Review Article

CARIES SUSCEPTIBILITY IN CHILDREN- A REVIEW

Kaushal Chaudhari[1], Jignesh Rudani[1], Jay Patel[2]

[1]Tulane University School of Public Health and Tropical Medicine, New Orleans, Louisiana, USA,
[2]Rutgers, The State University of New Jersey, Newark, New Jersey, USA.

ABSTRACT
Caries is a unique multifactorial infectious disease. Our understanding of etiological factors, the progress of the disease, and the effectiveness of prophylactic procedures have led us to believe that we understand the disease. However, we still have too few answers to many questions. Early childhood caries affects 28% of children aged 2–6 in the US and is not decreasing. There is a well-recognized need to identify susceptible children at birth. Assessing a child's risk of developing caries is a vital component of caries management. A comprehensive caries assessment should consider factors such as past and current caries experience, diet, fluoride exposure, presence of cariogenic bacteria, salivary status, general medical history, behavioral and physical factors, and medical and demographic characteristics that may affect caries development. The purpose of this review article is to throw a light on these factors.
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Corresponding Author: Dr. Kaushal Chaudhari, Tulane University School of Public Health and Tropical Medicine, New Orleans, Louisiana, USA.

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INTRODUCTION

Dental caries is the dissolution of the enamel and dentin in pits, fissures, and interdental regions of the teeth, eventually spreading to buccal and lingual surfaces. Since the release of "Oral Health in America: A Report of the Surgeon General" in May 2000, efforts have not advanced dental caries prevention, its risk of development, or its early detection.[1] Worse, the severity of caries has since been increasing in all socioeconomic groups.[2] Public preventive measures such as water fluoridation are not universally available, few rural populations have access to fluoridated water, and fluoridated dentifrices are only effective if the teeth are brushed regularly.[3] Childhood caries is a major reason for hospital visits,[4] and it may destroy the deciduous dentition disproportionately in disadvantaged ethnic and socioeconomic groups.[5] A better means of identifying and protecting these children needs to be developed,[6] but a simple method of identifying such individuals a priori at birth has proved elusive.[7],[8] The notion that dental caries in animals is an infectious, transmissible disease was first demonstrated by Keyes.[9] Since then, a group of phenotypically similar bacteria, collectively known as mutans streptococci, has been implicated as the principal bacterial component responsible for the initiation and the development of dental caries. The tooth surface is unique among all body surfaces in two ways. First, it is a non-shedding hard surface, and, second, this surface is introduced into the human mouth during the first years of life. The earliest point at which the cariogenic mutans streptococci may become established is when the first teeth erupt. Solid surfaces are required for both streptococcal colonization and multiplication.[10],[11]

The relationship between the establishment of mutans streptococci and the initiation of dental caries in young children has been extensively studied. Several studies have shown that children who experience colonization by mutans streptococci early in life are at greater risk of developing dental caries than those who are colonized later.[11],[12] The extent of colonization of mutans streptococci and also, to some degree, subsequent caries activity experience are often correlated with the mother's salivary levels of mutans streptococci.[13] Once mutans streptococci become established, they are considered difficult

to eliminate, and the caries process is made possible.[11]

It is disconcerting to see rampant caries in young children. The pattern of decay is typically that many teeth are affected, with caries developing rapidly, often soon after the teeth have erupted. Surfaces usually at low risk of developing caries are affected such as the buccal surfaces of maxillary incisors with the obvious consequence of affecting the child's facial appearance. It is this pattern of caries that has been labelled variously as 'baby bottle tooth decay', 'nursing caries' and 'night bottle mouth'.

However, since these terms suggest that the prime cause of such caries is inappropriate bottle feeding and current evidence suggests that although use of a sugar-containing liquid in a bottle at night-time may be an important aetiological factor, it may not be the only or the most important factor, it is now recommended that the term 'early childhood caries' be used when describing any form of caries in infants and pre-school children.[14]

MICRO ORGANISMS AS RISK FACTOR

Mutans streptococci and lactobacilli historically have captured the greatest interest among researchers and clinicians. However, the accuracy of salivary tests for mutans streptococci in predicting future caries in the whole population is less than 50 percent.[15],[16] In populations with low caries prevalence, the caries-predictive ability of microbiological tests is even lower.[16] In addition, lactobacilli microbiological tests are less sensitive in predicting caries than are the tests for mutans streptococci.[17] In the United States, dentists can purchase several types of saliva tests to measure the amount of cariogenic microorganisms in saliva.[18]

