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Adhesive Restorative Challenges in Sports Dentistry: A Substrate-Centered Narrative Review of Bonding to Caries-Affected, Eroded, and Sclerotic Dentin

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Abstract

Purpose. To synthesize substrate-specific evidence on bonding to caries-affected, eroded, and sclerotic dentin and integrate it with current evidence on the athlete oral environment to define implications for adhesive restorative care in sports dentistry.

Methods. A targeted narrative review used PubMed/MEDLINE, citation chaining, and full-text verification. Systematic reviews and meta-analyses were prioritized; a separate contextual search addressed athletes, sports nutrition, saliva, caries, erosive tooth wear, and restorative dentistry through 6 September 2026.

Results. Caries-affected and eroded dentin generally show lower laboratory bond strength than sound dentin, while management of sclerotic surfaces remains material- and protocol-dependent. Contemporary evidence does not support a universal additional phosphoric-acid etching rule. A 2025 meta-analysis estimated caries prevalence at 44.4% and dental erosion at 36.5% among elite athletes, with high heterogeneity; no athlete-specific bonding trial was identified.

Conclusions. Altered dentin should be managed according to substrate biology and adhesive chemistry. Sports-related exposures may increase the clinical likelihood of encountering carious or eroded substrates, but current evidence does not justify sport-specific bonding protocols.

Keywords: dentin bonding; caries-affected dentin; eroded dentin; sclerotic dentin; universal adhesives; sports dentistry; athletes; erosive tooth wear

Clinical significance. The sports-dentistry relevance is not that athletes require different adhesives, but that the oral-disease burden and sport-related exposure patterns may increase restorative encounters with altered dentin. Caries-affected, eroded, and sclerotic dentin present different interfacial barriers. Current evidence therefore supports substrate-aware, material-specific adhesive care combined with control of relevant oral exposures, rather than a sport-specific bonding protocol.

1. Introduction

Adhesive dentistry has transformed restorative treatment by enabling conservative preparations that depend on micromechanical and chemical interaction with dental hard tissues rather than macromechanical retention. Despite major advances in adhesive formulations, durable resin-dentin bonding remains less predictable than enamel bonding. Dentin is a hydrated biological composite in which mineralized collagen, dentinal fluid, tubular architecture, smear-layer characteristics, and regional variability all influence the interaction with adhesive systems. The hybrid layer created by resin infiltration of conditioned dentin is not an inert structure: residual water, incomplete monomer penetration, resin hydrolysis, phase separation, and endogenous proteolytic activity can progressively weaken the interface (Breschi et al. 2018; Breschi et al. 2025).

Much adhesive research has been conducted on standardized sound dentin, whereas clinical bonding surfaces may be modified by disease, chemical exposure, aging, or defensive tissue responses. This review uses the term "compromised dentin" only as an operational umbrella for three clinically relevant substrates—caries-affected dentin (CAD), eroded dentin, and sclerotic dentin—and not as a standardized biological taxonomy. These substrates are considered together because each can impair adhesion while doing so through fundamentally different structural alterations.

Contemporary carious tissue removal prioritizes pulpal health, preservation of tissue that can be maintained beneath a durable restorative seal, and avoidance of unnecessary excavation rather than complete removal of every zone of demineralized dentin (Schwendicke et al. 2016). Clinically retained dentin after selective carious tissue removal cannot, however, be assumed to correspond uniformly to histopathologically defined caries-affected dentin (CAD). Consensus terminology describes excavation endpoints primarily according to dentin hardness—soft, leathery, firm, or hard—because clinical appearance and tactile characteristics cannot be mapped directly onto histopathology (Innes et al. 2016). CAD nevertheless remains an experimentally relevant substrate model: its mineral content, porosity, collagen organization, and mechanical properties differ from those of sound

dentin, and systematic evidence demonstrates a laboratory bonding disadvantage (Ekambaram et al. 2015; Isolan et al. 2018). Eroded dentin develops through repeated non-bacterial acid challenge, usually within the multifactorial process of erosive tooth wear, and contains a superficial demineralized collagen-rich layer that may exceed effective resin infiltration (Wiegand et al. 2021). By contrast, sclerotic dentin—frequently encountered in non-carious cervical lesions (NCCLs) and worn occlusal surfaces—may be hypermineralized and characterized by extensive tubular occlusion, producing a relatively impermeable surface that resists conventional conditioning (Tay et al. 2000; Kwong et al. 2002).

These substrate differences have opposing implications for conditioning. More aggressive acid treatment may be mechanistically plausible when a hypermineralized surface limits interaction, but the same intervention may deepen an already demineralized zone in CAD or eroded dentin. Likewise, collagen stabilization targets an altered organic matrix and therefore addresses a different limitation from the mineralized barrier of sclerosis. Adhesive performance consequently depends not only on whether a system is classified as etch-and-rinse (E&R), self-etch (SE), or universal, but also on substrate composition, pretreatment, adhesive chemistry, application technique, and aging.

Sports dentistry increasingly encompasses prevention, disease management, and restorative care within comprehensive athlete health. A 2025 systematic review and meta-analysis of elite athletes estimated pooled prevalence of dental caries at 44.4% and dental erosion at 36.5%, although heterogeneity was high; contemporary reviews similarly identify caries and erosive tooth wear as recurring concerns in high-performance sport (Campana Zamudio et al. 2025; Needleman et al. 2026). These findings create a clinically relevant restorative case mix in which clinicians may encounter CAD after selective carious tissue removal or eroded dentin after progression of erosive tooth wear.

Potential sports-related contributors include frequent carbohydrate exposure during fueling, acidic sports and energy products, and exercise-related reductions in salivary protection associated with dehydration, high airflow, or mouth breathing. The strength of association varies across studies: a 2025 systematic review found inconsistent associations between sports-drink consumption and dental erosion, and observational athlete studies cannot establish causal pathways (Memon et al. 2025; Schulze and Busse 2024; Tripodi et al. 2025). The relevant conceptual link is therefore not a distinct "athlete dentin" phenotype. Rather, sport-associated behaviors and physiology may influence the probability and severity of oral disease or wear, whereas the adhesive response remains governed by the residual substrate and material chemistry.

This narrative review critically synthesizes current evidence on adhesion to CAD, eroded dentin, and sclerotic dentin. The objectives are to (1) compare the structural and biochemical alterations that make these substrates difficult to bond, (2) evaluate conventional and contemporary substrate-specific bonding strategies, (3) distinguish immediate bond-strength effects from evidence of durability, (4) examine the limits of translating laboratory outcomes into clinical recommendations, (5) propose a substrate-centered interpretive framework for restorative decision-making, and (6) integrate that framework with evidence on athlete oral-health exposures and restorative needs while explicitly separating contextual sports evidence from direct bonding evidence.

2. Materials and Methods

This article was prepared as a targeted narrative review rather than a registered systematic review. PubMed/MEDLINE served as the principal bibliographic database. Full texts were checked through PubMed Central and publisher platforms where available, and retrieval was supplemented by citation chaining from relevant systematic reviews and meta-analyses and by reference-list screening of key primary studies. The literature was updated through 6 September 2026.

Separate targeted search frameworks were used for caries-affected dentin, eroded dentin, and sclerotic dentin so that terminology specific to one substrate would not dominate retrieval. Search concepts combined substrate terms with adhesion, adhesive strategy, durability, and pretreatment terms; additional targeted searches addressed hybrid-layer degradation, matrix metalloproteinases, nanoleakage, aging, thermocycling, water storage, clinical performance in NCCLs, and recently published studies. A separate contextual search combined terms related to sports dentistry, athletes, elite sport, sports nutrition, dietary carbohydrates, sports and energy drinks, salivary flow and pH, dehydration, mouth breathing, dental caries, erosive tooth wear, and restorative dentistry. Priority was given to systematic reviews, meta-analyses, and athlete-specific clinical studies. These sports sources were used to characterize oral-disease burden, relevant exposure patterns, and external clinical context; they were not treated as evidence that athletic status modifies resin-dentin bonding. Exact search syntax, hit counts, and screening logs were not prospectively archived and are therefore not retrospectively reconstructed as if they had been generated within a systematic-review workflow. This limits reproducibility and is acknowledged as a methodological limitation.

