Disease Progression in Periodontally Healthy and Maintenance Subjects

R.P. Teles,* M. Patel,* S.S. Socransky,* and A.D. Haffajee*

Background: The aim of this study was to determine whether the rate of attachment loss in periodontally healthy subjects in a prevention regimen would differ from the rate of disease progression in periodontitis subjects enrolled in a maintenance program.

Methods: Fifty-five periodontally healthy subjects and 57 periodontitis subjects were clinically and microbiologically monitored at baseline and at 1, 2, and 3 years. Clinical parameters measured at six sites per tooth included bleeding on probing, visible plaque, probing depth, and attachment level. Subgingival plaque samples were taken from the mesiobuccal aspect of every tooth and were analyzed for the levels of 40 bacterial species using checkerboard DNA-DNA hybridization. The significance of differences over time in the clinical parameters was determined using repeated-measures analysis of variance, whereas the significance of differences between groups was determined using the unpaired t test. The Mann-Whitney test was used for microbial analyses, and P values were adjusted for multiple comparisons.

Results: Mean clinical parameters improved for both groups over time. By the end of the study, 4% of the sites in maintenance subjects lost ≥2 mm of attachment, whereas in the prophylaxis subjects only 1% of the sites lost ≥2 mm of attachment. Maintenance subjects lost attachment primarily at shallow buccal and lingual sites. The maintenance subjects harbored significantly higher levels of most test species throughout the study. The maintenance program did not reduce the levels of red complex species to those typical of healthy subjects.

Conclusions: Treated periodontitis subjects under maintenance displayed more rapid attachment loss than periodontally healthy subjects in a preventive regimen. The greater propensity to disease progression may be related to an elevated exposure to periodontal pathogens. J Periodontol 2008; 79:784-794.

KEY WORDS
Bacteria; biofilm; gingivitis; health; maintenance; periodontitis.

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One of the main goals of anti-infective periodontal therapy is to alter clinical and microbiologic parameters to those more typical of periodontal health. If this were achieved, it is likely that the rates of attachment loss in periodontitis subjects receiving periodontal maintenance would be similar to those detected in periodontally healthy subjects. Haffajee et al.1 compared the subgingival microbiota of periodontally healthy individuals, “well-maintained” elder subjects, and periodontitis subjects. They found that the subgingival microbiota of the well-maintained elder subjects was more similar to that of the periodontally healthy subjects than the periodontitis subjects. They suggested that periodontal therapy followed by regular professional maintenance and scrupulous self-performed home-care procedures can result in a subgingival microbiota in subjects treated for periodontitis that resembles that found in healthy subjects. However, some studies2-4 investigating the rate of disease progression in subjects enrolled in periodontal maintenance programs reported further deterioration of the periodontium in well-maintained periodontitis subjects. Although supportive periodontal therapy based on self-performed supragingival plaque removal, professional supragingival prophylaxis, and scaling and root planing of residual pockets can slow down the progression of periodontal disease, it may not halt the periodontal disease process. Conversely, studies5,6 on the effects of periodontal preventive
programs on the clinical parameters of periodontally healthy subjects indicated that these individuals had a minimal increased tendency to lose attachment. It was believed that studies directly comparing clinical and microbiologic characteristics of periodontitis subjects under maintenance to those of periodontally healthy subjects might reveal potential mechanisms that explain the higher susceptibility for disease progression in maintenance subjects. The goal of the present investigation was to test the hypothesis that the rate of periodontal attachment loss is lower in periodontally healthy subjects in a prevention program compared to treated periodontitis subjects on periodontal maintenance. Further, the microbial changes over time were examined to determine whether the rates of disease progression in the two groups were related to subgingival microbial profiles.