Since the earlier the infection of mouth with Streptococcus Mutans, the greater is the caries risk of the deciduous dentition, and that since salivary transfer is required to spread the infection, nurturing habits such as cleaning a pacifier by putting in the mother's mouth before it is given to the child, kissing the child directly on the mouth, and pre-tasting food before it is given to the child have been studied.[19] However these studies have shown that a frequent transfer of saliva to the mouth of the baby from the mother is actually protective. Children with a high frequency of maternal salivary contact before tooth eruption had lower numbers of Streptococcus Mutans and less dental caries than those with rare contact, possibly because the infant's exposure to cariogenic bacteria prior to tooth eruption might have increased the child's immunological resistance to the infection.[14]

DIET AS A RISK FACTOR

Although the decline in tooth decay in many countries has been largely linked to fluoride exposure and improved dental hygiene, eating habits still affect the risk of tooth decay. The major dietary factors which influence caries in children include: **(i) Fermentable carbohydrates:** For many years the simplified message to prevent tooth decay was 'don't eat too much sugar and sugary foods'. Over the last few decades sugar intake in many countries has remained constant whilst caries levels have declined. This suggests that where appropriate oral hygiene is practiced (i.e. regular tooth brushing using fluoride toothpaste) the role of sugars in tooth decay is less manifest; **(ii) Food characteristics:** The physical characteristics of a food, particularly how much it clings to the teeth also influence the tooth decay equation. Foods that adhere to the teeth increase the risk of tooth decay compared to foods that clear from the mouth quickly; **(iii) Frequency of eating:** There is some debate over the relative importance of the frequency of consuming carbohydrate foods and its link with dental caries. As with the relationship between diet and caries, the link appears to have been weakened with the adoption of good oral hygiene and fluoride; and **(iv) Protective foods:** some foods help protect against tooth decay. For example hard cheese increases the flow of saliva. Cheese also contains calcium, phosphate and casein, a milk protein, which protects against demineralisation. Finishing a meal with a piece of cheese helps counteract acids produced from carbohydrate foods eaten at the same meal.[20]

SALIVA AS A RISK FACTOR

Whole saliva is a dilute, viscous solution whose electrolytes and proteins control the microbiota and prevent tooth enamel from dissolving. Major gland secretions are obtained using devices that are held tightly by suction to the orifice of the parotid gland duct in the cheek opposite the second upper molar, or to the orifices of the submandibular and sublingual glands together beneath the tongue.[21] Human parotid saliva secretions contain small quantities of urea, free amino acids, and peptides that could interact with bacterial metabolism in whole saliva to neutralize acids in the dental biofilm in situ. Indeed, caries-free subjects produce more ammonia from urea in

their biofilm, although they secrete the same amount of urea in parotid and whole saliva as caries-susceptible subjects. On the other hand, arginine and lysine contents are increased in the parotid saliva from caries-free individuals, but ammonia from arginine is increased only in whole saliva and has to diffuse into the biofilm to neutralize acids.[8] The greater lysine content of caries-free subjects in parotid saliva may be converted to cadaverine (a strong base) in the biofilm, but amounts are small, and only marginally greater than in caries-susceptible subjects. Small peptides containing lysine and arginine may also be metabolized to release ammonia in the biofilm. Nevertheless, bacterial metabolism seems inadequate to explain the greater neutralization of biofilm acids in caries-free subjects. Differences in saliva protein composition between caries-free and cariessusceptible individuals may provide a different and more satisfactory explanation that also accounts for intrinsic genetic differences.[8]

CULTURAL INFLUENCES AS A RISK FACTOR

Because the behavior of individual persons is an important determinant of caries and caries risk, it is clear that cultural and social influences play a role as well. When the prevalence of caries increased in the 16th century, the wealthy upper class was affected first because they could afford and used sucrose. A comparable pattern is now seen in African countries, where the prevalence and severity of dental caries tends to be higher in affluent urban areas, where sugars are more available than in rural communities. In newly industrialized countries, the incidence of caries increases when people switch from a dependence on traditional starchy staple foods to a dependence on refined carbohydrates. In most industrialized countries, persons with a relatively high risk of caries are found in the lower socioeconomic and immigrant groups, although differences seem to diminish in older age groups. In the Netherlands, children of Turkish or Moroccan mothers had a higher incidence of caries than did the native Dutch children. Cultural influences diminished as the children grew older.[22]

