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Review Article

Novel therapeutic targets for the treatment of periodontal diseases: Emerging concepts and strategies -A narrative review

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Abstract

Periodontal disease is a complex multifactorial disease. Progression and initiation of periodontal disease are related to multiple etiologic and risk factors, characterized by a disruption in the equilibrium between the host's immune response and the role of the human microbiome. The understanding of the pathogenesis of periodontal disease has improved over the past few years. Consequently, the main objective of periodontal treatment has recently transitioned toward reestablishing homeostasis within the oral microbiome and promoting a balanced interaction with the host's periodontal tissues. This review explores recent advancement in alternative therapeutic strategies and novel compounds that offer promising potential in the prevention and management of periodontal conditions.

Keywords: Recent therapeutic targets, Periodontal diseases, Host modulation, Immunomodulation, Inflammasomes, Quorum sensing, Quorum quenching, Sclerostin, Anabolic agents.

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1. Introduction

Up to 90% of people worldwide suffer from periodontal disease, one of the most prevalent chronic inflammatory conditions, caused by bacterial infections such as dental plaque.[1] Gingivitis and periodontitis are the two most prevalent types of periodontal disease. Up to 95% of people worldwide suffer from gingivitis, a common and curable inflammation of the gingiva. [2] The alveolar bone and the periodontal ligaments are usually not damaged in gingivitis. Periodontitis, on the other hand, is characterised by the breakdown of alveolar bone and periodontal ligaments, and it affects half of the global population.[2] It is caused by bacterial invasion or toxins, but the pathology is primarily determined by the host immune response. The response of a given host to these microorganisms is determined by genetic, immunological, and environmental factors.[3] Microbial toxins and enzymes, excessive bacterial growth, and local phagocytic response all contribute to chronic inflammation, periodontal destruction and the development of periodontal pockets. Periodontitis begins and progresses as a host's imbalanced immune response to an organized dysbiotic biofilm. If local stimulation causes a minor host immune response, immunological surveillance and an adequate immune response take precedence. Conversely, if the pathogenicity of the local microbiota is increased by the colonization of keystone pathogens that stimulate the host immune response, tissue damage occurs. In recent years, understanding the etiology of periodontal diseases has sparked interest in exploring new treatment strategies for the long-term management of periodontitis. Therefore, the purpose of this review is to summarise and evaluate the current literature on recent advancements in alternative methods and therapeutic approaches that target the factors that influence susceptibility to periodontal infection for the treatment and prevention of periodontal diseases as a whole.

1.1. Search strategy

A literature search for published works was carried out utilizing both electronic and manual methods, such as PUBMED, MEDLINE, and Google Scholar **(Figure 1).** The search phrases included recent therapeutic targets in periodontal disease, host modulation, immunomodulation,

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inflammasomes, quorum sensing, quorum quenching, sclerostin and anabolic agents.

