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Pathophysiological Cascades of Chlorine-Induced Dental Erosion and Associated Dentin Hypersensitivity in Competitive Swimmers

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Abstract

Background : Dentin hypersensitivity is a condition that is becoming more prevalent and can significantly diminish health-related quality of life. A recognized cause of this condition is dental erosion, which is defined as the irreversible loss of dental enamel due to acid exposure in the absence of bacterial involvement. The acids responsible for dental erosion may originate from intrinsic or extrinsic sources.

Aim : This narrative review summarizes current evidence regarding the impact of exposure to chlorinated swimming pool water on the development of erosive dental lesions and the occurrence of dentin hypersensitivity among swimmers.

Material and methods : This review article was based on scientific publications published in PubMed, Quality in Sport, and MDPI databases between 2003 and 2026. The literature search focused on studies investigating the prevalence of dentin hypersensitivity and dental erosion among swimmers.

Results : Prolonged exposure to chlorinated swimming pool water may constitute a significant risk factovr for the development of erosive lesions in dental hard tissues in swimmers. Regular contact between enamel and water with improperly controlled pH levels and chlorine compounds promotes demineralization of the tooth surface, resulting in the progressive loss of hard dental tissues. Consequently, elite swimmers show an erosive tooth wear (ETW) prevalence of 26–90%, much higher than non-swimming athletic controls (25.6%).

Conclusions : Extended competitive swimming poses a clear occupational risk to dental tissues. Addressing this issue requires urgent interdisciplinary reforms in sports medicine, strict regulation of pool chemistry saturation, targeted behavioral guidance, such as avoiding mechanical brushing immediately after training, and mandatory systematic remineralization screening.

Key words : Dental erosion; dentin hypersensitivity; competitive swimming; athletic performance; sports dentistry.

1. Introduction

Dental erosion, also known as erosive tooth wear (ETW), involves the chronic, pathological, and irreversible loss of dental hard tissues through a chemical process that occurs independently of bacterial activity (Wiegand and Attin 2007, Guldag et al. 2008, Buczkowska-Radlińska et al. 2013, Nijakowski et al. 2022, Favero et al. 2024, Chojnowska et al. 2025). ETW differs from dental caries; while caries result from local acid production within cariogenic biofilms, erosive demineralization is caused by direct exposure of the tooth surface to acidic substances (Wiegand and Attin 2007, Czapiński et al. 2026). The etiology of ETW is multifactorial and is typically categorized into intrinsic and extrinsic risk factors that determine the severity of tissue loss (Lussi and Jaeggi 2008, Boonviriya et al. 2017, Ali et al. 2024, Chojnowska et al. 2025).

Intrinsic risk factors arise from endogenous sources, primarily involving gastric acid present in the oral cavity (Guldag et al. 2008, Lussi and Jaeggi 2008, Ali et al. 2024). Chronic digestive disorders, such as gastroesophageal reflux disease (GERD), and psychiatric disorders characterized by binge or purge behaviors, including bulimia and anorexia nervosa, expose dental structures to highly concentrated hydrochloric acid (Wiegand and Attin 2007, Lussi and Jaeggi 2008, Chojnowska et al. 2025). Biological factors, such as hyposalivation and reduced salivary buffering capacity, further increase intrinsic risk by failing to neutralize acid challenges (Dawes 2008, Lussi and Jaeggi 2008). In contrast, extrinsic risk factors originate from external environmental sources, most notably diets, fruit juices, and sports drinks (Nijakowski et al. 2022). Occupational exposures, such as contact with acidic fumes in battery factories or prolonged exposure to inadequately maintained swimming pool water, also constitute significant external triggers that can result in rapid and extensive enamel dissolution (Wiegand and Attin 2007, Buczkowska-Radlińska et al. 2013).

