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A STUDY OF THE FLUORIDE ION
RELEASE FROM THREE PROPRIETARY
ORTHODONTIC BONDING AGENTS

STEPHEN LEONARD DUNCAN
BDS
(SYDNEY)

A Thesis Submitted in Partial Requirement for the
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Department of Preventive Dentistry
Faculty of Dentistry
University of Sydney
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DEDICATION

This thesis is dedicated to my loving wife, Natalie, and parents, Leonard and Adella, all of whom have allowed me every opportunity in life.
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INTRODUCTION

The problem of enamel decalcification associated with fixed orthodontic appliances is well recognised (Zachrisson and Zachrisson 1971, Sadowsky et al 1981, Mizrahi 1982, Gorelick et al 1982). The difficulty in cleaning around cemented bands and, more recently, resin-bonded brackets may cause teeth with orthodontic attachments to be highly susceptible to decay (Douglass et al 1991). Consequently, it would be a desirable property of an orthodontic cement and bonding agent to possess a fluoride-releasing potential, considering the well-documented anti-caries effect offered by fluoride (Valk and Davidson 1987).

The silicate cements present a copious fluoride ion concentration to the surrounding oral environment, yet are unsuitable for use due to their excessive solubility (Wilson and Batchelor 1967). Wilson and Kent (1972) developed glass ionomer cement which, like the silicate cements, has a long term fluoride-releasing capability but with an acceptable dissolution rate. Glass ionomer cement is currently the material of choice for the cementation of orthodontic bands because of its anti-caries capability (Valk and Davidson 1987). Unfortunately the bond strengths offered by glass ionomer cement for bracket placement are insufficient to be clinically viable at this stage (Cook and Youngson 1988).
The bracket cementation method used today is based on the acid-etch principle first introduced by Buonocore (1955). The luting agent is a BIS-GMA resin complex. Although the bond strengths achieved by the acid-etch resin technique are clinically adequate, the risk of enamel demineralisation around the bracket base persists.

Recently, several manufacturers have begun marketing orthodontic bonding resins claiming to have a fluoride-ion-releasing potential. It is the purpose of this study to examine the absolute fluoride-release from three different proprietary "fluoride-containing resins" over a period of 84 days.

HYPOTHESIS
That fluoride is released from FluoroBond*, Northern Lights ** and Light-Bond*** when polymerised specimens are placed in a deionised water environment.

* FluoroBond – Visible light-cure orthodontic sealant. Ormco, California, USA.
** Northern Lights – Visible light-cure orthodontic bonding system. Rocky Mountain Orthodontics, Colorado, USA.
*** Light-Bond – Visible light-cure orthodontic bonding system. Reliance, Illinois, USA.
REVIEW OF LITERATURE

CHAPTER ONE

FLUORIDE : ITS MODE OF ACTION

The beneficial effects of fluoride as an anti-caries agent have been well documented since Dean wrote on the matter in 1938. Fluoride is available to the population in many different forms (e.g., fluoridated water supplies, fluoridated dentrifices, fluoride tablets, fluoridated mouthrinses, fluoridated gels, fluoridated topical solutions, etc).

Fluoride-based preventive programmes have caused a varied reduction in caries incidence, with the majority ranging between a 40-70 per cent lowering of DMFS scores (Naylor and Murray 1989.p151, Thylstrup et al 1982). It is generally accepted that fluoridation of the local water source to the value of 1ppm will cause an approximate 50 per cent reduction in caries incidence (Backer Dirks et al 1978). The cariostatic mechanism does not seem to be determined by the concentration or mode of fluoride application. The most important factor in caries reduction appears to be the frequency of fluoride exposure, with increasing frequency offering greater caries protection (Ericsson 1977).
The exact mechanisms of fluoride action are still largely unknown. Clinical and experimental data indicate that the mode of action is not singular (Luoma et al 1986.p299, Mellberg 1977).

The suggested mechanisms of action include -

1. Decrease in enamel solubility.
2. Improvement in enamel crystal structure.
3. Improvement in tooth morphology.
5. Improvement of remineralisation.

1.1. DECREASE in ENAMEL SOLUBILITY

Enamel is composed mainly of an hydroxyapatite crystal (crystalline calcium phosphate) lattice. The susceptibility of this structure to dissolution in a low pH environment forms the chemical basis of a carious attack (Larsen and Bruun 1986.p189).

Fluoride may become incorporated in the enamel inorganic structure in place of an hydroxyl-ion. This may occur due to systemic acquisition of fluoride during enamel formation, or by surface contact following crown emergence (Aasenden et al 1973). The resultant fluoroapatite is less soluble in an acid environment than hydroxyapatite (Brown et al 1977, Newbrun 1986.p157).
Fluoroapatite contains $38 \times 10^3$ ppm fluoride, compared to the average enamel fluoride content of 500-1500 ppm. Therefore, one can only expect partial enamel hydroxyl substitution when an individual is exposed to physiologically acceptable levels of fluoride (Newbrun 1986.p157). The greatest concentration of enamel fluoride, up to 5000 ppm, is found in the outermost 10-50 micrometers (Luoma et al 1986.p318, Newbrun 1986.p157, Schamshula et al 1982). The formation of an enamel surface layer with the characteristics of fluoroapatite is possible and this may be an important mechanism in the anti-caries effect of fluoride (Brown et al 1977).

Even with complete fluoride-ion for hydroxyl-ion substitution, however, the solubility difference between hydroxyapatite and fluoroapatite seems too small to explain the enamel protection offered by fluoride (Newbrun 1986.p157). Brown et al (1977) suggested that the solubility of enamel is far more complicated than that presented by the gravimetric means of testing solubility.

Brudevold, Amdur, Vogel and Spinelli (1965) indicated that although many substances are known to reduce the solubility of enamel, only fluoride reduces the caries incidence. Additionally, studies tend to illustrate a poor correlation between the enamel fluoride content and caries incidence (Retief et al 1987, Nasir et al 1985). It appears, therefore, that the fluoride content of enamel
may play only a limited role in the protection of enamel from carious attack.

1.2. IMPROVEMENT in ENAMEL CRYSTAL STRUCTURE

It has been suggested that the incorporation of fluoride during enamel formation produces a more stable crystal structure which is more resistant to acid dissolution, less impure and possibly dimensionally bigger (Beltran and Burt 1988).

One proposed mechanism is that the presence of fluoride eliminates the formation of octacalcium phosphate. The hydroxyapatite thus formed is considered thermodynamically more stable (Brown et al 1977).

Van der Lugt et al (1970) suggested that fluoride might stabilise the enamel crystal by the formation of stronger hydrogen bonds.

1.3. IMPROVEMENT in TOOTH MORPHOLOGY

Certain studies indicate that teeth formed in a fluoridated environment acquire a more favourable morphology to resist plaque retention. Cooper and Ludwig (1965) reported that distinct changes occur in the shape of teeth that are formed in the presence of fluoride. The cusps are more rounded and the fissures are shallower than
in unfluoridated teeth (Forrest 1956, Cooper and Ludwig 1965). According to Luoma et al (1986.p314), the more rounded cusps may be due to the wearing of the surface enamel of "snow-capped" cusp tips (the result of mild fluorosis) instead of a change in natural morphology.

1.4. ANTI-BACTERIAL ACTION

Fluoride has the ability to act as an anti-bacterial agent in a number of ways -

2. Enzyme inhibition.
3. Reducing the ability of bacteria to colonise enamel.

1.4.1. BACTERICIDAL ACTION

Fluoride, at high concentration, has an immediate bactericidal effect. The concentration required is far in excess of the normal daily fluoride experience (eg, 1ppm fluoride in H₂O, fluoridated toothpastes, fluoridated mouth-rinses etc.) but may occur with a professionally applied fluoride application, such as an acidulated phosphate fluoride gel containing 12,300ppm (Newbrun 1986.p163, Loesche 1977). This high quantity of fluoride will cause an immediate reduction in the population of the oral flora. Unfortunately, however, a long term bactericidal action of such an application has not been
demonstrated due to the subsequent dilution or reduction in fluoride ion concentration by saliva or mechanical removal (Loesche 1977).

In a study of children exposed to high and low levels of water fluoridation, Kilian et al (1979) found no basic difference in the microbial composition of plaque between the two groups.

1.4.2. ENZYME INHIBITION

It has been proposed that fluoride exposure has an inhibitory effect on the carbohydrate metabolism of plaque bacteria (Kilian et al. 1979, Hamilton 1977). NaF mouthrinses, containing a minimum concentration of 0.05% fluoride, have been shown to drastically reduce or even stop acid production from plaque bacteria for up to 60 minutes (Hata et al. 1991).

In a review article, Hamilton (1977) stated that fluoride may inhibit carbohydrate metabolism by the following mechanisms:

1. Inhibits enolase and therefore the production of phosphoenolpyruvate (PEP) in the glycolytic pathway. By depleting the quantities of PEP, fluoride indirectly interferes with the glucose transport into the bacterial cell by affecting the phosphoenolpyruvate-hexose phospho-transferase transport system.
2. Inhibits sugar translocation in membranes.
3. Inhibits cation transport and accumulation in cells.
4. Inhibits cellular phosphatases which dephosphorylate sugar-phosphatases resulting from transport.

1.4.3. REDUCTION OF BACTERIAL COLONISATION of ENAMEL

The initial stage in the bacterial colonisation of enamel is the formation of the acquired pellicle which is followed by bacterial penetration, or adherence, and subsequent multiplication to form the bacterial plaque (Sonju 1986.p46).

Fluoride may theoretically influence this colonisation by:
1. Interfering with the adherence of the acquired pellicle.
2. Interfering with the aggregation and multiplication of bacteria (Newbrun 1986.p164).

Ericson and Ericsson (1967) showed that protein has less affinity for fluoroapatite than for hydroxyapatite. This decreased affinity for fluoridated enamel may be explained by Glantz (1969) who found that fluoroapatite has a lower surface energy than normal apatite.

Rolla and Melsen (1975) indicated that fluoride decreases the adsorption of albumin to enamel and may even cause the desorption of protein from the apatite surface. The
mechanism suggested involves competitive inhibition in that the fluoride and monofluorophosphate ions displace the acidic protein groups adsorbed to calcium sites on the enamel surface.