Evidence was selected purposively according to relevance to permanent dentin and to the specific substrate under discussion. Systematic reviews and meta-analyses were prioritized to establish the direction, consistency, and limitations of the evidence. Primary studies were retained when they examined natural human altered dentin,

clarified substrate-specific mechanisms, included meaningful aging or durability assessment, evaluated clinically relevant pretreatments, or represented contemporary biomodification or adhesive strategies. Clinical trials and systematic clinical reviews were incorporated when they provided patient-level context, particularly for NCCLs.

The principal synthesis focused on permanent CAD, eroded dentin, and naturally sclerotic dentin. Sound dentin was used as a comparator and mechanistic reference. Evidence from artificially induced caries, experimental erosion, bovine dentin, or artificially hypermineralized dentin was retained selectively as mechanistic evidence but was not assumed to be biologically equivalent to naturally altered human tissue. Enamel-only studies, primary dentin, irradiated dentin, endodontically treated dentin, contamination studies, and unrelated restorative substrates were outside the principal scope unless required to clarify a directly relevant mechanism.

Because this review was designed as a narrative synthesis, study identification and selection were iterative rather than conducted within a prospectively locked systematic-review workflow. No PRISMA flow diagram or retrospective record counts are reported because such reconstruction would imply screening documentation that was not generated prospectively. No de novo meta-analysis, duplicate independent screening, formal risk-of-bias scoring, or formal certainty-of-evidence grading was performed. When risk-of-bias judgments are mentioned, they refer to assessments reported in the cited systematic reviews unless explicitly stated otherwise. Interpretation emphasized source hierarchy, substrate model, aging protocol, biological and statistical unit, consistency across studies, and the distinction between surrogate laboratory endpoints and clinical restorative outcomes.

3. Narrative Evidence Synthesis and Sports-Dentistry Context

The retrieved evidence was organized into five linked domains: the biological basis of substrate-dependent resin-dentin adhesion, bonding to CAD, bonding to eroded dentin, bonding to sclerotic dentin, and the sports-dentistry context in which altered dentin may be encountered. A comparative evidence map was then used to identify where apparently similar reductions in laboratory bond strength arise from different substrate-level mechanisms.

3.1. Biological Basis of Resin-Dentin Adhesion and Substrate Dependency

Resin-dentin adhesion depends on controlled interaction with both the mineral and organic phases of dentin. In etch-and-rinse (E&R) systems, phosphoric acid removes the smear layer and demineralizes superficial dentin, exposing a collagen scaffold that must be infiltrated by primer and adhesive resin. In conventional wet-bonding protocols, controlled moisture is required to avoid collapse of the demineralized collagen network; excessive residual water, however, can impair complete resin replacement and leave zones of denuded or incompletely infiltrated collagen at the base of the hybrid layer (Breschi et al. 2018).

Self-etch (SE) systems use acidic monomers to demineralize and infiltrate dentin simultaneously and may reduce the mismatch between demineralization depth and resin penetration. Universal adhesives (UAs) increase application flexibility because the same material may be used in SE, E&R, or selective-etch approaches and commonly contains functional monomers such as 10-methacryloyloxydecyl dihydrogen phosphate (10-MDP), which can interact chemically with residual hydroxyapatite. However, the label "universal" does not denote uniform chemistry. Contemporary products differ in acidity, solvent composition, water content, hydrophilicity, filler content, functional monomers, initiator systems, and recommended application procedures (Breschi et al. 2025).

Immediate micromechanical retention is only one component of adhesive performance. Hybrid-layer degradation can occur through hydrolysis and leaching of resin components as well as degradation of incompletely protected collagen. Hydrophilic monomers improve infiltration of moist dentin but also increase water sorption and permeability after polymerization. Endogenous dentinal enzymes, including matrix metalloproteinases (MMPs) and cysteine cathepsins, can contribute to collagen degradation when the organic matrix is exposed and susceptible to proteolysis. These mechanisms explain why strategies that appear equivalent at 24 hours may diverge after water storage, thermocycling, mechanical loading, or other forms of artificial aging (Breschi et al. 2018; Breschi et al. 2025).

Substrate composition changes the relative importance of each mechanism. When mineral has already been lost, as in CAD or eroded dentin, additional demineralization can increase the thickness of collagen that requires resin infiltration and may reduce the amount of accessible hydroxyapatite available for functional-monomer interaction. When mineralization is excessive, as in sclerotic dentin, conventional acidic monomers may fail to penetrate the hypermineralized surface and occluded tubules. Consequently, the same adhesive may encounter opposite barriers in different clinical lesions (Breschi et al. 2018; Breschi et al. 2025).

This substrate dependency also limits interpretation of bond-strength data generated on sound dentin. Sound coronal dentin provides a relatively standardized experimental surface, whereas natural CAD and sclerotic dentin are biologically heterogeneous and eroded dentin is highly protocol dependent. The clinically relevant question

is therefore not whether an adhesive has a high average bond strength under ideal conditions, but whether its chemistry and application protocol can create a stable interface on the specific tissue that remains after disease or wear (Breschi et al. 2018; Breschi et al. 2025).

3.2. Caries-Affected Dentin

3.2.1. Structural and biochemical characteristics

Contemporary caries management has shifted from routine non-selective excavation toward preservation of tissue when this can be achieved without compromising pulpal health or the restorative seal (Schwendicke et al. 2016). In this review, CAD refers to the experimental or histopathologic substrate used in the adhesion literature; it should not be treated as a direct chairside excavation category. Clinically retained dentin after selective removal may overlap biologically with CAD, but contemporary consensus terminology intentionally uses hardness-based descriptors because clinical manifestations cannot be mapped reliably onto histopathology (Innes et al. 2016).

Traditional histopathologic descriptions distinguish CAD from an outer, more heavily infected and structurally disrupted carious dentin zone. CAD is generally less bacterially contaminated, less structurally damaged, and more capable of remineralization, but the boundary is not absolute and should not be used as a chairside excavation endpoint. Caries-related mineral loss, dissolution and redistribution of apatite, increased intertubular porosity, altered tubule contents, and changes in the collagenous matrix reduce hardness and can impair uniform hybridization. Evidence derived exclusively from sound dentin therefore cannot be extrapolated uncritically to CAD (Ekambaram et al. 2015; Innes et al. 2016; Schwendicke et al. 2016; Isolan et al. 2018).

Natural CAD is also intrinsically variable. Lesion activity, depth, hardness, mineral distribution, bacterial history, collagen integrity, and the method used to remove infected tissue all affect the final bonding surface. Grinding, bur excavation, chemomechanical removal, and other preparation methods may expose different depths of dentin and produce different smear layers. Artificial caries models reduce this variability but may reproduce only selected features of natural lesions. This distinction should be explicit when experimental adjuncts are proposed for clinical use (Ekambaram et al. 2015; Isolan et al. 2018).

3.2.2. Bonding performance and adhesive strategy

The most consistent conclusion in the CAD literature is that laboratory adhesion is generally inferior to that achieved on sound dentin. A systematic review of 29 laboratory studies on natural CAD found higher bond strength to sound dentin in most comparisons (Ekambaram et al. 2015). A subsequent meta-analysis of 40 studies favored sound dentin for both E&R adhesives (mean difference 10.04 MPa; 95% CI, 8.14–11.94 MPa; I^2 = 95%) and SE adhesives (mean difference 6.76 MPa; 95% CI, 5.30–8.23 MPa; I^2 = 89%). When analysis was restricted to CAD, E&R adhesives were favored over SE adhesives by 3.13 MPa (95% CI, 1.82–4.44 MPa; I^2 = 72%) (Isolan et al. 2018). The high heterogeneity and predominantly unclear risk of bias limit the precision and generalizability of these pooled estimates.

