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Er:YAG laser in the treatment of periodontal sites with recurring chronic inflammation: a 12-month randomized, controlled clinical trial

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Abstract

Aim: The objective of this randomized, controlled clinical trial was to compare the clinical and microbiological effects of pocket debridement using erbium-doped: yttrium, aluminium and garnet (Er:YAG) laser with conventional debridement in maintenance patients.

Material & Methods: Fifteen patients, all smokers, having at least four teeth with residual probing depth (PD) ≥ 5 mm were recruited. Two pockets in two jaw quadrants were randomly assigned to subgingival debridement using an Er:YAG laser (test) or ultrasonic scaler/curette (control) at 3-month intervals. Relative attachment level (RAL), PD, bleeding on probing and dental plaque were recorded at baseline and at 6 and 12 months. Microbiological subgingival samples were taken at the same time points and analysed using a checkerboard DNA-DNA hybridization technique.

Results: A significant decrease in PD took place in both treatments from baseline to 12 months ($p < 0.01$). In the control, the mean initial PD decreased from 5.4 to 4.0 mm at 12 months. For the test, a similar decrease occurred. No significant between-treatment differences were shown at any time point. The mean RAL showed no overall significant inter- or intra-treatment differences ($p > 0.05$). No significant between-treatment differences were observed in subgingival microbiological composition or total pathogens.

Conclusion: The results failed to support that an Er:YAG laser may be superior to conventional debridement in the treatment of smokers with recurring chronic inflammation. This appears to be the first time that repeated Er-YAG laser instrumentation has been compared with mechanical instrumentation of periodontal sites with recurring chronic inflammation over a clinically relevant time period.

Key words: chronic periodontitis; clinical trial; Er:YAG laser; maintenance; periodontal pathogens; planing; scaling; smoking

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Conflict of interest and source of funding statement

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Laser devices with controlled penetration depth, and with power and wavelength suitable for ablation of soft and hard tissues, have increased the range of potential applications of

lasers in periodontal therapy. Whereas, the carbon dioxide (CO_2) and the neodymium-doped: yttrium, aluminium and garnet (Nd:YAG) lasers have shown non-favourable outcomes when used on the root surface (Spencer et al. 1992, Wilder-Smith et al. 1995), the erbium-doped: yttrium, aluminium and garnet (Er:YAG) laser appears to hold greater promise. This is closely related to its capacity for ablating soft and hard deposits on the root surface with minimal thermal side effects (Schwarz et al. 2001a, Eberhard et al. 2003). As the Er:YAG laser has a wavelength (2.94 μm) close to the maximum absorption coefficient for water, absorption of the energy by water and hydrous organic components occurs rapidly, resulting in evaporation of water, microexplosive ablation and reduced heat accumulation (Walsh et al. 1989, Walsh & Cummings 1994, Aoki et al. 2004, Ishikawa et al. 2004). Furthermore, the Er:YAG laser may possess bactericidal effect (Ando et al. 1996) and the potential to remove bacterial endotoxins from the root surface because of the high coefficient of absorption of the applied light frequency by lipopolysaccharides (Yamaguchi et al. 1997, Folwaczny et al. 2003), properties one can envision being of great importance considering the pathogenesis of periodontal disease.

Controlled clinical trials have indicated that the Er:YAG laser is as effective as hand instruments (Watanabe et al. 1996, Schwarz et al. 2001b, Derdilopoulou et al. 2007, Sgolastra et al. 2012) or ultrasonic device (Sculean et al. 2004, Derdilopoulou et al. 2007) for subgingival instrumentation in the treatment of chronic periodontitis as evaluated by clinical and microbiological parameters.

Regarding care of maintenance patients with the Er:YAG laser, a key point might be its potential to ablate the root surface with minimal loss of tooth substance. Repeated root planing may lead to excessive removal of root substance (Lie & Leknes 1985, Schmidlin et al. 2001). Furthermore, sites with persistent chronic inflammation and residual pockets ≥ 5 mm after treatment have been associated with greater risk for periodontal disease progression and tooth loss (Claffey & Egelberg 1995, Renvert & Persson 2002, Matuliene et al. 2008), and therefore represent a particularly demanding and complex challenge in periodontal therapy.

