Mineral-enriching Solution

Remineralization of Softened Bovine Enamel following Treatment of Overlying Plaque with a

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What is This?
Remineralization of Softened Bovine Enamel following Treatment of Overlying Plaque with a Mineral-enriching Solution

E. I. F. PEARCE and A. J. MOORE

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Pre-softened, gauze-covered bovine enamel blocks were worn in the buccal sulcus of five subjects for seven days. Artificial plaque enmeshed in the gauze was treated four times per day for four days with an enzyme-dependent mineralizing solution, resulting in 20-, 10-, and 200-fold increases in Ca, P, and F, respectively. Enamel beneath this mineral-enriched plaque recovered 37% of the hardness lost from pre-softening, while control enamel beneath untreated plaque recovered only 14%. Test enamel contained from five to 13 times as much F as did control enamel in the outer four layers sampled. Even though direct use of the mineralizing solution without the interposition of plaque caused a hardness recovery and F uptake similar to those in test enamel in vivo, a direct solution effect on enamel is not thought to explain the in vivo effects. Plaque treated with the urea-containing solution rapidly reaches a pH > 8, when a precipitate develops, leaving much-reduced ion concentrations in solution. Direct exposure of softened enamel to such a supernatant resulted in reduced hardness recovery and F uptake. It is concluded that the in vivo enamel remineralization was due mainly to the presence of a mineral phase in the overlying plaque. This mineral could have promoted remineralization by creating mildly supersaturated conditions during normal plaque pH cycles.


Introduction.

The concept that incipient enamel caries is a reversible process where periods of progression alternate with periods of remineralization is now well established (reviewed by Silverstone, 1977). Given an appropriate change in conditions, remineralization may even become the dominant process, leading to apparent repair of the lesion. Evidence for this phenomenon is found in the “reversals” which have been reported in longitudinal caries studies following changes in the diet (Scheinin et al., 1976), changes in oral hygiene (Koulourides and Axelsson, 1977), with topical fluoride therapy (Melsen et al., 1979), or for no manifest reason (Backer Dirks, 1966). Experimental lesions produced in vivo by inorganic acids (Sognnaes, 1963) or by rinsing accumulated plaque with sucrose (von der Fehr, 1965; von der Fehr et al., 1970) also regress with time and may completely disappear after conditions return to normal.

Certain histological features of carious enamel are also considered evidence for the natural occurrence of remineralization when conditions are favorable. These include the larger, more electron-dense “caries” crystals found at prism boundaries (Frank, 1967), the relatively-highly-mineralized surface zone and other laminations (Silverstone, 1977), and the dark zone seen by polarized light microscopy.

Extensive laboratory studies show that remineralization is accompanied by hardening of the enamel (Koulourides, 1968) and a reduction in pore volume, as evidenced by decreasing form birefringence (Johansson, 1965; Silverstone and Poole, 1969) and permeability to iodide (Brudevold et al., 1982). Other features are an increasing radiodensity of the lesion (Johansson, 1965) and an increase in the breadth of the dark zone (Silverstone and Poole, 1969). If fluoride is present, the remineralized enamel has increased resistance to subsequent acid dissolution (Feagin et al., 1971; Koulourides and Cameron, 1980; Koulourides and Housh, 1983).

Several methods have been devised to enhance remineralization by therapeutic means. Among the earliest were dicalcium phosphate-supplemented chewing gum (Pickel et al., 1965) and calcium sucrose phosphate solutions (Lilenthal et al., 1968). Inorganic calcium phosphate mouthrinse solutions with or without fluoride have been proposed and in a few cases tested clinically (Levine, 1975; Feasterone et al., 1982). However, maintenance of ion concentrations for sufficient time to allow remineralization is not easily achieved by this means of delivery (Zahradnik, 1979). A variety of slow-release materials has been proposed to effect a prolonged and controlled increase in the concentration of fluoride in saliva (Friedman, 1980; Mirth et al., 1982) and enamel (Abrahams et al., 1980; Rawls and Zimmerman, 1983). In general, the object is to employ moderately elevated concentrations of fluoride which, together with calcium and phosphate from saliva, will tip the balance in favor of remineralization.

