



# CURRENT FAILURES IN THE CONSUMPTION OF SUGAR IN THE PRODUCTION OF DENTAL CARIES

ALBERTINA STĂNILĂ<sup>1</sup>, MONA IONAȘ<sup>2</sup>

<sup>1,2</sup>“Lucian Blaga” University of Sibiu

**Keywords:** dental caries, sugars, biofilm, sucrose, natural sweeteners, fluoride

**Abstract:** Inadequate dietary intake as well as increased intake of sugar and carbohydrates are among the etiological factors of dental caries. The aim of the study is to synthesize current research on the effect of sugar-rich nutrition on dental tissues. An electronic search in Science Direct and PubMed databases was conducted with search words such as dental caries, sugars, biofilm, sucrose, and natural sweeteners, a total of 362 articles were found, of which 59 items met the inclusion criteria. Following the evaluation, we can say that the contact between sucrose and oral biofilm determines its use as a substrate in order to generate increased amounts of organic acids, thus inducing a marked decrease in pH, sucrose and monosaccharides have a cariogenic effect. Natural sweeteners alter the microbiome of the oral cavity and are involved in preventing the transmission of *Streptococcus Mutans* in early childhood, fluoride has a safe and scientifically proven prophylactic effect, which through local and general administration contributes to the prophylaxis of dental caries.

## INTRODUCTION

Poor diet and increased sugar and carbohydrates are among the etiological factors of tooth decay.(1,2)

Maintaining the health of the oral cavity at optimal parameters is achieved by its microflora because of a balanced dietary intake. A high-CARB diet containing several kinds of sugars influences both inhibition and bacterial plaque formation.(3) The oral cavity is a complex, dynamic system. It houses over 700 distinguished bacterial species that normally live in symbiosis.(4) Dental caries is considered one of the most common health problems recorded globally, with a multifactorial etiology (5,6,7), which can be prevented, is irreversible, and does not present an increased severity. Untreated dental caries causes a number of complications that can lead to an alteration or a decrease in the quality of life.(3,8)

## AIM

The aim of the study is to synthesize current research on the effect of a high-sugar diet on dental tissues.

## MATERIALS AND METHODS

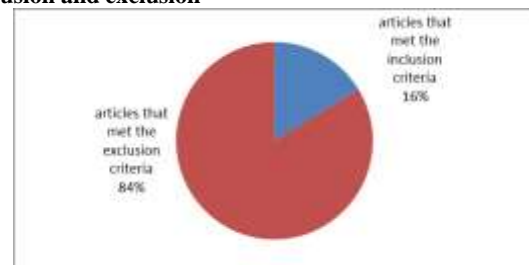
We researched literature data on the effects of sugar consumption on oral health using Science Direct and PubMed databases. We also consulted exclusively English publications, published in the period 1975-2022, using keywords such as dental caries, sugars, biofilm, sucrose, natural sweeteners, and fluoride. We have selected from the Science Direct and PubMed databases only original articles that correspond to search keywords and provide solid evidence of the research on the effects of sugar consumption on oral health.

Articles that were selected based on keywords by the database search engine and which we only had access to the summary and conference summaries and letters to the editor were excluded.

## RESULTS

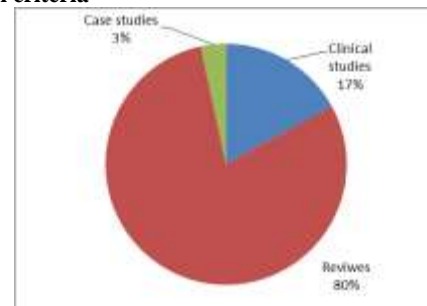
We examined 362 articles of which, 59 articles (16%) met the inclusion criteria, 303(84%) met the exclusion criteria (figure no. 1).

**Figure no. 1. Distribution of the articles through the basis of inclusion and exclusion**



Of the total number of articles that met the inclusion criteria: 3% were case studies, 17% clinical studies and 80% reviews (figure no. 2).

