Rheumatoid arthritis and periodontitis: shared mechanisms and integrated care

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Abstract

Sharing inflammatory pathways, microbial dysbiosis, and systemic immune responses are the foundations of the considerable reciprocal link between rheumatoid arthritis (RA) and periodontitis, two chronic inflammatory illnesses. Both conditions significantly impact patients' quality of life: periodontitis causes gradual loss of oral tissue and tooth loss, while RA causes systemic autoimmune inflammation and joint deterioration. There is growing evidence that these standard inflammatory processes facilitate treatment synergy and worsen the disease. In addition to disease-modifying anti-rheumatic medications (DMARDs), non-surgical periodontal therapies (NSPT) have demonstrated promise in lowering systemic inflammation and alleviating RA symptoms. Additionally, the function of microbial dysbiosis, specifically Porphyromonas gingivalis, emphasizes how crucial it is to address systemic and local inflammation to treat these disorders adequately. The importance of multidisciplinary treatment solutions that combine dental and medical practices is emphasized in this paper. The dual burden of these diseases can be fully addressed by developing therapeutic paradigms and encouraging cooperation between periodontists and rheumatologists. Future research should concentrate on patient-centered care models, novel therapies, and customized treatment techniques to maximize results and enhance the general quality of life for those impacted.

Keywords: Rheumatoid Arthritis; Periodontitis; Microbial Dysbiosis; Non-Surgical Periodontal Treatment; Disease-Modifying Anti-Rheumatic Drugs; Curcumin-Based

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Formulations; Photodynamic Therapy; Probiotics; Systemic Inflammation; Biomimetic Scaffolds

Introduction

The systemic autoimmune disease known as rheumatoid arthritis (RA) mainly damages synovial membranes, resulting in stiffness, joint discomfort, persistent inflammation, and eventually deformity (1–9). With a global frequency of about 1%, RA presents serious problems for both public health and quality of life (10–13). In addition to joint pathology, it is linked to systemic signs such as osteoporosis, cardiovascular issues, and increased vulnerability to infections (14–18). These systemic effects highlight RA's broad health implications, which go beyond musculoskeletal symptoms (19–25). Because of the chronic inflammatory character of the condition, therapy must be comprehensive and consider both systemic and local effects (24,26–30).

In contrast, 62% of individuals worldwide suffer from periodontitis, a common mouth disease. It is characterized by a gradual breakdown of the alveolar bone and periodontal ligament, which frequently leads to tooth loss and has been connected to systemic diseases like RA, diabetes, and cardiovascular disorders (28–32). A dysbiotic oral microbiome and a dysregulated host immunological response are the main causes of periodontitis, which leads to tissue damage and chronic inflammation (31–37). In addition to its high incidence, it is linked to other systemic inflammatory illnesses, making it a serious public health concern (38–43).

Although the main targets of RA and periodontitis differ, they both have inflammatory cascades that are remarkably similar and are primarily mediated by cytokines, including IL-1β, IL-6, TNF-α, and IL-17 (44–48). Because they promote tissue death, bone resorption, and persistent inflammation, these cytokines are essential to the pathological processes of both disorders (42–44,49–51). Researchers have looked at the interaction between RA and periodontitis because of the overlap in these pathways, and they have found a complicated reciprocal link (52–57). Severe periodontitis is more common in RA patients, according to recent research, and persistent periodontitis may raise the likelihood and severity of RA (58,58–61). Standard pathogenic processes, such as systemic inflammation, microbial dysbiosis, and genetic predispositions like HLA-DRB1 alleles, are assumed to be the source of this interaction (62–65). Additionally, environmental factors, most notably smoking, exacerbate this link by intensifying inflammatory responses and making both illnesses more severe (66–70).

The recognition of this interaction highlights the significance of interdisciplinary approaches to care. To address this dual burden, better comprehending their interactions and coordinated therapy approaches considering systemic and local inflammatory processes are necessary (71–74). Additionally, halting the advancement of the disease may be significantly aided by early therapies that focus on modifiable risk factors like smoking cessation, dental hygiene, and dietary changes (75–81). This emphasizes the need for holistic disease management in treating chronic inflammatory disorders, urges cooperation between dental and medical experts to provide comprehensive care, and enhances patient outcomes (82–84). Dysbiosis in periodontal pockets creates an environment conducive to the proliferation of pathogenic bacteria such as Porphyromonas gingivalis (Pg), Tannerella forsythia (Tf), and Treponema denticola (Td), collectively known as the "red complex" (Figure 1) (85–89).

