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Enamel Demineralization in Adolescents Undergoing Fixed Orthodontic Treatment: Pathogenesis, Digital Detection, and Emerging Preventive Strategies

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Abstract. Enamel demineralization is a common complication of fixed orthodontic treatment in adolescents. It usually appears as chalky white spot lesions on the tooth surface, most visibly around bracket margins. These lesions are not merely cosmetic. If left untreated, they may develop into caries and may interfere with the long-term goals of orthodontic treatment. Clinicians can choose among several standard approaches, such as plaque-control routines, various fluoride formulations, and calcium-phosphate remineralizing agents. Even so, clinical outcomes vary. Compliance differs from patient to patient, lesions are at different stages, and individual risk factors can push results in opposite directions. This review brings together current understanding of orthodontically induced enamel demineralization, covering how it develops, what drives it, and where conventional treatment falls short. It also looks at the role of digital detection tools and risk prediction models in earlier diagnosis, and reviews newer remineralizing, antibacterial, and stimuli-responsive materials. Finally, it considers why these newer options have not yet entered routine clinical use, and provides practical guidance for screening, risk stratification, and tailored intervention.

Keywords: enamel demineralization, fixed orthodontic treatment, adolescents, digital diagnosis

1. Introduction

Malocclusion is common among adolescents. For this age group, fixed orthodontic appliances are a common treatment option, since they are reliable and predictable in most cases. Their downside is well documented, however. Brackets, archwires, and auxiliary attachments sit on tooth surfaces and get in the way of the mouth's natural self-cleaning. Food debris and plaque become trapped in retention areas, and enamel is repeatedly exposed to acids from plaque metabolism. A chalky white lesion around bracket margins is the classic sign [1]. Such lesions appear most often where brushing is difficult, particularly on maxillary anterior teeth and cervical regions.

Streptococcus mutans and other acid-producing bacteria in plaque ferment dietary carbohydrates into lactic acid and other metabolites. These acids slowly dissolve hydroxyapatite crystals in enamel. When demineralization exceeds saliva's repair capacity, early enamel lesions form. If

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nothing is done, they can progress to cavitated caries. That outcome is bad for aesthetics, bad for treatment results, and a poor experience for the patient.

This is not a single-cause problem [2]. It involves patient behavior, oral environmental conditions, and treatment-related variables. Poor daily oral hygiene, high sugar intake, and weak adherence to instructions all increase plaque and acid production. Reduced salivary flow, weaker buffering, and disturbed microbial homeostasis impair the tooth's natural defense and repair. Fixed appliances also make cleaning harder. Because adolescent orthodontic treatment often lasts a long time, persistent plaque retention becomes an ongoing risk, not a temporary one.

Current prevention relies mainly on mechanical cleaning, topical fluoride, and calcium-phosphate supplements. These approaches are safe, straightforward, and easy to deliver. Their effectiveness, though, depends on long-term patient compliance. For lesions that are already chalky and established, single interventions may have limited effects. And strategies tailored to individual risk profiles are still underdeveloped.

In recent years, digital dental technologies and new biomaterials may help address some of these gaps. Intraoral three-dimensional scanning, fluorescence imaging, and artificial intelligence (AI)-based image analysis may support earlier detection of subtle demineralization and make longitudinal monitoring easier [3]. Risk prediction models built on oral hygiene, salivary markers, and microbial profiles may give a better sense of individual risk. Bioactive materials that deliver calcium and phosphate are entering clinical discussion, and smart antibacterial materials allow more targeted modulation of the oral microecology. Still, most of this work is early. Long-term safety data are thin, and standardized clinical evaluation is lacking.

Enamel demineralization is one of the most common complications of fixed orthodontics in adolescents. It directly affects dental health and can undermine the purpose of treatment. Prevention and management remain fragmented, and no standardized, personalized framework exists. Drawing on domestic and international research, this review examines pathogenesis and risk factors, weighs the pros and cons of conventional prevention, and focuses on digital diagnostics and new bioactive materials. Clinical value, evidence gaps, and barriers to translation are also critically assessed. The aim is to provide a theoretical basis for enamel protection during treatment and practical guidance for screening, early prevention, and individualized care.