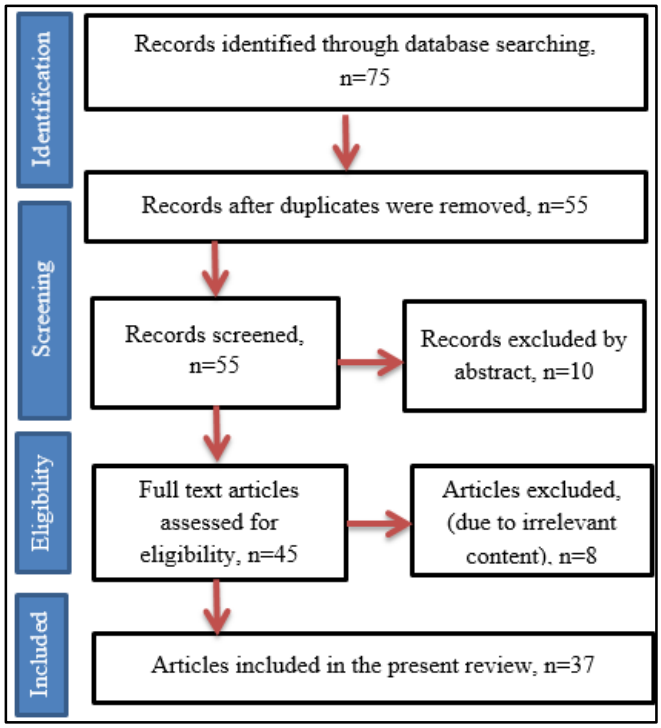

Figure 1: Prisma flow diagram of narrative review

1.2. What are the therapeutic targets for periodontal disease?

Therapeutic targets for periodontal disease include the elimination or significant reduction of pathogenic bacteria, particularly those associated with the red and orange complexes, also aims to control inflammation, restore the balance of the oral microbiome, regenerate lost periodontal tissues and prevent further attachment loss. Targeting pathogenic bacteria's virulence factors and regulatory systems, as well as host-mediated inflammatory response and other variables that influence susceptibility to periodontal infection, is a promising alternative to traditional therapy for better periodontal disease management. Some of the factors that influence periodontal disease aetiology and thus targets for periodontal therapy are mentioned below:

1.2.1. Pathogen associated - virulence factors, bacterial toxins

Over 700 bacterial species inhabit the oral cavity, with Gram-negative bacteria playing a central role in the development of periodontal diseases.[4] These bacteria release endotoxins through outer membrane vesicles during growth, contributing to tissue destruction. Key virulence factors such as gingipains, collagenases, lectins, proteases, superoxide dismutase (SOD), and lipopolysaccharides (LPS) further promote disease progression. LPS not only helps maintain bacterial structural integrity but also facilitates adhesion to surfaces, supporting biofilm formation.[5] As a result, recent therapeutic strategies have focused on targeting these virulence factors and neutralizing bacterial toxins to interrupt the pathogenic mechanisms of periodontal bacteria.

1.2.2. Host immune response

Periodontal disease behaves more like an opportunistic infection rather than a classic one. While bacteria are essential for initiating the disease, it is primarily the host's inflammatory response to the microbial presence that causes damage to the periodontal tissues.[6] The earliest comprehensive model of the host response in periodontitis was proposed in the 1970s by Roy Page and Hubert Schroeder.[7] By 1985, the focus of research had shifted significantly toward exploring the complex interactions among bacteria and the host, marking the beginning of the "Host-Bacteria Inter-Relationship Era." During this period, it became evident that although specific bacterial species trigger the disease, the host's immune response plays an important role in connective tissue breakdown and bone loss. This evolving understanding led to the development of host-modulatory therapies aimed at enhancing treatment effectiveness, slowing disease progression, improving patient management, serving as preventive interventions against periodontitis.

1.2.3. Genetic and enviromental factors

An individual's resistance or susceptibility to periodontitis seems to be influenced by multiple factors, such as genetic, epigenetic, environmental factors, aging, and systemic conditions etc, all of which can alter the host's immune response in either a protective or destructive direction.[8] Risk factors influencing the onset, progression, severity of periodontal disease, and its response to treatment include:

1. Heredity
2. Smoking
3. Hormonal changes
4. Systemic diseases
5. Immunocompromise
6. Stress
7. Nutrition
8. Certain medications
9. Faulty dentistry and a previous history of periodontal disease.

Some of these risk factors contributing to periodontitis are modifiable, offering opportunities to reduce a patient's susceptibility to the disease. These include smoking cessation, better management of systemic conditions, stress reduction, and nutritional support. Additionally, the use of targeted pharmacological agents specifically designed to enhance the treatment of periodontitis is gaining attention as a supportive strategy for managing high-risk individuals.

1.3. Advances in therapeutic target identification

1.3.1. Quorum sensing, quorum quenching

Microorganisms regulate their population density by releasing chemical signaling compounds a process known as

"Quorum Sensing (QS)". This mechanism allows bacteria to detect changes in their population and adjust gene expression accordingly. Since bacterial communication plays a crucial role in biofilm development, disrupting QS pathways offers a strategic approach to limiting biofilm formation, reducing virulence and suppressing the growth and toxin production of periodontal pathogens. Compounds that block QS pathways by interfering with signal detection or synthesis can inhibit microbial growth, disrupt virulence, and prevent biofilm formation. This approach is known as "Quorum Quenching", which includes enzymatic breakdown of the signaling molecules (autoinducers). Gram-negative bacteria generally use N-acyl homoserine lactones (AHLs) as signaling molecules, whereas peptides are used in gram-positive bacteria. Quorum sensing inhibitors (QSIs) often derived from natural sources like plants, algae, and fungi are being explored as promising agents for disrupting microbial communication.[9] Emerging evidence suggests that QSIs can effectively reduce plaque biofilm formation and help in managing periodontal diseases.[10]