Sodium fluoride seems to have a minimal effect on the initial colonisation of teeth whereas stannous fluoride causes significant plaque inhibition (Kilian et al 1979, Svantun et al 1977, Tinanoff et al 1976). The main mechanism suggested is an alteration of adhesive properties between enamel and bacteria, and between bacteria themselves caused by the covalent tin ion (Tinanoff et al 1976).

1.5. IMPROVEMENT of REMINERALISATION

The major mechanism by which fluoride exerts its anticariogenic activity appears to be in the remineralisation of the early acid demineralised enamel (Brudevold, Gron & McCann 1965, Silverstone 1985.p153, Mellberg 1977). The necessary requirements for remineralisation to occur include an apatite surface that is intact (without cavitation), a substrate containing the necessary ions and a favourable pH.

A caries-affected enamel surface may undergo remineralisation in the presence of saliva alone, provided the above conditions are satisfied (Dijkman et al 1986).
The ability for enamel to remineralise is considered a natural defence mechanism (Beltran and Burt 1988). What fluoride appears to do, is to enhance this remineralising process (Borsboom et al 1985). The fluoride enhanced reaction shows a greater level of remineralisation than other substrate combinations with the resultant repaired enamel incorporating more fluoride than normal enamel (Koulourides et al 1980, Silverstone 1983.p202).

Silverstone (1983.p201) reported that remineralised enamel demonstrated crystal growth, greater than that found in normal enamel, in two zones of the carious lesion (surface zone & dark zone). He continued (p204):

"..it appears that the initiation of a small sub-clinical lesion in the outer enamel paradoxically aids in preventing its progression. Firstly, by preferentially taking up significantly more fluoride than adjacent sound enamel. The lesion therefore acts as a fluoride reservoir and, during dissolution when calcium and phosphate ions are released, the presence of fluoride ions within the lesion favours remineralisation."

Contemporary thought is that the most important factor in the potential for remineralisation and subsequent protection of tooth structure from caries, is a constant or frequent supply of the fluoride ion (Silverstone 1983.p204, Mellberg 1977). The concentration required is not necessarily high (Silverstone 1983.p204). For example,
water fluoridation with 1ppm provides an average 50 per cent decrease in DMFT whereas bi-annual professional topical applications (usually high concentrations), in non-fluoridated areas, generally result with a 30 per cent decrease (Horowitz and Heifetz 1986, p73).

When large levels of sodium fluoride are used, much of the fluoride is precipitated on the tooth surface in the form of calcium fluoride (Saxegaard and Rolla 1989). Calcium fluoride is relatively insoluble and provides a reservoir of fluoride for use in the local environment (Saxegaard et al 1988). This proposed mechanism may explain the long term benefit of professional sodium fluoride topical applications. As time progresses, however, the alkali-soluble fluoride source is depleted and the caries risk increases.

There is also the suggestion that a simple ionic fluoride (eg, sodium fluoride) reacts more rapidly with the enamel than will a phosphate fluoride (eg, sodium monofluorophosphate) (Mellberg and Mallon 1984). The rapid remineralisation may block diffusion channels in the enamel surface, inhibiting penetration into the body of the lesion and therefore a less than ideal remineralisation may occur. Mellberg and Mallon (1984) suggested that a solution combining sodium fluoride and sodium monofluorophosphate will provide the most rapid and penetrating type of enamel remineralisation.
Published data support the concept that acid-primed enamel will remineralise more rapidly and to a greater extent than normal apatite (Koulourides et al 1980, Featherstone et al 1981). The cariogenic challenge is suggested to initiate local enamel changes which result in an increased caries resistance. This mechanism, if allowed to occur, should help in the protection of the acid-primed enamel surfaces encountered in the acid-etched bonding of orthodontic appliances. If the cariogenic challenge is too great, however, enamel cavitation will occur (Mellberg 1977, Koulourides and Cameron 1980).
CHAPTER 2

ORTHODONTICS and DEMINERALISATION

It is generally accepted that the placement of fixed orthodontic appliances makes plaque removal more difficult (Huser et al 1990). As a result, there is an increase in the levels of cariogenic bacteria adherent to tooth surfaces in orthodontic patients (Bloom and Brown 1964, Corbett et al 1981, Mattingly et al 1983, Arneberg et al 1984, Scheie et al 1984). Longitudinal and cross-sectional studies have confirmed that a higher incidence of demineralisation occurs in orthodontic patients (Stratemann and Shannon 1974, Mizrahi 1982, Gorelick et al 1982, Douglass et al 1991). As a result of the greater caries challenge faced by orthodontic patients, stringent preventive programmes are recommended (Shannon and West 1979).

In vivo studies, involving teeth designated for extraction, show that early signs of enamel demineralisation can occur within three to four weeks after bracket or band placement (Ogaard, Rolla and Arends 1988, Glatz and Featherstone 1985, O'Reilly and Featherstone 1987, von der Fehr et al 1970). Using hardness testing Glatz and Featherstone (1985) reported demineralisation to 75µm with 25 per cent mineral loss after four weeks. O'Reilly and Featherstone (1987)
demonstrated a 15% per cent mineral loss to 50µm, which was not clinically visible, over the same time period.

Ogaard, Rolla and Arends (1988) reported two types of early demineralisation:

1) A surface-softened lesion - Reflects a low surface concentration of mineral in comparison with normal enamel.

2) A subsurface lesion - A porous but mineral rich surface layer covering a subsurface region showing mineral loss.

The surface-softened lesion apparently represents an area of high cariogenic challenge, which has not allowed remineralisation of the surface layer to occur.

Borsboom and Arends (1985) showed that demineralisation without fluoride resulted in enamel defects without a surface layer (softened), whereas with small amounts of fluoride present (0.12ppm) the demineralisation resulted in subsurface lesions.

The following areas will be addressed in this chapter:

* Caries incidence in orthodontic patients.
* Benefits of fluoride based preventive programmes.
* The nature and treatment of white spot formation.
* Bacterial flora and orthodontics.
* Enamel loss during cementation/bonding/debonding procedures.
2.1. INCIDENCE of DEMINERALISATION ASSOCIATED WITH FIXED-APPLIANCES

The nature of the fixed appliance including bands, brackets, arch wires, auxiliaries and elastics, makes normal home oral hygiene procedures difficult (Zachrisson and Zachrisson 1971;183-192, Mizrahi 1983, Artun and Thylstrup 1986). In orthodontic patients plaque removal may therefore be less than desirable and the caries challenge is thus increased (Zachrisson and Zachrisson 1971;394-401). Zachrisson and Zachrisson (1971;394-401) found a definite correlation between oral health and caries incidence in an orthodontic group. With increasing Plaque and Gingival Index scores there was an almost linear increase in the mean Caries Index. They also suggested that females performed the oral hygiene tasks better than the males in the sample. Mizrahi (1982) in a cross-sectional study reported that the male patients experienced a greater increase in the severity of white spot lesions, than did females. Geiger et al (1988) found no significant differences in the incidence and severity of white spot according to sex.

The majority of studies reveal that the incidence of demineralisation in patients receiving orthodontic treatment is higher than the incidence found in a control population without fixed appliances (Douglass et al 1991, Gorelick et al 1982, Shannon and West 1979, Stratemann and
Shannon 1974). This incidence decreases with improvement of oral hygiene and the use of fluoridated preventive programmes (Stratemann and Shannon 1974).

Douglass (1991) examined, longitudinally, 50 patients from a non-fluoridated area undergoing multi-bonded orthodontic treatment. An 84 per cent increase in identifiable decalcification was registered.

Gorelick et al (1982) found that 50 per cent of patients incurred an increase in the number of white spots. There was no difference in the incidence of demineralisation between banded and bonded attachments.

Shannon and West (1979) demonstrated a post-treatment decalcification incidence of 64.1 per cent for orthodontic patients.

Stratemann and Shannon (1974) reported a decalcification incidence of 58 per cent in their control group not using a fluoride mouth rinse. The test group was instructed to use an 0.4 per cent stannous fluoride gel once daily. Those patients who complied with the daily use (51 of 99) experienced a 2% incidence in decalcification.
2.2. CARIES INCIDENCE and TOPICAL FLUORIDE PROGRAMMES

The use of fluoride in conjunction with orthodontic treatment has been shown to decrease the caries risk (Zachrisson 1971, Stratemann and Shannon 1974, Shannon et al 1977, Wisth and Nord 1977, Hirschfield 1978, Magness et al 1979, Dyer and Shannon 1982, Artun and Brobakken 1986, Geiger et al 1988). Topical fluoride procedures involving the use of varied concentration, chemical compound type, and frequency of application have been reported. The success or failure of these programmes seems largely dependent on the frequency of fluoride application and patient compliance (Stratemann and Shannon 1974).

Artun and Brobakken (1986) compared three groups; two orthodontic and one control. Both orthodontic groups were instructed to use a daily fluoride rinse (0.05% NaF). The first orthodontic group received more supervision and demonstrated no significant difference in white spot formation to the control group. The other orthodontic group, whose preventive programme was not monitored as closely showed significantly more demineralisation than the other two groups.

Dyer and Shannon (1982) compared the effects of two one minute rinses per day with sodium monofluorophosphate (0.184% F⁻) and stannous fluoride (0.1% F⁻) on multi-
banded patients. The clinical results between the two were similar with a nil and 1.5 per cent increase in demineralisation respectively. The assessment of decalcification was performed after 12 months treatment time with the bands still in situ.

Hirschfield (1978) demonstrated a statistically significant decrease in decalcification using a daily rinse with acid-phosphate-fluoride on banded individuals. In this study, only the maxillary right lateral incisor and mandibular left first permanent molar were scored.

Magness et al (1979) employed stringent oral hygiene conditions and a professionally applied acidulated phosphate fluoride (0.31% F⁻) followed by a 0.4 per cent stannous fluoride rinse. The average time between appointments was 3.15 weeks and the patients wore full bands. 1.3 per cent of teeth showed signs of new or increased decalcification. This represented an increase in the percentage of patients with increased demineralisation of 18.2 per cent. All orthodontic bands were in situ during the final examination for decalcification.