MATERIALS AND METHODS

Subject Population, Study Protocol, and Clinical Monitoring

Sixty-five periodontally healthy subjects and 62 chronic periodontitis subjects were recruited at The Forsyth Institute. The periodontally healthy subjects participated in a 3-year randomized clinical study evaluating the effects of different preventive protocols, whereas the periodontitis subjects were enrolled in a 3-year randomized clinical trial that examined the effects of different maintenance programs. Both studies started in 2000 and finished in 2005. Fifteen of the 127 subjects were lost to follow-up during the 3-year study period, five (8%) from the maintenance group and 10 (15%) from the prophylaxis group, resulting in 57 and 55 individuals in each group, respectively. The Institutional Review Board at The Forsyth Institute approved the study protocols, including the periodontal therapies used and the taking of clinical measurements and plaque samples. Each subject signed an informed consent before entering the respective studies. To be included in the study, the periodontally healthy subjects had to be ≥20 years of age, have ≥24 natural teeth, and have no probing depth (PD) >3 mm. The maintenance subjects had to be ≥20 years of age and have ≥15 natural teeth. For entry into the maintenance study, the subjects were required to have a minimum of four teeth with PD ≥5 mm. Subjects were excluded if they had any systemic condition that would influence the course of periodontal disease or treatment or any medical condition that would require antibiotic prophylaxis for routine dental procedures. Individuals who had taken antibiotics in the previous 3 months or who were pregnant or nursing were also excluded. The baseline clinical parameters of the study subjects are presented in Table 1.

Subjects were clinically and microbiologically monitored at baseline and at 1, 2, and 3 years. The clinical measurements were taken at six sites per tooth (mesio-buccal, buccal, disto-buccal, mesio-lingual, lingual, and disto-lingual). All teeth (excluding third molars) were measured at each visit (maximum of 168 sites per subject) by a calibrated examiner. Plaque accumulation (0 or 1), gingival redness (0 or 1), bleeding on probing (BOP, 0 or 1), PD (in millimeters), and clinical attachment level (CAL, in millimeters) were measured at each monitoring visit. PD and CAL were measured twice at each monitoring visit using a University of North Carolina probe.† Pairs of measurements were averaged and rounded to the nearest upper millimeter. The periodontally healthy subjects received dental prophylaxis performed by a dental hygienist as well as instructions in and reinforcement of proper home care procedures at the beginning of the study and every 6 months for 3 years. The maintenance subjects were provided supportive periodontal therapy (SPT) at baseline and every 3 to 6 months during the 3-year study period. The SPT included supra- and subgingival instrumentation, polishing with a rubber cup and a low-abrasive paste, and reinforcement in oral hygiene. No subgingival treatment was carried out within 3 months prior to the annual examinations.

Table 1.

Baseline Clinical Parameters of Maintenance and Prophylaxis Subjects

<table>
<thead>
<tr>
<th></th>
<th>Maintenance</th>
<th>Prophylaxis</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>62</td>
<td>65</td>
<td></td>
</tr>
<tr>
<td>Age (years; mean ± SD)*</td>
<td>57 ± 10</td>
<td>37 ± 11</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Males (%)†</td>
<td>52</td>
<td>31</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Smokers (%)†</td>
<td>13</td>
<td>5</td>
<td>NS</td>
</tr>
<tr>
<td>Sites with:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visible plaque (%; mean ± SD)*</td>
<td>60 ± 22</td>
<td>41 ± 17</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Gingival redness (%; mean ± SD)*</td>
<td>53 ± 21</td>
<td>33 ± 17</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>BOP (%; mean ± SD)*</td>
<td>22 ± 14</td>
<td>9 ± 7</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>PD (mm; mean ± SD)*</td>
<td>2.8 ± 0.4</td>
<td>2.2 ± 0.3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>CAL (mm; mean ± SD)*</td>
<td>3.3 ± 0.9</td>
<td>1.7 ± 0.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Missing teeth (n; mean ± SD)*</td>
<td>3.1 ± 2.9</td>
<td>0.5 ± 1.2</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

NS = not statistically significant.
* Unpaired t test.
† Fisher exact test.

