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Correlation between the PH of saliva, plaque and buffering capacity of saliva

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Abstract

Background of the study: pH of saliva and plaque will result in white spot lesions on the tooth surface which are considered initialization of caries because of demineralization. The buffering capacities of saliva and plaque will reduce the pH and result in the reduction of white spot lesions.

Aims of the study: To evaluate the correlation among the pH of saliva and plaque and buffering capacity of saliva.

Introduction: Dental caries is a disease where bacterial processes cause damage to the hard tooth structure, characterized by acid demineralization (represented by pH) of the tooth enamel. Changes to the microflora within the oral cavity result in an overgrowth of various bacteria like mutans streptococci and Lactobacilli that cause Dental Caries by producing acids.

Methodology: A study designed with 50 subjects from whom stimulated saliva samples and plaque were collected to measure the buffering capacity with using chair-side kits (CRT Buffer, Ivoclar Vivadent AG, Schaan, Liechtenstein) and pH with a chair side test strip, Hydriion (9800) Spectral 0-14 Plastic pH Strip (Micro Essential Laboratories, USA). The data were processed statistically with the Statistical Package for Social Sciences (version 20.0, SPSS Inc., Chicago, Illinois, USA).

Results: No significant correlation exists among the pH of saliva and plaque. Buffering capacity of saliva reduces the pH of saliva to some extent.

Conclusions: Buffering capacity of saliva reduces the pH of saliva to some extent, but have not much role in the reduction of pH of plaque.

Keywords: saliva, plaque, buffering capacity, lactobacilli

Introduction

Saliva is a biologic fluid in the oral cavity, composed of a mixture of secretory products from the major and minor salivary glands. Saliva plays key roles in lubrication, mastication, taste perception, prevention of oral infection and dental caries. Saliva is one of the most important factors in prevention of dental caries. Therefore, physical and chemical changes in saliva composition and particularly changes in its buffering capability play an important role on development and progression of caries [1]. By constantly bathing the teeth and oral mucosa with saliva functions as a cleansing solution, a lubricant, a buffer and ion reservoir of calcium and phosphate which are essential for re-mineralization of initial carious lesions.

Subjects and methods

Study Group

The group comprising of 50 healthy children (30 female, 20 males), 5–15 years of age, who had been screened for the dental caries and brief case history is recorded to rule out any drug allergies and systemic disorders, before the commencement of the study. The patients volunteered after verbal and written information, and informed consent was obtained from their guardian of the orphanage. Special children and those who were not willing to participate in the study were also excluded.

Stimulated saliva samples were collected to measure the buffering capacity, pH, Salivary samples were collected everyday between 9 and 10 am to avoid any diurnal variation. Plaque samples were collected to measure the pH and its buffering capacity. All the samples were collected under the supervision and guidance of the same clinician. A single clinician did the evaluation of the samples to overcome the inter-personnel bias.

Evaluation of salivary buffering capacity

The stimulated saliva collected was used to check the buffering capacity of saliva of all the children using chair-side kits (CRT Buffer, Ivoclar Vivadent AG, Schaan, Liechtenstein). The buffer strip is removed carefully from the packaging without touching the yellow field. Test strip is placed on a stable, absorbent paper with the yellow test field facing upwards. Saliva is transferred on to the strip with a small Pipette and after 5 min, there results are compared to the model chart provided by the manufacturer.

Evaluation of salivary pH

pH of saliva is estimated using a chair side test strip, Hydrion (9800) Spectral 0-14 Plastic pH Strip (Micro Essential Laboratories, USA). The strip is dipped into the saliva and the color of the strip change instantly. The resulting color is compared with the matching pH color chart provided by the manufacturer.

Evaluation of plaque pH

pH of plaque is estimated using the same chair side test strip, Hydrion (9800) Spectral 0-14 Plastic pH Strip (Micro Essential Laboratories, USA) similar to saliva. The collected plaque samples were transferred to sterile distilled water on a sterile glass slab and the strip is dipped into the plaque solution and the color of the strip change instantly. The resulting color is compared with the matching pH color chart provided by the manufacturer.

Results

Statistical Analysis

The data were processed statistically with the Statistical Package for Social Sciences (version 20.0, SPSS Inc., Chicago, Illinois, USA). P-value of less than 0.05 was considered statistically significant.

Table 1

pH of saliva with Buffering capacity of saliva	N=50	
	Mean	SD
	7.75	0.55
p-value	0.054	

*p<0.05, # applied dependent t test

Table 2

pH of plaque with Buffering capacity of saliva	N=50	
	Mean	SD
	6.39	1.05
p-value	0.5214	

*p<0.05, # applied dependent t test

Table 3

pH of saliva with pH of plaque	N=50	
	Mean	SD
	2.8	0.4
p-value	0.8604	

*p<0.05, # applied dependent t test

Discussion

Many studies showed a correlation between low salivary buffer capacity and caries.

