Effect of Repeated Acid Challenges on the Color Stability of Resin-Infiltrated Enamel White Spot Lesions

by

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CLINICAL RELEVANCE STATEMENT

While the literature clearly supports the potential effectiveness of resin infiltration in masking white spot lesions and inhibiting caries, few studies have examined the durability of resin-infiltration against acid challenges. We have found only one study (Torres & colleagues, 2011) that evaluated potential effect of an acid solution on the color stability of a resin-infiltrated lesion. However, the study subjected specimens to only one acid challenge and evaluated its effect immediately. Therefore, the purpose of this study is to evaluate the color stability of resin-infiltrated white spot lesions following repeated acid challenges.

ABSTRACT

INTRODUCTION: Repeated bacterial acid challenges to tooth enamel result in loss of the mineral component. The resulting spaces decrease the refractive index to light giving the appearance of a white lesion. Resin infiltration fills subsurface enamel voids and increases the refractive index of white spot lesions to more closely match the surrounding enamel and improve esthetics. Currently, there is a lack of information regarding the color stability of resin-infiltrated enamel following multiple acid challenges.

OBJECTIVE: This pilot study evaluated the effect of multiple acid challenges on the color stability of resin-infiltrated enamel white spot lesions in vitro.

METHODS: For this study CIE L*a*b* (L*defines lightness, a* denotes red/green value and b* the yellow/blue value) values were used to numerically describe tooth color. Four de-identified human molar teeth were sectioned and mounted in resin to expose the buccal surface. A spectroradiometer was used to record 1931 CIE values (Yxy) of the buccal surfaces which were then converted to 1976 CIE L*a*b* values. Specimen 1 served as an internal control for spectroradiometer calibration. Specimen 2 was subjected to 37% H_3PO_4 challenge only. Specimens 3 and 4 were etched with 15% HCl to created white spot lesions. Specimens 3 and 4 were then infiltrated with Infiltration Concept (ICON) resin according to manufactures instructions. Specimens 4(etch/ICON/etch) was then exposed to an additional 37%H_3PO_4 challenge. L* values were recorded for all specimens after each step.

RESULTS: Acid application decreased L* for specimens 2, 3 and 4, resin infiltration led to an increase of L* for specimens 3 and 4, and resin infiltration appeared to have a protective effect for specimen 4 from further acid insult.

CONCLUSION: The preliminary data indicated that resin infiltrated tooth demonstrated better color stability after acid challenge than tooth without resin infiltration.

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LIST OF ABBREVIATIONS

1.	HCl	Hydrochloric Acid
2.	HPO	Phosphoric Acid
3.	CDC	Center of Disease Control
4.	ADA	American Dental Association
5.	CIE	International Commission on Illumination

CHAPTER I: INTRODUCTION

While the literature clearly supports the potential effectiveness of resin infiltration in masking white spot lesions and inhibiting caries, few studies have examined the durability of resin-infiltration against acid challenges. We have found only one study (Torres & colleagues, 2011) that evaluated potential effect of an acid solution on the color stability of a resin-infiltrated lesion. However, the study subjected specimens to only one acid challenge and evaluated its effect immediately. Therefore, the purpose of this study is to evaluate the color stability of resin-infiltrated white spot lesions following repeated acid challenges.

CHAPTER II: REVIEW OF LITERATURE

Enamel and its Dissolution

Tooth enamel is made up of 4% water and organic material and 96% mineral hydroxyapatite, a crystalline calcium phosphate. These crystals are organized into long, parallel, tightly packed rods extending from the dentin-enamel junction (DEJ) to the surface of the tooth. In between these rods exists interrod enamel, which is made up of less organized hydroxyapatite crystals. Where the rods and interrods meet there is a hypomineralized open space called the rod sheath, which is filled with proteins. These spaces are also referred to as pores. Another type of hypomineralized space within enamel is the striae of Retzius. These relatively large incremental growth spaces between the rods are theorized to be the primary entry point of hydrogen ions during caries development (Nanci, 2012).

Stephan (1944) categorized a pH 5.0 - 5.5 within dental plaque to be the “critical pH above which calcium salts would not be dissolved from teeth, under mouth conditions, and below which decalcification would take place.” When plaque produces a local environmental pH below 5.5, calcium and phosphate dissolve into solution and are lost from the surface enamel. Hydrogen ions also demineralize sub-surface enamel by way of the striae of Retzius and rod sheaths (Nanci, 2012).

