Journal of Clinical Periodontology
Official Journal of The European Federation of Periodontology
Founded by the British, Dutch, French, German, Scandinavian, and Swiss Societies of Periodontology

J Clin Periodontol 2009; 36: 650–660 doi: 10.1111/j.1600-051X.2009.01433.x

Journal of Clinical Periodontology

Immediate efficacy of diode laser application in the treatment of dentine hypersensitivity in periodontal maintenance patients: a randomized clinical trial

Alberto Sicilia[1,2], Susana Cuesta-Frechoso[2,3], Alfonso Suárez[2], Jorge Angulo[1,2], Armando Pordomingo[1,2] and Pablo De Juan[1,2]

[1]Clinica Sicilia, Oviedo, Spain; [2]Section of Periodontology, Department of Surgery, University of Oviedo, Oviedo, Spain; [3]Clinica Tejerina, Gijón, Spain

Sicilia A, Cuesta-Frechoso S, Suárez A, Angulo J, Pordomingo A, De Juan P. Immediate efficacy of diode laser application in the treatment of dentine hypersensitivity in periodontal maintenance patients: a randomized clinical trial. J Clin Periodontol 2009; 36: 650–660. doi: 10.1111/j.1600-051X.2009.01433.x.

Abstract

Background: To evaluate the immediate efficacy in the reduction of dentine hypersensitivity (DH) when applying an 810 nm diode laser (DL), and a 10% potassium nitrate bioadhesive gel (NK10%).

Material and Methods: Forty-five consecutive periodontal maintenance patients of both sexes, with a DH $\geq$ 2 on the verbal rating scale (VRS) in one or more teeth, were randomly allocated into three equal groups: 15 patients received DL and placebo gel; 15 patients were tested with a placebo laser and NK10%; and the remaining 15 received a placebo laser and placebo gel. The DH was evaluated at the start of the study, 15 and 30 min. after the laser application, and on days 2, 4, 7, 14, 30 and 60 by a blind examiner.

Results: After 15 min., observations showed a reduction in DH after an evaporative stimulus (ES) of 36.9% (0.86), three times greater than that of the control group (0.23) ($p = 0.008$). After 14 days, this effect was even greater [DL 71.7% (1.67)/NK10% 36.3% (1.73)/control 28.1% (0.73); $p = 0.004$], and lasted until day 60 [65.7% (1.53)/ 30.4% (0.73)/25.8% (0.67); $p = 0.01$].

Conclusions: The DL and NK10% gel were proven effective in the treatment of DH. A significantly greater immediate response was observed with DL.

Key words: bioadhesive gel; dentine hypersensitivity; diode laser; periodontal patients; potassium nitrate

Accepted for publication 23 April 2009

Conflicts of interest and sources of funding statement

The present project has been carried out with the research funds of the Section of Periodontology of the University of Oviedo and with the selfless support of two private clinics specialized in Periodontology. No external financing source has provided any funds. The Spanish company Lacer S.A., has cooperated by means of its research laboratory, in the design of the trial and providing the 10% potassium nitrate (Sensilacer Gel®) and placebo gels. For the fulfilment of the study, Biolase Technology Inc. (San Clemente, CA, USA) donated a LaserSmile® unit to the Section of Periondontology of the University of Oviedo.

Disclosure of any conflicts of interest: There are no conflicts of interest to disclose in the present study.

Dentine hypersensitivity (DH) is a painful response of the tooth to different stimuli such as brushing, acid diets, occlusal overload and thermal changes (Addy & Urquhart 1992). It is characterized by an acute, non-spontaneous, short- or long-lasting pain that appears suddenly in a specific location, which cannot be attributed to any other dental pathology (Holland et al. 1997). It is a highly common occurrence (Addy 2002) that is easy to diagnose with a routine examination.

The DH mechanism could perhaps be explained by a combination of two theories: the ''hydrodynamic theory'' (Brannstrom & Astrom 1972) and the ''neural theory'' (Seltzer et al. 1963). These suggest that various external stimuli cause a movement of fluids in the dentinal tubules, producing a stretching or compressing of the outermost odontoblasts of the pulp and of the nerve endings connected to them, causing pain (Holland et al. 1997, Dababneh et al. 1999, Kimura et al. 2000, Walters 2005). DH therefore depends on the existence of open dentinal tubules and pulp vitality, although further study is needed to determine the exact mechanism through which the flow of fluid in the dentinal tubules stimulates the nerve endings (Dababneh et al. 1999).

Multiple treatments have been proposed, showing variable results (Hodosh 1974, Kanapka 1990, Echeverría et al. 1991, Sicilia et al. 1996, Frechoso et al. 2003, Pillon et al. 2004, Yates et al. 2004), the most commonly used today being potassium salts (Jacobsen & Bruce 2001). An oxidizing effect, or blocking of tubules through crystallization, have been proposed as possible mechanisms of action for potassium nitrate in reducing DH (Hodosh 1974, Addy & Dowell 1983). Various clinical studies have proven the efficacy of potassium nitrate, in the form of toothpaste, mouthwash and bioadhesive gel, as a desensitizing agent (Hodosh 1974, Green et al. 1977, Tarbet et al. 1980, Nagata et al. 1994, Sicilia et al. 1996, Frechoso et al. 2003).

Another recent proposal is the irradiation of affected teeth with different types of laser, having tested low (He–Ne, diode) and middle output power (CO_2, Nd:YAG) lasers for the reduction of DH, although there is little documentation on their efficacy (Renton-Harper & Midda 1992, Gelskey et al. 1993, Lan & Liu 1996, Zhang et al. 1998, Kimura et al. 2000, Marsilio et al. 2003, Ladalardo et al. 2004).

Low output power (low-level) lasers have been proven to have significant anti-inflammatory effects (Karu 1989, Zanirato et al. 2007), while middle output power lasers have excessive effects on pulp (Launay et al. 1987).

The diode laser (DL) (gallium/aluminium/arsenide – GaAlAs) is able to generate a continuous wave without overheating (Kimura et al. 2000). This type of technology has been tested with different output power levels, combining wavelengths ranging from 660 to 900 nm, and application periods from 60 to 150 s, on a single tooth per patient (Iida et al. 1993, Ladalardo et al. 2004). Within this range, tests to date have failed to demonstrate a greater efficacy using higher energy levels (Kimura et al. 2000, Ladalardo et al. 2004).

DL irradiation has enabled a DH reduction equal or superior to conventional treatments such as potassium nitrate, sodium fluoride, stannous fluoride and fluoride varnish (Yamaguchi et al. 1990, Iida et al. 1993, Ladalardo et al. 2004). However, the lack of an authentic control group (with a placebo laser) in these studies means that its efficacy cannot be accurately evaluated.

The main hypothesis that has been tested in the current study was that a 60-s application of an 810 nm DL to a tooth with DH would lead to significantly greater immediate reduction of DH [measured by means of an evaporative stimulus (ES)] than with placebo. At the same time and secondarily, the efficacy of DL with respect to an already tested treatment (NK 10% bioadhesive gel) was studied, as well as the efficacy of the various types of treatment evaluated by tactile stimulation (TS) and by a global subjective evaluation (GSE); the appearance of adverse effects was also assessed.

Material and Methods

Forty-five consecutive patients with DH (18 men and 27 women) between the ages of 19 and 70 (mean age: 41.67 years) were selected from April to July 2006, from the maintenance programme of the Section of Periodontology of the Oviedo Faculty of Odontology and two private clinics specialized in Periodontology in Asturias (Spain).

The patients had followed the maintenance programme for at least more than 1 year. They were initially diagnosed with chronic periodontitis (moderate or advanced), although some patients with gingivitis or mild periodontitis with gingival recession were included. None of the participants in our sample suffered from aggressive periodontitis. The mean recession of the teeth selected was 2.16 mm (1.06 mm), 2 mm, also being the most frequently observed value. Three cases presented no recession (6.7%) and only one (2.2%) had a recession of 5 mm, the maximum value.

