Microbiologic Results After Non-Surgical Erbium-Doped:Yttrium, Aluminum, and Garnet Laser or Air-Abrasive Treatment of Peri-Implantitis: A Randomized Clinical Trial

G. Rutger Persson,*†‡§ Ann-Marie Roos-Jansåker,‖ Christel Lindahl,*‖ and Stefan Renvert*‖¶#

Background: The purpose of this study is to assess clinical and microbiologic effects of the non-surgical treatment of peri-implantitis lesions using either an erbium-doped:yttrium, aluminum, and garnet (Er:YAG) laser or an air-abrasive subgingival polishing method.

Methods: In a 6-month clinical trial, 42 patients with peri-implantitis were treated at one time with an Er:YAG laser or an air-abrasive device. Routine clinical methods were used to monitor clinical conditions. Baseline and 6-month intraoral radiographs were assessed with a software program. The checkerboard DNA–DNA hybridization method was used to assess 74 bacterial species from the site with the deepest probing depth (PD) at the implant. Non-parametric tests were applied to microbiology data.

Results: PD reductions (mean ± SD) were 0.9 ± 0.8 mm and 0.8 ± 0.5 mm in the laser and air-abrasive groups, respectively (not significant). No baseline differences in bacterial counts between groups were found. In the air-abrasive group, *Pseudomonas aeruginosa*, *Staphylococcus aureus*, and *Staphylococcus anaerobius* were found at lower counts at 1 month after therapy (P <0.001) and with lower counts in the laser group for *Fusobacterium nucleatum naviforme* (P= 0.002), and *Fusobacterium nucleatum nucleatum* (P = 0.002). Both treatments failed to reduce bacterial counts at 6 months. *Porphyromonas gingivalis* counts were higher in cases with progressive peri-implantitis (P<0.001).

Conclusions: At 1 month, *P. aeruginosa*, *S. aureus*, and *S. anaerobius* were reduced in the air-abrasive group, and *Fusobacterium* spp. were reduced in the laser group. Six-month data demonstrated that both methods failed to reduce bacterial counts. Clinical improvements were limited. *J Periodontol 2011;82:1267-1278.*

KEY WORDS

Clinical trial; infection control; laser; microbiology.

* Department of Oral Sciences, School of Health and Society, Kristianstad University, Kristianstad, Sweden.
† Department of Oral Medicine, University of Washington, Seattle, WA.
‡ Department of Periodontics, University of Washington.
§ Department of Periodontology, University of Bern, Bern, Switzerland.
‖ Department of Periodontology, Public Dental Health Service, Kristianstad, Sweden.
¶ Blekinge Institute of Technology, Karlskrona, Sweden.
# School of Dental Sciences, Trinity College, Dublin, Ireland.

The infectious etiology of peri-implantitis is well established.[1-6] Data suggest that the prevalence of peri-implantitis is in the range of 16% to 25%.[7-9] If not successfully treated, peri-implantitis may lead to a complete disintegration and implant loss.[10-12] The current principles for the treatment of peri-implantitis were primarily derived from principles established for the therapy of periodontitis.[13] However, recent studies[4,5,14-17] that evaluated non-surgical intervention using traditional methods of subgingival mechanical debridement did not demonstrate significant clinical improvements or significant microbiologic changes. Thus, other effective methods for the treatment of peri-implantitis by managing the infection must be established.

Data from an in vitro study[18] suggested that, at low-energy densities, the erbium-doped:yttrium, aluminum, and garnet (Er:YAG) laser had a high bactericidal potential on common implant surfaces without causing morphologic changes of the implant surface or inducing excessive heat. Favorable formation of new bone was observed from a histologic analysis[19] in animal experimental peri-implantitis studies demonstrating that a laser-treated implant surface

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showed a tendency to produce a greater bone-to-implant contact than after treatment with curets only. Other studies[20,21] showed that, after the use of an Er:YAG laser as an adjunct to mechanical debridement in patients with periodontitis, a decrease in gingival fluid levels of proinflammatory cytokine levels were documented but without concomitant clinical improvements or reductions in bacterial counts. There are several different laser systems that have been suggested for the treatment of peri-implantitis.

An in vitro study[22] also showed that Er:YAG laser intervention may have had bacterial-killing effects. A study[23] suggested that the bacteria in pockets around implants were influenced by bacteria from teeth in adjacent pockets. Data have suggested that the microbiota at inflamed and infected implants was similar to the subgingival microbiota (biofilm) in periodontal pockets.[24] A similar microbiota was also identified at implant sites in patients who were edentulous, suggesting that such bacteria can transfer from locations other than from periodontal pockets to the pockets around dental implants.[25]

Peri-implant bone loss was associated with several pathogens that, otherwise, were associated with periodontitis.[26] However, progressive peri-implantitis may also be associated with the total infectious burden rather than with the presence of individual bacterial species in pockets around failing titanium implants.[27] Reports[3,11,28] also suggested that bacteria not primarily associated with periodontitis, such as *Staphylococci*, *Enterics*, and *Candida* species can be identified at high prevalence rates at failing dental implants.

In a previous study,[16] we identified that the treatment of non-advanced peri-implantitis lesions using either designated titanium curets or an ultrasound device did not result in clinical improvements or microbiologic changes. A new air-abrasive device for the management of infection around teeth and implants has been introduced.[29] A small, thin, disposable plastic nozzle is placed subgingivally dispersing a glycine-based grain of aluminum oxide powder to remove the biofilm. Recent data have suggested that this treatment is safe and provides comparable results to those obtained by the subgingival debridement of teeth alone.[29] In vitro data have also shown that the use of an air-abrasive device on titanium surfaces may change the titanium-surface characteristics.[30] Thus, treatment of a titanium surface with an air-abrasive device may have an impact on the future biofilm development at implant surfaces.

The objectives of the present longitudinal, single-masked, randomized clinical trial over a study period of 6 months were to assess the clinical and microbiologic effects of non-surgical treatment of established peri-implantitis lesions using either an Er:YAG laser or a subgingival abrasive method. We hypothesized that there were no differences in the treatment efficacy in the ability to improve clinical conditions and reduce bacterial counts between the two therapeutic interventions tested.

MATERIALS AND METHODS

The Ethics Committee, Lund University, approved this study. All enrolled patients signed a written approved consent to participate in the study. Patients were enrolled if they presented with ≥1 dental implant with radiographic evidence of bone loss ≥2 mm at the implant and a probing depth (PD) ≥5 mm with bleeding on probing (BOP) and/or pus on probing as assessed by a 0.2-N probing force at the same sites from which microbiologic samples were taken. All implants within the patient were treated with the same method. The study was conducted from October 2007 to September 2009 at the Specialty Clinic for Periodontology, Region Skåne, Kristianstad, Sweden.

