J Clin Periodontol 2004; 31: 141–148
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Journal of Clinical Periodontology

Quadrant root planing versus same-day full-mouth root planing

II. Microbiological findings

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Apatzidou DA, Riggio MP, Kinane DF: Quadrant root planing versus same-day full-mouth root planing II. Microbiological findings. J Clin Periodontol 2004; 31: 141–148. © Blackwell Munksgaard, 2004.

Abstract

Objectives: The aim of this study was to test the hypothesis that over a period of 6 months, same-day full-mouth scaling and root planing (FM-SRP) resulted in greater reductions in the detection frequency of five putative periodontal pathogens compared with quadrant scaling and root planing (Q-SRP) in chronic periodontitis patients.

Materials and Methods: Forty patients were recruited into this study. Subjects were randomised into two groups. The FM-SRP group received full-mouth scaling and root planing completed within the same day, while the Q-SRP group received quadrant root planing at 2-weekly intervals over four consecutive sessions. Selected-site analyses were performed on the deepest site in each quadrant before and after therapy, at approximately 3 and 6 months from baseline (R1 and R2) and clinical indices were recorded with an electronic pressure-sensitive probe. In addition, subgingival plaque samples were collected from these sites at baseline (BAS), at reassessment 1 (R1), approximately 6 weeks after the completion of therapy and at reassessment 2 (R2), 6 months from baseline. Polymerase chain reaction (PCR) was used to determine the presence of *Porphyromonas gingivalis*, *Actinobacillus actinomycetemcomitans*, *Prevotella intermedia*, *Treponema denticola* and *Bacteroides forsythus* in plaque.

Results: Both therapies resulted in significant improvements in all clinical indices both at R1 and R2. A marked reduction in the presence of all candidate periodontal pathogens was noted after both treatment modalities, reaching statistical significance for the majority of the test organisms. These improvements were maintained over a period of 6 months. When the two treatment groups were compared, a significantly higher percentage of Q-SRP patients was positive for *P. intermedia* at R1 compared with FM-SRP patients ($p<0.05$). In addition, a greater reduction in the patient prevalence for *T. denticola* was found for the FM-SRP group than the Q-SRP group at R1 and R2 from baseline ($p<0.005$), but the significance of this is questionable given the skewed detection frequency of this organism at baseline between the two treatments ($p<0.01$).

Conclusion: This study failed to confirm that same-day FM-SRP resulted in greater microbiological improvements compared with Q-SRP at 2-weekly intervals over a 6-month period, as determined by PCR.

Key words: chronic periodontitis; full-mouth periodontal therapy; periodontal pathogens; quadrant scaling and root planing

Accepted for publication 15 April 2003

Periodontitis is a multifactorial inflammatory disease process, leading to destruction in the periodontium of the tissues supporting the teeth. Periodontitis lesions are caused by mixed infections with the subgingival microbiota in a state of continual flux. No sole pathogen can therefore be implicated in the aetiology and pathogenesis of periodontitis. Haffajee & Socransky (1994) suggested the following subgingival species: *Actinobacillus actinomycetemcomitans*, *Porphyromonas gingivalis*, *Prevotella intermedia*, *Bacteroides forsythus* and *Treponema denticola* as putative periodontal pathogens.

Numerous investigations have sought to determine the beneficial effects of scaling and root planing on both the clinical and microbiological parameters and have consistently reported marked changes in the subgingival microflora and clinical indices following non-surgical instrumentation (Slots et al. 1979,

Pedrazzoli et al. 1991, Shiloah & Patters 1994, 1996, Darby et al. 2001). Simonson et al. (1992) demonstrated that *T. denticola* and, to a lesser extent, *P. gingivalis* levels decreased in successfully treated sites. Several suspected periodontal pathogens, including *P. gingivalis*, *T. denticola* and *B. forsythus,* were found at higher levels before than after treatment in the subjects or sites that responded well to therapy (Haffajee et al. 1997b). Nevertheless, it is unlikely that scaling and root planing can eradicate and permanently clear all the microorganisms from a site. The microbes that re-colonise a subgingival pocket post-therapy could be either residual microorganisms following incomplete instrumentation or the extension of a growing and maturing supragingival plaque. It has been shown that in the presence of supragingival plaque, it takes 42 days for organisms to re-colonise a previously scaled site (Mousquès et al. 1980).

