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DIETARY PATTERNS, LIFESTYLE FACTORS, AND THEIR COMBINED IMPACT ON PERIODONTAL HEALTH: AN INTEGRATIVE REVIEW

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Abstract

Periodontal disease is a chronic inflammatory condition driven by a dysregulated host response to a dysbiotic biofilm, with increasing evidence highlighting the influence of modifiable lifestyle factors. This integrative review synthesizes current evidence on the role of dietary patterns, specific nutritional strategies, and lifestyle behaviors in the prevention and management of periodontal disease. Data from systematic reviews, meta-analyses, randomized controlled trials, and population-based studies suggest that overall diet quality significantly influences systemic inflammation, immune function, and microbial homeostasis, all of which are crucial to periodontal pathophysiology. Anti-inflammatory dietary models, particularly the Mediterranean diet and high-quality plant-based diets, are consistently associated with improved periodontal parameters and reduced disease severity. Key nutrients and bioactive compounds, including omega-3 fatty acids and essential micronutrients, demonstrate adjunctive benefits when integrated with conventional periodontal therapy. Emerging evidence further implicates the oral-gut axis as a mechanistic link between diet-induced microbial alterations and systemic immune regulation influencing periodontal outcomes. Despite promising associations, the current literature is limited by heterogeneity of study designs and a predominance of observational data. Nonetheless, the findings support the integration of personalized nutritional counseling and lifestyle optimization into comprehensive periodontal care, emphasizing a systems-based approach to disease prevention and long-term periodontal stability.
Keywords: periodontitis, dietary patterns, lifestyle factors, systemic inflammation, oral–gut axis

Introduction

Periodontitis is a highly prevalent, chronic, biofilm-associated inflammatory disease characterized by irreversible destruction of the periodontal supporting tissues and, ultimately, tooth loss. Contemporary concepts emphasize that periodontal breakdown is not merely a consequence of bacterial accumulation, but the result of a dysregulated host-microbe interplay in which a dysbiotic biofilm triggers and perpetuates an exaggerated immune-inflammatory response. In this model, shifts in microbial ecology ("dysbiosis") and the host inflammatory

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phenotype are reciprocally reinforced, sustaining connective tissue degradation and alveolar bone resorption even when classical plaque levels alone do not fully explain disease severity or progression [6]. From a public health perspective, the burden is substantial: pooled epidemiological analyses over the past decade confirm that periodontitis remains a common worldwide condition, with significant implications for quality of life and healthcare systems [5].

Beyond oral hygiene and professional therapy, interest has accelerated in modifiable determinants that influence periodontal susceptibility and inflammatory load. Among these, dietary patterns and lifestyle factors stand out because they act upstream on systemic inflammation, oxidative stress, immune competence, and nutrient-dependent tissue homeostasis, processes that converge on periodontal pathogenesis. A recent systematic analysis focusing on dietary patterns and periodontitis supports the concept that global diet quality, rather than single nutrients alone, relates meaningfully to periodontal outcomes, encouraging a pattern-based approach (e.g., anti-inflammatory vs. pro-inflammatory dietary models) when interpreting clinical risk and designing interventions [1]. This perspective is clinically relevant because periodontal inflammation is increasingly viewed as part of a broader inflammatory network, where metabolic and nutritional signals modulate immune set-points and tissue resilience.

Among micronutrients, vitamin C has received particular attention due to its central role in collagen synthesis, wound healing, and antioxidant defense, functions directly relevant to gingival integrity and periodontal connective tissue turnover. Mechanistically, vitamin C contributes to maintaining epithelial barrier competence and controlling oxidative stress within inflamed tissues, supporting the premise that inadequate vitamin C exposure could favor exaggerated inflammatory responses and impaired repair. Narrative and mechanistic syntheses outline beneficial roles of vitamin C in maintaining oral health, framing it as a biologically plausible modulator of periodontal inflammation and healing capacity [2]. However, the clinical literature is heterogeneous: systematic evidence assessing supplementation and periodontal outcomes raises the practical question of whether vitamin C intake confers a measurable protective effect at the population level, beyond confounding behaviors such as smoking, socioeconomic status, and overall diet quality [3]. Lifestyle and nutritional epidemiology further supports this link: analyses based on large representative datasets report associations between dietary vitamin C intake and periodontitis, consistent with a dose–response hypothesis in which inadequate intake may correspond to poorer periodontal status [9]. Complementing population-level findings, pilot clinical observations have explored vitamin C levels among patients with periodontosis, highlighting the need to interpret biochemical status (not only reported intake) when discussing individualized risk and nutritional counseling [10].