Thus, the purpose of this randomized, prospective, controlled clinical trial is to compare in a split-mouth design, the clinical and microbiological effects of repeated Er:YAG laser treatment with conventional mechanical debridement (ultrasonic/curette scaling) at 3-month intervals in maintenance patients.

Material and Methods

Study subjects

Fifteen maintenance patients (three women), with recurring chronic inflammation were recruited to this prospective, controlled, single-masked, clinical trial between November 2007 and September 2008 from a list of patients under periodontal maintenance care at the Faculty of Medicine and Dentistry, University of Bergen, Bergen, Norway (Table 1). The patients were examined for eligibility and included if they fulfilled the inclusion criteria. The inclusion criteria included healthy subjects who had no systemic diseases and were not taking medication that would affect periodontal healing, must have received supportive therapy every 3–6 months for 2–3 years following primary periodontal treatment, had not used antibiotics in the 6 months prior to the treatment, had four teeth with probing depth (PD) ≥ 5 mm, two teeth each in different jaw quadrants with bleeding or pus on probing and no signs of apical pathology, and ≥ 6 months since last session of subgingival scaling. To simplify the study design, teeth with furcation involvement were excluded. The mean age of the recruited patient sample was 57.7 (range: 43–74 years). All patients were smokers (≥ 10 cigarettes/day for ≥ 5 years; Table 1).

The study protocol was approved by the Institutional Medical Research Ethics Committee (07/8298 – 129.07), University of Bergen, Norway. All patients were provided with detailed information about the study before signing an informed consent form. The study was in accordance with the Helsinki Declaration of 1975, as revised in 1983 and 2008.

Periodontal treatment

In each patient, the two deepest non-adjacent pockets in each jaw quadrant with bleeding or pus on probing were selected as experimental sites. Following baseline examination, the jaw quadrants were randomly assigned (by coin toss) to either laser debridement (test) or ultrasonic/curette instrumentation

Table 1. Patient and site characteristics

Patients	Gender	Age (years)	Smoking (years)	Ultrasonic/curette treatment* Tooth no.	Laser treatment† Tooth no.
1	M	66	51	45, 47	22, 23
2	F	54	30	23, 25	13, 15
3	M	50	30	46, 45	24, 25
4	M	59	40	14, 15	24, 25
5	M	44	28	12, 15	21, 22
6	M	51	35	21, 23	12, 15
7	M	56	25	14, 15	23, 25
8	M	60	15	24, 26	12, 15
9	M	57	30	22, 24	13, 15
10	M	43	30	33, 32	22, 25
11	M	71	58	11, 13	22, 25
12	F	64	15	22, 21	14, 15
13	M	63	35	12, 15	21, 25
14	F	74	45	21, 23	11, 13
15	M	54	30	21, 25	22, 23

*Ultrasonic/curette: Piezon Master 400 Perio Slim Tip/Gracey SAS, Hu-Friedy.
†Laser: Erbium-doped:yttrium,aluminium, and garnet (Er:YAG) laser.

(control). The sequence of the treatment was randomized in a similar way. Test as well as control sites were treated by one operator at the same appointment under local anaesthesia (Fig. 1).

The Er:YAG laser unit (KEY 1234 with handpiece P2061, KaVo Dental GmbH, Warthausen, Germany) was used with a chiselled tip with a rectangular end (1.1 × 0.5 mm). The energy level was set to 160 mJ/pulse and the pulse frequency rate to 10 Hz (26). The fibre tip was inserted into the pocket, slightly angulated at 15–20° to the root surface (Folwaczny et al. 2003), and the laser was activated with simultaneous supply of water spray and slow movement of the prism in the apical direction until bottom of the pocket was reached. The feed-back option of the unit was disabled (Krause et al. 2007), and instrumentation was terminated when the operator judged debridement to be adequately clean and smooth. Periodontal pockets assigned to the control instrumentation were first mechanically debrided using an ultrasonic scaler (Piezon Master 400 Perio Slim Tip; Electro Medical System, Nyon, Switzerland) with power set to 75% and water as coolant, and then root planed with sharp curette (Gracey SAS, Hu-Friedy, Chicago, IL, USA). Instrumentation was terminated when the operator judged debridement to be adequately clean and smooth. If the included teeth showed no residual pocket and bleeding on probing, only polishing was performed.

