Effect of Relief Amorphous Calcium Phosphate Versus Routine Fluoride Post Bleaching Agents on Enamel Surface Structure (Qualitative and Quantitative Analysis)

Thesis

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DEDICATION

I wish to dedicate this thesis to my parents, my husband and to all my family with a special thanks to all who helped me to bring this effort to light.

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INTRODUCTION AND REVIEW OF LITERTURE

Turkon, 2002 found that bleaching vital teeth has become one of the most popular esthetic dental services offered to patients and has been shown to be an efficient and safe technique with predictable results.

Whitening teeth for esthetic purposes has been dated back to the "Ancient Egyptians", where a mixture of ground pumice and wine vinegar was brushed on the teeth with a rudimentary toothbrush. The "Ancient Romans" used human urine by the belief that it kept the teeth white and firmly in place, a practice that continued on to the eighteenth century. In Biblical times it was considered “that the person who whitens the teeth of his neighbor is better than the person who gives him milk to drink.”Whitening in the middle ages was done by barbers, where the teeth were filed down and nitric acid applied to the teeth. However, this was a dangerous procedure, considering the massive tooth damage this practice caused **(Greenwall, 2001).**

The first known article on bleaching was published by **Haywood and Heymann** in **1989.** Since then, manufacturers have rapidly introduced numerous bleaching products.

Methods to improve the esthetics of the dentition by tooth whitening are of interest to dentists, their patients and the public. In the past 20 years, research on bleaching and other methods of removing tooth discolorations has dramatically increased. Now, products are available to solve a variety of tooth discoloration problems without restorative intervention.The indications for appropriate use of tooth-whitening methods and products are dependent on correct diagnosis of the discoloration reasons (**Sarrett, 2002).**

Watts and Addy, 2001 investigated the causes of discoloration. They found that exposure to high levels of fluoride, tetracycline administration**,** inherited developmental disorders and trauma to the developing tooth might result in pre-eruptive discoloration. After eruption of the tooth, discoloration might be caused either from extrinsic or intrinsic factors. Coffee, tea, red wine, carrots, oranges and tobacco, pigment generating bacteria and discoloration of restorative materials (mainly composite resins) gave rise to extrinsic stain. On the other hand, intrinsic discoloration may be due to: wear of the tooth structure, deposition of secondary dentin due to aging process or as a consequence of pulp inflammation and dentin sclerosis affected the light-transmitting properties of teeth, resulting in gradual darkening of the teeth.

Moreover incorporation of chromatogenic material into dentin and enamel either during tooth development or after eruption represented intrinsic tooth discoloration (**Christensen, 1997**).

When patients have their teeth bleached, they often become interested in veneers, replacing old restorations, diastema closures, or other esthetic procedures. In addition, bleaching may be indicated before placing an esthetic composite resin restoration to obtain a more pleasing final shade for the patient **(Swift, 1997**).

Tooth color can be improved by a number of methods and approaches including whitening toothpastes, professional cleaning by scaling and polishing, internal bleaching of non-vital teeth, external bleaching of vital teeth, microabrasion of enamel and placement of crowns and veneers. Whitening or tooth bleaching procedures in toothpastes attempt to lighten a tooth's color either chemically or mechanically. They have mild abrasives which aid in the removal of stains on enamel. Although this can be an effective method, it does not alter the intrinsic color of teeth. On other hand, micro abrasion techniques employ both methods where an acid is used first to weaken the outer 22–27 µm of enamel for subsequent abrasive force. This allows removal of the superficial stains in enamel, but if the discoloration is deeper or in dentin, this method of tooth whitening will not be successful. Chemically, a bleaching agent is used to carry out an oxidation reaction in enamel and dentin (**Dahl and Pallesen, 2003).**

Gerlach et al., 2000 found that there were currently numerous different whitening systems available, ranging from those based on carbamide peroxide to those with hydrogen peroxide. Though the product names and marketing were slightly different, the underlying mechanism of action was essentially the same.

