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Effect of Topacal C-5 on enamel adjacent to orthodontic brackets. An in vitro study

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Background: Enamel demineralisation leading to 'white spots' can occur during orthodontic treatment. Casein phosphopeptides may reduce demineralisation during orthodontic treatment.

Aims: To determine if a casein phosphopeptide preparation, Topacal C-5 (Enamel Improving Cream, NSI Dental Pty Ltd, Leighton, Hornsby, Australia), will inhibit demineralisation of enamel adjacent to orthodontic brackets.

Methods: Twenty-four pairs of human premolars from 24 subjects were used. One premolar in each pair was randomly assigned to the control group and the contralateral premolar was assigned to the experimental group. A stainless steel orthodontic bracket was bonded to the buccal surface of each tooth and a window of enamel (4 mm x 1 mm) left open to acid attack. The teeth were cycled alternately through an artificial saliva medium (11 hours) and an acid medium (1 hour) for 31 days. Topacal C-5 was applied to the exposed enamel windows in the experimental group after immersion in the acid medium. After 31 days the teeth were sectioned longitudinally and the depths of the enamel lesions measured by polarised light microscopy.

Results: Significantly deeper demineralisation occurred in the control teeth not protected by Topacal C-5 and at sites close to the brackets in both groups.

Conclusions: In this in vitro system, Topacal C-5 partially reduced the depth of enamel demineralisation compared with teeth not covered with Topacal C-5. Topacal C-5 may reduce enamel demineralisation in patients with fixed orthodontic appliances.

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Introduction

The prevalence of white spots in patients treated with fixed orthodontic appliances varies between 12 and 26 per cent.[1–3] The ability of fluoride compounds to prevent enamel demineralisation is well known.[4–8] For example, Basdera et al. reported that fluoride released from orthodontic bonding agents partially protected the enamel surface from demineralisation.[8] Topacal C-5, a milk protein-based formulation supersaturated with calcium and phosphate, has been shown to remineralise enamel lesions in humans.[9] An in vitro study also demonstrated that casein phosphopeptide – stabilised calcium phosphate solution (CPP-ACP) will remineralise subsurface lesions in human enamel,[10] and incorporation of CPP-ACP in a self-cure glassionomer cement will protect the dentine against acid attack.[11] Since no quantitative information has been reported on the effect of CPP-ACP on the enamel surrounding orthodontic brackets this study was designed to determine if Topacal C-5 inhibits enamel demineralisation adjacent to orthodontic brackets in vitro.

Materials and methods

Twenty four paired right and left non-carious human premolars with no visible enamel defects were used in this study. The teeth (N = 48) were extracted from 24 orthodontic patients living in a low fluoride area (0.4 ppm). The patients were between 10 and 14 years of age at the time of the extractions. Any soft tissue, calculus and/or bone remaining on the teeth following extraction were removed with a dental scaler and the teeth stored in deionised water until required.

One premolar in each pair was randomly assigned to the control group and the contralateral premolar was assigned to the experimental group. The buccal surfaces of the crowns were polished with a mixture of non-fluoridated pumice and water and masked with

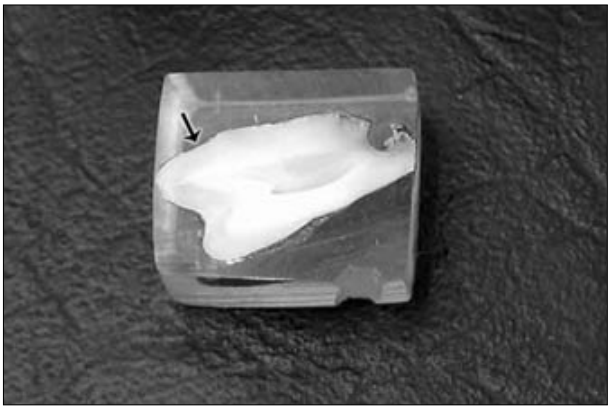

Figure 1. Longitudinal section of tooth embedded in polyester. The lesion is indicated by the arrow.