1.3.2. Inflammasome

When the human body experiences injury, it initiates a complex sequence of immune and inflammatory reactions. A key player in this process is the inflammasome, a cytosolic multiprotein complex within the innate immune system that regulates inflammation. Inflammasomes are basically intracellular pattern recognition receptors (PRRs) that recognize pathogen-associated or danger-associated molecular patterns (PAMPs and DAMPs).[11] Different types of inflammasomes can form, each named after the specific sensor PRR that activates it. These sensor molecules span several PRR families; including nucleotide-binding domain leucine-rich repeat-containing proteins (NLRs or NOD-like receptors), AIM2-like receptors (ALRs), RIG-I-like receptors (RLRs) and Pyrin inflammasome are associated with periodontal disease pathogenesis.[12] Persistent or inappropriate inflammasome activation, leading to continuous cytokine production, is thought to be a key factor in the development of numerous chronic diseases, including periodontal disease. As such, targeting inflammasome pathways either by inhibiting its components, modulating activation, or reducing cytokine output has shown potential as an effective approach for managing periodontal disease.

1.3.3. Anabolic agents

1. **Teriparatide:** Teriparatide, a synthetic analog comprising the active fragment of parathyroid hormone (PTH), was the first anabolic drug approved by the FDA for osteoporosis treatment. PTH plays a vital role in modulating the Wnt/β-catenin signaling pathway, where it inhibits sclerostin, a natural suppressor of Wnt-LRP5/6 signaling, thereby enhancing bone formation.[13] This pathway also triggers a signaling cascade involving cyclic AMP, protein kinase A, and protein kinase-1, all of which contribute to its anabolic effects on bone.

2. **Sclerostin Antibody:** The Wnt/β-catenin signaling pathway, second only to bone morphogenetic proteins (BMPs), is essential for osteoblast differentiation and bone growth.[14] Like many signaling pathways, Wnt activity is finely balanced by both stimulatory and inhibitory molecules. Sclerostin, a protein encoded by the *SOST* gene and predominantly produced by osteocytes, acts as a potent Wnt pathway antagonist, thereby limiting bone formation. Previously thought to function as a BMP inhibitor, recent studies now indicate that blocking sclerostin can reduce bone resorption and encourage bone regeneration.[15] Therefore; targeting sclerostin has emerged as a potentially innovative strategy in managing periodontal disease.

1.4. Conventional therapies for the management of periodontal diseases

The success of periodontal treatment, both in the short and long term, relies significantly on the mechanical disruption of supra- and subgingival pathogens found within the biofilm.[16] The initial causal therapy mainly consisted of oral hygiene instruction and professional scaling and root planing **(**SRP**)**. The mechanical disruption of both supra- and subgingival pathogens, along with calculus, is achieved through a combination of periodontal debridement and patient-administered oral hygiene instructions, resulting in a synergistic therapeutic effect.[17] In this context, SRP remains the gold standard for non-surgical periodontal treatment. However, SRP alone is insufficient to ensure beneficial long-term outcomes. This can be explained in part by mechanical debridement's intrinsic limitations in complex anatomical sites such as tortuous periodontal pockets, furcation areas, and vertical bone defects. Moreover, SRP alone may have limited effectiveness against certain periodontal pathogens, particularly bacteria from Socransky's red complex, and is insufficient in eradicating periodontopathogens residing in non-typical oral sites such as the oral mucosa or tongue.[18] These therapeutic limitations can be partially addressed by incorporating various alternative technologies. If non-surgical periodontal therapy (NSPT) proves ineffective or in cases of advanced periodontal defects and severe periodontitis, surgical intervention may be considered at a later stage.