Hydrogen peroxide acts as a strong oxidizing agent through the formation of free radicals. The reactive oxygen molecules and hydrogen peroxide anions attack the long-chained, dark-colored chromophore molecules and split them into smaller, less colored and more diffusible molecules (**Gregus and Klaassen, 1996).**

Hydrogen peroxide may be applied directly, or produced in a chemical reaction from sodium perborate or carbamide peroxide. However, carbamide peroxide is an extremely unstable solution; when it comes in contact with saliva it breaks down into 70% urea and 30% hydrogen peroxide. The latter being considered to be the active agent of the bleaching reaction. Theoretically urea can be further decomposed to carbon dioxide and ammonia. The high pH of ammonia facilitates the bleaching procedure **(Sun, 2000).**

To accelerate the bleaching process, the bleaching agent can be additionally heat-activated. Application of heat, light or lasers is used to increase the temperature of a bleaching agent applied to the tooth surface.

If light is projected onto a bleaching product, such as bleaching gel, a small fraction is absorbed and its energy is converted into heat. Most likely, this is the main mechanism of action of all light-activated bleaching procedures **(Luk et al., 2004).**

It was in the year 1916 that Dr. Walter Kane used hydrochloric acid to remove the fluorosis stains. In 1937, Ames reported an alternative for removing fluorosis using hydrogen peroxide instead of hydrochloric acid. Later, McInnis reported a technique where hydrogen peroxide, hydrochloric acid and ethyl ether were used. This technique has been found to be successful for bleaching the teeth of patients with endemic fluorosis (**Darshan and Shashikiran, 2008**).

Four different approaches for tooth whitening have been recognized and reviewed by **Barghi** in **1998**. These were **Dentist-administered bleaching** using high concentration of hydrogen peroxide (35-50%) or carbamide peroxide (35-40%), often supplemented with a heat source. **Dentist-supervised bleaching** by means of a bleaching tray loaded with high concentrations of carbamide peroxide (35- 40%) placed in the patient's mouth for 30 min- 2 hrs while the patient was in the dental office. **Dentist-provided bleaching** known as "at-home" or "night-guard" bleaching and administered by the patient through application of 5-22% solution of carbamide peroxide in a custom-made tray. Finally, **Over-the-counter products**: based on carbamide peroxide or hydrogen peroxide of various concentrations placed in a pre-fabricated tray, or by the recently introduced strips both to be adjusted by the user.

Leonard et al., 1998 found that the increased frequency of exposure to highly concentrated bleaching agents might enhance side effects, such as mucosal irritation, alteration of enamel surface and tooth sensitivity.

Morphological alteration of enamel following tooth bleaching including: pitting, erosion, porosity, reduced fracture strength, dissolution, and effects similar to those of initial caries has been addressed in several studies. It was also found that the demineralization altered enamel micro hardness. Thus, a reduction in surface micro hardness of enamel might be directly associated with a mineral loss under conditions of mild demineralization. This reduction has been related to the presence of a urea-releasing reaction which kept the pH close to the critical level for enamel demineralization that is 5.2-5.8**(Shannon,1993).**

The effect of bleaching treatment on the abrasion resistance of human enamel was shown to be significant by **Seghi and Denry 1992**, who found that bleached enamel showed a greater loss of tooth structure when abraded against both a harshly and mildly abrasive substrate than did in the unbleached enamel. They also detected decrease in the resistance of crack propagation related to the organic matrix structural integrity and its interfacial relationship to the crystalline phase in enamel.

Chemical processes caused by uncontrolled reaction of the peroxide radical might degrade the enamel structural component and decrease its ability to resist crack propagation. This is achieved by the small sized molecules which diffused into the interprismatic spaces of this semi-permeable tissue causing changes in its mechanical properties **(Seghi and Denry 1992).**

Furthermore**,** a clinical implication to these morphological alteration of the enamel were suggested by **Bitter**, **1998** who stated that "The teeth were more susceptible to extrinsic discoloration after bleaching due to increased surface roughness".

