# **MODERN PEDIATRIC DENTISTRY** SCIENTIFIC ADVANCES IN THE PRESERVATION OF CHILDREN'S ORAL HEALTH





#### **BIDGE Publications**

# MODERN PEDIATRIC DENTISTRY: SCIENTIFIC ADVANCES IN THE PRESERVATION OF CHILDREN'S ORAL HEALTH

Editor: DOÇ. DR. SACIDE DUMAN

ISBN: 978-625-372-781-9

Page Layout: Gözde YÜCEL 1st Edition: Publication Date: 25.06.2025 BIDGE Publications,

All rights of this work are reserved. It cannot be reproduced in any way without the written permission of the publisher and editor, except for short excerpts to be made for promotion by citing the source.

Certificate No: 71374

Copyright © BIDGE Publications

www.bidgeyayinlari.com.tr - bidgeyayinlari@gmail.com

Krc Bilişim Ticaret ve Organizasyon Ltd. Şti.

Güzeltepe Mahallesi Abidin Daver Sokak Sefer Apartmanı No: 7/9 Çankaya / Ankara



#### CONTENTS

CARIES ALARM IN THE EARLY YEARS OF LIFE: EARLY CHILDHOOD CARIES WITH ITS CAUSES, CONSEQUENCES, AND SOLUTIONS
MERVE BİLMEZ SELEN4
HATİCE AYDOĞDU4
DENTAL EROSION IN CHILDREN
HATİCE AYDOĞDU32
BERİL DEMİRCAN32
REMINERALIZATION AGENTS IN PEDIATRIC DENTISTRY
BERİL DEMİRCAN59
MERVE BİLMEZ SELEN59
THE ROLE OF PROBIOTICS IN PEDIATRIC ORAL HEALTH: BALANCING THE ORAL MICROBIOTA IN CHILDREN85
FATMA NUR KIZILAY85
TURKAN MAHYADDINOVA85
BRUXISM IN CHILDREN: CLINICAL FINDINGS, RİSK FACTORS, AND MANAGEMENT STRATEGIES FOR PEDIATRIC DENTISTS
TURKAN MAHYADDINOVA98
FATMA NUR KIZILAY98

### CARIES ALARM IN THE EARLY YEARS OF LIFE: EARLY CHILDHOOD CARIES WITH ITS CAUSES, CONSEQUENCES, AND SOLUTIONS

## MERVE BİLMEZ SELEN<sup>1</sup> HATİCE AYDOĞDU<sup>2</sup>

#### Introduction

Dental caries is one of the most common chronic infectious diseases affecting all age groups and different socioeconomic levels. The presence of cariogenic bacteria, consumption of fermentable carbohydrates, inadequate oral hygiene, and unbalanced dietary habits, along with various social and environmental factors, are among the main contributors to caries formation (Su et al., 2018: 111; Anil et al., 2017: 157; Galán et al., 2017: 946). The prevalence of dental caries is higher in developing countries compared to developed ones and is more common in Asia than in Europe and Latin America (Anil et al., 2017: 157; Kassebaum et al., 2015: 658). According to the 2022 Global Oral Health Status Report, it is

 <sup>1</sup>Asst. Prof. Dr. Merve BİLMEZ SELEN, Inonu University, Faculty of Dentistry, Department of Pedodontics, Malatya/Türkiye. Orcid:0000-0003-0726-4992
<sup>2</sup>Asst. Prof. Dr. Hatice AYDOĞDU, Hacıbektaş Veli University, Department of Pedodontics, Nevşehir/Türkiye. Orcid:0000-0002-4089-3507 estimated that 514 million children worldwide have caries in their primary teeth (WHO, 2022).

Early Childhood Caries (ECC) is defined as the presence of at least one decayed, missing (due to caries), or filled tooth surface in any primary tooth in a child younger than 72 months (under 6 years). If smooth surface caries is observed in a child younger than 3 years, it is classified as Severe Early Childhood Caries (SECC). Furthermore, having dmft (decayed, missing, filled teeth) scores  $\geq$ 4 at age 3,  $\geq$ 5 at age 4, and  $\geq$ 6 at age 5 also qualifies as SECC (AAPD, 2018).

The global prevalence of ECC is approximately 48% (Uribe et al., 2021: 830). Although this disease is largely preventable, diagnosis and treatment are often delayed (Tinanoff et al., 2019: 248). Dental caries is an infectious disease that develops through the colonization of cariogenic microorganisms, frequent contact with fermentable carbohydrates, and host factors. *Streptococcus mutans* (MS) is the most common causative microorganism of ECC (Holve et al., 2021). The classical "caries triangle" consists of microbial agents, diet (especially sugar content), and host factors.

In the early stage of ECC, demineralization appears as white opaque lesions on the enamel surfaces of the upper anterior teeth. Although these lesions are reversible, without proper hygiene, they can quickly develop into cavities and lead to enamel fractures. As the disease progresses, teeth may remain at the root level and dentoalveolar abscesses can form (Baltaci, Baygin & Korkmaz, 2017: 202). If the caries progresses to the dentin or pulp tissue, pain and discomfort arise. Untreated ECC can negatively impact a child's sleep and feeding patterns and hinder growth and development (Chen et al., 2019). Additionally, tooth loss due to ECC can cause malocclusion and decrease oral health-related quality of life. Research shows that children with ECC tend to have lower body weight and height (Li et al., 2015: 63).

Untreated ECC can lead to consequences such as hospitalization, emergency dental visits, school absenteeism, and reduced academic performance (Neves et al., 2016: 72; Allareddy et al., 2014: 399). In severe cases, these complications can even become life-threatening (Chen et al., 2019). Therefore, ECC is not merely an oral health issue but also a systemic problem that affects overall health and quality of life.

Global data indicate that a significant proportion of children under six years of age are affected by ECC. Despite a decrease in caries prevalence among children in Western countries, ECC remains a major public health concern during the preschool years in both developed and developing countries (Anil & Anand, 2017: 157). The prevalence of ECC varies based on factors such as race, ethnicity, socioeconomic status, lifestyle, dietary habits, and oral hygiene practices. Literature reports that ECC prevalence in developed countries generally ranges from 1% to 12% (Congiu, Campus & Lugliè, 2014: 76). Studies conducted in various countries between 2010–2015 reported prevalence rates of 11–21.2%, while studies from 2016-2020 reported rates ranging from 4.3-8.2% (Wigen et al., 2011: 317; Mantonanaki et al., 2013: 79; Olatosi et al., 2015: 501; Nobile et al., 2014: 12; Folayan et al., 2020: 804; Colombo et al., 2019: 273). In a comprehensive study evaluating 193 countries from 2007–2017, ECC prevalence was reported at 23.8% in children under 36 months and 57.3% in those aged 36–71 months (El et al., 2017: 1072).

In Turkey, studies conducted over the past decade show that ECC prevalence ranges from 17.7% to 63.1% (Bilmez et al., 2024: 243; Aydınoğlu & Kuşgöz, 2019: 596; Ozer et al., 2011: 103; Doğan et al., 2013: 325). Prevalence can reach as high as 70% in

underdeveloped countries and disadvantaged groups in developed nations. For example, rates have been reported as 11.4% in Sweden, 7–19% in Italy, 76% in Palestine, and 83% in the United Arab Emirates (Nobile et al., 2014: 12; Strömberg et al., 2012: 7; Azizi, 2014: 839419; El, Hassab & Al, 2010: 55). Other countries such as Greece (36%), Brazil (45.8%), India (51.9%), and Israel (64.7%) also report significant ECC prevalence (Anil & Anand, 2017: 157; Oulis et al., 2012: 32; Koya et al., 2016: 251; Ismail & Sohn, 1999: 191).

European studies show average dmft scores between 0.9–2.0 in Germany, the UK, and Italy, while in Indonesia, this score can be as high as 7.5 (Chen et al., 2019). In Australia, ECC prevalence is 44.4%, while in Sudan it is reported at 56%. A study in Turkey by Kuvvetli et al. found a caries prevalence of 45.7% in 300 five-year-olds with an average dft (decayed, filled teeth) score of 1.93 (Kuvvetli et al., 2008: 163). In another study conducted by Bilmez Selen et al. in 2024, the SECC rate was found to be 59.8% with an average dmft score of 5.75 (Bilmez et al., 2024: 243).

All these findings indicate that ECC is a highly contagious, serious infectious disease and still constitutes a significant public health problem globally.

Etiological Factors for ECC

#### **Host-Related Factors**

In the etiology of ECC, the child's biological and behavioral characteristics play an important role. These factors include:

- Low salivary flow during the night,
- Newly erupted immature permanent teeth,
- Hypoplastic defects commonly seen in primary teeth,
- Certain systemic and medical conditions.

Conditions such as maternal stress during pregnancy, health problems, complications during childbirth, postnatal illnesses, and inadequate nutrition of the baby can lead to developmental defects in the teeth. These defects make the tooth surface more permeable and vulnerable, increasing susceptibility to caries. In addition, individual factors such as the child's oral hygiene habits, dietary structure, past dental history, genetic predisposition, and access to preventive dental care also determine the risk of ECC (WHO, 2017).

Although the etiology of ECC resembles that of smooth surface caries, some biological differences exist. In young children, the oral microbiota is not fully developed, and their immune system is more vulnerable compared to adults. Moreover, newly erupted teeth appear with immature enamel, which completes its full mineralization over time after eruption. Therefore, the period between eruption and full maturation is when teeth are most susceptible to caries (WHO, 2017).

Teeth with developmental defects have a higher risk of developing caries (Bilmez Selen & M, 2024: 218). Studies have shown that the majority of children with widespread caries experienced various medical issues during infancy. Children with chronic illnesses may be prone to enamel hypoplasia. Additionally, these children are often fed with bottles containing sugary liquids or exposed to sweetened medications. Especially antihistamines and  $\beta$ -adrenoceptor-containing drugs reduce salivary flow, causing xerostomia, which increases caries risk (WHO, 2017).

Although some studies suggest that antibiotics may reduce caries risk, there is no conclusive evidence. On the contrary, commonly used pediatric agents such as iron supplements, sugarcontaining antibiotic suspensions, and powdered inhalation medications can increase the risk of caries (WHO, 2017).

#### **Microbial Factors**

- Early colonization of mutans streptococci (MS)
- Lack of oral hygiene habits

MS plays an active role in the development of carious lesions, especially in the initial stages. Transmission of these microorganisms to the baby typically occurs from the mother or caregiver upon tooth eruption. Vertical transmission of infected saliva from mothers with untreated caries can infect the baby. This is associated with actions such as kissing the baby on the mouth or sharing utensils like spoons and cups. For example, testing the temperature of food with their mouth and feeding the baby with the same spoon poses a risk for MS transmission. Parents or caregivers should be educated on this matter. It is extremely important for parents to maintain their own oral and dental health. Therefore, in addition to brushing, the use of antibacterial agents like chlorhexidine and fluoride, and chewing xylitol-containing gum, is especially important in the neonatal period when the mother-child interaction is intense. More than 30% of the plaque flora in children with ECC consists of MS. Frequent and often constant exposure to cariogenic foods increases the microbial load to pathogenic levels. The "window of infectivity" associated with initial MS exposure, usually between 19-31 months, has been reported to occur even earlier, possibly from the 6th month onward (WHO, 2017; Douglass & Clark, 2015: 274).

#### **Environmental Factors**

Fermentable and low molecular weight carbohydrates are more cariogenic than complex carbohydrates. For example, sucrose is more cariogenic than glucose, lactose, and fructose. Saliva, which has a cleansing effect on tooth surfaces, supports remineralization through protective factors such as flow rate, buffering capacity, salivary proteins, and mineral content. Low salivary flow rate, cariogenic diet, nighttime sugar intake, and inadequate oral hygiene are associated with ECC (Douglass & Clark, 2015: 274; AAPD, 2016: 54). Parenting style, socioeconomic status, and parental education level are also closely related to ECC (AAPD, 2016: 54).

#### Infant and Child Nutrition

- Frequent nighttime feeding with sugary drinks, milk, or formula
- Frequent consumption of sugary beverages and soft foods
- Use of bottles, pacifiers, and similar sucking habits
- Prolonged use of bottles and/or breastfeeding are risk factors for ECC.

The United Nations Children's Fund (UNICEF) has reported that 101 million children under the age of five are undernourished, 165 million show stunted growth, 45 million are obese or overweight, and over 30% of children under five suffer from vitamin deficiencies (UNICEF, 2013). Furthermore, it is stated that breastfeeding and complementary feeding could reduce the mortality rate in children under five by 19%. Breast milk is the preferred and supported method of feeding for infants. Breastfeeding for the first six months and continuing alongside complementary foods until of two provides immunological, psychological, the age developmental, social, economic, and environmental benefits for both infants and mothers. According to the American Academy of Pediatrics (AAP), breastfeeding is considered the ideal method of nutrition. It is stated that in children who are breastfed for longer periods, the consumption of sugary foods is delayed, reducing the risk of caries (UNICEF, 2013; Melis, Güven & Aktören, 2015: 70).

However, many studies comparing the cariogenic potential of cow's milk and breast milk have found that breast milk contains

higher lactose and lower mineral and protein content. Additionally, breast milk causes a greater pH drop than cow's milk, making it more cariogenic. Some researchers have suggested that frequent night breastfeeding or breastfeeding seven or more times per day after the age of one can increase both the likelihood and frequency of ECC (Weber-Gasparoni et al., 2007: 61). While breastfeeding is the best way to provide optimal nutrition to infants, the AAPD warns that frequent nighttime breastfeeding after tooth eruption may contribute to ECC development (AAPD, 2016: 54).

Feeding with a bottle is a contributing factor to the development of ECC. The nipple part of the bottle prevents saliva from reaching the upper incisors. With decreased salivary flow and buffering capacity at night, the use of bottles leads to prolonged exposure to fermentable carbohydrates in the mouth. Additionally, children with ECC often have less sleep, wake up easily, and are fed more often at night to reduce sleep disturbances (Melis, Güven & Aktören, 2015: 70). Results from the 1991 National Health Interview Survey's Child Supplement show that the majority (95%) of children aged 6 months to 5 years had a history of bottle use, and about 16.6% had a history of nighttime bottle use with contents other than water (Kaste & Gift, 1995: 791). Another study linked the use of infant formula with early and high levels of MS colonization, a risk factor for ECC (O'Sullivan & Tinanoff, 1993: 1580). Although prolonged and especially nighttime bottle feeding increases caries risk, bottle use alone is not the only effective factor in early childhood caries development.

The effect of sugar on caries development is related to socioeconomic status, frequency of feeding, and oral hygiene habits. The World Health Organization recommends reducing sugar intake throughout life and suggests that it should not exceed 10% of total energy intake for both adults and children. Complementary foods introduced after the first six months of breastfeeding should be energy-dense and meet 90% of the infant's iron requirements. Sweetened foods should never be added to bottles or pacifiers. Foods and beverages containing free sugars should be limited to main meals (WHO, 2017). High consumption of sugary snacks and drinks between meals, eating more than six times a day, extended breastfeeding beyond 24 months, eating before sleep, and nighttime feeding are dietary factors that increase ECC risk (Pierce et al., 2019: 328; Tsang et al., 2019: 2456).

Sugar consumption is one of the most significant risk factors in the development of ECC. Therefore, many studies have evaluated the relationship between the frequency and amount of sugar intake and ECC. Some of these studies have shown statistically significant correlations between increased frequency and quantity of sugar intake and ECC. For example, Warren et al. (2008: 75) reported that the consumption of sugar-sweetened beverages was associated with ECC in a study involving 128 children aged 18 months. On the other hand, other studies have shown weak or no correlation between sugar consumption frequency and ECC. For instance, Gibson et al. (1999: 113) examined the relationship between sugary diets and caries experience in 1.5 to 4.5-year-old children in England and found no correlation between caries experience and the percentage of energy intake from each sugary diet.

#### **Social and Behavioral Factors**

Parental education

Socioeconomic status are etiological factors for ECC.

Family income, parents' educational level, number of children in the family, ethnicity, and social environment affect the development of ECC. The acquisition of consistent behavioral habits begins at home during childhood. Parents serve as role models for their children. It is unrealistic to expect good oral hygiene from children whose parents have poor oral hygiene habits. Therefore, parents should be informed that their oral health habits directly affect their children's oral health and, thus, their quality of life (AAPD, 2016: 54). Children generally lack the knowledge and experience needed for proper oral hygiene and cannot effectively brush their teeth. Hence, parents and caregivers play a crucial role in promoting their children's oral health. Evidence shows that parental involvement during the perinatal period is essential for effectively preventing ECC (Chen et al., 2019).

#### ECC Prevention Strategies

#### **Prenatal Oral Health Care**

Pregnancy is an ideal time to promote the primary prevention of ECC in children, given the profound impact of maternal health and behaviors on children's oral health outcomes. ECC is a multifactorial bacterial disease caused primarily by the cariogenic bacterium *Streptococcus mutans* and is strongly influenced by diet. The presence of untreated maternal caries and high levels of MS in saliva have been associated with an increased risk of ECC in children. Children's nutrition and oral hygiene behaviors are influenced by the oral health knowledge and habits of their parents or caregivers (Xiao et al., 2019: 421).

Initiating ECC prevention during the prenatal period is optimal. Considering the evidence of maternal-to-child transmission of cariogenic bacteria, routine oral health evaluations, preventive dental care, oral hygiene education, optimal prenatal nutrition, and the use of fluoride toothpaste by pregnant women are strategies that can help prevent ECC in their children (Dentistry, 2016: 154). Recent guidelines have concluded that dental care during pregnancy is necessary to protect the child from caries-causing microorganisms (California Dental Association Foundation, 2010: 440; Kumar & Samelson, 2006).

#### **Oral Health Education and Community-Based Strategies**

Through public health education events and practices, community awareness of oral health should be increased to develop appropriate behaviors and reduce emergency visits to dental centers, thereby fostering healthier generations. The public should be informed about the importance of preventive oral and dental care. Education during the prenatal period, guidance for families of infants regarding minimizing sugary drink consumption, avoiding adding sugar to bottle-fed liquids—especially at night—and cleaning the baby's gums with a soft brush or moist gauze from birth will help instill lifelong healthy oral hygiene habits (Baltaci, Baygin & Korkmaz, 2017: 202).

Dental and pediatric associations approve the safe and effective use of fluoride in caries prevention (Holve et al., 2021; Ajiboye et al., 2018:1296; The use of fluoride in infants and children, 2002: 582; Preventive oral health intervention for pediatricians, 2008). All mentioned organizations recommend that children brush their teeth twice daily with fluoride toothpaste. Children under two years should use a smear (rice-sized amount) of 1000 ppm fluoride toothpaste, and children aged 2 to 6 years should use a pea-sized amount of 1450 ppm fluoride toothpaste (Holve et al., 2021; Toumba et al., 2019: 516).

Community water fluoridation is safe, effective, inexpensive, and does not require daily compliance (Riley, Lennon & Ellwood, 1999: 305). In populations consuming fluoridated water, a 40% reduction in caries incidence has been observed (CDC, 2011). Professionally applied topical fluorides have also been shown to be effective in caries prevention (Holve et al., 2021). These fluoride applications come in varnish and gel forms. Commonly used gel agents include 2% sodium fluoride (NaF), 1.32% acidulated phosphate fluoride (APF), and 8–10% stannous fluoride (SnF). Fluoride varnishes typically contain 5% NaF (22,000 ppm fluoride ion). Professional fluoride applications should be administered to children at high and moderate risk of caries. Frequency depends on risk category but usually requires application at least once every six months. Due to ease of application and low swallowing risk, fluoride varnishes are preferred especially for younger children. For children at high risk, mouth rinses and dental floss are recommended in addition to brushing. However, mouth rinses are not recommended for children under five due to the risk of swallowing (AAPD, 2020: 243).

Improving ECC prevention strategies requires understanding how social class or living environment can positively or negatively affect health. One correlation of individual class status is the type of area a person is likely to inhabit. While it may not be feasible to make everyone middle class, it is possible to improve the social and physical environments of poor areas through health-promoting efforts. Therefore, local and national health promotion policies should directly address features of local, social, and physical environments that promote or threaten health, considering both place characteristics and individual traits.

