

EFFICACY OF ENAMEL SEALANTS IN PREVENTION OF DEMINERALISATION



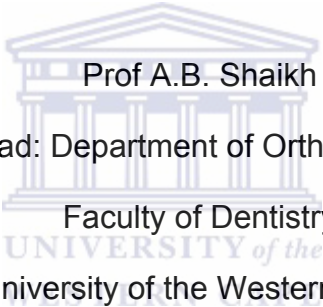
2008

**EFFICACY OF ENAMEL SEALANTS IN PREVENTING
DEMINERALISATION**

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A thesis submitted in partial fulfillment of the requirements for the degree of Magister Chirurgiae Dentium in the discipline of Orthodontics in the Faculty of Dentistry, University of the Western Cape.

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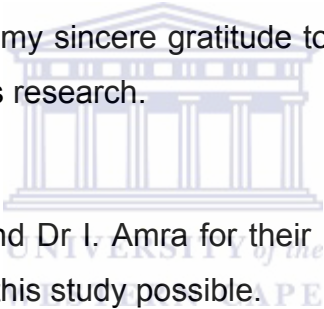
I Paul Mandla Nkosi declare that this thesis titled;

“Efficacy of enamel sealants in preventing demineralisation” is my own work and that all sources quoted have been indicated and acknowledged by means of references.

Signed:

_____ **ACKNOWLEDGEMENTS**

I wish to acknowledge my sincere gratitude to the following people for the assistance to me in this research.

- 
- A. Prof A.B. Shaikh and Dr I. Amra for their invaluable guidance and assistance in making this study possible.
- B. Prof SR Grobler, Drs R.J. Rossouw and N.J. Basson for their advice and assistance with laboratory and photographic work.
- C. Dr MT Ferguson for his advice, encouragements, motivations and for his support over the last 4 years and his assistance with setting out the statistics and help with the computer.
- D. Drs Theunis J W Kotze and Geetesh Solanki for their advice and assistance with statistics.

E. Mr. L Rayner for his help with mounting of the specimens.

Last but not least to Mr. Duncan Robinson from Orthotek (American Orthodontics) who donated the Fluor-Sure used in the study.DEDICATION

- A. To my wife, Masontaha, my best friend and love of my life. You have worked so hard to get us through my mid-life crisis. You have handled the stress and strain of juggling so many jobs. Without you, I could never achieve success. I promise that now this is over, it's your time to relax. I love you.
- B. To my daughters, Thandeka and Ntokozo, who made me, get up in the morning. I look forward to watching both of you develop into beautiful ladies. Thank you for helping mom at home and supporting me for the past four years. I am very proud of all your accomplishments.
- C. To my son, Simphiwe, we had very little contacts in the past four years but I promise it is pay back time from me now.
- D. To my parents, Mom and Dad, your support continues to keep our family strong. Without you both, this would not mean a whole lot.

To my brothers and sisters Mbhekeni, Mfanimpela, Kholekile, Ntombana and

Agnes, you have supported me and my family and that means the world to me. ABSTRACT

Enamel decalcification remains, in the absence of proper oral hygiene a common negative sequelae of orthodontic treatment (Todd *et al*, 1999). A new product, FluorSure (Orthotek, American Orthodontics) is claimed to protect the enamel against demineralisation during orthodontic treatment with fixed appliances. This sealant is a lightly filled (silica/glass mixture) light cured resin containing 31 percent leechable sodium fluoride (NAF). The manufacturers claim that it ensures abrasion resistance, provides a protective barrier between the enamel and brackets, and allows for a longer period of fluoride release.

Aims of the study: To compare the efficacy of two fluoride containing materials, namely, FluorSure and Duraphat, in protecting the enamel around and underneath the orthodontic brackets against decalcification.

Materials and Methods: Sixty freshly extracted human premolars with intact buccal enamel were selected for the study. The teeth were cleansed and polished with pumice slurry and prophylactic rubber cups. Roots of the teeth were sectioned below the cemento-enamel junction using a diamond disc and the crowns were embedded in self-curing acrylic resin in PVC pipes (Amra *et al*, 2007). The specimens were then allowed to stand until complete polymerization of the resin had occurred. The protruding enamel surfaces of the teeth were ground with an 800 and 1000 grade carborundum papers under running water to a smooth, flattened area. The teeth were then divided into 3 groups of 20 teeth each. In the control group the teeth were not sealed while in experiment groups the teeth were either sealed with FluorSure or with Duraphat. The baseline enamel microhardness tests were carried out with a Zwick/Roell (Indentec, hardness tester, Germany) at a load of 300g applied for 15 seconds. After baseline microhardness, lingual attachments were bonded. In FluorSure group, Fluor-

Sure was applied on the buccal surface before bonding while in Duraphat group, Duraphat was applied after bonding. Demineralisation/remineralisation solutions were prepared by adapting the method used by Hu and Featherstone (2005). The specimens were immersed in 300ml of demineralisation solution for six hours. The specimens were then removed from the demineralisation solution, rinsed with distilled water for few seconds then were brushed daily for 5 seconds with an Oral-B® soft bristled two heads electric toothbrush (CrossAction® PowerMax, B1011, Whitening, Oral B Laboratories, Germany) with no dentifrice. After the brushing the specimens were immersed in 300ml of remineralisation solution at 37°C overnight. After 14 days of this demineralisation/ remineralisation cycle, debonding was carried out using a double bladed debonding pliers and any remnant cement was carefully removed with a scalpel blade. The enamel microhardness tests were repeated, as close as possible to the previous indentations.

Results: Visible white spot formations were seen after the first day of demineralisation in the control group. At the last day, in the control group almost all the specimens were chalky white. The experimental groups showed varied levels of white spot formation as well, the mean decalcification in FluorSure group was 67.66 percent and ranged from 23.1 percent and to 100 percent. The mean decalcification in the Duraphat group was 63.95 and ranged from 43 percent to 98 percent at the end of the brushing process. Enamel microhardness also showed that enamel was much softer in the control group. There was no statistical difference between the FluorSure and Duraphat groups.

Conclusions: There was a significant difference between the FluorSure treated teeth and teeth in the control group with respect to demineralisation but FluorSure

**was not any better than Duraphat
thus either could be used to pre-
vent enamel
demineralisation. TABLE OF
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INTRODUCTION



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Orthodontists are still challenged by an "old problem" in their clinics, that of enamel demineralisation around orthodontic appliances (Gontijo *et al*, 2007). Although orthodontists have long recognised this problem, and most take active steps to minimise it, demineralisation continues to be a challenge (Todd *et al*, 1999). Enamel demineralisation is an undesirable side effect of orthodontic treatment with fixed appliances (Hu and Featherstone, 2005) and the demineralisation of enamel adjacent to orthodontic brackets is a significant clinical problem (Sudjalim *et al*, 2006).

Enamel demineralisation and white lesions occur during and sometimes remain after orthodontic treatment (Ärtun and Brobakken, 1986). Gorelick *et al* (1982) reported a significant increase in the incidence of white spot lesions following the placement of orthodontic appliances when compared with a control group of untreated individuals. O'Reilly and Featherstone (1987) and Øgaard *et al* (1988) have shown that visible white lesions can develop within 4 weeks of fitting a fixed bonded orthodontic appliance.



According to Geiger *et al* (1988), there has been general agreement that the development of white spots seems to be related to (1) the retention of plaque on the gingival side of brackets or bands, (2) lack of oral hygiene efficiency and (3) the inherent resistance of the individual. White spot lesions develop as a result of prolonged plaque accumulation on the affected surface, commonly due to inadequate oral hygiene (Sudjalim *et al*, 2006). It has been reported by Gwinnett and Ceen (1979) that plaque is accumulated in association with resin-bonded orthodontic brackets and some of the resins used to bond them.

Clearly, the best approach during orthodontic treatment is the prevention of the formation of white spot lesions (Willmot, 2004). Fluoride is known to inhibit lesion development during fixed appliance treatment and to enhance remineralisation following treatment (Stratemann and Shannon,

1974; O'Reilly and Featherstone, 1987; Øgaard *et al*, 1988; Geiger *et al*, 1992; Millett *et al*, 1999; Willmot, 2004; Hu and Featherstone, 2005)). It has been shown that the daily use of a fluoride rinse combined with oral hygiene instruction can lead to a significant reduction in decalcification during orthodontic treatment (Gorelick *et al*, 1982; Millett *et al*, 1999). Duckworth *et al* (1987) found that following the use of a sodium fluoride mouthrinse over a two week period, with one rinse per day, fluoride concentration in the saliva increased significantly.

Although topical fluorides have been shown to be effective, their main disadvantage is that they require patient compliance. Unfortunately, patient co-operation with home-use of topical fluoride agents and the maintenance of optimal oral hygiene levels is frequently inadequate (Stratemann and Shannon, 1974; Shannon, 1981; Geiger *et al*, 1988; Geiger *et al*, 1992). Geiger *et al* (1988) found that 50 percent of their patients were not compliant in maintaining optimal oral hygiene levels. They also reported on a clear association in increased white spot incidence with a decreasing fluoride dose and decreasing oral hygiene compliance.

A method of protecting the susceptible area adjacent to bonded attachments, independent of patient compliance, would be extremely beneficial (Hu and Featherstone, 2005). These strategies include enamel sealants (Ceen and Gwinnett, 1981; Joseph *et al*, 1992; Joseph *et al*, 1994; Banks and Richmond, 1994; Hicks *et al* 2000), fluoride containing or releasing banding and bonding agents (Marcusson *et al*, 1997; Schmit *et al*, 2002) and fluoride varnish application (Koch and Petersson, 1975; De Bruyn and Arends, 1987; Helfenstein and Steiner, 1994; Todd *et al*, 1999; Gillgrass *et al*, 2001; Øgaard *et al*, 2001; Demito *et al*, 2004; Sudjalim *et al*, 2006).

Previous studies have shown that the use of sealants prior to bonding brackets provides caries protection and also increases resin bond strength

(Banks and Richmond, 1994; Ceen and Gwinnett, 1981; Hicks *et al* 2000). The application of fluoride varnishes has also been shown to decrease enamel demineralisation (Koch and Petersson, 1975; De Bruyn and Arends, 1987; Helfenstein and Steiner, 1994; Todd *et al*, 1999). It has also been reported that fluoride varnishes have the benefit of adhering to the enamel surface longer than other topical fluoride products (Arends *et al*, 1980).

Duraphat (Colgate-Palmolive, New York) has been available in the market for more than 30 years and has been thoroughly studied. It presents high fluoride concentrations (Gontijo *et al*, 2007). According to Beltrán-Aguilar *et al* (2000), Duraphat contains five percent sodium fluoride. It remains adhered to enamel for a significant period of time and its use does not require patient cooperation (Gontijo *et al*, 2007).

The manufacturers of a new product, FluorSure, claim that this sealant protects the enamel against demineralisation during orthodontic treatment with fixed appliances (American Orthodontics information pamphlet). This sealant is a lightly filled (silica/glass mixture) light cured resin containing thirty one percent leechable sodium fluoride (NAF). The manufacturers claim that it ensures abrasion resistance, provides a protective barrier between the enamel and brackets, and allows for a longer period of fluoride release.

The purpose of this study was to investigate the efficacy of two methods of fluoride application in non-compliant patients in protecting the enamel around and underneath orthodontic brackets against decalcification and to determine the abrasion resistance of FluorSure and Duraphat. However quantifying FluorSure proved to be difficult during the pilot study as it was not possible to visualize FluorSure due to its clear colour. A dye could not be added as it might affect its properties. However white spot lesion for-

mations were seen and the study was therefore modified to include the observation and quantification of these formations.



LITERATURE REVIEW



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INTRODUCTION

Enamel demineralisation is an inconsistent but nevertheless undesirable side effect of orthodontic treatment with fixed appliances (Hu and Featherstone, 2005). It can be attributed in part to increased plaque accumulation around fixed orthodontic appliances, due to difficulty of plaque removal, as well as a significant increase in oral bacteria during orthodontic treatment (Zachrisson and Zachrisson, 1971). Additionally the acid from these bacteria result in the reduction of the mineral content of the tooth structure (Diedrich, 1981; O'Reilly and Featherstone, 1987).

The early carious lesions appear clinically as opaque, white spots caused by mineral loss in the surface and subsurface of the enamel (Øgaard *et al*, 1988). When further reduction takes place, decalcification continues and cavitation may occur (Zachrisson and Zachrisson, 1971; Arends and Christoffsen, 1986; Mitchell, 1992). Development of carious lesions during fixed orthodontic appliance therapy is an extremely rapid process and may present an aesthetic problem, even more than five years after treatment (Mizrahi, 1982).

The prevention of demineralisation during orthodontic treatment is therefore essential for aesthetic reasons and to circumvent the onset of caries. Strategies to minimise or eliminate decalcification include better oral hygiene, diet modification, use of dentifrice with fluoride, as well as the use of self applied topical fluoride rinses (Tanna, 2003). Other methods mentioned in the literature are: fluoride containing or releasing banding and bonding agents, in-office topical fluoride, fluoride varnish application and enamel sealant application (Tanna, 2003). Many of the methods of preventing decalcification require patient compliance and demand constant reinforcement and motivation.

DEMINERALISATION AND CARIES

The Illustrated Stedman's Medical Dictionary (Stedman, 1992) defines demineralisation or decalcification as a loss or decrease of the mineral constituents of the body or individual tissues, especially of bone and teeth.

Dental caries covers the continuum from the first atomic level of demineralisation, through the initial enamel or root lesion, through dentinal involvement, to eventual cavitation (Featherstone, 2004). The dynamic balance between demineralisation and remineralisation determines the end result. The disease is reversible, if detected early enough. Since demineralisation can be quantified relatively early before frank cavitation, intervention methods can be tested by short-term clinical trials. Intervention in the caries process can occur at any stage, either naturally or by the application of some procedure or treatment (Featherstone, 2004).

Zachrisson (1978) described the progression of caries from intact enamel surface as going through 3 distinct stages:

- Whitish decalcification without a cavity forming on the enamel,
- Whitish decalcification with a cavity beginning to form on the enamel,
- Enamel cavities that cannot be removed by cautious grinding.

Predisposing Factors to Caries Development

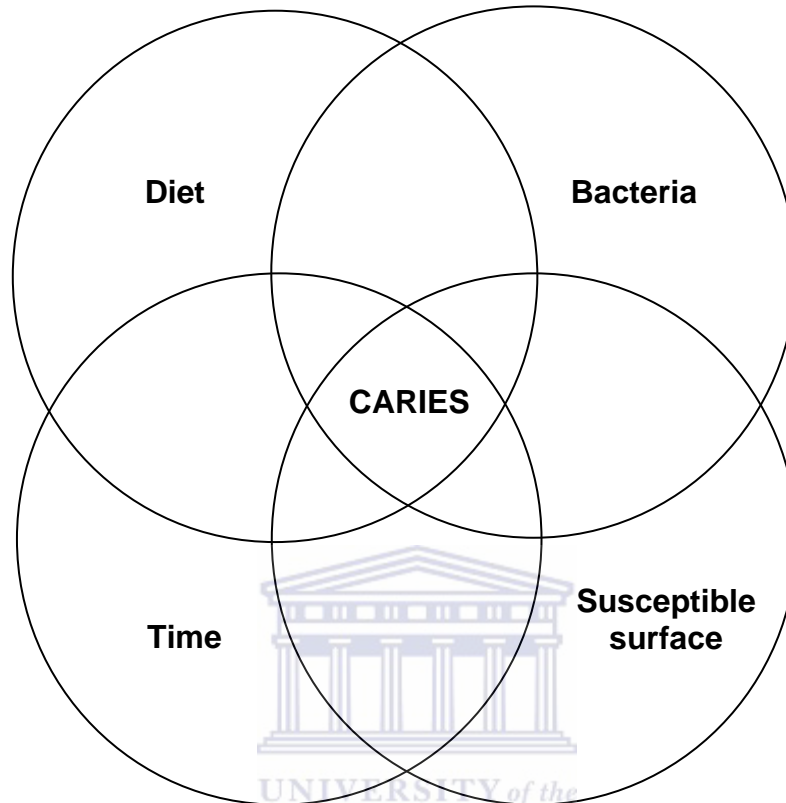
Harris *et al* (2004) emphasized that dental caries is widely recognised as an infectious disease induced by diet. They named the contributing factors in the aetiology of the disease as; cariogenic bacteria, fermentable carbohydrates, a susceptible tooth and host, and time (Fig, 1).

Possible interventions:

- Reduce intake of cariogenic sugars, particularly sucrose

Possible interventions:

- Reduce *S. mutans* numbers by;
- reduction in sugar intake
 - active or passive immunisation



Possible interventions:

- Avoid frequent sucrose intake (snacking)
- Stimulate salivary flow and sugar clearance

Possible interventions

- Water and other types of fluoridation
- Prevention during post-eruptive maturation
- Fissure sealing
- Remineralisation solutions
- Properly contoured restorations

Figure 1 - The four major factors in the aetiology of dental caries (Cawson and Odell, 2002).