Stratemann and Shannon (1974) used 0.4 per cent stannous fluoride gel daily on 99 patients banded for 18-24 months. The patients were instructed to record the frequency of gel usage. The control sample showed a 58 per cent increase in the incidence of decalcification. The
The incidence of post-treatment decalcification as a function of compliance follows:

<table>
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<th>Gel use</th>
<th>Patient No.</th>
<th>Decal.%</th>
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<tr>
<td>0-1 per week</td>
<td>29</td>
<td>66</td>
</tr>
<tr>
<td>2-3 per week</td>
<td>19</td>
<td>26</td>
</tr>
<tr>
<td>Daily as directed</td>
<td>51</td>
<td>2</td>
</tr>
</tbody>
</table>

The importance of frequency of fluoride exposure in the reduction of caries incidence is well demonstrated by these findings. It also documents the problems faced with patient compliance in that only 50 per cent of the participants followed the instructions completely.

Shannon et al (1977) charted the band margins (orthodontic bands in situ) after 12 months of orthodontic treatment. The study was designed to compare the protection offered by the daily use of stannous fluoride and sodium fluoride gels and rinses. Stannous fluoride (0.4%) offered total protection from decalcification whereas sodium fluoride showed decalcification in 3 per cent of teeth or 30 per cent of patients.

In a cross-sectional study Wisth and Nord (1977) reported a decreased caries experience with orthodontic treatment. The orthodontic patients were instructed to rinse daily with an 0.05 per cent sodium fluoride solution.
Geiger et al (1988) conducted a longitudinal study of 101 multi-bonded orthodontic cases instructed to use an 0.05 per cent sodium fluoride mouthrinse daily. At the completion of treatment 34 patients exhibited one or more teeth with white spot formation. They found that the incidence of decalcification was time-dependant with a definite increase after 18 months. Additionally, patient compliance was registered. White spot incidence in excellent co-operators (26.7% of sample) was 11.8 per cent, whereas those with poor compliance (52.5%) was 64.7 per cent. This relationship between poor compliance and increased risk of demineralisation supports the findings of Stratemann and Shannon (1974).

Fluoride is effective at low pH in reducing the extent of enamel demineralisation (Jeansonne and Feagin 1979). Enamel fluoride uptake is greatest when the vehicle of fluoride application is acidic (Kajander et al 1987). An increased fluoride concentration in the enamel surface has been demonstrated in caries-like lesions treated with fluoride solutions (Koulourides et al 1980).

Kajander et al (1987) recommended the use of a 4 per cent acidified sodium fluoride topical treatment following bonding procedures on the basis that the acidified vehicle raised the enamel fluoride concentrations more than neutral sodium fluoride.
Ogaard, Rolla, Arends and ten Cate (1988) demonstrated the efficiency of one topical application of 0.6 per cent sodium fluoride at pH 1.9 compared to daily rinsing with a neutral sodium fluoride solution in retarding enamel demineralisation. The neutral sodium fluoride significantly decreased decalcification whereas the one acidified application completely inhibited lesion formation. The acidified solution results in the formation of large amounts of calcium fluoride which if not removed act as a reservoir of fluoride ion.

Dyer and Shannon (1982) reported that an 0.1 per cent stannous fluoride solution is a more effective anti-caries agent than an 0.184 per cent monofluorophosphate solution. Stannous fluoride, however, has certain disadvantages in that the taste is unpleasant, the metallic ions tend to cause discoloration of hypomineralised regions of teeth, and it lacks stability in aqueous media (Zachrisson 1975).

Shannon et al (1977) and Shannon (1981) favour stannous fluoride (0.1% solution or 0.4% gel) before neutral sodium fluoride for preventive use in orthodontic patients.
O'Reilly and Featherstone (1987) compared four preventive regimes on bonded premolars scheduled for extraction.

- **Group 1** - Brushing only with fluoridated dentifrice.
- **Group 2** - Brushing plus nightly 0.05% NaF rinse.
- **Group 3** - Brushing + weekly 1.23% APF gel.
- **Group 4** - Brushing + daily NaF rinse + weekly APF gel.

Group 1, who brushed with a normal fluoridated toothpaste, showed early evidence of enamel surface softening. Nightly sodium fluoride rinsing (group 2) revealed a normal enamel profile. Group 3 possessed an hypermineralised outer layer to a depth of 25μm. A similar but slightly more extensive hyper-mineralisation was seen in Group 4. The authors concluded that daily toothbrushing coupled with an 0.05 per cent sodium fluoride rinse is sufficient to prevent demineralisation occurring in orthodontic patients.

### 2.3. CARIES INCIDENCE and OTHER FLUORIDE-BASED PREVENTIVE PROGRAMMES

Apart from topical fluoride applications, fluoride has been incorporated into band-cementing agents and more recently bonding resins. Glass ionomer cements have become the orthodontic cement of choice due to their prolonged fluoride-releasing capacity. The benefit of a fluoride-releasing orthodontic cement or bonding agent has been clearly demonstrated by Valk and Davidson (1987) and

Sonis and Snell (1989) and Underwood et al (1989) investigated fluoride-releasing bonding resins. Underwood et al (1989) found that a fluoride-exchanging resin (FER) experienced a 2.3 per cent incidence of dark zone formation compared to 33.5 per cent for Concise™. 1 Sonis and Snell (1989) compared FluorEver® (fluoride-releasing resin)² to a non-fluoride-releasing bonding resin (Aurafill™)³. After an average treatment period of 25 months, those teeth bonded with FluorEver® exhibited no decalcification whereas the teeth bonded with Aurafill™ demonstrated a 12.6 per cent incidence of white spot formation.

Adriaens et al (1990) examined Fluor Protector™ (polyurethane varnish with 0.7% fluoride) applied to molars prior to banding. The in vitro study revealed that the fluoride varnish was very effective in inhibiting demineralisation.

Non-fluoridated resins and varnishes have been used to seal the enamel surface prior to the placement of orthodontic appliances with limited success (Lee et al 1973, Hughes et al 1979, Zachrisson 1979, Ceen and

¹3M Dental Products, St Paul, Minn.
²Macrochem Corp., Woburn, Mass.
³Johnson & Johnson Dental Care Co., East Windsor, N.J.

2.4. WHITE SPOT LESIONS

According to Artun and Thylstrup (1986), the early carious lesion appears as a "dull white stripe" or a large "greyish-white" spot. The surface is still patent and the white spot "is caused by changes in the optical properties of the enamel due to subsurface demineralisation".

The incidence of visible decalcification at the completion of orthodontic treatment in orthodontic patients with good oral hygiene and excellent compliance in the use of a topical fluoride rinse is low (Zachrisson 1971). Stratemann and Shannon (1974) and Geiger et al (1988) indicate that some patients will perform below the standards required to prevent white spot formation.

Certain teeth seem to be more likely to undergo demineralisation associated with orthodontic treatment than others. The most susceptible tooth appears to be the maxillary lateral incisor followed by the maxillary canine and the mandibular canine and posteriors (Gorelick et al 1982, Mizrahi 1983, Geiger et al 1988). The area of the tooth surface most susceptible to demineralisation is the
gingival third of the labial or buccal surface (Artun and Brobakken 1986, Mizrahi 1983).

In patients exhibiting visible enamel decalcification, appliance removal usually results in cessation of the high cariogenic challenge and arrest of further demineralisation (Artun and Thylstrup 1989). With time, there is a gradual improvement in the appearance of these lesions. Artun and Thylstrup (1989) investigated the changes in white spot lesions for a period of 3 years following orthodontic treatment. They reported that, after three years, only remnants of the original opacities remained.

Ogaard (1989) examined 51 orthodontic patients more than five years out of treatment. The higher incidence of white spot lesions in this group, compared to an untreated control group, led the author to report that the aesthetic problem of decalcification associated with orthodontics may continue for more than five years following treatment.

The degree of repair experienced by the white spot depends on the extent of the lesion and the environmental influences on the tooth surface (Artun and Thylstrup 1989).

Repair can occur by two means:

1 - Remineralisation.
2 - Wear or abrasion of the enamel surface.
Ideal repair would result from complete remineralisation of the enamel lesion. Fluoride enhances the rate of enamel remineralisation (Mellberg and Mallon 1984). There is some evidence, however, that the presence of high concentrations of fluoride causes a rapid remineralisation of the surface layer which blocks diffusion channels leading to the deeper areas of the lesion. In such circumstances only the superficial layers are repaired (Ogaard, Rolla, Arends and ten Cate 1988).

Featherstone et al (1982) tested a remineralising solution designed specifically to penetrate early carious enamel. The solution, which contained a low concentration of fluoride, caused rapid remineralisation of artificially produced decalcification.

Hicks and Silverstone (1984) recommended the acid-etching of white spot lesions in order to create a porous surface that is more reactive in the presence of a remineralising solution. Simon and Geurtsen (1991) studied the morphological changes of artificially produced white spot lesions subjected to acid-etch procedures. They concluded that the acid-etching may cause a loss of the intact surface layer and thus cavitation.

Saliva, itself, is capable of causing remineralisation (Featherstone et al 1982; Ogaard, Rolla, Arends and ten Cate 1988). Ogaard, Rolla, Arends and ten Cate (1988)
considered the rate of enamel remineralisation in the presence of saliva and in the absence of high fluoride concentrations to be relatively fast.

Artun and Thylstrup (1989) considered that the gradual regression of white spots is primarily due to surface wear. For this reason, the more extensive lesions (ie, the deeper lesions) still remained 3 years following appliance removal.

2.5. BACTERIAL FLORA CHANGES ASSOCIATED WITH FIXED ORTHODONTIC APPLIANCES

Following the placement of fixed orthodontic appliances, the plaque and salivary levels of cariogenic bacteria is reported to increase. Raised levels of lactobacilli and streptococcus mutans in these patients have been documented by Bloom and Brown 1964, Adams 1967, Corbett et al 1981, Arneberg et al 1984, Scheie et al 1984 and Sinclair et al 1987.

Scheie et al (1984) found a decrease in the levels of streptococcus mutans soon after the placement of attachments. This decrease was only transient with elevated quantities of streptococcus mutans re-established by three months.
Increased levels of plaque accumulation in orthodontic patients is not uncommon (Huser et al 1990). The interesting finding, concerning the nature of this bacterial flora, is that a statistically significant increase is found preferentially in the cariogenic species in many studies (Bloom and Brown 1964, Corbett et al 1981, Mattingly et al 1983, Arneberg et al 1984, Scheie et al 1984 and Sinclair et al 1987).

