RESEARCH PAPER

Clinical, Microbiological and Biochemical Effects of Probiotic Supplementation as an Adjunct to Scaling and Root Planing in Patients with Periodontitis: A Randomized Controlled Study

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ABSTRACT

Background: Periodontitis is a chronic inflammatory disease driven by dysbiotic subgingival biofilms and a host-mediated response. Scaling and root planing (SRP) is the foundation of nonsurgical therapy, but residual microorganisms and recolonization may limit outcomes. Probiotics may provide adjunctive benefits by modifying microbial ecology and host responses.

Aim: To evaluate the clinical, microbiological and biochemical effects of probiotic supplementation as an adjunct to SRP in mild-to-moderate periodontitis.

Materials and methods: A single-blinded, randomized, placebo-controlled study included 60 participants allocated to SRP plus placebo lozenges (Group I) or SRP plus probiotic lozenges twice daily for 3 weeks (Group II). PI, GI, SBI, PPD and CAL were assessed at baseline, 3 and 6 weeks. GCF MMP-8 and subgingival P. gingivalis were assessed at baseline and 3 weeks.

Results: Both groups improved, with greater changes in the probiotic group. At 6 weeks, GI decreased from 2.09 ± 0.53 to 1.12 ± 0.42, PI from 2.15 ± 0.48 to 1.04 ± 0.51, SBI from 2.24 ± 0.42 to 1.13 ± 0.49, and PPD from 5.19 ± 0.14 mm to 1.69 ± 0.57 mm in Group II. CAL improved from 3.05 ± 0.13 to 0.50 ± 0.47 mm. MMP-8 decreased from 40.9 ± 0.36 to 20.2 ± 0.67 ng/mL, with marked reduction in P. gingivalis detection.

Conclusion: Probiotic supplementation with SRP was associated with greater clinical changes, lower MMP-8 and reduced P. gingivalis detection. Larger, longer-term trials with standardized strains are required.

Keywords: Periodontitis; probiotics; scaling and root planing; gingival index; probing pocket depth; clinical attachment level; matrix metalloproteinase-8; Porphyromonas gingivalis.

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INTRODUCTION

Periodontitis is a chronic inflammatory disease of the tooth-supporting tissues in which dysbiotic microbial communities interact with the host immune-inflammatory response, producing clinical attachment loss, periodontal pocket formation and, in susceptible individuals, progressive destruction of connective tissue and alveolar bone.[1,2] The subgingival pocket contains a complex polymicrobial community. Porphyromonas gingivalis has been extensively studied because of its virulence factors and capacity to influence host inflammation, while Tannerella forsythia, Treponema denticola, Prevotella intermedia and Aggregatibacter actinomycetemcomitans may also contribute to dysbiosis and disease progression.[3]

The primary objective of periodontal treatment is to control the microbial challenge and arrest disease progression. SRP remains a key component of nonsurgical periodontal therapy because it disrupts subgingival biofilm and removes calculus and contaminated root-surface deposits.[4,5] Nevertheless, complete elimination of pathogenic microorganisms is difficult and recolonization can occur. Systemic or locally administered antimicrobials may provide additional benefits in selected cases, but concerns regarding adverse effects, antimicrobial resistance and recolonization have encouraged investigation of biological approaches that modify the oral ecosystem.[4,5]

Probiotics are live microorganisms that, when administered in adequate amounts, confer a health benefit on the host.[6] In the oral cavity, proposed actions include competition with pathogens for adhesion sites and nutrients, production of

Table 1. Schedule of clinical, biochemical and microbiological assessments

Outcome	Baseline	3 weeks	6 weeks
Plaque Index (PI)	✓	✓	✓
Gingival Index (GI)	✓	✓	✓
Sulcus Bleeding Index (SBI)	✓	✓	✓
Probing Pocket Depth (PPD)	✓	✓	✓
Clinical Attachment Level (CAL)	✓	✓	✓
GCF MMP-8	✓	✓	—
Subgingival P. gingivalis	✓	✓	—

antimicrobial substances, interference with biofilm formation, modification of local environmental conditions and modulation of host inflammatory responses.[7] Clinical studies involving Lactobacillus reuteri have reported improvements in periodontal parameters when probiotics are used with conventional periodontal therapy.[8]

