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Evaluation of the Effect of Probiotics as an Adjunct to Non-Surgical Periodontal Therapy

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Abstract

Background: Periodontitis is a biofilm-mediated inflammatory disease, and non-surgical periodontal therapy (scaling and root planing, SRP) remains the standard treatment. Probiotics have recently been explored as adjunctive agents to modulate the oral microbiome and enhance periodontal healing. This study evaluated the clinical efficacy of Lactobacillus paracasei GMNL-33 (Sporolac®-DG) as an adjunct to SRP in patients with Stage I and Stage II periodontitis.

Materials and Methods: This randomised controlled clinical trial included 32 systemically healthy adults diagnosed with Stage I or II periodontitis. Participants were randomly allocated into two equal groups (n = 16 each) using a computer-generated sequence. Group I (test) received SRP along with daily administration of Sporolac®-DG probiotic tablets for four weeks, while

Group II (control) received SRP alone. All participants were provided standardised oral hygiene instructions. Clinical parameters such as Plaque Index (PI), Gingival Index (GI), Probing Pocket Depth (PPD), and Clinical Attachment Level (CAL) were recorded at baseline and four weeks by calibrated examiners using standard periodontal probes. Data were analysed using appropriate statistical tests (paired t-test, unpaired t test), with significance set at $p < 0.05$.

Results: Both groups demonstrated statistically significant improvements in PI, GI, PPD, and CAL following SRP ($p < 0.05$). However, the probiotic group exhibited significantly greater reductions in gingival inflammation and probing depths, along with superior gains in clinical attachment compared to the control group ($p < 0.05$). These findings indicate enhanced periodontal healing when Lactobacillus paracasei GMNL-33 was used as an adjunct to mechanical debridement.

Conclusion: Adjunctive administration of Lactobacillus paracasei GMNL-33 with SRP resulted in superior clinical improvements compared to SRP alone in Stage I and II periodontitis. Probiotic supplementation appears to offer additional anti-inflammatory and tissue-healing benefits and may serve as an effective adjunct in early-stage periodontal therapy.

Keywords: Probiotics; Lactobacillus paracasei GMNL-33; Periodontitis; Scaling and root planing; Clinical attachment level.

Introduction

Periodontitis is a chronic, multifactorial inflammatory disease characterised by the progressive destruction of the periodontal ligament, cementum, and alveolar bone, ultimately leading to tooth mobility and potential tooth loss. The condition arises from a complex interplay between oral microbial dysbiosis and an exaggerated host immune-inflammatory response, which together drive the transition from gingival health to periodontal breakdown. Microbial biofilms dominated by anaerobic Gram-negative organisms trigger sustained inflammation, contributing to connective tissue degradation and bone resorption through the release of cytokines, proteolytic enzymes, and reactive oxygen species.[1] Conventional management centres on non-surgical periodontal therapy (NSPT), particularly scaling and root planing (SRP), which reduces microbial load and disrupts subgingival biofilms. While NSPT is effective in most early to moderate cases, its outcomes may be limited in deeper periodontal pockets, sites with complex anatomy, and in patients whose healing capacity is compromised by systemic conditions or dysregulated immune responses.[1] These limitations have prompted exploration of adjunctive therapeutic strategies that can complement mechanical debridement.

Probiotics have emerged as a promising adjunctive approach in periodontal therapy. Defined as "live microorganisms which, when administered in adequate amounts, confer a health benefit to the host",[2] they have gained increasing attention for their potential to modulate the oral microbiome and restore microbial balance. Their mechanisms of action are multifaceted: probiotics can directly inhibit the growth of pathogenic bacteria through competitive exclusion, coaggregation, and production of antimicrobial substances, or exert indirect effects by modulating the host immune response, down regulating pro-inflammatory mediators, and enhancing the anti-inflammatory milieu.[3] Such properties position probiotics as a biologically driven strategy to augment periodontal healing beyond the mechanical benefits of SRP.

The probiotic formulation evaluated in this study, Sporlac DG, represents a novel advancement in the field of dental probiotics. It is a mouth-dissolving, oro-dental

tablet containing heat-killed Lactobacillus paracasei GMNL-33 combined with Cichorium intybus, a prebiotic containing inulin-type fructans.[4] Unlike traditional probiotics that rely on live organisms, Sporlac DG employs heat-killed *L.* paracasei, which retains immunomodulatory and pathogen-interference properties without the viability constraints associated with live cultures. The inclusion of *C.* intybus acts synergistically by supporting beneficial microbiota and enhancing biological activity within the oral environment. Sporlac DG has demonstrated the ability to coaggregate and agglutinate major periodontal pathogens such as Streptococcus mutans and Porphyromonas gingivalis. This antimicrobial interaction has been associated with reductions in plaque accumulation, gingival inflammation, periodontal probing depths, and halitosis following 4–6 weeks of daily administration.[5]

Despite the expanding evidence supporting probiotics in periodontal therapy, this is the first clinical investigation to specifically assess the adjunctive effects of Sporlac DG in individuals with Stage I and Stage II periodontitis. Evaluating a formulation combining heat-killed *L.* paracasei GMNL-33 with *C.* intybus introduces a novel therapeutic approach that may offer advantages in terms of stability, safety, and targeted antimicrobial activity. This study aims to bridge a critical knowledge gap by determining whether Sporlac DG enhances the clinical outcomes of NSPT, potentially contributing to a new paradigm in biologically supported periodontal treatment.