**Figure no. 2. Distribution of articles according to the inclusion criteria**



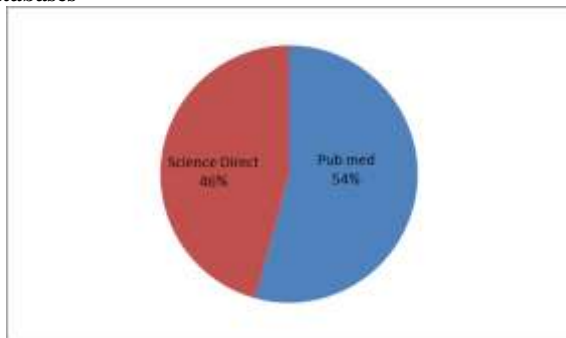
Out of the total of 59 articles examined, 32 articles

<sup>1</sup>Corresponding author: Albertina Stănilă, Str.Octavian Goga, Nr.43, Șelimbăr, România, Email:albertina.stanila@ulbsibiu.ro, Phone: +40757 060347  
Article received on 14.09.2022 and accepted for publication on 02.09.2022

## CLINICAL ASPECTS

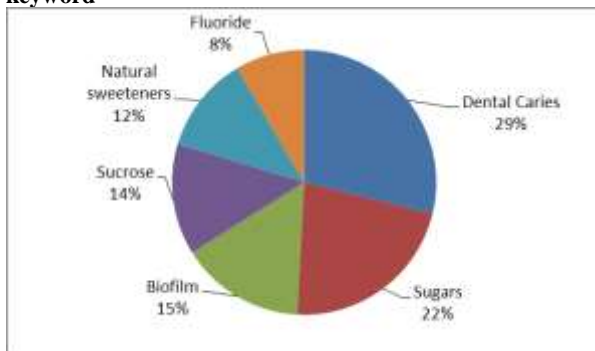
(54%) were selected from the Pub Med database, 27 articles (46) from the Science Direct database (figure no. 3).

**Figure no. 3. Distribution of articles according to selected databases**



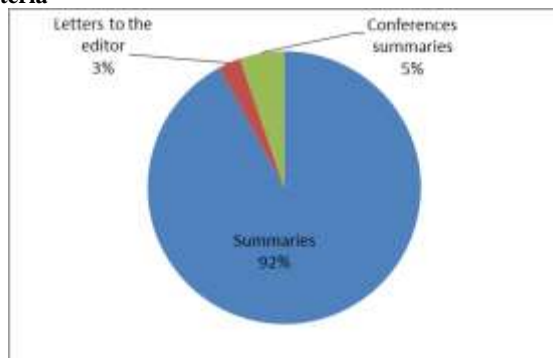
Using keywords in the search engine of the Science Direct and Pub Med databases such as dental caries, sugars, biofilm, sucrose, natural sweeteners, and fluoride we identified a number of relevant articles by the keyword used, namely: 29% of the total articles using the dental caries word, 13% using the word sugars, 15% using the word biofilm, 14% using the word sucrose, 12% using the word natural sweeteners and 8% using the word fluorine (figure no. 4).

**Figure no. 4. Distribution of articles according to the search keyword**



Of the 303 articles that met the exclusion criteria, 92% were summaries, 5% were conference summaries, and 3% were letters to the editor (figure no. 5).

**Figure no. 5. Distribution of articles on the basis of exclusion criteria**



## DISCUSSIONS

It is estimated that around 80% of adult people with permanent teeth will have dental caries by the age of 34.(8) Dental caries is a set of dynamic and complex demineralization and remineralisation processes that take place at the level of dental structures. The main etiological factor of dental caries is

considered to be the consumption of sugars in different forms. Ingestion of carbohydrates or sugars causes the flora of the oral cavity to continuously produce acids via the glycolytic pathway, with the stimulation of the long-term demineralization of enamel and the appearance of dental caries.(3) Dental caries results from the repeated demineralisation of the inorganic portion of the enamel and the alteration of its organic portion with the appearance of cavity and eventually tooth decay.(9,10) The resulting amplitude of the generated cariogenesis is influenced by the efficiency with which carbohydrates or sugars ingested by food intake by the oral flora are processed and metabolized.(10)

