Longitudinal changes in gingival crevicular fluid after placement of fixed orthodontic appliances

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Introduction: Bacterial plaque is an etiologic factor in the development of gingival inflammation and periodontitis. The presence of orthodontic bands and brackets influences plaque growth and maturation. The purposes of this research were to monitor microbiologic and periodontal changes after placement of orthodontic attachments over a 1-year period and to link these changes to alterations in cytokine concentrations in the gingival crevicular fluid (GCF). Methods: This longitudinal split-mouth trial included 24 patients. Supragingival and subgingival plaque composition, probing depth, bleeding on probing, and GCF flow and composition were assessed at baseline (Tb) and after 1 year (T52). A statistical comparison was made over time and between the banded and bonded sites. Prognostic factors for the clinical reaction at T52 in the GCF at Tb were determined. Results: Between Tb and T52, the pathogenicity of the plaque and all periodontal parameters increased significantly, but intersite differences were not seen, except for bleeding on probing. The cytokine concentrations in the GCF did not differ significantly between the sites or between Tb and T52. The interleukin-6 concentration in the GCF at Tb was a significant predictive value for the GCF flow at T52 (P < 0.05). The same relationship was found between the interleukin-8 concentration at Tb and the increase in probing depth at T52 (P < 0.05). Conclusions: Interleukin-6 and interleukin-8 concentrations before orthodontic treatment were shown to be significant predictive factors for some potential inflammatory parameters during treatment. (Am J Orthod Dentofacial Orthop 2011;139:735-44)

It is well established that bacterial plaque is the primary etiologic factor in the development of gingival inflammation and periodontitis.1 The quantity and the quality of plaque are known to be influenced by many factors, including surface characteristics.2-4 Especially surface roughness and high surface free energies were found to be positively correlated with plaque growth and maturation.5 Gingival inflammation is known to further increase this.5-8 In addition to the total amount of bacteria, the ratio between the aerobic and anaerobic bacteria is also an important marker for plaque pathogenicity.9 The placement of orthodontic bands and brackets influences plaque growth and maturation because of the above-mentioned factors. Significant differences in biofilm formation on bonded teeth compared with control teeth were reported.10 Most studies reporting on gingival changes after bracket placement suggested only temporary reversible periodontal changes.11,12 Another study, however,
reported significant attachment loss during orthodontic treatment.  

Another way of studying the changes during orthodontic therapy is by analysis of the composition of the gingival crevicular fluid (GCF). GCF reflects the immune and inflammatory reactions from host–parasite interactions and biomechanical stresses. It is a noninvasive method, and, until now, many substances involved in the inflammatory process and produced by the periodontal ligament cells in sufficient amounts to diffuse into the GCF have already been studied. Giannopoulou et al compared the GCF composition of orthodontic patients with that of nonorthodontic controls. However, the wide intersubject differences in GCF composition made reasonable comparisons difficult and warrant prospective intra-subject studies. Among many inflammatory and immune mediators identified in GCF, cytokines have attracted particular attention.

Interleukin-6 (IL-6) is known to be a major modulator of inflammation in chronic local inflammatory reactions. It regulates the immune cell recruitment in the transition from the acute (recruitment of neutrophils) to the chronic (recruitment of monocytes) form of inflammation. Interleukin-8 (IL-8), produced by various cells (polymorphonuclear leukocytes, monocytes, macrophages, and fibroblasts), plays a key role in the accumulation of leukocytes at the sites of inflammation. Interleukin-10 (IL-10) is an inhibitor of inflammation that downregulates the production of proinflammatory cytokines, including IL-1β and TNF-α. IL-10 also plays a role in inhibiting the proliferation of fibroblasts and neutrophils.

Our aims in this study were to monitor the microbiologic and clinical periodontal changes after placement of orthodontic attachments over a 1-year period and to link these changes to alterations in cytokine concentrations in the GCF. The null hypothesis was that the cytokine concentrations before orthodontic treatment have no predictive value for the clinical periodontal reaction during treatment. The dependant variables were change in probing depth, number of sites bleeding on probing, and GCF flow.