† Hu-Friedy, Chicago, IL.
**Microbiologic Assessment**

Subgingival plaque samples were taken at baseline and at 1, 2, and 3 years from the mesio-buccal aspect of all teeth, excluding third molars. The subgingival plaque samples were collected using sterile Gracey curets after removal of supragingival plaque (if present). The samples were placed in separate microcentrifuge tubes containing 0.15 ml TE buffer (10 mM Tris-HCl; 1 mM EDTA, pH 7.6), and 0.1 ml 0.5 M NaOH was added. All samples were processed at The Forsyth Institute. Samples were individually analyzed for their content of 40 bacterial species using the checkerboard DNA-DNA hybridization technique. In brief, the samples were lysed, and the DNA was placed in lanes on a nylon membrane using a minislot. After fixation of the DNA to the membrane, the membrane was placed in a miniblotters, with the lanes of DNA at 90° to the lanes of the device. Digoxigenin-labeled whole-genomic DNA probes to 40 subgingival species were hybridized in individual lanes of the miniblotters. After hybridization, the membranes were washed at high stringency, and the DNA probes were detected using antibody to digoxigenin, conjugated with alkaline phosphatase. Signals were detected using a chemiluminescent substrate and analyzed using a computer-linked instrument that read the intensity of the fluorescence signals resulting from the probe-target hybridization. Two lanes in each run contained standards at the concentration of $10^5$ and $10^6$ cells of each species. The sensitivity of the assay was adjusted to permit the detection of $10^4$ cells of a given species by altering the concentration of each DNA probe. Signals were evaluated using a fluorimeter and converted to absolute counts by comparison with standards on the same membrane. Failure to detect a signal was recorded as zero. A total of 13,288 (3,322/visit) subgingival samples were evaluated for an average of 105 samples per subject (26.2 samples/subject/visit).

**Data Analyses**

Data analyses were performed using only data from complete cases. Fifty-seven subjects in the maintenance group and 55 individuals in the prophylaxis group had data for all four monitoring visits. The outcome variables evaluated in this study were the changes in mean clinical parameters from baseline to 1, 2, and 3 years. Values for each clinical parameter were averaged within a subject and then averaged across individuals in each group for each time point. The differences in mean clinical parameters between the maintenance and prophylaxis groups at baseline were determined using the unpaired $t$ test, whereas differences in the distribution of males and smokers were examined using the Fisher exact test. The significance of differences over time (baseline and 1, 2, and 3 years) within each group was determined using repeated-measures analysis of variance (ANOVA).

Changes in PD and CAL from baseline to 3 years were also described. The baseline PD and CAL for each site was subtracted from the corresponding 3-year site value. The resulting values were averaged within a subject and then across subjects in each group. The significance of differences between groups was determined using the unpaired $t$ test.

Sites were assigned to baseline PD categories $<3$, 3 to 4, and $>4$ mm to examine the changes in mean PD and CAL from baseline to 3 years at sites with different initial PDs. The mean changes in PD and CAL were averaged within a subject for each of the baseline PD categories and then averaged across all subjects within each PD category for each clinical group. The change in mean PD and CAL from baseline to 3 years was also determined at sites categorized according to site location: buccal, lingual, and interproximal. The changes in data for the different site locations were averaged within a subject and across subjects within each group. The significance of differences between healthy and maintenance subjects for the mean change in PD and CAL for each baseline PD category and each site location category was determined using the unpaired $t$ test.

The percentage of sites with loss of CAL $\geq 2.0$ mm at each time point was also determined for each clinical group. The significance of differences between healthy and maintenance groups in the percentage of sites with loss of CAL $\geq 2.0$ mm was determined using the unpaired $t$ test.

