faces barriers including the need for specialized clinician training, the multifactorial complexity of periodontal disease, limited long-term clinical validation, high cost, and workflow-integration challenges. Continued longitudinal research and biomarker validation will be essential to translate precision periodontics from concept to routine clinical practice[3,4]

REFERENCES

1. Loos BG, Van Dyke TE. The role of inflammation and genetics in periodontal disease. Periodontology 2000. 2020 Jun;83(1):26-39.
2. Bartold PM. Lifestyle and periodontitis: the emergence of personalized periodontics. Periodontol 2000. 2018 Oct;78(1):7-11.
3. Bartold PM, Ivanovski S. P4 medicine as a model for precision periodontal care. Clin Oral Investig. 2022 Sep;26(9):5517-5533.
4. Rakic M, Pejcic N, Perunovic N, Vojvodic D. A roadmap towards precision periodontics. Medicina (Kaunas). 2021 Mar 3;57(3):233.
5. Kikuchi T, Hayashi J, Mitani A. Next-Generation Examination, Diagnosis, and Personalized Medicine in Periodontal Disease. J Pers Med. 2022 Oct 20;12(10):1743.
6. Knight ET, Murray Thomson W. A public health perspective on personalized periodontics. Periodontol 2000. 2018 Oct;78(1):195-200.
7. Kornman KS, Giannobile WV, Duff GW, Van Dyke TE. Quo vadis: what is the future of periodontics? How will we get there? Periodontol 2000. 2017 Oct;75(1):353-371.
8. Sheilesh D. Risk factors for periodontitis. J Int Acad Periodontol. 2005 Jan;7(1):3-7.
9. Heaton B, Dietrich T. Causal theory and the etiology of periodontal diseases. Periodontol 2000. 2012 Feb;58(1):26-36.
10. Peeran SW, Ramalingam K, Ahmed FMA. Periodontal risk assessment. In: Essentials of Periodontics and Oral Implantology. 2021.
11. Christodoulides N, Floriano PN, Miller CS, et al. Lab-on-a-chip methods for point-of-care measurements of salivary biomarkers of periodontitis. Ann N Y Acad Sci. 2007;1098:411-428.
12. Giannobile WV, Beikler T, Kinney JS, Ramseier CA, Morelli T, Wong DT. Saliva as a diagnostic tool for periodontal disease: current state and future directions. Periodontol 2000. 2008;50:52-64.
13. Cafiero C, Matarasso S. Predictive, preventive, personalised and participatory periodontology: 'the 5Ps age' has already started. EPMA J. 2013 Jun 14;4(1):16.
14. Gundelly M, Pusuluri SV, Koduganti RR, Ambati M, Chiluveru S, Chandaka M. Precision medicine in periodontics: a literature review. Cureus. 2024 Sep 8;16(9):e68952.
15. Dong A, Proctor G, Zaric S. Diagnostic accuracy of microbiome-derived biomarkers in periodontitis: systematic review and meta-analysis. J Periodontal Res. 2025 Jan 13. doi: 10.1111/jre.13377.

