Early childhood caries: a New Zealand perspective

Katie Bach BDS (Otago);[1] **David J Manton** BDSc (Melb), MDSc, PhD, FRACDS, FICD, FADI[2]

ABSTRACT

Dental caries, primarily a preventable disease, remains the most common chronic disease of childhood and one of the most common reasons for hospital admissions for children in New Zealand. The most vulnerable children are shouldering the burden of the disease, with Maori and Pacific children having greater experience and severity of dental caries. Early childhood caries has deleterious effects on a child's oral and general health and significant numbers of preschool-aged children experience pain and infection. Early identification by primary health care providers of children at high risk of developing early childhood caries can ensure these children are referred to the appropriate oral health services to receive appropriate and timely management.

KEYWORDS: Dental care for children; dental caries; New Zealand; preschool child

[1] Postgraduate student in Paediatric Dentistry (Health Workforce New Zealand Advanced Trainee Fellowship), Melbourne Dental School, Faculty of Medicine Dentistry and Health Sciences, The University of Melbourne, Melbourne, Australia

[2] Elsdon Storey Chair of Child Dental Health, Melbourne Dental School, Faculty of Medicine Dentistry and Health Sciences, The University of Melbourne

Epidemiology

Early childhood caries (ECC) is classified as the presence of one or more decayed, missing (due to caries) or filled teeth in a child under the age of 72 months.[1] Early childhood caries is a significant problem in New Zealand, with school dental data showing a prevalence of around 50% in children at age five.[2] The disease has a skewed distribution, with over 58% of Maori and 64% of Pacific children experiencing caries by age five.[2] Whereas many societies are experiencing a decrease in caries experience for their general populations, this is not the case for preschoolers. Over the past decade in New Zealand, there has been no decrease in prevalence or severity of ECC in the preschool population. The management of ECC requires considerable cooperation from a young child; therefore, general anaesthesia is often required to provide high quality comprehensive treatment for these children. Between 2000 and 2009, dental-based admissions made up 7.3% of all New Zealand hospital admissions for children aged 29 days to 14 years; additionally, dental conditions were the leading cause of potentially avoidable hospital admissions.[3]

Significant health inequalities exist in New Zealand and there is a definite socioeconomic gradient for the burden of ECC in the preschool population. In Australia, the number of decayed, missing and filled teeth is 70% higher in children in the lowest socioeconomic status households, as compared to those of the highest socioeconomic status.[4] In New Zealand, Maori and Pacific children in general have poorer health outcomes and this certainly applies with ECC; 88% of five- to six-year-old children in Te Tai Tokerau had dental caries, with an average of 5.6 decayed, missing or filled teeth per child.[5] Almost one in four of these children had already had a tooth extracted due to dental caries.[5] In the early 1990s, non-Maori children were three times more likely to have no caries than Maori children.[6] Unfortunately, although it has been commonly accepted that Maori and Pacific children have poorer health outcomes, there has been little change in this trend. More recent data from Christchurch and Wellington found that the mean decayed, missing or filled surfaces (DMFS) of Maori five-year-olds was double that of the 'other' ethnicities group, and the mean DMFS for Pacific children was three times that of the 'other' group.[7]

Children with a greater number of decayed, missing or filled teeth have decreased oral health-related quality of life.[8] Children in lower socioeconomic status households begin brushing

J PRIM HEALTH CARE 2014;6(2):169–174.

CORRESPONDENCE TO:
Katie Bach
Melbourne Dental School, Faculty of Medicine Dentistry and Health Sciences, The University of Melbourne, 720 Swanston Street, Melbourne 3010, Australia.
k.bach@student.unimelb.edu.au

their teeth at a later age, have their teeth brushed less frequently and are more likely to access dental services only when teeth become symptomatic.[9] Experience of dental caries in the primary dentition is a strong indicator that an individual will continue to experience dental caries into adulthood. Having more than two surfaces of the primary molars affected by caries at age five identifies a child as high risk for developing caries in the permanent dentition.[10] There is now increasing evidence that poor oral health in adults is associated with poor general health and systemic conditions, such as cardiovascular disease, stroke, and respiratory diseases, such as aspiration pneumonia.[11–13]