MATERIAL AND METHODS

Twenty-four subjects (10 boys, 14 girls) aged 14.6 ± 1.1 years (mean ± SD) referred to the postgraduate clinic of the Department of Orthodontics of the Catholic University of Leuven in Belgium were included in the study. The subjects and their parents were given a written explanation of the background of the study, its objectives, and their involvement. After screening for suitability and after good comprehension of the protocol, the parents all gave their written informed consent. This study was approved by the ethical committee of the same university. The patients were selected if they fulfilled the following inclusion criteria: no smoking, no orthodontic treatment with extractions, no extensive dental restorations or adhesive fixed partial dentures, a sulcus bleeding index of less than 0.3, no periodontal disease, and no use of antibiotics during or up to 4 months before the study. The patients were asked whether they were right- or left-handed; in the right-handed patients, the right quadrants might be brushed more thoroughly, leading to a healthier gingival condition. All patients were right-handed. Fourteen subjects (6 boys, 8 girls) of the 24 subjects were treated with headgear (headgear group) and received bands on the maxillary first molars for 18 weeks before bonding the brackets to the remaining maxillary teeth. In the headgear group, it was possible to make intrasubject comparisons between the bonded and banded sites. The other 10 subjects were treated with brackets only (nonheadgear group).

The study had a longitudinal prospective design. During the study period, the subjects were periodontally analyzed 2 or 3 times (Table I). The first time, at minus 18 weeks (T−18), was to record the status praesens of the periodontium, to sample the subgingival and supragingival plaque, and to place the molar bands. At the second visit, after 18 weeks (T0), the measurements and samples were repeated, and brackets were bonded on the remaining maxillary teeth (headgear group). For the nonheadgear group, this was the first visit. At T0, the initial orthodontic archwire was also placed (0.014-in nickel-titanium alloy). T−18 was considered the baseline for the banded sites, and T0 was the baseline for the bonded sites. The orthodontic therapy was performed by using Generus full edgewise brackets with an 0.018-in slot (GAC International, Bohemia, NY). The extraction therapies were excluded; after 1 year (T52) of orthodontic treatment, all subjects were in the treatment phase with 0.016 × 0.022-in stainless steel wires. At T52, the final measurements were made. The 1.4 and 1.6 were sampled; for the headgear groups, the first molar was a banded site, and the first premolar was a bonded site. For the nonheadgear group, both teeth were bonded. Standardized oral hygiene instructions with an orthodontic toothbrush (Oral-B, Kirkland, Quebec, Canada) with the Bass technique and a single tufted brush (Oral-B) were instructed. Interdental cleaning was taught with extra-fine interdental wood sticks (Oral-B). The youngsters were able to use these wood sticks also after placement of the bands, brackets, and orthodontic wires. The patients were told to always brush their teeth for 3 minutes. The hygiene protocol was taught with a model; then the subjects’ brushing was analyzed and improved by a clinician (J.v.G.) to achieve good comprehension. At each visit, the teeth were stained with erythrosine disclosing solution (4% erythrosine in water solution) to show the patients how to remove the remaining plaque.
For band placement at T–18, only the patients from the headgear group received orthodontic bands on their maxillary first molars (Table I). The teeth were pumiced with a rubber cup, the orthodontic bands were fitted, and the correct size was selected. The gingival band margins were trimmed to be placed supragingivally. After disinfecting the bands with alcohol and drying them, Transbond Plus glass ionomer cement (Multi-Cure Ionomer Orthodontic Band Cement, 3M Unitek, Monrovia, Calif) was mixed according to the manufacturer’s instructions. The bands were placed, and any excess cement was removed from the occlusal and cervical margins of the bands and the teeth. All band selections and cementations were performed by the same clinician (J.v.G.). The cement was light cured (QHL75 halogen curing light, Dentsply, Addlestone, Surrey, United Kingdom) for 30 seconds from the occlusal side. The preformed headgear was adjusted, and the patients were instructed to wear it for 14 hours a day.

At T0, all patients received brackets in the maxilla (Table I). For the nonheadgear group, these were the first orthodontic attachments in the mouth. The headgear group received brackets on all remaining teeth in the maxilla. The teeth were pumiced with a rubber cup, and the quadrant to bond was isolated with cotton rolls and saliva suction. A 1-step adhesive (Transbond Plus Self Etching Primer, 3M Unitek) was applied with a microbrush, and the excess was blown away with dry air in the incisal-occlusal direction to prevent contact with the gingiva. The composite bonding material (Transbond Plus color change adhesive, 3M Unitek) was applied to the bracket base, the bracket was pressed firmly onto the enamel surface, and any excess adhesive was removed. Then the composite was light cured (Dentsply QHL75 halogen curing light, Dentsply) for 30 seconds from the occlusal and gingival directions. After placement of the brackets, an initial nickel-titanium orthodontic wire was placed and ligated to the brackets with elastic ligatures.