Traditional restorative and surgical management of ECC often requires general anesthesia, is costly, and does not prevent caries; it only treats the outcome of the disease. A recurrence rate of 79% has been reported in patients treated with conventional methods. Therefore, ECC management focuses on preventive measures such as parental involvement, active surveillance, preventive programs, interim restorations, and deferral of extensive restorations (Tungare & Paranjpe, 2021).

#### Oral Hygiene Habits and Their Relationship with ECC

Maintaining good oral health from an early age is critically important for children's development, overall health, and well-being (Selwitz, Ismail & Pitts, 2007: 59). Epidemiological studies have revealed that dental caries is the most common chronic disease worldwide and represents a costly burden on health services (Elamin, Garemo & Gardner, 2018: 9).

Dental caries is a multifactorial disease with numerous contributing risk factors for its initiation and progression. These risk factors can be categorized as biological, environmental, or sociobehavioral (Selwitz, Ismail & Pitts, 2007: 59). During the preschool period, excessive sucrose intake, consumption of sweetened beverages, high sugar intake during snacks, and frequent snacking are associated with dental caries (WHO, 2017). The quality of a child's oral hygiene habits and the family's ability to limit cariogenic snacks are also important determinants for dental caries (Elamin, Garemo & Gardner, 2018: 9).

Numerous studies have evaluated the impact of tooth brushing and the use of fluoride toothpaste on the prevalence and incidence of ECC. Many of these studies have shown that tooth brushing is a significant risk-related factor in ECC. Peltroche and colleagues (2015) found that among children aged 3 to 6 years with high caries risk, inadequate oral hygiene and lack of dental visits were the most common contributing factors. In Taiwan, 981 children under 6 years were examined by three pediatric dentists using WHO criteria. Multiple logistic regression analyses showed a statistically significant difference in ECC prevalence between children who brushed their teeth before bed every night and those who did not (p<0.05).

A 2009 case-control study conducted in Australia by Seow and colleagues (2009: 35) examined ECC risk factors including maternal psychological influences in 617 children aged 0–4 years. The sample consisted of 461 children without ECC and 156 children with ECC, selected from childcare facilities and public/private dental clinics. Children were evaluated for ECC, enamel hypoplasia, and MS presence. Meanwhile, mothers were asked to complete dental and psychological questionnaires, as well as provide medical, dietary, and oral hygiene histories. A multivariate logistic modeling approach was used to analyze the data. The results indicated that children without ECC had fewer problems related to tooth brushing compared to those with ECC (p=0.01).

#### Socioeconomic Status and Oral Health Relationship

National statistics in the United States report that ECC is most prevalent among children from families with low socioeconomic status (SES) (Dye et al., 2007). Moreover, national surveys have found that ECC is more common among specific racial and ethnic groups such as African Americans, Hispanics, and Native Americans (Dye et al., 2007).

Many studies have been conducted to evaluate the relationship between SES and ECC prevalence. Some of these studies have shown statistically significant relationships. For example, Warren et al. (2008: 75) evaluated the association between annual income and ECC in 212 one-year-old children. This study, published in 2008, showed that children from low-income households had a higher prevalence of ECC (p=0.04).

In a 2020 study conducted in Turkey by Yiğit et al. investigating the relationship between ECC and oral hygiene status in 530 children, it was found that children of civil servant parents had lower dmfs (decayed, missing, filled surfaces) scores, while children of unemployed and low-income families had higher dmfs scores (p<0.001). As parents' education levels increased, children's dmfs scores decreased, toothbrushing frequency increased, and the age of initiating brushing decreased (p<0.001) (Yiğit & Küçükeşmen, 2020: 372). On the other hand, Kumarihamy et al. (2011: 6) conducted a cross-sectional study to evaluate ECC prevalence in 422 children aged 1 to 2 years who visited health centers in semi-urban areas of Colombo, Sri Lanka. Relationships between mean dmft scores (classified by presence or absence of cavities) and various sociodemographic factors were assessed. A one-way ANOVA showed no statistically significant difference in mean dmft scores among lowincome (1.52), middle-income (2.28), and high-income (1.50) groups (p=0.10).

#### Systemic Disease and Oral Health Relationship

Bone metabolism and the development of teeth are closely related to systemic health; systemic diseases can affect the formation and regulation of these tissues. Often, the first signs of systemic diseases may appear in the oral cavity. As systemic illnesses impact the body's metabolic, endocrine, and circulatory systems, they can also affect the teeth, jaws, oral mucosa, and the temporomandibular joint (TMJ). In addition to providing structural support, bones serve as a calcium reservoir for the hematopoietic system. Disorders of calcium metabolism affect the entire skeletal system, including the jaws. Likewise, the stages of tooth formation, development, mineralization, and eruption are governed by systemic factors (Yıldırım & Bilgir, 2017).

The relationship between oral diseases and general health is multifaceted and complex. Systemic diseases can directly impact oral health through pathological mechanisms or indirectly through behavioral changes related to illness or treatment. Changes in oral health can, in turn, affect systemic health. Tooth loss is closely associated with increased all-cause mortality, cardiovascular diseases, and reduced quality of life. Components of pathogenic biofilm can enter the body via the periodontal ligament from inflamed areas or be inhaled or ingested, thus increasing the risk of pneumonia and gastritis (Dörfer et al., 2017: 18).

Bacteremia defined as the presence of bacteria in the bloodstream is a risk factor for endocarditis and can be triggered by any mechanical effect on the skin or mucosa. The frequency and intensity of bacteremia originating from the oral cavity depend on the invasiveness of the mechanical effect and the degree of inflammation at the interface between hard and soft tissues. Although bacteremia does not typically lead to endocarditis, even in high-risk patients, the probability increases with the frequency and intensity of bacteremia (Dörfer et al., 2017: 18).

The relationship between diabetes mellitus and periodontitis has long been recognized, with periodontitis considered one of the complications of uncontrolled diabetes mellitus. Moreover, acute inflammation is known to impair glycemic control. This occurs because infections reduce glucose uptake by cells and because endotoxins and inflammatory mediators reduce insulin effectiveness. For these reasons, periodontitis is regarded as a risk factor for prolonged poor glycemic control in patients with diabetes (Dörfer et al., 2017: 18).

Various studies have shown that long-term use of beta-2 agonists in asthma treatment reduces salivary flow (Thomas et al., 2010: 133). In one study, Sağ et al. (2007: 2242) found that one month after initiating treatment with a beta-2 agonist and inhaled corticosteroid in asthmatic children, there was a significant decrease in salivary flow and a significant increase in plaque indices on buccal tooth surfaces. Asthmatic patients may also experience increased oral dryness due to bronchodilator use and mouth breathing. Changes in salivary flow rate and content can increase the risk of erosion and caries (Thomas et al., 2010: 133). Ryberg et al. (1991: 218) attributed the high caries risk in asthmatic children treated with beta-2 agonists to increased levels of lactobacilli and mutans streptococci, as well as reduced salivary flow. Furthermore, the fermentable carbohydrates found in asthma medications are believed to contribute to the increased frequency of dental caries (Thomas et al., 2010: 133; Aral, Aral & Kalkan, 2016: 46).

#### **Conclusion and Recommendations**

Early childhood caries (ECC) is a preventable but contagious disease that, if left untreated, can lead to serious complications not only on an individual level but also with significant impacts at the societal level. This book chapter has thoroughly examined the causes, consequences, and preventive measures for ECC. Individuals with low socioeconomic status and communities with inadequate oral hygiene are particularly at risk. Future research should emphasize individual awareness and family-based prevention strategies. Additionally, community-wide education programs should be expanded, and epidemiological studies should be encouraged to better understand the relationship between systemic diseases and oral health.

#### **Recommendations for ECC Prevention:**

- Avoid putting babies to sleep with a bottle in their mouth at night
- Eliminate the habit of feeding to sleep
- Do not add sweeteners such as sugar, honey, or molasses to milk in bottles
- Ensure that babies or children drink water after feeding
- Once the first teeth erupt, clean them after nighttime and morning feedings
- Avoid constant feeding outside of main and snack meals; reduce snack frequency to allow saliva buffering

- Reduce the frequency and amount of sugar-containing food consumption (Tungare & Paranjpe, 2021; Pierce et al., 2019: 328; Tsang et al., 2019: 2456)
- Begin regular dental check-ups with the eruption of the first tooth
- Impact on Quality of Life (ECOHIS Scale): Various scales have been developed to assess the impact of ECC on children's quality of life. One of the most widely used is the "Early Childhood Oral Health Impact Scale" (ECOHIS). ECOHIS is a 13-item questionnaire designed to evaluate the impact of children's oral health on both themselves and their parents. It includes factors such as child symptoms, functional limitations, psychological effects, and social interactions. Since it relies on parental reporting, it is suitable for young children and is also effective in monitoring post-treatment changes (Kurt et al., 2025: 210).

#### References

Su, H., Yang, R., Deng, Q., Qian, W., & Yu, J. (2018) Deciduous dental caries status and associated risk factors among preschool children in Xuhui District of Shanghai, China. *BMC Oral Health, 18*(1), 111. https://doi.org/10.1186/s12903-018-0565-8

Anil, S., & Anand, P. S. (2017) Early childhood caries: Prevalence, risk factors, and prevention. *Frontiers in Pediatrics*, *5*, 157. https://doi.org/10.3389/fped.2017.00157

Galán, C. A., Shaw, D. S., Dishion, T. J., & Wilson, M. N. (2017) Neighborhood deprivation during early childhood and conduct problems in middle childhood: Mediation by aggressive response generation. *Journal of Abnormal Child Psychology*, 45(5), 935–946. https://doi.org/10.1007/s10802-016-0209-x

Kassebaum, N. J., Bernabé, E., Dahiya, M., Bhandari, B., Murray, C. J., & Marcenes, W. (2015) Global burden of untreated caries: A systematic review and metaregression. *Journal of Dental Research*, 94(5), 650–658. https://doi.org/10.1177/0022034515573272

World Health Organization. (2022) *Global Oral Health Status Report: Towards Universal Health Coverage for Oral Health by 2030.* https://www.who.int/publications/i/item/9789240061484

American Academy of Pediatric Dentistry(AAPD). (2018) Policy on early childhood caries (ECC): Classifications, consequences, and preventive strategies. *Pediatric Dentistry*, 40(6), 60–62.

Uribe, S. E., Innes, N., & Maldupa, I. (2021) The global prevalence of early childhood caries: A systematic review with metaanalysis using the WHO diagnostic criteria. *International Journal of Paediatric Dentistry*, *31*(6), 817–830. https://doi.org/10.1111/ipd.12783. Tinanoff, N., Baez, R. J., Diaz Guillory, C., Donly, K. J., Feldens, C. A., McGrath, C., ... Twetman, S. (2019) Early childhood caries: Epidemiology, aetiology, risk assessment, societal burden, management, education, and policy: Global perspective. *International Journal of Paediatric Dentistry*, *29*(3), 238–248. https://doi.org/10.1111/ipd.12484.

Holve, S., Braun, P., Irvine, J. D., Nadeau, K., Schroth, R. J., Bell, S. L., ... & Fraser-Roberts, L. (2021) Early childhood caries in indigenous communities. *Pediatrics*, *147*(6), e2021051481.

Baltaci, E., Baygin, Ö., & Korkmaz, F. M. (2017) Erken Çocukluk Çağı Çürükleri: Güncel

Literatür Derlemesi. Turkiye Klinikleri. Dishekimligi Bilimleri Dergisi, 23(3), 191-202.

Chen, K. J., Gao, S. S., Duangthip, D., Lo, E. C., & Chu, C. H. (2019) Prevalence of early childhood caries among 5-year-old children: A systematic review. *Journal of investigative and clinical dentistry*, *10*(1), e12376.

Li L-W, Wong HM, Peng S-M, McGrath CP. (2015) Anthropometric measurements and dental caries in children: a systematic review of longitudinal studies. Advances in Nutrition,6(1):52-63.

Neves ÉTB, Firmino RT, de França Perazzo M, Gomes MC, Martins CC, Paiva SM, et al. (2016) Absenteeism among preschool children due to oral problems. Journal of Public Health,24(1):65-72.

Allareddy V, Nalliah RP, Haque M, Johnson H, Rampa SB, Lee MK. (2014) Hospital-based emergency department visits with dental conditions among children in the united states: nationwide epidemiological data. Pediatric dentistry,36(5):393-9.

Anil S, Anand PS. (2017) Early childhood caries: prevalence, risk factors, and prevention. Frontiers in pediatrics,5:157.

Congiu G, Campus G, Lugliè PF. (2014) Early childhood caries (ecc) prevalence and background factors: a review. Oral Health Prev Dent,12(1):71-6.

Wigen TI, Espelid I, Skaare AB, Wang NJ. (2011) Family characteristics and caries experience in preschool children. A longitudinal study from pregnancy to 5 years of age. Community dentistry and oral epidemiology, 39(4):311-7.

Mantonanaki M, Koletsi-Kounari H, Mamai-Homata E, Papaioannou W. (2013) Prevalence of dental caries in 5-year-old greek children and the use of dental services: evaluation of socioeconomic, behavioural factors and living conditions. International dental journal,63(2):72-9.

Olatosi O, Inem V, Sofola O, Prakash P, Sote E. (2015) The prevalence of early childhood caries and its associated risk factors among preschool children referred to a tertiary care institution. Nigerian journal of clinical practice, 18(4):493-501.

Nobile CG, Fortunato L, Bianco A, Pileggi C, Pavia M. (2014) Pattern and severity of early childhood caries in southern italy: a preschool-based cross-sectional study. BMC Public Health,14(1):1-12.

Folayan MO, Oginni AB, El Tantawi M, Alade M, Adeniyi AA, Finlayson TL. (2020) Association between nutritional status and early childhood caries risk profile in a suburban nigeria community. International journal of paediatric dentistry,30(6):798-804.

Colombo S, Gallus S, Beretta M, Lugo A, Scaglioni S, Colombo P, et al. (2019) Prevalence and determinants of early

childhood caries in italy. European journal of paediatric dentistry,20(4):267-73.

El Tantawi M, Folayan MO, Mehaina M, Vukovic A, Castillo JL, Gaffar BO, et al (2017). Prevalence and data availability of early childhood caries in 193 united nations countries, 2007–2017. American journal of public health,108(8):1066-72.

Bilmez Selen M, Demir P, Eden E, Inceoğlu F. (2024) Relationship between parental adverse childhood experiences and the prevalence of early childhood caries. Clinical Oral Investigations, 28(5), 243.

Aydinoğlu S, Kuşgöz A. (2019) Prevalence of early childhood caries and associated risk factors among 3-6-year-old children in trabzon. Atatürk Üniversitesi Diş Hekimliği Fakültesi Dergisi,29(4):589-96.

Ozer S, Sen Tunc E, Bayrak S, Egilmez T (2011). Evaluation of certain risk factors for early childhood caries in samsun, turkey. European journal of paediatric dentistry. 2011,12(2):103.

Doğan D, Dülgergil ÇT, Mutluay AT, Yıldırım I, Hamidi MM, Çolak H. (2013) Prevalence of caries among preschool-aged children in a central anatolian population. Journal of natural science, biology, and medicine,4(2):325.

Early childhood caries in low-income african american young children. Pediatric dentistry,30(4):289-96.

Strömberg U, Holmn A, Magnusson K, Twetman S. (2012) Geo-mapping of time trends in childhood caries risk a method for assessment of preventive care. BMC Oral Health. 2012,12(1):1-7. Azizi, Z. (2014). The Prevalence of Dental Caries in Primary Dentition in 4-to 5-Year-Old Preschool Children in Northern Palestine. *International journal of dentistry*, 2014(1), 839419.

El Nadeef M, Hassab H, Al Hosani E. (2010) National survey of the oral health of 5-year-old children in the united arab emirates. EMHJ-Eastern Mediterranean Health Journal, 16 (1), 51-55.

Oulis C, Tsinidou K, Vadiakas G, Mamai-Homata E, Polychronopoulou A, Athanasouli T. (2012) Caries prevalence of 5, 12 and 15-year-old greek children: a national pathfinder survey. Community dental health,29(1):29-32.

Koya S, Ravichandra K, Arunkumar VA, Sahana S, Pushpalatha H. (2016) Prevalence of early childhood caries in children of west godavari district, andhra pradesh, south india: an epidemiological study. International journal of clinical pediatric dentistry,9(3):251.

Ismail AI, Sohn W. (1999) A systematic review of clinical diagnostic criteria of early childhood caries. Journal of public health dentistry,59(3):171-91.

Kuvvetli S, Cildir S, Ergeneli S, Sandalli N. (2008). Prevalence of noncavitated and cavitated carious lesions in a group of 5-year-old turkish children in kadikoy, istanbul. Journal of Dentistry for Children, 75(2):158-63.

World Health Organization. (2017) WHO expert consultation on public health intervention against early childhood caries: report of a meeting, Bangkok, Thailand, 26-28 January 2016. In WHO expert consultation on public health intervention against early childhood caries: report of a meeting, Bangkok, Thailand, 26-28 January 2016. Bilmez Selen M B , Demir P, & Inceoglu F. (2024) Evaluation of possible associated factors for early childhood caries: are preterm birth and birth weight related? BMC Oral Health, 24(1), 218.

Douglass JM, Clark MB. (2015) Integrating oral health into overall health care to prevent early childhood caries: need, evidence, and solutions. Pediatric dentistry,37(3):266-74.

AAPD. (2016) Policy on early childhood caries (ECC): classifications, consequences, and preventive strategies. Pediatr Dent,38:52-4.

New York (NY): UNICEF. (2013) Improving child nutrition: the achievable imperative for global progress.

Feldens CA, Giugliani ERJ, Duncan BB, Drachler MdL, Vítolo MR. (2010) Long-term effectiveness of a nutritional program in reducing early childhood caries: a randomized trial. Community dentistry and oral epidemiology, 38(4):324-32.

Melis A, GÜVEN Y, AKTÖREN O. (2015) Bebeklerde beslenme modelleri ve erken çocukluk çaği çürükleri. Atatürk Üniversitesi Diş Hekimliği Fakültesi Dergisi,25:64-70.

Erickson PR, Mazhari E. (1999) Investigation of the role of human breast milk in caries development. Pediatric Dentistry,21:86-90.

Weber-Gasparoni K, Kanellis MJ, Levy SM, Stock J. (2007) Caries prior to age 3 and breastfeeding: a survey of la leche league members. Journal of Dentistry for Children,74(1):52-61.

Kaste LM, Gift HC. (1995. Inappropriate infant bottle feeding: status of the healthy people 2000 objective. Archives of pediatrics & adolescent medicine,149(7):786-91.

O'sullivan D, Tinanoff N. (1993) Maxillary anterior caries associated with increased caries risk in other primary teeth. Journal of dental research,72(12):1577-80.

Pierce, A., Singh, S., Lee, J., Grant, C., Cruz de Jesus, V., & Schroth, R. J. (2019) The burden of early childhood caries in Canadian children and associated risk factors. *Frontiers in public health*, *7*, 328.

Tsang, C., Sokal-Gutierrez, K., Patel, P., Lewis, B., Huang, D., Ronsin, K., ... & Gurung, S. (2019) Early childhood oral health and nutrition in urban and rural Nepal. *International journal of environmental research and public health*, *16*(14), 2456.

Warren JJ, Weber-Gasparoni K, Marshall TA, Drake DR, Dehkordi-Vakil F, Kolker JL, et al. (2008) Factors associated with dental caries experience in 1-year-old children. Journal of public health dentistry,68(2):70-5.

Gibson S, Williams S. (1999) Dental caries in pre-school children: associations with social class, toothbrushing habit and consumption of sugars and sugar-containing foods. Caries research,33(2):101-13.

Xiao J, Alkhers N, Kopycka-Kedzierawski DT, Billings RJ, Wu TT, Castillo DA, et al. (2019) Prenatal oral health care and early childhood caries prevention: a systematic review and meta-analysis. Caries research,53(4):411-21.