16. Bostanci N. Precision periodontal care: from omics discoveries to chairside diagnostics. Clin Oral Investig. 2023 Mar;27(3):971-978.
17. Radu CM, Radu CC, Zaha DC. Salivary and microbiome biomarkers in periodontitis: advances in diagnosis and therapy—a narrative review. Medicina (Kaunas). 2025 Oct 11;61(10):1818.
18. Hajishengallis G, Darveau RP, Curtis MA. The keystone-pathogen hypothesis. Nat Rev Microbiol. 2012 Oct;10(10):717-725.
19. Lamont RJ, Hajishengallis G. Polymicrobial synergy and dysbiosis in inflammatory disease. Trends Mol Med. 2015 Mar;21(3):172-183.
20. Apatzidou DA. The role of cigarette smoking in periodontal disease and treatment outcomes of dental implant therapy. Periodontol 2000. 2022 Oct;90(1):45-61.
21. Alhajj M, Halboub E, et al. Periodontal disease and smoking: systematic review. J Pharm Bioallied Sci. 2023 Jul;15(Suppl 1):S85-S89.
22. Stöhr J, Barbaresko J, Neuenschwander M, Schlesinger S. Bidirectional association between periodontal disease and diabetes mellitus: a systematic review and meta-analysis of cohort studies. Sci Rep. 2021 Jul 1;11(1):13686.
23. Genco RJ, Sanz M. Clinical and public health implications of periodontal and systemic diseases: an overview. Periodontol 2000. 2020 Jun;83(1):7-13.
24. Tran AH, Zaidi AH, Bolger AF, et al; American Heart Association Cardiovascular Disease Prevention Committee. Periodontal disease and atherosclerotic cardiovascular disease: a scientific statement from the American Heart Association. Circulation. 2026 Feb 10;153(6):e73-e88.
25. Sanz M, Marco Del Castillo A, Jepsen S, et al. Periodontitis and cardiovascular diseases: consensus report. J Clin Periodontol. 2020 Mar;47(3):268-288.
26. Bui FQ, Almeida-da-Silva CLC, Huynh B, et al. Association between periodontal pathogens and systemic disease. Biomed J. 2019 Feb;42(1):27-35.
27. Genco RJ, Graziani F, Hasturk H. Effects of periodontal disease on glycemic control, complications, and incidence of diabetes mellitus. Periodontol 2000. 2020 Oct;83(1):59-65.
28. de Molon RS, Rossa C Jr, Thurlings RM, Cirelli JA, Koenders MI. Linking periodontal disease and rheumatoid arthritis: a systematic review. Int J Mol Sci. 2019 Sep;20(18):4541.
29. Wegner N, Wait R, Sroka A, et al. Peptidylarginine deiminase from Porphyromonas gingivalis citrullinates human fibrinogen and α-enolase: implications for autoimmunity in rheumatoid arthritis. Arthritis Rheum. 2010 Sep;62(9):2662-2672.
30. Martinez-Herrera M, Silvestre-Rangil J, Silvestre FJ. Association between obesity and periodontal disease: a systematic review of epidemiological studies and controlled clinical trials. Med Oral Patol Oral Cir Bucal. 2017 Nov 1;22(6):e708-e715.
31. Suvan J, D'Aiuto F, Moles DR, Petrie A, Donos N. Association between overweight/obesity and periodontitis in adults: a systematic review. Obes Rev. 2011 May;12(5):e381-e404.
32. Decker AM, Kapila YL, Wang HL. The psychobiological links between chronic stress and periodontitis. Periodontol 2000. 2021 Jun;86(1):101-115.
33. Peruzzo DC, Benatti BB, Ambrosano GM, et al. A systematic review of stress and psychological factors as possible risk factors for periodontal disease. J Periodontol. 2007 Aug;78(8):1491-1504.
34. Larvin H, Kang J, Aggarwal VR, Pavitt S, Wu J. Periodontitis and risk of immune-mediated systemic conditions: a systematic review and meta-analysis. Community Dent Oral Epidemiol. 2023 Aug;51(4):705-716.
35. Barros SP, Fahimipour F, Tarran R, Kim S, Scarel-Caminaga RM, Justice A, North KE. Epigenetic modifications and periodontal disease. Curr Oral Health Rep. 2016;3(4):337-345.
36. Baptista NB, Portinho D, Casarin RC, Vale HF, Casati MZ, De Souza AP, Andia DC. DNA methylation levels of SOCS1 and LINE-

1 in oral epithelial cells from aggressive periodontitis patients. Arch Oral Biol. 2014 Jul;59(7):670-678.
37. Kornman KS, Crane A, Wang HY, et al. The interleukin-1 genotype as a severity factor in adult periodontal disease. J Clin Periodontol. 1997 Jan;24(1):72-77.
38. Lin PL, Huang PW, Huang PY, Hsu HC. Alveolar bone-loss area localization in periodontitis radiographs based on threshold segmentation with a hybrid feature fused of intensity and the H-value of fractional Brownian motion model. Comput Methods Programs Biomed. 2015 Oct;121(3):117-126.

HOW TO CITE: Nowfiya M, Deepa S, Jenifer Cynthia R A, Shanmuga priya R, Arunmozhi U, Personalized Periodontics: Review, Int. J. of Pharm. Sci., 2026, Vol 4, Issue 9, 2206-2215. https://doi.org/10.5281/zenodo.22842324