Supragingival cleaning was performed using a rubber cup and a low abrasive polishing paste before the initiation of subgingival instrumentation. All patients received oral hygiene instructions at each appointment. The test and the control sites were treated at baseline, 3, 6 and 9 months by the same operator.

Clinical assessments

The clinical examination was done by an experienced operator who was not involved in the instrumentation procedures and was masked with regard to the treatment assignment. The following clinical parameters were recorded at baseline and at 6 and 12 months postoperatively:

Full mouth dental plaque (FMDP): percentage of tooth surfaces with visible plaque following staining with disclosing soultion (O'Leary et al. 1972) at the mesial, buccal, distal and lingual surfaces.

Full mouth gingival bleeding (FMGP): percentage of sites showing bleeding on gentle probing assessed at the mesial, buccal, distal and lingual surfaces (Ainamo & Bay 1975).

Probing depth at experimental and control sites: measured as the

Instrumentation à l'aveugle

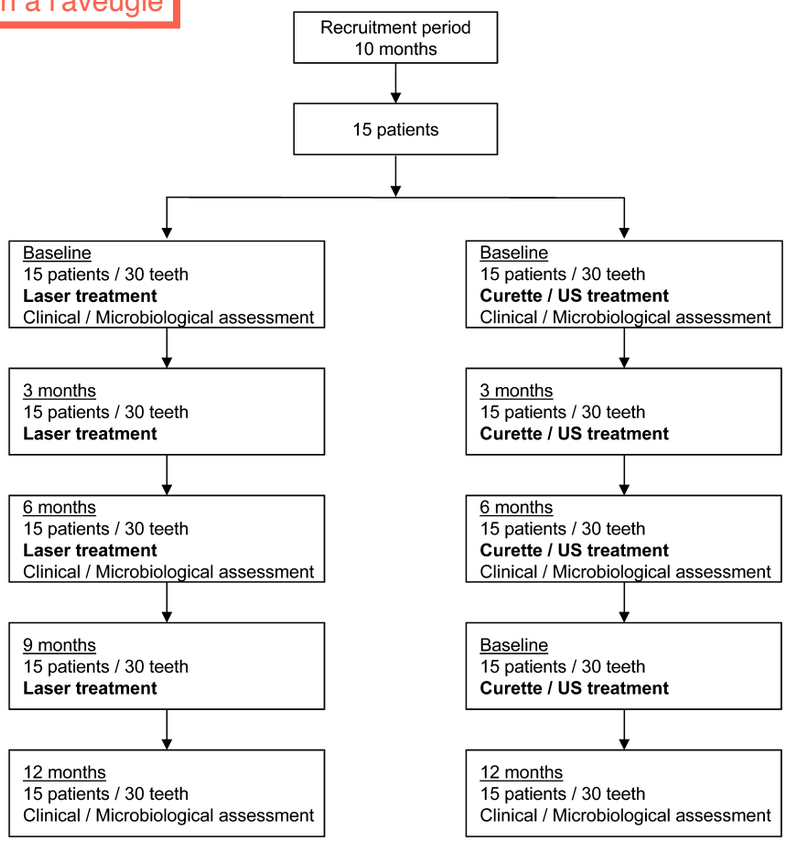

Fig. 1. Flow chart of the study design.

distance in millimetres from the gingival margin to the base of the probeable pocket using the Florida Probe (Florida Probe Corporation, Gainesville, FL, USA) with 0.1 mm increments and a probe tip diameter of 0.4 mm (Gibbs et al. 1998). The standard probing force was set to 25 *g* and a single pass measurement was done.

Relative attachment level at experimental and control sites: measured as the distance in millimetres from the occlusal disc located at the cusp/incisal edge to the base of the probeable pocket using the Florida Probe.

A separate sample of 17 subjects were measured twice 5–7 days apart to determine the intra-examiner reliability for the primary clinical outcome variables PD and RAL.