The older E&R-versus-SE comparison should not be converted into a universal rule for contemporary universal adhesives. A 2024 systematic review and meta-analysis of 26 studies found no consistent improvement from additional phosphoric-acid pretreatment when mild or ultra-mild universal adhesives were used on CAD; aging reduced performance, and CAD remained less favorable than sound dentin regardless of application mode (Karadas et al. 2024). More recent primary evidence reinforces material dependence. In a 2026 study of three universal adhesives, phosphoric-acid pre-etching negatively affected bonding to CAD for two tested materials after aging (Bilce et al. 2026). Another 2026 study found that one-year bonding to CAD varied according to adhesive formulation and conditioning mode, with degradation observed across tested interfaces (Garfias & De Goes 2026). These data are better interpreted as evidence of adhesive-specific interaction than as support for a single etching category.

More aggressive conditioning is therefore not automatically beneficial on a substrate that is already partially demineralized. Additional acid treatment may enlarge the demineralized zone that requires resin infiltration and may reduce residual mineral available for functional-monomer interaction. At the same time, adhesive acidity, solvent behavior, hydrophilicity, and application protocol matter, so the absence of a pooled advantage cannot be translated into a rule that phosphoric acid is never useful. The current evidence supports material-specific interpretation rather than category-wide recommendations (Breschi et al. 2018; Karadas et al. 2024; Breschi et al. 2025; Bilce et al. 2026; Garfias & De Goes 2026).

3.2.3. Bond durability and biomodification

CAD appears not only to reduce immediate bond strength but also to create a more degradation-prone interface. A long-term study of natural CAD bonded with E&R and SE adhesives found lower microtensile bond strength than on sound dentin, together with a significant decrease in CAD bond strength after six months of

water storage, increased exposed collagen, and poorer adhesive infiltration (Erhardt et al. 2008). These findings support the concept that the altered substrate affects both initial hybridization and subsequent interface stability.

Several adjunctive approaches have therefore targeted collagen degradation or matrix stability. In a 12-month experimental study, incorporation of chlorhexidine (CHX) into an ethanol-wet bonding protocol preserved bond strength to CAD and reduced nanoleakage with a hydrophobic adhesive (Ekambaram et al. 2014). Chemical cross-linkers have also enhanced bond strength and collagen stability in experimental CAD models (Macedo et al. 2009). A two-year aging study under simulated intrapulpal pressure reported preservation effects of chlorhexidine pretreatment in a specific SE protocol (Mobarak 2011). These results are mechanistically informative, but the techniques are highly protocol specific and have not established routine clinical benefit with contemporary universal adhesives.

Recent biomodification research has shifted from relatively simple enzyme inhibition toward direct modification of collagen chemistry and adhesive composition. A collagen-reactive isocyanate-based primer increased microtensile bond strength, reduced nanoleakage, produced more homogeneous hybrid layers, and reduced matrix metalloproteinase activity in CAD immediately and after aging (Tang et al. 2023). A 2026 study evaluated a dual-reactive functional monomer termed ITCM as a 5-second primer and reported improved immediate and aged bonding to CAD, together with findings consistent with chemical bridging, deeper adhesive infiltration, greater collagen enzymatic resistance, and MMP inhibition (Tang et al. 2026). Although biocompatibility experiments were also reported, restoration-level clinical efficacy has not been established.

Other experimental approaches modify the adhesive itself. Nanoencapsulated quercetin or naringin incorporated into universal adhesives improved aged microtensile bond strength and reduced nanoleakage in artificially induced CAD after six months of water storage (Falconi-Páez et al. 2025). Zinc oxide/copper nanoparticles did not increase immediate bond strength but improved two-year bond-strength preservation and reduced nanoleakage, suggesting a greater effect on degradation kinetics than on initial micromechanical retention (Gutiérrez et al. 2026). In a 2026 study, incorporation of 1 wt% chitosan/hydroxyapatite nanoparticles into an ultra-mild universal adhesive increased 24-hour and six-month microtensile bond strength to CAD and increased surface mineral deposition without a detectable reduction in degree of conversion (El-Demellawy et al. 2026). Surface mineral deposition should not, however, be equated with clinically validated dentin remineralization. The microtensile data were analyzed with a multilevel approach appropriate for the nested beam-within-tooth structure, although the findings remain preclinical.

Biomimetic remineralization is another developing direction. A 2026 study found that a higher concentration of the self-assembling peptide P11-4 improved immediate bonding to CAD, whereas adding dentin matrix protein 1 to low-dose P11-4 did not clearly outperform P11-4 alone. No tested pretreatment restored bond strength to the level of sound dentin, and only immediate outcomes were assessed (de Alencar Pinto Magalhães et al. 2026). P11-4 should therefore be described as promising rather than clinically validated.

A 2026 systematic review focused specifically on pretreatment strategies for natural CAD identified nine eligible in vitro studies. Cross-linking and biomodulation strategies showed the most consistently favorable bond-strength results, remineralization approaches had condition-dependent effects, and fluoride-based strategies were more variable. Methodological heterogeneity prevented meta-analysis; six studies were judged to have moderate risk of bias and three low risk of bias (Costa et al. 2026). This synthesis supports a mechanistic rationale for matrix-directed pretreatment while confirming that the evidence remains laboratory based.

Taken together, CAD is supported by a qualitative hierarchy of evidentiary strength rather than a formal certainty grading: the laboratory bonding disadvantage relative to sound dentin is consistent; the preferred etching approach for contemporary adhesives remains material dependent; and biomodification strategies remain experimental despite increasingly sophisticated mechanistic data (Isolan et al. 2018; Tang et al. 2023 2026; Karadas et al. 2024; Falconi-Páez et al. 2025; Bilce et al. 2026; Costa et al. 2026; de Alencar Pinto Magalhães et al. 2026; El-Demellawy et al. 2026; Garfias & De Goes 2026; Gutiérrez et al. 2026).

3.3. Eroded Dentin

3.3.1. Structural alterations and bonding impairment

Erosive tooth wear is a multifactorial process in which chemical dissolution of dental hard tissues by non-bacterial acids interacts with mechanical wear. Once dentin is exposed, repeated acidic challenges dissolve the mineral phase and leave a superficial organic matrix enriched in exposed collagen. The thickness and morphology of this layer depend on acid type, pH, exposure time, cycling pattern, saliva, and concomitant abrasion. Experimental erosion models therefore vary substantially and may not reproduce the cumulative physicochemical processes that characterize clinical erosive tooth wear (Wiegand et al. 2021).

The collagen-rich surface creates both a physical and biochemical limitation. Adhesive monomers may not penetrate the full depth of the demineralized matrix, leaving a zone of incompletely infiltrated collagen that is mechanically weak and susceptible to hydrolytic and enzymatic degradation. Additional phosphoric-acid etching

may further demineralize the superficial layer, although the clinical importance of this theoretical effect depends on adhesive chemistry and the severity of erosion (Wiegand et al. 2021).

A systematic review and meta-analysis synthesized 47 studies, of which 45 were in vitro and two were in situ. Erosion reduced dentin bond strength by 10.2 MPa compared with sound dentin (95% CI, 8.6–11.9 MPa lower), whereas eroded enamel was not significantly impaired (Wiegand et al. 2021). A second systematic review and meta-analysis including 23 studies likewise supported a bonding disadvantage for eroded dentin and found that selected mechanical or chemical pretreatments may improve laboratory performance (de Rossi et al. 2022). Both syntheses point in the same direction, although their concordance should not be treated as independent replication because overlap of primary studies is likely.

3.3.2. Adhesive mode and management of the altered surface

Pooled evidence does not support a general superiority of E&R or SE bonding on eroded dentin. The largest meta-analysis found no significant difference between the two application modes (Wiegand et al. 2021). This finding is relevant to clinical hypothesis generation, but direct clinical evidence remains very limited; adhesive mode alone does not appear to overcome the altered-substrate problem demonstrated in laboratory models.

