

# Effect of Er, Cr: YSGG Laser Output Power on Enamel Caries Prevention: An in vitro study

Running Title: Effect of Er, Cr: YSGG Laser on caries

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## ABSTRACT

**Aims of the study:** The purpose of this study was to evaluate caries-preventive potential of Er, Cr: YSGG laser and to study the effect of power in order to determine the optimal output power for caries prevention.

**Materials and Methods:** The study samples were upper first premolars obtained from patients who required orthodontic extraction. Sample size was a forty-eight freshly extracted, caries-free upper first premolars of patients aged 15-20 years old. The crowns of teeth were covered with nail varnish leaving a square window (approximately 3mm×3mm) on the buccal surface at the middle third of the crown. Teeth were randomly assigned to four groups with 12 samples in each group, these groups were: group A (irradiated with laser power 0.25 watt for 10 seconds), group B (irradiated with laser power 0.50 watt for 10 seconds), group C (irradiated with laser power 0.75 watt for 10 seconds) and control group (no laser treatment). Er, Cr: YSGG laser with 2.78µm in wavelength and 20Hz repetition rate was used. Teeth were subjected to caries-like formation by immersion in lactic acid (pH=3.5) and incubation for twenty-one days at 37°C. Sections of 250µm in thickness were then obtained. Mean lesion depth was measured using polarized microscope.

**Results:** Post hoc (Duncan's test) confirmed that there was a high significant difference among lesion means of group A, B and C ( $P \leq 0.00$ ) where samples were irradiated at different powers and a constant time. Results indicated that treatment with Er, Cr: YSGG laser could cause caries inhibition up to 56%.

**Conclusions:** It is obvious that increasing the laser power (up to 0.75 watt) could result in an increase in caries inhibition and reduction in mean lesion depth.

**Keywords:** Enamel, Caries, Er, Cr: YSGG laser.

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## INTRODUCTION

Dental caries is a multifactorial disease and one of the most worldwide health problems affecting children and young adults pain causing and eventual tooth loss.<sup>(1)</sup> It is the result of interplay of three main factors over time which are: susceptible hard tooth surface, cariogenic bacteria within dental plaque and dietary carbohydrates. The most acceptable theory of dental caries is (acidogenic theory) which involves metabolism of fermentable carbohydrates and possible production of organic acids by acidogenic bacteria. Such acids can spread from dental plaque and infiltrate through enamel porous surface and dissociate to produce hydrogen ions.<sup>(2)</sup>

Primary prevention of such consequence is aimed toward intake restriction of cariogenic diet and increasing acid resistance of tooth surface through proper individual plaque control and hygiene measures which can counteract possible progression of caries and eventual tooth destruction.<sup>(3)</sup>

The first use of laser in maximizing enamel resistance to acid dissolution and reducing enamel solubility was demonstrated in the 1970s by using CO<sub>2</sub> laser irradiation.<sup>(4)</sup> Different wavelength lasers have been used for caries prevention such as Nd: YAG, Er: YAG, Er, Cr: YSGG, Ho: YAG, Argon and CO<sub>2</sub>.<sup>(5)</sup>

Er, Cr: YSGG laser that works at a wavelength of (2.78 $\mu$ m) has been used for cavity preparation by process called (ablation).<sup>(6)</sup>For caries prevention, it is important that laser does not ablate treated tissue surface but alters enamel chemically (i.e., irradiation at sub-ablative level).<sup>(7)</sup>

The purpose of this study was to evaluate caries-preventive potential of Er, Cr: YSGG laser and to study the effect of power in order to determine the optimal output power for caries prevention.

### **Materials and Methods**

**Samples Size and Criteria:** The study samples were obtained from patients who required orthodontic extraction of upper first premolars. Sample size was a forty-eight freshly extracted, caries-free upper first premolars of patients aged 15-20 years old.