MMP-8 is a collagenolytic enzyme involved in periodontal connective tissue breakdown and is considered a useful biomarker of periodontal inflammation. Increased GCF MMP-8 has been associated with active inflammation, while successful periodontal therapy may reduce its concentration.[7] Probiotics may also influence periodontal pathogens through ecological competition and production of inhibitory metabolites.[7]

Systematic reviews and meta-analyses generally indicate short-term improvements in PPD, CAL, gingival inflammation and bleeding when probiotics are used as adjuncts to nonsurgical periodontal therapy, although studies differ substantially in strains, formulations, dose and duration.[8] Effects appear to be strain-specific and therefore should not automatically be generalized across probiotic preparations.[8] Probiotics have also been investigated for oral malodour. Available evidence suggests possible improvement in some halitosis outcomes, but objective volatile sulfur compound findings remain inconsistent and protocols are heterogeneous.[8] The present study did not objectively assess halitosis; therefore, it cannot establish a direct probiotic effect on oral malodour.

The present study evaluated the clinical efficacy of probiotic supplementation as an adjunct to SRP and investigated associated changes in GCF MMP-8 and subgingival P. gingivalis in patients with periodontitis.

MATERIALS AND METHODS

Study design and participants

A single-blinded, randomized, placebo-controlled prospective clinical study was conducted over 6 weeks in 60 participants with mild-to-moderate periodontitis. Participants were randomly allocated into two groups of 30. The source study defined eligible periodontal sites as having probing depths

≥4 mm and ≤7 mm with generalized interproximal attachment loss. Institutional ethical clearance was obtained, the study followed the Declaration of Helsinki and Good Clinical Practice principles, and written informed consent was obtained.

Randomization and intervention Participants were allocated by lottery into Group I (SRP + placebo lozenges; n=30) and Group II (SRP + probiotic lozenges; n=30). Both groups received full-mouth ultrasonic scaling followed by root planing with area-specific Gracey curettes. Group II received probiotic lozenges twice daily for 3 weeks, while Group I received identical placebo lozenges on the same schedule.

Probiotic formulation: The supplied study used BIFILAC lozenges containing Lactobacillus sporogenes (100 million), Streptococcus faecalis T-110 JPC (60 million), Clostridium butyricum TO-A (4 million) and Bacillus mesentericus TO-A JPC (2 million).

Clinical periodontal assessment

PI, GI, SBI, PPD and CAL were recorded at baseline, 3 weeks and 6 weeks. These outcomes were used to evaluate changes following SRP and adjunctive supplementation. Clinical assessments were performed at baseline, 3 weeks and 6 weeks. Gingival crevicular fluid and plaque sampling. The site with the greatest inflammatory signs or attachment loss was selected for GCF collection. After isolation and gentle air drying, a sterile 2-µL glass microcapillary tube was placed at the pocket opening. Samples were transferred to labelled Eppendorf tubes containing 500 µL phosphate-buffered saline and stored at −20°C. Subgingival plaque was collected from the selected site with sterile Gracey curettes and similarly stored for microbiological analysis.

Biochemical and microbiological analysis

GCF MMP-8 was measured by ELISA at baseline and 3 weeks. Subgingival plaque was analyzed by conventional PCR for detection of P. gingivalis at baseline and 3 weeks.

Statistical analysis. Data were analyzed using SPSS version 19. Within-group comparisons were performed using Student's paired t-test, with $p<0.05$ considered statistically significant. The supplied study report described significant between-group differences for several clinical and biochemical outcomes.

