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Clinical effects of scaling and root planing on untreated teeth

Adrian P. Pawlowski[1], Allen Chen[1], Beth M. Hacker[2], Lloyd A. Mancl[3], Roy C. Page[2], and Frank A. Roberts[2]

[1]Private Practice, Seattle WA, USA; [2]Department of Periodontics and [3]Department of Dental Public Health Sciences, University of Washington, Seattle, WA 98195, USA

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Abstract

Objective: The aim of this report is to examine whether scaling and root planing (SRP) in one area of the mouth may affect periodontal improvement in untreated areas in the same patient, possibly through systemic effects of treatment.

Material and Methods: Twenty patients diagnosed with generalized aggressive periodontitis were randomized into treatment ($n = 11$) and no treatment ($n = 9$) groups. Within the treatment group, three quadrants were treated by SRP at week 0, 3, 12, and 24, while a single experimental quadrant remained untreated throughout the study. The outcome for all teeth was assessed using clinical parameters, subtraction radiography, and pathogenic bacteria levels in the subgingival flora over the 24-week study period.

Results: Compared with sites in no treatment patients, the treated sites in the treated patients showed a 1 mm decrease in probing depth (PD) ($p < 0.01$) and a 0.5 mm increase in bone height ($p < 0.01$) by 24 weeks. In untreated sites within treated subjects, however, PDs tended to improve ($p = 0.09$) but at a reduced rate compared with treated sites. The levels of *Actinobacillus actinomycetemcomitans*, *Porphyromonas gingivalis*, and *Tannerella forsythensis* (*Bacteroides forsythus)* remained unchanged in untreated sites while levels of *Prevetolla intermedia* and *Treponema denticola* tended to decrease as compared with controls but did not reach significance.

Conclusions: This study indicates that untreated sites in treated periodontitis patients show a trend towards clinical improvement and exhibit reductions in some but not all periodontopathic bacterial species tested.

Key words: generalized aggressive periodontitis; microbiology; non-surgical therapy; scaling and root planing; treatment effects

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Thorough scaling and root planing (SRP) of patients with moderate-to-severe periodontitis results in a marked clinical improvement in the resolution of visible manifestations of inflammation, decreased probing depth (PD), and either a gain or stabilization of attachment levels (AL) (Haffajee et al. 1997, Morrison et al. 1980, Badersten et al. 1981, Hung & Douglass 2002). Although the traditional explanation for the clinical improvement associated with SRP result solely from the removal of bacterial deposits (Waerhaug 1978, Cobb 2002), alternate hypotheses should be entertained. One such hypothesis is that the bacteremia known to occur during SRP can activate the immune response (Sjöström et al., 1994), resulting in additional clinical improvement. Anecdotal evidence that SRP of some teeth may result in clinical improvement around untreated teeth in the same mouth could support this theory. Such observations are empirical and have not been subjected to rigorous conditions that either measure or control for placebo effects and observer bias.

Several investigators have evaluated the effect of SRP and other treatments on humoral immunity. In patients with periodontitis such simple procedures as chewing, tooth brushing, and the use of dental floss induce transient bacteremia that could thereby activate host defense mechanisms (Sconyers et al. 1973, Silver et al. 1977, Carroll & Sebor 1980, Li et al. 2000). While some studies have reported a small reduction in serum antibody titers to antigens of periodontopathic bacteria following therapy (Ishikawa et al. 1997, Naito et al. 1987), others report an initial post-therapy rise in specific antibody titers followed by a decrease within 1 year in some of the patients and a lack of any response in others (Ebersole et al. 1985, 1987, Aukhil et al. 1988, Murray et al. 1989, Darby et al. 2001). These conflicting results were explained in part when other researchers reported that the effects of treatment on the humoral immune response depended in part on the serostatus of the patient prior to treatment (Chen et al. 1991, Kinane et al. 1993, Mooney et al. 1995). Following therapy, most previously seronegative patients seroconverted with increases in both titer and antibody avidity (strength of binding), while seropositive patients manifested enhanced antibody

avidity and either no change or decrease in titers. Furthermore, relative to pre-treatment samples, sera from patients who had seroconverted were found to enhance in vitro neutrophil opsonization and bacteriocidal activity (Sjöström et al. 1994).

The effects of non-surgical debridement on the microbiological population in periodontal patients has been investigated (Haffajee et al. 1997, Mousques et al. 1980, Fujise et al. 2002). While personal oral hygiene can help manage the bacterial load, SRP results in significant bacterial reduction. *Actinobacillus actinomycetemcomitans*, *Porphyromonas gingivalis*, and *Tannerella forsythensis* (*Bacteroides forsythus*), *Prevetolla intermedia*, and *Treponema denticola* are all pathogenic bacteria implicated in periodontal disease. Several studies (Simonson et al. 1992, Lowenguth et al. 1995) discovered that *P. gingivalis* levels decreased after SRP, with *T. denticola* levels also being affected. Haffajee (Haffajee et al. 1997) monitored bacterial flora in 2900 subgingival plaque samples from 57 periodontitis patients prior to and 3 months after SRP. The results showed that mean levels of *T. forsythensis*, *T. denticola*, and *P. gingivalis* dropped significantly by 3 months. However, *A. actinomycetemcomitans* and other subgingival species did not show a significant post-therapy population difference.