California Dental Association Foundation; American College of Obstetricians and Gynecologists, District IX. Oral health during pregnancy and early childhood: evidence-based guidelines for health professionals. (2010) J Calif Dent Assoc. Jun;38(6):391-403, 405-40. PMID: 20645626. Kumar J, Samelson R. (2009) Oral health care during pregnancy recommendations for oral health professionals. N Y State Dent J. Nov;75(6):29-33. PMID: 20069785.

Ajiboye A, Dawson III D, Fox C, Committee ASI (2018) American association for dental research policy statement on community water fluoridation. Journal of dental research,97(12):1293-6.

The use of fluoride in infants and children. (2002) *Paediatr Child Health*,7(8):569-82.

Preventive oral health intervention for pediatricians. (2008) *Pediatrics*,122(6):1387-94.

Toumba K, Twetman S, Splieth C, Parnell C, Van Loveren C, Lygidakis N. (2019) Guidelines on the use of fluoride for caries prevention in children: an updated EAPD policy document. *European Archives of Paediatric Dentistry*,20(6):507-16.

Riley, J. C., Lennon, M. A., & Ellwood, R. P. (1999) The effect of water fluoridation and social inequalities on dental caries in 5-year-old children. *International Journal of Epidemiology*, 28(2), 300-305.

Centers for Disease Control and Prevention (CDC). (2011) Dental caries in rural Alaska native children---Alaska, 2008. *MMWR: Morbidity & Mortality Weekly Report*, 60(37).

Dentistry AAPD. American Academy of Pediatric Dentistry. (2020) Caries-risk

assessment and management for infants, children, and adolescents. The Reference Manual of Pediatric Dentistry Chicago, Ill: American Academy of Pediatric Dentistry, (7):243.

Tungare S, Paranjpe AG. Early Childhood Caries. StatPearls. Treasure Island (FL): StatPearls Publishing Copyright © 2021, StatPearls Publishing LLC.; 2021.Selwitz RH, Ismail AI, Pitts NB. (2007) Dental caries. *The Lancet*, 369(9555):51-9.

Elamin A, Garemo M, Gardner A. (2018) Dental caries and their association with socioeconomic characteristics, oral hygiene practices and eating habits among preschool children in Abu Dhabi, United Arab Emirates the NOPLAS project. *BMC oral health*, 18(1):1-9.

Peltroche NO, Gabrielli E, Vásquez M, Castro A. (2015) Riesgo de caries dental en pacientes de tres a seis años que acuden a la clínica de la de la Universidad Nacional Federico Villarreal. *Cátedra Villarreal*,3(1).

World Health Organization (WHO). (2013) Oral health surveys: basic methods. World Health Organization.

Seow W, Clifford H, Battistutta D, Morawska A, Holcombe T. (2009) Case-control study of early childhood caries in Australia. *Caries research*, ,43(1):25-35.

Dye, B. A., Tan, S., Smith, V., Barker, L. K., Thornton-Evans, G., Eke, P. I., & Beltrán-Aguilar, E. D. (2007) Trends in oral health status; United States, 1988-1994 and 1999-2004.

Delgado-Angulo EK, Hobdell MH, Bernabé E. (2009) Poverty, social exclusion and dental caries of 12-year-old children: a cross-sectional study in Lima, Peru. *BMC oral health*, 9(1):1-6.

Yiğit, T., & Küçükeşmen, Ç. (2020). Ebeveynlerin Sosyo-Ekonomik Durumunun ve Oral Hijyen Alişkanliklarinin Erken Çocukluk Çaği Çürüklerine Etkisi. *Atatürk Üniversitesi Diş Hekimliği Fakültesi Dergisi*, 30(3), 366-372.

Kumarihamy SL, Subasinghe LD, Jayasekara P, Kularatna SM, Palipana PD. (2011) The prevalence of Early Childhood Caries

in 1-2 yrs olds in a semi-urban area of Sri Lanka. *BMC research notes*, 4(1):1-6.

Yıldırım, D., & Bilgir, E. (2017) Oral bulgu veren sistemik hastalıklar. *Medical Journal of Süleyman Demirel University*, 24(2).

Dörfer C, Benz C, Aida J, Campard G. (2017) The relationship of oral health with general health and NCDs: a brief review. *International dental journal*, 67:14-8.

Thomas MS, Parolia A, Kundabala M, Vikram M. (2010) Asthma and oral health: a review. *Australian Dental Journal*, 55(2):128-33.

Sag C, Ozden FO, Acikgoz G, Anlar FY. (2007) The effects of combination treatment with a long-acting  $\beta$ 2-agonist and a corticosteroid on salivary flow rate, secretory immunoglobulin a, and oral health in children and adolescents with moderate asthma: a 1-month, single-blind clinical study. *Clinical therapeutics*, 29(10):2236-42.

Ryberg M, Möller G, Erigson T. (1991) Saliva composition and caries development in asthmatic patients treated with  $\beta$ 2adrenoceptor agonists: a 4-year follow-up study. *European Journal* of Oral Sciences, 99(3):212-8.

Aral K, Aral CA, Kalkan RE. (2016) Astım ve ağız sağlığı. *EÜ Diş hek Fak Derg*, 37(2):42-6.

Kurt, A., Bolat, D. & Hatipoğlu, Ö. (2025) Impact of the severity and extension of dental caries lesions on Turkish preschool children's oral health-related quality of life: a cross-sectional study. *BMC Oral Health*, 25, 210. https://doi.org/10.1186/s12903-025-05549-7.

#### **DENTAL EROSION IN CHILDREN**

# HATİCE AYDOĞDU<sup>1</sup> BERİL DEMİRCAN<sup>2</sup>

#### Introduction

Dental wear describes irreversible losses occurring in teeth due to physiological and pathological destruction, excluding the formation of cavities(Devletli Özyiğit & Bulut Eyüboğlu, 2024:72). The term erosion resulting in dental wear is defined as the loss of the tooth enamel layer as a result of demineralization and exposure to acid due to chemical and mechanical factors(Wegehaupt et al.,2012:416). A study indicated that this chemical erosion, which does not contain bacteria, occurs due to hydrogen ions released from acids or anions that can bind to or form compounds with calcium. (Bartlett & et al., 1996:125).

The pH of the oral environment falls below critical levels, resulting in the demineralization and dissolution of dental enamel tissue, which creates a suitable environment for erosion. Dietary factors, especially the consumption of acidic foods and beverages,

<sup>&</sup>lt;sup>1</sup>Asst. Prof. Dr. Hatice AYDOĞDU, Hacıbektaş Veli University, Department of Pedodontics, Nevşehir/TürkiyeOrcid:0000-0002-4089-3507

<sup>&</sup>lt;sup>2</sup> Asst. Prof. Dr. Beril DEMİRCAN, Osmangazi University, Faculty of Dentistry, Department of Pedodontics, Eskişehir/Türkiye. Orcid: 0000-0002-2865-7843

play a significant role in the development of dental erosion(Taji & Seow, 2010:358). In addition, gastroesophageal reflux disease (GERD), oral breathing, and low saliva flow are also significant risk factors in the formation of erosion(Jha, Yavagal & Prabhakar, 2024:28).

The severity and progression rate of erosion varies depending on exposure time, the type of acidic agents, and individual defense mechanisms. If left untreated, dental erosion in children can lead to functional problems such as dentin hypersensitivity, aesthetic issues, and loss of vertical dimensions, negatively impacting the child's oral health and quality of life(Chan& et al., 2020:713).

Individuals with erosion in their deciduous teeth also have an increased risk of erosion in their permanent teeth, so early diagnosis and preventive treatments applied at an early age will help prevent damage to the permanent teeth(Ganss, 2001:264). In this case, understanding the risk factors and making an early diagnosis is crucial to prevent and manage dental erosion. The World Health Organization (WHO) has recognized dental erosion as a public health issue and has included it in the 5th edition of its guidelines(Peres, & et al.,2005:249).

#### Etiology

Dental erosion is an oral health issue that primarily affects children and adolescents due to recent lifestyle changes(Jaeggi & Lussi, 2014:55). The etiology of dental erosion is complex and multifactorial. It can be of intrinsic origin when related to the presence of gastric acid in the oral cavity or primarily of extrinsic origin due to the high consumption of acidic foods and beverages(Schlueter, Jaeggi & Lussi, 2012:68Dental erosion occurs as a result of the softening of the dental surface due to the effects of abrasive substances of either internal or external origin, or both together(Carvalho, & et al., 2015:1557).

Not all acidic products exhibit corrosive effects. For a risk to be clinically significant, the frequency, severity, and/or duration of exposure must be high(Carvalho, & et al., 2015:1557).

#### **Intrinsic Factors**

When intrinsic factors are taken into account, it is necessary to examine various eating and drinking habits, genetic factors, and systemic disorders related to the patient. It is essential to know that the individual's eroded tooth wear may not only be due to poor nutrition but could also be linked to a broader medical issue. Primarily, identifying the underlying medical condition that causes erosion is crucial to prevent further deterioration of the individual's oral health(Moazzez & Austin, 2018:326).

#### Systemic Disease

Gastroesophageal reflux disease (GERD) is a gastrointestinal system disease that is very common in the community but whose serious dental damages go unnoticed(Dündar & Şengün, 2015:67). Although it is a common condition, there is limited literature on the oral health of children with GERD( Taji & Seow, 2010:358). In a study, GERD was associated with increased erosion rates, and erosion was found to be 66.7% in children with GERD and 26.3% in the healthy group(Gońda-Domin, & et al., 2013:180). In other studies, the prevalence of dental erosion was found to be statistically higher in children diagnosed with GERD than in healthy individuals(Linnett, & et al., 2002:156, Ersin, & et al., 2006:279). . Dental erosion is a well-known symptom of GERD, and the prevalence of erosion in GERD patients has been reported to range from 5% to 47% (Moazzez & Austin, 2018:326).

Differences in the results among studies in the literature may arise from variations in age, type of dentition, and sample sizes. However, the most important factor is the duration of exposure of the teeth to gastric acid(Gońda-Domin, & et al., 2013:180). The saliva properties characteristic for GERD can reduce the natural saliva protection from internal and external acids(Gońda-Domin, & et al., 2013:180), and when stomach acid reaches the mouth, erosion is seen on the paltine surfaces of tooth enamel(Tolia & Vandenplas, 2009:258, Pace & et al., 2008:1179).

#### **Nutritional Disorders**

The most common nutritional disorders related to dental erosion, such as bulimia nervosa and anorexia nervosa, which cause conditions associated with frequent vomiting or serious stomach disorders, can also lead to dental erosion(Dündar & Sengün, 2015:67). The dental team has a fundamental role in the management of eating disorders. The risk of developing any type of eating disorder is highest for young men and women between the ages of 13 and 17(Moazzez & Austin, 2018:326). The connection between poor oral health and eating disorders has been shown in many studies; the main pathology is damage to enamel, which occurs with the ingress of gastric juices into the oral cavity and, accordingly, dental erosion; However, the lack of a healthy and balanced diet can also lead to tooth decay(Meyer, & et al., 1986:131; Vakil, & et al., 2007:1125). Most commonly, the main oral feature of an eating disorder is the erosion of the palatine (lingual) surfaces of the maxillarv anterior teeth, creating а flat and shinv appearance(Bartlett,2006:119; Sweis & et al., 2011:419). This form of erosion, referred to as perimyolysis, is characterized by the appearance of indentations and protrusions on incisal surfaces, loss at the enamel margins, a raised appearance in restorations, and loss of contours in teeth that have not yet been restored(Birmingham & Beumont, 2007:52).

Rumination is a rare condition that can cause severe dental erosion, similar to other eating disorders. Affected people

consciously remove the swallowed stomach contents and then swallow them again. Rumination is often seen in people with learning disabilities but can also affect other members of the population(Gilmour & Beckett, 1993:368). It is generally thought to be a psychological disorder, but it has also been suggested that patients may suffer from GERD.

Saliva is considered the most important biological factor in preventing the formation of dental erosion(Hara & Zero, 2014:195). Saliva dilutes acidic substances and removes them from the mouth. It buffers and neutralizes acidic products, thereby shortening periods of erosion. Finally, salivary proteins are the source of the acquired salivary membrane, which reduces acid erosion(Hara & Zero, 2014:195). For these reasons, erosion is directly proportional to the decreased saliva flow rate. The flow rate may be reduced, for example, due to systemic diseases and certain medications (e.g., antidepressants) or high levels of exercise(Carvalho, & et al., 2015:1557). Mouth breathing also causes dry mouth, reducing the buffering capacity of saliva, paving the way for erosion. In one study, a decrease in unstimulated saliva flow rate and a decrease in bicarbonate levels were reported in patients with blumia(Rytömaa, & et al., 1998:36). This situation increases the susceptibility to erosion in two ways.

#### **Extrinsic Factors**

#### Diet

A large part of erosion problems stems from extrinsic dietary acids(Taji & Seow, 2010:358, Carvalho, & et al., 2015:1557). Drinks such as cola, fruit juice, and energy drinks, frequently consumed by children, have a low pH value and can cause damage to the tooth surface(West, Hughes & Addy, 2000:875). The greater the amount and frequency of consumption of erosive products per day, the greater the risk of erosion. Furthermore, keeping an acidic drink in
the mouth before swallowing, especially if swished around the teeth, increases the risk(Carvalho, & et al., 2015:1557). Consuming acidic drinks with a straw or a retractable drinking lid positioned in front of the teeth increases the risk of wear on the incisors (Carvalho et al., 2015:1557). Conversely, narrow straws positioned as far back as possible will largely prevent contact between the incisors and molars and the liquid, thus reducing the risk of erosion(O'Sullivan & Curzon, 2000:186). Consuming drinks before bedtime, after exercise that reduces saliva flow rate, keeping them in the mouth for a long time, and brushing immediately after consumption are factors that facilitate the formation of erosion(Ganss & Lussi, 2008:1).

It has been reported that traditional tea and herbal teas also have erosive effects because their pH is lower than the critical pH value(Arat, 2012:86). It has been reported that mineralized beverages have a minimum erosion potential compared to these beverages(Brunton & Hussain, 2001:517).

It has been stated that some acids found in foods affect erosion seen in young people(Millward, Shaw & Smith, 1994a:263). Citric acid and malic acid found in fruits and vegetables have high erosive properties(Arat, 2012:86). It has been reported that erosion is 37 times more common in people who consume citrus fruits 2 or more times a day compared to those who consume less(Järvinen, Rytömaa & Heinonen, 1991:942).

#### **Oral Hygiene Products and Acidic Medications**

It has been found that the pH values of mouthwashes, which are oral care products, vary between 3.4 and 8.3, and that the acid titratable levels of these products also differ (Bhatti, Walsh, & Douglas, 1994, p. 71).Many oral care products, such as toothpastes, have a low pH. In this case, frequent brushing with abrasive oral hygiene products can increase tooth erosion(Lussi & Jaeggi, 2008:5). In addition, brushing immediately after exposure to acidic products also increases the risk of erosion. However, even without brushing, softened enamel is still eroded because it is not remineralized by saliva in short time periods. Therefore, delaying brushing after eating acidic foods is not a useful preventive measure (Carvalho, & et al., 2015:1557).

Excessive brushing to keep teeth clean also increases the risk of erosion. Dental erosion occurs more frequently in areas without plaque(Maupome, et al., 1998:148).

Long-term and frequent use of acidic medications can also cause erosive lesions on teeth(Hellwig & Lussi, 2006:112). ome medications (e.g., acidic saliva stimulants or preparations containing acetylsalicylic acid) and dietary supplements (e.g., vitamin C tablets) have been found to lower the pH in the mouth below 2.0, as do chewable tablets or effervescent drinks(Carvalho, & et al., 2015:1557). A positive relationship has been found between vitamin C use and the prevalence of erosion(Al-Malik, Holt & Bedi, 2001:430). The long-term use of chewable aspirin tablets may result in the development of erosion(McCracken & O'Neal 2000:95). At the same time, many medications that cause vomiting as a side effect also cause dental erosion(Ganss & Lussi, 2008:1). In addition, some medications can indirectly increase the risk of erosion because they have the side effect of reducing saliva flow.

Several pediatric medications have shown direct erosion potential in vitro, but clinical evidence of erosion occurring after the use of these medications is still lacking(Hellwig & Lussi, 2014:155). However, regular and long-term use of these medications may carry a risk of causing erosion(Hellwig & Lussi, 2014:155). For example, it has been reported that amino acid supplements used in the treatment of children with phenylketonuria increase the risk of erosion due to their low pH and high titratable acidity(Kilpatrick & et al., 1999:433).

#### Sport

Especially during exercise, the increased mouth breathing leads to less saliva cleaning of acid and carbohydrates in the oral environment(Arat, 2012:86). In addition, special food and fluid intake for athletes is fundamental for optimal athletic performance, but it can also be a risk factor for dental erosion(Broad & Rye, 2015:18). A study has detected dental erosion in athletes at a rate of 42-59%(Schulze & Busse, 2024:319). The average prevalence of erosion in athletes has been estimated to be 47%( de Queiroz Gonçalves, & et al.,(2020:207).

The first study showing that swimmers in poorly maintained swimming pools may be susceptible to acid erosion of enamel was published in 1982(Savad, E. N. (1982:32). An epidemiological study reported that 3% of non-swimmers, 12% of swimmers, and 39% of swimming team members were suffering from dental erosion(Centerwall, &et al., 1986:641). In a study, a case was published of a swimmer who had complete enamel loss on their upper incisors and general wear on other teeth after swimming intensively in improperly chlorinated gas water for 27 days(Geurtsen, 2000:291). It has been determined that the prevalence of dental erosion has increased in professional swimmers due to the low pH of pool water(Geurtsen, 2000:291).

#### **Risk Factors**

Dental erosion is increasingly recognized as a significant oral health issue, particularly among young populations. Understanding the risk factors associated with dental erosion is essential for effective prevention.

To initiate appropriate preventive treatments for dental erosion, a definitive diagnosis must be achieved. It is important to identify the relative contributions of acid sources causing erosion. Biological factors such as the buffering capacity of saliva, flow rate, pH, viscosity and composition, acquired pellicle and tooth structure, as well as behavioral factors such as eating and drinking habits, swimming habits, hydration during exercise, and oral hygiene should be evaluated(Lussi & Jaeggi, 2008:5). To identify relevant risk factors, a detailed dietary diary should be completed by the caregiver over six days, and the physician should review it. Because vegetarian diets also lead to dental erosion outside of acidic eating and drinking habits(Smits, Listl & Jevdjevic, 2020:7).

The medical history should consider any medication that could have an acidic effect or reduce the saliva protection mechanism. Patients should be questioned about the presence of reflux, frequency of vomiting, and any history of GERD. In places where intrinsic acids are identified, it is important to ensure that the patient receives further evaluation from healthcare professionals for this condition(Hellwig & Lussi, 2006:112). Unfortunately, children are the first to be affected by media foods due to unfavorable changes in societal lifestyles and easier access to fast food. (Peker & Arıkan, 2023:59).

Socioeconomic status, particularly middle-class families, dietary habits, and gastroesophageal reflux are cited as prevalent risk factors(Jha, Yavagal & Prabhakar, 2024:28).

#### **Prevalence Of Dental Erosion**

Dental erosion has a high prevalence in many regions, including Europe(Jaeggi & Lussi, 2014:55). For example, a European study showed that approximately 30% of the general population aged 18-35 visiting dental clinics had advanced erosive tooth wear on at least one tooth(Bartlett, et al., 2013:1007). Another study reported a dental erosion prevalence of 30-100%. (Kreulen, & et al., 2010:151). A study in Davangere city found a 4.5% prevalence

of dental erosion among children aged 6-12, with a higher prevalence in deciduous teeth(Jha, Yavagal & Prabhakar, 2024:28). Erosive tooth wear is a common condition among children in many industrialized countries, occurring at a percentage ranging from 14% to 87%(Pace & et al.,2008:1179).