Materials and Methods

The trial was structured as a randomised controlled clinical study involving 32 patients diagnosed with Stage I or Stage II periodontitis. After screening for eligibility and obtaining informed consent, participants were randomly allocated via a computer-generated sequence into two equal arms of 16 each. Group I (test) received the probiotic along with scaling and root planing, while Group II (control) received scaling and root planing alone. Both groups were provided with standardised oral hygiene instructions. While exact details of masking aren't specified here, many similar periodontal trials employ double-blind or parallel group designs to minimise bias.[6]

Participants were adults diagnosed with Stage I or II periodontitis, criteria likely involving specific thresholds for pocket depth and attachment loss. Typically, Inclusion criteria cover systemic health, absence of recent periodontal or antibiotic therapy, and non-smoking status. Exclusion criteria further eliminate confounding variables like systemic conditions, medication use, or pregnancy, ensuring a more homogeneous sample.

At baseline, clinical parameters including Plaque Index (PI), Gingival Index (GI), Probing Pocket Depth (PPD), and Clinical Attachment Level (CAL) were measured using standardized periodontal probes. These assessments were conducted at multiple sites per tooth to provide a comprehensive evaluation of periodontal health. Where applicable, examiners were likely calibrated to ensure measurement consistency. All participants first received non-surgical periodontal therapy scaling and root planing (SRP) performed using both ultrasonic scalers and hand instruments to debride subgingival calculus thoroughly. In group I (Test group), in addition to SRP, participants received Sporolac®-DG probiotic tablets once daily for 4 weeks, while group II (Control group) received SRP without probiotic supplementation. Both groups were also given standardised oral hygiene instructions and follow-up recommendations.

After four weeks of intervention, the same clinical parameters (PI, GI, PPD, CAL) were reassessed by the

study examiner. This allowed for direct comparison of changes from baseline in both groups.

Statistical Analysis

Data were presented as mean ± standard deviation, comparing within-group changes from baseline and between-group differences at four weeks. Paired and unpaired t tests done for inter and intragroup comparisons with a significance threshold set at $p < 0.05$.

Results

Significant intragroup improvements were observed in both treatment groups over the 4-week period. In Group I, mean PI, GI, PPD, and CAL scores showed highly significant reductions from baseline to 4 weeks ($p < 0.001$ for all)(Table 1). PI decreased from 2.3 ± 0.2 to 0.9 ± 0.2, and GI reduced from 2.1 ± 0.3 to 0.8 ± 0.2, indicating marked improvements in plaque control and gingival health. Periodontal parameters also improved substantially, with PPD reducing from 4.2 ± 0.4 to 3.1 ± 0.3 and CAL from 4.4 ± 0.3 to 3.2 ± 0.4. Group II also demonstrated significant reductions in all parameters ($p < 0.001$), though the magnitude of improvement was comparatively smaller than Group I (Table 2). PI reduced from 2.2 ± 0.3 to 1.3 ± 0.3, GI from 2.0 ± 0.3 to 1.2 ± 0.4, PPD from 4.1 ± 0.5 to 3.6 ± 0.4, and CAL from 4.3 ± 0.3 to 3.8 ± 0.5, reflecting moderate clinical improvement within the group.

Between-group comparisons further highlighted the superior performance of Group I at 4 weeks. At baseline, there were no statistically significant differences between the groups for any clinical parameters ($p > 0.05$), confirming comparability (Table 3). However, at 4 weeks, Group I exhibited significantly lower PI, GI, PPD, and CAL values than Group II ($p \leq 0.001$ for all) (Table 4). The greater reductions in Group I suggest a more pronounced therapeutic effect compared with Group II, indicating that the intervention provided to Group I was more effective in improving both gingival inflammation and periodontal parameters within the 4-week period.

Discussion

Sporlac-DG Oro-Dental Tablet (Fresh Mint) is an oral dispersible tablet containing Lactobacillus paracasei GMNL-33 (heat-killed probiotic), developed for the supportive management of Oro-dental conditions, inflammation of the gums and teeth, bad breath, and dental caries. This probiotic strain may exert immunomodulatory and anti-inflammatory[7] properties that help restore oral microbial balance, control harmful bacteria, and support oral immunity.[4]

Previous studies have shown that L. paracasei GMNL-33 can suppress cariogenic and periodontal pathogens. Chuang et al. conducted a randomized, double-blind, placebo-controlled study in healthy adults aged 20-40, administering L. paracasei GMNL-33 tablets three times daily for two weeks. While there was no immediate reduction in salivary Streptococcus mutans counts at the end of treatment, a significant decline emerged two weeks post-treatment ($p = 0.016$), suggesting that the probiotic effect may manifest after discontinuation rather than during active dosing. Chuang et al. reported delayed but significant reductions in S. mutans after probiotic administration in healthy adults.[8]