**Samples Grouping:** The samples were randomly divided into three treatment groups and one control group with 12 samples in each group.<sup>(7)</sup> These groups were: Group A(samples irradiated with laser power 0.25watt for 10 seconds), Group B(samples irradiated with laser power 0.50watt for 10 seconds), Group C(samples irradiated with laser power 0.75watt for 10 seconds), Control group were samples receive no laser treatment. The distance was adjusted to (4mm) allowing the entire surface to be irradiated at the same distance.<sup>(8)</sup>

**Samples preparation:** Teeth were scaled and polished using fluoride-free pumice and rubber cup, stored in thymol solution(0.1%, pH=7).<sup>(9)</sup>In addition, teeth were examined under polarized light microscope with 10X magnification power to exclude those with cracks or any defects.<sup>(10)</sup>The crowns were separated from the roots at cemento-enamel junction in bucco-lingual direction using high speed hand piece with diamond disc under continuous water irrigation to prevent dehydration. A square adhesive tape of (3 $\times$ 3 mm<sup>2</sup>) was fixed on the middle third of the crowns. Then, the crowns were painted with acid-resistant varnish except for a (3 $\times$ 3mm<sup>2</sup>) square window.<sup>(11)</sup>The samples were dried for 24 hours and tapes were removed. The specimens were fixed on microscopic slides with the aid of adhesive (super glue) which was applied to the palatal surface of crowns.

**Surface Treatment:** The window of exposed enamel for each sample was irradiated with Er, Cr: YSGG laser device (Waterlase i-Plus, Biolase Technology Inc., San Clemente, CA, U.S.A).<sup>(11)</sup>This equipment emitting photons at a wavelength (2.78 $\mu$ m). The repetition rate was (20Hz) for all groups. Control group receive no treatment. The MZ10 tips of (6mm length) and (1mm spot diameter) were used for samples irradiation. Water and air flow were set to (40%, 60%) respectively. The slides were positioned on a movable stage of the microscope and a laser hand piece was coupled by specially fabricated acrylic tool. With the laser tip fixed, homogenous irradiation was made by moving the stage horizontally and vertically in scanning mode over the entire area of window within a pre-determined time for each group (10 seconds).

**Induction of Caries-like Lesion in Vitro:** After laser treatment, the samples were subjected to acid challenge. This was done by immersing the samples of each sub-group in lactic acid (pH=3.5) placed in plastic containers and incubated for 21 days at 37 °C.<sup>(12)</sup>

**Microscopical Evaluation of Demineralization:** At the end of the incubation period, the samples were washed with deionized water and embedded in epoxy-resin using specially fabricated molds, then the samples were cut in cross sections through the center of window using thin section cut-off saw. Each cut-sample was fixed on a microscopical slide from the cutting side using Canada balsam and hot plate. The non-cutting side of each sample was subjected to grinding by polishing grinding machine until the enamel slice with a thickness of 250 $\mu$ m was obtained and verified using digital caliper.<sup>(13)</sup>

Water was used as an imbibition solution for the enamel slices which were examined under polarized light microscope (Optika, B-600POL-1, Italy) with 10X magnification power. Lesion depths in ( $\mu$ m) were measured in three different points for each slide using eyepiece graticule supplied with polarized microscope. The lesion depths included the surface zone and the body of lesion.<sup>(14)</sup> The three measurements were averaged to identify the lesion depth of each sample, photomicrographs were captured using a special digital camera and images were digitalized on computer.

**Statistical Analysis:** The null hypothesis tested assumed that there was no difference in lesion depth means between the groups with different parameters. The lesion depth means in ( $\mu$ m) obtained were analyzed using IBM® statistical package for social science V.20 software (SPSS Inc., Chicago, Illinois, U.S.A.). The lesion depth was considered the dependent variable whereas the experimental parameters were considered the independent variables.