If the acid insult is balanced by the remineralizing and buffering functions of saliva, ions restore those lost and no lesion results. This demineralization-remineralization process is theorized to be a natural part of homeostasis, occurring numerous times per day (Silverstone, 1977). In fact, this process can actually increase the resistance to acid dissolution if fluoride is incorporated during the remineralization phase. In the presence of fluoride, calcium and phosphate ions not only precipitate more readily on damaged enamel surfaces, they also form fluorapatite, rather than hydroxyapatite. Fluorapatite contains a fluoride ion replacing the hydroxyl group of hydroxyapatite (Figure 1). This new crystal is more resistant to acid attack, effectively lowering the critical pH to 4.5 - 5.0 (Featherstone, 2000).

Figure 1. Chemical change of hydroxyapatite to fluorapatite.

$$\underset{\text{(Hydroxyapatite)}}{Ca_{10}(PO_4)_6(OH)_2 + 2F^-} \longrightarrow \underset{\text{(Fluorapatite)}}{Ca_{10}(PO_4)_6F_2 + 2OH}$$

When the acid demineralization overwhelms the remineralization potential of saliva, dental decay results. Silverstone (1973) examined enamel caries lesions under a polarizing light microscope and described four distinct zones (Figure 2). These zones are the result of a dynamic process involving calcium-phosphate dissolution, hydrogen ion diffusion, and the reparative effects of the saliva (Silverstone, 1977).

The most superficial zone is an approximately 30-micron thick area exhibiting a mineral content similar to the surrounding healthy enamel. It remains highly mineralized because it receives calcium and phosphate ions from both the saliva on the surface and the dissolved enamel from the subsurface tooth structure. Also, it is in direct contact with salivary fluoride, which inhibits demineralization and promotes remineralization (Silverstone, 1973, 1983; Featherstone, 2000). Because of the relatively normal mineralization, only very small pores between the enamel rods are seen on the polarized microscope (Silverstone, 1973).

The subsurface zone, known as the body of the lesion, is the most demineralized portion. It is relatively structure-less, as large amounts of the mineral structure of the parallel rods are lost. This results in very large pores that may contain bacteria.

The following layer is the dark zone. It is the result of demineralization with loss of enamel structure followed by mineral deposition from diffusing ions released by the body-zone. Because this is a dynamic process, there exist larger and smaller pores all in different orientations, which appear in the microscope as dark areas (Silverstone, 1973; Nanci, 2012).

The deepest zone is the translucent zone. It represents the advancing front of the hydrogen ions. Here the pores are enlarged, but the enamel structure and orientation are retained. When water is allowed in these spaces, the zone appears to be translucent in polarized light (Silverstone, 1973; Nanci, 2012).

Dentin Demineralization

Dentin is composed of 70% mineral, 20% organic material, and 10% water. Its microstructure is based around odontoblasts. Each odontoblastic cell body resides in the dental pulp, with a process that extends outward, toward the DEJ. The structure immediately surrounding each process is a mineralized tube structure called peritubular dentin. These tubules are arranged in a matrix structure and are separated by the collagen-dense, less mineralized, intertubular dentin (Nanci, 2012).

By the time acids reach the DEJ, a large area of structureless, bacteria laden enamel exists in the body of the caries lesion. This front of acid and bacteria forms the outer zone of infected dentin. In this superficial layer of dentinal caries, the acids cause dissolution of the mineral content, while the bacterial proteolytic enzymes break down the collagen matrix. This creates a structureless irreparable zone saturated with bacteria (Kennedy, Teuscher & Fosdick 1952; Pashley, 1991; Roberson, Heymann & Swift, 2006; Nanci, 2012).

The next layer represents the advancing front of bacteria, which are present within the tubules. This "turbid dentin" is composed of structured, yet still irreparable, demineralized and denatured collagen. The odontogenic process has retreated and deposited mineral crystals in attempts to slow the bacterial progression down the tubule (Kennedy, Teuscher & Fosdick 1952; Pashley, 1991; Roberson, Heymann & Swift, 2006; Nanci, 2012).

Just beyond this layer exists "transparent dentin," which is demineralized as a result of acid diffusion, but contains no bacteria. Its collagen structure is intact and tubules are completely occluded with large crystal deposits (Kennedy, Teuscher & Fosdick 1952; Pashley, 1991; Roberson, Heymann & Swift, 2006; Nanci, 2012).

Deeper within the dentin, the "subtransparent dentin" is only slightly demineralized and contains damaged, yet vital, odontoblastic processes. The deepest layer is normal dentin.

No appreciable demineralization or collagen is present (Kennedy, Teuscher & Fosdick, 1952; Pashley, 1991; Roberson, Heymann & Swift, 2006; Nanci, 2012).