Studies have concluded that the prevalence of dental erosion increases proportionally with age. (Wiegand, & et al., 2006:117; Nunn, & et al., 2003:98). A meta-analysis study indicated that dental erosion in children reaches up to 80% at the end of deciduous dentition and decreases to approximately 30% in adolescents with permanent dentition(Corica & Caprioglio, 2014:385; Kreulen, & et al., 2010:151). This suggests that wear on deciduous teeth may be more common and severe than on permanent teeth(Gatou & Mamai-Homata, 2012:923). Studies in our country have shown a higher prevalence of erosion in deciduous dentition(Arıkan, Vapur & Oba, 2017:194). The higher prevalence of wear on deciduous teeth compared to permanent teeth is attributed to the thinner enamel layer and lower degree of mineralization of deciduous teeth compared to permanent teeth, making them more susceptible to erosion(Holbrook & Ganss, 2008: 33; Taji & Seow, 2010:358). When evaluating gender, a meta-analysis has shown that the probability of erosive tooth wear in boys is significantly higher compared to girls(Yip, Lam & Yiu, 2022:491).

In a study, dental erosion was found in 83% of 24 children with GERD confirmed by endoscopic examination(Dashan, & et al., 2002:474). In another study, within a patient group of 17 children who were found to have pathological reflux during a 24-hour esophageal pH monitoring, the prevalence of dental erosion was found to be 87%(Aine, Baer & Maki,1993:210). Other studies have found that the prevalence of dental erosion in children diagnosed with GERD is statistically higher than in healthy subjects(Linnett, & et al., 2002:156; Ersin, & et al., 2006:279).

Prevalence has been studied worldwide, and results have varied significantly. Prevalence ranges from 5.7% to 78% depending on workplace, age, methodology, and definitions and criteria of tooth wear due to erosion(Zhang, & et al., 2014:14; Luo, &et al., 2005:115). Differences in the results between studies may arise from variations in age, tooth type, and sample sizes. However, the most important factor is the duration of the teeth's exposure to gastric acid. There are several factors that can alter the erosion process. These include diet, swallowing habits, general diseases, salivary capacity, contact time with teeth, and the surfaces exposed to acidic liquids. According to Hellström, erosive tooth wear is likely to become clinically apparent after two years of gastric acid exposure to tooth surfaces(Bartlett & et al., 1996:125).

#### **Diagnosis And Clinical Findings**

For a complete diagnosis of dental erosion, a detailed medical history, dental examination, and saliva analysis, if necessary, are required. Because it is difficult to diagnose dental erosion early, as it presents with few or less noticeable symptoms. At this stage, erosion does not cause softening or discoloration on the surface(Peker & Arıkan, 2023:59). Since there is no device available in routine dental practice for the detection and progression of dental erosion, clinical examination is the most important feature for dentists in the early stages(Lussi & Jaeggi, 2008:5 ve Lussi, 2006b:1). The clinical appearance, however, appears when the teeth are exposed to acid for several months(Scheutzel, 1996:178). Lesions at this level have an "orange peel" appearance(Lussi, 1996:191). When erosive agents affect the enamel actively, the eroded enamel appears brighter while the enamel lacks shine(Peker & Arıkan, 2023:59). The morphology caused by erosion can vary depending on the predominant cause(Carvalho, & et al., 2015:1557). Generally, dental erosion is characterized by the loss of natural surface morphology and contour.

Typical signs on occlusal surfaces are the cupping of protrusions and the flattening of occlusal structures(Carvalho, & et al., 2015:1557).

When the wear progresses to the dentin, the tooth takes on a yellow-brown color and becomes more sensitive to hot and cold changes(Peker & Arıkan, 2023:59). In advanced stages, all occlusal morphology may be lost and cavity surfaces may develop(Carvalho, & et al., 2015:1557). In this case, dentin takes on a round, messy, flattened shape. Thus, dark yellow-brown sclerotic dentin forms(Peker & Arıkan, 2023:59). On smooth surfaces, the typical signs are the flattening of the surface and the presence of a solid edge along the gum line(Carvalho, & et al., 2015:1557). As the condition becomes chronic and untreated, it even affects the buccal surfaces of the posterior teeth. The lower incisors are usually not affected, as the position of the tongue and the saliva flow rate provide some protection.

In all cases, these lesions are seen in both permanent and deciduous teeth and can extend to the dentin. Erosive wear in deciduous teeth may occur more rapidly and, in some cases, lead to pulp exposure(Gillborg, Åkerman & Ekberg, 2020:235). Microradiness studies have also shown that the erosion process is faster in deciduous teeth than in permanent teeth. Lesions may be localized (single tooth, buccal/lingual), generalized, or asymmetrical depending on the etiology(Carvalho, & et al., 2015:1557). The most commonly affected areas have been reported as the occlusal surfaces of molars and the palatal and incisal surfaces of incisors(Peker & Arıkan, 2023:59). In more severe cases, as erosion progresses, the palatal surfaces of maxillary premolars and molars are affected, and eventually the erosion pattern becomes more widespread, also involving the occlusal and facial surfaces of the teeth(Bartlett, 2006:119).

The BEWE (Basic Erosive Wear Examination) score can be used to assess the severity of erosion. This score is designed to provide a simple scoring system that can be used with the diagnostic criteria of all existing indices and aims to translate their results into a single unit, the BEWE score total(Bartlett, Ganss & Lussi, 2008:65). The aim is to establish a simple, reproducible and transferable scoring system for recording clinical findings and aiding the decision-making process for the management of erosive tooth wear. The main objective includes identifying and eliminating the main etiological cause, prevention and monitoring, and treating symptomatically and operatively where necessary(Bartlett, Ganss & Lussi, 2008:65). Assessment of progression is also important, as it determines whether preventive measures are needed or if implemented interventions are successful and can help in deciding when and how to restore worn teeth(Carvalho, & et al., 2015:1557).

While dental erosion is an urgent problem in pediatric dentistry, some argue that its impact may be overshadowed by more prevalent conditions such as dental caries, which also require significant attention in children's oral health management(Taji & Seow, 2010:358). Early intervention is critical to prevent irreversible damage and necessitates referral to pediatric dentists for children with GERD or significant erosion(Gońda-Domin et al., 2013).

#### Treatment

While dental erosion poses a serious risk to children's oral health, it is important to consider that not all children are equally affected. Factors such as genetics, individual dietary choices, and oral hygiene practices may lead to varying degrees of susceptibility to this condition. Therefore, the observed differences in the severity of dental erosion among patients with similar dietary habits can be explained this way(Zero, 1996:162). Biological factors such as saliva composition, flow rate, and acquired pellicle, along with

behavioral factors like dietary habits, fluid intake during exercise, and children's oral hygiene, should be carefully evaluated moving forward(Lussi & Jaeggi, 2008:5).

The first step in diagnosis and treatment is to notice the progression of erosion as early as possible. At this point, a determination should be made as to whether the primary factor is intrinsic or extrinsic. If these findings are confirmed, appropriate prevention and treatment strategies can be adopted, followed by restorative treatment. Preventive measures include suitable educating families about the erosive potential of certain foods and drinks, well promoting proper hygiene as as oral practices(Ludovichetti, & et al., 2022:262). Consumption of acidic beverages should be limited, use of straws should be recommended, and teeth should not be brushed immediately after consuming acidic products. The use of fluoride-containing toothpaste and mouth rinses should be encouraged.

The patient's medical history should also be questioned to determine if they are using any medication that contains acid or reduces saliva secretion(Taji & Seow, 2010:358). In patients diagnosed with reflux, medical treatment should be initiated, and nighttime feeding and food consumption in a lying position should be prevented. If there is oral breathing, an expert medical opinion should be sought for causes such as nasal congestion and adenoids. If there are symptoms indicating that dental erosion is caused by intrinsic acid sources and a medical diagnosis has not yet been made, the patient should be referred to a specialist. When determining existing risk factors, a comprehensive dietary analysis should be created by the person caring for the child and reviewed by a physician. Consumption of beverages high in calcium, phosphate, and xylitol should be recommended, and consumption of foods that neutralize the pH after meals (such as cheese and milk) should be advised. Rinsing the mouth or drinking water after consuming acidic foods and beverages should be advised(Magalhães, & et al., 2009:75; O'sullivan & Milosevic, 2008:29).

Studies recommend that patients at risk of dental erosion use soft-bristled toothbrushes, low-abrasive, and high-fluoride toothpastes(Kırzıoğlu & Yetiş, 2015:81). However, some studies have shown that fluoride-containing toothpastes are not effective in preventing this condition and have not found a significant relationship between the type of toothbrush used and the frequency of use(Ludovichetti, & et al., 2022:262). It is healthiest to tell the patient suffering from erosion to wait at least 30 minutes after erosive events before brushing their teeth(Kırzıoğlu & Yetiş, 2015:81). In addition, it has been proven that high-fluoride gels provide surface hardening in erosive lesions and protect the enamel against a second acid exposure thanks to the calcium fluoride layer(Gaffar, 1999:27).

It has been shown that chewing sugar-free gum, which increases saliva flow, also increases remineralization in the erosion phenomenon (Rios, & et al., 2006:218). Another method that can be used to increase the enamel's resistance to erosion is casein calcium phosphopeptide-amorphous phosphate (CPP-ACP) applications. CPP-ACP applications provide a significant advantage in the prevention and management of dental erosion in pediatric patients, especially when used in conjunction with topical fluoride agents due to their synergistic effect(Reynolds, 1998:8). It has been reported that the use of fluoride toothpastes containing potassium citrate (5.5%) and potassium nitrate can alleviate tooth sensitivity as a result of the precipitation of calcium carbonate in the dentin tubules(Arat, 2012:86).

The approach to dental erosion treatment should be tailored to the severity and extent of the lesion, as well as the patient's age and general health. Before starting operative treatment, it is important to identify and primarily stop the cause of the erosion. If the patient has no complaints, restorative treatment is not indicated during the deciduous dentition period. If the patient complains of sensitivity, small areas with erosion can be covered with resin materials such as dentin bonding agents. Glass ionomer cements are not recommended for use in teeth with erosion because they are susceptible to dissolution as a result of erosion(Aliping-McKenzie, Linden & Nicholson, 2004:1046).

If erosion affects larger areas, composite crowns may be necessary for anterior teeth and stainless steel crowns for posterior teeth. In cases of dentin exposure or significant substance loss, composite resin restorations, indirect restorations, and crown applications may be performed as needed. In cases of severe symptoms, extraction of the affected tooth may be considered as a treatment option(O'sullivan & Milosevic, 2008:29).

#### **Conclusion and Recommendations**

In children, dental erosion is a condition that can be controlled with early diagnosis and appropriate measures. It is of great importance for parents and healthcare professionals to be aware of this issue. Successful results can be achieved through risk factor identification, education, preventive applications, and restorative approaches when necessary. The regulation of eating habits, regular dental check-ups, and appropriate treatment methods can ensure that children have a healthy oral structure. It should not be forgotten that dental health is an integral part of overall health and should not be neglected.

#### References

Aine, L., Baer, M., & Mäki, M. (1993). Dental erosions caused by gastroesophageal reflux disease in children. ASDC journal of dentistry for children, 60(3), 210–214.

Al-Malik, M. I., Holt, R. D., & Bedi, R. (2001). The relationship between erosion, caries and rampant caries and dietary habits in preschool children in Saudi Arabia. International journal of paediatric dentistry, 11(6), 430–439

Aliping-McKenzie, M., Linden, R. W., & Nicholson, J. W. (2004). The effect of Coca-Cola and fruit juices on the surface hardness of glass-ionomers and 'compomers'. Journal of oral rehabilitation, 31(11), 1046–1052. https://doi.org/10.1111/j.1365-2842.2004.01348.x

Arat, E. M. (2012). Dental erozyonda tanı ve tedavi yöntemleri. Gülhane Tıp Dergisi, 54(1),86-91.

Arıkan, V., Vapur, K. N., Oba, A. A. (2017). Kırıkkale ilinde yaşayan 3-6 yaşları arasındaki çocuklarda süt dişi dental erozyon prevalansının değerlendirilmesi. Kırıkkale üniversitesi tıp fakültesi dergisi, 19(3),194-203. doi.org/10.24938/kutfd.324245

Bartlett, D. W., Evans, D. F., Anggiansah, A., & Smith, B. G. (1996). A study of the association between gastro-oesophageal reflux and palatal dental erosion. British dental journal, 181(4), 125–131. https://doi.org/10.1038/sj.bdj.4809187

Bartlett D. (2006). Intrinsic causes of erosion. Monographs in oral science, 20, 119–139. https://doi.org/10.1159/000093359

Bartlett, D. W., Lussi, A., West, N. X., Bouchard, P., Sanz, M., & Bourgeois, D. (2013). Prevalence of tooth wear on buccal and lingual surfaces and possible risk factors in young European adults.

Journal of dentistry, 41(11), 1007–1013. https://doi.org/10.1016/j.jdent.2013.08.018

Bartlett, D., Ganss, C., & Lussi, A. (2008). Basic Erosive Wear Examination (BEWE): a new scoring system for scientific and clinical needs. Clinical oral investigations, 12 Suppl 1(Suppl 1), S65–S68. https://doi.org/10.1007/s00784-007-0181-5

Birmingham CL, Beumont P. Medical managment of eating disorders. First published(2007). İngiltere, Cambridge University Press 2004;52-3.

Bhatti, S. A., Walsh, T. F., & Douglas, C. W. (1994). Ethanol and pH levels of proprietary mouthrinses. Community dental health, 11(2), 71–74.

Broad, E. M.,& Rye, L. A. (2015). Do current sports nutrition guidelines conflict with good oral health?. General dentistry, 63(6), 18–23

Brunton, P. A., & Hussain, A. (2001). The erosive effect of herbal tea on dental enamel. Journal of dentistry, 29(8), 517–520. https://doi.org/10.1016/s0300-5712(01)00044-6

Carvalho, T. S., Colon, P., Ganss, C., Huysmans, M. C., Lussi, A., Schlueter, N., Schmalz, G., Shellis, R. P., Tveit, A. B., & Wiegand, A. (2015). Consensus report of the European Federation of Conservative Dentistry: erosive tooth wear--diagnosis and management. Clinical oral investigations, 19(7), 1557–1561. https://doi.org/10.1007/s00784-015-1511-7

Centerwall, B. S., Armstrong, C. W., Funkhouser, L. S., & Elzay, R. P. (1986). Erosion of dental enamel among competitive swimmers at a gas-chlorinated swlmmtng pool. American journal of epidemiology, 123(4), 641-647.

Chan, A. S., Tran, T. T. K., Hsu, Y. H., Liu, S. Y. S., & Kroon, J. (2020). A systematic review of dietary acids and habits on dental erosion in adolescents. International journal of paediatric dentistry, 30(6), 713–733. https://doi.org/10.1111/ipd.12643

Corica, A.,& Caprioglio, A. (2014). Meta-analysis of the prevalence of tooth wear in primary dentition. European journal of paediatric dentistry, 15(4), 385–388.

Dahshan, A., Patel, H., Delaney, J., Wuerth, A., Thomas, R., & Tolia, V. (2002). Gastroesophageal reflux disease and dental erosion in children. The Journal of pediatrics, 140(4), 474–478. https://doi.org/10.1067/mpd.2002.123285

de Queiroz Gonçalves, P. H. P., Guimarães, L. S., de Azeredo, F. N. A., Wambier, L. M., Antunes, L. A. A., & Antunes, L. S. (2020). Dental erosion'prevalence and its relation to isotonic drinks in athletes: a systematic review and meta-analysis. Sport Sciences for Health, 16, 207-216.

Devletli Özyiğit L, Bulut Eyüboğlu G. Diş Aşınmalarına Genel Bir Bakış. Akd Dent J. 2024;3(2):72-80.

Dündar, A.,& Şengün, A. (2015). Dental erozyonun etiyolojisi ve tedavi yaklaşımları. Atatürk Üniversitesi Diş Hekimliği Fakültesi Dergisi, 24(Supplement 8), 67-73. https://doi.org/10.17567/dfd.67179

Ersin, N. K., Onçağ, O., Tümgör, G., Aydoğdu, S., & Hilmioğlu, S. (2006). Oral and dental manifestations of gastroesophageal reflux disease in children: a preliminary study. Pediatric dentistry, 28(3), 279–284.

Gaffar A. (1999). Treating hypersensitivity with fluoride varnish. Compendium of continuing education in dentistry (Jamesburg, N.J. : 1995), 20(1 Suppl), 27–35.

Ganss, C., Klimek, J., & Giese, K. (2001). Dental erosion in children and adolescents--a cross-sectional and longitudinal investigation using study models. Community dentistry and oral epidemiology, 29(4), 264–271. https://doi.org/10.1034/j.1600-0528.2001.290405.x

Ganss, C.,& Lussi, A. (2008). Current erosion indicesflawed or valid?. Clinical oral investigations, 12 Suppl 1(Suppl 1), S1–S3. https://doi.org/10.1007/s00784-007-0182-4

Gatou, T.,& Mamai-Homata, E. (2012). Tooth wear in the deciduous dentition of 5-7-year-old children: risk factors. Clinical oral investigations, 16(3), 923–933. https://doi.org/10.1007/s00784-011-0586-z

Geurtsen W. (2000). Rapid general dental erosion by gaschlorinated swimming pool water. Review of the literature and case report. American journal of dentistry, 13(6), 291–293.

Gillborg, S., Åkerman, S., & Ekberg, E. (2020). Tooth wear in Swedish adults-A cross-sectional study. Journal of oral rehabilitation, 47(2), 235–245. https://doi.org/10.1111/joor.12887

Gilmour, A. G., & Beckett, H. A. (1993). The voluntary reflux phenomenon. British dental journal, 175(10), 368–372. https://doi.org/10.1038/sj.bdj.4808327

Gońda-Domin, M., Lisiecka, K., Rojek, R., Mokrzycka, M., Szymanowicz, J., & Glura, B. (2013). Dental manifestations of gastroesophageal reflux disease in children. Gastroenterology Review/Przegląd Gastroenterologiczny, 8(3), 180-183. https://doi.org/10.5114/pg.2013.36332

Hara, A. T.,& Zero, D. T. (2014). The potential of saliva in protecting against dental erosion. Monographs in oral science, 25, 197–205. https://doi.org/10.1159/000360372

Hellwig, E.,& Lussi, A. (2006). Oral hygiene products and acidic medicines. Monographs in oral science, 20, 112–118. https://doi.org/10.1159/000093358

Hellwig, E.,& Lussi, A. (2014). Oral hygiene products, medications and drugs - hidden aetiological factors for dental erosion. Monographs in oral science, 25, 155–162. https://doi.org/10.1159/000359942

Holbrook, W. P.,& Ganss, C. (2008). Is diagnosing exposed dentine a suitable tool for grading erosive loss?. Clinical oral investigations, 12 Suppl 1(Suppl 1), S33–S39. https://doi.org/10.1007/s00784-007-0174-4

Jha, G., Yavagal P. & Prabhakar A. R (2024). Prevalence and Associated Risk Factors of Dental Erosion among 6-7-Year-Old Children in Davanagere city: A Cross Sectional Survey. Acta Scientific Dental Sciences, 8(6),28-36. 10.31080/ASDS.2024.08.1841

Jaeggi, T.,& Lussi, A. (2014). Prevalence, incidence and distribution of erosion. Monographs in oral science, 25, 55–73. https://doi.org/10.1159/000360973

Järvinen, V. K., Rytömaa, I. I., & Heinonen, O. P. (1991). Risk factors in dental erosion. Journal of dental research, 70(6), 942– 947. https://doi.org/10.1177/00220345910700060601

Kırzıoğlu, Z.,& Yetiş, C. (2015). Çocuklarda dental erozyon ve koruyucu uygulamalar. Atatürk Üniversitesi Diş Hekimliği Fakültesi Dergisi, 25, 81-90. https://doi.org/10.17567/dfd.39974

Kilpatrick, N. M., Awang, H., Wilcken, B., & Christodoulou, J. (1999). The implication of phenylketonuria on oral health. Pediatric dentistry, 21(7), 433–437.