A more consistent laboratory signal concerns management of the superficial organic layer. Pretreatments designed to remove or stabilize the demineralized matrix have improved bond strength in selected protocols, with favorable signals reported for collagen cross-linkers, sodium hypochlorite (NaOCl), silver diamine fluoride, selected laser treatments, and diamond-bur preparation (Wiegand et al. 2021). A second meta-analysis also supported selected pretreatments but identified a somewhat different pattern of agent-specific effects (de Rossi et al. 2022). These differences illustrate that conclusions for individual conditioners depend on the materials, aging protocols, and treatment sequences studied and should not be reduced to a universal clinical recipe.

Mechanical surface preparation has a clear mechanistic rationale because it can remove the most heavily altered layer and restore a morphology closer to intact dentin. A long-term comparison of several surface-preparation methods found that erosion reduced bond strength in all groups, but the disadvantage was less pronounced after superficial diamond-bur preparation. After one year of water storage, eroded interfaces showed more adhesive failures and greater silver infiltration (Zimmerli et al. 2012). The result illustrates the balance between adhesion and tissue preservation: minimal roughening may improve the interface, but unnecessary removal of tooth structure conflicts with minimally invasive principles.

Chemical removal of the organic matrix has been investigated primarily with NaOCl. In a three-year in vitro study, deproteinization influenced the durability of resin-eroded dentin interfaces, demonstrating that effects may persist well beyond the immediate test period (de Siqueira et al. 2018). These findings support substrate modification as a rational strategy while underscoring the dependence on adhesive chemistry and treatment sequence.

Stabilization rather than removal of the collagen layer represents a complementary approach. A two-year water-storage study found that collagen cross-linking agents could improve long-term bonding behavior on eroded dentin, with protocol-dependent effects (de Siqueira et al. 2020). Cross-linking is biologically plausible because it may increase matrix stiffness and resistance to proteolysis, but formulation, concentration, carrier, application time, and compatibility with the adhesive remain unresolved.

A 2024 study found that 6% NaOCl, particularly when paired with an arylsulfinate-containing activator, improved bonding of a two-step SE adhesive to experimentally eroded dentin and increased resistance to thermocycling; performance did not simply become equivalent to sound dentin (Yang et al. 2024). The result supports management of the altered surface but should not be generalized beyond the tested material and deproteinization protocol.

A 2026 six-year in vitro study provided additional durability evidence for silver diamine fluoride (SDF) pretreatment. In an experimentally eroded human-dentin model, 12% SDF, 38% SDF, and 38% SDF combined with potassium iodide improved immediate microtensile bond strength; after six years, SDF reduced bond degradation, with 38% SDF producing the most favorable aged values, whereas potassium iodide attenuated the protective effect (Moreira et al. 2026). Importantly, the tooth—not the individual beam—was defined as the statistical unit (n = 8 per group), beams from each tooth were distributed across aging times, and the analysis used a mixed-model ANOVA with time as a repeated measure. The study therefore addressed within-tooth dependence more appropriately than a beam-level independent-observation analysis would. Its endpoint nevertheless remained laboratory bond strength rather than restoration survival.

Collectively, the eroded-dentin literature provides a more consistent preclinical signal for management of the altered surface than for any single named pretreatment. The altered collagen-rich layer is a central interfacial barrier, and simply switching from SE to E&R—or vice versa—is unlikely to eliminate the substrate deficit (Zimmerli et al. 2012; de Siqueira et al. 2018; de Siqueira et al. 2020; Wiegand et al. 2021; de Rossi et al. 2022; de Siqueira et al. 2023; Yang et al. 2024; Moreira et al. 2026).

3.3.3. Durability and translational limitations

Durability is particularly important in erosive tooth wear because the bonding interface may be placed on a substrate that has already undergone repeated chemical challenge. The largest systematic synthesis found that erosion impaired both immediate and aged dentin bonding; 32 of 47 included studies had medium risk of bias, and heterogeneity was substantial across erosion protocols, aging procedures, and bond-strength tests (Wiegand et al. 2021). The predominance of in vitro evidence limits direct clinical extrapolation.

Long-term experiments are valuable because they show that interventions can influence degradation trajectories, but they remain surrogate evidence. One-year water storage, two-year cross-linker studies, three-year deproteinization experiments, and six-year aging studies are more informative than 24-hour bond strength, yet none directly establishes superior restoration survival in patients with erosive tooth wear. A six-year laboratory evaluation of 10 universal adhesives on experimentally eroded human dentin showed no overall advantage of E&R over SE application. Bond strength generally decreased and nanoleakage increased with aging, and degradation varied among formulations; 10-MDP-containing adhesives showed more favorable long-term behavior in that experiment (de Siqueira et al. 2023). A separate six-year study showed concentration-dependent preservation of bonding after SDF pretreatment, again supporting a durability signal without establishing patient-level benefit (Moreira et al. 2026).

3.4. Sclerotic Dentin

3.4.1. Morphology of natural sclerotic dentin

Sclerotic dentin presents an adhesive problem that is almost the inverse of superficial demineralization. It develops through progressive mineral deposition in dentinal tubules and at the exposed surface, often in association with chronic cervical or occlusal wear. The resulting tissue may be less permeable, more acid resistant, and more difficult to hybridize than sound dentin (Tay et al. 2000).

Classical ultrastructural work on natural human cervical lesions described a hypermineralized surface layer that could be devoid of intact banded collagen, with dentinal tubules blocked by acid-resistant sclerotic casts and resin tags rarely observed. Overall bond strength to natural sclerotic dentin was approximately 20% lower than to sound cervical dentin in that experimental system (Tay et al. 2000). These features provide a direct morphological explanation for why an adhesive that performs well on freshly prepared sound dentin may interact incompletely with a longstanding NCCL surface.

A subsequent study of natural NCCLs found higher bond strength to sound dentin than to sclerotic dentin regardless of the tested conditioning approach. Phosphoric-acid pretreatment produced localized benefit but did not uniformly overcome the hypermineralized barrier; microscopy showed that the self-etching primer often failed to penetrate beyond the surface layer (Kwong et al. 2002). The finding cautions against interpreting stronger acid conditioning as a simple, universally effective solution.

3.4.2. Surface conditioning and adhesive strategy

Both surface treatment and adhesive architecture can influence bonding to sclerotic dentin, although findings are system specific. A comparison of one-step and two-step SE adhesives on natural human sclerotic dentin found lower microtensile bond strength than on normal dentin for both materials; after thermocycling, the two-step system showed better durability than the one-step system (Xie et al. 2010). This supports the broader concept that simplified hydrophilic adhesives may be more vulnerable to aging, but it does not establish superiority of all two-step systems on sclerotic dentin.

An investigation of human occlusal sclerotic dentin found that phosphoric-acid preconditioning opened more tubules and produced higher microtensile bond strength than simply doubling the etch-prime time in the tested SE system (Kwansirikul et al. 2020). Such findings strengthen the mechanistic argument for disrupting an acid-resistant surface but remain dependent on the specific substrate and material combination.

A 2026 in vitro study evaluated three adhesives after no pretreatment, aluminum-oxide air abrasion, or bioglass air abrasion on human dentin categorized as sclerotic (del Cid Rodríguez et al. 2026). Aluminum-oxide air abrasion increased microtensile bond strength in an adhesive-dependent manner, most prominently with the tested two-step SE adhesive, whereas bioglass did not consistently improve bonding; most groups remained stable after six months of water storage. Interpretation requires substantial statistical caution: only 16 teeth yielded 461 beams across nine experimental combinations, and individual beams were treated as analytical observations in a four-way ANOVA without a tooth-level random effect despite acknowledged within-tooth dependence. Nominal precision and p values may therefore be anti-conservative because of clustering. In addition, the classification of sclerosis and beam position was not supported by a validated tooth-level microscopic grading procedure. These findings should be regarded as exploratory and material-specific rather than as precise tooth-level evidence.

