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# Dental Caries

## From Prevention to Restoration

*Edited by Efka Zabokova Bilbilova*

# Dental Caries - From Prevention to Restoration

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# IntechOpen Book Series

# Dentistry

## Volume 26

## Aims and Scope of the Series

This book series will offer a comprehensive overview of recent research trends as well as clinical applications within different specialties of dentistry. Topics will include overviews of the health of the oral cavity, from prevention and care to different treatments for the rehabilitation of problems that may affect the organs and/or tissues present. The different areas of dentistry will be explored, with the aim of disseminating knowledge and providing readers with new tools for the comprehensive treatment of their patients with greater safety and with current techniques. Ongoing issues, recent advances, and future diagnostic approaches and therapeutic strategies will also be discussed. This series of books will focus on various aspects of the properties and results obtained by the various treatments available, whether preventive or curative.

# Meet the Series Editor

Dr. Sergio Alexandre Gehrke is a doctorate holder in two fields. The first is a Ph.D. in Cellular and Molecular Biology from the Pontificia Catholic University, Porto Alegre, Brazil, in 2010 and the other is an International Ph.D. in Bioengineering from the Universidad Miguel Hernandez, Elche/Alicante, Spain, obtained in 2020. In 2018, he completed a postdoctoral fellowship in Materials Engineering in the NUCLEMAT of the Pontificia Catholic University, Porto Alegre, Brazil. He is currently the Director of the Postgraduate Program in Implantology of the Bioface/UCAM/PgO (Montevideo, Uruguay), Director of the Cathedra of Biotechnology of the Catholic University of Murcia (Murcia, Spain), an Extraordinary Full Professor of the Catholic University of Murcia (Murcia, Spain) as well as the Director of the private center of research Biotecnos – Technology and Science (Montevideo, Uruguay). Applied biomaterials, cellular and molecular biology, and dental implants are among his research interests. He has published several original papers in renowned journals. In addition, he is also a Collaborating Professor in several Postgraduate programs at different universities all over the world.

# Meet the Volume Editor

Dr. Efka Zabokova Bilbilova is a Specialist in Paediatric and Preventive Dentistry, Associate Professor at the Department of Pediatric and Preventive Dentistry at the Faculty of Dentistry, University "Ss Cyril and Methodius" in Skopje. Dr. Efka Zabokova Bilbilova's MSc thesis was entitled "The role of salivary urea and bicarbonate on the appearance of dental caries.", while her Ph.D. thesis was entitled "Inhibition of demineralization on enamel around the orthodontic braces during the treatment: in vivo and in vitro study." She was a visiting researcher at the Institute of Biomedical Research, Medical Faculty, University of Nis, Serbia (2008 and 2010), where she worked on a project to qualitatively evaluate the remineralization potential of different topical materials on early enamel lesions. She has held several educational lectures and presentations at symposia and conferences in Macedonia and abroad. Her complete professional and scientific experience is reflected in numerous scientific papers published in national and international journals, book chapters, and monographs in the fields of pediatric and preventive dentistry, as well as in saliva buffer systems and demineralization-remineralization in teeth. Associate Prof. Dr. Efka Zabokova Bilbilova is an author of the book, "Dental Caries", May 2021; coauthor on Chapter, "Oral Systemic Linkage" in the book, "Science of Sea Salt", Taylor Specialty Books, 1550 W. Mockingbird Ln. Dallas, Texas, USA, March 2023, and she is a coauthor of the textbook "Pediatric Dentistry", published in 2023. She is currently the head of the Clinic for Pediatric and Preventive Dentistry at the University Dental Clinical Center "St. Panteleimon" in Skopje, Macedonia.

# Contents

# Preface

Dental caries remains one of the most common diseases in dentistry. Modern approaches focus on early detection and minimally invasive treatment. The use of fluoride for prevention and laser technology in therapy offer new possibilities for more effective, comfortable care. This book provides an overview of current knowledge on the role of saliva, fluoride and laser applications in the prevention and treatment of dental caries.

In the chapter "Dental Caries and Fluoride", the authors examine that among the many factors influencing dental health, fluorine holds a unique and indispensable role. It is essential not only for the formation of hard dental tissues but also for the proper development of the skeletal system.

Although fluorapatite accounts for only a small portion of tooth enamel's crystalline structure, its impact is profound. Even in these minute amounts, it strengthens enamel and provides remarkable resistance to acids, a critical feature, as dental tissues are constantly exposed to mechanical, thermal, and chemical challenges. This underscores the importance of adequate fluoride intake during both the pre-eruptive and post-eruptive stages of tooth development for long-term dental health.

Research over the decades has demonstrated that fluoride, when used responsibly and in controlled preparations, is the only mineral element consistently linked to increased resistance of dental hard tissues to caries.

Topical fluoride therapy plays a central role in the prevention of dental caries. In contrast to systemic fluoride, which is incorporated into developing teeth during pre-eruptive stages, topical fluoride acts directly on the tooth surface, enhancing enamel resistance to acid demineralization and promoting remineralization of early lesions. Fluoride preparations, such as gels, varnishes, mouth rinses, and toothpastes, have been extensively studied and proven effective in reducing caries incidence across all age groups. Their controlled application ensures maximum benefit while minimizing potential risks, making topical fluoride a cornerstone of modern preventive dentistry.

This chapter explores the mechanisms, clinical applications, and protocols for topical fluoride use, highlighting its critical role in maintaining long-term oral health.

In the chapter "The Role of Saliva in Preventing Tooth Caries", Hülya Erten Can and Pınar Güvenç provide information that Saliva is a remarkable and complex fluid essential for maintaining oral health. It is a heterogeneous mixture of proteins, glycoproteins, electrolytes, and small organic molecules that traverse the bloodstream. Produced primarily by the salivary glands, this clear, slightly acidic, mucous exocrine secretion performs a multitude of physiological functions. Beyond its biochemical composition, saliva plays a central role in preserving the ecological balance within the oral cavity, safeguarding both hard and soft tissues, and supporting overall oral homeostasis. As it flows from the salivary glands through their ducts, saliva mixes with crevicular fluid, serum, blood cells, microorganisms, and their metabolic products, creating a dynamic environment crucial for oral health and protection.

This chapter highlights the significance of saliva, not only as a biological secretion but also as a cornerstone of preventive and therapeutic strategies in dentistry. Understanding its composition, functions, and interactions is fundamental to advancing both research and clinical practice in oral health.

In the chapter "Development of Strategies Based on the Recent Evidence for the Management of Dental Caries in Children: A Systematic Overview", Shivani Dhonde and Ashwin Jawdekar describe that an evidence-based approach is fundamental to standardizing pediatric dental care and improving outcomes. While international guidelines such as those from the AAPD, EAPD, and SDCEP provide structured recommendations, a unified global approach for children remains lacking. This chapter summarizes and critically appraises current evidence on pediatric caries management and proposes context-specific recommendations. Key strategies include dietary guidance, fluoride use, sealants, non-invasive treatment approaches, and risk- and activity-based treatment planning. Selective caries removal and biological approaches are preferred in primary teeth, with tricalcium silicate materials reducing the need for invasive pulp therapies. Behavior modification and appropriate recall schedules remain essential for long-term success.

In the chapter "Early Childhood Caries: Comprehensive Action for the Quality of Life of Children", the authors discuss that early detection and management are particularly critical in children, as dental caries can begin at a very young age. Early Childhood Caries (ECC) is a prevalent condition. Preventive strategies for ECC include parental education, community-based programs, prevention of bacterial transmission, home and professional preventive approaches, and the reduction of barriers to care. Diagnostic tools such as Caries Risk Assessment, Digital Imaging Fiberoptic Transillumination, and DIAGNOcam facilitate early detection, enabling timely interventions. Effective treatment of ECC restores proper dental form, function, and esthetics while preserving physiological integrity, with operative dentistry combining evolving materials and techniques to achieve long-lasting outcomes.

Finally, "Advanced Laser Applications for Dental Caries Management" provides information that highlights the integration of laser technology into contemporary caries management, emphasizing its diagnostic, preventive, and therapeutic potential. By combining scientific evidence with clinical applications, it aims to provide dental professionals with a comprehensive understanding of how laser therapy can enhance outcomes, promote minimally invasive care, and improve patient experience in the treatment of dental caries.

Laser therapy has emerged as a promising and accessible treatment modality in modern dentistry, increasingly complementing or even competing with conventional approaches. Its role in managing dental caries spans the entire spectrum of care, from early diagnosis and preventive measures to monitoring pulp vitality and performing definitive restorative treatments.

Surgical ablative lasers, such as Er:YAG and Er,Cr:YSGG, are applied in cavity preparation and surface modification, enhancing enamel resistance to caries while simultaneously decontaminating and conditioning the tooth surface for optimal adhesion of restorative materials. Clinical reports indicate high patient acceptance of laser therapy, with reduced need for anesthesia and decreased postoperative discomfort, making it an attractive and patient-friendly alternative to traditional dental treatments.

I acknowledge the authors' desire to share their knowledge with the public. As a Professor of Pediatric and Preventive Dentistry, I am especially pleased that dental students at all levels will read this book and learn about dental caries and preventive measures. As a comprehensive and up-to-date summary of current thinking in the field, this volume is essential reading not only for dental students but also for researchers and dental practitioners.

I hope that the scientific evidence and experience presented in this book will enable dental providers to deliver evidence-based dental care in cariology and preventive dentistry.

Once again, I would like to thank my colleagues for their contributions to this book.

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To my father Nikola and my mother Cveta, for their love, sacrifice, and unwavering support. Thank you!

Chapter 1

# Dental Caries and Fluoride

*Elena Stepco and Tatiana Gonța*


## Abstract

Fluorine (F) is an important chemical element, the presence of which is indispensable for the formation and harmonious development of hard dental tissues, as well as skeletal bones. Although fluorapatite constitutes only a small part of the crystalline components, its presence gives tooth enamel hardness and resistance to the action of acids, which is particularly important for hard dental tissues, which are constantly subjected to mechanical, thermal, and chemical actions. From this point of view, the optimal intake of fluorine in the body is extremely important both in the pre-eruptive and post-eruptive periods. Controlled use of fluoride preparations has shown that fluoride is the only mineral element unanimously correlated with the resistance of dental hard tissues to dental caries.


**Keywords:** caries, fluoride, mechanism of action, fluoride metabolism, fluoridation

## 1. Introduction

Dental caries represents the result of a dynamic imbalance between the demineralization and remineralization processes of the dental enamel, processes that occur continuously in the oral environment [1]. When factors that favor demineralization prevail—such as frequent consumption of fermentable sugars, poor oral hygiene, or decreased salivary flow—the balance shifts toward a net loss of minerals, eventually leading to the development of carious lesions [2]. The progression of dental caries is influenced by a multitude of local and systemic factors, including the composition and volume of saliva [3, 4], the presence of fluoride, the oral microbial flora, frequency of acid exposure, and the individual resistance of dental tissues [5, 6].

Dental caries is among the most common chronic diseases worldwide, affecting individuals of all ages, with particularly high prevalence in children and adolescents. This multifactorial disease significantly impacts the quality of life, influencing eating, sleep, communication, and overall health status [2]. According to the World Health Organization (WHO), nearly 60–90% of school-aged children and almost 100% of adults have carious lesions, highlighting the endemic nature of this condition across most global populations.

One of the most effective strategies for preventing dental caries is to support the natural processes of enamel remineralization and to slow down demineralization. In this context, caries prevention becomes a major priority in both public health and dental medicine. Among the recognized preventive measures, fluoride plays a central role due to its proven efficacy in reducing the incidence of caries [7–10]. The introduction of water fluoridation in the 1940s marked a turning point in caries control, resulting in significant reduction in the prevalence of caries in many geographic regions [11–13].

Fluoride acts through multiple synergistic mechanisms, including the strengthening of enamel structure, inhibition of demineralization, and stimulation of remineralization in early lesions [14–17]. It also exerts an indirect antimicrobial effect on cariogenic microflora, particularly *Streptococcus mutans*, by reducing their acid-producing capacity.

However, the use of fluoride in caries prevention is not without controversy, especially regarding optimal dosing, the risk of dental fluorosis, and potential systemic effects from chronic exposure [18–21]. These considerations justify the need for a balanced analysis based on current scientific data regarding both the benefits and risks associated with fluoride use [19–22].

This chapter aims to provide a comprehensive exploration of fluoride's role in the prevention and control of dental caries, offering a synthesis of its mechanisms of action, administration strategies, and the scientific evidence supporting its efficacy, alongside a critical discussion of the limitations and ethical considerations associated with its public health application.

## 2. Fluoride: General overview

Fluoride (symbol F, atomic number 9) is a chemical element from group XVII of the periodic table (halogens), known as the most electronegative and thus extremely reactive. Under standard conditions, fluoride exists as a pale-yellow diatomic gas with a high degree of toxicity. Due to its reactivity, it is not found in free form in nature, but only as inorganic or organofluoride compounds—fluorides—distributed in soil, water, air, plants, animals, and human tissues [11, 13, 21, 23].

### 2.1 Biological role and importance in odontogenesis

Fluoride is an essential element for the proper formation and development of hard dental tissues and bone. Although fluorapatite represents only a small fraction of the crystalline components of enamel, its presence significantly increases enamel's resistance to acid attack. Thus, fluorapatite crystals are a key factor in ensuring durability in teeth constantly exposed to mechanical, thermal, and chemical challenges [14, 16, 17]. Fluoride incorporation into dental structures occurs at two main stages:

- *Pre-eruptive phase*, during enamel formation and mineralization, through uptake from tissue fluids.
- *Post-eruptive phase*, through topical action from saliva, drinking water, food, or oral hygiene products that can penetrate even superficial enamel fissures.

The systemic influence of fluoride on dental tissues is restricted to a specific time window corresponding to the active stages of enamel and dentin formation. These periods vary depending on the type of tooth but generally range from the 13th week of gestation (for primary incisors) to approximately age 8 (for permanent molars).

The most critical period for systemic fluoride action is *amelogenesis*, the process during which specialized epithelial cells—*ameloblasts*—produce the enamel matrix and regulate its mineralization. Amelogenesis consists of many phases, but two of them are distinct:

- *Secretory phase*: Ameloblasts actively synthesize and deposit the organic enamel matrix, incorporating small amounts of fluoride.

- *Maturation phase*: Ameloblasts undergo structural and functional changes, ensuring an active influx of ions (calcium ion ($Ca^{2+}$), phosphate ion ($PO_4^{3-}$), hydrogen ion ($H^+$). During this stage, fluoride is intensely incorporated into the forming enamel crystals as fluorapatite [14, 16, 17, 21, 24].

Regarding dentinogenesis, odontoblasts can incorporate fluoride into primary dentin crystals, although this is less significant compared to enamel due to dentin's higher permeability and different mineral compositions [14, 21, 25].

Fluoride influences odontogenesis through several biological mechanisms, both direct and indirect, such as:

- Incorporation into the mineral structure.

- Alteration of crystal morphology.

- Regulation of gene expression.

- Modulation of ion transport.

The presence of fluoride favors the formation of larger, more uniform, and more densely organized crystals with stable orientation and reduced porosity. This results in enamel with increased structural integrity.

Molecular studies have shown that fluoride modulates the expression of key genes involved in amelogenesis, including:

- *AMELX*—encodes amelogenin, the main protein of the enamel matrix.

- *ENAM*—encodes enamelin, involved in initiating crystal nucleation.

- *MMP-20 (matrix metalloproteinase-20) and KLK-4 (kallikrein-related peptidase 4)*—proteases responsible for the controlled degradation of the organic matrix.

Through these effects, fluoride contributes to the coordination of mineralization and protein resorption processes during enamel development [16, 17, 24].

Fluoride also modulates ameloblast function by influencing ion channels and membrane pumps (e.g., $Ca^{2+}$-ATPases, bicarbonate, and anion channels), thereby regulating local pH and creating favorable conditions for crystal growth [14].

An adequate fluoride intake during active odontogenesis is essential for development of a caries-resistant dentition. The recommended daily dose for children is 0.05–0.07 mg fluoride/kg body weight/day, with sources including fluoridated drinking water, food, supplements, and unintentional ingestion from toothpaste [26].

However, *excessive* and sustained exposure to fluoride—typically above 0.1 mg/kg/day—can lead to dental fluorosis, a mineralization disorder caused by ameloblast dysfunction during the maturation phase. Clinically, fluorosis is characterized by opaque white spots, horizontal white lines, or, in severe cases, pitting and surface defects. Once the teeth have erupted and the enamel is fully mineralized, systemic fluoride overexposure can no longer cause fluorosis [21, 22, 27–29].

Fluorosis severity is not only directly proportional to the level and duration of fluoride exposure but is also influenced by individual variations in absorption, metabolism, and excretion. The intensity of fluorosis correlates closely with plasma fluoride concentration, which is in equilibrium with tissue fluid surrounding the developing enamel organ. Plasma concentration depends on multiple factors, including total fluoride intake, route of administration (oral or inhaled), renal function, bone metabolism dynamics, and overall metabolic activity [19, 21, 23, 30].

The most commonly observed form of fluorosis today is mild, characterized by subtle white spots that are often difficult to detect. Moderate forms exhibit more prominent spots and occasional brown discolorations, while severe forms involve significant structural defects, surface erosions, increased fragility, and even crown fractures [27–29].

It is important to note that not all children exposed to the same fluoride levels develop fluorosis to the same degree. This reflects interindividual differences related to:

- Total fluoride intake (including processed foods, supplements, or accidental toothpaste ingestion).
- Duration and timing of exposure (especially between 15 and 30 months of age for permanent upper incisors).
- Genetic susceptibility of ameloblasts to fluoride-induced metabolic stress.
- Nutritional status (intake of calcium, phosphorus, vitamin D, etc., can modulate fluoride's effect on enamel).

For instance, some children may exhibit mild fluorosis while consuming water containing only 1 part per million (ppm) fluoride, whereas others may develop moderate or severe forms of fluorosis if chronically exposed to higher levels, such as 4 ppm. Cumulative exposure from multiple sources—including supplements, tablets, or mouth rinses—can also significantly increase risk [23, 29, 30].

*Sources and absorption of fluoride.*

The most common sources of fluoride are:

- *Fluoridated drinking water*—This is the most effective source from a public health perspective.
- *Fluoridated toothpaste*, gels, varnishes, and other dental products.
- *Black tea, oceanic fish, fluoridated salt*, and processed foods made with fluoridated water.
- *Oral fluoride supplements*—These supplements are recommended in areas with non-fluoridated water.

About 80–90% of ingested fluoride is absorbed in the gastrointestinal tract through passive diffusion. Approximately 25% of this absorption occurs in the stomach, with the remaining 65–77% taking place in the small intestine. After absorption, fluoride is transported in the blood, where it circulates partially bound to plasma proteins. Its concentration in plasma is low (0.01–0.06 ppm) and is not homeostatically regulated [21, 23, 30].

Following absorption, roughly 90% of fluoride is distributed to hard tissues (bones and teeth), with about 1% remaining in soft tissues. Fluoride preferentially accumulates in areas of active bone formation and in developing dental enamel. Adults retain about 36% of ingested fluoride, while the retention rate in children can be as high as 50%, which underscores the need for caution in its administration.

The body primarily excretes fluoride through *urine*, accounting for about 50% of the total elimination. Unabsorbed fluoride is passed through feces. Excretion via saliva, sweat, or breast milk is considerably lower. The kidneys are the primary organ for fluoride excretion and for maintaining its balance in the body [21, 23].

## 3. Factors influencing fluoride metabolism

Fluoride metabolism is influenced by various physiological and pathological factors, including:

- acid-base balance
- kidney function
- age
- level of physical activity
- altitude
- diet (especially foods high in calcium, magnesium, aluminum, and iron, which can form insoluble compounds that are difficult to absorb).

In pregnant women, fluoride crosses the placenta based on its plasma concentration. At low levels, transfer is possible, but at concentrations of 0.4 ppm or higher, the blood-placental barrier limits the transfer, thereby protecting the fetus from excessive exposure.

### 3.1 Considerations for systemic *versus* topical use

For over 75 years, public health authorities in many countries have supported water fluoridation as a primary method for preventing dental caries. However, some recent studies have raised questions about the safety of this intervention, particularly for young children who are at a higher risk of excessive fluoride ingestion.

There is strong scientific evidence supporting the effectiveness of *topical fluoride* in preventing tooth decay. In contrast, the benefits of *systemic administration* are less supported in today's context, where topical sources are widely available. The unequal access to oral hygiene products and dental care also introduces an element of inequity into community fluoridation policies. Furthermore, evidence suggests that ingesting fluoride is not essential for caries prevention and may lead to dental fluorosis (a cosmetic condition causing enamel discoloration) or, in some cases, neurodevelopmental issues, especially in children and during the prenatal period.

### 3.2 Fluoride fixation in hard tissues: Teeth and bones

Following gastrointestinal absorption, fluoride circulates in plasma as either a free ion or bound to proteins, and is rapidly distributed to mineralized tissues. Bone tissue, particularly during periods of growth, is the primary storage site, capable of accumulating up to 99% of the body's total fluoride. The fixation of fluoride in bone is a dynamic process influenced by age, bone remodeling rate, and daily intake [23].

Depending on the physiological stage, the balance of fluoride fixation in bone can be classified into three phases:

1. Positive balance: This phase is characteristic of childhood and adolescence, when bone fixation is at its maximum, reaching 50–70% of daily intake. Fluoride is actively incorporated into the mineralizing bone matrix.

2. Steady-state balance: This phase occurs during adulthood when intake and excretion are balanced. The bone maintains a slow remodeling activity, with a gradual replacement of old ions with new ones. In this stage, typically between the ages of 20 and 40 years, bone fluoride concentration can increase from around 800 to 1600 ppm.

3. Negative balance: This phase arises when fluoride intake falls below the necessary level. The body temporarily compensates for this deficit by maintaining constant renal elimination, mobilizing fluoride from its bone reserves. This redistribution can lead to a gradual decrease in bone fluoride concentration and, in severe cases, can affect bone structure.

Thus, bone serves as a crucial metabolic reservoir for fluoride and also as an indicator of chronic exposure, with both beneficial and pathological implications in cases of excessive intake (skeletal fluorosis) [23, 27, 31].

Regarding dentition, fluoride fixation is directly correlated with the developmental stages of dental hard tissues. Fluoride is incorporated primarily into the enamel and dentin, and to a lesser extent, into the cementum. The fixation process follows three distinct physiological stages:

1. *During amelogenesis and dentinogenesis:* In the hard tissue formation phase, fluoride fixation is at its maximum. Fluoride is delivered to the mineralizing dental matrix via the bloodstream and peridental interstitial fluid.

2. *During the pre-eruptive period:* After the crown has completed mineralization, fluoride absorption is limited but still possible through the diffusion of ions from pericoronal fluids. This process leads to a slow accumulation of fluoride in the enamel.

3. *After eruption:* Fluoride fixation into the dental structure becomes exclusively superficial and dependent on topical exposure. Fluoride from saliva, fluoridated drinking water, or oral hygiene products (toothpaste, gels, varnishes) is fixed in the enamel exposed to the oral environment. At this stage, remineralization is local and does not affect the entire volume of enamel.

The fluoride content in enamel varies significantly depending on geographical intake: in areas with a fluoride deficiency, the dental concentration is 800–900 ppm, while in areas with an adequate daily intake (1 mg/day), it can reach up to 1600 ppm. It is worth noting that, unlike enamel, dentin contains roughly four times more fluoride, with the maximum concentration located near the odontoblasts—an area that remains constant as long as the pulp is vital [21].

Remarkably, enamel cannot accumulate fluoride through active local mechanisms after it has fully mineralized. Post-eruptive fixation is strictly limited to interactions with the external environment, and deep structural remodeling is no longer possible. Therefore, the duration of exposure is a critical factor in the efficiency of dental fluoride fixation.

As a result, the controlled use of fluoride—both systemic and topical—is universally recognized as an essential protective factor against dental caries. It works by strengthening the mineralization of the dental structure and increasing its resistance to bacterial acids [7–10, 14].

## 4. Mechanisms of action of fluoride in caries prevention

The prophylaxis of dental caries through fluoride use is based on three essential topical mechanisms:

1. inhibition of enamel demineralization,

2. stimulation of remineralization processes, and

3. antibacterial action on the dental biofilm (dental plaque).

Accordingly, fluoride administered *via* fluoridated drinking water, toothpaste, mouthwash, or fluoride varnishes acts predominantly through these mechanisms. Systemic administration (tablets, drops, lozenges) has relatively lower effectiveness in caries prevention [12, 17, 22].

### 4.1 Inhibition of enamel demineralization

Enamel demineralization is initiated by a drop in oral pH, caused by acids produced by cariogenic bacteria. Under such acidic conditions, hydroxyapatite—the main mineral constituent of enamel—becomes soluble. Fluoride does not significantly alter enamel solubility during tooth development, as systemically formed fluorapatite is present only in low concentrations. However, topically applied fluoride, present in the dental biofilm, can protect hydroxyapatite crystals from dissolution [7, 8, 14–16, 32].

When bacterial acid is produced, the fluoride present in the plaque (as a result of regular topical exposure) diffuses with the acid toward the enamel surface, where it becomes incorporated into the crystal structure of hydroxyapatite, forming fluorapatite. This inhibits further demineralization.

Fluorapatite ($Ca_5(PO_4)_3F$), formed by substituting the $OH^-$ group in hydroxyapatite ($Ca_5(PO_4)_3OH$) with $F^-$, is significantly more resistant to acid dissolution than calcium hydroxyapatite.

### 4.2 Stimulation of remineralization

During the natural remineralization process, bacterial acids are neutralized by the buffering action of saliva, allowing the re-precipitation of minerals (calcium and phosphate ions) into the enamel structure. These minerals are present in saliva in a supersaturated state, which promotes the regeneration of apatite crystals.

Fluoride accelerates the growth of fluorapatite crystals in submicroscopic, partially demineralized enamel regions. Adsorption of fluoride onto these crystal surfaces attracts calcium and phosphate ions from saliva, rebuilding the mineral structure with enhanced acid resistance. The result is a more stable mineral surface with lower solubility in the acidic environment generated by dental plaque [8, 33].

### 4.3 Inhibition of bacterial activity

Fluoride also has antimicrobial properties, especially under low pH conditions. In such environments, fluoride ions ($F^-$) penetrate bacterial cell walls in the form of hydrofluoric acid (HF). Once inside the cell, HF dissociates into $F^-$ and $H^+$, acidifying the bacterial cytoplasm and inhibiting key metabolic enzymes such as enolase, thereby disrupting glycolysis and reducing acid production. Additionally, fluoride:

- inhibits phosphoenolpyruvate phosphotransferase, an enzyme involved in carbohydrate fermentation, reducing lactic acid production;

- interferes with glucose transport into bacterial cells and inhibits the synthesis of extracellular polysaccharides that form the plaque matrix, reducing *Streptococcus mutans* adhesion to enamel surface; and

- disrupts microbial adherence to tooth surfaces by interfering with salivary proteins and glycoproteins, hindering plaque formation.

The antimicrobial effect of fluoride is cumulative, increasing over time with repeated exposures to fluoride.

Importantly, fluoride ions present in saliva, the gingival sulcus, and topical products are retained in the dental biofilm, where they form a localized, slow-release reservoir. This fluoridated microenvironment provides continuous protection at the enamel surface and facilitates remineralization of early carious lesions [7–9].

### 4.4 Fluoride and post-eruptive enamel maturation

After eruption, enamel is not fully mineralized and remains vulnerable to acid attack. Continuous fluoride exposure during this critical *post-eruptive phase* contributes to enamel maturation by strengthening the crystal structure and increasing resistance to demineralization. Topical fluoride can be incorporated into the superficial enamel layer as fluorapatite, offering substantial protection against future acid challenges [14, 15, 17, 24].

Fluoride is most effective when present frequently in low concentrations in the oral environment. Numerous studies have shown that constant exposure to fluoride has a more pronounced dental caries preventive effect than infrequent high-dose applications. This mechanism relies on maintaining a constant concentration of fluoride in both saliva and the dental biofilm, which contributes to a dynamic balance between demineralization and remineralization processes [1, 16].

Fluoride effectiveness is influenced by several local factors, including salivary composition (buffering capacity, calcium and phosphate content), oral pH, and diet. For example, in cases of hyposalivation or persistent oral acidity, fluoride's protective effect is diminished. Moreover, in the absence of sufficient calcium and phosphate ions, fluoride-induced remineralization is limited. Therefore, modern fluoride-containing formulations often include calcium-phosphate complexes—such as amorphous calcium phosphate (ACP) or tricalcium phosphate (TCP)—to enhance remineralization [14, 16].

The fixation of fluoride in both bone and teeth occurs through similar mechanisms involving ionic exchange in hydroxyapatite and carbonated hydroxyapatite crystals, substituting hydroxide ion ($OH^-$) and carbonate ion ($CO_3^{2-}$) groups with $F^-$, forming acid-resistant fluorapatite. In enamel, this transformation is limited: even with high fluoride doses, conversion does not exceed 10% of enamel mass or about 50% of its $OH^-$ sites. Fluoride accumulates mainly in the superficial layers, reaching concentrations of up to 1000 ppm at a depth of 30 μm [21].

Fluoride ions, along with other ions and water molecules, may bind to the surface of apatite crystals. At higher local concentrations, this leads to the formation of calcium fluoride ($CaF_2$), a temporary fluoride reservoir according to the reaction:

$$Ca_{10}(PO_4),(OH)_2 + 20F^- \rightarrow 10CaF_2 + 6PO_4^{3-} + 2OH^-$$

Calcium fluoride is unstable and gradually dissolves in contact with saliva, releasing bioavailable fluoride.

In addition to these primary mechanisms, fluoride also has several indirect effects, particularly during the formation of dental hard structures:

- It increases the growth rate of crystals, making them larger, more regular, and less soluble.

- It reduces occlusal relief and tooth dimensions, aspects that contribute favorably to a lower incidence of cavities.

- It normalizes protein and mineral metabolism when administered enterally.

- It has an optimal cariostatic effect at daily doses of 2 mg, beyond which the benefit does not increase proportionally.

Ultimately, fluoride fixation in hard tissues depends on two main factors: the *concentration of fluoride ingested* and *age*. Thus, while bone tissue is a permanent reservoir of fluoride ions that constantly remodels its content, the tooth benefits from a limited period of assimilation [21, 23].

## 5. Side effects of fluoride: Toxicological and clinical aspects

Under certain conditions of excessive or prolonged exposure, fluoride may induce a range of adverse effects with implications for general health. These side effects vary from structural changes in dental enamel to alterations in bone tissue, thyroid function, and mineral metabolism. A critical evaluation of these effects is essential for establishing safety thresholds and guiding fluoroprophylaxis strategies.

The most common adverse effect of fluoride is dental fluorosis, which occurs exclusively during the development of permanent teeth, prior to their eruption into the oral cavity (generally up to age 8). It is caused by excessive accumulation of fluoride in the enamel matrix, which disrupts the normal processes of mineralization and maturation [21, 27–30, 34].

At the opposite end of the age spectrum lies skeletal fluorosis, a chronic condition resulting from the slow but progressive accumulation of fluoride in bone tissue. This disorder is observed in endemic regions where daily fluoride intake exceeds 10 mg over several years, such as in parts of China, India, and East Africa. Bones affected by chronic fluorosis exhibit increased density (osteosclerosis) but lose elasticity and resilience, resulting in joint pain, stiffness, and, in advanced stages, skeletal deformities and ankylosis [19, 21, 27].

The underlying pathology involves abnormal bone remodeling, in which fluoride replaces hydroxyl groups in hydroxyapatite crystals to form fluorapatite. Although fluorapatite is less soluble, it differs biomechanically from hydroxyapatite, leading to altered physiological properties, especially in response to mechanical stress.

The impact of fluoride on neurological development remains a subject of ongoing research and debate. Some epidemiological studies conducted in China, Iran, and other countries have suggested an association between chronic exposure to high fluoride concentrations in drinking water (>2 ppm) and a slight decrease in intelligence quotient (IQ) scores among children. A meta-analysis published by Choi et al. (Harvard School of Public Health, 2012) reviewed 27 studies and found a mean difference of 0.45 standard deviations in IQ scores favoring children from low-fluoride areas [13]. However, the authors highlighted the limitations of the included studies, including the lack of control for other environmental factors, methodological variability in cognitive testing, and absence of individual-level data.

Fluoride has the potential to influence thyroid function by competing with iodine and inhibiting the activity of the enzyme 5′-deiodinase, which is responsible for converting thyroxine (T4) into triiodothyronine (T3). Some studies suggest that chronic high-dose fluoride exposure (>4 mg/day) may lead to increased serum thyroid-stimulating hormone (TSH) levels and decreased triiodothyronine/thyroxine (T3/T4) concentrations, particularly in individuals with iodine deficiency. Although current evidence is insufficient to establish a clear causal relationship, these findings underscore the need to monitor endocrine function in regions with naturally high fluoride levels in water [18–20, 35].