Arneberg et al (1984) showed that loose orthodontic bands created a cariogenic environment that suited aciduric species such as streptococcus mutans and lactobacilli.

Sinclair et al, in 1987, could not demonstrate a significant increase in plaque levels following 12 months of fixed appliance wear. The authors, however, were able to document a significant increase in the sub-gingival levels of streptococcus mutans.

Huser et al (1990), in contrast to Sinclair et al (1987), noted a decrease in streptococci found in plaque associated with orthodontic bands.

Composite resins are almost universally used for the bonding of brackets to teeth. The resin surface is not smooth in nature. The roughened texture has been shown to facilitate the adherence of plaque (Weitman and Eames 1975, Gwinnett and Ceen 1979, Oliver and Howe 1989).
Gwinnett and Ceen (1979) found that plaque accumulated on the surface of composite resins in patients, even with good oral hygiene performance. The surface area of the resin was considered most important in relation to the amount of plaque present.

The area of particular concern for plaque accumulation is the enamel-resin interface (Gwinnett and Ceen 1979, Oliver and Eames 1989). The presence of composite ridges, overhangs, folds and voids at the interface, demonstrated by scanning electron microscope, makes this site more prone to plaque accumulation. Mature plaque was routinely associated with these mechanically retentive regions (Gwinnett and Ceen 1979).

Forss et al (1991) compared the streptococcus mutans grown on a glass ionomer cement and a non-fluoride-releasing composite resin in vivo. They were able to demonstrate an intracellular increase in fluoride levels and a reduction in the numbers of streptococcus mutans associated with the glass ionomer cement.

2.6. ENAMEL LOSS DURING CEMENTATION, BONDING and DEBONDING PROCEDURES

The fluoride content of the outermost micrometers of the enamel surface is considered important in caries resistance (Brown et al 1977). The loss of the enamel
surface layer will result in an exposed hydroxyapatite crystal surface whose fluoride concentration is dramatically reduced (Schamschula et al 1982). Surface preparation for acid-etch bonding procedures has been shown to cause enamel loss (Fitzpatrick and Way 1977, Lee Brown and Way 1978, Pus and Way 1980, Sampson et al 1987). This enamel loss, according to some, may cause a reduced caries resistance due to a decreased fluoride concentration.

Routine bonding or banding procedures require the polishing of the enamel surface in order to remove any remnants of the acquired pellicle or plaque. In bonding procedures this is followed by acid-etching.

Using zirconium silicate Lee Brown and Way (1978) found that the median enamel loss using a bristle brush for a 10-15 seconds polish was 26.0µm. Pus and Way (1980), in a similar study, demonstrated 10.7µm enamel loss with a bristle brush and 5.0µm with a rubber cup.

A 90 seconds etch with 37 per cent phosphoric acid caused a mean enamel loss of 3µm (Lee Brown and Way 1978); 9.9µm (Fitzpatrick and Way 1977); 6.9µm (Pus and Way 1980). Sampson et al (1987) found that the enamel loss with etchant times under 60 seconds ranged from 1.1µm to 7.4µm. Etchant times greater than 60 seconds eliminated the surface fluoride and risked significant loss of enamel.
Lehman and Davidson (1981) found that fluoridated enamel has a highly resistant surface layer approximately 2-4μm thick. They estimated that a mean enamel loss of 0.7μm is caused by an etchant duration of 60 seconds.


Debonding procedures also cause loss of surface enamel. Pus and Way (1980) found that the enamel loss depended on the technique used. A mean loss of 19.2μm occurred using high speed drills whereas an 11.3μm loss was associated with a low speed drill employing a tungsten carbide bur.

The total enamel loss due to placement and removal of orthodontic appliances is approximately 40μm to 50μm (Fitzpatrick and Way 1977, Lee Brown and Way 1978, Pus and Way 1980).
CHAPTER 3

FLUORIDE-RELEASING COMPOSITE RESINS

The development of dental composite resins was encouraged by the less than adequate performance of silicate cements in the oral environment (Braden 1978). Early composite resins used methyl methacrylate as the monomer. These resins, however, possessed unsuitable properties (high shrinkage, heat generation, pulpal toxicity) for use as restorative materials (Braden 1978).

In 1963, Bowen introduced a composite resin based on the co-monomer bisphenol A bis 2 hydroxy propyl methacrylate. Variations of this monomer system currently form the basis of the composite resins used in dentistry.

Silicate cements incorporate fluoride in their manufacturing process (Wilson and Batchelor 1967). This fluoride is readily available due to the high solubility of silicates in the oral environment. In contrast to silicate cements, composite resins are relatively insoluble and (generally) have not exhibited a significant fluoride-release potential until recently (Swift 1989).

Fluoride may be released from a material by two methods –

1- The material itself may dissolve resulting in the
release of incorporated fluoride, or

2- Fluoride ions may diffuse directly out of the material (Forsten & Paunio 1972, Shen 1985). According to Shen (1985), fluoride ion diffusion depends on the diffusion co-efficient of fluoride and the ability of water to penetrate the composite resin to aid the process.

Soderholm et al (1984) referred to the hydrolytic degradation of composite resins. Soderholm previously demonstrated that barium, silicon and strontium are released from composite resins in an aqueous medium (1983). Hydrogen ions enter the resin to maintain the charge balance leaving an excess of hydroxyl ions at the surface. This increase in concentration of hydroxyl ions begins to disrupt the silica network allowing ion exchange to occur (Charles 1958).

Apart from allowing ion-exchange to occur, hydrolytic degradation causes crack propagation which leads to an increase in the opacity of the composite resin. Soderholm (1984) established that microfilled composites resist crack formation more so than macrofilled resins and therefore possess better optical qualities.

Following the reported fluoride-release from an amalgam (Jerman 1970), Forsten and Paunio (1972) decided to investigate the availability of fluoride from other
relatively insoluble dental materials, including composite resins. One composite resin, TD 71®, demonstrated fluoride-release. The authors concluded that composite resins may possess the potential for fluoride-release similar to that of silicate cement.

Rawls and co-workers developed an experimental fluoride-exchanging composite resin which was first reported in 1979. The fluoride was incorporated as an amine-HF salt. The experimental resin demonstrated significantly less demineralisation than did Concise® in an artificial caries environment (Turpin-Mair et al 1982). Enamel fluoride-uptake from the resin was demonstrated in 1983 by Benton et al. The anti-caries effect of this fluoride-releasing resin was well recognised and modifications were made to enable testing of the resin as an orthodontic adhesive (Sansing et al 1984). Shear bond strengths were comparable to contemporary bonding adhesives (System 1® & Concise®) and the fluoride-release was calculated at 0.01mg/bonded surface (experimental duration of 30 days). The resin was further modified in order to decrease the setting time, increase the initial hardness and to improve the viscosity of the mix to prevent bracket drift (Bassett et al 1986). The amount of fluoride incorporated was also doubled. A near linear rate of fluoride-release (0.01mg/day) was maintained over a trial period lasting 12 weeks. In 1989,

Dental Fillings Ltd., England.

Ormco Corp., Glendora, Calif.
Underwood, Rawls and Zimmerman reported a clinical trial using the fluoride-exchanging resin as an orthodontic adhesive. The formulation of the composite follows:

1.3 % w/w Benzoyl peroxide
0.6 % w/w Dihydroxyethyl-paratoluidene
2.1 % w/w Bis-phenol-A dimethacrylate
22.5 % w/w Triethyleneglycol dimethacrylate
4.3 % w/w t-butylaminoethyl methacrylate hydrogen fluoride
11.7 % w/w Ethoxylated Bis-GMA
2.5 % w/w Fumed silica
55.0 % w/w Silica 1.6μm

The fluoride-releasing resin was compared to Concise®. Bracket failure rates were 10.8 per cent and 7.3 per cent respectively. Ten patients requiring orthodontic extractions consented to have brackets bonded to these teeth prior to treatment. The teeth were then extracted after 60 days. Those bonded with the fluoride-releasing resin demonstrated a 93 per cent reduction in the initial stages of demineralisation compared to those cemented with Concise®.

In recent years several commercial brands of fluoride-releasing resins have been tested for fluoride-release. Products examined include FluorEver® (restorative and orthodontic), Direct® (orthodontic only), Heliomolar®

*Ortho-Care, Bradford, West Yorkshire.
FluoroBond™ (orthodontic only).

**FluorEver® Restorative**

Cooley et al (1988) investigated Fluorever® light-cured restorative material for fluoride-release over a 6 months period. There was a rapid decline in fluoride-release during the first few days followed by a more gradual decline.

Temin and Csuros (1988) reported on the fluoride-release from FluorEver® (test duration of 4 years). A similar "burst effect" of fluoride-release as reported by Cooley et al (1988) was found. A steady state of fluoride-release (approximately 0.2µg/cm²/day) was reached after 8 months. The authors also carried out an 8 days fluoride-release study comparing FluorEver® to a silicate cement and a GIC (Ketac-Cem™). The average fluoride-release scores for the 8 days follow:

- FluorEver®: 9.9 µg F/cm²/day
- Ketac-Cem™: 10.2 µg F/cm²/day
- Silicate: 16.9 µg F/cm²/day

Temin, Csuros and Mellberg (1989) demonstrated that the fluoride released from FluorEver® was available for enamel uptake and caused an increased enamel surface fluoride concentration.

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Vivadent Inc.

Espe, Seefeld/Oberbay, W.Germany.
Swift (1989) looked at the fluoride-release from FluorEver® and Heliomolar® and compared the two composite resins to a glass ionomer cement (Ketac-Fil®) over a period of two weeks. The amount of fluoride released from FluorEver® and Ketac-Fil® were the same for the first day. Thereafter, the glass ionomer cement released more fluoride than FluorEver®. The pattern of fluoride-release was similar to previous studies (Cooley et al 1988, Temin and Csuros 1988). The fluoride-release from Heliomolar® was negligible, which was also supported by Forsten (1990).

FluorEver® Orthodontic Adhesive

Sonis and Snell (1989) conducted a clinical trial comparing FluorEver® against a conventional composite bonding system, Aurafill®. 22 patients were involved, with an average treatment period of 25 months. At the completion of active treatment, 26 teeth in the Aurafill® group demonstrated signs of decalcification (12.6%) whereas zero teeth bonded with the fluoride releasing resin exhibited decalcification.