Competitive swimmers are recognized as a high-risk group due to occupational exposure to chlorinated water. Although disinfection is necessary for pool safety, the specific method used determines the erosive potential (Zebrauskas et al. 2014, Favero et al. 2024). Sodium hypochlorite generally maintains an alkaline pH, whereas gas chlorination, commonly used in high-performance facilities, generates hydrochloric acid (HCl) as a byproduct (Dawes and Boroditsky 2008, Buczkowska-Radlińska et al. 2013). International standards recommend a pH range of 7.2 to 7.8; however, insufficient buffering can cause a rapid decrease in pH to as low

as 3.0 (Dawes and Boroditsky 2008, Rao et al. 2019, Abdelrahman et al. 2023). When the pH drops below the critical thresholds of 5.5 for enamel and 6.0 for dentin, the environment becomes highly aggressive, leading to rapid dissolution of hydroxyapatite crystals (Zebrauskas et al. 2014, Boonviriya et al. 2017, Rao et al. 2019).

This damage builds up over years of intense training, made worse because athletes breathe primarily through their mouths during tough sessions. (Chojnowska et al. 2025). Loss of protective enamel frequently exposes dentinal tubules, resulting in dentin hypersensitivity (DH). DH, characterized by short, sharp pain in response to thermal or chemical stimuli, affects approximately 69.6% of professional swimmers and represents a significant yet often overlooked consequence of the sport (Rao et al. 2019, Abdelrahman et al. 2023).

Research Objective. This review aims to synthesize current scientific evidence regarding the chemical and biological mechanisms through which exposure to chlorinated pool water leads to dental erosion and subsequent dentin hypersensitivity in competitive swimmers.

Research Problems. This review examines the correlation between inadequate buffering capacity in swimming pool water and the accelerated dissolution of tooth minerals. It evaluates whether cumulative years of training are the primary predictor for the onset of dentin hypersensitivity. Additionally, it examines how exercise-induced changes in salivary flow rates and biological buffering capacity influence athletes' susceptibility to acid-induced lesions during high-intensity aquatic training.

Research Hypotheses. The primary hypothesis is that the marked difference in dental erosion rates between competitive swimmers and the general population is attributable to prolonged exposure to training environments. In these settings, pool water is chemically undersaturated with respect to tooth minerals. The secondary hypothesis asserts that dentin hypersensitivity in competitive swimmers is a direct physiological consequence of the erosive opening and increased patency of dentinal tubules resulting from inadequately maintained swimming pool environments.

2. Research materials and methods

This narrative review analyzes existing scientific literature and does not involve live human subjects. The research draws from a rigorously selected set of peer-reviewed documents sourced from international biomedical and sports science databases and publisher platforms, including PubMed, Quality in Sport, and MDPI. The literature search focused on publications from 2003 to 2026 that specifically examine the prevalence, etiology, and clinical manifestations of dental erosion (ETW) and dentin hypersensitivity (DH) in competitive swimmers. Only original research articles, systematic reviews, and detailed clinical case reports were included.

A structured protocol was used to extract and synthesize data from the selected studies, starting with environmental and chemical metrics. This included monitoring swimming pool pH fluctuations, especially drops below the critical enamel threshold of 5.5, caused by hydrochloric acid formation in gas-chlorinated systems. The protocol also synthesized data on training volume, such as average weekly hours often 15-20 and total years of competitive experience, to assess cumulative dental risk (Buczkowska-Radlińska et al. 2013, Zebrauskas et al. 2014, Rao et al. 2019, Abdelrahman et al. 2023).

Clinical and biological data were collected on dental parameters, emphasizing lesion severity and distribution using standardized indices such as the Basic Erosive Wear Examination (BEWE) and the Lussi scale, as well as diagnostic criteria for dentinal tubule patency (Zebrauskas et al. 2014, Nijakowski et al. 2022, Favero et al. 2024).

Artificial intelligence (AI) tools were used for basic data analysis, text formatting, and language improvement during manuscript preparation. The authors thoroughly reviewed and verified the final manuscript to ensure academic integrity and take full responsibility for its content.