Acute fluoride intoxication is rare but can have serious or even fatal consequences. The estimated lethal dose for adults is 5–10 g of sodium fluoride (NaF) (equivalent to 32–64 mg fluoride/kg body weight), while in children, symptoms may appear with an intake exceeding 5 mg/kg. The pathophysiological mechanisms of acute toxicity are multifactorial and involve four primary actions:

- *Local corrosive effect* – Fluoride reacts with moisture in mucosal tissues to form hydrofluoric acid, which is highly caustic and causes gastrointestinal tissue burns.

- *Neurological dysfunction* – Fluoride has a strong affinity for calcium, an essential ion for neural impulse transmission; its binding disrupts normal neuronal activity.

- *Enzyme inhibition* – Fluoride acts as a metabolic toxin by forming complexes with metal ions (e.g., magnesium ion ($Mg^{2+}$), calcium ion ($Ca^{2+}$)) critical for enzymatic activity, thereby impairing cellular function.

- *Cardiac compromise* – Fluoride-induced electrolyte imbalances, especially hypocalcemia and hyperkalemia, can lead to arrhythmias and circulatory collapse.

Clinical manifestations include nausea, vomiting, abdominal pain, hypocalcemia, seizures, cardiac arrhythmias, and, in severe cases, cardiopulmonary arrest. Emergency treatment involves prompt administration of calcium salts (e.g., calcium gluconate), correction of electrolyte disturbances, and intensive supportive care.

Fluoride is eliminated primarily *via* the renal route (approximately 90%). In patients with chronic kidney disease, fluoride clearance is reduced, leading to progressive accumulation and potential bone and systemic toxicity. Additionally, fluoride may disrupt calcium and phosphate metabolism, causing hypocalcemia and hypomagnesemia in both acute and chronic exposure scenarios.

Fluoride has also been investigated in relation to pineal gland function, melatonin secretion, male fertility, and DNA integrity. However, the available evidence remains limited and inconclusive, with most data originating from experimental or observational studies of low epidemiological validity. Further research is required to clarify these potential associations [18, 23, 35].

## 6. Methods of endogenous prevention of dental caries

Endogenous prophylaxis, also referred to as systemic or general prevention, encompasses the internal, physiological, and metabolic means by which the body can prevent or reduce the incidence of dental caries. This approach is based on the adequate intake of substances essential for the mineralization of dental tissues during their development and maturation, as well as on maintaining a general metabolic state conducive to oral health [21, 23].

The essential substances involved in endogenous prophylaxis include:

- fluoride *(primarily from drinking water and supplements)*
- calcium
- phosphorus
- vitamin D
- other trace elements (magnesium, zinc, iodine).

One of the most important components of endogenous prophylaxis is the presence of systemic fluoride, obtained from sources, such as fluoridated drinking water, food, nutritional supplements, or controlled pharmaceutical administration. Ingested fluoride reaches the developing dental germs via systemic circulation, positively influencing the mineral structure of enamel from the earliest developmental stages.

*Water fluoridation* remains the most effective and cost-efficient method of systemic caries prevention and is endorsed by the World Health Organization (WHO). The recommended optimal level is 0.5–1 ppm (mg/L), adjusted based on local climatic conditions (lower requirements in warmer climates due to higher water intake). Soluble fluorides (of sodium, calcium, magnesium, or silicon) are added at water treatment plants to reach target concentrations of:

- 0.7–0.8 mg/L in warm climates
- 1.0 mg/L in temperate climates
- 1.2 mg/L in cold climates [11, 12, 19].

Epidemiological studies conducted in various regions of the world have shown that the introduction of water fluoridation has led to a 40–60% reduction in caries incidence in children compared to populations in non-fluoridated areas [9–11].

Conditions for the application of this method include: a DMF (Decayed, Missing, Filled) index >3 in 12-year-old children, fluoride levels below 0.5 ppm in drinking water, and the absence of other systemic fluoride sources. It is recommended to begin use from birth to positively influence enamel structure during odontogenesis. The reduction in caries incidence ranges from 25 to 80%, with maximum effect on the smooth surfaces of molars and less impact on pits and fissures [9, 10, 12].

In regions where drinking water contains low-fluoride levels (<0.3 ppm), *controlled supplementation* in the form of tablets, drops, or chewable lozenges is indicated—especially for children living in areas without natural or artificial fluoridation.

The recommended daily fluoride doses, based on age, are:

- 0–6 months: 0.01 mg/day
- 7–12 months: 0.5 mg/day
- 1–3 years: 0.7 mg/day
- over 4 years: 1.0 mg/day.

Daily administration is recommended after breakfast for 200–250 days per year. The main advantage is precise dosing tailored to age and metabolic needs. Concomitant use of calcium supplements is contraindicated, and simultaneous use of two systemic fluoridation methods (e.g., water and tablets) is not advised. The primary contraindication is a high natural fluoride content in drinking water (>50% of the optimal dose) [23, 26, 30].

In addition to water and supplements, other dietary or pharmacological sources can contribute to endogenous prophylaxis, including:

- fluoridated dairy products;
- fluoridated salt (available in some countries as an alternative to water fluoridation);
- certain vegetables and fruits, depending on soil content; and
- black or green tea, which may naturally contain moderate amounts of fluoride.

An alternative method, particularly suited for communities where water fluoridation is not feasible, is milk fluoridation—especially effective in children's diets. Fluoride is added as sodium fluoride or sodium monofluorophosphate ($Na_2PO_3F$) at a dose of 0.5 mg per glass. This method is recommended for children aged 3–12 years, administered over 250 days per year. It has been implemented in Bulgaria, the Czech Republic, Hungary, Russia, and other countries, demonstrating proven effectiveness for both primary and permanent dentitions [11, 12].

*Fluoridated table salt* is another accessible and cost-effective method, used in Switzerland, Hungary, and Colombia. Its fluoride content is typically 250 mg/kg of salt. The main disadvantage is the inability to personalize the dosage and the wide variability in salt consumption. Nevertheless, studies indicate an average 40–50% reduction in dental caries incidence [11, 12].

*Saliva* plays a fundamental role in maintaining oral homeostasis and in the prevention of dental caries. Its protective actions include:

- buffering the acidity of the oral cavity;
- remineralizing areas of initial enamel demineralization;
- transporting essential ions (calcium, phosphate, fluoride); and
- mechanically cleansing the tooth surfaces.

An adequate salivary flow is crucial to maintaining a protective oral microenvironment [1, 3, 16].

*Diet al*so significantly and indirectly influences enamel resistance. Vitamins A, C, and D, along with sufficient intake of protein, calcium, and phosphorus, are essential during odontogenesis. In particular, vitamin D enhances intestinal absorption of calcium and phosphorus and is essential for efficient mineralization of hard tissues. Vitamin D deficiency during odontogenesis may lead to enamel hypoplasia, a condition that predisposes to caries. Similarly, an insufficient intake of calcium and phosphorus, especially in childhood, results in poor mineralization and increased caries risk [21, 23].

Endogenous prophylactic methods, when implemented early and adapted to the local context (fluoride levels in water, supplement availability, dietary habits), offer effective and long-term protection against dental caries. National oral health strategies should prioritize these methods, particularly for pediatric populations [11, 12].

## 7. Methods of topical fluoridation

Topical fluoridation represents a central pillar in the prevention of demineralizing diseases of hard dental tissues, playing a predominant role in the post-eruptive period by maintaining a balance between demineralization and remineralization processes at the enamel surface. These methods involve the direct application of fluoride to tooth surfaces, increasing local fluoride concentration and forming deposits of calcium fluoride ($CaF_2$), which act as a biological reservoir. Topical fluoridation is effective, regardless of age [9, 10, 14–16, 36].

This approach is widely used in both individual and community-based prevention, especially among children and adolescents. Numerous clinical studies support its efficacy, showing a 30–40% reduction in caries incidence among individuals who use fluoride products as recommended [9, 10, 14].

Compared to systemic fluoridation, topical administration offers several advantages:

- *Enhanced safety*, especially in children, due to the minimal risk of dental fluorosis when properly applied.
- *Targeted administration* and precise dosage based on individual needs.
- *Increased effectiveness* in post-eruptive caries prevention through direct action on enamel.
- *Low cost and wide accessibility*, facilitating large-scale implementation.

However, topical fluoridation also has certain limitations:

- It requires *patient cooperation*, particularly in maintaining regular brushing and consistent use of fluoridated products.
- Its *efficacy is significantly reduced* in the context of poor oral hygiene.
- It *cannot replace systemic fluoridation* during the formative period of tooth development and mineralization, being more effective after tooth eruption.

### 7.1 Fluoridated agents used in topical application

Over time, numerous fluoride-based substances have been used locally with remarkable results in caries prevention. The most commonly used include:

- Sodium fluoride (NaF)
- Stannous fluoride ($SnF_2$)
- Amine fluorides (AmFs)
- Acidulated phosphate fluoride (APF)
- Sodium monofluorophosphate ($Na_2PO_3F$)
- Stannous hexafluorozirconate ($SnFrZ_6$)
- Fluorosilanes.

*Sodium fluoride (NaF)* is frequently used as a 2% aqueous solution. For effective caries protection, four series of applications are recommended, coordinated with the eruption of primary and permanent dentitions at ages of 3, 7, 10, and 13 years.

Advantages:

- Stable in plastic containers
- Tasteless
- Does not stain teeth or irritate soft tissues.

*Stannous fluoride ($SnF_2$)* is applied as an 8–10% aqueous solution every 6 months, starting at age 3. The frequency may be increased in cases of high caries activity or elevated caries risk [9, 10, 14].

Disadvantages:

- Unstable in solution—must be freshly prepared before use.
- Unpleasant taste, often poorly tolerated by children.
- Stains hypomineralized and demineralized enamel brown, as well as silicate or glass ionomer restorations.
- May cause gingival irritation.

*Acidulated phosphate fluoride (APF)* is currently one of the most widely used topical fluoride agents. It is available as an aqueous solution, gel, or thixotropic gel. Its composition typically includes:

- 2% sodium fluoride
- 3% hydrofluoric acid (HF)
- 1 M orthophosphoric acid ($H_3PO_4$).

Application is recommended every 6 months or more frequently based on clinical need.

Advantages:

- Good stability in plastic containers.
- More pleasant taste than stannous fluoride, improving patient compliance.
- Does not stain enamel or irritate oral mucosa.
- High prophylactic efficacy.

Orthophosphoric acid facilitates fluoride ion penetration into enamel and prevents the formation of insoluble calcium fluoride. Although some free fluoride is lost, the remainder contributes to the formation of fluorapatite and fluorohydroxyapatite.

*Precaution:* In patients with ceramic restorations, protection is required prior to APF application, as hydrofluoric acid may damage such materials [14, 17].

*Amine fluorides* reduce enamel solubility and inhibit bacterial plaque formation. Studies show that bacterial glycolysis is inhibited by 30% at 0.3 ppm and up to 90% at 3.2 ppm. The amine component has bactericidal effects by disrupting cell membranes, inhibiting aerobic and anaerobic glycolysis, and blocking intracellular polysaccharide synthesis. The surfactant properties of the amine increase fluoride contact time with enamel and plaque by up to fourfold, enhancing the anticaries effect [6, 14, 37].

Topical fluoride agents with high prophylactic efficacy include acidulated phosphate fluoride and amine fluorides, applied in various forms. These agents also contribute to remineralizing decalcified enamel areas and halting demineralization in early carious lesions [7, 8, 16].

Integrating topical fluoridation into daily oral hygiene, along with a balanced diet and proper brushing, is among the most effective strategies for dental caries prevention—especially for children and adolescents.

Topical fluoride products are available in the following forms:

- Solutions (e.g., NaF, $SnF_2$, APF).
- Gels or thixotropic gels (e.g., APF, amine fluorides).
- Fluoride varnishes (e.g., Duraphat®).
- Prophylactic pastes and fluoridated toothpaste.
- Mouth rinses containing 0.05–0.2% fluoride.
- Fluoride tablets and drops, sometimes used for both topical and systemic delivery.

These products may contain one or more fluoridated agents, and the same agent can be included in multiple formulations.

Methods of topical fluoridation

i. At-home topical fluoridation

- Fluoridated toothpaste
- Fluoridated mouthwash
- Fluoride gels

ii. Professional topical fluoridation

- Fluoride gels (e.g., 2% NaF, 1.23% APF)
- Fluoride foams
- Fluoride varnishes (e.g., Duraphat®—5% NaF)
- Concentrated solutions (e.g., stannous fluoride—$SnF_2$).

## 8. Topical application of fluoride preparations for the prevention of dental caries

The topical application of fluoride preparations is based on the principle of direct delivery of fluoride ions to tooth surfaces exposed to the oral environment. This preventive strategy exerts a dual effect: on one hand, it promotes the remineralization of early carious lesions, and on the other, it forms a superficial fluoride reservoir that releases fluoride ions during episodes of pH drop in the dental biofilm, thereby preventing further demineralization [14–17, 21, 24].

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Topical fluoride can be applied in various forms, tailored to the patient's age, caries risk level, and degree of cooperation. The main methods include both professional and individual use of varnishes, gels, mouth rinses, toothpaste, and fluoride-releasing dental materials [9, 10, 26].

*Fluoride varnishes* offer prolonged fluoride release and strong adherence to enamel surfaces, with the added benefit of significantly reducing the risk of accidental ingestion, especially in young children. The most commonly used formulations contain 5% sodium fluoride (equivalent to 22,600 ppm fluoride). Their efficacy has been particularly demonstrated in the prevention of occlusal caries in children and root caries in the elderly.

Application is typically carried out professionally, 2 to 4 times per year, by a dental practitioner. The procedure involves thorough drying of the teeth followed by application of the varnish using a brush or applicator on clean tooth surfaces. Fluoride varnishes are especially indicated in the following situations:

- in children and adolescents with high caries risk;
- in patients with dentin hypersensitivity;
- in the context of orthodontic treatment or poor oral hygiene; and
- in individuals with systemic conditions associated with xerostomia.

*Topical fluoride gels* (1.23% sodium fluoride or 0.4% stannous fluoride) and *fluoride foams* are applied professionally in the dental office or prescribed for home use. They are recommended as a preventive measure in patients with dentin hypersensitivity, multiple active carious lesions, or those undergoing orthodontic treatment. Fluoride gels exert remineralizing and prophylactic effects, reducing enamel solubility. They are applied using custom-fitted soft acrylic trays or a spatula and are kept in the oral cavity for 1 to 4 minutes, once or twice weekly.

Recommended application technique:

1. Clean the teeth using a nonabrasive toothpaste.
2. Line the trays with absorbent paper.
3. Apply 2–2.5 mL of gel per tray.
4. Insert the trays and maintain saliva suction for 5–15 minutes.
5. No eating or drinking for 30 minutes after the procedure.

Fluoride foams, due to their aerated consistency, are more readily accepted by children and may contain the same fluoride concentrations as gels, but require smaller volumes per application—thereby reducing the risk of ingestion [9, 14].

*Fluoride mouth rinses* typically contain fluoride in the form of sodium fluoride, silicofluoric acid, or amine fluoride. These rinses represent an accessible method of prophylaxis, with documented efficacy in reducing caries by up to 26–30%. The anticaries effect persists for 2–3 years after use. They may be used daily or weekly, depending on fluoride concentration and individual caries risk.

Common fluoride concentrations in mouth rinses:

- Sodium fluoride (NaF) 0.05% (200 ppm)—for daily use.
- Sodium fluoride (NaF) 0.2% (900 ppm)—for weekly use.
- Stannous fluoride ($SnF_2$) 0.1%—for daily use.
- Amine fluoride (AmF) 0.4% daily or 0.2% weekly use.

Mouth rinses are especially recommended for:

- children over 6 years of age;
- adolescents;
- individuals with orthodontic appliances;
- patients with xerostomia syndromes; and
- persons with frequent exposure to fermentable carbohydrates.

They are not recommended for children under 5 years due to the risk of accidental ingestion.

*Professional toothpaste* is the primary source of fluoride used worldwide. Typically employed during routine professional dental cleanings, it contains high fluoride concentrations and contributes to the reformation of the surface fluoride layer. Its effectiveness is directly proportional to the fluoride concentration (measured in ppm—parts per million) and the brushing frequency.

According to international guidelines:

- Children aged 6 months to 3 years: use toothpaste containing 1000 ppm fluoride, in an amount the size of a grain of rice.
- Children aged 3 to 6 years: use 1000–1450 ppm fluoride, in an amount the size of a pea.
- Adolescents and adults: use 1450–5000 ppm fluoride, depending on the severity of carious lesions.

## 8.1 Professional fluoridation in the dental office

Professional fluoride application is recommended at regular intervals (2–4 times per year) depending on the patient's caries risk level. This category includes:

- *high-concentration gels*: (1.23% sodium fluoride—12,300 ppm; 0.4% stannous fluoride—1000 ppm);
- *prolonged-release varnishes*, capable of slow fluoride release, enhancing localized protection—especially around dental restorations; and

- *solutions applied via brushing* directly onto the teeth—an effective method particularly for patients with increased exposure to risk factors (e.g., orthodontic appliances, multiple restorations, poor oral hygiene).

These procedures are painless, quick, and do not require anesthesia, making them well tolerated even by young children and patients with special needs [9, 26].

### 8.2 Other methods of topical fluoride application

*Fluoride-releasing discs* represent a modern and controlled form of topical fluoride delivery, designed to ensure a slow and sustained release of active fluoride ions at the surface of the tooth. These are small, typically round or oval devices containing sodium fluoride or other fluoride compounds embedded in a biodegradable or bioinert polymer matrix.

The mechanism of action relies on direct adhesion to dental enamel, especially in high-risk areas for caries development—such as the buccal surfaces of molars or proximal surfaces that are difficult to access through brushing. Once applied, the discs firmly adhere to enamel without requiring cementation and begin to release fluoride ions into the saliva and directly onto the enamel surface [14, 17, 24].

The controlled release occurs over a period ranging from 1 to 6 months, depending on the composition of the material and local intraoral conditions. The released fluoride contributes to the remineralization of early lesions and serves as a localized reservoir to prevent demineralization during periods of acidic challenge. Additionally, it provides indirect antibacterial effects by inhibiting the acidogenic metabolism of cariogenic bacteria [5, 6, 37].

Fluoride discs are generally well tolerated, odorless, and tasteless, and they do not interfere with speech or mastication, making them especially suitable for children. They are applied professionally and are indicated for patients with high caries risk, particularly those with poor oral hygiene, orthodontic appliances, or disabilities that prevent regular toothbrushing [9, 10, 26].

Clinical studies have shown that fluoride discs can significantly reduce the incidence of new carious lesions and halt the progression of incipient decay, especially when used in conjunction with other fluoridation methods (e.g., toothpaste, gels, or varnishes). Therefore, they represent a valuable adjunct in modern preventive dentistry.

### 8.3 Fluoridation via superficial demineralization

This method is based on the use of acidic agents to induce controlled demineralization of the enamel's superficial layer, thereby opening the crystalline structure and enhancing fluoride penetration into the subsurface enamel [7, 14, 16, 17].

The rationale is that partially demineralized enamel becomes more receptive to the formation of stable fluorinated mineral compounds, particularly fluorapatite, which is significantly more resistant to acid attack than hydroxyapatite. This technique is typically performed once every 6 months, considered a high-intensity topical fluoridation method, and is indicated for patients with high caries risk or incipient enamel lesions [9, 10].

A specific example is EPOXIT 9070, a product in which fluoride is present as disodium fluorophosphate ($Na_2PO_3F$), incorporated into a polyurethane matrix. This formulation allows for easy application without complicated procedures and ensures a sustained release of fluoride into the enamel, with long-lasting retention. Application is carried out professionally *via* brush-on techniques or swabs, and a major advantage is that repeated treatments are generally not necessary.

**8.4 Fluoride-releasing dental materials**

In modern dental practice, the use of fluoride-releasing restorative materials serves as an additional and effective strategy for preventing secondary caries, particularly around restoration margins or prosthetic work. These materials continuously release fluoride ions, creating a local microenvironment with antimicrobial and remineralizing potential, thereby limiting the occurrence of recurrent caries near restorations.

Commonly used fluoride-releasing materials include:

- Glass ionomer cements (GICs).
- Compomers.
- Certain fluoride-modified composite resins.

GICs are particularly noted for their ability to release fluoride over time and to recharge from external sources (e.g., fluoride toothpaste, gels, or varnishes), making them active fluoride reservoirs. This makes them ideal in pediatric dentistry, in patients with poor oral hygiene, or for the management of cervical lesions.

The advantage of fluoride-releasing materials lies in their dual role: restorative (replacing lost tooth structure) and preventive (providing long-term fluoride release). They are especially favored in areas where hygiene is difficult to maintain (e.g., interproximal zones, gingival margins), as they inhibit bacterial colonization and protect adjacent enamel [6, 14, 16].

**8.5 Sealants for pits and fissures**

Sealants are a highly effective preventive method for occlusal caries, widely used in pediatric and orthodontic dentistry. This technique involves the application of a low-viscosity resin into the deep pits and fissures on the occlusal surfaces of molars and premolars—areas that are hard to clean and prone to plaque retention due to anatomical morphology.

Some sealant materials are fluoridated, offering the additional benefit of fluoride release, which helps inhibit bacterial colonization. The application is performed in the dental office and typically involves:

1. *cleaning* the tooth surface
2. *isolating* with cotton rolls
3. *etching* with phosphoric acid
4. *rinsing and drying*
5. *applying* the sealant and *curing* it.

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Sealants are especially recommended for children and adolescents immediately after eruption of permanent molars, to prevent caries in vulnerable areas. The lifespan of a sealant ranges from 1 to 3 years, depending on the material, application technique, and oral hygiene habits. Regular checkups are essential to evaluate sealant integrity and reapply as needed [9, 10, 17].

By combining a physical barrier effect with fluoride's chemical protection, sealants represent a cost-effective preventive strategy that significantly reduces caries incidence during childhood and adolescence [6, 24].

It is important to note that no single topical fluoridation method provides complete protection against dental caries. The maximum effectiveness of fluoride is achieved when used in combination with other preventive approaches and a rigorous oral hygiene regimen. The choice of method depends on patient age, caries risk, compliance, and access to dental care. Regular applications under professional supervision significantly increase the level of protection against tooth decay [9, 14, 16].

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Chapter 2

# The Role of Saliva in Preventing Tooth Caries

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## Abstract

Dental caries is the most common chronic, multifactorial, and infectious disease worldwide, which progresses under the influence of factors such as plaque microorganisms, tooth structure, saliva, dietary habits, and time, and is caused by the interaction of these factors with each other. Saliva is a complex biological fluid composed of secretions from the major salivary glands—the parotid, submandibular, and sublingual glands—as well as contributions from minor salivary glands and gingival crevicular fluid. It plays a critical role in maintaining oral health by protecting against dental caries, aiding in chewing and swallowing, preserving the integrity of the oral mucosa, and supporting taste perception and wound healing. The solid 1% of saliva, which is 99% water, contains various organic and inorganic components, shed epithelial cells, blood cells such as leukocytes and lymphocytes, microorganisms and their products, and food residues. Theoretically, saliva affects the incidence of dental caries in four ways. It provides less accumulation of plaque because it mechanically cleans the teeth. With the calcium, phosphate, and fluoride it contains, it reduces the solubility of enamel and helps remineralization. It provides neutralization by buffering acids produced by cariogenic bacteria or taken directly from the diet. It has antibacterial activity. In conclusion, although saliva is not among the primary factors that directly affect the formation of dental caries, it prevents the formation of caries thanks to the organic and inorganic compounds it contains and helps the remineralization of initial caries.

**Keywords:** saliva, dental caries, antioxidant capacity, bacterial plaque, biomarkers

## 1. Introduction

### 1.1 Structure and functions of saliva

Saliva can be defined as a heterogeneous fluid composed of proteins, glycoproteins, electrolytes, and small organic molecules (compounds that pass through the blood). It is a complex, clear, slightly acidic, mucoserous, exocrine secretion filling the oral cavity. Saliva, which is largely composed of secretions from the salivary glands, has been found to contain different substances, including water, electrolytes, glucose, and proteins, the most important of which are amylase, lipase, mucin, lysozyme, and secretory IgA. It plays a major role in maintaining the ecological balance in the oral cavity, maintaining oral health, and protecting hard and soft tissues.

Saliva secreted by the salivary glands passes through the ducts, opens into the oral cavity, and mixes with crevicular fluid, serum, blood cells, microorganisms and their products, oral epithelial cells and their products, food debris, and upper respiratory tract secretions. In some individuals, acidic fluids from the stomach also mix with saliva. Sometimes, liquid forms of food and beverages are also added to the oral fluids. For this reason, saliva is often referred to as whole saliva, which includes all oral fluids, rather than duct saliva, which is secreted by the salivary glands.

Digestive juices are usually isotonic. Saliva is also isotonic during its formation but becomes hypotonic during its journey through the duct network. In the mouth of a healthy adult, 1 ml of saliva spreads over a surface of approximately 200 $cm^2$ and forms a thin film only 10–100 microns thick.

Saliva consists of two main components: macromolecules and water. Approximately 99% of its content is water, and 1% is composed of organic and inorganic substances.

## 1.2 Organic content

The entire organic content of plasma is observed in trace amounts in saliva. Saliva contains enzymes, immunoglobulins (Ig), antibacterial proteins, mucin, albumin, polypeptides, hormones, urea, DNA, and viruses.

Many of the salivary proteins, present at a concentration of approximately 3% of plasma, have antimicrobial properties. The well-known major salivary glycoproteins (mucins, proline-rich glycoprotein, and immunoglobulins) and minor salivary glycoproteins (agglutinins, lactoferrin (Lf), cystatins, and lysozyme) are involved in the defense mechanism in the oral cavity. Salivary protein type and concentration vary in different secretions [1].

*Glycoproteins* are named according to the cell structures from which they originate. Mucous glycoproteins called mucins have low solubility, high viscosity, high elasticity, and strong adhesive properties. Mucins constitute 26% of salivary proteins. Mucins (MG1, MG2) in saliva constitute a large part of the viscous layer covering the soft and hard tissues of the oral cavity [2].

It contributes to controlling bacterial and fungal colonization by selectively regulating the adhesion of microorganisms to oral tissue surfaces, thus fulfilling an antibacterial role.

It is found in the secretions of the submandibular, sublingual, and small salivary glands. The mucin content of sublingual saliva is higher than that of submandibular saliva, whereas parotid saliva lacks mucin. There are two types of mucins, defined as MG1 and MG2 [3].

They differ in function and structure. High-molecular-weight MG1 contributes to the enamel pellicle. MG1 is found in higher concentrations in unstimulated saliva than in stimulated saliva. This explains the high viscosity of unstimulated saliva. Small-molecular-weight MG2 is more useful in washing away oral bacteria, including *S. mutans*. MG2 is present in similar concentrations in stimulated and unstimulated saliva.

*Proline-rich proteins (PRPs)* are a complex group of proteins. These proteins contain high levels of proline and are therefore called proline-rich proteins. They constitute 70% of the total protein content of human parotid saliva. Proline-rich proteins have a high affinity for hydroxyapatites. They rapidly adsorb to the hydroxyapatite surface from saliva and inhibit the spontaneous precipitation of calcium phosphate salts. They are also involved in the lubrication of oral tissues. They are secreted from the parotid and submandibular glands [4].

Proline-rich proteins encoded by genes called salivary protein complex constitute 37% of salivary proteins. Proline-rich proteins facilitate mutans streptococci to colonize the tooth surface and form a biofilm layer. Therefore, the relationship between caries susceptibility and mutans streptococcal colonization is largely explained by genetic variation. The polymorphic, acidic proline-rich proteins in saliva, which cause individuals to have different past caries experiences, are encoded by the PRH1 and PRH2 genes. The absence of the Db allele of the PRH1 gene in Caucasians has increased caries rates. In addition, having the Pa and Pr22 genotypes increases the risk of caries formation in permanent teeth.

*Immune factors* originate from saliva (secretory IgA) or serum and gingival groove fluid (IgG, IgA, IgM). IgG, IgM, IgA, and secretory IgA (sIgA) form the basis of the salivary defense system against oral microbial flora, including *S. mutans*. IgA and IgM inhibit *S. mutans* adhesion to salivary hydroxyapatite and epithelial surfaces. sIgA is the biggest immunoglobulin. sIgA is the largest immunological component of saliva. It can neutralize viruses, bacteria, and bacterial enzymes. Antibodies against bacterial antigens precipitate bacteria and prevent their adhesion to oral tissues. IgM and IgG are less abundant. sIgA is the prominent immunoglobulin in whole saliva and is considered to be the main specific defense mechanism in the oral cavity. sIgA helps in the prevention of dental caries by inhibition of bacterial adherence, reduction of hydrophobicity, agglutination of bacteria, and inactivation of bacterial enzymes and toxins. Several studies on the role of sIgA in the prevention of dental caries showed contradictory results. We compared the sIgA levels in the unstimulated whole saliva of caries-free and caries-active children to determine the role of sIgA in protection from dental caries [5].

*Lysozyme (muramidase)* is an enzymatic protein with antimicrobial activity. It is secreted more from the sublingual gland than from the submandibular and parotid glands. Gingival groove fluid also contributes lysozyme.

Lysozyme is an enzyme that cleaves the β(1:4) bonds between N-acetyl muramic acid and N-acetyl glucosamine and thus causes the death or lysis of the bacteria by the destruction of these molecules in the bacterial wall. If a bacterial wall lacks these polysaccharides, lysozyme is ineffective there. Gram-negative bacteria are more resistant to this enzyme because they have an external lipopolysaccharide layer with a protective function. The activity of lysozyme in saliva is probably inversely related to the presence of mucin, since mucin is thought to inhibit lysozyme [6]. Lysozyme is a small protein with a molecular weight of 14.6 kDa, is cationic, and has an isoelectric point (pI). Lysozyme can be found in fluids such as tears, breast milk, gastric fluid, nasal secretions, serum, saliva, urine, and cerebrospinal fluid. In the mouth, it is produced from salivary glands (major and minor), gingival sulcular fluid, and salivary leukocytes [7].

*Lactoferrin (Lf)* is a nonenzymic antimicrobial protein secreted from the serous cells of the major and minor salivary glands. It exerts bacteriostatic or bactericidal effects by binding to free iron, which is essential for the survival of bacteria (especially *S. mutans*). When it does not bind to iron, it binds directly to the bacterial cell surface, leading to their precipitation. Lactoferrin is also a multifunctional protein with fungicidal, antiviral, and anti-inflammatory properties. It also plays a role in inhibiting the initial stages of *S. mutans* colonization of tooth surfaces, competing with bacteria for attachment to salivary agglutinin, which results in reduced pathogen adhesion to the pellicle formed by saliva. Furthermore, Lf strongly prevents the formation of bacterial biofilms, a compact community of microorganisms attached to natural or artificial surfaces, which is associated with higher resistance of pathogens to unfavorable external factors and antibiotics [8].

*Peroxidase (PO) systems* consist of two enzymes: sialoperoxidase originates from acinar cells and myeloperoxidase (MPO) from gingival leukocytes. The main function of the system is to protect host proteins and cells from oral bacteria through antimicrobial activity.

Hydrogen peroxide toxicity is produced by *S. mutans* and Lactobacilli. *In vitro* studies have shown that peroxidases are effective against *S. mutans* and Lactobacilli. Peroxidase is an enzyme that catalyzes the oxidation of thiocyanate to convert $H_2O_2$ to hypothiocyanite, an antimicrobial agent.

As a result of the study conducted by Gornowicz et al. [9] to compare slgA, histatin-5, and lactoperoxidase (LPO) levels between adolescents with low and high caries activity, it was reported that compared to adolescents with low caries activity, adolescents with high caries activity had significantly increased salivary slgA, histatin-5, and lactoperoxidase levels. In their study, they found that salivary peroxidase levels were high in individuals with low caries activity and that there was an inverse relationship between caries formation and this enzyme [10]. In a study by Gudipaneni et al. [11], as a result of a clinical trial evaluating the antimicrobial efficacy of a toothpaste containing lactoferrin, lysozyme, and lactoperoxidase in children with severe childhood caries, they reported that this paste (S-ECC) significantly reduced the levels of *S. mutans* and *L. acidophilus* in the saliva of children using it. Lactoperoxidase (LPO), a heme-containing peroxidase enzyme in saliva, catalyzes the oxidation of electron-donating substrates through hydrogen peroxide-mediated reactions. Salivary peroxidase activity originates from two primary sources: human salivary lactoperoxidase (HS-LPO), produced by salivary glands, and myeloperoxidase (MPO) derived from polymorphonuclear leukocytes (PMNs) in gingival crevicular fluid. MPO contributes 30–75% of total salivary peroxidase activity, varying with individual health status. Notably, children with Down syndrome exhibit reduced stimulated salivary peroxide levels (1.53 µg/mg) compared to healthy children (2.58 µg/mg). LPO demonstrates broad catalytic activity, oxidizing both inorganic (bromide, iodide) and organic compounds, classifying it as a haloperoxidase. Its interaction with thiocyanate generates potent bactericidal oxidation products. Collectively, the lactoperoxidase system—comprising LPO, thiocyanate, hydrogen peroxide, and their reaction products—forms a critical antimicrobial defense mechanism in oral immunity, protecting against pathogenic microorganisms through oxidative biocidal activity [12].

