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Article

Dental Caries

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Abstract: Dental caries is one of the most prevalent chronic infectious diseases globally, affecting nearly 100% of adults and a large proportion of children. It is a multifactorial condition resulting from the interaction between cariogenic bacteria, dietary sugars, and susceptible tooth surfaces. Streptococcus mutans and other acidogenic bacteria metabolize sugars to produce acids that demineralize tooth enamel and dentin. The ecological plaque hypothesis explains how frequent sugar intake alters the oral microbiota, favoring aciduric species and initiating caries formation. Clinically, caries manifests as white spots, cavitations, and lesions, which can be classified using the International Caries Detection and Assessment System (ICDAS). Histologically, carious lesions exhibit distinct zones based on demineralization and microbial activity. Fluoride, dietary control, oral hygiene, and regular dental assessments are critical in prevention. Diagnostic advancements, including laser and light-induced fluorescence, have improved early detection. Root caries and secondary caries also pose unique challenges, particularly in aging populations. A multidisciplinary approach involving dental professionals, public health experts, and patient education is essential to control and prevent dental caries. Despite preventive efforts, disparities in caries prevalence persist due to socioeconomic factors and limited access to care. Future strategies should focus on personalized prevention, minimally invasive treatment, and promoting equity in oral healthcare.

Keywords: dental caries, Streptococcus mutans, ecological plaque hypothesis, demineralization, remineralization, fluoride, enamel, dentin, ICDAS, white spot lesion, cariogenic bacteria, dietary sugars, oral microbiota, early detection, root caries

1. Introduction

Dentistry traces its origins to as early as 5000 B.C., when the prevalent belief was that dental caries were caused by a "tooth worm." [1]. The term "dental caries" was first introduced in medical literature in 1634[2], derived from the Latin word caries, meaning "decay." Originally, the term referred to visible holes or lesions in the teeth. Today, dental caries is recognized as one of the most ancient and widespread chronic diseases affecting humankind. It is classified as a chronic infectious disease caused by tooth-adherent cariogenic bacteria. These microorganisms metabolize dietary sugars to produce acids, which over time, lead to the demineralization of the tooth structure[3].

Etiology:

Dental caries refers to both the pathological process and the resulting lesions. The initiation of caries occurs within the biofilm that continuously covers the teeth and becomes active during every fluctuation in pH. A shift from a balanced oral microbiome to one dominated by acid-producing and acid-tolerant bacteria leads to caries development[4]. This shift is often driven by the frequent intake of fermentable carbohydrates such as glucose, fructose, maltose, and sucrose.

In its early stages, caries may not be clinically visible, although mineral loss is occurring within the tooth structure. Over time, this loss may lead to visible cavitation. Thus, dental caries is considered a dietary-microbial disease. Behavioral, psychological, and socioeconomic factors also contribute significantly to its occurrence[5]. Additionally,

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inadequate exposure to fluoride—a well-established protective agent against caries—further heightens disease risk.

Epidemiology:

Dental caries is the most prevalent disease globally, affecting nearly 100% of the adult population at some point in their lives[6]. The disease is particularly concentrated among individuals from lower socioeconomic backgrounds. Despite numerous prevention strategies and public health efforts, the global prevalence of dental caries has not seen a marked reduction over the past three decades.

Pathophysiology:

Microbial Role

Research has shown a higher incidence of bacteria such as Streptococcus mutans, Streptococcus sobrinus, and Lactobacillus species in advanced carious lesions. This observation initially led to the specific plaque hypothesis, which suggested that only specific bacteria caused dental caries. However, further evidence has shown that caries can develop even in the absence of these specific organisms. As a result, the nonspecific plaque hypothesis emerged, proposing that caries result from the collective metabolic activity of the oral microbiota[7].

The currently accepted ecological plaque hypothesis posits that caries result from a dysbiosis, or imbalance, within the biofilm microbiota[8]. Frequent sugar consumption creates acidic conditions that favor the growth of cariogenic bacteria while suppressing benign species.

Demineralization and Remineralization:

Cariogenic bacteria metabolize dietary sugars into organic acids—chiefly lactic acid—which lowers the pH of the biofilm and initiates enamel demineralization. This process increases enamel porosity and allows acids to penetrate deeper into the tooth. Minerals such as calcium and phosphate, released during demineralization, accumulate on the surface and can contribute to remineralization, particularly when aided by fluoride[9]. Saliva plays a critical role in buffering acids and facilitating the return of the biofilm to a neutral pH, enabling remineralization to occur.

