# EARLY CARIES IN CHILDREN: ETIOLOGY, DIAGNOSIS AND TREATMENT. A NARRATIVE REVIEW.

**Mihaela Anchidic[1†], Carmen Nicoleta Savin[2†], Monica Tatarciuc[3†], Oana Bejan[4*], Oana Maria Butnaru[5*],Cristina Cenusa[6†], Ioana Martu[3]**

[1]MSc Pediatric Dentistry, Zahnklinik Fur Kinder und jugendliche, Uster, Switzerland,; miha.anchidin@gmail.com
[2] UMPh "Grigore T. Popa" 700115, Iasi, Romania; Department of Surgery, Discipline of Pediatric Dentistry, Faculty of Dental Medicine, carmen.savin@umfiasi.ro
[3] UMPh "Grigore T. Popa" 700115, Iasi, Romania; Department of Oral Implantology, Removable Dentures and Technology, Faculty of Dental Medicine, monica.tatarciuc@umfiasi.ro, ioana.martu@umfiasi.ro,
[4] UMPh "Grigore T. Popa" 700115, Iasi, Romania; Orthodontic Resident, Faculty of Dental Medicine, bejanoana@gmail.com
[5] UMPh "Grigore T. Popa", 700115, Iași, Romania; Department of Surgery, Discipline of Basics of Physics and Biophysics in Dental Medicine, Faculty of Dental Medicine, oana.maria.butnaru@umfiasi.ro
[6]UMPh "Grigore T. Popa", 700115, Iași, Pediatric Dentistry Resident, Faculty of Dental Medicine, Romania; Cris_cenusa@yahoo.com

***** Corresponding author: Butnaru Oana Maria. e-mail: oana.maria.butnaru@umfiasi.ro
Bejan Oana. e-mail: bejanoana@gmail.com
† *Authors with equal contributions as the first author*

**Abstract:**
Dental caries remain a major oral health problem affecting children of small ages although it is a perfectly preventable disease. According to the American Association of Pediatric Dentistry, early childhood decay remains a major public health problem (AAPD, 2018). [1]
The definition of severe early childhood caries (S-ECC) is any sign of smooth-surface caries in a child younger than three years of age, and from ages three through five, one or more cavitated, missing (due to caries), or filled smooth surfaces in primary maxillary anterior teeth or a decayed, missing, or filled score of greater than or equal to four (age 3), greater than or equal to five (age 4), or greater than or equal to six (age 5)." The risk factors involved in the development of caries such as an imbalanced diet and high daily sugar intake are also involved in generating other diseases, such as diabetes or cardiovascular disease (WHO Report, 2003).[2] Reducing these risk factors could result in an overall improved quality of life which should be a priority for the medical health professionals. Reducing these risk factors should be a priority not for the dentist but also for all the medical health professionals as it can result in an overall improvement of the quality of life.

**Keywords:** *early caries in children, etiology, diagnosis, treatment.*

## Introduction

The term of early childhood caries (ECC) [3] was first proposed at a workshop held by the Center for Disease Control and Prevention (Kaste and Gift, 1995). According to AAPD (2016) ECC [4,5] "is defined as the presence of one or more decayed (non cavitated or cavitated lesions), missing (due to caries), or filled tooth surfaces in any primary tooth in a child under the age of six. The World Health Organization (WHO) [6] defines dental caries as "a localized, posteruptive microbial pathological process of external origin, which produces a softening of the dental hard

tissues, resulting in the formation of a cavity" [7].
The association between early childhood caries and socioeconomic status has been well documented.[8] Studies revealed that ECC is more prevalent in children with a low socioeconomic status.[9] The possible influence of socioeconomic status on dental health may also be a consequence of differences in dietary habits and the role of sugar in the diet.[10]

### *1. Etiology of caries in children:*

Over the years, hundreds of theories have emerged that tried to explain the development of caries, some of them are based on links in the etiopathogenic chain, others have only a historical value, as time proved them to be false [7]. Today dental caries is considered to be an infectious disease with chronic evolution, a chronic destructive process of multicausal nature [11].
Carious disease is considered a condition acquired as a result of the simultaneous interaction of at least three factors, which constitute Keyes' triad:

a) The host represented by the quality of dental hard structures and the quantity and quality of saliva
b) Food diet or fermentable nutrient substrate
c) Microbial flora colonizing the tooth surface
d) To these three factors is added a fourth represented by the exposure time of dental surfaces to the action of fermentation acids to produce demineralization. [12]