There is substantial evidence that indicates that *streptococci* are essential for development of caries, particularly of smooth surfaces. These viridans *streptococci* which are a heterogeneous group include: *Streptococcus mutans* (*S. mutans*), *S. sobrinus*, *S. salivarius*, *S. mitior* and *S. sanguis*. Viridans *streptococci* vary in their ability to attach to different types of tissues, their ability to ferment sugars (particularly sucrose), and the concentra-

tions of acid thus produced. They also differ in the types of polysaccharides that they form (Ekstrand *et al*, 1983; Cawson and Odell, 2002).

The relationship between sugar consumption and caries incidence shows that frequent consumption of sugars is directly associated with caries (Featherstone *et al*, 1983; O'Reilly and Featherstone, 1987; Fontana *et al*, 1996; Gaffar *et al*, 1998). The acid produced by the fermentation of sugars results in a plaque pH drop, which initiates decalcification of the enamel (Featherstone *et al*, 1983; O'Reilly and Featherstone, 1987; Fontana *et al*, 1996; Gaffar *et al*, 1998).

The intake of dietary sucrose has two effects on plaque; firstly, the frequent ingestion of foods containing sucrose provides a strong potential for colonisation of *S. mutans*, thereby enhancing the caries potential of plaque. Secondly, frequent exposure of mature plaque to sucrose results in its rapid metabolism to organic acids. This results in a profound and prolonged drop in plaque pH (Clark, 1982).

Pathogenesis

Plaque accumulation encourages colonisation and growth of cariogenic bacteria such as *Streptococcus mutans* and *Lactobacillus acidophilus* (Sakamaki and Bahn, 1968; Balenseifen and Madonia, 1970; Lundstrom and Krasse, 1987; Rosenbloom and Tinanoff, 1991; Chang *et al*, 1999; Turkkahraman *et al*, 2005). The organic acids produced by these types of bacteria cause the dissolution of calcium and phosphate ions from the enamel surface. In a matter of four weeks, this process can lead to white spots or early carious lesions (Diedrich, 1981; O'Reilly and Featherstone, 1987).

The amount of enamel demineralisation, the rate of demineralisation and the likelihood of enamel remineralisation is influenced by salivary factors such as pH, rate of flow and buffer capacity (Mitchell, 1992; van Palenstein 1996). Evidence shows that salivary flow rate can influence both caries risk and caries activity (Papas *et al*, 1993). Adequate flow of saliva is considered an important factor in the prevention of enamel demineralisation (Andersson *et al*, 1974).

The pH and buffering capacity of the saliva is maintained by the rate of salivary secretion (Andersson *et al*, 1974). An intraoral environment with low pH favours colonisation of the cariogenic bacteria, particularly *Streptococcus mutans*, whereas a high salivary pH maintains a higher buffering capacity. There is also a significant negative correlation between the salivary buffering capacity and the frequency of caries (Russell *et al*, 1990).

Dietary sugars play an important role in the development of enamel caries and sucrose is necessary for *Streptococcus mutans* to cause significant smooth surface caries (Ekstrand *et al*, 1983). The acid produced by the fermentation of sugars results in a drop in pH of the plaque which initiates decalcification of the enamel (Featherstone *et al*, 1983; O'Reilly and Featherstone, 1987; Fontana *et al*, 1996; Gaffar *et al*, 1998).

O'Reilly and Featherstone (1987) found a close association between the frequency of ingestion of sucrose-containing foods, the duration that sugars are retained in the mouth and the prevalence of enamel demineralisation.

Pathological Features of Caries

Macroscopic Features

Early enamel caries manifest clinically as a white spot lesion (Gorelick *et al*, 1982). According to Cawson and Odell (2002), the enamel, despite having a chalky appearance, is hard and smooth to the probe (Fig.2). Once bacteria have penetrated the enamel, they reach the amelodentinal junction and spread laterally to undermine the enamel. First, the enamel loses the support of the dentine and is therefore greatly weakened. Second, it is attacked from beneath. Third, spread of bacteria along the amelodentinal junction allows them to attack the dentine over a wide area (Cawson and Odell, 2002).

The primary lesion thus provides the bridgehead for the attack on enamel, but undermining of the enamel determines the area of a cavity. Clinically this is frequently evident when there is no more than a pinhole lesion in an occlusal pit, but cutting away the surrounding enamel shows it to be widely undermined. As undermining of the enamel continues, it starts to collapse under the stress of mastication and to fragment around the edge of the cavity (Cawson and Odell, 2002).



Figure 2 - Early enamel caries (Cawson and Odell, 2002).

Microscopic Features

Cawson and Odell (2002) state that the microscopic changes in the early white spot lesion may be seen in undecalcified sections, but more readily when polarised light is used. The initial lesion is conical in shape with its apex towards the dentine, and a series of four zones of differing translucency can be discerned (Fig. 3). These zones are the translucent zone; the dark zone; the body of the lesion and the surface zone.

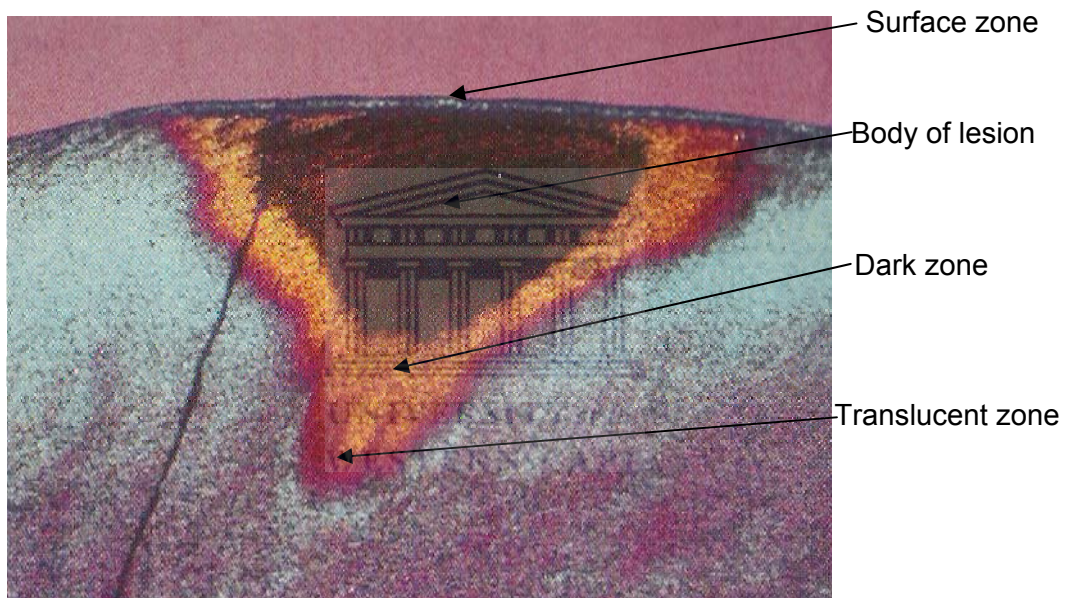


Figure 3 - Early enamel lesion (Cawson and Odell, 2002).

The translucent zone is the first observable change. The appearance of the translucent zone results from formation of submicroscopic spaces or pores apparently located at prism boundaries and other junctional sites such as the striae of Retzius (Cawson and Odell, 2002).

The dark zone is fractionally superficial to the translucent zone. Polarised light microscopy shows that the volume of the pores in this zone has in-

creased to between two and four percent of the enamel volume (Cawson and Odell, 2002).

The body of the lesion forms the bulk of the lesion and extends from just beneath the surface zone to the dark zone. When viewed under transmitted light, the body of the lesion is comparatively translucent compared with normal enamel and sharply demarcated from the dark zone. Within the body of the lesion the striae of Retzius appear enhanced, particularly when the section is mounted in quinoline and viewed under polarised light. Polarised light examination also shows that the pore volume is five percent at the periphery but increases to at least twenty five percent in the centre (Cawson and Odell, 2002).

The surface zone represents one of the most important changes in enamel caries in terms of prevention and management of the disease. It shows the paradoxical feature that it has not merely remained intact during this stage of the attack but remains more heavily mineralised and radiopaque than the deeper zones (Cawson and Odell, 2002).

ENAMEL DEMINERALISATION IN ORTHODONTICS

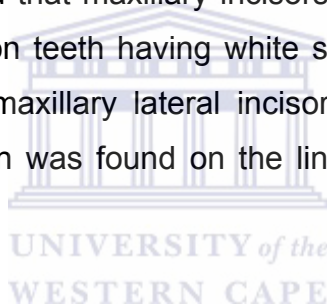
Prevalence

The prevalence of enamel demineralisation (white spot lesions) in orthodontic patients has reported to be up to ninety six percent in patients undergoing fixed appliance therapy (Mizrahi, 1982; Gorelick *et al*, 1982; Mitchell, 1992). A cross-sectional study by Gorelick *et al* (1982) found that fifty percent of individuals undergoing orthodontic treatment had white spot lesions compared with twenty five percent of controls.

O'Reilly and Featherstone (1987) indicated the following common areas for plaque accumulation that may then lead to enamel decalcification: gingival margins under bands where the luting agents have washed out and the junction of the bonding agent and etched enamel surface.

In a study by Banks and Richmond (1994) on enamel decalcification in orthodontically treated patients, the incidence and distribution in these patients were recorded using a modified index by direct clinical observation. Their results showed that seventy percent of patients were affected by some form of decalcification.

Mizrahi (1982) showed that maxillary incisors and mandibular first molars were the most common teeth having white spot formation. Gorelick *et al* (1982) reported that maxillary lateral incisors were most often affected while no decalcification was found on the lingual surfaces of mandibular incisors.



Mizrahi (1983) did a study to determine the prevalence and severity of enamel opacities occurring on different surfaces of the dentition and also the distribution of these lesions on individual teeth following orthodontic treatment. His results showed that there was a significant increase in the prevalence of enamel opacities on the vestibular and lingual surfaces of the teeth. The increase was greater on the cervical and middle third of crowns. Among individual teeth, there was a statistically significant increase in the prevalence and severity of enamel decalcification on the maxillary and mandibular first molars, maxillary lateral incisors and the mandibular lateral incisors and canines. This increase in prevalence was greatest on the cervical and middle thirds of the vestibular surfaces of these teeth.

Øgaard (1989) showed that even five years after treatment, orthodontic patients had a significantly higher incidence of white spot lesions than a control group of patients who had not had orthodontic treatment. The teeth most commonly affected are molars, maxillary lateral incisors, mandibular canines and premolars.

Vorhies *et al* (1998) found that there was a significant increase in the incidence of white spot lesions following the placement of fixed orthodontic appliances when compared with a control of untreated individuals. Their study showed that 49.6 percent of the patients developed areas of decalcification. The frequency of white spot formation on bonded teeth was found to be in the following order: maxillary lateral, mandibular canine, mandibular first premolar, and mandibular first molar, mandibular second premolar, maxillary canine and maxillary first premolar (Vorhies *et al*, 1998).

Boersma *et al* (2005) undertook a study to determine the caries prevalence on the buccal surfaces of teeth in orthodontic patients using Quantitative Light-induced Fluorescence and visual examination immediately after removal of fixed appliances. The results of their study found 97 percent of all subjects had white spot formations and on average, 30 percent of the buccal surfaces were affected. Furthermore, they found that there were more white spot lesions in males (40%) when compared with the females (22%). Prevalence of white spots was lower in incisors and canines than in molars and premolars.

More recently Lovrov *et al* (2007) examined fifty-three patients with fixed orthodontic appliances at the Erlangen-Nuremberg University (Germany) in 2007. They found that of the dentitions examined, 2.5 percent of teeth before and 26.4 percent of the teeth after treatment had white spot lesions. Of all teeth, 24.9 percent developed either new white spot lesions or a rise in the number of lesions. There was higher incidence of white spot

lesion in the upper and lower premolars (34.4%) compared with the front teeth (28.1%). The molars seemed to be the least affected (11.8%).

Predisposing Factors

Decalcification or demineralisation of the enamel is caused by ineffective oral hygiene and subsequent retention of bacterial plaque for an extended period of time on the enamel surface (Gorelick *et al*, 1982).

It is generally accepted that the presence of fixed orthodontic appliances contributes to the accumulation of plaque which, in turn, may lead to the development of areas of enamel demineralisation presenting clinically as enamel opacities (Mizrahi, 1982) and a generalised gingivitis (Zachrisson and Zachrisson, 1971).

A number of studies have shown an increase in the prevalence of enamel opacities following multibanded or bonded orthodontic therapy (Bach, 1953; Bach, 1954; Zachrisson and Zachrisson, 1971; Gorelick *et al*, 1982; Mizrahi, 1982; 1983). The presence of archwires complicates cleaning and makes access to plaque retaining areas difficult, especially when multiple loops, auxiliary archwires and different types of elastics are used (Forsberg *et al* 1991; Sukontapatipark *et al*, 2001; Turkkahraman *et al*, 2005). As a consequence of this, new sites susceptible to enamel demineralisation are created next to the bands and brackets during orthodontic treatment with fixed appliances (Forsberg *et al* 1991; Sukontapatipark *et al*, 2001; Turkkahraman *et al*, 2005).

Features

Clinically, formation of white spots around orthodontic attachments can occur as early as 4 weeks into treatment (O'Reilly and Featherstone, 1987; Øgaard *et al*, 1988).

According to Boyd (2001) mild decalcification due to orthodontic treatment is evidenced by a clinical colour change (white or white-yellow stains) with possible surface roughness (Fig.4). Moderate decalcification is usually seen as larger areas of colour changes (yellow-brown stain) with definite surface roughness. Severe decalcification is characterised by large areas of darker, yellow-brown stains with lost enamel.



Figure 4 - Extensive white spot lesion development during active orthodontic treatment (Sudjalim *et al*, 2006).

PREVENTION OF DEMINERALISATION IN ORTHODONTICS

Prevention of demineralisation can be achieved with the use of either mechanical methods like toothbrushing and flossing (Brightman *et al*, 1991; Heasman *et al*, 1998; Ramaglia *et al*, 1999) or by using chemical methods

such as fluoride mouthrinse, fluoride gels, fluoride varnishes and chemical barriers like sealants (Øgaard *et al*, 1992; Mitchell, 1992; Frazier *et al*, 1996; Kindelan, 1996; Todd *et al*, 1999; Demito *et al*, 2004, Hu and Featherstone, 2005).

Mechanical Methods

A. Toothbrushing

Toothbrushing is the most common form of mechanical plaque removal and can be performed with manual or electric toothbrushes. Toothbrushing twice daily is recommended by many clinicians as an essential part of a daily plaque control programme for all orthodontic patients (Sudjalim *et al*, 2006). Many toothbrushes are available on the market. Specifically designed orthodontic toothbrushes are also available and are said to be more effective than regular toothbrushes in removing plaque deposits around orthodontic brackets (Boyles, 2007).

According to Boyd (2001), there are few well-controlled, long-term studies comparing toothbrushing as the only effective method to prevent enamel decalcification. He further states that studies have demonstrated that 20 percent to 40 percent of orthodontic patients with fixed appliances show less than ideal plaque removal with conventional toothbrushes even with repeated instructions (Boyd, 2001). Sudjalim *et al*, (2006) stated that there are conflicting reports on the effectiveness of both the manual and the electric toothbrushes.

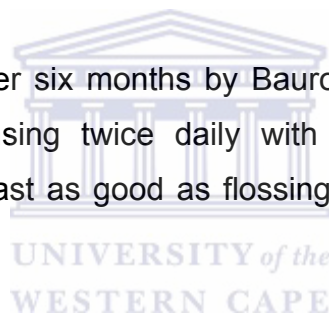
A study by Wilcoxon *et al* (1991) demonstrated that electric toothbrushes were found to be more effective in plaque removal than regular manual toothbrushes. The electric toothbrush with the short pointed bristles is

most effective in minimising plaque accumulation in orthodontic patients and thereby preventing enamel demineralisation (Boyd *et al*, 1989; Boyd and Rose, 1994).

B. Flossing

Although dental floss has been the mechanical device most widely recommended for purposes of interproximal plaque control, surveys of oral hygiene practices have shown that only 10 to 40 percent of respondents reported the daily use of floss, in contrast to the close to 100 percent who reported daily toothbrushing (Bauroth *et al*, 2003) .

Results of a study over six months by Bauroth *et al* (2003) showed that toothbrushing and rinsing twice daily with an essential oil-containing mouthrinse, was at least as good as flossing daily in reducing interproximal plaque.



Tufekci *et al* (2007) states that considerable clinical trial evidence is available showing that the oral hygiene status is significantly improved when antibacterial mouthrinses are added to daily oral hygiene measures (toothbrushing and flossing) compared with toothbrushing and flossing alone.

Chemical methods

Fluoride administration has been proposed as a method of reducing enamel susceptibility to decalcification (Mitchell , 1992). Fluoride affects the caries process by enabling the formation of high quality fluorapatite that aids remineralisation and inhibits glycolysis of plaque micro-

organisms (Zachrisson, 1976; Schwaninger and Vickers-Schwaninger, 1979; Mitchell, 1992).

Several fluoride regimens with varying fluoride concentrations, pH, and delivery systems (varnish, gel, rinse, dentifrice) have been shown to be effective in preventing demineralisation (O'Reilly and Featherstone, 1987; Geiger *et al*, 1992). Clinical studies have been conducted to evaluate the effectiveness of different methods of fluoride administration (Fischer *et al*, 1954; Howell *et al*, 1955; O'Reilly and Featherstone, 1987; Geiger *et al*, 1988; Alexander and Ripa, 2000; Benson *et al*, 2005).