Microbiological assessments

At baseline and at follow-up examinations after 6 and 12 months, microbial samples were taken with sterile paper points in both test and control sites. Before sampling with four sterile paper points for each site, the area was carefully cleaned of supragingival plaque and kept dry during the sampling procedure. The paper points were placed gently to the bottom of the pocket and kept for 30 s before being removed and immersed into a pre-reduced transport medium (PRAS Dental Transport Medium, Morgan Hill, CA, USA). All sample tubes, separately pooled by treatment, were sent to Microbiological Diagnostic Service at the Institute for Oral Biology, Faculty of Dentistry, University of Oslo, Norway for analysis by the checkerboard technique (Socransky et al. 2004). The analyses of all samples were performed according to standard procedure at the Microbiological Diagnostic Service. The results of the microbiological parameters were reported separately for each sample, showing both qualitative and quantitative results.

The samples were analysed for detection of the species in Socransky's red complex (Socransky et al. 1998), *Porphyromonas gingivalis, Treponema denticola and Tannerella forsythia*. In addition, the detection included the following species: *Aggregatibacter actinomycetemcomitans, Prevotella intermedia, Fusobacterium nucleatum* subsp. *polymorphum, Fusobacteriuem nucleatum* subsp. *vincentii* and *Prevotella nigrescens*.

Statistical analysis

Primary clinical outcome variables were changes in PD and RAL. A difference of 0.5 mm between the treatments from baseline to 12 months was considered to be clinically relevant for both variables. Assuming that the standard deviation of the differences between the changes in each variable (PD and RAL) is 0.7 mm, the power analysis based on a sample of 15 subjects and with the level of significance (α) set at 0.05 resulted in 86% power to detect a true difference of 0.5 mm.

Descriptive statistics (mean, standard deviation, minimum and maximum) were calculated separately for the test and control treatments at baseline, 6 and 12 months for clinical variables. A repeated measure ANOVA with two within factors (time and treatment) was used to analyse PD and RAL, with time and treatment considered as fixed factors. The overall analysis was first employed, followed by the Bonferroni approach of multiple comparisons if any significant difference was detected. For PD, the ANOVA revealed no overall significant difference between treatments ($p = 0.261$) or interaction ($p = 0.261$), but along time ($p = 0.001$). The corresponding p values for RAL were 0.533, 0.014 and 0.855. The secondary outcome variables, plaque, bleeding on probing and total number of bacteria were analysed by the Friedman test for each treatment separately, followed by the Wilcoxon signed rank test to make comparisons of paired data if a significant overall result was found ($p < 0.05$). When conducting the Wilcoxon test, the level of significance was reduced according to the number of comparisons.

To compare the binomial proportions between the treatments on each time point and between different time points for each treatment, the McNemar test for matched-pair data was used. This was done for each pathogen separately. The level of significance was set at 0.05. In all analyses, the patient represents the experimental unit.

Results

All 15 patients returned for all scheduled maintenance visits. For repeated measurements of PD and RAL of the separate sample of 17 subjects, the intra-class correlation coefficients (ICCs) were calculated separately for every site. The ICC ranged between 0.61 and 0.85 for PD and between 0.94 and 0.97 for RAL. The better reproducibility of the RAL measurements was related to greater degree of variance.

Clinical findings

The number of experimental teeth exhibiting supragingival plaque was high for both treatments throughout the study with no significant difference between test and control. At baseline, all 30 teeth (100%) in the control treatment showed visible plaque following staining. By 6 and 12 months, the numbers were reduced to 25 (83%) and 23 (77%) respectively. In the test treatment, the number of teeth with visible plaque at baseline and at 6 and 12 months was 27 (90%), 27 (90%) and 25 (83%) respectively.

At baseline, 26 teeth (87%) in the control showed bleeding on probing, whereas at 6 and 12 months, the numbers were 23 (77%) and 25 (83%) respectively (Fig. 2). In the test, the number of teeth with bleeding on probing at baseline and at 6 and 12 months was 26 (87%), 20 (67%) and 22 (73%) respectively.

For the variable PD, a statistically significant reduction occurred in both treatments from baseline to 6 months and from baseline to 12 months ($p < 0.01$). The difference in mean PD between 6 and 12 months was not significant ($p = 0.52$ and $p = 0.95$ for control and test respectively). In the control, the mean initial PD decreased from

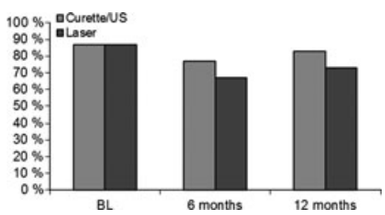

Fig. 2. Percentage of sites with bleeding on probing by observation period.