Aetiology

Dental caries is a complex multifactorial disease that occurs when there is a microbiological shift in the commensal oral microflora, promoting a biofilm favouring acidogenic and aciduric bacteria due to frequent exposure to fermentable carbohydrates.[14] Once the acidic environment has been established, if the plaque is left undisturbed and provided with a continued supply of fermentable carbohydrates, aciduric bacteria can promote the development of the carious lesion, as they are able to sustain an environment that is characterised by net mineral loss from the tooth.[15] Many of the micro-organisms in the plaque can produce acids that cause demineralisation of the tooth surfaces when exposed to dietary sugars. However, if these episodes are mild and infrequent, the homeostatic mechanisms in the plaque may restore the mineral balance to an overall mineral gain that will lead to remineralisation of the tooth.[15]

The areas of demineralised enamel, known as 'white spot lesions', are the beginning of the carious lesion. With continued supply of fermentable carbohydrates, the lesion can progress rapidly and become cavitated—a point at which surgical intervention is often required. Early childhood caries often begin as white spot lesions on the upper maxillary incisor teeth. These can be identified by lifting the lip of a young child and inspecting the upper incisor teeth for areas that appear whiter than the surrounding enamel, or for brown discoloration and or cavitations. This 'lift the lip' technique is especially important in young children, as in children from nine months until 18 months of age, white spot, non-cavitated lesions are most prevalent. After 18 months of age, cavitated lesions become more prevalent.[16]

Risk factors

Diet

The main dietary factors increasing a child's risk of developing ECC are the frequency, amount and timing of consumption of sugars, especially sucrose. There is a strong association between the frequency of consumption of sugar and the prevalence of caries, with a weaker association with the amount of sugar consumed.[17] Children with ECC are more likely to consume sugar between meals, especially in the form of fruit juices and soft drinks. Daily bottle-feeding with juices, cordials and soft drinks greatly increases the prevalence of ECC.[18]

New Zealand children are consuming increasing amounts of sucrose in their diets, with the main sources being powdered drinks, soft drinks and cordials and fruit drinks.[19] The 2002 National Children's Nutrition Survey found beverages were the most common vectors of sucrose delivery in New Zealand children, with 26% of sucrose in the diet coming from beverage consumption,[20] and this is likely to be higher now due to increasing soft drink consumption. It was reported that soft drinks featured six times in the top 30 selling supermarket products, with two sizes of cola drinks featuring twice in the top 10 selling products.[21] Non-diet soft drinks made up 32% of sales of non-alcoholic beverages (excluding milk), powdered cordials making up 23% and fruit juices 20%.[21] Increased intakes of soft drinks is associated with decreased intake of milk and other dairy products.[22]

Infant feeding

Infant feeding practices greatly influence a child's risk of developing ECC. Infant formulas have a higher cariogenic potential than regular bovine milk, as the lactose in bovine milk is not fermented to the same degree as other sugars, such as sucrose and fructose.[23,24] Mammalian milks

contain casein, calcium and phosphate, which inhibit demineralisation and promote remineralisation of enamel.[25] Although milk itself is not a highly cariogenic food (unless provided *ad libitum* in a feeding bottle overnight), it is often used as a vector for other sugars, such as those found in flavourings.[23] Soy-based beverages have significantly more cariogenic potential than bovine milk and limited buffering ability; the added calcium in most commercially available soy-based beverages has low solubility and, therefore, low bioavailability, especially due to phytate chelation of calcium.[26]

Developmental defects

Developmental defects of enamel (DDEs), both hypomineralisation (less mineral, normal thickness) and hypoplasia (normal mineral, decreased thickness), are often seen in primary and permanent teeth. These defects result from inherited and acquired systemic conditions that can cause damage or disruption to the developing enamel organ.[27] There is variability in the appearance of hypomineralised teeth; hypomineralisation can present as a localised, well-demarcated area on the tooth, or can affect the entire tooth and the colour varies from creamy white-yellow to brown. Primary second molars are developing in a similar time-period to first permanent molars and the two share possible risk factors for hypomineralisation; children with hypomineralisation on their primary second molars have a greater risk of developing molar-incisor hypomineralisation.[28] As yet, there is no published information on the prevalence of DDEs in primary teeth in New Zealand. However, we do have information on the prevalence of hypomineralisation in permanent teeth; 18.8% of children in a Wellington cohort were found to have hypomineralisation of their first permanent molars.[29] Hypomineralisation and hypoplasia in primary teeth has a positive correlation with increased dental caries and poorer outcomes.[30]

