Caries risk prediction — the way of the future

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Dental caries almost never kills anyone but it certainly causes pain and treatment is expensive. Loesche[1] 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.[2] Incredibly, this cost was only for the approximately 50% of people who consulted a dentist. In England, Miller *et al.*[3] 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.[4]

Prevention of dental caries was promoted by Miller,[5] 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.[2] 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[6,7] 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[8] 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[9] and McMay,[10] 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,[11] 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.*[8] 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 fluorotic staining of teeth is limited to about 10% of individuals. In 1947 water fluoridation studies were begun in several American and Canadian cities;[12] these continue to this day and have confirmed benefit to the community at low cost. In South Africa, a Commission of Inquiry[13] 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.[14] Fluoride has been placed in toothpastes, rinses, gels and tablets. Methods to improve oral hygiene have been introduced as has the sealing, with adhesives, of the fissures of posterior teeth, where most caries usually begins.

Microbiological research has progressed since Miller's time and mutans streptococci have been designated the prime bacterial aetiological factor.[2] These organisms produce dextrans which help them to cling to teeth and ferment carbohydrate there, just as Miller[5] had suggested. An enzyme, dextranase, which broke down dextrans,[15,16] enjoyed a brief vogue, but has not been widely used because administration is inefficient given the short contact between enzyme and substrate.

Diet has been implicated in caries development since the time of Hippocrates who associated the eating of figs with the disease.[17] White sugar was labelled as 'pure white and deadly'[18] or as the 'arch criminal' of dental caries[19] and pressure to reduce sugar intake increased. Walker and Cleaton-Jones[20-22] have urged a realistic evaluation of the evidence concerning diet and caries interaction. Certainly sugar intake is one of the factors in caries development, but the expression of its effect varies from individual to individual and is influenced by individual foods eaten. To clarify this concept scientists in Switzerland developed sophisticated ways of measuring pH in tooth *in vivo* using telemetry; many foods have been studied in this way. Some did not lower pH below the critical level of 5,7 needed to decalcify teeth and initiate caries. Such foods are labelled '*zahnschonend*' — friendly to teeth — indicated by a smiling tooth with an umbrella over it on the packaging.[23] Consumption of these foods is recommended to reduce caries risk.

By 1973 knowledge on dental caries had increased to the point where Carlos[24] of the National Institute for Dental Research in the USA, confidently predicted the elimination of caries as a public health problem. But it was not to be. Certainly the incidence of caries diminished in those who heeded the preventive message, and where water fluoridation was introduced. The number of children free of caries has increased in developed countries to the extent that some dental schools have closed. In developing countries, the number of children with dental caries has increased. Also, as teeth have been retained for longer, dental caries of the tooth root in older individuals has appeared as an additional problem.[25] Rather than aim at its elimination, we should focus on control of the disease.[26]

Gradually, realism replaced earlier euphoria. It has become clear that 60 - 80% of caries occurs in about 20% of the population,[2,27] many of whom may not regularly attend for dental services. Secondly, there is concern over the worldwide increase in the cost of health care. To cope with these, there has been a move towards appropriateness of care[2] in medicine and dentistry. A cornerstone of this philosophy is the identification of risk attached to a disease so that scarce resources may be concentrated on those who need them most. In some countries such as South Africa there has been a call to increase the numbers of dental and paradental graduates to service previously neglected communities.[28]

How should caries risk be predicted and in whom? One must first consider the natural history of the disease.[29] 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 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.[30] Swedish researchers have been in the forefront of caries risk assessment,[31,32] but the most comprehensive review of the problem by Stamm *et al.*[2] 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.[33] 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

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the household, decay in primary posterior teeth, decay in permanent teeth, race (white) and gender (male).[2] Several other factors had weak effects. Other work by the same group,[34] emphasised 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.[35]

In South Africa, epidemiological studies have been undertaken in different communities since 1910,[36] and classic studies are those of Oranje *et al.* in 1935[37] and Staz in 1935.[38] 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[39] 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.[40] 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.[41] 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.[42] 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.[43] Those in lower social classes, whose parents were without tertiary education, had more caries. Three other risk factors have been studied. Salivary mutans streptococci > 10^6 CFU/ml was significantly associated with dental caries in 3 - 5-year-olds.[44] Also, in an Indian community primary dentition caries may be a reasonable indicator of future caries in the permanent dentition.[45] The third factor was oral hygiene and here a modified version of the Community Periodontal Index of Treatment Needs (CPITN) was found to have reasonable specificity, sensitivity and predictor values for primary dentition caries.[46]

There is still much work to be done. In Africa, 'risk' is unclear. Manji *et al.*[47] 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.[48] 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.[49] We are attempting to do just that in respect of dental caries.

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