3. Research results

3.1. Pool Water Chemistry and the Mechanisms of Enamel Demineralization

Improper management of disinfection systems is the primary risk factor for erosive tooth wear (ETW) in aquatic environments. In large facilities, water is usually treated by gas chlorination, in which chlorine gas reacts with water to form hypochlorous acid, a germicidal agent, and hydrochloric acid (HCl), a highly acidic byproduct (Buczkowska-Radlińska et al. 2013, Chojnowska et al. 2025). The World Health Organization recommends maintaining pool water pH between 7.2 and 7.8. However, insufficient buffering, often due to inadequate soda ash (sodium carbonate) addition, can cause pH to drop rapidly to decalcifying levels as low as 3.0 (Buczkowska-Radlińska et al. 2013, Chojnowska et al. 2025, Rao et al. 2019, Abdelrahman et al. 2023). These acidic conditions promote hydroxyapatite dissociation and inhibit the mineralization of dental hard tissues (Rao et al. 2019).

Aggressive chemical dissolution occurs when pool water pH drops below the critical thresholds for tooth mineral stability, approximately 5.5 for enamel and 6.0 for dentin (Zebrauskas et al. 2014, Rao et al. 2019, Abdelrahman et al. 2023). However, pH alone does not determine erosivity; the concentrations of calcium, phosphate, and fluoride ions also influence the degree of saturation (Buczkowska-Radlińska et al. 2013, Lussi and Carvalho 2014). Consequently, pool water with a neutral pH (e.g., 7.2) but undersaturated with respect to hydroxyapatite can still cause significant surface demineralization due to a persistent chemical driving force (Buczkowska-Radlińska et al. 2013).

3.2. Training Volume and Cumulative Exposure Risks

Epidemiological studies show that dental erosion affects 26% to 90% of competitive swimmers, a rate much higher than in recreational swimmers (10%) and non-swimming athletes (25.6%) (Buczkowska-Radlińska et al. 2013, Abdelrahman et al. 2023, Chojnowska et al. 2025). This increased risk is closely linked to both the intensity and cumulative duration of pool exposure. Elite athletes often spend 15 to 20 hours per week in chemically treated water during high-intensity training (Buczkowska-Radlińska et al. 2013, Abdelrahman et al. 2023). Clinical data

show that swimmers training more than 4 hours per session have a 92.3% prevalence of ETW, compared to only 20% among those training less than 2 hours (Favero et al. 2024).

The risk of irreversible dental tissue loss increases throughout an athlete's career. Swimmers with more than three years of competitive experience have 5.3 times higher odds of developing erosive lesions (Rao et al. 2019, Abdelrahman et al. 2023). Those with over 10 years of practice face an even greater risk (Zebrauskas et al. 2014). This damage is worsened by physiological responses to strenuous exercise, such as mouth breathing, which leads to oral cavity dehydration and a significant decrease in stimulated salivary flow (hyposalivation) (Abdelrahman et al. 2023, Chojnowska et al. 2025, Czapiński et al. 2026). The resulting loss of the protective salivary pellicle, which normally acts as a diffusion barrier, makes the labial surfaces of the anterior teeth especially vulnerable to repeated acid exposure and permanent loss of tooth volume (Buczkowska-Radlińska et al. 2013, Chojnowska et al. 2025).

