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Review Article

PRECISION PERIODONTICS: A CRITICAL REVIEW OF PERSONALIZED APPROACHES

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ABSTRACT

Periodontal care has traditionally followed a one-size-fits-all model the same diagnostic criteria, the same treatment steps, the same antibiotics, applied more or less uniformly regardless of who the patient is. Personalized periodontics pushes back against that. Borrowing from the wider movement toward personalized medicine, it argues that effective periodontal care must account for who the patient actually is their genetics, their oral microbiome, their lifestyle, their systemic health. The goal is better outcomes with less overtreatment. The problem with conventional approaches is not that they are wrong, but that periodontitis itself is far more variable than a single protocol can capture. Microbial imbalances differ between patients, risk factors like obesity, chronic stress, and poor nutrition compound the disease in different ways, and older adults bring their own set of clinical challenges. Routine antibiotic prescriptions, handed out with little regard for individual microbial profiles, are a particular point of concern. To examine how well the science actually supports this shift, relevant literature published through 2024 was gathered from PubMed-Medline, Web of Science, Scopus, and Google Scholar. This review works through that evidence and asks a direct question is personalized periodontics a genuinely evidence-based advancement, or is it an appealing idea still waiting for the science to catch up?

Keywords: Precision periodontics, Genetic profiling, Biomarkers, Targeted therapy, Customized treatment.

INTRODUCTION

"Personalized medicine" is not a new idea dressed in modern clothing. The concept has been quietly reshaping clinical practice for years, under different names precision medicine, individualized care, stratified treatment all circling the same uncomfortable truth: give the same treatment to ten patients, and you will not get ten identical outcomes. The old assumption that a single protocol could work for everyone has largely been abandoned. In its place is a more honest acknowledgment that effective care depends on understanding the individual, not just the disease. Personalized periodontics follows this same logic. Rather than slotting every patient into the same treatment pathway, it sorts them into meaningful groups and builds clinical decisions around what is actually known about each person their phenotype, their lifestyle, their genetic makeup, their microbial environment, their immune profile. The aim is practical: fewer side effects, better outcomes, less wasted treatment. Whether that promise holds up under scrutiny is another question. This essay works through the evidence, looks at where personalized periodontics is already being applied clinically, and considers where the field might realistically go from here.

The Origin of Personalized Periodontics

Dr. Leroy Hood proposed the term 'Personalised periodontics', which is a part of P4 medicine. It is a radical shift to proactive healthcare from reactive methods, which focuses more on the patient's overall well-being rather than being solely occupied with fighting against ailments and their associated symptoms. This approach is built on following four principles (Bartold & Ivanovski, 2022). P4 medicine integrates holistic and collaborative practices, combining precision medicine, novel diagnostics, risk measurements, patient involvement, and technological advancements to enhance healthcare delivery. P4 Medicine

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has evolved into a multilevel healthcare model for managing chronic diseases. Given that periodontitis is among the most prevalent chronic conditions affecting humans, the term "P4 periodontics" has been introduced. This proposed model aligns closely with the 2018 Classification of Periodontal Diseases (Bartold & Ivanovski, 2022) and logically extends this classification into treatment paradigms (Figure 1).

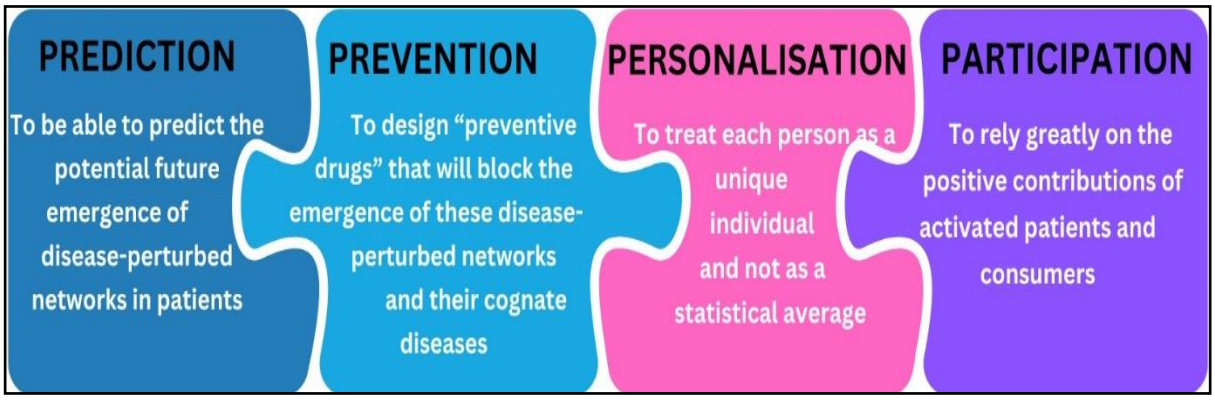

Figure 1. Principle of P4 medicine.

The Need of Personalised Periodontics

The staging and grading classification of periodontitis considers the varying clinical signs, symptoms, risk factors, but these aspects are not addressed in traditional periodontal therapy, which only targets 10-15 out of 700 species linked to the onset and progression of periodontal dysbiosis. This reactive approach, focuses on plaque control, pocket depth reduction, and gingival bleeding elimination, overlooking individual patient needs and the complexities of human biology, allostasis and genetic factors (Tonetti *et al.,* 2018). Hence Personalised periodontics is necessary to overcome all the limitations in today's world (Figure 2).