In this study, we explored whether the treatment of periodontal disease-affected teeth by SRP produced any observed clinical and microbiological changes in untreated sites in the same patient. Changes in subgingival flora composition, radiographic bone height, and clinical parameters were monitored for treated and untreated teeth in periodontitis patients over a 24-week period. Understanding changes in assessment indices in periodontal patients will allow us to more completely define the benefits of SRP and explore the mechanisms behind these changes.

Material and Methods

Patient screening and clinical protocol

Patients diagnosed with untreated generalized aggressive periodontitis (GAP; formerly rapidly progressive periodontitis) attending the Regional Clinical Dental Research Center (RCDRC) and the Graduate Periodontics Clinic at the University of Washington as well as patients from the private practices of Seattle periodontists were recruited for the study using protocols approved by the UW Human Subjects Division. Patients between the ages of 18 and 35 who had been diagnosed with GAP (Ranney et al. 1982, Page et al. 1983, Armitage 2003), had a minimum of 20 teeth with at least one tooth in each posterior sextant, and at least one posterior sextant with a minimum of three natural teeth were selected for this study. The subjects also needed $\geqslant 5$ mm attachment loss around at least seven teeth with other than first molars and central incisors involved. Finally, the patients must have tested positive for at least one of the following putative periopathogens: *A. actinomycetemcomitans*, *P. gingivalis*, *T. forsythensis*, *P. intermedia,* or *T. denticola*. Patients were excluded if one or more of the following conditions applied: (1) periodontal treatment in the last 6 months; (2) antibiotic treatment in the last 3 months; (3) antimicrobial oral rinse use on a regular basis; (4) regular use of aspirin (ASA) or non-steroidal anti-inflammatory drugs in the last 6 months; (5) systemic diseases known to affect periodontal health; (6) women of child-bearing age not on birth control during the study; or (7) a history of smoking within the last 5 years.

Twenty-four subjects who met the diagnostic criteria were enrolled, and experimental and control quadrants were designated (Fig. 1). In the three control quadrants of each subject the posterior tooth with the deepest pocket was designated as the analysis site. In the experimental quadrant, the three teeth with the deepest pockets were designated as analysis sites. Sites could include the distal aspect of the canines, but third molars and the distal aspects of the lower second molars were excluded. Preference was given for sites that were not in the same interproximal space. Following site selection the subjects were randomized using a computer-generated randomization code. Subjects were assigned to either treatment ($n = 12$) or no-treatment groups ($n = 12$) without stratification.

Treatment and safety considerations

For subjects in the treatment group, all teeth in the mouth except those in the experimental quadrant were treated by SRP under local anesthetic using a combination of hand instruments (Hu–Friedy, Chicago, IL, USA). Treatment was performed by two investigators (A.P. and A.C.) and was standardized to the extent possible. Teeth in two quadrants were treated at baseline or as soon as practical thereafter, but in all cases before week 3; teeth in the third quadrant were treated at week 3. The three control quadrants were root planed again at week 12. At 6 months the entire mouth was treated, and the patients exited from the study. For patients in the no-treatment group, no treatment was provided for any teeth except for safety reasons until completion of the study. At that time each subject received full-mouth SRP, and they then were exited from the study.

For safety, full-mouth probing was recorded at baseline and at 3 and 6 months on all subjects. Any site, whether experimental or control, manifesting an increase in pocket depth of 3 mm or more was treated by SRP. Patients with non-responding sites were given systemic antibiotic therapy. Any patient receiving remedial therapy continued with the study protocol, and all study measures were collected as scheduled.

Clinical and radiographic data collection

Prior to treatment and at specified time points thereafter (see Table 1), plaque index (PI), bleeding on probing (BOP), PD, and relative AL were recorded for all test sites. Subgingival plaque sam-

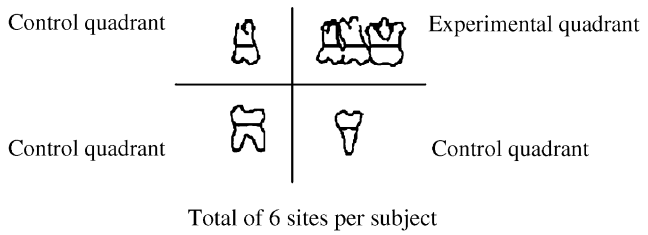

Fig. 1. Test site diagram. All subjects had control and experimental quadrants selected before randomization into treatment and no treatment groups. In the treatment group, the three control quadrants received scaling and root planing.