The following criteria were used to exclude patients from entering the study: 1) serum glycated hemoglobin >7.0; 2) a prescription of anti-inflammatory medications, or antibiotics within the preceding 3 months or during the study period; 3) the use of medications known to have an effect on gingival growth; and 4) patients who required prophylactic antibiotics. The outline of the study is described in a flowchart (Fig. 1; Consolidated Standards of Reporting Trials [CONSORT] statement flowchart). To be included in the study, implants must have presented with bone loss with the equivalence of ≥3 implant threads or ≥1.8 mm as defined from the immediate postplacement and radiographs from the study baseline. Changes in bone levels during the study were defined from averaged measurements of the mesially and distally assessed distances between the base of the bony defect and the abutment–implant level reference. Thus, the implant mean bone level–change value was used for additional analysis.

Before enrollment, periodontal lesions at remaining teeth were treated. Patients were randomly assigned to one of the two treatment regimens. The randomized allocation was performed using a computer software program.** An investigator (SR), who did not collect data or perform the procedures allocated the study patients to intervention group. When performing their study tasks, the study examiner (Margareta Nilsson, Public Dental Health Specialty Clinic for Periodontology, Region Skåne, Kristianstad, Sweden) and therapist (CL) were not jointly present with the study participants. Study participants were instructed not to discuss the therapy with the study

** PASW 18.0 for Macintosh, IBM, Chicago, IL.

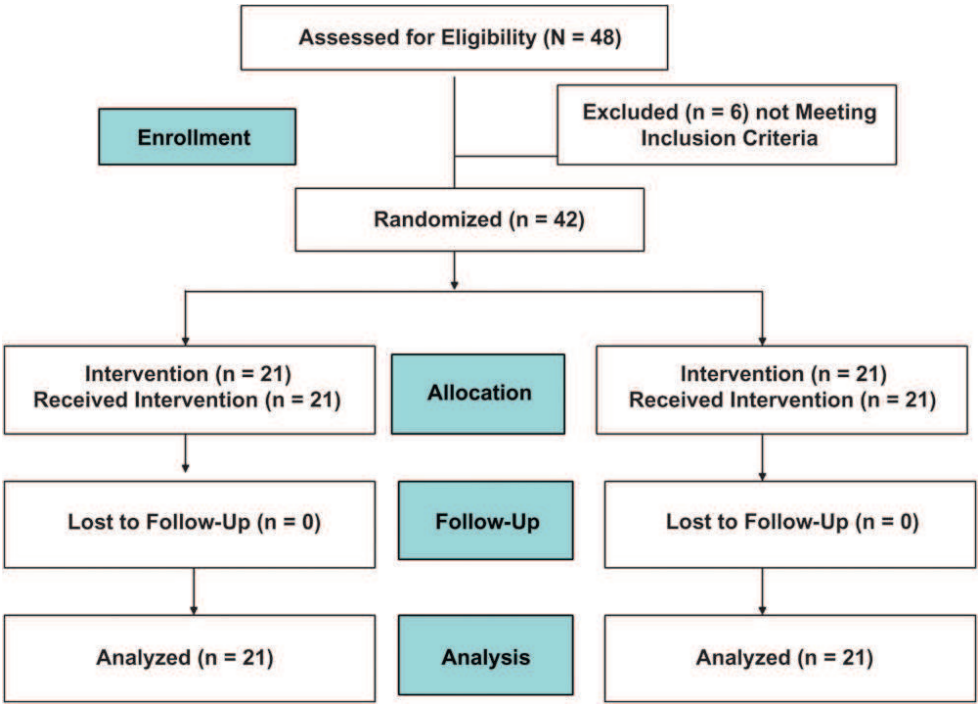

Figure 1.
CONSORT statement flowchart.

examiner. The study examiner was unaware of the study treatment allocations and performed all clinical measurements. The clinician performing treatments had >10 years clinical experience in the mechanical treatment of implants with a diagnosis of peri-implantitis.

Clinical Measurements and Procedures

Before clinical measurements and bacterial samples were taken, all superstructures were removed to enhance assessments of PD and BOP and to improve the ability to collect bacterial samples. In addition, the removal of superstructures allowed for the proper instrumentation of implants diagnosed with peri-implantitis. At 6 months, the same procedures were repeated before sampling and measurements were made.

PD and BOP measurements were performed using a standardized probing force of 0.2 N and with a plastic probe.†† To get proper access for clinical measurements and treatments, the superstructures were removed and repositioned after assessments and treatments. The following clinical assessments were performed at baseline and 6 months: 1) PD at four sites per implant, 2) bleeding scores, 3) suppuration, 4) intraoral radiographs, and 5) a composite outcome index was used to define treatment outcome. Implants demonstrating a PD reduction ≥0.5 mm and with no change or gain in bone levels were defined as responding to treatment. Implants with evidence of bone loss were defined as non-responders.

Intraoral radiographs of sites of interest were obtained at baseline and 6 months. Eggen holders were used for standardization purposes. Radiographs were analyzed to assess bone heights by one of the study investigators (GRP) who was masked to the study assignment. Radiographs were assessed from digital images using a software program.‡‡ A double analysis of bone-height assessments by the examiner from 30 randomly selected digitized images with a time interval of 1 day between assessments identified a Cronbach $\alpha = 0.96$ with an intraclass correlation of 0.96 (95% confidence interval [CI]: 0.91 to 0.98; $P < 0.001$). To control for potential differences in the alignment of radiographs at baseline and 6-month radiographs, distances among three implant threads of the implants in question from the two radiographs were measured and used to normalize the digital measurements at the two time points in millimeter values.

A composite-outcome index was used to identify the treatment outcome as a positive outcome with evidence of bone fill and combined with a decrease in PD ≥0.5 mm. In the event of no PD decrease or increase combined with no gain or increasing bone levels (loss), cases were identified as unsuccessfully treated.

Checkerboard DNA–DNA Hybridization, Microbiologic Sampling, and Analyses

Before any tissue manipulation, subgingival bacterial samples were taken immediately before intervention and at 1, 3, and 6 months after treatment. The sampled area was isolated with cotton rolls to prevent contamination with saliva. Samples were taken with two sterile endodontic paper points (size 55)§§ that were inserted at the same time at the site of the treated implant with the deepest PD. Endodontic paper points were kept in situ for 20 seconds. Efforts were made to

†† Click-Probe, KerrHawe, Bioggio, Switzerland.
‡‡ Image J software program 1:43r, National Institutes of Health, Bethesda MD.
§§ Absorbent Paper Points, DENTSPLY/Maillefer, Ballaigues, Switzerland.

