Why Does Supragingival Calculus Form Preferentially on the Lingual Surface of the 6 Lower Anterior Teeth?

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SOMMAIRE

Selon de nombreux auteurs, le tartre sus-gingival a tendance à se former surtout sur la face linguale des 6 dents antérieures inférieures, parce que la salive qui provient des conduits submandibulaires adjacents est une source d'ions calcium et phosphate et que la diminution du CO_2, sous l'effet de l'apport de salive dans la bouche, entraîne une élévation du pH. Cependant, la phase liquide de la plaque dentaire est sursaturée par rapport à la teneur en phosphates de calcium du tartre, lequel a toujours tendance à se déposer, sauf après la consommation de sucre alors que le pH de la plaque dentaire peut descendre sous le seuil critique et que la phase liquide de la plaque devient non saturée. C'est toutefois sur la face linguale des dents antérieures inférieures que le pH de la plaque risque le moins de descendre sous le seuil critique, car la plaque est très mince à cet endroit et que la concentration en sucre après l'ingestion de sucres y est la plus faible alors que son élimination est la plus rapide. De plus, la rapidité de formation du film salivaire à cet endroit favorise l'élimination de tout acide formé dans la plaque et a aussi pour effet d'accroître l'apport d'urée salivaire, ce qui facilite l'alcalinisation de la plaque. Ces facteurs contribuent à la formation de courbes de Stephan de faible amplitude et de courte durée et fournissent une explication plus plausible du dépôt plus rapide de tartre sus-gingival sur la face linguale des dents antérieures inférieures.

Mots clés MeSH : dental calculus/etiology; dental calculus/prevention & control; dental plaque; surface properties

Cet article a été révisé par des pairs.

Calculus is mineralized dental plaque and mineralization can only occur if the fluid phase of plaque is supersaturated with the components of calculus. Saliva and plaque fluid are normally supersaturated with respect to various calcium phosphates, except when fermentable carbohydrates are being consumed, and thus most people are susceptible to calculus deposition, albeit at different rates. The degree of supersaturation of plaque fluid increases when its pH is high. This occurs in patients who are tube fed, as their plaque is not exposed to fermentable carbohydrates.[1] It also occurs in patients on dialysis for renal disease, as their salivary urea levels are high and the urea can be converted by plaque bacteria to ammonia, which increases plaque pH.[1,2] Both classes of patients are very susceptible to calculus deposition.[1]

All dentists and dental hygienists will have noted that supragingival calculus almost always occurs predominantly on the lingual surface of the 6 lower anterior teeth, with lesser amounts on the buccal surface of the upper molars. In contrast, there is little or no site specificity in the deposition of subgingival calculus.[3] The proportion of supragingival

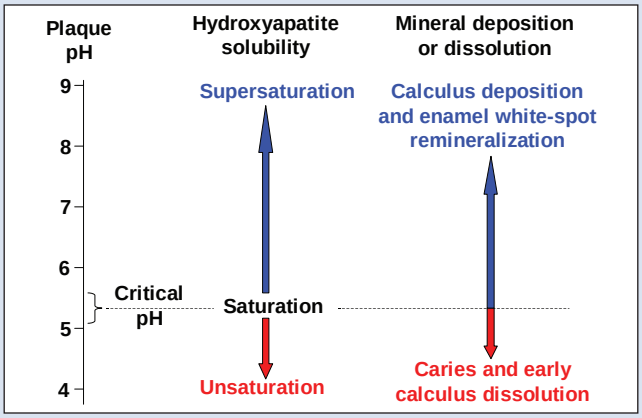

Figure 1: The effect of plaque pH, which may range from about 4–9, on hydroxyapatite solubility and on the tendency for mineral deposition or dissolution. At the critical pH, which varies slightly among individuals, plaque fluid is just saturated with respect to hydroxyapatite and there is no tendency for mineral to deposit or dissolve.

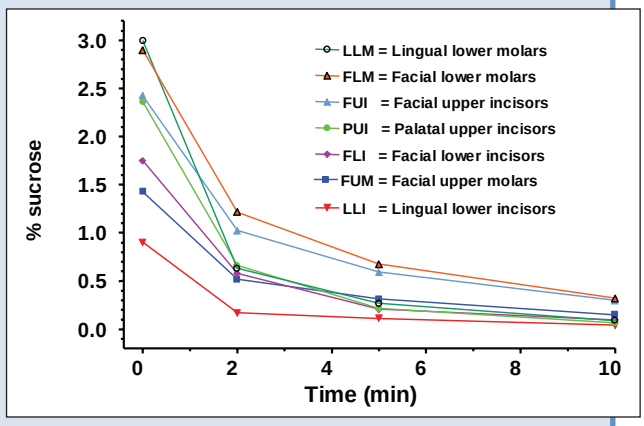

Figure 2: Mean changes in the percentage of sucrose in saliva at different sites in the mouth over 10 minutes beginning a few seconds after the mouth was rinsed for 1 minute with a 10% sucrose solution, and after consumption of a doughnut, mint candy or orange juice.

calculus on the lingual surface of the 6 lower anterior teeth has been reported to range from 63%[3] to 88%[4]; the amount of calculus on the lateral incisors and canines is 70.2% and 44.5%, respectively, of that on the central incisors.[5] Wirthlin and Armitage[6] and many previous authors have attempted to explain this site specificity on the basis that saliva leaving the submandibular–sublingual and parotid ducts provides a source of calcium and phosphate to dental plaque and that loss of CO_2 as the saliva enters the mouth increases the local pH. They state, correctly, that this will make the saliva more supersaturated with calcium phosphates, which might promote calculus deposition in dental plaque on the teeth in locations close to the salivary ducts.