In a pilot study, Quirynen et al. (1995) introduced the one-stage full-mouth disinfection therapy as a means of preventing re-infection of treated sites from the remaining untreated pockets and/or other intra-oral niches. In their study, plaque samples were collected from the upper right quadrant of 10 patients at baseline and after 1 and 2 months. At 1 month, differential phase-contrast microscopy revealed a significantly greater reduction in the proportion of potential periodontal pathogens (sum of spirochaetes and motiles) in the test group (periodontitis patients who received the one-stage full-mouth disinfection treatment) rather than the control group (patients who received quadrant root planing at 2-weekly intervals), whereas at 2 months the differences were no longer significant. The culture data showed that there were no differences in the relative proportion of aerobic colony forming units (CFUs) for both treatments. Nevertheless, when the relative proportions of potential pathogenic and beneficial species were compared, the test group showed significantly less pathogenic organisms at 1 month and more beneficial species at 2 months. Post-treatment, *P. gingivalis* was eliminated from the test group although this was not the case for *A. actinomycetemcomitans*, whose proportion increased following treatment, which is consistent with the poor treatment findings of others (Slots et al. 1986, Renvert et al. 1990, Pedrazzoli et al. 1992).

In follow-up studies by the same investigators using a similar study protocol, subgingival plaque samples were analysed by differential phase-contrast microscopy and consistently showed that the spirochaetes and motile organisms were markedly reduced in the full-mouth treated patients as compared with the quadrant-treated patients; in addition the culture results were also markedly improved (Bollen et al. 1996, 1998, Quirynen et al. 1999). A later study compared the effects of the one-stage full-mouth disinfection and quadrant scalings on the subgingival microflora by the checkerboard DNA–DNA hybridisation technique (De Soete et al. 2001). The results of this study showed that the one-stage full-mouth disinfection resulted in a greater reduction in the detection frequency and levels of the ''red'' and ''orange'' complex species (Socransky et al. 1998), and in the numbers of patients in whom these complexes of species were undetectable.

Polymerase chain reaction (PCR) is a rapid and highly sensitive technique that has been used for the detection of bacterial DNA sequences. PCR is highly specific and relatively inexpensive. It is not as labour-intensive as other methods and it does not rely on viable cells. With these characteristics in mind, PCR can be more advantageous than other techniques such as culture methods immunodiagnostic methods, and chromosomal and cloned DNA probes (Ashimoto et al. 1996, Riggio et al. 1998).

The aim of the current study was to determine whether same-day full-mouth scaling and root planing (FM-SRP) would show greater microbiological reductions in five putative periodontal pathogens detected by PCR over a 6-month period compared with typical consecutive quadrant scaling and root planing (Q-SRP) in chronic periodontitis patients.

Materials and Methods

Forty untreated chronic periodontitis patients, aged 31–70 years, were recruited from new referrals to Glasgow Dental Hospital and School. Each patient had at least two non-adjacent sites per quadrant with pocket depths (PDs) of 5 mm or over and radiographic evidence of bone loss, with no history of systemic disease or antibiotic therapy within the last 3 months or during the course of the study. All patients gave informed consent. The demographic details of the participants and the study design and treatment protocol are described in our earlier published report (Apatzidou & Kinane 2004). Briefly, the present investigation is a randomised controlled study of Q-SRP against FM-SRP. No disinfection, i.e. antiseptics such as chlorhexidine, was used during the 6-month period of observation. Q-SRP was performed at 2-weekly intervals, while FM-SRP was completed within 12 h. Table 1 depicts the periodontal status and disease severity for each treatment group.

Sample Collection

Selected-site clinical readings and site-specific subgingival plaque samples were performed at one site per quadrant with the deepest PD, but not shallower than 5 mm, with no endodontic or furcation involvement. These selected-site samples were obtained at three time points: (1) baseline (BAS); (2) reassessment 1 (R1), 6 weeks after the last session of scaling and root planing; and (3) reassessment 2 (R2), 6 months after baseline. Twenty subjects comprised each treatment group. Two subjects in the FM-SRP group failed to attend R2 and therefore the analysis at this time point was based on 18 subjects.