Chan et al (1990) examined the in vitro fluoride-release (duration of 6 weeks) and bond strength of FluorEver®. The resin samples demonstrated a high early fluoride-release which decreased with time:

°Espe, Seefeld/Oberbay, W.Germany.
181 ± 25.9μg F/cm² on day 3.

1.5 ± 0.4μg F/cm² on day 43.

The fluoride-releasing resin also demonstrated a lower tensile bond strength than did Concise®. Microhardness testing showed that some of the FluorEver® resin which was located underneath the metal bracket bases, was not totally polymerised. According to the authors, this may have contributed to the weaker bond strengths.

**Direct® Orthodontic Adhesive**

Direct® is a hybrid composite resin and glass ionomer cement (acrylic resin with silico-alumino-phospho glass filler). Cook and Youngson (1989) showed that the shear-peel bond strength of Direct® was favourable when compared to the conventional composite resin, Right-On®.

Fox (1990) concluded that the fluoride-release from Direct® was very small and unlikely to have any clinical anti-caries effect. He also found that Ketac-Cem® stopped releasing fluoride after 10 weeks which contradicts most studies (Cranfield et al 1982). Fox assumed, perhaps incorrectly, that the test solutions would not become saturated and used this assumption to justify not changing the solutions.

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10TP Laboratories Inc., LaPorte, Ind.
FluoroBond® Orthodontic Primer

According to the manufacturer, FluoroBond® contains a boron tri-fluoride complex which allows ion exchange to occur, resulting in the release of fluoride into the environment. Capilouto (1988) demonstrated enamel-fluoride uptake in teeth which had been coated with the fluoride-releasing sealant.
CHAPTER 4

METHODOLOGY

4.1. ORTHODONTIC BONDING AGENTS

The following brands of light-cured orthodontic bonding resins and sealant were examined for their fluoride-ion-release in an aqueous environment.

1- FLUOROBOND® (see figure 1)

Fluorobond is a BIS-GMA-based light-cured orthodontic sealant that may be applied prior to bracket placement and/or around the brackets after placement. The manufacturer states that the resin contains a "methacrylate-boron trifluoride complex which allows a controlled release of fluoride for an extended period of time."

2- NORTHERN LIGHTS® (see figure 2)

Northern Lights is a BIS-GMA-based fluoride-containing visible light-activated bonding system. It consists of a bonding agent and adhesive paste.
3- LIGHT-BOND® (see figure 3)

Light Bond is also a BIS-GMA-based fluoride-containing visible light activated bonding system. The manufacturer claims that the fluoride is an "ion exchange organic fluoride, primarily in the monomer. However, as the paste also contains monomer in the packaging process the fluoride is also contained in the paste."

Unfortunately the local distributor for Reliance products provided me with a sample of Light-Bond® paste which, at the end of the study, proved to contain no fluoride. The non-fluoride release from the Light Bond® paste samples should be viewed in this light.

Figure 1. FluoroBond.
Figure 2. Northern Lights.

Figure 3. Light-Bond.
4.2. CHEMICALS

Fluoride standards were prepared from Orion 0.1000 ± 0.0005 moles/litre sodium fluoride solution. An ionic strength buffer (TISAB with CDTA) was used to dissociate metal fluoride complexes. 1 molar HCl was diluted with equal volumes of distilled-deionised water for use in the cleaning of all experimental plasticware. Distilled-deionised water was obtained through the Institute of Dental Research (United Dental Hospital of Sydney). 15 litres of distilled-deionised water was further distilled and deionised by being filtered through a Barnstead Water 1 system (Sybron Corporation, Boston, USA). This 15 litres of double-distilled deionised water was stored in a single 20 litre container and formed the sole source of water for the experiment and in the cleansing of the fluoride ion meter during analysis of fluoride concentration.

4.3. APPARATUS

* Plastic sterile containers were obtained through Bacto Laboratories, Sydney, Australia.
* All volumetric measurement was performed with a Pipetman Precision Air Displacement Pipette model number P5000.
* Orion Combination Fluoride Electrode model 9609-00 (Orion Research Inc., Boston, USA).
* Digital pH-Meter, model number PW 9409
(Phillips).

* Mettler balance (model H16, E. Mettler, Zurich).
* Qualtex Solidstat Incubator (Watson.Victor Ltd., Australia).
* Visilux 2 Visible Light Curing Unit, 3M.
* PTFM mould.
* Stop watch.

4.4. EXPERIMENTAL PROCEDURE

The methodology is primarily based on that used by Wilson et al (1967 & 1985) and Turek (1984), with some minor variations.

The possibility existed that the primers and pastes for Northern Lights® and Light-Bond® would contain different quantities of fluoride. If the components were not mixed in exact proportions and distributed evenly throughout the sample the possibility of a wide-ranging fluoride-release for the two brands would exist. For this reason it was decided to test primers and pastes separately.

Each sample was produced by placing the uncured resin into a polytetrafluoroethylene mould of diameter 7mm and height 3mm (see figure 4). A perspex sheet was placed on top of the mould and the sample was then polymerised for 20 seconds using a visible light curing source. The perspex sheeting was then removed and the sample was cured for a
further 20 seconds at which time the sample was removed from the mould, turned upside down and cured for a final 20 seconds to ensure polymerisation.

Figure 4. Polytetrafluoroethylene mould.

Five sample discs of each resin grouping were prepared according to the method described (see figures 5 and 6). For ease of labelling, the primer samples were designated the title of bond, eg Northern Lights® primer sample number one was labelled Northern Lights® Bond One (NB1). The resin groupings were as follows:

<table>
<thead>
<tr>
<th>FluoroBond sample number</th>
<th>1) F1</th>
<th>2) F2</th>
<th>3) F3</th>
<th>4) F4</th>
<th>5) F5</th>
</tr>
</thead>
<tbody>
<tr>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
</tbody>
</table>
2- NL primer sample number
   1) NB1
   2) NB2
   3) NB3
   4) NB4
   5) NB5

3- NL paste sample number
   1) NP1
   2) NP2
   3) NP3
   4) NP4
   5) NP5

4- LIGHTBOND primer sample no.
   1) LB1
   2) LB2
   3) LB3
   4) LB4
   5) LB5

5- LIGHTBOND paste sample no.
   1) LP1
   2) LP2
   3) LP3
   4) LP4
   5) LP5

6- Five 20 ml samples of double distilled de-ionised water serving as the control.

Figure 5. Five NL paste sample disks.

Figure 6. Resin samples (L to R) NP, NB, LB, LP and F.
Each sample was weighed using a Mettler balance. The samples were then tied with 100 per cent pure cotton which was sealed through a container lid with a cyanoacrylic acid ester glue. Each specimen was then placed in a separate container holding 20mls of double-distilled de-ionised water. The containers were then sealed and placed in an incubator at 37° Celsius (see figure 7).

Figure 7. Samples placed in Qualtrex incubator.
At the following times:*11
(day 1, day 2, day 3, week 1, week 2, week 4, week 8 and finally week 12)—
each sample was removed from the incubator, rinsed with
2mls of double-distilled deionised water (the 2mls rinse
solution was added to the liquid remaining in the
container, raising the volume to 22mls), allowed to air-
dry for 30 minutes (see figure 8) and then re-immersed in
20 ml of fresh solution. The new solution was then sealed
and replaced in the incubator. The old solution was sealed
and placed in a refrigerator awaiting assay.

Figure 8. Samples air-drying for 30 minutes.

*11These times represent experimental periods 1-8 respectively.
At the completion of the experimental period (12 weeks), each resin disc was air-dried for 30 minutes and reweighed. All test solutions were analysed for their fluoride ion concentration using a fluoride ion-specific electrode and digital pH meter.

A 2 mls aliquot of each solution was mixed with an equal volume of total ionic strength adjustment buffer (TISAB). The pH readings were recorded at precisely 2 minutes and then at intervals of 30 seconds until 4 minutes. The fluoride electrode was then rinsed with double-distilled deionised water until an "off-the-scale" reading was recorded. The electrode was blotted dry with tissue paper and now ready for the next recording.

Calibration of the electrode, using known fluoride concentrations, was performed at room temperature. The room temperature during assaying was maintained within one degree (Celsius) of that recorded during calibration. Calibration was performed twice daily.

The pH readings were plotted on Time Response Paper in order to establish the millivoltage reading at infinity. This reading was then used to establish the fluoride molarity from the calibration plottings on 4 cycle semi-logarithmic paper.
The fluoride-ion-release in micrograms was calculated using the following procedure:

Number of moles = Molarity × Volume (22 × 10⁻³ L)
Grams of fluoride = Molarity × 22 × 10⁻³ × Mol. weight F
μg's of fluoride = Molarity × 22 × 10⁻³ × 19 × 10⁶
" " " = Molarity × 4.18 × 10⁶

The reproducibility of readings was tested by assaying five replicate samples from a specimen selected at random. No variation in these recordings was found. (See Appendix 1).

All readings performed during the fluoride-ion analysis that gave a milli-volt reading less than -180mV were considered to represent nil fluoride presence.

4.5. STATISTICAL METHOD

The results were placed into the following similar time groupings for analysis:

GROUP 2 - Week 1 (accumulative)/ Week 2
GROUP 3 - Month 1 (accumulative)/ Month 2/
Month 3.¹²

¹²Month 1, 2, and 3 represent weeks 0–4, 4–8, and 8–12 respectively.
The data was analysed using an analysis of variance. A completely random design was used. Due to the heterogeneity of variances the recordings were transformed to logarithmic values. Individual comparisons between log means were made at the 5 per cent level using the least significant difference.
CHAPTER 5

RESULTS

5.1. FLUORIDE-ION RELEASE

The deionised water samples (and therefore the whole deionised water source) showed no measurable presence of fluoride. Fluoride-ion-release was demonstrated from all samples of FluoroBond®, Northern Lights® paste, and Light-Bond® primer. No detectable fluoride-release was recorded for Northern Lights® primer and Light-Bond® paste. Due to the lack of fluoride-release from Northern Lights® primer and Light-Bond® paste, it was deemed unnecessary to include their results in the statistical analysis.