A critical nuance is that vitamin C is not a “pure” antioxidant in all contexts; depending on the biochemical milieu, it may exert pro-oxidant effects, underscoring why simplistic supplementation assumptions may be clinically misleading. This dual behavior has been described in detail, with implications for interpreting supplementation trials and tailoring dosage to patient context (e.g., inflammation severity, redox-active metals, systemic conditions) [7]. Translationally, experimental work testing local vitamin C application in periodontitis models suggests potential anti-inflammatory and tissue-protective effects at the periodontal site, supporting exploration of targeted adjunctive approaches alongside conventional therapy [11]. Taken together, these data justify positioning vitamin C not as a standalone “treatment,” but as a biologically relevant variable within a broader dietary-host-response framework.

In parallel, vitamin D has emerged as a key candidate linking lifestyle to periodontium through its immunomodulatory actions and its role in mineral homeostasis and bone metabolism. Vitamin D can shape innate and adaptive immune responses, potentially influencing periodontal inflammatory tone and tissue catabolism. The immunomodulatory effects of supplementation have been demonstrated in deficient populations, supporting the plausibility that correcting deficiencies could alter inflammatory profiles relevant to periodontal disease [12]. Updated systematic syntheses and meta-analyses report associations between vitamin D status and

periodontitis, collectively suggesting that lower vitamin D exposure may correlate with worse periodontal parameters, although causality and effect size remain under active investigation [13]. Observational clinical data connect vitamin D levels to periodontal attachment loss, strengthening the clinical relevance of vitamin D status when discussing structural periodontal outcomes rather than gingival inflammation alone [14]. Earlier meta-analytic work similarly evaluated the vitamin D–periodontitis relationship, contributing to the evidence base but also illustrating heterogeneity in definitions, baseline status, and confounder control [15]. Focused reviews further integrate vitamin D biology with periodontal health and disease, providing a mechanistic rationale for how vitamin D pathways may modulate periodontal immunity and bone remodeling [16]. At a biomarker level, recent clinical research reports inverse associations between serum vitamin D concentration and MMP-9 levels, an enzyme implicated in extracellular matrix degradation, supporting a mechanistic bridge between vitamin D status and tissue-destructive pathways in periodontal disease [17]. Additionally, assessment of vitamin D binding protein in serum and gingival crevicular fluid in health versus chronic periodontitis adds granularity to the vitamin D axis, suggesting that carrier proteins and local bioavailability may be relevant, not only total circulating vitamin D [18]. Importantly, integrative discussions also consider the combined relevance of vitamins C and D in periodontal pathogenesis and therapy, reinforcing the need for multi-nutrient reasoning rather than isolated nutrient claims [8].

Lifestyle behaviors beyond diet also matter. Physical activity is increasingly recognized as an anti-inflammatory exposure that can reduce systemic inflammatory burden and improve metabolic regulation, factors that plausibly translate into periodontal benefit. Meta-analytic evidence supports an association between exercise and lower periodontitis prevalence, motivating combined lifestyle strategies in periodontal prevention frameworks [4]. Finally, interest in omega-3 fatty acids reflects the modern emphasis on resolution biology and host modulation. Systematic reviews and meta-analyses indicate that omega-3s may be beneficial as adjuncts in periodontal management, aligning with mechanisms related to pro-resolving mediators and inflammatory control [19]. Evidence specifically addressing omega-3 effects during active periodontal therapy similarly supports adjunctive potential, reinforcing omega-3s as a plausible component of integrative periodontal care rather than a substitute for mechanical biofilm control [20].