Table 1. Clinical protocol for therapy of treated quadrants and data collection for all sites in all subjects

Time	Clinical data	Plaque sample	GCF	Blood sample	Bite wings	SRP
week 0	×		×	×	×	half mouth
week 3	×	×	×	×		third quadrant
week 6	×	×	×	×		
week 9	×	×	×	×		
week 12	×	×	×	×	×	all three quadrants
week 16	×	×	×	×		
week 24	×	×	×	×	×	full mouth

GCF, gingival crevicular fluid; SRP, scaling and root planing

ples were harvested from the same locations. Gingival index (GI) was scored according to Loe & Silness (1963) while the PI was determined according to Silness & Loe (1964). PD was measured using a hand-held Michigan O probe to the nearest 1.0 mm. Relative AL was measured to the nearest 0.1 mm with an automated probe (Florida Probe Corp., Gainesville, FL, USA), making two passes with a third probing in cases where the two differed by more than 0.5 mm and averaging the two closest values. Data were collected by a hygienist (K.N.) who was blinded to the design and purpose of the study.

Standardized radiographs of each test site at baseline were taken 3 and 6 months using the technique described by Jeffcoat & Williams (1984). Measurements of alveolar bone height were made from computer-digitized images of the radiographs using a digital X-ray processing analysis method described previously (Jeffcoat & Williams 1984, Tonetti et al. 1993). The radiographs were coded to mask their origin and analyzed by one of the investigators (A.P.) at the University of Washington RCDRC Imaging Laboratory using image analysis software (Periopro version 3.1, University of Alabama at Birmingham, Birmingham, AL, USA) (Jeffcoat & Williams 1984). The distance from the cemento-enamel junction or restoration margin to the bony crest or the bottom of the intrabony defect (if present) was measured in the digitized images and converted to millimeters by the software.

Subgingival microflora was sampled by placing two filter paper strips (Proflow Inc., Amityville, NY, USA) into each test site for 30 s. The six paper points from the three sites in the treatment quadrants in the treated patients were combined to provide the necessary volume for analysis. The paper point samples from the untreated test sites (three sites) were also combined. Samples from the non-treatment groups were collected and combined in exactly the same manner as in the treatment group, as the collector was blinded to assignment. The samples were analyzed for bacterial presence using a DNA probe-based system (Affirm DP processor, MicroProbe Corp., Bothell, WA, USA) following the instructions of the manufacturer. Briefly, the assay involves an automated DNA probe sandwich system. The probes were bound to beads mounted on cards, exposed to plaque samples, developed in the processor, and the resultant color reflectance was read and compared with both a positive and negative control. Differences in reflectance between test samples and positive controls were converted to bacterial counts by comparison with standard curves with known quantities of *A. actinomycetemcomitans*, *P. gingivalis*, *T. forsythensis*, *P. intermedia*, and *T. denticola*.

Statistical analysis

For all oral data collected, a mean value was calculated for the three experimental sites and the three control sites in treated and all sites in no-treatment patients. The change in these mean values from baseline to each data gathering point was calculated and served as the basis for all subsequent statistical analyses. Where necessary, the data were logarithmically transformed prior to calculating change from baseline and mean values to satisfy assumptions of normality or equal variances, e.g. for bacterial levels that were highly skewed. We compared the mean values for treated teeth with untreated teeth in the same subject (treatment group) using paired *t*-tests. Mean values for treated and untreated sites in the treatment group were compared with mean values for sites in no-treatment group subjects using two-sample *t*-tests. All study data were subjected to statistical analyses both including (according to intent-to-treat principles) and excluding data from patients receiving remedial therapy.

Results

Twenty-four patients were initially enrolled in the study and randomized. Of this initial group, four patients did not start the protocol. The remaining 20 patients consisted of 11 treatment patients and nine no treatment patients comprising 12 Caucasians (seven male, five female), four Asian males, two African-Americans (one male, one female), and two Native American males. At week 9, a no treatment patient required remedial root planing at control tooth #14 and at week 12 another no treatment patient received remedial therapy on teeth #3, 4, 12, and 13 (neither experimental nor control teeth). A total of three patients received antibiotics during the course of the study. Two treatment patients were given erythromycin for respiratory infections (one at week 6, one at week 12) and one no treatment patient was given penicillin for an abscess between #3 and 4 (neither experimental nor control teeth) at week 12. All figures represent all subjects at all time points including those who needed antibiotic therapy or rescue treatment (intention-to-treat approach). Data were also re-analysed without subjects receiving remedial therapy, but there was no significant change other than reported below (data not shown).

Study sites treated by SRP in treatment group patients resulted in the expected clinical improvement relative to both baseline values and no treatment patient group values. By 12 weeks, there was a slight decrease in mean PD at sites in no treatment patients followed by stabilization over the remaining 12 weeks (Fig. 2). By week 24, the decrease in PD at treated sites in the treatment group was approximately 1 mm relative to sites in the no treatment group ($p < 0.01$). Although both treated and untreated sites within treated subjects showed virtually identical improvement in PD over the first 12 weeks of the observation interval, there was a divergence apparent at 24 weeks with untreated sites within treated subjects continuing to improve but at a reduced rate compared with treated sites ($p = 0.09$). Compared with sites in no

treatment subjects and baseline values, ALs at treated sites (Fig. 3) also improved by week 12 roughly by 0.5 mm ($p < 0.05$), which decreased by week 24 ($p = 0.13$). Mean AL at sites in subjects in the no treatment group remained unchanged during the first 12 weeks of the observation period or showed a trend toward attachment loss when subjects receiving antibiotics or remedial treatment were excluded (three in treatment group, one in no treatment group).