After isolating the teeth from the saliva with cotton rolls and gently drying them to prevent contamination, the supragingival plaque was carefully removed with sterile curettes without traumatizing the gingiva, because this would increase the production of crevicular fluid. The supragingival plaque was transferred into flip-capped vials containing 2.0 mL of prereduced transport medium to be processed within 2 hours. Each sample was homogenized by vortexing for 30 seconds and coded. The coding was not revealed until all analyses were completed, leading to blinded microbiologic analyses.

The subgingival plaque was sampled after collecting the GCF without traumatizing the crevice, because this would increase the GCF flow. To sample the subgingival plaque per tooth, 6 sterile medium paper points (RoekoA, Roeko, Langenau, Germany) were inserted into the sulcus (3 mesially, 3 distally) and kept in place for at least 10 seconds. The subgingival plaque samples were transferred into flip-capped vials containing 2.0 mL of prereduced transport medium to be processed within 2 hours. The mesiobuccal and distobuccal sites of the maxillary right first premolar and first molar were sampled. For the headgear group, the first molar was a banded site, and the first premolar was a bonded site; the samples were separately analyzed. For the nonheadgear group, both teeth were bonded, and the samples were pooled.

To minimize the effect of tooth movement on the composition of the crevicular fluid, we did not perform any orthodontic activations during the last 2 months before sampling, and intermaxillary elastics were not worn. After removing all supragingival plaque as described, the

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Tb, Baseline; T–18, baseline for the headgear group; T0, baseline for the nonheadgear group; T52, end of the study after 52 weeks of treatment.
GCF was sampled. The absence of plaque is important because dental plaque has also been shown to have a marked effect on the recorded volume of crevicular fluid in the strip.26–28

Periopaper absorbent strips (593525, Ora Flow, Amityville, NY) were placed into the sulcus until light resistance was felt.27 After keeping the strip in place for 30 seconds, the absorbed volume was measured with the Periotron 6000 device (Ora Flow), which was calibrated before each measurement according to the standard curve obtained with bovine serum.29 Strips with blood contamination were discarded. The volume measurements were performed within 5 seconds after removal of the strip from the crevice to minimize evaporation.30 Per site, 3 strips were used. After measuring the collected volumes, the Periopaper strips were placed in coded sterile screw-capped vials and stored at −70°C.

The mesiobuccal and distobuccal sites of the maxillary right first premolar and first molar were sampled. In the headgear group, the first molar was a banded site, and the first premolar was a bonded site; the samples were analyzed separately. For the nonheadgear group, both teeth are bonded, and the strips were pooled.

After collecting all GCF samples, the fluid was extracted from the strips by adding 50 μL of phosphate-buffered saline solution containing 0.05% Tween 20 (Fluka, Buchs, Switzerland) and storing the vials at 4°C for 12 hours. After centrifuging the vials, the fluid was collected, and again 50 μL of phosphate-buffered saline solution containing 0.05% Tween 20 was added, and the whole procedure was repeated. The fluid extracted from the strips was pooled and stored at −70°C until analysis.

A Bio-Plex human cytokine assay for simultaneous quantification of interleukin-2 (IL-2), interleukin-4 (IL-4), interleukin-6 (IL-6), interleukin-8 (IL-8), interleukin-10 (IL-10), granulocyte monocyte colony stimulating factor (GM-CSF), interferon-γ (IFN-γ), tumor necrosis factor-α (TNF-α), monocyte chemotactic protein-1 (MCP-1), and interferon-inducible protein-10 (IP-10) was run according to the procedure recommended by the manufacturer (Bio-Rad Laboratories, Hercules, Calif). For each cytokine, a standard curve (1/4 dilutions) ranging from 0.5 pg per milliliter (IL-4), 2 pg per milliliter (IL-2, IL-6, IL-8/CXCL8, IL-10, GM-CSF, IFN-γ, MCP-1/CCL2, and IP-10/CXCL10) or 8 pg per milliliter (TNF-α), to 32,000 pg per milliliter was prepared. The filter plate was prewet with Bio-Plex assay buffer (Bio-Rad Laboratories), and 50 μL of beads coupled to specific monoclonal antibodies against the analyzed cytokines was added per well. Subsequently, the standards and samples (12.5 μL/sample) were prepared by using the Bio-Plex serum diluent kit (Bio-Rad Laboratories), and 50 μL per tube was transferred to the test plate, followed by incubation (30 minutes) at room temperature on a shaker (300 rpm). After 3 washes with Bio-Plex wash buffer (Bio-Rad Laboratories), detection antibody was added, and the plate was again incubated (30 minutes, room temperature, 300 rpm) to allow interaction with the beads. After removal of any excess detection antibody (3 washes with Bio-Plex wash buffer), a final incubation with streptavidin phycoerythrin was performed (10 minutes, room temperature, 300 rpm). The beads were washed 3 times and resuspended in 125 μL of Bio-Plex assay buffer before analysis in the Bio-Plex suspension array system. The data were analyzed by using the Bio-Plex Manager software (version 4.1) with 5-parameter logistic regression curves.