Toothbrushing

The presence of plaque in the mouth is a significant risk factor for ECC. Preschoolers who have a visible accumulation of plaque at 12 months of age are more likely to have ECC at three years of age than children who are plaque-free at 12 months.[31] Consistent with this finding, children in whom toothbrushing was commenced before 12 months have lower caries experience than children who began brushing after 13 months.[18] Parents who are dentally aware and motivated are likely to begin brushing habits earlier and show appropriate oral hygiene behaviours and oral health education levels.[18] Children under the age of six years have limited manual dexterity skills and are not able to adequately remove plaque from their teeth unassisted. It is no surprise that preschool-aged children who brush their own teeth have much higher levels of visible plaque than children who have their teeth brushed by a parent or guardian.[31] One suggestion to quell arguments relating to child independence is that the child brushes the teeth in the morning, and the parent brushes the teeth after dinner before bed.

Hyposalivation

Saliva is an important protective factor that contains many inorganic and organic components that can contribute to prevention of the development of carious lesions. When there is a disruption or alteration in saliva flow or quantity, there is an increased risk of dental disease.[32] Children with respiratory diseases tend to have altered salivary function both due to their tendencies to mouth breath and some prescribed drug therapies.[32]

Consequences of early childhood caries

Early childhood caries significantly affects a child's oral and general health and quality of life. Common outcomes include pain, infection, loss of sleep, alterations in behaviour and missed school. Dental caries can have significant effects on the growth and development of preschool-aged children and may have a role in failure-to-thrive in otherwise healthy children.[33] Children with at least one decayed tooth involving the pulp (nerve) were reported to be on average one kilogram lighter than the healthy controls.[33] Health improves with treatment and children typically undergo a period of catch-up growth after comprehensive treatment, as a pain-free,

functional dentition is required for good nutritional intake.[33]

Oral infections can compromise a child's general health, with 47–50% of facial cellulitis episodes in the paediatric population being due to an underlying odontogenic infection.[34] Localised infection and sepsis is a common consequence of ECC. It was found that approximately 4% of Scottish five-year-olds had evidence of dental sepsis and, when findings were stratified for socioeconomic status, 11% of children in low socioeconomic status households had evidence of sepsis, significantly higher in children with untreated decay.[35]

With increasing severity of ECC comes decreased oral health–related quality of life.[8] Comprehensive treatment of dental caries under general anaesthesia improves a preschool-aged child's quality of life significantly, as illustrated in multiple studies across New Zealand.[36–38] In particular, Maori children were found to have significant improvements in oral symptoms post-treatment compared to their counterparts from other ethnic groups.[37]

Fluoride exposure

The exposure to fluoride provides an important protective factor against dental caries. Fluoride has three major effects on teeth. It inhibits demineralisation and drives remineralisation by incorporating into the enamel crystals at the tooth surface as fluorapatite, which is less soluble than hydroxyapatite. Fluoride enhances remineralisation of enamel by attaching to the surface and adsorbing calcium and phosphate ions from the saliva. In high concentration, it inhibits the plaque bacteria's metabolism, therefore decreasing acid production.[39] Fluoride is most effective when used topically, and a lifetime exposure to fluoride is important, as it is a key component in driving the remineralisation, demineralisation process.[40]