The clinical assessments of the selected sites were performed with the Florida probe for PD and relative attachment level (RAL) measurements. Florida probes were wiped with 70% isopropyl alcohol wipes between measurements to reduce bacterial contamination of the sites for subsequent collection of subgingival plaque samples. After ensuring that no supragingival plaque deposits were present, the sites were isolated with cotton rolls and gently air-dried in an apico-coronal direction. Subgingival plaque samples were taken with a single vertical stroke, using a sterile curette for each sample to prevent cross-contamination. Care was taken to access the most apical part of the pocket with the instrument and it was a pooled sample including gingival, mid-pocket and apical samples. Samples were stored on ice in coded sterile microcentrifuge tubes containing 0.5 ml sterile MilliQ grade H_2O (Millipore UK Limited, Watford, UK). Plaque samples were vortex mixed for 30 s and stored at $-70^{\circ}C$ until required. The PCR analysis was performed blindly. Once thawed the plaque sample was vortex mixed for

Table 1. Descriptive characteristics of disease severity in the Q-SRP ($N = 20$) and FM-SRP groups ($N = 20$) (mean ± SD)

Whole-mouth clinical indices	Baseline	Selected-site clinical indices	Baseline
PD (mm)		PD (mm)	
Q-SRP	4.4 ± 0.7	Q-SRP	6.2 ± 0.7
FM-SRP	4.4 ± 0.6	FM-SRP	5.9 ± 0.8
CAL (mm)		RAL (mm)	
Q-SRP	5.0 ± 0.9	Q-SRP	14.0 ± 1.7
FM-SRP	5.1 ± 1.0	FM-SRP	13.9 ± 1.3
BOP (%)		MGI	
Q-SRP	71.0 ± 19.0	Q-SRP	2.5 ± 0.7
FM-SRP	68.0 ± 17.0	FM-SRP	2.4 ± 0.5
No. of sites ⩾ 5 mm		PI	
Q-SRP	69.0 ± 20.0	Q-SRP	1.9 ± 0.6
FM-SRP	68.0 ± 26.0	FM-SRP	1.9 ± 0.6

Mean number (min–max) of teeth present: 27 (22–31) for the Q-SRP group and 26 (19–32) for the FM-SRP group.
no. = number; PD = pocket depth; CAL = attachment level; BOP = clinical bleeding on probing; RAL = relative attachment level; MGI = modified gingival index; PI = plaque index; Q-SRP = quadrant scaling and root planing; FM-SRP = full-mouth scaling and root planing.

30 s and an aliquot was taken, for subsequent use in the PCR analysis. Lysates of plaque samples were prepared by boiling for 10 min.

PCR

Table 2 lists the PCR primers used in the current study. Species-specific primers that were previously tested for cross-reactivity with other closely related species targeted the 16S rRNA of the bacteria. The specificity of the amplified products and therefore of the primers was confirmed in previous studies by Ashimoto et al. (1996) for *P. gingivalis*, *A. actinomycetemcomitans*, *T. denticola* and *B. forsythus*, and by Riggio et al. (1998) for *P. intermedia*. All primers were obtained from MWG-Bistech (Milton Keynes, UK). Primers were supplied in a lyophilised form and were re-suspended in sterile, molecular biology grade water at 1 μg/ml (MilliQ grade H_2O).

PCR amplification reactions were carried out in a reaction mixture of 100 μl consisting of 10 μl sample lysate and 90 μl of reaction mixture containing 1 × PCR buffer (10 mM Tris-HCl (pH 9.0 at 25°C), 1.5 mM $MgCl_2$, 50 mM KCl, 0.1%Triton® X-100; Invitrogen, Carlsbad, CA, USA), 2 U of Taq DNA polymerase (Promega, Madison, WI, USA), 0.2 mM dNTPs (dATP, dCTP, dGTP, dTTP) and 50 pmol of each primer. The primers were separated from the other components of the reaction mixture by a layer of wax (DyNAwax, Flowgen, Lichfield, UK). The wax layer prevented the PCR reaction from starting until the wax had melted upon commencement of PCR cycling (''hot start'' PCR). PCR cycling was carried out in an OmniGene thermal cycler (Hybaid, Teddington, UK).

The cycling conditions for *P. intermedia* and *A. actinomycetemcomitans* comprised an initial denaturation step at 94°C for 5 min, 40 amplification cycles of denaturation at 94°C for 1 min, annealing of primers at 55°C for 1 min and primer extension at 72°C for 1.5 min, followed by a final extension step at 72°C for 10 min. The cycling conditions for *P. gingivalis*, *T. denticola* and *B. forsythus* were as follows: initial denaturation at 94°C for 5 min, 35 amplification cycles of denaturation at 94°C for 1 min, annealing of primers at 60°C for 1 min and primer extension at 72°C for 1.5 min, followed by a final extension step at 72°C for 10 min as previously described by Ashimoto et al. (1996). The reaction products were either stored at −20°C or analysed immediately.

