

## The effect of sealing white spot lesions on lesion progression in vitro

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### Abstract

*The resistance of white spot lesions to acid demineralization when etched with phosphoric acid and sealed with a resin material was studied. Artificially created white spot lesions were sealed and re-exposed to an artificial caries medium along with unprotected white spot lesions. Examination with polarized light microscopy revealed continued progression of the unprotected white spot lesions, whereas no lesion progression was evident under the resin sealant. New lesions formed on unprotected sound enamel adjacent to the margin of the resin sealant over sound enamel tangent to the white spot lesions. The sealed sound enamel adjacent to the white spot lesions did not undergo demineralization indicating protection by the resin tags. Sealing white spot lesions following etching with a 37% phosphoric acid may be beneficial in inhibiting lesion progression.*

Pit and fissure sealing, a process of acid etching enamel to create a surface acceptable for the mechanical bonding of a caries-resistant resin, has become an effective procedure for caries prevention (Horowitz et al. 1977; Houpt and Shey 1979). Sealants also have been shown to inhibit the progression of the carious lesion by reducing the number of cultivable microorganisms by 99.9% after 2 years (Handelman et al. 1976). Mertz-Fairhurst et al. (1979a, b) demonstrated arrested lesion progression via reduced microorganism viability, and confirmed inhibition of lesion progression by depth and radiographic measurements following sealant application. The solubility rate of sealed sound enamel when exposed to organic acids has been shown by Silverstone (1973) to be greatly reduced when compared to the solubility rates for sound enamel. When the clinically detectable bulk of sealant was lost, Hinding (1974) demonstrated a continued 83% caries reduction when compared to the contralateral control. Hicks and Silverstone (1982) also demonstrated the caries resistance of enamel following the loss of sealant

material, which they attributed to the presence of resin tags remaining in the enamel.

Etching of enamel with phosphoric acid in the concentration range of 30-40% (w/w) produces the most favorable surface for resin bonding. Silverstone et al. (1975) described the effects of etching on sound enamel as a 10- $\mu$ m surface layer loss, which removes the pellicle and plaque, leaving a porous enamel surface which exhibits 3 basic etching patterns. Silverstone (1975) reported that etching sound enamel results in resin tag lengths of approximately 50  $\mu$ m following the application of a sealant. A comparison of etching patterns on various tooth surfaces by Conlon and Silverstone (1982) revealed that the mesial and distal proximal surfaces showed the most uniform etch, demonstrating that these surfaces would produce a successful bond with resin materials.

During lesion formation, 4 distinct zones of enamel caries can be observed. The translucent zone and the body of the lesion represent areas of demineralization, whereas the dark zone and surface zone represent areas of remineralization (Silverstone 1973). The surface zone of the white spot lesion appears relatively unaffected when compared with adjacent sound enamel clinically. However, the surface zone has a pore volume of between 1 and 5% and is between 10 and 50 times more porous than sound enamel. Utilizing a 60-sec etch with 30% phosphoric acid on white spot lesions, Hicks and Silverstone (1984 a, b; 1985) produced an etching pattern of the surface zone which was similar to that seen in sound enamel. They demonstrated the possibility of rendering the surface zone porous while maintaining a relatively intact surface which might facilitate a more rapid mineralization of the enamel or be suitable for the placement of a resin material to prevent lesion progression.

The purpose of this study was to evaluate the effectiveness of placing a resin sealant over white spot lesions to arrest lesion progression.

## Materials and Methods

Fifteen caries-free human teeth were cleaned thoroughly with a fluoride-free pumice paste. Prior to exposure to the artificial caries system (Silverstone 1967), an acid-resistant varnish was applied to the teeth, leaving windows of sound enamel exposed on the buccal and lingual surfaces measuring 2 mm occlusocervically and 5 mm mesiodistally which were available for lesion formation. The teeth were suspended in a 15% acidified gel brought to pH 4.0 with the addition of lactic acid. Following 12 weeks of exposure to the acid gel, the teeth were removed. The white spot lesions on each surface then were divided into 3 segments: baseline lesion, re-exposed lesion, and sealed lesion by applying acid-resistant varnish over the middle one-third of the original window (Fig 1). One of the windows on each surface was enlarged and the surrounding enamel was etched for 60 sec with a 37% phosphoric acid gel. The etched area was rinsed for 30 sec and then dried for 30 sec. Then unfilled, light-cured resin sealant was placed over the etched white spot and surrounding etched sound enamel surface and cured

with a standard curing light for 40 sec. This window was further enlarged to expose sound enamel adjacent to the sealant. The other window with the white spot lesion was unaltered. The center of the original window which was covered with acid-resistant varnish preserved a portion of the original lesion to serve as a baseline. The teeth were then re-exposed to the acidified gel for an additional 6 weeks. Following the second exposure to the acidified gel, the teeth were removed, rinsed, and sectioned mesiodistally into buccal and lingual segments. The buccal and lingual segments then were sectioned buccolingually through the lesions and examined under polarized light to assess lesion depth and lesion progression. A total of 25 segments were available for examination — each consisting of baseline lesion, sealed lesion, progressed lesion, and new lesion components for comparison. The number of lesion components available varied from 22 to 24 due to loss during the sectioning process.

Lesion depths were measured on the sections imbedded in water with polarized light microscopy using a standardized grid. Lesion depth was measured from the outer enamel surface to the advancing front of the body of the lesion. Where irregular advancing fronts were encountered, the deepest portion was measured.