In the current context and following studies conducted over time, current oral prevention programmes are directed at Streptococcus Mutans, considered to be the most notable bacteria with cariogenic potential.(11,12,13,14,15) Streptococcus mutans are considered responsible for producing carious processes at the substance level.(16) Bacterial involvement in the production of dental caries multiple and involves in addition to Streptococcus Mutans other bacterial species such as the Streptococcus Sobrinus and lactobacilli. Oral bacteria with high cariogenic potential are therefore represented by Streptococcus Mutans and Streptococcus Sobrinus.(17)

There is the question of a window of infectivity in children aged 2-4 years, with temporary dentition, which shows most colonization of Streptococcus Mutans considered the primary etiological agent of dental carious processes.(18)

The increased cariogenesis of Streptococcus Mutans is due to the fact that it rapidly ferments sucrose in order to produce acids that in turn alter the mineral substances present in hard dental tissues, generating extracellular polysaccharides that increase the adhesion of bacterial species to the teeth and decrease the permeability of the bacterial plaque.(19,20) The adhesion, as well as the accumulation of Streptococcus Mutans, is due to the matrix composed mainly of insoluble glucans.(21)

As seen through molecular studies of the human microbiome, the oral ecosystem is composed of immeasurable bacterial species. It has also been found that oral pathogenic bacterial species can be found even in healthy people, who do not show carious processes.(22)

The frequency of occurrence of carious processes depends on the type of dietary carbohydrate consumed, the amount as well as the frequency of consumption.(23,24) A daily dietary intake rich in sucrose, ingested at least 3 times a day is associated with a 179% increase in the occurrence of carious processes.(25)

The World Health Organization for dental caries prevention recommends that sugar consumption should account for 5, a maximum 10% of daily caloric intake.(26,27)

A sugar intake of less than 10% of daily caloric intake is associated with low cariogenesis but does not exclude the formation of carious processes. The cariogenic risk decreases as the intake of sugars decreases.(28,29)

A decrease in sucrose intake below 15–20 kg/person/year or 40–55 g/day causes a reduction in the risk of developing carious processes.(30) The contact between sucrose and oral biofilm determined the use of sucrose as a substrate in order to generate increased amounts of organic acids, thus inducing a marked decrease in pH, (31,32) with demineralisation of dental tissue and change in the composition and virulence of oral biofilm.(31,33,34) Also, alterations in the bacterial composition of supragingival dental tartar are considered to be responsible by some authors for initiating carious processes. In this context, a transformation of the composition of oral biofilm takes place, incorporating more and more bacterial species when continuous and systematized deposition of microbes and bacteria occurs on dental

## CLINICAL ASPECTS

surfaces.(35,36)

Biofilm is the extracellular matrix of polymeric substances made up of commensal and pathogenic microorganisms in the form of colloidal dispersion which protects bacteria present in the oral cavity from the external environment and allows them to grow and become multi involved.(37) In order to obtain a mature dental biofilm, it is necessary the adhesion of the bacteria to the hard dental tissues.(38)

Dental plaque can be compared to a biofilm because like the biofilm it contains a very large number of bacteria in the form of a polymer matrix (39,40) but unlike the biofilm which is less susceptible to antimicrobial agents, dental plaque exhibits improved pathogenicity.(41)

Carbohydrates, the fuels necessary for the functioning of the human body, are consumed frequently. They are chemically classified into polyhydroxy aldehydes, ketones, alcohols, or acids, and can be found in the form of sugars, oligosaccharides, or polysaccharides with acetyl bonds.(42)

Of all these types of carbohydrates, sucrose holds the highest cariogenic potential due to the structural changes it induces in the oral biofilm, making the oral biofilm thinner, porous and more adherent to hard dental surfaces when is present as a sublayer. The association of sucrose with other carbohydrates causes an increase in its cariogenic potential.(43)

A dietary intake rich in sucrose and monosaccharides has an increased cariogenic potential because it causes a rapid decrease in salivary pH. (44,45,46,47,48,49) Numerous studies on the effects of high-CARB diets on oral health describe fructose and glucose as carbohydrates with a lower cariogenic potential than sucrose.(50)

As for the use of natural sweeteners over sugars, xylitol, which comes from the hydrogenation of xylose, is preferred among them and possesses about 95% of the sweetness of sucrose, being also a natural compound present in many fruits and vegetables (51) and erythritol. Both sweeteners alter the microbiome of the oral cavity are involved in preventing the transmission of Mutans streptococcal in early childhood, possessing both oral and systemic benefits.(52)

