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The acidogenic potential of plaque from sound enamel, white spot lesions, and cavities in children

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Abstract

An ion-sensitive field-effect transistor was used to monitor changes in pH following exposure of plaque samples to sucrose. A total of 90 plaque samples were removed from sound enamel, incipient white spot lesions, or cavities of 84 children and placed in a microholder. Sucrose (5% solution) was placed onto the plaque sample and the pH followed for 30 min by inserting the microelectrode into the plaque. Plaque samples from the white spot lesions showed faster decreases in pH and significantly greater quantities of acid were produced in comparison to the control plaques. These results demonstrate that the plaque found over white spot lesions in children has greater acid-producing activity than plaque from nondisease-associated sites.

Dental caries results from the production of acids from dietary fermentable carbohydrate by cariogenic plaque bacteria. Minah and Loesche (1977) demonstrated that plaque from carious tooth surfaces contained elevated levels of *Streptococcus mutans* and that the plaque fermented sucrose to acid more rapidly than plaque samples from control noncariogenic plaque. Duchin and van Houte (1978) analyzed for the presence of *S. mutans* and lactobacilli in plaque removed from incipient white spot lesions on buccal tooth surfaces and cavitated carious lesions on buccal and approximal tooth surfaces in children. The plaque from the carious sites frequently showed a 100-fold elevation in the presence of *S. mutans* compared to adjacent control sites.

The purpose of the present study was to compare the acid-producing potential of plaque from sound enamel, white spot lesions, and cavitated caries in children.

Methods and Materials

Human Subjects

Eighty-four subjects were selected at random from patients receiving periodic exams at the University Dental Hospital outpatient clinic. The patients ranged in age from 4 years, 4 months to 8 years, 7 months with a mean of 7 years, 1 month. Final diagnosis of sound enamel or white spot lesions without cavitation was made using a mirror and probe. White spot lesions were defined as slightly roughened, chalky areas not yet at the stage of clinically detectable cavitation (Duchin and van Houte 1978). Cavitated primary teeth had carious lesions not involving pulp.

Plaque Sampling and Acid Production from Sucrose

Plaque samples were removed from the buccal surface of maxillary permanent central incisors, lateral incisors or first molars and from the lingual tooth surface of mandibular first permanent molars. Also, plaque was obtained from the labial and buccal surfaces of primary maxillary anterior (from right canine to left canine) or primary second molars, or the lingual surface of mandibular second primary molars. A total of 90 samples were obtained from the various sites (Table 1).

In each procedure a small amount of plaque (~ 2.0 mg wet weight) was obtained using a sterile wooden stick and the plaque was transferred into a plastic tube[a] (inner diameter 1.0 mm; depth 1.0 mm) which had been sealed at the bottom with silicon rubber.[b] Approximately 5 µl of sterile sucrose (5% w/v in 10 mM phosphate buffer solution, pH 6.86 at 25° C) was added onto the plaque (Fig 1). A combination hydrogen-ion sensitive field-effect transistor electrode[c] was inserted slowly into the sucrose solution and then lowered into the plaque with the aid of a mechanical manipulator.[d] The pH responses were recorded continuously for 30 min at room temperature with a strip chart recorder[e] connected to a pH meter.[f]

[a] Applicator tube — Kuraray Co Ltd; Kurashiki, Japan.
[b] Silicon impression material — G-C Dental Industrial Corp; Tokyo, Japan.
[c] pH sensor, pH-2135 — Kuraray Co Ltd; Kurashiki, Japan.
[d] M-3 — Narishige Science Institute; Tokyo, Japan.
[e] R-2 — Rikadenki Kogyo Co Ltd; Tokyo, Japan.
[f] pH/pCO_2 Monitor, KR-500 — Kuraray Co Ltd; Kurashiki, Japan.