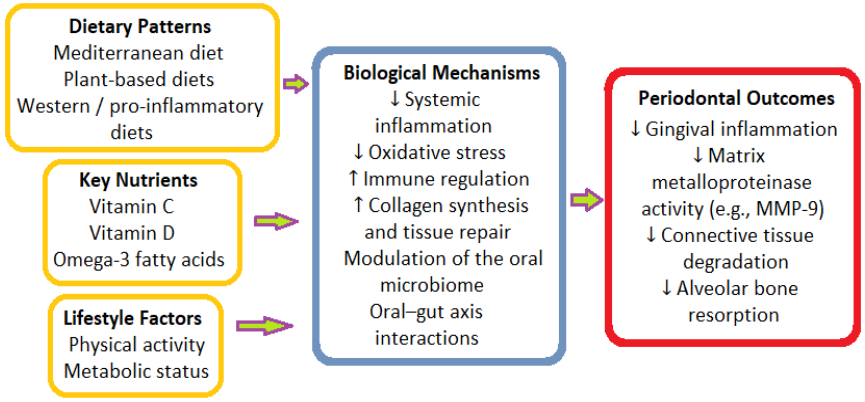

Figure 1. Integrative framework linking dietary patterns, lifestyle factors, and periodontal disease [1-4,6-8,13,19-20].

Figure 1 presents an integrative conceptual model illustrating how dietary patterns, key micronutrients, and lifestyle factors influence periodontal disease through modulation of systemic inflammation, oxidative stress, immune regulation, collagen turnover, and oral microbiome homeostasis. These interconnected pathways collectively affect gingival

inflammation, extracellular matrix degradation, and alveolar bone resorption, contributing to periodontal disease progression or stability.

Methodological framework

The present work was designed as an integrative review, chosen to allow the simultaneous inclusion and synthesis of evidence derived from heterogeneous study designs, including systematic reviews, meta-analyses, randomized controlled trials (RCTs), observational studies, and large population-based datasets. This approach is particularly suitable for investigating diet–periodontium relationships, where complex exposures (dietary patterns, nutrients, lifestyle behaviors) interact with biological, behavioral, and socioeconomic confounders that a single methodological paradigm cannot adequately capture. Integrative reviews enable a comprehensive appraisal of both quantitative outcomes and mechanistic interpretations, facilitating clinically relevant conclusions.

Scope and conceptual boundaries

The methodological framework focused on studies evaluating the association between dietary components, dietary patterns, and lifestyle-related factors and periodontal health outcomes, either as primary endpoints (e.g., probing depth, clinical attachment loss, gingival inflammation) or as adjunctive outcomes in periodontal therapy. Particular emphasis was placed on evidence addressing anti-inflammatory and host-modulatory mechanisms, consistent with the current paradigm that periodontitis is driven by dysregulated inflammation rather than bacterial load alone. Within this context, omega-3 fatty acids were considered a reference nutrient due to their well-established role in inflammation resolution and frequent use as adjuncts in periodontal interventions [20].

Evidence hierarchy and study types

Priority was given to systematic reviews and meta-analyses, as these provide the highest level of synthesized evidence regarding nutritional and lifestyle interventions in periodontology. Recent meta-analytic data examining omega-3 fatty acids in active periodontal therapy were included as benchmarks for methodological rigor, highlighting standardized periodontal endpoints and well-defined intervention protocols [20]. Additionally, controlled clinical trials evaluating dietary patterns, particularly the Mediterranean diet, were incorporated, as these studies offer higher internal validity compared to observational designs while maintaining translational relevance [21].

Observational cross-sectional and cohort studies were also included, particularly those derived from large-scale datasets such as NHANES, which allow robust statistical adjustment for confounders and enhance external validity. For example, studies examining the association between serum calcium levels and periodontitis in representative populations provided valuable insights into mineral metabolism and periodontal attachment outcomes, despite their inherent limitations regarding causality [22].

2.3 Inclusion and exclusion criteria

Studies were considered eligible if they met the following criteria:

- Evaluated adult populations with clinically defined periodontal outcomes
- Assessed dietary intake, dietary patterns, or lifestyle-related biochemical markers
- Reported quantitative periodontal parameters or clearly defined disease classifications
- Were published in peer-reviewed journals

Exclusion criteria included case reports, expert opinions without original data, and studies lacking standardized periodontal assessment. Narrative reviews were selectively included only when they provided a critical synthesis of emerging concepts, such as systemic

interactions or understudied nutrient–periodontium pathways, without duplicating evidence already captured by systematic analyses.

Dietary patterns as methodological constructs

Dietary patterns were operationalized using predefined indices or adherence scores, such as Mediterranean diet scores, plant-based diet quality indices, or pattern-derived classifications. Systematic syntheses comparing multiple dietary models were particularly valuable, as they enabled cross-pattern comparisons under a unified analytical framework. Meta-analytic evidence evaluating four major dietary patterns in relation to periodontal disease risk was used to illustrate the methodological strengths and limitations of pattern-based nutrition research, including exposure misclassification and residual confounding [25].