Table 2. Probing depth (mm) at baseline and 6 and 12 months ($N = 15$)

Treatment*	Baseline		6 Months		12 Months		Δ_{0-6}	*p* value	Δ_{0-12}	*p* value	Δ_{6-12}	*p* value
	Mean	SD	Mean	SD	Mean	SD						
Curette/US	5.4	0.3	3.6	1.5	4.0	1.5	−1.8 ± 1.4	0.001†	−1.4 ± 1.5	0.009†	0.4 ± 0.9	0.517
Laser	5.3	0.5	3.7	0.8	3.4	1.1	−1.6 ± 0.7	<0.001†	−1.9 ± 0.9	<0.001†	−0.3 ± 1.1	0.949

*No treatment differences were detected at any time point ($p = 0.416$).
Δ_{0-6} = difference between baseline and 6 months; Δ_{0-12} = difference between baseline and 12 months; Δ_{6-12} = difference between 6 months and 12 months.
†Significant differences between baseline and 6 months and baseline and 12 months ($p < 0.01$).

5.4 to 3.6 mm at 6 months and then increased to 4.0 mm at 12 months (Table 2). For the test, the initial PD decreased from 5.3 mm to 3.7 mm at 6 months and further to 3.4 mm at 12 months. No discernible treatment differences were detected at any time point ($p = 0.416$; Table 2).

The mean RAL showed no overall significant inter – ($p = 0.533$) or intra-treatment differences ($p = 0.855$). For both treatments, only minor changes in RAL occurred over time (Table 3). From baseline to 12 months, the control group gained 0.2 mm, whereas no change in mean RAL was recorded for the test group.

Microbiological findings

Total bacterial scores showed an overall significant difference by time for the control treatment ($p = 0.002$) and a borderline difference for the test group ($p = 0.05$). The difference between treatments was not significant ($p > 0.05$). Comparable values of bacterial counts were recorded at different time points for the two treatments (Fig. 3). In the control treatment, a significant reduction in bacterial counts was observed from baseline to 6 months ($p = 0.008$) and to 12 months ($p = 0.003$), whereas the reduction between 6 months and 12 months was not significant following Bonferroni approach of multiple comparisons ($p = 0.037$). The reduction in bacterial counts for the test treatment from baseline to 6 and 12 months was close to significant ($p = 0.022$ and $p = 0.$ 0.019, respectively), whereas the reduction between 6 months and 12 months was not significant ($p = 0.115$).

Figure 4a,b and c show the number of patients harbouring different proportions of target pathogens at baseline (a), and at 6 (b) and 12 (c) months. For both treatments, the prevalence of *P. gingivalis* decreased significantly from baseline to 6 months ($p = 0.016$ and $p = 0.039$ for control and test, respectively), and within the test treatment, a significant reduction was observed from baseline to 12 months ($p = 0.016$). At 12 months, *P. gingivalis* was totally eradicated in the control, whereas one patient in the test harboured *P. gingivalis*.

The prevalence of *T. forsythia* decreased significantly from baseline to 6 months within the test ($p = 0.021$) and the pathogen was not detected at 12 months. In the control, the reduction from baseline to 6 and 12 months was not significant ($p = 0.289$ and $p = 0.$ 250 respectively).

For both treatments, the prevalence of *T. denticola* showed a non-significant change from baseline to 6 months ($p = 0.688$ and $p = 0.453$ for control and test respectively). A rebound was observed from 6 to 12 months for the test ($p = 0.289$; Fig. 4b,c).

A significant reduction in prevalence of *A. actinomycetemcomitans* from baseline to 6 months was only achieved for the control ($p = 0.008$; $p = 0.109$ for the test group). However, from 6 to 12 months, a rebound was observed for both treatments (Fig. 4b,c).

Discussion

To our knowledge, this is the first time that repeated Er:YAG laser instrumentation has been compared with repeated mechanical instrumentation of periodontal sites with recurring chronic inflammation over a 12-month period of time. It also appears to be the first study evaluating the effect of Er:YAG laser treatment in a sample of only smokers. In short, clinical outcomes and microbiological results throughout the 12-month period did not demonstrate in non-furcated teeth, any distinct advantage in using the Er:YAG laser for repeated subgingival debridement compared with conventional mechanical debridement.