Kreulen, C. M., Van 't Spijker, A., Rodriguez, J. M., Bronkhorst, E. M., Creugers, N. H., & Bartlett, D. W. (2010). Systematic review of the prevalence of tooth wear in children and adolescents. Caries research, 44(2), 151–159. https://doi.org/10.1159/000308567

Linnett, V., Seow, W. K., Connor, F., & Shepherd, R. (2002). Oral health of children with gastro-esophageal reflux disease: a controlled study. Australian dental journal, 47(2), 156–162. https://doi.org/10.1111/j.1834-7819.2002.tb00321.x

Ludovichetti, F. S., Signoriello, A. G., Colussi, N., Zuccon, A., Stellini, E., & Mazzoleni, S. (2022). Soft drinks and dental erosion during pediatric age: a clinical investigation. Minerva dental and oral science, 71(5), 262–269. https://doi.org/10.23736/S2724-6329.22.04662-9

Lussi, A.,& Jaeggi, T. (2008). Erosion--diagnosis and risk factors. Clinical oral investigations, 12 Suppl 1(Suppl 1), S5–S13. https://doi.org/10.1007/s00784-007-0179-z

Lussi A. (2006). Erosive tooth wear - a multifactorial condition of growing concern and increasing knowledge. Monographs in oral science, 20, 1–8. https://doi.org/10.1159/000093343

Lussi A. (1996). Dental erosion clinical diagnosis and case history taking. European journal of oral sciences, 104(2 (Pt 2)), 191–198. https://doi.org/10.1111/j.1600-0722.1996.tb00067.x

Luo, Y., Zeng, X. J., Du, M. Q., & Bedi, R. (2005). The prevalence of dental erosion in preschool children in China. Journal of dentistry, 33(2), 115–121. https://doi.org/10.1016/j.jdent.2004.08.007

Magalhães, A. C., Wiegand, A., Rios, D., Honório, H. M., & Buzalaf, M. A. (2009). Insights into preventive measures for dental erosion. Journal of applied oral science : revista FOB, 17(2), 75–86. https://doi.org/10.1590/s1678-77572009000200002

Maupomé, G., Díez-de-Bonilla, J., Torres-Villaseñor, G., Andrade-Delgado, L. C., & Castaño, V. M. (1998). In vitro quantitative assessment of enamel microhardness after exposure to eroding immersion in a cola drink. Caries research, 32(2), 148–153. https://doi.org/10.1159/000016445

McCracken, M.,& O'Neal, S. J. (2000). Dental erosion and aspirin headache powders: a clinical report. Journal of prosthodontics : official journal of the American College of Prosthodontists, 9(2), 95–98. https://doi.org/10.1111/j.1532-849x.2000.00095.x

Meyer, G. W., Austin, R. M., Brady, C. E., 3rd, & Castell, D. O. (1986). Muscle anatomy of the human esophagus. Journal of clinical gastroenterology, 8(2), 131–134. https://doi.org/10.1097/00004836-198604000-00005

Millward, A., Shaw, L., & Smith, A. (1994). Dental erosion in four-year-old children from differing socioeconomic backgrounds. ASDC journal of dentistry for children, 61(4), 263– 266.

Moazzez, R.,& Austin, R. (2018). Medical conditions and erosive tooth wear. British dental journal, 224(5), 326–332. https://doi.org/10.1038/sj.bdj.2018.166

Nunn, J. H., Gordon, P. H., Morris, A. J., Pine, C. M., & Walker, A. (2003). Dental erosion -- changing prevalence? A review of British National childrens' surveys. International journal of paediatric dentistry, 13(2), 98–105. https://doi.org/10.1046/j.1365-263x.2003.00433.x

O'Sullivan, E. A.,& Curzon, M. E. (2000). A comparison of acidic dietary factors in children with and without dental erosion. ASDC journal of dentistry for children, 67(3), 186–160.

O'Sullivan, E., Milosevic, A., & British Society of Paediatric Dentistry (2008). UK National Clinical Guidelines in Paediatric Dentistry: diagnosis, prevention and management of dental erosion. International journal of paediatric dentistry, 18 Suppl 1, 29–38. <u>https://doi.org/10.1111/j.1365-263X.2008.00936.x</u>

Pace, F., Pallotta, S., Tonini, M., Vakil, N., & Bianchi Porro, G. (2008). Systematic review: gastro-oesophageal reflux disease and dental lesions. Alimentary pharmacology & therapeutics, 27(12), 1179–1186. https://doi.org/10.1111/j.1365-2036.2008.03694.x

Peker O & Arıkan R. (2023) Dental Erosion in Primary Teeth. J Dent Fac Usak Univ.;2(3):59-64.

Peres, K. G., Armênio, M. F., Peres, M. A., Traebert, J., & De Lacerda, J. T. (2005). Dental erosion in 12-year-old schoolchildren: a cross-sectional study in Southern Brazil. International journal of paediatric dentistry, 15(4), 249–255. https://doi.org/10.1111/j.1365-263X.2005.00643.x

Reynolds E. C. (1998). Anticariogenic complexes of amorphous calcium phosphate stabilized by casein phosphopeptides: a review. Special care in dentistry : official publication of the American Association of Hospital Dentists, the Academy of Dentistry for the Handicapped, and the American Society for Geriatric Dentistry, 18(1), 8–16. https://doi.org/10.1111/j.1754-4505.1998.tb01353.x

Rios, D., Honório, H. M., Magalhães, A. C., Delbem, A. C., Machado, M. A., Silva, S. M., & Buzalaf, M. A. (2006). Effect of salivary stimulation on erosion of human and bovine enamel subjected or not to subsequent abrasion: an in situ/ex vivo study. Caries research, 40(3), 218–223. https://doi.org/10.1159/000092229

Rytömaa, I., Järvinen, V., Kanerva, R., & Heinonen, O. P. (1998). Bulimia and tooth erosion. Acta odontologica Scandinavica, 56(1), 36–40. https://doi.org/10.1080/000163598423045

Savad, E. N. (1982). Enamel erosion... multiple cases with a common cause (?). Journal of the New Jersey Dental Association, 53(1), 32-35.

Schulze, A.,& Busse, M. (2024). Sports Diet and Oral Health in Athletes: A Comprehensive Review. Medicina (Kaunas, Lithuania), 60(2), 319. https://doi.org/10.3390/medicina60020319

Schlueter, N., Jaeggi, T., & Lussi, A. (2012). Is dental erosion really a problem?. Advances in dental research, 24(2), 68–71. https://doi.org/10.1177/0022034512449836

Scheutzel P. (1996). Etiology of dental erosion--intrinsic factors. European journal of oral sciences, 104(2 (Pt 2)), 178–190. https://doi.org/10.1111/j.1600-0722.1996.tb00066.x

Smits, K. P. J., Listl, S., & Jevdjevic, M. (2020). Vegetarian diet and its possible influence on dental health: A systematic literature review. Community dentistry and oral epidemiology, 48(1), 7–13. https://doi.org/10.1111/cdoe.12498

Sweis, R., Fox, M., Anggiansah, A., & Wong, T. (2011). Prolonged, wireless pH-studies have a high diagnostic yield in patients with reflux symptoms and negative 24-h catheter-based pHstudies. Neurogastroenterology and motility, 23(5), 419–426. https://doi.org/10.1111/j.1365-2982.2010.01663.x

Taji, S.,& Seow, W. K. (2010). A literature review of dental erosion in children. Australian dental journal, 55(4), 358–475. https://doi.org/10.1111/j.1834-7819.2010.01255.x

Tolia, V.,& Vandenplas, Y. (2009). Systematic review: the extra-oesophageal symptoms of gastro-oesophageal reflux disease in children. Alimentary pharmacology & therapeutics, 29(3), 258–272. https://doi.org/10.1111/j.1365-2036.2008.03879.x

Vakil, N., van Zanten, S. V., Kahrilas, P., Dent, J., Jones, R., & Globale Konsensusgruppe (2007). Die Montreal-Definition und gastroösophagealen Refluxkrankheit: Klassifikation der Ein evidenzbasiertes Konsensus-Papier The Montreal globales definition and classification of gastroesophageal reflux disease: a global, evidence-based consensus paper]. Zeitschrift fur Gastroenterologie, 45(11), 1125-1140. https://doi.org/10.1055/s-2007-963633

West, N. X., Hughes, J. A., & Addy, M. (2000). Erosion of dentine and enamel in vitro by dietary acids: the effect of temperature, acid character, concentration and exposure time. Journal of oral rehabilitation, 27(10), 875–880. https://doi.org/10.1046/j.1365-2842.2000.00583.x

Wegehaupt, F. J., Tauböck, T. T., Sener, B., & Attin, T. (2012). Long-term protective effect of surface sealants against erosive wear by intrinsic and extrinsic acids. Journal of dentistry, 40(5), 416–422. https://doi.org/10.1016/j.jdent.2012.02.003

Wiegand, A., Müller, J., Werner, C., & Attin, T. (2006). Prevalence of erosive tooth wear and associated risk factors in 2-7year-old German kindergarten children. Oral diseases, 12(2), 117– 124. https://doi.org/10.1111/j.1601-0825.2005.01167.x

Yip, K., Lam, P. P. Y., & Yiu, C. K. Y. (2022). Prevalence and Associated Factors of Erosive Tooth Wear among Preschool Children-A Systematic Review and Meta-Analysis. Healthcare (Basel, Switzerland), 10(3), 491. https://doi.org/10.3390/healthcare10030491 Zhang, S., Chau, A. M., Lo, E. C., & Chu, C. H. (2014). Dental caries and erosion status of 12-year-old Hong Kong children. BMC public health, 14, 7. https://doi.org/10.1186/1471-2458-14-7

Zero D. T. (1996). Etiology of dental erosion--extrinsic factors. European journal of oral sciences, 104(2 ( Pt 2)), 162–177. https://doi.org/10.1111/j.1600-0722.1996.tb00065.x

## REMINERALIZATION AGENTS IN PEDIATRIC DENTISTRY

## BERIL DEMIRCAN<sup>1</sup> MERVE BILMEZ SELEN<sup>2</sup>

#### Introduction

Dental caries is one of the most common chronic diseases in childhood, progressing rapidly especially in primary and young permanent teeth, leading to tooth loss and a decline in quality of life (Kiatipi, Davidopoulou, Arapostathis, & Arhakis, 2021). Although traditional caries treatment approaches are usually based on restorative methods, interest in minimally invasive and preventive applications has been increasing in recent years (Frencken, 2017). In this context, agents that enable the remineralization of demineralized dental hard tissues have gained an important place in pediatric dentistry (Arifa, Ephraim, & Rajamani, 2019). These agents make it possible to manage early-stage carious lesions without the need for invasive intervention, thereby protecting the dental tissue and positively shaping the child's dental experience. In this chapter, the

<sup>&</sup>lt;sup>1</sup> Asst. Prof. Dr., Eskischir Osmangazi University, Faculty of Dentistry, Pediatric Dentistry, Orcid: 0000-0002-2865-7843

<sup>&</sup>lt;sup>2</sup> Asst. Prof. Dr. Merve BİLMEZ SELEN, Inonu University, Faculty of Dentistry, Department of Pedodontics, Malatya/Türkiye. Orcid:0000-0003-0726-4992

main remineralization agents used in pediatric patients, their mechanisms of action, and clinical applications will be comprehensively discussed in light of current literature.

#### **Enamel and Dentin Remineralization: Biological Foundations**

Although enamel and dentin differ in structural and biochemical properties among dental hard tissues, both are susceptible to acidic attacks in the oral environment. Organic acids produced by bacterial metabolism cause the dissolution of hydroxyapatite crystals within these tissues, initiating the process (Hemagaran demineralization & Neelakantan, 2014). However, this process is reversible under appropriate environmental conditions. Saliva creates a natural environment for remineralization by providing calcium, phosphate, and bicarbonate ions (Hicks, Garcia-Godoy, & Flaitz, 2003). Remineralization is a re-crystallization process that begins at the surface and progresses into deeper layers, playing a critical role especially in the management of early carious lesions. To support this biological balance, various remineralizing agents have been developed to enhance the existing ion balance and reverse mineral loss. In this way, both the integrity of the dental tissue is preserved and the need for more invasive treatment is reduced (Hicks, Garcia-Godoy, & Flaitz, 2004).

Demineralization is a reversible chemical process defined as mineral loss in the enamel due to acid attacks resulting from dietary intake or bacterial activity. When the oral environment is favorable, minerals such as calcium and phosphate re-precipitate on the enamel surface in a process known as "remineralization" (Featherstone, 2008). This process is supported by minerals in saliva and fluoride present in dental plaque. Saliva provides a saturated medium with calcium, phosphate, and fluoride ions, facilitating their deposition on the enamel surface following a drop in pH. Additionally, some proteins in saliva (e.g., statherin, histatin) help maintain these ions in a dissolved state. Dental plaque also contributes to remineralization through its mineral content; however, continued acidic conditions can hinder this process. Therefore, regular oral hygiene and the use of fluoride play a critical role in promoting remineralization (Hicks et al., 2003).

The remineralization process, which is the natural defense mechanism of dental hard tissues against mineral loss, is not only limited to endogenous factors but can also be supported by external therapeutic agents. In recent years, various remineralization agents that mimic or optimize this biological process have been developed and gained widespread clinical use. These agents aim to re-establish ion balance in enamel and dentin, reversing mineral loss and also caries progression. this classifying limiting In context, remineralization agents based on their chemical composition, mechanisms of action, and application methods is essential for understanding their effectiveness and guiding clinical decisions (Pitts et al., 2017).

#### **Remineralization Agents**

Remineralizing agents developed to support the remineralization process are categorized into various subgroups based on their composition, mechanism of action, and clinical usage (Arifa et al., 2019). Among the most commonly used are fluoride compounds, which enhance enamel resistance to acid attacks by reducing the solubility of hydroxyapatite crystals. In addition, products that directly provide mineral support by delivering calcium and phosphate ions such as CPP-ACP (casein phosphopeptideamorphous calcium phosphate) and tricalcium phosphate stand out as non-fluoride alternatives. Silver diamine fluoride has emerged as a dual-action agent that both promotes remineralization and inhibits caries progression due to its antimicrobial effect.

Newer generation agents include nano-hydroxyapatite particles, biomimetic peptides, and polyphenol-based natural compounds. These agents go beyond the classical fluoride mechanisms by targeting more complex biological pathways and show promising potential, particularly in pediatric patients with high caries risk. Such classification allows clinicians to develop effective and safe remineralization strategies tailored to patient-specific risk factors.

#### **Fluoride Agents**

Fluoride is one of the most extensively studied and widely used agents in dental remineralization processes. Since the mid-20th century, fluoride has been employed to prevent and manage dental caries and is widely recognized as a major contributor to the significant decline in caries prevalence (Oh et al., 2017). Fluoride can be delivered systemically or topically. Systemic intake typically occurs through fluoridated drinking water, fluoride-containing tablets, and certain foods. This approach is particularly effective during tooth development, promoting the formation of fluorapatite in enamel and increasing resistance to caries (Carey, 2014).

Topical fluoride, on the other hand, is administered directly to the tooth surface through toothpaste, mouth rinses, gels, and professional varnish applications. Fluoride-containing products enhance the formation of fluoroapatite crystals, which are less soluble than hydroxyapatite, thus providing a more acid-resistant enamel structure. In doing so, fluoride not only helps restore existing mineral loss but also prevents future demineralization (Simmer, Hardy, Chinoy, Bartlett, & Hu, 2020).

The effectiveness of fluoride varies depending on the method of application, concentration, and duration of exposure. Clinical use includes fluoride varnishes, gels, rinses, and toothpastes in various concentrations. In pediatric patients, lower-dose but more frequent applications are preferred to minimize systemic intake and maximize topical benefits. Additionally, formulations combining fluoride with calcium and phosphate have demonstrated a synergistic increase in remineralization efficacy, guiding the development of modern fluoride products olmaktadır(Elsayad, Sakr, & Badr, 2009).However, inappropriate or excessive use of fluoride can lead to adverse effects such as dental fluorosis, highlighting the importance of careful dose management in pediatric dentistry.

## Multifaceted Mechanisms of Fluoride in Dental Remineralization

Fluoride enhances the resistance of dental tissues to acidic challenges through a multifaceted mechanism. The fluoride ion replaces the hydroxyl group in the hydroxyapatite crystal lattice, resulting in decreased crystal volume and increased structural stability. Primarily, fluoride binds to hydroxyapatite crystals on the enamel surface, converting them into fluoroapatite, a more acid-resistant form (Fazzi, Vieira, & Zucas, 1977). This transformation reduces the solubility of the crystal structure and lowers the critical pH threshold for demineralization.

Fluoride also facilitates the precipitation of calcium and phosphate ions in the oral environment, promoting the formation of new mineral layers on the enamel surface. Furthermore, fluoride exhibits antimicrobial properties by inhibiting the metabolic activity of plaque bacteria. The fluoride ion ( $F^-$ ) inhibits the bacterial enzyme enolase, thereby disrupting the production of phosphoenolpyruvate (PEP) a key intermediate in the glycolytic pathway essential for energy and phosphate uptake in many bacteria. Even at concentrations of 10–100 ppm, fluoride significantly suppresses acid production by plaque bacteria, indirectly supporting the remineralization process. Thus, fluoride functions both as a passive mineralizing agent and as an active protective compound (Soi, Vinayak, Singhal, & Roy, 2013).

## Therapeutic Uses and Clinical Modalities of Fluoride in Dental Practice

Fluoride is utilized in dental applications in various concentrations forms. Toothpastes containing and low concentrations of fluoride are suitable for daily use, whereas varnishes, gels, and solutions with higher fluoride levels are designed for professional use tasarlanmıştır (Beltrán-Aguilar, Goldstein, & Lockwood, 2000; Twetman & Keller, 2016). Fluoride varnishes are particularly favored in pediatric patients due to their ability to provide high efficacy at low doses, ensuring both safety and effectiveness. In individuals with high caries risk, more frequent and targeted fluoride applications are recommended, while for those in the low-risk category, preventive-level applications are typically sufficient. Fluoride mouth rinses, on the other hand, are used as supplementary agents, especially in individuals undergoing orthodontic treatment or those with poor oral hygiene. When determining the form and frequency of fluoride application, factors such as the patient's age, caries activity, systemic fluoride exposure, and level of cooperation should be considered.

Although fluoride is generally regarded as safe when used in appropriate doses, it is crucial to control systemic exposure, especially in children during developmental stages. The most common adverse effect is dental fluorosis, which arises from excessive systemic fluoride intake and manifests as opacities and staining in the developing enamel structure of the teeth (Warren et al., 2009).For topical applications, the key concern is to avoid ingestion of fluoride following administration. Fluoride protocols in pediatric patients should be tailored according to age and body weight, and caregivers should be properly instructed on appropriate usage.

Fluoride is a scientifically proven agent employed in various chemical forms to support dental remineralization (Twetman & Keller, 2016). Different fluoride compounds are categorized based on their composition and clinical indications. These forms vary in terms of effectiveness, solubility, bioavailability, and application protocols.

It is known that gels and varnishes contribute to remineralization by accumulating glycoprotein-stabilized calcium fluoride (CaF<sub>2</sub>) on the enamel surface, serving as a mineral reservoir and releasing fluoride during acidic attacks. Among the topical fluoride products formulated for professional and home use such as solutions, gels, rinses, and toothpastes the most commonly preferred compounds include sodium fluoride (NaF), stannous fluoride (SnF<sub>2</sub>), and acidulated phosphate fluoride (APF). These are more frequently used compared to other fluoride compounds (e.g., titanium tetrafluoride [TiF<sub>4</sub>], amine or ammonium fluorides), due to their clinical effectiveness and practical application profiles (Elsayad et al., 2009)

#### Sodium Fluoride (NaF)

Sodium fluoride is the most commonly used form of fluoride in topical applications. It provides a highly reactive fluoride ion, which promotes the conversion of hydroxyapatite crystals into fluoroapatite in mildly acidic environments, thereby enhancing the enamel's resistance to caries. In clinical practice, sodium fluoride is safely and effectively used in toothpastes, rinses (0.05% NaF), and high-concentration varnishes (5% NaF – 22,600 ppm) (Gao, Zhang, Mei, Lo, & Chu, 2016; Oh et al., 2017).