The etiology of ECC is multifactorial and has been frequently associated with a poor diet and bad oral health habits.[10] The factors that contribute to the development of ECC are the well-known factors leading to dental caries: cariogenic microorganisms, fermentable carbohydrates and susceptible tooth surface/host [13].
In the mouths of young children are present some particularities: newly established bacterial flora, immaturity of the host defense system, dietary substrate, which lead to severe and accelerated development of caries [13-15]. Inappropriate use of baby bottles (prolonged bedtime use of bottles with sweet content) has a central role in the etiology and severity of ECC. Most of the studies have shown significant correlation between ECC and bottle-feeding and sleeping with a bottle [10].
Also the constant negotiation and modeling of the diet by the parents, the transition from natural to artificial nutrition as well as the transition from liquid to semi-solid and solid nutrition, the shaping of personality and individual preferences. These elements bring the uniqueness of the etiological characteristics of dental caries in the case of temporary and mixed dentition .[16] Genetic factors have little influence in the production of dental caries compared to the factors listed above. It is a well-known fact that parents decisively influence children's eating habits and how to maintain oral hygiene, as they tend to imitate the behavior of adults.
Parents will blame the genetic factor for the appearance of carious processes in children, ignoring the food factor and oral hygiene. Heredity could be blamed in the case of dento-maxillary anomalies with crowding that favors plaque retention in areas with crowding, morphological anomalies, but there are not enough data to support this hypothesis.

### *2. Quality of hard dental structures*

Carious disease appears because of an imbalance that is produced at the level of tooth enamel. It is considered a disease with delayed expression because numerous acid bacterial attacks over a long period of time

are necessary for the appearance of cavity caries in the enamel. Dental enamel plays an essential role in the occurrence of the carious process. It is made up of hydroxyapatite crystals surrounded by a matrix made up of water, proteins and lipids. Through this matrix, which represents 10-15% of the enamel volume, continuous exchanges are made between the enamel surface and the oral environment[11]. Brudevold, in 1965,[17] studies the chemical composition of enamel and demonstrates that the enamel surface is much more resistant to caries than its subsurface. The enamel surface is more strongly mineralized and tends to accumulate an increasing amount of F, Zn, Pb and Fe than the immediately underlying layer. The surface is more difficult to dissolve in the acidic environment, contains less water and has more inorganic material than the adjacent substrate. These factors contribute to caries resistance and are partly responsible for the poor decay of the enamel surface compared to the adjacent substrate in the case of the initial carious lesion [7,18].

In children, the morphological characteristics of the teeth can be considered adjuncts in the initiation of the carious process:

- certain morphological features: deep and narrow occlusal grooves and pits, flat and wide interdental contacts thus favoring food retention and implicitly bacterial retention [11]
- deficiencies during the formation period of the dental organ that generate hypoplasias or hypocalcifications on which caries are more easily grafted [11]. Mallenby: enamel hypoplasia predisposes to the development of dental caries and the more teeth are affected by dystrophy, the more extensive the caries will be [7,19].
- the immaturity of recently erupted teeth, the teeth being more susceptible to caries in the first two years posteruptive, a period necessary for posteruptive maturation [20].

### *3. The nutritional substrate*

Nutrition affects the teeth during their development (food deficiencies of pregnant women affecting the quality of the child's dental structures), and malnutrition can exacerbate periodontal diseases as well as infectious diseases located in the oral cavity. The prenatal period is of limited importance, it is interested in the complete formation of the temporary dentition and the fetus always secures its needs from the maternal economy, in harsh conditions even by spoiling it. Protein intake is decisive for the formation of organic matrices, the quality of which is dependent on certain amino acids, provided to the greatest extent by natural nutrition from the first year of life, which artificial nutrition cannot imitate.