However, as calcium and phosphate concentrations in plaque fluid are higher than those in saliva,[7,8] there is normally no concentration gradient to allow these ions to move from saliva into dental plaque. In addition, the loss of CO_2 into air present in the mouth is far from instantaneous, and one might expect that as saliva travels further away from the duct openings, more CO_2 will be lost and salivary pH will become even higher. If the explanation provided by Wirthlin and Armitage[6] were correct, more calculus would be expected at sites further from the salivary ducts. Thus their explanation for the site specificity of supragingival calculus deposition seems unlikely.

The Mineral Phase of Calculus and Its Solubility

The mineral phase of calculus is composed of calcium phosphate.[9] Although 4 different forms (dicalcium phosphate dihydrate, octacalcium phosphate, tricalcium phosphate and hydroxyapatite) have been detected,[10] hydroxyapatite is the predominant one[10] and the least soluble.[9] All are more soluble at an acid pH and less soluble at an alkaline pH. The concept of a critical pH for hydroxyapatite solubility has been discussed recently in this journal[11] and **Fig. 1** illustrates that above the critical pH, plaque fluid will be supersaturated with respect to hydroxyapatite, while below the critical level it will be unsaturated. In addition, when plaque pH is above the critical level, there is a tendency for calculus to deposit and for remineralization of white-spot enamel lesions to occur, although a considerable degree of supersaturation is necessary before these processes take place.[9] Conversely, when plaque pH is below the critical level, caries will tend to occur and any small calcium phosphate crystals that have not been integrated into the 3-dimensional network present in calculus will tend to dissolve. Thus calculus formation is most likely to occur when plaque pH remains well above the critical level for long periods.

Factors Influencing the Depth and Duration of the Stephan Curve

Exposure to Fermentable Carbohydrate

The main reason for a fall in plaque pH to below the critical level is exposure to fermentable carbohydrate, which allows the formation of acid by plaque bacteria. The fall in pH and its subsequent rise as acid is removed is termed the Stephan curve. The higher the carbohydrate concentration in the saliva over the plaque, the greater the amount that will diffuse into plaque and be available as a substrate for acid formation. Ingested sugar is not uniformly distributed in the saliva,[12,13] and **Fig. 2** shows that, after the intake of various sources of sucrose (doughnut, mint candy, orange juice

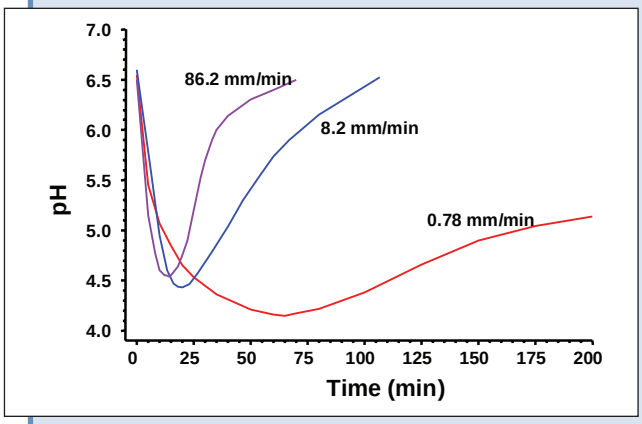

Figure 3: The effect of the velocity of a salivary film, 0.1 mm deep, on the Stephan curve developed in artificial plaque, 0.5 mm deep, after simulation of a rinse with 10% sucrose for 1 minute. The velocities of 8.2 and 0.78 mm/min are close to the maximum and minimum unstimulated rates, while that of 86.2 mm/min is about 10 times higher than the maximum when salivary flow is unstimulated. pH is the mean at the enamel surface of a plaque 6 mm in length.

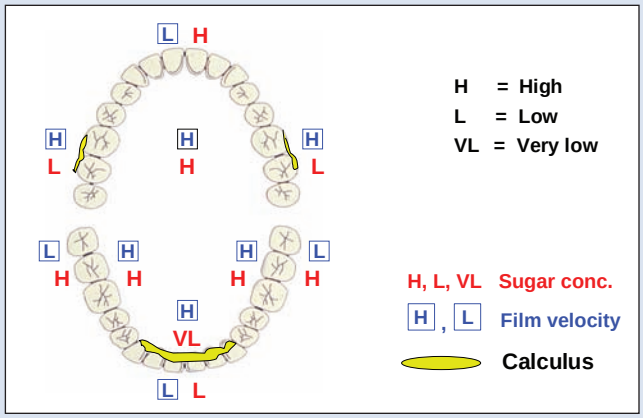

Figure 4: The site specificity of supragingival calculus deposition, the film velocity when salivary flow is unstimulated and the salivary sucrose concentration after consumption of sucrose from any source.