Shared Pathophysiological Mechanisms

Cytokine Dysregulation

A delicate imbalance between pro-inflammatory and anti-inflammatory cytokines is the root cause of both periodontitis and RA (90,90–96). Key cytokines such as IL-6, TNF-α, IL-1β, and IL-17 are elevated and are crucial in mediating the inflammatory cascades that define both disorders (97–102). These cytokines coordinate synovial inflammation in RA, which leads to progressive bone erosion, joint swelling, and cartilage degeneration (102–106). Likewise, in the case of periodontitis, these pro-inflammatory mediators prolong chronic inflammation, promote alveolar bone resorption, and worsen the breakdown of periodontal tissues (107–110). Interestingly, they have extensive systemic effects that contribute to increased immune activation and a vicious cycle of inflammation that connects the two illnesses (111–114). Chronic bloodstream cytokine release might intensify systemic immune responses, potentially escalating pre-existing inflammatory diseases or initiating new autoimmune pathways (114–118). This systemic diffusion highlights the importance of adequately targeting cytokine control to manage both diseases (87,119–123).

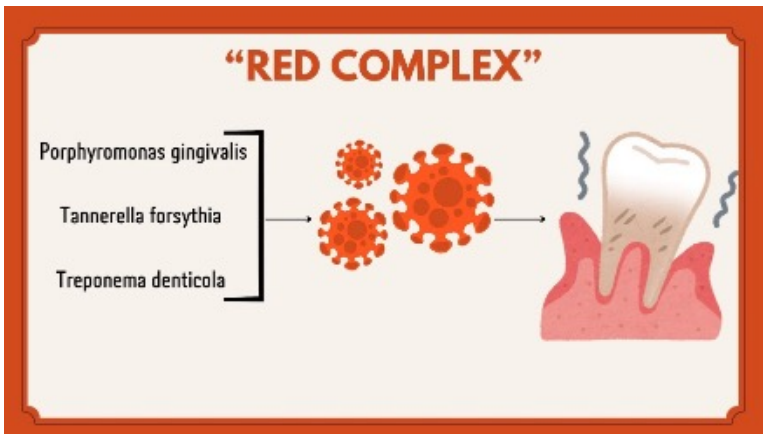

Figure 1. Pathogenic bacteria such as P*orphyromonas gingivalis (Pg*), T*annerella forsythia (Tf)*, and *Treponema denticola (Td)*, collectively known as the "*red complex*."

Microbial Dysbiosis and Citrullination

Porphyromonas gingivalis (Pg), an oral pathogen, has become a crucial link between periodontitis and RA because of its distinct pathogenic processes (124–127). The enzyme catalyzes protein citrullination and peptidyl arginine deiminase, which Pg can produce (128–134). Neoantigens produced by this process are identified by autoantibodies specific to RA, including anti-citrullinated protein antibodies (ACPAs) (135–141). These autoantibodies indicate the severity and course of the disease and are a diagnostic tool for RA (136–138,142–144). The function of Pg is further demonstrated by the discovery of its DNA in RA patients' synovial fluid, which offers strong proof of microbial translocation and its impact on systemic autoimmune (136–139,145). A possible mechanism by which periodontitis can function as a risk factor for the development of RA is established by Pg-induced dysbiosis, a disturbed balance of the oral microbiota that sets off systemic inflammatory responses (90,146–153). Additionally, Pg is a key player in the pathogenic nexus between RA and periodontitis because of its capacity to elude host immune systems, which enables it to sustain chronic inflammation (128,154–159).

Immune Cross-Reactivity

There is a notable overlap between the immune responses in RA and periodontitis, especially when generating autoantibodies such as rheumatoid factors (RF) and ACPAs (130,160–166). Although these autoantibodies are essential to the pathophysiology of RA, they are also high in those with periodontitis, indicating that the two diseases have a similar immunological foundation (133,135–137,167–173). Those who are genetically susceptible may experience an increased autoimmune reaction due to chronic periodontal inflammation that sensitizes the immune system (174). Molecular mimicry, in which antigens from periodontal pathogens trigger immune responses that cross-react with host tissues, maybe the mechanism underlying this immune cross-reactivity (104). Further connecting these disorders is the systemic spread of inflammatory mediators and oral infections, which raises immune activation (175). These interrelated immune pathways highlight how therapeutic approaches targeting one ailment may assist the other (176,177). For example, RA medications may lessen periodontal tissue damage, while treatments that target periodontal inflammation may reduce systemic immune dysregulation (178). To address the intricate interactions between RA and periodontitis, these findings highlight the significance of a cohesive and integrated therapeutic approach (63). The similar pathophysiological pathways between RA and periodontitis highlight a complex interaction between cytokine dysregulation, microbial dysbiosis, and immunological cross-reactivity (179). Clinicians can improve patient outcomes by managing these interrelated disorders with focused, interdisciplinary methods if they know these pathways better (105).