insert the paper points to the bottom of the space between the implant and soft tissue lining. Collected paper points were placed in dry Eppendorf tubes (1.5-ml natural flat cap D Nas- and R Nas-free microcentrifuge tubes).‖‖ All samples were stored in batches at –20°C and forwarded on a monthly basis on dry ice by overnight carrier to the oral microbiology laboratory at the University of Bern. At the laboratory, 0.15 mL Tris EDTA buffer (10 mM Tris-HCL, 1.0 mM EDTA, pH 7.6), and 0.10 mL 0.5 M NaOH was added to each Eppendorf tube. The tubes were vortexed for 20 seconds, and paper points were removed. The remaining content was pipetted on to slots on each of two panels and processed as described for the checkerboard DNA–DNA hybridization method.[30-33] Species assessed by the checkerboard DNA–DNA hybridization method are presented in Table 1. Thus, in addition to species commonly studied in cases with periodontitis (i.e., *Porphyromonas gingivalis*, *Tannerella forsythia* [previously *T. forsythensis*], and *Treponema denticola*), we included a broad array of species that were comprised of other bacteria that were identified in cases with peri-implantitis.[3,5,11,14,17,28]

The digitized information was analyzed by a software program,¶¶ which allowed for the comparison of signals against standard lanes of known bacterial amounts (10^4 and 10^5 cells) in the appropriate checkerboard slot. Signals were converted to absolute counts by comparisons to these standards and studied as the proportion of sites defined as having $\geq 1.0 \times 10^5$ bacterial cells. Cross-reactivity was routinely tested in the microbiology laboratory among known pure bacterial reference bacterial strains purchased or obtained from other laboratories (Table 1). Our quality-control results were consistent with those reported elsewhere.[31] Thus, our analysis failed to identify measurable amounts of bacteria that could be explained as examples of cross-reactivity. The oral microbiology laboratory at the University of Bern is certified by the Swiss Health Authorities (Federal Agency of Health), Bern, Switzerland, to conduct clinical and laboratory microbiologic diagnostics.

Treatment Procedures

Implants in the air-abrasive group were treated with the air-abrasive device.## The nozzle was placed in the pocket mesially, lingually, distally, and buccally and used for ≈15 seconds in each position. Careful attempts were made to cover the full circumference of the implant. Implants in the laser group were treated using an Er:YAG laser*** at an energy level of 100 mJ/pulse and 10 Hz (12.7 J/cm^2) using a cone-shaped sapphire tip. This sapphire tip was provided by the manufacturer as the appropriate tip for the management of titanium-implant surfaces. The instrument tip was used in a parallel mode using a semicircular motion around the circumferential pocket area of the implant.

A topical local anesthesia was used as needed, whereas some patients (three of 21 patients in the laser group) received an infiltrative local anesthesia. Thus, the laser efficacy should not have been affected by the need for pain control. All study participants received comprehensive oral hygiene instructions. In addition, all patients received a sonic toothbrush††† and instructions on how to use it. The patients were supplied with new brush heads after 3 months.

Statistical Methods

If a PD difference in the change between methods of 1 mm was to be detected at $\alpha = 0.05$ and a power of $\beta = 0.20$, the appropriate number of participants per group was 20 patients. The PD change was defined as the primary outcome. The secondary outcome was a change in individual bacterial counts.

The Kolmogorov-Smirnov one-sample test identified that none of the baseline bacterial counts presented with a normal distribution pattern. Clinical data were analyzed using repeated univariate analysis of variance adjusting for participant factors and the number of implants treated in each patient. Data were analyzed by using Kruskal-Wallis analysis of variance to assess microbiologic changes with time within study groups and then by repeat Mann-Whitney *U* tests at different time points between study groups. The Wilcoxon test was used to assess within-site bacterial counts. Correlations between PDs and bacterial counts were assessed by Spearman rank correlation. The relationship between the clinical outcome and smoking status was assessed by χ^2 analysis. A statistical software package‡‡‡ was used for the analysis. To control for multiple observations, statistical differences were defined as $P < 0.001$.

RESULTS

All patients completed the study, and no implants were lost. No adverse events were diagnosed in either group, suggesting a low risk for air embolism by air-abrasive treatment or temperature development by the ER:YAG-laser treatment. PD reductions (mean ± SD) were 0.9 ± 0.8 mm and 0.8 ± 0.5 mm in the laser and air-abrasive groups, respectively (not significant). In the laser group, 21 patients had a total of 55 implants (41 machined surfaces and 14 medium-rough surfaces). Each patient in this group had an average ± SD of 2.6 ± 0.2 implants (range: one to eight implants). In the air-abrasive group, 21 patients had an average

‖‖ Starlab, Ahrensburg, Germany.
¶¶ ImageQuant, Amersham Pharmacia, Piscataway, NJ.
PERIO-FLOW, EMS – Electro Medical Systems, Nyon, Switzerland.
*** Key Laser 3 Perio KaVo, Biberach, Germany.
††† Philips Oral Healthcare, Snoqualmie, WA.
‡‡‡ PASW 18.0 for Macintosh, IBM.

Table 1.

Bacteria Included in the Checkerboard DNA–DNA Hybridization Assays

Bacteria	Collection	Bacteria	Collection
A. actinomycetemcomitans (a)	ATCC29523	*Alloscardovia omnicolens*	GUH071026
A. actinomycetemcomitans (b)	ATCC43718	*Actinomyces neuii*	GUH550898
A. israelii	ATCC1201	*Aerococcus christensenii*	GUH070938
A. naeslundii	ATCC121045	*Anaerococcus vaginalis*	GUH290486
A. odontolyticus	ATCC17929	*Atopobium parvulum*	GUH160323
C. gingivalis	ATCC33612	*Atopobium vaginae*	GUH010535
C. ochracea	ATCC33596	*Bacteroides ureolyticus*	GUH080189
Capnocytophaga sputigena	ATCC33612	*Bifidobacterium bifidum*	GUH070962
C. gracilis	ATCC33236	*Bifidobacterium breve*	GUH080484
C. rectus	ATCC33238	*B. longum*	GUH180689
C. showae	ATCC451146	*Corynebacterium nigricans*	GUH450453
E. corrodens	ATCC238345	*Corynebacterium aurimucosum*	GUH071035
Eubacterium saburreum	ASTCC33271	*Dialister* sp.	GUH071035
F. nucleatum naviforme	ASTCC49256	*Enterococcus faecalis*	GUH170812
F. nucleatum nucleatum	ATCC25586	*Enterococcus faecalis*	ATCC29212
F. nucleatum polymorphum	ATCC10953	*Escherichia coli*	GUH070903
Fusobacterium periodonticum	ATCC33993	*Gardnerella vaginalis*	GUH080585
L. acidophilus	ATCC11975	*Haemophilus influenzae*	ATCC49247
L. buccalis	ATCC14201	*Helicobacter pylori*	ATCC43504
N. mucosa	ATCC33270	*Lactobacillus crispatus*	GUH160342
P. micra	ATCC19696	*Lactobacillus gasseri*	GUH170856
Prevotella intermedia	ATCC25611	*Lactobacillus iners*	GUH160334
P. melaninogenica	ATCC25845	*Lactobacillus jensenii*	GUH160339
Prevotella nigrescens	ATCC33563	*Lactobacillus vaginalis*	GUH0780928
P. gingivalis	ATCC33277	*Mobiluncus curtisii*	GUH070927
Propionybacterium acnes	ATCC11827/28	*Mobiluncus mulieris*	GUH070926
Selenomonas noxia	ATCC43541	*Peptoniphilus* sp.	GUH550970
Streptococcus anginosus	ATCC33397	*Peptostreptococcus anaerobius*	GUH160362
S. constellatus	ATCC27823	*P. endodontalis*	ATCC35406
S. gordonii	ATCC10558	*P. bivia*	GUH450429
S. intermedius	ATCC27335	*Prevotella disiens*	GUH190184
S. mitis	ATCC49456	*Proteus mirabilis*	GUH070918