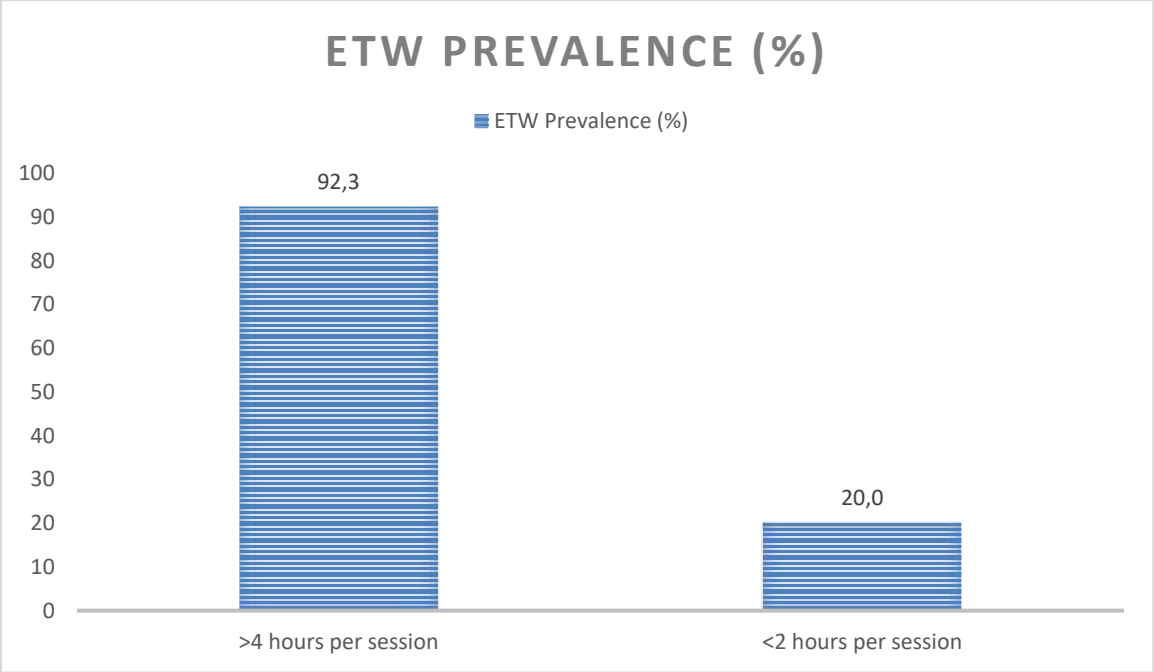

Figure 1. Comparison of dental erosion prevalence based on daily training volume (based on data from Favero et al. 2024).

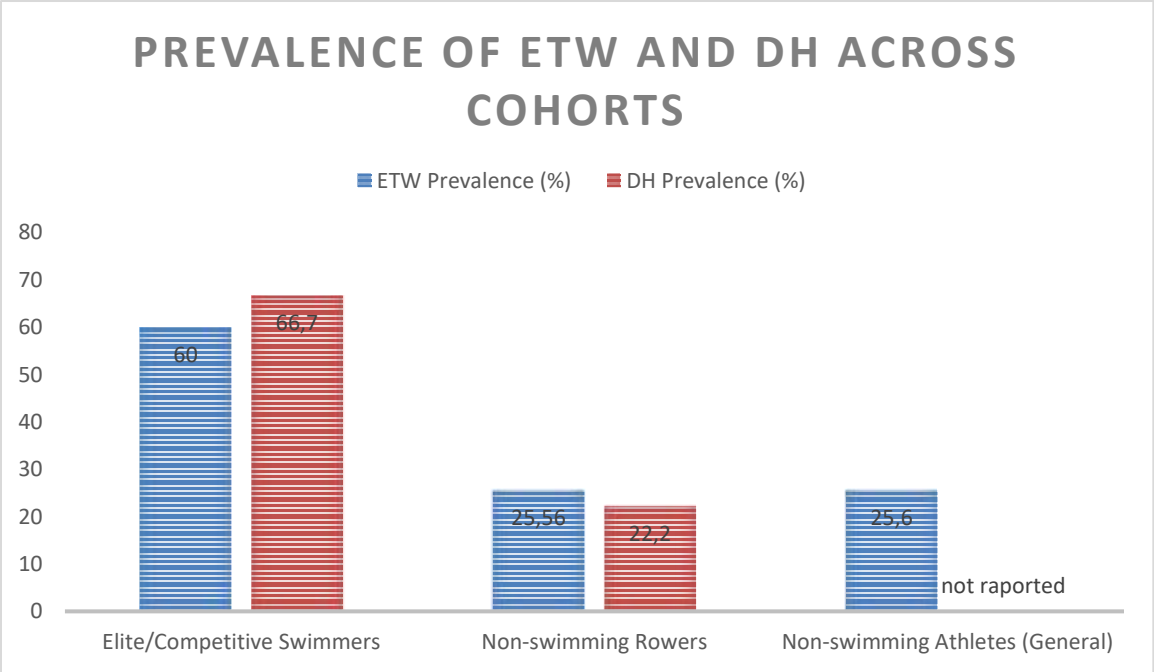

Figure 2. Comparative analysis of erosive tooth wear (ETW) and dentin hypersensitivity (DH) prevalence between elite/competitive swimmers and non-swimming athletic controls (based on data from Abdelrahman et al. 2023)