Kuusela et al.[23] studied odds ratios in 20 European countries, Canada, and Israel for the association between the self-reported family socioeconomic status of 11-y-old children and the consumption of soft drinks and sweets more than once per day. In general, self reported family socioeconomic status (good compared with poor and average) was positively associated with soft drinks or sweets consumed more than once daily; in the countries where the association was significant, the odds ratios varied between 2.9 and 20.2 for soft drinks and between 1.8 and 5.6 for sweets. These findings do not support a relation between the consumption of soft drinks and sweets and more caries in the lower socioeconomic class.[22] Freeman *et al.*[24] investigated determinants of reported snack consumption in adolescents in Belfast, Northern Ireland, and in Helsinki. Adolescents in Belfast had significantly higher levels of oral health knowledge, despite higher rates of consumption of snacks sweetened with sugars, than did Helsinki adolescents. In contrast, the adolescents in Helsinki had a more positive attitude toward their oral health. This study showed that knowledge may play a lesser role than attitude as a determinant of oral health behaviors.[22]

ORAL HYGIENE AS A RISK FACTOR

Studies have collected information on oral hygiene habits either by means of reported behaviour or more directly by using a plaque or debris index. There is evidence in more studies that toothbrushing once a day or more as opposed to less than once daily and the presence of visible plaque is important, than for other factors such as the frequency of toothbrushing comparing brushing three, twice and once daily, age at which toothbrushing was started, parental supervision of toothbrushing, not having teeth brushed at bed-time and the use of a fluoride as opposed to a non-fluoridated toothpaste.[14] Gibson and Williams (1999),[25] comparing children in Britain from families where the head of the household is employed in manual (Manual) as opposed to non-manual work (Non-Manual), found that the impact of toothbrushing frequency did not reach significance among children from the Manual group. A relationship was however evident among children from Non-Manual groups. Children from families with a Non-Manual head of household were more likely to have parental help in toothbrushing and the authors suggest that toothbrushing undertaken in these families was a more effective means of plaque removal and this was more important a factor than how many times a day toothbrushing was carried out.[14]

PAST CARIES EXPERIENCE

Epidemiological studies have shown a positive strong correlation between past caries experience

and future caries development. This single caries risk indicator provides the greatest predictive ability.[15],[26] The presence of caries in the mother increases a young child's risk.[15] Caries prevalence in primary teeth can help predict future caries in permanent teeth. In adults, there is an association between existing caries and the risk of developing root caries.[18]

MEDICAL & DEMOGRAPHIC FACTORS

Epidemiological surveys show that caries prevalence increases with age. In addition, newly erupted teeth are more susceptible to caries than are teeth that have erupted and have had a chance to mature in the oral cavity.[16],[18] Also, until the newly erupted teeth have reached the occlusal plane, they are difficult to clean, especially at pit and fissure sites. Socioeconomic status is a stronger predictor of caries risk in children than it is in adults.4 Because caries generally is more prevalent in lower socioeconomic groups than in higher socioeconomic groups, the dentist should consider social variables such as the patient's education and occupation. One example of how social variables can play a role in the determination of caries risk was presented in a study that showed that bakery workers have a higher prevalence of caries than do workers in other industries.[18],[27]

CONCLUSION

Whilst many studies have looked for predictors of caries in young children, many have not used the optimum study design, which is a longitudinal study. There is also a shortage of high quality studies, particularly those using validated measures for dietary and oral hygiene habits. A wide range of risk factors have been found to be significantly related to early childhood caries, and whilst factors relating to breast and bottle-feeding do feature, they are by no means the only factors. Further studies, conducted in different countries, on different social and ethnic groups, but using standardised data collection will help in understanding how socio-economic background and ethnicity help determine which young children develop dental caries.[14]

