VIEW POINT

Precision Periodontology: Integrating Microbiome, Biomarkers, and Patient-Specific Risk Assessment

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ABSTRACT

Periodontitis is a prevalent, polyetiological inflammatory disease in which subgingival dysbiotic communities interact with a vulnerable host. The current stage and grade classification only reflects severity and progression risk, but not the differences in responses to the same treatment for patients with similar presentations. According to this perspective, precision periodontology would require three complementary layers of data, including microbiome profiling, patient-specific risk factors, and host-derived biomarkers. Using an appraisal of the literature based on a personal narrative, it argues that any single layer is an incomplete predictor and that a more convincing pathway to individualized risk stratification, treatment choice, and maintenance planning will be through integrated and longitudinally updated models. This viewpoint suggests a conceptual outline and discusses challenges to implementation: standardization of the assays, validation in a variety of populations, cost, and data governance. Moving from population-average care to individually tailored care will require prospective trials that test biomarker- and microbiome-guided decisions against standard care.

Keywords: Periodontitis; Precision Medicine; Risk Assessment; Biomarkers; Gingival Crevicular Fluid; Microbiota.

VIEW POINT

Periodontitis is a chronic inflammatory disease which affects the tooth supporting apparatus, and is a major cause of loss of teeth in adults. The 2017 World Workshop classification replaced the previous distinction between chronic and aggressive periodontitis with a unified classification based on disease stage, which reflects severity and management complexity, and grade, which estimates progression rate and incorporates risk factors. However, these categories primarily describe clinical disease characteristics and do not fully capture individual biological variation [1]. They use radiographic bone loss, clinical attachment loss, and a couple of modifiers like glycaemic control, smoking, etc. These do not explain in detail the biological mechanisms underlying disease in a particular patient.

There has also been a change in the pathogenic model. Periodontitis is not considered to be a disease caused by a few "red complex" organisms [2]. The same bacterial attack can cause only slight change in one individual, and attachment loss in another and this limit of change, the loss of attachment, is an exaggerated inflammatory response that is poorly resolving in the susceptible person [3]. The clinical issue that precision medicine is intended to solve is variability. Precision periodontology can be said to be the ability to predict disease onset and progression, and select a successful intervention for that individual and patient, based on individual level microbial, host, and clinical data. This perspective poses three questions. What is provided by each data layer? What are some ways they might be integrated? What is the difference between concept and practice ? This is a narrative viewpoint. Literature related to the sequencing of the periodontal microbiome, host-response biomarkers in gingival crevicular fluid (GCF), saliva, serum, and multivariable risk assessment tools was reviewed. Sources were identified by targeted searching of PubMed, Google Scholar, and Embase using MeSH and Emtree terms (periodontitis, microbiota, biomarkers, gingival crevicular fluid, precision/personalized medicine, risk assessment), supplemented by consensus reports and guidelines. Longitudinal and interventional studies were preferred to cross-sectional association studies. There was no formal quality scoring or meta-analysis, and conclusions were interpreted as an expert synthesis. Findings were grouped into three areas (microbiome, biomarkers, patient-specific risk) and incorporated into a conceptual framework.

Microbiome Profiling: Studies based on sequencing show that in periodontitis, the oral microbiome shifts to become more diverse, with higher levels of pathogens such as *Porphyromonas gingivalis*, *Tannerella forsythia*, *Treponema denticola*, *Filifactor alocis*, and various Gram-positive anaerobes, and a loss of beneficial health-associated bacteria [4]. Overall features of the community, like total bacterial load, inflammatory burden, and functional genes, seem to be more informative than any single species. Metagenomic and meta-transcriptomic data indicate that the functional activity of the community is just as important as which species are present. Nevertheless, the data from microbiomes have their limitations. Cross-sectional profiles differentiate health from advanced disease well, but do not predict future progression very well. Results are dependent on sampling site, DNA extraction methods, sequencing platform, bioinformatic pipeline, and do not have a consensus reference range [5]. Furthermore, there is uncertainty about the ability of microbial signatures to be used because they will also change after therapy. They may be most useful in monitoring the response and for the detection of persistent dysbiosis following debridement.

Host-Derived Biomarkers: Host-response markers in blood or saliva are indicators of inflammation, tissue breakdown, and bone turnover. Innate immune recognition also contributes to host–microbe interactions through pattern recognition receptors (PRRs), which recognize microbial components and activate downstream inflammatory signaling. Toll-like receptor-mediated pathways and antigen-presentation mechanisms are important components of this response, although their specific roles vary according to the microbial environment and disease context [6]. Active MMP-8 point-of-care assays have been evaluated in the chairside setting, and panels that include combinations of assays perform better than single assays [7]. Several challenges persist. There are numerous markers that are elevated in any inflammatory condition, thus not specific to periodontitis. The concentration is site-, time-of-day-, sampling method-, and gingival inflammation-dependent. The conclusions

about prognosis from most validation studies are based on cross-sectional or short-term designs. When used for the diagnosis of active inflammation and response to treatment, biomarkers hold greater promise of usefulness than for the prediction of long-term attachment loss.