Table 1. Site and Number of Plaque Samples and Mean Age of Subjects

	Site of Sampling					
	Sound		White Spot		Cavity	
	Tooth Types	Number of Teeth Sampled	Tooth Types	Number of Teeth Sampled	Tooth Types	Number of Teeth Sampled
Permanent	7, 8, 9, 10	21	7, 8, 9, 10	10		
	3, 14	12				
	30, 19	10				
Primary	C, D, E, F, G, H	12			C, G, H, K	5
	A or J	10				
	T or K	10				
Number of samples:		75		10		5

Strip charts were used to calculate the change in pH (Δ pH) illustrated in Figure 2. The pH electrode touched the buffered sucrose solution and immediately provided a pH reading of 6.86. This stage was designated stage I. The electrode then was inserted into the plaque and the pH adjusted to the pH of the plaque (stage II). The pH then falls due to the fermentation of the sucrose (stage III). The difference in pH between stages II and III (Δ pH) was determined at 1-min intervals to determine the rate of acid production during the first 5 min and then at 5-min intervals.

The unpaired Student's *t*-test was used to evaluate the difference in pH between plaque from sound enamel and white spot lesions or cavities.

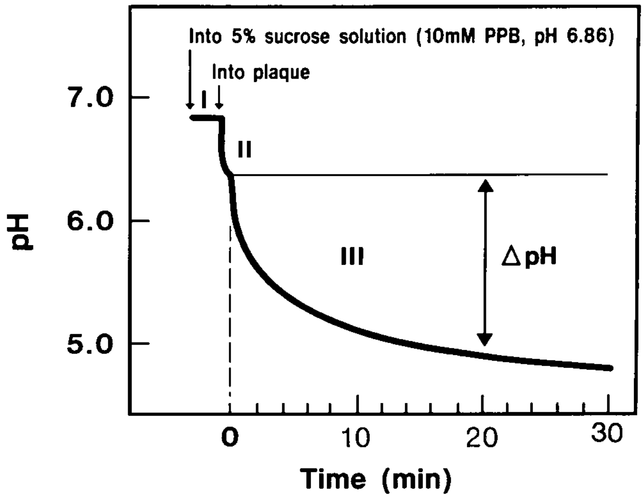

Fig 2. Representative plaque pH curve obtained with the micro-electrode-plaque sampling technique. The pH electrode touches the buffered sucrose solution (stage I); the electrode then is inserted into the plaque (stage II); and the pH falls due to the fermentation of the sucrose (stage III). The difference in pH between stages II and III (Δ pH) was determined.

Results

A typical plaque pH curve showed 3 distinct stages (Fig 2). In the first, or resting stage, the pH was 6.86 due to the buffer-sucrose solution. When the electrode was inserted into the plaque the pH normally dropped based on the individual plaque sample (stage II). When fermentation commenced the pH rapidly dropped (stage III).

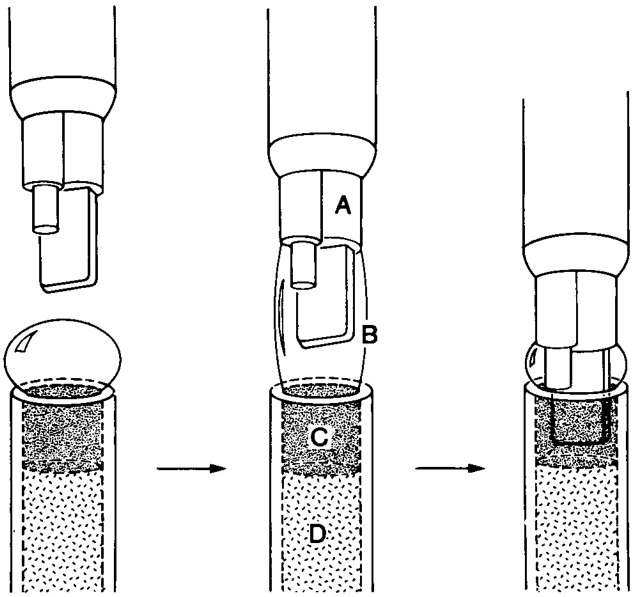

Fig 1. Schematic illustration of the method. Plaque (C) was put into a tube sealed at the bottom with silicon rubber (D), and a sucrose solution (B) was placed onto the plaque. A pH electrode (A) was moved down into the sucrose solution (B) and then into the plaque.