Recent systematic syntheses emphasize how limited the evidence remains. A 2025 systematic review identified only eight eligible in vitro studies evaluating pretreatment of cervical non-carious sclerotic dentin. Results were inconsistent; some ethylenediaminetetraacetic acid (EDTA) or NaOCl protocols showed no benefit or inferior outcomes, whereas prolonged phosphoric-acid conditioning and combined mechanical-chemical approaches appeared most promising, although the underlying evidence was judged low quality (Hamdi et al. 2025). A 2026 systematic review restricted to human sclerotic dentin likewise reported generally lower bond strength than sound dentin. Phosphoric acid, mechanical roughening, EDTA, and selected two-step SE or universal adhesives were frequently associated with improvement, but heterogeneity prevented meta-analysis (Holiela et al. 2026).

Accordingly, surface conditioning should be interpreted as an interaction among sclerosis severity, surface morphology, adhesive acidity, functional monomers, and resin hydrophilicity. The term "universal adhesive" alone is not sufficiently informative to predict behavior on a heavily sclerotic lesion. Broader meta-analytic evidence on universal adhesives also indicates that etching effects vary with adhesive acidity and substrate rather than being determined by the category label alone (Cuevas-Suárez et al. 2019). Statements such as "two-step self-etch is best" or "phosphoric acid is mandatory" exceed the current evidence.

3.4.3. Clinical context of non-carious cervical lesions

NCCLs provide an important clinical model because restorations often have limited macromechanical retention and may involve substantial areas of sclerotic dentin. However, an NCCL trial is not a pure experiment on the sclerotic dentin interface. Clinical performance also depends on enamel margins, lesion geometry, cervical moisture control, restorative material, occlusal loading, patient behavior, and the degree of sclerosis. This distinction is necessary when laboratory surface-treatment findings are compared with patient-level retention data.

The limitation is illustrated by a double-blind randomized clinical trial in which dentin roughening before application of a universal adhesive in E&R or SE mode did not improve retention, marginal adaptation, or discoloration at 18 months (Loguercio et al. 2018). A mechanical pretreatment that appears logical from ultrastructural or bond-strength data therefore does not necessarily produce a detectable short-term clinical advantage. This discordance is not evidence that morphology is irrelevant; it shows that restoration performance is a multifactorial endpoint and reinforces the need for clinical validation of substrate-specific interventions.

3.5. Sports-Dentistry Context: Why Altered Dentin Matters in Athletes

Sports-related relevance is best conceptualized as a two-stage pathway: athlete-associated exposures may contribute to oral disease or tooth wear, whereas substrate biology determines adhesive behavior once restorative treatment is required. These pathways should not be conflated. A 2025 systematic review and meta-analysis of 10 observational studies estimated pooled prevalence of caries at 44.4% (95% CI, 33.9–55.1%) and dental erosion at 36.5% (95% CI, 22.6–51.7%) among elite athletes, with high heterogeneity and important limitations in participant recruitment and sample-size adequacy (Campana Zamudio et al. 2025). Needleman et al. (2026) likewise concluded that oral diseases are common in elite athletes and should be integrated into the wider sports-medicine duty of care. These data support a meaningful restorative workload in sport, but they do not demonstrate an athlete-specific dentin phenotype.

Sports nutrition and exercise physiology provide plausible exposure pathways. Frequent carbohydrate intake around training and competition can increase cariogenic challenge, while acidic sports or energy products may contribute to erosive exposure. Dehydration, high airflow, and mouth breathing during prolonged exercise may reduce salivary protection and prolong oral clearance of acids or carbohydrates. However, the causal evidence is heterogeneous. A 2025 systematic review of sports drinks and dental erosion included only five cross-sectional studies and found inconsistent associations overall (Memon et al. 2025). A comprehensive review of sports diet and oral health emphasized interactions among carbohydrate frequency, acidic products, saliva, and oral hygiene (Schulze and Busse 2024), while a study of young athletes reported sport- and diet-associated differences in salivary and microbiological measures but remained observational (Tripodi et al. 2025). Accordingly, these factors should be treated as contextual risk modifiers rather than as proof that sport itself causes altered dentin.

The most direct restorative connection concerns eroded dentin. Repeated acid challenge can progress from enamel loss to dentin exposure, leaving the collagen-rich demineralized surface described in the bonding literature. Caries can likewise lead to clinically retained altered dentin after appropriate selective tissue removal. Sclerotic dentin is less specifically linked to sport but may coexist with longstanding cervical or occlusal wear. Athlete-specific oral-care trials demonstrate that prevention and management of erosion can be studied directly in sporting populations; for example, a randomized trial in rugby players evaluated hydroxyapatite-based home-care protocols, but it did not assess adhesive bonding or restoration survival and did not demonstrate clear intergroup superiority across the reported clinical indices (Butera et al. 2022). Thus, the current sports-dentistry evidence informs case mix and prevention more strongly than adhesive protocol selection.

3.6. Comparative Evidence Map

The three substrates converge at the level of reduced predictability but diverge at the level of mechanism. CAD is a partially demineralized, mechanically altered residual tissue in which both mineral availability and collagen quality are changed. Eroded dentin is dominated by a superficial demineralized organic matrix created by repeated acid exposure. Sclerotic dentin presents a mineralized, tubule-occluded barrier that resists interaction. The same laboratory outcome — lower bond strength — therefore has different biological meanings (Tay et al. 2000; Kwong et al. 2002; Isolan et al. 2018; Wiegand et al. 2021).

This comparison explains why the direction of a rational pretreatment differs. In CAD, preservation of clinically appropriate retained tissue is consistent with contemporary caries management, whereas matrix-stabilizing adjuncts remain predominantly experimental. In eroded dentin, removal or stabilization of the altered collagen-rich layer has the strongest preclinical support, but no single surface-management protocol has been clinically validated as a routine standard. In sclerotic dentin, controlled chemical or mechanical modification of the mineralized barrier is mechanistically plausible but remains incompletely validated clinically. Tables 1 and 2 summarize these contrasts and map representative strategies to the level and limitations of the available evidence (Innes et al. 2016; Schwendicke et al. 2016; Wiegand et al. 2021; Hamdi et al. 2025; Costa et al. 2026; Holiela et al. 2026).

Table 1. Structural characteristics and dominant interfacial barriers of the three target dentin substrates

Substrate	Dominant structural alteration	Main interface consequence	Mechanistic treatment target	Key evidence limitation
Caries-affected dentin	Partial demineralization, increased porosity, altered apatite/collagen, reduced mechanical quality	Less predictable hybridization and greater susceptibility to degradation	Preserve clinically appropriate retained tissue; optimize infiltration and matrix stability rather than assume stronger etching is always beneficial	Predominantly in vitro evidence; heterogeneous natural/artificial lesion models; laboratory CAD is not identical to a chairside excavation category
Eroded dentin	Superficial demineralized collagen-rich layer produced by repeated non-bacterial acid exposure	Altered organic layer may exceed effective resin infiltration and degrade with aging	Remove or stabilize the altered superficial matrix while preserving tooth structure	Erosion and aging protocols vary markedly; very limited in situ and clinical evidence
Sclerotic dentin	Hypermineralized surface, intratubular mineral deposition, tubular occlusion	Reduced permeability and resistance to conventional acidic conditioning	Controlled chemical and/or mechanical disruption of the mineralized barrier when clinically justified	Small, heterogeneous laboratory evidence base; NCCL clinical outcomes do not isolate dentin sclerosis

Table 2. Evidence map of representative substrate-specific bonding strategies

Substrate / strategy	Key sources	Main finding	Interpretation	Major limitation
CAD: etching strategy	(Isolan et al. 2018; Karadas et al. 2024; Bilce et al. 2026; Garfias & De Goes 2026)	CAD generally bonds less effectively than sound dentin. Older evidence favored E&R, whereas contemporary universal-adhesive evidence shows material-dependent effects and no consistent benefit from additional phosphoric acid.	Adhesive chemistry and generation matter; no universal etching rule.	High heterogeneity; predominantly laboratory outcomes.
CAD: durability / anti-degradation	(Erhardt et al. 2008; Macedo et al. 2009; Mobarak 2011; Ekambaram et al. 2014)	CAD interfaces show aging-related deterioration; selected CHX-based or cross-linking protocols preserved bond strength in specific systems.	Supports vulnerability of CAD and mechanistic value of degradation control.	Protocol specific; limited modern clinical translation.