All of the patients were invited to volunteer in a randomized, triple-blind, placebo-controlled study, lasting for 60 days (ADA 1986).

In an external research centre (R+D Laboratory, Laboratorios Lacer, S.A., Barcelona, Spain), a computer-generated randomization list was created [BIOMOS 1.0-1995 (Biométrica Médica y Social S.L.)], and 15 patients were randomly allocated to each type of treatment:

1. Test group (15 patients): treatment with DL and placebo gel.
2. Positive control group (15 patients): treatment with placebo laser and NK10% gel.
3. Placebo group (15 patients): treatment with placebo laser and placebo gel.

A set of specifically designed patient dedicated folders was used in the study in order to preserve patients' privacy. The folders were only identified with the initials corresponding to the name and two surnames of each patient. According to the randomized procedure carried out, the material that enabled the application of the randomization and the protection of the blinding codes was prepared in the external research centre by personnel who were not to be involved in the study clinical proceedings, in the statistical analysis or in the writing of the manuscript. The aforementioned material included:

1. Forty-five transparent plastic bags with an envelope marked with the number assigned to the patient in the study and containing the card specifying the information regarding the laser treatment to be applied (''laser'' or ''placebo laser''), and a tube with no description of its contents (10% potassium nitrate or placebo gels), only bearing the number of the patient.
2. A second set of envelopes, also marked with the number of the patient only, containing full information about the treatment received (laser and gel) that was not opened until the study was completed.

In order to protect the blinding procedure, a three-member team in each clinic joined the study. One of the members, the examiner, was in charge of carrying out the initial assessment and the monitoring of patients, as well of teaching the way the gel should be

applied (NK10% or placebo) and the oral hygiene techniques. At all times, the examiner was not aware of the type of treatment applied. A second member of the team, with no information whatsoever on the patients' or study's characteristics, was in charge of opening the envelope containing the type of laser treatment to be used and of applying the said treatment. Finally, the third team member was in charge of the custody of the second set of envelopes with detailed information about the treatment that was applied. At the end of the study, those envelops were opened to build the study's data matrix.

The study included consecutive patients of both sexes, all above 18 years of age, who followed a periodontal maintenance programme, for at least 1 year, and showed a DH level of 2 or higher on the verbal rating scale (VRS) following the application of ES (cold air from a distance of 1 cm) to at least one tooth. The study excluded patients with systemic diseases or those having taken medication in the previous 30 days, those either pregnant or lactating, those with hypersensitivity to any of the components of the medications used in the study, those having used DH treatment in the previous 30 days and those with cavities, restorations, occlusal overload or recent occlusal adjustment to the tooth under study. Once accepted into the study, the patient was scheduled for an initial examination. The first member of the team recorded the DH level with ES, TS and GSE. At this session, patients were given instructions in oral hygiene techniques, avoiding aggressive cleaning habits, and were given a soft [Cepillo Lacer Technic Suave® (Lacer S.A., Barcelona, Spain)] toothbrush and prohibited from using any toothpaste or mouthwash.

After examining each patient, the second member of the team was appointed to open the envelope containing the allocation of the patient to the DL or the placebo laser group (DL placebo). This envelope also contained an unmarked tube with the NK10% or placebo gels. The selected tooth (with DH) was then subjected to irradiation treatment in a single session. The test group was treated using a DL with a wavelength of 810 nm and an inactive fibre, at an output power of 1.5–2.5 mW, for 1 min. (DL), while a placebo laser was used with the rest of the patients (positive control and placebo groups).

Once the DL or the placebo laser treatment was applied, relevant records were taken at 15 and 30 min. by the first member of the team, unaware of the applied treatment. This member also performed the first gel application (NK10% or placebo gels), and gave instructions to the patient about its use – apply at home twice a day for 14 days and return the randomly assigned unmarked tube (Fig. 1).

Patients were prohibited from daily brushing with a fluoride toothpaste. Further records were taken (ES, TS and GSE) on days 2, 4, 7, 14, 30 and 60.

After finishing the study, the second set of envelopes was opened. None of the participants, researchers or personnel performing statistical evaluations were aware of the treatment group to which each patient belonged.

All patients concluded the 2-month study, and were analysed according to the group they were randomly allocated in.

Clinical measures

The clinical efficacy of the treatments was evaluated using the following procedures:

1. ES was used as the main variable for the study. The selected tooth was isolated, dried and a jet of air was applied using a dental syringe from a distance of 1 cm for 1 s (Tarbet et al. 1979, Collins et al. 1984). Patient responses were recorded according to the VRS scale:

- 0: No discomfort, but patient felt stimulus.
- 1: Slight discomfort, but not painful.
- 2: Painful during application of stimulus.
- 3: Painful during application of stimulus and immediately afterwards.

2. To evaluate TS the stimulus was applied by scraping the exposed radicular surface of the tooth by means of periodontal probing (Collins et al. 1984, Silverman 1986). The patient's response was classified according to the aforementioned VRS scale.

3. The GSE (Tarbet et al. 1980, Silverman 1986, Clark et al. 1987, Minkoff & Axelrod 1987) was evaluated by recording the patient's level of sensitivity to common stimuli experienced in his or her daily life. The patient recorded his or her sensitivity level on a scale of 0–5 points, 0 for no sensitivity and 5 for maximum sensitivity.

Patients were monitored for the appearance of adverse effects. In case of an adverse effect, the protocol was to record its diagnosis, a description of its intensity and its accountability. The intensity of each effect was graded as light, moderate or intense.

The degree of treatment compliance was evaluated by measuring the weight of the tube of bioadhesive gel returned by the patient at the check-up on day 14, according to the following table:

- Correct (acceptable): Approximate actual consumption $>80\%$ of supply.
- Regular (acceptable): Approximate actual consumption 60–80% of supply.

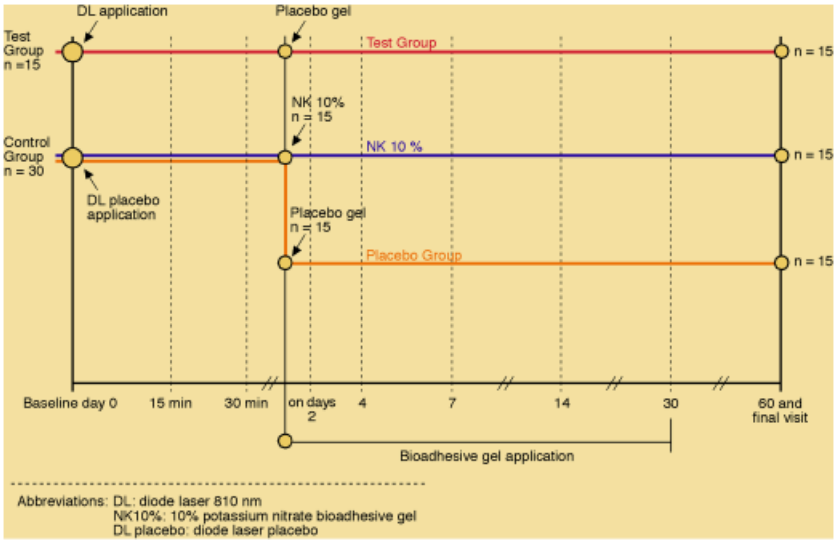

Fig. 1. Study diagram.

- Insufficient (unacceptable): Approximate actual consumption $<60\%$ of supply.

A detailed guide of the DH evaluation proceedings was written, and the personnel in charge of collecting data were specifically trained in clinical examination proceedings, treatment application and survey conduction (SC and AS).

Sample size calculation

Sample size was calculated in order for us to identify as significant a clinical difference in DH reduction of 30% between DL and placebo laser patients (power = 0.8, $\alpha = 0.05$, two-sided). According to this, the estimated sample size to assess the study's main hypothesis was 15 patients per group (30 patients). A 15-patient additional group was enrolled in the study, which was to be treated with NK10% gel in order to evaluate the secondary hypothesis.