Changes in bone height followed the same ordering as those of relative AL (Fig. 4). Sites in no treatment subjects were observed to undergo a slight but continuous trend toward mean loss of bone height of 0.24 mm over the 24-week observation period. However, the treatment patients' treated site bone heights increased about 0.5 mm relative to baseline at both week 12 ($p < 0.05$) and week 24 ($p < 0.01$). Untreated sites in the treated subjects showed a trend toward continuous improvement in bone height over the entire 24-week observation period. Although the extent of improvement for untreated sites in treatment patients relative to sites in the no treatment group did not reach statistical significance at the $p < 0.05$ level, there was a strong trend for improvement in all of the outcome measures with p-values for PD, AL, and bone height at 24 weeks of 0.09, 0.11, and 0.18, respectively.

Other clinical parameters were not appreciably affected by SRP. PI values for the no treatment subjects were significantly higher ($p < 0.05$) as compared with treated controls by week 12 and to both treated and untreated sites in week 16 (Fig. 5). By week 24, however, the PI for no treatment subjects had returned to near baseline levels. Gingival index and BOP were used as measures of gingival inflammation. Values for treated sites relative to baseline and to sites in no treatment patients showed a trend toward improvement, but differences were not statistically significant (data not shown).

All patients exhibited detectable levels of all of the five periodontal pathogens being monitored. However, some bacterial counts were less than the 10^4 cells necessary for accurate DNA probe test measurements. Bacterial counts $\geqslant 10^4$ were observed for *A. actinomycetemcomitans* in three of 20 patients, for *T. forsythensis* in 18 of 20 patients, for *P. gingivalis* in 13 of 20 patients, for *P. intermedia* in 13 of 20 patients, and for *T. denticola* in 20 of 20 patients. Bacterial counts for *T. forsythensis*, *P. gingivalis*, *P. intermedia*, and *T. denticola* were all significantly reduced and remained at reduced levels over the entire 24-week observation period at treated sites in treatment group subjects (Figs 6 and 7). Significance values for the various time points for reduction in bacterial counts for treated sites relative to sites in untreated patients were at or below the $p < 0.05$ level for all of the species except *A. actinomycetemcomitans* (data not shown) where the sample size was minimal. Values for untreated sites in treatment group subjects tended to fall between values for treated sites and sites in no-treatment group subjects for *P. intermedia* and *T. denticola*, although they did not reach statistical significance. Lesser effects were observed for untreated sites in treated patients for *P. gingivalis*, *T. forsythensis*, and *A. actinomycetemcomitans*.

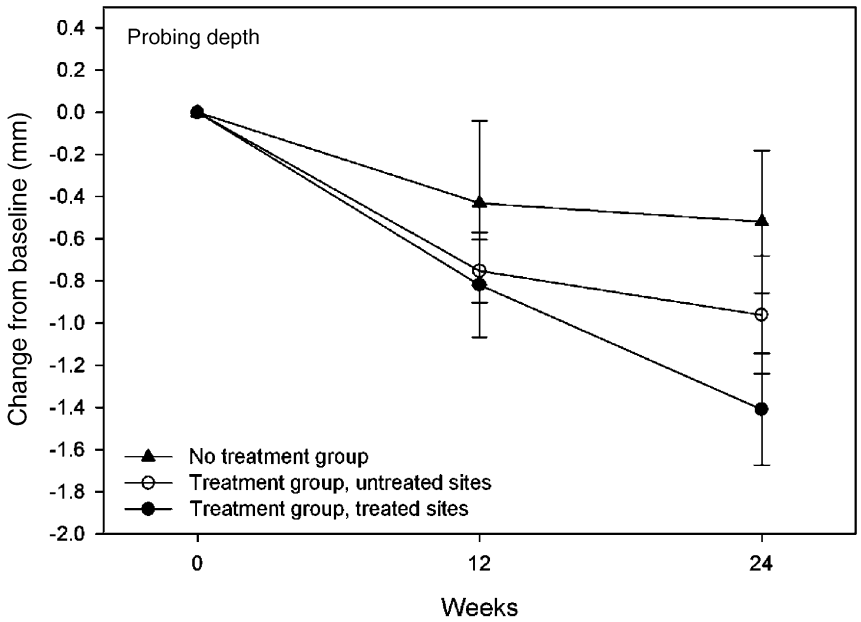

Fig. 2. Probing depth changes at baseline, 12, and 24 weeks in treatment ($n = 11$) and no-treatment groups ($n = 9$) (mean ± SEM).

