

# Periodontal Care Through Precision Biology And Clinical Judgement

Dr. Poonam kedar<sup>1</sup>, Dr. Pradumna Niturkar<sup>2</sup>, Dr. Raghvendra Metri,<sup>3</sup> Dr. Gauri Ugale<sup>4</sup>, Dr. Mithila Kakade<sup>5</sup>, Dr. Trupti Giri<sup>6</sup>

1,3,4,5,6 Department of Periodontology, MIDS Dental College, Latur.

2 Postgraduate student, Department of Periodontology, Tatyasaheb Kore Dental College & Research Centre, New Pargaon, Kolhapur.

**Abstract:** Periodontitis is not a uniform disease entity but a spectrum of biologically and clinically distinct conditions that share a common phenotype of inflammatory tissue destruction. While conventional periodontal therapy has achieved substantial success at the population level, it inadequately accounts for the marked inter-individual variability observed in disease susceptibility, progression, and response to treatment. Personalized periodontics applies the principles of precision medicine to periodontal care, integrating genetic and epigenetic predisposition, host immune–inflammatory behavior, microbial ecology, systemic modifiers, and behavioral factors to guide clinical decision-making. This narrative review critically examines the scientific foundations and translational relevance of personalized periodontics, synthesizing evidence from molecular biology, microbiome research, biomarker discovery, and digital diagnostics.

**Keywords:** Personalized periodontics; precision dentistry; periodontal medicine; host response; microbiome; biomarkers

**Corresponding Author:** Dr. Pradumna Niturkar, Postgraduate student, Department of Periodontology, Tatyasaheb Kore Dental College & Research Centre, New Pargaon, Kolhapur.

Email: [pradumna.niturkar@gmail.com](mailto:pradumna.niturkar@gmail.com)

## 1. Introduction

Despite decades of refinement in preventive strategies and therapeutic protocols, periodontitis remains highly prevalent and continues to exhibit unpredictable clinical behavior. One of the enduring paradoxes in periodontology is that patients exposed to comparable microbial challenges often demonstrate strikingly different disease trajectories. Some individuals maintain periodontal stability despite heavy plaque accumulation, whereas others experience rapid attachment loss under apparently

modest local challenges. This variability cannot be adequately explained by biofilm quantity alone.

The contemporary understanding of periodontitis recognizes it as a host-mediated, multifactorial inflammatory disease in which microbial dysbiosis acts as a necessary but insufficient trigger. Genetic susceptibility, immune regulation, systemic health, and environmental exposures collectively determine disease expression. Traditional population-based treatment algorithms, although evidence-based, inherently assume biological

homogeneity and therefore fail to address individual risk and response variability. Personalized periodontics emerges from this clinical and biological reality, proposing a shift from standardized care pathways to biologically informed, patient-specific strategies.

## 2. From Population-Based Therapy to Personalized Periodontics

Conventional periodontal care is largely reactive, initiated after clinical signs of tissue destruction become evident. Personalized periodontics, in contrast, prioritizes anticipation and interception of disease pathways. Conceptually aligned with precision medicine, it emphasizes prediction of risk, prevention of breakdown, personalization of therapy, and active patient participation.

This transition does not imply abandonment of established therapeutic principles such as biofilm control or surgical reconstruction. Rather, it reframes these interventions within a broader biological context, allowing clinicians to tailor timing, intensity, and adjunctive measures according to individual disease drivers.

## 3. Biological Basis for Individualized Periodontal Risk

### 3.1 Genetic Predisposition

Epidemiologic and twin studies consistently demonstrate a substantial heritable component in periodontitis, with estimates ranging between 30% and 50%. Polymorphisms in genes regulating innate immunity, cytokine signaling, and bone metabolism influence how individuals respond to microbial challenge. While single genetic variants rarely dictate

disease outcome, their cumulative effect contributes meaningfully to overall risk and may partially explain why certain patients exhibit severe disease at a young age.

### 3.2 Epigenetic Influences

Epigenetic mechanisms provide a biological link between environmental exposure and sustained inflammatory behavior. Smoking, hyperglycemia, and psychosocial stress can induce epigenetic modifications that amplify pro-inflammatory gene expression, even after the initiating stimulus is reduced. These changes may account for disease persistence and incomplete resolution following otherwise adequate therapy, underscoring the need for individualized risk modification strategies.