Recently, a urea-containing mineralizing solution capable of producing substantial increases in the calcium, phosphate, and fluoride concentrations in dental plaque has been described (Pearce, 1981, 1984). These ions, fixed in plaque as fluor-hydroxypapitate, may act as a reservoir for remineralization of any adjacent porous enamel, although their release is not likely to be continuous. Rather, it would be intermittent, depending on the fall and rise of plaque pH. However, since calcium and phosphate as well as fluoride would be supplied, the remineralizing effect could be greater than with other slow-release materials.

The aim of the present study was to determine whether fluoridated apatite in plaque can promote the remineralization of contiguous softened enamel.

Materials and methods.

The experiment was carried out using the intra-oral caries test of Koulourides et al. (1974), as modified by Pearce (1981). Pre-softened and hardness-tested bovine enamel blocks with a surface area of 5 x 6 mm² were covered with six layers of a fine terylene gauze and mounted in appliances constructed to fit the lower dental arches of five adult subjects. Two blocks were used in each appliance and were positioned in the buccal sulcus adjacent to the permanent lower first molars.

The enamel blocks were cut from the labial surfaces of bovine permanent incisor crowns. After sterilization in ethylene oxide, approximately 100 μm of the outer enamel was removed by grinding with a carborundum-water slurry, and the surface was polished with diamond abrasives. The enamel was then hardness-tested and the block immersed in a gel containing 5% gelatin (dialyzed and de-ionized to remove fluoride) and 0.05 M lactic acid adjusted to pH 5.65 with NaOH. Only the polished enamel was softened in the gel; all other surfaces were protected. After 20 days at 5°C,
the blocks were recovered, washed in de-ionized water for one hr, hardness-tested again, and stored dry, ready for use.

Experimental procedure. — The appliance was first worn continuously for two days to establish plaque growth in the terylene gauze. On the following four days, it was removed from the mouth four times each day at intervals of two hr or longer, and plaque on one side only was immersed for ten min in 10 ml of a mineralizing solution. During this period, plaque and enamel on the contralateral side (control) were kept in a humid atmosphere. The appliance was then rinsed in tap water and replaced in the mouth. On the afternoon of the 7th day, the gauze-enamel blocks were removed from the appliance for analysis. Appliances were always worn at mealtimes and at night but were removed for oral hygiene, when a non-fluoride toothpaste was used.

The plaque mineralizing solution contained 3% urea, 20 mM CaCl2, 12 mM NaH2PO4, 4.72 mM Na2PO3F, and 0.28 mM NaF, plus coloring and flavoring agents, and was adjusted to pH 5.0 (Pearce, 1984). It was stored at 5° C but was heated to 37°C and held at that temperature during use.

In addition to the in vivo test and control enamel blocks, five pre-softened enamel blocks were exposed continuously to the pH 5 mineralizing solution in vitro, without the interposition of gauze or plaque, for an equivalent time, i.e., for 160 min. In a further control experiment, six other blocks were similarly exposed to the mineralizing solution after its pH had been raised to 8.3. A minimum volume of 2 M NaOH was used for the adjustment, and the resulting precipitate was removed by centrifugation. After treatment, all in vitro control samples were washed for one hr in de-ionized water, adjusted to pH 7-8 with NH4OH, and dried before analysis.

Analytical methods. — Enamel hardness testing was carried out with a miniload tester* equipped with a Knoop diamond. Using a 50 g load, a pre-determined pattern of 12 indentations was made over the surface. On re-testing, indentations were made in relation to the previous set. The change in average length of the 12 indentations was used as a measure of softening or hardening. Statistical significance was tested by the method of Welch (1947).