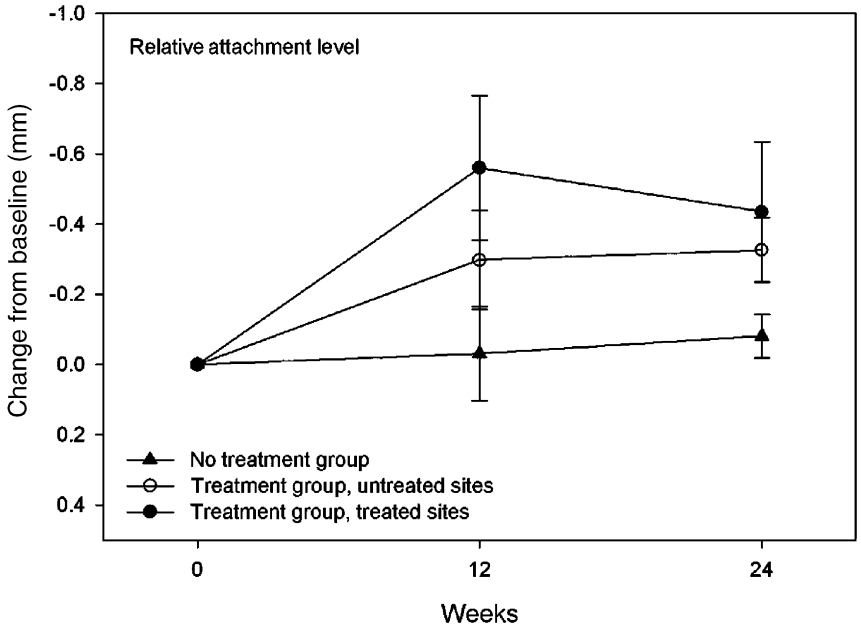

Fig. 3. Relative attachment level changes at baseline, 12, and 24 weeks in treatment ($n = 11$) and no-treatment groups ($n = 9$) (mean ± SEM).

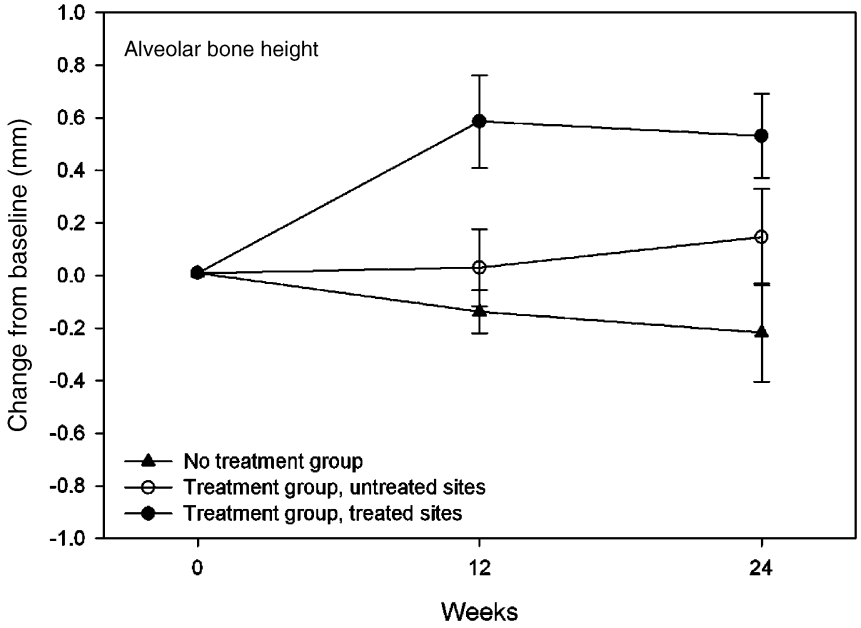

Fig. 4. Alveolar bone height changes at baseline, 12, and 24 weeks in treatment ($n = 11$) and no-treatment groups ($n = 9$) (mean ± SEM).

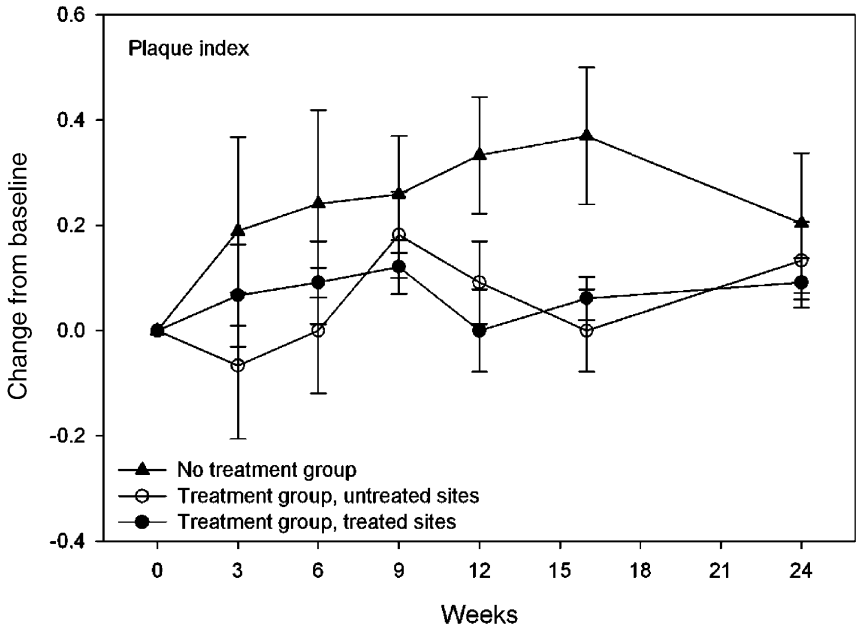

Fig. 5. Plaque index change every 3 weeks over the 24 weeks of the study in treatment ($n = 11$) and no-treatment groups ($n = 9$) (mean ± SEM).