Primary prevention of dental caries is achieved by excluding at least one of the etiological factors, fluoride having a special place. The administration of fluoride and the use of food sweeteners are effective means of achieving widespread prevention of carious processes.(53) *In situ* studies have highlighted the quality of the fluoride to mitigate the consequences of dietary intake rich in sucrose.(54)

Although fluoride administration is an effective method of preventing dental caries when the intake of sucrose is high and frequent, the prophylactic effect of fluoride decreases considerably.(55)

Over time, multiple measures to prevent dental caries have been tested. To date there are the balanced diet, administration of fluoride, and sealing of the grooves and fosses of permanent teeth.(56,57)

Dental brushing and flossing are mechanical means used to remove plaque in order to remove potentially cariogenic bacterial species present in the oral cavity.(58,59)

## CONCLUSIONS

Dental caries is a set of dynamic and complex demineralization and remineralisation processes that take place at the level of dental structures. The main etiological factor of dental caries is considered to be the consumption of sugars in various forms. The contact between sucrose and oral biofilm causes it to be used as a substrate in order to generate increased amounts of organic acids, thus inducing a marked decrease in pH. Sucrose and monosaccharides have an increased cariogenic effect. Natural sweeteners alter the microbiome of the oral

cavity and are involved in preventing the transmission of Streptococcus Mutans in early childhood. Fluoride has a safe and scientifically proven prophylactic effect, which through local and general administration contributes to the prophylaxis of dental caries.

## REFERENCES

1. Ruottinen S, Karjalainen S, Pienihäkkinen K, Lagström H, Niinikoski H, Salminen M, Rönnemaa T, Simell O. Sucrose intake since infancy and dental health in 10-year-old children. *CariesRes*. 2004;38:142–148.
2. Sheiham A, James WP. Diet and dental caries: the pivotal role of free sugars reemphasized. *J Dent Res*. 2015;94:1341–1347.
3. Asma G, Benahmed AG, Maryam D, Maria A, Geir B. The role of sugar-rich diet and salivary proteins in dental plaque formation and oral health, *Journal of oral biosciences*. 2021;63(2):134-141.
4. Amy ES, William MW, Targeting. S. mutans biofilms: a perspective on preventing dental caries, *Medchemcomm*. 2019;10(7):1057–1067.
5. Eby A, Anil VA. Effectiveness of xylitol and polyol chewing gum on salivary streptococcus mutans in children: A randomized controlled trial, *Indian J Dent Res*. 2018;29(4):445-449.
6. Marsh PD. Microbial ecology of dental plaque and its significance in health and disease., *Adv Dent Res*. 1994;8:263-271.
7. Vinay P, Marie S, Carla H, Elisabeth T, Reinhard H, Joachim H, Jan K. Association of sugar-sweetened drinks with caries in 10- and 15-year-olds, *BMC Oral Health*. 2020;20:81.
8. Felix A, Terrick A, Kristen MA. Nanotechnology-based therapies for the prevention and treatment of Streptococcus mutans-derived dental caries, *Journal of oral biosciences*. 2021;63(4):327-336.
9. Smith DJ, Taubman MA. Experimental immunization of rats with a Streptococcus mutans 59 kDa glucan binding protein protects against dental caries, *Infect Immun*, 1996;64:3069–73
10. Johansson I, Witkowska E, Kaveh B, Lif Holgersson P, and Tanner ACR. The Microbiome in Populations with a Low and High Prevalence of Caries, *J Dent Res*. 2016;95(1):80–86.
11. Aurea SS, Alex M. Solving the etiology of dental caries, *Trends in microbiology*. 2015;23(2):76-82.
12. Loesche WJ, et al. Association of Streptococcus mutans with human dental decay, *Infect. Immun*. 1975;11:1252-1260.
13. Plonka KA, et al. Mutans streptococci and lactobacilli colonization in prenatate children from the neonatal period to seven months of age. *Caries Res*. 2012;46:213-220.
14. Kt S, et al. Dental caries vaccine – a possible option? *J Clin Diagn Res*. 2013;7:1250-1253.
15. Islam B, et al. Dental caries: from infection to prevention, *Med Sci Monit*. 2007;13:196-203.
16. Thar's MP, Carolina SO, Cristiane D, Regina Celia RP, Lidiany Karla AR, Marine's Nobre-dos-S. Relationship among microbiological composition and presence of dental plaque, sugar exposure, social factors and different stages of early childhood caries, *Archives of oral biology*. 2010;55:36–373.
17. Kazemtabrizi A, Haddadi A, Shavandi M, Harzandi N. Metagenomic investigation of bacteria associated with dental lesions: a cross-sectional study, *Med. Oral, Patol. Oral Cirugía Bucal*. 2020;25(2):240-251.