Thus, the diet can be the source of some changes in the dental structures that can favor the early grafting of the carious process. However, protein deficiencies must be accompanied by an increased consumption of carbohydrates to cause caries to appear (Zarnea). The most important effect of food on the oral cavity is the local action. Nutrition is the second determining factor in the genesis of caries, its implications being dominated by the harmful effect of carbohydrates and the lack of harmfulness of lipids and proteins.

Research has concluded that fats have a cariostatic effect, as the addition of lipids to cariogenic products reduces the incidence of caries.[18] Thus, chocolate containing cocoa butter is less cariogenic. The mechanism of the inhibitory effect is unknown, assuming that dietary lipids change the surface properties of enamel.[7] The harmful effect of carbohydrates has been proven by observations made on populations that have a

diet of exclusively vegetable origin (Polynesian islands) or exclusively of animal origin (Eskimos) and who have healthy teeth if they do not consume carbohydrates. With their introduction into the food ration, after the establishment of military bases and ports, caries appeared with greater frequency. This was also observed during the period of food restrictions during the Second World War, when the consumption of carbohydrates decreased greatly, leading to a decrease in the frequency of dental caries, and after the end of the war and the resumption of normal life, the frequency increased (Zarnea).

### *4. Microbial flora of caries in children*

Carious disease occurs when an imbalance occurs in the microbial community on the surface of the tooth, so that microorganisms with cariogenic potential become dominant. The microbial flora responsible for the production of dental caries is represented by various species of *Streptococcus* (*mutans, sanguis, salivarius, millieri*), *Lactobacillus* (*acidophilus* and casei), some types of *Actinomycetes*. *Streptococcus mutans,* found in all children with dental caries, refers to a heterogeneous group of streptococcus of which *S.Mutans* and *S.Sobrinus* are the most common microorganisms associated with caries. These microorganisms are commonly found in the oral cavity and are found in an ecological balance .[11]

*Streptococcus mutans* is considered to be the main bacterial species responsible for initiating the carious process. Although *S. mutans* is not detectable in the oral cavity of children before tooth eruption, several studies have shown that the age at which the child is infected with this microorganism is an important indicator of caries risk. The presence of *Lactobacillus* is also important. High level of *S. mutans* was correlated with increased number of caries. The impossibility of correlating the number of *Lactobacillus* with carioactivity shows that *Lactobacillus* cannot initiate the carious process alone but only correlated with *Streptococcus mutans*, by increasing plaque acid production (Toi et al. 1999).

At birth, the child's oral cavity is sterile. The process of population with micro-organisms is initially done through passive contamination from the mother, food, milk and water and from the saliva of individuals in the immediate vicinity of the child. Until now, the exact age at which infection with *Streptococcus mutans* occurs is unknown. Caufield et al. (1993)[16] show that this infection ("window of infectivity") occurs at 19 to 31 months of age. It is appreciated that the sooner the population with *Streptococcus mutans* is done, the sooner the carious experience of the individual will begin [11]. Studies by Aaltonen and Tenovuo (1994) have shown that frequent saliva transfer between mother and child has a protective effect. Children with an increased frequency of contact with maternal saliva before tooth eruption had lower numbers of *Streptococcus mutans* and fewer cavities compared to those with infrequent contact with maternal saliva. The possible explanation is that the child's exposure to cariogenic bacteria before tooth eruption increased the immunological resistance to caries.

### *5. Pathogenesis of caries in children*

The carious process is characterized by the demineralization and dissolution of hard dental structures caused by the significant drop in pH at the bacterial plaque-tooth interface and the demineralization of the tooth. The local decrease in pH is the result of plaque metabolism, but only plaques with high content of *Streptococcus mutans* and *Lactobacillus* can cause a pH low enough for tooth demineralization to occur. A single exposure to a glucose solution of the cariogenic plaque results in the metabolism

of the nutrients into organic acids (lactic acid) that dissociate, lowering the local pH. A single decrease in local pH is not sufficient to produce significant changes in the mineral content of the tooth surface.[18] Several such episodes, spent over a long period of time, will cause caries to appear. Frequent exposure to sucrose is the most important factor in maintaining a low pH in the tooth, frequently resulting in demineralization of its surface[21].