Microbiologic data available for each subject were the counts of each of the 40 test species for up to 28 subgingival plaque samples per subject at baseline and at 1, 2, and 3 years. The data for each species were expressed as counts $\times 10^5$ at each site, averaged within each subject, and then averaged across subjects at each time point separately. The significance of differences in mean counts for each species between the prophylaxis and maintenance subjects at each time point was determined using the Mann-Whitney test. All microbiologic analyses were adjusted for 40 comparisons.

**RESULTS**

**Baseline Examination**

The study involved 127 participants: 62 individuals had chronic periodontitis and were enrolled in a supportive periodontal maintenance program, and 65 subjects were periodontally healthy individuals. Points included.

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† Hu-Friedy.
§ Minislot device, Immunetics, Cambridge, MA.
¶ Miniblotters 45, Immunetics.
‖ AttoPhos, Amersham Life Sciences, Arlington Heights, IL.
¶ Storm Fluorimager, Molecular Dynamics, Sunnyvale, CA.
participating in a prophylaxis regimen. Table 1 presents the baseline characteristics of the two groups of subjects. The mean ages of the maintenance and prophylaxis subjects were 57 and 37 years, respectively ($P < 0.001$). The maintenance group also had a statistically significantly higher percentage of males compared to the prophylaxis subjects (52% versus 31%, $P < 0.05$). All periodontal clinical parameters, including the percentage of sites with plaque, redness, or BOP; mean PD; mean CAL; and the mean number of missing teeth, were statistically significantly greater in the maintenance group compared to the prophylaxis group ($P < 0.001$). A greater number of maintenance subjects were current smokers compared to prophylaxis subjects (13% versus 5%), but the difference was not statistically significant. The differences in baseline clinical parameters between groups remained statistically significant, even after adjusting for confounding variables such as age and the percentage of males, using analysis of covariance (data not shown).

Changes in Clinical and Microbiologic Parameters Over Time in Maintenance and Prophylaxis Subjects

Data analyses were performed using only data from complete cases: 57 subjects in the maintenance group and 55 individuals in the prophylaxis group. Figure 1 describes the changes in periodontal parameters over the 3 years of observation for maintenance and prophylaxis subjects. There was a statistically significant decrease in the percentage of sites with BOP ($P < 0.001$) over time for both groups. Mean PD decreased over the 3 years from 2.8 to 2.5 mm in the maintenance group ($P < 0.001$) and from 2.2 to 1.9 mm in the prophylaxis group ($P < 0.001$). There was a statistically significant reduction in mean CAL over time only in the prophylaxis group ($P < 0.01$).

The groups also differed with regard to the rates of tooth loss. During the 3 years of the study, the maintenance group lost 0.12 teeth/subject/year, whereas the prophylaxis group lost 0.02 teeth/subject/year.

Figure 1. Full-mouth mean ± SEM values for clinical parameters at baseline and at 1, 2, and 3 years in prophylaxis and maintenance subjects. Values for each parameter were measured at up to 168 sites in each subject. Significance of differences over time was tested using the repeated-measures ANOVA test. NS = not statistically significant.

Twelve of 57 (21%) patients in the maintenance group lost a total of 20 teeth, whereas only two of 55 (4%) individuals in the prophylaxis group lost a total of three teeth. In the maintenance group, the mean baseline PD and CAL for the teeth lost during the study were statistically significantly higher (4.1 and 5.4 mm, respectively; $P < 0.001$) than the mean values for teeth maintained throughout the study (2.8 and 3.2 mm, respectively). The extraction of 10 teeth in this group was due to their periodontal condition. In the prophylaxis group, the mean baseline PD and CAL values for teeth lost (2.4 and 2.6 mm, respectively) and teeth maintained (2.2 and 1.7 mm, respectively) were not statistically significantly different, and teeth were extracted because of prosthetic and orthodontic considerations.