The overall compliance towards oral hygiene instructions and information was poor throughout the course of the study as revealed by the consistently high plaque and bleeding scores. This is in contrast to other clinical studies, which all have reported significant improvement in BOP (Schwarz et al. 2001b, Schwarz et al. 2003, Tomasi et al. 2006, Lopes et al. 2010). The exact reason for this lack of compliance in the study population may be hard to pinpoint, but presumably reflects weariness and loss of motivation after years of treatment without lasting results.

Both SRP and laser treatment showed significant reduction in PD from baseline to 12 months without any significant difference between the two treatments. This is in accordance with results of earlier studies (Sculean et al. 2004, Crespi et al. 2007, Lopes et al. 2010). Other studies have reported both short term (Tomasi et al. 2006) and long term (Crespi et al. 2007) results in favour of treatment with the Er:YAG laser. Tomasi et al. (2006) found significantly greater PD reduction in favour of the test sites at 1 month ($p < 0.01$), but this difference was not significant at the 4-month examination. For the Er:YAG group, Crespi et al. (2007) reported statistically significant and consistently greater reduction in PD in a 2-year study period for pockets of 5–6 mm ($p < 0.01$) and ≥ 7 mm ($p < 0.001$).

Compared to other studies (Schwarz et al. 2003, Sculean et al. 2004,

Table 3. Attachment level (mm) at baseline and 6 and 12 months ($N = 15$)

Treatment*	Baseline		6 Months		12 Months		Δ_{0-6}	p value	Δ_{0-12}	p value	Δ_{6-12}	p value
	Mean	SD	Mean	SD	Mean	SD						
Curette/US	11.9	2.7	11.5	2.9	11.7	2.8	−0.4 ± 0.8	0.138	−0.2 ± 1.2	>0.9	0.2 ± 0.8	0.855
Laser	12.0	2.1	12.2	1.7	12.0	1.8	0.2 ± 0.9	0.958	0.0 ± 1.2	>0.9	−0.2 ± 0.7	0.882

*No treatment differences at any time point ($p = 0.533$).
Δ_{0-6} = difference between baseline and 6 months; Δ_{0-12} = difference between baseline and 12 months; Δ_{6-12} = difference between 6 months and 12 months.

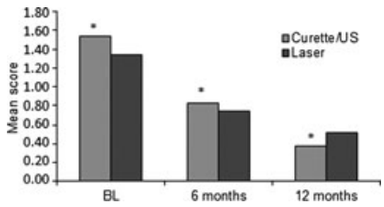

Fig. 3. Total bacterial scores for the control and test treatment at baseline and at 6 and 12 months. *Significant difference between baseline and 6 months and baseline and 12 months ($p < 0.01$).

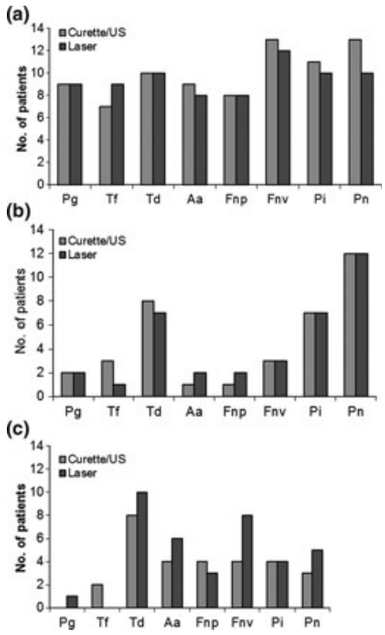

Fig. 4. (a) Number of patients harbouring different pathogens at baseline. (b) Number of patients harbouring different pathogens at 6 months. (c) Number of patients harbouring different pathogens at 12 months.