Statistical analysis

The data were analysed using the SPSS 15.00 program. The relation between the treatment type and the reduction of DH, measured using ES, tactile stimulation and global subjective evaluation, was analysed using the Mann–Whitney *U*-test and the Kruskal–Wallis test, or Student's *t*-test and ANOVA test when normality of variables and homogeneity of variances could not be ruled out. The intra-group response to a specific treatment (before/after) was evaluated using paired Student's *t*-test.

According to the main and secondary hypothesis formulation, the analyse of the relationship between the main variable (DH reduction, measured by ES) and the type of treatment (DL *versus* placebo laser, or DL *versus* NK10%) are to be considered as pre-specified analyses. The remaining analyses should be considered as exploratory.

Results

42.2% of the participants emphasized the inclusion of citric acid in their daily diet, and 33.3% drank coca-cola every day. The tooth showing the most DH was the first upper right molar. The treatment was correctly completed by all patients, and no adverse events were detected in any of the three treatment groups during the entire 60-day study. In the initial phase of the study we had a group of 30 patients in the positive control and placebo groups, who were given treatment with an inactive laser, and a test group of 15 patients who received DL treatment (Fig. 1). In this first period, 15 min. after the laser was applied, we observed a reduction in the response to ES of 36.9% (mean = 0.86, SD = 0.92) in the test group, four times greater than that observed in the control group, which was 9.2% (mean = 0.23, SD = 0.43) ($p = 0.008$). Between 15 and 30 min. this effect was not so evident, with the gap remaining the same between the 2 groups. No baseline differences were observed between them (Table 1). Altogether, DH reduction in the DL group from baseline to 30 min. is 41.6% and that in the DL placebo group is 9.2%, showing a 32.4 percentage point greater reduction for the group treated with DL [95% confidence interval (CI): 19.4–45.4]. In this period, significant reductions were observed in both groups (Fig. 2). As for the response to tactile stimulation, the test group showed a reduction of 46% (mean = 0.40, SD = 0.74), five times greater than that observed in the control group, which was 9.7% (mean = 0.10, SD = 0.48), although this is not statistically significant. Between 15 and 30 min. after the laser application, the reduction was 3.3 times greater in the test group (57.4% in the test group compared with 17.2% in the control group) (Table 2). The reduction of tactile stimulation was significant in the test group after the 15- and 30-min. intervals (baseline), while this was lower yet significant after 30 min. (baseline) in the placebo group (Fig. 3).

Following the laser application treatment, patients received either the NK10% or the placebo gel according to the group they were in (Fig. 1), the two groups becoming three according to the combination of laser and gel applied.

Patients in the test group (DL), despite being given a single laser application at baseline, experienced a continuous improvement when ES was applied, lasting for up to 14 days (30-min.–day 14 reduction of 52.1%, $p = 0.003$). The positive control group (NK10%) experienced a clinical improvement from 30 min. up to day 7 (38.3%), displaying a small turnaround between days 7 and 14, with a 30-min.–day 14 reduction of 32.6% (non-significant) (Table 3, Fig. 4). Both reductions were much greater (52.1% and 32.6%) than that obtained in the placebo group (17.6%), although these differences are not statistically significant (Table 3).

After 30 min., baseline differences were observed between the groups, which may affect comparisons between them. This is due to the greater significant reduction observed between 0 and 30 min. in the DL group. To prevent this, the ES response was calculated from baseline through to day 14 for all groups, that is, from the start of the study and the laser treatment to the end of the application of the NK10% or placebo gels. Observations showed a significantly greater DH reduction after ES in the laser-treated patients (71.7%) (mean = 1.67, SD = 0.72), compared with 36.3% (mean = 0.87, SD = 1.73) and 28.1% (mean = 0.73, SD = 0.80),

Table 1. Reduction in dentine hypersensitivity response to evaporative stimulus in the group treated with diode laser and in control groups (placebo laser)

Group	Baseline $\bar{x}$ (SD)	15 min. $\bar{x}$ (SD)	RED 0–15 $\bar{x}$ (SD) %	30 min. $\bar{x}$ (SD)	RED 15–30 $\bar{x}$ (SD) %
Test (DL)	2.33 (0.49)	1.47 (0.83)	0.86 (0.92)	1.40 (0.73)	0.07 (0.59)
$n = 15$			36.9%		4.7%
Control (placebo)	2.50 (0.51)	2.27 (0.64)	0.23 (0.43)	2.27 (0.69)	0 (0.37)
$n = 30$			9.2%		0%
	$t = -1.065$	$t = -3.26$	UMW = 132	$t = -3.796$	UMW = 211
	$p = 0.3$	$p = 0.003$	$p = 0.008$	$p = 0.001$	$p = 0.629$

t, Student's *t*-test; UMW, Mann–Whitney *U*-test.
RED 0–15: DH reduction from baseline to 15 min.
RED 15–30: DH reduction from 15 to 30 min.

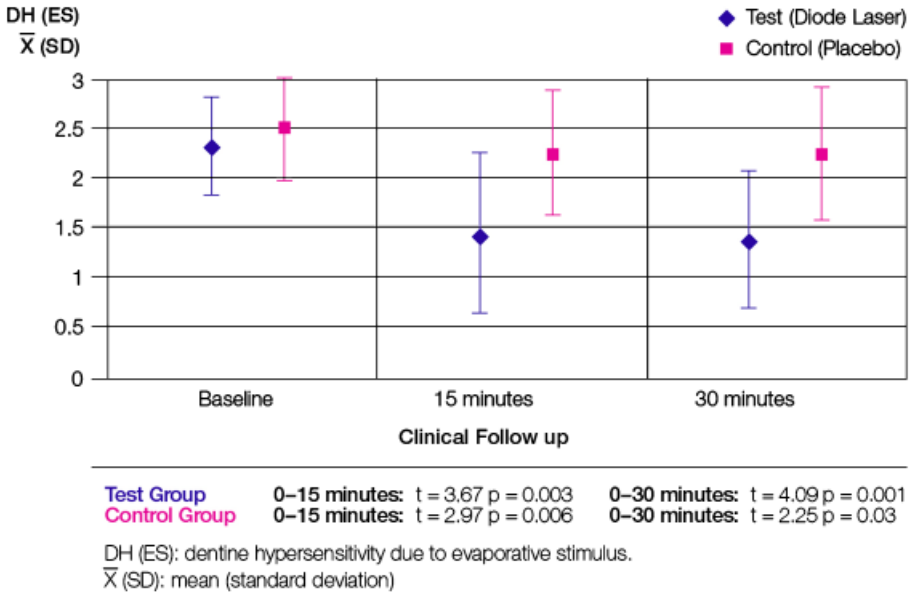

Fig. 2. Evolution of dentine hypersensitivity response to an evaporative stimulus in test and control groups (placebo laser), from baseline to 30 min.

Table 2. Reduction in dentine hypersensitivity response to tactile stimulation in the group treated with diode laser and in control groups (placebo laser)

Group	Baseline $\bar{x}$ (SD)	15 min. $\bar{x}$ (SD)	RED 0–15 $\bar{x}$ (SD) %	30 min. $\bar{x}$ (SD)	RED 15–30 $\bar{x}$ (SD) %
Test (DL)	0.87 (1.06)	0.47 (0.52)	0.40 (0.74)	0.20 (0.41)	0.27 (0.46)
$n = 15$			46%		57.4%
Control (placebo)	1.03 (0.85)	0.93 (0.83)	0.10 (0.48)	0.77 (0.73)	0.16 (0.38)
$n = 30$			9.7%		17.2%
	$t = -0.53$	$t = -2.32$	UMW = 186.5	$t = -2.79$	UMW = 202
	$p = 0.60$	$p = 0.03$	$p = 0.22$	$p = 0.008$	$p = 0.43$

t, Student's *t*-test; UMW, Mann–Whitney *U*-test.
RED 0–15: DH reduction from baseline to 15 min.
RED 15–30: DH reduction from 15 to 30 min.

in the positive control and placebo groups, respectively. As a result, at 14 days, the observed DH reduction in the DL group is 43.6 percentage points greater than the one observed in the placebo group (95% CI: 25.6–61.6) and 35.4 points greater than the one detected in the NK10% group (95% CI: 18.4–52.4).