The lactoperoxidase system represents a promising natural defense mechanism against cariogenic biofilms. This system demonstrates enhanced antimicrobial activity. These modifications disrupt critical pathogenic processes in *Streptococcus mutans*, primarily by interfering with carbohydrate transport and metabolism. As a result, the expression of key virulence factors is significantly reduced, particularly the bacteria's ability to form biofilms through polysaccharide matrix production and lactic acid generation. This targeted action makes the lactoperoxidase system a potential therapeutic strategy for caries prevention, offering a natural approach to inhibit the development and progression of dental decay while maintaining oral microbial balance [13].

*Agglutinins* were identified as a glioprotein precipitating *S. mutans* isolated from parotid saliva but were also found to be present in submandibular and sublingual saliva. It has now been described to bind to other bacteria besides *S. mutans*. Agglutinins enable bacteria to aggregate and then be ingested with saliva.

*Cystatin* is a cysteine-containing protein. Cystatin has proteinase inhibitory activity and is also involved in mineralization control.

*Statherin* is a protein that contributes to the stabilization of calcium and phosphate salts in saliva. It inhibits the spontaneous precipitation of calcium and phosphate salts. The statherin molecule is too large to enter the enamel pores. Therefore, it remains bound to hydroxyapatite on the surface, allowing the passage of minerals for remineralization and controlling the crystalline growth of enamel. Recent studies have shown that statherin can contribute to the initial colonization of certain bacteria (such as Actinomyces viscosus) on the tooth surface. It is found in the secretions of both the submandibular and parotid glands [14].

*Histatin* is a histidine-rich peptide (HRP) with antimicrobial properties found in parotid and submandibular secretions. It plays an important role in inflammation by inhibiting histamine secretion from mast cells. It also contributes to pellicle formation.

*Amylase* is a major component of parotid secretion. The enzyme α-amylase (ptyalin) plays a minor role in the early stages of digestion, breaking down starch into maltose and dextrin. The main function of salivary amylase is thought to be to digest starch residues left in the mouth after a meal, rather than to contribute to digestion.

*Sialin* is a small tetrapeptide with the ability to increase plaque pH in saliva. It is degraded to ammonia and polyamines by the enzymatic activity of bacteria. It is present in parotid secretion.

*Urea* is an organic compound that increases pH. The urea molecule is converted into ammonia and carbon dioxide by the action of the enzyme urease, increasing the pH of the acidic environment.

### *1.2.1 Saliva proteins*

It has been reported that there may be a relationship between genes encoding salivary proteins and caries susceptibility. To date, genes encoding salivary proteins carbonic anhydrase 6 (CA6), proline-rich protein, mucin, and aquaporin have been investigated in relation to caries.

*Carbonic anhydrase 6 (CA6)* is the secretory form of the carbonic anhydrase enzyme found in saliva and other body fluids. This enzyme was first identified in saliva produced by the serous cells of the parotid and submandibular glands. It is stated that carbonic anhydrase 6 in saliva penetrates dental plaque and facilitates acid neutralization with salivary bicarbonate. The bicarbonate system is the main buffer that contributes to the buffering capacity of saliva. Bicarbonate ions have been reported to neutralize lactic and acetic acid produced by plaque bacteria, thereby reducing demineralization. Carbonic anhydrase 6 is encoded by the CA6 gene. Although there is no association between dental caries and CA6 gene polymorphism, salivary buffering capacity may be affected by polymorphism in the CA6 gene [15].

Salivary defense elements play an important role in maintaining oral health and caries prevention. Antimicrobial peptides (AMPs) in saliva are natural antibiotics that form the first line of defense against a broad spectrum of microorganisms. The importance of these peptides is especially prominent in the mouth, where microbial flora is constantly dominant.

According to their structural properties, antimicrobial peptides can be classified as defensins, histatins, and cathelicidins. The levels of human neutrophil peptides (HNP1-3), which are in the alpha defensins group, in saliva are genetically controlled. Low levels of HNP1-3 can be considered as useful data for assessing caries risk in children [16].

Another salivary protein that has been examined for the relationship between salivary components and caries formation is mucin. The most important function of mucin protein is to prevent the colonization of bacteria on the tooth surface. Studies have not found a relationship between caries formation and the MUC7 gene encoding mucin protein.

*Aquaporin proteins* in saliva play a role in tooth development during water uptake from the extracellular matrix. Aquaporin 5 protein is encoded by the AQP5 gene and is involved in salivary secretion. AQP5 deficiency in mice has been associated with caries susceptibility by decreasing salivary flow, while in humans, alleles in this gene with caries-protective properties have been identified.

### 1.3 Inorganic content

Sodium, potassium, chlorine, and bicarbonate are abundant in saliva, while calcium, inorganic phosphate, fluoride, magnesium, and iodine ions are less abundant. All of these are of serum origin and actively transit through the striated ducts and acini. Electrolyte composition has been found to be affected by flow rate. pH and ion concentration can affect the activation of salivary organic components. Sodium and chlorine concentrations change in proportion to the flow rate. Potassium concentration decreases slightly with increasing salivary flow rate.

Calcium is an ion released by salivary proteins. It increases in parallel with the salivary flow rate. Salivary calcium levels are not affected by diet. Depending on salivary pH, salivary calcium can be ionized or nonionized. Ionized calcium is particularly important in caries episodes as it maintains the balance between enamel calcium phosphate and surrounding fluids. No association between salivary calcium and dental caries has been demonstrated. However, it has been suggested that increased levels of plaque calcium passing through saliva are beneficial.

Inorganic phosphates in saliva consist of phosphoric acid ($H_3PO_4$), primary ($H_2PO_4$), secondary ($HPO_4$), and tertiary ($PO_4$) inorganic phosphates. The concentration of inorganic phosphate decreases with increasing salivary flow rate. High phosphate levels are associated with low caries rates [17]. But the opposite has also been reported. The most important function of these ions is to maintain the dental structure. The other function is buffering.

Fluoride is the most effective caries preventive agent known today. This property of fluoride was first recognized by Dean in the 1930s when he observed the relationship between fluoride in drinking water and endemic fluorosis and dental caries. The salivary fluoride concentration is independent of the salivary flow rate. Fluoride enters saliva directly from ingestion or topical treatments (drinking water, beverages, foods, toothpastes, and mouthwashes) or indirectly from the bloodstream *via* saliva or gingival groove fluid. Fluoride ion reduces the solubility of hydroxyapatites, making them more resistant to demineralization. Fluoride in the fluid surrounding enamel crystals has been shown to have the potential effect of reducing enamel demineralization and increasing remineralization. Fluoride also reduces acid production in dental plaque. It does this by inactivating the carbohydrate-metabolizing enzymes of acidogenic microorganisms [17].

Bicarbonate ($HCO_3$) ion is important for the buffering effect of saliva. As the amount of $HCO_3$ increases with increased saliva flow, the pH rises.

## 2. Functions of saliva

Saliva is an important protective factor in the performance of oral functions and in maintaining the integrity of oral hard and soft tissues. The components of saliva perform many functions together, complementing and reinforcing each other. Each of these functions is multifaceted.

### 2.1 Lubrication effect

Saliva lubricates and protects tissues against irritants (proteolytic and hydrolytic enzymes produced in bacterial plaque, smoking, or various carcinogenic agents, dryness caused by mouth breathing) by coating the oral cavity in the form of a continuous ceremucosal film. This layer, called biofilm, protects teeth against erosion and abrasion and aids chewing, speaking, and swallowing.

### 2.2 Washing and dilution effect

The most important caries-protective function of saliva is to dilute carbohydrates and bacterial products that enter the oral cavity with food. After ingestion of food and drink, sugar reaches high concentrations in saliva. Regulates sugar levels in the plaque by eliminating high levels of carbohydrates. With the cleaning process referred to as oral clearance, microorganisms are removed from the mouth along with dietary sugar intake.

Saliva continuously washes the teeth and oral mucosa; mechanically cleanses the existing bacteria, cells, and food residues; ensures their removal from the mouth; and slows down plaque formation. A high flow rate is a high diluting and washing capacity. A change in health status that reduces salivary flow also causes marked changes in oral hygiene.

Studies have found that there is a relationship between the clearance of carbohydrates and the salivary flow rate, and that carbohydrate clearance decreases in individuals with decreased salivary flow rate. Carbohydrate clearance is faster in certain areas of the mouth. For example, on the lingual surfaces of the lower anterior teeth. This is directly related to the salivary flow rate. Clearance is also related to the frequency of swallowing and is faster in individuals with frequent swallowing habits. In addition, organic acids formed as a result of the breakdown of carbohydrates by cariogenic microorganisms are diluted with saliva, and this event is reported to be directly related to the salivary flow rate. Animireddy et al. [18] reported that the salivary flow rate increased statistically significantly and viscosity decreased in children without dental caries compared to children with few and severe caries. Gopinath et al. [19] reported that salivary parameters such as salivary flow rate, viscosity, pH, and buffering capacity decreased in individuals with an increased number of dental caries.

### 2.3 Buffering capacity

The ability to resist pH changes that may vary depending on H and OH ions in the environment is called buffering power. Saliva has a neutral pH (6.5–7.5) and is an effective buffer system. Buffering capacity functions to protect tissues against acids from nutrients and bacteria and to maintain pH within physiological limits. If pH is 5.5 and below, buffer components buffer acids and maintain pH at 6.0–7.5. The buffering capacity of saliva plays a major role in caries protection by neutralizing extrinsic and intrinsic acids that will cause tooth erosion. The buffering systems in saliva are bicarbonate-carbonic acid, phosphates, and proteins.

Bicarbonate is the most important buffering agent that primarily determines the buffering capacity of saliva. Saliva is rich in bicarbonate. It neutralizes acids formed as a result of the activities of acidogenic microorganisms. When acids and bicarbonates react, acids are decomposed into water and $CO_2$. Carbonic anhydrase, an enzyme found in the secretions of the parotid and submandibular glands, also contributes to the production of bicarbonate and is effective in the neutralization of acids.

It has been reported that the carbonic acid-bicarbonate buffer system is important in stimulated saliva, while the inorganic phosphate buffering system is important in unstimulated saliva. Inorganic phosphate ($HPO_4$) binds the H ion in the $H_2PO_4$ form and performs the buffering mechanism. Proteins do not act as buffers in environments above physiological pH but contribute when pH drops below.

Buffering capacity is related to the salivary flow rate. Decreased salivary flow rate leads to decreased buffering capacity. Buffering capacity is increased by the stimulation of saliva. Since the buffering capacity usually increases after a meal, it is reported that saliva for the test should be collected approximately 2 hours after a meal. It has been reported that buffering capacity may be reduced by decreased salivary flow rate, decreased response to caries-causing agents, malnutrition, and pregnancy. The thickness of the bacterial plaque and the number of bacteria determine the effectiveness of salivary buffers. The pH and buffering capacity of saliva are thought to be closely related to caries. Two methods are used to measure salivary buffering capacity:

#### *2.3.1 Ericsson method*

Without waiting, 1 ml of the collected saliva is taken into another container, and 3 ml of 0.005 molar HCl is added to it. The container is gently vibrated to remove carbon dioxide. The samples are kept for 10–20 minutes. At the end of the time, the pH is measured. Measurement can be done using a pH meter and pH indicator paper.

#### *2.3.2 Dentobuff strip method*

Special kits are used in this method to measure saliva buffering capacity. Such kits contain a special tube containing a weak acid. 1 ml of stimulated saliva is transferred into this test tube and allowed to stand. According to the color difference, information about the saliva buffering capacity is obtained. If the color is yellowish brown, it can be said to have a low buffering capacity of pH < 4; if it is green, it can be said to have a moderate buffering capacity of pH < 4.5–5.5; and if it is bluish, it can be said to have a high buffering capacity of pH > 6.0. Buffering capacity and pH are very important in the oral environment. When saliva is first secreted, its pH is slightly acidic. However, pH increases with salivary flow due to the increase in sodium and bicarbonate. Salivary pH can decrease to 5.3 at low flow rate and increase to 7.8 at high flow rate [20].

The value at which the fluid on the tooth surface is unsaturated with respect to hydroxyapatite and allows the separation of calcium and phosphate from the enamel is called the critical pH value. This is a pH of 5.5 or below. Below the critical pH, the dissolution of tooth enamel begins. This is an important reason for the onset of dental caries. The pH is low in the morning and on an empty stomach, and it increases later. The pH is low in women and during pregnancy. A reduced salivary flow rate leads to an increase in the number of acidogenic microorganisms in dental plaque and saliva, leading to a decrease in pH.

#### *2.3.3 pH measurement can be done by the following methods*

##### *2.3.3.1 Determination of pH with indicator*

This is the easiest way to determine pH. Indicators are ionized weak acids or structures that change color depending on pH. The major disadvantage is that the change is not visually detectable when it is less than 10%.

*2.3.3.2 Determination of pH by electrical pathway*

This method uses an instrument called a pH meter. It has a glass electrode with a platinum electrode impregnated with 1 M HCl. It is made of a special glass that conducts electricity at the end covered with thin-walled glass. The glass electrode, together with the standard calomel, forms a cell. The electromotive force of this cell at constant temperature depends entirely on the H ion concentration of the solution in which the glass electrode is placed.

### 2.4 Effect on remineralization

Saliva plays an important role in maintaining the physicochemical integrity of enamel by balancing remineralization and demineralization. Remineralization is the replacement of lost minerals by the organic matrix of the enamel, and this property of saliva is an important defense mechanism against caries. Maintaining the stability of the mineral structure of the enamel depends on saliva being saturated with calcium, phosphate, and fluorine salts. The mineral saturation of saliva plays an important role in posteruptive maturation. This maturation increases the surface hardness. Creates a structure that is more resistant to decay. Calcium and phosphate ions can stop decay by remineralizing demineralized enamel. It was investigated why a spontaneous mineral formation does not occur on enamel surfaces.

It was investigated why a spontaneous mineral formation does not occur on enamel surfaces. As a result, it has been determined that selective remineralization occurs with two phosphoproteins found in saliva. One is a proline-rich phosphoprotein, and the other is a trozine-rich phosphoprotein called statherin. As a result, it has been determined that selective remineralization occurs with two phosphoproteins found in saliva. These molecules' calcium-binding capacities. When pH decreases, the proteins bind calcium and phosphate ions and transport them to the tooth surface, increasing remineralization.

The so-called initial caries lesions refer to superficial soft enamel, moderately demineralized enamel, demineralization lesions beneath the superficial layer, and remineralized areas that have become resistant to acid attacks. These lesions can be remineralized by saliva rich in calcium, phosphate, and fluoride.

Pandey et al. [21] stated that compared to carious individuals, the salivary calcium content of caries-free individuals is higher, and the amount of calcium increases as the age of the individual increases. When the balance of remineralization, that is, why a spontaneous mineral formation does not occur on enamel surfaces, it was determined that selective remineralization occurs with two phosphoproteins in saliva. One is anionic PRP, and the other is TRP called statherin. In addition, HRP and cystatin S also play an auxiliary role. In studies, it has been determined that the levels of protein in saliva are higher in individuals with active caries compared to caries-free individuals.

### 2.5 Antimicrobial effect

Numerous antimicrobial factors in the oral defense system influence oral microbial composition and growth conditions. Saliva contains antimicrobial, non-immunological, and immunological proteins. Nonimmunologic salivary proteins include enzymes (lysozyme, lactoferrin, and peroxidase), mucin glycoproteins, agglutinins, histatin, proline-rich protein, staterin, and cystatin. Immunologic factors are IgA, IgG, and IgM [22]. These factors found in saliva control the oropharyngeal flora and protect oral tissues from viruses, fungi, and bacteria.

sIgA from saliva and IgG from gingival crevicular fluid are salivary antibodies that have the ability to selectively agglutinate microorganisms. Studies have shown that there is an inverse relationship between the level of sIgA in saliva and dental caries. As a result of the increase in salivary sIgA level, the adhesion of oral microorganisms to tooth surfaces, growth, and colonization decreased. It has also been reported that sIgA inhibits glycosyltransferase enzyme activity. Glycoproteins called agglutinins with high molecular weight in saliva also have antimicrobial properties like sIgA. Apart from salivary antibodies, components such as lactoperoxidase, lactoferrin, lysozyme, and histidine-rich peptides (HRPs) also have antimicrobial effects in saliva.

### 2.6 Formation of the bolus

Saliva plays an important role in preparing food for swallowing and in the act of swallowing. Saliva, with its mucin and abundant water content, allows food to be bitten and swallowed easily without causing any trauma to the soft tissues in the mouth. Saliva continuously keeps the oral cavity moist, moistening dry foods and cooling hot foods.

### 2.7 Effect on digestion

Saliva is responsible for the initial digestion of starch. This process occurs with the presence of the digestive enzyme alpha-amylase found in saliva. The activity of the enzyme is inactivated in acidic parts of the gastrointestinal tract. Therefore, its effect is limited to the mouth. Saliva, with its mucin and large amount of water, allows the food to be bitten and swallowed easily without traumatizing the soft tissues in the mouth. Saliva continuously keeps the oral cavity moist, moistening dry foods and cooling hot foods. Although these enzymes are present in saliva, oral digestion is not of significant importance. In contrast, the effect of lipase, which breaks down fat, is considered more important because it is not inhibited by the stomach's acid.

### 2.8 Influence on taste

Saliva is essential for optimal function of the taste buds. The hypotonic nature of saliva (low levels of glucose, sodium, chlorine, and urea) allows for the dissolution and transport of taste substances, allowing the digestive buds to perceive different tastes. Foods must be dissolved in water to be palatable. Foods dissolve in saliva, making their absorption easier. The taste buds on the tongue are cleaned with saliva and made ready for new stimuli.

Again, in the perception of the taste of the consumed foods, in addition to the tongue, there is also a component called gustin, which is a peptide structure found in saliva, that plays a role. Gustin is a salivary protein essential for the growth and maturation of taste buds.

### 2.9 Effect on speech

Saliva has an important function during speech as well. Wetting of the buccal and pharyngeal mucosa is essential for speech.

### 2.10 Excretory function

Saliva is the site of excretion for hormones, urea, iodine, fluoride toxins, mercury, lead, iron, gold, bismuth, thiocyanate, morphine, and many antibiotics.

### 2.11 Water regulation effect

When the body loses water in hot weather, saliva flow decreases, and the desire to drink water is activated.

### 2.12 Hormonal effect

A protein called parotin has been identified in saliva. This hormone helps the development of mesenchymal tissue and plays an important role in bone development.

### 2.13 Tissue repair

In studies conducted, although the amounts in saliva are very low, it has been determined that specific growth factors such as epidermal growth factor and nerve growth factor have a healing-promoting effect on oral mucosal injuries.

### 2.14 Other duties

Blood group substances found in saliva help forensic identification.

## 3. Factors affecting saliva secretion

Salivary gland function is regulated by the autonomic nervous system, with the medulla oblongata serving as the primary secretory control center. While both sympathetic and parasympathetic stimulation trigger saliva production, parasympathetic input exerts a more dominant effect. Secretion can be initiated either through direct autonomic innervation or by substances that mimic these neural signals. The process occurs in three key phases: the cephalic phase begins with the mere thought or smell of food, activating the cerebral cortex to stimulate salivation; the buccal phase is triggered when taste or tactile receptors in the mouth are stimulated; and the gastrointestinal phase involves reflexes from the stomach and upper digestive tract, where stretching of the esophagus or gastric mucosal irritation further promotes saliva secretion. This multiphase regulation ensures optimal saliva production for digestion and oral protection.

Saliva secretion exhibits significant variability in both flow rate and composition depending on physiological and pathological conditions. At rest, minimal secretion occurs—just enough to maintain mucosal moisture. However, salivary output and biochemical properties fluctuate based on age, gender, sleep patterns, hydration status, emotional state, systemic diseases (e.g., infections or neurological disorders), medications, and stimulus type. Mastication represents the most potent stimulant for salivation, followed closely by olfactory and gustatory cues. Increased secretion occurs during gum chewing, metal intoxication, pain responses, smoking, acute stomatitis, parasitic infections, and nausea—nocturnal hypersalivation in children may even indicate intestinal parasites. Notably, salivary enzyme and electrolyte concentrations dynamically adjust with flow rate changes. After age 20, secretory capacity gradually declines, reaching 0.025–0.034 mL/min by age 60, with men typically maintaining higher baseline rates than women. These variations underscore saliva's sensitivity to both internal and external influences, reflecting its vital role in oral and systemic homeostasis [23].

## 3.1 Saliva flow

Saliva flow varies depending on the individual's physiological condition and is also influenced by many factors throughout the day. Daily flow variability affects not only the flow rate but also the balance of electrolytes and proteins. The secretion content released from different glands shows many variations depending on the type of stimulus, diet, age, gender, any disease condition, and some pharmacological agents. In healthy individuals, under resting conditions without a stimulus from chewing, there is a constant flow of saliva that helps protect the tongue and teeth (oropharynx and oral mucosa) and moistens the environment.

### *3.1.1 Saliva flow rate*

The stimulated saliva flow rate is the expression of the amount of saliva obtained after the application of stimulation in ml/min. While the stimulated saliva sample is being taken, 1.5 g of paraffin or sugar-free gum is chewed for a few seconds. The first saliva formed is swallowed by the patient. Then, the chewing movement is continued using both sides of the jaw for 5 minutes. The saliva formed is spat into the sterile container tube. In patients with many missing teeth, stimulated saliva can be collected by tongue, lip, and cheek movements. The amount of saliva obtained is calculated as ml/min. Stimulated and unstimulated saliva flow rate values are shown in moments of body rest; a small amount of saliva with a relatively thick structure is secreted.

In the case of any stimulus, a relatively thin saliva begins to flow abundantly. In adults, the unstimulated salivary flow rate is 0.3–0.5 ml/min. The normal flow rate of stimulated saliva was given by the FDI in 1992 as 1–2 ml/min on average. There are many factors that affect the unstimulated salivary flow rate in healthy individuals. Light, smelling, thinking about food, chewing gum, metal poisoning, pain and irritations, some medications (such as pilocarpine, citric acid, and choline), acute stomatitis, intestinal parasites, nausea, and mechanical stimuli (such as dental instruments) cause an increase in secretion. It decreases in psychic states such as fear, excitement, depression, and stress, and physical states such as dehydration, increased ambient temperature, hospitalization, darkness, exercise, and fatigue. Saliva flow rate is calculated as unstimulated and stimulated [23].

### *3.1.2 Unstimulated salivary flow rate*

The patient is seated upright, and the head is bent forward. The arms and shoulders should be freely extended, and the forearm should be in contact with the leg up to the hands. The tip of the tongue should rest against the back surface of the lower teeth and remain motionless. The lips should be slightly open, and saliva should flow from the corner of the mouth. Spit into a sterile container for 5–10 minutes. The amount of saliva obtained is calculated in ml/min. It is difficult to maintain the standard.

## 4. Aging and salivation

Saliva secretion and its components, which play an important role in maintaining oral health, change with age. Xerostomia and related complications are seen in approximately 50% of the elderly population. With decreased salivary function, chewing, tasting, and swallowing functions are impaired, and various oral diseases, including dental caries and periodontal diseases, occur. This leads to malnutrition in the elderly. Hyposalivation causes difficulties in eating, tasting, and speaking, seriously affecting quality of life. Therefore, good salivary gland function is one of the important requirements of healthy aging. Regardless of the underlying mechanism, a decrease in salivary volume and a change in its composition are clinically very important. Saliva secretion becomes quite dense due to the decrease in its volume, its rheological properties deteriorate, and its functions, such as lubricating and moisturizing oral surfaces, decrease. This reduces the protective capacity of saliva. At the same time, the amount of various salivary proteins, such as enzymes and immunoglobulins, which are natural protectors, also decreases. Compared with young people, older people have been reported to have a 28–71% decrease in total salivary constituents and a 62% decrease in salivary flow rate compared with young people. It has also been reported that the decrease in salivary flow rate dominates the slight increase in relative concentrations [24]. Fluid consumption also decreases due to a decrease in the sense of thirst, which is not related to medications and systemic diseases but is seen due to aging. As a result of frequent urination and a decrease in the body's water storage capacity, the amount of water in the body may fall below the required level. This can lead to dry mouth. The use of diuretic or laxative drugs is also effective.

Salivary gland aging has been investigated from structural and functional aspects. Significant structural changes due to aging affect all salivary glands, large and small. Histologic changes occur in the salivary glands as a result of the replacement of acinar cells with fat, connective tissue, and duct-like epithelial structures. Acinar cell atrophy, cytoplasmic vacuolization, and lymphocyte infiltration have been detected in the submandibular glands of aged mice. In humans, an increase in adipose tissue is accompanied by a progressive decrease in the volume of acinar cells of the sublingual salivary gland. Changes in salivary function on oral and dental health have been observed with the effect of aging. The increase in stroma and decrease in parenchyma observed in the sublingual salivary gland is a common feature of aging in human salivary glands [25].

Although xerostomia and salivary gland hypofunction seem to be related due to their common features, there are important differences between them. Xerostomia is a subjective dry mouth. Oral surfaces may appear moist, but the patient's subjective feeling of dry mouth indicates xerostomia. Hyposalivation, a quantitative finding, can be determined by measuring the salivary flow rate.

A stimulated salivary flow rate of less than 0.7 ml/min or an unstimulated salivary flow rate of less than 0.1 ml/min in the resting state is called hyposalivation [12]. As a result of hyposalivation, cervical and root caries, erosion, mucosal lesions, and opportunistic infections develop rapidly. Therefore, the elderly who are thought to have salivary gland hypofunction should be diagnosed as soon as possible, and preventive measures should be taken. The risk factor that often accompanies aging in the development of dry mouth is polypharmacy. Over 400 medications have the potential to reduce salivary secretion, causing both subjective and objective dry mouth and subsequently various oral diseases. Medication is the most common cause of xerostomia. It has been reported that the incidence of xerostomia in patients taking 4–5 medications per day is 50%.

Antidepressants, anticholinergics, antihistamines, diuretics, antihypertensives, sedatives, and antiemetics are the most commonly prescribed drugs in the elderly that cause xerostomia. In addition, the use of mouthwashes containing alcohol and caffeinated beverages due to their diuretic effects can also lead to increased dryness in the mouth.

The viscosity of saliva is determined by the mucopolysaccharides in its composition. Mucins are mostly found in areas of mucosal secretion. The effect of aging-related changes in salivary function on oral and dental health provides moisture and lubricity to the environment with their water-retaining properties. Mucins contain high levels of carbohydrates. Sialic acid is a negatively charged carbohydrate that is hydrophilic and is found in the structure of some mucins. The strong electronegative charge of sialic acids increases the viscosity of mucin-containing secretions by retaining water molecules. This property is due to the repulsion of the negative charges of sialic acids.

Aging is one of the causes of xerostomia, which is characterized by a decrease in salivary flow rate and an increase in salivary viscosity. In the elderly, mucosal defense decreases, and sensitivity to stimuli deteriorates with the change in salivary rheology and loss of mucin function. It has been reported that the amount and glycosylation of salivary mucins decrease in the elderly, and especially the desialylation and deglycosylation of MU7 negatively affect mucin function.

In addition, the interaction of taste elicitors with their receptors is impaired with decreased mucoadhesion. Among mucins, acidic mucins are thought to play a key role in increasing the viscosity of saliva.

The determining factor is the rate of salivary flow. With a continuous flow of saliva, unwanted components and cells are removed without adhering to oral surfaces. Some animal studies have shown that with aging, protein synthesis in the salivary glands decreases by 60%. This may result in changes in the amount and/or activity of the organic components of saliva. In human studies, the concentration of secretory sIgA, especially in labial saliva, and the concentrations of high- and low-molecular-weight mucins in mucous saliva decreased with age. Since sIgA and mucins are important for immunological and non-immunological defense of the oral cavity, both defense systems decrease in healthy elderly people. Salivary antibodies play an important role in defending the oral surfaces against pathogens. Changes in salivary antibodies with aging may contribute to the development of mucosal immunosenescence and the risk of infection. sIgA is the most important immunoglobulin in saliva. The rate of salivary secretion of IgA has been shown to be significantly lower in older individuals and to decrease with advancing age [26].

MUC5B and MUC7, which are mucins in saliva, have functions such as lubricating and protecting oral surfaces and modulating oral microorganisms. The effects of salivary mucins, which are not components of the immune system, are transient and are removed from the mouth by swallowing after secretion. Large and small salivary glands as well as oral epithelial cells produce MUC1, a membrane-bound mucin. MUC1 participates in the mucus gel structure and is the first line of defense in the oral cavity. MUC1 on the cell surface is the most important component of the mucosal barrier against infection, and its synthesis has been reported to increase in infections caused by bacterial pathogens. Therefore, decreased synthesis of MUC1 in the oral mucosal epithelium due to aging increases susceptibility to infections. The presence of MUC1 in residual saliva covering the oral cavity provides both hydration of oral surfaces and an antibacterial effect [27].

## 5. Conclusion

In conclusion, although saliva is not among the primary factors that directly affect the formation of dental caries, it prevents the formation of caries thanks to the organic and inorganic compounds it contains and helps the remineralization of initial caries. We see an example of this in individuals who do not have caries in their mouth despite never brushing their teeth.

In addition, the importance of saliva in the protection of oral-dental health is much better understood in individuals with decreased saliva flow rate or no secretion for any reason, both by disruption of the integrity of oral soft and hard tissues and difficulties in performing activities such as swallowing, tasting, and speaking.

For this reason, we believe that we should not forget the importance of saliva, which is one of our natural resources in maintaining oral and dental health, and what its protective functions are.

Studies on saliva are increasing day by day. We think that saliva will play a key role not only in oral and dental diseases, such as dental caries, but also in the early diagnosis and monitoring of many diseases.

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**Chapter 3**

# Development of Strategies Based on the Recent Evidence for the Management of Dental Caries in Children: A Systematic Overview

*Shivani Dhonde and Ashwin Jawdekar*


## Abstract

An evidence-based approach to caries management is crucial for standardizing pediatric dental care and improving outcomes. While international guidelines like those from AAPD, EAPD, and SDCEP offer structured recommendations, a unified approach for children still lacks. This initiative aims to fill that gap by developing standardized strategies relevant to children. This paper aims to summarize and critically appraise current evidence on caries management in children from global perspectives and to propose context-specific recommendations. A structured literature search using keywords such as "caries," "guidelines," and "management" was conducted across PubMed, the Cochrane Library, and Google Scholar. Two independent reviewers (SSD and AMJ) screened articles published in English between January 2014 and mid-September 2024. Older studies were included only if more recent updates were unavailable. Recommendations were categorized as strongly recommended, conditionally recommended, or not recommended based on the quality and applicability of the evidence. Inclusion criteria included literature published in English or with full-text English translations. Articles had to be peer-reviewed and published between 2014 and mid-2024. Excluded were non-peer-reviewed sources or guidelines lacking authenticity or universal relevance. Key strategies in caries prevention include dietary guidance, fluoride use, sealants, NFTRAs, and non-invasive care. Risk-based and activity-based models guide treatment planning. Selective caries removal and biological approaches are preferred in primary teeth. Tricalcium silicate materials have reduced the need for invasive pulp therapies. Emphasis on behavior change and recall scheduling remains essential for long-term success.


**Keywords:** evidence-based guidelines, caries management, global perspective, pediatric dentistry, preventive strategies, children

## 1. Introduction

Dentistry has evolved significantly with the emphasis on evidence-based approaches, fostering a culture of continuous improvement and innovation that enhances both patient outcomes and professional standards of care within the field, with dental caries management in children being no exception. Dental caries continues to be a global concern, and several evidence-based approaches have been successful in reducing the burden, particularly in rich countries. Several guidelines and summary recommendations have been developed by professional bodies and government organizations for caries management such as the AAPD reference manual (USA), Delivering Better Oral Health Toolkit (UK), the most recent caries pathways approach of IAPD, and so on.

This is an attempt undertaken to formulate evidence-based recommendations for caries management. In alignment with these global efforts, this study seeks to adapt existing evidence-based recommendations to provide a comprehensive framework for effective caries management.

*Objectives*

- To appraise the evidence gathered from different sources over the past decade (2013–2024) pertaining to management of caries.

- To formulate strategies for implementation/to recommend interventions for dental practice.

A following research question was formulated to address the above objectives:

*Which specific evidence-based recommendations for caries management can be made for children?*

PIOS:

*Population*- Children.

*Interventions*- Evidence-based recommendations for caries management.

*Outcome*- Development of strategies for management of dental caries.

*Study Design*- Systematic Overview.

## 2. Sources

### 2.1 Qualification of researchers

Dr. Ashwin Jawdekar, Professor and Head of the Department of Pediatric and Preventive Dentistry, conceptualized the study and supervised data collection. Dr. Shivani Dhonde, Postgraduate Student in the same department, conducted the literature review, performed data analysis, and drafted the manuscript.

### 2.2 Search strategy

This appraisal involves a critical analysis of research publications including systematic reviews, guidelines, conference summaries, invited authors' papers in top journals, and so on, aiming to identify key trends, innovations, and best practices that have emerged in caries management during this period. The goal is to provide a comprehensive understanding of how caries management strategies have evolved and to highlight the most effective approaches supported by the latest evidence.