The mean counts of the 40 test species in subgingival plaque samples at baseline and at 1, 2, and 3 years in maintenance and prophylaxis subjects are presented in Figure 2. At baseline (first panel), the maintenance subjects showed statistically significantly higher levels of the three red complex species *Porphyromonas gingivalis*, *Tannerella forsythia* (previously *T. forsythensis*), and *Treponema denticola* compared to the prophylaxis subjects. In addition, levels of the orange complex species *Eubacterium nodatum* and *Prevotella intermedia* were also statistically significantly elevated in the maintenance group. By year 2 (third panel), both groups demonstrated reductions in the mean levels of most species, and
the number of significant differences between the groups increased. Twenty of the 40 taxa examined exhibited statistically significant differences between the groups at year 2. Levels of members of the purple, yellow, green, orange, red, and other complexes were significantly elevated in the maintenance group. At the end of the study (last panel), the maintenance group still had statistically significantly higher levels of a number of bacterial species than the prophylaxis group, including *Actinomyces* spp. and several members of the orange and red complexes.

Mean total DNA probe counts decreased significantly between baseline and the end of the study from $37.7 \times 10^5$ to $20.9 \times 10^5$ ($P < 0.01$) and from $23.6 \times 10^5$ to $13.7 \times 10^5$ ($P < 0.001$) in the maintenance and prophylaxis groups, respectively. The significance of differences in the mean counts of each species from baseline to 3 years was tested in each clinical group. In the maintenance group, the species with statistically significantly lower levels by year 3 were *Capnocytophaga gingivalis*, *Capnocytophaga sp*., *Campylobacter gracilis*, *Campylobacter rectus*, *Fusobacterium periodonticum*, *Prevotella nigrescens*, *Gemella morbillorum*, *Leptotrichia buccalis*, and *Neisseria mucosa*. In the prophylaxis group, the species that were statistically significantly reduced over the 3 years were *Actinomyces naeslundii* genospecies 1, *C. gingivalis*, *C. sputigena*, *C. rectus*, *Campylobacter showae*, *E. nodatum*, *Parvimonas micra*, and *L. buccalis*.

Because one goal of periodontal therapy and maintenance is to convert the subgingival microbiota of periodontitis subjects to one approximating periodontal health, the mean microbial profile of the prophylaxis subjects at baseline was compared to the mean microbial profile of the maintenance subjects at year 3 (Fig. 3). Most species were reduced in the maintenance group to levels comparable to those in the periodontally healthy subjects of the prophylaxis.

**Figure 2.** Mean counts ($x 10^5$) of the 40 test species in up to 28 subgingival biofilm samples from prophylaxis and maintenance subjects at baseline and at 1, 2, and 3 years. The significance of differences between clinical groups was determined using the Mann-Whitney test. *P < 0.05; †P < 0.01; ‡P < 0.001. The species were ordered and grouped according to the complexes described by Socransky et al.26 A. gerencseriae = Actinomyces gerencseriae; A. israelii = Actinomyces israelii; A. naeslundii 2 = Actinomyces naeslundii genospecies 2; A. odontolyticus = Actinomyces odontolyticus; V. parvula = Veillonella parvula; S. gordonii = Streptococcus gordonii; S. intermedius = Streptococcus intermedius; S. mitis = Streptococcus mitis; S. sanguinis = Streptococcus sanguinis; A. actinomycetemcomitans = Aggregatibacter actinomycetemcomitans; C. ochracea = Capnocytophaga ochracea; E. corrodens = Eikenella corrodens; F. nucleatum ss. polymorphum = Fusobacterium nucleatum ss. polymorphum; F. nucleatum ss. vincentii = Fusobacterium nucleatum ss. vincentii; S. constellatus = Streptococcus constellatus; E. saburreum = Eubacterium saburreum; P. acnes = Propionibacterium acnes; P. melaninogenica = Prevotella melaninogenica; S. anginosus = Streptococcus anginosus; S. noxia = Selenomonas noxia.
However, even after 3 years in a supportive periodontal care program, the mean counts of the three members of the red complex, *P. gingivalis*, *T. forsythia*, and *T. denticola*, as well as *Streptococcus oralis*, *Fusobacterium nucleatum ss nucleatum*, *N. mucosa*, and *Treponema socranskii* in the maintenance group were statistically significantly higher compared to the baseline values in the prophylaxis group. Conversely, the mean counts of species such as *C. gingivalis* were statistically significantly lower in the maintenance subjects at 3 years compared to the baseline values for periodontally healthy subjects.