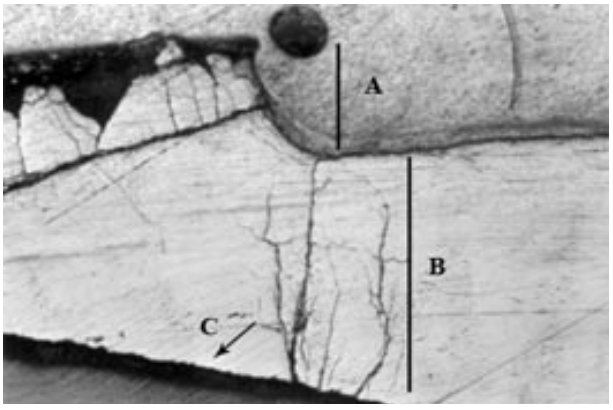

Figure 2. Enamel lesion in the control group viewed by polarised light microscopy. A, demineralised lesion; B, residual enamel; C, amelo-dentinal junction (4x magnification).

tape. A small window was left in the tape to accommodate a premolar bracket with 1 mm clearance on all sides of the base. A premolar stainless steel bracket (3M Unitek, Monrovia, California, USA) was bonded to the buccal surface of each tooth with a fluoride-free composite resin (Prime-dent, Dent World, Chicago, USA). The masking tape was removed and any residue of the tape wiped away with ethanol. Acid resistant nail varnish (Nivea, Beiersdorf, Hamburg, Germany) was then painted on the enamel surface leaving a rectangular window (4 mm × 1 mm) extending occlusally from each bracket.

The teeth in each group were immersed separately in an artificial saliva solution for 11 hours and an acid solution for 1 hour. Both solutions were agitated constantly and maintained at room temperature. The artificial saliva solution had a neutral pH and contained 20 mmol/L $KHCO_3$, 3 mmol/L KH2PO4 and 1 mmol/L $CaCl_2$. After 11 hours the teeth were removed and immersed in the acid solution for 1 hour. The acid solution contained 2.2 mmol/L Ca_2+, 2.2 mmol/L PO_4- and 50 mmol/L acetic acid at 4.4 pH. After each acid challenge, the surface layers in the exposed enamel windows in both groups were removed by brushing for 5 seconds with a soft toothbrush (Oral-B Laboratories, Belmont, California, USA). Topacal C-5 was applied to the exposed enamel in the experimental group before immersion in the saliva solution. The teeth were immersed alternately in the saliva and acid solutions for 31 days. The solutions were changed twice a week and the pH of each solution was monitored.

After 31 days the brackets were removed and the teeth mounted in polyester resin. The teeth were sectioned longitudinally through the buccal windows with a hard tissue microtome (Figure 1). Two sections, each approximately 0.5 mm thick, were obtained from each specimen. The sections were thinned and polished with increasing finer grades of aluminum oxide powder (Buehler, Evanston, Illinois, USA).

The sections were mounted in water and photographed with a polarised light microscope (Zeiss, Oberkochen, Germany) at 4x magnification (Figure 2). To facilitate measurement of the photographs a straight line was drawn between the intact enamel areas on the buccal surface of each specimen. The depths of demineralised enamel (μm) in each section were measured at three sites: near the gingival margin and close to the bracket, d1; in the middle of the demineralised area, d2; and near the occlusal margin, d3. The observer was blinded to the identity of the sections (i.e. experimental or control). The means and standard deviations of the depths at the three sites (d1, d2, d3) were calculated.

The *t*-test for unpaired data was used to compare the depths of the lesions in the control and the experimental groups and the paired *t*-test was used to compare the depths of the lesions at d1 and d3 in the same specimens. Statistical significance was set at the 5 per cent level.

Results

The results are given in Table I and Figure 3. Significantly deeper demineralisation occurred in the

Table I. Comparison of enamel demineralisation (μm) in the experimental and control groups.

Site		Experimental	Control	*p*
d1	N	24	24	**0.000**
	Mean	5.17	11.25	
	SD	3.77	3.10	
d2	N	24	24	**0.000**
	Mean	2.79	8.37	
	SD	1.82	3.16	
d3	N	24	24	**0.000**
	Mean	1.58	6.92	
	SD	1.21	3.68	

Significant values in bold
Experimental group, d1 versus d3, $p < 0.000$
Control group, d1 versus d3, $p < 0.019$