Table 1. (continued)

Bacteria Included in the Checkerboard DNA–DNA Hybridization Assays

Bacteria	Collection	Bacteria	Collection
S. mutans	ATCC25175	*Pseudomonas aeruginosa*	ATCC33467
Streptococcus oralis	ATCC35037	*S. aureus*	ATCC25923
Streptococcus sanguinis	ATCC10556	*S. aureus* yellow strain	GUH070921
T. forsythia	ATCC43037	*S. aureus* white strain	GUH070922
T. denticola	ATCC354405	*S. epidermis*	DSMZ20044
Treponema socranskii	D40DR2	*S. haemolyticus*	DSMZ20263
Veillonella parvula	ATCC10790	*Streptococcus agalactiae*	GUH230282
		Varibaculum cambriense	GUH070917

ATCC = American Type Culture Collection (Manassas, VA); GUH = Ghent University Hospital Collection (Ghent, Belgium); D = sample from Forsyth Institute (Boston, MA); DSMZ = The German Resource Center for Biologic Materials (Braunschweig, Germany).

of 45 implants (29 machined surfaces and 16 medium-rough surfaces). Each participant in this group had an average ± SD of 2.0 ± 0.2 implants (range: one to five implants). Patients in the laser group had significantly more implants (mean ± SE difference: 0.6 ± 0.2 implants; 95% CI: 0.1 to –0.2 implants; $P < 0.05$).

The average age and SD of the patients in the laser and air-abrasive groups were 68.5 ± 6.4 and 68.9 ± 12.5 years, respectively ($P = 0.91$). Twenty-nine of the patients were women. Statistical analyses failed to demonstrate study-group sex differences ($P = 0.34$), differences in the number of smoking years ($P = 0.58$), differences in medications used ($P = 0.17$), or differences in the number of implants in the maxilla ($P = 0.54$) or mandible ($P = 0.19$) between study groups.

Summary of Clinical Results at Sites From Which Microbiologic Samples Were Taken

At baseline, the mean ± SD PDs in the laser and air-abrasive groups were 5.9 ± 1.7 mm and 6.2 ± 1.9 mm, respectively ($P = 0.99$). BOP was identified at all implants. Using data from sites where the microbial samples had been taken, statistical analysis failed to demonstrate baseline differences in the extent of BOP ($P = 0.41$), presence of suppuration ($P = 0.97$), or PDs ($P = 0.25$) among implants assigned to the laser or air-abrasive treatment. The proportion of sites with bleeding and suppuration decreased similarly in both groups between baseline and 6 months ($P < 0.001$). Statistical analyses failed to demonstrate differences in PD reduction and bone-level changes between baseline and 6 months. At 6 months, 49% of sites from which bacterial samples were taken presented with a PD >5 mm. Statistical analyses also failed to demonstrate differences in the BOP at 6 weeks after treatment ($P = 0.52$), at 3 months after treatment ($P = 0.16$), and at the study endpoint by treatment modality ($P = 0.39$). At the study endpoint, 68.4% of implants presented with BOP at microbiologic test sites.

Microbiology

The pretreatment microbiologic screening for the presence of 74 bacterial species identified that many of these bacterial species were found at low proportions or below the threshold level of $\geq 1.0 \times 10^5$ bacterial cell defined for the checkerboard method. Statistical analyses failed to demonstrate baseline study-group differences in bacterial counts for all 74 species assayed. Statistical analyses also failed to demonstrate intervention-group differences in changes of bacterial levels between baseline and 6 months for all 74 bacterial species studied.

In the air-abrasive treatment group, significantly lower counts of *Pseudomonas aeruginosa*, *Staphylococcus aureus*, and *Staphylococcus anaerobius* were found at 1 month after therapy ($P < 0.001$) (Fig. 2). Mean values and SD for select bacteria in samples from the two study groups are presented in Table 2. At 1 month after treatment and in comparison to baseline levels, lower bacterial counts in the laser-treated group were found for *Fusobacterium nucleatum naviforme* ($P < 0.001$) and *Fusobacterium nucleatum nucleatum* ($P < 0.001$). At 3 months and at the final examination at 6 months, counts of bacteria increased. At the 6-month examinations, the total bacterial load, defined as the sum of bacterial counts of all species studied, was significantly higher than at baseline ($P < 0.001$).

Microbiologic Findings at 3 and 6 Months in Relation to BOP

At 3 months after therapy, neither treatment resulted in differences by bacterial counts for any of the 74

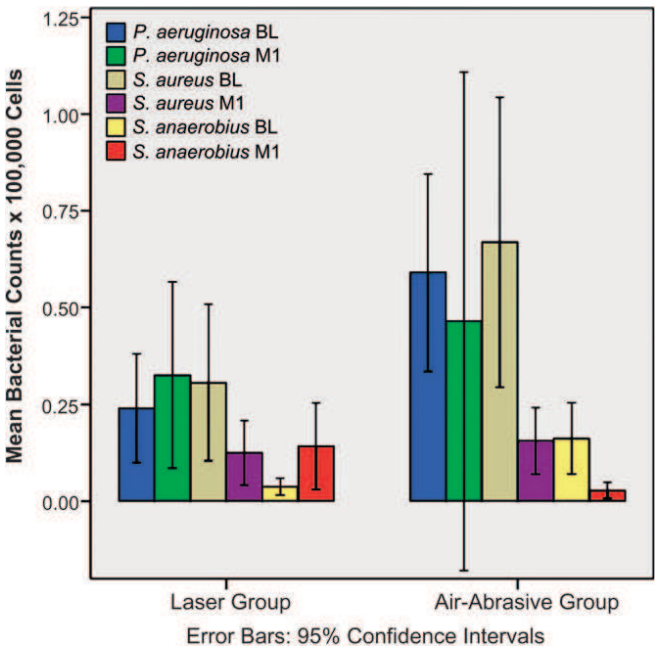

Figure 2.
Box-plot diagram presenting 25th percentiles (lower end of bar), medians, 75th percentiles (upper end of bar), and 95% confidence intervals (error bars) for counts of P. aeruginosa, S. aureus, *and* S. anaerobius *at baseline (BL) and at 1 month (M1) at implants in the laser-and air-abrasive-treated groups, respectively.*

studied species as an effect of BOP at the microbiologic test site. Statistical analyses demonstrated that, independent of the treatment modality, sites with BOP at 6 months presented with higher bacterial counts for the following species: *Aggregatibacter actinomycetemcomitans* (previously *Actinobacillus actinomycetemcomitans* (Y4), *Campylobacter gracilis*, *Campylobacter rectus*, *Eikenella corrodens*, *Leptothrichia buccalis*, *S. anaerobius*, *Staphylococcus haemolyticus*, *Streptococcus gordonii*, *Streptococcus mutans*, and *T. forsythia*.