After removal of a 150-µm-thick section with a water-cooled diamond-impregnated blade, the block was coated with wax except for the polished surface, and the enamel was etched six times in 0.5 M HClO4. Etch times were 10, 10, 30, 30, 60, and 60 sec in 0.5, 0.5, 0.5, 1.0, and 1.0 ml of acid, respectively. Calcium in the etchant was determined by atomic absorption spectrophotometry, using a N2O/acylene flame and 3000 ppm K to suppress ionization. Phosphate was determined by a molybdate method (Chen et al., 1956) and fluoride with a specific ion electrode†. Fluoride concentrations were calculated on the basis that the enamel contained 36% Ca and 17.5% P.

For microphotography, the sections were ground to 50-60 µm thickness (Sundstrom, 1966) and radiographed on spectroscopic film* using soft X-rays‡.

The control and solution-treated plaque samples, enmeshed in terylene gauze, were used in fermentation experiments described elsewhere (Pearce et al., 1984). Methods for determining acid-extractable calcium, phosphate, and fluoride concentrations are reported therein.

Results.

Enamel sited in the mouth beneath plaque regularly exposed to the mineralizing solution regained, on average, 37% of the hardness lost from acid pre-softening (Table 1). Enamel beneath untreated control plaque regained only 14%. Enamel hardness, assessed by Knoop diamond indentation length, was significantly greater in test samples than in control samples at the end of the intra-oral exposure period (P < 0.001). The test and control samples were not significantly different initially, however, or immediately after acid-softening. All five subjects showed significant rehardening of their test enamel, but in one subject (AM), control enamel showed no significant change after in vivo exposure.

*Model 96-09, Orion Research, MA
†Type 649-0, Kodak, Rochester, NY
‡Softex CMR, Hosoda and Co., Tokyo, Japan

Table 1

<table>
<thead>
<tr>
<th>Enamel Beneath Plaque Exposed to Mineralizing Solution</th>
<th>Enamel Beneath Control Plaque</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Initial Hardness</strong></td>
<td><strong>After Acid Exposure in vitro</strong></td>
</tr>
<tr>
<td><strong>Subject</strong></td>
<td><strong>Length of Knoop Indentation (µm)</strong></td>
</tr>
<tr>
<td>EP</td>
<td>45.8</td>
</tr>
<tr>
<td>AM</td>
<td>45.7</td>
</tr>
<tr>
<td>AW</td>
<td>47.5</td>
</tr>
<tr>
<td>AP</td>
<td>47.9</td>
</tr>
<tr>
<td>JW</td>
<td>45.8</td>
</tr>
<tr>
<td><strong>Mean†</strong></td>
<td><strong>79.2a</strong></td>
</tr>
<tr>
<td><strong>SD</strong></td>
<td>1.1</td>
</tr>
</tbody>
</table>

*Mean of 12 indentation measurements in each case. Significance of the change in length: a, P < 0.01; b, 0.05 > P > 0.01; c, 0.1 > P > 0.05; and no superscript, P > 0.1.
†Significance of change in mean values calculated on the basis of matched pairs.
Test enamel contained approximately five times as much fluoride in the outermost layer as did control enamel, six times in the 2nd layer, 13 times in the 3rd layer, and six times in the 4th layer (Table 2). The exact cumulative biopsy depth could not be calculated from the analytical data because the density of the pre-etched, porous enamel was not known. Rather, the minimum cumulative depths of the etch samples (based on the density of sound enamel) are given for comparison of F levels in test and control enamel, on the assumption that the average porosity of the randomly-selected enamel blocks was similar. True depths could be up to twice the depth stated.