Below a critical pH of 5.5, the tooth minerals act like a buffer system, losing Ca and phosphate ions to the plaque. At a low pH of 3 or 4 the enamel surface is etched and rough. At pH 5, the surface of the tooth remains intact, while the minerals in the subsurface are dissolved.[18] This initial lesion confined to the enamel is incipient caries and is characterized by a virtually intact surface but a porous subsurface. This intact surface and the underlying porous substrate are responsible for the clinical features of the early lesion: the smooth intact surface becomes chalky white, opaque, especially when dry. When the porous body of the incipient lesion is hydrated, the lesion is not observed clinically, because the area remains translucent, a translucency that disappears after the removal of water from the underlying layer by drying, the area becoming opaque and whitish. The initial lesion may disappear through remineralization, restoring the enamel to health. If fluoride ions participate in remineralization, not only will the enamel be restored, but it increases its resistance to new acid attacks.

## *6. Diagnosis of early caries in children*

Determining individual caries risk is the key factor in modern dental caries management. It is imperative to establish the risk in order to find an optimal prevention strategy.[22] The best indicator of future carious activity, especially in children, is the detection of the earliest sign of caries which is demineralization.

Establishing individual caries risk guides provides choosing a reliable diagnostic and therapeutic method.[23] As no diagnostic method is perfect, overseeing or underestimating a lesion is inevitable. In these cases reevaluating the lesions after 6 months is advisable. In a few cases the lesion will expand a lot in 6 months (rampant caries) and the prognosis of the tooth will be compromised. In case we overestimate a carious lesion and place a restoration without it being necessary, the attitude is irreversible [24].

The diagnostic methods currently used for the diagnosis of early caries and cavitation are:

a) inspection of dental surfaces
b) bitewing radiography
c) fiber optic transillumination (FOTI)
d) laser fluorescence
e) ECM – diagnosis based on electrical measurements
f) infrared radiation
g) caries risk assessment
h) enameloplasty as a diagnostic and treatment method

## *7. Treatment of early caries in children*

The preventive treatment aims on the one hand to correct oral hygiene, [25] control and correct the diet, general fluoride supplements, to which the parent and the child contribute, and on the other hand the application of preventive measures by the dentist in the office: periodic professional hygiene, the application of fluorinated gels and varnishes, sealing of the pits and fissures. The actual curative treatment includes: remineralization through the topical application of fluorinated compounds, infiltration of lesions with resins, ozone therapy.[26]

***7.1. The role of fluoride in the prevention and remineralization of early lesions***

At the level of tooth enamel, ionic exchanges are constantly occurring in both directions from the saliva to the tooth and vice versa. Enamel is constantly exposed to demineralization and remineralization processes dependent on the ionic composition of saliva. In order for caries not to occur, there must be a perfect balance between these processes. [27,28] Immediately posteruptive the enamel is the most vulnerable as it is completely mineralized in the first two years posteruptive, recently erupted teeth are more vulnerable to caries. [29-33]. Knowing these, a series of measures can be taken that favor the formation of healthy and resistant dental structures. The main exogenous sources of fluoride are: water and foods such as cheese, meat, cereals, tea leaves, fish, paddy rice. The amounts of fluoride in food are small, the intake from water can be important in some areas where the water is fluoridated [34].

***Fluoride toxicity:***The reference dose for fluoride poisoning is 5 mg F/kg body weight. Signs of acute fluoride poisoning are: nausea, vomiting, abdominal pain, convulsions, paresthesias. The emergency treatment of poisoning involves the induction of vomiting and the protection of the digestive mucosa. To reach this lethal dose, a child would need to ingest 200 fluoride tablets of 0.25 mg or a tube of toothpaste. Cases of acute poisoning are relatively rare. The most frequent adverse reaction is dental fluorosis that occurs due to the consumption of an inadequate amount of fluoride until the age of 8-9 [34,35]

***Mouthwash:***

To optimize the protective effect of the toothpaste, it is recommended to use mouthwash 2 times a day. [36] Most mouthwashes contain sodium fluoride in varying amounts (0.2% NaF that is 225 ppm fluoride indicated for weekly rinses; 0.05% NaF that is 900 ppm fluoride for daily rinses), but they may also contain stannous fluoride, amino fluorides, acidulated fluoro orthophosphate (APF). The mouthwash is indicated for rinsing the oral cavity of children who have learned to spit. Studies by Poulsen have shown a 20-50% reduction in caries when using fluoride mouthwashes [24].