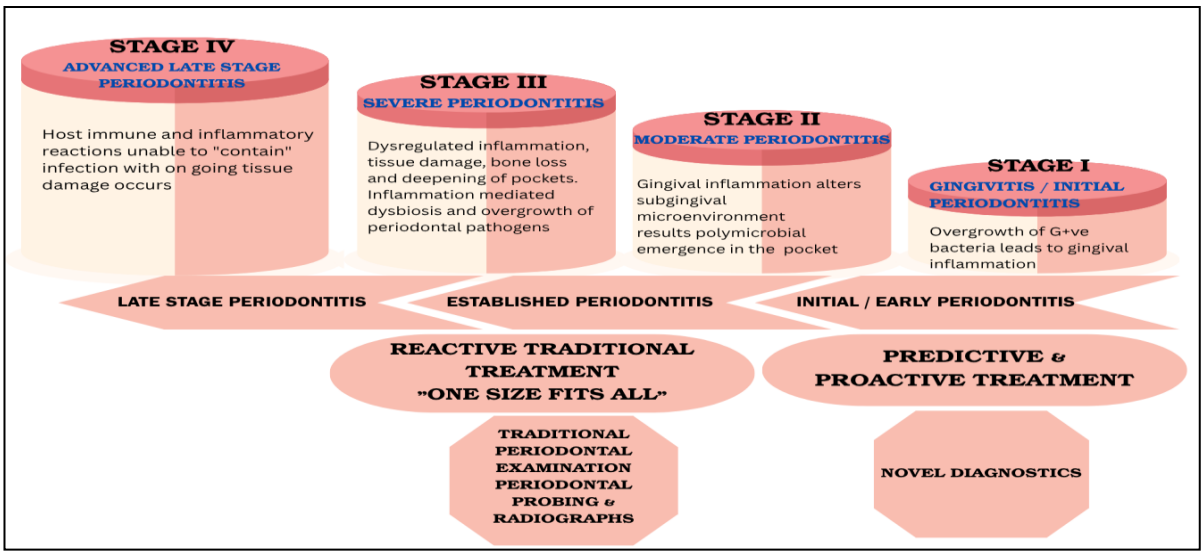

Figure 2. Reactive to a proactive clinical approach for patient care.

Individual Risk Factor Variability

Periodontitis is a multifaceted disease influenced by diverse risk factors but assuming that all adults have the same risk level for periodontal disease leads to uniform preventive and interceptive care, resulting in inadequate treatment for high-risk individuals and over-treatment for low-risk individuals. Key candidates include tobacco users, particularly moderate to heavy smokers who are often resistant to quitting, patients with aggressive periodontitis, medical comorbidities. Additionally, patients with a family history or genetic predisposition to periodontal disease, and those who do not respond to conventional periodontal therapy, are ideal candidates for personalised approaches to improve treatment effectiveness and long-term oral health. Research from the University of Michigan and the University of Gothenburg found that periodontitis isn't solely due to bacterial build-up. Most people develop only

mild to moderate periodontitis without routine oral hygiene, while some develop severe periodontitis despite minimal calculus (Kornman, 2018). Rarely treating periodontitis is straightforward. It also claims attention to microbial imbalances, systemic conditions, behavioural habits, local risk factors and not isolation, but together. Oral cavity should be kept free of calculus, but so is understanding what is driving disease in that particular patient, and treatment plan is builded around that.

Pitfalls of Uniform Antibiotic Prescriptions

Prescribing the same antibiotics to every periodontitis patient without considering particular micro-organisms can cause antibiotic resistance, disruption of useful microbiota, allergic effects and insufficient targeted therapy for complex infections. (Ximenez fyvie *et al*, 2000) highlighted the difference in subgingival microbiota in periodontitis patients, it shows the need for tailored antibiotic therapy. Kilian *et al* (2016) wanted that random antibiotics use can disrupt useful oral and the gut bacteria. The risk of adverse reaction is more due to varying patient sensitivities it makes the blanket prescription risky.

Lifestyle and epigenetic modifications

Lifestyle modifications like poor diet, obesity, environmental toxins, exposure to metal and chemical, tobacco smoke, polluted water, infections cause epigenetic modification such as DNA methylation histone, modification, non-coding RNAs and cheomatin restricting, which considerably give changes in periodontitis (Bauos *et al*, 2018). These problems are frequently over looked in the clinical practice and just address symptoms with medication without addressing the underlying lifestyle cause is not effective. Exaggerated inflammatory response is triggered by obesity, it worsens periodontal inflammation and delays healing (Suvan *et al*, 2018) and it induces insulin resistance and hyper glycemia, it develops the growth of periodontal pathogens and aggravates periodontitis. It emphasizes the need for personalized periodontitis treatment in the obese individuals and also the importance of diet nutrition and micronutrients (Vitamin A, B-complex, C, D, E, Coenzyme Q10, Minerals) in periodontal health has got attention, poor nutrition, decreases wound healing and tissue repair when the micronutrients are associated with periodontitis prevalence, their effect on therapy outcomes is under researched (Dommisch *et al*, 2018). This underscores the requirement for personalized periodontics to address the micronutrient deficiencies, that can improve treatment effectiveness.