Boyd (2001) states that according to literature the best way to prevent decalcification in orthodontic patients is by using a daily self-applied, topical low-concentration stannous fluoride (SnF₂) gel. The fluoride rinse or gel protocol should continue for 6 months after appliance removal to remineralise areas of decalcification that may have occurred during treatment (Boyd, 2001).

Light-cured sealants containing fluoride could also be used on the entire labial surface (Frazier *et al*, 1996). The sealants can also be reapplied during treatment if demineralised areas appear (Boyd, 2001). Other products that contain fluoride such as cements, elastomeric chains, or fluoride varnishes may reduce the incidence of decalcification (Frazier *et al*, 1996). However Boyd (2001) states that most studies of these products do not show an actual reduction in the frequency of decalcification but only that there is a short-term fluoride release of 4 to 8 weeks.

A. Toothpastes

Regular use of fluoride toothpaste is a very common recommendation by orthodontists as a means preventing plaque accumulation (Zachrisson, 1977; Gorelick *et al*, 1982; Øgaard *et al*, 1992). Studies have shown that the use of fluoride toothpaste combined with a regime of not rinsing with water after toothbrushing to be more effective against plaque formation (Chesters, 1992; Sjögren and Birkhed, 1994; Attin and Hellwig, 1996).

Other studies have shown that the use of toothpastes alone has been proven to be insufficient to prevent lesion development around orthodontic brackets (Zachrisson, 1977; Gorelick *et al*, 1982; Øgaard *et al*, 1992).

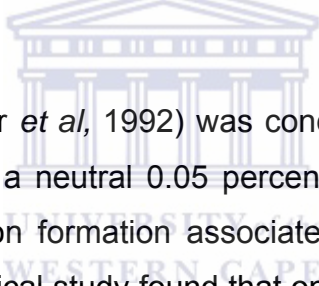


B. Mouthrinses

Fluoride mouthrinse is an effective adjunct to mechanical cleaning (Bauroth *et al*, 2003). Its topical effect reduces enamel decalcification and gingival inflammation, and enhances the remineralisation of enamel adjacent to orthodontic brackets (Denes and Gabris, 1991; Boyd, 1993). A review article of 30 studies on the effectiveness of using fluoride mouthrinses for the reduction of caries during orthodontic treatment estimated a success rate of around 30 percent (Horowitz, 1980).

Hirschfield (1978) advocated the use of an acidulated phosphate fluoride (APF) mouthrinse to make enamel more resistant to orthodontic induced decalcification. Duckworth *et al* (1987) found that following two weeks use of sodium fluoride mouthrinse, with one rinse per day, fluoride concentration in the saliva increased significantly. They concluded that the ability of fluoride treatments to sustain elevated oral fluoride levels between daily applications may be of major importance in caries control.

O'Reilly and Featherstone (1987) undertook a study to determine quantitatively the amount of demineralisation and the ability of commercially available products to inhibit or reverse demineralisation related to orthodontic treatment. The control group brushed only with the supplied dentifrice. In addition to brushing with the dentifrice, one group rinsed once each night with a sodium fluoride (0.05 percent) mouthrinse another group received a weekly topical APF treatment (1.2 percent fluoride) and the last group received a weekly topical APF treatment and rinsed once each night with the sodium fluoride mouthrinse. Their study demonstrated that measurable demineralisation occurred around orthodontic appliances after only one month and that demineralisation could be completely inhibited and/or reversed by the use of commercially available fluoride products.

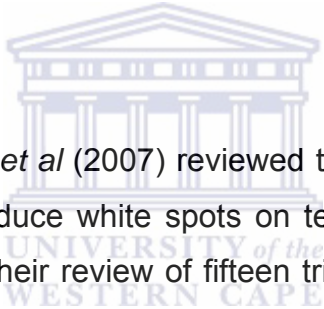


A clinical study (Geiger *et al*, 1992) was conducted to determine whether rinsing frequently with a neutral 0.05 percent sodium fluoride rinse influenced white spot lesion formation associated with orthodontic brackets. The results of their clinical study found that only 13 percent of the 206 participants fully complied with the rinse protocol; 42 percent of the subjects used 10 ml approximately every other day; and 45 percent used the rinse less frequently. A significant dose-response relationship was noted in that those who rinsed at least once every other day had fewer lesions than those who rinsed less frequently. Geiger *et al* (1992) concluded that a significant reduction in enamel white spot lesions can be achieved during orthodontic therapy through the use of a 10 ml neutral sodium fluoride rinse at least once every other day.

Paraskevas *et al* (2005) found that the combined use of amine fluoride/stannous fluoride (Amy/ SnF₂) mouthrinse did not decrease gingivitis at a significant level in comparison with the regular regime of two times daily brushing with a NaF-containing dentifrice. It did result in greater

plaque reduction than that observed with the use of the conventional dentifrice only.

A prospective, randomized, double-blind study with 115 orthodontic patients was designed by Øgaard *et al* (2006) to determine the effect of combined use of a toothpaste/mouthrinse containing amine fluoride/stannous fluoride on the development of white spot lesions, plaque, and gingivitis on maxillary anterior teeth in orthodontic patients. They concluded that the use of an amine fluoride/stannous fluoride (AmF/ SnF₂) toothpaste together with a mouthrinse had a slightly more inhibitory effect on white spot lesion development, plaque and gingivitis on maxillary anterior teeth during fixed orthodontic treatment compared than did sodium fluoride.



More recently Benson *et al* (2007) reviewed the literature on the ability of fluoride products to reduce white spots on teeth during fixed orthodontic appliance treatment. Their review of fifteen trials found some evidence to support the use of a daily 0.05 percent neutral sodium fluoride rinse in reducing the severity of white spot lesions.

Brightman *et al* (1991) showed a dramatic reduction in plaque (65 percent) and gingival bleeding (77%) during a 3 month regimen of daily use of a 0.12 percent chlorhexidine mouthrinse. This was supported by Boyd (2001) who suggested that chlorhexidine is the best product for optimum management of plaque accumulation in adolescent orthodontic patients.

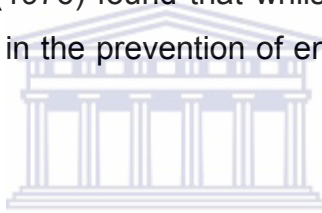
Fluoride Gels

Wefel and Harless (1981) have shown that topical fluoride treatment with either neutral or acidulated NaF preparations results in fluoride incorpora-

tion into intact enamel. Lehman *et al* (1981) have also shown that topical fluoride treatment produced a more acid resistant outer layer.

Some investigators have advocated the application of topical fluoride before etching (Zachrisson, 1975; Byrant *et al*, 1985), while others have suggested that acid-etching the enamel before fluoride application increases fluoride uptake (Mellberg and Loertscher, 1973).

Stannous fluoride gels have also been recommended for patients during orthodontic treatment due to their ability to decrease enamel decalcification (Stratemann and Shannon, 1974; Shannon and West, 1979). Furthermore, Zachrisson (1976) found that whilst professionally applied fluoride gel was beneficial in the prevention of enamel caries; it was not cost-effective.



Boyd (1993) compared the use of a 1100ppm fluoride toothpaste alone with either a daily 0.05 percent sodium fluoride rinse or a 0.4 percent stannous fluoride gel applied twice daily with a toothbrush. He found that both the gel and rinse provided additional protection against decalcification when compared to toothpaste alone, but neither was superior.

Øgaard *et al* (1988) have suggested that visible white spots on facial surfaces that develop during orthodontic treatment should not be treated topically with concentrated fluoride agents since this procedure may prevent complete repair.

The above methods of preventing decalcification demand patient compliance and require constant reinforcement and motivation which can be a problem in some patients.

Bonding Materials

In an attempt to achieve a compliance-free, constant exposure to topical fluoride, fluoride-releasing bonding agents were developed. In the late 1980's, glass ionomer cements were proposed as an alternative to the more commonly used composite material for bracket bonding (Sudjalim *et al*, 2006). The glass ionomer cements have been widely used for cementing orthodontic bands due to their ability to release fluoride (Bassham, 1999).

Fluoride release, whether short-term or long-term, from dental restorative materials is related to their matrices, setting mechanisms and fluoride content. Fluoride releasing materials may act as a fluoride reservoir and may increase the level of fluoride in saliva, plaque, and dental hard tissues (Boyles, 2007).

Other proposed benefits of using glass ionomer cements include: the cements do not need pretreatment of the enamel with phosphoric acid to create conditions for mechanical bonding; they release fluoride over several months and they may contribute to the possible development of a modified, less cariogenic microflora (Matalon *et al*, 2005). However, Cook and Youngson (1990) found that glass ionomers have significantly weaker bond strengths and are therefore questionable as orthodontic bonding adhesives.

In-vitro studies of glass ionomer cements have demonstrated a one to two year sustained fluoride release and evidence exists that these cements may reduce decalcification (Vorhies *et al*, 1998; Millett *et al*, 1999; Chung *et al*, 1999).

In a study by Voss *et al* (1993), orthodontic brackets bonded onto teeth with resin-modified glass ionomer (RMGI) cement, Ketac-Fil (Espe, Seefeld, Germany) were found to demonstrate a 50 percent reduction in lesion depth whether or not a fluoride varnish, Visiobond (Espe, Seefeld, Germany) was applied. RMGI adhesives have been demonstrated to sustain fluoride release long after initial application.

McNeill *et al* (2001) reported that light-cured, fluoride- containing orthodontic bonding materials release enough fluoride to prevent white spot lesions six months after bonding. The Fuji Ortho LC (RMGI) cement released the most fluoride after six months, followed by a polyacid-modified composite resin, Assure (Reliance Orthodontic products, Inc, Itasca, III). The modified bisphenol-A-diglycidylmethacrylate (Bis-GMA) composite resin, Python (TP Orthodontics, LaPorte, Indiana) released the least fluoride.

An *in-vitro* study by Corry *et al* (2003) was undertaken to compare the cariostatic potential of a resin modified glass ionomer cement (Fuji Ortho LC) with that of a resin control, Transbond (3M/Unitek), for bracket bonding and to compare the effect of extrinsic fluoride application on the cariostatic potential of each material. Fluoride release from Fuji Ortho LC alone fell to minimal values, but with the addition of extrinsic fluoride the levels fell initially and then followed an upward trend. There was minimal fluoride release, from Transbond alone, but with daily addition of extrinsic fluoride, subsequent fluoride release was increased. Significant differences existed in the amount of fluoride released between all groups, except comparing Fuji Ortho LC alone and Transbond with added fluoride. The authors then concluded that the creation of white spot inhibition could best be achieved by the use of resin-modified glass ionomer cement, supplemented with fluoride exposure.

In a review on fluoride-releasing restorative materials, Wiegand and co-workers (2007) found that glass ionomers and compomers, which are fluoride releasing dental materials, do show cariostatic properties. However, it was not proven that the incidence of secondary caries can be significantly reduced due to the release of fluoride. Glass ionomers have been shown to decrease decalcification within 1mm of the orthodontic attachment due to the slow release of fluoride.

Fluoride Releasing Elastomeric Ligatures

Several authors have suggested that fluoride releasing elastomeric modules are effective in reducing plaque accumulation and enamel decalcification around orthodontic brackets (Wiltshire, 1999; Banks *et al*, 2000; Mattick *et al*, 2001).

Wiltshire (1996) did an *in-vitro* study to determine fluoride release from 200 fluoride-containing elastomeric ligature ties. He reported that the release of fluoride is sufficient to inhibit demineralisation and promote remineralisation even after six months. On the contrary, Joseph *et al* (1993) reported that fluoride release from a fluoride containing elastic chain was high for the first week and decreased significantly thereafter.

Banks and his co-workers (2000) evaluated in an *in-vitro* prospective study the effectiveness of stannous fluoride-releasing elastomeric modules and chains in the prevention of enamel decalcification during fixed appliance therapy. They found that fluoride-releasing elastomerics appear to provide a clinically worthwhile reduction in enamel decalcification during fixed appliance therapy when they are changed at each treatment visit.

This was further supported by Mattick *et al* (2001). They reported that fluoride releasing elastomeric modules reduced the incidence of decalcification around orthodontic brackets during a complete course of orthodontic treatment. They concluded that the use of fluoride releasing elastomeric modules reduced the degree of decalcification experienced during orthodontic treatment.

However, Doherty *et al* (2002) found that fluoride releasing ligatures do not provide a significant anti-cariogenic benefit in patients undergoing orthodontic treatment. Benson *et al* (2004) also concluded that fluoridated elastomers had no effect on the quantity of disclosed plaque around orthodontic brackets.

Miura *et al* (2007) evaluated the efficacy of fluoride-releasing elastomers in the control of *Streptococcus mutans* levels in the oral cavity. They found that there were no significant differences in the numbers of *Streptococcus mutans* in saliva or plaque in the area surrounding the fluoride-releasing or conventional elastomeric ligature ties. They thus concluded that other means of prevention against enamel decalcification should, therefore, be indicated for orthodontic patients.

Fluoride Varnishes

Fluoride varnish is a thin coating of resin that is applied to the tooth surface to protect it from decay, to retard, arrest, and reverse the process of cavity formation (Governor, 2005). According to the Food and Drug Administration (FDA), fluoride varnish falls under the category of “drugs and devices” that presents minimal risk and is subject to the lowest level of regulation (Governor, 2005).

Fluoride-containing varnishes were developed during the late 1960's and early 1970's in an effort to improve shortcomings of the existing topical fluoride vehicles, such as fluoride gels and mouthrinses. The beneficial effect is thought to be derived from the prolonged contact of the fluoride varnish with tooth enamel (Beltrán-Aguilar *et al*, 2000). The varnish has been shown to provide prolonged fluoride release when compared with a mouthrinse, resulting in an increased enamel fluoride uptake (Petersson, 1993).

History

The use of varnishes as a protective coating is discussed in the literature as early as 1940 (Lee *et al*, 1973). Fluoride varnishes were first introduced in Europe in 1964 under the trade name Duraphat (Pinkham *et al*, 2005). It first became available in the United State of America in 1991 when Duraflor received approval from the FDA for its use as a cavity varnish. In 1997 Duraphat also became available in the USA (Pinkham *et al*, 2005).

A clinical test was done by Meyers in 1952, in which teeth were coated with copal varnish (cavity liner and dentinal tubuli sealer, contains resin) prior to banding. The incidence of new demineralisation in 263 uncoated teeth was 27.4 percent; whereas in 275 coated teeth, only 5.9 percent of the teeth exhibited new demineralised lesions (Meyers, 1952).

Tillery *et al* (1976) tested a polymeric protective coating, Protecto (Lee Pharmaceuticals, South El Monte, Calif) and found it to provide more protection against decalcification of teeth under loose orthodontic bands than did either acidulated phosphate fluoride gel or stannous fluoride mouthrinse.

Øgaard *et al* (1996) reported a 48 percent reduction in enamel demineralisation when using a fluoride varnish. This was later supported by Todd *et al.* (1999) who found that the application of fluoride varnish (Duraflor; Pharmascience Inc., Montreal, Canada) decreased the incidence of enamel demineralisation in orthodontic treated teeth by 50 percent.

Types of fluoride varnishes

There are basically three types of fluoride varnishes available in the United States: Duraphat (Colgate Oral Pharmaceuticals) which contains 5 percent sodium fluoride (22,600 parts per million), Duraflor (Pharmascience Inc) containing 5 percent NaF (22,600 parts per million) and Fluor Protector (Ivoclar-Vivadent) which contain 1 percent difluorsilane (1000 parts per million) (Beltrán-Aguilar *et al*, 2000).

Other fluoride varnishes available on the market are: Bifluoride (Voco, Germany) which contains 6 percent fluoride (Sköld-Larsson *et al*, 2000) and CavityShield (OMNII Oral Pharmaceuticals, West Palm Beach, Fla). CavityShield contains 5 percent sodium fluoride in a natural resin (Shen and Autio-Gold, 2002). Duraphat, Duraflor and CavityShield set to a light yellow film, while Fluor Protector sets to a thin transparent film (Shen and Autio-Gold, 2002).

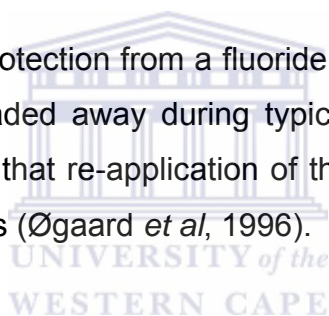
Research studies

Fluoride varnishes have been reported to be superior to sodium fluoride and monofluorophosphate dentifrices in their ability to increase fluoride uptake in enamel (Pettersson, 1976; Arends *et al*, 1980).

Petersson (1976) found that there was an increase in fluoride uptake in saliva after 3 weeks when comparing a fluoride varnish with 2 percent sodium fluoride gel applied weekly, 2 percent acidulated phosphate fluoride gel applied weekly, or 0.25 percent sodium fluoride rinse used daily.

Seppä *et al* (1995) compared the caries-preventive effect of Duraphat and an acidulated phosphate fluoride gel, Nupro (Dentsply Professional, York, Pa). They concluded that fluoride varnish is as effective as fluoride gel at least in preventing approximal caries. Therefore they suggested that taking into account the shorter treatment time, using fluoride varnish for professional applications seems justified.