Similarly, the use of a commercially available probiotic toothpaste containing L. paracasei (PerioBiotic) demonstrated efficacy in reducing monospecies biofilm of S. mutans via plaque regrowth modeling. However, the effect was short-lived, especially under high sucrose exposure. While probiotic toothpaste containing L. paracasei also demonstrated inhibitory effects on plaque regrowth, albeit short-lived under high sucrose exposure.[9]

Beyond clinical outcomes, in vitro evidence supports the antimicrobial potential of L. paracasei. It has been shown

to inhibit *S.* mutans growth and other pathogens, reinforcing its usefulness in oral biofilm modulation. In vitro studies confirm that L. paracasei exhibits antimicrobial and biofilm-modulating properties.[10]

Products like Sporlac-DG, which utilize heat-killed (postbiotic) L. paracasei GMNL-33, have been developed with the intention of correcting oral dysbiosis and shifting the microbial balance toward health. The postbiotic components, such as lipoteichoic acid and peptidoglycan, are designed to co-aggregate with pathogens like S. mutans and P. gingivalis, potentially reducing plaque buildup, gingival inflammation, periodontal pocket depths, and halitosis, especially with consistent use over several weeks.

Importantly, Sporlac DG uses a postbiotic approach (heat-killed strain), which offers advantages of safety, stability, and resistance to antimicrobial resistance transfer, while still exerting beneficial effects via bacterial cell wall components (lipoteichoic acid, peptidoglycan).[11,12] These findings, combined with the synbiotic action from *C.* intybus prebiotic, may explain the observed improvements in clinical parameters in the present study.

Altogether, these findings illustrate that *L.* paracasei GMNL-33, whether live or heat-inactivated, offers a multifaceted oral health benefit. It not only demonstrates delayed but sustained reductions in cariogenic *S.* mutans, but also directly inhibits pathogenic biofilms and may adjust microbial communities in favor of oral health. Such attributes make it a promising adjunct in periodontal therapy, reinforcing the effectiveness of conventional mechanical treatments.

Conclusion

This study supports the use of heat-killed *L.* paracasei GMNL-33 with *C.* intybus (Sporlac DG) as an effective adjunct to mechanical therapy in managing Stage I and II periodontitis. Incorporating probiotics/postbiotics into periodontal treatment protocols may enhance clinical outcomes and provide a safer, novel adjunctive approach.

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Legend Tables

Table 1: Intragroup comparison of PI, GI, PPD, CAL from baseline to 4 weeks in the test group

Parameter	Group I baseline	Group I 4 weeks	t statistic	P value
PI	2.3 ± 0.2	0.9 ± 0.2	28.0	<0.001*
GI	2.1 ± 0.3	0.8 ± 0.2	19.7	<0.001*
PPD	4.2 ± 0.4	3.1 ± 0.3	12.2	<0.001*
CAL	4.4 ± 0.3	3.2 ± 0.4	13.3	<0.001*

Paired t test; p≤0.05 considered statistically significant; * denotes significance

Table 2: Intragroup comparison of PI, GI, PPD, CAL from baseline to 4 weeks in the control group

Parameter	Group II baseline	Group II 4 weeks	t statistic	P value
PI	2.2 ± 0.3	1.3 ± 0.3	12.3	<0.001*
GI	2.0 ± 0.3	1.2 ± 0.4	8.88	<0.001*
PPD	4.1 ± 0.5	3.6 ± 0.4	4.36	<0.001*
CAL	4.3 ± 0.3	3.8 ± 0.5	4.59	<0.001*

Paired t test; p≤0.05 considered statistically significant; * denotes significance

Table 3: Intergroup comparison of PI, GI, PPD, CAL at baseline

Parameter	Group I baseline	Group II baseline	t statistic	P value
PI	2.3 ± 0.2	2.2 ± 0.3	1.11	0.28
GI	2.1 ± 0.3	2.0 ± 0.3	0.94	0.35
PPD	4.2 ± 0.4	4.1 ± 0.5	0.62	0.54
CAL	4.4 ± 0.3	4.3 ± 0.3	0.94	0.35

Unpaired t test; p≤0.05 considered statistically significant

Table 4: Intergroup comparison of PI, GI, PPD, CAL at 4 weeks

Parameter	Group I 4 weeks	Group II 4 weeks	t statistic	P value
PI	0.9 ± 0.2	1.3 ± 0.3	4.44	<0.001*
GI	0.8 ± 0.2	1.2 ± 0.4	3.58	0.001*
PPD	3.1 ± 0.3	3.6 ± 0.4	4.00	<0.001*
CAL	3.2 ± 0.4	3.8 ± 0.5	3.75	<0.001*

Unpaired t test; p≤0.05 considered statistically significant; * denotes significance