2. Pathogenesis and risk factors of enamel demineralization in adolescents receiving fixed orthodontic treatment

Enamel demineralization is one of the most common early complications in adolescents undergoing fixed orthodontic treatment. It is not merely an aesthetic problem. Advanced lesions can progress to frank caries, which may disrupt the treatment schedule. At its core, the condition reflects a persistent shift in the balance between demineralization and remineralization at the tooth surface, with demineralization gaining the upper hand [4]. Three groups of factors drive this shift: the appliances, changes in oral microecology, and individual patient characteristics.

Brackets, archwires, and auxiliary attachments blunt the mouth's self-cleansing function. This creates many hard-to-clean zones: bracket edges, interdental spaces, gingival margins. Plaque builds up there continuously. Acid-producing and acid-tolerant organisms, including *Streptococcus mutans* and *Lactobacillus* species, ferment fermentable carbohydrates into lactic acid and other acidic by-products. Local pH may repeatedly fall below the critical level for enamel dissolution, which varies with the oral environment. Calcium and phosphate ions leach from enamel hydroxyapatite, and chalky white lesions form over time.

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Saliva is the main regulator of the oral microenvironment. If flow rate is low, buffering is weak, or calcium and phosphate concentrations are inadequate, remineralization cannot keep up. The oral microbial community may also change during orthodontic treatment. Cariogenic organisms can become more prominent, which further disturbs homeostasis.

Individual factors matter too. Poor oral hygiene, frequent high-sugar intake, poor treatment compliance, a history of recurrent caries, and enamel hypoplasia all raise risk significantly. Appliance type, bonding technique, and treatment duration also correlate with plaque retention and demineralization risk. Most studies, however, show statistical associations rather than hard causation.

3. Conventional prevention and management of enamel demineralization

For a long time, clinical management has revolved around two goals: control plaque and promote remineralization. That has produced the standard system of mechanical removal plus chemical intervention, widely used in clinics. Mechanical plaque control is the foundation. Daily Bass brushing, orthodontic-specific toothbrushes, interdental brushes, and oral irrigators can target plaque around brackets and between teeth, cutting acid production at the source. These measures are only as good as the technique and the consistency with which they are used. Adolescents frequently struggle to keep plaque control at an ideal level, since their ability to manage their own care is still developing.

Fluoride is widely used for chemical prevention [5, 6]. It may be delivered as toothpaste or mouthwash, which keep oral fluoride at a low level; as varnish, which forms a high-concentration reservoir on the tooth surface; or through bonding materials that release fluoride slowly around brackets. Fluoride can reduce mineral dissolution and support the formation of more acid-resistant mineral at the enamel surface. Fluoride also promotes calcium and phosphate deposition, which supports remineralization of early lesions. Concentration, application frequency, and patient use all affect how well it performs.

Calcium-phosphate agents include casein phosphopeptide-amorphous calcium phosphate (CPP-ACP) and bioactive glass [7]. They release calcium and phosphate ions gradually, supplying minerals needed for early lesion repair. In most cases, they are added to fluoride rather than used alone. Product type and study design account for much of the variation in reported results.

Overall, conventional strategies are safe, easy to use, and not expensive. However, important limitations remain: adherence is uneven, established chalky lesions are hard to reverse, and individualized risk stratification is largely absent. These limitations motivate more individualized prevention strategies.

4. Digital diagnosis, monitoring, and risk prediction

Early detection and monitoring are important to prevention and management, but conventional methods have limitations. Visual and tactile examination is subjective, may miss subtle subsurface changes and does not reliably quantify mineral loss or lesion depth. Tracking progression over time is therefore difficult. Conventional radiography has limited sensitivity to early demineralization, and because it involves ionizing radiation, it is not suitable for repeated monitoring of white spot lesions. As shown in Figure 1, the clinical spectrum extends from faint white opacities to more extensive lesions with surface breakdown.

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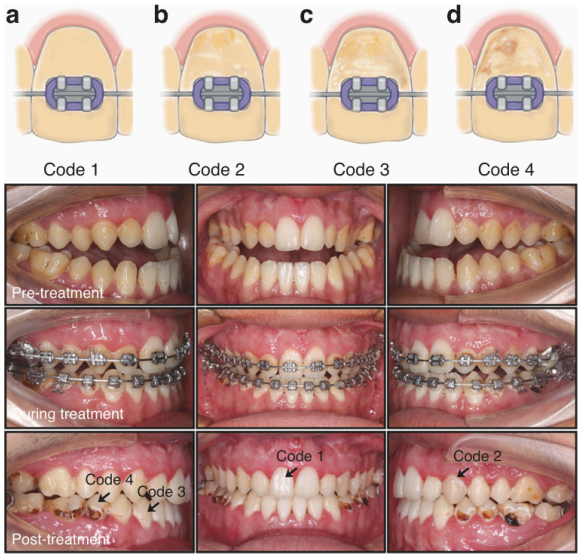