The physical barrier protection from a fluoride varnish is short-lived as this material is easily abraded away during typical toothbrushing. In view of this, it was suggested that re-application of this material is recommended at least every 3 months (Øgaard *et al*, 1996).



Adriaens *et al* (1990) tested Fluor Protector, a fluoride varnish, applied to molars before orthodontic banding for the prevention of white spot formation. They found that Fluor Protector was very effective in the prevention of white spot formation under molar bands. However, van der Linden and Dermaut (1998) found that the application of Fluor Protector in combination with Aquacem did not contribute to a reduction of white spot formation underneath molar bands when compared with the use of only Aquacem for banding.

Kindelan (1996) attempted to measure *in-vitro* demineralisation around orthodontic brackets bonded using five different bonding agents; Concise (3M,/Unitek, Bradford), Bondfast orthodontic composite (Orthocare, Bradford), Rely-a- Bond (Forestadent), Pulpdent O.B.A. (Ortho-care, Bradford),

Ketac-cem (ESPE, London) whilst a sixth group utilized a fluoride varnish (Duraphat) after bonding. The results of the study showed that Concise with Duraphat, Ketac- cem, and Pulpdent O.B.A. performed statistically significantly better than Concise, Bond-fast, and Rely-a-bond alone in resisting enamel demineralisation.

In 1999, Todd and co-workers evaluated the ability of Duraflor to inhibit demineralisation of enamel surrounding orthodontic brackets. Those teeth treated with Duraflor exhibited 50 percent less demineralisation than the control teeth and an even greater difference when compared to the non-fluoridated placebo cavity varnish (Pharmascience Inc., Montreal, Canada) group.

Sköld-Larsson *et al* (2000) did a study to measure the fluoride concentration in plaque after a single topical application of different fluoride varnishes (Bifluoride, Duraphat and Fluor Protector) with contrasting levels of fluoride. Their study found that fluoride varnish treatments resulted in elevated fluoride levels in plaque adjacent to fixed orthodontic appliances for a period of up to one week. The result further showed that the fluoride concentration in plaque was back to baseline levels for all participants in the Duraphat group after 7 days, while some individuals in the Bifluoride and Fluor Protector groups still registered slightly increased levels after 30 days.

A study was done by Gillgrass *et al* (2001) in which experimental Polymer coating; Odyssey (3M, Unitek, Monrovia, CA, USA) was compared with Duraphat and a chlorhexidine-containing varnish: Cervitec (Vivadent, Liechtenstein). The findings were as follows: Duraphat group exhibited the lowest mean lesion depth; the Duraphat and Odyssey groups had significantly less lesion depth when compared with the control. The efficacy of

Duraphat application in preventing demineralisation was demonstrated in that study.

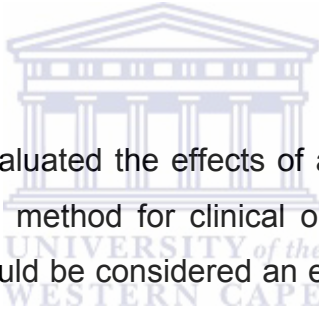
In 2001, Castillo and co-workers conducted a study to evaluate the fluoride released from two fluoride varnishes: Duraphat and Duraflor. The authors found a greater variability in the release of fluoride from the Duraflor samples than from the Duraphat samples. They concluded that both varnishes released fluoride for five to six months. However, the two products exhibited differences in their release kinetics (Castillo *et al*, 2001).

A randomised prospective clinical study was conducted by Øgaard *et al* (2001) to test whether the application of antimicrobial varnish (Cervitec) in combination with Fluor Protector was significantly more effective in reducing white spot lesions on the labial surfaces than the application of the fluoride varnish alone. Their results showed that the antimicrobial varnish significantly reduced the number of *S. mutans* in plaque during the first 48 weeks of treatment. This effect did not significantly decrease the development of white spot lesions on the labial surfaces when compared with the group receiving the Fluor Protector application only. In that study there was, however, a clear trend indicating that the combination of the antimicrobial and fluoride varnishes was more effective in reducing the amount of new lesions on maxillary incisors.

An *in-vitro* study was done by Schmit *et al* (2002) to evaluate the effect of Duraflor on inhibition of enamel demineralisation adjacent to orthodontic brackets bonded with either RMGI cement, Fuji Ortho LC (GC America Inc, Alsip, Ill) or composite resin cement, Transbond (3M/ Unitek, Monrovia, Calif). Teeth bonded with RMGI cement showed no significant differences in lesion depth between varnish and non-varnish groups. However, teeth bonded with composite showed a 35 percent reduction in decalcification when Duraflor was applied. They concluded that clinicians

should consider applying Duraflor on areas of enamel that exhibit demineralisation or are at risk of demineralisation in patients with poor oral hygiene.

Demito *et al* (2004) tested the hypothesis that a fluoride varnish (Duraflor) is effective in reducing demineralisation (white spot) lesions adjacent to bonded orthodontic brackets. They found that teeth that had been treated with two applications of a fluoride varnish (one at the outset and another 15 days later) demonstrated about 38 percent less mean lesion depth than teeth where no varnish had been applied. They concluded that orthodontists may wish to consider the application of fluoride varnish during fixed orthodontic therapy to help reduce the development of enamel white spot lesions.



Gontijo *et al* (2007) evaluated the effects of a fluoride varnish application as a caries prevention method for clinical orthodontics. They concluded that fluoride varnish could be considered an efficient preventive method to enhance enamel resistance against the cariogenic challenges during orthodontic therapy.

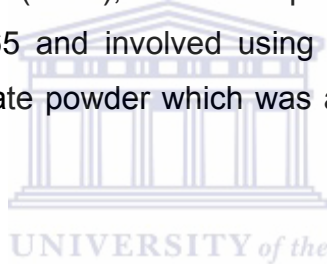
Stecksén-Blicks and co-workers (2007) evaluated the efficacy of topical fluoride varnish applications on white spot lesion formation in adolescents during treatment with fixed orthodontic appliances. The results of their study showed that regular topical fluoride varnish applications during treatment with fixed appliances may reduce the development of white spot lesions adjacent to the bracket base. The authors therefore concluded that application of fluoride varnish should be advocated as a routine measure in orthodontic practices during treatment.

Sealants

Sealants are available as either partially filled or unfilled (De Spain, 1999). Inert filler particles are added to the resin to increase resistance to wear. Unfilled sealants flow easily on the tooth surface and have little effect on occlusion after placement. Sealants may be clear, tinted or opaque. Tinted or opaque sealants are easier to re-evaluate for retention than clear sealants, and are well-accepted by patients (De Spain, 1999).

1. History

According to Bassham (1999), the technique of sealing the enamel was first introduced in 1965 and involved using ethyl-2-cyanoacrylate mixed with methyl methacrylate powder which was applied to pit and fissures of posterior teeth.



Buonocore and his co-worker published their paper on the successful application of sealants to pits and fissures in 1967 (Buonocore *et al*, 1968). Since that time, sealants have been developed into chemical cured and light cured bisphenol-A-diglycidylmethacrylate (Bis-GMA) resins (Bassham, 1999).

2. Types

Sealants are divided into different categories based on mode of curing: light cured, chemical cured or dual cured (Tanna, 2003). Chemically cured sealants come in two separate solutions. Light cured sealants have diketone comphoroquinone and an aliphatic amine as initiator and visible light at 460 nm as an accelerator (Tanna, 2003). Light cured systems come in one container and the curing process or working time is controlled by the

operator using a curing light. Dual cure sealants use a combination of chemical and light cure mechanisms. Though the curing mechanisms are different, retention rate and bond strength are similar for the chemical cured and light cured sealants (Tanna, 2003).

3. Composition

The components of sealants are similar to those of composite resin restorative material (De Spain, 1999). The most common materials used today are bisphenol-A-diglycidylmethacrylate (Bis-GMA), which polymerise when exposed to a visible light source. This third-generation material replaced the second-generation methacrylates which were autopolymerised. Self curing sealant resin contains a chemical catalyst and accelerator that harden when mixed (De Spain, 1999).

Phillips and Swartz (1970) stated that most of the sealants used in dentistry are based on the bisphenol-A-diglycidylmethacrylate (Bis-GMA) resin. These sealants contain inorganic fluoride compounds and polyacrylate materials. The chemistry of the Bis-GMA types of sealants is essentially the same as that of the composites. The principal difference is that the Bis-GMA sealants must be made more fluid to penetrate into pits and fissures and also into etched areas produced on the enamel.

Filler particles are added to sealants for increased wear resistance. Compressive strength, modulus of elasticity, hardness, and water sorption properties of the sealant are improved when filler particles are added. Tensile strength is the only property that is not affected by the addition of filler particles (Bassham, 1999).

Fluoride is added to sealants to enhance their anti-caries benefits. The greatest amount of fluoride is released within the first 24 hours after sealant placement, and continues, in lessening degrees over time (Cooley *et al*, 1990). This release results in an immediate increase in salivary fluoride levels in the region of the mouth closest to the sealant. Fluoride in sealants may act in its usual manner by enhancing remineralisation of the tooth surface, or it may affect cariogenic bacteria at the enamel-sealant interface through its antibacterial properties (De Spain, 1999).

4. Research studies

Lee *et al* (1973) began looking at utilising a polymeric adhesive coating, Enamalite (Lee Pharmaceuticals) that could prevent demineralisation in the orthodontic patient. Their rationale was that decalcification could be avoided or minimised by using a protective coating prior to the placement of the orthodontic bands and later removed on completion of the orthodontic treatment. They reported that the use of this polymeric adhesive coating was an effective orthodontic prophylaxis which could eliminate the problem of decalcification.

Zachrisson (1978) discussed four distinct advantages to using sealants in orthodontic bonding, each of them important enough alone to merit their use: (1) caries protection, (2) increased bond strength, (3) moisture control no longer extremely important once the sealant is applied to the etched surface, (4) the debonding action is facilitated, since less adhesive is needed and the break occurs more easily in the enamel/adhesive interface. For these reasons, he believed that all bonding systems should include a sealant. It should be mentioned, however, that different sealants do not have the same chemical properties and may therefore not exhibit all the properties mentioned above.

Self Curing sealants

Brauer (1978) cautioned that the sealant coating should be thin and even, because excess sealant may induce bracket drift and unnatural enamel topography when polymerised. However, according to Zachrisson (1978), a particular problem in orthodontics was that the sealant film on a facial tooth surface was so thin that oxygen inhibition of polymerisation was likely to occur throughout the film with autopolymerising sealants.

Zachrisson *et al* (1979) tested the effects of five different sealants on the smooth buccal surfaces of premolar teeth. The study demonstrated the inadequacy of some conventional sealants to polymerise in a thin film on smooth tooth surfaces. The main reasons postulated were non polymerisation (due to oxygen inhibition) and flow (viscosity of sealant). Furthermore it was found that sealants to which acetone was added were able to polymerise in thin films due to the formation of acetone vapour, thereby preventing the ingress of oxygen.

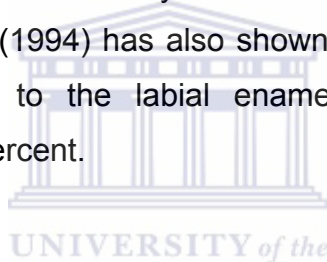
Later Ceen and Gwinnett's (1980) study confirmed Zachrisson and co-worker's (1979) findings. They showed that most of the chemically cured sealants do not effectively seal smooth enamel surfaces because of oxygen inhibition of polymerisation especially when the sealant is in contact with the air in a thin layer.

Joseph and co-workers (1992) and Joseph *et al* (1994) also found that the chemically cured sealants do not effectively seal smooth enamel surfaces, because of oxygen inhibition of polymerisation when the sealant is in contact with the air in a thin layer. Only "islands" of cured sealant were found, and these were in relation to areas of resin.

Light Cured Sealants

Ceen and Gwinnett (1981) found that light polymerised sealants protected the enamel adjacent to brackets from dissolution and subsurface lesions, whereas chemically cured sealants polymerise poorly, exhibited drift and had low resistance to abrasion.

The light-cured sealants have also proven to cure completely on smooth enamel surfaces and to prevent enamel demineralisation effectively *in-vitro* (Joseph *et al*, 1992; 1994). The authors concluded that with the "indirect" or light polymerised system, albeit it with an unfilled resin, will act as a cariostatic barrier for at least 2 years after application. A clinical trial by Banks and Richmond (1994) has also shown that the application of light-cured resin sealants to the labial enamel surface can reduce demineralisation by 13 percent.



An *in-vitro* study by Frazier *et al* (1996) showed that sealing the exposed labial surfaces of teeth already bonded with orthodontic brackets using a light-cured unfilled resin on teeth with previously placed orthodontic brackets results in a significant reduction in the incidence of enamel demineralisation. When examining for the presence or absence of any demineralisation, an eighty percent reduction was found when sealants were used.

Fluoride Releasing Sealants

Jensen *et al* (1990) found a 30 percent reduction in the depth of outer surface lesions in primary teeth with the use of fluoride-releasing sealants. On the other hand, Simonsen (1991) noted that nonfluoridated resin sealants have been effective in reducing caries, as long as they remain intact and

no leakage occurs. Adequate isolation and lack of contamination increase the retention, which has been documented to be as long as four years after placement.

Work was done by Hu and Featherstone (2005) to evaluate the efficacy of applying a light-cured filled, fluoride containing sealant, Pro Seal (Reliance Orthodontic Products, Itasca, Ill) onto the buccal tooth surfaces to prevent demineralisation. The results of their study showed that demineralisation in the Pro Seal group was significantly less than in the other groups: control, CavityShield, and light Bond Sealant Reliance (Orthodontic Products) groups. The study concluded that Pro Seal can be considered for use as a preventive method to reduce enamel demineralisation adjacent to orthodontic attachments, particularly in patients who exhibit poor compliance with oral hygiene and home fluoride use.

Soliman *et al* (2006) conducted a study to measure the rate and amount of fluoride ions released from Pro Seal over a period of 17 weeks and to determine whether the fluoride-releasing sealant had a recharging ability when fluoride ions are reintroduced into the environment. The results showed that Pro Seal released fluoride ions in sustained but significantly decreasing amounts. Furthermore it was found that Pro Seal had the ability to be recharged with fluoride ions introduced from a foaming solution of acidulated phosphate fluoride.

Salar *et al* (2007) subsequently conducted a study to examine the effect Pro Seal had on enamel demineralisation in an *in-vitro* artificial caries system. Their study showed that Pro Seal provided increased protection against demineralisation compared with a conventional sealant containing no fluoride, but it was less than that shown by a glass ionomer sealant.

Bond strength

Wang and Tarng (1991) conducted a study to compare bond strengths with and without sealants in orthodontic bonding. The results of their study found no significant differences in bond strengths between groups with unfilled bonding resin and groups without such resin. They concluded that the use of a sealant may offer extra protection to enamel from possible damage during debonding without affecting the bond strength.



AIMS AND OBJECTIVES



AIMS OF THE STUDY

The purpose of this study was to compare the efficacy of two fluoride containing materials, namely, FluorSure and Duraphat, in protecting the enamel around and underneath the orthodontic brackets against decalcification.

OBJECTIVES OF THE STUDY

The objectives of the study were to:

- Establish the microhardness of the enamel before and after any demineralisation.
- Assess the extent of white spot formation.
- To compare these data secured from teeth treated with either FluorSure or Duraphat.



MATERIALS AND METHODS



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STUDY DESIGN

This is an *in-vitro* study on extracted human premolars using microhardness testing to determine the extent of enamel demineralisation. Photographs were also taken and white spot lesions were quantified.

SAMPLING

Freshly extracted intact premolars were collected for the study and stored in distilled water with thymol crystals. Sixty teeth (n=60) were randomly selected and were divided into 3 groups of 20 each:

- Group A: (Control group); the teeth were not sealed.
- Group B: the teeth were sealed with FluorSure.
- Group C: the teeth were sealed with Duraphat.

SELECTION CRITERIA

All the teeth were visually inspected for any:

- Anatomical defects
- Carious lesions
- Restorations
- Exposed dentine
- Damaged buccal enamel surface caused by extraction process

Teeth that showed any of these were excluded from the study.



MATERIALS

Sealants

A. FluorSure (Orthotek, American Orthodontics)

According to the manufacturer FluorSure is:

- Visible light curable fluoride releasing sealant
- Contains 31% leechable sodium fluoride (NaF)
- Lightly filled with a silica/glass mixture for enhanced abrasion resistance

B. Duraphat (Colgate-Palmolive, UK)

One millilitre of Duraphat contains (according to the manufacturer):

- 50 mg of the active ingredient Sodium Fluoride equivalent to 22.6 mg of Fluoride
- Other ingredients are Ethanol, White wax (E901), Shellac (E9g4), Colophony, Mastic, Saccharin (E954) and Raspberry Essence.
- Duraphat is a brown/yellow opaque suspension.