## CLINICAL ASPECTS

18. Pucc ST, Ueda S, Kuramitsu HK. Sequence analysis of the *gtfB* gene from *Streptococcus mutans*. *J Bacteriol*. 1987;169:4263–70.
19. Aubrey A. Sugars and dental decay, *The Lancet*; 1983. p. 282-284
20. Megha G. Sugar Substitutes: Mechanism, Availability, Current Use and Safety Concerns-An Update, *Open Access Maced J Med Sci*. 2018;25;6(10):1888–1894.
21. Parisotto TM, Stipp R, Rodrigues LKA, Mattos-Graner RO, Costa LS, Nobre-dos-Santos M. Can insoluble polysaccharide concentration in dental plaque, sugar exposure and cariogenic microorganisms predict early childhood caries? A follow-up study. *Archives of oral biology*. 2015; 60:109–1097.
22. Aurea Sn-S, Alex M. Solving the etiology of dental caries, *Trends in Microbiology*. 2015;23(2):76-82.
23. Moynihan P, Petersen PE. Diet, nutrition and the prevention of dental diseases, *Public Health Nutr*. 2004;7:201–26.
24. Bradshaw DJ, Lynch RJ. Diet and the microbial aetiology of dental caries: New paradigms., *Int Dent J*. 2013;63(2):64–72.
25. Ismail AI, Burt BA, Eklund SA. The cariogenicity of soft drinks in the United States. *J Am Dent Assoc*. 1984;109:241–5.
26. Cor van L. Sugar Restriction for Caries Prevention: Amount and Frequency. Which Is More Important? *Caries Res*. 2019;53(2):168–175.
27. Augustinho do Nascimento C, Ricci R Ruis Ferrari C, Martines de Souza B, Braga AS, Magalhães AC. Effect of sweetener containing Stevia on the development of dental caries in enamel and dentin under a microcosm biofilm model, *Journal of Dentistry*. 2021;115:103835.
28. Moynihan P. Sugars and Dental Caries: Evidence for Setting a Recommended Threshold for Intake. 2016;7(1):149–156.
29. Moynihan PJ. Effect on caries of restricting sugars intake: Systematic review to inform WHO guidelines. *J Dent Res*. 2014;93:531.
30. Sheiham A, James WP. A reappraisal of the quantitative relationship between sugar intake and dental caries: The need for new criteria for developing goals for sugar intake. *BMC Public Health*. 2014;14:863.
31. Natalia DG, Carla L, Rodrigo AG. Frequency of sucrose exposure on the cariogenicity of a biofilm-caries model, *Eur J Dent*. 2016;10(3):345–350.
32. Paes Leme AF, Koo H, Bellato CM, Bedi G, Cury JA. The role of sucrose in cariogenic dental biofilm formation – New insight. *J Dent Res*. 2006;85:878–87.
33. Marsh PD. Dental plaque as a biofilm and a microbial community – Implications for health and disease. *BMC Oral Health*. 2006;6(1):S14.
34. Takahashi N, Nyvad B. The role of bacteria in the caries process: Ecological perspectives. *J Dent Res*. 2011;90:294–303.
35. Rina Rani R. Dental biofilm: Risks, diagnostics and management, *Biotechnology*. 2022;43(C):102381.
36. Marsh PD. Dental plaque as a biofilm and a microbial community – implications for health and disease, *BMC Oral Health*. 2006;6(6):S14.
37. Adelaida Esteban-F, Irene Zorraquín-P, Dolores Gonz alez de L, Bego~na B, Victoria MA. The role of wine and food polyphenols in oral health, *Trends in Food Science & Technology*. 2017;69:118-130.
38. Koo H, Falsetta ML, Klein MI. The exopolysaccharide matrix: A virulence determinant of cariogenic biofilm. *J Dent Res*. 2013;92:1065–73