Strict procedures were employed when carrying out PCR. Separate rooms were used for sample preparation, setting up of PCR reactions and analysis of PCR products. Filter tips were used at

Table 2. Sequences, expected product size, target and references for PCR primers

Primer pairs (5′–3′)	Base position amplicon length (bp)	Target	Reference
Porphyromonas gingivalis AGG CAG CTT GCC ATA CTG CG ACT GTT AGC AAC TAC CGA TGT	729–1132 (404)	16S rRNA	Slots et al. (1995)
Actinobacillus actinomycetemcomitans ATG CCA ACT TGA CGT TAA AT AAA CCC ATC TCT GAG TTC TTC TTC	478–1034 (557)	16S rRNA	Ashimoto et al. (1996)
Prevotella intermedia CCT AAT ACC CGA TGT TGT CCA CA AAG GAG TCA ACA TCT CTG TAT CC	1028–1006 (855)	16S rRNA	Riggio et al. (1998)
Treponema denticola TAA TAC CGA ATG TGC TCA TTT ACA T TCA AAG AAG CAT TCC CTC TTC TTC TTA	193–508 (316)	16S rRNA	Slots et al. (1995)
Bacteroides forsythus GCG TAT GTA ACC TGC CCG CA TGC TTC AGT GTC AGT TAT ACC T	120–760 (641)	16S rRNA	Slots et al. (1995)

all stages of the experiment, except when adding the sample to the reaction mixture when a positive displacement pipette was used. Negative and positive controls were included in each batch of samples being analysed by PCR. The negative control was a 90 μl reaction mixture with the sample replaced by 10 μl of sterile water. The positive control, with the exception of *T. denticola*, contained 10 ng of genomic DNA from the relevant organism in 90 μl of reaction mixture, with sterile water added to bring the volume to 100 μl.

For the *T. denticola* positive control, a synthetic *T. denticola* positive control was constructed. A small fragment of the *T. denticola* 16S rRNA gene (79 bp) was amplified using the following nucleotide primers (from 5′–3′): TAA TAC CGA ATG TGC TCA TTT ACA TAA AGG TAA ATG AGG AAA GGA GCT (base position from 193 to 244) and T CAA AGA AGC ATT CCC TCT TCT TCT TA (base position from 508 to 482).

Analysis of PCR Products

Ten microlitres of each reaction product was added to 1.5 μl of gel loading dye (0.25% bromophenol blue, 50% glycerol, 100 mM EDTA pH 8.0), electrophoresed on a 2% agarose gel containing ethidium bromide (0.5 μg/ml), and visualised and photographed using an ImageMaster video documentation system (Pharmacia Biotech, St Albans, UK). A 100 bp DNA ladder (Pharmacia Biotech) was used as a molecular weight marker.

Statistical analysis

SPSS (version 9.0, SPSS Inc., Chicago, IL, USA) statistical package was used for the analyses of data. Statistical significance was set at the 95% confidence level ($p<0.05$ for hypothesis testing). The patient was used as the experimental unit for analysis.

Differences in the prevalence of the microorganisms between Q-SRP and FM-SRP groups were analysed using the χ^2-test, except when expected counts were less than 5 where Fisher's exact test was used. The data were analysed for differences at each visit (BAS, R1, R2) and for comparison of changes with treatment (BAS to R1 and BAS to R2). For each group, the McNemar test was used to compare the detection frequency of specific organisms before and after treatment. The patient was scored positive for an organism if at least one out of the four sites harboured this organism.

Results

Figs. 1–5 depict the percentage of patients and the frequency of sites (from 1 to 4) positive for the five organisms tested. The prevalence of the five putative periodontal pathogens presented here is relatively consistent with the range of values reported by other studies (Ashimoto et al. 1996, Riggio et al. 1996, 1998, Meurman et al. 1997,, Umeda et al. 1998). Table 3 summarises the percentage of patients in each treatment group positive for the five putative periodontal pathogens. Marked reductions in all test bacteria were noted post-treatment, reaching statistical significance for *P. gingivalis*, *T. denticola* and *B. forsythus* in the Q-SRP group ($p<0.05$) and for the majority of the pathogens in the FM-SRP group ($p<0.05$). These improvements were maintained at the 6-month reassessment and this appeared to be more pronounced in the Q-SRP group.