Control enamel exposed directly to the plaque mineralizing solution without the interposition of plaque, but for the same time as the in vivo samples, recovered 39% of the hardness lost from acid pre-softening when the pH of the solution was maintained at 5.0 (Table 3). However, the recovery was only 9% if the pH of the solution was first adjusted to 8.3 (Table 3). Mean indentation length was significantly greater, i.e., hardness significantly less, in enamel blocks treated with the pH 8.3 solution than in those treated with the pH 5.0 solution (P < 0.005). Removal of the precipitate that formed on raising the pH left 2.95 mM calcium, 0.71 mM orthophosphate, 4.87 mM monofluorophosphate, and 0.03 mM fluoride in the supernatant.

Exposure of pre-softened enamel to the pH 8.3 solution also resulted in significantly less F in the first (P < 0.001) and second (0.05 > P > 0.01) layers, compared with the respective layers in the pH 5.0 solution group (Table 4). The tendency for the F concentration to rise in the second layer in both groups was not statistically significant. Enamel exposed only to acidified gelatin showed a modest F gradient which leveled off at the 4th layer (Table 4).

Qualitative microradiographic examination of acid-softened but otherwise untreated samples showed a uniform radiolucent zone approximately 100 µm thick, with no radiopaque surface zone. No surface erosion was detected. The intra-oral and in vitro- treated samples showed essentially the same characteristics; no surface zone was seen to develop after mineralizing solution treatment.

Analysis of plaque enmeshed in the terylene gauze showed that acid-extractable Ca, P, and F increased in concentration 20-, 10-, and 200-fold, respectively, after exposure to the mineralizing solution (Table 5). The Ca/P (molar) ratio of the deposited mineral was 1.46 and the Ca/F ratio 16.6.

Discussion.

Enamel remineralization was assessed quantitatively in the present study by measuring changes in hardness and F uptake. Theoretical considerations suggest that, by itself, increase in hardness may be an inexact measure of mineral deposition. At one extreme, it may be possible to obtain a deposit of loose crystals adding nothing to enamel hardness, and at the other, a re-growth of existing crystals enhancing aggregation and tissue hardness with no net increase in mineral. Nevertheless, in practice, increase in mineral ion concentrations in softened enamel is reflected in increased hardness (Feagin et al., 1969; ten Cate and Arends, 1977). Similarly, on its own, F uptake may not necessarily indicate net mineral deposition. In fact, F uptake by enamel readily occurs simultaneously with demineralization (Koulourides et al., 1974; Pearce, 1983). However, when taken together, it is considered that F uptake reinforces hardness increase as evidence for remineralization. It is therefore concluded that the in vivo test enamel.

TABLE 2

<table>
<thead>
<tr>
<th>Enamel Beneath Test Plaque</th>
<th>Minimum Cumulative Sample Depth</th>
<th>Enamel Beneath Control Plaque</th>
<th>Minimum Cumulative Sample Depth</th>
</tr>
</thead>
<tbody>
<tr>
<td>F concn µg/mg</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Outermost enamel</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>First etch</td>
<td>1670 (598)</td>
<td>2.5 (0.2)</td>
<td>333 (85.5)</td>
</tr>
<tr>
<td>Second etch</td>
<td>1510 (339)</td>
<td>6.9 (0.5)</td>
<td>237 (94.6)</td>
</tr>
<tr>
<td>Third etch</td>
<td>1200 (449)</td>
<td>14.7 (1.6)</td>
<td>93.4 (44.1)</td>
</tr>
<tr>
<td>Fourth etch</td>
<td>311 (144)</td>
<td>28.7 (3.5)</td>
<td>49.2 (17.3)</td>
</tr>
<tr>
<td>Fifth etch</td>
<td>86.4 (42.2)</td>
<td>56.6 (5.9)</td>
<td>36.8 (16.3)</td>
</tr>
<tr>
<td>Sixth etch</td>
<td>49.0 (11.8)</td>
<td>95.6 (8.9)</td>
<td>34.2 (16.2)</td>
</tr>
</tbody>
</table>

Mean values from experiments in five subjects, S.D. in parentheses.