Discussion

Many clinicians treating periodontal disease with SRP have made the observation that after treating several areas of the mouth, clinical improvement can be seen in untreated areas. Whether this observation was because of some enhancement of the host defense mechanism or to placebo effect is much difficult to answer, as there are many factors to consider. We selected subjects with GAP in order to maximize our ability to see change over the 24-week study intervals. Patients undergoing routine periodontal therapy are generally placed on strict oral hygiene programs and are expected to be highly motivated in their compliance by virtue of their involvement in therapy. In the present study, we attempted to control for these variables by making no effort to modify oral hygiene and by comparing the response of untreated teeth in treated subjects not only to treated teeth in the same subjects but also to teeth in subjects who were monitored but received no treatment. In this way, we were able to better manage the placebo effect.

Changes in mean AL showed a significant improvement at treated sites over the first 12 weeks of the study period followed by a trend toward slight regression over the remaining 12 weeks. Although it is difficult to interpret this trend given that the observed differences were not significant, we speculate AL gains observed at untreated sites within treated patients may maximize with 12 weeks of initial therapy followed by stabilization and failure to respond further despite additional therapy. This minimal trend toward AL loss after 12 weeks is consistent with clinical observations that support the clinical efficacy of the 3-month recall intervals in periodontal maintenance (Schallhorn & Snider 1981, Shick 1981).

Treated sites gained significant bone height from baseline to 12 weeks and then leveled out with no further change by 24 weeks. The loss of significance at the 24-week observation interval taken together with the increase in variability would suggest that between 12 and 24 weeks some sites had begun to regress slightly while others continued to gain.

Clinical conditions at untreated sites in treated patients also improved over the course of the study, but to a lesser extent than improvement at treated sites in the same patients. The pattern of improvement was the same for PD, AL, and bone height (Figs 2–4). Treatment did not significantly affect the GI or BOP of untreated teeth in the same mouth (data not shown) although for no-treatment patients, there was an increase in the PI through week 16 which then decreased to a level comparable with treated patients and untreated sites in treated patients.

Consistent with the work of others (Renvert et al. 1990, Haffajee et al. 1997, Shiloah et al. 1997, Takamatsu et al. 1999, Doungudomdacha et al. 2001, Fujise et al. 2002) SRP therapy in our patient population resulted in significant declines in bacterial counts harvested from treated sites in treated patients for *T. forsythensis*, *P. gingivalis*, *P. intermedia*, and *T. denticola*, but not for *A. actinomycetemcomitans*. No conclusions about the response of *A. actinomycetemcomitans* to therapy could be drawn since we detected threshold levels of *A. actinomycetemcomitans* in