Therapeutic Implications

Non-Surgical Periodontal Treatment (NSPT)

The foundation of periodontal therapy is Non-Surgical Periodontal Treatment (NSPT), which includes scaling and root planing (SRP) (177,178,180–184). Its ability to minimize periodontal pocket depth, reduce microbial load, and reduce inflammation is well known (176,181,185). In addition to these local effects, NSPT has systemic benefits, especially in treating rheumatoid arthritis (RA) (177,182–184). NSPT helps to lower systemic inflammatory markers and overall disease activity by treating periodontal inflammation (175,186–188). This dual effect emphasizes how important it is for preserving oral health and affecting more general systemic diseases (189,190). In the context of managing RA, clinical trials consistently show several noteworthy effects of NSPT, such as:

- Notable drops in the Disease Activity Score 28 (DAS28): research has demonstrated that regular periodontal therapy improves this composite metric, which is used to assess RA activity and Reflects a wider systemic benefit (85,86,191–194).
- Decreased levels of systemic inflammatory markers: Two critical indicators of inflammation, erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP), are linked to NSPT (195–199).
- ower autoantibody titers: Anti-citrullinated protein antibodies (ACPAs) and rheumatoid factor (RF), two autoantibodies associated with the severity and course of RA, can be inhibited by periodontal treatment (200–205).

The long-term advantages of NSPT highlight how crucial routine periodontal care is to managing RA and periodontal disease (206,207). This integrative method demonstrates how treating a modifiable risk factor, such as periodontal disease, can enhance patient outcomes (208). Clinicians may improve their patients' systemic health and disease control by integrating periodontal therapy into RA care strategies (209). NSPT significantly impacts systemic inflammation reduction and its direct effects on dental health. It is, therefore, positioned as a valuable supplement to traditional RA treatments. Improved methods have increased the potential advantages of NSPT even more (210). Examples include supportive periodontal treatment, the supplementary use of antibiotics, and minimally invasive SRP (211–214). Because of these cutting-edge methods, which enhance treatment effectiveness and yield long-lasting effects, NSPT is a crucial part of comprehensive care for patients with periodontitis and RA (215–218).

Disease-Modifying Anti-Rheumatic Drugs (DMARDs)

Modifying Diseases Anti-rheumatic drugs (DMARDs) are the mainstay of RA treatment since they prevent joint degeneration and address systemic inflammation (219–222). Recent data raises the possibility that these medications may also improve periodontal health, offering patients who treat both illnesses a double benefit (223,224). Several important DMARDs and their effects on periodontal health include:

- Methotrexate (MTX): This often prescribed first-line treatment for RA improves periodontal metrics and lowers systemic inflammation. According to studies, MTX can lessen the damage to periodontal tissue, mainly when used alone (225). Its ability to effectively treat RA and periodontal disease highlights how systemic and local inflammatory management is interdependent (226).
- Anti-TNF medications (such as adalimumab and in-

fliximab): TNF-α, a crucial cytokine in both RA and periodontitis, is the target of these biological treatments (209). Through the inhibition of TNF-α, these medicines successfully decrease bleeding on probing, clinical attachment loss, and probing depths in periodontal tissues. Both illnesses can be effectively managed by addressing inflammation at its root cause (161).

- Tocilizumab: An IL-6 receptor antagonist, tocilizumab has shown notable effectiveness in lowering clinical attachment levels and periodontal inflammation (227,228). These results demonstrate its dual function of supporting oral and systemic health, making it a potentially useful component of integrated treatment plans (227).

DMARDs significantly benefit RA patients with periodontitis by regulating inflammatory pathways systemically. However, because these medications inhibit the immune system, they must be closely monitored for infections and side effects. Ongoing research aims to balance localized periodontal benefits with systemic efficacy to optimize medication regimens. Thanks to ongoing research into customized interventions that reduce dangers while improving therapeutic results, future treatments may be more accurate and successful (Figure 2).