At all visits, digital color photographs were taken to ascertain the status praesens of the periodontium and the dental plaque accumulation.

The probing depths were measured at the proximal buccal sides of the teeth with a Merrit B probe.
Horse blood. The number of specimens forming units (CFU) were counted. From these data, the total numbers of anaerobic and aerobic colony-counting, the pure cultures were identified at all visits, and the examiner (J.v.G.) was blinded to the previous scores.

All samples were transferred to the laboratory and processed within 2 hours. Serial 10-fold dilutions were prepared in the prereduced transport medium. Dilutions of 10:2 to 10:4 were plated in duplicate with a spiral platter (Spiral Systems, Cincinnati, Ohio) onto nonselective blood agar plates (Blood Agar Base II, Oxoid, Basingstoke, United Kingdom), supplemented with hamine (5 μg/mL), menadione (1 μg/mL), and 5% sterile horse blood.

After 7 days of anaerobic incubation (80% N₂, 10% CO₂, and 10% H₂) in an anaerobic chamber and 3 days of aerobic incubation in an aerobic incubator at 37°C, the total numbers of anaerobic and aerobic colony-forming units (CFU) were counted. From these data, the CFU ratio (CFU aerobe:CFU anaerobe) was also calculated. The number of specific dark pigmented colonies (black pigmented bacteria) on a nonselective anaerobic plate, containing approximately 100 colonies, was counted. From the black-pigmented bacteria in the plaque samples, every third colony was subcultured on a blood agar plate. After 48 hours of anaerobic incubation, the pure cultures were identified by means of a series of biochemical tests (including N-acetyl-β-D-glucosaminidase, α-glucosidase, α-galactosidase, α-fucosidase, esculine, indole, and trypsin activity) to differentiate Porphyromonas gingivalis and P intermedia from other pigmented porphyromonas and prevotella species.

### Statistical analysis

To compare the groups, a linear mixed model was fit to the data, with time and treatment as the fixed factors. The data at Tb were compared with those at T52 (for the bonded sites, Tb was T=18; for the bonded sites, Tb was T0). The repeated measurement aspect of the data was modeled by taking the patient factor as the random variable. Residual analysis indicated that CFU counts, crevicular fluid, and cytokine concentrations needed a log transformation before analysis to achieve normal distribution. Multiple comparisons between treatments and time groups were corrected for simultaneous hypotheses according to the general linear hypothesis model, resulting in global confidence of 95% for all comparisons per variable. For variables with values below quantification limits, a 1000-fold Monte Carlo simulation was built up, replacing values below the quantification limit with a uniform, randomly distributed value between 0 and the quantification limit. Only P values for which 95% of the simulations were below 0.05 were considered significant.

To find relationships between the cytokine concentrations at baseline and the severity of the inflammatory reaction (i.e., worsening of the periodontal parameters) after 1 year of treatment, a stepwise regression was performed. The periodontal parameters (bleeding on probing, probing depth, GCF flow) after 1 year of orthodontic treatment were used as response and cytokine concentrations (IL-6, IL-8, IFN-γ, IP-10) at the start as explanatory variables. Values were entered in the model in the forward step and omitted from the model in the backward step by using a threshold P value of 0.2. The variable modeling of the difference in the models was by default used as the first variable in the model. Only variables having a P value below 0.05 were considered significant. A correlation coefficient would be difficult to interpret for the relationship between the cytokine concentrations at baseline and the severity of inflammatory reaction after 1 year of treatment because the data were not bivariately normally distributed and not independent. Instead, we used the P values of the regression to reflect the strength of the relationship.