Figure 1. Enamel demineralization grading and intraoral appearance during fixed orthodontic treatment: (a) smooth enamel, code 1; (b) linear lesions, code 2; (c) striped lesions, code 3; and (d) cavitation, code 4 [4]

Digital detection and analysis have drawn growing interest for exactly this reason. Among non-invasive options, optical and fluorescence-based methods have been studied most. Quantitative light-induced fluorescence and laser fluorescence work by comparing fluorescence intensity in demineralized enamel with that in healthy enamel, which allows an estimate of mineral loss. Readings are not always clean, however; surface staining, plaque, and device settings may all interfere.

Near-infrared imaging and optical coherence tomography offer another route. They can reveal internal changes in enamel without invasive procedures and may be useful for early detection and repeated follow-up. Their diagnostic cutoffs and reproducibility in clinical settings, though, are not yet standardized. Intraoral three-dimensional scanning combined with digital image registration permits close comparison of tooth surface morphology over time. When computer vision or AI image analysis is added, these tools may assist in lesion identification and in measuring changes in area and color. These surface-imaging methods do not reliably measure mineral loss or lesion depth directly.

Multifactor risk prediction models are beginning to appear as well. Such models draw on oral hygiene behaviors, salivary function markers, microbial profiles, and imaging data. Few have undergone validation in large external cohorts. For routine use, they would need better calibration, clearer clinical benefit, stronger interpretability, and evidence that they perform well across different populations. A recent review summarizes prognostic factors and models for white spot lesions [8].

5. Bioactive materials and precision prevention

As understanding of demineralization mechanisms has improved and digital tools have advanced, the limits of conventional strategies, especially around adherence and personalization, have become harder to ignore. Novel bioactive materials and precision prevention are now major research areas. Their common goal is to combine local targeted intervention with dynamic risk management.

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Newer remineralizing materials are intended to place active ions at the target site and hold them there longer. In shape and composition, nano-hydroxyapatite is close to natural enamel crystals. It may plug surface micropores and create nucleation sites, which can support mineral deposition [9]. Newer bioactive glasses liberate calcium, phosphate, and other ions; these ions encourage the formation of apatite-like layers. Biomimetic mineralization peptides take a different route, controlling mineral nucleation and crystal growth. Whether this translates into meaningful clinical repair is still unclear and needs larger, better-designed studies.

Several approaches are being explored to regulate the oral microecology. These include quaternary ammonium-modified materials, nano-metal particles, antimicrobial peptides, and probiotics [10]. Their shared aim is to lower acidic metabolite production, whether by inhibiting cariogenic bacteria, disrupting biofilms, or restoring microbial balance. A few of these agents have already been added to bracket adhesives and orthodontic attachments.

Smart drug delivery systems deserve particular attention. Platforms that respond to pH or enzymes, along with multifunctional adhesive materials that combine antibacterial and remineralizing effects, are built to detect shifts in the local microenvironment [11]. They aim to release active ingredients in response to relevant local conditions. This suggests a different model of intervention—one that reacts to the surrounding environment.