Persistent acidic conditions, however, prevent this recovery and result in progressive mineral loss. Clinically, this appears as a white spot lesion, which can either be reversed or arrested through behavioral and preventive measures. If unaddressed, microcavities and ultimately macroscopic cavities form, which although still arrestable, leave irreversible structural damage[10].

Histopathology:

Histologically, carious lesions display four distinct zones, three of which are clinically visible. The outermost zone comprises necrotic and contaminated tissue, heavily populated with microorganisms and presenting as soft dentin. The next layer is the demineralization zone, characterized by fewer microbes and a nutrient-deprived, anaerobic environment—clinically corresponding to leathery dentin[11]. The innermost translucent zone, located near the dental pulp, is composed of firm but demineralized dentin and is typically free of microorganisms due to their inability to penetrate this depth.

2. Materials and Methods

Study Design:

This study was designed as a comprehensive literature review combined with an observational analysis of current data regarding the etiology, pathophysiology, histopathology, and prevention of dental caries. The objective was to identify and analyze the mechanisms by which dental caries develops, with a particular emphasis on microbial interaction, dietary influences, fluoride exposure, and the role of social and behavioral determinants in disease prevalence and prevention.

Data Collection: The data used in this study were collected through a systematic search of scientific databases, including PubMed, MEDLINE, ScienceDirect, and the Cochrane Library[12]. The search focused on articles published between 1990 and 2024. Keywords used included "dental caries," "etiology of caries," "Streptococcus mutans,"

"demineralization," "remineralization," "fluoride and caries prevention," "caries epidemiology," and "plaque hypothesis." Boolean operators such as "AND" and "OR" were used to refine the search and ensure the inclusion of highly relevant articles[13].

Inclusion criteria consisted of peer-reviewed articles, clinical trials, systematic reviews, and meta-analyses that addressed caries formation, biofilm composition, enamel demineralization processes, and fluoride's role in dental health. Excluded from the review were articles focusing exclusively on rare diseases, pediatric anomalies unrelated to caries, or publications lacking English translations[14].

Study Population (For Observational Review):

Although the primary focus was literature-based, secondary observational data were integrated from publicly available datasets such as the World Health Organization (WHO) oral health reports and data from national health surveys (e.g., NHANES and KNHANES)[15]. These provided global and national statistics on dental caries prevalence across various demographic groups.

The observational component focused on children aged 6–12, adolescents aged 13–18, and adults aged 19–60 across different socioeconomic and geographic backgrounds. These data helped evaluate how demographic factors influence the distribution and severity of dental caries.

3. Results

The systematic literature review and observational data analysis revealed consistent findings across microbial, dietary, environmental, and social variables, all contributing to the onset and progression of dental caries. These results were categorized into microbial profiles, environmental and behavioral influences, lesion characteristics, and population-level patterns.

Microbial Composition and Biofilm Activity:

Multiple studies confirmed the central role of acidogenic and aciduric bacteria in the development of dental caries. Streptococcus mutans was present in over 80% of advanced carious lesions examined in clinical samples. Lactobacillus species and Streptococcus sobrinus were also found in elevated numbers, particularly in deep dentinal lesions. PCR-based studies revealed a consistent microbial shift from commensal to pathogenic species in individuals with high sugar intake, particularly in low-pH environments[16]. The ecological plaque hypothesis was supported by data demonstrating that acid-tolerant organisms became dominant when biofilm pH repeatedly dropped below 5.5 due to frequent sugar consumption.

Demineralization and Remineralization Patterns:

Advanced imaging and surface hardness studies demonstrated a clear cycle of demineralization following sugar exposure. Enamel specimens subjected to sucrose solutions showed significant mineral loss within 30 minutes. In vivo studies using QLF showed early subsurface lesions forming within days of repeated sugar intake. Remineralization, in contrast, was observed in individuals with sufficient salivary flow, fluoride exposure, and reduced sugar intake. Controlled trials indicated that fluoride application, either via toothpaste (1,000–1,500 ppm) or professional varnishes, resulted in 25–40% reduction in demineralization compared to non-fluoride controls[17].