After the 2 weeks of the study, a small, non-significant change was observed in all groups between days 30 and 60 (Fig. 4). A global evaluation of the 30-min.–60-day period showed a reduction of 45% in the test group, compared with 25.8% in the positive control group and 13.5% in the placebo group, none of which reached levels of statistical significance (Table 3). In agreement with the 30-min.–14-day reduction analysis, the data were affected by differences between the groups after 30 min.; therefore, we analysed the reduction from baseline through to the end of the study (day 60). With this approach we found the differences to be much clearer and statistically significant, the reduction in the laser-treated group reaching 65.7% (mean = 1.53, SD = 0.74), compared with 30.4% (mean = 0.73, SD = 1.10), and 25.8% (mean = 0.67, SD = 0.82), in the positive control and placebo groups, respectively (Table 4). As a result, at 60 days, the observed DH reduction in the DL group was 39.9 percentage points greater than the one observed in the placebo group (95% CI: 22.4–57.4) and 35.3 points greater than the one detected in the NK10% group (95% CI: 18.2–52.4).

With tactile stimulation, we observed that patients in the test group (DL) experienced a notable improvement up to 14 days, with the total disappearance of all symptoms, although this was not significant (30-min.–14-day reduction of 100% $p = 0.008$), and the patients underwent a turnaround after 30 days, yielding a final 30-min.–60-day reduction of 65% (Fig. 5). The positive control group (NK10%) experienced a clinical improvement from 30 min. to day 30, which remained unchanged until the end of the study on day 60 (30-min.–60-day reduction of 83% $p = 0.001$). Both reductions were far greater (65% and 83%) than that obtained in the placebo group (31%) and are statistically significant ($p = 0.017$) (Table 5). When trying to analyse the data, with baseline as a reference point, we discovered baseline differences for tactile stimulation, with the DH in the positive control group being significantly higher, which affected the interpretation of our

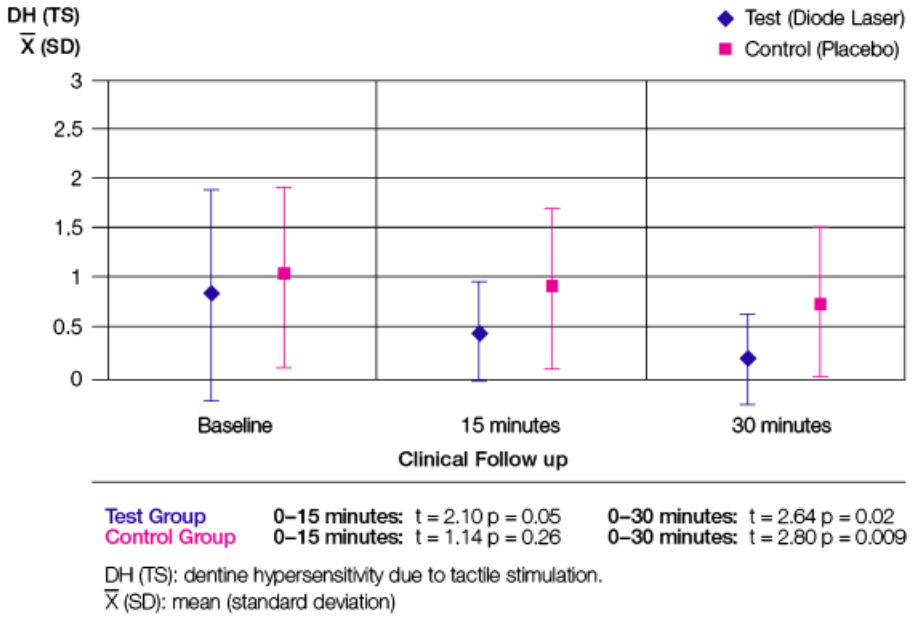

Fig. 3. Evolution of dentine hypersensitivity response to tactile stimulation in test and control groups (placebo laser), from baseline to 30 min.

Table 3. Reduction in dentine hypersensitivity response to evaporative stimulus in the three treatment groups from 30 min. to day 60 (end of the study)

Group	30 min. $\bar{x}$ (SD)	2nd day $\bar{x}$ (SD)	4th day $\bar{x}$ (SD)	7th day $\bar{x}$ (SD)	14th day $\bar{x}$ (SD)	RED 30 –14 $\bar{x}$ (SD) (%)	30th day $\bar{x}$ (SD)	60th day $\bar{x}$ (SD)	RED 30–60 $\bar{x}$ (SD) (%)
DL	1.40	1.27	1.07	1.0	0.67	0.73 (0.80)	0.67	0.80	0.60 (0.83)
$n = 15$	(0.73)	(0.88)	(0.59)	(0.65)	(0.62)	52.1%	(0.49)	(0.56)	45.1%
NK10%	2.27	1.80	1.53	1.40	1.53	0.74 (1.57)	1.53	1.67	0.60 (0.98)
$n = 15$	(0.69)	(0.77)	(1.06)	(1.12)	(1.51)	32.6%	(1.06)	(0.90)	25.8
Placebo laser and gel	2.27	2.20	2.0	1.93	1.87	0.40 (0.63)	1.93	1.93	0.33 (0.62)
$n = 15$	(0.69)	(0.77)	(0.75)	(0.80)	(0.91)	17.6%	(0.89)	(0.88)	13.5%
	$\chi^2 = 11.12$ gl = 2 $p = 0.04$	$\chi^2 = 8.55$ gl = 2 $p = 0.014$	$\chi^2 = 8.56$ gl = 2 $p = 0.014$	$\chi^2 = 7.44$ gl = 2 $p = 0.024$	$\chi^2 = 9.61$ gl = 2 $p = 0.008$	$\chi^2 = 1.38$ gl = 2 $p = 0.5$	$\chi^2 = 13.44$ gl = 2 $p = 0.01$	$\chi^2 = 12.80$ gl = 2 $p = 0.002$	$\chi^2 = 0.79$ gl = 2 $p = 0.67$

KW, Kruskall–Wallis test; df, degrees of freedom.
RED 30–14: DH reduction from baseline to 30 min. and on day 14.
RED 30–60: DH reduction from 30 min. to day 60.

results. However, a significantly greater reduction was observed in the test and positive control groups (92% and 89%) compared with the placebo group (34%) at the end of the study (Table 6).

In the global subjective evaluation, after 2 weeks, we observed a significant reduction with both treatments (DL: 69%, $p = 0.002$; NK10%: 58%, $p = 0.032$), which was non significant in the placebo group (41% $p = 0.29$) (Fig. 6). After 48 h, a greater reduction was observed in the DL (48%) and NK10% (35%) groups than in the placebo group (20%), although this was not statistically significant. This pattern reoccurred up to day 14, with more notable differences at the end of the study, displaying a baseline – 60-day reduction of 65% in the DL group, 58% in the NK10% group and 31% in the placebo group ($p = 0.04$) (Table 7).

Discussion

The application of a DL at a wavelength of <780 nm and at an output power below 30 mW, with an application time of <3 min., is a safe treatment with regard to pulp. Various studies have reported a lack of significant pulp damage or thermal alterations after irradiation of the radicular surface (Matsumoto et al. 1985, Arrastia et al. 1994, Kimura et al. 2000, Goharkhay et al. 2007). In our study, none of the 15 laser-treated patients showed secondary effects, which confirms the safety of this type of treatment.