Stress and allostatic load

Sabbath *et al.* (2018) spotlighted the impact of stress in periodontal tissues that leads to systemic effect known as allostatic load and demonstrated how the composite biological indicators related with the allostatic load may serve as important risk factor for the periodontal diseases. Stress causes behavioural changes like the neglected oral hygiene, smoking, alcohol usage, that leads to increased plaque accumulation, gingival inflammation and increased systemic inflammation markers like cortisol, IL-6, TNF, CRP and insulin like growth factor. Personalized Periodontics targets to tailor treatment plans to address these particular factors that enhancing oral health and overall wellbeing.

Genetic heterogeneity

Every single individual has a particular human genome influenced by the genetic variations in multiple loci. Genetic differences involve polygenic effects, gene-gene interactions and variable genetic risk variants, all potentially converting host responses. In current, variants in at least 65 genes, mainly IL genes, TNF-α and FcγR Polymorphisms, are studied are correlated with periodontitis (Loos & Van Dyke, 2020). Conventional periodontal therapy frequently looks these genetic insights though extensive research. And epigenetic modifications are derived or acquired meantime life cause single nucleotide polymorphism, converts DNA structure, immune function and inflammatory effects. Patients with these alterations frequently fail to respond to traditional therapies, highlighting the need for personalized periodontal treatment.

Challenges in older adults

Ageing is associated with biological changes, immune senescence, and comorbidities that complicate periodontal disease. They face extreme risk of cognitive decline, with senility and memory loss, which can worsen the gingival inflammation from poor oral hygiene and tooth loss (person, 2018). The diagnostic tool is to differentiate between normal ageing and pathological conditions in periodontitis, which are lacking. As compared to the younger patients, the treatment strategies for older adults are not fully developed. Its an useful need for research and personalised approaches in the periodontics tailored to the specific needs of ageing populations.

The requirement of personalised dental implants

Every patient has particular anatomical characteristics along with differences in bone density, jaw structure, biting and chewing functions and gum health. Individuals with particular health conditions like osteoporosis, diabetes might require specialized implant materials (Refai, 2023) so custom implants must be crafted to incorporate continuously with the existing oral structures reducing discomfort. Decreasing the risk of complicated and elevating of fasters, less painful recovery.

FUNDAMENTAL CONSIDERATIONS FOR A PERSONALISED APPROACH

Disease severity

Identifying the severity of periodontitis is essential for determining the appropriate level of treatment, and it serves as the foundation for initial staging in the 2018

classification of periodontitis. Advanced imaging, real-time microbiome assessment, proteomic profiling of gingival punch biopsies and biomarker profiling are needed to accurately assess tissue damage severity from periodontitis.

Disease activity

Crucial aspect of managing periodontitis is the ability to determine whether the disease is currently active or in a phase of inactivity or remission. This distinction enables the implementation of different management protocols based on the disease's activity and ongoing tissue destruction. Personalised periodontal therapy can be given after detecting the disease activity through by hi-tech diagnostic tools.

Disease control

Current strategies to control periodontitis focus on understanding it as an opportunistic infection. Key _ approaches include addressing both inflammation and infection, and identifying factors that predispose and modify disease development and progression. Stabilized disease is managed with regular reassessment and supportive periodontal care. Personalised therapy requires sophisticated diagnostic tools to accurately and quantitatively assess disease control post-treatment.

Response to treatment

Several studies showed 20% of patients show inadequate outcomes, while the remaining 80% respond favourably to conventional periodontal treatment. Identifying non-responsive patients is crucial, especially for those who shows poor correlation with plaque exposure duration. Personalized periodontics seeks to assess individual responses, considering factors such as microbiome, resistome, inflammasome, and allostatic load.

Diagnostic tools for personalised periodontics

To make personalized periodontics a reality in clinical practice, advanced diagnostic tools are essential for early, integrated diagnosis, aiming to prevent and detect the disease earlier for more effective treatment. This approach proposes five diagnostic levels (Cafiero & Matarasso, 2013) to identify specific pathogens, genetic and inflammatory biomarkers, and assess an individual's vulnerability and stratify patients into different risk categories to provide tailored treatment.

High-tech diagnostic tools

High-tech diagnostic tools identify lesions before they become clinically detectable. These technologies enable timely intervention during the 'active phase' of periodontitis, improving patient outcomes.

Lab on a chip

Lab-on-a-chip (LOC) consolidates laboratory tasks on a millimetre-sized chip, handling tiny fluid volumes (microfluidics). It enables DNA amplification, sequencing, proteomic analysis, and single-cell gene expression profiling with minimal sample and reagent requirements, offering immediate periodontal health indications for dental operators (Srinivasan, 2004).