3.3. Dentin Hypersensitivity Prevalence and Clinical Mechanisms

The chronic progression of erosive tooth wear (ETW) in the aquatic training environment serves as a primary precursor to the development of dentin hypersensitivity (DH), a condition defined as short, sharp pain arising from exposed dentin in response to external stimuli (Consensus Board 2003, West et al. 2013, Liu et al. 2020). The pathogenesis of DH in swimmers follows a biphasic process: lesion localization, involving the cumulative loss of protective enamel due to chlorinated water exposure, and lesion initiation, characterized by the subsequent opening and patency of the underlying dentinal tubules (Davari et al. 2013, West et al. 2013, Chojnowska et al. 2025). While general population estimates of DH prevalence range from 11.5% to 33.5%, clinical data targeting high-performance aquatic athletes indicate a significantly higher burden, with 69.6% of professional swimmers exhibiting acute hypersensitivity symptoms (Rao et al. 2019, Zeola et al. 2019, Abdelrahman et al. 2023).

The physiological triggers for this pain are comprehensively explained by the hydrodynamic theory, which suggests that various external stimuli—thermal, tactile, osmotic, or chemical—

cause the quick movement of fluid within the patent dentinal tubules (Davari et al. 2013, Liu et al. 2020, Scribante et al. 2025). In the context of competitive swimming, frequent contact with cold pool water or the inhalation of cool air during mouth breathing induces an outward flow of dentinal fluid, which excites the intradental myelinated A-β and A-δ sensory fibers located at the pulp-dentin border (Davari et al. 2013, West et al. 2013, Liu et al. 2020). This neurosensory activation causes the characteristic rapid-onset pain that professional swimmers report as a consistent annoyance. (Rao et al. 2019, Chojnowska et al. 2025).

Ultrastructural analysis of the dentin morphology in sensitive teeth stresses the severity of the erosive challenge faced by swimmers. Clinical investigations using scanning electron microscopy (SEM) have demonstrated that sensitive dentin surfaces contain eight times as many open tubules per unit area compared to non-sensitive surfaces (Consensus Board 2003, Davari et al. 2013). Furthermore, the diameter of these tubules is approximately twice as large in sensitive teeth, measuring 0.83 μm versus 0.43 μm in non-sensitive controls (Consensus Board 2003, West et al. 2013). Following to Poiseuille's Law, which states that fluid flow is proportional to the fourth power of the tubule radius, these morphological changes lead to a 16-fold increase in fluid flow from diameter changes alone, potentially culminating in a total fluid shift 100 times greater than that of nonsensitive dentin (Consensus Board 2003, West et al. 2013).

3.4 Impact of Dental Pathologies on Training Quality and Performance

The manifestation of dental pain and erosive lesions is a key factor to the "elite-athlete oral health paradox," in which individuals in peak physical condition paradoxically exhibit poor oral health that directly compromises athletic performance (Czapiński et al. 2026). Approximately one-third of elite athletes report that their oral condition negatively affects training, sleep, and competitive focus (Czapiński et al. 2026). When a swimmer experiences a sudden, sharp pain from cold pool water splashing against sensitized anterior teeth, it triggers a sympathetic nervous system reflex (Czapiński et al. 2026). This physiological response, characterized by increased peripheral vascular resistance and heart rate, can distract the athlete from critical tactical cues and disrupt the precise technical execution required during intense-level training intervals (Liu et al. 2020, Abdelrahman et al. 2023, Czapiński et al. 2026).