The data obtained throughout the course of study were analyzed using Excel; Microsoft office 2010, Windows 7 Ultimate. Descriptive statistics including mean, standard deviation, minimum and maximum were calculated for each

group. The parametric tests including single factor One-Way ANOVA and the post hoc Duncan's test designated at ( $\alpha$ -level=5%) were executed to locate the significant difference among groups. Caries-inhibition percentages of experimental samples were estimated using the following formula.<sup>(14)</sup>

$$\text{Caries-inhibition Percentage} = \frac{\mu_C}{\mu_C - \mu_S} \times 100$$

Where  $\mu_C$  = Total mean lesion depth of control samples,

$\mu_S$  = Total mean lesion depth of experimental samples.

### RESULTS

The descriptive statistics of treatment groups(A,B,C) and control group including mean, standard deviation and standard error were calculated (Table 1), then a comparison was held between one treatment group and control by using Levene's test for equality of variance (Table2). According to this test, it was clear that there was a high significance between each treatment group and control group.

In order to explain the effect of the study parameter (power variable) on the mean lesion depth of different treatment groups, One-Way ANOVA (F-test) and post hoc Duncan's test were used for comparison between these groups. Table (3) illustrates the effect of power on mean lesion depth by making a comparison among sample means of group (A, B, C) at different powers and specific exposure time (10 seconds).Duncan's test confirmed that there was a high significant difference among the lesion means of these groups.

According to the specific equation, caries inhibition percentage corresponding to each group was calculated (Table 4). The results show that increase in the mean depth of different groups corresponding to a decrease in caries inhibition percentage.

Figure (1) explains the effect of output power on caries inhibition percentage. Increasing the output power lead to an increasing in caries inhibition percentage for each set of constant time. Figures (2,3) illustrate the colored photomicrographs of a caries-like lesion of control and C samples under 10X magnification power.

### DISCUSSION

The Er, Cr: YSGG laser (2.78  $\mu\text{m}$ ) wavelength coincides with the maximum peak of the hydroxyl ion ( $\text{OH}^-$ ) present in the hydroxyl apatite structure.<sup>(15)</sup> In order for the Er, Cr: YSGG laser to be used for the preventive treatment of caries without ablating the enamel surface, sub-ablative parameters of irradiation have been suggested.<sup>(15,16)</sup>

Table (3) has shown a comparison between the sample means of group (A, B, C) at different powers and specific exposure time (10 second) to test the effect of power using post hoc Duncan's test where  $p \leq 1\%$ . The results confirmed that there was a highly significant difference between the lesion means of these groups where the maximum sample mean in A was 345 $\mu\text{m}$  and the minimum sample mean in C was 220 $\mu\text{m}$ . According to these results, the increase in laser power within a specific time results in a decrease of lesion depth and consequently an increase in caries inhibition percentage.

Irradiation with 0.75 watt power for 10 seconds (as in group C) has led to higher reduction in lesion depth (200 $\mu\text{m}$ ) and consequently, lesser mineral loss and higher caries resistance (56%). The lased samples in the three treatment groups show an increase in caries inhibition percentage and reduction in demineralization depth compatible with the increase of irradiation power, thus suggesting the direct relation between irradiation power and degree of caries inhibition. This could be attributed to the fact that the increase in the power output of laser beam could lead to an increase of total energy deposited per unit area of evident beam. Consequently, the chemical composition of the exposed enamel will be affected, especially when the absorbed energy is high enough to drive organics and carbonate out of the enamel crystals which will lead to the formation of a more acid resistant apatite crystals.<sup>(17,18)</sup>

Featherstone et al. (1998) showed that the temperature at which carbonate is driven from the carbonated apatite is in the range of 400-600°C. They also found a direct correlation between carbonate loss in laser treated dental enamel and corresponding reduction in the rate of acid dissolution. The loss of carbonate resulted in a mineral phase which more closely resembles hydroxyapatite and is therefore less soluble than normal enamel at any given pH. Increasing the

power up to 0.8 watt ( $9 \text{ J/cm}^2$ ) led to ablation. This interesting finding was agreed with Apel et al. (2002) who reported that ablation threshold ranges between  $9\text{-}11 \text{ J/cm}^2$ .

