EVALUATION OF SALIVARY FLORA ACIDOGENICITY UNDER ACIDIC CONDITIONS FOR PREDICTION OF CARIOGENIC POTENTIAL DURING FIXED ORTHODONTIC TREATMENT

Submitted in partial fulfilment to the degree of Doctor of Clinical Dentistry (Orthodontics)

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Abstract

Orthodontic treatment is a common occurrence with up to 29.7% of the adolescent population (Bollen, Cunha-Cruz *et al*., 2007) and 1% of the adult population (Whitesides, Pajewski *et al*., 2008) receiving fixed braces. This type of treatment poses significant risks to the hard and soft tissues. One of the most common complications of fixed orthodontic appliance treatment is the demineralization and subsequent white spot lesion development in the enamel (Travess, Roberts-Harry *et al*., 2004. White spot lesions are the early sign of dental caries and the incidence of white spot lesions in orthodontic patients has been reported as being as high as 50 per cent (Gorelick, Geiger *et al*., 1982; Lundström and Krasse, 1987; Lovrov, Hertrich *et al*., 2007) with white spot lesions sometimes occurring as early as 1 month after banding (Ogaard, Rølla *et al*., 1988).

Currently available chair-side saliva tests measure bacterial counts or acid production of the entire oral microflora. These tests tend to be able to predict patients who are at a low risk of demineralization more accurately than those at an increased risk. There is no single test to suit all individuals that can reliably identify at risk patients (Hausen, 1997;Reich, Lussi *et al*., 1999; Zimmer, Bizhang *et al*., 2008).

The aim of this short-term study was to evaluate a technique to predict white spot lesion development in patients undergoing fixed appliance orthodontic treatment, and to analyse salivary bacteria to determine any differences in their metabolism.

Fifty-two patients due to start fixed appliance orthodontic treatment agreed to participate in the study. Saliva samples collected before braces were placed

and during treatment at six-eight week intervals, were mixed with a potassium phosphate buffer solution containing sucrose (10% w/v), at pH 5.7, and rate of pH change was measured over 30 minutes. Demineralisation development was determined from standardized intra-oral photographs.

Subjects whose samples showed the greatest pH change towards acid production were selected for further salivary analysis. Ten of the higher risk individuals were further analysed, along with ten low risk individuals. Samples were grown on TSY20B plates, and pure strains of mutans streptococci were isolated and re-grown. These were then suspended in tryptone-soya broth and after 48 hours optical density, terminal pH and acid analysis using HPLC were measured and analysed.

Of the fifty-two participants, three developed demineralisation. Two were identified as high risk from their initial saliva test, one as low risk, giving the test a sensitivity of 67% and specificity of 94%. There was no statistically significant change over time in the subjects, which indicated the risk status is unlikely to change.

There was no statistically significant difference between the high and low risk groups in salivary microflora metabolism. The major acid produced in each case was lactic acid, with acetic acid being produced at lower concentrations.

This test has the potential to be developed into a commercial chair-side saliva test. However, further testing is continuing and aims to follow the cohort of patients through the entirety of their treatment.

Declaration

This work contains no material which has been accepted for the award of any other degree or diploma in any university or other tertiary institution to Sara Roberts and, to the best of my knowledge and belief, contains no material previously published or written by another person, except where due reference has been made in the text.

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Dr Sara Roberts

Dated

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