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Review Article

Rosemary as an Adjunctive Therapy for Periodontitis: A Minireview

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Abstract

Periodontitis is a chronic, multifactorial inflammatory disease in which dysbiotic subgingival biofilms drive gradual loss of the structures that anchor the teeth. While non-surgical periodontal therapy (NSPT) remains the gold-standard initial approach for reducing microbial load and inflammation, mechanical debridement alone has limitations in deep periodontal pockets and inaccessible root concavities. Consequently, adjunctive local drug delivery (LDD) systems have been increasingly explored to target periodontal pathogens directly, achieve higher local concentrations, and minimize systemic side effects. In recent years, natural herbal alternatives such as Rosmarinus officinalis (rosemary) have gained considerable scientific interest due to their potent antimicrobial, antioxidant, and anti-inflammatory properties, favorable safety profiles, and high patient acceptability. The aim of this mini-review is to highlight recent studies demonstrating the therapeutic potential of Rosmarinus officinalis as an adjunct to NSPT in the management of periodontitis.

Keywords: Rosemary; Periodontitis; NSPT; Herbal Therapy

Abbreviations

NSPT: Non-surgical Periodontal Therapy; LDD: Local Drug Delivery; TNF-α: Tumor Necrosis Factor Alpha; IL: Interleukin; MIC: Minimum Inhibitory Concentration; MBC: Minimum Bactericidal Concentration; TLR: Toll-Like Receptor; LPS: Lipopolysaccharide; MMPs: Matrix Metalloproteinases.

Introduction

Periodontitis is a chronic inflammatory disorder with multiple causes, linked to dysbiotic plaque biofilms. It gradually destroys the teeth's supporting apparatus, namely the periodontal ligament and alveolar bone. Clinical signs include periodontal pockets, bleeding gums, loss of clinical attachment, and bone loss on radiographs,

and untreated disease may end in tooth loss. Its high prevalence and its toll on chewing, appearance, and quality of life make it a major public health concern [1].

The 2017 classification merged the former "chronic" and "aggressive" categories into a single diagnosis of "periodontitis," characterized by staging (Stage I to IV, reflecting severity and complexity) and grading (Grade A to C, reflecting progression rate and risk factors such as smoking and diabetes) [1].

Organized communities of subgingival bacteria trigger an exaggerated immune-inflammatory response in the host, which breaks down connective tissue and resorbs bone. NSPT is the gold standard step of treatment, yet instruments cannot reach every surface in deep pockets and anatomically complex areas, so highly virulent pathogens frequently persist [2,3]. Systemic antibiotics and conventional LDD systems can improve results but may cause adverse reactions, hypersensitivity, and bacterial resistance, prompting a search for safer, biocompatible, lower-cost herbal options [4,5]. Plant extracts have a long traditional use with few reported adverse effects, but only a small number have been rigorously tested in periodontal therapy [6]. Agents investigated so far include Aloe vera, Neem, Green tea, Tea tree oil, Lemongrass, Curcumin, Coriander, Oak, Bakul, and Babul [7].

Rosemary (*Rosmarinus officinalis*) has drawn particular interest because its phenolic constituents, notably carnosic acid, rosmarinic acid, and carnosol, provide antioxidant, anti-inflammatory, and antimicrobial activity [8,9]. Preclinical and *in vitro* work report activity against major periodontal pathogens and lower levels of inflammatory biomarkers, pointing to its potential in managing periodontitis [10,11].

Etiopathogenesis and microbial dysbiosis in periodontitis

Periodontitis arises from complex interactions among microbial, host, environmental, and genetic factors. Modifiable risk factors include smoking, poorly controlled diabetes, poor oral hygiene, stress, and obesity, while genetic predisposition, age, and sex influence severity and treatment response [12,13].

Models of microbial etiology have evolved from the non-specific and specific plaque hypotheses to the ecological plaque hypothesis, and more recently to the Keystone-Pathogen and Polymicrobial Synergy and Dysbiosis (PSD) models, which hold that certain organisms, particularly *Porphyromonas gingivalis (P. gingivalis)*, can disrupt host immune regulation and drive dysbiosis even at low abundance [2,14]. Meyle and Chapple (2015) described progression as a shift from symbiosis to dysbiosis in which persistent biofilm and inflammation create a self-sustaining cycle [15], and the IMPEDE model similarly emphasizes a bidirectional relationship between inflammation and dysbiosis [16]. Together, these models show that tissue destruction results not from infection alone but from a dysregulated host immune-inflammatory response to a dysbiotic biofilm, leading to connective tissue degradation and alveolar bone resorption [17].