### 3.3 Immune–Inflammatory Phenotypes

Clinical destruction in periodontitis is driven less by bacterial virulence per se and more by the magnitude and regulation of the host inflammatory response. Hyper-inflammatory phenotypes generate excessive cytokines, proteases, and osteoclast-activating signals, leading to disproportionate tissue breakdown. Recognizing such phenotypes has direct therapeutic implications, particularly when considering adjunctive host-modulatory approaches.

## 4. Microbiome Heterogeneity and Clinical Relevance

Advances in sequencing technologies have transformed the understanding of periodontal microbiology. Disease-associated biofilms are now recognized as functionally dysbiotic ecosystems rather than collections of discrete pathogens. Importantly, the composition and metabolic activity of these communities

vary substantially between individuals and over time.

From a personalized care perspective, this variability explains inconsistent responses to uniform antimicrobial strategies.

Targeted modulation of microbial communities—guided by risk, disease activity, and systemic context—offers a more rational approach than indiscriminate antibiotic use.

## 5. Biomarkers and Omics: Translational Opportunities and Limitations

### 5.1 Molecular and Protein Biomarkers

Salivary and gingival crevicular fluid biomarkers, including active matrix metalloproteinase-8, interleukin-1 $\beta$ , and tumor necrosis factor- $\alpha$ , provide insight into ongoing tissue breakdown and inflammatory burden. Unlike traditional clinical parameters, these biomarkers reflect current disease activity rather than historical damage, making them attractive tools for monitoring and risk stratification.

### 5.2 Multi-Omics Integration

Genomic, transcriptomic, proteomic, and metabolomic datasets offer unprecedented resolution of disease biology. However, their clinical value lies not in isolated measurements but in integrated interpretation. Systems-level approaches that contextualize omics data within clinical findings represent the most realistic pathway toward meaningful personalization.

## 6. Digital Dentistry and Artificial Intelligence

Artificial intelligence has demonstrated considerable potential in periodontal diagnostics, particularly in radiographic

bone loss assessment and risk prediction. When used appropriately, AI functions as a clinical decision-support tool rather than a substitute for professional judgment. Its greatest value in personalized periodontics lies in synthesizing complex datasets and highlighting patterns that may not be immediately apparent to the clinician.

## 7. Personalized Therapeutic Decision-Making

### 7.1 Risk-Based Prevention and Maintenance

Individualized recall intervals and preventive strategies are central to personalized care. Patients with stable biological profiles may require less intensive maintenance, whereas high-risk individuals benefit from closer monitoring and earlier intervention.

### 7.2 Precision Use of Adjunctive Therapies

Adjunctive antimicrobials and host-modulatory agents should be prescribed selectively, guided by inflammatory burden, systemic modifiers, and previous treatment response. This approach maximizes benefit while minimizing unnecessary exposure and adverse effects.

### 7.3 Surgical Personalization

Decisions regarding regenerative versus resective surgery must consider not only defect morphology but also patient-specific healing potential, systemic health, and behavioral factors. Personalized periodontics encourages surgical restraint when biological conditions are unfavorable and supports regeneration when host factors are conducive.

## 8. Periodontal Medicine and Systemic Context

The bidirectional associations between periodontitis and systemic diseases such as diabetes and cardiovascular disease further reinforce the need for personalized care. Shared inflammatory pathways and common risk modifiers provide opportunities for integrated management and interprofessional collaboration.

## 9. Practical and Ethical Considerations

While the promise of personalized periodontics is substantial, its implementation is constrained by cost, accessibility, data interpretation challenges, and ethical concerns related to genetic information. Pragmatic adoption requires careful validation, clinician education, and development of scalable models that complement, rather than complicate, routine care.

## 10. Future Perspectives

Future progress in personalized periodontics will depend on longitudinal studies linking biological profiles to long-term clinical outcomes, validation of chairside diagnostic tools, and development of evidence-based decision frameworks. Importantly, success will be measured not by technological sophistication alone, but by demonstrable improvements in patient-centered outcomes.

## 11. Conclusion

Personalized periodontics represents an evolutionary, rather than revolutionary, step in periodontal care. By integrating biological insight with clinical expertise, it enables a more nuanced understanding of disease behavior and therapeutic response.

As evidence continues to mature, personalized approaches have the potential to enhance predictability, efficiency, and long-term stability in periodontal practice.

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