Adjuvant Therapies

The possibility of emerging adjuvant medicines to supplement traditional care in managing periodontitis and RA is drawing attention (41,42,229–235). These cutting-edge methods improve treatment results using cutting-edge technologies and unique biological mechanisms (189,236–241). Notable instances consist of:

- Curcumin-Based Formulations: Curcumin, a naturally occurring substance with strong antibacterial and anti-inflammatory qualities, has demonstrated potential for improving periodontal health. It may offer further advantages by lowering inflammation and promoting tissue healing in periodontal therapy regimens (242,243).
- Photodynamic Therapy (PDT): This method uses light-activated chemicals to target inflammatory mediators and microbial biofilms. It has shown promise in enhancing periodontal health and could be a valuable supplement to NSPT and DMARDs (225,244–252).
- Probiotics: One new tactic is using probiotics to help the oral cavity's microbiota return to equilibrium. By modifying the microbiome, probiotics may lessen harmful bacteria and promote general periodontal health (227,253–259).
- Low-Level Laser Therapy (LLLT): LLLT has demonstrated promise in lowering inflammation and encouraging tissue regeneration. This strategy offers a viable way to improve clinical results and promote periodontal healing (228,260–266).
- Furthermore; cutting-edge technologies are being developed to alter how RA and periodontitis are treated completely. Among these are:
- Personalized Pharmacological Agents: Drugs customized for each patient's unique profile are meant to maximize effectiveness and reduce side effects (263,267–271).
- Biomimetic Scaffolds: Developed for tissue healing, these scaffolds promote periodontal structure regeneration and have demonstrated promise in preclinical research (272–276).

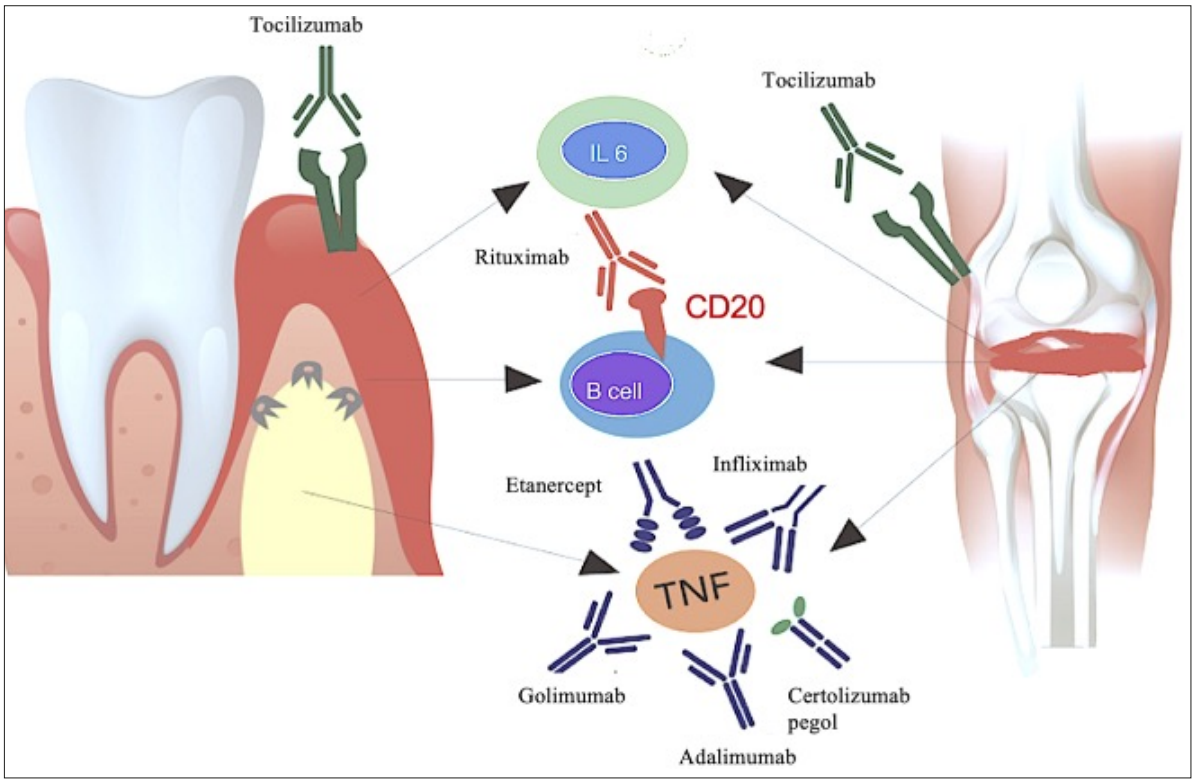

Figure 2. The bidirectional effect of biological disease-modifying anti-rheumatic drugs (bDMARDSs) on common therapeutic targets of RA and periodontitis. (IL-6: interleukine-6; TNF: tumor necrosis factor)