Caries Progression

The early caries lesion is confined to the subsurface enamel only and appears as a white chalky area when dried. These lesions are rarely cavitated. Pitts and Rimmer (1992) compared the radiographic depth of 1,468 interproximal lesions to the presence of clinical cavitation in the permanent teeth of 211 five- to fifteen-year-old children. When lesions were confined to the outer half of enamel, no clinical cavitation was evident; however, 10.5% of the lesions within the inner half of enamel were cavitated. The frequency of cavitation increased sharply as lesions progressed deeper into the dentin: cavitation was evident in 40.9% of the lesions in the outer half and 100% of those in the inner half of dentin.

Foster (1998) evaluated the progression of 65 interproximal caries lesions over three years in 65 adult patients, who received only oral hygiene and diet instructions. The patients were radiographically evaluated after 12 and 36 months. After 12 months, only 20% of the lesions that initially extended less than 0.5 mm into dentin had progressed. In contrast, 70% of the lesions initially extending 0.5-1mm into dentin had progressed. After 36 months, only 50% of the initially shallow lesions had progressed, while over 90% of the deeper lesions showed measurable progression.

Based on the findings of these studies, it suggested that caries progression and clinical cavitation are directly related to the depth of the lesion. It appears that if clinical cavitation is significant and the outer enamel surface is broken, remineralization of the tooth structure is not possible (Featherstone, 2000).

Tipping the Balance Toward Health and Regeneration

When pathologic factors such as acid-producing plaque or exogenous acid sources (e.g. sugar and soda) outweigh the protective factors of saliva (calcium, phosphate, antibacterial factors, and acid buffers), tooth structure is starting to disintegrate (Marsh, 1989). As the oral environment returns to a homeostasis, restoration of tooth mineral can occur. This process is known as remineralization (Silverstone, 1983).

While remineralization can occur on its own, additional variables must often be introduced to help reduce acids and aid in mineral deposition. One of these factors is fluoride exposure. Fluoride functions in three ways: (1) it inhibits key metabolic enzymes of cariogenic bacteria; (2) it inhibits dissolution of tooth mineral; and (3) it promotes deposition of calcium and phosphate ion on the tooth surface (Feathersone, 2000, 2004; Featherstone, Glena, Shariati & Shields, 1990).

Fluoride functions best in acidic environments, like those that exist in bacterial plaques. Fluoride is well absorbed into the bacterial cell membrane in its non-ionic form (hydrogen fluoride). Once inside the bacterial cell, it returns to its negatively charged ionic form and inhibits key metabolic enzymes, such as enolase. This effectively retards the production of waste acids (Whitford, Schuster & Pashley, 1977; Van Louvern, 1990; Feathersone, 2000).

Fluoride in solution can inhibit the loss of calcium and phosphate from tooth structure. Featherstone and colleagues (1990) demonstrated that as little as 1 part per million (ppm) of fluoride in an acid solution significantly reduced the dissolution rate of carbonated hydroxyapatite (a more soluble form of enamel that is present in teeth at eruption), and was more effective than fluoride incorporated into the enamel surface. Further increases in the fluoride solution concentration decreased the solubility rate logarithmically. As a comparative reference, the Centers for Disease Control and Prevention (CDC) have previously recommended 0.7-1.2 ppm fluoride in drinking water. These recommendations were reviewed and the new recommendation was updated to 0.7 ppm. Most over-the-counter fluoride dentifrices contain 1000-1100 ppm fluoride (CDC, 2001; Featherstone, Glena, Shariati, & Shields 1990, 2000; Robinson, Kirkham, Weatherell 1996; HHS, 2011; Ten Cate & Featherstone, 1991).

Fluoride not only prevents the loss of calcium and phosphate from tooth structure, but aids in their deposition. In an acidic environment, fluoride ions adsorb to the tooth surface and attract calcium and phosphate ions, where they form new crystals on the demineralized tooth surface. The result is the reconstitution of the tooth mineral structure incorporating fluoride in the form of fluorapatite, which is more resistant to acid dissolution than hydroxyapatite (Featherstone, Glena, Shariati, & Shields 1990; Featherstone, 2000; Nelson, Featherstone, Duncan & Cutress, 1983; Ten Cate & Featherstone, 1991).

The White Spot Problem

Early demineralization lesions are confined to the subsurface enamel. They frequently appear white when dried due to a reduction in their refractive index. For this reason they are often called white spot lesions (Figure 3) (Houwink, 1974; Brodbelt, O'Brien, Fan, Frazer-Dib & Yu, 1981). Not only do these lesions represent a disease state that can progress into the deeper tooth structure, they pose an esthetic problem when present on the facial surfaces of anterior teeth. White spot lesions in the esthetic zone are commonly seen in patients with xerostomia, poor oral hygiene, cariogenic diet, eating disorders, and orthodontic patients where bonded brackets provide retentive areas for plaque to accumulate

(Mota, Enoki, Ito, Elias & Matsumoto, 2008). There are four traditional methods of correcting these enamel lesions.