Past immediate tactical distractions, dental pathologies exert a deleterious effect on mandatory hydration and nutritional protocols. Many competitive athletes utilize chilled isotonic sports drinks to maintain electrolyte and glucose levels during long sessions, thereby compounding the risk of erosive lesion formation. What is more, the consumption of these beverages can be accompanied by dental pain in athletes. (Nijakowski et al. 2022).

Furthermore, the chronic nature of DH can lead to sleep disturbances, compromising a recovery phase that is crucial for hormonal regulation and tissue repair in elite populations (Czapiński et al. 2026). Despite these risks, over 80% of swimmers do not perceive these dental sensations as a serious health concern, leading to significant underutilization of preventive dental services and a progressive decline in training quality (Rao et al. 2019, Chojnowska et al. 2025, Scribante et al. 2025).

Table 1. Summary of epidemiological studies evaluating the prevalence of erosive tooth wear (ETW) and dentin hypersensitivity (DH) in swimming cohorts.

Study (Author & Year)	**Sample Size (N) and Cohort Type**	**Dental Erosion / ETW Prevalence (%)**	**Dentin Hypersensitivity / DH Prevalence (%)**	**Key Operational Variables & Findings**
Buczkowska-Radlińska et al 2013	62 competitive swimmers; 69 recreational swimmers	26% (Competitive) / 10% (Recreational)	0%	Training: **>19 hours/week**; median pool pH 7.2 but chemically undersaturated with tooth minerals.
Zebrauskas et al 2014	132 regular swimmers (Lithuania)	35.6% (Total) / 50% (Adults)	17%	**>10 years** of practice correlated with a **3-fold greater risk** of developing erosive lesions.

Rao et al 2019	56 competitive swimmers (India)	48.2%	69.6%	Swimmers with **>3 years experience** had **5.3 times higher odds** of erosion.
Abdelrahman et al 2023	90 competitive swimmers; 90 rowers	60% (Swimmers) / 25.6% (Rowers)	66.7% (Swimmers)	Pool water pH dropped to **3.24**; average practice: **16.1 hours/week** over 8 years

3.5 Preventive Protocols and Oral Hygiene Behavioral Patterns

The behavioral and oral hygiene practices of competitive swimmers are characterized by a high frequency of oral hygiene maneuvers that, paradoxically, often accelerate the progression of erosive tooth wear (ETW) due to improper timing and technique (Buczkowska-Radlińska et al. 2013, Rao et al. 2019, Chojnowska et al. 2025). While competitive athletes typically show higher toothbrushing frequencies—with 40% of professional swimmers brushing more than twice daily compared to 27% of recreational peers—these habits often occur immediately after acid challenges (Buczkowska-Radlińska et al. 2013, Chojnowska et al. 2025). Furthermore, the frequent consumption of low-pH sports drinks (ranging from pH 2.4 to 4.5) during sessions of exercise-induced hyposalivation creates an integrated environment where chemical demineralization outpaces the biological protective capacity of the acquired salivary pellicle (Nijakowski et al. 2022, Abdelrahman et al. 2023).

Exposure to chlorinated water with a pH below the critical limit of 5.5 causes a temporary softening of the superficial enamel structure through the dissolution of hydroxyapatite crystals (Dawes 2003, Rao et al. 2019, Favero et al. 2024). When mechanical toothbrushing is performed on this acid-softened substrate, the resulting abrasive force rapidly removes tooth volume because sufficient time for salivary remineralization and rehardening has not elapsed (Guldag et al. 2008, Rao et al. 2019). Current clinical recommendations suggest that athletes should strictly avoid toothbrushing for at least 60 minutes after aquatic training to allow

initiation of natural remineralization at the enamel interface (Nijakowski et al. 2022, Chojnowska et al. 2025).

