Caries risk prediction — the way of the future

PETER E. CLEATON-JONES

Dental caries almost never kills anyone but it certainly causes pain and treatment is expensive. Loesche has pointed out that in the USA, the annual cost of treating dental caries was about 10 billion dollars in 1977, an amount that had increased to 22 billion dollars by 1984 and 29 billion dollars by 1988. Incredibly, this cost was only for the approximately 50% of people who consulted a dentist. In England, Miller et al. estimated that 5 million days were disturbed by toothache and 1 million nights of sleep were lost, associated with the loss of some 12 million working days each year.

Prevention of dental caries was promoted by Miller, the father of modern cariology. In 1890 he linked oral bacteria to the disease and demonstrated that organic acids fermented from foods by oral bacteria initiated the disease. This concept is still accepted today. Miller explained that prevention would require oral hygiene, limiting the consumption of fermentable substances and the intelligent use of antiseptics to control or destroy oral bacteria.

Earlier this century Mellanby postulated that it was the quality of tooth substance that made one less or more susceptible to caries. This concept fitted well with her research into vitamin deficiency, rickets and quality of diet. She postulated a classification of enamel hypoplasia with varying risks for the development of caries. Mellanby’s belief was on the right track. During the late 1930s and early 1940s, a United States Public Dental Service Officer, Trendley Dean, and colleagues’ used epidemiology to confirm that as the water concentration of fluoride increased so dental caries decreased. Fluoride in the drinking water had been known to be associated with stained and pitted teeth since the work of Black and McMay, among others. The description of an inverse relationship between caries and fluoride was a significant advance in dental science. Multifocal discoveries are common in science and around the same time, Ockerse, a school dentist in South Africa, showed a low prevalence of caries in the north-western Cape, where the fluoride concentration in the water was high. Dean et al. suggested restoring fluoride to drinking water at a level of 1 ppm F. to reduce caries. At this level the caries rate is low and a protection of about 10% of individuals. In 1947 water fluoridation studies were begun in several American and Canadian cities; these continue to this day and have confirmed benefit to the community at low cost. In South Africa, a Commission of Inquiry recommended in 1967 that water fluoridation be introduced in this country.

Sadly, this has not yet happened.

As well as water fluoridation, other preventive measures have been adopted. Fluoride has been placed in toothpastes, rinses, gels and tablets. Methods to improve oral hygiene have been introduced as has the sealing, with stained teeth, of fissures of posterior teeth, where most caries usually begins.

Microbiological research has progressed since Miller’s time and understanding of caries etiology has been augmented by evidence of bacterial aetiological factor. These organisms produce dextranase which help them to cling to teeth. Lactic acid and other cariogenic products are produced and enamel dissolution occurs.

Diet has been implicated in caries development since the time of Hippocrates who associated the eating of figs with the development of disease. In 1947 water fluoridation studies were begun in several American and Canadian cities; these continue to this day and have confirmed benefit to the community at low cost. In South Africa, a Commission of Inquiry recommended in 1967 that water fluoridation be introduced in this country.

Sadly, this has not yet happened.

How should caries risk be predicted and in whom? One must consider the natural history of the disease. Caries may only appear once teeth have erupted, so in the primary dentition the prevalence increases from zero, prior to tooth eruption, until about the age of 5 years when primary teeth are exfoliated and the prevalence drops. Then, erupted permanent teeth may develop caries, the prevalence of which increases up to the age of 5 years, when primary teeth are exfoliated and the prevalence drops. Then, erupted permanent teeth may develop caries, the prevalence of which increases until late adolescence or early adulthood when, for ill-understood reasons, the disease rate slows. From this pattern it is clear that the child must be the initial target, although there is debate about whether the primary or permanent dentition or both should be concentrated on.

