Teles RP, Bogren A, Patel M, Wennstrom JL, Socransky SS, Haffajee AD. A three-year prospective study of adult subjects with gingivitis II: microbiological parameters. J Clin Periodontol 2007; 34:7-17. (37 refs.)

**Purpose:** To investigate whether the clinical benefits attained through periodontal prevention programs based on meticulous supragingival plaque control would be accompanied by a shift to a more host-compatible subgingival microbiota, in subjects with periodontal health or minimal disease.

Materials and Methods: 160 subjects were recruited from two locations: Landskrona, Sweden (n=80) and Boston. To be included in the study, subjects had to be > 20 years of age, have at least 24 natural teeth, < 2 sites with PPD > 4mm and no proximal sites with clinical attachment loss. Subjects were also excluded if they had any systemic condition which would influence the course of periodontal disease or treatment, and medical conditions requiring prophylactic antibiotic coverage. Subjects were clinically and microbiologically monitored at baseline, 1, 2, and 3 years. Clinical measurements were taken at 6 sites per tooth. Subgingival plaque samples were taken from the mesiobuccal aspect of every tooth with sterile curettes and were analyzed for the levels of 40 bacterial species using checkerboard DNA-DNA hybridization. The mean counts of each of the 40 test species were calculated for each subject at each time point. Significance of differences over time was sought using the Friedman test and p values were adjusted for multiple comparisons. All subjects received a dental prophylaxis as well as oral hygiene instructions at the beginning of the study at every 6 months for 3 years. A selfperformed plaque removal program was introduced that included brushing 2x day using a manual or powered toothbrush and fluoride toothpaste. Interdental cleaning with dental floss and/or toothpicks was also performed for the duration of the study. New supplies were provided at each 6-month recall.

**Findings:** 124 subjects had microbiological data for all four visits, resulting in a total of 13,477 samples (27 samples/visit/subject). There was considerable variability in the total counts of bacteria among subjects at different time points. All clinical parameters, at the microbiologically sampled sites, improved over time. The clinical changes were accompanied by statistically significant decreases in the mean counts of 35/40 test species. Major reductions occurred by year 2 for members of the

genera *Actinomyces*, *Capnocytophaga*, *Fusobacterium*, and *Prevotella* species. At year 3, there was a modest re-growth of the majority of the species.

**Conclusion:** Well-performed patient-initiated prevention procedures can lead to both clinical and microbiological improvements for prolonged periods of time in periodontally healthy subjects and those with minimal disease.

Broggini N, Mcmanus LM et al. Peri-implant inflammation defined by the implant-abutment interface. J Dent Res 2006; 85(5):473-478.

**Purpose:** To determine whether increasing apical position of the implant-abutment interface leads to a proportionally greater magnitude of inflammatory cells and associated bone loss under simulated clinical conditions.

**Materials and Methods**: Following approval by the Institutional Animal Care and Use Committeen (UTHSCSA, San Antonio, TX, USA), two-piece, submerged implants varying in the apico-coronal location of the implant-abutment interface were placed either 1 mm coronal to, at, or 1 mm apical to the alveolar bone crest (supracrestal, crestal, or subcrestal, respectively). In brief, 30 implants fabricated of grade IV commercially pure titanium and with a screw-type sand-blasted, largegrit, acidetched (SLA) surface were placed in duplicate (left and right) in partially edentulous mandibles of 5 male foxhound dogs. Abutments were connected 3 months later. At 4, 8, and 10 wks following abutment connection, abutments were loosened and then immediately tightened, to imitate typical clinical procedures. Six months following initial implant placement, specimens were obtained and prepared for non-decalcified histology.

## Histomorphometric analysis:

Histomorphometric software (Image-Pro Plus®, Media Cybernetics, Silver Spring, MD, USA) was used for digital image capture, enumeration of individual inflammatory cells, and measurement of peri-implant tissue areas.

## Data Analysis:

Test fields were evaluated individually and collectively as three different zones: the entire apicocoronal distance, coronal to the original bone crest, and apical to the original bone crest. The rationale for this subdivision was the hypothesis that a subset of cell accumulation could be correlated with bone loss.

## Findings and Conclusion:

As the apical position of the implant-abutment interface was progressively increased, the total number of peri-implant inflammatory cells was increased in parallel, i.e., the deeper

the interface, the greater the magnitude of peri-implant inflammation. Further, regardless of the implant-abutment interface position relative to the original alveolar bone crest, the highest concentration of inflammatory cells was consistently at or immediately coronal to the interface, and progressively decreased thereafter toward bone

or gingival. For both crestal subcrestal implants, the neutrophil was the predominant peri-implant inflammatory cell. For supracrestal implants, cumulative periimplant mononuclear cells were somewhat greater than neutrophils. The implant-abutment interface position significantly influenced peri-implant inflammatory cell accumulation apical to the original bone crest. In contrast, interface position had no significant effect on the cumulative collection of neutrophils or mononuclear cells coronal to the original bone crest (although there was a trend toward increased numbers of neutrophils with increased depth of the interface).

The current study demonstrated that moving the interface supracrestally, effectively changing the location of the inflammatory stimulus, also reduces peri-implant bone loss. Thus, minimal inflammation (and bone change) occurred when the interface was above the original bone crest, whereas the greatest inflammation (and bone loss) occurred when the interface was below the alveolar crest.

Steinebrunner L, Wolfart W, Bobmann K et al. In Vitro evaluation of bacterial leakage along implant-abutment interface of different implant systems. Int J Oral Maxillofac Implants 2005;20:875-81.

Purpose: to investigate bacterial leakage at implant-abutment interface using an in vitro model.