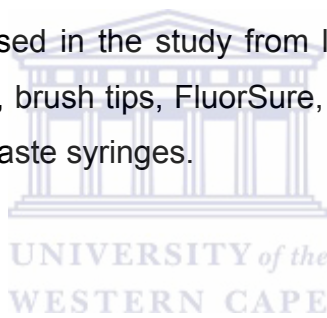
Bonding agents

A. Transbond™ XT Light Cure Orthodontic Adhesive

This bonding system comprises of the Light cure adhesive primer and the Adhesive Paste Syringes.



Figure 5 - Materials used in the study from left to right: Duraphat, brush holder with a brush tip, brush tips, FluorSure, Transbond™ XT primer and Transbond adhesive Paste syringes.



Lingual Attachments

Bondable Micro Lingual Buttons (HH Wire Company, USA) with a diameter of 3mm were used.

Demineralisation and remineralisation solutions

The demineralisation and remineralisation solutions were prepared by adjusting the method used by Hu and Featherstone (2005).

Three litres of the demineralisation solution were prepared as follows:

- Calcium Chloride – 0.8820g/l (2.0 mmol/l)

- Sodium Phosphate – 0.8577g/l (2.0 mmol/l)
- Sodium Acetate – 30.6120g/l (75 mmol/l)

The pH was adjusted with acetic acid to 4.3 at 37°C.

Three litres of remineralisation solution were prepared as follows:

- Calcium Chloride – 0.6615g/l (1.5-mmol/l)
- Sodium Phosphate – 0.3834g/l (0.9-mmol/l)
- Potassium Chloride – 33.5520g/l (150-mmol/l)
- Sodium Cacodylate – 12.8400 (20- mmol/l)

The pH was adjusted with Sodium Hydroxide and Hydrochloride acids to a pH of 7.0 at 37°C.



METHODOLOGY

All teeth were mounted in PVC pipes. They were then ground in preparation for setting the baseline for the microhardness. After baseline microhardness was determined, lingual attachments were bonded. No sealants were applied in group A. In group B FluorSure was applied on the buccal surface before bonding while in group C Duraphat was applied after bonding. The teeth were subjected to 14 days of demineralisation and remineralisation pH cycling process. All specimens were brushed once daily with an electric toothbrush. After day 14 they were debonded and cleaned. Microhardness was recorded in tabular form.

Mounting procedure

The teeth were prepared by initially debriding the soft tissue remnants. The teeth were then cleaned and polished using prophylactic rubber cups

with pumice slurry and then were thoroughly rinsed and dried. The roots of the teeth were sectioned below the cemento-enamel junction using a diamond disc and discarded.

The specimen crowns were then prepared using the procedure used by Amra et al (2007). They were embedded in self-curing acrylic resin (Orthocryl, Dentaaurum) in PVC pipes with dimensions of 15 mm high, 20 mm outer diameter, and a wall thickness of 2 mm. The mid-buccal surfaces of the teeth, the areas ear-marked for the bonding of the orthodontic bracket, were aligned parallel to the outer rim of the PVC pipe by supporting the specimens with periphery wax. Care was taken to ensure that the tooth surface projected above the rim of the pipe. The PVC pipe and periphery wax supported crowns were then placed on a glass surface and the chemically cured acrylic resin poured around the tooth specimen (Fig. 6).

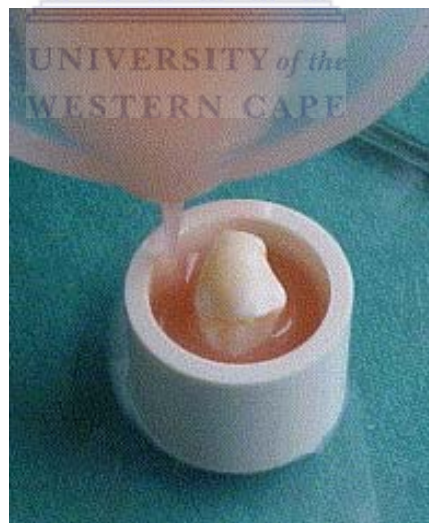


Figure 6 - Chemically cured acrylic resin was incrementally poured around tooth specimen.

This procedure was carried out incrementally until the teeth become embedded in the resin, whilst at the same time making sure that no resin contaminated the buccal enamel surface. The specimens were then allowed to stand until complete polymerisation of the resin had occurred.

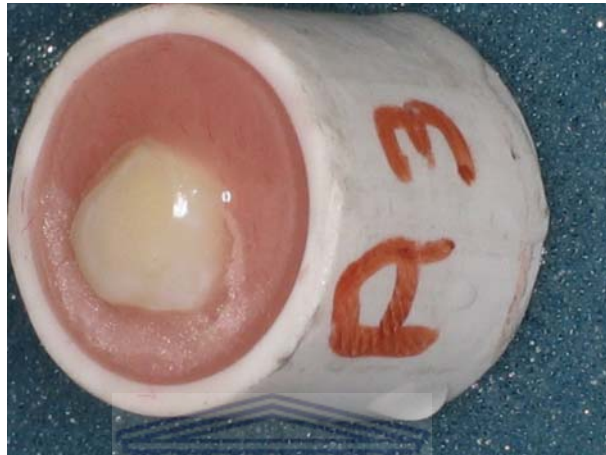


Figure 7 - Embedded tooth, note that the tooth surface projects above the rim of the pipe.

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After embedding, the enamel surfaces of all the teeth were ground using the Metaserv Universal Polisher (Surrey, England). They were ground under running water first with an 800 grade carborundum paper to flatten the enamel buccal surface. They were then polished to a smooth surface with a 1000 grade carborundum paper (Fig. 8 below).



Figure 8 - Metaserv Universal Polisher (Surrey, England).

Enamel microhardness tests for baseline values

The centre of each of the ground buccal enamel surfaces was marked with a small 0.9 μm fine grit round diamond bur. This was used as a reference point from which the different areas for indentations could be easily identified under the microscope.

The enamel microhardness tests were carried out using a Zwick/Roell hardness tester (Indentec, Germany) with a load of 300g applied for 15 seconds. The indenter was always advanced perpendicular to the enamel surface (at primary magnification of 40 times).

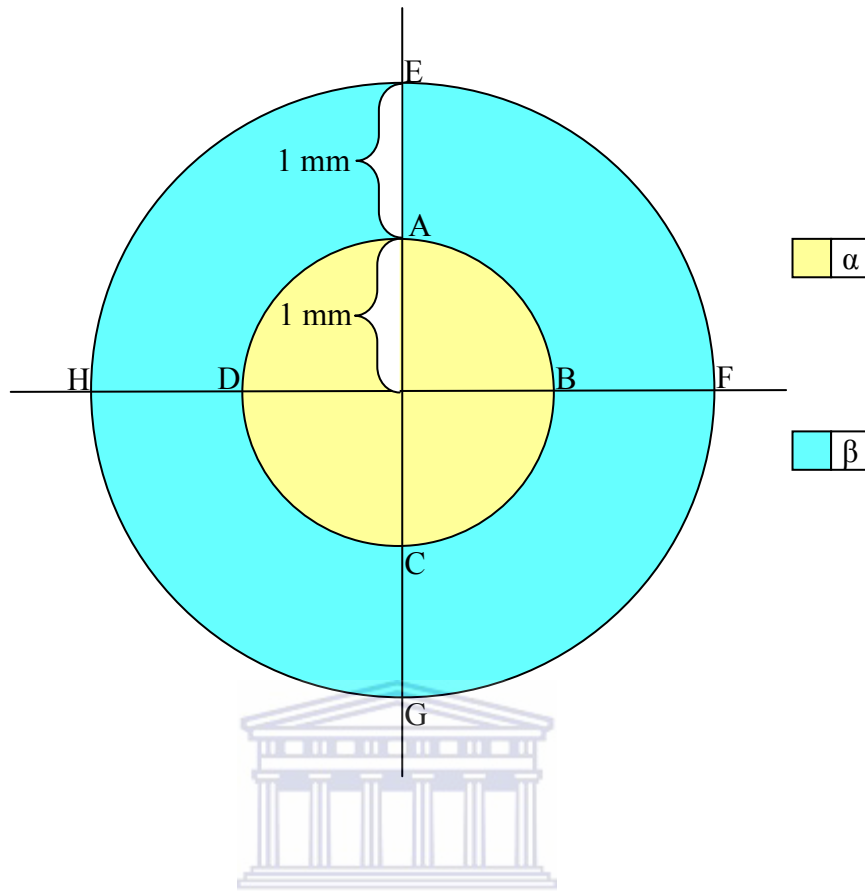


Figure 9 - A diagram of the buccal enamel surface with 8 areas where indentations were done.

Eight indentations were done across the enamel surfaces as indicated above (Fig. 9). The occlusal margin was always placed in the uppermost plane (12 o'clock position). Area α represents areas under the bracket (A; B; C and D) while area β represents areas (E; F; G and H) away from the bracket. The enamel microhardness values were recorded in a tabular form.

The specimens were then stored in distilled water until the bonding stage.



Figure 10 - Zwick/Roell hardness tester (Indentec, Germany).

Etching

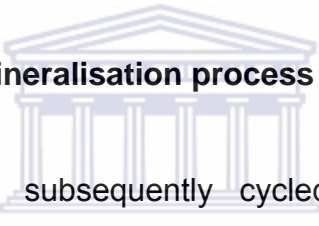
All the specimens were etched with 37 percent phosphoric acid gel which was carefully applied to the ground enamel surface of each tooth for 30 seconds. In Group A and C only the area of bonding of the button was etched; however in Group B the entire surface was etched (in group B FluorSure was to be applied over the entire buccal surface, as recommended by the manufacturer). They were then rinsed with a water spray for 20 seconds and dried with oil-free air until the etched enamel surface appeared chalky white.

Bonding procedure

In group B, FluorSure was applied to the entire buccal surface. A brush was used to ensure that the application was of a uniform thickness and the sealant was then exposed to a curing light for 15 seconds. In all the Groups, the Transbond™ XT bonding system was used for attaching the buttons, as per the manufacturer's instructions. Excess bonding material was removed using a small scaler and the adhesive cured for 20 seconds with a curing light at close range.

The specimens were then transferred to the distilled water for 24 hours.

Demineralisation/remineralisation process



The specimens were subsequently cycled through a demineralisation/remineralisation process. This involved immersing each group of the specimens in 300ml of demineralisation solution in an incubator (Lasec, RSA) for 6 hours at 37°C. After removal from the demineralisation solution, the specimens were rinsed with distilled water for few seconds, brushed for 5 seconds with an electric toothbrush (Oral-B soft bristle two head electric toothbrush, Oral B Laboratories, Germany) with no dentifrice. The solutions were changed every third day. After the brushing the specimens were immersed in 300ml of remineralisation solution and placed in an incubator at 37°C overnight.

This procedure was repeated on each specimen daily for 14 days.



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Figure 11 - Lasec Incubator used to keep the temperature at 37°.

Decalcification

After the brushing procedure, the specimens were viewed under a stereomicroscope (Nikon, Japan) at magnification of 50 times, before they were placed in the remineralising solution. Photographs were taken after day 5, 8, 11 and 14 with a Leica camera (Leica DFC 290 Microsystems, Germany) fitted onto the stereomicroscope. The ACDsee photo editing programme was used to transfer the photographs to a computer.



Figure 12 - Leica Camera fitted onto the stereomicroscope.



Post demineralisation microhardness test

After the demineralisation/remineralisation cycle, debonding was carried out according to the method described by Årtun and Bergland (1984), using double bladed debonding pliers (figure 13) and remnant cement was carefully removed with a scalpel blade. The specimens were then viewed under the microscope to ascertain that all the cement was removed and no visible enamel damage was present.

Enamel microhardness tests were carried out the same way as previously described.



Figure 13 - A double bladed debonding pliers and a scalpel blade.



Figure 14 - Enamel surface after the buttons were removed and cement cleaned with a scalpel blade.

EXAMINER VARIABILITY

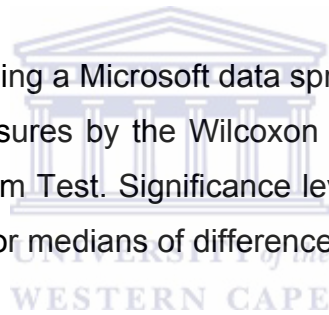
Scoring of the photographs was tested for intra-observer variability to ascertain whether the initial values obtained were repeatable and they were tested in order to ensure that bias was avoided in obtaining the values.

After each day of scoring of photographs for white spot lesions five (25%) photographs were randomly selected from each group using Microsoft Office Excel and re-scored by the researcher.

STATISTICAL ANALYSIS

Descriptive statistics including the mean, standard deviation, median and range were calculated for each of the three groups. The analysis of variance (ANOVA) was used to determine whether there were significant differences in the scoring of the white spot lesions. The level of significance was determined at the $p < 0.05$ level of confidence.

Data were captured using a Microsoft data spreadsheet and then analysed using descriptive measures by the Wilcoxon Signed Rank Sum Test and the Wilcoxon Rank Sum Test. Significance level was set at 5 percent and samples were tested for medians of differences and InterQuartile Range.



RESULTS



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The results of the study are presented in the form of tables, graphs and photographs. The table below illustrates the study sample.

Table 1 - Study sample.

Groups	Excluded teeth	Test Sample
Group A (20)	4,10, 12	17
Group B (20)	5, 7, 17	17
Group C (20)	1, 10	18

In all three groups there were teeth that were excluded due to exposed dentine or enamel cracks (Table 1).



DECALCIFICATION

Photographs were taken after days 5, 8, 11 and 14 and the white spot formation in all the groups were quantified and recorded in tabular form. It is important to note that every tooth, control and experimental, demonstrated some level of enamel demineralisation. The control group showed complete demineralisation in the areas around the bracket. Visible white spot formations were seen after the first day of demineralisation in the control (Group A) and FluorSure groups (Fig. 15 and 16). After the last day in the control group (Group A) almost all the specimens were chalky white (figure 17). The experiment groups also showed varied levels of white spot formation. The mean decalcification in the FluorSure group was 67.66 percent and ranged from 23.1 percent to 100 percent. The mean decalcification for Duraphat group was 63.95percent and ranged from 43 percent to 98 percent at the end of the brushing process. After debonding an observation was made of some specimens in the control and the experiment groups showing some white spots under the lingual buttons. Most speci-

mens in the experiment groups exhibited demineralisation at the occlusal margins of sealed teeth (Fig 15 and 16).

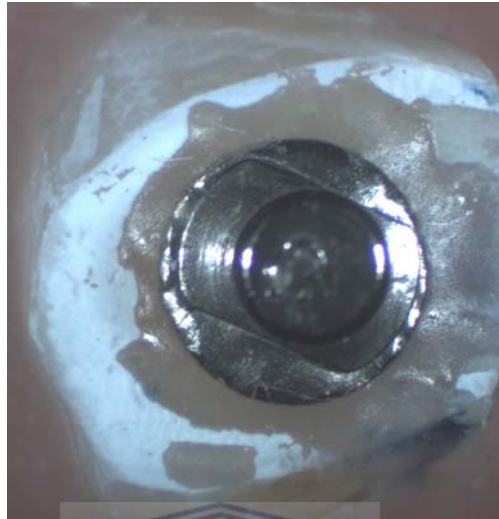


Figure 15 - Group A; photograph after day one of the cycling process.

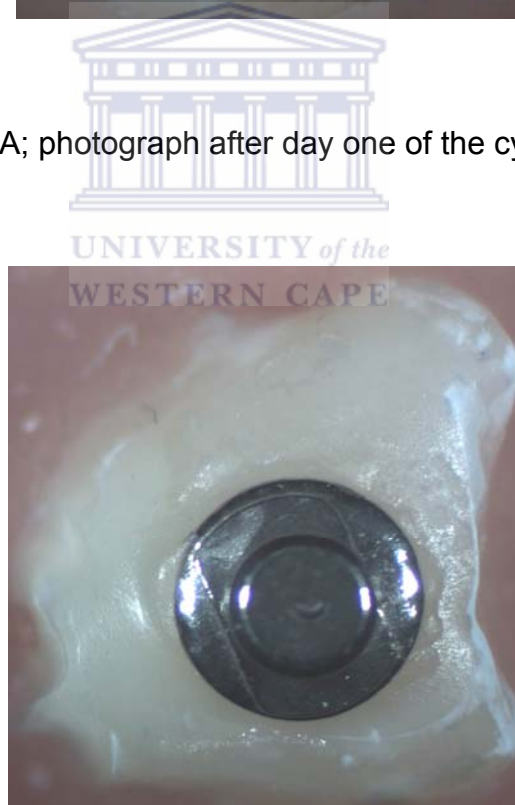


Figure 16 - Group B; photograph after day one of the demineralisation/remineralisation cycle.

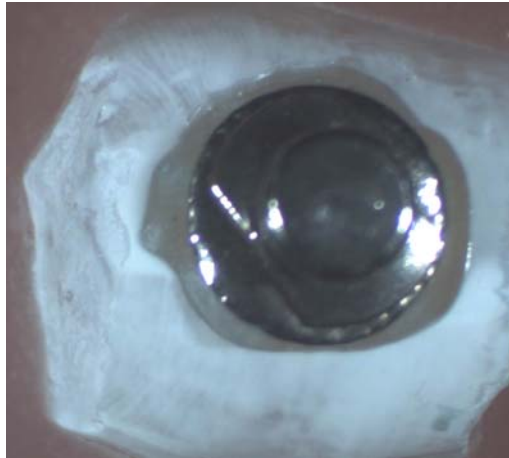


Figure 17 - Group A; photograph after day 14, showing chalky white enamel that was a common feature in the control group.