***Fluoride tablets:*** Fluoride tablets are not indicated if the drinking water has a fluoride concentration of more than 0.7 ppm fluoride. The tablets are taken orally, they are allowed to melt in the mouth, thus having a local and systemic action. Studies have shown that the use of fluoride tablets has reduced caries by 20-80% [24,35]

At home, weekly applications of 1.1% fluoride gels can be made by brushing followed by spitting out the excess, the method is addressed to children who have learned to spit.

Fluoride gels and fluoride varnishes are the local method of fluoride administration in the dental office.

***Gels with fluoride:*** Professional topical fluoridation is recommended to begin after age 2, with applications at least twice a year until age 15. It is recommended to fluoridate each newly erupted tooth as close as possible to the moment of eruption and mandatory in the first post-eruptive year [34].

***Fluoride-based varnishes:***They contain NaF 5%, it is applied simply and quickly, it requires a brief cleaning of the surfaces that can be dry or slightly wet, it is applied to the dental surfaces with a special applicator, it is left to evaporate for 1 minute. The child will avoid hard foods and the area will be brushed the next day. Two applications per year are recommended or in the case of early lesions 3 applications within a week [34].

The most used fluorine-based varnish is Duraphat, it contains a high concentration of

fluorine, 22600 ppm. The amount needed for both arches in a child up to 6 years old is the size of a pea, applied with a cotton swab. Studies in Scandinavia have shown a 50-70% reduction in caries [24].

**7.2.** ***Infiltration of early lesions with resins:***

Incipient carious lesions can be stopped from developing by sealing them with resins. The principle of the method is to create a resin coating on the surface of the lesion that prevents cariogenic acids, carbohydrates and bacteria from entering the lesion. The bacteria that can remain at the level of the lesion no longer receive a nutrient substrate and thus the lesion is stopped in its evolution or the evolution occurs more slowly.

Sealing aims to create a resin barrier on the surface of the lesion. Unlike sealing, the purpose of infiltration is to create a barrier within the lesion.

Early lesions are characterized by the presence of an apparently intact superficial layer of enamel, and beneath it is much more porous demineralized enamel.[37.38] These porosities are the pathway through which cariogenic acids penetrate. These pathways are closed by infiltration [31,32]. with resins so the progression of caries is slowed down or stopped. The affected enamel crystals are covered with resin and thus protected from dissolution, the fragile crystalline network of the lesion is stabilized by the resin matrix, thus stopping the cavitation process.

***7.3. Ozone therapy in the prevention and remineralization of early lesions*****:**

Ozone has proven to be a very powerful antimicrobial agent, is used in multiple medical fields, and since the 2000s is also available for dental use. Applying ozone for 10 seconds has been proven to eliminate over 99% of microorganisms associated with tooth decay. Ozone has an antimicrobial effect by oxidizing biomolecules of cysteine, methionine and histidine, interrupting bacterial metabolism and affecting the structure of bacteria at the level of the bacterial wall, causing their lysis. Thus, a link of the carious chain is removed and the remineralization of dental tissues is possible [38].

A study conducted in Germany in 1999 by Micheelis and Reich included 42 patients with 57 early carious lesions in the fissure system. The extent of initial lesions was assessed with DIAGNOdent. Ozone was administered through the HealOzone handpiece (KaVo) for 40 seconds to the fissure system affected by early caries (test tooth), and the contralateral tooth (control tooth) was not treated with any method. After the application of ozone, the test and control teeth were remeasured with DIAGNOdent; measurements were repeated at 1, 2, and 3 months and compared with baseline values before treatment. At the level of the test group, the analysis showed a significant improvement in the values, while at the level of the control group, no significant changes occurred [38].

Ozone therapy appears to be a promising alternative for the remineralization of early caries, but further studies are needed to introduce the widespread use of this method.

***8.*** **Conclusions:**

Dental caries remain a major oral health problem affecting children of small ages although it is a perfectly preventable disease. An in depth knowledge of the etiology and the early treatment is crucial for preventing dental caries in children. The risk factors involved in the development of caries such as an imbalanced diet and high daily sugar intake are also involved in generating other diseases. Reducing these risk factors should be a priority not for the dentist but also for all the medical health professionals as it can

result in an overall improvement of the quality of life.