Crystallographic and compositional changes of enamel due to the photo-thermal effect in which laser irradiation of dental hard tissue modifies the calcium-to-phosphorus ratio, reduces carbonate-to-phosphate ratio and leads to the formation of more stable and less acid-soluble compounds, thus reducing the susceptibility to acid attack and caries. It also includes thermal decomposition of carbonate due to the photo-thermal effect of laser. This mechanism seems to be the most acceptable one to explain laser induced caries prevention.<sup>(19,20)</sup>

## CONCLUSIONS

It is obvious that increasing the laser power (up to 0.75watt) could result in an increase in caries inhibition and reduction in mean lesion depth suggesting the direct proportion between output power and caries inhibition percentage. It is also clear that mean lesion depth decrease with the increase of laser power suggesting the inverse relationship between power and mean lesion depth.

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**Table (1): Descriptive statistics of treatment groups and control group**

| Group   | Sample Size | Total Mean $\mu$ m | Standard deviation | Standard Error |
|---------|-------------|--------------------|--------------------|----------------|
| A       | 12          | 395                | 22.8               | 6.5            |
| B       | 12          | 358                | 18.2               | 5.26           |
| C       | 12          | 290                | 30.2               | 8.72           |
| Control | 12          | 500                | 0.85               | 0.246          |

**Table (2): Independent Samples T-test**

| Group   | Sample Size | F                    | Sig. | Confidence interval |
|---------|-------------|----------------------|------|---------------------|
| A       | 12          | 53.106<br><br>39.612 | 0.00 | P $\leq$ 0.05       |
| Control | 12          |                      |      |                     |
| B       | 12          |                      |      |                     |
| Control | 12          |                      |      |                     |

**Table (3): A comparison among sample means of group A, B, C at different output powers and specific exposure time (10 seconds).**

| Group  | Sample size | Sample mean ( $\mu$ m) | Duncan's Test |
|--|-------------|------------------------|---------------|
| A  | 4           | 345                    | A             |
| B  | 4           | 305                    | B             |
| C  | 4           | 220                    | C             |
| F-test = 156.397, Significance = 0.00, P $\leq$ 0.01 |             |                        |               |

**Table (4): Corresponding mean lesion depth and caries inhibition percentage in each group**

| Group   | Mean depth ( $\mu$ m) | Caries inhibition percentage |
|---------|-----------------------|------------------------------|
| A       | 345                   | 31 %                         |
| B       | 305                   | 39 %                         |
| C       | 220                   | 56 %                         |
| Control | 500                   | 00                           |