CAD: biomodification / remineralization	(Tang et al. 2023 2026; Falconi-Páez et al. 2025; Costa et al. 2026; de Alencar Pinto Magalhães et al. 2026; El-Demellawy et al. 2026; Gutiérrez et al. 2026)	Collagen-reactive primers, dual-reactive primers, flavonoids, nanoparticles, and biomimetic strategies improved selected laboratory outcomes. A 2026 systematic review found the most consistent signal for cross-linking/biomodulation.	Promising preclinical direction; some recent studies also used longer aging and/or multilevel analysis.	Heterogeneous laboratory evidence; no restoration-level clinical trials.
Eroded dentin: E&R vs SE	(Wiegand et al. 2021; de Siqueira et al. 2023)	No significant pooled difference between E&R and SE modes; long-term laboratory data show aging- and material-dependent degradation.	Substrate alteration and adhesive formulation appear more important than application mode alone.	Predominantly laboratory evidence; erosion models and materials vary.
Eroded dentin: superficial bur preparation	(Zimmerli et al. 2012)	Diamond-bur preparation reduced the long-term bonding penalty in an erosion model.	Mechanistically plausible minimal removal of severely altered surface.	In vitro; tissue-removal trade-off.
Eroded dentin: matrix / surface pretreatment	(de Siqueira et al. 2018; de Siqueira et al. 2020; Yang et al. 2024; Moreira et al. 2026)	NaOCl, collagen cross-linking, or SDF can improve aged bonding under selected protocols; a six-year SDF study strengthened the durability signal.	Surface-matrix management is a rational preclinical target; no specific protocol is clinically validated as routine care.	Concentration, sequence, and adhesive compatibility remain protocol specific; no clinical survival evidence.
Sclerotic dentin: morphology	(Tay et al. 2000; Kwong et al. 2002)	Hypermineralized surface and sclerotic casts limit penetration; sound dentin generally bonds better.	Provides biological rationale for tailored conditioning.	Older material systems; lesion heterogeneity.
Sclerotic dentin: adhesive architecture / conditioning	(Xie et al. 2010; Kwansirikul et al. 2020; del Cid Rodríguez et al. 2026)	A two-step SE system showed better aging in one study; phosphoric-acid preconditioning and aluminum-oxide air-abrasion improved laboratory outcomes in selected material/substrate combinations.	Material- and substrate-specific benefit is possible.	Individual in vitro studies; very small tooth-level sample in the 2026 air-abrasion study, with multiple beams analyzed without a tooth-level random effect, creating a clustering/pseudoreplication concern.
Sclerotic dentin: systematic evidence	(Hamdi et al. 2025; Holiela et al. 2026)	Phosphoric acid and mechanical modification are often favorable, but results are inconsistent and heterogeneous.	Tailored interpretation is better supported than a single protocol.	Few studies; no robust pooled estimate.
NCCL: clinical roughening	(Loguercio et al. 2018)	Dentin roughening did not improve 18-month clinical performance in the tested universal-adhesive protocol.	Laboratory rationale does not guarantee clinical benefit.	NCCL is multifactorial; one material and follow-up period.

4. Discussion

4.1. Why One Adhesive Strategy Does Not Fit All

The central synthesis of this review is that "compromised dentin" can be a convenient umbrella term but becomes biologically misleading if it implies a common treatment. The three substrates reviewed here can all generate lower laboratory bond strength while presenting contrasting interfacial barriers. A substrate-centered interpretation therefore provides a more coherent explanation of the evidence than a hierarchy in which one adhesive category is assumed to be universally superior.

The CAD literature is particularly instructive. An older meta-analysis favored E&R over SE adhesives on CAD, whereas contemporary evidence on mild and ultra-mild universal adhesives does not demonstrate a

consistent benefit from additional phosphoric-acid etching (Isolan et al. 2018; Karadas et al. 2024). Recent 2026 primary studies further demonstrate adhesive-specific responses to conditioning mode (Bilce et al. 2026; Garfias & De Goes 2026). These findings can coexist because adhesive formulations, acidity, functional monomers, solvents, and study eligibility criteria differ. Category labels therefore cannot substitute for material-specific chemistry and substrate context.

Eroded dentin provides a second example. E&R and SE modes were not significantly different in the largest pooled analysis, whereas interventions addressing the altered collagen layer showed a more consistent laboratory benefit (Wiegand et al. 2021). In sclerotic dentin, by contrast, a resistant mineralized surface provides a plausible rationale for additional conditioning. Yet primary studies and recent reviews show variable and material-dependent benefit, and clinical roughening did not produce a clear retention advantage in at least one randomized trial (Kwong et al. 2002; Loguercio et al. 2018; Hamdi et al. 2025; del Cid Rodríguez et al. 2026). The direction of evidence is substrate dependent, while support for any specific protocol remains less robust than the evidence that the substrates themselves differ biologically.

4.2. Aged Laboratory Outcomes Add Information Beyond Immediate Bond Strength but Remain Surrogate Endpoints

Immediate microtensile or microshear bond strength remains useful for screening adhesive interactions, but restoration failure is a time-dependent process. Laboratory evidence is more informative when an intervention is evaluated after water storage, thermocycling, or other aging and when bond-strength findings are supported by nanoleakage or interfacial microscopy. Studies showing progressive collagen exposure and reduced CAD bond strength after water storage, long-term erosion experiments demonstrating increasing interface degradation, and nanoparticle modifications affecting aged rather than immediate strength all reinforce this principle (Erhardt et al. 2008; Zimmerli et al. 2012; de Siqueira et al. 2023; Gutiérrez et al. 2026).

Nevertheless, aged laboratory strength remains a surrogate outcome. A meta-analysis of dental adhesive bond-strength tests found no meaningful correlation between tensile bond-strength results and clinical retention loss or marginal integrity in cervical restorations, although some correlation with marginal discoloration was observed (Heintze et al. 2015). A pooled difference of approximately 10 MPa therefore represents a sizeable laboratory difference, but no validated clinical threshold permits conversion of such an effect into a predicted percentage of restoration failure. This distinction is critical because the literature on altered dentin is dominated by surrogate outcomes.

Bond-strength methodology also has statistical vulnerabilities. Microtensile testing commonly generates multiple beams from one tooth; these observations are biologically clustered and are not fully independent. A statistical evaluation of microtensile methodology demonstrated within-tooth correlation and showed that analytical conclusions can change when the tooth effect is accounted for (Eckert & Platt 2007). Future studies should report the numbers of teeth and beams separately, specify how premature failures are handled, and either use tooth-level summaries or hierarchical models that account for clustering. Apparent precision based on large numbers of bonded beams or specimens should not be confused with biological replication.

4.3. Clinical Translation and an Evidence-Informed Decision Framework

Patient-level evidence is most developed for NCCLs, but it must be interpreted carefully. A 2023 systematic review of universal adhesives found no significant overall difference between E&R and SE strategies for retention, marginal adaptation, marginal discoloration, secondary caries, or postoperative sensitivity over the evaluated follow-up periods (Doshi et al. 2023). A 2026 network meta-analysis of 74 trials found favorable performance for several strategies involving selective enamel etching or E&R combined with universal adhesives and nanocomposites, while a separate systematic clinical review found no single UA brand that could be considered a gold standard (Chaple Gil et al. 2026; D'Elia et al. 2026).