However, inappropriate laser use could result in potential tissue damage, causing thermal lesions on the radicular surface, gingival tissues, dental pulp and adjacent bone (Schwarz et al. 2008). In view of these possible side effects, together with the wide variety of laser types and application protocols (many of which have not been scientifically tested) in the market, the practitioner must read thoroughly the security and efficacy documentation of the laser protocol that she/he wants to apply.

Traditional DH treatment is based on the application of desensitizing substances, which reduce or eliminate pain and are capable of stimulating the formation of dentine, which obliterates the dentinal tubules exposed to the oral environment (Kim 1986). According to

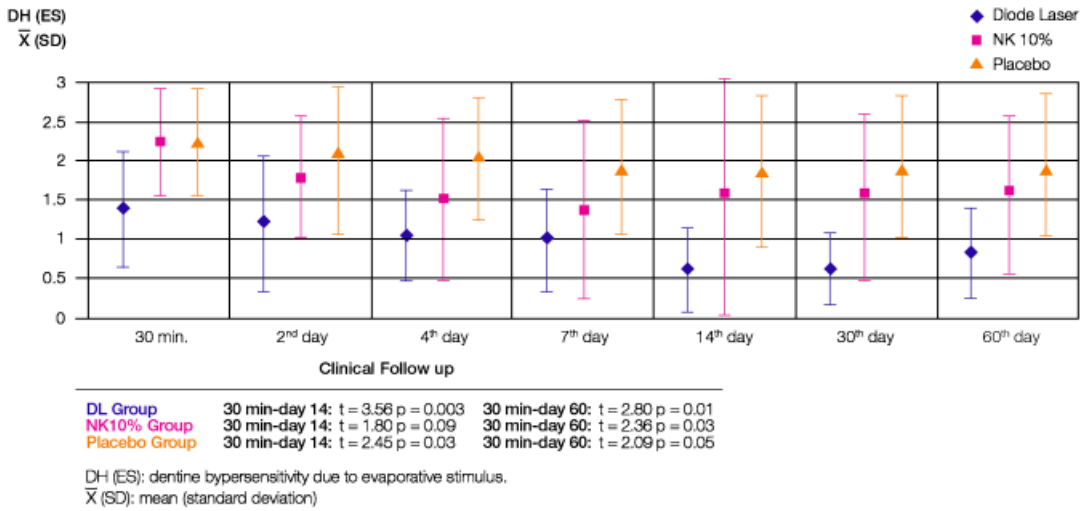

Fig. 4. Evolution of the response to an evaporative stimulus in the three treatment groups from 30 min. to day 60 (end of the study).

Table 4. Reduction in dentine hypersensitivity response to evaporative stimulus in the three treatment groups from the start of the study (baseline data) through to day 60 (end of the study)

Group	Baseline $\bar{x}$ (SD)	14th day $\bar{x}$ (SD)	RED 0–14 $\bar{x}$ (SD) (%)	60th day $\bar{x}$ (SD)	RED 0–60 $\bar{x}$ (SD) (%)
DL $n = 15$	2.33 (0.49)	0.67 (0.62)	1.67 (0.72) 71.7%	0.80 (0.56)	1.53 (0.74) 65.7%
NK10% $n = 15$	2.40 (0.51)	1.53 (1.51)	0.87 (1.73) 36.3%	1.67 (0.90)	0.73 (1.1) 30.4%
Placebo (laser and gel) $n = 15$	2.60 (0.51)	1.87 (0.91)	0.73 (0.80) 28.1% $\chi^2 = 6.43$ gl = 2 $p = 0.04$	1.93 (0.88)	0.67 (0.82) 25.8% $F = 6.96$ $p = 0.01$

KW, Kruskall–Wallis test; df: degrees of freedom.
ANOVA: Single-factor analysis of variance (ANOVA) test. *F*: Fisher–Snedecor *F*.
RED 0–14: DH reduction from day 0 (baseline) to day 14.
RED 0–60: DH reduction from day 0 (baseline) to day 60.

the literature, conventional treatment with potassium salts has demonstrated a significant reduction of DH in weeks (Tarbet et al. 1980, Reinhart et al. 1990, Nagata et al. 1994, Sicilia et al. 1996). However, because the elicitation of pain in DH patients is acute, the availability of a treatment that reduces or eliminates DH within a period of 24–48 h, or even earlier, would be ideal (Grossman 1935, Addy & Dowell 1983, Dababneh et al. 1999, Goharkhay et al. 2007). With this objective, a gel containing 10% potassium nitrate was designed, which produced led to DH reductions of 36% after 48 h and 66% after 96 h, significantly greater than with placebo (Frechoso et al. 2003). It was due to this proven, short-term clinical efficacy that we selected this gel as a positive control treatment in our study.

DL treatment is capable of partially sealing the dentinal tubules, as well as having an analgesic effect and stimulating the tropism of dental pulp (Kimura et al. 2000, Goharkhay et al. 2007). Low-level laser radiation acts generically on somatic pain such as hyperaesthesia, introducing an anaesthetic effect with surprising speed (Kimura et al. 2000). In this respect, previous studies, performed without a control group, have demonstrated a rapid reduction of DH in patients receiving DL treatment, fluctuating, excluding any methodological differences, between 50% and 70% after 15 min. and between 39% and 61% after 30 min. with DL (Ladalardo et al. 2004) and between 30% and 100% with methodological variants, such as the use of lasers with a wavelength of 830–900 nm (Yamaguchi et al. 1990, Iida et al. 1993).

From a methodological point of view, pain assessment in patients with DH gives rise to reproducibility issues that could hamper the correct assessment of results in clinical trials. Its reduced reproducibility causes a higher data variability; therefore, larger sample size studies must be designed (Ide et al. 2001). This aspect becomes more relevance when performing a subjective assessment of the patient's pain (Ide et al. 2001).When trying to assess the pain experienced by the patient during a relatively long procedure, as for instance an ultrasound prophylaxis on a group of teeth, an intermodal intensity comparison seems to be more adequate, as it creates a pain record whose timing matches exactly with pain peaks (Braun et al. 2007).

However, regarding pain recording from a short given stimulus, like periodontal probing (Braun et al. 2007) or an evaporative or a tactile stimulus on a tooth with hypersensitivity (Ide et al. 2001), the visual analogue scale is

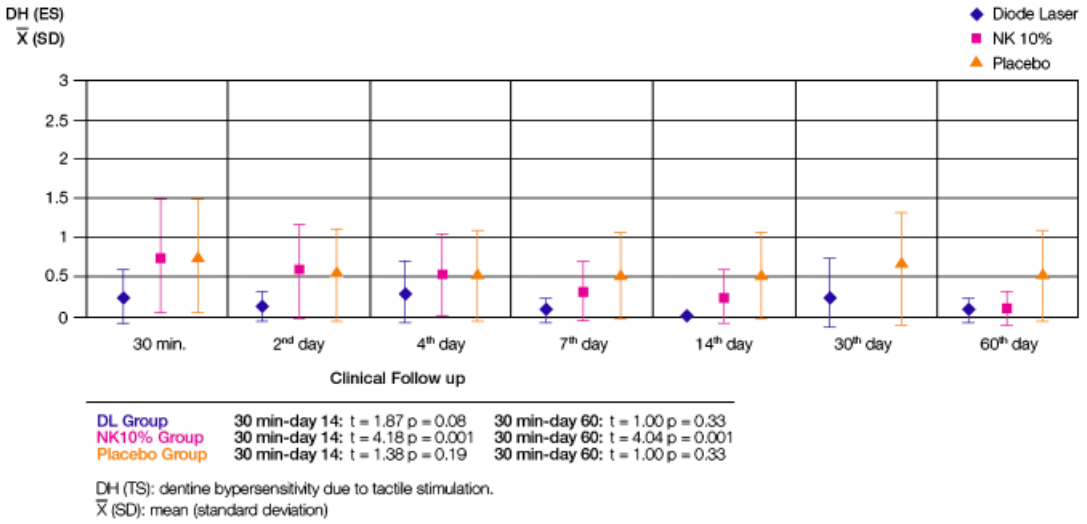

Fig. 5. Evolution of the response to tactile stimulation in the three treatment groups, from 30 min. to day 60 (end of the study).