Laser Doppler flowmetry

Laser Doppler flowmetry (LDF) is a non-invasive technique used to assess vascular flow in the gingiva. It employs two optical fibres to deliver and collect laser light, measuring blood flow in perfusion units (PU). Periodontal pathogens can enter the bloodstream, causing microcirculatory dysfunction and impaired tissue perfusion (Orekhova & Barmasheva, 2013). Inflammation-induced changes in vascular morphology can predict pathological events in gingival tissue, making blood flow dysfunction a valuable prognostic marker. Hence LDF is recommended to predict and prevent periodontal lesions.

Point of care

Point-of-care testing (POCT) enables rapid chairside diagnostics for immediate treatment decisions in periodontal care. Researchers are identifying periodontal disease signatures to identify inflammatory markers, pathogens, and genetic predispositions for precise diagnosis and treatment. High-throughput biomarker validation tools and miniature chemical processing units provide information on inflammation, tissue degradation, and bone loss, enhancing personalized periodontal care (Ivaturi *et al*., 2021) (Table 1).

Table 1. Chair side diagnostic kits.

Product Name	Purpose
My PerioPath	Determines the cause of periodontal infections.
Oral Fluid NanoSensor Test	Simultaneous and precise detection of multiple salivary protein and nucleic acids.
Electronic Taste Chips	Detects multiple biomarkers for early diagnosis of periodontal disease.
OraQuick	An antibody test that provides results in 20 minutes, usually detects HIV 1 and HIV 2.
Integrated Microfluidic Platform for Oral Diagnosis	Rapidly (3-10 min) measures the concentrations of MMP-8 and other biomarkers in small amounts (10 ml) of saliva.

Genetic susceptibility tests

These tests identify the concurrent presence of allele 2 at the IL1A+4845 and IL1B+3954 loci, with patients having both alleles being referred to as "genotype-positive" for severe chronic periodontitis. The viably available test finds individuals at higher risk before clinical signs and symptoms appear, permitting early intervention and personalized preventive schemes (Greenstein and Haet, 2002). The genetic testing helps to develop treatment plans tailored to particular genetic makeup, potentially enhancing outcomes, and motivates patients to adhere to preventive measures and maintain better oral hygiene. (Table 2).

Table 2. Genetic susceptibility tests.

Genetic Susceptibility Test Kits	Evaluation
Periodontal Susceptibility Test	IL-1α & IL-1β
MyPerioID	IL-1 polymorphism
Perio predict	IL-1 and TNF-α genes
Metagenomic Analysis	Investigates bacterial genes contained in plaque, profiled by gene function
Candidate gene approach	Gene mapping determines if a specific allele is frequently found in patients with a disease compared to those without it
Transcriptomics	Analyze gene expression at the level of the mRNA

Table 3. Microbial test kits.

Microbial Test Kits	Evaluation
EvaluSite	Employs sandwich type ELISA to detect Aa, Pg, Pi
Perioscan	Detects periodontal pathogens *B. forsythus*, Pg, Td, as well as certain *Capnocytophaga* species that produce trypsin-like enzyme
Omnigene (DMDx)	DNA probe for detection of Aa, Pg. A paper point sample of subgingival plaque is placed in the container and mailed for assay. Detects Aa, Pg, Pi, F. nucleatum, C. rectus, T. denticola, and E. corrodens. Reports provided within a short time.
IAI Pado Test	Oligonucleotide probe for detection of Aa, Pg, Tf, Td
BANA	Pg, Td, Tf have unique ability to hydrolyze trypsin substrate
Perio 2000 (Diamond Probe)	Detects sulfate-producing bacteria (Td, Pg, Pi, Tf)
Meridol Periodiagnostics	Real-time PCR for quantitative determination of 6 marker organisms of periodontitis & peri-implantitis (Aa, Pg, Td, Fn, Tf, Pi)
Microbiome Typing (16S rRNA)	Describes characteristics of individual plaques in terms of their bacterial species

Aa-Actinobacillus actinomycetemcomitans, Pg-Porphyromonas gingivalis, Td-Treponema denticola, Tf-Tanerella forsythia, Pi- Prevotella intermedia

Microbial tests

The patient's microbial status and treatment response are tracked by microbial assessment. Anaerobic culture tests or DNA-based chairside tests (semi-quantitative PCR) are the testing methods that offer essential insights to subgingival plaque composition, which elevates antibiotic selection, incorporates probiotics, inhibits resistance, and protects essential microbiota (Shankar *et al*., 2020). The following test kits assist in identifying the specific pathogens responsible for periodontal disease (Table 3).

Host response factors and tissue breakdown-derived products

Periodontal biomarkers related to the host immune response and tissue destruction can detect active disease sites and subclinical inflammation, enabling early intervention and also predict treatment response (Korte & Kinney, 2016). For example, persistent high levels of biomarkers like MMP-8 and IL-1β even after treatment indicate the need for personalized treatment adjustments to improve therapy effectiveness. Biomarkers can be classified into several types based on their role in disease management. Prognostic markers help assess disease progression, stage, and grade during the treatment planning phase. Predictive markers are measured in healthy individuals to aid in disease prevention. Diagnostic markers of disease onset help detect the beginning of a disease, while diagnostic markers and surrogate endpoints are used to evaluate patient compliance, therapy stability, and disease activity during the maintenance phase. Each type plays a vital role in personalized healthcare and treatment strategies (Table 4).