The search was conducted using a combination of the keywords “caries,” “guidelines,” and “management.” Two electronic databases—PubMed and Cochrane Library—were primarily used for the search, and the search was extended on Google Scholar. The process was independently carried out by SSD and AMJ to ensure a comprehensive and unbiased collection of relevant studies, reviews, and guidelines on caries management. In case of conflict, an independent third investigator was identified for resolution. The detailed search strategy is given on https://drive.google.com/drive/folders/16ueHxTFGW9eMccLP8D3s9kBUwLYXWhnj?dmr=1&ec=wgc-drive-globalnav-goto

## 3. Methodology

This systematic overview has been registered in PROSPERO (International Prospective Register of Systematic Reviews) under the registration number-CRD420251010746.

*Eligibility Criteria*: The selection of articles was based on the following inclusion and exclusion criteria.

*Inclusion criteria*

- Literature published in English or full text translations available in English language
- Articles published from January 2014 to 2024 until mid-September
- Earlier articles (published before 2014) were included only if there were no more recent guidelines providing the same recommendations.

*Exclusion criteria*

- The guidelines that cannot be considered universal or lacked authenticity and scientific merit
- Articles available through unreliable sources or without peer reviews.

The search strategy is shown in **Figure 1**. A total of 24 reports were included for the appraisal.

## 4. Results

The literature was assessed for the quality of reporting using AGREE checklist for the guidelines, PRISMA checklist for the SRMAs, PRISMA-ScR checklist for the Scoping Reviews, and SANRA checklist for the narrative review [1–4]. The findings are reported in **Tables 1–4**, respectively. Three studies could not be assessed due to restricted access. One study was a critical review [22], while two were position papers [22, 23], which could not be appraised as no standardized checklists are available for such study types [22–24].

The summary statements or conclusions of the included reports were carefully segregated pertaining to the intervention/s, specifications, source, and so on. The same are presented in **Table 5**.

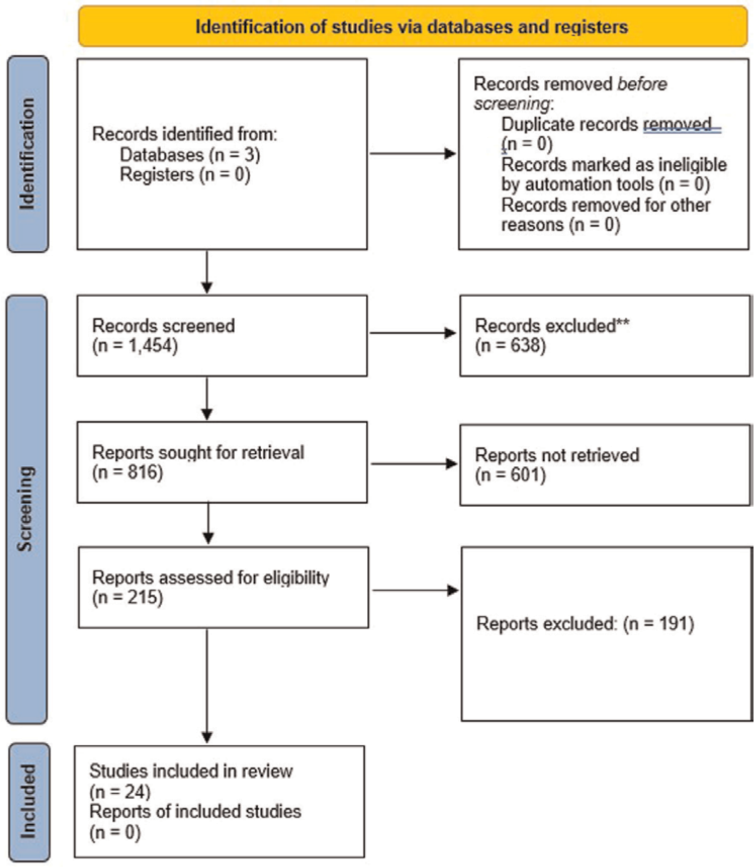


**Figure 1.**
*Search strategy.*

| | AGREE Checklist Item | Crall et al. [5] | Hurlbutt and Young [6] | Mejare et al. [7] | Do [8] | Featherstone et al. [9] | WHO [10] | Bulazaf [11] |
|---|---|---|---|---|---|---|---|---|
| Domain 1: Scope and Purpose | | | | | | | | |
| 1 | Objectives | Yes | Yes | Yes | Yes | Yes | Yes | Yes |
| 2 | Health Questions | Yes | Yes | No | No | Yes | Yes | No |
| 3 | Population | Yes | Yes | Yes | Yes | Yes | Yes | Yes |
| Domain 2: stakeholder involvement | | | | | | | | |
| 4 | Group Membership | Yes | Yes | Yes | No | No | Yes | No |
| 5 | Target Population Preferences and Views | No | Yes | No | Yes | Yes | Yes | Yes |
| 6 | Target Users | Yes | Yes | Yes | No | Yes | Yes | No |
| Domain 3: Rigor of Development | | | | | | | | |
| 7 | Search Methods | Yes | Yes | Yes | Yes | Yes | Yes | Yes |

| AGREE Checklist Item | | Crall et al. [5] | Hurlbutt and Young [6] | Mejare et al. [7] | Do [8] | Featherstone et al. [9] | WHO [10] | Bulazaf [11] |
|---|---|---|---|---|---|---|---|---|
| 8 | Evidence Selection Criteria | Yes | Yes | Yes | Yes | Yes | Yes | Yes |
| 9 | Strengths and Limitations of the Evidence | Yes | Yes | No | No | Yes | Yes | No |
| 10 | Formulation of Recommendations | Yes | Yes | Yes | Yes | Yes | Yes | Yes |
| 11 | Consideration of Benefits and Harms | No | Yes | No | Yes | Yes | Yes | Yes |
| 12 | Link Between Recommendations and Evidence | Yes | Yes | Yes | Yes | Yes | Yes | Yes |
| 13 | External Review | No | No | No | No | No | No | No |
| 14 | Updating Procedure | Yes | Yes | Yes | Yes | No | Yes | Yes |
| Domain 4: Clarity of Presentation | | | | | | | | |
| 15 | Specific and Unambiguous Recommendations | Yes | Yes | Yes | Yes | Yes | Yes | Yes |
| 16 | Management Options | Yes | Yes | Yes | No | Yes | Yes | No |
| 17 | Identifiable Key Recommendations | Yes | Yes | Yes | Yes | Yes | Yes | Yes |
| Domain 5: Applicability | | | | | | | | |
| 18 | Facilitators and Barriers to Application | No | Yes | No | No | No | Yes | No |
| 19 | Implementation Advice/Tools | Yes | Yes | No | No | Yes | Yes | No |
| 20 | Resource Implications | No | Yes | No | No | Yes | Yes | No |
| 21 | Monitoring/Auditing Criteria | No | No | No | No | No | No | No |
| Domain 6: Editorial Independence | | | | | | | | |
| 22 | Funding Body | Yes | Yes | Yes | No | Yes | Yes | No |
| 23 | Competing Interests | Yes | Yes | Yes | Yes | No | Yes | Yes |

**Table 1.**
*Appraisal of guidelines using the AGREE checklist.*

## 5. Discussion

Twenty-four articles provided useful guidance on different aspects of caries management. Appraisal of this evidence provided support for different caries prevention and management strategies. A total of 17 such strategies are summarized in **Table 5**.

This table information can be used as summary recommendations for caries prevention and management; the evidence is updated as of June 24.

We have explained our results as below:

| | PRISMA checklist item | Goodwin et al. [12] | Moynihan et al. [13] | Weynant et al. [14] |
|---|---|---|---|---|
| Title | | | | |
| 1 | Identify the report as a systematic review | Yes | Yes | Yes |
| Abstract | | | | |
| 2 | Structured summary of objectives, methods, results, and conclusions | Yes | Yes | Yes |
| Introduction | | | | |
| 3 | Rationale | Yes | Yes | Yes |
| 4 | Objectives | Yes | Yes | Yes |
| Methods | | | | |
| 5 | Eligibility criteria | Yes | Yes | Yes |
| 6 | Information sources | Yes | Yes | Yes |
| 7 | Search strategy | Yes | Yes | Yes |
| 8 | Selection process | Yes | Yes | Yes |
| 9 | Data collection process | Yes | Yes | Yes |
| 10a | Data items | Yes | Yes | Yes |
| 10b | Define all variables | Yes | Yes | Yes |
| 11 | Study risk of bias assessment | Yes | Yes | Yes |
| 12 | Effect measures | Yes | Yes | Yes |
| 13a | Synthesis methods | Yes | Yes | Yes |
| 13b | Preparation methods | No | Yes | No |
| 13c | Methods to display results | Yes | Yes | Yes |
| 13d | Synthesis methods description | Yes | Yes | Yes |
| 13e | Heterogeneity exploration methods | No | Yes | No |
| 13f | Sensitivity analyses | No | Yes | No |
| 14 | Reporting bias assessment | Yes | Yes | Yes |
| 15 | Certainty assessment | Yes | Yes | Yes |
| Results | | | | |
| 16a | Study selection | Yes | Yes | Yes |
| 16b | Study exclusion explanation | Yes | Yes | Yes |
| 17 | Study characteristics | Yes | Yes | Yes |
| 18 | Risk of bias in studies | Yes | Yes | Yes |
| 19 | Results of individual studies | Yes | Yes | Yes |
| 20a | Results of syntheses | Yes | Yes | Yes |
| 20b | Results of statistical syntheses | Yes | Yes | Yes |
| 20c | Investigation of heterogeneity | No | Yes | No |
| 20d | Results of sensitivity analyses | No | Yes | No |
| 21 | Reporting biases | Yes | Yes | Yes |
| 22 | Certainty of evidence | Yes | Yes | Yes |

**Table 2.**
*Appraisal of SRMAs using the PRISMA checklist.*

| PRISMA-ScR checklist item | Peres et al. [15] | Campbell et al. [16] | Veneri et al. [17] | Delgado et al. [18] | Corrêa-Faria et al. [19] |
|---|---|---|---|---|---|
| Title | | | | | |
| Identify the report as a scoping review | No | Yes | Yes | Yes | Yes |
| Abstract | | | | | |
| Structured summary of objectives, methods, results, and conclusions | Yes | Yes | Yes | Yes | Yes |
| Introduction | | | | | |
| Rationale | Yes | Yes | Yes | Yes | Yes |
| Objectives | Yes | Yes | Yes | Yes | Yes |
| Methods | | | | | |
| Protocol and registration | Yes | Yes | No | No | Yes |
| Eligibility criteria | Yes | Yes | Yes | Yes | Yes |
| Information sources | Yes | Yes | Yes | Yes | Yes |
| Search strategy | Yes | Yes | Yes | Yes | Yes |
| Selection of sources of evidence | Yes | Yes | Yes | Yes | Yes |
| Data charting process | Yes | Yes | Yes | Yes | Yes |
| Data items | Yes | Yes | Yes | Yes | Yes |
| Critical appraisal of individual sources of evidence | No | No | No | No | No |
| Synthesis of results | Yes | Yes | Yes | Yes | Yes |
| Results | | | | | |
| Selection of sources of evidence | Yes | Yes | Yes | Yes | Yes |
| Characteristics of sources of evidence | Yes | Yes | Yes | Yes | Yes |
| Critical appraisal within sources of evidence | No | No | No | No | No |
| Results of individual sources of evidence | Yes | Yes | Yes | Yes | Yes |
| Synthesis of results | Yes | Yes | Yes | Yes | Yes |
| Discussion | | | | | |
| Summary of evidence | Yes | Yes | Yes | Yes | Yes |
| Limitations | Yes | Yes | Yes | Yes | Yes |
| Conclusions | Yes | Yes | Yes | Yes | Yes |
| Funding | | | | | |
| Funding sources | Yes | Yes | Yes | Yes | Yes |

**Table 3.**
*Appraisal of scoping reviews using the PRISMA- ScR checklist.*

| SANRA criteria | Brookes et al. [20] | Garrocho-Rangel et al. [21] |
|---|---|---|
| Justification of the article's importance | Yes | Yes |
| Stating concrete aims or questions | Yes | Yes |
| Literature search comprehensiveness | No | Yes |
| Referencing (appropriate citations) | Yes | Yes |
| Scientific reasoning (logical argumentation) | Yes | Yes |
| Appropriate presentation of data | No | Yes |

**Table 4.**
*Appraisal of narrative reviews using the SANRA criteria.*

## 6. Systemic fluoride

Community water fluoridation (CWF) is the process of adding up to 1.5 mg/L of fluoride to drinking water. It is practiced in 25 countries and about 75% of the US population, but only a small proportion of the UK and the European population implement it [17].

The National Health and Medical Research Council (NHMRC) recommended in 2016 that fluoride levels in drinking water in fluoridated areas be kept between 0.6 and 1.1 mg/L, while the World Health Organization (WHO) recommended in 2019 that the optimal concentration should be between 0.5 and 1.0 mg/L. According to the NHMRC guidelines, bottled water in non-fluoridated locations should include about 1.0 mg/L of fluoride [11].

The efficacy and safety of community water fluoridation were thoroughly examined by the NHMRC in 2017. According to the review, fluoridating water at the present Australian levels effectively lowers dental caries in both adults and children. To balance preventing cavities and lowering the risk of fluorosis, the U.S. Department of Health and Human Services currently also suggests a fluoride concentration of 0.7 parts per million. Although it has been criticized for failing to consider all pertinent data, it took into account different fluoride sources and individual variations in fluoride intake [11].

Water fluoridation significantly lowers dental caries and social disparities in children's oral health, according to a 2000 review [3]. Although several studies were conducted before 1975, before the widespread use of fluoride toothpaste, a 2015 Cochrane study corroborated these advantages [45]. The review was also criticized for failing to account for fluoride from other sources and for excluding pertinent studies [11].

Fluoridated milk and salt are implemented in some countries as part of school health and nutrition programs. These strategies have shown improvement in preventing dental caries, especially among children, though the evidence supporting their effectiveness is of low quality [17].

Based on the appraisal of literature, there is no universal recommendation for water fluoridation. Due to the wide geographic variation, including fluorosis belts, diverse water supplies, and varying climatic conditions, implementing water fluoridation can be challenging.

## 7. Topical fluoride- home use

### 7.1 Toothpaste

The strategy for using fluoridated home products is to maximize the duration of fluoride contact with the tooth surface through a "low-dose, high-frequency"

| Sr. No | Measures | Specifications pertaining to | Guidelines | Conclusion statements | Remarks | Recommendations-strong/ conditional/not recommended |
|---|---|---|---|---|---|---|
| 1 | Systemic fluoride | Amount in fluoridated areas | NHMRC [11, 17]. | Fluoride levels in water supply should be maintained between 0.6–1.1 mg/L. | The ideal fluoride concentration in drinking water is 0.5–1.0 mg/L [11]. | Diverse climatic conditions, lack of centralized water supplies and endemic fluorosis in certain areas make water fluoridation impractical. *Not recommended*. |
| | | Amount in non-fluoridated areas | [11, 17, 25]. | Fluoride levels in bottled water should be about 1.0 mg/L. | | If packaged water industry is made appropriate recommendation; this could be feasible. *Conditionally recommended*. |
| 2 | Topical fluoride-Home-use: Toothpaste | Starting age for recommendations. | NHMRC, WHO [10, 11, 17]. | From tooth eruption (around 6 months) to 17 months, clean children's teeth with water only to avoid fluorosis. | | *Strongly recommended*<br>Use of non-fluoride toothpastes may be *conditionally recommended* if these are certified as safe to swallow. |
| | | Amount of toothpaste on brush (in ppm). | NHMRC [11]. | • Children of 18 months to 5 years: Use toothpaste with 500–550 ppm fluoride, twice daily.<br>• Ages 6 years and above: Use toothpaste with 1000–1500 ppm fluoride, twice daily or more.<br>• High-risk individuals: Use fluoride toothpaste with 5000 ppm, as advised by a dental professional. | | *Strongly recommended*; Use of >1000 ppm fluoride toothpaste may be *conditionally recommended* if these are certified as safe to swallow or under parental supervision.<br>These could be *conditionally recommended* only if prescribed by a dental practitioner. |
| | | Size of the toothpaste on the brush head. | NHMRC [11]. | • Children of 18 months to 5 years: Use a pea-sized amount of toothpaste.<br>• People over 6 years: Use a 2 cm strip of toothpaste. | Veneri et al. [17] recommends the use of rice-size toothpaste until 3 years [1]. | Either pea-size or rice-size can be *strongly recommended*. |
| | | Spitting out toothpaste foam without rinsing. | NHMRC [11]. | • Children of 18 months to 5 years: Spit out, do not swallow or rinse.<br>• People 6 years and above: Spit out, do not swallow or rinse. | | Since the ingredients in the toothpaste may vary across brands and the safety of all (if swallowed) cannot be considered at par, this can be *conditionally recommended*. |

| Sr. No | Measures | Specifications pertaining to | Guidelines | Conclusion statements | Remarks | Recommendations-strong/ conditional/not recommended |
|---|---|---|---|---|---|---|
| | Topical fluoride-Home use: Mouth rinses | Starting age for recommendations. | NHMRC, EAPD, AAPD [11, 26, 27]. | Recommended for children over 6 years. | | An additional daily mouth rinsing with fluoride mouthwash can be *strongly/conditionally recommended* based on factors such as caries risk/ susceptibility. |
| | | Concentration of the rinses. | AAPD [26, 27]. | Generally, contain 230 ppm F/10 mL of rinse volume at 0.05% sodium F. | | |
| | | With/without the toothpaste. | NHMRC [11]. | Use fluoride mouth rinse at a different time than brushing and do not substitute it for fluoridated toothpaste. | | *Strongly recommended*. |
| | | Spitting out with rinsing. | NHMRC [11]. | After rinsing, mouth rinse should be spat out, not swallowed. | | *Strongly recommended* |
| 3 | Topical fluoride-Office use: Varnish | Recommended age. | NHMRC, AAPD [11, 26, 28, 29]. | Fluoride varnish is recommended every 3 to 6 months for high-risk individuals, including children under 10 years. | | *Strongly recommended*; the 6 monthly applications can be continued for older children based on caries risk/ susceptibility and adequacy of home measures. |
| | | Concentration of the varnish. | AAPD [26, 28, 29]. | Typically, 22,600 ppm NaF. | | *Strongly recommended*. |
| | Topical fluoride-Office use: Gels | Recommended age. | NHMRC [11]. | High-concentration fluoride gels (over 1.5 mg/g fluoride) may be used for individuals aged 10 and older at high caries risk. | | *Strongly/conditionally recommended* based on factors such as the costs and settings since the gels are cheaper and usually available in the dental institutional set-ups where children from low socioeconomic backgrounds are treated. |
| | Fluoride supplements | Tablets. | AAP [30]. | 6 months to 3 years: 0.25 mg/day<br>3 to 6 years: 0.50 mg/day Used for high-risk patients or when topical fluoride is not feasible, and only if tap water fluoride is below 0.6 mg/L. | This is not a universal recommendation. | *Not recommended*. |

| Sr. No | Measures | Specifications pertaining to | Guidelines | Conclusion statements | Remarks | Recommendations-strong/ conditional/not recommended |
|---|---|---|---|---|---|---|
| 4 | Pit and fissure sealants | | Cochrane collaboration, ADA, AAPD [31, 32]. | Resin-based sealants are recommended for permanent first molars in children over 54 months to prevent carious lesions. | Sealants are recommended for permanent molars with sound or non-cavitated surfaces in children and adolescents, compared to not using sealants. | Pit and fissure sealants for first permanent molars in children during 6–9 years of age can be *strongly recommended*; however, the use of resin sealants or glass-ionomer sealants is a choice subject to settings, costs and skills of the provider and can be recommended in public health programmes. |
| 5 | NFTRA | | Garrocho-Rangel et al. [21]. | Various topical calcium phosphate (CaP) derivatives are available for enhanced remineralization and antimicrobial effects on pediatric caries. | Further research and trials are needed to confirm their efficacy. | NFTRAs can be *conditionally recommended* in children between 2 and 6 years, where high fluoride toothpastes or mouthrinses are not recommended. |
| 6 | Diet – Breastfeeding | Duration/ frequency of breastfeeding. | WHO [15, 33]. | Initiate breastfeeding within an hour of birth, exclusively for 6 months, and continue up to 2 years or longer. | | *Breast-feeding is strongly recommended* exclusively for 6 months, up to the age of 1 year, and can be continued up to 2 years or beyond with the assessment of individual needs and risks. |
| | | | Tham et al. [33]. | Breastfeeding up to 12 months is beneficial and may prevent dental caries in early childhood. | | |
| | | Replacement of breastfeeding. | Tham et al. [33]. | Replacement of breastfeeding with infant formula should not be recommended. | | *Not recommended* as formulas carry higher risk of caries and other health conditions. |
| | | Sugar intake in children and adults. | EAPD [34]. | Recommend limiting sugary snacks and drinks to main meal times and reducing the number of times sugar is consumed throughout the day. | | Limiting sugary snacks and drinks in both frequency and quantity is *strongly recommended*. |
| | | | NIN, AAPD, IAPD [26, 28, 35, 36]. | Avoid added sugars in children's diets under age two, limit sweet-tasting foods and drinks, and keep sugar intake below 5% of total energy to reduce the risk of weight gain and dental caries. | | |

| Sr. No | Measures | Specifications pertaining to | Guidelines | Conclusion statements | Remarks | Recommendations-strong/ conditional/not recommended |
|---|---|---|---|---|---|---|
| | | | WHO [10]. | Strongly recommended a reduced intake of free sugars throughout the life course.<br>Also recommended to lower free-sugar intake <10% E to reduce the risk of dental caries. | There may be benefit in limiting sugars to <5% E to minimize the risk of dental caries [10, 35]. | Strongly recommended a reduced intake of free sugars and delaying introducing sugar to children. |
| | | | ICMR-NIN [35]. | Children under the age of 2 should not consume any added sugar to ensure healthy development. | | *Strongly recommended* to avoid exposing children to any added sugars particularly if breast feeding is continued. |
| 7 | Caries detection | | Cochrane Database on Systematic Reviews [37]. | Visual classification systems like International Caries Detection and Assessment System (ICDAS) and Ekstrand-Ricketts Kidd (ERK) show reasonable sensitivity and specificity for detecting early caries. | Among various systems of caries detection, a few such as DIAGNOdent (DD), DIAGNOdent Pen (DD Pen), and bitewing radiographs (BW) are considered. | Visual method (of dry surface and good illumination) is highly recommended. Visual examination aided with magnification, probe (non-invasive, ball-ended), transillumination, etc. can be conditionally recommended. ICDAS for the categorization and identifying a threshold for intervention is highly recommended. |
| | | | Foros et al. [38]. | • Visual examination is the primary method for detecting caries on all surfaces and dentitions.<br>• In permanent teeth, supplement with DIAGNOdent (DD) for occlusal caries and bitewing radiographs (BW) for approximal surfaces.<br>• In primary teeth, use DIAGNOdent Pen (DD Pen) to enhance exams for both occlusal and approximal lesions. | | *Diagnodent is not routinely recommended*. |

| Sr. No | Measures | Specifications pertaining to | Guidelines | Conclusion statements | Remarks | Recommendations-strong/ conditional/not recommended |
|---|---|---|---|---|---|---|
| | | Bitewing radiography - Interval for low-risk | ADA [12]. | • Low-risk caries in primary dentition: Radiographs every 12–18 months.<br>• Low-risk caries in mixed dentition: Radiographs every 24 months.<br>• Adolescents with low-risk caries: Radiographs every 24 months.<br>• Adults with low-risk caries: Radiographs every 24–36 months. | | *Conditionally recommended* for children with proper access to services and costs not a barrier. Non-invasive caries risk assessment could be a better option. |
| | | Interval for moderate-risk | SDA [12]. | • Moderate-risk caries in children: Radiographs every 12 months.<br>• Moderate-risk caries in adults: Radiographs every 12–24 months. | | |
| | | Interval for high-risk | SDA [12]. | • High-risk caries in children: Radiographs every 6–12 months.<br>• High-risk caries in adults: Radiographs every 12 months. | | |
| 8 | Behavior modification | Dental home | AAPD [26, 28, 29]. | Recommends that a a dental home should provide safe, individualized, and accessible care, including disease assessments, evidence-based treatments, preventive plans, and guidance on hygiene and diet. It should manage conditions, offer specialist referrals, and ensure a smooth transition to adult care. | | First visit to a dentist within 6 months of eruption of first tooth and no later than first birthday is *strongly recommended*. |
| 9 | Recall intervals | | EAPD [34]. | *Low-Risk Patients*: Recall every 12 months.<br>*Medium-Risk Patients*: Recall every 6 months.<br>*High-Risk Patients*: Recall every 3 months. | As per CRAFT manual [39], recall intervals are: 12 months for very low/no risk, 6–12 months for low risk, 4–6 months for moderate risk, and 3–4 months for high risk [40]. | Risk-based approach for recall and preventive measures is *strongly recommended*. |

| Sr. No | Measures | Specifications pertaining to | Guidelines | Conclusion statements | Remarks | Recommendations-strong/ conditional/not recommended |
|---|---|---|---|---|---|---|
| | *Secondary measures* | | | | | |
| 10 | Caries risk assessment and management | | AAPD [26, 28, 36]. | Use of CAMBRA-CRA tool for assessing caries risk levels (low, moderate, high, extreme) regularly. | | Use of an India-specific tool CRAFT is strongly recommended. |
| 11 | Caries activity assessment and management | | EAPD, Loveren and Helderman [34]. | For non-active lesions with indicators, improve compliance and possibly shorten recall intervals; for active non-cavitated lesions, apply fluoride varnish or sealants, and for cavitated lesions use lining material. | | Caries risk and activity-based approaches are strongly recommended particularly for primary teeth and conditionally recommended for permanent teeth based on the need for esthetics and longevity of restorations. |
| 12 | Caries classification/ threshold/ ICDAS | | Gugnani et al. [41]. | ICDAS was developed to standardize detection and calibration, as caries detection can be subjective. | | ICDAS is strongly recommended as a threshold for surveys; however, it is *conditionally recommended* for restorative caries management as other factors (type of removal technique, restorative materials, etc.) predominate. |
| 13 | SDF | Concentration | AAPD, CEDACORE [19, 23, 42]. | For preventing and arresting carious lesions. | Indications for complete caries removal include high caries risk, difficulty in control, progressing lesions, and medical or additional care needs [6]. | SDF is *conditionally recommended* as an interim measure or where other restorative treatments are not suitable and esthetics is not a concern or objection; such as in children with limited cooperation, costs a prohibition for extensive treatment, settings/expertise not suitable for better management, etc. |
| | | Frequency | AAPD [23, 42]. | Two to four times per year for high-risk patients. | | Frequency of SDF applications (one or more) is *conditionally recommended* based on the need at the time of the follow-ups. |

| Sr. No | Measures | Specifications pertaining to | Guidelines | Conclusion statements | Remarks | Recommendations-strong/ conditional/not recommended |
|---|---|---|---|---|---|---|
| 14 | Caries removal technique | Selective caries removal | Dhar et al., [43]. | *Moderate Caries*:<br>*Primary/Permanent Teeth*: Use selective or nonselective removal, or no removal (seal).<br>*Advanced Caries*:<br>*Primary/Permanent Teeth*: Prefer selective, nonselective, or stepwise removal, or no removal (seal). | | |
| | | | SDCEP [19]. | Except for specific cases involving anterior teeth, complete caries removal is avoided. | | Selective caries removal is *strongly recommended* for intracoronal restorations in posterior teeth (both primary and permanent). |
| 15 | Restoration materials | Atraumatic restorative treatment (ART) | AAPD [23, 42]. | For single-surface cavities; for large symptomatic lesions, incomplete caries removal with glass ionomer cement and review in 3–7 days. | | ART is *conditionally recommended* only for open cavitated occlusal/ cervical caries depending on factors such as settings, child cooperation, cost, and provider expertise. |
| | | | Dhar et al. [43]. | *Primary Teeth Class III*: Use nanocomposite or hybrid RC.<br>*Anterior Primary Teeth Class V*: Use GIC (conventional, hybrid, or resin-modified).<br>*Posterior Primary Teeth Class I*: Use resin-modified GIC, RC, conventional GIC, crowns, or amalgam.<br>*FDA Advisory*: Avoid amalgam in children under six or pregnant individuals.<br>*Permanent teeth*<br>*Moderate/Advanced Caries: Class I (occlusal)*: Use GIC, hybrid RC, or resin-modified GIC. | | |

| Sr. No | Measures | Specifications pertaining to | Guidelines | Conclusion statements | Remarks | Recommendations-strong/ conditional/not recommended |
|---|---|---|---|---|---|---|
| | | | | *Class III (approximal)*: Use nanocomposite or hybrid RC.<br>*Class V (cervical)*: Use conventional GIC, hybrid RC, or resin-modified GIC.<br>*Class I Pit/Fissure*: Choose conventional GIC, dental amalgam, RC, or resin-modified GIC.<br>*Posterior Teeth Class II (approximal)*: Use amalgam, RC, or resin-modified GIC.<br>*Class V (cervical) on Posterior Teeth*: Choose conventional GIC, hybrid RC, or resin-modified GIC.<br>*FDA Advisory*: Avoid amalgam in children under six or individuals with mercury hypersensitivity.<br>*Root Caries on Anterior/Posterior Teeth*: Use resin-modified GIC or conventional GIC. | | |
| 16 | Biological management/ Hall technique | Pre-formed metal crowns. | SDCEP [19]. | For the restoration of multi-surface carious lesions; for managing lesions requiring intervention but no pulpal therapy. | | *Strongly recommended* for primary molars requiring extracoronal restorations. |
| | | | Coll et al. [44]. | For teeth with caries extending more than 50% into dentin, as it has comparable success at 24 months to selective or complete caries removal with filling or crown placement. | | |
| | | Non-restorative cavity control | SDCEP [19]. | For management of caries over 1/3 into dentine in primary teeth, only if no other treatment is possible and excellent oral hygiene is maintained at home. | | *Conditionally recommended* based on risk assessment and monitoring. |

| Sr. No | Measures | Specifications pertaining to | Guidelines | Conclusion statements | Remarks | Recommendations-strong/ conditional/not recommended |
|---|---|---|---|---|---|---|
| 17 | Pulp therapies | Pulp capping | AAPD [43]. | IPT- for permanent teeth with deep caries and vital pulp capable of healing.<br>DPT- for normal pulp with small exposures. | | |
| | | Pulpotomy | AAPD [23, 42]. | In vital primary incisors with deep caries for 12-month success, with the use of calcium silicate cement for higher success. | | *Strongly recommended* for vital and conditionally recommended for teeth with (early) irreversible pulpitis; most suitable agent being Calcium Silicate-based. |
| | | Pulpectomy | Coll et al. [44]. | For nonvital teeth when long-term success (over 24 months) is desired, provided no root resorption.<br>For long-term success (over 18 months), use zinc oxide eugenol/ iodoform/calcium hydroxide or ZOE fillers. | | *Strongly recommended* for irreversible pulpitis and non-vital teeth without root resorption and of strategic importance; most suitable agent being ZOE or Endoflas. |
| | | LSTR | AAPD [23, 42]. | For nonvital teeth with root resorption when the tooth needs to be retained in the arch for 12 months or less. | Based on 12-month results, pulpectomy is preferred over lesion sterilization and tissue repair for nonvital teeth without root resorption. | *Conditionally recommended* for non-vital teeth with root resorption and of strategic importance; typically, primary second molars prior to the eruption of the first permanent molars. |

**Table 5.**
*Summary statements of included publications.*

approach. This method has been shown to be effective in reducing the prevalence of dental caries in industrialized countries [17].

From the time of tooth eruption (around 6 months) to 17 months, the NHMRC [11] and WHO [10] recommend that children's teeth be cleaned by an adult without using toothpaste to minimize the risk of fluorosis. Once children reach 18 months of age, they should use a small pea-sized amount of toothpaste containing 0.5–0.55 mg/g fluoride (500–550 ppm) for brushing twice a day. For individuals aged 6 years and older, a "2 cm" strip of toothpaste with 1–1.5 mg/g fluoride (1000–1500 ppm) should be used twice daily or more frequently. Teenagers, adults, and older adults at elevated risk of caries may use a higher fluoride toothpaste containing 5 mg/g fluoride (5000 ppm) under the guidance of a dental professional. Additionally, children from 6 years of age and older, as well as those 18 months to 5 years old, should spit out toothpaste foam without rinsing it [11, 17].

For those who do not consume fluoridated water or who are at elevated risk of developing caries, toothpaste usage guidelines may be adjusted based on advice from a dental or trained health professional. This might include more frequent use of fluoridated toothpaste, beginning toothpaste use at a younger age, or starting the use of standard toothpaste earlier. This approach may be particularly relevant for preschool children at higher risk of caries [8].

The use of non-fluoride toothpaste can be recommended up to 17 months only if it is certified as safe to swallow. The use of toothpaste containing 500–550 ppm fluoride is strongly recommended. Toothpaste with more than 1000 ppm fluoride may be conditionally recommended in the age group between 18 months and 5 years, under parental supervision and if the child has the ability to spit out. Toothpaste containing 5000 ppm fluoride may be conditionally recommended, only if prescribed by a dental practitioner. The use of either a pea-sized or rice-sized amount of toothpaste can be strongly recommended. Since the ingredients in toothpaste may vary across brands and the safety of all formulations (if swallowed) cannot be considered equal, their use can be conditionally recommended.