When the two treatment groups were compared, a significantly higher percentage of Q-SRP patients was positive for *P. intermedia* at R1 compared with FM-SRP patients ($p<0.05$). No significant differences in the reduction of patients positive for this pathogen were found

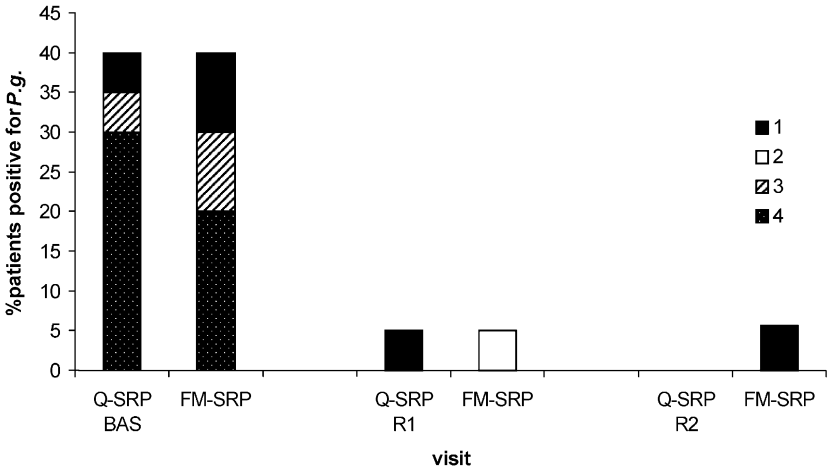

Fig. 1. Percentage of patients and the frequency of sites (from 1 to 4) that are positive for *Porphyromonas gingivalis* in each treatment group. Q-SRP = quadrant scaling and root planing; FM-SRP = full-mouth scaling and root planing; BAS = baseline; R1 = reassessment 1; R2 = reassessment 2.

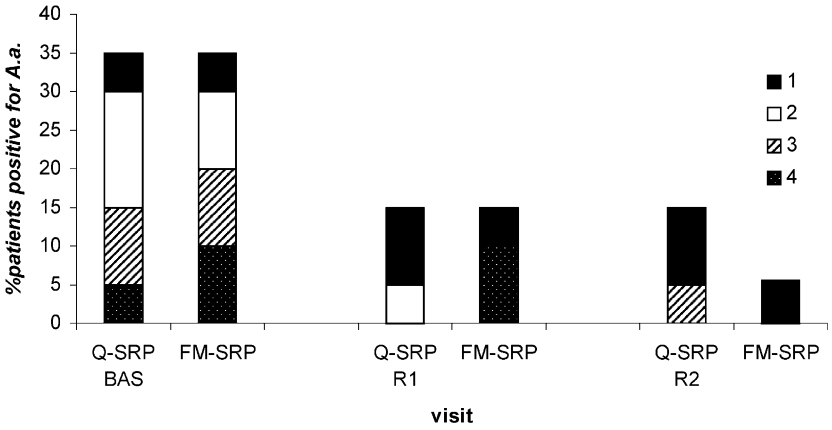

Fig. 2. Percentage of patients and the frequency of sites (from 1 to 4) that are positive for *Actinobacillus actinomycetemcomitans* in each treatment group. Q-SRP = quadrant scaling and root planing; FM-SRP = full-mouth scaling and root planing; BAS = baseline; R1 = reassessment 1; R2 = reassessment 2.

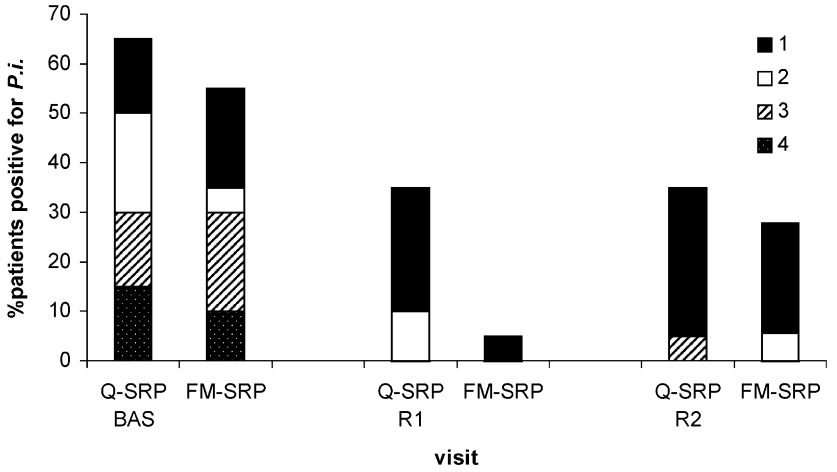

Fig. 3. Percentage of patients and the frequency of sites (from 1 to 4) that are positive for *Prevotella intermedia* in each treatment group. Q-SRP = quadrant scaling and root planing; FM-SRP = full-mouth scaling and root planing; BAS = baseline; R1 = reassessment 1; R2 = reassessment 2.