Table 1. Comparative Effects of Interventions

Intervention	Benefits in RA	Benefits in Periodontitis
NSPT	Reduced DAS28, CRP, ACPAs	Decreased probing depth, inflammation
Methotrexate	Improved RA symptomatology	Modulation of periodontal inflammation
Anti-TNF Agents	Decreased joint and periodontal damage	Reduced microbial burden
Curcumin Therapy	Enhanced systemic anti-inflammatory effects	Adjunctive control of biofilms

- Treatments involving cytokine modulation: These medicines target specific cytokines implicated in periodontitis and RA and have the potential to provide more accurate inflammatory control (277–282).

Incorporating these novel approaches into current treatment plans may have synergistic advantages that enhance patient outcomes even more (277,283–285). Clinicians who combine cutting-edge treatments with more conventional methods like NSPT and DMARDs might be better able to handle the intricate relationship between RA and periodontitis (Table 1).

Challenges and Future Directions

Research Gaps

Despite mounting evidence, current research is limited by small sample sizes, inconsistent study designs, and brief follow-up periods (286). Intense longitudinal research is required to clarify long-term results and improve treatment regimens (287). Furthermore, it would be easier to compare data if methods for evaluating how periodontal therapy affects RA activity were standardized (288). Future studies ought to investigate the molecular processes that underlie the reciprocal link, providing insight into how systemic treatments impact oral health and vice versa (228). Working together across disciplines is crucial to producing high-quality data that can direct clinical practice (178,227,289).

Clinical Integration

Rheumatologists and periodontists must collaborate interdisciplinaryly to close the gap between systemic and dental health care (290,290,291). RA management guidelines should include routine periodontal exams (72,292–297)to detect and treat periodontitis early on. Improving adherence to integrated treatment techniques requires educating patients and healthcare professionals about how these illnesses are interconnected (298). Structured care pathways that include routine screens and follow-ups (50)can guarantee better health outcomes and prompt treatments. Long-term advantages and adherence can be further improved by incorporating patient-centered treatment approaches that prioritize education and collaborative decision-making.

Emerging Technologies

Developments in precision medicine, biomarker identification, and microbiome-targeted treatments promise individualized treatment plans for periodontitis and RA (202). For instance, oral dysbiosis probiotics and prebiotics may be used in conjunction with traditional therapies, and new biomarkers may make it possible to detect and track the course of the disease early (113). Customizing therapies to each patient's unique profile and incorporating artificial intelligence (AI) into predictive modeling may improve treatment approaches. Innovations like AI-driven diagnostic tools and wearable biosensors for real-time inflammatory monitoring could revolutionize how doctors treat these chronic disorders by offering previously unheard-of levels of precision in disease management (138,299,300).

Conclusion

The necessity of comprehensive treatment techniques that treat both disorders at the same time is highlighted by the interaction between RA and periodontitis. Since periodontal health is a modifiable component that can have a substantial impact on the outcomes of RA, managing it is essential to providing comprehensive care. By combining periodontal therapy with systemic treatment approaches, healthcare professionals can improve overall patient quality of life, lower disease burden, and better regulate inflammation. For integrated illness management to reach its full potential, future research should concentrate on long-term studies, novel treatments, and interdisciplinary care models. To achieve patient-centered results, close care gaps, and establish new benchmarks for managing chronic inflammatory disorders, it will also be essential to promote collaboration between the medical and dentistry fields.

Abbreviations

ACPAs: Anti-Citrullinated Protein Antibodies
AI: Artificial Intelligence
CRP: C-Reactive Protein
DAS28: Disease Activity Score 28
DMARDs: Disease-Modifying Anti-Rheumatic Drugs
ESR: Erythrocyte Sedimentation Rate
HLA-DRB1: Human Leukocyte Antigen DRB1 (genetic allele associated with RA)
IL-1β: Interleukin-1 beta
IL-6: Interleukin-6
IL-17: Interleukin-17
LLLT: Low-Level Laser Therapy
MTX: Methotrexate
NSPT: Non-Surgical Periodontal Treatment
Pg: Porphyromonas gingivalis
PDT: Photodynamic Therapy
RA: Rheumatoid Arthritis
RF: Rheumatoid Factor
Td: Treponema denticola
Tf: Tannerella forsythia
TNF-α: Tumor Necrosis Factor-alpha

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Conflicts of Interest

The authors declare no conflicts of interest.

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