Table 2 (next page) presents results obtained with the plaque from the children's permanent teeth. The drop in pH during the first 2 min after exposure to sucrose was significantly greater in the plaque from the maxillary anterior white spot lesions when compared to the plaque from the maxillary anterior sound enamel. Beyond 2 min, acid production in plaque over white spots in anterior permanent teeth continued to be greater than that in plaque samples from sound anterior permanent teeth; however, the difference was not statistically significant. When the acid-producing potential of the white spot lesion plaque was compared to data from the plaque of teeth 3 or 14, the enhanced production of acid was significantly different for 4 min. It also was shown that acid production by plaque obtained from teeth 30 and 19 in the mandible is less rapid when compared to plaque ob-

TABLE 2. Acid Production by Resting Plaque from Permanent Teeth

Time (min)	*Source of Plaque Sample* Upper Anterior (White Spot)	Upper Anterior (Sound)	3 or 14 (Sound)	30 or 19 (Sound)
	Δ pH (mean ± SD)			
1	0.78 ± 0.35	0.45 ± 0.31	0.41 ± 0.24	0.22 ± 0.16
2	0.93 0.35	0.64 0.36	0.58 0.29	0.37 0.19
3	1.05 0.34	0.79 0.38	0.70 0.32	0.52 0.21
4	1.13 0.34	0.90 0.40	0.80 0.34	0.64 0.23
5	1.20 0.33	0.99 0.40	0.91 0.35	0.75 0.23
10	1.46 0.29	1.30 0.40	1.16 0.39	1.14 0.25
15	1.61 0.25	1.51 0.40	1.36 0.37	1.39 0.28
20	1.72 0.22	1.64 0.45	1.50 0.36	1.62 0.24
25	1.84 0.20	1.70 0.47	1.59 0.33	1.66 ± 0.14
30	1.87 ± 0.23	1.74 ± 0.37	1.84 ± 0.31	—

Statistical analysis: * $P < 0.05$, ** $P < 0.01$.

tained from all maxillary teeth after 1 min. This difference, except for teeth 3 and 14, was detectable for at least 4 min. Even after 10 min, there was a significant difference between white spot and mandible plaque.

When pooled plaque samples from primary teeth were analyzed for their acid-producing potential, it was found that maxillary posterior teeth A and J provided plaque which was much more active than plaque from maxillary anterior teeth and mandibular teeth T and K (Table 3). Acid production by the plaque from teeth A and J was very similar to production of plaque from the permanent dentition (Table 2). When plaque from carious lesions in primary teeth (Table 3) was analyzed for acid production, it was clear that this plaque had much greater potential than plaque from maxillary anterior and T or K sound enamel during the first 5 min of the experiment. Acid production by the cariogenic plaque was not significantly different from plaque from the maxillary posterior A or J sound enamel, and was similar to acid production observed with the plaque from white spot lesions in the permanent dentition (Table 2).

Discussion

Minah and Loesche (1977) used radioactive sucrose to demonstrate that the plaque from carious lesions can ferment the sugar to acid more rapidly than plaque from nondisease-associated sites. The results presented in this study extend these findings and demonstrate that the rate of acid production is greater in the plaque from white spot lesions than in that from the surface of sound maxillary anterior permanent teeth. Thus, the plaque over white spot lesions would respond more rapidly to the presence of dietary fermentable carbohydrate than plaque on sound enamel surfaces.