Figure 3. Clinical example of enamel white spot lesions.

First attempt is to remineralize, utilizing topical fluorides or casein phosphopeptide-amorphous calcium phosphate (CPP-ACP) (Kang, Lee & Lee, 2008). This technique does remineralize and strengthen the carious enamel; however, it does not predictably correct the appearance. This is because mineral deposition usually occurs only on the most superficial portion of the white spot. Therefore, the demineralized opaque body of the lesion continues to show through the outer enamel; however, it does not predictably correct the appearance. This is because mineral deposition usually occurs only on the most superficial portion of the white spot. Therefore, the demineralized opaque body of the lesion continues to show through the outer enamel (Ardu, Castioni, Benbachir & Krejci, 2007; Kang, Lee & Lee, 2008).

The second treatment option is bleaching the surrounding tooth structure with hydrogen peroxide. The goal in this treatment is to raise the value, or lightness, of the surrounding healthy enamel to better match that of the opaque demineralized region. However, like topical fluoride treatment, bleaching has proven to be unpredictable and can cause transient thermal sensitivity (Bussadori, Do Rego, Da Silva, Pinto, & Pinto, 2004; Bastin, Rodriques, Junior, & Serra, 2001).

The third treatment for white spot lesions is micro-abrasion. Micro-abrasion removes superficial enamel using an abrasive paste made from a strong acid (usually hydrochloric acid) combined with pumice. Both healthy and demineralized enamel are removed in attempts to reach a depth where only healthy, esthetic tooth structure exists. This treatment is invasive, can cause long-term post-operative sensitivity, and produces inconsistent results (Welbury, Carter, 1993; Wong & Winter, 2002).

Finally, the white spot lesion can be removed and replaced with resin composites or veneering porcelains. While this treatment produces more predictable results, it is the most invasive, requiring the removal of both healthy and demineralized enamel (Stahl & Zandona, 2007).

Resin Infiltration: A Restorative Alternative

Resin infiltration aims to fill the voids in the subsurface enamel caused by mineral loss. Infiltration of a low viscosity resin into incipient caries lesion may effectively prevent the diffusion hydrogen ions into the tooth during an acid attack, thereby inhibiting caries development and progression (Mueller, Meyer-Lueckel, Paris, Hopfenmuller & Kielbassa, 2006; Paris & Meyer-Lueckel, 2008, 2010). Resin infiltration also has the potential to improve the esthetics by way of increasing the refractive index of white spot lesions. This brings the refractive index closer to that of the surrounding enamel (Houwink, 1974; Brodbelt & colleagues, 1981).

Pre-Infiltration Etching. White spot lesions often have a thin hypermineralized enamel surface (approximately 30 microns) that has a smaller pore size than the demineralized subsurface (Bergman & Lind, 1966; Silverstone, 1973). This outer enamel veneer prevents the infiltration of light-cured resins, a process driven primarily by capillary action (Paris, Meyer-Lueckel, Hummel & Kielbassa 2006). To open these pores, an acid is used. Paris and colleagues (2007) showed that 15% hydrochloric acid (HCL) is more effective than the more common pre-restorative etchant, 37% phosphoric acid (H_3PO_4). They measured resin penetration depth in human enamel sections with natural white spot lesions following treatment with 15% HCL gel, 37% H_3PO_4 gel, or no acid treatment. Treatment with 15% HCL for 120 seconds allowed the infiltrative resin to penetrate to a mean depth of 58 µm, while 120 seconds of H_3PO_4 and no acid treatment showed penetration of 18 and 0 µm, respectively.

The Infiltrative Resin. The penetration coefficient (PC) measures the ability of a liquid (in this case, a low-viscosity, unfilled resin) to penetrate into a porous material (e.g., demineralized tooth structure). PC is the rate of penetration of a liquid into a capillary space in centimeters per second (Figure 4.). The PC of an infiltrative resin is directly related to its surface tension and viscosity. The higher the surface tension and the lower the viscosity, the greater is the resin's ability to penetrate into a caries lesion. Paris and colleagues (2007) calculated the PCs of five commercially available adhesive resins, five commercially available fissure sealant resins, and 66 experimental composite resins. They reported the highest PCs in resins containing triethylene glycol dimethacrylate (TEGDMA), 2-

hydroxyethyl methacrylate (HEMA), and 20% ethanol. This is one of the many studies conducted by Paris and colleagues that eventually led to the development of a proprietary infiltrative resin now commercially available as ICON® (DMG, Hamburg, Germany) (Paris, Meyer-Lueckel , Colfen & Kielbassa 2007a, 2007b, 2007c; Meyer-Lueckel & Paris 2008, 2010).