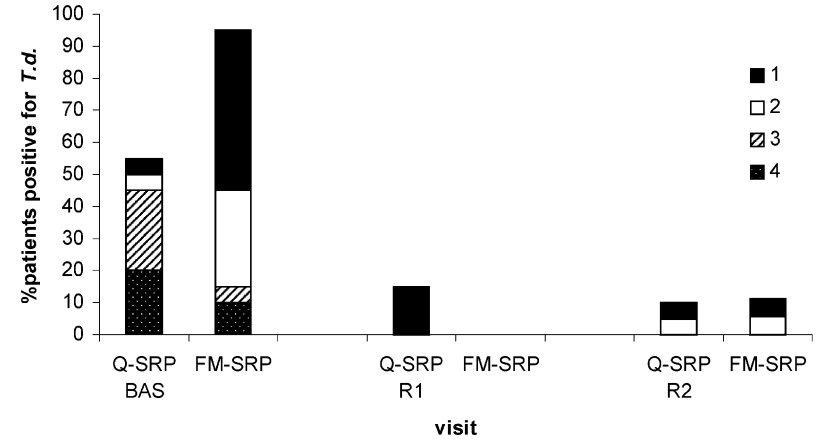

Fig. 4. Percentage of patients and the frequency of sites (from 1 to 4) that are positive for *Treponema denticola* in each treatment group. Q-SRP = quadrant scaling and root planing; FM-SRP = full-mouth scaling and root planing; BAS = baseline; R1 = reassessment 1; R2 = reassessment 2.

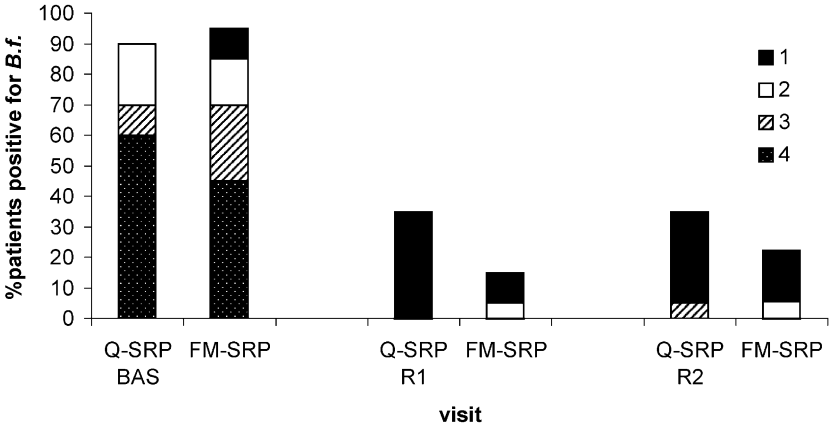

Fig. 5. Percentage of patients and the frequency of sites (from 1 to 4) that are positive for *Bacteroides forsythus* in each treatment group. Q-SRP = quadrant scaling and root planing; FM-SRP = full-mouth scaling and root planing; BAS = baseline; R1 = reassessment 1; R2 = reassessment 2.

between the two groups at R1 or R2 from baseline. On the contrary, a greater reduction in the patient prevalence for *T. denticola* was found for the FM-SRP group than the Q-SRP group at these time intervals ($p < 0.005$), but this is not a safe comparison given the significant difference in the detection frequency of this organism between the two treatment groups at baseline ($p < 0.01$).

Table 4 compares the prevalence of the test organisms between smokers and, non-smokers and, despite the differences in the detection frequencies of the bacteria between the two subgroups before and after treatment, these failed to reach statistical significance ($p > 0.05$).

Discussion

Both treatment modalities resulted in marked reductions in the percentage of subjects positive for the five putative periodontal pathogens, and this paralleled significant improvements in all clinical indices as was shown in a previously published study (Apatzidou & Kinane 2004), confirming the findings of previous reports (Simonson et al. 1992, Hafström et al. 1994, Shiloah & Patters 1994, Haffajee et al. 1997b). Despite the reductions in the percentage of patients positive for most of the bacteria tested following both treatment strategies, the majority of the organisms were still detected post-scaling but in significantly lower frequencies than baseline. This is in agreement with other reports which showed that scaling and root planing lowers the numbers of selected periodontal pathogens, but is unlikely to eliminate these species from any subject (Haffajee et al. 1997a, Cugini et al. 2000).