### 7.2 Mouthrinses

As recommended by NHMRC [11], European Academy of Pediatric Dentistry (EAPD) 2019, and the American Academy of Pediatric Dentistry (AAPD) 2018 guidelines, fluoride mouth rinses are to be used by children above 6 years old, under the supervision of an adult, due to the risk of accidental ingestion in younger children. These rinses contain 230 ppm fluoride per 10 mL of rinse volume, which corresponds to 0.05% sodium fluoride concentration. It must be used at a different time in relation to brushing time as it is not allowed to be a substitute for toothbrushing but rather to complement it. After using a fluoride mouth rinse, children should spit it out and not swallow it, to avoid excess fluoride ingestion [11, 26, 27].

Population data indicate that the use of fluoride mouth rinse increases among adolescents, suggesting it could be a beneficial supplementary fluoride source for those at higher risk of dental caries. However, it is crucial that fluoride mouth rinses do not replace regular toothbrushing with fluoridated toothpaste [8].

An additional daily mouth-rinsing with a fluoride mouthwash can be strongly or conditionally recommended in children above 6 years age, depending on factors such as caries risk and susceptibility.

## 8. Topical fluoride – office use

### 8.1 Varnish

Fluoride varnish typically contains a high concentration of fluoride, with a standard formulation of 22,600 ppm sodium fluoride (NaF). Because it adheres to the tooth surface and releases fluoride gradually over time, this high concentration is intended to have a considerable preventive impact against cavities [17].

It is a widely used topical treatment in an office-setting, especially for individuals at increased risk of developing caries. The AAPD [26, 28, 29,] and the NHMRC [11] recommend fluoride varnish for individuals who are at high risk for caries, including children under the age of ten. Current guidelines suggest that children at high risk should receive fluoride varnish applications every three to 6 months to effectively reduce their risk of caries [17, 26].

The six-monthly applications are strongly recommended from the time of eruption of the first tooth and can be continued for older children, depending on their caries risk, susceptibility, and the adequacy of home oral care measures.

### 8.2 Gels

High concentration fluoride gels are also a specialized form of topical fluoride treatment used in office settings. According to the NHMRC [11], these gels, which contain more than 1.5 mg/g fluoride ion, are recommended for individuals aged 10 years and older who are at an elevated risk of developing caries. These gels are designed to provide a concentrated dose of fluoride to help strengthen enamel and reduce the risk of dental caries [11].

High-concentration fluoride gels (over 1.5 mg/g fluoride) are strongly or conditionally recommended, depending on factors such as costs and settings, as these gels are more affordable and typically available in dental institutional setups, where children from low socioeconomic backgrounds are often treated, where varnishes are not routinely available due to higher costs.

### 8.3 Fluoride supplements

Fluoride supplements in the tablet form are advised at certain dosages depending on the age and fluoride concentration in the local water supply, according to the American Academy of Pediatrics [30].

The dose recommendations for children aged 6 months to 3 years are 0.25 mg daily, it increases to 0.50 mg daily for children ages three to 6 years. These supplements are generally reserved for high-risk patients or in situations where other topical fluoride applications are not feasible. They are specifically recommended only when the fluoride concentration in tap water is less than 0.6 mg/L.

To update Australia's Guidelines for 'Use of Fluorides,' a national workshop was held in 2019. The guidelines make an important note that fluoride supplements should not be administered in the form of drops or tablets that are chewed and/or swallowed [46].

The use of fluoride tablets is not recommended.

## 9. Pit and fissure sealants

AAPD [32] and SDCEP [19] recommends the use of pit and fissure sealants for sound occlusal surfaces and early carious lesions, because if early occlusal dentinal caries is sealed, it is unlikely to progress if the sealant remains intact. It is also suggested to examine the sealant for wear and integrity using a probe during recall visits and replace worn or non-adherent sealants and consider alternative management if the lesion has progressed. For partially erupted teeth or when the child's cooperation is insufficient, the use of glass ionomer material as a temporary sealant or restoration is suggested [26, 27, 30].

According to the Cochrane Collaboration [31], the American Dental Association (ADA) [32], and the AAPD [32], resin-based pit and fissure sealants are recommended for prevention of carious lesions on the occlusal surfaces of permanent first molars (PFM) in children above 54 months of age. The use of sealants on permanent molars with sound or non-cavitated occlusal surfaces is also advised compared to not using sealants. However, the EAPD [34] suggests that sealants should be applied to the occlusal surfaces of permanent molars as soon as they erupt to prevent caries. It also states although resin sealants are preferred for their effectiveness, they can be included in public health programs as a preventive measure [26, 27, 30].

According to the expert group convened for the AAPD Pediatric Dentistry Restorative Symposium, a low-viscosity, hydrophilic bonding layer, either as part of or beneath the sealant enhances long-term retention and effectiveness. They suggested that glass ionomer materials can serve as transitional sealants and may also be effective for longer-term use.

Pit and fissure sealants for first permanent molars in children aged 6–9 years are strongly recommended. However, the choice between resin-based sealants and glass-ionomer sealants depends on factors such as settings, costs, and the provider's skills.

## 10. NFTRA (non-fluoride topical remineralizing agents)

Different topical calcium phosphate (CaP) derivatives, such as tricalcium phosphate (TCP), casein phosphopeptide-amorphous calcium phosphate (CPP-ACP), functionalized β-tricalcium phosphate (fTPC), calcium sodium phosphosilicate, and CPP-amorphous calcium phosphate (CPP-ACFP), are commercially available in products like chewing gums, toothpastes, and drinks.

The use of CaP in conjunction with fluoride improves the potential for remineralization and exhibits an antimicrobial property against dental caries in children. However, more studies and clinical tests are needed to validate the effectiveness of these CaP derivatives in caries management [24].

Also, studies examining the effects of xylitol, chlorhexidine (CHX), and CPP-ACP vehicles, either alone or in combination with fluoride therapy, are few. Most of these studies did not demonstrate a statistically significant reduction in dental caries [32].

There is a need for further research to develop new preventive interventions, including innovative methods for delivering fluoride, and new strategies that do not rely on fluoride [8].

NFTRAs may be conditionally recommended for children aged from 2–6 years, especially when high-fluoride toothpastes or mouthrinses are not indicated.

## 11. Diet

### 11.1 Breastfeeding

According to the WHO [15], breastfeeding should be initiated within an hour of birth, with exclusive breastfeeding recommended for up to 6 months. Continued breastfeeding is advised up to 2 years and beyond. It is suggested that breastfeeding up to 12 months can be beneficial and may help prevent dental caries in early childhood. It also advises against replacing breastfeeding with infant formula, the former being caries-protective up to the age of one [15].

Dental professionals should provide personalized advice, balancing the benefits and risks of extended breastfeeding, while public guidelines should emphasize introducing complementary foods at 6 months, avoiding free sugars, and using fluoridated water and toothpaste [33].

Breastfeeding is strongly recommended exclusively for the first 6 months and up to the age of 1 year. It can be continued up to 2 years or beyond, with ongoing assessment of individual needs and risks. Formula feeding is not recommended, as it carries a higher risk of caries and other health conditions. Limiting sugary snacks and drinks, both in frequency and quantity, is strongly recommended.

### 11.2 Sugar

The ICMR-NIN [35] advises that children under the age of two should not consume any added sugar to ensure healthy development [35].

Similarly, the Brazilian Academy of Dentistry [23] recommends avoiding added sugars in children under 2 years, limiting intake to 25 g per day, and implementing educational interventions, promoting sugar-free environments, regulating food labeling, restricting advertising, increasing taxes on sugary products, and reformulating foods to reduce sugar content.

In line with these recommendations, the EAPD [34] suggests limiting sugary snacks and drinks to main meal times and reducing the frequency of sugar consumption throughout the day. The WHO [10] also strongly recommends reducing the intake of free sugars throughout life and suggests lowering free-sugar intake to less than 10% of total energy intake to reduce the risk of dental caries. For further reduction in risk, limiting sugars to less than 5% of total energy intake may be beneficial [28, 34, 35].

A reduced intake of free sugars and delaying the introduction of sugar to children are strongly recommended. It is also strongly recommended to avoid exposing children to any added sugars, particularly if breastfeeding is continued.

## 12. Caries detection

The Cochrane database on Systematic reviews [37] suggests that visual classification systems like International Caries Detection and Assessment System (ICDAS) and Ekstrand-Ricketts Kidd (ERK) show reasonable sensitivity and specificity for detecting early caries [37].

Visual examination should be the primary method for detecting caries on all surfaces and dentitions. In permanent teeth, it is suggested to supplement visual examination with DIAGNOdent (DD) for occlusal caries and bitewing radiographs (BW) for approximal surfaces, while in primary teeth, the DIAGNOdent Pen (DD Pen) can be used to enhance visual exams for both occlusal and approximal lesions [46].

The study by Jawdekar et al. [47] recommends replacing the conventional dental probe with a novel, non-invasive diagnostic tool to improve patient comfort and preserve tissues. Sharp probes are discouraged because they can cause damage to remineralizing lesions and may lead to misdiagnoses [47]. For visuotactile examinations, the use of periodontal probes with ball-ended tips is advised. Additionally, magnification and electronic devices should be explored for better caries detection and monitoring [48].

Furthermore, fiber-optic transillumination (FOTI), electrical conductivity, and laser fluorescence are frequently used to diagnose dental caries [21]. For caries detection using bitewing radiography, the recommended intervals vary based on risk levels. For individuals with low-risk caries, the American Dental Association (ADA) suggests the following intervals: children with primary dentition should have radiographs every 12–18 months, those in the mixed dentition stage should undergo radiographs every 24 months, and adolescents should be assessed every 24 months. Adults with low-risk caries should have radiographs at intervals of 24–36 months. For moderate-risk caries, the Swiss Dental Association recommends that children receive radiographs every 12 months, while adults should have them every 12–24 months, and for high-risk caries, children should undergo radiographs every 6–12 months and adults every 12 months [12].

Bitewing radiographs are conditionally recommended for children when there is proper access to dental services and costs are not a barrier. However, non-invasive caries risk assessment may be a preferable option in many cases.

## 13. Behavior modification

The AAPD [26, 28, 29] encourages parents and other care providers to help every child establish a dental home no later than 12 months of age. It also emphasizes that a dental home for pediatric patients should offer safe, culturally sensitive, and individualized care that is comprehensive, continuous, and accessible. This includes a thorough assessment for oral diseases, evidence-based care, personalized preventive programs based on caries and periodontal disease risk, and anticipatory guidance on oral hygiene, development, diet, injury prevention, and other health issues. It should also manage acute and chronic oral conditions, provide dietary counseling, and offer referrals to specialists if needed. Additionally, effective transition from pediatric to adult dental care is crucial, ensuring coordinated care [36].

According to Kannan et al. [49], Motivational Interviewing (MI), which is a patient-focused technique of behavior management, can also play an important role in improving the dental attendance in children. It is in accordance with another study by Manek et al. [50], which specifies the reduction in new carious lesions in children with early childhood caries due to MI [49, 50].

A disease-based management approach to dental caries, rather than a lesion-based approach, allows for more effective preventive recommendations by utilizing risk assessment tools. These tools serve two primary purposes: disease prediction and behavioral surveillance. Additionally, risk categorization helps determine the appropriate frequency of follow-up visits, enabling better monitoring of behavior changes related to diet, oral hygiene practices, and treatment-seeking behavior [49, 50].

It is strongly recommended that a child's first dental visit occurs within 6 months of the eruption of their first tooth and no later than their first birthday.

## 14. Recall intervals

According to the EAPD [34], recall intervals should be tailored to each patient's caries risk. For very low or no-risk patients, schedule recall visits every 12 months for routine check-ups and preventive care. Low-risk patients should have recall visits every 6–12 months, focusing on routine check-ups, preventive care, and oral hygiene reinforcement.

Moderate-risk patients require more frequent visits, ideally every 4–6 months, which should include fluoride varnish applications and dietary counseling. High-risk patients should have recall visits every 3–4 months to ensure frequent check-ups, intensive preventive and restorative care, behavior modification, and close monitoring. CRAFT [39] supports these recommendations by emphasizing the need for individualized recall intervals based on caries risk assessments. Caries risk assessment tools provide a framework for patient engagement and behavior surveillance [39].

Patient engagement and motivation are key factors for improving dental attendance and caries control. Techniques such as motivational interviewing help improve these outcomes [31].

A risk-based approach to recall visits and preventive measures is strongly recommended. The recall intervals are recommended to be three monthly for very high or high risk as against six monthly for low or very low risk.

## 15. Caries risk assessment and management

Caries risk assessment and management can be effectively conducted using the CAMBRA-CRA (Caries Management by Risk Assessment) tool, which regularly assesses caries risk levels as low, moderate, high, or extreme.

According to the AAPD [26, 28, 36], caries risk assessment, based on a child's age, social/behavioral/medical factors, protective factors, and clinical findings, should be done routinely in new and periodic exams by oral health and medical providers [36].

Caries risk assessment is essential for tailored, prevention-focused management. CRAFT (Caries risk assessment for treatment) is another valid and reliable chair-side tool that provides clinicians with a structured approach to preventive care while promoting active patient involvement in the process [41].

The use of the India-specific tool CRAFT is strongly recommended.

## 16. Caries activity assessment and management

According to the EAPD, van Loveren and van Palenstein Helderman [24], caries activity is determined by the presence or absence of signs indicating the progression of carious lesions. No caries activity is defined as the absence of new carious lesions or progression of existing ones between successive examinations. Inactive non-cavitated lesions typically appear smooth, shiny, and polished, while inactive cavitated lesions feel hard upon visuo-tactile examination [24].

EAPD also guides that indicators of caries activity include a dull, whitish appearance of the teeth, roughness, plaque accumulation, gingivitis, and a lack of parental interest in maintaining oral health. Additionally, partial eruption of teeth may also suggest caries activity. When caries activity is not present but indicators are, it is necessary to increase compliance with basic oral health programs, requiring shorter recall intervals. In cases of active caries, efforts should be directed toward improving compliance with oral health practices. If caries activity persists, fluoride varnish or pit and fissure sealants can be applied to non-cavitated active lesions. For active cavitated lesions, a lining material may be used to cover the surface, along with specific instructions for brushing the affected area [24].

It is important to note that micro-, minimal-, or invasive measures alone do not reduce caries activity.

Caries risk and activity-based approaches are strongly recommended for primary teeth and conditionally recommended for permanent teeth, depending on the need for esthetics and the longevity of restorations.

## 17. Caries classification/threshold/ICDAS

Caries detection can be subjective, leading to significant inter-examiner and intra-examiner variability. To address this and establish a threshold for examiner calibration, the ICDAS (International Caries Detection and Assessment System) was developed [41].

ICDAS is strongly recommended as a threshold recording caries in surveys but is conditionally recommended for restorative caries management, as other factors such as removal techniques and restorative materials play a more significant role in treatment decisions.

## 18. Silver diamine fluoride

Silver diamine fluoride (SDF) is a fluoride solution available at a concentration of 38% (44,800 ppm fluoride) and is used to prevent and arrest carious lesions. It is specifically recommended for use in high caries-risk children, those who are challenging to manage, individuals with actively progressing carious lesions, and patients who are medically compromised or have additional care needs [24].

Current guidelines suggest that for high-risk patients, SDF should be applied two to four times per year to maximize its preventive and arresting effects on carious lesions in at-risk individuals [24]. The main drawbacks are the discoloration of the carious surface and therefore acceptance from other children and parents [51]. A new development, the nano-silver fluoride (NSF), has shown potential for overcoming these drawbacks and claims higher efficiency. More research is underway on this agent [52].

SDF is conditionally recommended as an interim measure or when other restorative treatments are not feasible, and esthetics is not a concern. This may apply in cases where children have limited cooperation, treatment costs are prohibitive, or the setting and expertise are not suitable for more extensive management. The frequency of SDF applications (one or more) is also conditionally recommended, depending on the need during follow-up visits.

## 19. Caries removal techniques

### 19.1 Nonselective caries removal (complete caries removal)

This traditional approach involves removing both soft and firm dentin, regardless of proximity to the pulp, with the goal of removing all carious tissue and bacteria to prevent further spread. It provides a solid base for restorative materials but is associated with a higher risk of pulp exposure and is increasingly questioned for its nonconservative nature [48].

### 19.2 Selective caries removal

This method involves removing caries based on their proximity to the pulp, preserving soft or firm dentin as needed. It can be done in one or two steps. The one-step approach removes carious dentin and places a permanent restoration in a single visit, such as indirect pulp capping. The two-step method, or stepwise caries removal, involves two appointments, reducing the risk of pulp exposure but potentially leading to issues with restoration due to dentin shrinkage. Selective removal aims to maintain pulp vitality and has shown lower pulp exposure rates compared to nonselective methods [48], in agreement with the SDCEP [19], which also recommends it as the preferred technique, advising against complete caries removal except in specific circumstances, particularly for anterior teeth [48].

Selective caries removal is strongly recommended for intracoronal restorations in posterior teeth, both primary and permanent.

## 20. Restoration materials

### 20.1 ART (Atraumatic restorative technique)

Atraumatic Restorative Treatment (ART) is recommended by the AAPD [23, 42] for managing dental caries, particularly in settings where conventional restorative treatments may be challenging. It is deemed appropriate for single-surface cavities but is generally not recommended for multi-surface lesions. For large symptomatic carious lesions, this approach involves incomplete caries removal followed by the placement of a glass ionomer cement dressing, as it aims to manage the carious lesion conservatively while providing a temporary restoration. However, the patient should be reviewed for symptoms within three to seven days to assess the effectiveness of the treatment and determine if further intervention is necessary [42].

Intracoronal restorative materials supporting the selective caries removal are:

Dhar et al. [43] recommend specific restorative materials based on the cavity type and tooth location. For primary teeth, Class III lesions should use nanocomposite or hybrid resin composite, and Class V lesions on anterior teeth are best treated with glass ionomer cement (GIC). For posterior Class I lesions, suitable options include resin-modified GIC, resin composite, conventional GIC, crowns, or amalgam, although the FDA advises against amalgam in young children and pregnant individuals [43].

For permanent teeth, Class I restorations should use GIC, hybrid resin composite, or resin-modified GIC, while Class III lesions are suitable for nanocomposite or hybrid resin composite. Class V lesions can be treated with GIC or resin composites and root caries with resin-modified or conventional GIC. The FDA similarly recommends avoiding amalgam in children under six or those sensitive to mercury.

## 21. Biological methods

### 21.1 Hall technique

SDCEP [19] recommends the use of pre-formed metal crowns, specifically using the Hall Technique, for the restoration of multi-surface carious lesions. This technique is advocated for managing carious lesions that require intervention but do not necessitate pulpal therapy. It involves placing a pre-formed crown over the tooth without removing any carious tissue, which can be particularly effective in managing extensive carious lesions while preserving tooth vitality [19].

Coll et al. [44] also supports the use of this technique, especially for teeth with caries extending more than 50% into dentin. Their research indicates that the success rate of pulp vitality with Hall Technique crowns is comparable to that achieved through selective or complete caries removal followed by filling or crown placement. This makes it a viable option for managing significant carious lesions while maintaining pulp health [44].

The Hall technique is strongly recommended for primary molars requiring extracoronal restorations.

### 21.2 Non-restorative cavity control

For Non-Restorative Cavity Control (NRCC), the SDCEP [19] suggests it as a possible management strategy for carious lesions that extend more than one-third into dentin in primary teeth. This approach is recommended only when other treatment options are not feasible and provided that the child maintains excellent oral hygiene at home along with the use of fluoride treatments to halt lesion progression. NRCC involves monitoring the carious lesion and focusing on preventive care rather than immediate restorative intervention [19].

This method is guided by the lesion's shape and depth, the patient's oral hygiene habits, and esthetic needs. The cavity opening is widened for easier cleaning, enhancing the patient's ability to maintain hygiene and control plaque. Additionally, changing behaviors and educating patients about caries causes and prevention are crucial. This approach is recommended for early-stage root surface caries with shallow defects, where good oral hygiene and fluoride treatment are initially used to promote remineralization and manage caries [21].

Non-Restorative Cavity Control (NRCC) is conditionally recommended, based on risk assessment and regular monitoring.

## 22. Pulp therapies

### 22.1 Pulp capping

According to the AAPD [43], permanent teeth with deep caries, no pulpitis, or reversible pulpitis should undergo indirect pulp treatment (IPT) if the pulp is radiographically and clinically vital and has the capacity to heal from the carious insult, and the deepest carious dentin is preserved to prevent pulp exposure. Furthermore, if the pulp is normal, Direct Pulp Capping (DPT) is advised for permanent teeth with a small carious or mechanical pulp exposure [42].

### 22.2 Pulpotomy

For the management of deep carious lesions in vital primary incisors, AAPD [23, 42] recommends opting for pulpotomy rather than pulpectomy. Pulpotomy, a procedure that involves removing the affected part of the pulp and placing a medicament, is favored due to its significantly higher success rate at 12 months, for which the use of calcium silicate cement is advised as it has been shown to have the highest success rate at 24 months, ensuring better long-term outcomes [42].

According to the AAPD [43] it is also indicated for primary teeth with normal pulp or reversible pulpitis following caries removal or traumatic pulp exposure. However, it is necessary that there are no radiographic signs of infection or pathological resorption. It is further advised that after amputating the coronal pulp, the remaining radicular pulp must be vital, without signs of suppuration, necrosis, purulence, or uncontrollable hemorrhage after a few minutes of pressure with a cotton pellet.

Pulpotomy is strongly recommended for vital teeth and conditionally recommended for teeth with early irreversible pulpitis, with calcium silicate-based agents being the most suitable.

### 22.3 Pulpectomy

According to the AAPD [23, 42], in cases where the tooth is nonvital and long-term success is required, pulpectomy is the recommended treatment. It involves the complete removal of the pulp tissue and the filling of the root canals with a suitable material. For achieving long-term success (beyond 24 months), it is advised to use materials such as zinc oxide eugenol/iodoform/calcium hydroxide or ZOE fillers, as these materials have been found to perform better than iodoform fillers in maintaining the tooth's health over time [42].

Pulpectomy is strongly recommended for irreversible pulpitis and non-vital teeth without root resorption that are of strategic importance, with zinc oxide eugenol (ZOE) or Endoflas as the preferred agents.

### 22.4 Lesion sterilization and tissue repair

AAPD [23, 42] also suggests that when dealing with nonvital teeth that exhibit root resorption, Lesion Sterilization and Tissue Repair (LSTR) might be preferred over pulpectomy, especially if the tooth needs to be preserved in the arch for a period of 12 months or less. However, based on results from studies covering 12 months, pulpectomy is generally preferred over LSTR in cases where there is no root resorption, as it tends to offer more reliable outcomes for maintaining tooth function and health over time [42].

LSTR is conditionally recommended for nonvital teeth with root resorption that are of strategic importance, particularly primary second molars before the eruption of the first permanent molars.

An evidence-based approach is a dynamic process that requires continuous updating; moreover, applying evidence demands, in addition to the establishment of a standard of care, consideration of patient preferences and clinical judgment. Furthermore, recommendations can vary depending on the setting (clinical vs. community), socioeconomic status, context (urban, semi-urban, rural), and so on.

Recommendations made by us are more directed toward behavioral change; however, a few are also for environmental change. Despite the limitations of these approaches, such recommendations are valuable for day-to-day use.

Standardization of recommendations has been the mainstay of preventive care in the Western world, with examples such as the delivery of SDCEP (Scottish Dental Clinical Effectiveness Programme), AAPD (American Academy of Pediatric Dentistry), and EAPD (European Academy of Pediatric Dentistry) guidelines.

This is the first-of-its-kind attempt to appraise and synthesize evidence to formulate strategies for caries prevention and management for children.

## 23. Conclusion

Comparison of the literature led to the current evidence on the basis of which final recommendations were made (**Tables 6** and 7).

| | **No toothpaste** | **Non-fluoride toothpaste** | **Junior fluoride (approx. 500 ppm)** | **Regular toothpaste (1000–1500 ppm)** | **High fluoride (5000 ppm)** |
|---|---|---|---|---|---|
| From first tooth eruption till 17 months | YES (Only wet brushing is strongly recommended) | YES (If the toothpaste is safe to swallow, the same can be strongly recommended) | NO (Only if the child can spit out, conditionally recommended) | NO | NO |
| 18 months to 5 years | YES | YES (Only until the child can spit out) | YES (Strongly recommended) | NO (YES - if the child can spit out; in case of high-risk category and under the supervision of parents, conditionally recommended) | NO |
| 6 years and above | NO | NO | NO | YES (Strongly recommended) | NO (YES for high risk) |

**Table 6.**
*Age-appropriate recommendations for toothpastes.*

| |
|---|
| Appraisal of the literature led to the following final recommendations. |
| *Caries risk and activity*-based approaches are strongly recommended for primary teeth and conditionally for permanent teeth (such as the CRAFT approach). ICDAS is strongly recommended for caries surveys to set a threshold but conditionally for restorative management. |
| Exclusive *breastfeeding* is strongly recommended for the first 6 months and up to 1 year, with continued breastfeeding up to 2 years or beyond based on individual needs. Formula feeding is not recommended due to increased caries risk. |
| Limiting *sugary* snacks and drinks, reducing free sugar intake to less than 10% of total energy intake, and avoiding added sugars, especially during breastfeeding, are strongly advised. |
| *For home use, fluoride* toothpaste containing 500–550 ppm fluoride is strongly recommended for children, with non-fluoride toothpaste safe to swallow up to 17 months; toothpaste with 1000 ppm fluoride may be conditionally used for children 18 months to 5 years if they can spit, and higher fluoride concentrations (e.g., 5000 ppm) should only be used under dental supervision, with a pea-sized or rice-sized amount advised, while fluoride mouthwash may be recommended for children over six based on caries risk, especially in areas where toothpaste is capped at 1000 ppm fluoride. |
| *For office use*, fluoride varnish applications every 6 months are strongly recommended from the eruption of the first tooth and can continue based on caries risk and home care effectiveness, while high-concentration fluoride gels (over 1.5 mg/g fluoride) may be conditionally recommended over 10 years of age in settings with cost constraints, but fluoride tablets are not recommended. |
| *Non-fluoride topical remineralizing agent (NFTRAs*) can be conditionally recommended for children aged 2–6 years, particularly when high-fluoride toothpastes or mouthwashes are not advisable. |
| *Caries detection* with the help of visual examination should be the primary method for detecting caries on all surfaces and dentitions, with DIAGNOdent (DD) and bitewing radiographs (BW) supplementing visual exams for occlusal and approximal caries in permanent teeth, and the DIAGNOdent Pen (DD Pen) enhancing exams for both occlusal and approximal lesions in primary teeth. |
| *ICDAS* is strongly recommended for caries recording in surveys but conditionally recommended for restorative caries management, as other factors like removal techniques and restorative materials are more significant in treatment decisions. |
| *Silver Diamine Fluoride (SDF)* is conditionally recommended as an interim measure or when restorative treatments are not feasible and esthetics are not a concern, particularly in cases with limited cooperation, high costs, or inadequate settings, with the frequency of applications based on follow-up needs. |
| *Pit and fissure sealants* for first permanent molars in children aged 6–9 years are strongly recommended, with the choice between resin-based and glass-ionomer sealants depending on factors such as setting, cost, and provider expertise. |
| Selective *caries removal* is strongly recommended for intracoronal restorations in both primary and permanent posterior teeth, while no caries removal is strongly recommended for primary molars requiring extracoronal restorations using the Hall technique. Non-Restorative Cavity Control (NRCC) is conditionally recommended, based on risk assessment and regular monitoring. |
| Indirect pulp treatment is recommended for deep caries with reversible pulpitis and vital pulp, while direct pulp capping is recommended for small carious or mechanical pulp exposures in permanent teeth with normal pulp. *Pulpotomy* is strongly recommended for vital primary teeth and conditionally for early irreversible pulpitis with calcium silicate-based agents, while *pulpectomy* is recommended for irreversible pulpitis and nonvital teeth without root resorption using ZOE or Endoflas, and *LSTR* is conditionally recommended for non-vital teeth with root resorption, especially primary second molars before the eruption of permanent molars. |
| The *recall* intervals are recommended to be 3 monthly for very high or high risk as against 6 monthly for low or very low risk. |

**Table 7.**
*Summary of conclusions from caries management recommendations.*

## Conflicts of interest

The authors declare no conflict of interests.

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Chapter 4

# Early Childhood Caries: Comprehensive Action for the Quality of Life of Children

*Renie Kumala Dewi and Zakiah Husada Noor*

## Abstract

Early caries is often spotted in early childhood and is known as Early Childhood Caries (ECC). In children under 3 years of age, signs of fine surface caries are indicative of Severe Early Childhood Caries (S-ECC). Some of the prevention of early childhood caries include the Readiness Assessment of Parents concerning Infant Dental Decay (RAPIDD) scale, Community-Based Education, prevention of transmission of cariogenic bacteria, Home Professional and Home Prevention Approach, Barriers in Early Childhood Caries, and operative dentistry. Detecting caries at the practicing stage is essential to prevent significant tooth decay through some methods, such as Caries Risk Assessment, Digital Imaging Fiberoptic Translumination, DIAGNOcam, and other diagnostic aids in dental caries. ECC treatment must lead to essential results in the restoration of proper dental shape, function, and esthetics, and must be requisite with the physiological integrity of the teeth in a harmonious relationship through operative dentistry, which is a dynamic combination of ever-evolving ingredients and new techniques.

**Keywords:** DIAGNOcam, fiber optic, prevention, risk factor, white spot lesion

## 1. Introduction

Caries in infants and young children have long been known as a clinical syndrome, described as early as the middle of the last century. In 1962, Dr. Elias Fass published the first comprehensive description of caries in infants, which he referred to as *nursing bottle caries*. The first sentence of his paper begins with "Nothing is more surprising to a dentist than an examination of a pediatric patient suffering from rampant caries," and this is especially the thought we get when observing a child with *nursing caries*. Since its first explanation in 1962, the term *nursing bottle mouth* has been replaced by many names, but it is only recently that the original concept has been rethought. In 1994, a conference at the Centers for Disease Control and Prevention recommended the use of less specific terms, such as Early Childhood Caries (ECC), because there was a consensus among participants that the relationship between the habit of using bottles and caries was not absolute. Children who are exposed to sugary drinks early are more likely to develop Severe Early Childhood Caries (S-ECC). However, this term does not negate the basic reasons for dental demineralization in very young children, including excessive exposure to cariogenic foods and early infection with cariogenic bacteria [1, 2].

Prevention and treatment of ECC should be started in the prenatal period. Treating these severely damaged teeth is still a challenge for dentists. Toddlers and preschoolers are usually very uncooperative in dental care, so many poor oral hygiene practices are found that cause swollen, inflamed, and bleeding gingiva. Recommended treatment plans for severely damaged anterior and posterior primary teeth include restoration, crowning, or extraction, followed by the installation of a space maintainer. Treatment for dental conditions that experience ECC to S-ECC is recommended to carry out dental restoration to maintain the remaining tooth structure and pulp to prevent pain, maintain function, restore esthetics (if possible), facilitate oral cavity hygiene treatment, and maintain the length and space of the jaw arch for permanent tooth growth. Parents should be instructed and assured that they bear some responsibility for the success of treatment [1, 2].

## 2. Early childhood caries (ECC)

### 2.1 Background of ECC

The definition of ECC, according to the American Academy of Pediatric Dentistry (AAPD), is a condition in which one or more primary teeth have been decayed, missing teeth due to caries, and tooth surfaces in children less than ≤71 months old who have been restored [3]. All signs of damage to the smooth surface of a child's teeth under the age of 3 are predicted to indicate severe early childhood caries. The severity of ECC is classified into three types, based on clinical signs, causes, and age of the child, namely Type I (mild), Type II (moderate), and Type III (severe). The severity of caries in early childhood was lower in children who consumed unbottled milk than in children who consumed bottled milk [4]. ECC is used to replace terms such as nursing caries, bottle caries, baby bottle tooth decay, rampant caries, or night bottle mouth. The term ECC is used because it has a broad meaning, not only due to bottled milk consumption, but also as a multifactor disease caused by the interaction between microorganisms, prolonged exposure to carbohydrates (substrates), feeding foods that contain high carbohydrates, and various other factors [3]. Nursing caries are the most common cause of damage to the ECC, which has a distinctive pattern, namely regarding the four upper incisal teeth, while the four lower incisal teeth usually remain healthy because they are covered by the position of the tongue during breastfeeding and undergo self-cleansing by saliva from the submandibular gland. Primary molar teeth, canines, and second molars can be involved when tooth decay persists. The formation of dental caries is an active pathological process that requires three main components, namely cariogenic bacteria, fermentable carbohydrates, and a susceptible tooth/host surface [4].

Early childhood caries is associated with prolonged improper breastfeeding or bottle-feeding. Prolonged bottle use until a child falls asleep is believed to be associated with an increased risk of caries, but this may not be the only factor in the development of caries in early childhood. Risk factors for ECC include [5]: (1) ECC microbial factors, such as *Streptococcus mutans and Streptococcus sobrinus;* (2) Substrate (fermented carbohydrates; (3) Hosts are teeth, saliva, and immunological factors; (4) Dental plaque; and (5) Time. The estimated time for caries to develop into a cavity varies, ranging from 6 to 48 months.