### RESULTS

At Tb, there were no differences in microbial composition of the dental plaque between the bonded and the...
banded sites, and no differences in CFU ratios (aerobe:anaerobe) between the sexes at Tb were seen (Table II). The supragingival and subgingival CFU ratios (aerobe:anaerobe) decreased significantly over time for both the banded and the bonded sites (P < 0.05). The decrease in the ratio was not significantly different for the banded compared with the bonded sites. The presence of P gingivalis increased over time for both banded and bonded sites, but no intersite or intersex differences were seen.

No periodontal parameter differed significantly between the banded and bonded sites or between the sexes at Tb. GCF flow, probing pocket depth, and number of sites bleeding on probing increased significantly over time for both sites. Only for the bleeding-on-probing measurements, a significant difference (P < 0.05) in increase was seen between the banded and the bonded sites. The number of apical sites bleeding after probing was almost twice as high in the banded compared with the bonded sites. For the bleeding-on-probing increase over time, there was also a significant time-sex interaction (P < 0.05). The girls (n = 14) showed a significant difference (P < 0.05) in bleeding-on-probing increase between the banded and bonded sites over this 1-year period, but the boys did not (P = 0.32). The other parameters did not differ between the sexes.

Only for IL-6, IL-8, IFN-γ, IP-10, and MCP-1 did the Bio-Plex assay provide enough readings above the detection limit to apply statistical analyses. No significant alterations in the cytokine concentrations occurred over time for the banded or the bonded sites. Neither was there a significant difference in cytokine concentrations between the sites at Tb and T52. When the absolute cytokine values per strip (per 30 seconds) were statistically analyzed, a 2-fold increase for IL-8 was seen between Tb and T52 (P < 0.05). Significant differences between the materials were not seen. The girls showed a 10-fold higher concentration of IP-10 compared with the boys at baseline (P < 0.05).

The concentrations of some cytokines at Tb, however, seemed to be significant prognostic factors for the inflammation at T52 (Fig). More specifically, the concentration of IL-6 at Tb had a significant predictive value for the GCF flow at T52 (P < 0.05). The same relationship was found between the IL-8 concentration at Tb and the increase in probing depth at T52 (P < 0.05). The higher the cytokine concentrations at Tb, the higher the probability for increased measurements at T52 for GCF flow and probing depth.

DISCUSSION

Although the sample size was relatively small, the study provided meaningful statistical results with only 24 patients. Intrasubject rather than intersubject comparisons were performed, since more reliable results could be expected. This is especially the case because it is known that intrasubject differences in cytokine concentrations are rather high.12,33,34 The subjects were about the age of 14.5 years; at that age, differences between subjects, especially between boys and girls, could be expected. Girls are known to reach puberty earlier than boys, with possible hormonal differences as a result. It seems logical that these differences have an impact on the host response. Separate statistical analyses were performed for the sexes, but only a few differences were observed.

Before the bands and brackets were placed, the gingiva was healthy. This is important because several studies indicated increased plaque accumulation with gingival inflammation. At every visit, the standardized oral hygiene instructions were repeated, and oral hygiene was improved if necessary.

Although it was not possible to measure the periodontal parameters in a blinded manner, the researcher was blinded to the previous scores. All laboratory analyses were performed by using a coding system, which was only revealed after the completion of the study.

The increased probing depth recorded during this study was most likely caused by pseudopocket formation or by deeper penetration of the probe into the weakened connective tissues.35,36 Because these 2 processes could simultaneously contribute to the increased probing depth, a distinction cannot be made with the instruments used in this study. During this experiment, gingivitis was induced, but attachment loss probably did not occur.11,12

The microbiologic changes over time confirmed the results of a previous study of our research group.37 During the short-term follow-up (18 weeks after bracket bonding) of that study, significant elevations in microbial and clinical periodontal parameters were seen. Other authors showed comparable changes over time.38,39

Petti et al,40 however, did see a tendency toward the development of gingivitis, but significant changes did not occur. A microbial comparison between bands and brackets was by our knowledge not made by any other authors. Alexander41 found that the plaque index, the gingival index, and pocket depths at banded molars were significantly elevated compared with bonded molars. He assumed that these differences in clinical periodontal reactions were attributed to alterations in the composition of the bacterial biofilm without sampling it. In this study, we found no significant differences in microbiology between the bonded and banded sites. This is contrary to the general expectation of orthodontists, assuming that the microbial-periodontal impact of
the placement of a band is more harmful than that of a bracket.