#### Stannous Fluoride (SnF<sub>2</sub>)

Stannous fluoride offers a dual effect by contributing to remineralization through its fluoride content while also exerting antimicrobial action via stannous ions. It reduces plaque accumulation and gingival inflammation, and enhances the acid resistance of enamel surfaces. Stabilized formulations of stannous fluoride have shown improved bioavailability and demonstrated protective effects against erosion (Myers et al., 2019).

## Acidulated Phosphate Fluoride (APF)

APF accelerates fluoride penetration into mineralized tissue under low pH conditions. APF gel consists of 1.23% sodium fluoride (12,300 ppm fluoride) buffered with phosphate ions and 0.1% orthophosphoric acid. APF gels and foams rapidly saturate the enamel, thereby enhancing remineralization. However, they may interact with certain restorative materials (e.g., composites, glass ionomers) and cause surface degradation; therefore, material compatibility should be assessed prior to application (Keratibumrungpong, Trairatvorakul, Jirakran, & Govitvattana, 2024).

### Amine Fluoride (AmF)

Amine fluoride exhibits strong adhesion to the enamel surface due to its surface-active molecular structure, allowing for a prolonged fluoride effect. This form is widely used in Europe and has demonstrated comparable efficacy to other fluoride types in preventing dental caries; however, its use remains limited in other regions due to lower local adoption rates (Madléna, 2013).

### Titanium Tetrafluoride (TiF4)

Titanium tetrafluoride exerts its effect by forming a protective layer on the tooth surface that enhances resistance to

acidic challenges. In a study conducted by Medeiros and colleagues, TiF4-based varnishes were shown to produce a thicker and more resistant protective coating compared to NaF-containing varnishes, offering a more effective barrier against erosive acid attacks (Medeiros et al., 2016).Similarly, the research group led by de Castilho reported that the combined use of mouth rinses containing both TiF4 and NaF produced superior remineralization effects compared to their individual application (Castilho, Salomão, Buzalaf, & Magalhães, 2015).

#### Silver Diamine Fluoride (SDF)

Silver diamine fluoride (SDF) gained widespread clinical use following the pioneering work of Dr. Nishino and Dr. Yamaga in employing it to arrest dental caries (Nishino, Yoshida, Sobue, Kato, & Nishida, 1969).SDF is an alkaline, clear, and colorless solution composed of diammine-silver and fluoride ions complexed with ammonia. It is most commonly used at a 38% concentration. The formulation combines the antibacterial effects of silver with the remineralizing properties of fluoride. Silver ions interact with the thiol groups of essential bacterial enzymes, leading to enzyme inactivation, disruption of the electron transport chain, and ultimately bacterial cell death. SDF contains the highest fluoride concentration among all dental fluoride agents, approximately 44,800 ppm (Contractor, Girish, & Indira, 2021). SDF not only halts the progression of existing carious lesions but also significantly reduces the development of new lesions in both primary and permanent teeth. Its non-invasive application makes it a particularly effective and safe alternative for children with systemic health conditions or limited cooperation during dental procedures. The most notable drawback of SDF use is the prominent black staining it produces on carious lesions, which can be a source of concern for parents, particularly in cases where aesthetic expectations are high(Seifo et al., 2020).

#### Safety and Adverse Effects of Fluoride

The American Academy of Pediatric Dentistry (AAPD) considers the use of fluoride at preventive doses to be safe, while emphasizing that excessive systemic intake may lead to dental fluorosis. Therefore, the amount of fluoride used should be adjusted according to the child's age—for example, a smear (rice grain size) for children aged 0–3 years and a pea-sized amount for those aged 3–6 years. In pediatric patients, topical applications are encouraged over systemic exposure to minimize potential risks.

The AAPD guidelines recommend the use of fluoride in various forms (American Academy of Pediatric Dentistry [AAPD], 2024):

- Topical applications (for children ≥6 months): Brushing with fluoride toothpaste at least twice daily is the first-line recommendation.
- **Professional applications:** High-concentration products (5% NaF varnish 22,600 ppm fluoride) are considered safe for use especially in children under the age of six and should be applied at least twice a year.
- Rinses, gels, and foams (for children ≥6 years): Prescription forms containing 0.5%–1.23% fluoride may serve as supportive agents for children at high risk of caries.

## Classification of Fluoride Agents by Method of Application(American Academy of Pediatric Dentistry [AAPD], 2024)

Fluoride-containing products are offered in various forms depending on their intended use in either clinical or home settings. The method of application directly influences both their efficacy and safety. Accordingly, fluoride agents can be categorized under two main headings: professional (clinical) applications and at-home use.

## **Professional Applications**

These applications involve high-concentration fluoride products administered in a dental clinic under the supervision of a dental professional:

- Fluoride Varnishes (5% NaF 22,600 ppm): This is the most commonly used professional topical form. It is applied to the teeth as a thin layer using a brush. Due to its low risk of fluoride ingestion, it is particularly preferred in pediatric patients.
- Fluoride Gels (1.23% APF or 2% NaF): These are typically applied using a tray method for approximately 4 minutes. Because of the risk of ingestion, caution should be exercised when using fluoride gels in young children.

## • Fluoride Foams (1.23% APF)

These are applied in a manner similar to gels but contain less fluoride, making them a safer alternative for pediatric patients.

# Fluoride Mouth Rinses (0.2% NaF weekly / 0.05% NaF daily)

Used as a mouthwash. Not recommended for children under the age of 6 due to the risk of swallowing; suitable for children aged 6 and above.

This category includes low-dose products developed to provide

fluoride supplementation as part of daily oral hygiene routines (American Academy of Pediatric Dentistry [AAPD], 2024):

## • Fluoridated Toothpastes (500–1450 ppm)

Should be used in age-appropriate amounts for children. Brushing twice daily helps support the remineralization process.

## • Fluoride Tablets / Drops (Systemic)

Prescribed by healthcare professionals in areas where systemic fluoride supplementation is necessary. Due to the risk of dental fluorosis, dosage planning must be carried out with caution.

## • Fluoride Mouth Rinses

Daily-use products containing 0.05% NaF are recommended for individuals at high risk of caries. They should only be used by children with a well-developed swallowing reflex.

# Alpha-Tricalcium Phosphate ( $\alpha$ -TCP) and Beta-Tricalcium Phosphate ( $\beta$ -TCP)

Alpha and beta tricalcium phosphate ( $\alpha$ -TCP and  $\beta$ -TCP) have emerged as promising remineralizing agents in the biological repair of dental hard tissues. These calcium phosphate–based compounds dissolve in the oral environment, releasing calcium (Ca<sup>2+</sup>) and phosphate (PO4<sup>3-</sup>) ions. These ions promote the reformation of hydroxyapatite crystals, particularly in demineralized enamel regions, thereby supporting a remineralization process that begins at the surface and progresses into deeper layers (Pinto de Souza et al., 2018).

Due to its high solubility,  $\alpha$ -TCP dissolves rapidly, creating an ion-rich microenvironment and converting into hydroxyapatite in a short time. This property makes it a fast-acting agent for the passive repair of early carious lesions. In contrast,  $\beta$ -TCP has lower solubility and releases ions more slowly, which makes it preferable in applications where extended remineralization is desired (Karlinsey & Pfarrer, 2012).

exhibit a Both forms synergistic effect the on remineralization process, especially when combined with fluoride. Fluoroapatite crystals formed with the aid of fluoride not only compensate for mineral loss but also make enamel more resistant to future acid attacks. Furthermore, when the particle size of TCP derivatives is reduced to the nanometer scale, their ability to penetrate the tooth surface increases significantly, thereby enhancing their overall remineralization potential (Hamba, Nakamura, Nikaido, Tagami, & Muramatsu, 2020). Therefore, formulations containing α-TCP and  $\beta$ -TCP have become key components of topical products and restorative materials developed to support caries management in modern minimally invasive dentistry.

# Casein Phosphopeptide–Amorphous Calcium Phosphate (CPP-ACP)

Casein phosphopeptide–amorphous calcium phosphate (CPP-ACP) is regarded as one of the pioneering agents in the biological restoration of dental hard tissues (Reynolds et al., 2008). Caseinderived phosphopeptides stabilize amorphous calcium phosphate (ACP) particles, allowing them to remain bioavailable in the oral environment without undergoing premature crystallization. This complex structure forms localized calcium and phosphate reservoirs on enamel and dentin surfaces, enabling targeted remineralization.

CPP-ACP facilitates the regrowth of hydroxyapatite crystals by regulating the ionic balance, particularly in acid-induced demineralized regions. As a result, the progression of early carious lesions such as white spot lesions can be halted or even reversed. Additionally, CPP-ACP can exert a buffering effect during episodes of decreased oral pH, thereby limiting environmental acidity. Clinical studies have demonstrated that topical applications containing this agent show enhanced remineralization potential when combined with fluoride. This synergistic effect arises from the combined actions of fluoride in promoting crystal formation and CPP-ACP in stabilizing available ions. Therefore, CPP-ACP systems represent an effective biomimetic strategy in preventive dentistry, both as a standalone treatment and in combination with fluoride stratejidir (Cochrane, Cai, Huq, Burrow, & Reynolds, 2010).

#### **Calcium Sodium Phosphosilicate (Bioactive Glass)**

Bioactive glasses are classified among materials that are both chemically and biologically active in the remineralization process (Andersson & Kangasniemi, 1991).The most commonly used form, sodium calcium phosphosilicate, initiates an ion exchange process upon contact with the tooth surface, releasing free calcium, phosphate, and sodium ions into the surrounding environment. These ions interact with the buffering components of saliva to form a hydroxyapatite-like mineral layer on the tooth surface (Jagga et al., 2018).In this way, not only is the mineral loss caused by acidic challenges compensated, but the progression of lesions is also effectively arrested.

Bioactive glass particles also contribute to the occlusion of dentinal tubules, making them effective in the management of dentin
hypersensitivity (Al-Haddad, Alarami, Alshammari, & Madfa, 2025). This dual action provides therapeutic advantages in cases of both enamel and dentin demineralization. Clinical studies have shown that even fluoride-free formulations of this material possess notable remineralization potential; however, its efficacy significantly increases when used in conjunction with fluoride. The silicate backbone in its structure is considered a key determinant of bioactivity, facilitating the rapid formation of an apatite-like layer on the surface (Hench & Jones, 2015). Therefore, bioactive glasses are recognized as valuable minimally invasive agents, particularly in early lesion management and hypersensitivity control.

## Xylitol

Xylitol is a naturally occurring five-carbon sugar alcohol that has gained attention in oral health due to its antimicrobial and remineralizing properties (Ly, Milgrom, & Rothen, 2006).It specifically disrupts the metabolism of cariogenic bacteria such as *Streptococcus mutans*, which are unable to utilize xylitol as an energy source, thereby suppressing acid production (Söderling, 2009).The resulting reduction in acid levels allows the oral pH to remain near neutral for extended periods, creating a favorable microenvironment for remineralization.

Additionally, xylitol stimulates salivary flow, enhancing the delivery of calcium and phosphate ions to the tooth surface, which actively contributes to the remineralization process (Abd Rahman et al., 2020). In vitro studies have shown that when xylitol is used in combination with fluoride as a caries-preventive agent, it supports hydroxyapatite-like mineral formation on demineralized enamel and reduces lesion depth (Gargouri, Zmantar, Kammoun, Kechaou, & Ghoul-Mazgar, 2018). The co-administration of xylitol with fluoride can amplify remineralization, as fluoride directly initiates mineral

formation while xylitol optimizes the oral environment for fluoride efficacy (Sharda, Gupta, Goyal, & Gauba, 2021).

Due to its proven cariostatic and remineralizing benefits, the use of xylitol in the form of chewing gums, tablets, or rinses has been recommended, particularly for children and individuals at high risk of dental caries (Gasmi Benahmed et al., 2020; Siqueira et al., 2021).In this context, xylitol serves as a supportive agent that not only balances the biological environment but also aids in the management of pre-cavitated lesions.

#### Nanoparticles for Remineralization

Nanotechnological approaches in the reconstruction of dental hard tissues have garnered significant interest due to their enhanced surface interaction and bioavailability compared to conventional remineralizing agents. Agents in nanoparticulate form support mineral repair at both the surface and subsurface levels, primarily due to their controlled ion release rates and superior tissue penetration capabilities (Aggarwal, 2016).

Nano-sized calcium phosphate, nano-hydroxyapatite (nHAP), amorphous calcium phosphate (ACP), and bioactive glass particles contribute to crystal growth by delivering targeted ions to demineralized regions of the enamel (Arifa et al., 2019). When combined with fluoride, these agents accelerate the formation of more stable and acid-resistant crystal structures. Furthermore, nanoparticles can easily penetrate dentinal tubules, providing desensitizing effects as well (Kunam, Manimaran, Sampath, & Sekar, 2016).

#### Theobromine

Theobromine (3,7-dimethylxanthine) is a naturally occurring alkaloid found in cocoa beans and has gained increasing attention in recent years for its anticariogenic and remineralizing properties in oral health products (Amaechi et al., 2013). Abundant in chocolate and cocoa, theobromine belongs to the methylxanthine family and shares structural similarities with compounds such as theophylline and caffeine Cova, Leta, Mariani, Pantoni, & Pomati, 2019).

Theobromine binds to the surface of hydroxyapatite crystals, promoting crystal growth and thereby reducing the solubility of the crystalline structure (Premnath et al., 2019). This mechanism renders the enamel more resistant to acid dissolution. Numerous in vitro studies, along with some in vivo investigations, have demonstrated that theobromine application may lead to enamel remineralization and help prevent early surface caries lesions (Durhan, Ozsalih, Gokkaya, Kulan, & Kargul, 2021).

## Arginine

Arginine is a naturally occurring amino acid found in saliva and has recently garnered attention for its role in caries prevention and remineralization processes (Nascimento, 2018). In particular, formulations containing arginine bicarbonate and calcium carbonate help modulate the oral microflora by limiting acid production and neutralizing pH levels. This effect contributes to the creation of a biologically favorable environment for remineralization by suppressing acidogenic bacteria such as Streptococcus mutans(Zheng et al., 2017).

Moreover, arginine enhances the local concentration of calcium and phosphate ions, providing a conducive surface for mineral precipitation. Both in vitro and in situ studies have shown that toothpastes containing arginine are effective in reversing early enamel lesions and improving microhardness values (Altınışık & Erten, 2023; Cantore et al., 2013). When combined with fluoride, the remineralization effect is significantly enhanced, with synergistic outcomes observed even at low fluoride concentrations (Bijle, Ekambaram, Lo, & Yiu, 2018; Konagala et al., 2020). Thus, arginine serves as an important bioactive component that promotes remineralization by creating an antimicrobial oral environment and biologically supporting enamel tissue.

## **Self-Assembling Peptides**

Self-assembling peptides have emerged as promising agents in the biomimetic repair of early enamel lesions (Kind et al., 2017).These peptides spontaneously organize into a nanofibrillar structure in the presence of water when applied to demineralized enamel surfaces (Kind et al., 2017).This structure acts as a scaffold that mimics natural enamel development, promoting the localized accumulation of calcium and phosphate ions. These ions then crystallize in an oriented manner along the peptide matrix, ultimately forming a hydroxyapatite-like mineral layer.

This process not only provides a superficial coating but also supports subsurface mineral gain, thereby halting the progression of lesions (Alkilzy, Qadri, Splieth, & Santamaría, 2023). P11-4-based formulations contribute a biomimetic dimension to existing fluoride protocols, holding the potential to reshape clinical approaches in early caries management.

#### References

American Academy of Pediatric Dentistry (AAPD). (2024). *Oral health policies and clinical recommendations*. Retrieved June 24, 2025, from <u>https://www.aapd.org/research/oral-health-policies-recommendations/</u>

Abd Rahman, N. H., Jahim, J. M., Munaim, M. S. A., Rahman, R. A., Fuzi, S. F. Z., & Illias, R. M. (2020). Immobilization of recombinant Escherichia coli on multi-walled carbon nanotubes for xylitol production. *Enzyme and microbial technology*, *135*, 109495.

Aggarwal, S. (2016). Nanotechnology in endodontics: Current and potential clinical applications. In: Medknow.

Al-Haddad, A., Alarami, N., Alshammari, A. F., & Madfa, A. A. (2025). Efficacy of bioactive glass-based desensitizer compared to other desensitizing agents or techniques in dentin hypersensitivity: a systematic review. *BMC Oral Health*, 25(1), 899. doi:10.1186/s12903-025-06288-5

Alkilzy, M., Qadri, G., Splieth, C. H., & Santamaría, R. M. (2023). Biomimetic Enamel Regeneration Using Self-Assembling Peptide P(11)-4. *Biomimetics (Basel)*, 8(3). doi:10.3390/biomimetics8030290

Altınışık, H., & Erten, H. (2023). Evaluation of the activities of toothpastes with different contents in the prevention of enamel demineralization: An in vitro study. *Current Research in Dental Sciences*, 33(1), 27-34.

Amaechi, B. T., Porteous, N., Ramalingam, K., Mensinkai, P. K., Ccahuana Vasquez, R. A., Sadeghpour, A., & Nakamoto, T. (2013). Remineralization of artificial enamel lesions by theobromine. *Caries Res, 47*(5), 399-405. doi:10.1159/000348589

Andersson, O. H., & Kangasniemi, I. (1991). Calcium phosphate formation at the surface of bioactive glass in vitro. *J* Biomed Mater Res, 25(8), 1019-1030. doi:10.1002/jbm.820250808

Arifa, M. K., Ephraim, R., & Rajamani, T. (2019). Recent Advances in Dental Hard Tissue Remineralization: A Review of Literature. *Int J Clin Pediatr Dent, 12*(2), 139-144. doi:10.5005/jpjournals-10005-1603

Beltrán-Aguilar, E. D., Goldstein, J. W., & Lockwood, S. A. (2000). Fluoride varnishes. A review of their clinical use, cariostatic mechanism, efficacy and safety. *J Am Dent Assoc, 131*(5), 589-596. doi:10.14219/jada.archive.2000.0232

Bijle, M. N. A., Ekambaram, M., Lo, E. C., & Yiu, C. K. Y. (2018). The combined enamel remineralization potential of arginine and fluoride toothpaste. *J Dent*, 76, 75-82. doi:10.1016/j.jdent.2018.06.009

Cantore, R., Petrou, I., Lavender, S., Santarpia, P., Liu, Z., Gittins, E., . . . Utgikar, N. (2013). In situ clinical effects of new dentifrices containing 1.5% arginine and fluoride on enamel de- and remineralization and plaque metabolism. *J Clin Dent, 24 Spec no A*, A32-44.

Carey, C. M. (2014). Focus on fluorides: update on the use of fluoride for the prevention of dental caries. *J Evid Based Dent Pract*, *14 Suppl*, 95-102. doi:10.1016/j.jebdp.2014.02.004

Castilho, A. R., Salomão, P. M., Buzalaf, M. A., & Magalhães, A. C. (2015). Protective effect of experimental mouthrinses containing NaF and TiF4 on dentin erosive loss in vitro. *J Appl Oral Sci, 23*(5), 486-490. doi:10.1590/1678-775720150127

Cochrane, N. J., Cai, F., Huq, N. L., Burrow, M. F., & Reynolds, E. C. (2010). New approaches to enhanced

remineralization of tooth enamel. J Dent Res, 89(11), 1187-1197. doi:10.1177/0022034510376046

Contractor, I. A., Girish, M., & Indira, M. (2021). Silver diamine fluoride: extending the spectrum of preventive dentistry, a literature review. *Pediatric dental journal*, *31*(1), 17-24.