Fluoridated toothpaste

Use of fluoridated toothpaste is a well-accepted, widely used method of delivering topical fluoride to prevent dental caries. Toothpaste comes commercially in various strengths—a typical child's toothpaste has approximately 500 ppm fluoride (a low concentration to putatively reduce the prevalence of enamel fluorosis), whereas toothpaste formulated for children over the age of six years and adult strength toothpaste will have 1000–1450 ppm fluoride. A Cochrane systematic review has found that there is limited evidence regarding the prevention of caries after use of toothpastes with less than 1000 ppm fluoride.[41] Worldwide, there are conflicting guidelines regarding at what age children should begin using toothpaste and when the move to 1000 ppm fluoride toothpaste should occur. The New Zealand Ministry of Health guidelines recommend that 1000 ppm fluoride toothpaste be used for children of all ages, and notes that a smear should be used up until five years of age, and a pea-size amount used for children six years and older.[42]

Water fluoridation

Water fluoridation is a public health measure that reduces the prevalence of dental caries by approximately 30%.[7] New Zealand children residing in fluoridated areas have significantly improved oral health outcomes when compared to children residing in unfluoridated areas.[7] Water fluoridation provides the greatest improvements in the oral health of Maori and Pacific children and in those from lower socioeconomic groups.[7] Prior to the removal of fluoride from the water in Hamilton, it was estimated that only just over half of the New Zealand population had access to reticulated fluoridated water.[41]

Importance of an early oral examination

Early identification of high-risk children and detection of early lesions provides an opportunity to implement minimally invasive prevention and remineralisation programmes, to attempt to halt and ideally reverse or heal the disease process. The Australasian Academy of Paediatric Dentistry recommends that the first oral examination should occur after the eruption of the first primary tooth and should occur no later than 12 months of age.[43] This visit is to assess the child's growth and development, caries risk, and to provide parents with oral health education and anticipatory guidance.[43] Many families are not

aware of the benefits of this early dental visit and parents often do not access dental services for their preschool-aged children. Primary care providers or medical practitioners are likely to see infants and toddlers more frequently and are in a good position to identify children who may be at high caries risk and to ensure that they are seen by an oral health professional.

Oral health care services in New Zealand

Children in New Zealand are eligible for free dental care until their eighteenth birthday. Children are seen by dental therapists at their local school dental clinic until the end of year eight (approximately age 12–13 years); if the treatment is too complex for the therapist they may be referred to a dental practitioner for management. From year eight until 18 years of age, adolescents are eligible for free basic dental care provided by general dental practitioners who have signed a combined dental agreement with the local district health board (DHB). Specialist care, including general anaesthesia for comprehensive treatment, is available on a referral basis through the various DHBs, with the presence of a specialist paediatric dentist and the treatment provided varying between DHBs. Private specialist paediatric dentists are located in some cities in New Zealand. Parents can contact the school dental service directly on 0800 TALK TEETH (0800 825 583). Dental therapists will examine the children and will appropriately refer those who they consider in need of specialist care.

Early primary care identification needed

Early childhood caries is a complex public health problem. Dental caries in young children leads to pain, infection, adverse general health effects, and increased, preventable hospitalisations. Many young children at high caries risk do not access dental services until their teeth become symptomatic. If primary health care providers are able to identify children at high risk and refer these children early to oral health services, early preventive programmes can be implemented to decrease the burden of oral disease in New Zealand's preschool population.