Figure 4. Formula for Penetration Coefficient

(Biomaterials Properties Database, University of Michigan, 1996)

$$PC = \frac{(\text{Surface tension of liquid}) \times (\text{cosine of the contact angle})}{2 \times \text{viscosity}}$$

Inhibition of Caries Progression. As previously discussed, diffusion of acid ions into and mineral ions out of a caries lesion occurs through enamel pores. If these pores were to be occluded with resin, ion diffusion would effectively stop and thereby arrest the caries process (Mertz-Fairhurst, Curtis, Ergle, Rueggeberg & Adair, 1998; Fejerskov & Kidd, 2008).

A number of *in vitro* and *in situ* studies presented initial support for resin infiltration as a means to prevent caries progression (Mueller & colleagues, 2006; Paris & Meyer-Lueckel, 2008, 2010). More recently, a number of clinical trials of the ICON® (DMG, Hamburg, Germany) infiltration system have shown great reductions in caries progression. In an 18-month split-mouth, placebo-controlled, randomized clinical trial, Paris et al. (2010) compared 29 interproximal lesion pairs in 22 young adults. One tooth of each pair was treated with the ICON® protocol (Appendix E) and the other treated with an identical placebo protocol. All participants were given oral hygiene and diet instructions. Examiners, blinded to the groups, used bitewing radiographs and digital subtraction radiography to compare the lesions at 18 months. Results showed progression in only 7% of the lesions treated with ICON®, compared to 37% in those treated with placebo.

In a similar study, Martignon, Ekstrand, Gomez, Lara and Cortes (2012) conducted a randomized clinical trial with 39 individuals, each having three interproximal lesions. Each of the participants' three lesions was randomly treated with an infiltrative resin, a commercially available self-priming urethane dimethacrylate (UDMA)-based resin bonding agent, or placebo. The subjects were evaluated at one, location.[94] With the gingival margin more accessible, it can be isolated with a rubber dam. This allows the the dentist better access and moisture control.[95]

The PBE technique has been used to raise proximal dentin margins prior to scanning for indirect dental restorations. Using this composite placement technique, PBE could be two, and three years via digital subtraction radiography. The results of the study are summarized in Table 2.

Table 2. Progression of interproximal caries lesions following resin infiltration. Adapted from Martignon and colleagues (2012).

	Lesion progression at 1 year	Lesion progression at 2 years	Lesion progression at 3 years
Resin Infiltration	15.5%	28.9%	47.7%
Bonding agent	24.3%	40.5%	62.2%
Placebo	32.4%	40.5%	70.3%

Martignon and colleagues found that resin infiltration was effective in preventing caries progression, but there was little statistical difference between the infiltrative resin and the UDMA bonding agent (Martignon et al, 2012).

Literature supports that infiltration with low-viscosity, high penetration coefficient resin is effective in inhibiting caries lesion progression and may serve as an alternative to more invasive cavity preparation and replacement with restorative material (Paris and colleagues 2007).

Resin Infiltration's Esthetic Effect. Beyond reducing the likelihood of lesion progression, resin infiltration has a significant effect on the optical properties of demineralized enamel. The translucency of enamel is dependent on the size of the intercrystalline spaces. When enamel is decalcified, these spaces are enlarged and the enamel takes on an opaque appearance.

When the lesion is wet, water (refractive index 1.33) is able to fill the spaces, blending the decalcified enamel with the surrounding healthy enamel (refractive index 1.65). However, when the lesion is dried, or has advanced to the extent that water cannot adequately fill the intercrystalline spaces, air (refractive index 1.00) causes the surface to take on a white chalky appearance (Houwink, 1974; Brodbelt et al, 1981). DMG's infiltrative resin has a refractive index of 1.46. As a result, once infiltrated with resin, white spot lesions match the refractive index of surrounding healthy enamel (Paris & Meyer-Lueckel, 2009). Several *in vitro* and *in vivo* studies have confirmed the effectiveness and color stability of resin infiltration in masking white spot lesions.

In Vitro Effectiveness. Torres, Borges, Torres, Gomes and Oliveira (2011) evaluated 60 bovine enamel samples with artificially created white spot lesions. They compared the effect of daily 0.05% fluoride solution exposure, weekly 2% fluoride gel exposure, resin infiltration, and no treatment on color via spectrophotometer. Examinations were performed at baseline, after creation of artificial caries lesions, after four weeks, after eight weeks, and after new acid challenge. The color measurements were quantified in terms of the coordinate value L* (Commission International De L'Eclariage- CIE), which represents the degree of lightness within a sample and ranges from black to white. Specimens treated with resin infiltration exhibited the lowest ΔL (difference in L* value from baseline to examination point) immediately after treatment, at four weeks, and at eight weeks ($p = 0.001$). Also, this group had the lowest ΔL when values at eight weeks and immediately after acid challenge were compared. Therefore, the infiltration group showed the greatest color stability following acid challenge. Although this study suggests that resin infiltration may be more effective in masking white spot lesions than daily or weekly fluoride, the specimens were evaluated immediately after only a single acid challenge. Therefore, no conclusions can be made regarding the masking durability of infiltration resin following repeated acid challenges.