RESULTS

Participant safety and clinical outcomes

All 60 participants completed the intervention period. No adverse reactions, discomfort or complications related to the study product were reported. Both groups showed reductions in PI, GI, SBI and PPD, with greater improvement in the probiotic group.

According to Table 1, Both groups' GI, PI, and SBI readings significantly decreased; in Group I, GI decreased from 2.07 to 1.84, PI from 2.12 to 1.92, and SBI from 2.30 to 1.96. GI decreased from 2.09 to 1.12, PI decreased from 2.15 to 1.04, and SBI decreased from 2.24 to 1.13 in Group II. The statistical significance of the intergroup comparison was $p<0.001$.GI decreased from 2.07 ± 0.48 to 1.84 ± 0.47 in Group I and from 2.09 ± 0.53 to 1.12 ± 0.42.

Table 2. Changes in clinical inflammatory indices from baseline to 6 weeks

Parameter	SRP + placebo Baseline	SRP + placebo 6 weeks	SRP + probiotic Baseline	SRP + probiotic 6 weeks	p value
Gingival Index	2.07 ± 0.48	1.84 ± 0.47	2.09 ± 0.53	1.12 ± 0.42	<0.001
Plaque Index	2.12 ± 0.51	1.92 ± 0.43	2.15 ± 0.48	1.04 ± 0.51	<0.001
Sulcus Bleeding Index	2.30 ± 0.47	1.96 ± 0.43	2.24 ± 0.42	1.13 ± 0.49	<0.001

Table 3. Changes in probing pocket depth and clinical attachment level

Clinical parameter	SRP + placebo Baseline	SRP + placebo 6 weeks	SRP + probiotic Baseline	SRP + probiotic 6 weeks	p value
PPD (mm)	5.13 ± 0.10	2.50 ± 0.54	5.19 ± 0.14	1.69 ± 0.57	<0.001
CAL (mm)	3.09 ± 0.21	1.27 ± 0.38	3.05 ± 0.13	0.50 ± 0.47	<0.001

According to Table 2, Group I = SRP + placebo; Group II = SRP + probiotic; values are mean ± SD.PPD reduced from 5.13 ± 0.10 mm to 2.50 ± 0.54 mm in Group I and from 5.19 ± 0.14 mm to 1.69 ± 0.57 mm in Group II. PPD decreases of 51.7% and 77.4%, respectively, were found.

According to table 3, Clinical attachment level: CAL increased from 3.05 ± 0.13 mm to 0.50 ± 0.47 mm in Group II and from 3.09 ± 0.21 mm to 1.27 ± 0.38 mm in Group I.CAL reported increases of 56.7% and 84.4%, respectively.

According to table 4, MMP-8 levels: Group I and Group II had baseline mean GCF MMP-8 concentrations of 40.7 ± 0.61 ng/mL and 40.9 ± 0.36 ng/mL, respectively. After three weeks, values dropped to 20.2 ± 0.67 ng/mL and 32.3 ± 0.38 ng/mL, respectively, with a reported $p<0.05$.

Values are mean ± SD. MMP-8 = matrix metalloproteinase-8.

Microbiological findings

P. gingivalis was detected in both groups at baseline. After 3 weeks, a marked reduction in detection was reported in the probiotic group compared with the placebo group.

DISCUSSION

The present study assessed whether probiotic supplementation provides additional benefit when combined with nonsurgical periodontal therapy. Both groups improved, consistent with the established effect of mechanical debridement. SRP disrupts subgingival biofilm and removes calculus and contaminated root-surface deposits, reducing microbial burden and allowing resolution of periodontal inflammation.[9,10]

The greater improvement in the probiotic group may be related to competition with periodontal pathogens for adhesion sites and nutrients, interference with biofilm development, production of antimicrobial compounds and modification of the local microbial ecosystem.[7,11] the greater reductions in GI, PI and SBI are consistent with clinical studies reporting improved gingival inflammation and plaque-related parameters after probiotic use. Krasse et al. reported decreased gingival bleeding and gingivitis following L. reuteri administration, while other studies found improved periodontal parameters when probiotic lozenges were combined with nonsurgical treatment.[8,12]