**References**

1. The American Academy of Pediatric Dentistry (AAPD), Policy on Early Childhood Caries (ECC): Classifications, Consequences, and Preventive Strategies, Reference Manual, 2018, 40 (6):18-19
2. Petersen PE, The World Oral Health Report 2003: Continuous improvement of oral health in the 21st century - the approach of the WHO Global Oral Health Programme, Community Dent Oral Epidemiol. 2003, Dec;31 Suppl 1:3-23.
3. Congiu G, Campus G, Lugliè PF, 2014, Early Childhood Caries (ECC) Prevalence and Background Factors: A Review, Oral Health Prev Dent. 2014;12(1):71-6.
4. The American Academy of Pediatric Dentistry (AAPD), Policy on Early Childhood Caries (ECC): Classifications, Consequences, and Preventive Strategies, Reference Manual, 2016, 40 (6):18-19
5. The American Academy of Pediatric Dentistry (AAPD), Periodicity of Examination, Preventive Dental Services, Anticipatory Guidance/Counseling, and Oral Treatment for Infants, Children, and Adolescents, Reference Manual, 2018, 40 (6):18-19
6. WHO Expert Consultation on Public Health Intervention against Early Childhood Caries: Report of a Meeting, Bangkok, Thailand, 26–28 January, 2016
7. Carligeriu V, Bold A- Tratat de odontoterapie conservatoare și restauratoare, Timișoara, Ed. Mirton, 2002:137,154-172
8. Maxim, DC; Savin, CND; (...); Balan, A. Early Childhood Caries and the Quality of Life in Children. Rom. J. of Oral Rehab, 2013, 5 (1):50-55
9. The American Academy of Pediatric Dentistry (AAPD), 2011, Policy on Early Childhood Caries(ECC): Classifications, Consequences, and Preventive Strategies, Reference Manual, 2011, 40 (6):18-19
10. Colak H · 2013 · Early childhood caries update: A review of causes, diagnoses, and treatments. J Nat Sci Biol Med. **2013** Jan;4(1):29-38. doi: 10.4103/0976-9668.107257.
11. Luca Rodica-Pedodonție, vol.2, București, Cerma, 2003:5-21
12. Popa MB, Bodnar DC, Vârlan CM. Manual de odontoterapie restauratoare, București, Ed. Universitară Carol Davila, 2007:151-152
13. Vadiakas G. Of early childhood caries (ECC): A revisited review. Eur Arch Paediatr Dent. 2008 Sep;9(3):114-25. doi: 10.1007/BF03262622.
14. Solomon, SM; Matei, MN; (...); Iancu, LS. Evaluation of DNA Extraction Methods from Saliva as a Source of PCR - Amplifiable Genomic DNA. Rev Chimie, 2015, 66 (12):2101-3.
15. Popa, CG; Luchian, I; (...); Martu, S. ELISA Evaluation of RANKL Levels in Gingival Fluid in Patients with Periodontitis and Occlusal Trauma. Rev. de Chimie, 2018, 69 (6):1578-1580
16. Caufield PW, Cutter GR, Dasanayake AP. Initial Acquisition of Mutans Streptococci by Infants: Evidence for a Discrete Window . J Dent Res, 1993, 72: 37.