This study demonstrated that acid production is more rapid in plaque from maxillary teeth compared to permanent or primary mandibular teeth. This finding is consistent with the studies of Stephan (1944) and Kleinberg and Jenkins (1964) in which acid production was measured by touching electrodes to plaque in situ. This difference has been proposed (Kleinberg and Jenkins 1964) to reflect the flow and presence of saliva in the mouth. In the present study the plaque was removed from the mouth so that the influence of saliva was minimized.

Several studies have been performed on the microbiology of plaque obtained from incipient white spot lesions. Duchin and van Houte (1978) used a microsampling technique to obtain small samples of plaque from incipient white spot lesions and the surrounding sound enamel of children. The plaque from over the lesions was shown to contain markedly elevated levels of *S. mutans*. Recently, Boyer and Bowden (1985) and Milnes and Bowden (1985) demonstrated that the level of *S. mutans* and lactobacilli was at higher levels at sites where caries developed. It appears reasonable to project that the enhanced production of acid that the authors of this study observed in the plaque from white spot lesions reflected the presence of highly acidogenic cariogenic bacteria such as *S. mutans* and lactobacilli.

Plaque from decayed primary teeth produced more acid than from maxillary anterior and lower posterior sound enamel. However, there was no significant difference between maxillary posterior sound enamel and carious lesions. The reason might be that the data from decayed teeth included the maxillary and mandibular teeth and the number of decayed teeth were fewer than

TABLE 3. Acid Production by Resting Plaque from Deciduous Teeth

Time (min)	Upper Anterior (Sound)		A or J (Sound)		T or K (Sound)		C, G, H, or K (Decay)	Upper Anterior vs. C, G, H, or K
	Δ pH (mean ± SD)							
1	0.18 ± 0.14		0.33 ± 0.25		0.19 ± 0.14	*	0.64 ± 0.35	*
2	0.28 0.21	*	0.53 0.29	*	0.30 0.17	**	0.77 0.36	**
3	0.37 0.27	*	0.69 0.31	*	0.41 0.19	**	0.85 0.36	**
4	0.45 0.31	*	0.82 0.30	*	0.50 0.21	*	0.92 0.37	*
5	0.52 0.36	*	0.93 0.32	*	0.60 0.22	*	0.96 0.38	**
10	0.75 0.49	*	1.29 0.40	*	0.93 0.31		1.13 0.43	
15	0.92 0.57	*	1.59 0.36	*	1.12 0.34		1.54 0.16	
20	0.93 0.56	*	1.66 0.41		1.28 0.43		1.69 0.12	*
25	1.10 0.60	*	1.91 ± 0.13		1.63 0.41		1.77 ± 0.08	*
30	1.29 ± 0.71		—		1.73 ± 0.55		—	

Statistical analysis: * $P < 0.05$, ** $P < 0.01$.

that of maxillary sound enamel.

Since the production of acid by the test method in this study can be performed very quickly, it is possible that it could be used as part of a caries susceptibility test for sites in a child's dentition known to be prone to the development of carious lesions.

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Boyer RM, Bowden GHW: The microflora associated with the progression of incipient caries lesions in teeth of children living in a water-fluoridated area. Caries Res 19:298-306, 1985.

Duchin S, van Houte J: Relationship of Streptococcus mutans and lactobacilli to incipient smooth surface dental caries in man. Arch Oral Biol 23:779-86, 1978.

Kleinberg I, Jenkins GN: The pH of dental plaque in the different areas of the mouth before and after meals and their relationship to the pH and rate of flow of resting saliva. Arch Oral Biol 19:493-516, 1964.

Milnes AR, Bowden GHW: The microflora associated with developing lesions of nursing caries. Caries Res 19:289-97, 1985.

Minah GF, Loesche WJ: Sucrose metabolism in resting-cell suspensions of caries-associated and noncaries-associated dental plaque. Infect Immun 17:43-54, 1977.

Stephan RM: Intraoral hydrogen-ion concentration assessed with dental caries activity. J Dent Res 23:257-66, 1944.