In a similar *in vitro* study, Yuan and colleagues (2013) created white spot lesions in 52 human premolars and molars. Baseline spectrophotometry examinations were performed on the specimens, ensuring similar initial color values. Specimens then received one of four treatments:

1) NaF: specimens immersed in 330 ml of 500 ppm sodium fluoride for five minutes;
2) CPP-ACP: Casein phosphopeptide-amorphous calcium phosphate cream was applied to the tooth surface for five minutes;

3) Resin infiltration: specimens were subjected to the ICON® (DMG) resin infiltration protocol (Appendix E);

4) DDW: control group immersed in 300 ml distilled deionized water (DDW) for five minutes.

Following treatment, all specimens were rinsed with DDW and stored in remineralization solution (1.5 mM $CaCl_2$, 0.9 mM KH_2PO_4, 130 mM KCL, 1 mM NaN_3, and 20 mM *4-(2-hydroxyethyl)-1-piperazineethanesulfonic acid* [HEPES]). All specimens were evaluated with a spectrophotometer at baseline, after artificial lesion creation, immediately after treatment, and then at two, four, and six weeks.

Immediately after lesion creation, all specimens showed an average color difference (ΔE) of 12.5 compared to baseline. When evaluating all post-treatment time points, the resin infiltration group had the lowest mean ΔE (2.9) compared to baseline. The NaF, CPP-ACP, and DDW groups showed an average ΔE of 12.0, compared to baseline at all time points.

There was no statistically significant change within any group after treatment. The authors concluded that resin infiltration was much more effective in reducing the ΔE between white spot lesions and surrounding healthy enamel when compared to NaF, CPP-ACP, and no treatment.

In-Vivo Effectiveness. Kim and colleagues(2011) used an infiltrative resin to treat 38 teeth in 20 patients. Twenty teeth had hypo mineralization developmental defects in enamel (DDE), and 18 teeth exhibited post-orthodontic decalcification (POD). They used standardized clinical photography and image analyzing software to compare the air-dried lesions before and one week after treatment. Comparisons were classified as completely masked, partially masked, or unchanged. Table 3 illustrates their results.

Table 3. Percent of DDE and POD lesions masked by resin infiltration. Adapted from Kim and colleagues (2011).

Lesion Type	Completely Masked	Partially Masked	Unchanged
DDE	25%	35%	40%
POD	61%	33%	6%

DDE: developmental defect in enamel; POD: post-orthodontic decalcification.

They concluded that resin infiltration seems to be more predictable in masking post-orthodontic decalcifications than developmental defects of enamel. Senestraro and colleagues (2013) conducted a randomized clinical trial on the efficacy of the ICON® (DMG) resin infiltration protocol in masking post-orthodontic decalcifications. Twenty patients, with a total of 66 teeth exhibiting white spot lesions, were selected. Of these 66 teeth, 46 (approximately three per patient) were treated with the resin infiltration system, and 20 (one tooth per patient) were left untreated to serve as controls. Photographs were taken of each tooth at baseline, immediately after treatment, and eight weeks later. Five calibrated observers evaluated the photographs using a visual analog scale (VAS; 0 = no change, 100 = white spot completely disappeared). Results showed that the mean VAS of the infiltrated lesions was 67.7 immediately after treatment, compared to 5.2 for the control. There was no statistically significant change in the VAS at eight weeks for either group. The authors concluded that the ICON® resin infiltration protocol was more effective in decreasing the visibility of post-orthodontic white spot lesions when compared to no treatment. Also, the treatment group showed consistent appearance after eight weeks. While this study was based on qualitative evaluation, rather than objective quantitative analysis, it is one of the few studies that take into account the final visual result of resin infiltration.

Color Stability. Paris, Schwendicke, Keltsch, Dorfer and Meyer-Luekel (2013) studied 120 bovine enamel specimens, each with four artificially generated white spot lesions. For each specimen, two of these lesions were treated with an infiltrative resin (either ICON® [DMG] or one of five experimental resins with varying refractive indexes), and two lesions remained untreated. On each specimen, one resin-treated lesion and one untreated lesion were polished; the other two lesions were left unpolished. Specimens were stored in a remineralizing solution and subjected to a 1:1 mixture of red wine and coffee for 10 minutes per day for 50 days. Digital photographs of the lesions were analyzed via photographic analyzing software after initial white spot creation, after resin infiltration, and after 50 days of staining, analyzed via photographic analyzing software after initial white spot creation, after resin infiltration, and after 50 days of staining. The authors used six proprietary, experimental infiltrative resins with varying refractive indexes (RI) to evaluate their effect on white spot lesion masking. Varying the RI did not significantly affect the masking ability of the resin. They also found that polished infiltrated lesions showed less staining than the unpolished infiltrated lesions. However, the opposite was true of the non-infiltrated lesions, as polishing increased staining compared to unpolished.