17. Yoon. SH, Brudevold, F. Fluoride, calcium, phosphate, ash, and water. 1965**,** J;44:696-700. doi: 10.1177/00220345650440041601. https://pubmed.ncbi.nlm.nih.gov .
18. Nicolae V, Neamtu B, Picu O, Martu Stefanache MA, Ibric Cioranu VS. The Comparative Evaluation of Salivary Biomarkers (Calcium, Phosphate, Salivary pH) in Cario-resistance Versus Cario-activity. Rev.Chim.(Bucharest), 2016,67(4):821-824
19. Mihalas, E; Ogodescu, A; (...); Savin, C. In vivo Experimental Study on the Influence of Sodium Fluoride and Amoxicillin/Clavulanic Acid on the Minerals Composition of Dental Enamel. Rev. de Chimie, 2017, 68 (2):269-272
20. McDonald RE, Avery DR. Dentistry for the child and adolescent, Elsevier, 2022:163-219
21. Roberson TM, Heymann HO, Swift EJ. Sturdevant's Art and Science of Operative Dentistry, Ed. Mosby Elsevier, 70-94
22. Harris R, Nicoll AD, Adair PM, Pine CM. Risk factors for dental caries in young children- a systematic review of the literature, Community Dental Health , 2004, 21 (Supplement):71–85
23. Pierce KM, Rozier RG, Vann WF Jr., Accuracy of pediatric primary care providers' screening and referral for early childhood caries, Pediatrics. 2002 May;109(5):E82-2.
24. Splieth Christian. Revolutions in Pediatric Dentistry, Quintessence Publishing, 2011: 2-10, 38-47, 77-88, 103-117.
25. Bogdan, M; Tica, I; (...); Budacu, C. Effect of 0.2% Chlorhexidine's use for Treatment of Localized Gingival Lesions in Patients with Type 2 Diabetes. Rev. de Chimie, 2016, 67 (12):2651-2653
26. Sfedu, AM; Trandafir, A; (...); Gabriela, G. Treatment of Early Childhood Caries in Primary Frontal Teeth - Case Report. Rom. J of Oral Rehab, 2022, 14 (3):221-225
27. Gavrila, L; Balan, A; (...); Savin, C. In vitro Study Regarding the Effect of Various Commercial Remineralizing Products on Primary and Permanent Teeth Dentine Caries Lesions. Rev. de Chimie, 2016, 67 (11):2228-2230.
28. Gavrila, L; Maxim, A; (...); Savin, C. Comparative Study Regarding the Effect of Different Remineralizing Products on Primary and Permanent Teeth Enamel Caries Lesions. Rev. de Chimie, 2015, 66 (8):1159-1161
29. Balan, A; Andrian, S; (...); Stoleriu, S. Comparative Study Regarding the Effect of Remineralizing Products on Primary Teeth Dissolution Induced by Acidic Drinks Rev. de Chimie, 2015, 66 (4): 562-564
30. Petcu, A; Savin, C; (...); Vasilca-Gavrila, LM. Atraumatic Restorative Treatment in Temporary Dentition. Int. J. of Medical Dentistry, 2017, 21 (1):49-51
31. Savin, C; Murariu, A; (...); Balan, A. Dental Material Biocompatibility: A Cross-Sectional Study. Int. J. of Medical Dentistry, 2017,21 (2):94-97
32. Petcu, A; Savin, C; (...); Antohe, ME. Biomaterials Involved in Frontal Area Restorations in Pediatric Dentistry. Rev. de Chimie, 2018, 69 (12):3473-3476
33. Raducanu AM. Tratamentul cariei simple si complicate la copii si adolescenti, Bucuresti, Ed. Bren, 2011:132-139.
34. Cuculescu M. Prevenție primară în carie și parodontopatie, București, Ed. Didactica si Pedagogică, 2010:242-249, 254-257, 268-286, 395,396
35. Petersen PE, Lennon MA. Effective use of fluorides for the prevention of dental caries in the 21st century: the WHO approach , Community Dent Oral Epidemiol, 2004; 32: 319–21. Blackwell Munksgaard, 2004.

36. Ogodescu, AS; Morvay, AA; (...); Savin, C. Comparative Study on the Effect of Three Disinfection Procedure on the Streptococcus pyogenes Biofihn formed on Plastic Materials used in Paedodontics and Orthodontics. Mater. Plastice. 2017, 54 (1):116-118
37. Kim S, Kim EY, Jeong TS, Kim JW, The evaluation of resin infiltration for masking labial enamel white spot lesions, Int J of Paediatric Dentistry, 2011, 21(4):241-248
38. Balan, A; Stoleriu, S; (...); Savin, C. In vitro Study Regarding the Effect of Different Types of Etching Acids on the Morphology and Chemical Content of the External Surface of Enamel Carious Lesion on Primary Teeth. Rev. de Chimie, 2015, 66 (1):70-73
39. Lynch Edward-Ozone: The revolution in dentistry, Quintessence, 2004:120-122,199-209.