39. Socransky SS, Haffajee AD. Dental biofilms: difficult therapeutic targets, *Periodontology*. 2002;28:12-55.
40. Marsh PD. Dental plaque as a microbial biofilm, *Caries Res*. 2004;38:204-211.
41. van Steenberghe TJM, van Winkelhoff AJ, de Graaff J. Pathogenic synergy: mixed infections in the oral cavity, *Antonie van Leeuwenhoek*. 1984;50:789-798.
42. William RA. Lactose cariogenicity with an emphasis on childhood dental caries, *International dairy journal*. 2012;22(2):152-158.
43. Clarissa GS, Rodrigo AA, Lina NH. Effect of the association of maltodextrin and sucrose on the acidogenicity and adherence of cariogenic bacteria. *Archives of Oral Biology*. 2016;65:72–76.
44. Eun-JL, Hye-KyoungY, In-KyeongH, Kwang-Hak B, Bo-Hyoung J, Dai-ilP. Measurement of the cariogenicity of snacks using a radioisotope PAHA disc, *Archives of oral biology*. 2012;57(6):769-774.
45. Johansson I, Lif Holgersson P, Kressin NR, Nunn ME, Tanner AC. Snacking habits and caries in young children, *Caries Res*. 2010;44:421-430.
46. Seow WK, Clifford H, Battistutta D, Morawska A, Holcombe T. Case-control study of early childhood caries in Australia, *Caries Res*. 2009;43:25-35.
47. Van Houte J, Sansone C, Joshupura K, Kent R. Mutans streptococci and non-mutans streptococci acidogenic at low pH, and in vitro acidogenic potential of dental plaque in two different areas of the human dentition, *J Dent Res*. 1991;70:1503-1507.
48. Sansone C, Van Houte J, Joshupura K, Kent R, Margolis HC. The association of mutans streptococci and non-mutans streptococci capable of acidogenesis at a low pH with dental caries on enamel and root surfaces, *J Dent Res*. 1993;72:508-516.
49. Sheiham A, James WP. Diet and dental caries: The pivotal role of free sugars reemphasized. *J Dent Res*. 2015;94:1341–7.
50. Fatemeh AM, Loghman RS, Leila K, Mohammad YA, Jalal P, Masoud M. Effects of honey, glucose, and fructose on the enamel demineralization depth, *Journal of Dental Sciences*. 2013;8:147-150.
51. Marcio C, Patricia M, Isabel CFR. Sweeteners as food additives in the XXI century: A review of what is known, and what is to come, *Food and Chemical Toxicology*. 2017;10:302-317.
52. Zhan L. Rebalancing the Caries Microbiome Dysbiosis: Targeted Treatment and Sugar Alcohols, *Advances in dental research*. 2018;29(1):110–116.
53. Gabriella G, Maria D, Adele P. Food components with anticaries activity. *Current Opinion in Biotechnology*. 2012;23:153–159.
54. David JB, Richard JML. Diet and the microbial aetiology of dental caries: new paradigms. *International Dental Journal*. 2013;(63):64-72.
55. Ccahuana-Vásquez RA, Tabchoury CPM, Tenuta LMA, Del Bel Cury AA, Vale GC, Cury JA. *Caries Res*. 2007;41:9–15.
56. Andrew RG. Dental caries: Strategies to control this preventable disease. *Acta Medica Academica*. 2013;42(2):117-130.
57. Petersen PE, Ogawa H. *Community Dent. Health*. 2016;33:66–68.
58. Zero DT, Fontana M, Martinez-Mier EA, Ferreira-Zandona A, Ando M, Gonzalez-Cabezas C, Bayne SJ. *Am Dent. Assoc, JADA*. 2009;140:25S–34S.
59. Lee Y. *Diagnosis and Prevention Strategies for Dental Caries J. lifestyle Med*. 2013;3:107–109.