Microbiologic Findings in Relation to PDs ≥6 or <6 mm at 6 Months

Independent of the treatment modality, the Spearman rank correlation analysis identified significant correlations between PDs and bacterial counts at 6 months for the following species ($P<0.001$): *Actinomyces israelii*, *Actinomyces naeslundii*, *Actinomyces odontolyticus*, *Bifidobacterium longum*, *Capnocytophaga gingivalis*, *F. nucleatum nucleatum*, *Fusobacterium nucleatum polymorphum*, *Lactobacillus acidophilus*, *Neisseria mucosa*, *P. gingivalis*, *Prevotella melaninogenica*, *Streptococcus intermedius*, *and Streptococcus mitis*.

After laser treatment at 6 months and at implant test sites with PDs ≥6 mm, the following bacterial species were found at higher counts than at sites with PDs <6 mm: *Campylobacter showae*, *Capnocytophaga ochracea*, *P. melaninogenica*, *S. anaerobius*, *S. haemolyticus*, S. *intermedius*, and *S. mutans*. At 6 months after the air-abrasive treatment, sites with PD ≥6 mm presented with a trend of higher counts of *S. aureus* ($P=0.006$) than at sites with PD <6 mm.

Statistical analyses demonstrated that, independent of the treatment modality, sites with PDs ≥6 mm at 6 months presented with higher bacterial counts than at sites with PDs <6 mm for the following species: *A. odontolyticus*, *A. actinomycetemcomitans* (serotype a), *C. gingivalis*, *C. ochracea*, *E. corrodens*, *F. nucleatum polymorphum*, *P. melaninogenica*, *Streptococcus constellatus*, *S. intermedius*, *S. mitis*, *L. buccalis*, *S. anaerobius*, *S. haemolyticus*, *S. gordonii*, and *S. mutans*.

Using the composite outcome index (PD and bone-level changes) to assess the effect of treatment, 44% of implants in the laser-treatment group improved, and 47% of the implants in the air-abrasive group improved. Patient-based clinical (PD and BOP) and microbiologic data for *P. gingivalis* and *S. aureus* at baseline and 6 months are presented in Table 3. The χ^2 analysis failed to demonstrate differences in treatment outcomes as previously defined ($P=0.45$). Because of the fact that no differences in bacterial counts were found at any observation time between the two study groups, microbiologic data of the two study groups were merged. Using the composite-outcome index (improvement = no change or gain of bone height and a PD decrease ≥0.5 mm) to define a progressive disease, the total bacterial count was higher at implants with progressive disease ($P=0.001$). At 6 months, counts of *P. gingivalis* were higher at implants with progressive peri-implantitis than at stable/improving implants ($P=0.003$).

The data analysis revealed that 13 of 42 patients who were not successfully treated were current smokers. The statistical analysis using the χ^2 test failed to identify a difference in the treatment outcome by smoking status ($P=0.57$).

DISCUSSION

Consistent with other studies, the present study fails to demonstrate unequivocal improved clinical benefits as a result of clinical intervention using an Er:YAG laser or air-abrasive treatment of peri-implantitis.[20,34,35] Nevertheless, ≈50% of treated implants demonstrated some clinical and microbiologic improvements during the study period. A repeated treatment at month 3, for example, might have improved the results. The present study also failed to confirm results from an in vitro study[22] that demonstrated the efficacy in killing pathogens associated with periodontitis (i.e., *T. forsythia* and *P. gingivalis*) by lasers.

Table 2.

Bacterial Counts at Baseline and 1, 3, and 6 Months (study endpoint) After Treatment at Implants Treated in the Laser and Air-Abrasive Treated Groups (bacterial counts $\times 10^5$ cells)

	Baseline			1 Month			3 Months			6 Months		
Bacterial Species	Mean	SD	%	Mean	SD	%	Mean	SD	%	Mean	SD	%
Laser treatment												
F. nucleatum nucleatum	0.4	0.7	11.3	1.3	2.0	**28.8**	1.3	2.0	**30.8**	1.0	1.9	26.7
P. gingivalis	1.0	4.5	5.9	1.2	2.7	17.0	0.9	2.3	**17.0**	1.1	3.5	13.5
P. aeruginosa	0.2	0.5	13.2	0.2	0.4	5.8	0.5	1.5	19.2	0.8	2.1	19.2
S. anaerobius	0.0	0.1	0.0	0.2	0.9	**1.9**	0.6	2.1	7.7	0.3	0.8	8.9
S. aureus	0.3	0.7	11.3	0.2	0.6	3.8	0.5	2.2	7.3	0.3	0.8	11.1
Air-abrasive treatment												
F. nucleatum nucleatum	0.5	1.2	13.3	1.1	1.6	31.8	1.7	2.1	**44.5**	1.0	1.6	25.0
P. gingivalis	0.3	0.8	3.2	0.5	1.5	11.9	2.0	5.5	9.1	1.1	2.4	18.2
P. aeruginosa	0.6	0.8	28.9	0.2	0.6	**4.5**	1.2	3.7	18.2	0.5	0.9	18.2
S. anaerobius	0.2	0.3	2.2	0.0	0.0	**0.0**	0.2	0.4	2.3	0.4	1.7	9.1
S. aureus	0.7	1.2	17.8	0.1	0.5	**2.3**	0.3	0.6	2.3	0.4	1.0	4.5

Proportion (%) of bacteria are expressed as bacteria present at counts $\geq 1.0 \times 10^5$ bacterial cells. Bacterial values identified in bold type denote significant differences compared to baseline values.

Table 3.