Table 1. FLUORIDE-RELEASE: Means and Standard Deviations (micrograms)

<table>
<thead>
<tr>
<th>Sample (n=5)</th>
<th>FluoroBond®</th>
<th>Northern Lights® Paste</th>
<th>Light-Bond® Primer</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>sd</td>
<td>Mean</td>
</tr>
<tr>
<td>Day 1</td>
<td>376.2</td>
<td>41.8</td>
<td>135.5</td>
</tr>
<tr>
<td>Day 2</td>
<td>186.4</td>
<td>22.6</td>
<td>54.3</td>
</tr>
<tr>
<td>Day 3</td>
<td>161.4</td>
<td>28.6</td>
<td>36.8</td>
</tr>
<tr>
<td>Week 1</td>
<td>359.5</td>
<td>27.3</td>
<td>61.8</td>
</tr>
<tr>
<td>Week 2</td>
<td>182.2</td>
<td>16.1</td>
<td>42.6</td>
</tr>
<tr>
<td>Week 4</td>
<td>169.7</td>
<td>15.5</td>
<td>65.2</td>
</tr>
<tr>
<td>Week 8</td>
<td>217.4</td>
<td>15.6</td>
<td>148.0</td>
</tr>
<tr>
<td>Week 12</td>
<td>154.7</td>
<td>14.9</td>
<td>166.3</td>
</tr>
<tr>
<td>TOTAL</td>
<td>1807.5</td>
<td></td>
<td>710.5</td>
</tr>
</tbody>
</table>
Figure 9. Mean Fluoride-Release:

Experimental periods 1, 2, 3, 4, 5, 6, 7 & 8 correspond with samples taken at day 1, day 2, day 3, week 1, week 2, week 4, week 8 & week 12 respectively (see table 1).
Figure 10. Accumulative Mean Fluoride-Release
Table 2. MEAN DAILY FLUORIDE-RELEASE With Standard Deviations (µg) : [n=5]

<table>
<thead>
<tr>
<th>Time-Span</th>
<th>FluoroBond®</th>
<th>Northern Lights® Paste</th>
<th>Light-Bond® Primer</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>sd</td>
<td>Mean</td>
</tr>
<tr>
<td>Weeks 0-4 (month 1)</td>
<td>51.2</td>
<td>1.3</td>
<td>14.2</td>
</tr>
<tr>
<td>Weeks 4-8 (month 2)</td>
<td>7.8</td>
<td>0.6</td>
<td>5.3</td>
</tr>
<tr>
<td>Weeks 8-12 (month 3)</td>
<td>5.5</td>
<td>0.5</td>
<td>5.9</td>
</tr>
</tbody>
</table>

**MEAN F-RELEASE**

Month 1, 2 and 3

![Graph showing mean fluoride release over three months](image)

*Figure 11. Mean Daily Fluoride-Release.*
5.2. STATISTICAL ANALYSIS

The data was analysed using an Analysis of Variance (ANOVA). Due to the heterogeneity of the variances, the raw data was transformed to logarithmic values. The ANOVA gave highly significant F-values which allowed the utilisation of a Least Significant Difference test to determine individual treatment differences.

5.2.1. ANALYSIS OF VARIANCE (See Appendix 2)

The log value means and interaction means for Group 1 (day 1/2/3) were all significant at \( p < 0.001 \).

The log value means and interaction means for Group 2 (week 1/2) were all significant at \( p < 0.001 \).

The log value means and interaction means for Group 3 (month 1/2/3) were all significant at \( p < 0.001 \).

5.2.2. LEAST SIGNIFICANT DIFFERENCE (See Appendix 3)

The least significant differences at the 5 per cent level for the various log-converted means were as follows –:

- Group 1 - \( lsd = 0.169 \)
- Group 2 - \( lsd = 0.134 \)
- Group 3 - \( lsd = 0.19 \)

Using the \( lsd \) values, the following conclusions may be drawn–:

1) The log day 1, 2 and 3 means for FluoroBond™,
Northern Lights® paste and Light-Bond® primer were all significantly different at the 5 per cent level.

2) The log weeks 1 and 2 means for FluoroBond®, Northern Lights® paste and Light-Bond® primer were all significantly different at the 5 per cent level.

3) The log monthly means for FluoroBond®, Northern Lights® paste and Light-Bond® primer were all significantly different at the 5 per cent level except for the 3'rd monthly mean of FluoroBond® and Northern Lights® paste.

5.3. PRE- and POST- TRIAL WEIGHT DIFFERENCES

(See Appendix 4)

No outstanding changes in weight were noted in the samples tested. The largest variation occurred in one disk of Light-Bond® Primer which registered a 5 per cent gain in weight. The mean percentage loss or gain in the total sample (25 disks) was 1.6 per cent with a standard deviation of 1.2 per cent.
CHAPTER 6

DISCUSSION

It would appear, with reference to recognised epidemiological studies, that fluoride exposure may cause a decrease in caries incidence. Depending on the source, concentration and frequency of fluoride application, reported decreases in DMFT vary between a 20-90 per cent reduction (Naylor & Murray 1989, p151).

The mechanism, or mechanisms, by which fluoride imparts its anti-caries action are, at this stage, not precisely known. There is general agreement, however, that the mode of action is not singular (Luoma et al 1986). Several proposed means of action have been outlined in Chapter 1 of the literature review.

In the past, the major anti-cariogenic effect of fluoride was related to an increase in the enamel surface fluoride concentration. The increased fluoride concentration decreased the solubility of the enamel which, when exposed to a lowered pH (during a cariogenic attack), would resist demineralisation (Brown et al 1977). This explanation is partly supported by the lower solubility of fluoroapatite when compared to hydroxyapatite but is not supported by the poor correlation between enamel fluoride content and caries incidence (Retief et al 1987, Nasir et al 1985).
Current opinion considers that the major anti-cariogenic effect of fluoride relates to its influence on enamel remineralisation (Silverstone 1983,p204, Borsboom et al 1985). During a carious episode, the pH of the juxta-enamel environment drops favouring the dissolution of the apatite structure. Remineralisation of the caries-affected enamel may occur without the presence of fluoride, provided the necessary substrates and an intact surface are present. When fluoride is present, however, this remineralisation process occurs more rapidly and to a greater extent. The resultant enamel surface layer may contain a higher fluoride content than it did previously (Koulourides et al 1980).

The presence of large concentrations of fluoride does not seem necessary for optimal remineralisation to occur. What appears to be important is that sufficient fluoride is present during a period of demineralisation, enabling rapid remineralisation to occur when a more favourable pH is reached. The fluoride available for remineralisation comes from external sources and from that incorporated in the demineralised enamel (Silverstone 1983.p204). The amount of fluoride that is considered "sufficient" or "necessary" for a fluoride-enhanced remineralisation has not, as yet, been established.

The caries risk of orthodontic patients is greatly increased due to the increased difficulty in removing
plaque. The nature of band and bracket attachment procedures may additionally leave areas that are uncleanable, such as overhangs and voids (Gwinnett & Ceen 1979). These areas demonstrate mature plaque which create a caries risk for even the "best patient".

Unfortunately, the visualisation of "white-spot" lesions is not an uncommon occurrence on the removal of fixed appliances in patients whose oral hygiene maintenance has been less than desirable. Although there is a gradual improvement in the appearance of these lesions with time, the aesthetic problem associated with the more severe areas may persist for more than five years following treatment (Ogaard 1989).

The use of fluoridated preventive programmes have demonstrated significant reductions in the incidence of enamel decalcification among patients receiving fixed orthodontic treatment (Dyer and Shannon 1982, Artun and Brobakken 1986, Geiger et al 1988). The effectiveness of such programmes appears to be related to the frequency of fluoride application and is thus reliant on patient co-operation (Stratemann and Shannon 1974).

Stratemann & Shannon (1974) found that only 50 per cent of the patients included in their study followed the instructions (daily use of 0.4% stannous fluoride gel) completely. It would therefore be ideal if a substance
could be permanently placed on the teeth that would allow a continuous and long-term fluoride-release. This would remove the reliance on patient co-operation and provide the optimal fluoride delivery system for protection against "white-spot" formation.

It is well-recognised that dental restorative materials and luting agents, ideally, should possess a long-term fluoride-releasing potential. This property is especially relevant to orthodontic cements and bonding materials. Presently, glass ionomer cement is the material of choice for the cementation of orthodontic bands. Glass ionomer cement releases fluoride long-term and has been shown to prevent caries from developing in adjacent enamel (Valk & Davidson 1987).

Unfortunately, the bond strength of glass ionomer cement is not adequate for use in the bonding of orthodontic brackets (Cook and Youngson 1988). Composite resins, using the acid-etch technique are currently the material of choice. Unfortunately, these resins have not possessed a fluoride-releasing potential until recently (Swift 1989).

In this study, FluoroBond®, Northern Lights® paste and Light-Bond® primer demonstrated fluoride-release over the 12 weeks period. Fluoride, however, was not released from the Northern Lights® primer and the Light-Bond® paste (as supplied).
The nature of the fluoride-release was similar to that described by previous studies on fluoride-releasing composite resins (Cooley et al 1988, Temin and Csuros 1988). There was the initial "burst effect" (Cooley et al 1988) with rapidly reducing daily-release values for the first week (Table 1, Figures 9 & 10, Appendix 5). This was followed by a more steady decline in fluoride-release for the following 11 weeks. Northern Lights™ paste followed this pattern except for the third month where a slight increase in the mean fluoride release was demonstrated (18μg).

FluoroBond® demonstrated the greatest mean fluoride release. The mean log fluoride-release was significantly greater than the other brands except for weeks 8–12 where the release from Northern Lights™ paste was not significantly different.

The mean log fluoride-release from the Northern Lights™ paste was significantly greater than that from the Light-Bond® primer for all readings.

When the average daily fluoride-release measurements (Table 2 & Appendix 5) were inspected it appeared that the fluoride-release from the Light-Bond® primer (0.8μg/day 4–8 weeks; 0.4μg/day 8–12 weeks) was negligible after 4 weeks. FluoroBond's® average daily release was very high for the first week (154.8μg) dropping to a 5.5μg daily
value between weeks 8-12. Northern Lights® paste had an initial daily average fluoride release of 41.2μg which decreased to a 5.9μg daily rate for weeks 8-12. The fluoride release from FluoroBond™ and Northern Lights® paste was similar between weeks 8-12.

The results are encouraging in view of the potential for significant fluoride-release from composite resins now and in the future. The methacrylate-boron trifluoride complex of FluoroBond™ appears to be more effective than the "ion-exchange organic fluoride" incorporated in the Light-Bond™. Unfortunately, the nature of the fluoride-incorporation in Northern Lights® could not be elicited.