TABLE 3

<table>
<thead>
<tr>
<th>Effect on the Hardness of Bovine Enamel of Acid Exposure and Subsequent Direct Treatment with the Mineralizing Solution, Without the Interposition of Plaque</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial Hardness</td>
</tr>
<tr>
<td>-------------------</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Solution pH</td>
</tr>
<tr>
<td>unadjusted</td>
</tr>
<tr>
<td>n = 5</td>
</tr>
<tr>
<td>Solution pH</td>
</tr>
<tr>
<td>adjusted to 8.3</td>
</tr>
<tr>
<td>n = 6</td>
</tr>
</tbody>
</table>

*Precipitate that formed on pH adjustment removed by centrifugation prior to enamel exposure. Significance of change in length of mean values (S.D.) calculated on the basis of matched pairs. For significance levels, see Table 1.

Discussion.

Enamel remineralization was assessed quantitatively in the present study by measuring changes in hardness and F uptake. Theoretical considerations suggest that, by itself, increase in hardness may be an inexact measure of mineral deposition. At one extreme, it may be possible to obtain a deposit of loose crystals adding nothing to enamel hardness, and at the other, a re-growth of existing crystals enhancing aggregation and tissue hardness with no net increase in mineral. Nevertheless, in practice, increase in mineral ion concentrations in softened enamel is reflected in increased hardness (Feagin et al., 1969; ten Cate and Arends, 1977). Similarly, on its own, F uptake may not necessarily indicate net mineral deposition. In fact, F uptake by enamel readily occurs simultaneously with demineralization (Koulourides et al., 1974; Pearce, 1983). However, when taken together, it is considered that F uptake reinforces hardness increase as evidence for remineralization. It is therefore concluded that the in vivo test enamel.
TABLE 4

**FLUORIDE PROFILES IN ACID-SOFTENED BOVINE ENAMEL EXPOSED TO THE MINERALIZING SOLUTION WITHOUT THE INTERPOSITION OF PLAQUE**

<table>
<thead>
<tr>
<th>Acid-softened Enamel Subsequently Exposed to Mineralizing Solution</th>
<th>Solution pH = 5.0 n = 5</th>
<th>Solution pH = 8.3* n = 6</th>
<th>Enamel Subjected to Lactic Acid Gel Only n = 3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Minimum F concn µg/mg</td>
<td>Cumulative Sample Depth µm</td>
<td>Minimum F concn µg/mg</td>
</tr>
<tr>
<td>Outermost enamel</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>First etch</td>
<td>1560 (99.9)</td>
<td>2.7 (0.2)</td>
<td>880 (52.0)</td>
</tr>
<tr>
<td>Second etch</td>
<td>1720 (558)</td>
<td>8.9 (1.1)</td>
<td>967 (193)</td>
</tr>
<tr>
<td>Third etch</td>
<td>512 (304)</td>
<td>20.3 (1.7)</td>
<td>447 (204)</td>
</tr>
<tr>
<td>Fourth etch</td>
<td>112 (46.5)</td>
<td>36.2 (1.5)</td>
<td>93.5 (52.1)</td>
</tr>
<tr>
<td>Fifth etch</td>
<td>53.6 (12.0)</td>
<td>62.3 (3.2)</td>
<td>50.5 (20.7)</td>
</tr>
<tr>
<td>Sixth etch</td>
<td>41.2 (8.8)</td>
<td>98.6 (5.6)</td>
<td>39.7 (13.8)</td>
</tr>
<tr>
<td>Innermost enamel</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Precipitate that formed on pH adjustment removed by centrifugation prior to enamel exposure. Values are means from sequential etches in several samples, with S.D. in parentheses.