Lesion Classification and Histopathological Findings:

Studies employing ICDAS scoring found that early lesions were most common in children and adolescents, while more advanced lesions were more prevalent in adults. Histological evaluation confirmed four distinct zones within carious dentin: the necrotic zone filled with bacterial colonies, the demineralized zone with low bacterial content, the translucent zone near the pulp with no bacterial invasion, and the unaffected sound dentin. These zones correlated well with clinical tactile sensation—softness increasing with severity—and were key indicators in conservative treatment planning.

Population-Level Epidemiological Trends:

Global data from WHO and NHANES indicated that dental caries affects nearly 100% of adults worldwide and between 60–90% of school-aged children. Prevalence rates remained highest in countries with low-income populations, minimal fluoride use, and

limited access to dental services. For example, caries prevalence in children from low socioeconomic status (SES) backgrounds was 1.8 times higher than in children from higher SES groups[18]. Furthermore, studies from fluoridated communities reported significantly lower caries incidence compared to non-fluoridated areas, confirming the protective effect of fluoride.

Behavioral and Preventive Factors:

Preventive behaviors such as twice-daily brushing with fluoride toothpaste, regular dental visits, and dietary sugar control were associated with lower caries incidence. In contrast, frequent snacking, soft drink consumption, and poor oral hygiene were linked with higher caries risk. Fluoride exposure—both systemic (via water) and topical (via toothpaste or varnish)—was consistently associated with reduced demineralization and fewer carious lesions.

Behavioral intervention programs targeting children, particularly in schools, demonstrated measurable success. In studies involving school-based fluoride varnish applications and oral health education, the mean DMFT (Decayed, Missing, Filled Teeth) score was reduced by up to 35% within 12 months.

4. Discussion

Dental caries remains one of the most widespread and persistent chronic diseases affecting humans globally. The findings from this review reinforce that dental caries is a multifactorial disease involving a dynamic interaction between microbial communities, dietary habits, environmental exposures, and individual behaviors. Understanding the complexity of this interaction is key to both the treatment and prevention of caries.

Microbial Shifts and the Ecological Plaque Hypothesis:

The data strongly support the ecological plaque hypothesis, which proposes that caries results from an imbalance in the oral microbiome driven by environmental stress, such as frequent sugar intake. Rather than being caused by a single microorganism (Streptococcus mutans), the disease arises when the oral environment selectively favors aciduric and acidogenic bacteria capable of surviving at low pH. This shift in the microbial composition alters the biofilm from a state of symbiosis to dysbiosis, creating conditions conducive to enamel demineralization.

What differentiates the ecological plaque hypothesis from older models is its emphasis on the environmental drivers of microbial changes, rather than the mere presence or absence of specific pathogens. This perspective has significant implications for prevention strategies. It suggests that interventions should not only target bacterial elimination but should also aim to restore oral environmental balance—mainly by reducing sugar intake and promoting behaviors that increase salivary flow and pH buffering.

Dietary Sugars and pH Fluctuations:

One of the most consistent findings across studies was the role of fermentable carbohydrates—particularly sucrose, glucose, and fructose—in promoting acid production. These sugars are rapidly metabolized by cariogenic bacteria into organic acids like lactic acid, which significantly lower the pH within the biofilm. When pH levels fall below the critical threshold (~5.5), enamel demineralization is initiated.

Importantly, the frequency of sugar intake was more strongly associated with caries progression than the total amount of sugar consumed. Frequent snacking and consumption of sugary drinks throughout the day maintain low pH conditions, giving teeth little time to recover via remineralization. This underscores the need for public health campaigns not only to reduce total sugar consumption but also to address eating patterns and snacking habits, particularly in children and adolescents.

Demineralization and Remineralization Dynamics:

The demineralization process, as confirmed by both in vitro and in vivo studies, occurs when acids dissolve the calcium and phosphate from the enamel. However, this process is not irreversible. Saliva, through its buffering capacity and mineral content,

plays a crucial role in neutralizing acids and delivering calcium and phosphate back to the enamel surface.

Fluoride significantly enhances this natural remineralization process by forming fluorapatite, which is more resistant to acid attack than hydroxyapatite. Studies reviewed show that even low levels of fluoride can reduce demineralization and facilitate enamel repair. The preventive effect of fluoride is most effective when delivered consistently through multiple sources such as water fluoridation, toothpaste, mouth rinses, and professional applications.

The implication here is that prevention should focus on enhancing the body's own protective mechanisms. Promoting regular brushing with fluoride toothpaste, encouraging water consumption (especially fluoridated), and ensuring adequate salivary flow through hydration and sugar-free chewing gum can greatly aid in maintaining enamel integrity.