It is difficult to compare results of in vitro fluoride-release studies because the methodologies vary quite considerably. The fluoride-release from dental materials is surface-area-dependent and unfortunately no uniform size and shape of samples are used in published studies. Fortunately, this study used the identical teflon mould that Turek (1984) used in a fluoride-release study of various glass ionomer cements.

It was therefore possible to compare the fluoride-release of FluoroBond®, Northern Lights® paste and Light-Bond® primer to that of Ketac-Fil® and Fuji Type 11#13 (see figure 12).

---

13G.C. Dental Industrial Corp. Tokyo, Japan.
Accumulative Mean Fluoride

Figure 12. Fluoride Release: Composite Resins vs. GIC's.

Fuji 11 & Ketac-Fil from Turek (1984)
FluoroBond® and Northern Lights® paste compared favourably with both glass ionomer cements. What is encouraging is that FluoroBond® had the highest accumulative fluoride-release. The average daily fluoride-release for weeks 8-12 was largest for Fuji Type II®. The average daily values for FluoroBond® and Northern Lights® paste, however, are greater than that for Ketac-Fil®.

It would therefore appear that certain composite resins have the capability to compare favourably with glass ionomer cements with regard to early fluoride-release. Long-term fluoride studies are required before further comment can be made.

It is difficult to extrapolate the in vitro fluoride-release findings of FluoroBond®, Northern Lights® and Light-Bond® to the clinical situation. The primary problem is that the quantities of the materials used intra-orally are very small. Secondly, will the resins behave differently when in contact (or mixed) with the paste and/or primer system that they are partnered with? The ultimate question, of course, is whether the fluoride-release is sufficient to protect the enamel surface, adjacent to the brackets, from decalcification, and if so, for how long? The effectiveness of these materials can only be determined by future studies.
CHAPTER 7

RECOMMENDATIONS

There is clear evidence that patients wearing fixed orthodontic appliances are at a greater risk of incurring enamel decalcification than a non-orthodontic control group. The use of daily fluoride-preventive measures, specifically daily fluoride rinses, have demonstrated marked reductions in "white-spot" formation.

Unfortunately, we can expect only half our patients to follow instructions completely. Therefore, it is simply not sufficient to instruct the patients to use a daily fluoride rinse and clean their teeth meticulously because we know that a fair proportion of them will not perform these tasks adequately. To counteract the lack of cooperation the following recommendations are put forth:

1- Increasing patient awareness concerning the risks of enamel demineralisation prior to the commencement of fixed appliance therapy.

2- Increasing the time spent on the maintenance of oral hygiene and compliance with fluoride-based preventive programmes.

3- The use of a daily fluoride rinse. (An 0.05 % neutral sodium fluoride solution should be sufficient).

4- The use of fluoride-releasing orthodontic
cements and bonding resins. Considering the difficulty in determining future patient co-operation with regard to oral hygiene performance etc., it would seem logical to use a fluoride-releasing bonding system on all patients. This study has revealed two products that are worthy of consideration.
CHAPTER 8

CONCLUSION

The hypothesis that fluoride is released from FluoroBond®, Northern Lights® and Light-Bond® has been confirmed. The fluoride-release from FluoroBond® and Northern Lights® paste was similar after 8 weeks. Before this time, the fluoride-release from FluoroBond® was significantly greater. The fluoride-release from Light-Bond® primer was negligible after 4 weeks. No fluoride was released from Northern Lights® primer and Light-Bond® paste (as supplied).

Long-term fluoride-release studies need to be performed on the fluoride-containing composite bonding resins and sealants that are currently available. The resins also require clinical testing (with and without supplementary preventive procedures) to determine their effect on the incidence of decalcification at the completion of fixed-appliance therapy.
APPENDICES

APPENDIX 1

Reproducibility of Recordings

<table>
<thead>
<tr>
<th>Sample</th>
<th>Calibration to $\omega$ (mV)</th>
<th>Molarity</th>
</tr>
</thead>
<tbody>
<tr>
<td>NF2</td>
<td>78</td>
<td>$1.1 \times 10^{-4}$</td>
</tr>
<tr>
<td>NF2</td>
<td>78</td>
<td>$1.1 \times 10^{-4}$</td>
</tr>
<tr>
<td>NF2</td>
<td>78</td>
<td>$1.1 \times 10^{-4}$</td>
</tr>
<tr>
<td>NF2</td>
<td>78</td>
<td>$1.1 \times 10^{-4}$</td>
</tr>
<tr>
<td>NF2</td>
<td>78</td>
<td>$1.1 \times 10^{-4}$</td>
</tr>
</tbody>
</table>
APPENDIX 2

**Analysis of Variance (ANOVA)**

**INDEX—**

DF- Degree(s) of freedom.

SS- Sum of squares.

MS- Mean squares.

F- F-value.

P- Probability.

1- ANOVA for Days 1, 2, and 3 (log values)

<table>
<thead>
<tr>
<th>SOURCE</th>
<th>DF</th>
<th>SS</th>
<th>MS</th>
<th>F</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day</td>
<td>2</td>
<td>11.7966</td>
<td>5.8983</td>
<td>341.56</td>
<td>0.000</td>
</tr>
<tr>
<td>Sample</td>
<td>2</td>
<td>47.9576</td>
<td>23.9788</td>
<td>1388.55</td>
<td>0.000</td>
</tr>
<tr>
<td>Day/Sample</td>
<td>4</td>
<td>0.5055</td>
<td>0.1264</td>
<td>7.32</td>
<td>0.000</td>
</tr>
<tr>
<td>Error</td>
<td>36</td>
<td>0.6217</td>
<td>0.0173</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>44</td>
<td>60.8814</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### 2- ANOVA for Week 1 (accum1.) and Week 2 [log values]

<table>
<thead>
<tr>
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<th>DF</th>
<th>SS</th>
<th>MS</th>
<th>F</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Week</td>
<td>1</td>
<td>20.4035</td>
<td>20.4035</td>
<td>1938.37</td>
<td>0.000</td>
</tr>
<tr>
<td>Sample</td>
<td>2</td>
<td>24.3843</td>
<td>12.1922</td>
<td>1158.28</td>
<td>0.000</td>
</tr>
<tr>
<td>Week/Sample</td>
<td>2</td>
<td>0.6714</td>
<td>0.3357</td>
<td>31.89</td>
<td>0.000</td>
</tr>
<tr>
<td>Error</td>
<td>24</td>
<td>0.2526</td>
<td>0.0105</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>29</td>
<td>45.7118</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### 3- ANOVA for Months 1 (accum1.), 2 & 3 [log values]

<table>
<thead>
<tr>
<th>SOURCE</th>
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<th>SS</th>
<th>MS</th>
<th>F</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Month</td>
<td>2</td>
<td>30.589</td>
<td>15.295</td>
<td>705.46</td>
<td>0.000</td>
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<td>Sample</td>
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<td>46.329</td>
<td>23.164</td>
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<td>0.000</td>
</tr>
<tr>
<td>Month/Sample</td>
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<td>0.976</td>
<td>45.03</td>
<td>0.000</td>
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<tr>
<td>Error</td>
<td>36</td>
<td>0.780</td>
<td>0.022</td>
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<td></td>
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<tr>
<td>Total</td>
<td>44</td>
<td>81.604</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
APPENDIX 3

Least Significant Difference (1sd)

\[ 1sd = t_{\alpha/2} \sqrt{\frac{EMS \times 2}{\text{No. of replicates}}} \]

where EMS = error mean square.

and edf = error degree freedom.

1- \[ 1sd \] for Days 1, 2, and 3 [log values]

\[ 1sd = t_{5\%} \sqrt{0.0173 \times 2/5}, \text{ where } t_{5\%} = 2.03 \]

\[ 1sd = 0.169 \text{ at the 5 per cent level.} \]

<table>
<thead>
<tr>
<th>SAMPLE TYPE</th>
<th>DAY 1</th>
<th>DAY 2</th>
<th>DAY 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>FluoroBond</td>
<td>5.9252</td>
<td>5.2224</td>
<td>5.0709</td>
</tr>
<tr>
<td>NL-Paste</td>
<td>4.9081</td>
<td>3.9848</td>
<td>3.5921</td>
</tr>
<tr>
<td>LB-Primer</td>
<td>3.7123</td>
<td>2.6684</td>
<td>2.2519</td>
</tr>
</tbody>
</table>

Mean log fluoride-release scores for days 1,2 & 3.
2- *lsd for Week 1 (accuml.) and Week 2 (log values)*

\[ lsd = t_{24} \times \Phi (0.0105 \times 2/5), \text{ where } t_{24} = 2.064 \]

\[ lsd = 0.134 \text{ at the 5 per cent level.} \]

<table>
<thead>
<tr>
<th>SAMPLE TYPE</th>
<th>WEEK 1</th>
<th>WEEK 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>FluoroBond</td>
<td>6.9876</td>
<td>5.2021</td>
</tr>
<tr>
<td>NL-Paste</td>
<td>5.6623</td>
<td>3.7340</td>
</tr>
<tr>
<td>LB-Primer</td>
<td>4.5323</td>
<td>3.2980</td>
</tr>
</tbody>
</table>

Mean log fluoride-release scores for week 1 and 2.

3- *lsd for Months 1 (accuml.), 2 & 3 (log values)*

\[ lsd = T_{\Phi} \times \Phi (0.022 \times 2/5), \text{ where } t_{\Phi} = 2.03 \]

\[ lsd = 0.19 \text{ at the 5 per cent level.} \]

<table>
<thead>
<tr>
<th>SAMPLE TYPE</th>
<th>MONTH 1</th>
<th>MONTH 2</th>
<th>MONTH 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>FluoroBond</td>
<td>7.2689</td>
<td>5.3795</td>
<td>5.0376</td>
</tr>
<tr>
<td>NL-Paste</td>
<td>5.9769</td>
<td>4.9582</td>
<td>5.0939</td>
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<td>LB-Primer</td>
<td>4.9731</td>
<td>3.1361</td>
<td>2.4525</td>
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</tbody>
</table>

Mean log fluoride-release scores for months 1,2,3.
Figures 13, 14 & 15. Mean log fluoride-release.
APPENDIX 4

Pre- and Post-Immersion Weight Measurements.