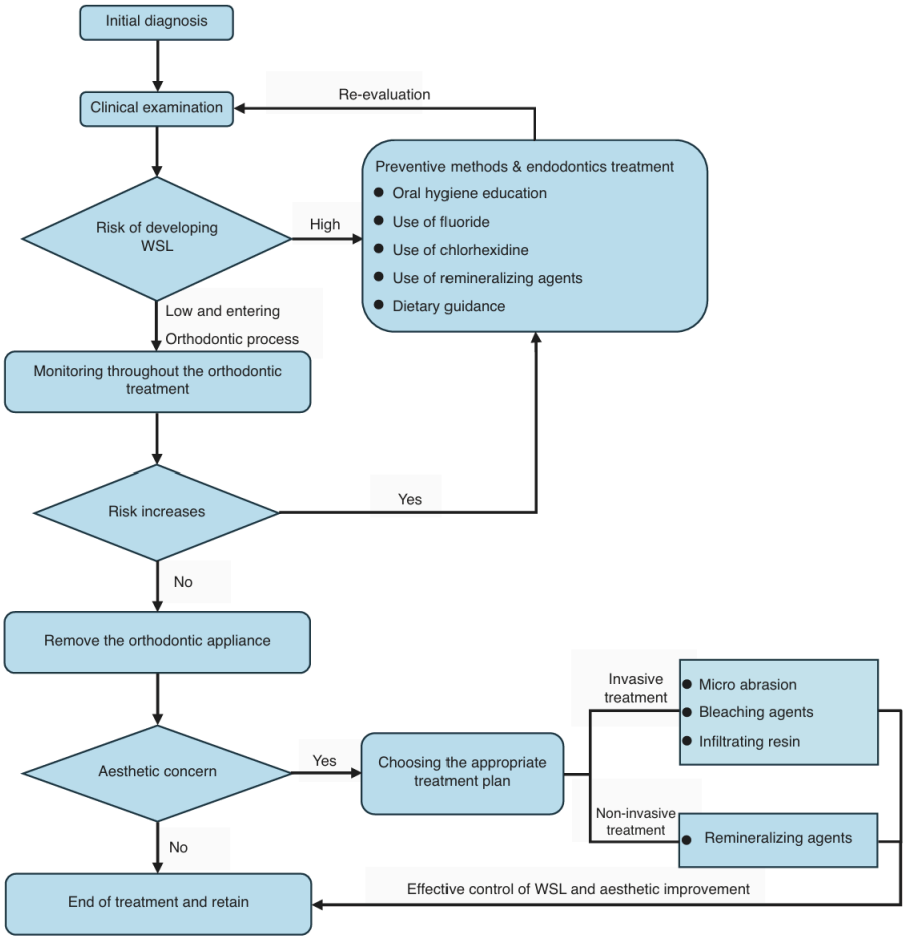


Figure 2. Risk-based pathway for preventing and managing white spot lesions during and after fixed orthodontic treatment [4]

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Looking ahead, personalized monitoring and intervention based on risk stratification is a sensible direction. The obstacle is evidence. Long-term biosafety data, robust clinical efficacy data, standardized evaluation frameworks, and cost-effectiveness analyses remain limited. Overcoming these barriers will require rigorous materials testing, long-term clinical follow-up, and well-designed clinical trials. Figure 2 summarizes a risk-based pathway: initial assessment, preventive intervention, continuous monitoring, and lesion-specific treatment after appliance removal.

6. Conclusion

The available evidence indicates that enamel demineralization during adolescent fixed orthodontic treatment is multifactorial. Appliances interfere with self-cleaning, promote plaque retention, and shift the oral microecology toward acid-producing and acid-tolerant bacteria. Salivary buffering and remineralization capacity influence how quickly lesions begin and how far they progress. Poor daily hygiene and individual differences in enamel susceptibility further increase risk. Current prevention and management still rely on mechanical plaque control, fluoride, and calcium-phosphate materials. Real-world results vary greatly among patients. Adherence is a major reason: consistent cleaning and proper product use make a substantial difference. Baseline differences in oral health also mean that patients respond differently to the same treatment.

Digital detection technologies are gradually entering clinical practice. They may improve the objectivity of early detection and longitudinal monitoring. Novel bioactive materials also show promise for enhancing local remineralization, targeting plaque, and delivering environment-responsive care. However, none of these approaches yet has sufficiently strong, consistent, high-quality clinical evidence to replace conventional prevention. The most reasonable current approach is to use them as adjuncts to traditional methods and build more comprehensive prevention and management strategies.

Research limitations remain substantial. Many studies are small or have relatively short follow-up. More importantly, there is no consensus on diagnostic thresholds for demineralization or on efficacy endpoints across studies. This makes direct comparison and evidence synthesis difficult. Some digital assessment methods, risk prediction models, and smart materials are still *in vitro* or at early clinical stages. Their long-term efficacy and safety are not well established.

Future research should prioritize multicenter, prospective, long-term follow-up studies and unified, standardized criteria for diagnosing demineralization and evaluating treatment. With such criteria, clinical data, salivary biomarkers, microbial profiles, and imaging information could be integrated into risk prediction models that are well calibrated, interpretable, and clinically useful. These models would need independent external validation before individualized prevention can move from theory into everyday practice.

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