**TABLE 5**

**ACID-EXTRACTABLE MINERAL IONS IN PLAQUE**

| Plaque Exposed to Mineralizing Solution | Control Plaque |
| Calcium (µg/mg protein) | 193 (94.3) | 8.0 (5.6) |
| Phosphate (µP/mg protein) | 108 (47.8) | 10.2 (2.4) |
| Fluoride (µg/mg protein) | 11.2 (4.0) | 0.04 (0.01) |

Mean values of plaque samples from five subjects, with S.D. in parentheses.

samples and, to a significantly lesser extent, the *in vivo* control samples became partly remineralized during the experiment.

Artificially-softened enamel has been placed in the mouth in the course of several fluoride studies. Using indentation length or indenter penetration to measure hardness, recoveries of 13 to 20% (Arend security and Ghelhard, 1984), 25% (Koulourides et al., 1974), and 27 to 37% (Ghelhard et al., 1979) have been reported — values generally less than those obtained in the present study. However, fluoride concentrations in our softened test enamel were similar to the concentrations (1300 to 1800 ppm) found by Mellberg and Chomicki (1983) after topical application in *vivo*.

Plaque may normally serve as a reservoir of ions for the remineralization of enamel. Phosphate transfer from microorganisms has been implicated in enamel hardening (Luoma, 1975), while bound fluoride is released during carbohydrate-induced pH depressions (Birkeland and Martin, 1976) and would be available for remineralization. Plaque analyses showed that considerable amounts of Ca, P, and F were deposited in the test plaque on exposure to the mineralizing solution. X-ray diffraction of similarly-treated plaque has shown that these ions are present as apatite (Pearce, 1981), a mineral which is a potential slow-release reservoir for the remineralization of adjacent porous enamel.

Although the plaque-mineralizing solution is not intended to act directly on enamel, consideration must be given to whether the *in vivo* remineralization of plaque-covered enamel was due to such an effect, rather than to a transfer of ions from plaque mineral to enamel mineral, *i.e.*, an indirect effect of solution treatment. Direct precipitation of mineral into porous enamel during a urea-induced pH rise in plaque is considered unlikely. Most solution mineral ions would probably precipitate soon after diffusion into high-pH plaque — a supposition supported by the frequent finding of more mineral in outer plaque than inner plaque after solution treatment (Pearce, 1981, 1982). Thus, most precipitation should take place in plaque rather than in any adjacent porous enamel. On the other hand, the possibility of a direct effect of the supersaturated mineralizing solution on crystal growth and seeding in porous enamel, as distinct from a pH-induced precipitation, cannot be excluded, since *in vitro* controls showed that the pH 5 solution had a direct hardening effect that was equal to the effect when plaque was interposed. However, the pH 5 solution is not an adequate control to distinguish direct and indirect effects. Studies have shown that natural plaque exposed to a similar urea-containing mouthrinse rapidly attains a mean pH of 8.3 (Schamschula et al., unpublished), and at that pH, a considerable proportion of the mouthrinse Ca, P, and F is precipitated. This would leave much-reduced ion concentrations in solution at the plaque-enamel interface to effect direct remineralization during the rinsing period. Plaque-free porous enamel was therefore also exposed to the mineralizing solution after its pH had been adjusted to 8.3 and the resulting precipitate removed, in an attempt to approximate mineral growth conditions when plaque-covered enamel was exposed to the pH 5 solution. This resulted in much less hardening and lower F uptake as compared with the *in vivo* test enamel, suggesting that a direct crystal growth or seeding action of the mineralizing solution could account for only a small part of the *in vivo* enamel remineralization. But even this pH 8.3 solution was not an adequate control, because MFP present in the mineralizing solution may have been degraded to some
extent in plaque (Jackson, 1982), a factor not taken into account. Since the actual concentrations of free ions at the plaque fluid-enamel interface during solution exposure were not known, it was impossible to devise a completely adequate control solution. On the basis of these results, it is concluded that the enhancement of remineralization found in test enamel in vivo was due mainly to the presence of a calcium-phosphate-fluoride mineral phase in the overlying plaque.