Table 5. Reduction in dentine hypersensitivity response to tactile stimulation in the three treatment groups from 30 min. through to day 60 (end of the study)

Group	30 min. $\bar{x}$ (SD)	2nd day $\bar{x}$ (SD)	4th day $\bar{x}$ (SD)	7th day $\bar{x}$ (SD)	14th day $\bar{x}$ (SD)	RED 30–14 $\bar{x}$ (SD) (%)	30th day $\bar{x}$ (SD)	60th day $\bar{x}$ (SD)	RED 30–60 $\bar{x}$ (SD) (%)
DL	0.20	0.13	0.27	0.07	0.00	0.20 (0.41)	0.20	0.07	0.13 (0.52)
$n = 15$	(0.41)	(0.35)	(0.46)	(0.26)	(0.00)	100%	(0.56)	(0.26)	65%
NK10%	0.77	0.60	0.53	0.27	0.20	0.57 (0.62)	0.13	0.13	0.64 (0.70)
$n = 15$	(0.73)	(0.63)	(0.52)	(0.46)	(0.41)	74%	(0.35)	(0.35)	83%
Placebo (laser and gel)	0.77	0.53	0.53	0.47	0.47	0.30 (0.56)	0.60	0.53	0.24 (0.52)
$n = 15$	(0.73)	(0.64)	(0.64)	(0.64)	(0.64)	39%	(0.73)	(0.64)	31%
	$\chi^2 = 7.48$	$\chi^2 = 5.85$	$\chi^2 = 2.36$	$\chi^2 = 4.65$	$\chi^2 = 7.46$	$\chi^2 = 6.37$	$\chi^2 = 5.88$	$\chi^2 = 8.02$	$\chi^2 = 8.204$
	gl = 2	gl = 2	gl = 2	gl = 2	gl = 2	gl = 2	gl = 2	gl = 2	gl = 2
	$p = 0.02$	$p = 0.54$	$p = 0.31$	$p = 0.09$	$p = 0.02$	$p = 0.04$	$p = 0.05$	$p = 0.02$	$p = 0.017$

KW, Kruskall–Wallis test; df, degrees of freedom.
RED30–14: DH reduction from 30 min. to day 14.
RED30–60: DH reduction from 30 min. to day 60.

widely used; in this way we have applied it in previous studies (Sicilia et al. 1996, Frechoso et al. 2003) and in the present study.

In our study, the use of an 810 nm DL enabled us to demonstrate a significant and clinically important reduction compared with placebo after 15 (37% *versus* 9%) and 30 min. (41% *versus* 9%). These results are slightly inferior to those published by Ladalardo et al. (2004), although the latter data were obtained without evaluating the placebo effect, which can be of great importance. For example, in our study, the effect of the placebo laser was a significant DH reduction of 9% with ES after 15 min., and of 26% at 60 days (Table 4). This effect is backed up by previous studies evaluating the role of placebo in clinical DH tests, reaching DH reduction levels of 20–60% (West et al. 1997, Dababneh et al. 1999, Kimura et al. 2000, Yates et al. 2004).

At 48 and 96 h, we obtained a DH reduction of 46% and 54% with DL application (ES) (data calculated from Tables 1 and 3). These results are greater than those obtained with NK10% (21% and 33%) and placebo (3% and 12%) (Table 3). The reduction observed in our NK10% positive control group was lower than that obtained in the study by Frechoso et al. (2003), in which these values reached 36% and 66%, respectively. However, it is important to bear in mind that the base DH data after ES were superior in the study by Frechoso et al. (2003), and that patients who received NK10% treatment in our study had already shown a 9% improvement 30 min. after receiving the placebo laser, which reduced the DH even more before the application of the gel, leaving a smaller margin for further improvement. The same effect can be observed in all other comparative evaluations; however, a simple analysis of the data reveals a significantly greater reduction for the DL group after 14 days (71.7%) compared with the NK10% and placebo groups (Table 4), which is even greater than the reduction seen in the NK10% group after 14 days in the study by Frechoso et al. (2003) (63% NK10% *versus* 59% placebo). Emphasis must be placed on the important effect observed in the control group after 14 days in this study.

Equally as important as the rapid reduction of DH (immediate efficacy) is the long-term duration of the effects (Grossman 1935, Addy & Dowell 1983, Dababneh et al. 1999). A previous study showed a DH reduction of 40.8% after 8 weeks following a treatment with a mouthwash containing 1% potassium nitrate and sodium

Table 6. Reduction in dentine hypersensitivity response to tactile stimulation in the three treatment groups from the start of the study (baseline) through to day 60 (end of the study)

Group	Baseline $\bar{x}$ (SD)	14th day $\bar{x}$ (SD)	RED 0–14 $\bar{x}$ (SD) (%)	60th day $\bar{x}$ (SD)	RED 0–60 $\bar{x}$ (SD) (%)
DL	0.87	0.00	0.87 (1.06)	0.07	0.80 (1.15)
$n = 15$	(1.06)	(0.00)	100%	(0.26)	92%
NK10%	1.27	0.20	1.07 (0.88)	0.13	1.13 (0.83)
$n = 15$	(0.88)	(0.41)	84%	(0.35)	89%
Placebo laser and gel	0.80	0.47	0.33 (0.49)	0.53	0.27 (0.46)
$n = 15$	(0.77)	(0.64)	41%	(0.64)	34%
			$F = 2.99$		$\chi^2 = 7.70$
			$p = 0.09$		gl = 2
					$p = 0.02$

KW, Kruskall–Wallis test; df, degrees of freedom.
ANOVA, single-factor analysis of variance (ANOVA) test; *F,* Fisher–Snedecor *F*.
RED 0–14: DH reduction from day 0 (baseline) to day 14.
RED 0–60: DH reduction from day 0 (baseline) to day 60.

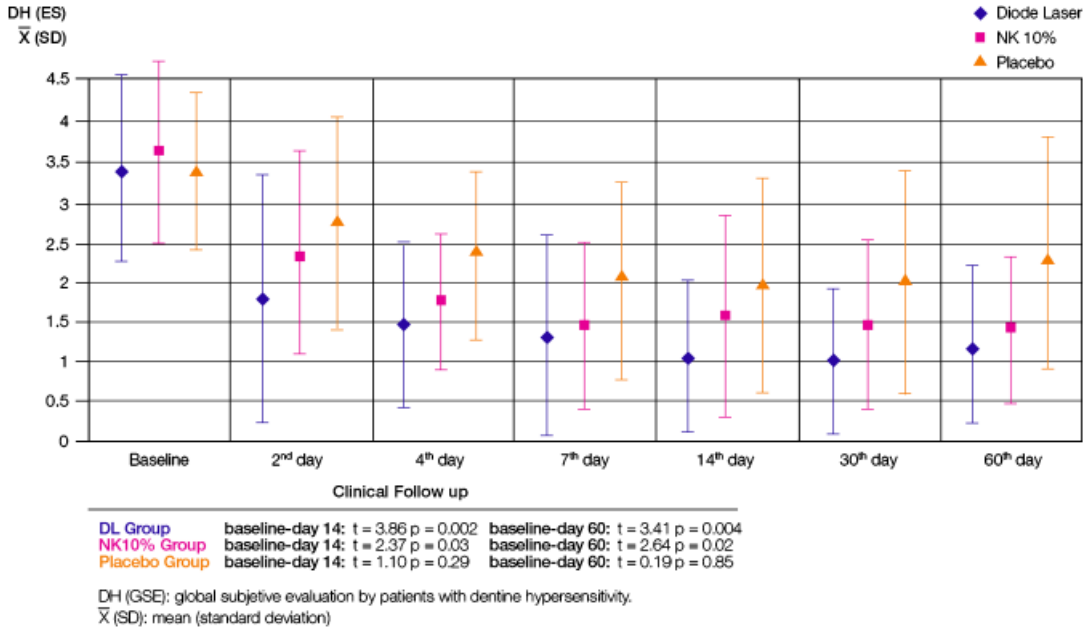

Fig. 6. Evolution of dentine hypersensitivity intensity as perceived subjectively by patient in the three treatment groups.