REFERENCES

1. S layton RL, Slavkin HC. Scientific investments continue to fuel improvements in oral health (May 2000- Present). Acad Pediatr 2009;9:383–5.
2. Milgrom P, Zero DT, Tanzer JM. An examination of the advances in science and technology of prevention of tooth decay in young children since the surgeon general's report on oral health. Acad Pediatr 2009;9:404–9.
3. Beltran-Aguilar ED, Barker LK, Canto MT *et al.* Surveillance for dental caries, dental sealants, tooth retention, edentulism, and enamel fluorosis—United States, 1988–1994 and 1999–2002. MMWR. Surveillance Summaries 2005;54:1–43.
4. Sheller B, Williams BJ, Lombardi SM. Diagnosis and treatment of dental caries-related emergencies in a children's hospital. Pediatr Dent 1997;19:470–5.
5. Azevedo TDPL, Bezerra ACB, De Toledo OA. Feeding habits and severe early childhood caries in Brazilian preschool children. Pediatr Dent 2005;27:28–33.
6. Abiko. Passive immunization against dental caries, and periodontal disease: development of recombinant and human monoclonal antibodies. Crit Rev Oral Biol Med 2000;11:140–58.
7. Aleksejuniene J, Holst D, Brukiene V. Dental caries risk studies revisited: causal approaches needed for future inquiries. Int J Environ Res Publ Health 2009;6:2992–3009.
8. Levine M. Susceptibility to dental caries and the salivary proline-rich proteins. Int J Dent 2011;2011:953412.
9. Keyes PH. The infectious and transmissible nature of experimental dental caries. Arch Oral Biol 1960;1:304-20.
10. Loesche WJ. Role of Streptococcus mutans in human dental decay. Microbiol Rev 1986;50:353-80.
11. Lenander-Lumikari M. Loimaranta V. Saliva and Dental Caries. Adv Dent Res 2000;14:40-7.
12. Alaluusua S, Renkonen O-V. Streptococcus mutans establishment and dental caries experience in children from 2 to 4 years. Scand J Dent Res 1983;91:453-7.
13. Li Y, Caufield PW. The fidelity of initial acquisition of mutans streptococci by infants from their mothers. Dent Res 1995;74:681-5.
14. Harris R, Nicoll AD, Adair PM, Pine CM. Risk factors for dental caries in young children: a systematic review of the literature. Commun Dent Health 2004;21 (Supplement):71–85.

15. National Institutes of Health. Diagnosis and management of dental caries throughout life. Bethesda, Md.: National Institutes of Health;2001.
16. Reich E, Lussi A, Newbrun E. Caries-risk assessment. Int Dent J 1999;49:15-26.
17. Wilson RF, Ashley FP. Identification of caries risk in schoolchildren: salivary buffering capacity and bacterial counts, sugar intake and caries experience as predictors of 2-year and 3-year caries increment. Br Dent J 1989;167:99-102.
18. Fontana M, Zero DT. Assessing patients' caries risk. JADA 2006;137:1231-9.
19. Aaltonen AS, Tenovuo J. Association between mother-infant salivary contacts and caries resistance in children: a cohort study. Pediatr Dent 1994;16:110–6.
20. Dental health. Available from: http://www.eufic.org/article/en/expid/basics-dental-health/.
21. Wolff A, Begleiter A, Moskona D. A novel system of human submandibular/sublingual saliva collection. J Dent Res1997;76:1782–6.
22. Touger-Decker R, van Loveren C. Sugars and dental caries. Am J Clin Nutr 2003;78(suppl):881S–92S.
23. Kuusela S, Kannas L, Tynjälä Jyväskylä J, Hinkala E, Tudor-Smith C. Frequent use of sugar products by schoolchildren in 20 European countries, Israel and Canada in 1993/1994. Int Dent J 1999;49:105–14.
24. Freeman R, Heimonen H, Speedy P, Tuutti H. Determinants of cariogenic snacking in adolescents in Belfast and Helsinki. Eur J Oral Sci 2000;108:504–10.
25. Gibson, S., and Williams, S. Dental caries in pre-school children: associations with social class, toothbrushing habit and consumption of sugars and sugar-containing foods. Caries Res 1999;33:101–3.
26. Disney JA, Graves RC, Stamm JW, Bohannan HM, Abernathy JR, Zack DD. The University of North Carolina Caries Risk Assessment study: further developments in caries risk prediction. Community Dent Oral Epidemiol 1992;20:64-75.
27. Petersen PE. Dental health among workers in a Danish chocolate factory. Community Dent Oral Epidemiol 1983;11:337-41.

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