Knosel, Eckstein and Helms (2013) evaluated the effectiveness and durability of resin infiltration in 20 subjects with 219 post-orthodontic white spot lesions; 111 teeth were treated with infiltrative resin and 108 served as controls. For each tooth, the white spot lesion was compared to the sound adjacent enamel with an intraoral spectrophotometer. This analysis provided a difference in color value (ΔE), which was calculated at baseline and after one day, four weeks, three months, and six months post-treatment. The mean baseline ΔE values for both the control group and the infiltrative group were statistically similar, at 7.38 and 7.88, respectively. Immediately after treatment, the resin infiltration group displayed a mean ΔE of 6.09. The mean ΔE for the treatment group remained consistent at each time interval, with a final ΔE of 5.64 at six months. The control group showed no statistically significant change in ΔE from baseline (7.38) to six months (7.59). There was a statistically significant decrease in ΔE values from baseline to six months for the treatment group.

Summary

White spot lesions are the visible sequelae of enamel mineral dissolution. This demineralization is caused by repeated bacterial acid challenges, over time, in the absence of sufficient protective factors. Once these lesions are present, it is difficult to restore the tooth structure to its previous appearance and structural integrity. Remineralization and bleaching techniques yield unpredictable results, while micro-abrasion and operative procedures involve the irreversible removal of both healthy and diseased tooth structure. The recently introduced resin infiltration technique may provide a less invasive, more predictable method for correcting white spot lesions.

Investigators have evaluated the caries inhibition potential of resin infiltration. They found that by occluding the enlarged pours present in demineralized enamel, diffusion of acid into tooth structure is prevented and caries progression is inhibited.

Investigators have also evaluated the ability of infiltrative resins to mask white spot lesions. By filling the demineralized enamel pours with resin (having a similar refractive index to that of healthy enamel), white spot lesions are effectively blended with surrounding tooth structure.

CHAPTER III: MATERIALS AND METHODS

Methodology

Four de-identified, caries-free extracted human molar teeth were obtained from WRNMMC Oral Surgery Clinic and stored in demineralized deionized water (DDW) since extraction for the duration of the study. Each tooth was positioned in the center of a mold such that the buccal surface of the clinical crown protruding out of the mold. The mold was filled with self-curing acrylic resin and allowed to bench cure. Once cured, the teeth in acrylic blocks were be stored in DDW until conduction of the spectroradiometer analysis procedures.

CIE L*a*b* (L*defines lightness, a* denotes red/green value and b* the yellow/blue value) values were used to numerically describe tooth color. The majority of the literature utilizes the 1976 International Commission on Illumination CIE L*a*b* system. The software and PC interface cable were not available at the time of the procedures, therefore, a color calculator (www.EasyRBG.com) was used to convert CIE 1931Yxy values from the spectroradiometer to CIE 1976 L*a*b* values. A custom stand with clamps was constructed to hold specimens at a set distance from the spectroradiometer to avoid refocusing and maintain reproducibility.

Each tooth specimen was assigned to one of four treatment groups. Specimen 1 served as an internal control for spectroradiometer calibration. Specimen 2 was subjected to 37% H_3PO_4 challenge only. Specimens 3 and 4 were etched with 15% HCl to created white spot lesions. Specimens 3 and 4 were then infiltrated with Infiltration Concept ICON® (DMG) resin according to manufactures instructions. Specimen 4(etch/ICON/etch) was then exposed to an additional 37%H_3PO_4 challenge. L* values were recorded for all specimens after each step.

CHAPTER IV: RESULTS

Pilot Study Results:

L* value of specimen 1 remained constant throughout the duration of the study confirming the calibration of the spectroradiometer. The L* value of Specimen 2 was constant through first three readings with a decreased L* value from 19.51 to 16.78 after a 60 second application of 37% phosphoric acid. 15% HCl was applied to specimen 3 and a decrease in L* from 19.38 to 17.41 was observed. The L* value rebounded to 18.54 after specimen 3 was infiltrated with ICON® (DMG) resin and remained constant in last two observations. Specimen 4 received treatment with 15% HCl and baseline L* of 22.90 decreased to 21.64 then recovered to near baseline 22.39 after ICON® (DMG) resin infiltration and finally fell to 21.85 after 37% phosphoric acid treatment.