These clinical data do not prove that aggressive dentin etching improves adhesion specifically to sclerotic dentin. Selective enamel etching primarily modifies the enamel margin, and NCCL retention reflects the whole restoration-tooth complex. Clinical trials are therefore essential for patient-level outcomes but relatively weak for isolating the molecular mechanism of the sclerotic interface. Conversely, laboratory sclerotic-dentin studies isolate the substrate but cannot reproduce the complexity of the cervical lesion. The two evidence streams should be considered complementary rather than interchangeable.

The framework proposed in Table 3 therefore summarizes evidence-informed considerations rather than clinical recommendations. For CAD, the most defensible approach is to preserve clinically appropriate retained tissue while avoiding the assumption that additional phosphoric acid is universally beneficial. For eroded dentin, the altered surface layer is a rational experimental target, but no management method has been established as a routine clinical standard. For pronounced sclerotic dentin, controlled surface modification may be a hypothesis-informed option when clinically indicated and supported by material-specific evidence, but no single protocol is mandatory. In all three settings, manufacturer instructions, isolation, adhesive application quality, solvent evaporation, light curing, and restorative design remain fundamental.

Table 3. Evidence-informed interpretive framework for bonding to altered dentin

Substrate	What is relatively well supported	Potential considerations	What should not be claimed	Evidence status
CAD	Laboratory bond strength is generally lower than to sound dentin; selective carious tissue removal may leave biologically altered dentin, but clinical hardness categories are not synonymous with histopathologic CAD.	Follow material-specific adhesive instructions; prioritize careful application and interface stability; do not add phosphoric-acid pretreatment solely because the substrate is CAD.	That E&R is universally superior, that all clinically retained dentin is CAD, or that experimental biomodifiers improve restoration survival.	Consistent laboratory substrate disadvantage; limited direct clinical evidence for adjunctive strategies.
Eroded dentin	Erosion reduces laboratory dentin bond strength; E&R and SE are not consistently different in pooled evidence.	When surface modification is clinically indicated, use the least destructive approach compatible with restorative needs; chemical deproteinization and collagen-stabilizing protocols remain predominantly preclinical.	That one pretreatment is a validated routine standard or that laboratory MPa gains prove longer restoration survival.	Consistent laboratory substrate disadvantage; very limited direct clinical evidence for substrate-specific pretreatment.
Sclerotic dentin	Natural sclerosis can impede conditioning and resin penetration.	Controlled roughening or enhanced chemical conditioning may be considered as a hypothesis-informed, material-specific option in pronounced sclerosis; no protocol is a validated universal standard.	That phosphoric acid, EDTA, roughening, or a specific adhesive class is mandatory for all NCCLs.	Limited and heterogeneous laboratory evidence; clinical NCCL evidence is indirect because outcomes are multifactorial.

A further limitation of a substrate-centered framework is that the dominant interfacial alteration cannot always be characterized directly at chairside. Clinical assessment based on lesion history, dentin hardness, surface appearance, location, and the presence of wear or sclerosis provides only a proxy for the microscopic substrates investigated in laboratory studies. This is particularly important in carious lesions, where consensus terminology explicitly cautions against direct mapping of clinical appearance to histopathology (Innes et al. 2016). The framework should therefore be interpreted as a mechanistic aid to reasoning, not as a diagnostic classification or clinical guideline.

4.4. Sports Dentistry: From the Athletic Oral Environment to Adhesive Restorative Care

For sports dentistry, altered-dentin adhesion is most relevant through the clinical pathway from athlete oral-health exposures to restorative need. Elite athletes have a substantial documented burden of caries and erosive tooth wear, but the available evidence does not support attributing those conditions uniformly to sport participation (Campana Zamudio et al. 2025; Needleman et al. 2026). The practical consequence is that sports-dentistry services should be prepared to diagnose and restore carious and erosive lesions while also addressing modifiable exposures that may perpetuate disease or wear.

The restorative decision pathway should remain substrate centered. For a carious lesion, disease control and clinically appropriate selective tissue removal come first; the residual dentin should then be bonded according to its characteristics and the selected adhesive rather than according to athletic status. For erosive tooth wear, control of intrinsic and extrinsic acid exposure, hydration and salivary protection, dietary counseling, and minimally invasive restorative planning are complementary to substrate-specific bonding. For pronounced sclerosis, controlled surface modification may be considered only when supported by lesion- and material-specific evidence. In each scenario, the athlete context changes prevention, exposure assessment, and follow-up priorities more plausibly than it changes the fundamental chemistry of the adhesive interface.

A useful distinction is between performance relevance and treatment efficacy. Oral disease may interfere with well-being, training, and availability, which justifies integration of dental care into sports medicine; however, there is no evidence that a particular bonding protocol improves athletic performance, recovery, or readiness. Likewise, the presence of frequent sports-drink use, a mouthguard, or high training load should not be used as a surrogate indication for stronger dentin etching or experimental biomodification. The causal chain must

remain explicit: sport-related exposure may influence lesion development; lesion biology determines the substrate; and the substrate–material interaction determines the bonding strategy.

Athlete-specific intervention evidence remains largely preventive rather than restorative. The rugby-player trial by Butera et al. (2022) evaluated home-care management of dental erosion, not bonding to eroded dentin, and therefore cannot be used to infer restoration longevity or substrate-specific adhesive benefit. No athlete-specific clinical trial of dentin bonding or restoration survival was identified in the targeted search. This is a genuine research gap and a more defensible sport-specific contribution than extrapolating a bonding effect from athletic status.

Table 4. Sports-related contexts and their relevance to altered-dentin adhesive care

Sports-related context	Supported oral-health link	Potential altered substrate	Implication for adhesive care	Evidence boundary
Frequent carbohydrate fueling (drinks, gels, bars, mouth-rinse strategies)	Can increase cariogenic challenge when exposure is frequent; overall athlete caries burden is substantial.	Caries-affected or clinically retained altered dentin after selective carious tissue removal.	Control caries risk and follow substrate- and material-specific bonding principles; athletic status does not select the adhesive.	Mostly observational/contextual evidence; confounding by hygiene, fluoride, diet, and baseline disease.
Acidic sports/energy products and repeated oral acid exposure	Biologically plausible contribution to erosive tooth wear; epidemiologic association with sports drinks is inconsistent.	Eroded dentin when wear progresses beyond enamel.	Control acid exposure and salivary risk factors; if restoration is indicated, manage the altered dentin surface according to substrate evidence.	Sports-drink reviews are based largely on cross-sectional studies; causality and product-specific effects remain uncertain.
Dehydration, high airflow, and mouth breathing during exercise	May reduce salivary availability and buffering, potentially amplifying cariogenic or erosive conditions.	Indirectly relevant to carious or eroded substrates.	Optimize hydration and preventive care; these factors are not indications for a different dentin-etching protocol.	Physiological and observational evidence; direct restoration-survival effects have not been established.
High burden of oral disease in elite sport	Pooled evidence indicates frequent caries and erosion in elite athletes.	Higher clinical probability of encountering altered dentin requiring adhesive care.	Integrate screening, prevention, timely restorative care, and recall within sports-medicine pathways.	Prevalence estimates are heterogeneous and do not prove sport causation.
Athlete-specific erosion-management studies	A rugby-player RCT evaluated hydroxyapatite-based home care for erosion.	Erosion context only; no direct bonding substrate outcome.	Supports athlete-specific preventive research but does not justify sport-specific adhesive protocols.	No athlete-specific dentin-bonding or restoration-survival trial identified.