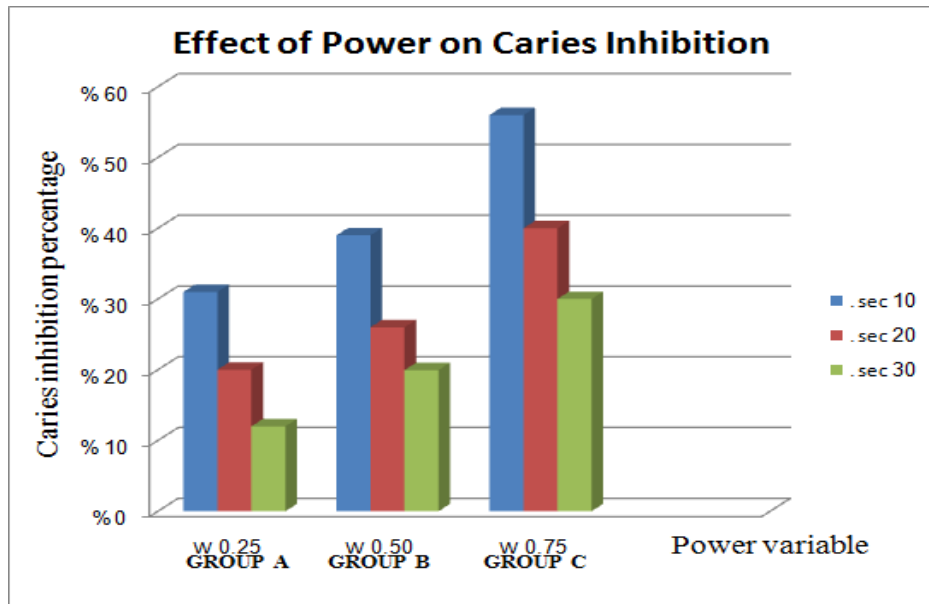


Figure (1): The relationship between power variable and caries inhibition percentage.

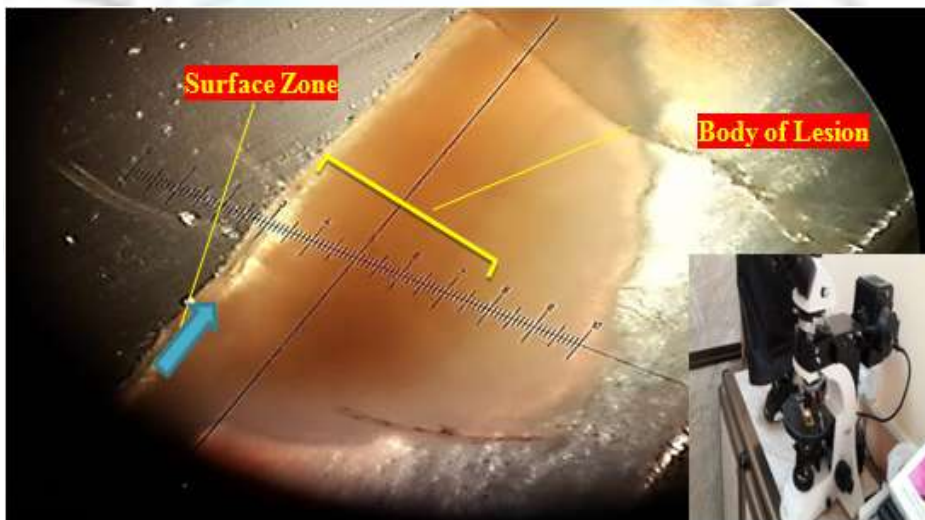
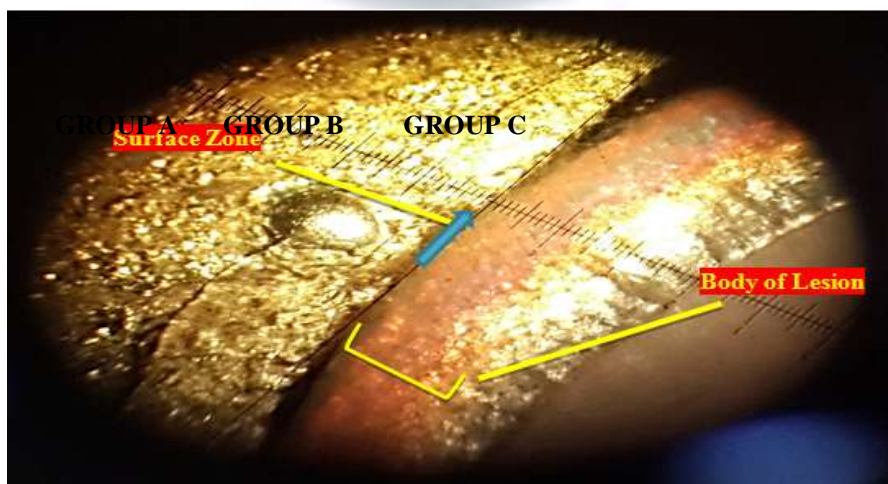


Figure (2): A colored photomicrograph of a caries-like lesion of control sample under 10X magnification power.



**Figure (3): A colored photomicrograph of a caries-like lesion of C sample under 10X magnification power.**