### 2.2 ECC classification

See **Table 1**.

| Type | Clinical Description | Clinical Image |
|---|---|---|
| Type I | Light to moderate |  |
| | The presence of isolated caries lesions involving molars and incisors | |
| | The number of carious teeth increases as cariogenic challenges continue | |
| | The cause is usually a combination of cariogenic semi-dense foods and a lack of oral hygiene | |
| | Seen at 2–5 years of age | |
| Type II | Moderate to severe |  |
| | Labiolingual caries lesions regarding the incisors of the maxilla | |
| | Mandibular incisors are not affected | |
| | Bottled use or breastfeeding at will or a combination of both, with or without poor oral hygiene | |
| | Visible immediately after tooth eruption | |
| Type III | Severe |  |
| | Caries lesions affect all teeth, including lower incisors | |
| | The cause is cariogenic food and poor oral hygiene | |
| | The condition is rampant | |

**Table 1.**
*ECC classification by Wayne H [1].*

## 3. Early childhood caries identification

### 3.1 Caries risk assessment

Caries risk assessment might be defined as a procedure to forecast the future development of caries before the onset of the disease clinically. The American Academy of Pediatric Dentistry (AAPD) recognizes that caries risk assessment is an essential element in contemporary clinical care for infants, children, and adolescents. The caries risk assessment according to AAPD is divided into two types:

1. Form for children aged 0–5 years old

2. Forms for children 6 years old or older.

Risks will be categorized into low-risk, medium-risk, and high-risk (**Figure 1**).

The treatment of caries can be based on biological principles, rather than coincidence or belief. Therefore, this procedure is recommended for anyone seeking to treat dental caries, not only through restorative measures such as fillings, but also through preventive and rehabilitative actions aimed at restoring function, preserving aesthetics, and improving the child's oral health [2, 6, 7]:

### *3.1.1 Microbial tests for mutant streptococci detection*

#### *3.1.1.1 Laboratory method*

Saliva samples or dental plaque were tested to spot the number of colonies of *Streptococcus mutans* bacteria through the media of mitis-salivarius-bacitracin (MSB) per mL of saliva.

**Table 1. Caries-risk Assessment Form for 0-5 Years Old[24]**

| Factors | High risk | Moderate risk | Low risk |
|---|---|---|---|
| ***Risk factors, social/biological*** | | | |
| Mother/primary caregiver has active dental caries | Yes | | |
| Parent/caregiver has life-time of poverty, low health literacy | Yes | | |
| Child has frequent exposure (>3 times/day) between-meal sugar-containing snacks or beverages per day | Yes | | |
| Child uses bottle or non-spill cup containing natural or added sugar frequently, between meals and/or at bedtime | Yes | | |
| Child is a recent immigrant | | Yes | |
| Child has special health care needs | | Yes | |
| ***Protective factors*** | | | |
| Child receives optimally-fluoridated drinking water or fluoride supplements | | | Yes |
| Child has teeth brushed daily with fluoridated toothpaste | | | Yes |
| Child receives topical fluoride from health professional | | | Yes |
| Child has dental home/regular dental care | | | Yes |
| ***Clinical findings*** | | | |
| Child has non-cavitated (incipient/white spot) caries or enamel defects | Yes | | |
| Child has visible cavities or fillings or missing teeth due to caries | Yes | | |
| Child has visible plaque on teeth | Yes | | |

Circling those conditions that apply to a specific patient helps the practitioner and parent understand the factors that contribute to or protect from caries. Risk assessment categorization of low, moderate, or high is based on preponderance of factors for the individual. However, clinical judgment may justify the use of one factor (e.g., frequent exposure to sugar-containing snacks or beverages, more than one decayed missing filled surfaces [dmfs]) in determining overall risk.

**Overall assessment of the child's dental caries risk: High ☐ Moderate ☐ Low ☐**

**Table 2. Caries-risk Assessment Form for ≥6 Years Old[25]**
**(For Dental Providers)**

| Factors | High risk | Moderate risk | Low risk |
|---|---|---|---|
| ***Risk factors, social/biological*** | | | |
| Patient has life-time of poverty, low health literacy | Yes | | |
| Patient has frequent exposure (>3 times/day) between-meal sugar-containing snacks or beverages per day | Yes | | |
| Child is a recent immigrant | | Yes | |
| Patient has special health care needs | | Yes | |
| ***Protective factors*** | | | |
| Patient receives optimally-fluoridated drinking water | | | Yes |
| Patient brushes teeth daily with fluoridated toothpaste | | | Yes |
| Patient receives topical fluoride from health professional | | | Yes |
| Patient has dental home/regular dental care | | | Yes |
| ***Clinical findings*** | | | |
| Patient has ≥1 interproximal caries lesions | Yes | | |
| Patient has active non-cavitated (white spot) caries lesions or enamel defects | Yes | | |
| Patient has low salivary flow | Yes | | |
| Patient has defective restorations | | Yes | |
| Patient wears an intraoral appliance | | Yes | |

Circling those conditions that apply to a specific patient helps the practitioner and patient/parent understand the factors that contribute to or protect from caries. Risk assessment categorization of low, moderate, or high is based on preponderance of factors for the individual. However, clinical judgment may justify the use of one factor (e.g., interproximal lesions, low salivary flow) in determining overall risk.

**Overall assessment of the dental caries risk: High ☐ Moderate ☐ Low ☐**

**Figure 1.**
*Caries risk assessment form according to AAPD [6].*

#### *3.1.1.2 Chair-side method*

The SM-Strip Mutans Dentocult Kit for estimating mutant streptococci in saliva contains test strips, bacitracin discs, test tubes with broth, paraffin for chewing, and standard charts for evaluating mutant levels after incubation. The level of streptococcal mutants is given as a "class" after comparing it with the graph, which shows low ("0") to high ("3") numbers, equivalent to 106 CFU mutants per mL of saliva. Colonies of mutant streptococci will appear on the strip as small blue dots, but the color can vary from dark blue to pale blue.

#### *3.1.1.3 Survey methods*

A sample of dental plaque is taken from one-third of the gingiva in the buccal opening of the tooth, which is placed in a Ringer solution through a toothpick, then beaten until homogeneous, placed on selective agar plates, and incubated at 37°C for 72 hours. The results about the presence or absence of *Streptococcus mutans* can be determined [1].

### *3.1.2 Saliva buffer capacity test*

The buffer capacity of saliva is significantly essential in maintaining the pH level in saliva and plaque, which counteracts mineral dissolution, but the buffer capacity of the overall stimulated saliva is weakly correlated with increased caries. There is a tendency for an inverse relationship between saliva buffering capacity and caries activity. The saliva of individuals with numerous carious lesions often shows a higher acid buffering capacity than the saliva of people who are relatively caries-free. However, this test does not sufficiently correlate with caries activity [1, 7].

### *3.1.3 Cariogram*

Computer programs could display graphic images that are able to illustrate the possible scenarios of caries risk as a whole. This program contains algorithms from data that has been input, especially biological factors. This reveals the extent to which different caries etiological factors affect caries risk. Cariograms identify risk factors for caries in individuals and provide examples of prevention and treatment strategies to dentists. The computerized version of the cariogram presents a graphical picture that illustrates the possible risk scenario of caries as a whole. A cariogram shows whether the patient is overall at high, moderate, or low risk of developing caries. It also indicates, for each individual examined, which etiological factors are thought to be responsible for the risk of caries. The results also show that targeted actions to improve the situation will have the best impact. Cariograms only show the risk of caries [1, 7].

The cariogram has five sectors in five color groups of factors related to carie-causing factors, including [7]:

1. Green sector: A real opportunity to avoid new caries.

2. Dark blue sector: A diet that is based on a combination of diet content and diet frequency.

3. Red sector: A combination of plaque and *Streptococcus mutans* amounts.

4. Light blue sector: Susceptibility, which is based on a combination of the fluoride program, saliva secretion, and saliva buffer capacity.

5. Yellow sector: A state based on a combination of past caries experiences and related diseases (**Figure 2**).

### 3.2 Digital imaging fiberoptic transillumination

Similar to Fiberoptic Transillumination (FOTI), this device has been used for caries detection in dentistry for many years. Repeated repairs have been made in such a way that the digital imaging fiberoptic translumination can be utilized to detect caries on the occlusal and proximal surfaces of the teeth, such as manual examination instruments, where the clinically observed condition is captured using a Digital Charge-Coupled Device (CCD) camera and sent to a computer for analysis with an algorithm. These images which have a sample obtained are then sent to a computer to be analyzed with a specific algorithm. This algorithm was developed to facilitate the location of caries and diagnosis of caries lesions and provide quantitative characterization to detect lesions (**Figure 3**) [1, 7].

### 3.3 DIAGNOcam

Near-infrared transillumination, particularly the DIAGNOcam, is a digital dental diagnostic tool to detect early enamel demineralization and the presence or absence of caries, which can then determine the diagnosis and subsequent treatment plan for restoration or not. DIAGNOcam is a new laser diode for recording images and displaying them directly on the computer layer, penetrating its laser beam at 780 nm to detect the earliest stage of enamel demineralization to perform "micro-minimally line-minimally." Demineralized teeth are shown as dark shadows. The images displayed on the computer layer can be recorded and stored, allowing for the determination of early demineralization without the use of radiography, thus significantly simplifying patient monitoring and communication [1, 7].

This method can prevent excessive removal of dental tissue and accurately diagnose the depth of caries, thereby reducing the risk of secondary caries after restoration. Compared to other methods, DIAGNOcam is more effective and convenient

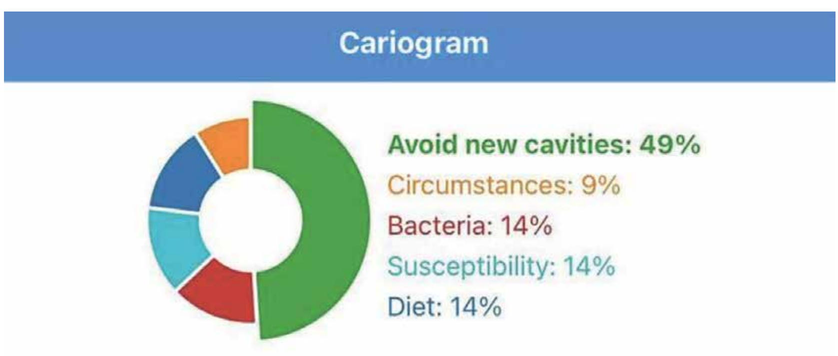


**Figure 2.**
*Cariogram analysis results.*

**Figure 3.**
*Digital imaging fiberoptic transillumination [1].*

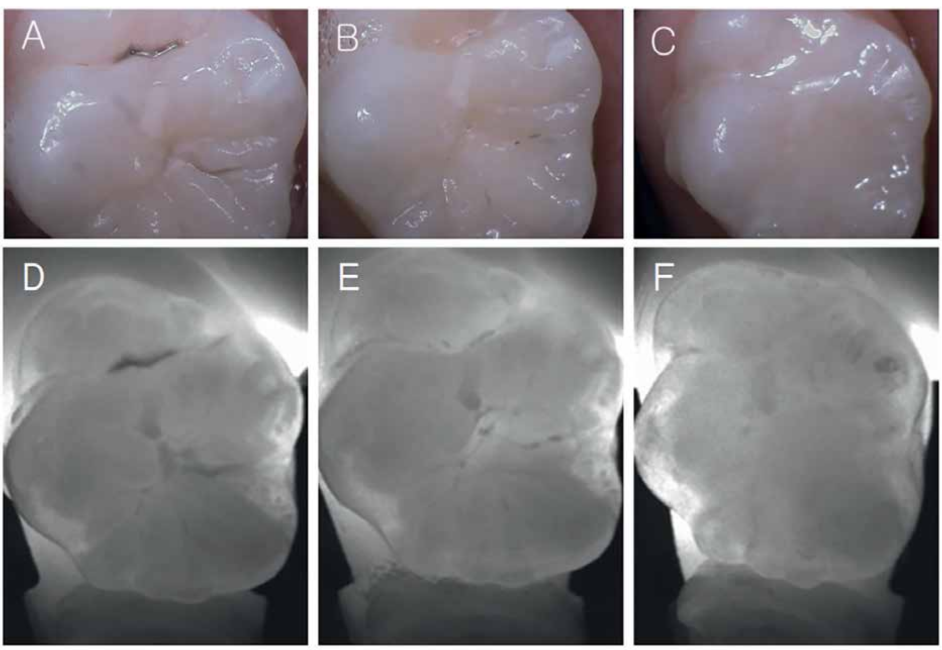


**Figure 4.**
*Picture results with DIAGNOcam: (a) Clinical before treatment, (b) Clinical during treatment, and (c) Clinical after treatment of dental caries of the maxillary molar. DIAGNOcam image of (d) Before treatment, (e) During treatment, and (f) After treatment of maxillary molar dental caries [8].*

**Figure 5.**
*Fluorescence image scoring [1].*

and can be applied in real time. However, a lot of research is still needed before DIAGNOcam can replace other methods (**Figure 4**) [6, 8].

### 3.4 Fluorescence image scoring

The device is an intraoral camera consisting of six blue light-emitting diodes (LEDs) that emit a 405-nm light sensor, a Charge-Couple Device (CCD), and DBSWIN software for analysis. This camera is able to digitize the video signal from the tooth surface during fluorescence emission using a CCD sensor. On the layer, areas of different colors appear; green appears as normal tissue, and red appears as caries tissue. This device has the advantage of making data storage, so it motivates patients for the next dental treatment (**Figure 5**) [1].

## 4. Comprehensive new treatment of early childhood caries (ECC)

### 4.1 Prevention and treatment of early childhood caries (ECC)

Early screening for signs of caries development, starting from the first year of life, can identify toddlers and infants who are at risk for *Early Childhood Caries* (ECC) and can help provide information to parents on how to improve oral health and prevent tooth decay. Prevention of ECC also requires addressing the social and economic factors faced by many families with endemic ECC. Educating mothers or caregivers to improve healthy eating habits in babies has become the main strategy used to prevent the occurrence of ECC. Three common approaches have been used to prevent ECC. The first is a community-based strategy by relying on education for mothers in the hope of influencing the eating habits of mothers and their babies; the second approach is based on the provision of screening services and preventive care at dental clinics; and the third approach involves the development of proper eating habits and self-care at home [1, 9].

#### *4.1.1 Readiness assessment of parents concerning infant dental decay (RAPIDD) scale*

The Readiness Assessment of Parents concerning Infant Dental Decay (RAPIDD) scale was developed to assess the stages of parental behavioral change, including precontemplation, contemplation, and action, about their child's oral health. The RAPIDD instrument is a tool used to determine the stage of change parents are in regarding their child's oral health. Parents in the econtemplative stage tend to exhibit low openness and low health scores, whereas those in the action stage tend to show high scores [1].

This instrument is based on the work of Prochaska and DiClemente to measure the pros and cons of parents being open about their children's dental care. Parents in the precontemplative stage exhibit low openness and low health scores, whereas those in the action stage display high scores [1]. The RAPIDD scale comprises 38 response items, each with a five-point scale ranging from "strongly agree" to "strongly disagree." The primary patient or nurse is instructed to select one of the options from five categories after the interviewer reads the questions in the language they use. Each of the 38 items is placed into one of four constructions [1].

1. Disclosure of health information

2. Valuing dental health

3. Convenience and difficulty of change

4. The child's permissive attitude. To categorize respondents as individuals who perform precontemplators, contemplators, or individual takers, responses to the questions in each construct are summed up, the scaled-down values are ranked, and the presentation is calculated for each individual in each construct.

The RAPIDD instrument is a tool used to determine the stages of change in parents regarding their children's dental and oral health. Once a particular stage of change has been established, the counselor will then determine the best approach to proceed to the next stage. Based on the transtheoretical/stages of change model (TTM), this tool is planned to measure the "readiness for change" of parents/caregivers. The Transtheoretical Model, also known as the Stages of Change Model (TTM) is a framework that explains how individuals modify their behavior. This model comprises several stages through which a person progresses during the change process, ranging from having no intention to change to maintaining a new behavior. In this context, it is helpful to understand how parents or caregivers develop readiness and establish consistent daily habits to support their children's oral health. Once a particular stage of change is established, the counselor then determines the best approach to move on to the next stage. RAPIDD's challenge is to assess whether the parent/caregiver is at one of the following stages: contemplative, preparing for action, or action concerning their child's oral health care [1, 10].

### *4.1.2 Community-based education*

In 2002, WHO and the United Nations Children's Fund (UNICEF) developed a global strategy on community-based interventions through healthy feeding and identification of dental and oral diseases in infants and children. The results of the intervention showed a reduction in the occurrence of caries and the experience of mouth pain in children aged 3–6 years, so this Community-Based Education program is highly recommended by the WHO and UNICEF [11].

Some of the Community-Based Education activities include conducting discussion sessions between parents and dentists, which are carried out in groups in the community; conducting counseling on dental and oral health to the community by dentists; hospitals or health facilities having programs to improve the oral health of pregnant women; and the important role of the center in maternal and infant oral health by carrying out several treatments in each session, such as during pregnancy at 1, 6, 12, and 18 months after childbirth. These program activities educate mothers about nutritional problems and oral health behaviors through promotive actions such as using posters, pamphlets, dental models, toothbrushes, and finger brushes. Social networking applications, such as Telegram, WhatsApp, Instagram, and Facebook, through oral health educational content in the form of videos, audio, and text messages [11, 12].

This community-based program has unique advantages and limitations. One of the important advantages is that families and communities are not only beneficiaries of the intervention but also become a resource to shape the intervention. The use of trained health workers from the community allows the implementation of health care close to where mothers, caregivers, and other children live, thereby improving the quality of life of children. Community-based programs can be delivered through a variety of weekly interactive sessions conducted by professional oral health care workers with a focus on the following three parameters: (a) Tooth brushing technique: Proper brushing technique with fluoride toothpaste appropriate for age; (b) Dietary Advice: Education on limiting foods and drinks high in sugar; (c) Oral hygiene habits: Strengthening the habit of brushing teeth and using daily dental floss is emphasized. The goal of education is to increase maternal knowledge about ECC and improve infant and maternal nutritional dietary habits. It is assumed that the increase in knowledge of the mother or caregiver will affect their self-care habits and dietary habits and will turn into the habit of improving the baby's diet and *oral hygiene* to prevent ECC [11, 12].

A decade ago, a study aimed to reduce early childhood caries (ECC) by 50% over five years in American Indian and Alaska Native communities through three levels of intervention intensity. High-intensity locations involve training community coordinators and parent volunteers to directly manage educational programs, medium-intensity locations include only training sessions for coordinators, and low-intensity locations only receive educational materials by mail without training. The program addresses feeding issues related to ECC, including the use of bottles and a lack of knowledge about ECC. The program features one-on-one counseling with the mother or caregiver. After three years, the prevalence of ECC decreased to 33% in high-intensity locations, 18% in medium-intensity locations, and 27% in low-intensity locations [1].

### *4.1.3 Prevention of transmission of cariogenic bacteria*

Dental caries is an infectious disease caused by cariogenic bacteria, including *Streptococci mutans*, the main cariogenic pathogen, along with lactobacilli that help in the development of caries. The acquisition of bacteria in the oral cavity occurs during the early years of childhood and has been linked to the occurrence of early childhood caries. Identifying the source of the cause and possible routes of transmission of the microorganisms that cause caries is also a strategy to prevent the occurrence of dental caries in children. Various studies have addressed the transmission of karyogenic organisms, with mother-to-child transmission being widely accepted as the primary route. However, some genotypes that are not related to the maternal genetic profile have been identified, thus indicating other possible sources of transmission (e.g., intrafamilial, such as father-to-child, sibling-to-child, caregiver-to-child, and extrafamilial, such as nurse and schoolmate) [13].

Transmission of karyogenic bacteria can be transmitted through vertical transmission, i.e., the occurrence of different generations (such as mother, father, caregiver-to-child), while horizontal transmission usually occurs between the same generation (such as siblings or classmates). There is evidence that cariogenic bacteria are transmitted from mothers to their babies. The genotype of streptococcal mutants in infants appeared to be identical to the maternal genotype in 71% of mother-infant pairs. A non-randomized study divided mothers who had at least 106 *Streptococci mutans* per mm of saliva into a test and control group. The test program includes the provision of dental education, oral hygiene instruction, dental care, dental cleaning, the use of 2% sodium fluoride, and fluoride varnish. The program begins when children are 3–8 months old and continues until they reach the age of 3. After reexamination, it was found that the children whose mothers were in the experimental group had a DMF-T of 5.2, which was much lower than the DMF-T of the control group, which was 8.6 [1, 13].

#### *4.1.4 Professional and home-based preventive approaches*

Some professionally applied and home-based approaches that can be used in ECC prevention are listed based on risk status. Professional care for early childhood caries ranges from dietary counseling to patient prosthodontic rehabilitation. Restoration was carried out with GIC, composite, and composite. Endodontic therapy was carried out according to the indications, followed by the installation of a crown, and severely damaged teeth were removed, followed by the installation of a space maintainer (**Figure 6**). The use of fluoride is carried out according to the fluoride content in the water (**Table 2**) [1].

Fluoride is beneficial in slowing down the demineralization process and increasing remineralization in teeth with the aim of prevention. Topical fluoride in toothpaste and fluoride drinking water can reduce dental caries by 29–51% in children and adults. However, fluoride supplements are not recommended for children with a low risk of caries under the age of 3 years. For children at high risk, fluoride tablets are recommended after 6 months of age. In all cases, before prescribing, it is important to evaluate risk factors for caries, make sure the child does not drink water that already contains fluoride or has taken fluorine supplements (in vitamins), adjust the dosage at each consultation under the supervision of the pediatrician, and evaluate other possible sources systemically (total daily consumption should not exceed 0.05–0.07 mg F−/kg). The success of fluoride therapy depends on parental motivation and participation, regular monitoring, and dose adjustment according to the dosage schedule. If used in excessive doses, it can cause dental fluorosis, which will appear as a pathological change in the teeth. The initial manifestation of dental fluorosis is the occurrence of increased enamel porosity along the Retzius fibers. If

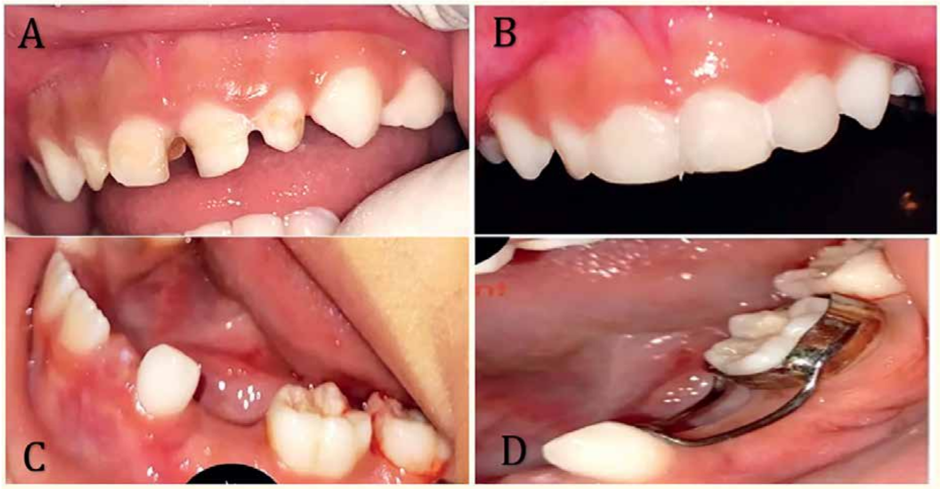


**Figure 6.**
*(A and B) Rehabilitation of the anterior teeth using compomer restoration; (C and D) Preventive orthodontics with a space maintainer.*

| Age (years) | Fluoride levels in water | | |
|---|---|---|---|
| | <0.3 | 0.3–0.7 | >0.7 |
| 0–2 | 0.25 | 0.00 | 0.00 |
| 2–3 | 0.50 | 0.25 | 0.00 |
| 3–16 | 1.00 | 0.50 | 0.00 |

**Table 2.**
*Recommended fluoride supplemental dosage schedule (mg F/day) [1].*

fluoride exposure occurs during tooth formation, porosity will appear on the enamel surface of the tooth. This porosity is formed due to the hypomineralization of enamel. Hypomineralization of enamel will increase as fluoride exposure increases during tooth development. According to the American Dental Association, fluoride in the form of varnish, which dentists should apply, is highly recommended for preventing and controlling early dental caries in high-risk children. Based on research carried out, it is known that the preventive effect is more substantial when fluoride varnish is applied before caries is formed [7, 14, 15].

### *4.1.5 Barriers in early childhood caries*

Any proposal to improve children's social, mental, and physical health will not succeed without adequate funding, political leadership, and support. Some potential barriers to providing optimal care for children are:

1. Lack of involvement and commitment from dental health organizations and others.

2. The dental community lacks a shared vision of what the problem is, how to prevent it, and who is responsible for planning and implementation.

3. There is no unified plan to resolve the social, economic, and nutritional problems faced by people in low socioeconomic groups.

4. There is no direct support for ECC epidemiology, etiology, and prevention research.

5. Dental health is not a priority for most insurance programs and plans [1].

#### *4.1.6 The role of parents*

Good oral health during pregnancy is achieved through eating habits, and it may reduce the rate of caries in children. Every pregnant mother should be introduced to the habit of cleaning her mouth. Prevention that can be done during pregnancy can be started with easy things, namely with daily treatments to prevent cavities and diseases of the gingiva, by brushing your teeth regularly twice a day using toothpaste containing fluoride and using dental floss, which aims to remove plaque, food residues, and drinks that contain sugar [15, 16].

Education about oral health is also essential to be given to mothers. The most important action to prevent ECC is to break the habit of consuming cariogenic foods, as several studies have shown that 78% of parents with children who have ECC give their children drinks that are cariogenic, such as using a bottle of juice or formula while the child is sleeping. To prevent the occurrence of ECC, parents should be encouraged to adopt healthy eating habits and instruct and supervise their children when brushing their teeth. Mothers should instruct their children on the “lift the lip” technique for spots in the form of white lesions, which can be an early sign of dental caries. Teeth that have recently erupted should be treated with fluoride agents, and if necessary, antimicrobial agents containing chlorhexidine and thymol can also be used. Based on several studies that have been carried out, it has been proven that fluoride can be used to reduce the occurrence of caries because fluoride is one of the ions that play a role in the mineralization process. Fluoride can be added to toothpaste, and it is a method that is often used to control caries while at home [13, 15, 16].

#### *4.1.7 Operative dentistry*

Treatment of ECC depends on the speed of disease progression, the age of the child, and the extent of the disease. The ideal treatment can be carried out for the child in the first year. Children at moderate risk need restoration of caries lesions and also *white spots*. As for high-risk children, immediate restoration and preventive measures to inhibit the development of caries aim to reduce the development of caries [15].

Operative dentistry is the art and science of diagnosis, treatment, and prognosis of tooth decay that does not require a thorough restoration for repair. The treatment should result in a restoration of shape, proper dental function, and esthetics while maintaining the physiological integrity of the teeth in a harmonious relationship with the surrounding hard and soft tissues, all of which should improve the general health and well-being of the patient. Pediatric operative dentistry is a dynamic combination of ever-evolving ingredients and new techniques**.** In 1924, G.V. Black outlined some steps for the preparation of permanent teeth that are ready to receive restoration. The same measures have been adopted, although slightly modified, for the restoration of primary teeth [1, 7].

The goal of pediatric operative dentistry is to keep the teeth in the arch in a healthy state so as to prevent loss and the development of further problems. It is important to understand the structural differences between primary teeth and permanent teeth before planning any procedure. The characteristics of primary teeth are that the crown is shorter because the total height of the clinical crown of the primary tooth is smaller, and therefore, the depth of the cavity should also be smaller. The horns of the mesial pulp of the tooth extend higher to the occlusal than the permanent teeth, so that the risk of accidental exposure increases during cavity preparation. The occlusal width of primary molars is very narrow as compared to permanent molars; therefore, the width of the cavity should also be smaller in primary teeth. A narrowed cervical section of the primary tooth is characterized by a more prominent cervical protrusion on the buccal aspect and a steep cervical narrowing of the cervix. There is a risk of opening the pulp at the location of the narrowing in the cervical area. The enamel and dentin layers are thinner so that caries can easily penetrate the pulp. The preparation of cavities must be done carefully because there is an increased risk of opening of the pulp. The enamel rod extends slightly occlusal from the Dentino-Enamel Junction at the gingival third. Hence, an enamel tilt at the angle of the cervico-gingival surface line is not necessary because there is no unsupported enamel rod. The contact area is large and flat, allowing caries to go undetected. It is wider than the occlusal. The primary teeth are lighter in color than the permanent teeth [1].

Principles of minimum intervention dentistry, based on the FDI World Federation, the five principles of minimum intervention must be applied, including [1]:

1. Control disease through the reduction of cariogenic flora.

   Control plaque and reduce carbohydrate foods.

2. Patient education

   Patients are educated on the causes of caries and ways to prevent caries. Dietary modification and maintaining dental and oral hygiene should be emphasized.

3. Remineralization of non-cavity lesions in enamel and dentin.

   Caries is a process of demineralization and remineralization. Early caries lesions can be stopped through remineralization. Saliva plays an important role in this process. The assessment of saliva for its quantity and quality is an important aspect.

4. Minimal intervention in cavity lesions

   Minimizing the amountcremoved to preserve the natural structure of the tooth, then restoring it with an adhesive material, such as GIC or composite resin.

5. Repairing damaged restorations

   Because the removal of old restorations results in the loss of healthy tooth structure, repairs should be considered as an alternative to replacement when possible, such as crown insertion.

The classification of cavity preparation consists of:

1. Initial preparation

   Initial preparation is carried out at a specific depth limits to provide access to caries, achieve healthy tooth structure (except for infected dentin on the pulp wall or crown wall), resist tooth fractures as well as restoration materials from the masticing force especially along the long axis of the tooth, and retain the restoration material in the tooth.

   a. *Outline form and initial depth* are carried out as a location where the periphery of the finished tooth preparation will be carried out.

   b. *Primary resistance form*, the shape of the preparation wall allows the restoration of the tooth to withstand, not fracture, the resulting chewing force evenly over the length of the tooth axis.

   c. *Primary retention form*, a form of preparation that is retained from the tipping force or lifting during the restoration process.

   d. *Convenience form*, a form of preparation that provides adequate observation, accessibility, and ease in the dental restoration process.

2. Final preparation

   a. Removing infected dentin tissue or old restoration material.

   b. Pulp protection, if needed.

   c. *Secondary resistance and retention forms,* many preparations require additional retention. When dental preparations include occlusal and proximal surfaces, each of those areas must have free retention and resistance. For example, for amalgam, *groove* for *cast metal*, fringe for restoration.

   d. *Finishing the external walls:* This is an additional measure implemented when the restoration material's maximal efficacy is demonstrated to be achieved through the utilization of a specific cavosurface design and the degree of smoothness or roughness.

   e. *The final procedure*: involves cleaning, checking, and sealing, which includes removing all residual impurities, drying the preparations, and performing final checks for infected dentin residues, poor enamel margins, or any conditions that render the preparation unacceptable for receiving restorative materials.

### 4.2 Restoration options of early childhood caries (ECC)

Caries control with early caries treatment must be carried out systematically and comprehensively, and in accordance with the principles of prevention and comprehensive care. Comprehensive treatments are quite numerous and complex, including

Class I and II fillings, Class III fillings, pulpotomy with composite indirect follow-up, retraction with infiltration anesthesia and Fisher blocks, and space management treatments such as space maintainer or space regainer. Patient cooperation greatly determines the success of treatment. In these patients, both the patient's parents and the patient behaved positively toward dental care, considering the many treatments that had to be done [17].

#### *4.2.1 Cosmetic restoration in primary teeth*

ECC treatment is usually limited to tooth extraction and restoration of teeth that have suffered from caries, but over the past half-century, the emphasis on ECC treatment has shifted broadly from extraction to restoration. Esthetic treatment of the first teeth that have experienced ECC to S-ECC is the biggest challenge for pediatric dentists. ECC to S-ECC restoration options for primary teeth should provide an esthetic appearance in addition to restoring function and durability. Involvement of the central incisive teeth, lateral incisive, and first molar posterior maxillary and mandibular teeth. The primary incisive teeth of the upper jaw are most affected by caries lesions involving open pulp. In extreme cases, ECC can even lead to the loss of the entire crown structure. Loss of primary anterior teeth can result in reduced chewing function, loss of vertical dimensions, oral bad habit (sticking out the tongue in the missing tooth area), esthetic problems, disturbed psychological, and behavioral development of children [1, 7].