Randomized dietary intervention trials, such as those assessing the Mediterranean diet's impact on gingival inflammation, were emphasized because they minimize confounding and provide stronger causal inference. These trials typically employed controlled dietary counseling, predefined adherence monitoring, and standardized periodontal outcome measures, supporting their methodological robustness [21].

Lifestyle and systemic modifiers

Lifestyle factors were incorporated as contextual modifiers rather than isolated exposures. Physical activity, metabolic status, and inflammatory biomarkers were considered insofar as they interacted with dietary variables or influenced periodontal outcomes indirectly. Large observational datasets linking mineral status or nutrient biomarkers to periodontal disease were particularly useful for evaluating systemic pathways, even though they remain susceptible to reverse causality [22].

In addition, evidence exploring the intersection between periodontal disease and systemic conditions, including hematological parameters, was reviewed to contextualize dietary effects within broader inflammatory and metabolic networks. Such studies underscore that periodontal health cannot be fully interpreted in isolation from systemic physiology, reinforcing the integrative rationale of the review [23].

Trace elements and micronutrients: methodological considerations

Trace elements such as zinc were included due to their immunological relevance and measurable systemic biomarkers. Population-based analyses examining serum zinc levels in relation to periodontitis in smokers and non-smokers provided methodologically robust models for stratified analysis and confounder control, illustrating best practices in nutritional epidemiology applied to periodontal research [24]. These studies informed methodological considerations regarding subgroup analyses, lifestyle stratification, and interaction testing.

Data synthesis strategy

Rather than pooling effect sizes quantitatively, a thematic qualitative synthesis was employed. Findings were grouped according to exposure type (dietary patterns, fatty acids, minerals, trace elements) and biological mechanism (anti-inflammatory effects, bone metabolism, immune modulation). This strategy allowed coherent integration of evidence across diverse methodologies while avoiding misleading numerical aggregation in the presence of heterogeneity.

Methodological limitations

Across the included literature, common methodological limitations were acknowledged, including reliance on self-reported dietary data, cross-sectional designs, and variable periodontal case definitions. These limitations were consistently considered when

interpreting associations and formulating clinical implications, ensuring that conclusions remained proportionate to the strength of the underlying evidence [21-25].

Pathophysiological basis

Periodontitis represents the local manifestation of dysregulated chronic inflammation, in which the host immune response, rather than bacterial presence alone, drives periodontal tissue destruction. Within this framework, diet and lifestyle exert a direct influence on the systemic inflammatory threshold, with downstream effects on periodontal tissues. Current evidence supports the concept that diet quality modulates systemic inflammation, which in turn affects the severity and progression of periodontal disease [26].

Dietary patterns rich in ultra-processed foods, refined carbohydrates, and pro-inflammatory fats promote a state of chronic low-grade inflammation, potentially amplifying the local immune response to a dysbiotic biofilm. Conversely, anti-inflammatory dietary models, particularly the Mediterranean diet, are associated with reduced systemic inflammatory markers and improved control of gingival inflammation [27,28]. This relationship is consistently supported by meta-analyses, observational studies, and interventional trials demonstrating a favorable association between adherence to the Mediterranean diet and periodontal clinical parameters [29,30].

Table 1. Pathophysiological mechanisms linking diet, lifestyle, and periodontal disease

Factor	Biological mechanism	Systemic effect	Periodontal implication	References
Poor diet quality (ultra-processed foods, refined carbohydrates, pro-inflammatory fats)	Promotion of chronic low-grade inflammation	Increased circulating inflammatory mediators	Amplified host immune response to dysbiotic biofilm and enhanced tissue destruction	[26]
Mediterranean dietary pattern	Anti-inflammatory and antioxidant effects; modulation of lipid mediators	Reduction of systemic inflammatory markers	Improved gingival inflammation control and periodontal clinical parameters	[27,28]
High adherence to the mediterranean diet	Sustained immune modulation and improved metabolic profile	Lower inflammatory burden	Reduced prevalence and severity of periodontitis	[29]
Mediterranean diet–based intervention	Modification of oral and salivary microbiota composition	Reduced microbial inflammatory stimulus	Decreased levels of periodontopathogenic bacteria	[30]
Diet–microbiome interaction	Alteration of microbial ecology and immune signaling	Attenuation of persistent immune activation	Improved periodontal inflammatory status	[30]
Physical activity and active lifestyle	Regulation of systemic inflammation and metabolic homeostasis	Reduced insulin resistance and inflammatory load	Lower periodontal severity and slower disease progression	[31]