**Materials and Methods:** 5 different implant systems with 8 standard implant-abutment combinations for single molar crowns were tested. The implants were mounted in the resin to mimic intraoral conditions. Base metal alloy crowns were luted to implant abutments with composite resin. The internal aspect of each implant was inoculated with E coli. Each abutment-crown combination was assembled according to manufacturers' protocols. The specimens were partially immersed in a soy broth media that came halfway up to the crown slightly above implant abutment interface. These implants then subjected to a dual axis chewing stimulator with a force of 120N for a 1.2 million cycles at 1 Hz. The number of completed chewing cycles was recorded when E coli colonization was detected. Data collected was analyzed statistically.

**Findings:** All specimens indicated bacterial leakage. Median chewing (loading) cycles until bacterial penetration were found- Branemark system with 172,800, Frialit/Hermetics system with 43,200, Replace-Select system with 64,800, Camlog system with 345,600, ScrewVent system with 24,300. Camlog system show bacterial leakage at significant higher chewing cycles than Frialit or Screw Vent system.

**Conclusions:** Bacterial leakage along the implant-abutment interface was found for all tested implant systems. The number of loading cycles until bacterial penetration occurred differed significantly between implant systems and their respective connection designs. This new in vitro model indicated bacterial penetration along implant abutment interface during dynamic loading.

Chin MY, Sandham A, de Vries J, et al. Biofilm formation on surface characterized micro-implants for skeletal anchorage in orthodontics. Biomaterials 2007;28 2032-2040.

**Purpose:** To quantitatively assess oral biofilm formation on commercially available micro-implant surfaces as well as the antimicrobial efficacy of chlorhexidine and fluoride containing mouthrinses on these biofilms.

Materials and Methods: Five commercially available, self-tapping orthodontic micro-implants were selected for the present study. All micro-implants were packaged individually by the manufacturers and required sterilization prior to surgical insertion. The elemental surface compositions of implants were characterized by X-ray photoelectron spectroscopy. To analyze the surface topography of as-received micro-implants, scanning electron microscopy (SEM) was performed. Overnight biofilms were grown on micro-implant surfaces by immersion in pooled human whole saliva. Student's t-test for independent samples was used to compare the amount of biofilm formed on the five types of micro-implant surfaces. The effects of chlorhexidine and fluoride mouthrinses on biofilm viability were analyzed by univariate two-way analysis of variance (ANOVA).

Findings and Conclusions: Generally, the as-received micro-implants demonstrated high amounts of C, indicative of high level of particulate contamination on the oxide surfaces. Traces of N, Ca, Fe, Cr, Cu, Pb, Zn, and Si were also detected. XPS analysis, after cleaning the micro-implants by sputtering with Ar<sup>+</sup> ions, of the oxide film showed that the oxygen content was marginally higher than that of the titanium for all Ti microimplants. Scanning electron microscopy revealed different surface topographies between manufacturers. In addition, many small protrusions were visible on a typical machined metal surface with very pronounced finishing grooves. Small protrusions and occasional pittings localized at the bottom of the thread were also noted. Biofilms prior to mouthrinse treatment on all micro-implant systems evaluated contained on average 57.0±4.5% viable organisms. In vitro exposure to the chlorhexidine and fluoride mouthrinses did not significantly reduce the amount of biofilm on the different types of micro-implant systems, but did decrease the viability of the biofilm organisms, with a mean of 11.7% and 10.5% viable organisms present post-treatment, respectively. This corresponds to a mean reduction of 80% in biofilm viability compared to the control. However there was no significant interaction between the effects of micro-implant type and antimicrobial treatment on the viability of biofilm organisms. Also there was no significant difference between the antimicrobial effects of chlorhexidine and fluoride mouthrinse on biofilm viability.

Aas JA, Barbuto SM, Alpagot T, et al. Subgingival plaque microbiota in HIV positive patients. J Clin Periodontol 2007;34:189-95.

**Purpose:** To describe and compare the predominant bacterial and fungal species associated with gingivitis, periodontitis, and linear gingival erythema (LGE), in HIV positive subjects with different immune status.

**Materials and Methods:** 14 HIV+ subjects participated in this study. Viral loads and CD4 levels determined HIV disease status. There were 5 gingivitis, 4 periodontitis subjects with low viral load and high CD4 level and 4 periodontitis subjects with high viral load. Subgingival plaque samples were collected using a Gracey curette. From pooled subgingival plaque, 16S and 18S rDNA were cloned and sequenced to determine species identity.

**Findings:** 109 bacterial species were identified and they fell into 7 bacterial phyla. These phyla consisted of Firmicutes, Actinobacteria, Cyanobacteria, Fusobacteria, Sygnergistes, Proteobacteria, and Bacteroides. Close to 50% of the species were not cultivable. Saccharomyces cerevisiae was the only fungal species detected in an LGE subject and in periodontitis subjects with high viral loads. In periodontitis patients with low viral loads, Candida albicans was predominant, but S. cerevisiae was only a minor component.

The classical putative periodontal pathogens, T. denticola, P. gingivalis and T. forsythia were below the limit of detection and were not detected. Species of Gemella, Dialister, Streptococcus and Veillonella were predominant. In one HIV positive subject with periodontitis and low viral load, Gemella morbillorum, a known opportunistic pathogen, constituted 84% of the clones.

**Conclusion:** The result from this study suggests that other bacterial species, rather than the classical periodontal pathogens, may be involved in periodontal diseases of HIV subjects. These bacterial profiles differed with HIV severity. These data are indicative of opportunistic infections in a highly susceptible immunocompromised host, and alternative therapies may be required.