References

1. American Academy of Pediatric Dentistry (AAPD). Policy on early childhood caries (ECC): classifications, consequences, and preventive strategies. In: Oral health policies reference manual. Chicago: American Academy of Pediatric Dentistry, Council on Clinical Affairs; 2011.
2. Ministry of Health, New Zealand. Age 5 and Year 8 time-series oral health data. Wellington: Ministry of Health; 2011. [cited 20 Feb 2012]. Available from: http://www.health.govt.nz/nz-health-statistics/publications-data-sets-and-stats/oral-health-data-and-stats/age-5-and-year-8-time-series-oral-health-data
3. Craig E, Anderson P, Jackson G, Jackson C. Measuring potentially avoidable and ambulatory care sensitive hospitalisations in New Zealand children using a newly developed tool. N Z Med J. 2012;125(1366):38–50.
4. Ha D. Dental decay among Australian children. Canberra: Australian Institute of Health and Welfare; 2011.
5. Gowda S, Thomson W, Foster PL, Croucher N. Dental caries experience of children in Northland/Te Tai Tokerau. N Z Dent J. 2009;105(4):116–20.
6. Thomson WM. Ethnicity and child dental health status in the Manawatu-Wanganui Area Health Board. N Z Dent J. 1993;89(395):12–4.
7. Lee M, Dennison PJ. Water fluoridation and dental caries in 5- and 12-year-old children from Canterbury and Wellington. N Z Dent J. 2004;100(1):10–5.
8. Wong H, McGrath C, King N, Lo E. Oral health-related quality of life in Hong Kong preschool children. Caries Res. 2011;45(4):370–6.
9. Van den Branden S, Van den Broucke S, Leroy R, Declerck D, Hoppenbrouwers K. Oral health and oral health-related behaviour in preschool children: evidence for a social gradient. Eur J Pediatr. 2013;172:231–7.
10. Skeie M, Raadal M, Strand G, Espelid I. The relationship between caries in the primary dentition at 5 years of age and permanent dentition at 10 years of age: a longitudinal study. Int J Paediatr Dent. 2006;16(3):152–60.
11. Azarpazhooh A, Leake JL. Systematic review of the association between respiratory diseases and oral health. J Periodontol. 2006;77(9):1465–82.
12. Meurman JH, Sanz M, Janket SJ. Oral health, atherosclerosis, and cardiovascular disease. Crit Rev Oral Biol Med. 2004;15(6)403–13.
13. Joshipura K. The relationship between oral conditions and ischemic stroke and peripheral vascular disease. J Am Dent Assoc. 2002;133(suppl 1):23S–30S.
14. Kleinberg I. A mixed-bacteria ecological approach to understanding the role of the oral bacteria in dental caries causation: an alternative to Streptococcus mutans and the specific-plaque hypothesis. Crit Rev Oral Biol Med. 2002;13(2):108–25.
15. Takahashi N, Nyvad B. The role of bacteria in the caries process: ecological perspectives. J Dent Res. 2011;90(3):294–303.
16. Drury TF, Horowitz AM, Ismail AI, Maertens M, Rozier R, Selwitz R. Diagnosing and reporting early childhood caries for research purposes: a report of a workshop sponsored by the National Institute of Dental and Craniofacial Research, the Health Resources and Services Administration, and the Health Care Financing Administration. J Public Health Dent. 2007;59(3):192–7.
17. Harris R, Nicoll AD, Adair PM, Pine CM. Risk factors for dental caries in young children: a systematic review of the literature. Community Dent Health. 2004;21(1):71–85.
18. Hallett K, O'Rourke P. Social and behavioural determinants of early childhood caries. Aust Dent J. 2003;48(1):27–33.
19. Thornley S, McRobbie H, Jackson G. The New Zealand sugar (fructose) fountain: time to turn the tide? N Z Med J. 2010;123(1311):58–64.