Data presented here showed that the microbial benefits of both treatment strategies were maintained over a 6-month period for the majority of the test species in a group of highly motivated patients with optimal plaque control. It has been shown that the improvement of the subgingival microflora after mechanical periodontal treatment is not dramatic and requires supportive periodontal therapy for its maintenance (Haffajee et al. 1997a). In contrast, the present study showed that conservative periodontal therapy resulted in marked microbiological and clinical improvements. Another study has demonstrated a decrease in the prevalence of *P. gingivalis*, *A. actinomycetemcomitans*

Table 3 Percentage of patients positive for the five putative periodontal pathogens before and after Q-SRP and FM-SRP

$N_{Q\text{-}SRP} = 20$ $N_{FM\text{-}SRP} = 20$	Baseline	R1	R2	Change (BAS–R1)	*p*-value	Change (BAS–R2)	*p*-value
Prevotella gingivalis							
Q-SRP	40.0	5.0	0.0	35.0	§	40.0	§§
FM-SRP	40.0	5.0	5.5	35.0	§	33.3	§
Actinobacillus actinomycetemcomitans							
Q-SRP	35.0	15.0	15.0	20.0		20.0	
FM-SRP	35.0	15.0	5.5	20.0		33.3	§
Prevotella intermedia							
Q-SRP	65.0	35.0*	35.0	30.0		30.0	
FM-SRP	55.0	5.0*	27.8	50.0	§§§	27.8	
Treponema denticola							
Q-SRP	55.0**	15.0	10.0	40.0***	§	45.0*	§§§
FM-SRP	95.0**	0.0	11.0	95.0***	§§§	83.3*	§§§
Bacteroides forsythus							
Q-SRP	90.0	35.0	35.0	55.0	§§§	55.0	§§§
FM-SRP	95.0	15.0	22.2	80.0	§§§	72.2	§§§

Note that at R2, two subjects in the FM-SRP group dropped out from the study.
*$p < 0.05$; **$p < 0.01$; ***$p < 0.005$; *p*-values represent differences between the Q-SRP and FM-SRP groups. §$p < 0.05$; §§$p < 0.01$; §§§$p < 0.005$; *p*-values represent longitudinal changes within each group.
Q-SRP = quadrant scaling and root planing; FM-SRP = full-mouth scaling and root planing; BAS = baseline; R1 = reassessment 1; R2 = reassessment 2.

Table 4. Percentage of smokers positive for the five putative periodontal pathogens before and after treatment

$N_{non\text{-}smokers} = 25$ $N_{smokers} = 15$	Baseline	R1	R2
Prevotella gingivalis			
non-smokers	36.0	8.0	0
smokers	46.7	0	6.7
Actinobacillus actinomycetemcomitans			
non-smokers	40.0	20.0	13.0
smokers	26.7	6.7	6.7
Prevotella intermedia			
non-smokers	68.0	24.0	34.8
smokers	46.7	13.3	26.7
Treponema denticola			
non-smokers	76.0	8.0	8.7
smokers	73.3	6.7	13.3
Bacteriodes forsythus			
non-smokers	96.0	20.0	30.4
smokers	86.7	33.3	26.7

No statistically significant differences were noted between non-smokers and smokers ($p > 0.05$).
Q-SRP = quadrant scaling and root planing; FM-SRP = full-mouth scaling and root planing; R1 = reassessment 1; R2 = reassessment 2.

and *P. intermedia* 12 months following scaling and root planing in the absence of supportive periodontal therapy (Shiloah & Patters 1996).

Several studies have shown that conventional periodontal therapy is ineffective at reducing the levels of *A. actinomycetemcomitans* (Kornman & Robertson 1985, Mombelli et al. 1994), and there is evidence that its present post-treatment is related to a compromised clinical outcome (Slots et al. 1986, Renvert et al. 1990). In addition, there are data demonstrating that the proportions of this organism increase after treatment (Renvert et al. 1990, Pedrazzoli et al. 1992), possibly as a result of higher sensitivity of the other subgingival species to debridement. The present study demonstrated that despite the reduction in the percentage of patients that possessed this pathogen after treatment, this was not found to be statistically significant, but the clinical significance of this finding is difficult to assess.