Cova, I., Leta, V., Mariani, C., Pantoni, L., & Pomati, S. (2019). Exploring cocoa properties: is theobromine a cognitive modulator? *Psychopharmacology (Berl), 236*(2), 561-572. doi:10.1007/s00213-019-5172-0

Durhan, M. A., Ozsalih, S., Gokkaya, B., Kulan, P. Y., & Kargul, B. (2021). Caries Preventive Effects of Theobromine Containing Toothpaste on Early Childhood Caries: Preliminary Results. *Acta Stomatol Croat*, *55*(1), 18-27. doi:10.15644/asc55/1/3

Elsayad, I., Sakr, A., & Badr, Y. (2009). Combining casein phosphopeptide-amorphous calcium phosphate with fluoride: synergistic remineralization potential of artificially demineralized enamel or not? *J Biomed Opt*, *14*(4), 044039. doi:10.1117/1.3210780

Fazzi, R., Vieira, D. F., & Zucas, S. M. (1977). Fluoride release and physical properties of a fluoride-containing amalgam. *J Prosthet Dent, 38*(5), 526-531. doi:10.1016/0022-3913(77)90028-2

Featherstone, J. D. (2008). Dental caries: a dynamic disease process. *Aust Dent J*, 53(3), 286-291. doi:10.1111/j.1834-7819.2008.00064.x

Frencken, J. E. (2017). Atraumatic restorative treatment and minimal intervention dentistry. *Br Dent J*, 223(3), 183-189. doi:10.1038/sj.bdj.2017.664

Gao, S. S., Zhang, S., Mei, M. L., Lo, E. C., & Chu, C. H. (2016). Caries remineralisation and arresting effect in children by

professionally applied fluoride treatment - a systematic review. *BMC Oral Health*, *16*, 12. doi:10.1186/s12903-016-0171-6

Gargouri, W., Zmantar, T., Kammoun, R., Kechaou, N., & Ghoul-Mazgar, S. (2018). Coupling xylitol with remineralizing agents improves tooth protection against demineralization but reduces antibiofilm effect. *Microb Pathog*, *123*, 177-182. doi:10.1016/j.micpath.2018.06.038

Gasmi Benahmed, A., Gasmi, A., Arshad, M., Shanaida, M., Lysiuk, R., Peana, M., . . . Bjørklund, G. (2020). Health benefits of xylitol. *Appl Microbiol Biotechnol*, *104*(17), 7225-7237. doi:10.1007/s00253-020-10708-7

Hamba, H., Nakamura, K., Nikaido, T., Tagami, J., & Muramatsu, T. (2020). Remineralization of enamel subsurface lesions using toothpaste containing tricalcium phosphate and fluoride: an in vitro  $\mu$ CT analysis. *BMC Oral Health, 20*(1), 292. doi:10.1186/s12903-020-01286-1

Hemagaran, G., & Neelakantan, P. (2014). Remineralization of the tooth structure-the future of dentistry. *International Journal of PharmTech Research*.

Hench, L. L., & Jones, J. R. (2015). Bioactive Glasses: Frontiers and Challenges. *Front Bioeng Biotechnol, 3*, 194. doi:10.3389/fbioe.2015.00194

Hicks, J., Garcia-Godoy, F., & Flaitz, C. (2003). Biological factors in dental caries: role of saliva and dental plaque in the dynamic process of demineralization and remineralization (part 1). *J Clin Pediatr Dent*, *28*(1), 47-52. doi:10.17796/jcpd.28.1.yg6m443046k50u20

Hicks, J., Garcia-Godoy, F., & Flaitz, C. (2004). Biological factors in dental caries enamel structure and the caries process in the dynamic process of demineralization and remineralization (part 2). J

*Clin Pediatr Dent, 28*(2), 119-124. doi:10.17796/jcpd.28.2.617404w302446411

Jagga, U., Paul, U., Padmanabhan, V., Kashyap, A., Guram, G., & Keswani, K. (2018). Comparative Evaluation of Remineralizing Effect of Novamin and Tricalcium Phosphate on Artificial Caries: An in vitro Study. *J Contemp Dent Pract, 19*(1), 109-112. doi:10.5005/jp-journals-10024-2221

Karlinsey, R. L., & Pfarrer, A. M. (2012). Fluoride plus functionalized  $\beta$ -TCP: a promising combination for robust remineralization. *Adv Dent Res, 24*(2), 48-52. doi:10.1177/0022034512449463

Keratibumrungpong, K., Trairatvorakul, C., Jirakran, K., & Govitvattana, N. (2024). A Comparison of the Fluoride 'Paint- On' vs Tray Application Techniques for Enamel Remineralisation. *Int Dent J*, *74*(5), 1006-1015. doi:10.1016/j.identj.2024.03.003

Kiatipi, M., Davidopoulou, S., Arapostathis, K., & Arhakis, A. (2021). Dental Neglect in Children: A Comprehensive Review of the Literature. *J Contemp Dent Pract, 22*(2), 199-204.

Kind, L., Stevanovic, S., Wuttig, S., Wimberger, S., Hofer, J., Müller, B., & Pieles, U. (2017). Biomimetic Remineralization of Carious Lesions by Self-Assembling Peptide. *J Dent Res, 96*(7), 790-797. doi:10.1177/0022034517698419

Konagala, R. K., Mandava, J., Anwarullah, A., Uppalapati, L. V., Karumuri, S., & Angadala, P. L. (2020). Synergistic Effect of Arginine on Remineralization Potential of Fluoride Varnish and Nanohydroxyapatite on Artificial Caries Lesions: An In Vitro Study. *J Contemp Dent Pract, 21*(9), 1048-1053.

Kunam, D., Manimaran, S., Sampath, V., & Sekar, M. (2016). Evaluation of dentinal tubule occlusion and depth of penetration of nano-hydroxyapatite derived from chicken eggshell powder with and without addition of sodium fluoride: An in vitro study. *J Conserv Dent, 19*(3), 239-244. doi:10.4103/0972-0707.181940

Ly, K. A., Milgrom, P., & Rothen, M. (2006). Xylitol, sweeteners, and dental caries. *Pediatr Dent, 28*(2), 154-163; discussion 192-158.

Madléna, M. (2013). Experiences with amine fluoride containing products in the management of dental hard tissue lesions focusing on Hungarian studies: a review. *Acta Med Acad, 42*(2), 189-197. doi:10.5644/ama2006-124.86

Medeiros, M. I., Carlo, H. L., Lacerda-Santos, R., Lima, B. A., Souza, F. B., Rodrigues, J. A., & Carvalho, F. G. (2016). Thickness and nanomechanical properties of protective layer formed by TiF4 varnish on enamel after erosion. *Braz Oral Res, 30*(1). doi:10.1590/1807-3107BOR-2016.vol30.0075

Myers, C. P., Pappas, I., Makwana, E., Begum-Gafur, R., Utgikar, N., Alsina, M. A., . . . Sullivan, R. J. (2019). Solving the problem with stannous fluoride: Formulation, stabilization, and antimicrobial action. *J Am Dent Assoc*, 150(4s), S5-s13. doi:10.1016/j.adaj.2019.01.004

Nascimento, M. (2018). Potential uses of arginine in dentistry. *Advances in dental research*, 29(1), 98-103.

Nishino, M., Yoshida, S., Sobue, S., Kato, J., & Nishida, M. (1969). Effect of topically applied ammoniacal silver fluoride on dental caries in children. *The Journal of Osaka University Dental School*, *9*, 149-155.

Oh, H. J., Oh, H. W., Lee, D. W., Kim, C. H., Ahn, J. Y., Kim, Y., ... Jeon, J. G. (2017). Chronologic Trends in Studies on Fluoride Mechanisms of Action. *J Dent Res*, 96(12), 1353-1360. doi:10.1177/0022034517717680

Pinto de Souza, S. C. T., Araújo, K. C., Barbosa, J. R., Cancio, V., Rocha, A. A., & Tostes, M. A. (2018). Effect of dentifrice containing fTCP, CPP-ACP and fluoride in the prevention of enamel demineralization. *Acta Odontol Scand*, *76*(3), 188-194. doi:10.1080/00016357.2017.1401658

Pitts, N. B., Zero, D. T., Marsh, P. D., Ekstrand, K., Weintraub, J. A., Ramos-Gomez, F., . . . Ismail, A. (2017). Dental caries. *Nat Rev Dis Primers*, *3*, 17030. doi:10.1038/nrdp.2017.30

Premnath, P., John, J., Manchery, N., Subbiah, G. K., Nagappan, N., & Subramani, P. (2019). Effectiveness of Theobromine on Enamel Remineralization: A Comparative In-vitro Study. *Cureus*, *11*(9), e5686. doi:10.7759/cureus.5686

Reynolds, E. C., Cai, F., Cochrane, N. J., Shen, P., Walker, G. D., Morgan, M. V., & Reynolds, C. (2008). Fluoride and casein phosphopeptide-amorphous calcium phosphate. *J Dent Res, 87*(4), 344-348. doi:10.1177/154405910808700420

Seifo, N., Robertson, M., MacLean, J., Blain, K., Grosse, S., Milne, R., . . . Innes, N. (2020). The use of silver diamine fluoride (SDF) in dental practice. *Br Dent J*, 228(2), 75-81. doi:10.1038/s41415-020-1203-9

Sharda, S., Gupta, A., Goyal, A., & Gauba, K. (2021). Remineralization potential and caries preventive efficacy of CPP-ACP/Xylitol/Ozone/Bioactive glass and topical fluoride combined therapy versus fluoride mono-therapy - a systematic review and meta-analysis. *Acta Odontol Scand*, *79*(6), 402-417. doi:10.1080/00016357.2020.1869827

Simmer, J. P., Hardy, N. C., Chinoy, A. F., Bartlett, J. D., & Hu, J. C. (2020). How Fluoride Protects Dental Enamel from Demineralization. *J Int Soc Prev Community Dent, 10*(2), 134-141. doi:10.4103/jispcd.JISPCD\_406\_19

Siqueira, V. L., Barreto, G. S., Silva, E. B. V., Silva, T. V. D., Nascimento, D. G. D., Veronezi, A., . . . Cardoso, C. A. B. (2021). Effect of xylitol varnishes on enamel remineralization of immature teeth: in vitro and in situ studies. *Braz Oral Res, 35*, e137. doi:10.1590/1807-3107bor-2021.vol35.0137

Soi, S., Vinayak, V., Singhal, A., & Roy, S. (2013). Fluorides and their role in demineralization and remineralization. *J Dent Sci Oral Rehabil, 14*, 19-21.

Söderling, E. M. (2009). Xylitol, mutans streptococci, and dental plaque. *Adv Dent Res, 21*(1), 74-78. doi:10.1177/0895937409335642

Twetman, S., & Keller, M. K. (2016). Fluoride Rinses, Gels and Foams: An Update of Controlled Clinical Trials. *Caries Res, 50 Suppl 1*, 38-44. doi:10.1159/000439180

Warren, J. J., Levy, S. M., Broffitt, B., Cavanaugh, J. E., Kanellis, M. J., & Weber-Gasparoni, K. (2009). Considerations on optimal fluoride intake using dental fluorosis and dental caries outcomes--a longitudinal study. *J Public Health Dent, 69*(2), 111-115. doi:10.1111/j.1752-7325.2008.00108.x

Zheng, X., He, J., Wang, L., Zhou, S., Peng, X., Huang, S., . . . Zhou, X. (2017). Ecological Effect of Arginine on Oral Microbiota. *Sci Rep*, 7(1), 7206. doi:10.1038/s41598-017-07042-w

# THE ROLE OF PROBIOTICS IN PEDIATRIC ORAL HEALTH: BALANCING THE ORAL MICROBIOTA IN CHILDREN

## FATMA NUR KIZILAY<sup>1</sup> TURKAN MAHYADDINOVA<sup>2</sup>

#### Introduction

The oral cavity is a dynamic and diverse microbial ecosystem, colonized by over 700 bacterial species, many of which are established early in life through vertical and environmental transmission. In children, the composition of the oral microbiota is shaped by multiple factors, including delivery mode, infant feeding practices, hygiene habits, dietary exposure, and antibiotic use (Dzidic & ark., 2018:2292). A balanced oral microbiota plays a crucial role in maintaining oral health by suppressing pathogenic organisms, stabilizing oral pH, and supporting mucosal immune responses(Deo & Deshmukh, 2019:122).

<sup>1</sup>Asst. Prof. Dr. Fatma Nur Kızılay, Inonu University, Faculty of Dentistry, Department of Pedodontics, Malatya/Türkiye. Orcid: 0000-0001-6588-663X

<sup>2</sup> Pediatric Dentist Turkan Mahyaddinova, Alanya Oral and Dental Health Center, Orcid: 0000-0001-5415-6084 Disruptions in this balance, referred to as dysbiosis, have been consistently associated with common pediatric oral diseases such as early childhood caries (ECC), gingivitis, and halitosis(Deo & Deshmukh, 2019:122). In this context, probiotics—defined by the World Health Organization as "live microorganisms which, when administered in adequate amounts, confer a health benefit on the host"—have emerged as a promising biocompatible strategy for modulating oral microbial communities. By interfering with pathogenic colonization and promoting beneficial microbial activity, probiotics may help prevent or manage oral infections in children(Hill & ark., 2014:506).

Recent evidence highlights the potential of specific probiotic strains to reduce the growth of cariogenic bacteria, alleviate gingival inflammation, and restore microbial equilibrium in the oral cavity(Laleman & ark., 2014:1539). Given the increasing awareness of antimicrobial resistance and the need for safe, non-invasive interventions in pediatric dentistry, probiotics have become a subject of growing clinical and scientific interest.

This chapter explores the role of probiotics in modulating the oral microbiota in children, their mechanisms of action, clinical applications, safety considerations, and future research directions.

## **Oral Microbiota in Children**

The colonization of the oral cavity begins at birth and is shaped by a complex interplay of vertical and environmental factors, particularly the mode of delivery and early feeding practices. Vaginal delivery facilitates the transmission of beneficial microorganisms such as Lactobacillus and Prevotella, while cesarean delivery is associated with delayed microbial diversity and increased colonization by skin-derived taxa (Dominguez-Bello & ark., 2010:11971). During infancy, breastfeeding supports the establishment of beneficial oral commensals like Streptococcus salivarius and Lactobacillus spp., whereas formula feeding has been linked to an overrepresentation of potentially cariogenic species, including Streptococcus mutans. As solid foods are introduced and primary teeth erupt, the microbial community becomes more diverse, enabling colonization by anaerobes such as Fusobacterium nucleatum and Porphyromonas spp (Holgerson & ark., 2013:127).

In healthy children, this microbiota maintains a dynamic equilibrium that regulates pH, inhibits pathogen overgrowth, and modulates mucosal immunity. However, frequent sugar intake, suboptimal hygiene, and repeated antibiotic exposure can lead to dysbiosis—a microbial shift dominated by acidogenic and aciduric bacteria, primarily S. mutans and Lactobacillus spp., which are strongly associated with early childhood caries (Tanner & ark., 2011:1464).

Moreover, dysbiosis in children has been linked not only to increased caries risk but also to altered levels of salivary antimicrobial peptides and pro-inflammatory cytokines, suggesting broader implications for systemic immune development (Simon-Soro & ark., 2013:616). Thus, early microbial colonization and the preservation of microbial homeostasis are vital for long-term oral and overall health in pediatric populations.

#### **Mechanism of Action of Probiotics**

Probiotics support oral health through multiple synergistic mechanisms that contribute to the stabilization of the oral microbiota and suppression of pathogenic species(Firmino & ark., 2016:242). A key mechanism is competitive exclusion, whereby probiotic strains adhere to oral epithelial surfaces, occupying ecological niches and preventing colonization by pathogens such as *Streptococcus mutans*.

This inhibition reduces biofilm formation and curtails the development of cariogenic environments(Sulieman, 2008:148).

In addition, many probiotics produce antimicrobial compounds, including bacteriocins, hydrogen peroxide, and organic acids, which create inhospitable conditions for cariogenic and periodontopathogenic bacteria. For instance, *Lactobacillus reuteri* synthesizes reuterin, a broad-spectrum antimicrobial agent that inhibits both *S. mutans* and *Porphyromonas gingivalis*(Sulieman, 2008:148).

Probiotics also play a role in modulating host immune responses, enhancing the production of secretory IgA and downregulating proinflammatory mediators such as IL-1 $\beta$  and TNF- $\alpha$ . These immunomodulatory effects are particularly relevant in reducing gingival inflammation in pediatric patients(Hove & ark., 2006:440).

Moreover, probiotics contribute to pH stabilization within the oral cavity by metabolizing carbohydrates into less acidic byproducts than those generated by pathogenic bacteria. Certain strains may also enhance salivary flow and buffer capacity, providing further protection against enamel demineralization(Hill & ark., 2014:506).

Collectively, these mechanisms foster a shift toward a healthassociated microbiota, promoting oral resilience during the formative stages of microbial development in children.

## Clinical Applications and Strain-Specific Evidence in Pediatric Oral Health

The clinical application of probiotics in pediatric dentistry has focused on conditions with a microbial basis, including early childhood caries (ECC), gingival inflammation, and halitosis. Selection of probiotic strains for these applications requires careful consideration of safety, colonization ability, and strain-specific efficacy, as not all probiotics demonstrate uniform biological activity(Caglar, Kargul & Tanboga, 2005:131).

Among the most widely studied strains is *Lactobacillus rhamnosus GG* (LGG), which exhibits strong adhesion to oral epithelium and suppresses *Streptococcus mutans* levels in saliva. Its incorporation into milk or chewable tablets has been shown in randomized controlled trials to reduce ECC incidence in preschoolaged children(Hong, Levy & Warren, 2009:345). Similarly, a double-blind, placebo-controlled study demonstrated that long-term consumption of LGG-supplemented milk significantly lowered caries risk, and a meta-analysis confirmed moderate yet significant caries-preventive effects when LGG was used alongside fluoride therapy (Cagetti & ark., 2013:2530).

*Lactobacillus reuteri* (DSM 17938 and ATCC PTA 5289) is another well-supported strain, known for producing reuterin—a broad-spectrum antimicrobial compound. Clinical trials have shown that *L. reuteri*-based lozenges reduce gingival bleeding and plaque scores, particularly in children undergoing orthodontic treatment or presenting with gingivitis(Vicario & ark., 2013:813). These effects are partly attributed to the strain's immunomodulatory properties, including the suppression of pro-inflammatory cytokines such as IL- $1\beta$  and TNF- $\alpha$ .

In addition, *Streptococcus salivarius* K12 and M18, derived from the oral cavity, have gained attention for their ability to produce bacteriocins like salivaricin A and B, which inhibit *S. mutans*, *S. pyogenes*, and *P. gingivalis*. These strains have been successfully formulated into lozenges for pediatric use and have demonstrated efficacy in reducing oral malodor by targeting volatile sulfur compound (VSC)-producing bacteria on the tongue dorsum (Wescombe & ark., 2012:1355). A systematic review supported their effectiveness in improving breath odor in both children and adolescents.

Emerging evidence also suggests potential roles for other strains such as *Weissella cibaria* and *Bifidobacterium animalis subsp. lactis*, although robust clinical data in pediatric populations remain limited (Kang & ark., 2020:243).

Despite promising outcomes, inconsistencies across clinical trials—in terms of strain selection, dosage, formulation, and treatment duration—underscore the need for well-controlled, long-term studies to establish standardized protocols. Current evidence supports a strain-specific approach, with *L. rhamnosus GG*, *L. reuteri*, and *S. salivarius* representing the most substantiated probiotics in pediatric oral care(Chauhan & ark., 2021:1635).

#### Safety, Dosage and Usage Duration

The administration of probiotics in children is generally considered safe, especially among healthy individuals with intact immune function. Strains such as *Lactobacillus rhamnosus GG*, *L. reuteri*, and *Streptococcus salivarius* K12 have been widely used in pediatric studies without significant adverse effects and are classified as GRAS (Generally Recognized As Safe) by authorities including the U.S. FDA and EFSA (Hove & ark., 2006:440).

Nevertheless, caution is advised in immunocompromised children, preterm infants, or those with invasive medical devices such as central venous catheters. In these populations, rare cases of probiotic-associated bacteremia or sepsis have been reported, although such events are typically related to systemic—not oral—use(Kuboniwa & Lamont, 2010:38). Therefore, safety evaluations should be both strain-specific and context-dependent, tailored to the child's underlying health status.