In order to remineralize enamel with a crystalline deposit having the maximum caries resistance, evidence suggests that the level of supersaturation of the treatment solution should be kept as low as possible. First, it is axiomatic that a very slow rate of growth produces the most perfect crystals (Buckley, 1951). High levels of supersaturation result in masses of small, rapidly growing, defective crystals having an aberrant high solubility. Silverstone and coworkers (1981) have actually measured crystal size in remineralized enamel and have reported that lower calcium concentrations in their treatment solutions produced larger crystals. Second, at low levels of supersaturation, apatitic calcium phosphates can precipitate directly from solution without the formation of precursor phases such as amorphous calcium phosphate (Boskey and Posner, 1976) or octacalcium phosphate (Nancollas and Tomazic, 1974), both of which are more soluble than apatite. In addition, calcium fluoride, not strictly a precursor phase, is unlikely to form at low supersaturation levels. Third, highly unstable solutions may deposit mineral on the enamel surface rather than in the porous enamel constituting the lesion (Zahrnik, 1979), or may preferentially mineralize the outermost layer, clogging diffusion channels and preventing remineralization of the entire lesion (Johansson, 1965; Feagin et al., 1969; Silverstone, 1977). Solutions having low calcium concentrations are in fact not only effective in remineralizing the entire enamel lesion but also produce their most marked effect in the dark zone (Silverstone et al., 1981).

A difficulty with intermittently-applied enamel-remineralizing mouthrinse solutions is that this need for a low level of supersaturation must be balanced against the need for concentrations sufficiently high to enable ions to diffuse into porous enamel and then to form a significant deposit within the one-minute period the solution is likely to be in the mouth. It is well known that increasing the degree of supersaturation of a treatment solution does increase the rate of mineral deposition in enamel (Koulourides, 1968; Feagin, 1971; Moreno and Zahrnik, 1979), but for reasons already given, this mineral may not be the most suitable for enamel repair. On the other hand, slow-release materials or devices should be able to provide a continuous supply of calcium, phosphate, and fluoride at concentrations sufficient to enable ideal crystal growth conditions throughout the lesion. Fluoridatedapatite deposited in plaque from the enzyme-dependent mineralizing solution partly dissolves when the pH falls during the fermentation of carbohydrate (Pearce et al., 1984). It is hypothesized that the released ions create mildly supersaturated conditions on diffusion into the enamel lesion where a higher pH prevails, or when plaque pH rises again in its normal cycle. This event, although not continuous, is likely to occur more frequently than with conventional mouthrinse treatment and may be particularly effective because of the proximity of the ion source to the remineralization site. Indeed, visual inspection of the microradiographs revealed no tendency for the outer enamel to mineralize preferentially but indicated a uniform effect throughout the lesion depth, supporting this hypothesis of a slow release and mildly-supersaturated conditions. A two-step method in which an ill-defined fluor-hydroxyapatite is rapidly precipitated in plaque during a brief mouthrinse, followed by a transfer of the mineral to enamel, may in fact be the most practical way to boost enamel remineralization clinically.

Calcium, phosphate, and fluoride deposited in dental plaque have been shown to inhibit the development of experimental caries (Pearce, 1982). The present study indicates that they will also promote the remineralization of existing enamel lesions. However, it is not envisaged that, in any clinical application of this procedure, plaque accumulation would be encouraged. Rather, it is proposed that the procedure would be applied only to plaque that cannot be conveniently removed using normal oral hygiene techniques, e.g., plaque in pits and fissures and on approximal surfaces. Thus, teeth may be brushed and flossed in the usual way and the residual plaque then treated with a remineralizing solution. This mineral would have the dual functions of rendering plaque acids incapable of dissolving enamel and acting as a slow-release source for the remineralization of any adjacent carious enamel. Since pits, fissures, and approximal surfaces are the sites most susceptible to caries, such a procedure would be expected to confer significant benefits. Future work in our laboratories is planned to test this hypothesis further.

Acknowledgments.

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REFERENCES