Table 7. Reduction in dentine hypersensitivity response perceived by the patient during daily life in the three treatment groups from the start of the study (baseline) through to day 60 (end of the study)

Group	Baseline $\bar{x}$ (SD)	2nd day $\bar{x}$ (SD)	RED 0–2 $\bar{x}$ (SD) (%)	14th day $\bar{x}$ (SD)	RED 0–14 $\bar{x}$ (SD) (%)	60th day $\bar{x}$ (SD)	RED 0–60 $\bar{x}$ (SD) (%)
DL	3.47	1.80	1.67 (1.80)	1.07	2.40 (1.40)	1.20	2.27 (1.44)
$n = 15$	(1.12)	(1.52)	48%	(0.96)	69%	(1.01)	65%
NK10%	3.67	2.40	1.27 (1.16)	1.53	2.13 (1.85)	1.43	2.13 (1.68)
$n = 15$	(1.17)	(1.24)	35%	(1.19)	58%	(0.94)	58%
Placebo (laser and gel)	3.40	2.73	0.67 (1.11)	2.00	1.40 (1.40)	2.33	1.07 (1.39)
$n = 15$	(0.99)	(1.33)	20%	(1.36)	41%	(1.50)	31%
			$F = 1.96$		$X^2 = 3.41$		$X^2 = 6.37$
			$p = 0.15$		gl = 2		g = 2
					$p = 0.18$		$p = 0.04$

KW, Kruskall–Wallis test; df, degrees of freedom.
ANOVA ,single-factor analysis of variance (ANOVA) test; *F*, Fisher–Snedecor *F*.
RED 0–2: DH reduction from day 0 (baseline) to day 2.
RED 0–14: DH reduction from day 0 (baseline) to day 14.
RED 0–60: DH reduction from day 0 (baseline) to day 60.

monofluorophosphate, where a 33.3% reduction was observed in the control group (Sicilia et al. 1996). Other authors have obtained equivalent results between 5 and 12 weeks (Echeverría et al. 1991, Nagata et al. 1994, West et al. 1997, Pereira & Chava 2001). In our study, results from treatment with DL remained stable until day 60, showing a 66% DH reduction after ES, compared with 30% with NK10% gel and 26% with placebo gel.

We can finally conclude that, in the current study, the application of DL has shown efficacy in rapid DH reduction compared with placebo laser in periodontal patients. This effect has become apparent at 15 and 30 min., at 2 weeks, and it remains stable until 2 months. Bearing in mind its rapid action and the stability of results, DL can be considered a useful tool for DH reduction in populations of a similar nature, as for instance, patients in periodontal practices.

An additional therapeutic option could be the combination of laser irradiation with the application of specific products for the treatment of DH, with the intention of achieving an accumulation of effects from both treatments. Combinations of different types of laser and fluorides have been evaluated, with promising results (Kumar & Mehta 2005, Goharkhay et al. 2007). In our study, and in previous works (Frechoso et al. 2003), the NK10% gel proved to be effective in the immediate treatment of DH, although its possible effect associated with the DL could not be evaluated. It would be recommendable to perform future studies combining both treatments.

Acknowledgements

The authors wish to thank Begoña Lozano, Patricia Roces, Charo Prieto and Catalina García for their collaboration in the collection of information and patient monitoring, Jordi Poblet, Gema García-Teresa and Javier Mira of LACER SA and Pedro Morales of Biolase Inc., for their selfless collaboration and María de la Fuente Latorre (www.visverbi.com) for the translation and review of the English manuscript.

References

ADA (1986) Recommendations for evaluating agents for the reduction of dentinal hypersensitivity. Ad Hoc Advisory Committee on Dentinal Hypersensitivity Council on Dental Therapeutics. *Journal of American Dental Association* **112**, 709–710.

Addy, M. (2002) Dentine hypersensitivity: new perspectives on an old problem. *International Dental Journal* **52**, 367–375.

Addy, M. & Dowell, P. (1983) Dentine hypersensitivity – a review. Clinical and in vitro evaluation of treatment agents. *Journal of Clinical Periodontology* **10**, 351–363.

Addy, M. & Urquhart, E. (1992) Dentine hypersensitivity: its prevalence, aetiology and clinical management. *Dental Update* **19**, 407–408, 410–402.

Arrastia, A. M., Machida, T., Smith, P. W. & Matsumoto, K. (1994) Comparative study of the thermal effects of four semiconductor lasers on the enamel and pulp chamber of a human tooth. *Lasers in Surgery and Medicine* **15**, 382–389.

Brannstrom, M. & Astrom, A. (1972) The hydrodynamics of the dentine; its possible relationship to dentinal pain. *International Dental Journal* **22**, 219–227.

Braun, A., Jepsen, S. & Krause, F. (2007) Subjective intensity of pain during ultrasonic supragingival calculus removal. *Journal of Clinical Periodontology* **34**, 668–672.

Clark, D. C., Al-Joburi, W. & Chan, E. C. (1987) The efficacy of a new dentifrice in treating dentin sensitivity: effects of sodium citrate and sodium fluoride as active ingredients. *Journal of Periodontal Research* **22**, 89–93.

Collins, J. F., Gingold, J., Stanley, H. & Simring, M. (1984) Reducing dentinal hypersensitivity with strontium chloride and potassium nitrate. *General Dentistry* **32**, 40–43.

Dababneh, R. H., Khouri, A. T. & Addy, M. (1999) Dentine hypersensitivity – an enigma? A review of terminology, mechanisms, aetiology and management. *British Dental Journal* **187**, 606–611; discussion 603.

Echeverría, J. J., Manau, C. & Sentís, J. (1991) Eficacia de un nuevo dentífrico para el tratamiento de la hiperestesia dentinaria: un estudio a doble ciego. *Archivos de Odonto-Estomatología* **7**, 385–390.

Frechoso, S. C., Menendez, M., Guisasola, C., Arregui, I., Tejerina, J. M. & Sicilia, A. (2003) Evaluation of the efficacy of two potassium nitrate bioadhesive gels (5% and 10%) in the treatment of dentine hypersensitivity. A randomised clinical trial. *Journal of Clinical Periodontology* **30**, 315–320.

Gelskey, S. C., White, J. M. & Pruthi, V. K. (1993) The effectiveness of the Nd:YAG laser in the treatment of dental hypersensitivity. *Journal of Canadian Dental Association* **59**, 377–378, 383–376.

Goharkhay, K., Wernisch, J., Schoop, U. & Moritz, A. (2007) Laser treatment of hypersensitive dentin: comparative ESEM investigations. *Journal of Oral Laser Applications* **7**, 211–223.

Green, B. L., Green, M. L. & McFall, W. T. Jr. (1977) Calcium hydroxide and potassium nitrate as desensitizing agents for hypersensitive root surfaces. *Journal of Periodontology* **48**, 667–672.

Grossman, L. I. (1935) A systematic method for the treatment of hypersensitive dentin. *Journal of American Dental Association* **22**, 592–602.

Hodosh, M. (1974) A superior desensitizer – potassium nitrate. *Journal of American Dental Association* **88**, 831–832.

Holland, G. R., Narhi, M. N., Addy, M., Gangarosa, L. & Orchardson, R. (1997) Guidelines for the design and conduct of clinical trials on dentine hypersensitivity. *Journal of Clinical Periodontology* **24**, 808–813.

Ide, M., Wilson, R. F. & Ashley, F. P. (2001) The reproducibility of methods of assessment for cervical dentine hypersensitivity. *Journal of Clinical Periodontology* **28**, 16–22.