Salivary buffer capacity, pH and Dental Caries:

The critical pH is the pH at which a solution is just saturated with respect to a particular mineral, such as tooth enamel. If the pH of the solution is above the critical pH, then the solution is supersaturated with respect to the mineral, and

more minerals will tend to precipitate out. Conversely, if the pH of the solution is less than the critical pH, the solution is unsaturated, and the mineral will tend to dissolve until the solution becomes saturated [2].

The concept of critical pH is applicable only to solutions that are in contact with a particular mineral, such as enamel. Saliva and plaque fluid, for instance, are normally supersaturated with respect to tooth enamel because the pH is higher than the critical pH, so our teeth do not dissolve in our saliva or under plaque. However, these fluids cannot be supersaturated with respect to individual ions, such as calcium or phosphate, as some authors state.

When HA is in contact with water, a small amount of HA dissolves, releasing calcium, phosphate and hydroxyl ions. This process continues until the water is saturated with respect to HA. At that point, the rate of the forward reaction (mineral dissolution) is equal to the rate of the backward reaction (mineral precipitation).

Plaque pH and Dental Caries

This new paradigm was a result of a concomitant reconsideration of the pathogenesis of dental caries lesions. Dental caries was always recorded in epidemiological studies as cavities [3]. As such, all clinical measurements (the DMFT/S) only comprised very late stages in the caries process. Thereby it was ignored that a clinically detectable lesion (even the non-cavitated white spot) is a result of innumerable pH fluctuations in the microbiota covering the enamel. The enamel surface is a sponge, which by no means is chemically inert. The constantly ongoing pH fluctuations taking place even in so-called 'resting plaque' and dramatically enhanced during exposure to fermentable carbohydrates will be associated with chemical exchange reactions between the tooth mineral and the surrounding plaque fluid.

Depending on the environmental conditions in the oral cavity, of the individual in general, or at specific sites within an individual, the physiological equilibrium between tooth and biofilm may be disturbed, resulting in a net loss of mineral. If a frank cavity is allowed to form, such a site represents an ecological niche where the biofilm composition gradually adapts to a declining pH environment

The production of acids by microorganisms within the dental plaque continues until the carbohydrate substrate is metabolized. It also is known that the plaque's pH goes from acidic to normal (or the resting level) within a few minutes and depends on the presence of saliva. This is due primarily to the carbonate and phosphate pH buffering agents in saliva. Thus, one can think of this process as being equilibrium. In essence, an equilibrium exists within the dental plaque whereby the pH of the plaque decreases each time the host ingests a snack or meal that contains fermentable carbohydrates; afterwards, the pH returns to the resting level because of saliva.

Stephan Haband, Engländer and colleagues reported plaque pH responses after plaque exposure to foods and beverages that include sucrose or other fermentable carbohydrates [4]. Within three to five minutes after such exposures, the pH of the plaque decreases below the so-called critical pH values of 5.5 and 6.0 for enamel and dentin, respectively, and demineralization of the underlying enamel or dentin is initiated. The duration of the demineralization depends on the time required for the pH of the plaque to increase above this lowered pH and is controlled primarily by the amount and composition of saliva. When the plaque's exposure to saliva is

restricted, the decrease in plaque pH is greater and the recovery period is longer than when normal exposure is allowed.

In addition to neutralizing the acids produced within the dental plaque, saliva also serves as the host's defense mechanism by repairing the demineralization that occurs when the plaque pH is below 5.5 to 6.0. Saliva's ability to remineralize enamel has been known for more than 40 years and has been the focus of investigations during the past 25 years⁵. Perhaps the first clinical evidence of the ability of saliva and the oral environment to remineralize enamel was reported by Backer Dirksin 1966.

According to the classification of WHO low caries risk group is one having 1.2-2.6 of DMF (t) index; while group having 4.6-6.5 of DMF(t) index is grouped into one with high caries risk [6].

There is clearly an association between low salivary buffer capacity and dental caries experiences. Patients having high caries risk have significant lower saliva pH compared to the patients with low caries risk; which is in accordance with the previous research.

Caries is caused because of the reduced pH in the oral environment resulting in increased demineralization. It's a known fact that microbes will release acid and result in increased pH, which in turn will be neutralized by the buffering capacity of saliva. As most of the microbes were preserved in the plaque and also the pH changes were more in the region of plaque accumulation, our study tried to correlate and observe the differences in salivary and plaque pH along with the buffering capacity of saliva using chair-side kit CRT Buffer manufactured by Ivoclar Vivadent AG, Schaan, Liechtenstein.

The findings must, for a number of reasons, be interpreted with caution. First, the sample size was limited and caries-associated bacteria in saliva should be regarded as an intermediate end point for caries. It remains unclear whether or not this really is beneficial for patients.

Conclusion

Buffering capacity of saliva reduces the pH of saliva to some extent, but have not much role in the reduction of pH of plaque.

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