Comparison of Microbiological Responses Between Q-SRP and FM-SRP Groups

A greater reduction for *T. denticola* was seen in the FM-SRP group than the Q-SRP group at R1 and R2 from baseline. However, it is difficult to make conclusions from this finding, given the unbalanced distribution of *T. denticola* between the two treatment groups at baseline. FM-SRP resulted in significantly lower percentages of patients positive for *P. intermedia* at R1 than Q-SRP. In addition, the selected-site analysis of FM-SRP patients showed less PD reduction between baseline and R1 than those of Q-SRP patients (Apatzidou & Kinane 2004), indicating that at 6 weeks, post-scaling healing is still occurring and that the microbiological improvements may precede clinical changes.

In the present study, marked microbiological improvements for all test organisms were seen after both treatments. Nevertheless, except for the short-term differences between the two treatment groups at R1, there were no significant differences in the detection of any organism at the 6-month reassessment. Therefore, these data do not confirm previous findings of Quirynen et al. (2000), which showed that over a period of 8 months FM-SRP with and without the use of chlorhexidine resulted in a less pathogenic microflora compared with the common therapy of consecutive sessions of Q-SRP. That study also showed that at 8 months the number of CFU/ml of specific periodontal pathogens returned to pretreatment values only for the Q-SRP group. Although the present results are based on detection frequencies of selected pathogens, no such deteriora-

tion was noted for the Q-SRP group at 6 months.

The findings of our previously published study demonstrated that smoking and treatment approach (Q-SRP and FM-SRP) over a period of 6 months had a significant effect on PD and RAL of the selected sites (Apatzidou & Kinane 2004). Although this three-way interaction is complicated and difficult to interpret, it appears to be that Q-SRP non-smokers had the greatest PD reduction between baseline and R1 compared with the other subgroups (Q-SRP smokers, FM-SRP smokers and non-smokers). In order to clarify whether smoking had different effects on the subgingival microflora of subjects, the detection frequencies of specific organisms were compared between smokers and non-smokers. This analysis found no significant differences between smokers and non-smokers who were positive for the test species at baseline and at 6 months, and this finding is in agreement with previous reports (Stoltenberg et al. 1993, Darby et al. 2000, Boström et al. 2001).

The rationale behind the one-stage full-mouth disinfection was to prevent re-colonisation of the treated sites by periodontal pathogens from the remaining untreated pockets and other intra-oral niches and therefore prevent any recurrence of disease (Quirynen et al. 1995). The one-stage FM-SRP without chlorhexidine was shown to be equally efficacious with the one-stage full-mouth disinfection therapy, and more beneficial than the classical treatment of consecutive sessions of scaling and root planing in terms of clinical and microbiological responses (Quirynen et al. 2000). These findings indicate that the primary source of bacterial translocation is the periodontal pocket. The authors suggested that these considerations should be taken into account when split-mouth clinical trials are designed. Another study from the same laboratory demonstrated that the composition of the microflora around teeth significantly influenced the formation of the subgingival flora around implants, and this was more pronounced when teeth and implants were in the same jaw (Quirynen et al. 1996). This finding highlights the role of the periodontal pockets as bacterial reservoirs.

In the present study, FM-SRP was completed in 12 h instead of 24 h as described by Quirynen et al. (1995). Although the time interval between initiation and completion of treatment was lessened, and therefore the chances for bacterial re-colonisation were also reduced, no microbiological or clinical differences were found between the two treatment groups 6 months post-scaling to support the hypothesis of Quirynen et al. (1995). It must be stressed, however, that the participants in this study were highly motivated patients and practised a high standard of plaque control measures. Several studies showed that meticulous plaque control can affect the clinical and microbiological parameters in moderate (McNabb et al. 1992, Ximénez-Fyvie et al. 2000a) and deep pockets (Smulow et al. 1983, Dahlén et al. 1992, Hellström et al. 1996). These data emphasise the magnitude of oral hygiene measures on the subgingival environment. Haffajee et al. (2001) suggested that the beneficial effects of supragingival plaque control on the composition of the supra- and subgingival microflora decreased the risk of disease initiation or recurrence in maintenance periodontitis patients.

Taking these factors into consideration, although there is a potential for translocation of periodontal pathogens from one pocket to another, carefully performed plaque control measures and changes in the subgingival environment and the host response induced by treatment may prevent re-infection of sites, and thus relapse of periodontal disease. In conclusion, the current study failed to provide evidence that FM-SRP resulted in greater reductions in the detection frequency of suspected periodontal pathogens compared with Q-SRP over a 6-month period.

Acknowledgments

We thank Dr. D. Lappin for his technical support to complete this work and Mr. A. Lennon for growing *P. gingivalis*, *A. actinomycetemcomitans*, *P. intermedia* and *B. forsythus* bacterial cells.

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