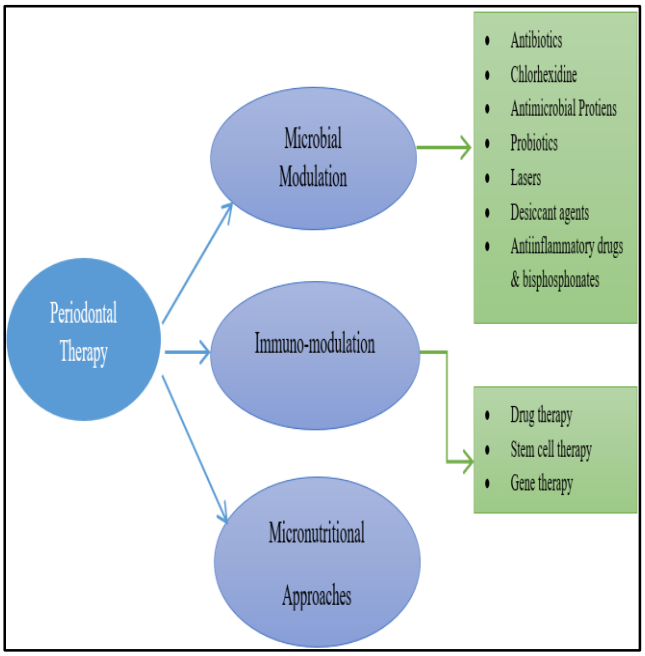

Figure 2. Emerging treatment strategies for periodontal disease management.

1.4.1. Microbial influence and therapeutic modulation of periodontitis

1. Antimicrobials: Most forms of periodontal diseases are driven by the presence of a pathogenic microbiome within the periodontal pocket, necessitating therapeutic strategies to manage and disrupt this microbial community. Pathogen-induced oral infections are generally difficult to manage using antibiotics alone; especially without prior mechanical removal of the biofilm.[19] Current consensus emphasizes that effective antimicrobial treatment must be preceded by thorough periodontal debridement to eliminate both supra- and subgingival biofilms.[20] Some clinical studies support the idea that in certain cases such as aggressive or rapidly progressing periodontitis, the existence of advanced periodontal pockets or distinct pathological conditions combining systemic antibiotics with SRP can offer meaningful clinical improvements both in medium and long run. Amoxicillin and metronidazole were originally prescribed in combination to eliminate Aggregatibacter actinomycetemcomitans from periodontal tissues.[21] When it comes to topical antibiotic in periodontics (such as Doxycycline, minocycline, metronidazole etc), studies have reported limited effectiveness, mostly in areas unresponsive to conventional SRP or in cases of localized periodontal lesions. Compared to systemic antibiotics, topical applications such as gels, chips, or rinses are associated with fewer side effects, reduced risk of bacterial resistance, and better patient tolerance. Current research is increasingly focused on alternative treatment approaches to minimize antibiotic use, especially systemically, reserving them for the most severe cases of periodontitis. One promising avenue involves antiviral therapies targeting leukotoxin A (LtxA), a virulence factor secreted by A. actinomycetemcomitans strains, which selectively destroys human white blood cells and compromises the host immune response.[22] Studies indicate that interfering with virulence factors like LtxA could serve as a viable method to treat periodontal disease. Catechins, a class of flavonoid, can block the function of LtxA in both its soluble and vesicle-bound form.[23]
2. Chlorhexidine: During the active phase of periodontal therapy, antiseptic solutions are widely utilized to manage biofilms. Chlorhexidine (CHX) gluconate is a broad-spectrum antiseptic agent with antifungal and bactericidal properties, effectively targeting pathogenic microbes involved in periodontal disease, encompassing both Gram-positive and Gram-negative strain. Apart from its bactericidal action, CHX also exhibits bacteriostatic properties that help inhibit bacterial growth.[24] This bisbiguanide compound is available in various slow-release formulations, such as gels, varnishes, and chips. Clinical studies have shown that using CHX mouthwash yields superior clinical and microbiological outcomes compared with SRP alone, SRP combined with clinical plaque control, or SRP with placebo.[25] Nonetheless, potential side effects such as tooth and mucosal staining, changes in taste perception and increased calculus formation must be weighed with cautions.
3. Antimicrobial Proteins: The host defense system relies heavily on antimicrobial peptides and proteins, which help control the growth and transmission of oral pathogens. Notably, several antimicrobial peptides such as the tissue-specific human beta-defensin-3 (hBD-3) and the cathelicidin LL-37 have been investigated for their effectiveness against various periodontal bacterial strains.[26] Additional antimicrobial agents, including lactoferrin and mucin-7; have also been linked to periodontal health. Significantly reduced levels, up to threefold of mucin-7 (MG2) and lactoferrin were observed in patients with periodontitis compared to healthy individuals, indicating their potential contribution to antimicrobial protection.[27]
4. Probiotics: The “probiotics” term was defined in 2005 as live microorganisms that, when administered in adequate amounts, confer health benefits to the host, have been studied as adjuncts in periodontal therapy.[28] In cases of periodontitis, probiotic strains have shown potential in preventing bacterial recolonisation when used alongwith SRP.[29] The majority of studies on probiotic research have focused on products derived from Lactobacillus, particularly Lactobacillus reuteri. A recent meta-analysis revealed that incorporating probiotics into periodontal treatment led to a clinical