Evidence-based preventive protocols prioritize the systematic application of high-efficacy remineralization therapies to fortify the dental hard tissues against chemical dissolution. The use of bioavailable gluconate-chelated stannous fluoride (*SnF*2) toothpastes has been shown to provide an 83% benefit in protecting enamel against erosive challenges by forming a stable, acid-resistant layer on the tooth surface (West et al. 2021). Additionally, the application of casein phosphopeptide-amorphous calcium phosphate (CPP-ACP) or amorphous calcium phosphate (ACP) pastes functions as an essential mineral reservoir, releasing high concentrations of calcium and phosphate ions that selectively integrate into demineralized areas (Boonviriya et al. 2017, Chojnowska et al. 2025, Scribante et al. 2025). Experimental data indicate that weekly treatment with high-concentration fluoride gels (12,500 ppm F^-) or daily use of remineralizing mousses (*F−APC*) containing 1,450 ppm F^- can effectively maintain enamel microhardness even amid simulated professional training loads, including 4 hours of daily pool immersion (Favero et al. 2024).

Behavioral strategies focused on pH neutralization and physical protection are critical adjuncts to chemical therapy. Swimmers are encouraged to rinse the oral cavity with neutral tap water or alkaline solutions, such as sodium bicarbonate (baking soda), immediately after training to promote the swift return of the oral environment to a physiological pH (Rao et al. 2019, Abdelrahman et al. 2023, Chojnowska et al. 2025). Furthermore, the use of custom-fitted mouthguards acts as a substantial physical barrier, limiting the volume of chlorinated water in direct contact with the dental surfaces and preventing the "swishing" behavior that accelerates mineral dissolution (Rao et al. 2019, Abdelrahman et al. 2023, Chojnowska et al. 2025). It has even been proposed that lining these mouthguards with alkaline agents can further neutralize any acidic fluids that may pool against the teeth during training (Abdelrahman et al. 2023).
Despite the clinical severity of these risks, there remains a considerable lack of awareness regarding aquatic-specific dental hazards among both athletes and coaching staff. In many high-performance settings, only 20% of athletes use mouthguards as a preventive measure against environmental erosion (Abdelrahman et al. 2023).

4. Discussion

This review identifies a pronounced disparity in oral health between competitive swimmers and the general population. Global meta-analyses report that the baseline prevalence of dentin hypersensitivity (DH) in non-aquatic populations ranges from 11.5% to 33.5% (Zeola et al 2019). In contrast, professional swimmers exhibit a markedly higher prevalence of DH, reaching up to 69.6% (Rao et al. 2019; Abdelrahman et al. 2023). A comparable pattern is evident in erosive tooth wear (ETW), with prevalence among elite swimmers ranging from 26% to 90%, significantly exceeding the approximately 25,6% prevalence observed in non-swimming athletic cohorts or general young adult populations (Buczkowska-Radlińska et al. 2013, Nijakowski et al. 2022, Abdelrahman et al. 2023).

These elevated statistics indicate that competitive swimming poses a significant occupational hazard to dental hard tissues (Wiegand and Attin 2007, Rao et al. 2019, Chojnowska et al. 2025). Evidence suggests that the risk of irreversible tissue loss is directly attributable to the aquatic training environment, particularly when pool facilities fail to maintain proper chemical balance (Dawes and Boroditsky 2008, Buczkowska-Radlińska et al. 2013, Favero et al. 2024). Furthermore, swimmers with more than three years of experience have 5.3 times higher odds of developing lesions, highlighting the cumulative nature of this risk (Rao et al 2019; Abdelrahman et al. 2023).

A notable statistical paradox arises when comparing clinical findings with athlete self-reports. Although clinical examinations detect DH in nearly 70% of professional swimmers, over 80% of these individuals do not perceive these dental sensations as significant medical concerns or threats to long-term health (Rao et al. 2019; Chojnowska et al. 2025). This lack of awareness often persists until advanced structural tissue loss becomes evident, such as "veneer-like" smoothing of labial surfaces or protrusion of dental restorations above the eroded enamel (Dawes and Boroditsky 2008, Buczkowska-Radlińska et al. 2013, Abdelrahman et al. 2023). This perception gap underscores the need for more proactive screening protocols, as the absence of patient complaints does not rule out pathological environmental demineralization (Rao et al.2019, Chojnowska et al. 2025, Czapiński et al. 2026).