PPD reduction was greater in the probiotic group, with an approximately 2.63-mm reduction in Group I and 3.50-mm reduction in Group II. These findings are compatible with randomized clinical trials and systematic reviews suggesting an additional short-term reduction in pocket depth with probiotics used alongside SRP.[16–19] CAL improvement was also greater in Group II. Teughels et al. and Ince et al. reported greater pocket-depth reduction and attachment gain with L. reuteri-containing preparations as adjuncts to nonsurgical periodontal therapy.[8,12]

The biochemical findings support a possible anti-inflammatory effect. MMP-8 contributes to collagen degradation and periodontal connective tissue destruction. In the present dataset, MMP-8 fell from approximately 40.9 to 20.2 ng/mL in the probiotic group, compared with a reduction to approximately 32.3 ng/mL in the placebo group. Similar reductions have been described after

Table 4. Gingival crevicular fluid MMP-8 concentrations at baseline and 3 weeks

Time point	SRP + placebo	SRP + probiotic	p value
Baseline (ng/mL)	40.7 ± 0.61	40.9 ± 0.36	—
3 weeks (ng/mL)	32.3 ± 0.38	20.2 ± 0.67	<0.05

probiotic supplementation.[13] Probiotics may influence inflammatory mediator production and tissue-destructive enzyme activity, providing a possible biological explanation for the observed changes.[7]The reduction in P. gingivalis detection is also consistent with proposed ecological effects of probiotics. Probiotic organisms may compete for ecological niches, adhesion sites and nutrients and may produce metabolites that inhibit pathogenic growth.[14,15] However, conventional PCR detects microbial DNA and does not necessarily demonstrate bacterial viability. In addition, assessment of a single pathogen cannot represent the complete subgingival microbiome. Periodontitis is recognized as a dysbiotic polymicrobial disease rather than a disease attributable to one organism.[1,2] Current evidence suggests a possible short-term adjunctive benefit, but heterogeneity among studies limits direct comparison. Meta-analyses have reported improvements in PPD, CAL, gingival inflammation and bleeding at selected follow-up periods, whereas long-term effects are less consistent.[16–19]

Differences in strain, dose, formulation, route and duration are important, and a systematic review of L. reuteri also emphasized strain-specific effects.[20]

The probiotic preparation in this study was a combination product rather than a single standardized periodontal strain. Therefore, the results cannot be generalized to all

probiotics. Standardized preparations and clearly defined treatment protocols are needed in future trials.[18,20]

Probiotics have also been studied for halitosis. Yoo et al. reported improvement in organoleptic halitosis scores, although evidence for volatile sulfur compound reduction was inconclusive.[21,22] Another systematic review found potential benefit but emphasized limited and heterogeneous trials, while more recent evidence suggests possible reduction in volatile sulfur compounds. [23,24] Because halitosis was not measured in the present study, these findings cannot establish efficacy for oral malodour. Future research should use validated organoleptic scores and/or objective volatile sulfur compound measurements.

CONCLUSION

Within the limitations of the supplied randomized clinical study, probiotic supplementation used with SRP was associated with greater short-term improvement in clinical periodontal parameters than SRP with placebo. The probiotic group showed greater reductions in plaque accumulation, gingival inflammation, sulcus bleeding and PPD, together with greater CAL improvement. GCF MMP-8 also decreased more markedly, and P. gingivalis detection was reduced. These findings support the potential role of probiotics as an adjunct to conventional nonsurgical periodontal therapy, but not as a substitute for mechanical debridement. Because halitosis was not objectively assessed, the study does not establish an effect on oral malodour. Larger randomized trials with standardized strains, defined dosing, longer follow-up, comprehensive microbiological assessment and objective halitosis measurements are needed.

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