Tomasi et al. 2006, Crespi et al. 2007, Lopes et al. 2010, Rotundo et al. 2010, Ratka-Krüger et al. 2012), a striking feature in this study was the almost complete lack of RAL gain. The recorded differences within and between treatments were not significant. A reason why the statistically significant reduction in PD is not reflected in attachment gain might be that the reduction observed was mainly due to gingival recession (GR), which has been reported to be greater in smokers (Albandar et al. 2000). One can also suspect that the low standard of oral hygiene among participants in this study has had a detrimental effect on attachment gain. Patients with a low frequency of plaque-free tooth surfaces reportedly have a higher frequency of sites showing inflammation and additional loss of attachment (Nyman et al. 1977, Lindhe et al. 1984). All participants in this study were smokers. Smoking, second to bacterial plaque, is the strongest modifying risk factor for periodontal disease (Johnson & Guthmiller 2007), is associated with a higher risk for periodontal attachment loss (Machtei et al. 1997, Bergstrom 2003), and has a negative influence on the response to periodontal therapy (Heasman et al. 2006).

Intra-group improvement in clinical attachment level (CAL) has been reported in several studies (Schwarz et al. 2003, Sculean et al. 2004, Tomasi et al. 2006, Crespi et al. 2007, Lopes et al. 2010, Rotundo et al. 2010, Ratka-Krüger et al. 2012), but only two (Schwarz et al. 2003, Crespi et al. 2007) have reported statistically significant differences between test and control groups for CAL gain.

It seems that because of a wavelength that is well absorbed by water and lipopolysaccharides, the Er:YAG laser has antimicrobial and detoxification abilities (Ando et al. 1996, Folwaczny et al. 2002, 2003). Even so, this study did not express any difference in values of total pathogens between the SRP and the laser treatment. We did find significant reduction in total pathogens within the treatment groups and also reduced numbers of patients harbouring target pathogens. The most noticeable change took place between baseline and the 6-month control. No significant differences between the treatments were

observed. Other studies (Schwarz et al. 2001b, 2003, Derdilopoulou et al. 2007, Lopes et al. 2010) have reported on microbiological data, but because these data have been obtained through different techniques and to some degree focus on different types of microbiota, it is difficult to draw valid conclusions. Considering bacteria belonging to the "red complex" and "orange complex" as defined by Socransky et al. (1998), this study presents some valid results. Both treatments showed a significant decrease in the prevalence of *P. gingivalis* from baseline to 6 months and within the test, a significant reduction was observed from BL to 12 months. Prevalence of *T. forsythia* decreased significantly from BL to 6 months in the test and the pathogen was not detected at 12 months. Both of these species belong to the "red complex". Smoking may strongly have influenced the microbiological results. Studies have reported less reduction in periodontal pathogens in smokers, compared with non-smokers, following scaling and root planing (Darby et al. 2005, Van der Velden et al. 2003). One can speculate that any advantageous effect of the Er:YAG laser on the microbiological composition was lost due to a strong adverse effect of smoking, masking any potential differences.

We acknowledge some limitations of this study. It should be noted that the sample size was relatively small and homogeneous, and as such, results must be interpreted with caution. Longitudinal changes in gingival inflammation cannot exclusively be attributed to plaque accumulation or biofilm dispersion, but are also influenced by factors such as hormones and dietary effects (Jönsson et al. 2011). This study as well as other studies evaluating the effectiveness of Er:YAG laser have utilized a split-mouth design. Carry-across effects between treatments may be an issue in such studies (Schwarz et al. 2008), contributing to biased results (Hujoel & Rouen 1992, Sgolastra et al. 2012). In addition, repeated recall visits with one examiner and one operator may potentially create a shortcoming in the masking procedure.

Due to heterogeneity in study design and currently only few published, well-designed clinical trials, definite conclusions regarding the use of the Er:YAG laser in non-surgical periodontal treatment cannot be drawn. Yet, our data indicate that there is no evidence for claiming superiority of the Er:YAG laser compared with SRP procedures in the treatment of smokers with recurring chronic periodontal inflammation.

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Clinical Relevance

Scientific rationale for the study: Due to its capacity for subgingival debridement with minimal thermal side effects and its antimicrobial potential, the erbium-doped: yttrium, aluminium and garnet (Er:YAG) laser appears to hold promise in the treatment of periodontal disease. However, whether these treatment effects are of clinical relevance needs to be critically validated.

Principal findings: The 12-month results revealed no significant between-treatment differences with respect to clinical and microbiological outcomes of subgingival debridement performed with Er:YAG laser and scaling and root planing in 15 maintenance patients with recurring chronic inflammation.

Practical implications: The two treatment approaches appear to have similar clinical and microbiological effects.