Table 1 presents the summary of key pathophysiological mechanisms through which dietary patterns and lifestyle factors influence periodontal inflammation and tissue destruction. The table highlights systemic inflammatory modulation, microbiome-related effects, and their downstream impact on periodontal clinical outcomes.

A central mechanistic pathway involves the interaction between diet, the microbiome, and host immunity. Dietary interventions can modify the composition of the oral and salivary microbiota, reducing the abundance of periodontopathogenic species and attenuating persistent immune stimulation [30]. Additionally, an active lifestyle contributes to the regulation of systemic inflammation and metabolic homeostasis, effects that translate into reduced periodontal severity and slower disease progression [31].

Dietary patterns and periodontal health

Dietary patterns represent complex exposures that integrate nutrient composition, food processing, and eating behaviors, exerting cumulative effects on systemic inflammation and periodontal susceptibility. Contemporary nutritional research increasingly favors pattern-based approaches over single-nutrient analyses, particularly in chronic inflammatory diseases such as periodontitis. Diet quality has been consistently associated with systemic inflammatory burden, suggesting that food choices can modulate host immune responses relevant to periodontal tissue breakdown [26].

Among the most extensively studied models, the Mediterranean diet has emerged as a reference anti-inflammatory dietary pattern. Characterized by high consumption of fruits, vegetables, whole grains, legumes, olive oil, and fish, this diet provides antioxidants, polyphenols, fiber, and omega-3 fatty acids that collectively downregulate inflammatory signaling. Randomized controlled trials demonstrate that adherence to the Mediterranean diet leads to significant reductions in gingival inflammation, even in the absence of changes in oral hygiene practices, supporting a direct host-modulatory effect [27]. These findings are reinforced by systematic reviews and meta-analyses showing that Mediterranean diet adherence is inversely associated with periodontitis prevalence and severity [28].

Observational evidence further supports these associations across diverse populations. Studies conducted in young adults and community-based cohorts report that higher adherence to the Mediterranean dietary pattern correlates with improved periodontal parameters, including reduced bleeding on probing and shallower probing depths [29]. Beyond clinical outcomes, interventional research indicates that Mediterranean diet–based interventions can alter the oral microbial ecosystem, reducing salivary levels of key periodontopathogens and thereby attenuating immune stimulation at the periodontal interface [30].

Dietary patterns should also be interpreted in conjunction with lifestyle behaviors. Cross-sectional analyses integrating dietary adherence and physical activity reveal that individuals who combine high-quality diets with higher levels of physical activity exhibit significantly lower periodontal disease severity. This suggests a synergistic interaction between nutritional anti-inflammatory effects and exercise-mediated metabolic regulation, rather than independent or additive influences [31-33]. Plant-based dietary patterns have gained attention for their potential anti-inflammatory benefits, largely attributed to high fiber intake, antioxidant density, and reduced consumption of saturated fats. However, emerging evidence emphasizes that diet quality within plant-based diets is critical. Diets rich in minimally processed plant foods are associated with lower periodontal risk, whereas poorly constructed plant-based diets high in refined carbohydrates may not confer similar benefits [33,34]. These findings align with broader nutritional epidemiology data linking diet quality to systemic inflammation and mortality risk [35].

Comparative analyses of vegan, vegetarian, and omnivorous diets suggest modest differences in oral health outcomes, with no consistent evidence of harm associated with well-

balanced plant-based diets. However, potential micronutrient inadequacies warrant consideration in periodontal risk assessment and dietary counseling [34]. Collectively, meta-analytic evidence comparing multiple dietary patterns confirms that anti-inflammatory, nutrient-dense diets are associated with reduced periodontal disease risk, while Western-style dietary patterns exert detrimental effects [25].