## Results

The mean lesion depths for each of the component lesions are listed in the Table. The general characteristics of each lesion component are as follows.

**Baseline lesions** — The baseline lesions achieved a mean lesion depth of 67  $\mu\text{m}$ .

**Sealed lesions** — The sealed lesions exhibited a mean lesion depth of 60  $\mu\text{m}$ .

**Progressed lesions** — The lesions re-exposed to the acid gel achieved a mean lesion depth of 100  $\mu\text{m}$ . This reflects a mean increase in lesion depth of 51% following the second acid challenge.

**New lesions** — New lesions that developed in sound enamel adjacent to the sealant achieved a mean depth of 57  $\mu\text{m}$ .

All lesions previously described exhibited the 4 classic zones of enamel caries as described by Silverstone (1973). Values for lesion depths were analyzed using a 2-way analysis of variance.

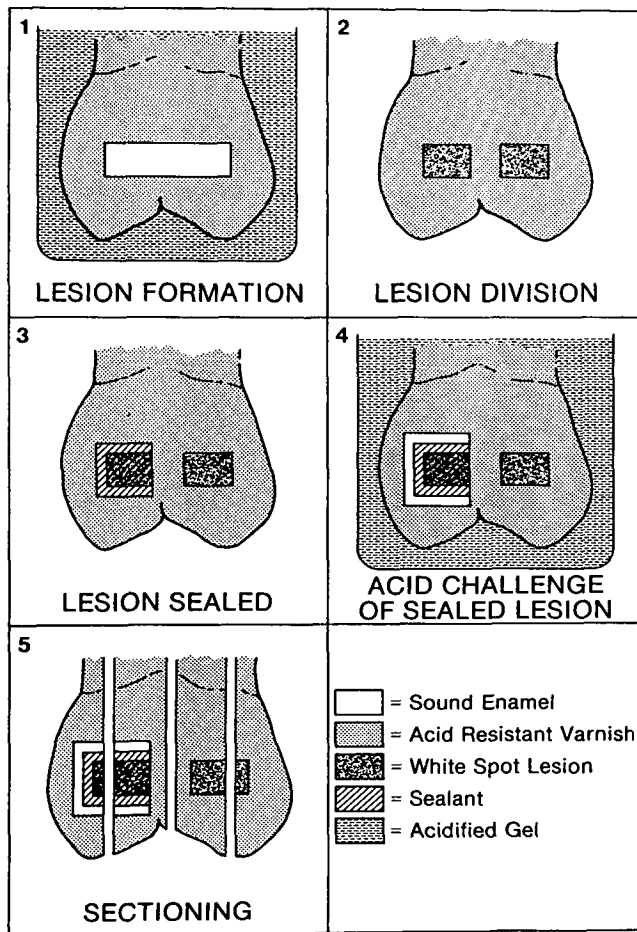


FIG 1. Schematic representation of experimental design.

TABLE. Mean Lesion Depths

	N	Mean Lesion depth ( $\mu\text{m}$ )	SD ( $\mu\text{m}$ )
Baseline lesions	24	67	13
Sealed lesions	22	60	13
Progressed lesions	23	100	18
New lesions	22	57	13

## Discussion

The depths of the baseline lesions and the sealed lesions did not differ significantly ( $P = .05$ ) from one another, which indicates that the white spot lesions that were protected with the sealant did not progress when subjected to further exposure to the acid gel. The unprotected lesions that were re-exposed to the acid gel progressed by an additional  $33 \mu\text{m}$  (51% depth increase), indicating that there was sufficient acid challenge to continue the demineralization process. The progressed lesions differed significantly from baseline and sealed lesions ( $P = .05$ ). Further indication of sufficient acid challenge to create enamel demineralization was the development of new lesions in sound enamel adjacent to the sealants. The new lesions reached a mean depth of  $57 \mu\text{m}$  during the 6-week acid exposure. In each case, the new lesion began on sound enamel at the junction of unprotected and sealed enamel, demonstrating the effectiveness of the resin tags in preventing acid demineralization of the underlying enamel (Fig 2).

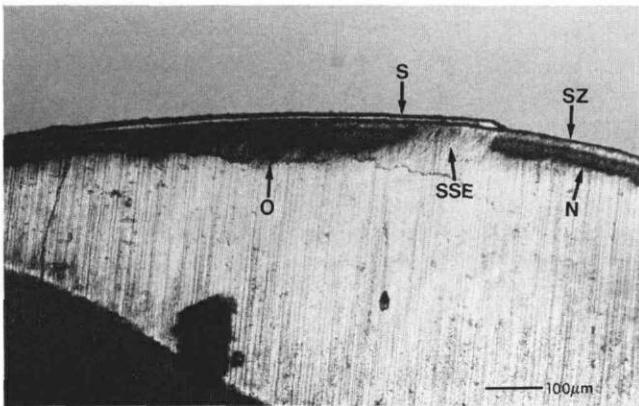


FIG. 2. Sealed lesion and new lesion following re-exposure to acidified gel. S = sealant, O = original lesion, N = new lesion, SE = sound enamel, SSE = sealed sound enamel, SZ = surface zone.

The ability of resin tags in sealed enamel to protect against acid demineralization *in vitro* is present in sealed white spot lesions as well. It would appear that acid etching of the white spot lesions followed by the application of a resin material may be a method for arresting the progression of white spot lesions.

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