Periodontitis-associated plaque differs from healthy plaque, commonly harboring the red complex bacteria (*P. gingivalis*, *T. forsythia*, *T. denticola*) [18], and the combination of *A. actinomycetemcomitans*, *P. gingivalis*, and *P. intermedia* shows the strongest link to disease [19,20]. Pathogen groups reshape their environment to persist in biofilms, producing the dysbiosis thought to drive the shift from health to disease [21]. *P. gingivalis*, a keystone pathogen, alters the local environment and host response through its fimbriae, which promote attachment and activate TLR2, its LPS, which increases invasiveness and activates TLR4, its gingipains, which dysregulate immunity and degrade fibrinogen and fibrin, and its protective capsule [22]. *A. actinomycetemcomitans*, once considered the main cause of aggressive periodontitis (now Grade C periodontitis), is an early colonizer that evades mucosal immunity through virulence factors such as leukotoxin and adhesins [1,23]. Its LPS stimulates macrophages to produce IL-1 and TNF [24], and the resulting cytokines, together with MMPs and RANK-L, drive attachment loss, collagen degradation, and osteoclast-mediated alveolar bone destruction [18].

Periodontal therapy

Newman., *et al.* (2018) describe five phases of periodontal therapy: preliminary (managing emergencies and extracting hopeless teeth), nonsurgical (phase I), surgical (phase II), restorative (phase III), and supportive periodontal care (phase IV), with re-evaluation after each treatment phase [25].

Phase I, NSPT, aims to control infection and inflammation through mechanical removal of supragingival and subgingival biofilm, patient involvement, and risk factor management [26]. It combines root debridement with chemotherapeutic aids such as

antiseptics, local drug delivery, or systemic antibiotics [27], and host modulation drugs can be added to regulate the inflammatory response [28]. Success is judged by reduced probing depth and gained clinical attachment, and patients who do not reach stability (no pockets over 4 mm with bleeding on probing) move to phase II, while the rest enter supportive care [29].

Phase II addresses poorly responding sites, such as intra-bony and furcation lesions, by repeated instrumentation or access flap, resective, or regenerative surgery, although these endpoints may not be achievable for all teeth in stage III and IV periodontitis [30]. Supportive periodontal care then maintains stability at risk-based intervals through preventive and therapeutic measures, retreatment for recurrence, and good oral hygiene and a healthy lifestyle [31].

Adjunctive aids in periodontal therapy

Mechanical debridement alone cannot fully eliminate bacteria in deep pockets (>5 mm), root concavities, and furcation defects, and bacteria can invade periodontal tissues, so adjunctive antimicrobials have been explored [32]. Systemic antibiotics studied for this purpose include amoxicillin (with or without clavulanic acid), azithromycin, clindamycin, doxycycline, metronidazole, spiramycin, and tetracycline [33]. To limit systemic exposure, LDD systems were developed to deliver tetracycline, minocycline, doxycycline, metronidazole, or chlorhexidine directly into periodontal pockets as fibers, gels, chips, or microspheres [7,34].

Systemic antibiotics, however, carry risks of allergic or anaphylactic reactions, superinfection by opportunistic bacteria, antibiotic resistance, drug interactions, and gastrointestinal effects. They also require high doses to reach adequate concentrations in gingival fluid, and cost and poor patient compliance further limit their use [35]. Therefore, herbal agents represent a reasonable alternative to investigate as adjuncts to NSPT.

Herbal therapy in periodontics

Many herbal agents are now used in LDD systems for periodontitis because of their anti-inflammatory, immunomodulatory, antimicrobial, and antioxidant properties [7]. A literature review identified several plants, including Aloe vera, Calendula officinalis, Magnolia officinalis, Rosmarinus officinalis, Cinnamomum zeylanicum, and propolis, that reduced plaque, controlled gingival bleeding, and relieved inflammation [6]. Agents commonly used for local delivery include neem, aloe vera, lemongrass, green tea, tea tree oil, curcumin, oak, coriander, babul, and bakul [7].