Specific dietary strategies and adjunctive nutritional interventions

Beyond global dietary patterns, specific dietary strategies and structured nutritional interventions have been investigated for their potential to modulate periodontal inflammation and clinical outcomes. Comparative analyses of vegan, vegetarian, and omnivorous diets indicate that overall oral health status is more strongly influenced by diet quality than by the exclusion of animal-derived foods per se. Well-balanced plant-based diets do not appear to adversely affect periodontal health; however, inadequate intake of key micronutrients may increase susceptibility if not appropriately managed [34]. These findings align with broader epidemiological data showing that high-quality diets, irrespective of dietary label, are associated with reduced systemic inflammation and improved long-term health outcomes, reinforcing their indirect relevance for periodontal disease modulation [35].

Table 2. Specific dietary strategies and adjunctive nutritional interventions in periodontal health

Dietary strategy	Key characteristics	Primary mechanism	Periodontal effect	References
Plant-based diets (vegan/vegetarian)	High intake of fruits, vegetables, fiber, and reduced animal products	Reduction of systemic inflammation dependent on diet quality	Neutral to beneficial effects on periodontal health if micronutrient intake is adequate	[34]
High-quality diets (independent of dietary label)	Balanced nutrient-dense food patterns	Lower systemic inflammatory burden	Indirect reduction of periodontal disease susceptibility	[35]
Dietary and nutraceutical adjuncts to periodontal therapy	Anti-inflammatory nutrients and bioactive compounds	Host modulation rather than antimicrobial action	Improved clinical periodontal outcomes when combined with mechanical therapy	[36]
Dietary modulation of oral–gut–brain axis	Microbiome-supportive dietary patterns	Maintenance of microbial homeostasis and immune regulation	Attenuation of systemic and periodontal inflammatory pathways	[37]
Low-carbohydrate and ketogenic diets	Carbohydrate restriction; high-fat intake	Improved glycemic control and metabolic regulation	Potential reduction in gingival inflammation; periodontal effects not fully established	[38,39]
Metabolically oriented low-carbohydrate interventions	Low carbohydrates with omega-3s, antioxidants, and fiber	Combined metabolic and anti-inflammatory effects	Improved gingival inflammation and periodontal indices in selected patients	[40]

Table 2 presents the overview of specific dietary strategies and adjunctive nutritional interventions investigated for their potential role in modulating periodontal inflammation and clinical outcomes, highlighting underlying metabolic and host-modulatory mechanisms.

Interventional research has increasingly explored dietary and nutraceutical approaches as adjuncts to non-surgical periodontal therapy. Systematic evidence indicates that dietary optimization, including increased intake of anti-inflammatory nutrients and bioactive compounds, can enhance clinical periodontal outcomes when combined with conventional

mechanical debridement. Such interventions act primarily through host modulation rather than direct antimicrobial effects, supporting their role as complementary, not substitutive, strategies [36].

Emerging mechanistic insights further expand the scope of dietary influence through the oral-gut-brain axis, highlighting how diet-induced changes in gut microbiota may interact with periodontal inflammation and systemic immune regulation. Experimental models suggest that periodontitis-associated dysbiosis can extend beyond the oral cavity, potentially contributing to systemic and neuroinflammatory pathways, thereby reinforcing the importance of dietary interventions that support microbial homeostasis [37].

Low-carbohydrate and ketogenic diets have attracted interest due to their effects on glycemic control and systemic inflammation. While ketogenic diets demonstrate efficacy in improving metabolic parameters, particularly in diabetic populations, their implications for periodontal health remain incompletely defined [38,39]. Pilot and controlled studies suggest that carbohydrate restriction combined with increased intake of omega-3 fatty acids, antioxidants, and fiber may lead to improvements in gingival inflammation and clinical periodontal indices, supporting a metabolically oriented dietary approach in selected patients [40].

Lifestyle factors and systemic interactions influencing periodontal health

Lifestyle-related factors interact closely with dietary exposures in shaping periodontal inflammation and disease expression. Controlled clinical evidence indicates that dietary interventions low in refined carbohydrates and enriched in omega-3 fatty acids, antioxidants, and fiber can lead to significant improvements in gingival inflammation and clinical periodontal indices, supporting the concept that metabolic control and anti-inflammatory nutrition act synergistically at the periodontal level [40]. These findings emphasize that lifestyle-driven metabolic status, particularly glucose homeostasis, is a critical modifier of periodontal inflammatory burden.