Table 4. Types of salivary biomarkers and their functions.

Type of Marker	Salivary Biomarker	Function
Inflammatory	IL-1β	Potent inflammatory stimulator. Strong correlation with periodontal disease progression.
	IL-6	Regulates immune and inflammatory responses, and stimulates osteoclast differentiation. Increased levels in periodontal disease.
	Macrophage inflammatory protein-1alpha	Synthesis and distribution of proinflammatory cytokines. Increased salivary concentration associated with periodontal disease.
Connective Tissue Degradation	TNF-α	Inhibits bone collagen synthesis. Induces collagenases.
	MMP-8	Degradation of interstitial collagens. Prevalent host proteinase in periodontal disease.
	MMP-9	Degradation of extracellular matrix proteins. Mediator of tissue destruction and immune responses in periodontal disease.
	TIMP-1	Inhibitor of matrix metalloproteinase-8 and collagenolytic activity. Decreased levels in periodontal disease.
	C-4-S (Chondroitin-4-sulphate)	Elevated glycosaminoglycan concentrations were found in aggressive periodontal diseases.
Alveolar Bone Turnover / Resorption	Aspartate aminotransferase	Released during cell injury or death. Increased levels during periodontal disease progression.
	Alkaline phosphatase	Associated with the calcification process. Increased levels in periodontal disease.
	Osteoprotegerin	Competitively inhibits osteoclast differentiation and activity.
	Carboxyterminal telopeptide of type I collagen	Collagen degradation. Released during periodontal disease progression.

Oral fluid diagnostics and rapid point of care test is supported by the American Dental Association. Saliva and gingival crevicular fluid are potent diagnostic medium, giving non-invasive sample collection and patient acceptance. The whole saliva is practical for rapid testing, though gingival crevicular fluid is optimal for specific analyses due to higher biomarkers concentration. (Table 5).

Table 5. biochemical test kits.

Biochemical Test Kits	Evaluation
Periocheck	Detects neutral proteases like collagenases in GCF
Prognostik	Detects serine proteinase & elastase in GCF
Pocket watch	Detects Aspartate aminotransferase in GCF
Periogard	Detects Aspartate aminotransferase in GCF
Perio 2000	Detects VSC (Volatile Sulfur Compounds)
TOPAS	Detects bacterial toxins and proteins
Dipstick	Detects MMP-8 in GCF
IMPOD	Saliva-based detection of MMP-8
OFNASET	Saliva-based detection of IL-1, IL-8
Electronic Taste Chips	Detects CRP (C-reactive protein)
Biolise	Detects elastase in GCF

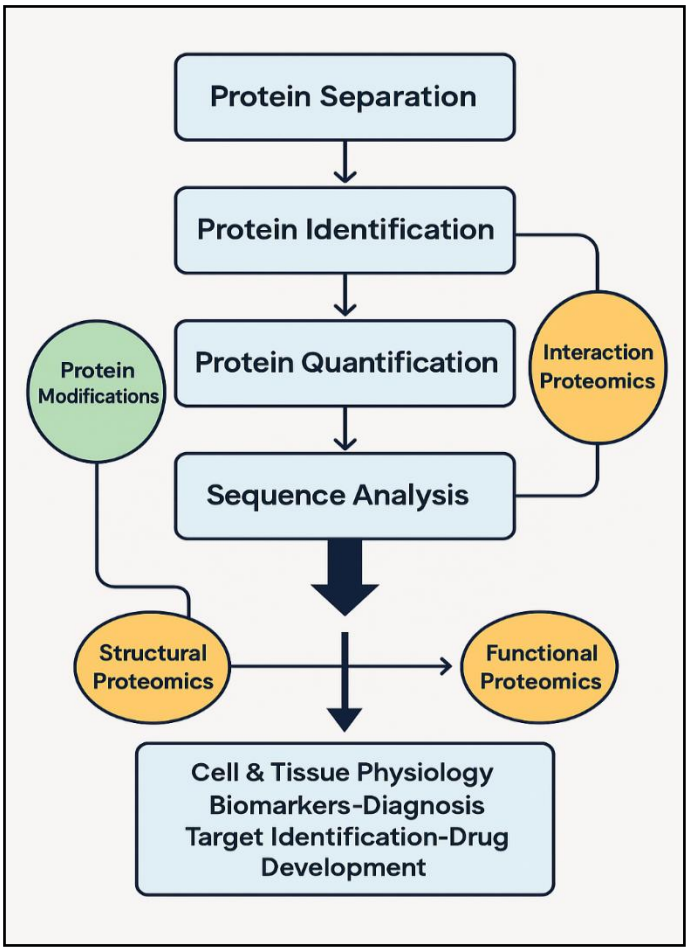

Figure 3. Steps of Proteome Analysis

Proteomics

Studying all the patients in a cell or organism with their adundance, distribution, functions and interactions is proteomics (Hu *et al*, 20025) used two- dimensional gel electrophoresis and a mass spectrometry to find 309 different proteins in human saliva. Liable to its biomarker properites, the salivary proteoma is an invaluable resources for early diagnosis and periodontal disease. (Figure 3).