The microbial burden during the 3 years of the study was determined by averaging the counts of the 40 test species across all visits in the two groups (Fig. 4). The maintenance subjects had statistically significantly higher mean levels of 27 species throughout the 3 years of the study compared to subjects in the prophylaxis group.

### Changes in Clinical Parameters at Sites With Different Baseline PDs and Different Locations

The mean changes in PD and CAL between baseline and 3 years for the two groups at sites with baseline PDs <3, 3 to 4, and >4 mm are presented in Figure 5. In the maintenance group, there was an increase in mean PD of 0.05 mm and a loss of mean CAL of 0.20 mm at the sites with PDs <3 mm at baseline. Conversely, in the prophylaxis subjects, these sites had a decrease in mean PD of 0.17 mm and a gain in mean CAL of 0.08 mm. At sites with baseline PDs of 3 to 4 mm, both groups showed a decrease in mean PD.
(0.39 and 0.60 mm for maintenance and prophylaxis groups, respectively) and an improvement in mean CAL (0.26 and 0.48 mm for maintenance and prophylaxis groups, respectively) over the 3 years of the study. In the maintenance group, the sites with baseline PDs >4 mm showed a decrease in mean PD of 1.16 mm and a gain in mean CAL of 1.01 mm.

Figure 6 presents the mean (±SEM) change in PD and CAL from baseline to 3 years at different site locations. There were statistically significant differences between the groups with regard to changes in PD and CAL at buccal and lingual sites, whereas the differences at the interproximal sites were not statistically significant between the groups. The loss of attachment in the maintenance group occurred primarily at buccal and lingual sites. Because there was a reduction in mean PD at these sites, for the most part, attachment loss seems to have occurred as a result of recession of the gingival margin.

**Figure 6.**
Mean counts (×10^5) of the 40 test species averaged across all visits in the prophylaxis and maintenance subjects. The significance of differences between clinical groups was determined using the Mann-Whitney test. *P <0.05; †P <0.01; ‡P <0.001. The species were ordered and grouped according to the complexes described by Socransky et al. A. gerencseriae = Actinomyces gerencseriae; A. israelii = Actinomyces israelii; A. naeslundii 1 = Actinomyces naeslundii gerospecies 1; A. naeslundii 2 = Actinomyces naeslundii gerospecies 2; A. odontolyticus = Actinomyces odontolyticus; V. parvula = Veillonella parvula; S. gordonii = Streptococcus gordonii; S. intermedius = Streptococcus intermedius; S. mitis = Streptococcus mitis; S. sanguinis = Streptococcus sanguinis; A. actinomycetemcomitans = Aggregatibacter actinomycetemcomitans; C. ochracea = Capnocytophaga ochracea; E. corrodens = Eikenella corrodens; F. nucleatum ss. polymorphum = Fusobacterium nucleatum ss polymorphum; F. nucleatum ss vincentii = Fusobacterium nucleatum ss vincentii; S. constellatus = Streptococcus constellatus; E. saburreum = Eubacterium saburreum; P. acnes = Propionibacterium acnes; P. melaninogenica = Prevotella melaninogenica; S. anginosus = Streptococcus anginosus; S. noxia = Selenomonas noxia.