GCF cytokines reflect the immune and inflammatory reactions from host-parasite interactions as well as from biomechanical stresses; it is not possible to make a distinction between these 2 processes. To minimize the effect of tooth movement, the wires were not activated until 2 months before the measurements were taken.

Fig. A, Relationship between the IL-6 concentration at Tb and the increase in GCF flow after 1 year of orthodontic treatment (T52-Tb). The predictive value is statistically significant ($P = 0.002$). B, Relationship between the IL-8 concentration at Tb and the increase in probing depth after 1 year of orthodontic treatment (T52-Tb). The predictive value is statistically significant ($P = 0.0001$).
The wire was not removed; thus, relapse will not occur. We wanted to measure the changes in GCF composition because of the increased plaque adhesion and the concomitantly provoked inflammatory reaction. To our knowledge, this has not been done so far. Before bracket placement, this was not a problem, because there was no tooth movement then. To minimize the effective tooth movement after 1 year of treatment, we did not do activations, and the patients were not allowed to use intermaxillary elastics during the 2 months before the measurements were made. Removing the archwire for a period was not considered because this would have certainly led to relapse and passive tooth movement. After 1 year of orthodontic treatment, the patients (none had extractions) were in their finishing stages, with only moderate activations. Başaran et al.42 followed the GCF concentrations of IL-2, 6, and 8 during orthodontic treatment that consisted of extraction of the maxillary right and left first premolars followed by fixed appliances.42 They concluded that, during leveling (low forces) and retraction (high forces) of the canines, IL-6 and IL-8 were not elevated at 21 days after activation. These data made us believe that, after the 2-month period of no activations, the contribution of tooth movement to the total amount of IL-6 and IL-8 could be ignored.

To detect differences in cytokine concentration over time and between the bonded and banded sites, these sites were followed for 1 year. Nevertheless, no significant differences over time or between the different materials were seen in this study when the cytokine concentrations were considered. This might be because increased inflammation would lead to increased GCF production and thereby dilute the cytokines. When the absolute IL values per strip (per 30 seconds) were statistically analyzed, a 2-fold increase for the chemotactic factor IL-8 was seen between T0 and T52 (P < 0.05). Significant differences between the materials were not seen.

In the periodontal tissues, the neutrophil follows the gradient of chemokines (such as IL-8) and crawls up the chemotactic gradient by using its receptors. Humoral factors, specific antibodies, and complements assist the neutrophils in their protective response, particularly in opsonization and phagocytosis of bacteria. It remains controversial whether an antibody is protective in periodontal disease, but an effective antibody response is a main mechanism by which the body generally responds to bacterial infection and invasion.43 On the other hand, in more chronic forms of inflammation, the persistence of excessive inflammatory mediators might lead to destruction of the tooth-supporting tissues and result in irreversible pathologic changes such as the clinical condition of periodontitis. Higher levels of this chemotactic factor IL-8 at baseline might reflect a more alert immune system, which according to our data results in increased probing depths, indicating more pronounced gingival inflammation after 1 year of treatment.

High levels of IL-6 in biologic fluids and blood have been found in infections and chronic inflammatory diseases.44 This cytokine is involved in the pathogenesis of several inflammatory diseases, therefore constituting a major mediator of the host response to injury and infection.44-46 Our results point out the significant value of IL-6 in the prediction of increased GCF flow after 1 year of treatment.

CONCLUSIONS

IL-6 and IL-8 concentrations before orthodontic treatment were shown to be significant predictive factors for some inflammatory parameters during treatment. Increased values of IL-6 at baseline were correlated with increased GCF flows after 1 year of orthodontic therapy (P < 0.05). High levels of IL-8 at baseline are predictive for higher probing depths at T52 (P < 0.05). To our knowledge, this information is new and could contribute to the decision of whether to start orthodontic treatment in susceptible patients. More research in this area is needed to confirm the predictive value of these biomarkers. They might allow orthodontists to screen patients before active treatment to determine whether it is indicated to start orthodontic treatment, especially in patients who want treatment for mild esthetic deviations. Until now, it has not been clear whether these worsened periodontal and microbial parameters will normalize after the orthodontic therapy. Only in a few retrospective studies was the periodontal situation of orthodontically treated patients and untreated controls compared.11 Prospective studies to elucidate this topic should be carried out. Especially when the changes are not completely reversible, screening for susceptibility with biomarkers IL-6 and IL-8 might be a helpful tool.

REFERENCES