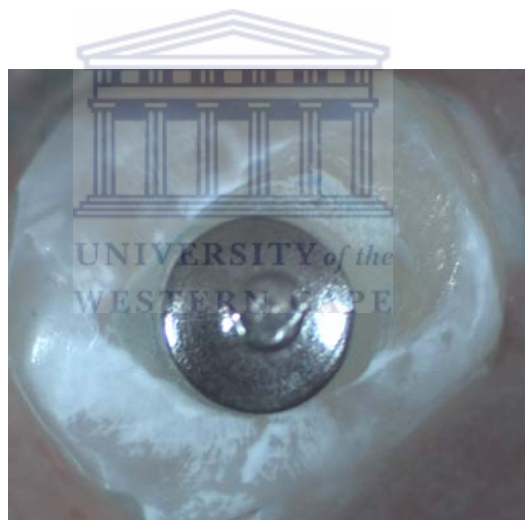


Figure 18 - Group B; photograph after day 14, showing white spots in the FluorSure group.

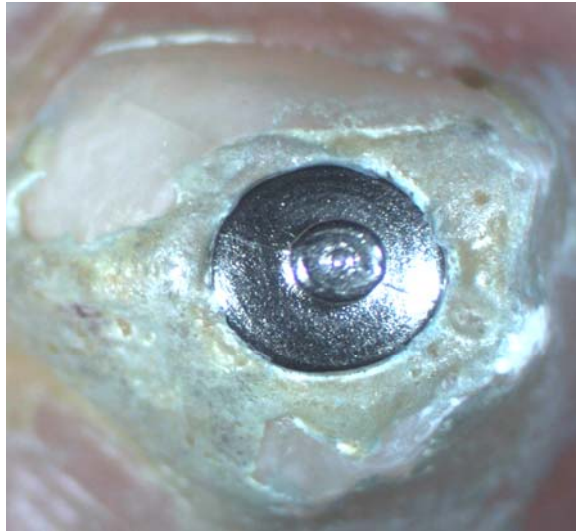


Figure 19 - Group C; photograph after 14 days showing some level of decalcification Duraphat

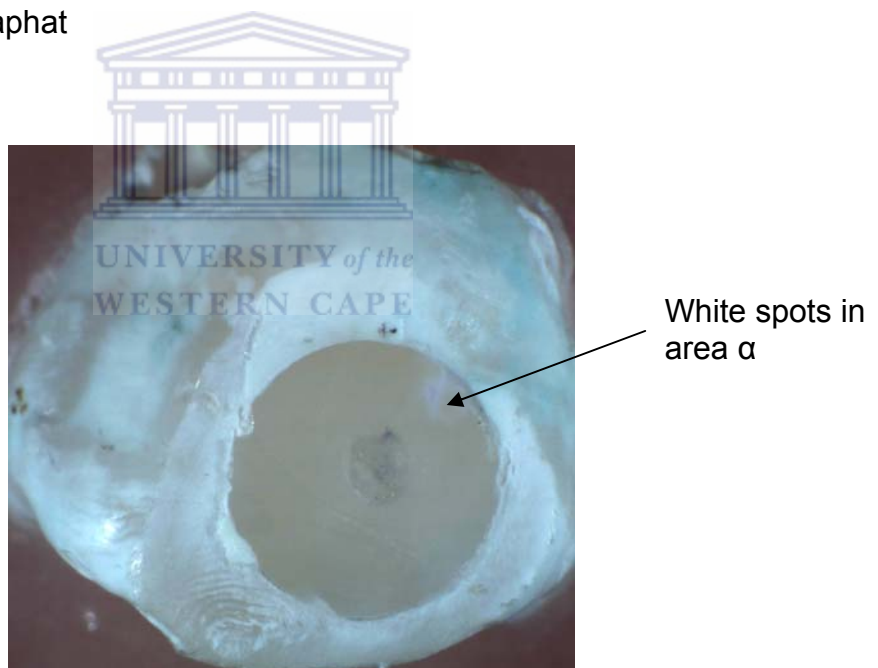


Figure 20 - Group A; photograph after debonding showing white spots in area α .

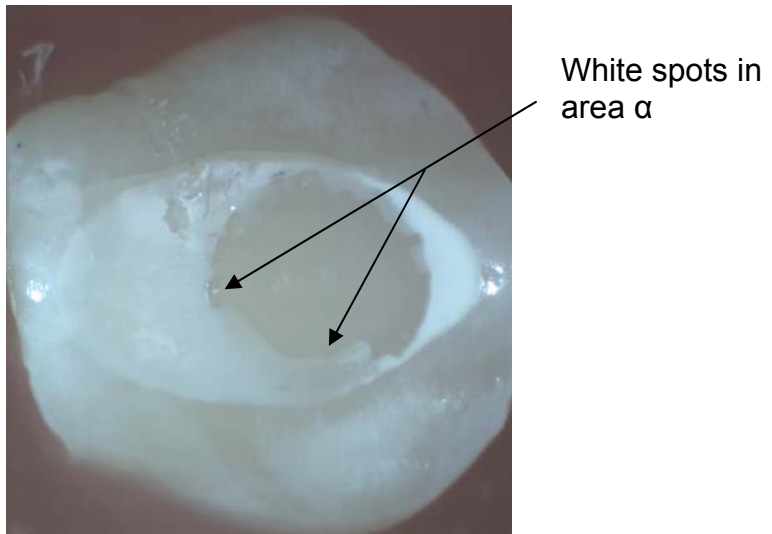


Figure 21 - Group B; photograph after debonding showing white spots in area α

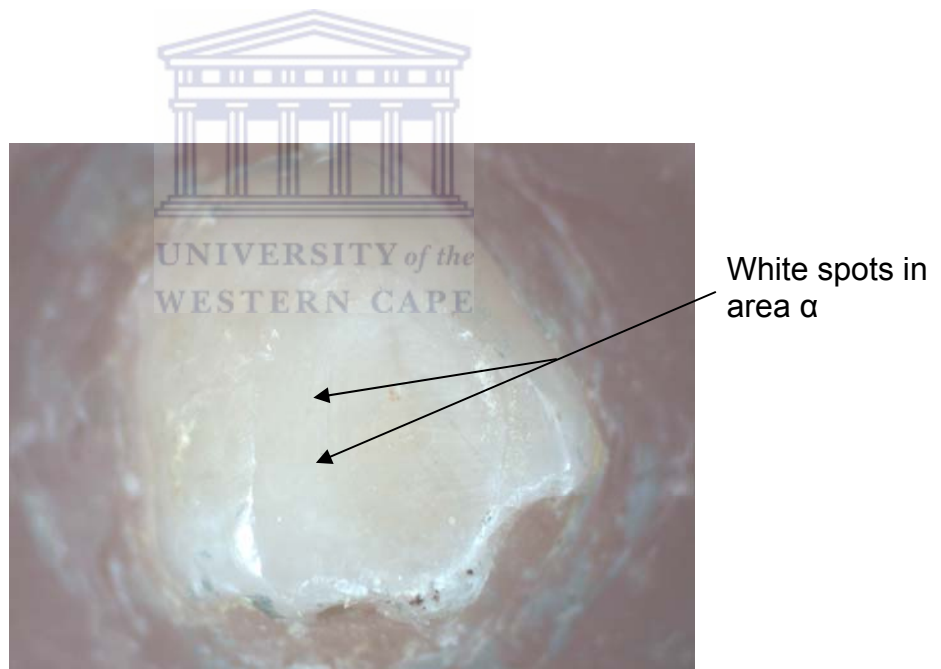


Figure 22 - Group C; photograph after debonding showing white spots in area α .

The tables below illustrate the comparison of white spot formation between the three groups.

Table 2 - FluorSure compared with the control.

Average white spot formation	Day 5	Day 8	Day 11	Day 14
Control %	68.49	75.04	81.94	88.50
FluorSure %	14.95	32.99	51.11	67.66
Significance	0.00	0.00	0.00	0.00
P Value	**	**	**	**

NS Not Statistically significant

* Statistically Significant: $P < 0.05$

** Statistically Significant $P < 0.01$

When comparing the FluorSure group with the control group, there was a highly significant difference in the amount of white spot formation between the two groups for all the days.

Table 3 - Duraphat compared with the control.

Average white spot formation	Day 5	Day 8	Day 11	Day 14
Control %	68.49	75.04	81.94	88.50
Duraphat %	11.40	39.75	53.05	63.95
Significance	0.00	0.00	0.00	0.00
P Value	**	**	**	**

NS Not Statistically significant

* Statistically Significant: $P < 0.05$

** Statistically Significant $P < 0.01$

When Duraphat was compared with the control group there was a highly significant difference in the amount of white spot formation. By day 14, the control group showed 88.5 percent of the examined area with white spots whereas in the Duraphat group there was only 63.95 percent white spot formation.

Table 4 - Duraphat compared with FluorSure.

Average white spot formation %	Day 5	Day 8	Day 11	Day 14
FluorSure %	14.95	32.99	51.11	67.66
Duraphat %	11.40	39.75	53.05	63.95
Significance	0.07	0.15	0.40	0.29
P Value	NS	NS	NS	NS

NS Not Statistically significant

* Statistically Significant: $P < 0.05$

** Statistically Significant $P < 0.01$

When FluorSure and Duraphat were compared, there was no statistically significant difference in the amount of white spots formed in the two groups for all the days.

The graph below (Fig. 23) illustrates the amount of white spot formation during the pH cycling process in all the groups

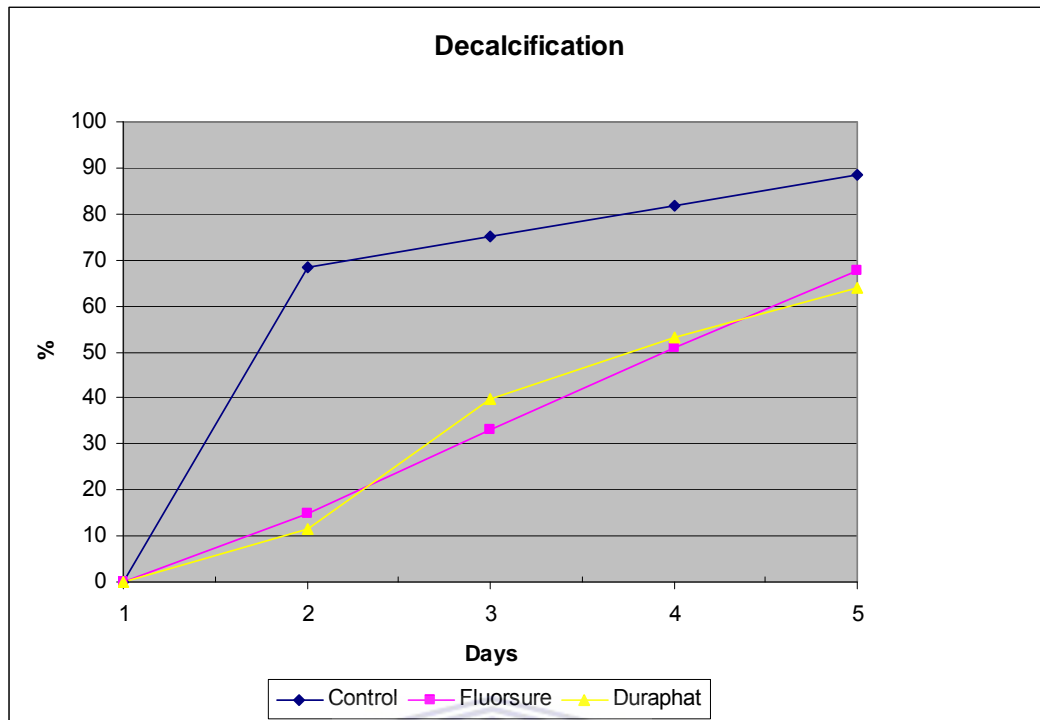


Figure 23 - Graph showing decalcification in the three groups. At the end the 14 days, the control group was 88% while FluorSure and Duraphat were 67.66% and 63.95% respectively.

The graph shows that there was rapid white spot formation in the control group while in the experiment groups the amount of white spot formation was steady.

DEMINERALISATION

The enamel demineralisation was determined using enamel microhardness. The graphs below illustrate the amount of demineralisation recorded in Vicker's hardness before and after the pH cycling.

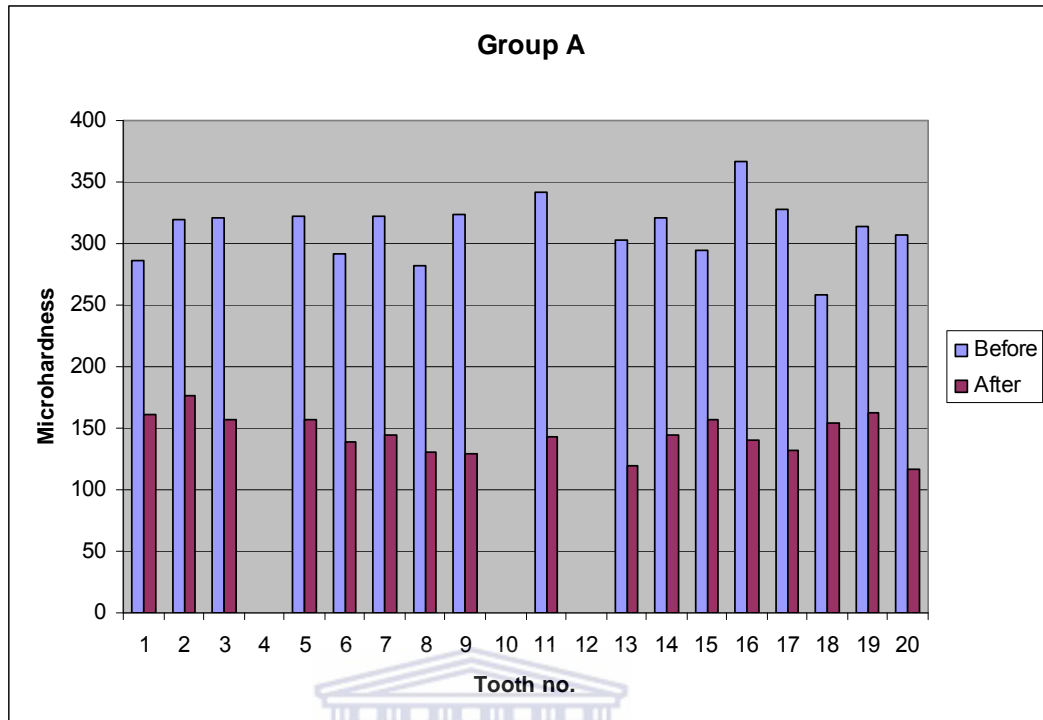


Figure 24 - Group A; Enamel microhardness before and after the demineralisation/remineralisation cycling process.

The graph (Fig. 24) above shows the enamel was highly demineralised in the control group (Group A). However there was a difference in the demineralisation in areas under the buttons, enamel microhardness values in area α (area under the bracket represented by areas A, B, C and D) and area β (area around the bracket, represented by areas E, F, G and H) after the cycling process showed that the areas around the bracket were highly demineralised (Fig. 27).

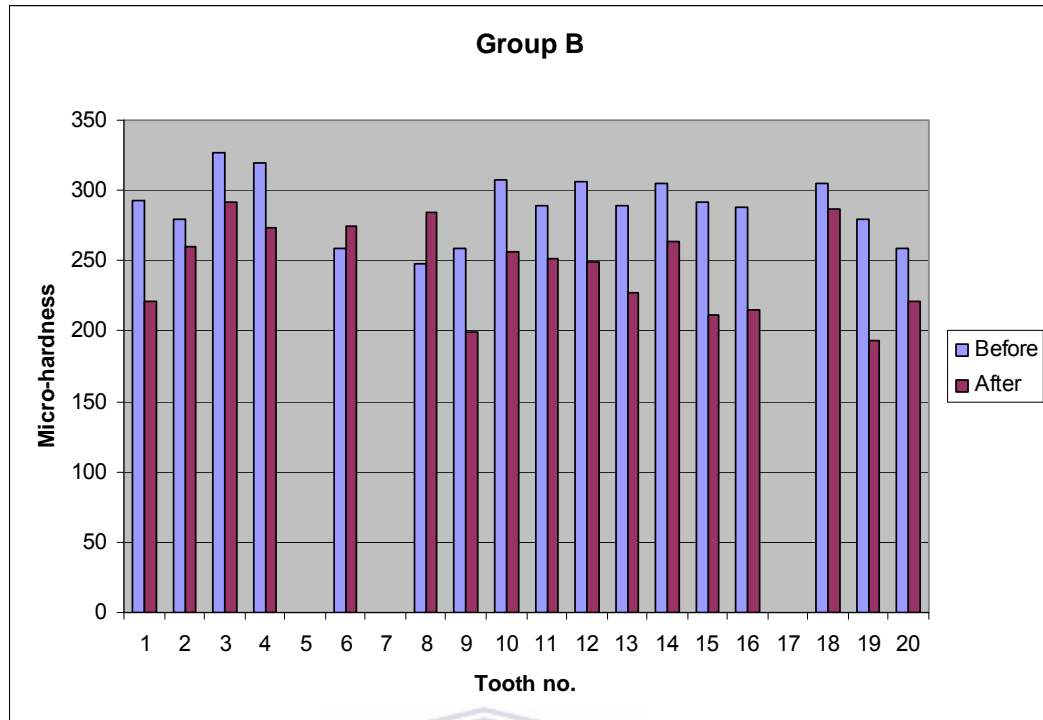
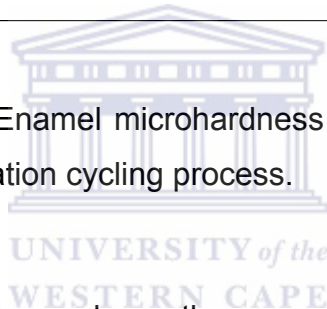


Figure 25 - Group B; Enamel microhardness before and after the demineralisation/remineralisation cycling process.