Recommended probiotic dosages for oral health in children generally range from  $10^8$  to  $10^{10}$  CFU/day. Clinical trials have employed daily doses of approximately  $10^9$  CFU for *L. rhamnosus GG*, while lozenges containing *S. salivarius* K12 have shown efficacy in halitosis and pharyngitis prevention at doses of  $10^8$ – $10^9$  CFU/day(Laleman & Teughels, 2015:255).

However, the beneficial effects of probiotics tend to be transient. Studies indicate that reductions in *S. mutans* levels and gingival inflammation occur primarily during active consumption and often return to baseline within weeks of discontinuation(Zaura & ark., 2014:85). Sustained and consistent intake may therefore be necessary to achieve and maintain clinical outcomes, especially in children at elevated risk for oral diseases.

Long-term safety and efficacy data remain limited. A 2024 systematic review highlighted the need for well-designed studies to define optimal usage duration, cumulative biological effects, and safety across varied pediatric subpopulations (Panchbhai & ark., 2024:10).

#### **Future Perspectives and Research Gaps**

Despite increasing interest and encouraging clinical findings, the routine integration of probiotics into pediatric dental practice remains limited due to unresolved scientific and clinical questions. One major challenge is the lack of standardized clinical protocols, including validated guidance on optimal strains, dosage, duration, and delivery vehicles (Twetman & Keller, 2012:98). Substantial heterogeneity across existing studies hampers the development of universal recommendations.

The mechanistic understanding of how probiotics influence the oral microbiome in pediatric populations is still evolving. Advances in sequencing and omics technologies offer insights, yet long-term, mechanistic trials in children remain limited (Twetman & Keller, 2012:98). Longitudinal, microbiome-based trials are necessary to deepen our understanding of host-microbe interactions.

An emerging research direction involves personalized probiotic strategies, tailored to an individual child's oral microbiota profile. Future clinical applications may include targeted selection of probiotic strains based on microbial composition, caries risk, or inflammatory status(Haukioja, 2010:348).

Finally, postbiotics—non-viable microbial metabolites or cell components—are gaining attention as safer and more stable alternatives to live probiotics, particularly for use in immunocompromised children(Aguilar-Toalá & ark., 2018:105). However, their role in pediatric oral care remains underexplored, as do health economic evaluations assessing cost-effectiveness in caries and gingivitis prevention among high-risk populations.

#### Conclusion

The use of probiotics in pediatric dentistry represents a promising adjunctive strategy for the prevention and management of common oral diseases such as dental caries, gingivitis, and halitosis. By modulating the composition and function of the oral microbiota, probiotics can promote microbial homeostasis, reduce pathogenic colonization, and support mucosal immunity in children.

Although clinical trials have demonstrated the efficacy of certain strains—such as *Lactobacillus rhamnosus GG*, *L. reuteri*, and *Streptococcus salivarius K12*—the benefits are strain-specific and transient, requiring consistent administration for sustained effects. Current evidence supports their short-term safety in healthy children, but further research is necessary to clarify long-term outcomes, especially in medically compromised populations.

Future directions point toward microbiome-guided, personalized probiotic applications and the incorporation of postbiotics as safer alternatives. With rigorous, standardized, and mechanistically driven studies, probiotics have the potential to become an integral component of pediatric preventive oral care.

#### References

Aguilar-Toalá, J., Garcia-Varela, R., Garcia, H., Mata-Haro, V., González-Córdova, A., Vallejo-Cordoba, B., & Hernández-Mendoza, A. (2018). Postbiotics: An evolving term within the functional foods field. *Trends in food science & technology*, 75, 105-114.

AM Sulieman, M. (2008). An overview of tooth-bleaching techniques: chemistry, safety and efficacy. *Periodontology 2000*, 48(1).

Cagetti, M. G., Mastroberardino, S., Milia, E., Cocco, F., Lingström, P., & Campus, G. (2013). The use of probiotic strains in caries prevention: a systematic review. *Nutrients*, *5*(7), 2530-2550.

Caglar, E., Kargul, B., & Tanboga, I. (2005). Bacteriotherapy and probiotics' role on oral health. *Oral diseases*, *11*(3), 131-137.

Chauhan, S., Kerr, A., Keogh, B., Nolan, S., Casey, R., Adelfio, A., . . . Wall, A. M. (2021). An Artificial-Intelligence-Discovered Functional Ingredient, NRT\_N0G5IJ, Derived from Pisum sativum, Decreases HbA1c in a Prediabetic Population. *Nutrients*, *13*(5), 1635.

Deo, P. N., & Deshmukh, R. (2019). Oral microbiome: Unveiling the fundamentals. *Journal of oral and maxillofacial pathology*, 23(1), 122-128.

Dominguez-Bello, M. G., Costello, E. K., Contreras, M., Magris, M., Hidalgo, G., Fierer, N., & Knight, R. (2010). Delivery mode shapes the acquisition and structure of the initial microbiota across multiple body habitats in newborns. *Proceedings of the National Academy of Sciences, 107*(26), 11971-11975.

Dzidic, M., Collado, M. C., Abrahamsson, T., Artacho, A., Stensson, M., Jenmalm, M. C., & Mira, A. (2018). Oral microbiome

development during childhood: an ecological succession influenced by postnatal factors and associated with tooth decay. *The ISME journal*, *12*(9), 2292-2306.

Firmino, R. T., Gomes, M. C., Clementino, M. A., Martins, C. C., Paiva, S. M., & Granville-Garcia, A. F. (2016). Impact of oral health problems on the quality of life of preschool children: a case–control study. *International journal of paediatric dentistry*, *26*(4), 242-249.

Haukioja, A. (2010). Probiotics and oral health. *European journal of dentistry*, 4(03), 348-355.

Hill, C., Guarner, F., Reid, G., Gibson, G. R., Merenstein, D. J., Pot, B., . . . Salminen, S. (2014). Activity of cecropin P1 and FA-LL-37 against urogenital microflora. *Nature Reviews Gastroenterology and Hepatology*, *11*(8), 506.

Holgerson, P. L., Vestman, N. R., Claesson, R., Öhman, C., Domellöf, M., Tanner, A. C., . . . Johansson, I. (2013). Oral microbial profile discriminates breast-fed from formula-fed infants. *Journal of pediatric gastroenterology and nutrition*, *56*(2), 127-136.

Hong, L., Levy, S., Warren, J., & Broffitt, B. (2009). Association between enamel hypoplasia and dental caries in primary second molars: a cohort study. *Caries research*, *43*(5), 345-353.

Hove, L., Holme, B., Øgaard, B., Willumsen, T., & Tveit, A. (2006). The protective effect of TiF4, SnF2 and NaF on erosion of enamel by hydrochloric acid in vitro measured by white light interferometry. *Caries research*, 40(5), 440-443.

Kang, M.-S., Lee, D.-S., Lee, S.-A., Kim, M.-S., & Nam, S.-H. (2020). Effects of probiotic bacterium Weissella cibaria CMU on periodontal health and microbiota: a randomised, double-blind, placebo-controlled trial. *BMC Oral Health, 20*, 1-12.

Kuboniwa, M., & Lamont, R. J. (2010). Subgingival biofilm formation. *Periodontology 2000, 52*(1), 38.

Laleman, I., Detailleur, V., Slot, D. E., Slomka, V., Quirynen, M., & Teughels, W. (2014). Probiotics reduce mutans streptococci counts in humans: a systematic review and meta-analysis. *Clinical oral investigations, 18*, 1539-1552.

Laleman, I., & Teughels, W. (2015). Probiotics in the dental practice: a review. *Quintessence International*, 46(3), 255-264.

Panchbhai, A. S., Khatib, M. N., Borle, R. M., Deolia, S. S., Babar, V. M., Vasistha, A. H., & Parida, R. P. (2024). Efficacy and Safety of Probiotics for Dental Caries in Preschool Children: A Systematic Review and Meta-analysis. *Contemporary Clinical Dentistry*, 15(1), 10-16.

Simon-Soro, A., Tomás, I., Cabrera-Rubio, R., Catalan, M., Nyvad, B., & Mira, A. (2013). Microbial geography of the oral cavity. *Journal of dental research*, *92*(7), 616-621.

Tanner, A., Mathney, J., Kent, R., Chalmers, N., Hughes, C., Loo, C., . . . Dahlan, M. (2011). Cultivable anaerobic microbiota of severe early childhood caries. *Journal of clinical microbiology*, *49*(4), 1464-1474.

Twetman, S., & Keller, M. (2012). Probiotics for caries prevention and control. *Advances in dental research*, 24(2), 98-102.

Vicario, M., Santos, A., Violant, D., Nart, J., & Giner, L. (2013). Clinical changes in periodontal subjects with the probiotic Lactobacillus reuteri Prodentis: a preliminary randomized clinical trial. *Acta Odontologica Scandinavica*, *71*(3-4), 813-819.

Wescombe, P. A., Hale, J. D., Heng, N. C., & Tagg, J. R. (2012). Developing oral probiotics from Streptococcus salivarius. *Future microbiology*, 7(12), 1355-1371.

Zaura, E., Nicu, E. A., Krom, B. P., & Keijser, B. J. (2014). Acquiring and maintaining a normal oral microbiome: current perspective. *Frontiers in cellular and infection microbiology*, *4*, 85.

# BRUXISM IN CHILDREN: CLINICAL FINDINGS, RISK FACTORS, AND MANAGEMENT STRATEGIES FOR PEDIATRIC DENTISTS

# TURKAN MAHYADDINOVA<sup>1</sup> FATMA NUR KIZILAY<sup>2</sup>

#### Introduction

Bruxism is defined as involuntary and non-functional teeth clenching or grinding movements of the masticatory system (Manfredini et al., 2013: 631). Although it is a common parafunction in childhood, its prevalence varies greatly depending on age and diagnostic criteria. Studies have reported a prevalence of 8–38% in children and adolescents; some reviews have reported a range from 3–5% to over 40% (Şentürk & Güzel, 2022). This is attributed to heterogeneity in results obtained using different methods and the fact that parents often fail to notice this habit. Bruxism is a condition that requires close monitoring by dentists, as it can affect both dental and temporomandibular joint tissues (Bulanda et al., 2021: 9544).

<sup>&</sup>lt;sup>1</sup> Pediatric Dentist Turkan Mahyaddinova, Alanya Oral and Dental Health Center, Orcid:0000-0001-5415-6084

<sup>&</sup>lt;sup>2</sup> Asst.Prof.Dr. Fatma Nur Kızılay, Department of Pedodontics, Faculty of Dentistry, Inonu University, Malatya, Turkey, Orcid:0000-0001-6588-663X

## **Definition and Classificationof Bruxism**

Bruxism is an involuntary jaw joint and muscle activity manifested by teeth clenching or grinding (Storari et al., 2023: 3-2023). It is classified as nocturnal (sleep) and diurnal (awake) bruxism depending on the time it occurs. Bruxism observed while awake is typically in the form of jaw clenching; during sleep, it can be both clenching and grinding. Additionally, it can be subdivided into idiopathic bruxism (occurring without a known organic cause) and iatrogenic bruxism (associated with factors such as neurological or psychiatric disorders, or medication use). While most cases of bruxism in children are of the nocturnal (sleep-related) type, both forms are important (Şentürk & Güzel, 2022).

#### **Etiology and Risk Factors**

Multiple factors play a role in the development of bruxism in children. The most commonly reported risk factors include malocclusion or TME-related local factors, psychosocial factors, genetic predisposition, and systemic conditions. For example, dental malocclusions, traumatic closures, or dental eruption anomalies can trigger bruxism as local stimuli (Huynh & Di Fabbro, 2024: 104). Psychologically, factors such as stress, anxiety, and hyperactivity increase the risk of bruxism; studies have shown that internal issues such as anxiety and unhappiness in preschool-aged children are associated with bruxism. A family history of bruxism may also indicate genetic predisposition. Additionally, respiratory issues such as asthma and allergies, systemic disorders such as gastroesophageal reflux, and even exposure to secondhand smoke have been linked to bruxism (Talebian et al., 2023: 81; Restrepo-Serna & Winocur, 2023: 1166091).

- Local factors (Dental/Oral): Factors such as malocclusion, premature contacts, high fillings, and eruption anomalies create dental-related stimuli (Şentürk & Güzel, 2022).
- Psychosocial factors: Stress, anxiety, anger, or emotional trauma in children facilitate the development of bruxism (Şentürk & Güzel, 2022; Talebian et al., 2023: 81).
- Systemic and environmental factors: Allergic conditions, digestive system problems (e.g., reflux), sleep apnea, and respiratory disorders are associated with bruxism (Restrepo-Serna & Winocur, 2023: 1166091).

#### **Clinical Findings**

The most typical finding in children with bruxism is wear on the tooth surfaces and flattening of the tooth ridges. This finding may appear locally or diffusely on the chewing surfaces. Additionally, enamel cracks, tooth sensitivity, tooth mobility, or traumatic lesions in the periodontal tissues may be observed (Bulanda et al., 2021: 9544). Hypertrophy or tenderness in the masticatory muscles (masseter, temporal muscles) is commonly reported, as well as jaw pain and headaches, especially in the morning. The sounds produced during teeth grinding are often noticed by parents. During the clinical examination, indirect signs such as lineal areas (linea alba) on the inner surface of the cheeks and tooth marks on the edges of the tongue may also be found (Emodi-Perlman et al., 2023: 2564).

#### **Diagnostic Process**

The diagnosis of bruxism is primarily based on a detailed medical history and physical examination. 1) Medical History: Parents and children are asked about sleep patterns, grinding sounds, jaw pain, and other symptoms. Psychological status and systemic disease history are also inquired about. 2) Clinical examination: Intraoral examination includes assessment of tooth wear, muscle sensitivity, and TME function. A detailed examination of the orofacial system is performed (Bulanda et al., 2021: 9544). 3) Additional methods if necessary: Polysomnography (PSG) is the gold standard in cases of sleep bruxism, but its use is limited in pediatric cases due to cost and difficulties. Instead, simplified measurement devices such as home-based EMG or devices such as BiteStrip® can be used (Huang & Tang, 2022: 825).

#### **Treatment and Follow-up Strategies**

Although bruxism in children is a condition that requires treatment, non-invasive approaches are preferred first. In cases with mild symptoms, the family should first be informed and stressreducing methods should be recommended. Relaxation exercises, behavioral modification techniques, and regular sleep hygiene can aid in treatment. A soft or hard night guard (occlusal splint) may be prescribed by the doctor to protect the tooth surfaces; these guards prevent tooth wear and may slightly reduce muscle activity (Ierardo et al., 2021: 58). If malocclusion or orthodontic abnormalities are present, appropriate orthodontic treatment may be planned. If underlying conditions such as anxiety, hyperactivity, or sleep apnea are identified, a multidisciplinary approach should be provided by referring the patient to the relevant specialists. Patient/parent education and follow-up: Since bruxism has a complex etiology, the family should be informed about the process, and the child's condition should be monitored at regular intervals. Behavioral and psychological methods: When necessary, support from a child psychologist or pedagogue may be sought; relaxation techniques and stress management training may be beneficial. Dental protective treatment: Night guards and friction appliances protect tooth surfaces (Scarpini et al., 2023: e006). Regular dental check-ups should be performed to monitor wear. Medical treatment: In very rare cases, short-term muscle relaxants or anxiolytic medications may be considered, but medication use in children should be avoided. Treatment of underlying disorders: If there are

accompanying pathologies such as sleep apnea, GERD, or chronic stress, medical treatment or surgery may be required (Nota et al., 2022: 1107).

## The Role of the Pediatric Dentist

The pediatric dentist is the first specialist to recognize bruxism in children at an early stage. During regular examinations, signs such as tooth wear patterns, muscle sensitivity, or jaw restriction should be looked for. The dentist should also assess risk factors by inquiring about the child's medical and psychosocial history (Şentürk & Güzel, 2022). In treatment planning, the patient and family are informed; recommendations to reduce stress are provided, and intraoral protective appliances (night guards) are prepared and used (Ierardo et al., 2021: 58). During the treatment process, the dentist coordinates the orthodontic or restorative corrections recommended to prevent bruxism. When necessary, the dentist collaborates with a pediatrician, neurologist, or psychiatrist to provide multidisciplinary care. Ultimately, the dentist plays a central role in the diagnosis, monitoring, and management of bruxism (Senff et al., 2023: e344).

## Conclusion

Childhood bruxism is a clinical condition that requires careful monitoring due to its multifactorial etiology, silent progression, and long-term effects. Although it often resolves spontaneously, in some cases it can lead to permanent dental damage and orofacial dysfunction. Therefore, it is crucial not to view bruxism as merely a temporary habit and to recognize the underlying physical, psychological, or systemic factors in a timely manner. Detailed history-taking and systematic clinical examination are the primary tools for diagnosis; in the treatment process, priority should be given to non-invasive, individualized, and holistic approaches. Multidisciplinary collaboration, along with family education, behavioral guidance, and preventive measures, are among the most critical factors determining treatment success.

The pediatric dentist is not only responsible for managing oral symptoms during this process but also serves as the first point of contact for the child's overall health. Effective management of bruxism begins with the dentist's clinical awareness; positive outcomes are achieved through proper guidance and consistent follow-up. Future controlled studies will contribute to the development of more evidence-based algorithms for the management of childhood bruxism.

#### References

Manfredini, D., Winocur, E., Ahlberg, J., Pekkan, G., & Lobbezoo, F. (2013). Prevalence of sleep bruxism in children: A systematic review of the literature. *Journal of Oral Rehabilitation*, *40*(8), 631–642.

Şentürk, Ö., & Güzel, K. G. U. (2022). Çocuklarda bruksizm: Derleme.

Bulanda, S., Szyszka-Sommerfeld, L., & Matthews-Brzozowska, T. (2021). Sleep bruxism in children: Etiology, diagnosis, and treatment—a literature review. *International Journal* of Environmental Research and Public Health, 18(18), 9544.

Storari, M., Garret-Bernardin, A., & Conti, P. C. R. (2023). Bruxism in children: What do we know? *European Journal of Paediatric Dentistry*, 24, 3–2023.

Huynh, N., & Di Fabbro, C. (2024). Sleep bruxism in children and adolescents—A scoping review. *Journal of Oral Rehabilitation*, 51(1), 103–109.

Talebian, A., Nouri, M., Sadeghi-Bazargani, H., & Alizadeh, M. (2023). Risk factors of bruxism in children and adolescents: A case-control study. *Iranian Journal of Child Neurology*, *17*(3), 81.

Restrepo-Serna, C., & Winocur, E. (2023). Sleep bruxism in children, from evidence to the clinic: A systematic review. *Frontiers in Oral Health*, *4*, 1166091.

Emodi-Perlman, A., Eli, I., & Winocur, E. (2023). Sleep bruxism in children—What can be learned from anamnestic information. *Journal of Clinical Medicine*, *12*(7), 2564.

Huang, M., & Tang, S. (2022). Sleep bruxism in children. *British Dental Journal*, 233(10), 825–825. Ierardo, G., Luzzi, V., & Polimeni, A. (2021). Treatments of sleep bruxism in children: A systematic review and meta-analysis. *CRANIO*, *39*(1), 58–64.

Scarpini, S., Varvara, G., & Ierardo, G. (2023). Associated factors and treatment options for sleep bruxism in children: An umbrella review. *Brazilian Oral Research*, *37*, e006.

Nota, A., Caruso, S., & Ehsani, S. (2022). Correlation between bruxism and gastroesophageal reflux disorder and their effects on tooth wear: A systematic review. *Journal of Clinical Medicine*, *11*(4), 1107.

Senff, J., Nunes, F. P., & Fagundes, N. C. F. (2023). Childhood and adolescents sleep bruxism treatment: A systematic review. *Sleep Science*, *16*(3), e344–e353.

# **MODERN PEDIATRIC DENTISTRY** SCIENTIFIC ADVANCES IN THE PRESERVATION OF CHILDREN'S ORAL HEALTH