Among these, neem showed better clinical results at a 10% extract and antibacterial activity against Gram-positive and Gram-negative bacteria at 5% [7,36]. Aloe vera shows antibacterial and anti-inflammatory activity, including against *A. actinomycetemcomitans* [37], and reduces gingival bleeding and swelling [7]. Green tea catechins combined with NSPT were more beneficial than NSPT alone [38], and its alcoholic extract inhibited and killed *P. gingivalis* [7]. A 5% tea tree oil gel with NSPT produced a greater reduction in GCF TNF-α at 90 days than mechanical therapy alone, with no adverse effects [39]. Curcumin is active against *P. gingivalis* and improved periodontal parameters with NSPT [40], and a 2% curcumin nanogel outperformed NSPT alone and NSPT with chlorhexidine gel and reduced key pathogens [41]. Tulsi with NSPT reduced disease parameters and eliminated red complex bacteria more effectively than curcumin gel, although clinical benefits were similar [42].

Rosemary (*Rosmarinus officinalis*) extract

Rosemary (*Rosmarinus officinalis*) is an aromatic Mediterranean plant of the Lamiaceae family, long used in folk medicine and valued as a spice and as an antioxidant that protects tissues from reactive oxygen species and free radicals. Its extracts are also used in food preservation because they inhibit oxidation and microbial contamination [43]. Its antimicrobial and antioxidant effects are attributed mainly to phenolic compounds such as carnosic acid, rosmarinic acid, carnosol, rosmanol, and ursolic acid [44]. It has also been reported to have hepatoprotective, anti-hyperglycemic, antifungal, antitumor, and anti-ulcerogenic activities, and has been explored topically for anti-inflammatory, wound-healing, and UV-protective effects [8]. In antioxidant assays, rosemary extract performed favorably compared with ascorbic acid [9].

Rosemary's antimicrobial activity extends to the main periodontal pathogens. Rosemary oil inhibited P. gingivalis at 1% and killed all the oral bacteria tested [45]. Leaf extract was active against P. gingivalis, F. nucleatum, T. forsythia, and A. actinomycetemcomitans, with MICs between 0.78 and 1.2 µg/ml. A. actinomycetemcomitans was the most susceptible and P. gingivalis the least [10]. It also acts on biofilms, the main target of NSPT: in an *ex vivo* study, 20 and 30 mg/ml extract significantly

lowered bacterial counts in initial oral biofilms, comparable to chlorhexidine [46]. It is active against drug-resistant bacteria such as *S. aureus* and *Salmonella* species, which is relevant given concerns over antibiotic resistance [47].

In a systematic review of preclinical *in vivo* models, rosemary treatment lowered TNF-α, IL-1β, IL-6, IL-10, and myeloperoxidase [11]. Its free-radical scavenging may aid tissue repair, as suggested by an Orabase paste combining rosemary extract with hyaluronic acid and metronidazole that healed traumatic oral ulcers [48].

In the oral cavity, rosemary mouthwash extracts inhibited *S. mutans* and *C. albicans* in a dose-dependent manner and showed synergy with conventional oral care substances [49]. In a randomized trial in gingivitis patients, a rosemary-containing polyherbal mouthwash lowered gingival and plaque scores relative to placebo, performing similarly to chlorhexidine with no adverse reactions [50]. A 5% rosemary toothpaste was comparable to conventional fluoridated toothpaste in controlling gingival bleeding and plaque accumulation [51].

Overall, rosemary could serve as a safe, natural adjunct to NSPT by targeting key pathogens and biofilm, reducing inflammation, and supporting healing, with fewer concerns about resistance and adverse effects than antibiotics and chlorhexidine [46].

Conclusion

Herbal products such as aloe vera, green tea, tea tree oil, and curcumin have shown antimicrobial and anti-inflammatory benefits as safe, affordable adjuncts to NSPT. Among them, rosemary is particularly promising because it acts against key periodontal pathogens, disrupts oral biofilms, and reduces inflammation. However, most evidence comes from *in vitro*, *ex vivo*, animal, and gingivitis studies, so *in vivo* research in periodontitis is still needed.

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Doesn't apply.

Conflict of Interest

There's no conflict of interest.

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