The graph (Fig. 25) above shows the enamel was less demineralised in the FluorSure group (Group B). There was also no difference in the enamel microhardness in areas under the buttons and the areas around them (Fig. 28). Figure 28 shows that in both areas α and β , the enamel hardness did not change significantly after the cycling process thus the enamel around the lingual buttons was not significantly softer after pH cycling process.

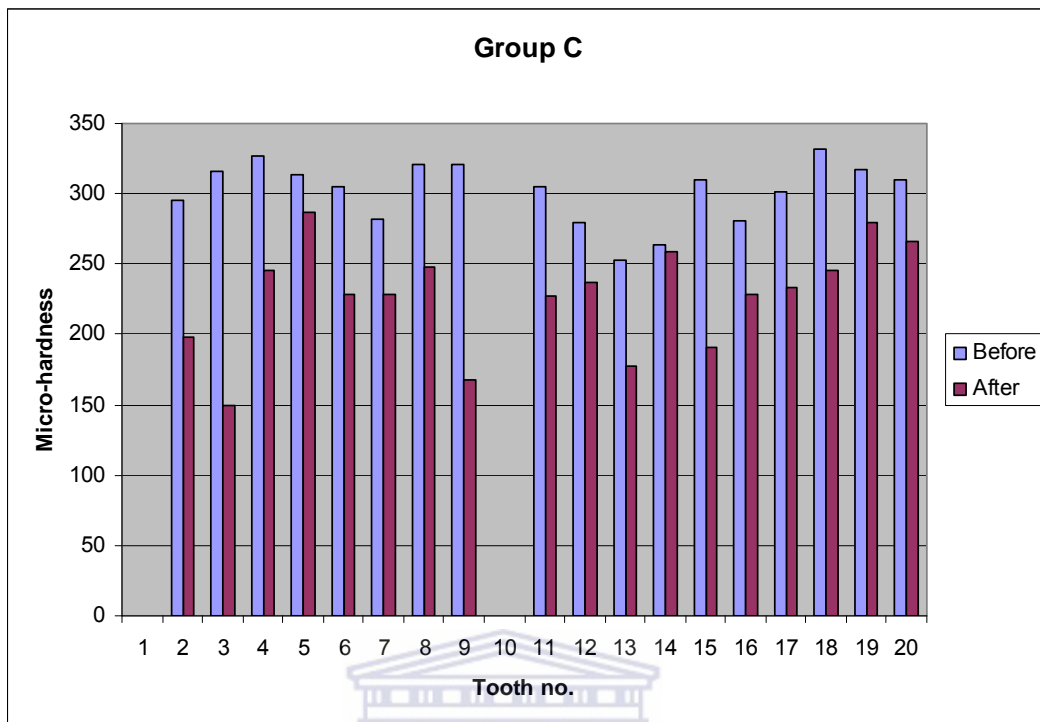


Figure 26 - Group C; Enamel microhardness before and after the demineralisation/remineralisation cycling process

The graph (Fig. 26) above shows the enamel was demineralised in the Duraphat group (Group C). There was an increase in enamel demineralisation around the buttons as can be seen in figure 29. This graph shows that in area α , the enamel hardness did not change significantly after the cycling process though there were areas under the lingual attachment which showed highly significant demineralisation. While area β showed a highly significant change thus the enamel was much softer around the buttons.

Comparison of demineralisation in the three groups

The tables below show comparison of enamel microhardness among the three groups.

Table 5 - FluorSure compared with the control

Changes in Enamel Microhardness Values	Area α	Area β	Entire Tooth
Control	-33.15	-300.74	-157.60
FluorSure	-38.00	-46.46	-42.51
Significance	0.37	0.00	0.00
P Values	NS	**	**

NS Not Statistically significant

* Statistically Significant: $P < 0.05$

** Statistically Significant $P < 0.01$

The table above shows that when enamel microhardness in the FluorSure groups and the control groups were compared, there was no statistical significant difference between the two groups in area α (Table 5). While the area β and the entire tooth surface showed highly significant differences between the two groups.

Table 6 - Duraphat compared with the control

Changes in Enamel Microhardness Values	Area α	Area β	Entire Tooth
Control	-33.15	-300.74	-157.60
Duraphat	-44.60	-103.91	-74.27
Significance	0.37	0.00	0.00
P Values	NS	**	**

NS Not Statistically significant

* Statistically Significant: $P < 0.05$

** Statistically Significant $P < 0.01$

When comparing the Duraphat group to the control group, there were no statistical differences in area α but there were highly significant differences in area β (Table 6).

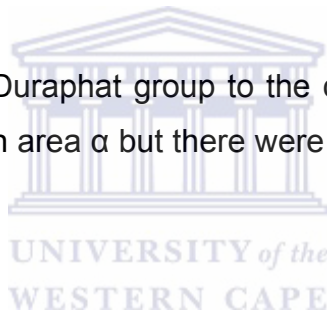


Table 7 - Duraphat compared with FluorSure

Changes in Enamel Microhardness Values	Area α	Area β	Entire Tooth
FluorSure	-38.00	-46.46	-42.51
Duraphat	-44.60	-103.91	-74.27
Significance	0.64	0.00	0.02
P Values	NS	**	**

NS Not Statistically significant

* Statistically Significant: $P < 0.05$

** Statistically Significant $P < 0.01$

When comparing the FluorSure group to the Duraphat group (Table 7), there were highly statistical significant differences in area β between the two groups. The entire tooth surface showed significant differences between the two groups while area α showed no statistical differences between the two groups.



DISCUSSION



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The application of a polymer coating, a fissure sealant, or a light-cured resin sealant to the labial enamel surface has been recommended to prevent decalcification of the enamel around the bonded bracket (Joseph *et al*, 1992; Banks and Richmond, 1994; Frazier *et al*, 1996; Gillgrass *et al*, 2001). Long-term sealing of enamel with sealant resin before bracket bonding does not require a patient's compliance to prevent or interrupt demineralisation related to orthodontic treatment. The duration of protection is influenced by the thickness and abrasion resistance of the sealant (Hu and Featherstone, 2005).

The present study was undertaken to compare the efficacy of FluorSure with that of Duraphat in protecting the enamel around and underneath the brackets against decalcification. These two materials are applied by an operator and do not require patient compliance. According to Wenderoth *et al*, (1999) a method to protect the susceptible area beneath and adjacent to bonded attachments, independent of patient compliance, would be extremely beneficial.

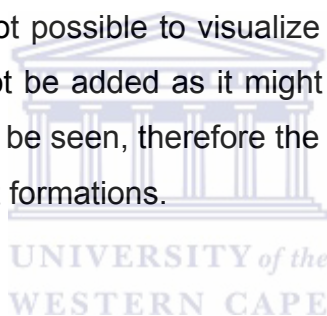
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The choice of techniques for the assessment of demineralisation and remineralisation depends strongly upon study protocols and laboratory capabilities (White *et al*, 1992). Microhardness indentation measurements have been used to determine demineralisation and remineralisation effects since the first *in situ* studies of Koulourides in 1966 (Arends and ten Bosch, 1992). In this method, a Knoop or Vickers diamond is positioned on the sample with a given load for a given time. The indentation length left by the diamond in the sample is determined microscopically in μm .

Enamel microhardness was used as the instrument to determine the amount of demineralisation. In group B the teeth were coated with Fluor-Sure while teeth in Group C were coated with Duraphat. The results of this *in-vitro* study showed that enamel under the brackets was not demineral-

ised in all three groups. However, in the control group the enamel around the bracket was completely demineralised. Both the FluorSure and Duraphat groups showed less demineralisation around the brackets which meant that Duraphat and FluorSure did protect the enamel around the bracket (Fig 27 and 29). However in spite of the positive results neither FluorSure nor Duraphat completely prevented enamel demineralisation. This is in agreement with a study by Hu and Featherstone (2005) who found that teeth treated with Pro Seal and fluoride varnish had the least amount of demineralisation, using a similar research method.

This study further aimed at determining the abrasion resistance of these sealants. However, quantifying FluorSure proved to be difficult during the pilot study as it was not possible to visualize FluorSure due to its clear in colour. A dye could not be added as it might have affected its properties. Only white spots could be seen, therefore the study was modified by quantifying these white spot formations.



The teeth were subjected to 14 days of tooth brushing to simulate mechanical wear in the oral environment. The use of tooth brushing is recommended in studies assessing wear of restorative materials (Hotta and Hirukawa, 1994; Donly *et al*, 1997; Todd *et al*, 1999; Gillgrass *et al*, 2001; Hu and Featherstone, 2005). Todd *et al* (1999) used manual tooth brushing twice daily without toothpaste for 37 days, while in the study by Gillgrass *et al* (2001), an Oral B electric toothbrush with a non-fluoridated toothpaste was used for the equivalent of 2 months toothbrushing. In a study by Hu and Featherstone (2005), a piston-action brushing machine with Oral B toothbrush and nonfluoridated toothpaste was used 3 times a day to simulate abrasion by everyday toothbrushing. In this study an Oral B electric toothbrush was used without toothpaste.

The control group showed white spot formations from the first day and this was not unexpected as this group was not protected. This finding conforms to the prevalence of white spot lesions in patients who are on orthodontic treatment reported to be in the range of 50-96 percent (Geiger *et al*, 1992; Geiger *et al* 1988; Øgaard *et al*, 1996). O'Reilly and Featherstone (1987) showed that white spot lesions develop in as little as one month after the placement of orthodontic appliances.

There was no statistical significant difference in the percentage of white spots formation between the teeth treated by FluorSure and by Duraphat (Table 4). The mean white spot formation in the FluorSure and Duraphat samples were 14.9 percent and 11.40 percent at the end of the fifth day of brushing and 67.66 percent and 63.95 percent respectively at the end of day 14 of brushing.

The physical barrier protection from a fluoride varnish is temporary as this material is easily abraded away during typical toothbrushing (Demito *et al*, 2004). Todd *et al* (1999) stated that once the varnish starts to break off through mechanical brushing, some enamel would be exposed. However a very high concentration of fluoride is still present in the remaining fluoridated varnish adjacent to these exposed enamel areas (Todd *et al*, 1999) thus offering protection to the enamel. According to Demito *et al* (2004), the protection from the fluoride which is incorporated into the surface of the enamel from the varnish, also diminishes with time and as a result, re-application of this material is recommended at least every 3 months (Øgaard *et al*, 1996).

A potential criticism of this study is that demineralisation could be found at the occlusal edges of some sealed teeth. These results concur with findings of Frazier *et al* (1996) who also found demineralisation at the incisal edge of sealed teeth. As in the study by Frazier *et al* (1996) it was impos-

sible to determine whether these areas of demineralisation represented sealant failure, breakdown of the varnish coating, or failure of the investigator to carry the varnish over the sealant margin. Furthermore, studies on incidence and location of demineralisation with orthodontic treatment would suggest incisal and occlusal edges to be at very low risk for demineralisation *in vivo* (Gorelick *et al*, 1982; Mizrahi, 1982; Øgaard, 1989).

Cycling between periods of demineralisation (caries solution) and remineralisation (artificial saliva solution) during this experiment was intended to simulate a clinical situation. Within the normal oral environment, there are periods of higher caries challenge; dependent upon the eating habits of each person. Demineralisation occurs when the pH of the mouth becomes more acidic but there is also subsequent remineralisation during the longer periods of exposure to saliva during the rest of the day.

FluorSure did not confer significant protection against decalcification during orthodontic treatment contrary to the manufacturer's claim. These findings are similar to the results of a study by Banks and Richmond (1994) of a sealant used around fixed appliances during orthodontic therapy, as they found it not significantly (13 percent) affecting the prevalence of decalcification compared with untreated teeth. They also reported a high incidence of decalcification (75 percent).

CLINICAL IMPLICATIONS



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Due to the busy pace of an average orthodontic practice, some orthodontists feel that applying a fluoride varnish is cumbersome and takes too long (Boyles, 2007). However this study has shown that FluorSure, a fluoride releasing sealant does have an advantage in the prevention of decalcification when applied under and around orthodontic brackets. An extra step and costs are added to the conventional bonding. However fluoridated sealants can be used during orthodontic treatment to cover the enamel surface therefore providing a protective barrier against caries causing bacteria. FluorSure is clear in colour when applied to the tooth thus it will be aesthetically acceptable to the patient.

Duraphat on the other hand is brownish in colour and it has a thick film which makes it aesthetically not appealing to the patient. The application Duraphat has three vital steps; (1) manual toothbrushing to remove surface plaque, (2) drying of the teeth, and (3) varnish application. An average of 5 minutes is needed to perform this service and it needs to be done every at least 4 months (Boyles, 2007). This can be time consuming and can be costly to the parents or the patient especially if a visit to a dentist is required every 3 to 4 months. Patients are instructed to avoid eating for 2 to 4 hours after application and to refrain from brushing the teeth the night of the application. Contact allergies have also been reported for Duraphat varnish (Isaksson *et al*, 1993 cited by Schmit *et al*, 2002), a reaction to the colophony component.

CONCLUSIONS



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In conclusion, sealant treatment results in a significant reduction of enamel demineralisation *in-vitro*. Such light cured resins can effectively seal large areas of smooth enamel surface after orthodontic bracket placement in those patients demonstrating poor oral hygiene (Frazier *et al*, 1996). Light cured sealant prevention holds promise for use in orthodontics and further investigations are warranted. More especially studies to investigate if the sealant can be used without a primer or conditioner. This will avoid an extra step in the bonding of orthodontic brackets.

Sealant use has a major advantage over more commonly used prevention modalities, that being, patient compliance is eliminated as a variable in its overall success or failure (Frazier *et al*, 1996). The following conclusions were made:

- There was a significant difference between the FluorSure treated teeth and teeth in the control group with respect to demineralisation.
- There was no significant difference between the FluorSure and Duraphat treated teeth with respect to demineralisation. Therefore either FluorSure or Duraphat has clinical benefits.

RECOMMENDATIONS



Based on the findings of this study, further *in-vitro* and clinical studies could be performed to determine the amount of fluoride released and the duration of fluoride release from FluorSure when applied under and around the bonded orthodontic bracket. Another *in-vitro* study could be performed to determine if FluorSure could be used without being followed by a primer or conditioner. This can benefit the practitioner in reducing or minimising the number of steps in bonding whilst at the same time adding protection against decalcification or white spot formation.