Some esthetic restoration options to improve ECC include resin restoration, both direct and indirect techniques, and fabrication of crowns. Primary teeth cannot accept occlusal force if restored with conventional cement, therefore requiring the use of an anterior crown as a proper option. It can be a polycarbonate crown, a strip crown, a stainless steel veneer crown, an artglass crown, or a zirconia crown. At the maximum level of physiological chewing force, teeth with ECC to S-ECC can be restored with a zirconia crown that has been formed can withstand stress better than teeth restored with Stainless Steel Crown (SSC), preveneered crowns, strip crowns, zirconia crowns are recommended for the restoration of the esthetics of the primary anterior and posterior teeth. Zirconia (Zirconium dioxide, $ZrO_2$), also referred to as "ceramic steel," has optimal properties for dental use—toughness, strength, and fatigue resistance, in addition to excellent wear properties and biocompatibility. This crown offers esthetically pleasing clinical features and has strong durability. An *in vitro* study has shown that natural teeth that oppose zirconia crowns have much more favorable wear than those opposite natural teeth (**Figure** 7) [1, 7].

Some restoration options, the use of Atraumatic Restorative Treatment (ART), are a basic procedure that aims to eliminate caries lesions using hand instruments due to the absence of other dental equipment available. This technique is not new in dentistry, but it is a very simple technique and is able to repair teeth that have suffered caries due to the development of glass ionomer cement, which is modified with resin so that it is more durable. Before the application of ART, sodium diamine fluoride can be applied to caries lesions to improve caries control in a technique known as the "silver modified atraumatic restorative technique" or SMART. Therefore, this technique is indicated to deal with ECC, especially in developing countries [7, 15].

#### *4.2.2 Simple treatment ECC with open pulp chamber*

Severe Early Childhood Caries (S-ECC) has a clinical picture in the form of extensive caries in the first tooth with rapid lesion development that occurs in

**Figure 7.**
*Zirconia crowns in pediatric dentistry [1].*

children under 3 to 5 years of age. Tooth decay due to caries has a great potential to disrupt optimal function and significantly affect esthetics. Primary teeth have an important function in the child's developmental process. For this reason, comprehensive treatment is needed in the rehabilitation of teeth affected by caries. Primary teeth that have extensive caries with the involvement of pulp and replacement permanent teeth that have not erupted can be treated with pulpotomy or pulpectomy, as for simple treatment on open pulp, namely Lesion Sterilization and Tissue Repair (LSTR). The concept was developed by Niigata University's School of Dentistry. LSTR is a noninstrumental endodontic treatment that can be used in a single-visit approach. LSTR provides a much faster treatment option, making it a better choice for patients with behavioral problems than conventional pulpectomy. Many studies have shown LSTR to be an effective treatment option for irreversible pulpitis and pulp necrosis in primary teeth. During LSTR treatment, ciprofloxacin and metronidazole were selected for their bactericidal properties against anaerobes and protozoa, and minocycline is used for its broad-spectrum activity against gram-positive and gram-negative microorganisms. The substance can produce changes in the blood vessels in the pulp. The remaining vital pulp cells multiply and develop new pulp tissue into the coronal pulp chamber, called pulp revascularization. The use of crown restoration with zirconia is recommended after treatment on teeth with open pulp [18–21].

### 4.3 Child psychology approach in dental care

In understanding children's behaviors and responses, children have different reactions and behaviors compared to adults, especially in clinical contexts. Factors such as age, previous experience with medical care, as well as their cognitive and emotional development greatly influence how they respond to clinical situations.

Clinical psychology treatment should consider the communication technique in accordance with the child's level of understanding and comfort. Using simple and easy-to-understand explanations, as well as appropriate metaphors or props, can help children understand the procedure they are going through, thus reducing anxiety. Children tend to be more sensitive to their surroundings. Therefore, creating a child-friendly clinical environment, such as a waiting room equipped with toys or fun decorations, can help reduce stress and increase their cooperation during treatment. Approaches in psychology to clinical management can be carried out among others [1, 7].

#### *4.3.1 Biological approach*

In a clinical context, this approach can be applied to understand how physiological responses to stress (such as increased heart rate or adrenaline) affect a child's behavior during treatment. This knowledge can guide the doctor in developing strategies to calm the child, such as using breathing techniques or other relaxation methods.

#### *4.3.2 Behavior approach*

Techniques such as positive reinforcement (reward) can be applied to encourage children's cooperative behavior. For example, giving a small gift after a medical procedure encourages the child to be calmer and cooperative during treatment.

#### *4.3.3 Cognitive approach*

In clinical management, understanding children's mindsets (especially their fears or misunderstandings about medical procedures) allows medical personnel to provide explanations that can turn negative perceptions into more positive ones. This can be applied through convincing narratives, role-playing, or the use of visuals that children can access.

#### *4.3.4 Implementation strategies in clinical management*

Communication with children must be implemented with empathy and in accordance with their cognitive development. In an individualized care approach, each child is unique and may require a different approach. Some children could be cooperative with detailed explanations, while others may respond better with distractions or game approaches. Therefore, medical personnel must be flexible and able to adapt their approach and handling based on the individual needs of the child. Children often feel scared or anxious when facing some medical procedures.

#### *4.3.5 Evaluation and adjustment of approach*

Continuous observation of the child's reactions and behavior during and after the procedure allows the medical personnel to evaluate and assess whether the approach is appropriate or needs to be adjusted or not. Based on the results of the evaluation, the clinical approach should be adjusted and improved as needed. For example, when the child shows excessive anxiety despite being explained completely already, additional approaches such as game therapy or brief counseling may be necessary.

## 5. Conclusions

ECC treatment is carried out comprehensively. Treatment from the mildest to the most invasive is carried out as one of behavior management. The success of early caries treatment depends on the coordination of dentists, parents, and children. Determining the right diagnosis and treatment plan can maximize ECC treatment outcomes. Preventive care in children is also very important. In this case, it includes educating how to take care of dental and oral health in children and directing dietary patterns, reducing the frequency of consuming sugary foods, consuming foods with balanced nutrition, and maintaining the cleanliness of the oral cavity. This is necessary to prevent the onset of caries, thus avoiding negative interference with the quality of life of children.

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## Conflict of interest

The authors declare no conflict of interest.

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Chapter 5

# Advanced Laser Applications for Dental Caries Management

*Ruxandra Elena Luca, Roxana Ioana Munteanu, Mariana Ioana Miron, Alexandra Roi and Ciprian Ioan Roi*

**Abstract**

Laser therapy is already a viable treatment modality, accessible to many doctors, which is coming to compete with or replace conventional treatments. Laser therapy in managing dental caries has applicability from the diagnosis and prevention stage to the monitoring of pulp vitality and the actual treatment of caries. There are several laser wavelengths, respectively types of laser radiation, that have therapeutic applications following international concepts of minimally invasive dentistry. Nonsurgical lasers have multiple applications: Laser fluorescence sustains the early diagnosis of carious lesions, laser Doppler flowmetry indicates variations in pulpal circulation, antimicrobial photodynamic therapy helps in reducing the microbial activity, and laser biomodulation has reparative effects and reduces postoperative pain and inflammation. Surgical ablative lasers, such as Er: YAG and Er,Cr: YSGG, are used in cavity preparation and surface modification to increase caries resistance, at the same time being able to decontaminate and condition the surface for the adhesion of restorative materials. Increased patient acceptability of laser therapy in the treatment of dental caries is reported, requiring less anesthesia and resulting in less postoperative pain.

**Keywords:** laser radiation, minimally invasive, laser fluorescence, diagnosis, treatment of dental caries

## 1. Introduction

Since Theodore Maiman introduced the first ruby laser (Light Amplification by Stimulated Emission of Radiation) device to the medical world in 1960, laser radiation has steadily and continuously gained ground in the dental field, with increasingly diversified applications in hard and soft tissues. Among the first investigations was the one conducted by Stern and Sognnaes [1], which showed that the dental enamel that was exposed to ruby laser radiation has enhanced resistance to acid.

Numerous physical phenomena involving the interaction of light with biological tissues, cells, and fluids serve as the foundation for the clinical application of laser irradiation. Nowadays, dental lasers range between 193 and 10,600 nm, with very distinct clinical applications and effects, depending on the wavelength. Their insufficient knowledge is also the main explanation underlying the still limited understanding of this technology compared to the traditional way of operating in dentistry.

The generation of monochromatic, coherent, and collimated radiation by a suitable laser medium in an optical resonator is the principle underlying this technology. These main characteristics of laser radiation ensure a very narrow wavelength, which targets a specific tissue with precision, without affecting neighboring tissues, having increased coherence, due to efficient photon collimation (the degree of divergence is approximately 1 mm for every m of generated radiation in the device). The result is a very well-focused radiation, on a small-sized spot, therefore a very precise treatment [2].

Depending on the emission mode and the thermal effect generated, the results of laser radiation on the target tissue may be different. High-power lasers are used to create the thermal effects required for therapeutic procedures such as cutting, coagulation, vaporization, and ablation of biological tissues. Since different tissues in the oral cavity have different optical qualities, the light energy from a laser can interact with the target tissues in four different ways: reflection, transmission, scattering, and absorption (**Figure 1**). These interactions are influenced by many variables, among which we include the wavelength, the pulse time, energy and repetition rate, beam spot size, delivery method, laser beam properties, and the optical properties of the tissue.

For a better understanding of laser-assisted dental treatments, we will establish the terminology used below [3]:

- Wavelength: the distance between two successive wave crests
- Watt: a metric unit of measurement of the intensity of power that gives rise to the production of energy at the rate of 1 J/s.
- Joule: measurement of energy

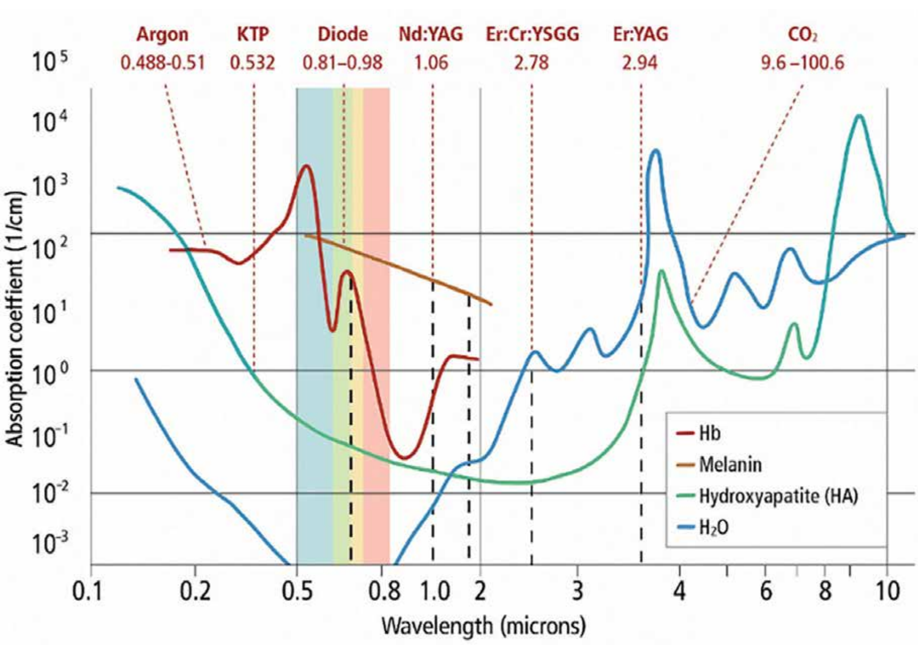


**Figure 1.**
*Absorption coefficient of different wavelengths used in dentistry.*

- Energy density: the total amount of energy per unit surface area, measured in J/cm$^2$.
- Hertz: measurement of frequency

Mainly, the processes by which laser radiation acts on tissues are the following: photochemical (includes molecular processes of stimulation and repair, as well as photodynamic therapy), photothermal (process underlying vaporization ablation), photomechanical (or photodisruption as a process of removing tissue through shock waves), and photoelectrical (photo-plasmolysis, electrically charged ions and particles) [2].

In restorative dentistry, the applications of laser radiation are the following **Figure 2** [2]:

- Caries prevention
- Diagnosis of dental caries
- Treatment of dentin hypersensitivity
- Cavity preparation
- Conditioning of tooth surface
- Removal of restorative materials
- Photopolymerization

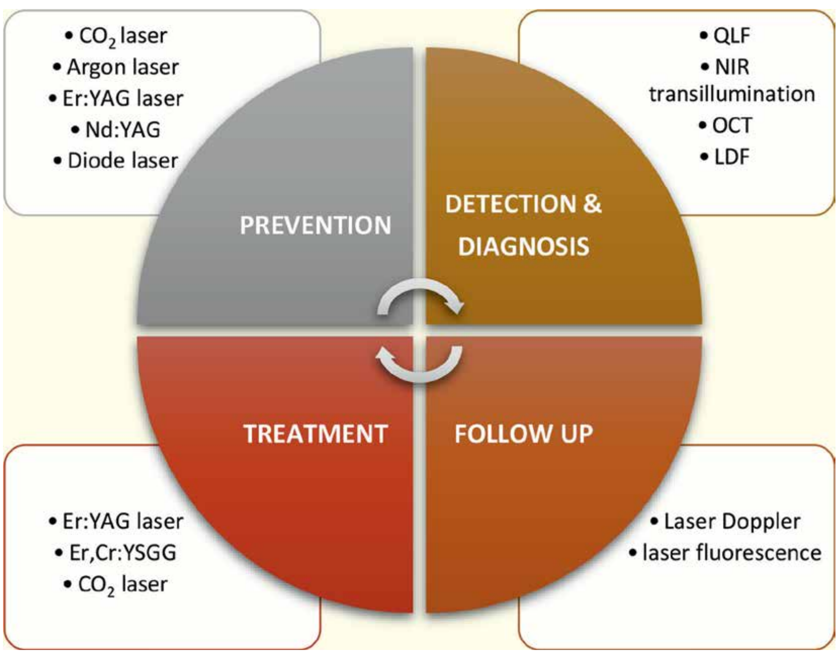


**Figure 2.**
*Overview of different wavelengths applications in caries management.*

- CAD/CAM technology
- Restoration, pit, and fissure sealants
- Traumatology and vitality testing
- Preservation of pulp vitality
- Disinfection and decontamination
- Pain-relieving effects, relief of pain and uneasiness
- Pulp capping

## 2. Lasers in preventive dentistry

### 2.1 Global context

Despite the fact that modern dentistry has undergone remarkable improvements, a 2018 study conducted in the USA brings into question a high prevalence of total and untreated dental caries in primary or permanent teeth among youth aged 2–19 years of 45.8 and 13.0%, respectively [4, 5]. On the other side of the world, in China, a 2023 study reports worrying percentages: The caries rate of 12-year-old children's permanent teeth is 34.5% and the caries rate of 5 years old children's temporary teeth is 70.9%, with an increasing pattern [6]. These percentages are worrying and show the limited effectiveness of policies to prevent and treat incipient caries lesions.

Other studies show that the overwhelming majority of carious lesions in children (90%) form in areas with morphological complexity, like pits and fissures [7]. These numbers and multiple clinical observations have been, over the years, the basis for the recommendation to seal these anatomical areas in recently erupted teeth, in an attempt to limit biofilm retention and therefore the possibility of caries development. In this direction, the interaction of different laser wavelengths with dental hard tissues has been studied, some of which, such as Argon (488–514 nm) and Nd:YAG (1064 nm), have shown their inefficiency regarding enamel absorption. Regarding the radiation of the Nd:YAG laser, attempts have been made to associate this with a photoabsorber, such as India ink, to increase the absorption of the laser beam at the surface of the enamel, with the positive results [8].

### 2.2 $CO_2$

Irradiation with microsecond pulsed $CO_2$ lasers resulted in enhanced caries resistance of the enamel. The emergence of this type of laser is attributed to Patel, in 1964 [9], and since then, it has been considered to be one of the most popular wavelengths in the infrared spectrum, having different emission possibilities, ranging from 9 to 11 μm. The 10.6 μm $CO_2$ laser is the strongest one and the most commercially available [3].

In order to have favorable effects on the dental enamel, laser radiation must be absorbed at this level, in such a way as to modify the structural composition or properties of the dental hard tissue without having unwanted secondary thermal effects on the surrounding tissues. It is therefore essential to understand and know the absorption and scattering coefficients of different biological tissues.

On enamel, the mode of action of $CO_2$ laser radiation has been explained in various ways, from modifying the solubility of enamel [10, 11] to some degree of its melting and recrystallization [12, 13] or carbonate loss, which is a soluble mineral within the enamel [14, 15]. On dentin, the effects of $CO_2$ irradiation were less investigated and reported. Even so, some hypotheses have been put forward, such as re-crystallization and an increase of inorganic content [16, 17], but the optimal parameters and effects of the $CO_2$ laser are not yet elucidated at the dentin level; factors to consider are the much higher content of water and protein than enamel and a bigger scattering effect with the possibility of negative outcomes.

When comparing 10.6-μm $CO_2$ laser wavelength with 9.3- and 9.6-μm $CO_2$ laser wavelengths, a study concluded that the latter are absorbed up to 10 times stronger in enamel [5, 18]. Following increased absorption, carbonates are removed from the natural carbonate hydroxyapatite, which increases the resistance to hydroxyapatite`s resistance to acid. If immediately after this process, fluorides are added, the resistance to acid attack is even more increased due to fluorapatite formation [19–21]. The conclusions of several studies support the fact that $CO_2$ laser irradiation in combination with the fluoride treatment is more effective in inhibiting caries demineralization than laser irradiation or fluoride alone; $CO_2$ laser and fluorides can be used together in order to decrease both the fluoride levels and laser energy density [3]. An extensive review conducted in 2020 [22] concluded that the combination of therapies, traditional prophylactic interventions combined with the use of sub-ablative lasers, boosted the efficiency of caries prevention, with the absence of side effects such as irreversible dental pulp pathologies. A more recent review [23] addresses the effects and potential use of a 9300 nm carbon dioxide laser on dental hard tissue. The authors clearly and concisely expose the differences in absorption of $CO_2$ wavelengths, which underlie the more frequent use of the 10,600 nm wavelength in surgery, while 9300 nm and 9600 nm are, with increased penetrability in enamel and dentin, the wavelengths of choice for dental procedures. Of the latter, the 9300 nm $CO_2$ laser has the best energy transfer to the hydroxyapatite level, which means that lower energy is needed to obtain the same therapeutic effects, with the risk of heating being diminished. At the level of hard dental structures, this type of laser has the ability to modify the chemical structure or to ablate, the effects being different and complex, depending on the parameters used (power, pulse frequency, etc.). Compositional changes at the enamel level refer to the alteration of the microstructure and the decrease of carbonate composition, resulting in purer apatite crystals, which are more resistant to acid attack. At the same time, there is a slight surface melting and an increase in calcium and phosphorus content, this effect being visible also at the dentinal level. Therefore, laser irradiation produces a surface that is homogeneous, smooth and with reduced organic content, a "hypercrystalline" structure. If the appropriate parameters are not selected, there is a risk of causing cracks and loss of the collagen matrix. Temperature control during laser treatment is also crucial, as it should be limited to a maximum increase of 5.5°C. This is an additional argument for the need for good training of the doctor in the use of laser radiation in dentistry, considering the variability of the oral soft and hard tissues, as well as the complex interaction between the different wavelengths and them [6, 24].

### 2.3 Argon laser

Modifying the behavior of dental hard tissues to acid attack can be a useful weapon in the fight against white spot lesions, often associated with the orthodontic treatments. The increase in micro-hardness can be the viable solution in these situations, and multiple wavelengths showed the promising results in this respect: argon lasers, $CO_2$, Nd-YAG, and Er:YAG. Some studies report a decrease in demineralization extent by 30–50% when Argon laser radiation is applied [2, 25].

### 2.4 Erbium lasers

Prior research has demonstrated that the combination of fluoride and Er:YAG laser can also alter the chemical composition of dental enamel, facilitating remineralization. Before and after the synergistic application of Er:YAG laser and fluoride, there were no statistically significant alterations in the main microbial composition; nevertheless, the proportion of microorganisms altered and the diversity of microflora increased: bacteria associated with caries occurrence, like Proteobacteria, Fusobacteria, and Bacteroidetes, declined, while other species increased. The laser and fluoride combination treatment groups also showed lower abundances of Firmicutes, Streptococcus, Fusobacterium, and Veillonella than the fluoride treatment group. In addition to being straightforward and noninvasive, the combined use of fluoride and Er:YAG laser promotes the microecological balance of plaques [26]. Fluoride and Er:YAG laser irradiation worked together to improve fluoride absorption and encourage the development of a "fluoride reservoir." Compared to using each technique alone, this combination showed more noticeable synergistic results [27, 28].

### 2.5 Diode lasers

Laser diodes have also been investigated in the prevention of dental caries. Promising results in this regard were demonstrated by the 940 nm diode, whose radiation of 5 W was for 15 sec. After two types of fluoride, varnish applications led to increased fluoride release [29].

The scientific foundation of preventing cavities behind this type of studies refers to fluoride treatments, which can be added to the enamel matrix to generate fluor-hydroxyapatite, which is less soluble in acidic environments.

The development of fluoride reservoirs on the tooth surface is the primary determinant of the effectiveness of this topical fluoride treatment. When used in conjunction with the topical fluoride, laser radiation enhanced fluoride binding to the enamel and boosted fluoride incorporation into the hydroxyapatite structure. The combination of these techniques increased the formation of calcium fluoride and fluor-hydroxyapatite on the surface of the enamel, acting as a fluoride reservoir [30, 31].

## 3. Laser-assisted diagnosis

The global spread of dental caries and the limited success of already known methods of preventing it have led to the exploration of new diagnostic and prevention techniques. Added to these are the limitations of current visual and tactile methods of diagnosing dental caries, which are still largely based on inspection, palpation with a probe, and radiological control. Perhaps one of the most sensitive issues is that diagnostic capacity varies greatly between individuals, with significant consequences for treatment and progression of the lesion, since it is a matter of subjective interpretation of sometimes very subtle clinical or radiologic findings [32, 33]. The same type of lesion could be monitored and remineralized by one practitioner, while another would decide to restore it. An objective assessment of the dental surface would help to standardize the diagnostic stage and eliminate individual errors.

Radiographs show that demineralized tissue has lower X-ray attenuation compared to normal tissue but cannot measure mineral changes over time. Bitewing radiography helps detect and diagnose early caries on the proximal surface. CBCT is unnecessary and contradicts the "as low as reasonably achievable" (ALARA) principles, as two-dimensional radiography is already an acceptable diagnostic tool [34].

Diagnostic problems can appear at any age, but they are more frequently encountered in young patients. To these, we add the current knowledge we have regarding dental caries, namely that this is a dynamic process that has phases of demineralization and remineralization, the dynamics of which depends on the static or progressive character of the carious lesion and, therefore, its treatment modality.

These challenges have led to the emergence of new tools for diagnosing dental caries, including methods based on fluorescence, transillumination, and electrical impedance. Among the optical based methods, DIAGNOdent (KaVo Dental GmbH, Germany) has become the most well-known adjuvant tool in the diagnosis of dental caries [32].

### 3.1 Laser fluorescence detection of occlusal caries

When we refer to optical methods for diagnosing dental caries, we must understand the physical interaction between light and the tooth surface, which is based on the four essential processes: absorption, transmission, reflection, and scattering. Fluorescence methods are based on the absorption capacity of the targeted tissue, most likely of organic components and bacterial metabolites (especially porphyrin IX) [35].

After being properly activated, materials can release light through a variety of processes, including fluorescence. One of the main characteristics of fluorescence is that the light emission corresponds to the excitation mode; therefore, if the light is pulsed in a specific manner, the fluorescence emissions will exhibit the same pattern. When a fluorophore absorbs light, the molecule is stimulated to higher energy levels, where it decays to lower energy levels through thermal relaxation and radiation emission. Fluorescence is highly helpful for quantification because the strength of the emission is linearly proportional to the concentration of the fluorophore in the target [36].

Several imaging approaches for detecting caries use the fluorescence response of organic components in teeth. The color of emitted fluorescence light differs from that of excitation light due to differences in energy, wavelength, and photon energy. Various systems available on the market can be categorized based on the light-based system color, such as [37]:

- red fluorescence is characteristic of 655 nm wavelength, which is employed in the DiagnoDent laser device.
- blue fluorescence is employed with wavelengths of 400–450 nm, of which the most well-known are the DIAGNOcam (Kavo, Germany), VistaProof (Durr Dental), and SoproLife (ACTEON, France).
- green fluorescence is identified with a spectrum around 370 nm, known as quantitative light-induced fluorescence (QLF) devices.

### 3.2 655 nm diode laser

The DIAGNODent laser device (KaVo) uses laser fluorescence to identify incipient caries lesions. It consists of a diode laser at a wavelength of λ 655 nm and 1 mW peak power, which generates a laser beam that is absorbed at the occlusal surface and then re-emitted (backscattered excitation) as infrared fluorescence [38]. The device will separate, through modulation, the fluorescence from the surrounding light and the result will be displayed on a screen as a number between 0 and 99. In order to limit the errors, in accordance with the manufacturer's instructions, differences in the laser's output power should be routinely corrected for by calibrating the device against the standard fluorescence and the sound enamel. To minimize reading errors, care must be taken in calibrating the device and in the way the measurement is performed; the surface must be smooth and clean of bacterial plaque [35].

One major advantage of this technology is when detecting dentinal caries that are not clinically visible, and this is due to the transparency of enamel for light in the near-infrared spectrum. Another aspect would be the completely non-invasive verification of non-cavitary lesions and of those that have reached the enamel-dentin junction, with high reproducibility in readings [36].

In terms of presentation, there are three variants of this device: the DIAGNOdent Classic (the first version, launched in 1999, **Figure 3a**), the DIAGNOdent Pen (appeared in 2005 and has applicability for approximal surfaces also; **Figure 3b**,**c**), and the KEY-3 laser (fluorescence added to an Er:YAG laser, in order to diagnose and treat carious lesions simultaneously).

Some studies reported acceptable clinical accuracy of the device, in comparison with the traditional diagnostic methods, encouraging the adjuvant use of fluorescence in conjunction with classical examination and diagnostic tools [35]. Other studies highlighted the increased sensitivity of the method, in comparison with traditional diagnosis methods, supporting its integration in daily clinical use [39, 40] and some researchers increased the awareness regarding the sources of error if the technique is not mastered correctly [41–43].

### 3.3 Quantitative light-induced fluorescence (QLF)

The QLF approach measures changes in teeth using autofluorescence generated by 405-nm visible light from the blue spectrum. The approach works by changing


**Figure 3.**
*DIAGNOdent classic (a) and DIAGNOdent Pen (b,c).*

the autofluorescence of a specific lesion in the tooth dependent on its mineral content. The reduction of fluorescence in teeth was highly linked with mineral loss. Furthermore, demineralization of enamel in carious lesions decreases autofluorescence and darkens the enamel. This method detects and monitors minute changes in demineralization/remineralization of early carious lesions without cavities.

Dental caries, plaques, and calculi exhibit red fluorescence, which are caused by porphyrin-induced metabolites produced by oral microbes [44]. When used for proximal caries, the QLF ability to detect caries is limited by the fact that light is already reflected before reaching the lesion; therefore, the accuracy of detection depends on the extent and type of lesion. When compared to the clinical and radiological traditional ways of detecting caries, QLF showed improved detection of occlusal dental cavities and cracks compared to traditional methods. Bitewing radiography had a better detection rate for proximal caries. Combining these complementary technologies could reduce missed lesions and improve diagnosis accuracy without requiring unneeded radiation [44].

QLF images can be visualized, saved, and sent as digital files with the appropriate equipment. They can also be processed using AI models. AI is already used to diagnose different diseases. Convolutional neural networks (CNNs), a type of AI model, excel at computer vision and can be utilized for image analysis [45, 46].

There are recent studies [46] revealing that QLF images may successfully diagnose dental cavities using AI applications. This work could contribute to a thorough examination of oral health risk factors, such as dental caries and plaque, using the QLF approach.

In addition to the diagnostic stage, fluorescence is also applicable in the treatment stage of the carious lesion, namely the verification of the walls of the prepared cavity in relation to the persistence of the carious lesion. This technique is also known as fluorescence-assisted caries excavation (FACE) and is achieved using a 405 nm diode and a 530 nm high-pass filter, which will show the carious dentine in an orange-red appearance. In comparison with conventional caries excavation, the FACE technique showed superiority in terms of bacterial reduction (**Figure 4**) [36, 47, 48].

What is interesting to note about this wavelength is that Lactobacilli, the secondary colonizers of carious lesions, produce more noticeable red fluorescence than *Streptococcus mutans*, and *Actinomyces odontolyticus* demonstrates significant porphyrin fluorescence, which is a sign of caries involvement, since this component is absent in healthy enamel [37, 49].

The clinical applicability of this wavelength has been expressed in the emergence of numerous pen/camera/handpiece-type devices, which helps in the diagnosis of

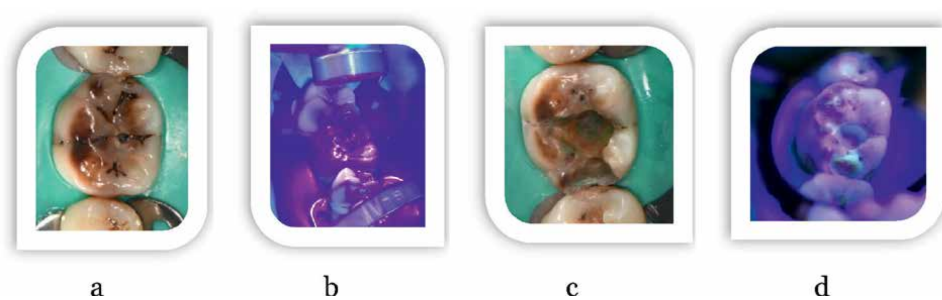

**Figure 4.**
*Caries detection mode of a 380 ~ 420 nm wavelength device (Coxo, COXO Medical Instrument Co., Ltd., China). Green light: healthy; Red light: infected tissue (a—initial situation, clinical views, b—initial situation-caries detection mode; c—cavity walls verification using Sable Seek, Ultradent; d—cavity wall verification using fluorescence).*

carious lesions, offering information about the size of the lesion and also about bacterial content, in the form of the intensity of the light spot. The same clinical application can be extended to the detection and evaluation of mature bacterial plaque, with high porphyrin content [50].

### 3.4 Near-infrared (NIR) transillumination

Transillumination is a long-standing alternative to radiography for detecting cavities. The concept is based on the enamel's optical characteristics, which are altered by the slightest variation in porosity, resulting in enhanced scattering as light passes through the enamel.

During light scattering, a photon's orientation changes without losing energy. When incident light encounters tissue impediments, it deviates from its route, resulting in enamel demineralization [51]. Scattering is dependent on wavelength, with shorter wavelengths scattering more than longer ones [52]. Caries detection methods using visible wavelengths (400–700 nm) are limited due to scattering [37].

In the 1970s, fiber optic transillumination (FOTI) was introduced to detect proximal caries, meaning early changes in enamel structures. This technique was used in conjunction with radiography and clinical examination, but the results proved to be significantly different among examiners. In the 1990s, digital fiber optic transillumination imaging (DIFOTI) was developed to capture images and track lesion progression over time. In 2012, the DIAGNOcam (KaVo, Biberach, Germany) was introduced using a 780 nm near-infrared transillumination technology. In 2021, a newer version, DIAGNOcam Vision Full HD, combines near-infrared transillumination with clinical features and fluorescence, offering clinicians an undeniable advantage in formulating an accurate diagnosis and treatment plan [37]. These merged technologies are the basis for the development, in recent years, of 3D oral scanners, which represent a paradigm shift in conventional dentistry.

NIR imaging at 1310 nm is more effective than dental X-rays for detecting early demineralization. The approach can identify not only proximal cavities, but also occlusal caries and cracks [53]. Several investigations have indicated that NIR has high sensitivity and it may emerge as a valuable alternative to radiography for the early detection of proximal caries, with the advantage of a better localization of the caries lesion in the bucco-oral direction, offering the possibility of a minimally invasive approach.

### 3.5 Optical coherence tomography (OCT)

Optical coherence tomography represents a nondestructive three-dimensional imaging technique that can generate high-resolution cross-sectional images of heterogeneous samples like biological tissue. Essentially, OCT is similar to ultrasound imaging, but it employs light instead of sound [54, 55]. It was first utilized in ophthalmology, and due to the very good results in this medical branch, the applications have been expanded, including many domains in dentistry: restorative dentistry (detection of demineralization, enamel defects, carious lesions), orthodontics (surface characteristics after debonding), prosthodontics (assessment of marginal fit, defects detection), implantology (assessment of implant-abutment interface, periimplant mucositis), periodontology (visualization of periodontal structures and dental biofilm), oral pathology (detection of different degrees of dysplasia) [56], and even bone regeneration [57].

OCT uses a low-coherence light source and a Michelson interferometer to create cross-sectional images of tissue structures. The images are formed by the interaction of a partially coherent stream of optical radiation with the tissue components. Evidence suggests that the infrared region (780–1550 nm) is ideal for imaging enamel due to minimal scattering and absorption, particularly around 1310 nm [58].

Colston et al. [59] performed the first dental OCT in 1998. Using an OCT prototype they have created, they captured images of porcine periodontal tissue, in which enamel and cementum were clearly visible, marking the first use of OCT for imaging biological hard tissue [55]. OCT imaging depth on a tooth varies based on the structure being imaged, but typically ranges from 2 to 3 mm in many circumstances [58].