Patient-Specific Risk Assessment: Smoking, plaques, glycaemic control, age, socioeconomic and behavioural context were determined as likely modifiable or non-modifiable risk factors. Multifactorial tools, including the periodontal risk assessment model, which uses clinical and systemic factors to create a risk category, have been used to guide recall intervals and investigated for several years, but have small effect sizes and lack clinical utility. Polygenic and epigenetic techniques are still nascent [8].

Integration: The results of a single layer cannot be used to predict results at the individual level. The three domains are focused on different questions. A challenge is defined by the microbiome, a host response is defined by the biomarkers, and the context, compliance or response to treatment is defined by clinical and systemic factors [9]. Multi-layer models generally perform well in discriminating and are more likely to include the inclusion of inflammatory markers or biomarker panels in addition to the microbial load; however, most are small, single-center, and do not have external validation [10]. **Table I** compared the layers with schematic approach.

Table I: Comparison of Data Layers in Precision Periodontology

Data layer	Typical sample	Main strength	Main limitation	Most plausible use
Microbiome [4,5]	Subgingival plaque, saliva	Describes the microbial challenge and functional potential	No consensus thresholds; method-dependent; weak prognostic evidence	Monitoring response; identifying persistent dysbiosis
Host biomarkers [6,7]	GCF, saliva, serum	Reflects inflammation and tissue breakdown; chairside tests exist	Limited specificity; sampling variability; few prognostic studies	Detecting active disease; tracking treatment response
Patient-specific risk [8]	Clinical examination, medical history, genetics	Inexpensive; some factors modifiable	Genetic markers weak; tools need broader validation	Baseline risk; recall planning; targeting modifiable factors
Integrated model [9,10]	Combination of the above, repeated over time	Captures complementary information; adapts to change	Largely unvalidated; complex and costly	Individualized care pathways (future)

DISCUSSION

The central argument of this viewpoint is that, despite the development of the concept of the "average patient," periodontal care continues to be organized around that concept. Stage and grade provide a general reference, but individuals in each category have varying levels of systemic context, intensity of host response, and microbial ecology. The idea of precision periodontology is to take advantage of that variability to make decisions. There are three potential applications. Patterns of biomarkers along with clinical, systemic data may help determine whether a patient with stage II disease may do well with an aggressive approach, or whether the patient's inflammation is controlled with stable pockets. Intensive prevention might be warranted if there is early identification [11, 12]. This could also avoid the need for patients to undergo unnecessary intervention if they were accurately classified as low risk. Systemic antimicrobials as an adjunct are currently indicated only for selected cases and are not recommended as routine therapy, in part due to concerns regarding antimicrobial resistance [13]. Microbial and host profiling may help identify the small subset of patients most likely to benefit, for example, those with persistent dysbiosis following mechanical debridement, and could provide a rationale for host-modulation approaches. At present, this is a hypothesis, because trials that randomize patients to profile-guided versus standard therapy are scarce. The recall interval usually determined by trial and error. Dynamic intervals may be possible, using longitudinal markers of subclinical inflammation, alongside clinical findings [14].

There are four hindrances that are significant. First, there is the issue of standardization; that is, sampling methods, storage, assay and analysis vary between studies, and the results obtained cannot be compared or pooled if they are not standardized. Secondly, validation: many reported signatures come from small, cross-sectional, often single-population cohorts. Prognostic models should be validated in future in a variety of different populations, and not only with respect to discrimination, but calibration should be considered as well [15]. Third, clinical utility and cost: if a test does not alter management and/or outcomes at reasonable cost, it will not be justified. There is still no evidence of improved patient-centered outcomes, such as maintaining teeth. Fourth, data governance and equity: genetic and microbial data are sensitive, and models built on small populations can have poor performance in other populations, thereby perpetuating existing inequalities in oral health [16]. The fact that periodontitis is already over-represented in disadvantaged groups means that any precision approach needs to be evaluated for its potential to enhance access for these groups, but not to increase needless layers of cost for those who are well off [17].

Future research should prioritize large, prospective, multicenter cohorts employing standardized sampling protocols and repeated longitudinal measures to capture disease dynamics. In parallel, there is a need for pre-registered biomarker and microbiome studies with external validation to improve reproducibility and generalizability. To translate these insights into practice, pragmatic randomized trials comparing stratified, profile-guided care with standard care are essential. Finally, transparent reporting of prediction models and a greater emphasis on health-economic outcomes will be critical for assessing clinical utility and informing implementation.

CONCLUSION

Periodontitis arises from the interaction of a dysbiotic microbial community, a variable host response, and patient-level risk factors, so a strategy relying on any one of these in isolation is unlikely to deliver true precision. Microbiome profiles characterize the microbial environment, host-derived biomarkers provide insight into inflammatory activity, and clinical and systemic factors establish the broader patient context. Their integration, supported by longitudinal assessment, may provide a foundation for more individualized risk assessment and treatment planning. Today there is enough evidence for these tools to be mostly used for monitoring and research purposes rather than for regular decision-making. There is a need to standardize the approaches, coupled with strong prospective validation, trials that produce better outcomes in patients, and a clear focus on the cost and equity of precision periodontology. The basics of practice with careful clinical staging and grading, good control of risk factors, and careful supportive care will continue to serve as the cornerstones of treatment for the time being.