CHAPTER V: DISCUSSION

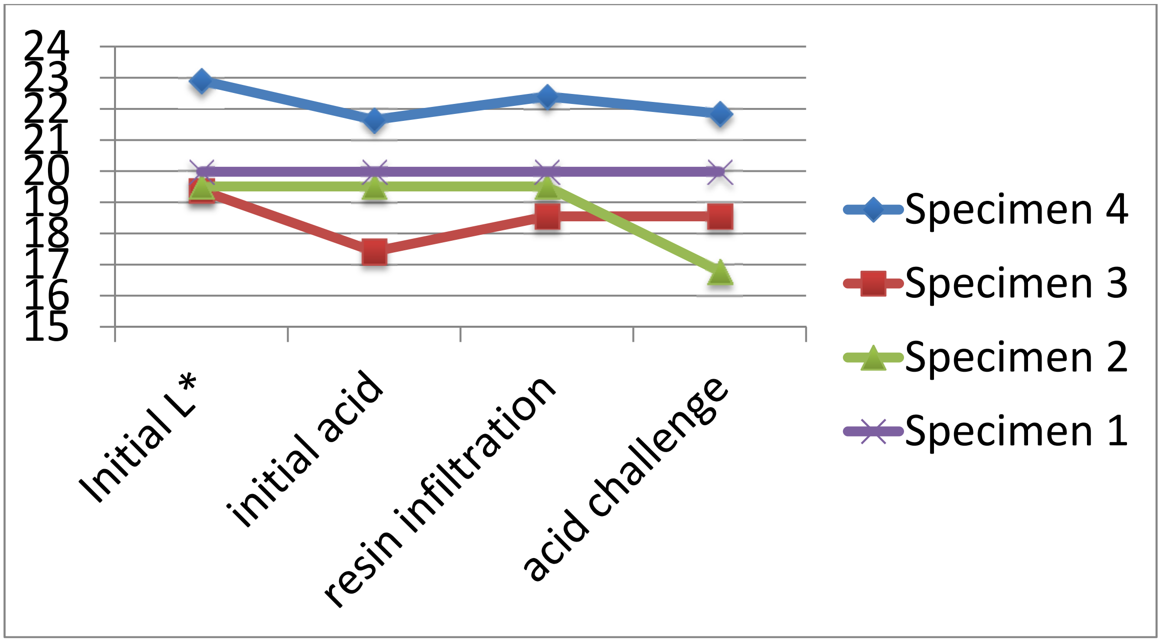
The masking effects of ICON® (DMG) resin infiltration has been established in the dental literature by Senestraro and colleagues (2013), Kim and colleagues (2011). This pilot study examined the durability of resin-infiltration against acid challenge and the initial results suggest that may be effective. Due to funding issue, the sample size in this study was one tooth per group A study that involves color analysis should pay strict attention to light sources. This study was conducted in commercial fluorescent lighting that was energized for at least 30 minutes before any spectroradiometer readings were obtained. Continuation of this study should consider using color corrected lighting to more closely compare natural daylight.

CHAPTER VI: CONCLUSION

Modern dentistry continues to advance toward minimally invasive and preventative approaches to combat the caries process. Esthetics is also of paramount importance in the outcomes of clinical practice (Mota, Enoki, Ito, Elias & Matsumoto, 2008). The treatments for white spot lesions historically either do not mask the lesion or remove healthy tooth structure in the process. Resin infiltration aims to fill the voids in the subsurface enamel caused by mineral loss. Infiltration of a low viscosity resin into incipient caries lesion mayeffectively prevent the diffusion hydrogen ions into the tooth during an acid attack, thereby inhibiting caries development and progression (Mueller, Meyer-Lueckel, Paris, Hopfenmuller & Kielbassa, 2006; Paris & Meyer-Lueckel, 2008, 2010). Resin infiltration also has the potential to improve the esthetics by way of increasing the refractive index of white spot lesions. This brings the refractive index closer to that of the surrounding enamel (Houwink, 1974;Brodbelt & colleagues, 1981). What has not been proven is the color stability of ICON® (DMG) after multiple acid challenges. The preliminary data indicated that resin infiltrated tooth demonstrated better color stability after acid challenge than tooth without resin infiltration.

APPENDIX A: DATA COLLECTION SHEET – PILOT STUDY

Specimen	**Initial L***	**Acid Challenge L***	**ICON**	**Acid Challenge L***
1	19.99	19.9	19.99	19.99
2	19.51	19.51	19.51	16.78
3	19.383	17.48	18.54	18.54
4	22.90	21.64	22.39	21.85



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