Baseline and 6-Month Clinical (PD and BOP) and Microbiologic (*P. gingivalis* and *S. aureus)* Statistics at Successfully and Non-Successfully Treated Implants in the Two Study Groups (patient-based data)

Study Groups and Outcomes (n patients in each subgroup)	PD at BL	PD at 6 Month	BOP at BL	BOP at 6 Months	*P. gingivalis* at BL ($\geq 10^4$ cells)	*P. gingivalis* at Month 6 ($\geq 10^5$ cells)	*S. aureus* at BL ($\geq 10^4$ cells)	*S. aureus* at 6 Months ($\geq 10^4$ cells)
	Mean ± SD	Mean ± SD	%	%	%	%	%	%
Laser group; positive outcome (8)	6.9 ± 1.4	5.8 ± 1.5	100	37.5	37.5	14.3	37.5	14.3
Laser group; no or negative outcome (13)	6.7 ± 2.1	5.9 ± 1.8	100	77.8	44.4	55.6	32.3	44.4
Air-abrasive group; positive outcome (9)	6.5 ± 2.1	5.2 ± 1.5	100	66.7	16.7	40.0	33.3	0.0
Air-abrasive group; no or negative outcome (12)	6.1 ± 1.9	5.3 ± 2.1	100	75.0	49.7	41.7	50.0	50.0

BL = baseline.

The cases selected in the present study represented established lesions at titanium implants as identified from intraoral radiographs. At baseline, a majority of the implants presented with deep PDs. Although the suprastructures were removed before the intervention, this effort may not have been enough to get clinical access to the bottom of the lesions to remove the bacterial biofilm with the laser or the air-abrasive devices.

The study baseline microbiologic data identified several species that were recognized as pathogens in studies of periodontitis, peri-implantitis, and other diseases[1,3,4,6,10,11,17,20,24-28,32] and consistent with findings in periodontitis, the present study of

peri-implantitis demonstrates that *T. forsythia* was the most prevalent microorganism before and after therapy. Other bacteria of concern, which were found at high prevalence rates, included *P. aeruginosa*, *A. actinomycetemcomitans*, *F. nucleatum*, and *S. aureus*.

The development of a bacterial biofilm on teeth, implants, and oral soft tissues is a major factor in the development of oral infections.[36] A biofilm is a microbial-derived sessile community characterized by cells that are irreversibly attached to a substratum or interface to each other and embedded in a matrix of extracellular polymeric substances that they have produced. *S. aureus* may play an important role in the development of a biofilm.[37] In addition, in vitro data have shown that *S. aureus* has a high affinity to titanium surfaces.[38] Another clinical study[39] showed that if *S. aureus* was identified at dental implants shortly after insertion, *S. aureus* was also identified with a high level of predictability 1 year later. A study[40] showed that when *S. aureus* was positively identified at titanium dental implants, *S. aureus* was also found in periodontal pockets of teeth. Serologic data suggested that humoral immunity and functional antibodies against *S. aureus* and *T. forsythia* may explain successful outcomes in dental implant therapy.[41]

The composite clinical outcome index based on PD and bone-level changes should be tested in a larger study population. This outcome index was developed to search for microbiologic factors that might have explained the clinical outcome results. Using a 0.5-mm PD can be considered a questionable marker given the difficulties in assessing the PD at implants. However, in the present study, the superstructures were removed at baseline and 6 months, which should have facilitated assessments. The outcome index was based on a relation between the bone gain, loss, or no change and the PD change. The levels of *P. gingivalis* and *S. aureus* appeared to be explanatory to the clinical outcomes. High levels of BOP in non-successfully treated cases suggested the failure of the plaque control. Obviously, this had an impact on the subgingival development of a pathogenic microbiota/biofilm. We also identified that current smoking status did not have an impact on the treatment outcome. The *P* value obtained was such that it would most likely require a very large study population to assess the impact of smoking on the treatment outcome using any of the treatment modalities studied.

Data suggested that the most common bacteria in superficial and deep cultures from transcutaneous osseointegrated titanium implants included *S. aureus* and coagulase-negative staphylococci.[42] In addition, acute infections by *Enterobacteriaceae* and *Pseudomonas* spp. are common in knee and hip infected implants and may be successfully treated in 75% of cases by open debridement and antibiotics.[43]

P. aeruginosa, *Porphyromonas endodontalis*, *Prevotella bivia*, and *S. aureus* were also found in subgingival plaque samples from subjects with periodontitis.[44] Another study[45] showed that the presence of *Parvimonas micra* in periapical lesions was often associated with positive detection of *P. aeruginosa* and *S. aureus*. This finding was consistent with the findings in the present study. Although interventions resulted in a shift of these species, *P. micra, P. aeruginosa*, *S. aureus*, and *T. forsythia* were commonly found 6 months after study interventions. The early colonization of *P. aeruginosa* may be most important in the further establishment of a pathogenic biofilm in peri-implantitis. Data suggested that *P. aeruginosa* easily established early biofilm structures on titanium surfaces.[46]

Consistent with the current perception that the oral cavity comprises a large diversity of bacteria (>700 different species), and the implant surface has different characteristics than a tooth surface, we decided to study a broader panel of species than currently assessed in most other studies.[47] Our data demonstrate that several well-known pathogens that have not been routinely studied in dental studies can be found at implants with a diagnosis of peri-implantitis and that some of those species appeared to be affected differently by the treatment modalities assessed. The present study also suggests that pathogens other than those commonly studied in cases with periodontitis are found in cases with peri-implantitis, and such bacteria can also be affected through mechanical treatment.

An in vitro study[48] suggested that a *P. aeruginosa* biofilm can be disrupted by a special miniature Q-switched neodymium-doped:yttrium, aluminum, and garnet laser. Such a device was not used in the present study. The wavelength used by different Er:YAG lasers may explain differences in the efficacy of eradicating bacteria from titanium surfaces.[49] It is also possible that the intervention with the laser at one time point was not sufficient to eliminate key pathogens from the infected implant surfaces. Furthermore, the penetration of the laser energy may not have been optimal to kill bacteria in large biofilm structures. Thus, repeated treatments may be required.

An in vitro study[50] suggested that an air-abrasive device could remove bacteria from rough titanium implant surfaces. The particle type used in the air-abrasive treatment of contaminated implant surfaces may explain the ability to remove pathogens from infected dental implants.[30] The air-abrasive method used in the present study demonstrated some advantages over the Er:YAG-laser method used in reducing

the counts of *P. aeruginosa*, *S. aureus*, and *S. anaerobius* at 1 month after the intervention. A clinical advantage with the air-abrasive method is that it may be used without removing the suprastructure because the plastic nozzle is flexible.

In vitro data have suggested that, in contrast to a continuous-wave diode and CO_2-laser irradiation, a pulsed Er:YAG-laser irradiation can cause alterations if the power setting for the ER:YAG laser exceeds 300 mJ/10 Hz on the sand-blasted acid-etched surface and 500 mJ/10 Hz on the polished surface.[51] In the present study, the power setting for the ER:YAG laser was set at an energy level of 100 mJ/pulse and 10Hz (12.7 J/cm^2), which thus was at a much lower range than what might have induced implant-surface alterations. However, it is possible that our power settings might have been too conservative to effectively eradicate the pathogens. We used the power setting that was suggested by other authors.[35] Although the impact of the microbiota in peri-implantitis[52] was not studied, in a review of the current literature on CO_2 lasers, the authors[53] concluded that a CO_2 laser in combination with augmentative techniques may effectively decontaminate implants with peri-implantitis. An in vitro study[54] showed that a 308-nm excimer laser light on peri-implantitis-associated bacteria specifically had an impact on Gram-negative anaerobes. Thus, lasers other than the Er:YAG laser may be effective in the treatment of peri-implantitis.[54] To our knowledge, there are no clinical trials that have assessed the efficacy of such laser treatments.