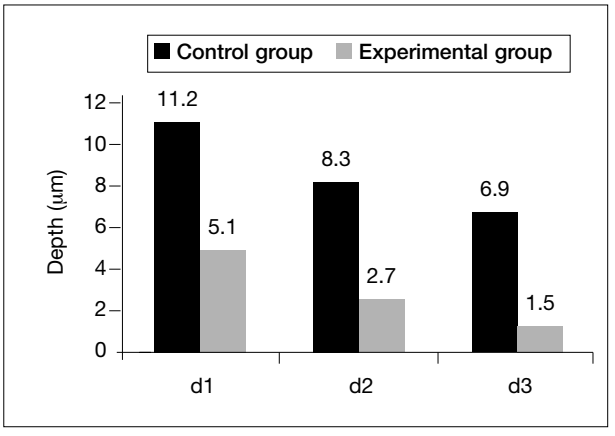

Figure 3. Mean depths of the enamel lesions in the control and experimental groups.

control teeth compared with the experimental teeth and at sites (d1) close to the brackets in both groups. In the experimental group the enamel was demineralised to a depth of 5.17 μm close to the bracket (d1), 2.79 μm in the centre of the demineralised area (d2) and only 1.58 μm at d3, where it was furthest from the base of the bracket. In the experimental group the difference between d1 and d3 was statistically significant.

Deeper demineralisation occurred in all sites in the control group compared with the experimental group. In this group demineralisation extended 11.25 μm into the enamel close to the bracket (d1), 8.37 μm in the mid-zone (d2) and 6.92 μm at d3. Furthermore, deeper demineralisation occurred at d3 in the control group than at d1 in the experimental group. In the control group the difference between d1 and d3 was also statistically significant.

Discussion

The results confirmed that regular applications of Topacal C-5 to teeth subjected to acid attack in vitro had less enamel demineralisation compared with teeth not treated with Topacal C-5. The deepest demineralisation occurred close to the bracket bases in both the Topacal C-5 treated teeth and untreated teeth. Casein phosphopeptide preparations, such as Topacal C-5, inhibit enamel demineralistion and enhance remineralisation of subsurface enamel lesions in vivo by creating a supersaturated zone of calcium and phosphate ions in dental plaque.[8–10] Although Topacal C-5 conferred partial protection against acid attack in our in vitro model, it still needs to be tested clinically.

Our finding that the deepest demineralisation in both groups occurred close to the bracket bases and the shallowest demineralisation occurred towards the occlusal surface may be due to morphological differences in the enamel, or it may be due to the different environmental conditions in our in vitro model. It could be postulated that fluid movement and/or the chemical conditions in our model reduced either the amount or the effectiveness of the Topacal C-5 close to the brackets. The former could be due to agitation and the latter to components in the artificial saliva, the bracket and/or the bonding agent. These factors require further investigation.

Regular applications of Topacal C-5 to sites at risk may prove to be an effective method of reducing the effects of demineralisation in susceptible patients, such as those with poor oral hygiene or salivary gland dysfunction.[4] However, Topacal C-5 is a milk product and should not be used on patients allergic to milk protein. The effectiveness of Topacal C-5 in preventing white spot lesions may be further enhanced if it is combined with fluoride-releasing cements, bonding materials and varnishes.[6] It has been postulated that fluoride-releasing materials may inhibit enamel demineralisation adjacent to orthodontic brackets by forming a protective deposit of calcium fluoride-like particles on the enamel surface.[8] Although these materials may not prevent

demineralisation they may reduce the formation of enamel lesions and remineralise subsurface enamel lesions.

Previous studies have reported white spot lesions in patients treated with fixed orthodontic appliances.[1–3] These lesions can occur rapidly in susceptible patients with fixed appliances. They invariably occur in areas on the tooth surface that are difficult to clean, and often on the labial surfaces of the incisors and canines. Materials such as Topacal C-5[9,10] and fluoride varnishes[6,7] that promote remineralisation of existing lesions or prevent small lesions from becoming larger, should be used if possible. In comparison with the effect of fluoride varnish on demineralised enamel adjacent to orthodontic brackets,[7] the casein phosphopeptide preparation we used also reduced demineralisation by more than 50 per cent.

Conclusions

In this in vitro study Topacal C-5 reduced the demineralisation of enamel adjacent to orthodontic brackets compared with teeth not covered with Topacal C-5.

Topacal C-5 may reduce enamel demineralisation in patients with poor oral hygiene or patients susceptible to enamel demineralisation, but it may not be appropriate in patients allergic to milk products.

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