References

1. Ravidà A, Travan S, Saleh MH, Greenwell H, Papapanou PN, Sanz M, et al. Agreement among international periodontal experts using the 2017 World Workshop classification of periodontitis. J Periodontol. 2021;92(12):1675-86. https://doi.org/10.1002/JPER.20-0825
2. Kwon T, Lamster IB, Levin L. Current concepts in the management of periodontitis. Int Dent J. 2021;71(6):462-76. https://doi.org/10.1111/idj.12630
3. Abdulkareem AA, Al-Taweel FB, Al-Sharqi AJ, Gul SS, Sha A, Chapple IL. Current concepts in the pathogenesis of periodontitis: from symbiosis to dysbiosis. J Oral Microbiol. 2023;15(1):2197779. https://doi.org/10.1080/20002297.2023.2197779
4. Nearing JT, DeClercq V, Van Limbergen J, Langille MG. Assessing the variation within the oral microbiome of healthy adults. mSphere. 2020;5(5):e00451-20. https://doi.org/10.1128/mSphere.00451-20
5. Hashimoto K, Shimizu D, Ueda S, Miyabe S, Oh-Iwa I, Nagao T, et al. Feasibility of oral microbiome profiles associated with oral squamous cell carcinoma. J Oral Microbiol. 2022;14(1):2105574. https://doi.org/10.1080/20002297.2022.2105574
6. Ahmed E, Azhar F, Akhter M. Host Immune Recognition, Pattern Recognition Receptor Signaling, and Antigen Presentation Pathways Initiating Chronic Host–Pathogen Interactions. J Biomol Pathog Ther. 2026;2(2):102–110. https://doi.org/10.68041/jbpt.v2i2/03
7. Brandt E, Keskin M, Räisänen IT, Tervahartiala T, Mäkitie A, Harmankaya İ, et al. Induction of collagenolytic MMP-8 and -9 tissue destruction cascade in mouth by head and neck cancer radiotherapy: a cohort study. Biomedicines. 2023;12(1):27. https://doi.org/10.3390/biomedicines12010027
8. Asimakopoulou K, Nolan M, McCarthy C, Newton JT. The effect of risk communication on periodontal treatment outcomes: a randomized controlled trial. J Periodontol. 2019;90(9):948-56. https://doi.org/10.1002/JPER.18-0385
9. Li Q, Wu R, Du C, Miao Q. Impact of continuity of care under the model of healthcare integration on treatment outcomes and prognostic recovery of orthodontic patients. Front Med (Lausanne). 2025;12:1632725. https://doi.org/10.3389/fmed.2025.1632725
10. Song J, Zhao H, Pan C, Li C, Liu J, Pan Y. Risk factors of chronic periodontitis on healing response: a multilevel modelling analysis. BMC Med Inform Decis Mak. 2017;17(1):135. https://doi.org/10.1186/s12911-017-0533-2
11. Bostanci N, Manoil D, Van Holm W, Belibasakis GN, Teughels W. Microbial markers for diagnosis and risk assessment for periodontal diseases: A systematic literature search and narrative synthesis. J Clin Periodontol. 2025;52(Suppl 29):125–154. https://doi.org/10.1111/jcpe.14183
12. Saleh MH, Dukka H, Troiano G, Ravidà A, Qazi M, Wang HL, et al. Long term comparison of the prognostic performance of PerioRisk, periodontal risk assessment, periodontal risk calculator, and staging and grading systems. J Periodontol. 2022;93(1):57-68. https://doi.org/10.1002/JPER.20-0662
13. Yuan X, Zhou F, Wang H, Xu X, Xu S, Zhang C, et al. Systemic antibiotics increase microbiota pathogenicity and oral bone loss. Int J Oral Sci. 2023;15(1):4. https://doi.org/10.1038/s41368-022-00212-1
14. Hammami C, Nasri W. Antibiotics in the treatment of periodontitis: a systematic review of the literature. Int J Dent. 2021;2021(1):6846074. https://doi.org/10.1155/2021/6846074
15. Weinert L, El Sayed N, Wensing M, Dannewitz B, Listl S. Exploring barriers to periodontal treatment adherence in patients with periodontitis to inform development of a digital companion: a qualitative study. J Clin Periodontol. 2026;53(1):58-71. https://doi.org/10.1111/jcpe.70044
16. Mills A, Levin L. Inequities in periodontal disease prevalence, prevention, and management. Quintessence Int. 2022;53(2):122. https://doi.org/10.3290/j.qi.b1763677
17. Jönsson B, Abrahamsson KH. Overcoming behavioral obstacles to prevent periodontal disease: behavioral change techniques and self-performed periodontal infection control. Periodontol 2000. 2020;84(1):134-44. https://doi.org/10.1111/prd.12334

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