20. Ministry of Health. NZ food NZ children: key results of the 2002 National Children's Nutrition Survey. Wellington: Ministry of Health; 2003.
21. Hamilton S, Mhurchu CN, Priest P. Food and nutrient availability in New Zealand: an analysis of supermarket sales data. Public Health Nutr. 2007;10(12):1448–55.
22. Vartanian LR, Schwartz MB, Brownell KD. Effects of soft drink consumption on nutrition and health: a systematic review and meta-analysis. Am J Public Health. 2007;97(4):667–75.
23. Tinanoff N, Palmer CA. Dietary determinants of dental caries and dietary recommendations for preschool children. J Public Health Dent. 2000;60(3):197–206.
24. Peres RC, Coppi LC, Volpato MC, Groppo FC, Cury JA, Rosalen PL. Cariogenic potential of cows', human and infant formula milks and effect of fluoride supplementation. Br J Nutr. 2009;101(3):376–82.
25. Reynolds EC. Anticariogenic complexes of amorphous calcium phosphate stabilized by casein phosphopeptides: a review. Spec Care Dentist. 1998;18(1):8–16.
26. Dashper SG, Saion BN, Stacey MA, Manton DJ, Cochrane NJ, Stanton DP, et al. Acidogenic potential of soy and bovine milk beverages. J Dent. 2012;40(9):736–41.
27. Salanitri S, Seow WK. Developmental enamel defects in the primary dentition: aetiology and clinical management. Aust Dent J. 2013;58:133–40.
28. Elfrink M, Ten Cate J, Jaddoe V, Hofman A, Moll H, Veerkamp J. Deciduous molar hypomineralization and molar incisor hypomineralization. J Dent Res. 2012;91(6):551–5.
29. Mahoney E, Morrison D. Further examination of the prevalence of MIH in the Wellington region. N Z Dent J. 2011;107(3):79.
30. Elfrink ME, Schuller AA, Veerkamp JS, Poorterman J, Moll H, Ten Cate J, et al. Factors increasing the caries risk of second primary molars in 5-year-old Dutch children. Int J Paediatr Dent. 2010;20(2):151–7.
31. Habibian M, Roberts G, Lawson M, Stevenson R, Harris S. Dietary habits and dental health over the first 18 months of life. Community Dent Oral Epidemiol. 2001;29(4):239–46.
32. Widmer RP. Oral health of children with respiratory diseases. Paed Resp Rev. 2010;11(4):226–32.
33. Sheiham A. Dental caries affects body weight, growth and quality of life in pre-school children. Br Dent J. 2006;201(10):625–6.
34. Unkel J, Fenton SJ. Comparison of odontogenic and non-odontogenic facial cellulitis in a pediatric hospital population. Pediatr Dent. 1997;19(8):476–479.
35. Pine CM, Harris RV, Burnside G, Merrett MC. An investigation of the relationship between untreated decayed teeth and dental sepsis in 5-year-old children. Br Dent J. 2006;200(1):45–7.
36. Anderson H, Drummond B, Thomson WM. Changes in aspects of children's oral health related quality of life following dental treatment under general anaesthesia. Int J Paediatr Dent. 2004;14(5):317–25.
37. Malden P, Thomson W, Jokovic A, Locker D. Changes in parent-assessed oral health-related quality of life among young children following dental treatment under general anaesthetic. Community Dent Oral Epidemiol. 2008;36(2):108–17.
38. Gaynor W, Thomson WM. Changes in young children's OHRQoL after dental treatment under general anaesthesia. Int J Paediatr Dent. 2012;22(4):258–64.
39. Featherstone JD. The science and practice of caries prevention. J Am Dent Assoc. 2000;131(7):887–900.
40. Slade GD, Sanders AE, Do L, Roberts-Thomson K, Spencer AJ. Effects of fluoridated drinking water on dental caries in Australian adults. J Dent Res. 2013;92(4).376–82.
41. Walsh T, Worthington HV, Glenny AM, Appelbe P, Marinho VC, Shi X. Fluoride toothpastes of different concentrations for preventing dental caries in children and adolescents. Cochrane Database Syst Rev. 2010 (1):CD007868.
42 New Zealand Guidelines Group. Guidelines for the use of fluoride. Wellington: Ministry of Health; 2009.
43. Australasian Academy of Paediatric Dentistry (Inc). Standards of Care. Perth, Western Australia: Australasian Academy of Paediatric Dentistry (Inc.); 2002.

COMPETING INTERESTS
None declared.

LETTERS TO THE EDITOR

Influencing medical students' career decisions

As a family of three generations of medical professionals, including two general practitioners (GPs) and a current fifth-year medical undergraduate, all of whom have spent part of their careers within the UK and New Zealand (NZ) health care systems, we read with interest the articles discussing the influences on medical students' career decisions.

Our collective personal experiences of undergraduate general practice exposure involved two lectures and a final-year voluntary attachment in the 1950s, to a week of public health and primary care lectures and a fifth-year, one-week stay with a GP in the 1980s, to the current-day regular general practice attachments from first year onwards.

Over our collective 57 postgraduate years, we have seen the role of the GP change greatly. It has gone from mainly one of a sole practitioner working from home, with 24/7 individual contractual responsibility with limited access, to open-access appointments and health advice through patient portals and e-health. Many salaried doctors now work in larger partnerships, using formalised referral pathways and stricter surveillance of prescribing, in purpose-built group health centres. There has been the development of undergraduate and postgraduate general practice attachments and the establishment of professo-