Many steps toward using OCT in caries detection have been made by the research group at the University of California, San Francisco [60], whose initial results showed the technology's ability to detect incipient demineralization, then showed its applicability in the detection of proximal, occlusal, and secondary caries, as well as various stages of de-/re-mineralization [55]. The caries detection capability of OCT was compared with other existing technologies, such as micro-CT, QLF, and near-infrared transillumination, showing promising results.

OCT enables early diagnosis of caries lesions without exposing patients or personnel to ionizing radiation. Additionally, it can monitor the early progression of dental caries and assess the effectiveness of restorative treatments. Even with these important advantages, there are still several issues that limit the practical application of OCT, such as small penetration depth, patient movements, and other disruptive intraoral environments during image acquisition and optimal image processing that must be addressed [55]. Furthermore, it requires customization for dental use and is not currently commercially available. It appears that this technology could be combined with other optical devices in the near future and even enhanced with deep learning and AI diagnostics methods.

### 3.6 Laser Doppler flowmetry (LDF)

The use of various types of lasers for the diagnosis and treatment of carious and non-carious lesions of teeth has become a common practice in dentistry [61]. Laser can be used as a suitable alternative for many conventional procedures in dentistry, both in pediatric dentistry and in adult and senior patients [62, 63]. However, there are certain dental hard tissue conditions whose correct treatment necessarily involves an initial assessment of the pulp vitality status in order to establish a diagnosis of certainty. This is the case of teeth with cracks or coronal fractures, either due to chronic occlusal trauma, as in the case of bruxism, or due to accidental trauma [64, 65]. Deep carious lesions may be accompanied by reversible or irreversible damage to the coronary pulp, whether they are located occlusally, proximally, or in the cervical or root area. Excessive consumption of acidulated and citrus juices can cause demineralization of the enamel, accompanied by the presence of dental sensitivity. On the other hand, performing dental treatments, such as teeth whitening, pulp capping, or photobiomodulation, can produce effects at the level of the dental pulp [66]. Even the incorrect use of diagnostic and treatment procedures for dental lesions, either through conventional or alternative techniques, can cause reactions of the odontoblasts extensions and, implicitly, at the level of the dental pulp microcirculation [67].

Therefore, procedures for establishing the diagnosis of various dental lesions, as well as treatment techniques, whether classical or alternative, must take into account the assessment of the vitality of the tooth that will be diagnosed and treated.

For this purpose, LDF is used in dentistry as a diagnostic tool, and essentially, it is a noninvasive method of measuring the blood perfusion quantitatively at micro-vascular level [68, 69]. The phenomenon on which it is based was first described by Christian Andreas Doppler and states that when a wave (e.g. laser light) meets an object in motion (e.g. blood cells) a Doppler frequency shift occurs, its amplitude being dependent on the speed of the moving object.

Laser Doppler flowmetry is recognized as an accurate, reproducible and reliable method for assessing pulp vitality, which allows continuous and real-time recordings of the microdynamics of pulpal blood flow (PBF). The major advantage of the LDF technique is its non-invasiveness and the ability to record changes in pulp microcirculation flow during the action of irritating factors and before the appearance of clinical symptoms. It is also painless and well tolerated even by children.

However, it is well known that many factors can affect tissue blood flow, so precautions must be taken in order to standardize measurement technique (**Figure 5**) and to eliminate other variables that may influence the blood flow signal and reduce the signal from the surrounding tissues (**Figure 6a** and **b**). Essential aspects for validating the LDF signal are the level of the signal as well as its pulsatile character. Moreover, the device software allows the keeping and statistical analysis of the data and the presentation of the results in scientific format (**Figure 7**).

According to the results of our previous study [70], LDF could be an objective instrument to evaluate and quantify the effects of dentin hypersensitivity (DH) treatment. LDF can capture the contribution of many concurrent factors and their interactions, in contrast to the subjective one-dimensional pain scales used in current practice.

Consequently, the laser Doppler flowmetry method can be used in dentistry as *an important tool for the diagnosis and prognosis of dental lesions*, due to several relevant advantages: noninvasive medical investigation; objective method; painless (does not cause discomfort to the patient); allows continuous measurements in real time; provides the quantitative determination of the pulp blood flow expressed in arbitrary units, compared to the subjective classical techniques that evaluate the painful, sensorial answer when applying a stimulus; provides data that evaluate the evolution of the vascular flow dynamics before the appearance of the clinical signs; and has software that allows the keeping, and statistical analysis of the data and the presentation of the results in scientific format.

**Figure 5.**
*The laser Doppler device, acquisition system and laser probe holder, which ensures the stability of the laser probe and allows repeated measurements.*


**Figure 6.**
*a—Application of a viscous light-curing resin around the evaluated teeth to reduce the signal from nearby gingival tissues; b—LDF signal acquisition system, positioned in the oral cavity.*

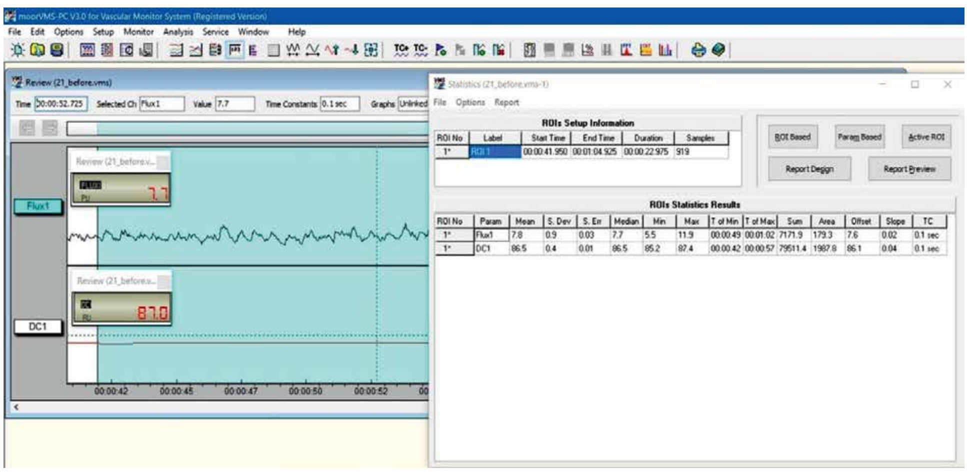


**Figure 7.**
*Laser Doppler signal and statistical results for the selected signal part.*

LDF would also be effective in *reliably monitoring the oral health status thus*, it is of *clinical relevance*. Moreover, as an objective, sensitive and reliable instrument, it could be employed in *screening and prevention programs*.

However, at the moment the LDF evaluation is not commonly used in dental offices, due to the lack of standardization of the acquisition technique and due to the sensitivity of the signal to any movement or factors, local, or general, that may influence the patient's local hemodynamics.

## 4. Laser-assisted treatment of dental caries

Traditionally, caries is removed using mechanical treatments such as hand instruments, dental drills or rotary instruments to eliminate the decayed tooth structure. While effective, this strategy may induce discomfort, worry, and dread in certain patients, resulting in dental phobia and avoidance of critical procedures. Complete caries removal, also known as "traditional" or "conventional" caries removal, entails fully removing diseased and damaged dentin and enamel during cavity preparation. Selective caries removal, often called "minimally invasive" or "conservative" caries removal, is a more cautious approach, in which only diseased soft dentin is removed, leaving the more mineralized dentin untouched. The decision between total and selective caries removal is based on factors such as decay severity, dental health, risk of recurrent caries, and the dentist's clinical judgment [71]. The goal of a minimally invasive dentistry (MID) approach to caries removal is to maximize the tooth's potential for health by halting the disease process and restoring lost tooth structure and function. Preserving as much of the dental tissue as possible is one of the key ideas of MID.

In order to address the conventional techniques limitations, researchers are studying other possibilities to solve these challenges and to find the potential benefits that represent the nowadays goals for caries treatment modalities [71]:

- minimally invasive techniques
- methods to reduce the anxiety, the pain and some patient's phobia
- preservation of tooth vitality
- less vibration and heat

In this way, different alternatives to the traditional approach appeared, among them the most well-known being chemical softening, mechanical abrasion, laser ablation, ultrasonic method, and the utilization of novel agents like ozone gas [71]. Alternative excavation techniques, such as lasers, may reduce dental discomfort and anxiety, and offer a less invasive, very precise, and more comfortable option.

Lasers that are employed for cavity preparation and caries removal use the ablation process, which removes dental hard tissue *via* thermal and/or mechanical effects during laser irradiation. This mechanism is dependent on the type of tissue to be treated as well as the properties of laser equipment. Knowledge of laser wavelength, emission, pulse duration, energy, repetition rate, beam spot size, delivery method, laser beam characteristics, and tissue optical properties, such as refractive index, scattering coefficient (μs), absorption coefficient (μa), and scattering anisotropy, is necessary to ensure better clinical results without thermal or mechanical damage to the dental hard tissue [72].

The most commonly utilized laser systems for irradiation of dental hard tissues include: Nd:YAG ($\lambda$ = 1.064 μm), Argon ($\lambda$ = 0.488 μm), Ho:YLF ($\lambda$ = 2.065 μm), Ho:YAG ($\lambda$ = 2.100 μm), Er:YAG ($\lambda$ = 2.940 μm), Er,Cr:YSGG ($\lambda$ = 2.780 μm), diode ($\lambda$ = 0.810 μm), and $CO_2$ ($\lambda$ = 9.300 μm, 9.600 μm, or 10.600 μm). With the exception of the argon laser, these lasers radiate in the infrared band of the electromagnetic spectrum. Most equipment runs in free-running mode, with pulse durations of microseconds. Lasers must be absorbed by enamel and dentin to effectively remove caries and prepare cavities; therefore, the most effective laser systems for this purpose are erbium and $CO_2$ [72].

Thermal ablation in dental hard tissues is sometimes referred to as explosive (water-mediated) tissue destruction, as infrared laser irradiation causes rapid heating of subsurface water within the hard tissue matrix. Heating water molecules causes a rise in molecular vibration and subsurface pressures, which can exceed tissue strength. Finally, there occurs an "explosion" of tissue, which results in hard tissue ablation, at temperatures below the melting point of tooth hard tissues (about 1200°C), and varies with the laser wavelength.

The efficacy of Er:YAG laser is based on the quick absorption of the 2940-nm wavelength laser by the water molecules and hydroxyl groups in dental tissue, causing water vaporization and fast heating. Erbium laser light destroys enamel due to the trapped water inside the mineral substrate, which expands quickly beneath the surface. In contrast to the rotary bur's action strategy, which mostly uses a side-cutting method, surface ablation eliminates tissue gradually (**Figure 8**) [73].


**Figure 8.**
*a, b: Initial situation revealing occlusal caries; c, d: different preset programs of Er:YAg lasers (Elexxion, Elexxion AG Dental Laser Singen, Germany, Fotona, Fotona d.o.o., Ljubljana, Slovenia); e: Er:YAG handpiece with sapphire tip; f, g, h, I, j: Er:YAG laser settings for enamel and dentin caries preparation and excavation (Elexxion, Fotona); k, l, m: clinical aspect after laser irradiation with set parameters; n, o, p: Er:YAG laser settings for conditioning dentin and enamel (Elexxion, Fotona); q,r: clinical aspect after laser irradiation with set parameters s, t: dentin hypersensitivity treatment parameters on 2 diodes (Elexxion 810 nm, Woodpecker, Guilin Woodpecker Medical Instrument Co., China, 650 nm).*

Since bacteria and viruses cannot survive at temperatures above 60°C, the dental laser destroys any bacteria or viruses that come into contact with it. It directly damages cell walls, modifies DNA, changes metabolic processes, and dissolves the biofilm's polysaccharide structure, among other biological structures. Two advantages of using burs as opposed to laser treatment include reduced postoperative pain and a lower chance of caries recurrence. Studies show that the laser lowers caries-causing bacterial strains such as *Streptococcus mutans* in addition to other strains like *Escherichia coli* and *Enterococcus faecalis*. Even while "total" sterilization is impossible, employing lasers to reduce microorganisms may reduce postoperative discomfort and the chance of recurring decay by lowering tactile trauma [73].

Microscopic studies reveal that laser ablation produces unique surface patterns with unequal micro-retention that are characterized by exposed enamel prisms, wide-open dentinal tubes, and the absence of a smear layer. Furthermore, it was shown that, in contrast to bur-cut surfaces, an even thicker hybrid layer with a more noticeable resin tag fabrication could be created on dentin surfaces that had been laser-prepared. Because laser treatment creates rough, uneven surfaces without a smear layer, self-etch adhesive methods are recommended. Phosphoric acid treatment of laser-cut tooth surfaces, however, seems to be advantageous as well. In this regard, Kiryk et al. clearly showed that combining laser-based preparation with Er:YAG with the conventional conditioning for acid results in improved adhesion qualities [74, 75].

The risk of postoperative pain and subsequent caries can be decreased by reducing bacterial populations as a result of surface cleaning. At the same time, the surface that is created can strengthen the bonding to the restorative material. Since the laser tip does not come into direct contact with the tooth surface, vibration, conventional noise, and pressure are eliminated. The Er:YAG laser preparation yields more conservative results due to the accuracy and selective removal of cavities [73].

In primary teeth, a useful clinical study [76] compared four different techniques of caries excavation in terms of efficacy, efficiency, and pain experienced during the procedure: Air rotor, Carisolv, Papacarie, and Er:YAG laser. The authors concluded that chemomechanical and laser irradiation were less unpleasant procedures for young children, but air rotor and laser irradiation were more effective and time-efficient methods of caries excavation in primary teeth. Another similar but more recent clinical study [77], conducted in 2024, compared the Er:YAG laser with a conventional drill in removing caries in children. The study was a split-mouth one, with conventional dental turbine drilling on the control side and an Er:YAG laser for the observed side. The study assessed the frequency and severity of tooth fractures during caries removal, as well as the usage of anesthetics, pain thresholds, and tooth hypersensitivity. The children's cooperative behavior and clinical anxiety were also noted, as was the amount of time needed to prepare the cavities, as well as the treatment success rate at 1-year follow-up. The findings showed that using an Er:YAG laser significantly decreased the need for anesthetics, discomfort, and the frequency and intensity of tooth hypersensitivity. There was no discernible difference in the two groups' rates of tooth fractures. For the side treated with Er:YAG laser, patients showed improved cooperative behavior and lower clinical anxiety levels. However, using the Er:YAG laser resulted in a longer cavity preparation time. There was no discernible difference between the two groups' treatment success rates at the 12-month follow-up. In conclusion, the use of the Er:YAG laser decreases the requirement for intraoperative anesthetics and enhances treatment comfort and cooperation in children with caries as compared to the conventional dental turbine. Similar results were reported in other studies [78, 79].

A very recent review article, from 2025, studied the performance of lasers versus burs in randomized clinical trials [80]. After analyzing the literature, the authors concluded that compared to lasers, conventional burs showed considerably lower excavation times. The two approaches did not significantly differ in terms of pulp vitality or restoration survivability. In contrast, compared to traditional preparation methods, laser-based cavity preparation offered significant patient-centered benefits, such as a decreased requirement for anesthesia and a decreased sense of pain. When comparing laser preparation to traditional drilling, all clinical investigations that examined pain perception found that both adults and children had noticeably less discomfort. Anesthesia requirements were shown to be similar for both the adult population and children/adolescents, with fewer patients having laser cavity preparation needing anesthesia than those treated with the traditional rotary devices. In an attempt to address the time issue when comparing with conventional techniques, the authors propose using newer and faster laser systems with shorter pulse durations (<150 μs) and higher frequency.

## 5. Laser-based antimicrobial photodynamic therapy in caries management

Recently, laser-based antimicrobial photodynamic therapy (aPDT) has emerged as a promising minimally invasive adjunctive treatment modality of caries lesions. Laser-PDT utilizes a combination of a *photosensitizer* (PS), *laser light source*, and *tissue oxygen* to generate reactive oxygen species (ROS) that selectively target and destroy cariogenic microorganisms. Among the commonly used lasers, indium gallium aluminum phosphide (InGaAlP) and diode lasers have gained attention due to their suitable wavelengths (typically 630–660 nm) that efficiently activate photosensitizers such as methylene blue (MB), curcumin, and toluidine blue O (TBO) [81].

Clinical studies have demonstrated that laser-PDT protocols can achieve significant microbial reductions. Alves et al. conducted a randomized split-mouth trial in primary molars and reported reductions exceeding 90% of *S. mutans* colonies after treatment with diode laser-PDT, without negatively affecting restorative outcomes [82]. Similarly, Reis et al. supported the antimicrobial efficacy of PDT, highlighting its safety and potential to reduce reliance on systemic antibiotics [83]. Beyond individual studies, meta-analytic reviews have reinforced the role of PDT in managing deep carious lesions by significantly lowering bacterial loads within infected dentin [84]. This evidence supports the integration of laser-PDT into minimal intervention dentistry frameworks that prioritize tooth preservation and infection control [85]. Such innovations may improve the depth of bacterial eradication and overall clinical efficacy [86].

### 5.1 What is laser photodynamic therapy (laser-PDT)?

Laser photodynamic treatment is a minimally invasive procedure that incorporates [87]:

- A photosensitizer, a light-sensitive chemical substance that accumulates in particular cells or bacteria.
- Laser light source—typically a laser emitting a certain wavelength that activates the photosensitizer.

- Oxygen present in tissues reacts with the activated photosensitizer to produce reactive oxygen species (ROS). These reactive oxygen species facilitate targeted destruction of pathogens or diseased cells while safeguarding surrounding healthy tissue.

The Mechanism of Laser Photodynamic Therapy in the Treatment of Dental Caries [88]:

1. Targeting cariogenic bacteria: Dental caries predominantly arise from acidogenic bacteria such as *Streptococcus mutans* and *Lactobacilli* that inhabit tooth surfaces and establish biofilms. Laser-PDT employs a photosensitizer that specifically attaches to these microorganisms within the carious lesion.

2. Application of photosensitizer: A photosensitizing chemical, typically methylene blue or toluidine blue, is directly administered to the carious lesion. These chemicals infiltrate the biofilm and amass within bacterial cells.

3. Activation by laser light: A laser, calibrated to the absorption wavelength of the photosensitizer (often red light at 630–660 nm), is aimed at the lesion. The laser light stimulates the photosensitizer molecules, elevating them from a ground state to an excited one.

4. Generation of reactive oxygen species (ROS): The energized photosensitizer conveys energy to adjacent oxygen molecules, resulting in the formation of reactive oxygen species, including singlet oxygen and free radicals.

5. Bacterial cell destruction: Reactive oxygen species (ROS) are extremely reactive and induce oxidative damage to bacterial membranes, proteins, and DNA, thereby effectively eradicating cariogenic bacteria within the lesion. This procedure breaks the biofilm and decreases bacterial burden without damaging adjacent tooth structure or healthy tissues.

6. Supplement to traditional treatment: Laser-PDT may be utilized in conjunction with mechanical caries removal or as an independent antimicrobial intervention for early lesions, fostering minimally invasive dentistry by safeguarding tooth structure.

7. Advantages beyond antimicrobial activity: Decreases the necessity for antibiotics, hence mitigating the risks of resistance. Facilitates wound healing and reduces inflammation in surrounding tissues. Can be safely repeated without cumulative tissue damage.

Laser-PDT is a targeted and localized technique for caries control, focusing on germs while preserving healthy enamel and dentin. It is non-thermal, hence preventing thermal injury or discomfort. The effectiveness of photosensitizers depends on their type and concentration, requiring careful selection of laser parameters. They are chiefly beneficial for superficial infections and require specific methodologies and comprehensive longitudinal studies [89].

## 5.2 Photodynamic therapy (PDT) in diagnosis of carious lesions

The diagnostic application of PDT involves the use of photosensitizers that preferentially accumulate in carious tissues. Upon exposure to specific wavelengths of light, these photosensitizers emit fluorescence, which can be captured using specialized imaging systems. This fluorescence highlights areas of demineralization and microbial activity, allowing for the detection of early carious lesions and assessment of lesion depth and activity. For instance, a study by de Oliveira et al. [90] investigated the use of PDT-mediated fluorescence for detecting early carious lesions in primary teeth. The results indicated that PDT fluorescence imaging provided enhanced visualization of carious areas compared to traditional visual examination, facilitating early intervention and more accurate treatment planning [90].

Additionally, research by Diniz et al. [91] explored the application of PDT fluorescence in differentiating between active and arrested carious lesions. Their findings suggested that PDT fluorescence could serve as a valuable adjunct to conventional diagnostic methods, aiding clinicians in distinguishing between lesions that require treatment and those that may be monitored over time [91].

### *5.2.1 Limits and considerations*

PDT has limits in caries diagnosis despite its benefits. The kind and concentration of photosensitizer, light exposure settings, and restorative materials can affect PDT fluorescence. Maintaining consistent and trustworthy findings across clinical contexts requires standardizing these factors.

Gonçalves and Braga [92] examined advancements in fluorescence diagnostic instruments and emphasized the incorporation of PDT photosensitizers as a promising approach to enhance early caries diagnosis. They emphasized the necessity for consistent methods regarding photosensitizer selection, concentration, and light activation parameters to enhance diagnostic accuracy and repeatability [92].

In summary, photodynamic therapy-based fluorescence diagnostics provide a new and less invasive method that improves the early diagnosis and monitoring of carious lesions, as presented in subchapters 3.1 and 3.3. As data accumulates, PDT possesses the potential to enhance current diagnostic instruments, facilitating improved patient outcomes *via* prompt and targeted caries therapy.

## 5.3 PDT in preventive dentistry

Since dental caries represents a persistent condition resulting from a disruption in the equilibrium between the processes of tooth demineralization and remineralization, primarily influenced by acid generation from *Streptococcus mutans* found in dental plaque, eliminating dental plaque and the harmful bacteria it contains is crucial for halting the advancement of caries [93, 94]. Photodynamic antimicrobial chemotherapy (PACT), a specific form of photodynamic therapy (PDT), employs lasers with particular wavelengths to activate photosensitizers that specifically target and eliminate cariogenic bacteria while also disrupting biofilms [95].

Despite being overlooked in favor of antibiotics for many years, PDT has recently attracted significant attention owing to the increasing prevalence of multidrug-resistant microorganisms, particularly those found in biofilms that exhibit greater resistance to traditional therapies [96]. PDT induces microbial death through reactive oxygen species (ROS) like singlet oxygen ($^1O_2$), which inflicts damage on various bacterial targets, including extracellular molecules, thereby rendering the development of resistance highly improbable [97]. PDT damages biofilm polysaccharides, disrupting the matrix and enhancing antimicrobial effects beyond traditional antibiotics. With its multi-target action, low resistance risk, and ability to break down biofilms, PACT is a promising adjunct or alternative for managing oral infections of the teeth or skin [97–100].

Indications for use:

- *Pit and fissure decontamination prior to sealant placement*: PDT involves applying a photosensitizer into the fissures followed by activation with a low-level diode laser, leading to targeted bacterial destruction, especially *Streptococcus mutans* and *Lactobacilli*, without damaging enamel [98]. PDT may be especially useful on newly erupted molars where enamel is immature and fissures are deeper and more susceptible to decay. da Silva et al. demonstrated that pre-sealant disinfection using PDT significantly reduced the microbial load and improved sealant longevity compared to conventional cleaning alone [100].

- *Early non-cavitated carious lesions* (white spot lesions): PDT reduces bacterial load surrounding orthodontic appliances and local acid generation to treat and prevent white spot lesions non-invasively. PDT targets *Streptococcus mutans* and *Lactobacilli* to stop demineralization and promote remineralization, especially when paired with fluoride [101] and repeated every 4–6 weeks during orthodontic treatment, especially in patients with poor oral hygiene or visible decalcification, for reducing gingival inflammation and microbial levels without damaging appliances or enamel and arresting early enamel demineralization and contributed to the stabilization of white spot lesions [101, 102].

- *Part of minimally invasive caries therapy (MICT)*: PDT employs a photosensitizer that is activated by low-power laser light, generating reactive oxygen species that effectively eliminate residual bacteria in the prepared cavity, all while maintaining the integrity of collagen structure and mineral content [102], proving to be particularly beneficial in situations involving deep caries that are nearing the pulp, as complete excavation could potentially lead to exposure

- *High caries risk patients* (e.g., xerostomia, special needs, orthodontic): PDT is appropriate for partially erupted molars or moderate to high caries risk patients where sealants may not prevent decay. da Silva et al. found that PDT before sealant implantation in pediatric patients significantly reduced *S. mutans* colonies and enhanced sealant retention and effectiveness [100]. PDT can be incorporated into standard oral maintenance visits in geriatric care settings to decrease the presence of *Streptococcus mutans* and other cariogenic bacteria in high-risk areas, including root surfaces, cervical margins, and interproximal spaces. Patients with dementia, Parkinson's disease, or post-stroke disabilities, who may struggle with conventional hygiene practices, can benefit from PDT as a safe and painless alternative for periodic disinfection [103].

- *Adjunct to fluoride application*: Combining photodynamic therapy (PDT) with the fluoride treatments provides a synergistic effect in caries prevention by

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| Step | Procedure |
|---|---|
| 1. Assessment | Identify high-risk areas or lesions suitable for PDT (e.g., occlusal pits, white spots, around brackets). |
| 2. Preparation | Isolate the area; clean plaque/debris using a prophylaxis brush (no polishing paste needed). |
| 3. Photosensitizer application | Apply a suitable dye (e.g., methylene blue, toluidine blue O, erythrosine) to the lesion or fissure surface. Let it sit for 1–3 minutes. |
| 4. Laser activation | Use a low-level diode laser (e.g., 630–660 nm wavelength) to activate the photosensitizer for 30–60 seconds [98] |
| 5. Irrigation | Rinse thoroughly with water or air-water spray. |
| 6. Follow-up care | Proceed with fluoride varnish application, sealant placement, or restorative procedure, as indicated. |

**Table 1.**
*Clinical protocol for PACT in caries lesions.*

addressing both microbial elimination and enamel remineralization. When fluoride (in the form of varnish, gel, or solution) is applied afterward, it reduces acidity, helping fluoride ions to better restore the enamel surfaces and deeper areas. A photosensitizer is applied to these areas, allowing bacterial uptake. A low-power diode laser is then applied for 30–60 seconds, ensuring safety protocols. The area is thoroughly rinsed with sterile water or air-water spray to remove residual dye and debris. Topical fluoride is applied immediately, avoiding eating or drinking for 30-min post-application. Reapplication is recommended every 3 to 6 months, based on individual caries risk assessment as seen in **Table 1** [104].

### 5.4 Types of lasers and photosensitizers used in photodynamic therapy (PDT) for carious lesions

In treating dental cavities, laser-assisted photodynamic therapy (PDT) mainly uses low-level lasers to activate a photosensitizer that is applied to the decayed tooth material. The choice of laser depends on the photosensitizer's activation wavelength, tissue penetration needs, and safety considerations. Diode lasers are the most common, with wavelengths of 630–660 nm (red light), which are ideal for methylene blue (MB) or toluidine blue (TB).

The success of aPDT mainly relies on the photosensitizer (PS) used, which, when exposed to light at certain wavelengths, creates reactive oxygen species (ROS) that kill harmful bacteria like *Streptococcus mutans* and Lactobacillus spp. [105, 106].

The most common photosensitizers used for in dental PDT are as follows:

#### *5.4.1 Methylene blue (MB)*

- Type: Synthetic phenothiazinium dye
- Activation wavelength: 660 nm (red diode laser)
- Typical concentration: 0.01% (100 μg/mL)
- Mechanism: Produces singlet oxygen, penetrates deeply into dentinal tubules

- Advantages: Highly effective against *S. mutans* and other Gram-positive bacteria
- Disadvantages: Can cause visible blue staining of the dentin or enamel if not adequately rinsed
- Used extensively in clinical and pediatric settings [105, 107, 108]

*5.4.2 Toluidine blue O (TBO)*

- Type: Synthetic phenothiazinium dye (closely related to MB)
- Activation wavelength: 630–660 nm
- Typical concentration: 100 μg/mL – 0.1%
- Advantages: Less staining than MB; good antimicrobial activity
- Disadvantages: Slightly less depth of penetration than MB
- It is preferred in cases that are sensitive to esthetics [109].

*5.4.3 Curcumin*

- Type: Natural polyphenol from *Curcuma longa*

*5.4.4 Riboflavin (vitamin B2)*

- Type: Water-soluble vitamin [106]

Among available photosensitizers, methylene blue (MB) and toluidine blue (TB) are the most widely studied and clinically used in laser aPDT for caries. MB is preferred for its deep antimicrobial activity, especially in pediatric cases, while TB is more suitable for anterior or esthetic areas due to its minimal staining. Natural alternatives like curcumin and riboflavin show promise but require further clinical validation. MB is effective in pediatric settings where discoloration is less critical, while TB is favored for esthetic restorations due to lower staining (**Table 2**) [107, 110].

| Photo-sensitizer | Type | Wavelength (nm) | Color | Staining | Effectiveness | Clinical Use |
|---|---|---|---|---|---|---|
| Methylene Blue | Synthetic | 660 | Blue | High | Strong | Pediatric, deep caries |
| Toluidine Blue | Synthetic | 630–660 | Blue | Low | Very strong | Esthetic regions |
| Curcumin | Natural | 400–450 | Yellow | None | Moderate | Experimental |
| Riboflavin | Vitamin-based | 445–470 | Yellow | None | Low–Moderate | Research stage |

**Table 2.**
*Comparison table of photosensitizers.*

| Advantages | Limitations |
|---|---|
| • Minimally invasive | • Equipment cost |
| • Broad-spectrum antibacterial action | • Potential for discoloration (especially with MB) |
| • Pulp preservation | • Variability in protocols |
| • Improved bonding conditions *via* bacterial reduction | • Requires practitioner training |

**Table 3.**
*Advantages and limitations of laser photodynamic therapy.*

Incorporating laser aPDT into carious lesion treatment represents a promising step toward minimally invasive, biologically friendly dentistry. With proper case selection and adherence to protocol, it offers enhanced bacterial control and preservation of healthy dental tissues (**Table 3**). However, further standardization and long-term clinical trials are needed to optimize its routine use.

Innovative advancements like home-use dual-light devices and biocompatible natural photosensitizers suggest that the application of PDT is poised for significant growth in the near future. Nonetheless, the standardization of protocols, the training of practitioners, and an increase in randomized clinical trials are crucial for wider clinical implementation.

## 6. Conclusion

Various dental lasers are being used to create a wide range of novel treatments thanks to recent technological advancements and wavelengths. The contemporary practitioner must be conversant with these tools and comprehend their respective potential and constraints. Despite decades of research into the mechanism and clinical usage of lasers, their use has been somewhat questioned because of the potential for heat and cutting damage while increasing enamel's resistance to caries. The appropriate power and wavelength selection of different types of lasers in treating dental surfaces requires the right expertise and understanding in order to minimize the trauma of lasers to dental hard tissues while preventing caries. Optimizing caries control techniques and enhancing patient outcomes require ongoing education and technological adaption.

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## Conflict of interest

The authors declare no conflict of interest.

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## Acronyms and abbreviations

| | |
|---|---|
| Er:YAG | erbium-doped yttrium-aluminum-garnet |
| Er,Cr:YSGG | erbium, chromium-doped yttrium, scandium, gallium garnet |
| Laser | light amplification by stimulated emission of radiation |
| QLF | quantitative light-induced fluorescence |
| NIR | near-infrared |
| OCT | optical coherence tomography |
| LDF | laser Doppler flowmetry |
| CAD/CAM | computer-aided design/computer-aided manufacturing |
| Nd:YAG | neodymium-doped yttrium aluminum garnet |
| CBCT | cone beam computed tomography |
| ALARA | as low as reasonably achievable |
| AI | artificial intelligence |
| CNNs | convolutional neural networks |
| FACE | fluorescence-assisted caries excavation |
| FOTI | fiber optic trans-illumination |
| DIFOTI | digital fiber optic trans-illumination imaging |
| PBF | pulpal blood flow |
| MID | minimally invasive dentistry |
| DH | dentin hypersensitivity |
| aPDT | antimicrobial photodynamic therapy |
| PS | photosensitizer |
| ROS | reactive oxygen species |
| InGaAlP | indium gallium aluminum phosphide |
| MB | methylene blue |
| TBO | toluidine blue O |
| PACT | photodynamic antimicrobial chemotherapy |
| MICT | minimally invasive caries therapy |
| TB | toluidine blue |

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In the era of minimally invasive dentistry, the prevention and early detection of caries are central to maintaining long-term oral health. The book discusses risk assessment models, preventive strategies such as fluoride application and sealants, patient education, and the role of diet and oral hygiene. At the same time, it addresses advanced restorative concepts and materials, including adhesive systems, composite resins, glass ionomer cements, and novel bioactive restorative technologies that aim to preserve as much natural tooth structure as possible. Each chapter is carefully structured to bridge the gap between theory and clinical application. Through this integration of prevention and restoration, *Dental Caries -From Prevention to Restoration* aims to foster a holistic approach to oral health, prioritizing early intervention, minimally invasive principles, and lifelong maintenance of dental function and aesthetics. This book is intended for dental students, educators, and practitioners who seek a deeper understanding of caries management, from preventive approaches to comprehensive restorative care.

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