By infra-red spectroscopic analysis, **Oltu and Gürgan in 2000** were able to find that in vitro treatment of extracted teeth with 35% carbamide peroxide for 30 min/day for 4 days lead to changes in the inorganic composition of the enamel causing a reduction in the calcium and phosphorus content, whereas 10% and 16% concentrations did not.

Areas of superficial erosion, characterized by an evident “peeling” of the enamel surface with increased superficial porosity characterized by a greater quantity of Tomes’ process pits were observed on the enamel fragments treated by 35% hydrogen peroxide. **Spalding et al., 2003** stated that "This morphologic aspect seemed to be a result of the partial removal of the superficial layer of enamel, which is related to the removal of organic precipitates, organic matrix of enamel and surface mineral from hypomineralized areas of the enamel ".

Hosoya et al., 2003 found that loss of minerals induced by bleaching agents seemed to be an increase in the space between enamel prisms, an increase in surface roughness of the enamel and in the increased tendency to adhesion of streptococcus mutans. This reaction was similar to that of initial white stain caires lesion.

It is hypothesized that the peroxide-containing bleaching agents affect the organic phase of enamel. Peroxides can affect not only the surface but also the inner structure of enamel as a result of its low molecular weight. Thus, inner oxidative effects are more likely to occur in the subsurface enamel where more organic material is present and oxidation is capable of altering the outer and inner enamel surface **(Kobakhidze et al., 2006**).

However, **Meireles et al.**, **2008** stated that "Changes caused by peroxide bleaching agents to enamel including altered surface morphology, decreased microhardness and sensitivity, during and following bleaching treatment were reversible side effects".

Tooth sensitivity was the main side effect of bleaching, and might be caused by the penetration of hydrogen peroxide and urea through the intact enamel and dentin into the pulp in a matter of minutes. Sensitivity takes the form of a reversible pulpitis caused from the dentin fluid flow and pulpal contact of the material, which changed osmolarity without apparent harm to the pulp. Sensitivity depends on inherent patient sensitivity, frequency of application and peroxide concentration **(Haywood, 2002).**

In **2002**, **Pohjola et al.,** evaluated the different commercially available bleaching systems and found that all of them produced similar change in shade but none of them was sensitivity free. It has been speculated that in all forms of bleaching (in-office, with or without light activation) some of their bleaching ingredients enter the dentinal tubules and cause reversible pulpitis, which in turn might result in an increase of thermal sensitivity in the teeth.

Consequently, **Jorgensen and Carroll, 2002** found that patients with a previous history of tooth sensitivity might thus have a higher risk for such an adverse effect from external tooth bleaching.

Haywood, 2005 has used several approaches to reduce incidence of bleaching sensitivity; like reduction in tray wearing time or frequency of application and temporary interruption of whitening. Initial prophylaxis by pre-brushing with desensitizing tooth paste having potassium nitrate as an active ingredient could reduce bleaching sensitivity.

Rosenstiel et.al., 1991 discovered that most bleaching agents which have been developed for combined use with light sources included the addition of an activator or colorant to improve light absorption or to reduce tooth heating.

It is possible that there is an optimum amount of time for light exposure that will enhance the whitening effect without excessively heating the tooth. Theoretically, the pulpal temperature increase associated with light application could be lessened by: reducing the duration of light irradiation, increasing the thickness of the applied whitening agent and increasing the absorption of light by the bleach, thereby decrease the transmission of the light energy through the tooth **(Karen et al., 2004**).

Armênio et al., 2008 have used fluoride as an active agent to reduce sensitivity, but unfortunately they applied fluoride only after the patient had experienced tooth sensitivity. They stated that "although the use of sodium fluoride gel does not treat the experience of tooth sensitivity, yet its use after each bleaching regimen reduces the overall intensity of tooth sensitivity".