attachment level (CAL) gain of 0.42 mm and a probing depth (PD) reduction of 0.18 mm in moderate periodontal pockets, and 0.67 mm in deeper pockets after periodontal therapy.[30] Furthermore, consistent probiotic consumption has been associated with the most favorable outcomes when used as an adjunct to SRP.

5. Lasers: Lasers have been introduced into medical practice for treating a variety of conditions over the past century. The laser device emits electromagnetic radiation at specific wavelengths and low-intensity beams, producing significant effects on biological tissues. In both soft and hard tissues, medical lasers can induce vaporization, microbial destruction, hemostasis, and tissue biostimulation, all contributing to favorable therapeutic outcomes. The primary function of most surgical lasers lies in their bactericidal effect, primarily mediated through photothermal mechanisms. Bacterial inactivation or destruction typically occurs during the evaporation or protein denaturation phases triggered by laser irradiation.[31] As a result, the disinfection effect provided by laser treatment proves beneficial during surgical procedures.[32] Owing to their added decontaminating and detoxifying properties, lasers have been shown to support improved wound healing when compared to traditional methods applied in periodontal pocket therapy. Furthermore, it has been suggested that laser irradiation of the root surface may exert a microbial inhibitory effect, reducing bacterial adhesion and colonization an essential factor in maintaining the long-term stability of periodontal healing. Different types of lasers used in the treatment of periodontal diseases are summarized in **Table 1.**

Table 1: Different laser types used in the treatment of periodontal diseases.

Laser Type	Wavelength	Use
Nd:YAG	1064 nm	For soft tissue surgeries (ablating, incising, excising, coagulating) aphthous ulcers treatment, pocket debridement, new attachment procedure-LANAP.
Er:YAG	2940 nm	For soft tissue surgery, sulcular debridement, cutting, contouring and resection of bone tissues, osseous surgery and osteoplasty.
Carbon dioxide	10,600 nm	For soft tissue surgery, debridement, coagulation, LANAP.
Diode	800-980 nm	For soft tissue surgery, sulcular debridement, coagulation, osseous crown lengthening, osteoplasty, removal of subgingival calculus.
Er,Cr:YSGG	2780 nm	Soft tissue surgery, sulcus debridement, cutting, contouring and resection of bone tissues, root decontamination, removal of subgingival calculus.

6. Desiccant Agents: Considering the pivotal role of biofilms in the development and persistence of periodontitis, several adjunctive therapies have been implemented to improve the effectiveness of SRP by specifically targeting periodontal pathogens. One such approach involves the use of desiccant agents, initially developed for managing oral aphthous stomatitis.[33] More advanced desiccant formulations were later created specifically for use in periodontal pockets. These desiccants, typically in liquid or gel form, are composed of a mix of sulphonic and sulfuric acid components. The sulfate groups within the desiccant carry a negatively charged surface because of their inherently polar structure and the presence of oxygen atoms on the surface. This characteristic enables the solution to bind the water within the oral bacterial biofilm matrix, facilitating adhesion and detachment, leading to rapid disruption and complete removal of the biofilm from both the gingival and root surfaces.[34]
7. Anti-Inflammatory Drugs and Bisphosphonates: Various cells in the periodontal tissue, including neutrophils, macrophages, fibroblasts, and epithelial cells, exhibit responses to bacterial lipopolysaccharides (LPS) by releasing prostaglandins, which are powerful pro-inflammatory mediators linked to the advancement of periodontal disease. Research indicates that both local and systemic administration of non-steroidal anti-inflammatory drugs (NSAIDs) can reduce gingival bleeding in the short term, while longer-term usage has been associated with decreased bone loss.[35] Bisphosphonates primarily prescribed for conditions like osteoporosis and bone metastases, function by inhibiting bone resorption. However, their major side effect is osteonecrosis of the jaw, especially in patients receiving the drug intravenously.[36] Due to their anti-resorptive effects, bisphosphonates have gained attention in periodontal research as a possible adjunctive therapy, with some encouraging outcomes reported in both animal and human trials.[37] However, while bisphosphonates may have the potential to influence the severity of periodontal disease, their application remains controversial due to risk of