Metabolomics

Horning implemented 'metabolic profiling' and in 1971, Pauling *et al*, recommended that the metabolome, particular to individuals, can be changed for health and disease treatment. It also identifies and quantifies small metabolites in cells, tissue and bodily fluids glancing dynamic biochemical changes (Mathews *et al*., 2023). Combined with other omics technologies, it offers tailored therapy for periodontal disease. It can be categorized into untargeted metabolomics, which provides an overall profile of many metabolites, and targeted metabolomics, which focuses on analysing specific subsets of known metabolites or pathways. Biological samples such as saliva, gingival crevicular fluid (GCF), dental plaque, oral rinse, tongue swabs, plasma, and serum are commonly used. Analytical techniques include Nuclear Magnetic Resonance (NMR), Gas Chromatography-Mass Spectrometry (GC-MS), and Liquid Chromatography-Mass Spectrometry (LC-MS). Metabolomics is useful for early diagnosis, tracking disease progression, distinguishing types of periodontitis, evaluating therapy and drug efficacy, and identifying periodontitis associated with systemic diseases (Table 6).

Table 6. Metabolites and its association with periodontitis.

Metabolites	Association with Periodontitis
Volatile Sulfur Compounds (VSC)	Higher levels associated with increased pocket depth and tissue destruction
Lactic acid	Elevated in periodontitis, correlates with *P. gingivalis*-positive sites
Glycerol	Reduced levels in periodontitis, used by bacteria for osmoregulatory functions
8-hydroxy-deoxyguanosine	Elevated levels signify oxidative DNA damage in periodontal disease
Lysophosphatidic Acid (LPA)	Higher levels associated with inflammation and bone resorption

4-hydroxynonenal	Increased levels reflect oxidative stress and tissue damage
Neopterin	Elevated levels indicate immune activation and inflammation
Glutathione (oxidized and reduced)	Reduced levels in periodontitis, indicating oxidative stress
Histidine	Higher sensitivity and specificity in diagnosing moderate to severe periodontitis
Phenylacetate	Positively correlates with probing pocket depth
Arachidonic acid	Altered metabolism linked to oxidative stress and inflammation
Linolenic acid	Altered metabolism linked to oxidative stress and inflammation

Patient Risk Assessment and Stratification

The American Academy of Periodontology highlights the importance of including risk assessment in routine periodontal evaluations. In-order to provide tailored preventive and therapeutic strategies, the following multifactorial risk assessment models (Rhea Kiran & Seema, 2016) are designed to identify individuals at elevated risk for periodontal disease (Table 7).

Table 7. Risk assessment models.

Author	Risk Assessment Model	Objective
Fors & Sandberg (2001)	Health Improvement in Dental Practice Model (HIDEP)	Evaluate a tool for oral health overviews and risk identification.
Page ***et al.*** (2003)	Periodontal Risk Calculator (PRC)	Provide a risk score for periodontal progression susceptibility from 1 (lowest) to 5 (highest).
Lang & Tonetti (2003)	Periodontal Risk Assessment Model (PRA)	Classify patients as low, medium, or high risk based on hexagonal risk diagram.
Trombelli ***et al.*** (2009)	University of Ferrara (UniFe)	Provide a risk score from 1 (lowest) to 5 (highest) for periodontal progression.
Chandra (2007)	Modified Periodontal Risk Assessment Model (Modified PRA)	Classify individuals by risk level for periodontal disease, with a focus on diabetes assessment.
Teich (2013)	Risk Assessment-Based Individualized Treatment (RABIT)	Recommend maintenance intervals based on risk classification.
Busby ***et al.*** (2014)	Oral Health Status (OHS) as a part of DenPlan Excel / Previsor Patient Assessment (DEPPA)	Provide patient-level risk scores for periodontal disease, caries, and oral cancer.

INDIVIDUALIZED TREATMENT APPROACH

Integration of precision antibiotic selection

After identifying the disease-causing microbiome with microbial test kits, selective antibiotics are prescribed to target the specific bacteria, while maintaining healthy microbial balance. If pathogens resist first-line antibiotics, alternative treatments based on susceptibility profiles can be provided to ensure effectiveness against specific strains in the patient's oral cavity. Personalized antibiotic prescriptions are especially beneficial for patients with refractory periodontitis, atypical pathogen infections, peri-implantitis, and immunocompromised patients (Žiemytė *et al.,* 2023).

Pharmacogenomics and pharmacomatabonomic therapy

Pharmacogenomics studies the genetic factors whereas Pharmacometabonomics, investigates the dual impact of genetic and environmental factors on drug safety, metabolism, safety, transport, and pharmacokinetics through metabolic profiles of biological fluids. Combining pharmacogenomics and pharmacometabonomics enhances the sensitivity and specificity of drug efficacy and safety (Freires *et al.,* 2012). The advantages of this approach to medicine include avoiding adverse drug reactions and improving overall drug safety. It helps reduce trial-and-error in prescriptions by identifying the optimal dose for each individual. Additionally, it serves as an effective alternative to conventional medicine, offering more personalized and targeted treatment options that enhance therapeutic outcomes.