A distinct chemical chain reaction within pool disinfection systems initiates the pathophysiological progression of dental erosion in swimmers. Large-scale competitive facilities primarily employ gas chlorination, where chlorine gas reacts with water to generate hypochlorous acid (HOCl) as a disinfectant and hydrochloric acid (HCl) as an acidic byproduct (Buczkowska-Radlińska et al. 2013, Chojnowska et al. 2025). When maintenance staff does not provide sufficient sodium carbonate buffering, the HCl byproduct rapidly lowers the pool water pH to decalcifying levels of 3.0 or 2.7 (Dawes and Boroditsky 2008, Rao et al. 2019, Abdelrahman et al. 2023). This environmental change surpasses the critical thermodynamic thresholds for mineral stability, set at pH 5.5 for enamel and 6.0 for dentin (Guldag et al. 2008, Zebrauskas et al. 2014, Abdelrahman et al. 2023). When water pH drops below these critical thresholds, the fluid becomes chemically aggressive, causing rapid dissociation of the hydroxyapatite crystals that constitute dental structures (Rao et al. 2019, Abdelrahman et al. 2023). This process leads to continuous leaching of calcium and phosphate ions from the tooth surface, systematically removing the protective smear layer and superficial enamel (Davari et al. 2013, West et al. 2013, Favero et al. 2024). Experimental models demonstrate that such exposure significantly reduces microhardness and increases the patency of the underlying dentinal tubules (Boonviriya et al. 2017; Favero et al. 2024).

The culmination of this demineralization process is the onset of pain, as explained by the Hydrodynamic Theory. This widely accepted mechanism posits that exposure of patent dentinal tubules permits external stimuli, such as pressure changes from swimming strokes or thermal input from cold pool water, to induce rapid fluid shifts within the tubules (Davari et al. 2013, West et al. 2013 Liu et al. 2020). In competitive swimming, continuous exposure to cool water and mouth breathing during inhalation trigger outward fluid flow, thereby stimulating intradentally myelinated A-δ sensory fibers. (Consensus Board 2003, Davari et al. 2013, West et al. 2013). Hypersensitivity serves as a physiological stressor; sharp pain from cold pool water contacting eroded anterior teeth can activate the sympathetic nervous system, leading to increased heart rate and peripheral vascular resistance (Chojnowska et al. 2025, Czapiński et al. 2026). This neurosensory interference may compromise an athlete’s technical focus and mental concentration during high-intensity training and critical competitive events (Abdelrahman et al. 2023, Chojnowska et al. 2025, Czapiński et al. 2026).

5. Conclusions

In conclusion, this review demonstrates that chemical mismanagement of gas chlorination systems is the primary environmental factor contributing to irreversible dental tissue loss in competitive aquatic environments. When the acidic byproducts of water disinfection are not adequately buffered, pool acidity falls consistently below the critical thermodynamic thresholds required for enamel and dentin stability. This persistent, non-bacterial exposure accelerates the dissociation of hydroxyapatite crystals, positioning competitive swimming as a distinct occupational hazard with a markedly higher prevalence of erosive wear and dentin hypersensitivity compared to non-aquatic athletes.

Importantly, this substantial clinical burden is obscured by a significant perception gap, as athletes often normalize dental pain and sensitivity. Such subjective tolerance leads to delayed clinical reporting, permitting environmental demineralization to advance untreated until severe structural damage develops. In addition to oral health consequences, these dental pathologies directly impede optimal sports performance.

Therefore, mitigating this occupational risk requires the prompt adoption of multilevel preventive strategies and interdisciplinary organizational reforms. Comprehensive educational initiatives are essential to address common behavioral errors, such as mechanical toothbrushing immediately after training, while enamel remains chemically softened. Integrating dental professionals into multidisciplinary sports medicine teams is critical for establishing mandatory screening protocols and systematic remineralization strategies, thereby safeguarding the oral health of elite swimmers and supporting their athletic performance.

Disclosure

Supplementary Materials

Not applicable

Author Contributions

All authors have read and agreed to the published version of the manuscript.

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The authors deny any conflict of interest

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