osteonecrosis and for the potential equivocal data related to the fact that osteoporosis could influence the progression of periodontitis.[38]

1.4.2. Immunomodulation of periodontitis

The immune activity in the periodontal microenvironment is a key factor in the onset and progression of the disease. Immunomodulatory therapy, which aims to regulate the immune response, has recently attracted significant attention as a promising approach for managing periodontal diseases.[39] By controlling both the inflammatory and bone-resorbing processes, this therapy helps limit bone loss and prevent the periodontal disease progression. To influence these processes, various strategies have been explored, including immunomodulatory drugs, stem cell-based treatments and gene therapy.

1. Drug therapy- Targeting the immune system through drug therapy is especially promising for periodontal treatment. For example, Resveratrol, a natural polyphenol, suppresses NF-κB activation in macrophages and modulates the immune response to Fusobacterium nucleatum by enhancing antioxidant pathways.[40] While statins are mainly used for lowering cholesterol, their anti-inflammatory properties have potential relevance in periodontal therapy. Metformin also has shown effectiveness by reducing nitric oxide synthase production in monocytes and regulating IL-1β, which helps decrease inflammation. Evidence has demonstrated that it can promote osteoblastic differentiation and enhance bone formation.[41] In clinical studies, the local application of 1% metformin gel into periodontal pockets has shown significant improvements in probing depth, clinical attachment level and reduction of intrabony defects when used as an adjunct to SRP.[42] Similarly, Catechin, another polyphenol, functions as an immunomodulatory agent. Studies revealed that curcumin promotes the release of anti-inflammatory agents and combats periodontal pathogens.[43] Trans-cinnamic aldehyde targets Aggregatibacter actinomycetemcomitans by down regulating cytokines like TNF-α and IL-1β. Other compounds, such as carnosic acid, glyburide, and bismuth-based drugs, contribute to modulation of immune response by inhibiting infiltration of inflammatory cells and reducing level of pro-inflammatory cytokines like IL-6, IL-1β, and TNF-α.[44]
2. Stem cell therapy holds significant promise in advancing periodontal health. Various mesenchymal stem cell (MSC) types have demonstrated the capacity to influence the local immune microenvironment by interacting with leukocytes and cytokines that drive periodontal inflammation. These cells are being actively explored as novel therapeutic strategies for managing periodontitis. For example, periodontal ligament stem cells (PDLSCs) have shown the ability to communicate with immune cells and modulate their functions, positioning them as a potential treatment option for periodontitis.[45] Gingiva-derived mesenchymal stem cells (GMSCs) promote the polarization of macrophages from the pro-inflammatory M1 phenotype to the anti-inflammatory M2 state, while also decreasing neutrophil infiltration and reducing levels of pro-inflammatory cytokines.[46] Stem cells from human exfoliated deciduous teeth (SHEDs) have exhibited immunomodulatory properties by lowering levels of TNF-α, IFN-γ, and IL-2, and enhancing anti-inflammatory responses in macrophages.[47] Similarly, dental follicle stem cells (DFSCs) have been shown to influence peripheral blood mononuclear cells by increasing IL-10 while decreasing IFN-γ and IL-4, underscoring their potential role in immune modulation during periodontal therapy.[48] Additionally, bone marrow-derived MSCs and dental pulp stem cells (DPSCs) have demonstrated the ability to suppress pro-inflammatory cytokines such as TNF-α, IFN-γ, and IL-17 in response to periodontal pathogens.
3. Immuno-related gene therapy is an innovative strategy that targets host immunity. It has been demonstrated that periodontal disease is impacted by the down regulation of the T-cell immune response, which results in fewer T-cells in gingival tissue. Research has shown that inhibiting T-cell immune response cDNA7 (TIRC7) lowers the quantity of T-cells in gingival tissue, which affects the course of periodontal disease. Certain studies have shown that an isoform of Atp6i, known as T-cell immune response cDNA7 (TIRC7), is closely linked to the regulation of T cell and B cell activation.[49] Additionally, injecting plasmid DNA expressing miR-200c into the gingival tissue has been found to eliminate gingival inflammation.[50]