Consistent with another study,[29] the present study fails to identify cases with air emphysema after treatment with an air-abrasive device. The question raised by other authors[55] regarding whether the powder or the fluid rinse of the pocket has an impact on the microbiota is another concern to be addressed.

CONCLUSIONS

The air-abrasive method appeared to have some advantages at 1 month after therapy because the counts of pathogens (i.e., *P. aeruginosa*, *S. aureus*, and *S. anaerobius*) associated with severe infections were significantly reduced by this method. The presence of *P. gingivalis* may explain progressive peri-implantitis. The air-abrasive or Er:YAG laser method in the treatment of severe peri-implantitis did not result in adverse clinical events.

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REFERENCES

1. Rutar A, Lang NP, Buser D, Bürgin W, Mombelli A. Retrospective assessment of clinical and microbiological factors affecting periimplant tissue conditions. *Clin Oral Implants Res* 2001;12:189-195.
2. Lindhe J, Meyle J; Group D of European Workshop on Periodontology. Peri-implant Diseases: Consensus Report of the Sixth European Workshop on Periodontology. *J Clin Periodontol* 2008;35(Suppl. 8):282-285.
3. Renvert S, Roos-Jansåker AM, Lindahl C, Renvert H, Rutger Persson G. Infection at titanium implants with or without a clinical diagnosis of inflammation. *Clin Oral Implants Res* 2007;18:509-516.
4. Renvert S, Lessem J, Dahlén G, Renvert H, Lindahl C. Mechanical and repeated antimicrobial therapy using a local drug delivery system in the treatment of peri-implantitis: A randomized clinical trial. *J Periodontol* 2008;79:836-844.
5. Heitz-Mayfield LJ, Lang NP. Comparative biology of chronic and aggressive periodontitis vs. peri-implantitis. *Periodontol 2000* 2010;53:167-181.
6. Aoki M, Takanashi K, Matsukubo T, Yajima Y, Okuda K, Sato T, Ishihara K. Transmission of periodontopathic bacteria from natural teeth to implants. [published online ahead of print December 11, 2009]. *Clin Implant Dent Relat Res*. doi: 10.1111/j.1708-8208.2009.00260.x
7. Fransson C, Lekholm U, Jemt T, Berglundh T. Prevalence of subjects with progressive bone loss at implants. *Clin Oral Implants Res* 2005;16:440-446.
8. Roos-Jansåker AM, Lindahl C, Renvert H, Renvert S. Nine- to fourteen-year follow-up of implant treatment. Part I: Implant loss and associations to various factors. *J Clin Periodontol* 2006;33:283-289.
9. Koldsland OC, Scheie AA, Aass AM. Prevalence of peri-implantitis related to severity of the disease with different degrees of bone loss. *J Periodontol* 2010;81:231-238.
10. Quirynen M, De Soete M, van Steenberghe D. Infectious risks for oral implants: A review of the literature. *Clin Oral Implants Res* 2002;13:1-19.
11. Leonhardt A, Dahlén G, Renvert S. Five-year clinical, microbiological, and radiological outcome following treatment of peri-implantitis in man. *J Periodontol* 2003;74:1415-1422.
12. Esposito M, Grusovin MG, Coulthard P, Worthington HV. The efficacy of interventions to treat peri-implantitis: A Cochrane systematic review of randomised controlled clinical trials. *Eur J Oral Implantology* 2008;1:111-125.
13. Lang NP, Wilson TG, Corbet EF. Biological complications with dental implants: Their prevention, diagnosis