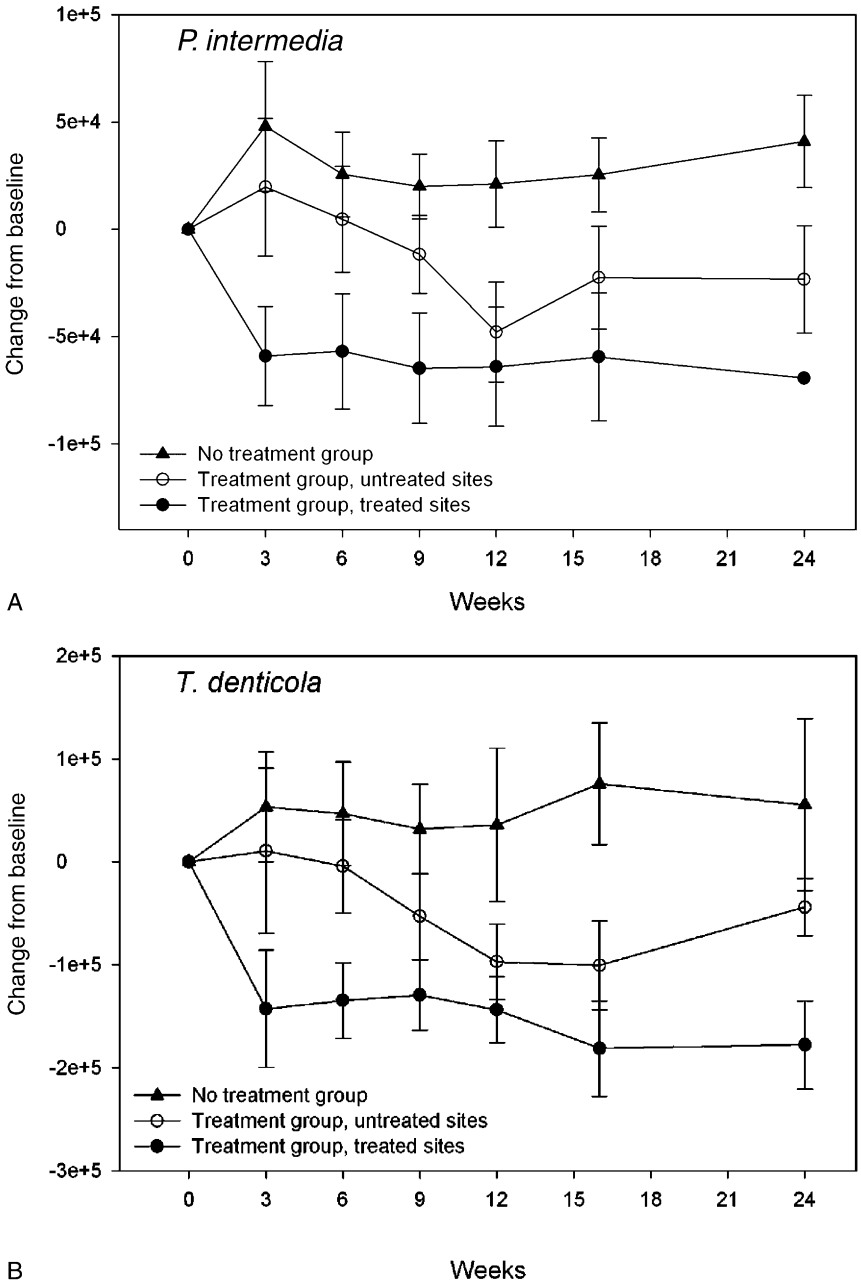

Fig. 6. Bacterial counts of (A) *Prevotella intermedia* and (B) *Treponema denticola* over 24 weeks in treatment ($n = 11$) and no-treatment groups ($n = 9$) (mean ± SEM).

only three subjects, two of whom were in the treatment group. The balance of our subjects all had low levels of *A. actinomycetemcomitans* which may reflect the age of the subject population, as *A. actinomycetemcomitans* is more often prevalent in juveniles with localized aggressive periodontitis (Zambon 1985). Bacterial counts at untreated sites in treated patients remained similar to those at sites in no treatment patients with the exception of *P. intermedia* and *T. denticola*. A trend was observed toward decreased mean bacterial counts at treated sites as well as untreated sites in treated patients following SRP therapy for some of the species (particularly *P. intermedia* and *T. denticola*). The ordering of the response to SRP in these species reflected the same pattern observed in the clinical parameters, such as PD. This may indicate a differential response among species to SRP therapy (Haffajee et al. 1997, Ximenez-Fyvie et al. 2000), although it is difficult to draw conclusions given the limitations of our sampling technique and sample size. Each individual's immune system may preferentially react to any of the periopathogens. It is possible that there was a correlation between serum antibody titer, avidity to a particular periodontal bacteria, and periodontal disease severity. Work by Lamster (Lamster et al. 1998) showed that *A. actinomycetemcomitans* and *P. gingivalis* produced different antibody responses in periodontitis patients. Both antibody titer and avidity to *A. actinomycetemcomitans* negatively correlated with the presence of the bacteria in patients, while *P. gingivalis* produced the opposite effect, with a positive correlation to higher bacterial infection.

This study found trends of improvement in untreated sites in the same mouth as treated teeth demonstrating a positive effect on untreated sites. However, there have been several studies examining factors associated with reinfection of periodontal sites from untreated sites following SRP. As an alternative to the traditional quadrant SRP (Q-SRP), full-mouth SRP (FM-SRP) has been explored as a means of decreasing the translocation of periodontopathogens in the mouth (Quirynen et al. 1999, 2001, 2003). In FM-SRP, SRP is performed on all quadrants in a 24 h period along with the use of chlorhexidine resulting in clinical and microbiological benefits that were reported to last longer and for a greater duration than Q-SRP treatment. Therefore, additional improvements may have been detected if all three quadrants had been debrided on the same day. Recently, experiments by Apatzidou et al. (Apatzidou & Kinane 2004, Apatzidou et al. 2004) were not able to detect a difference between FM-SRP and Q-SRP in either clinical or microbial measures. As both procedures seem to provide at least equal periodontal treatment efficacy, clinicians are now employing this additional option for treating periodontitis patients.