Epidrugs

Epidrugs are drugs that target epigenetic proteins to treat or prevent diseases. The US FDA has approved DNA methylation inhibitors (DNMTi) like 5-azacytidine and 5-aza-2′-deoxycytidine, and Histone deacetylase inhibitors (HDACi) such as Vorinostat and Romidepsin. Research by Kim *et al.* (25) showed that the HDAC inhibitor sodium

butyrate regenerates periodontium by inducing periodontal ligament fibroblasts to become osteoblasts and also treatment with 5-azacytidine increased TNF-α mRNA expression in periodontitis patients (Abraham *et al.,* 2017). Combining DNMT and HDAC inhibitors could have synergistic effects. Thus, epidrugs can be employed to treat periodontitis by customizing therapy according to personalized genetic profiles.

Gene therapy

Identifying genetic markers linked to periodontal disease can guide personalized treatment. Targeted gene delivery systems, using viral (e.g., adenoviruses, lentiviruses) and non-viral vectors (e.g., liposomes, nanoparticles), introduce therapeutic genes into affected periodontal tissues. Elangovan *et al* (2013) showed that nano-sized calcium phosphate particles (NCaPP) efficiently transfected PDGF plasmids into fibroblasts and exhibited high biocompatibility under in vitro conditions. CRISPR\Cas9 are gene editing technologies that correct genetic mutations or modulate the gene expression, improving tissue regeneration and also controlling inflammation. Periodontal regeneration is stimulated by gene encoding bone morphogenetic proteins (BMPs), vascular endothelial growth factor (VEGF) and platelet derived growth factor (PDGF).

Personalised Tobacco Cessation

Personalised tobacco cessation is a patient-cantered approach it tailors the quitting strategies stationed on individual readiness, preference and needs. Instead using a one-size -fits -all-method, it merges tools like motivational interviewing, 5A and 5R models, evidence -based alternatives and change talk. This improves success rates by addressing physiological and psychological aspect of the tobacco dependence, it guides the patient from intention to lasting change (Ryder *et al*., 2018) (Figure 4).

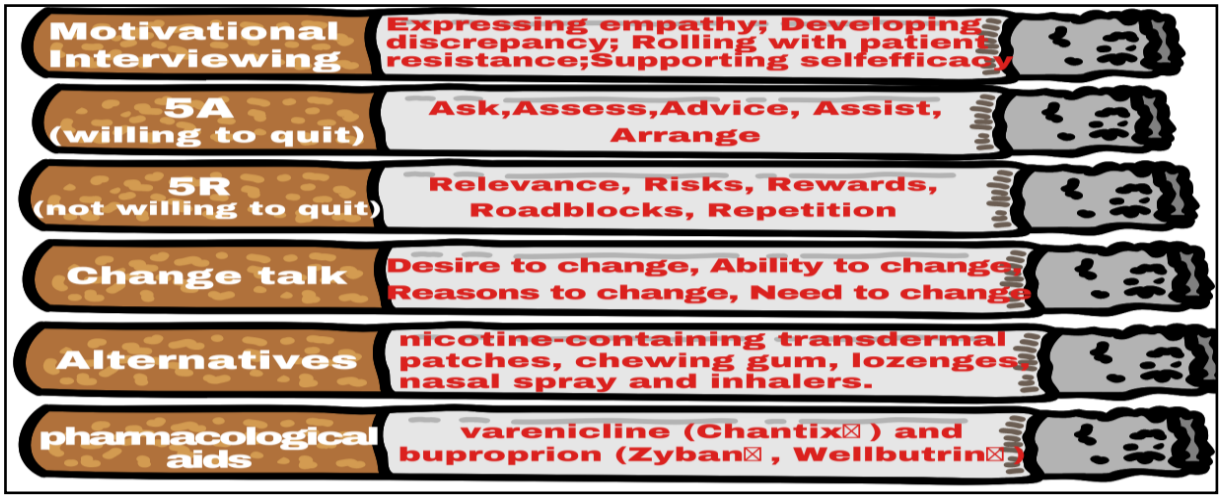

Figure 4. Personalised tobacco cessation

Nutraceuticals for the lifestyle impacts

Based on nutrigenomics, nutraceuticals are developed as health-promoting foods, which studies individual response to dietary compounds with post-genomic technologies. By acting as ligands for transcription factors the nutrients can influence gene expression, and being metabolized or altering signal pathways (Varela-López *et al*.,2018). To enhance disease prevention and improve periodontal health the personalized dietary regimens can be prescribed. (Table 8).

Table 8. Nutraceuticals for periodontal health.

Nutrient	Role in Periodontal Health
Vitamin C	Vital for collagen synthesis, crucial for gum health.
Vitamin D & Calcium	Ensure the structural integrity of alveolar bone.
Antioxidants	Combat oxidative stress in periodontal tissues, including vitamins C and E.
Omega-3 Fatty Acids	Have anti-inflammatory properties, can reduce the depth of periodontal pockets and decrease gum bleeding.
Zinc	Alter periodontal disease progression through changes in expression of the ZnT8 transporter gene.
Probiotics	Help manage periodontal disease by outcompeting pathogenic bacteria and modulating the host immune response.

Customized risk management

Patients are stratified using risk assessment tools, and personalized treatment is tailored according to their modifiable and non-modifiable factors (Suvan *et al.,* 2022) (Figure 5).