Ketogenic and low-carbohydrate dietary models have been investigated primarily in the context of metabolic disease. While ketogenic diets demonstrate consistent benefits in glycemic control and insulin sensitivity, especially in diabetic populations, their periodontal implications appear to be indirect and mediated through systemic metabolic improvements rather than direct effects on periodontal tissues [38,39]. Pilot oral health studies suggest that reductions in carbohydrate intake may decrease gingival inflammation, but the long-term periodontal safety and sustainability of strict ketogenic regimens remain insufficiently defined [41,42].

Beyond metabolic regulation, emerging research highlights the importance of systemic microbial interactions, particularly the oral-gut axis, in periodontal pathophysiology. Periodontal disease has been shown to influence gut microbial composition, potentially through translocation of oral pathogens and inflammatory mediators, thereby contributing to systemic immune dysregulation [44]. Clinical and experimental data indicate that alterations in gut microbiota composition are associated with changes in intestinal barrier function and mucosal immunity, processes that may amplify systemic inflammation and indirectly exacerbate periodontal tissue destruction [45].

These bidirectional interactions reinforce the notion that periodontal disease is embedded within a broader network of lifestyle-dependent systemic processes. Dietary habits, metabolic control, and microbial homeostasis collectively shape host immune responses,

influencing both local periodontal outcomes and extraoral inflammatory pathways [41-45]. Consequently, lifestyle optimization, encompassing balanced nutrition, metabolic health, and systemic inflammatory regulation, should be considered an integral component of comprehensive periodontal prevention and management strategies, rather than an adjunctive or secondary consideration.

Oral-gut axis and systemic implications in periodontal disease

Increasing evidence positions periodontitis within a bidirectional oral-gut axis, in which oral dysbiosis, systemic inflammation, and gut microbiota alterations interact to influence disease expression beyond the oral cavity. Conceptual analyses of ketogenic and low-carbohydrate dietary models emphasize that profound metabolic and microbial shifts induced by diet may indirectly affect periodontal inflammation through systemic immune modulation, rather than through direct effects on the periodontium itself [42]. These observations underscore the relevance of considering distal microbial ecosystems when evaluating dietary interventions in periodontal patients.

Experimental and translational studies have demonstrated that periodontal inflammation can induce gut dysbiosis, linking oral microbial imbalance to intestinal microbial alterations and systemic immune activation. In particular, experimental models of periodontitis reveal associations between oral inflammation, gut microbiota disruption, and neuroinflammatory pathways, supporting a broader oral-gut-brain axis framework [37]. Such findings expand the pathophysiological relevance of periodontitis beyond localized tissue destruction, implicating systemic inflammatory networks.

Clinical and mechanistic investigations further substantiate the oral-gut connection. Periodontal disease has been associated with gastrointestinal disorders, suggesting that translocation of oral bacteria and inflammatory mediators may contribute to intestinal mucosal inflammation and barrier dysfunction [44]. Studies analyzing colonic mucosa-associated microbiota demonstrate that poor oral health correlates with altered gut microbial profiles, reinforcing the concept that oral dysbiosis can influence distal microbial niches [45].

At a mechanistic level, molecular and animal studies provide compelling evidence that oral pathogens can directly reshape gut microbiota composition. Periodontitis has been shown to induce gut dysbiosis through salivary microbiota, altering intestinal microbial balance and immune signaling [47]. Specific pathogens, such as *Porphyromonas gingivalis*, have been implicated in promoting intestinal inflammation by disrupting epithelial barrier integrity and modulating $CD4^+$ T-cell responses, particularly IL-9-producing subsets, thereby linking periodontal infection to systemic immune dysregulation [48].

Collectively, these findings support a systems-based model in which periodontal disease is both a contributor to and a consequence of systemic microbial and inflammatory alterations. Dietary patterns and lifestyle factors that promote microbial homeostasis and barrier integrity may therefore exert periodontal benefits not only locally but also through modulation of the oral-gut axis. Integrating this perspective into periodontal research and clinical practice highlights the importance of comprehensive lifestyle-based strategies aimed at restoring immune and microbial equilibrium across interconnected biological compartments [42-48].