**DISCUSSION**
The present investigation compared the longitudinal clinical and microbial changes in periodontally healthy
subjects enrolled in a preventive regimen (prophylaxis group) for 3 years to the changes in the same parameters in periodontitis subjects in a 3-year supportive periodontal maintenance program (maintenance group). The maintenance and prophylaxis groups showed improvements in most clinical parameters, including reductions in the mean percentage of sites with BOP, mean PD, and mean CAL, over time. Although such changes were small, for the most part, the supportive periodontal regimens and the preventive programs were successful in stabilizing the clinical status and preventing loss of attachment. However, there were clear differences between the two groups in the rate of periodontal disease progression over the 3 years of the study. The maintenance subjects lost 0.12 teeth/subject/year, whereas the rate for the prophylaxis group was 0.02 teeth/subject/year. The rate of tooth loss in our periodontal maintenance group is comparable to other reports of annual tooth loss rates for periodontitis patients under maintenance and lower than the rates described for untreated populations. Our data also indicated that the periodontal condition of the extracted teeth was the primary cause of tooth loss in the maintenance group. Teeth that were lost during the trial had statistically significantly higher mean baseline PD and CAL compared to the mean baseline values for teeth maintained throughout the study. In fact, 10 of 20 teeth lost in the maintenance group were removed because of periodontal problems. Conversely, in the prophylaxis group, the mean baseline values of PD and CAL for the teeth that were extracted during the study did not differ significantly from the mean values for the other teeth. Reasons for tooth extractions were related to prosthetic and orthodontic treatments.

The maintenance subjects also had a statistically significantly higher percentage of sites losing ≥2 mm of clinical attachment than the prophylaxis group at all follow-up visits. By the end of the study, 4% (365 of 9,088 sites) of the sites in the maintenance group and 1% (137 of 10,683 sites) of the sites in the prophylaxis group had lost ≥2 mm of attachment. When the sites were characterized according to baseline PD, the shallow sites in the maintenance group showed an increase in mean PD and a mean loss of CAL, whereas the same category in the prophylaxis group had a mean gain in CAL. This observed trend for shallower sites at baseline to display a higher rate of disease progression compared to initially deeper sites in the maintenance subjects.
is consistent with several previous reports\textsuperscript{14-18} on the clinical changes that occur during maintenance.

By examining the changes in buccal, lingual, and interproximal sites separately, it was observed that, on average, buccal and lingual sites in the maintenance group lost attachment despite a mean pocket reduction at those sites, suggesting that recession of the gingival margin was the primary mechanism of attachment loss. Therefore, disease progression tended to occur primarily in the shallow buccal and lingual sites of the maintenance subjects. Recession of the gingival margin at buccal and lingual surfaces has been described as a consequence of trauma induced by zealous self-performed tooth cleaning.\textsuperscript{4,15,19-21} However, the same phenomenon affected a much smaller number of sites in the prophylaxis subjects. Longitudinal studies\textsuperscript{5,6} also demonstrated that gingivitis subjects show a low tendency to develop recession. A possible explanation for the greater recession of the gingival margin in maintenance subjects lies in the statistically significantly greater gingival recession present at baseline at buccal and lingual surfaces in the maintenance group compared to the prophylaxis group (data not shown). This preexisting marginal recession might have resulted in a thinner gingival margin that was more susceptible to mechanical trauma and more prone to further recession.\textsuperscript{22}