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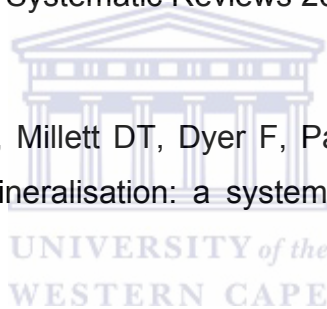
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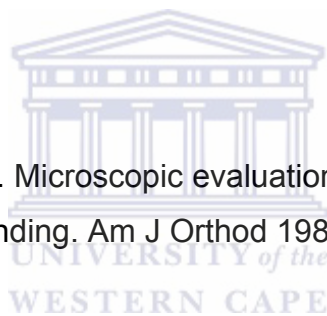
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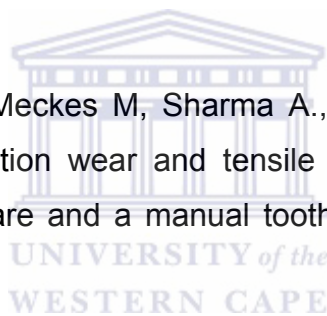
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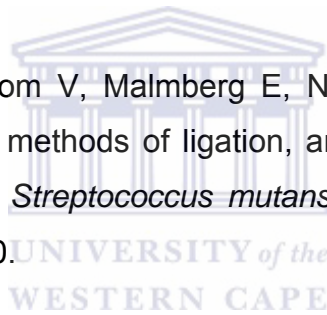
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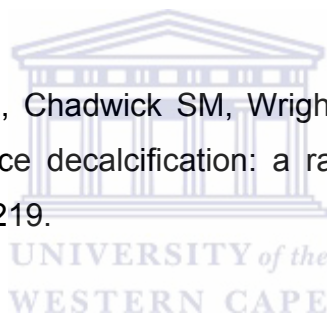
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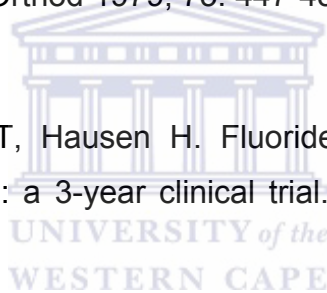
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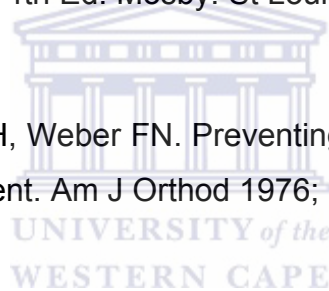
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APPENDIX



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A. DECALCIFICATION

Table 8 - Group A: Table recording incidence of white spots formation

No	Day 0	Day 5	Day 8	Day 11	Day 14
1	0	53.6	61.5	62.3	78.1
2	0	69.5	73.4	88.1	96.9
3	0	72.8	91.0	96.6	96.0
4	0	55.8	60.4	65.8	61.7
5	0	68.9	73.2	69.8	71.0
6	0	84.1	84.9	87.2	92.2
7	0	77.8	80.1	83.6	83.0
8	0	70.0	76.3	94.3	98.6
9	0	77.8	93.3	97.4	100.0
10	0	86.6	89.0	93.2	88.3
11	0	63.5	65.1	70.0	76.9
12	0	56.2	61.1	79.0	93.1
13	0	68.5	73.9	75.0	94.0
14	0	72.2	89.1	91.0	95.3
15	0	87.7	87.9	95.0	94.7
16	0	92.1	95.8	99.0	95.2
17	0	57.6	53.0	57.9	77.2
18	0	24.4	47.5	56.1	79.2
19	0	88.2	91.0	98.4	100.0
20	0	42.6	53.2	79.1	98.6

Table 9 - Group B: Table recording incidence of white spots formation

1	0	7.9	10.6	18.6	23.1
2	0	8.4	15.2	26.2	28.3
3	0	7.7	81.5	88.2	95.6
4	0	7.5	12.3	20.4	55.1
5	0	16.0	19.9	80.7	85.5
6	0	13.8	19.0	25.2	30.7
7	0	11.4	17.5	19.7	48.3
8	0	14.6	25.0	40.5	70.2
9	0	22.2	19.0	24.0	30.5
10	0	15.0	38.0	57.2	55.1
11	0	12.3	60.0	52.0	86.6
12	0	26.7	39.7	80.5	92.0
13	0	7.3	12.5	21.9	44.6
14	0	19.7	29.5	90.2	96.0
15	0	13.1	91.1	92.8	100.0
16	0	8.8	22.9	37.8	75.0
17	0	8.8	25.0	44.5	66.0
18	0	35.7	40.0	42.1	90.0
19	0	12.0	32.0	73.0	93.4
20	0	30.0	49.0	86.7	87.1

Table 10 - Group C: Table recording incidence of white spots formation

No	Day 0	Day 5	Day 8	Day 11	Day 14
1	0	17.3	64.1	65.0	83.0
2	0	6.0	22.0	44.6	60.0
3	0	20.2	80.3	89.2	98.0
4	0	11.7	40.7	50.5	55.0
5	0	9.5	23.0	33.1	50.3
6	0	5.6	20.0	25.6	60.0
7	0	13.4	33.4	46.8	75.0
8	0	5.0	27.2	38.5	55.0
9	0	5.7	27.0	55.2	55.6
10	0	11.9	55.0	62.3	70.0
11	0	6.3	42.0	53.0	60.0
12	0	8.4	26.0	42.0	53.0
13	0	11.6	51.0	55.1	57.0
14	0	22.5	56.0	67.6	70.0
15	0	27.3	62.0	89.5	96.0
16	0	4.8	17.1	26.2	43.0
17	0	15.0	43.0	54.8	70.0
18	0	9.8	40.0	60.0	60.0
19	0	9.6	26.2	53.0	55.0
20	0	6.4	38.9	49.0	53.0

B. ENAMEL MICROHARDNESS

Table 11 - Group A; Enamel microhardness before and after the pH cycling

	No	Before							
		A	B	C	D	E	F	G	H
	1	249	337	228	321	229	348	241	331
	2	287	302	315	289	302	396	321	341
	3	307	297	346	331	249	311	370	360
	5	219	323	348	366	323	391	315	298
	6	289	201	286	325	289	302	325	312
	7	331	315	276	323	336	373	263	364
	8	249	344	282	275	276	282	328	225
	9	341	298	299	350	329	301	334	334
	11	334	325	293	323	357	364	381	360
	13	256	307	282	271	308	336	269	398
	14	285	346	323	302	301	346	323	344
	15	297	315	316	263		229	315	323
	16	383	406	366	337	406	404	304	331
	17	302	318		341	302	393	318	318
	18	252	223	249	239	253	308	280	
	19	305	309	320	339	240	302	357	337
	20	197	286	331	346	286	329	323	357
	No	After							
		A	B	C	D	E	F	G	H
	1	239	265	290	237	44	45	77	97
	2	299	254	315	272	89	72	69	46
	3	272	290	277	277	49	28	30	31
	5	197	305	305	326	28	30	35	30
	6	255	255	261	272	60	4	2	6
	7	283	271	290	290	6	6	6	6
	8	263	238	245	276	6	6	6	6
	9	291	234	262	223	6	6	6	6
	11	266	265	293	293	6	6	6	6
	13	265	221	238	206	6	6	6	6
	14	238	217	305	295	6	9	9	76
	15	249	312	335	337	6	6	6	6
	16	253	253	253	325	10	10	10	10
	17	250	211	270	257	22	6	6	36
	18	279	275	336	312	6	6	8	6
	19	326	307	308	317	26	6	6	6
	20	323	283	251	49	6	6	6	6

Figure 27 - Group A; Enamel microhardness values in area α (area under the bracket represented by areas A, B, C and D) and area β (area around the bracket, represented by areas E, F, G and H) before the cycling process.

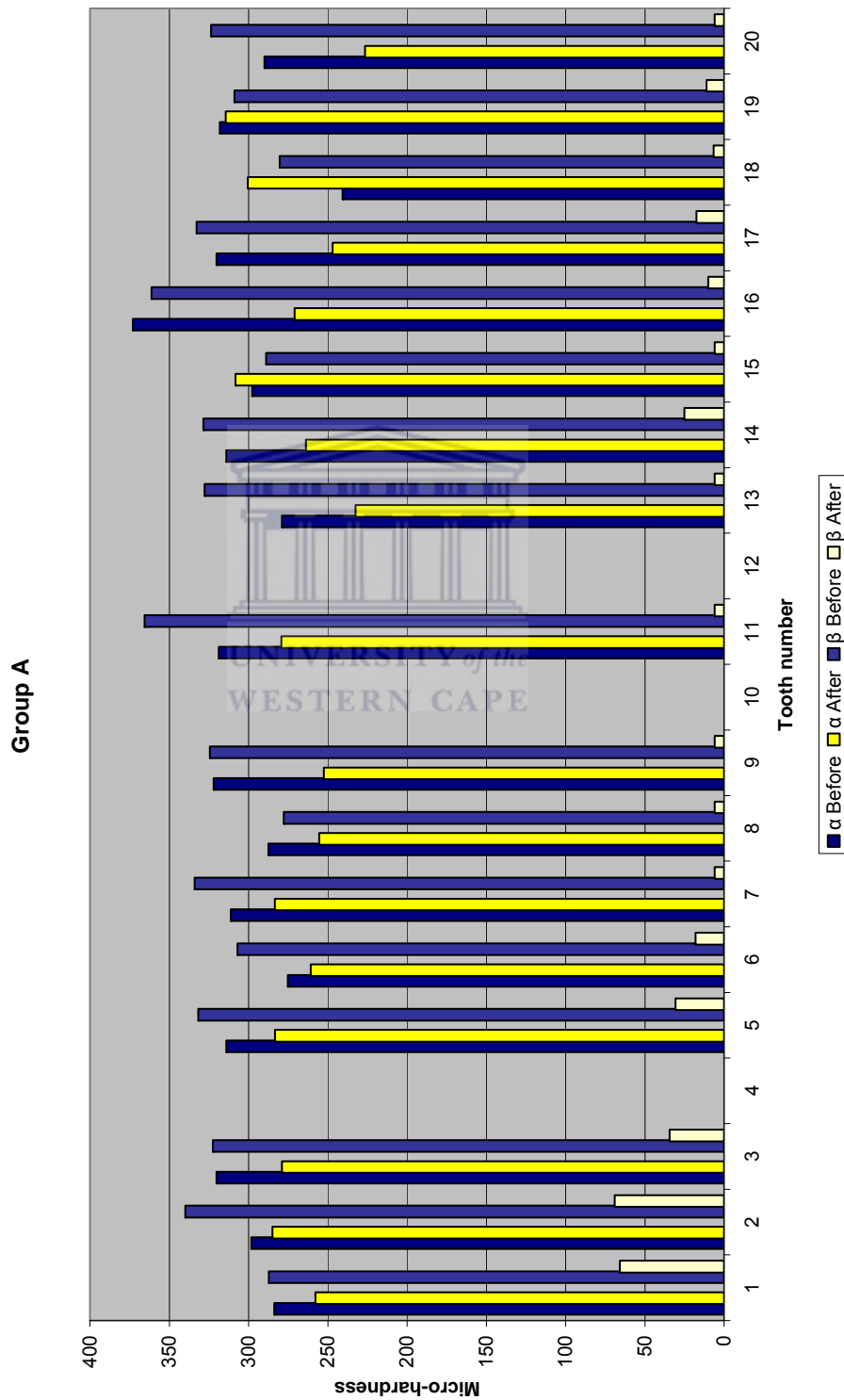


Table 12 - Group B; Enamel microhardness before and after the pH cycling.

	No	A	B	C	D	E	F	G	H	
Before	1	271	277	270	276	298	323	315	312	
	2	201	239	261	304	312	287	297	331	
	3	333	371	315	289	287	391	339	289	
	4	307	333	283	304	371	297	325	341	
	6	168	218	270	228	249	308	318	308	
	8	252	230	168	257	314	235	212	317	
	9	268	227	290	245	174	249	282	339	
	10	299	238	304	308	317	315	337	337	
	11	276	253	325	349	223	253	337	295	
	12	264	341	312	291	237	344	329	333	
	13	253	243	315	283	253	315	339	314	
	14	317	321	281	305	260	323	317	317	
	15	286	297	294	282	287	293	275	317	
	16	255	295	295	209	309	314	349	276	
	18	320	346	264	282	289	323	309	311	
	19		243	274	228	243	299	336	333	
	20	209	237	205	290	181	298	293	355	
	After	1	200	217	200	240	212	226	234	240
		2	257	276	260	250	207	235	349	245
		3	301	305	290	310	245	333	240	308
4		242	245	240	235	336	275	302	315	
6		235	200	246	210	239	375	331	360	
8		314	300	315	320	262	257	250	260	
9		163	220	183	189	185	299	149	210	
10		226	216	320	230	301	218	221	321	
11		234	249	225	230	194	321	301	263	
12		221	252	230	240	237	252	260	298	
13		290	213	177	230	141	177	215	373	
14		271	315	251	230	315	320	201	205	
15		229	239	171	191	92	287	302	179	
16		191	174	48	198	251	260	315	281	
18		260	276	280	258	310	299	304	310	
19			202	195		180	191	199		
20		241	230	260	255	200	189	194	200	

Figure 28 - Group B; Enamel microhardness values in area α (area under the bracket represented by areas A, B, C and D) and area β (area around the bracket, represented by areas E, F, G and H) before the cycling process.

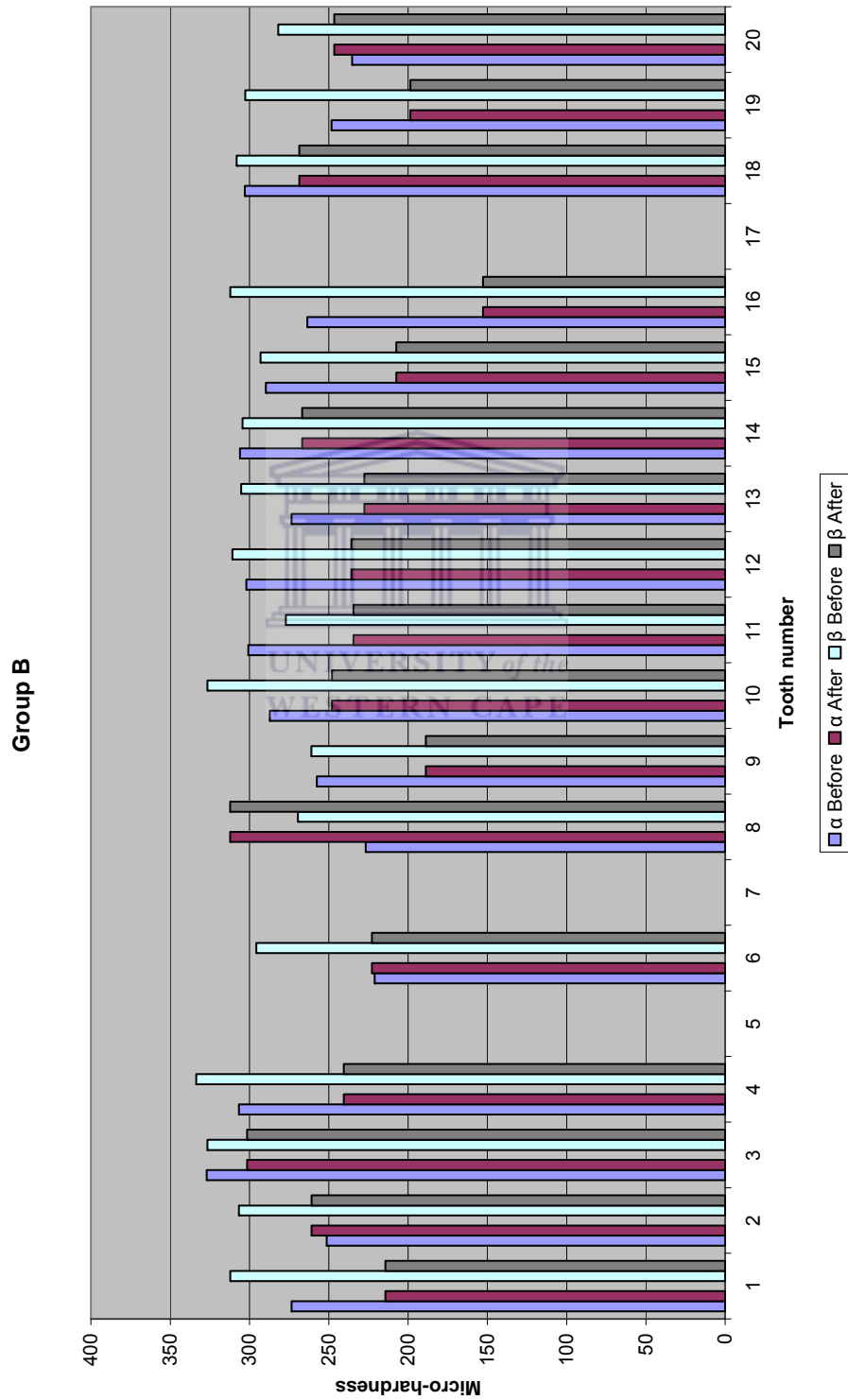


Table 13 - Group C; Enamel microhardness before and after the ph cycling process

	No	A	B	C	D	E	F	G	H	
Before	2	311	253	237	304	253	349	301	358	
	3	315	314	315	299	255	355	315	358	
	4	295	277	355	351	285	355	343	351	
	5	289	298	312	309	321	329	329	323	
	6	252	294	318	294	224	346	366	349	
	7	271	255	277	294	280	265	285	329	
	8	299	320	344	325	289	299	360	333	
	9	281	299	341	328		341	337		
	11	302	294	346	299	258	311	312	320	
	12	250	253	315	269		297	294		
	13	236	242	254	250	242	314	254	233	
	14	192	283	260	251		307	297	257	
	15	297	353	346	314	334	211		314	
	16	242	221	375		207	308	355	258	
	17	265	287	304	308	323	351	290	280	
	18	295	366	344	309	256		355	360	
	19	358	326	312	274	326	349	279		
	20	302	287	280	287	370	355	286	311	
	After	2	272	255	269	177	24	290	123	177
		3	262	270	270	207	60	63	21	43
4		290	302	271	253	274	223	225	124	
5		321	301	287	288	200	287	287	325	
6		270	253	219	329	293	142	307	19	
7		237	238	314	230	31	223	242	312	
8		270	211	309	314	120	206	366	190	
9		214	214	280	37	46	214	280	54	
11		27	268	248	274	186	270	248	294	
12		348	214	230	28		294	281	263	
13		225	250	237	200	34	280	84	114	
14		242	239	263	215		281	293	280	
15		246	240	276	242	94	250	24	154	
16		243	266	214	220	270	302	272	42	
17	301	266	271	280	179	180	195	190		
18	30	274	320	280	294	209	346	215		
19	362	340	269	301	68	315	258	320		
20	197	252	287	323	215	294	219	340		

Figure 29 - Group C; Enamel microhardness values in area α (area under the bracket represented by areas A, B, C and D) and area β (area around the bracket, represented by areas E, F, G and H) before the cycling process.