1.4.3. Micronutritional approaches

The development and progression of periodontal disease are influenced by a fine balance between microbial factors and the immune response of the host. Nutrition plays a significant role in maintaining this balance, as it is linked to several chronic inflammatory diseases such as cardiovascular disease, diabetes mellitus, rheumatoid arthritis, and inflammatory bowel disease that are also associated with periodontitis. There are six fundamental categories of nutrients: carbohydrates, fats, proteins, vitamins, minerals, and water. These are generally divided into macronutrients and micronutrients. Micronutrients which include vitamins, minerals, trace elements, amino acids, and polyunsaturated fatty acids (PUFAs), are required in small amount, (typically micro or milligrams per day) but are vital for health, growth, and metabolic function. In the context of periodontal health, several micronutrients have been recognized for their supportive roles. According to Medical Subject Headings

(MeSH), relevant compounds including vitamins A, B-complex, C, D, E, and K, as well as biotin, carotenoids, flavonoids, glutathione, melatonin, polyphenols, polyunsaturated fatty acids (like omega-3s), macrominerals, and trace minerals all of which contribute to preventing or managing periodontal disease.[51]

2. Discussion

Periodontitis is a bacterial-associated and host-mediated multifactorial inflammatory disease. Keystones in periodontitis onset and disease progression are biofilm dysbiosis coupled with sustained inflammation in the periodontium.[52] The treatment of periodontal disease in its various forms has evolved over the past century. In periodontology, it has been recognized that the removal of supra and subgingival deposits had a favourable influence on periodontal tissues. The biofilm removal, both through periodontal therapy using SRP and thorough home oral hygiene, has become the milestone of modern periodontal treatment.[17] However, it has been shown that changing the proportions of periodontopathogenic bacteria and the speed of recolonisation of the biofilm is regulated and improved by the use of additional agents to traditional mechanical procedures. The addition of chemotherapeutic agents administered systemically or locally, measured by clinical, microbial, and inflammatory outcomes has been demonstrated to possess good results.[17] Novel responses to periodontitis will require an understanding of individual molecular pathogenesis and the development of target-oriented therapeutic drugs which can be used as therapeutic options aside from the traditional treatment approaches. The previously mentioned novel treatment strategies including microbial modulation, immunotherapy and nutritional adjunct have been studied for their effects on the periodontium and are stipulated to be alternative therapeutic strategies for personalized medicine in periodontal disease management.[51] Experimental evidence shows that immunotherapy is effective in the repair and regeneration of periodontal tissue and can be used as a treatment for periodontitis.[39] Incorporating novel concepts of immunomodulation and gene therapy into the existing concepts of pathogenesis will highlight the importance of genetics and immune cells, which could further impact periodontal diseases. In addition, recent focus on inhibiting plaque biofilm formation with QSIs, anabolic agents, targeting inflammasomes production may result in the development of new drugs for periodontal treatment.[52] Therefore; this review highlights the need for future research to prioritize integrated treatment approaches tailored to each patient's unique microbial profile and individual response.

3. Conclusion

Periodontitis involves a dynamic interaction between pathogenic bacteria and the host's immune response, contributing to its multifactorial inflammatory nature. Traditional treatment approaches often fall short when excessive or dysregulated immune reactions compound the local inflammation triggered by bacterial colonization. As a result, future strategies for managing periodontitis must focus on understanding the molecular mechanisms behind the disease and developing targeted therapies. The direction of upcoming research in periodontology is to design personalized treatment plans, tailored not only to the patient's microbial profile but also to their unique immune response. This personalized approach aims to improve treatment outcomes by addressing both the microbial and host-specific factors that contribute to the disease.

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5. Conflicts of Interest

Author declared no conflicts of interest

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All contributors have been included as authors.

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