Attin et al., 1995 assumed that some of the fluorides support remineralization of enamel by formation of a calcium fluoride-like layer. This deposit was later dissolved, allowing fluoride to diffuse into the underlying enamel, the saliva or the plaque layer covering the tooth.

In **2005, Tanizawa** agreed that the fluoridated bleaching agent presents the potential to alter the demineralization–remineralization cycles. The addition of sodium fluoride (NaF) to the bleaching agents demonstrated accelerated remineralization by precipitating mineral crystals containing fluoride within the demineralized layer.

Addition of fluoride to a 10% carbamide peroxide whitening gel does impart some potential for remineralization of demineralized enamel. Once the proteins that adhere to the enamel crystallites are removed by the bleaching agent, the reactive fluoride ion could interact with the freshly cleaned crystallites, thereby enhancing the potential for remineralization. As therapeutic quantities of fluoride can influence remineralization properties of commercially available tooth whitening gels without altering whitening properties, it is also possible that the addition of fluoride to a whitening gel that enhances mineral deposition could help to reduce the tendency for teeth to re-stain and darken over time. Increased mineral deposition would reduce the inter crystallite spaces in enamel, thereby reducing the potential for the reintroduction of proteins that change the enamel optical properties (**Gladwell, 2006).**

Gaffar ,1998 studied the beneficial effect of fluoride regarding the precipitation of calcium fluoride crystals (approximately 0.05 micrometers) in dentin, thus reducing the functional radius of the dentinal tubules. Theoretically, this precipitation can reduce the penetration of hydrogen peroxide into the pulp by reducing the permeability of dentinal tubules, without affecting the oxidizing potential of the active bleaching agent.

However**, Gladwell, 2006** studied the remineralization potential of a fluoridated carbamide peroxide whitening gel and found that high concentration of fluoride added to the whitening gel could potentially be enough to cause fluorosis of the dentition if used for a long period of time during tooth development.

The use of added fluorides apparently also was not the answer. The problem with this approach was that the most effective anti hypersensitivity fluoride formulations used 0.4 ٪ stannous fluoride. This approach frequently caused products to be somewhat unstable and slow-acting and can also result in significant tooth discoloration. Hence, this ingredient was not favored for bleaching gels. Consequently manufacturers offered sodium fluoride formulations, but these were weak and slow-acting alternatives (**Jacobsen and Bruce, 2001**).

In an attempt to limit the discomfort felt during the tooth bleaching process, manufacturers added 5٪ potassium nitrate (KNO_3) to their "sensitive-formula" gels. This is because addition of KNO_3 is the most typical way of creating anti hypersensitivity toothpaste. However, this strategy fails to take into consideration that prolonged and sustained use of KNO_3 is needed to cause a noticeable regression of pain (**West et al., 1997**).

Haywood et al. ,2001 reported that using potassium nitrate-fluoride gel applied in the bleaching tray in patients with tooth sensitivity reduced the sensitivity in the majority of the patients and produced some degree of pain relief by reducing nerve excitation. This was done by bathing the nerve cells with high concentrations of potassium, the threshold potential increased so that an impulse won't occur no matter how strong the stimulus was. With no impulse elicited, no pain is felt.

Some studies showed that KNO_3 can obliterate tubules, and hence obliterating any stimulus from the dentin surface. However, a meta-analysis has shown that KNO_3 is better used with an osmotic agent, such as dimethylisosorbide (DMI), since the agent enhances the distribution of the KNO_3 into the pulp. The microtubules have constrictions and tight junctions that may block or further slow down the transport of KNO_3 alone. When DMI was added into the solution, there was significant improvement in the delivery, resulting in more rapid pain relief from tooth hypersensitivity. The resultant comfort was profound and lasting. Toothpaste containing 5% potassium nitrate, demonstrated a pronounced anesthetizing effect, it can be used successfully for treatment of dentinal hypersensitivity of teeth with clinically intact enamel, erosions and exposed root surfaces due to gingival recessions (**Frechoso et al., 2003, Tzanova et al., 2005 and Bartold, 2006).**