Histopathological Understanding for Clinical Management:

Understanding the histological progression of dental caries helps clinicians tailor interventions based on lesion depth and severity. The presence of four distinct zones—from necrotic to translucent—allows for a precise evaluation of how far the infection has advanced. This, in turn, informs whether a lesion can be remineralized or requires surgical intervention.

ICDAS scoring provides a reliable clinical tool for this purpose, correlating well with microscopic findings. Early lesions (ICDAS 1–2) can often be reversed with non-invasive techniques, such as fluoride varnishes and dietary changes, while more advanced lesions (ICDAS 5–6) may require restoration. Importantly, even severe lesions can be arrested, demonstrating that caries is a dynamic and potentially reversible disease if timely interventions are applied.

Epidemiological and Socioeconomic Considerations: Despite being preventable, dental caries continues to be the most prevalent disease worldwide. Almost all adults and a large majority of children are affected, indicating that current prevention and intervention strategies are not reaching all populations equally. The burden of disease is particularly high in communities with low socioeconomic status, poor oral health literacy, and limited access to dental care.

Social determinants of health—such as income, education, and access to fluoride—emerge as powerful predictors of caries prevalence. Children from low-income families are more likely to have untreated caries, higher DMFT scores, and fewer dental visits. This disparity underscores the need for population-level strategies that go beyond individual behaviors to address systemic barriers to oral healthcare. Community water fluoridation has consistently been shown to reduce dental caries across socioeconomic groups and remains one of the most effective public health measures. School-based programs that provide oral health education, fluoride varnishes, and dental screenings can also help bridge the gap for underserved populations. However, more investment is needed in preventive infrastructure, especially in rural and low-income areas.

Behavioral Factors and Preventive Strategies: Behavioral factors—including oral hygiene habits, dietary control, and dental visit frequency—play a crucial role in either promoting or preventing caries. Studies reviewed in this paper confirm that brushing twice a day with fluoride toothpaste, limiting sugary foods and drinks, and seeking professional dental care are associated with significantly lower caries risk. However, behavior change is challenging and requires sustained education and motivation. Public health interventions must be culturally sensitive, age-appropriate, and continuous. Digital tools such as mobile apps and reminders, community outreach through schools and clinics, and the involvement of parents and caregivers are all strategies shown to improve adherence to preventive behaviors.

Integrating Prevention with Treatment: Another important consideration is the integration of prevention with routine dental treatment. Too often, dental care focuses on surgical interventions (fillings, extractions) without addressing the underlying causes of the disease. Every dental visit should be seen as an opportunity to educate patients about caries prevention and to apply fluoride or sealants where needed. Moreover, early

diagnosis through visual inspection, radiographs, or QLF should be emphasized to detect caries at the non-cavitated stage. Early intervention not only prevents further damage but also reduces treatment costs and improves patient outcomes.

5. Conclusion

Dental caries is a globally prevalent chronic disease with a multifactorial etiology involving microbial dysbiosis, frequent intake of fermentable carbohydrates, insufficient fluoride exposure, and socioeconomic determinants. The transition from a symbiotic oral microbiome to a dysbiotic state—dominated by acidogenic and aciduric bacteria—is primarily driven by frequent sugar consumption, leading to a persistent low pH and enamel demineralization.

According to the ecological plaque hypothesis, caries results from biofilm adaptation to environmental stress rather than infection by specific pathogens. This supports preventive strategies focused on maintaining ecological balance rather than eradication of individual microbes. Evidence confirms that dental caries is a dynamic, reversible process. Non-cavitated lesions (ICDAS 1–2) can undergo remineralization through topical fluoride, improved oral hygiene, and dietary modification. In contrast, cavitated lesions may require restorative care but can still be stabilized under appropriate management protocols.

Fluoride is central to caries prevention. Its widespread use via dentifrices, varnishes, rinses, and fluoridated water effectively inhibits demineralization and promotes remineralization. Ensuring equitable fluoride access remains essential, especially in underserved communities. Public health approaches—including school-based interventions, water fluoridation, and oral health education—are critical in reducing disparities. Moreover, personalized, risk-based dental care involving caries risk assessment, behavioral counseling, and tailored fluoride therapy should be integrated into routine dental practice. In conclusion, dental caries is both a biological and societal concern. Long-term control necessitates addressing its root causes through population-level, preventive, and patient-centered strategies that promote oral and systemic health.

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