4.5. Limitations of the Current Evidence and of This Narrative Review

Several limitations constrain substrate-specific interpretation. First, the evidence base is predominantly laboratory based: high-level syntheses for CAD and eroded dentin largely pool in vitro bond-strength studies rather than clinical restoration outcomes, and even in sclerotic dentin, where NCCL trials are abundant, the trials evaluate complex restorations rather than the sclerotic dentin interface in isolation (Isolan et al. 2018; Wiegand et al. 2021; Hamdi et al. 2025; Costa et al. 2026; Holiela et al. 2026).

Second, substrate definition is inconsistent. Natural CAD varies biologically and does not correspond perfectly to hardness-defined clinical excavation endpoints; erosion protocols differ in acid type and cycling severity; and experimental hypermineralization does not reproduce natural sclerosis. Studies using the same diagnostic label may therefore test biologically different tissues (Innes et al. 2016; Wiegand et al. 2021). Third, aging protocols are highly heterogeneous. Twenty-four-hour water storage, months or years of immersion, thermocycling, simulated pulpal pressure, mechanical loading, and enzymatic challenges cannot be treated as equivalent measures of durability (Wiegand et al. 2021; Karadas et al. 2024).

Fourth, material heterogeneity complicates category-level conclusions. UAs, SE adhesives, and E&R systems encompass products with substantially different acidity, hydrophilicity, solvent systems, functional monomers, and polymerization characteristics. A statistically pooled "adhesive mode" effect may therefore obscure material-specific behavior. Fifth, unit-of-analysis problems can materially distort precision in microtensile studies: multiple beams from one tooth are clustered observations, and analyses that treat them as fully independent risk pseudoreplication unless the tooth is used as the experimental unit or the nesting is modeled explicitly (Eckert & Platt 2007).

This review has additional methodological limitations. It was designed as a targeted narrative synthesis rather than a registered systematic review. Study identification was purposive, exact search syntax and record

counts were not prospectively archived, no duplicate independent screening or de novo risk-of-bias assessment was performed, and no formal certainty grading was applied. The review may therefore be affected by selection and citation bias and cannot claim exhaustive retrieval or reproducibility equivalent to a systematic review. Its purpose is to integrate selected high-level syntheses with mechanistically important primary studies while making the boundaries between preclinical and clinical inference explicit.

The sport-focused component has additional limitations. Athlete oral-health evidence is dominated by observational prevalence and exposure studies across heterogeneous sports, and the contextual sports search in this review was targeted rather than a separate systematic review. Accordingly, pooled prevalence estimates are used to establish clinical relevance, not to infer that sport caused the substrate. No athlete-specific dentin-bonding or restoration-survival study was identified, so sport-specific effect modification of adhesive performance remains untested.

4.6. Future Research Priorities

Future studies should begin with better characterization of the substrate. Natural human lesions should be used whenever feasible, with objective description of lesion origin, location, hardness, degree of sclerosis or demineralization, surface morphology, and the procedure used to expose the bonding surface. Artificial models remain valuable, but their similarity to clinical lesions should be demonstrated rather than assumed.

Durability experiments should move beyond immediate strength and use aging protocols that match the proposed mechanism. An MMP-inhibiting or collagen-cross-linking strategy, for example, should be evaluated over sufficiently long storage and with complementary interfacial endpoints. Studies should also predefine the tooth as the biological experimental unit or use statistical models appropriate for nested beams. Reporting should include premature failures, the number of teeth, the number of beams per tooth, and exact aging conditions.

Head-to-head comparison is also needed. Many promising interventions are evaluated only against an untreated control, making cross-study ranking unreliable. Competing strategies should be tested on the same natural substrate with the same adhesive, aging protocol, and analytical framework. Emerging collagen-reactive primers, flavonoids, nanoparticles, and biomimetic peptides additionally require characterization of degree of conversion, solvent behavior, shelf stability, cytotoxicity, mechanical properties, and manufacturing reproducibility before clinical translation (Tang et al. 2023; Falconi-Páez et al. 2025; Costa et al. 2026; de Alencar Pinto Magalhães et al. 2026; Gutiérrez et al. 2026).

Most importantly, substrate-specific prospective clinical trials are required. Studies following selective carious tissue removal should document clinically relevant hardness-based excavation endpoints and lesion characteristics rather than assuming that the retained dentin represents a uniform histopathologic CAD category (Innes et al. 2016). Trials in erosive tooth wear should compare minimally invasive surface-management strategies in patients rather than only in pH-cycled specimens. NCCL trials should stratify or document the degree of dentin sclerosis when the purpose is to test a sclerotic-dentin hypothesis. Outcomes should include retention, marginal adaptation, discoloration, postoperative sensitivity, secondary caries, repair, and survival, with follow-up sufficient to detect degradation-related differences (Doshi et al. 2023; Chaple Gil et al. 2026).

Within sports dentistry, athlete-specific research should prioritize prospective restoration cohorts and pragmatic trials rather than post hoc subgroup claims. Studies could enroll athletes with confirmed erosive tooth wear or caries requiring adhesive restorations, prospectively record sport discipline, training load, carbohydrate and acid exposure, hydration, salivary flow and pH, mouth breathing, and mouthguard use, and follow retention, marginal integrity, postoperative sensitivity, repair, secondary caries, and restoration survival. Athlete-level exposures must be analyzed at the participant level; multiple teeth or restorations within one athlete are clustered observations and require participant-level summaries or multilevel methods such as mixed-effects models or generalized estimating equations. Matched non-athlete comparators may improve external-validity assessment, but causal claims about sport require careful control of diet, oral hygiene, fluoride exposure, socioeconomic factors, and baseline disease. Randomization to alternative bonding strategies is justified only when there is substrate-specific mechanistic rationale and clinical equipoise, not merely because participants are athletes.

The translational sequence should be explicit: biological rationale, reproducible laboratory effect, persistence after aging, independent replication, material feasibility, and finally meaningful clinical benefit. A statistically significant increase in microtensile bond strength is a useful experimental signal, not the endpoint of evidence-based restorative dentistry.

5. Conclusion

Caries-affected, eroded, and sclerotic dentin are clinically relevant but biologically distinct bonding substrates. CAD is characterized predominantly by partial demineralization, reduced mechanical quality, and alteration of the residual organic matrix; eroded dentin by a superficial demineralized collagen-rich layer; and sclerotic dentin by hypermineralization and tubular occlusion. These differences explain why a single adhesive or conditioning strategy cannot be expected to perform optimally across all compromised dentin surfaces.

For CAD, the laboratory bonding disadvantage relative to sound dentin is well established, but current evidence does not justify routine phosphoric-acid pre-etching across all mild or ultra-mild universal adhesives. For eroded dentin, pooled laboratory evidence suggests that management of the altered surface may be more relevant than selection of E&R versus SE mode alone, although no pretreatment has sufficient clinical evidence to qualify as a universal standard. For sclerotic dentin, enhanced chemical or mechanical conditioning is mechanistically plausible, but available studies are sparse and heterogeneous, and patient-level evidence remains indirect (Isolan et al. 2018; Wiegand et al. 2021; Karadas et al. 2024; Hamdi et al. 2025; Bilce et al. 2026; Costa et al. 2026; del Cid Rodríguez et al. 2026; Garfias & De Goes 2026; Holiela et al. 2026; Moreira et al. 2026).

The synthesis therefore supports a substrate-centered conceptual approach: consider the dominant structural barrier, use material-specific adhesive instructions, avoid unnecessary tissue damage, and interpret laboratory improvements within their translational limits. In sports dentistry, the strongest rationale is case-mix and exposure related rather than adhesive-chemistry related: elite athletes show a substantial burden of caries and erosive tooth wear, while sports nutrition and exercise-associated salivary conditions may contribute to clinically relevant oral exposures. These associations are heterogeneous and do not establish a distinct athlete dentin phenotype. No athlete-specific bonding protocol can currently be recommended. Future progress should link prospectively measured sport exposures, well-characterized substrates, clustered patient-level analyses, and clinically meaningful restoration outcomes.

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Writing - review and editing: [NLH], [WJ], [KKu], [KKe];
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