Clinical implications

Current evidence supports diet and lifestyle optimization as adjuncts to conventional periodontal therapy, rather than standalone interventions. Anti-inflammatory dietary patterns, adequate micronutrient intake, and metabolically favorable lifestyles can reduce systemic inflammatory burden and modulate host immune responses, thereby enhancing periodontal tissue stability and improving response to non-surgical therapy [26-36]. From a clinical perspective, this underscores the importance of addressing modifiable risk factors beyond plaque control, particularly in patients with recurrent periodontitis, slow healing, or systemic comorbidities.

Personalized nutritional counseling represents a key translational step. Rather than promoting restrictive or trend-based diets, clinicians should focus on overall diet quality, inflammatory potential, and patient-specific risks, including metabolic status, smoking, and nutritional deficiencies. Tailored counseling allows realistic dietary modifications that improve adherence and long-term sustainability, while minimizing the risk of micronutrient imbalance observed in poorly planned restrictive diets [34,35]. Importantly, dietary advice should be framed within evidence-based periodontal goals, reinforcing its role as supportive host modulation.

Integration of dietary and lifestyle assessment into preventive and maintenance protocols is increasingly justified. Periodontal maintenance visits offer structured opportunities to reinforce nutritional guidance, monitor lifestyle behaviors, and identify patients who may benefit from interdisciplinary referral. This approach aligns with a preventive, patient-centered model of periodontal care and reflects the growing recognition of periodontitis as a disease influenced by systemic inflammatory and metabolic context [31,36].

Limitations of current evidence

Despite growing interest, the current body of evidence is constrained by several methodological limitations. A major challenge is the heterogeneity of study designs, including variability in dietary assessment tools, periodontal case definitions, and outcome measures, which limits direct comparison across studies [25-35].

Observational data are predominant, particularly cross-sectional analyses, which preclude robust causal inference. While such studies are valuable for hypothesis generation and population-level insights, they remain susceptible to reverse causality and residual confounding. Confounding lifestyle variables, such as smoking, physical activity, socioeconomic status, and access to dental care, are difficult to fully control and may partially explain observed associations between diet and periodontal outcomes. These factors necessitate cautious interpretation of results and conservative translation into clinical recommendations [26-35].

Future research directions

Future investigations should prioritize long-term interventional trials with adequate sample sizes, standardized periodontal outcomes, and rigorous dietary adherence monitoring. Such studies are essential to clarify causality and quantify the true adjunctive benefit of dietary modification. There is also a need for multimodal lifestyle interventions that simultaneously address diet, physical activity, and metabolic control, reflecting real-world patient behavior and the multifactorial nature of periodontal disease [31,36]. Finally, microbiome-targeted dietary strategies represent a promising frontier. Advances in oral and gut microbiome research suggest that diet-induced microbial modulation may play a central role in periodontal inflammation and

systemic interactions. Integrating microbiome endpoints into future clinical trials will be critical for advancing precision periodontal medicine [37-48].

Conclusions

Periodontal disease emerges from a complex interplay between microbial dysbiosis, host immune response, and modifiable systemic factors. The evidence synthesized in this review indicates that dietary patterns and lifestyle behaviors substantially influence periodontal inflammation and tissue resilience by modulating systemic inflammatory tone, oxidative stress, and immune regulation. Anti-inflammatory, nutrient-dense diets and metabolically favorable lifestyles consistently align with improved periodontal parameters, supporting their relevance as adjunctive determinants of periodontal health.

Beyond isolated nutrients, overall diet quality and lifestyle context appear more influential in shaping periodontal outcomes than single dietary components. Mediterranean and high-quality plant-based dietary patterns, together with adequate intake of key micronutrients and omega-3 fatty acids, demonstrate coherent biological plausibility and clinical associations with reduced periodontal disease severity. Importantly, these effects are mediated through host modulation rather than direct antimicrobial action, reinforcing the paradigm that periodontal therapy should extend beyond mechanical biofilm control to address systemic inflammatory drivers and patient-specific risk profiles.

From a translational perspective, integrating dietary and lifestyle considerations into periodontal care reflects a shift toward systems-based and personalized management strategies. Periodontitis should be conceptualized within interconnected metabolic, inflammatory, and microbial networks, including the oral-gut axis. While current evidence is limited by heterogeneity and a predominance of observational designs, it provides a strong foundation for multidisciplinary preventive approaches. Future research integrating long-term interventions, microbiome analyses, and personalized nutritional strategies will be essential to refine evidence-based recommendations and fully harness the therapeutic potential of lifestyle optimization in periodontal medicine.

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