Iida, M., Ando, Y., Watanabe, H. & Ishikawa, I. (1993) Effect of GaAlAs-semiconductor laser irradiation on dentin hypersensitivity of exposed root surface and influence to microflora in dento-gingival region. *Journal of Japanese Society for Laser Dentistry* **4**, 3–7.

Jacobsen, P. L. & Bruce, G. (2001) Clinical dentin hypersensitivity: understanding the causes and prescribing a treatment. *Journal of Contemporary Dental Practice* **2**, 1–12.

Kanapka, J. A. (1990) Over-the-counter dentifrices in the treatment of tooth hypersensitivity. Review of clinical studies. *Dental Clinics of North America* **34**, 545–560.

Karu, T. (1989) Photobiology of low-power laser effects. *Health Physics* **56**, 691–704.

Kim, S. (1986) Hypersensitive teeth: desensitization of pulpal sensory nerves. *Journal of Endodontics* **12**, 482–485.

Kimura, Y., Wilder-Smith, P., Yonaga, K. & Matsumoto, K. (2000) Treatment of dentine hypersensitivity by lasers: a review. *Journal of Clinical Periodontology* **27**, 715–721.

Kumar, N. G. & Mehta, D. S. (2005) Short-term assessment of the Nd:YAG laser with and without sodium fluoride varnish in the treatment of dentin hypersensitivity – a clinical and scanning electron microscopy study. *Journal of Periodontology* **76**, 1140–1147.

Ladalardo, T. C., Pinheiro, A., Campos, R. A., Brugnera Junior, A., Zanin, F., Albernaz, P. L. & Weckx, L. L. (2004) Laser therapy in the treatment of dentine hypersensitivity. *Brazilian Dental Journal* **15**, 144–150.

Lan, W. H. & Liu, H. C. (1996) Treatment of dentin hypersensitivity by Nd:YAG laser. *Journal of Clinical Laser Medicine and Surgery* **14**, 89–92.

Launay, Y., Mordon, S., Cornil, A., Brunetaud, J. M. & Moschetto, Y. (1987) Thermal effects of lasers on dental tissues. *Lasers in Surgery and Medicine* **7**, 473–477.

Marsilio, A. L., Rodrigues, J. R. & Borges, A. B. (2003) Effect of the clinical application of the GaAlAs laser in the treatment of dentine hypersensitivity. *Journal of Clinical Laser Medicine and Surgery* **21**, 291–296.

Matsumoto, K., Wakabayashi, H., Funato, A. & Shirasuka, T. (1985) Histopathological findings of dental pulp irradiated by GaAlAs laser diode. *Japanese Journal of Conservative Dentistry* **28**, 1361–1365.

Minkoff, S. & Axelrod, S. (1987) Efficacy of strontium chloride in dental hypersensitivity. *Journal of Periodontology* **58**, 470–474.

Nagata, T., Ishida, H., Shinohara, H., Nishikawa, S., Kasahara, S., Wakano, Y., Daigen, S. & Troullos, E. S. (1994) Clinical evaluation of a potassium nitrate dentifrice for the treatment of dentinal hypersensitivity. *Journal of Clinical Periodontology* **21**, 217–221.

Pereira, R. & Chava, V. K. (2001) Efficacy of a 3% potassium nitrate desensitizing mouthwash in the treatment of dentinal hypersensitivity. *Journal of Periodontology* **72**, 1720–1725.

Pillon, F. L., Romani, I. G. & Schmidt, E. R. (2004) Effect of a 3% potassium oxalate topical application on dentinal hypersensitivity after subgingival scaling and root planing. *Journal of Periodontology* **75**, 1461–1464.

Reinhart, T. C., Killoy, W. J., Love, J., Overman, P. R. & Sakumura, J. S. (1990) The effectiveness of a patient-applied tooth desensitizing gel. A pilot study. *Journal of Clinical Periodontology* **17**, 123–127.

Renton-Harper, P. & Midda, M. (1992) NdYAG laser treatment of dentinal hypersensitivity. *British Dental Journal* **172**, 13–16.

Schwarz, F., Aoki, A., Becker, J. & Sculean, A. (2008) Laser application in non-surgical periodontal therapy: a systematic review. *Journal of Clinical Periodontology* **35**, 29–44.

Seltzer, S., Bender, I. B. & Ziontz, M. (1963) The dynamics of pulp inflammation: correlations between diagnostic data and actual histologic findings in the pulp. *Oral Surgery Oral Medicine Oral Pathology* **16**, 846–871 , contd.

Sicilia, A., San Narciso, J., Buitrago, P., Enrile, F., Tejerina, J. M. & Clerch, L. (1996) Ensayo clínico sobre la eficacia de un colutorio de nitrato potásico al 1% y monoflúorfosfato sódico al 1,13% en el tratamiento de la hipersensibilidad dentinaria. *Periodoncia* **6**, 247–256.

Silverman, G. (1986) The effectiveness of two dentifrices in relieving dentinal hypersensitivity. *Journal of Dental Restorative* **65**, 297, abstract 1150.

Tarbet, W. J., Silverman, G., Stolman, J. M. & Fratarcangelo, P. A. (1979) An evaluation of two methods for the quantitation of dentinal hypersensitivity. *Journal of American Dental Association* **98**, 914–918.

Tarbet, W. J., Silverman, G., Stolman, J. M. & Fratarcangelo, P. A. (1980) Clinical evaluation of a new treatment for dentinal hypersensitivity. *Journal of Periodontology* **51**, 535–540.

Walters, P. A. (2005) Dentinal hypersensitivity: a review. *Journal of Contemporary Dental Practice* **6**, 107–117.

West, N. X., Addy, M., Jackson, R. J. & Ridge, D. B. (1997) Dentine hypersensitivity and the placebo response. A comparison of the effect of strontium acetate, potassium nitrate and fluoride toothpastes. *Journal of Clinical Periodontology* **24**, 209–215.

Yamaguchi, M., Ito, M., Miwata, T., Horiba, N., Matsumoto, T., Nakamura, H. & Fukaya, M. (1990) Clinical study on the treatment of hypersensitive dentin by GaAlAs laser diode using the double blind test. *Aichi Gakuin Daigaku Shigakkai Shi* **28**, 703–707.

Yates, R. J., Newcombe, R. G. & Addy, M. (2004) Dentine hypersensitivity: a randomised, double-blind placebo-controlled study of the efficacy of a fluoride-sensitive teeth mouthrinse. *Journal of Clinical Periodontology* **31**, 885–889.

Zanirato, R., Curti, F., Prezzotto, G., De Carvalho, E., Pelizon, J. E. & Bagnato, V. S. (2007) Clinical effects of low-intensity laser vs light-emitting diode therapy on dentin hypersensitivity. *Journal of Oral Laser Applications* **7**, 129–136.

Zhang, C., Matsumoto, K., Kimura, Y., Harashima, T., Takeda, F. H. & Zhou, H. (1998) Effects of CO_2 laser in treatment of cervical dentinal hypersensitivity. *Journal of Endodontics* **24**, 595–597.

Supporting Information

Additional Supporting Information may be found in the online version of this article:

Appendix S1. CONSORT Statement – 2001.

Please note: Wiley-Blackwell are not responsible for the content or functionality of any supporting materials supplied by the authors. Any queries (other than missing material) should be directed to the corresponding author for the article.

Address:
Alberto Sicilia
Clínica Sicilia
Uría 5
Oviedo. 33003
Spain
E-mail: asicilia@clinicasicilia.com

Clinical Relevance

Scientific rationale for study: The DL has proved to have the potential to reduce DH, although it has not been assessed in clinical trials.
Principal findings: DL application has shown an immediate and relevant reduction of DH in periodontal patients, ranging from 42% at 30 min. to 72% at 14 days. These results remain stable until day 60.
Practical implications: A single DL application is an effective and clinically relevant way to reduce DH in periodontal patients. The use of NK10% for 14 days has been shown to be effective, although to a lesser extent. Simultaneous application of NK 10% and DL has not been assessed.