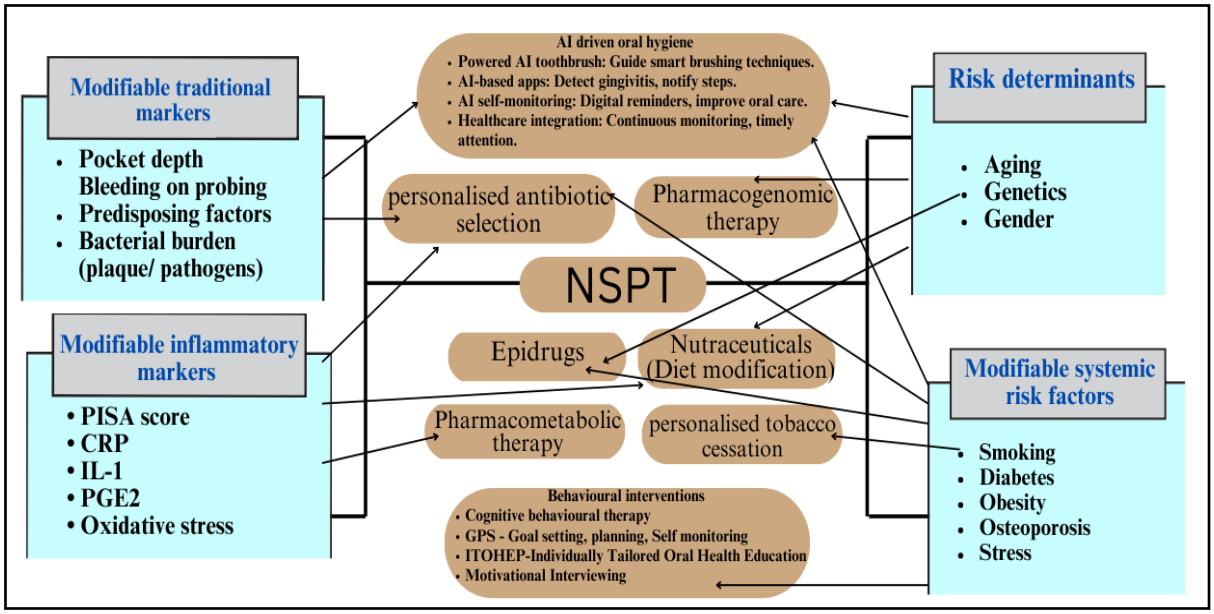

Figure 5. Patient stratification and personalised risk management.

Personalized scaffold-guided tissue regeneration

Periodontal destruction is unique to each individual, and advanced imaging and 3D printing enable personalized regenerative treatments through customized scaffolds. These porous scaffolds are essential for tissue infiltration and vascularization, crucial for regeneration. The "guided tissue regeneration (GTR)" concept uses occlusive membranes to control tissue regrowth in defects, aligning with personalized scaffolds. Additionally, controlling scaffolds internal structures allow for temporo-spatial control over healing, surpassing the capabilities of membrane-based GTR. The effectiveness of this approach has been proven clinically by Rasperini *et al.* (2015) with customized 3D-printed scaffolds for advanced periodontal defect regeneration.

Personalised dental implants

Personalized dental implant therapy goal is to give the correct treatment to the right patient at the right position and time. It is assisted by developments in multi-omics data, biotechnologies, growth and transcriptional factors, and patient-cantered care. Health status, lifestyle, environment, and electronic records also play a role. And bioinformatics and artificial intelligence enhance precision by examining complex data for more treatment planning. Genomics and materiomics are integrated by the concept of personalized dental implants and implantogenomics, it customises implants using advanced technologies to various conditions such as syndromic cases, systemic diseases and elderly individuals with the compromised jaw structures (Refai,2023).

Challenges in Customized Periodontal Care

According to Rakic *et al*. (2021) several challenges like high costs and technical complexity is faced by personalized periodontics, it limits accessibility and necessitate extensive training. Lack of standard protocols, data privacy, and limited long-term evidence complicate its implementation. By the difficulty of identifying causal variants, requiring large collaborative efforts the genetic research in periodontics in hindered. Gene therapy provides extra hurdles, such as need for rigorous clinical trials to make sure safety and efficacy, with ethical considerations. Against these limitations, with continued development and research, the personalized periodontics has the potential to considerably improve clinical outcomes.

CONCLUSION

Personalised periodontics is far from being a myth, it is a burgeoning reality in the modern dentistry. The customization of periodontal treatments to the unique needs of each patient is enabled by the implementation of advanced diagnostic tools and techniques. Success of personalized healthcare in countries like US and UK shows its viability. When a developing country like India, it's able to conquer the dark side of moon, it's not a big task to

achieve the global adaptation of the personalised periodontics with technology adaptation and current education, personalised periodontics is poised to turn a standard in clinical practice, assuring better patient outcomes.

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CONFLICT OF INTERESTS

The authors declare no conflict of interest

ETHICS APPROVAL

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AI TOOL DECLARATION

The authors declare that no AI and related tools are used to write the scientific content of this manuscript.

DATA AVAILABILITY

Data will be available on request

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