and a sucrose rinse), there are marked differences in salivary sucrose concentrations at different sites.[12,13] Particularly striking is the fact that sugar concentrations in saliva lingual to the lower incisors and, to a lesser extent, buccal to the upper molars are much lower than at the other sites tested; this occurred with all 4 products[13] and when gum containing sucrose was chewed.[12] Thus, plaque on the lingual surfaces of the lower anterior teeth will have the least amount of substrate available for acid formation compared with other locations in the mouth. Tube-fed patients will, of course, have no oral exposure to fermentable carbohydrate.[1]

Plaque Thickness

Computer modelling studies[14] suggest that very thin plaque layers, particularly those less than 0.5 mm, will develop only shallow Stephan curves on exposure to sugar. This is primarily because the acid formed in the plaque diffuses out into the overlying saliva at a rate that is inversely proportional to the square of the plaque thickness. Although no data appear to be available on plaque thickness at different sites in the mouth, my own clinical experience is that little plaque accumulates on the lingual surface of the lower anterior teeth compared with other oral sites, and thus plaque thickness there is very low.

Rate of Salivary Sugar Clearance

Not only is the initial salivary sugar concentration lowest on the lingual surface of the lower anterior teeth, but clearance there is also the most rapid (**Fig. 2**), presumably because of the proximity to the ducts of the submandibular salivary glands.

Salivary Film Velocity and Acid Clearance from Dental Plaque

The primary acids formed from sugar by dental plaque are lactic and acetic acids. For these to be removed from the plaque, they must diffuse out into the overlying film of saliva, which averages just under 0.1 mm in thickness,[15] and the salivary film must move across the plaque to keep acid from accumulating there and reducing the diffusion gradient for further acid removal from the plaque. The critical factor influencing the loss of acid from plaque is the velocity of the salivary film. This has been demonstrated in a computer model[16] and confirmed in a physical model[17] in which velocity of the salivary film over the surface of artificial plaque could be varied after exposure to sucrose. When salivary flow is unstimulated, the velocity of the salivary film varies from 0.8 to 7.6 mm/minute,[18] but even this relatively small range has a huge effect on the depth and duration of the Stephan curve. **Figure 3** shows Stephan curves for velocities close to the highest and lowest unstimulated levels (which occur lingual to the lower incisors and on the labial surfaces of the upper incisors, respectively) after exposure of plaque to a 10% sucrose rinse for 1 minute. The latter is deeper and much more prolonged than that associated with the highest unstimulated velocity. Although salivary film velocities on the lingual and palatal aspects of the teeth and on the buccal surfaces of the upper molars are greatly increased when salivary flow is stimulated by sugar consumption, within a minute of salivary sugar concentration falling below the taste threshold, the salivary flow rate falls to its unstimulated value. Thus, while the stimulated salivary flow rate will initially have a large influence on the rate of salivary sugar clearance, the

unstimulated flow rate will have a longer-term effect and will be particularly important for removal of acid from plaque. Because the highest salivary film velocity occurs lingual to the lower incisors, that is where plaque acid will be most readily removed.

Patients in whom salivary flow has been reduced by the action of their medications will presumably have a reduced salivary film velocity. It is of interest that such patients are less susceptible to calculus formation,[19] probably because acid is cleared less readily from their plaque.

Effect of Salivary Urea on Plaque pH

The urea in saliva diffuses into plaque and can be converted by certain bacteria into ammonia and CO_2, causing a rise in plaque pH.[20] The urea levels in saliva (about 3 mmol/L)[21] are such that, in the fasted state, the pH of plaque may be higher than that of the saliva flowing over it, particularly at sites with good access to saliva.[22] Using a physical model of plaque with ureolytic ability, it has also been shown that the pH of the plaque is positively correlated with the salivary film velocity at normal levels of salivary urea.[21] Plaque in regions of the mouth with better access to saliva also contains higher proportions of ureolytic bacteria,[23] which will facilitate development of a more alkaline plaque.

Figure 4 illustrates the site specificity of supragingival calculus deposition,[3,4] the salivary sugar concentration after consumption of different forms of sugar[12,13] and the salivary film velocity when salivary flow is unstimulated.[18]

Conclusions

In summary, several factors seem to explain why supragingival calculus forms most readily on the lingual surface of the lower anterior teeth. First, because plaque is thin in that region, any acid formed will diffuse out easily. Second, when sugar is ingested, its concentration is lowest there and is cleared most rapidly. Third, salivary film velocity is highest in that region, which promotes acid clearance from plaque. When sugar is ingested, all of these factors lead to the development of Stephan curves that are shallow and of short duration and, because the pH of the plaque lingual to the lower anterior teeth will be above the critical level for much longer than in other regions of the mouth, calculus will deposit there most readily. In addition, the high salivary film velocity will bring more urea to that region, leading to an elevated plaque pH.

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