<table>
<thead>
<tr>
<th>SAMPLE</th>
<th>BEFORE(g)</th>
<th>AFTER(g)</th>
<th>DIFFERENCE(g)</th>
<th>% CHANGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>F1</td>
<td>0.128</td>
<td>0.127</td>
<td>Loss 0.001</td>
<td>- 0.8</td>
</tr>
<tr>
<td>F2</td>
<td>0.125</td>
<td>0.122</td>
<td>Loss 0.003</td>
<td>- 2.4</td>
</tr>
<tr>
<td>F3</td>
<td>0.129</td>
<td>0.127</td>
<td>Loss 0.002</td>
<td>- 1.6</td>
</tr>
<tr>
<td>F4</td>
<td>0.126</td>
<td>0.123</td>
<td>Loss 0.003</td>
<td>- 2.4</td>
</tr>
<tr>
<td>F5</td>
<td>0.129</td>
<td>0.125</td>
<td>Loss 0.004</td>
<td>- 3.1</td>
</tr>
<tr>
<td>NB1</td>
<td>0.122</td>
<td>0.124</td>
<td>Gain 0.002</td>
<td>+ 1.6</td>
</tr>
<tr>
<td>NB2</td>
<td>0.113</td>
<td>0.115</td>
<td>Gain 0.002</td>
<td>+ 1.8</td>
</tr>
<tr>
<td>NB3</td>
<td>0.117</td>
<td>0.115</td>
<td>Loss 0.002</td>
<td>- 1.7</td>
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<tr>
<td>NB4</td>
<td>0.118</td>
<td>0.120</td>
<td>Gain 0.002</td>
<td>+ 1.7</td>
</tr>
<tr>
<td>NP1</td>
<td>0.260</td>
<td>0.260</td>
<td>Nil</td>
<td>Nil</td>
</tr>
<tr>
<td>NP2</td>
<td>0.253</td>
<td>0.254</td>
<td>Gain 0.001</td>
<td>+ 0.4</td>
</tr>
<tr>
<td>NP3</td>
<td>0.256</td>
<td>0.256</td>
<td>Nil</td>
<td>Nil</td>
</tr>
<tr>
<td>NP4</td>
<td>0.256</td>
<td>0.251</td>
<td>Loss 0.005</td>
<td>- 2.0</td>
</tr>
<tr>
<td>NP5</td>
<td>0.269</td>
<td>0.268</td>
<td>Loss 0.001</td>
<td>- 0.4</td>
</tr>
<tr>
<td>LB1</td>
<td>0.124</td>
<td>0.127</td>
<td>Gain 0.003</td>
<td>+ 2.4</td>
</tr>
<tr>
<td>LB2</td>
<td>0.122</td>
<td>0.126</td>
<td>Gain 0.004</td>
<td>+ 3.3</td>
</tr>
<tr>
<td>LB3</td>
<td>0.121</td>
<td>0.127</td>
<td>Gain 0.006</td>
<td>+ 5.0</td>
</tr>
<tr>
<td>LB4</td>
<td>0.124</td>
<td>0.127</td>
<td>Gain 0.003</td>
<td>+ 2.4</td>
</tr>
<tr>
<td>LB5</td>
<td>0.122</td>
<td>0.124</td>
<td>Gain 0.002</td>
<td>+ 1.6</td>
</tr>
<tr>
<td>LP1</td>
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<td>0.234</td>
<td>Gain 0.001</td>
<td>+ 0.4</td>
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<tr>
<td>LP2</td>
<td>0.236</td>
<td>0.238</td>
<td>Gain 0.002</td>
<td>+ 0.9</td>
</tr>
<tr>
<td>LP3</td>
<td>0.234</td>
<td>0.235</td>
<td>Gain 0.001</td>
<td>+ 0.4</td>
</tr>
<tr>
<td>LP4</td>
<td>0.239</td>
<td>0.241</td>
<td>Gain 0.002</td>
<td>+ 0.8</td>
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<tr>
<td>LP5</td>
<td>0.227</td>
<td>0.228</td>
<td>Gain 0.001</td>
<td>+ 0.4</td>
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</table>
APPENDIX 5

Mean Daily Fluoride-Release for Week-1 Accumulative with Standard Deviations (µg) : [n=5]

<table>
<thead>
<tr>
<th></th>
<th>FluoroBond®</th>
<th>Northern Lights Paste®</th>
<th>Light-Bond Primer®</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>154.8</td>
<td>41.2</td>
<td>13.3</td>
</tr>
<tr>
<td>sd</td>
<td>4.0</td>
<td>3.0</td>
<td>0.2</td>
</tr>
</tbody>
</table>
BIBLIOGRAPHY

Aasenden, R. Moreno, E. and Brudevold, F.

Adams, R.

Adriaens, M. Dermaut, L. and Verbeeck, R.

Arneberg, P. Ogaard, B. Scheie, A. and Rolla, G.

Artun, J. and Brobakken, B.

Artun, J. and Thylstrup, A.

Artun, J. and Thylstrup, A.

Backer Dirks, O. Kunzel, W. and Carlos, J.

Bassett, R. Zimmerman, B. and Rawls, H.

Beltran, E. and Burt, B.
Benton, J. Zimmerman, B. Rawls, H. and Turpin-Mair, J.
(1983) Enamel fluoride uptake from an experimental
919.

Bloom, R. and Brown, L.
(1964) A study of the effects of orthodontic
appliances on the oral microbial flora. J. Oral

Borsboom, P. Mei, H. and Arends, J.
(1985) Enamel lesion formation with and without 0.12

Bowen, R.
(1963) Properties of a silica-reinforced polymer for

Braden, M.
(1978) The formulation of composite filling

Brown, W. Gregory, T. and Chow, L.
(1977) Effects of fluoride on enamel solubility and
cariostasis. Caries Res. 11(Suppl.1):118-141.

Brudevold, F. Amdur, B. Vogel, J. and Spinelli, M.
(1965) Effect of ingested supplementary phosphate on
the tooth surface. J. Dent. Res. 43(Suppl):1168-1176.

Brudevold, F. Gron, P. and McCann, H.
(1965) Physico-chemical aspects of the enamel-saliva
system. Advances Fluorine Res. 3:63-78.

Buonocore, M.
(1955) A simple method of increasing the adhesion of
acrylic filling materials to enamel surfaces. J.

Capilouto, M.
(1988) A clinical study of a slow-release fluoride
tooth coating material. [Masters Thesis] Boston,
Massachusetts, Harvard School of Dental Medicine.

Chan, D. Swift, E. and Bishara, S.
(1990) In vitro evaluation of a fluoride-releasing

Ceen, R. and Gwinnett, A.
(1981) White spot formation associated with sealants

Charles, R.
29:1549-1553.
Cook, P. and Youngson, C.  

Cook, P. and Youngson, C.  

Cooley, R. Sandoval, V. and Barnwell, S.  

Cooper, V. and Ludwig, T.  

Corbett, J. Brown, L. Keene, H. and Horton, I.  

Cranfield, M. Kuhn, A. and Winter, G.  

Dean, H.  

Diedrich, P.  

Dijkman, A. Schuthof, J. and Arends, J.  

Douglas, J. DeVol, E. and Reid, S.  

Dyer, J. and Shannon, I.  
Ericson, T. and Ericsson, Y.

Ericsson, S.

Featherstone, J. Cutress, T. Rodgers, B. and Dennison, P.

Featherstone, J. Rodgers, B. and Smith, M.

Fitzpatrick, D. and Way, D.

Forrest, J.

Forss, H. Alakuijala, P. Seppa, L. and Luoma, H.

Forsten, L.

Forsten, L. and Paunio, I.

Fox, N.

Garcia-Godoy, F. Dodge, W. Dansby, M. and O’Quinn, J.


Jeansonne, B. and Feagin, F.  

Jerman, A.  

Kajander, K. Uhland, R. Ophaug, R. and Sather, A.  

Kilian, M. Larsen, M.Fejerskov, D. and Thylstrup, A.  

Kochavi, D. Simkin, A. Gedalia, I. and Laibu, E.  

Koulourides, T. and Cameron, B.  

Koulourides, T. Keller, S. Manson-Hing, L. and Lilley, V.  

Larsen, M and Bruun, C.  

Lee, H. Orlowski, J. and Kobashigawa, A.  

Lee Brown, C. and Way, D.  

Legler, L. Retief, D. and Bradley, E.  
Lehman, R. and Davidson, C.

Loesch, W.

Luoma, H. Fejerskov, O. and Thylstrup, A.

Magnesian, W. Shannon, I. and West, D.
(1979) Office-applied fluoride treatments for orthodontic patients. 58:1427.

Mattingly, J. Sauer, G. Yancey, J. and Arnold, R.

Mellberg, J.

Mellberg, J. and Mallon, D.

Mizrahi, E.

Mizrahi, E.

Nasir, H. Retief, D. and Jamison, H.

Naylor, M. and Murray, J.
Newbrun, E.

Newbrun, E.

Ogaard, B. Rolla, G. and Arends, J.

Ogaard, B. Rolla, G. Arends, J. and ten Cate, J.

Ogaard, B.

Oliver, R. and Howe, G.

O’Reilly, M. and Featherstone, J.

Pus, M. and Way, D.

Rawls, H. and Farris, C.

Retief, D. Harris, B. and Bradley, E.


Shannon, I. and West, D.  

Shen, C.  

Silverstone, L.  

Silverstone, L.  

Simon, M. and Geurtsen, W.  

Sinclair, P. Berry, C. Bennett, C. and Israelson, H.  

Soderholm, K.  

Soderholm, K. Zigian, M. Ragan, M. Fischlschweiger, W. and Bergman, M.  

Sonis, A. and Snell, W.  

Sonju, T.  

Stratemann, M. and Shannon, I.  

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Von der Fehr, F. Loe, H. and Theilade, E.  

Weitman, R. and Eames, W.  

Wilson, A. and Batchelor, R.  

Wilson, A. and Kent, B.  

Wilson, A. Groffman, D. and Kuhn, A.  

Wisth, P. and Nord, A.  

Zachrisson, B.  

Zachrisson, B. Heimgard,E. Ruyter, I. and Mjor, A.  

Zachrisson, B. and Zachrisson, S.  

Zachrisson, B. and Zachrisson, S.  