At present most researchers concentrate on the permanent dentition for the obvious reason that there is no succeeding dentition, and because of the relative availability of children at schools. Choice of predictor is difficult, a fact emphasised in a recent, extensive review of caries predictors suitable for mass screening in children. Swedish researchers have been in the forefront of caries risk assessment, but the most comprehensive review of the problem by Stanm et al. has come from the USA. They discuss advantages and disadvantages of various risk assessment models. Initial research on longitudinal caries data from a no-treatment group of children in the United States National Preventive Dentistry Demonstration Programme used correlation and multiple regression techniques. Factors with a significant influence on caries development were socio-economic status, caries scores and urgency of a child’s need for immediate dental care. More recent work has identified the following risk predictors: prediction of caries increment by an examining dentist, morphology of permanent molar teeth, lactobacilli in saliva, educational level of head of DENTAL RESEARCH INSTITUTE OF THE MEDICAL RESEARCH COUNCIL AND UNIVERSITY OF THE WITWATERSRAND, Jhb.

PETER E. CLEATON-JONES, B.D.S., M.B. B.CH., PH.D., D.SC.

DENT., D.P.H., D.T.M. & H, D.A.

SUPPLEMENT TO SAMJ, JULY 1994

47
the household, decay in primary posterior teeth, decay in permanent teeth, race (white) and gender (male). Several other factors had weak effects. Other work by the same group, emphasized how variables may have a strong association with caries in univariate analysis but may lose this association in multivariate analyses. Comprehensive details of individual risk factors have been noted.

In South Africa, epidemiological studies have been undertaken in different communities since 1910, and classic studies are those of Oranje et al. in 1935 and Statz in 1935. These early researchers showed different rates of caries in different races. Walker has applied his epidemiological skills to dental caries because of his interest in the role of diet in dental caries aetiology. A paper of his some 20 years ago showed levels of caries in sucrose consumption groups at variance with the postulated simple direct causative link between caries and sucrose intake. The teeth of urban black children were excellent in spite of a relatively high total sugar intake.

An examination of possible risk factors in South African children was begun several years ago. Among young adolescents (mean age 16.3 years) in Johannesburg, no statistically significant differences in caries rates were found between white English- or Afrikaans-speakers. However, caries scores were significantly higher in lower socio-economic areas of Johannesburg. In younger children conflicting observations have been made. No significant effects of social class on caries rates were seen when within-racial-group comparisons were made for 5 groups of 11-year-old children. More recently, in a study of 12-year-old Indian children from Lenasia and white children from Johannesburg, no significant effects on caries in the permanent dentition were seen among the Indian community with regard to the following factors: social class, parental education level, family income, room-to-person ratio (to measure home crowding) or parental occupation. In contrast, in the white community, social class, parental occupation, family income and room-to-person ratio were significantly associated with dental caries. In the same study multiple regression analysis showed significant effects for race (Indians had more caries than whites) and sex (females had more caries than males).

Younger white children (4-5 years old) manifested significant influences on caries of social class and parental education. Those in lower social classes, whose parents were without tertiary education, had more caries. Three other risk factors have been studied. Salivary mutans streptococci > 10⁵ CFU/ml was significantly associated with dental caries in 3-5-year-olds. Also, in an Indian community primary dentition caries may be a reasonable indicator of future caries in the permanent dentition. The third factor is fluoride and hygiene. We have modified a version of the Community Periodontal Index of Treatment Needs (CPTTN) was found to have reasonable specificity, sensitivity and predictor values for primary dentition caries.

There is still much work to be done. In Africa, 'risk' is a useable concept. Many et al. have said that there is little evidence that variations in caries can be attributed to the existence of 'high- or low-risk groups or individuals' who have the same 'risk factor' in common. Social and behavioural factors have been shown to have associations with dental caries but there is little evidence of their use in caries prediction. I believe that such social and behavioural factors may prove of practical use as caries risk predictors in South African communities, provided that suitable social measures relevant to South African conditions are developed. We are attempting to do just that in respect of dental caries.

REFERENCES