The observed trend for further improvements in mean PD and mean CAL demonstrated by the maintenance subjects attests to the efficacy of the supportive periodontal therapy and was in accord with other reports on the outcome of carefully executed maintenance programs. Serino et al.\textsuperscript{2} examined the clinical outcome in 64 subjects treated for “advanced periodontal disease” with surgical and non-surgical mechanical therapy. Patients were enrolled in a maintenance program and were reexamined after 1, 3, 5, and 13 years of supportive periodontal therapy. As observed in the present study, the investigators detected loss of attachment in shallow pockets, whereas deeper sites showed clinical attachment gain, at least during the first 5 years of follow-up. Axelsson et al.\textsuperscript{23} recently described the clinical outcome of 30 years of periodontal maintenance in 257 subjects; with the exception of buccal sites, the subjects had no signs of attachment loss. The mesial sites had a mean gain of clinical attachment between 0.3 and 0.5 mm during the observation period. Tonetti et al.\textsuperscript{3} reported on the changes of residual pockets in treated periodontal subjects during supportive maintenance therapy and described an increase in the number of new pockets of 4 to 5 mm over time. Conversely, the numbers of pockets that were 6 to 7 mm and >8 mm remained relatively stable during maintenance, suggesting that the new pockets were derived from previously shallow sites. Figure 8 illustrates the fate of sites categorized into three baseline PD categories over the 3 years of the current study. The shallow sites demonstrated a trend toward deepening (11% of these sites became deeper), whereas the intermediate and deep sites demonstrated a clear trend toward reductions in PD (36% and 64% of intermediate and deep sites became shallower, respectively).

Other longitudinal studies\textsuperscript{2,4,17} that followed subjects on maintenance demonstrated that clinical improvements obtained during initial periodontal therapy remained relatively stable thereafter. There were no further gains in attachment during maintenance, and there was a small trend for further deterioration of the periodontium over time. There are several possible explanations for the discrepancies among the findings of these studies and the current investigation. First, differences in patterns of disease progression during maintenance can be affected by differences in the level of residual disease after initial therapy. This concept was demonstrated by Rosling et al.,\textsuperscript{4} who described different rates of attachment loss during maintenance therapy between subjects with “normal susceptibility” and “high susceptibility” to
periodontal disease. Another important variable when assessing the outcome of maintenance studies is the length of time that subjects have been on supportive periodontal care. Tonetti et al. found a statistically significant association between the time on maintenance and an increase in the number of bleeding pockets and tooth loss. Other investigators reported that the deterioration of the periodontal status of subjects on maintenance tended to occur only after prolonged periods of time in a supportive periodontal care program.

Both clinical groups showed statistically significant reductions in the total mass of subgingival biofilms as measured by the total DNA probe counts. Although there were differences in the species affected in the two groups, the maintenance and prophylaxis regimens resulted in statistically significant reductions primarily in members of the green and orange complexes. Members of the red complex were noticeably less affected by the preventive and maintenance programs; the levels of these species were not significantly reduced over time in either group. This was not surprising, considering the extremely low levels in the prophylaxis subjects at baseline and the relatively low levels of these species in the maintenance group. Compared to previous data generated in our laboratories, the levels found at baseline in the maintenance group corresponded to approximately half of the levels found in untreated chronic periodontitis subjects.

The higher rate of disease progression in the maintenance subjects was accompanied by a statistically significantly higher microbial burden of 34 of the 40 test species over the 3 years (Fig. 4). Even after 3 years of supportive therapy, the maintenance subjects had statistically significantly higher levels of the three members of the red complex and other expected pathogenic species, such as *T. socranskii*, compared to the baseline levels of these microorganisms in the prophylaxis group (Fig. 3). Other investigators also reported that the mean microbial proportions in treated periodontitis subjects did not return to levels present in periodontally healthy subjects. Hence, the maintenance therapy did not reduce the levels of all subgingival pathogenic species to those usually found in periodontal health. This may have increased the propensity for loss of attachment in the maintenance group. As noted in a study on maintenance subjects of longer duration, it is also conceivable that exposure to this “elevated” level of subgingival pathogenic species for longer periods of time might result in further loss of attachment.

**CONCLUSIONS**

The results from the present study supported the notion that treated chronic periodontitis subjects on periodontal maintenance have a higher rate of periodontal deterioration than periodontally healthy subjects in a prevention program. Over the 3 years of the study, this elevated susceptibility to periodontal disease progression in the maintenance subjects was associated with a higher exposure to subgingival biofilm species, particularly members of the red complex.

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