Although every effort was made to minimize methodological error during this study, the difficulty in achieving statistically significant differences between samples may be because of variances in data measurement. Potential sources of error include the difference in the angulation of radiographs, accuracy in measurement because of changes in inflammation, and normal fluctuations in bacterial counts. There were challenges in retaining subjects for 24 weeks because of patient compliance with the intensive clinical appointments, hence the relatively small study size. Any future studies of this type would

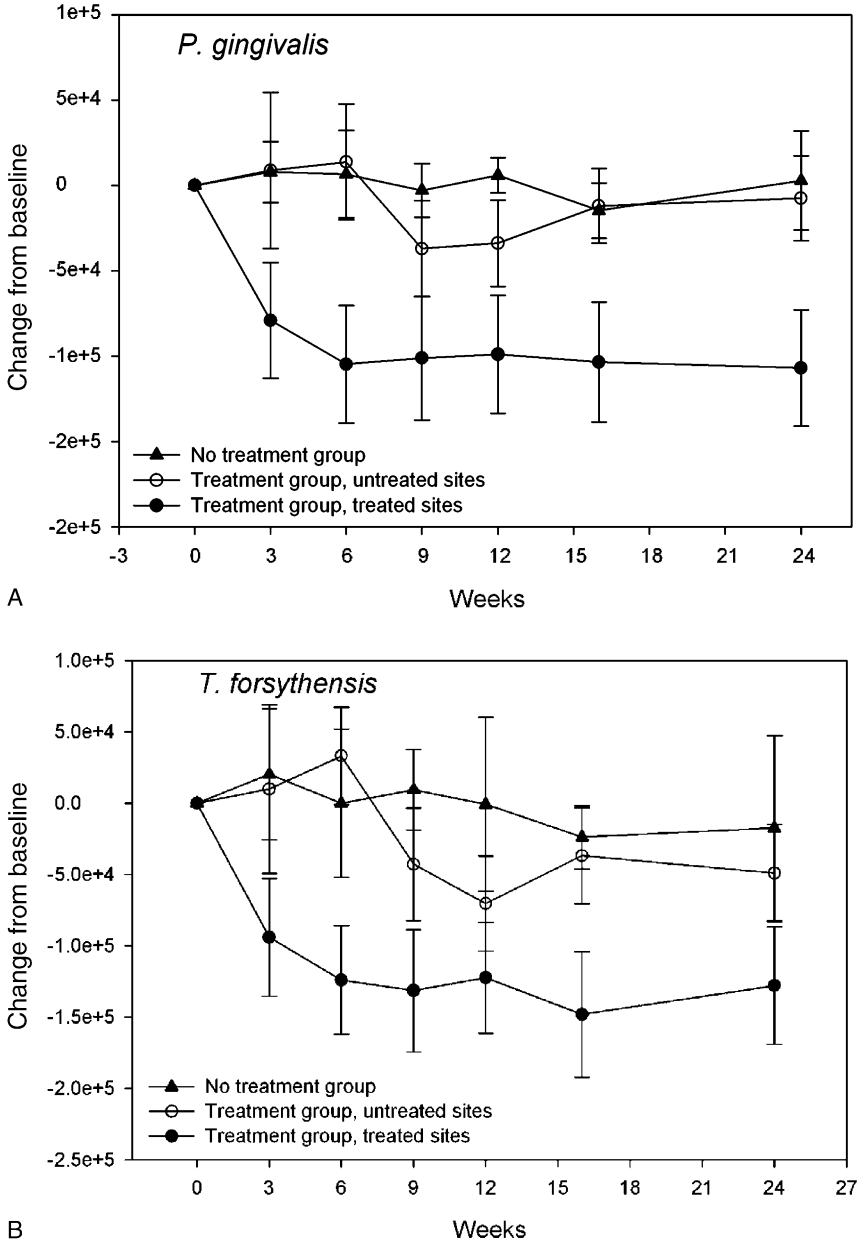

Fig. 7. Bacterial counts of (A) *Prevotella gingivalis* and (B) *Treponema forsythensis* over 24 weeks in treatment ($n = 11$) and no-treatment groups ($n = 9$) (mean ± SEM).

likely need to be larger, concentrate on measuring fewer parameters, include longer spacing between appointments, and extend for 9–12 months.

The aim of this paper was to examine the hypothesis that SRP therapy results in clinically detectable effects at untreated sites within the same mouth. The changes at untreated sites following SRP therapy suggest an increase in host resistance to periodontal breakdown, although the mechanism is not clear at this time. In the untreated sites of treated patients, we see approximately 30% improvement in clinical parameters including PD and bone loss. The systemic effect from root planing of the other quadrants causes an activation of the host response that may play a role in this improvement. We hope to shed light on the relationship between this immune effect and the role of systemic and local antibody responses as well as local inflammatory mediator expression through analysis of gingival crevicular fluid and serum specimens collected during this study. Data from this subject population support but do not definitively prove the hypothesis that SRP therapy results in clinically and radiographically detectable protective effects at untreated teeth in an otherwise treated mouth. The effect was manifested in gains in AL and bone height, decreases in PD, and in decreased counts in some bacterial species. Further studies employing larger sample sizes are warranted to demonstrate and quantify this effect conclusively.

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Address:
Frank Roberts
Department of Periodontics
University of Washington
Box 357444
Seattle, WA 98195-7444
USA
Fax: +1 206 616 7478,
E-mail: froberts@u.washington.edu