and treatment. *Clin Oral Implants Res* 2000;11(Suppl. 1):146-155.
14. Salvi GE, Persson GR, Heitz-Mayfield LJ, Frei M, Lang NP. Adjunctive local antibiotic therapy in the treatment of peri-implantitis II: Clinical and radiographic outcomes. *Clin Oral Implants Res* 2007;18:281-285.
15. Renvert S, Roos-Jansåker AM, Claffey N. Non-surgical treatment of peri-implant mucositis and peri-implantitis: A literature review. *J Clin Periodontol* 2008;35(Suppl. 8): 305-315.
16. Renvert S, Samuelsson E, Lindahl C, Persson GR. Mechanical non-surgical treatment of peri-implantitis: A double-blind randomized longitudinal clinical study. I: Clinical results. *J Clin Periodontol* 2009;36: 604-609.
17. Persson GR, Samuelsson E, Lindahl C, Renvert S. Mechanical non-surgical treatment of peri-implantitis: A single-blinded randomized longitudinal clinical study. II. Microbiological results. *J Clin Periodontol* 2010;37: 563-573.
18. Kreisler M, Kohnen W, Marinello C, et al. Bactericidal effect of the Er:YAG laser on dental implant surfaces: An in vitro study. *J Periodontol* 2002;73:1292-1298.
19. Takasaki AA, Aoki A, Mizutani K, Kikuchi S, Oda S, Ishikawa I. Er:YAG laser therapy for peri-implant infection: A histological study. *Lasers Med Sci* 2007; 22:143-157.
20. Gómez C, Domínguez A, García-Kass AI, García-Nuñez JA. Adjunctive Nd:YAG laser application in chronic periodontitis: Clinical, immunological, and microbiological aspects. *Lasers Med Sci* 2011;26:453-463.
21. Domínguez A, Gómez C, García-Kass AI, García-Nuñez JA. IL-1beta, TNF-alpha, total antioxidative status and microbiological findings in chronic periodontitis treated with fluorescence-controlled Er:YAG laser radiation. *Lasers Surg Med* 2010;42:24-31.
22. Kranendonk A, van der Reijden W, van Winkelhoff A, van der Weijden G. The bactericidal effect of a Genius Nd:YAG laser. *Int J Dent Hyg* 2010;8:63-67.
23. Sumida S, Ishihara K, Kishi M, Okuda K. Transmission of periodontal disease-associated bacteria from teeth to osseointegrated implant regions. *Int J Oral Maxillofac Implants* 2002;17:696-702.
24. Shibli JA, Melo L, Ferrari DS, Figueiredo LC, Faveri M, Feres M. Composition of supra- and subgingival biofilm of subjects with healthy and diseased implants. *Clin Oral Implants Res* 2008;19:975-982.
25. Emrani J, Chee W, Slots J. Bacterial colonization of oral implants from nondental sources. *Clin Implant Dent Relat Res* 2009;11:106-112.
26. Tabanella G, Nowzari H, Slots J. Clinical and microbiological determinants of ailing dental implants. *Clin Implant Dent Relat Res* 2009;11:24-36.
27. Persson GR, Salvi GE, Heitz-Mayfield LJ, Lang NP. Antimicrobial therapy using a local drug delivery system (Arestin) in the treatment of peri-implantitis. I: Microbiological outcomes. *Clin Oral Implants Res* 2006;17:386-393.
28. Leonhardt A, Renvert S, Dahlén G. Microbial findings at failing implants. *Clin Oral Implants Res* 1999;10: 339-345.
29. Moëne R, Décaillet F, Andersen E, Mombelli A. Subgingival plaque removal using a new air-polishing device. *J Periodontol* 2010;81:79-88.
30. Schwarz F, Ferrari D, Popovski K, Hartig B, Becker J. Influence of different air-abrasive powders on cell viability at biologically contaminated titanium dental implants surfaces. *J Biomed Mater Res B Appl Biomater* 2009;88:83-91.
31. Socransky SS, Smith C, Martin L, Paster BJ, Dewhirst FE, Levin AE. "Checkerboard" DNA-DNA hybridization. *Biotechniques* 1994;17:788-792.
32. Socransky SS, Haffajee AD, Smith C, et al. Use of checkerboard DNA-DNA hybridization to study complex microbial ecosystems. *Oral Microbiol Immunol* 2004;19:352-362.
33. Persson R, Hitti J, Verhelst R, et al. The vaginal microflora in relation to gingivitis. *BMC Infect Dis* 2009;9:6.
34. Schwarz F, Bieling K, Bonsmann M, Latz T, Becker J. Nonsurgical treatment of moderate and advanced periimplantitis lesions: A controlled clinical study. *Clin Oral Investig* 2006;10:279-288.
35. Schwarz F, Bieling K, Nuesry E, Sculean A, Becker J. Clinical and histological healing pattern of peri-implantitis lesions following non-surgical treatment with an Er:YAG laser. *Lasers Surg Med* 2006;38:663-671.
36. Busscher HJ, Rinastiti M, Siswomihardjo W, van der Mei HC. Biofilm formation on dental restorative and implant materials. *J Dent Res* 2010;89:657-665.
37. Costerton JW, Montanaro L, Arciola CR. Biofilm in implant infections: Its production and regulation. *Int J Artif Organs* 2005;28:1062-1068.
38. Harris LG, Meredith DO, Eschbach L, Richards RG. *Staphylococcus aureus* adhesion to standard microrough and electropolished implant materials. *J Mater Sci Mater Med* 2007;18:1151-1156.
39. Salvi GE, Fürst MM, Lang NP, Persson GR. One-year bacterial colonization patterns of *Staphylococcus aureus* and other bacteria at implants and adjacent teeth. *Clin Oral Implants Res* 2008;19:242-248.
40. Renvert S, Lindahl C, Renvert H, Persson GR. Clinical and microbiological analysis of subjects treated with Brånemark or AstraTech implants: A 7-year follow-up study. *Clin Oral Implants Res* 2008;19:342-347.
41. Kronström M, Svenson B, Hellman M, Persson GR. Early implant failures in patients treated with Brånemark System titanium dental implants: A retrospective study. *Int J Oral Maxillofac Implants* 2001;16:201-207.
42. Tillander J, Hagberg K, Hagberg L, Brånemark R. Osseointegrated titanium implants for limb prostheses attachments: Infectious complications. *Clin Orthop Relat Res* 2010;468:2781-2788.
43. Martínez-Pastor JC, Muñoz-Mahamud E, Vilchez F, et al. Outcome of acute prosthetic joint infections due to gram-negative bacilli treated with open debridement and retention of the prosthesis. *Antimicrob Agents Chemother* 2009;53:4772-4777.
44. Persson GR, Hitti J, Paul K, et al. *Tannerella forsythia* and *Pseudomonas aeruginosa* in subgingival bacterial samples from parous women. *J Periodontol* 2008;79: 508-516.
45. Geibel MA, Schu B, Callaway AS, Gleissner C, Willershausen B. Polymerase chain reaction-based simultaneous detection of selected bacterial species associated with closed periapical lesions. *Eur J Med Res* 2005;10:333-338.
46. Upritchard HG, Yang J, Bremer PJ, Lamont IL, McQuillan AJ. Adsorption to metal oxides of the *Pseudomonas aeruginosa* siderophore pyoverdine and implications for bacterial biofilm formation on metals. *Langmuir* 2007;23:7189-7195.

47. Paster BJ, Olsen I, Aas JA, Dewhirst FE. The breadth of bacterial diversity in the human periodontal pocket and other oral sites. *Periodontol 2000* 2006;42:80-87.
48. Krespi YP, Stoodley P, Hall-Stoodley L. Laser disruption of biofilm. *Laryngoscope* 2008;118:1168-1173.
49. Gonçalves F, Zanetti AL, Zanetti RV, et al. Effectiveness of 980-mm diode and 1064-nm extra-long-pulse neodymium-doped yttrium aluminum garnet lasers in implant disinfection. *Photomed Laser Surg* 2010;28: 273-280.
50. Duarte PM, Reis AF, de Freitas PM, Ota-Tsuzuki C. Bacterial adhesion on smooth and rough titanium surfaces after treatment with different instruments. *J Periodontol* 2009;80:1824-1832.
51. Stubinger S, Etter C, Miskiewicz M, et al. Surface alterations of polished and sandblasted and acid-etched titanium implants after Er:YAG, carbon dioxide, and diode laser irradiation. *Int J Oral Maxillofac Implants* 2010;25:104-111.
52. Schwarz F, Sculean A, Rothamel D, Schwenzer K, Georg T, Becker J. Clinical evaluation of an Er:YAG laser for nonsurgical treatment of peri-implantitis: A pilot study. *Clin Oral Implants Res* 2005;16:44-52.
53. Romanos GE, Nentwig GH. Regenerative therapy of deep peri-implant infrabony defects after CO_2 laser implant surface decontamination. *Int J Periodontics Restorative Dent* 2008;28:245-255.
54. Deppe H, Horch HH, Schrödl V, Haczek C, Miethke T. Effect of 308-nm excimer laser light on peri-implantitis-associated bacteria: An in vitro investigation. *Lasers Med Sci* 2007;22:223-227.
55. Petersilka G. Re: "Subgingival plaque removal using a new air-polishing device." Moëne R, Décaillet F, Andersen E, Mombelli A. *J Periodontol* 2010;81:962-963; comment: 79-88.

Correspondence: Dr. Stefan Renvert, Department of Oral Sciences, School of Health and Society, Kristianstad University, SE-29135 Kristianstad, Sweden. Fax: 46-44-20-40-18; e-mail: stefan.renvert@hkr.se.

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