CME

Dental caries, sugars, plaque and fluoride

Peter E Cleaton-Jones

BDS, MB BCh, PhD, DSc (Dent), DTM&H, DPH, DA (SA)

Professor and Director

Dental Research Institute

Medical Research Council and

University of the Witwatersrand

Johannesburg

Peter Cleaton-Jones has a broad background in dentistry, medicine and science. For 26 years he has been a researcher who has published widely, in the fields of cariology, pulp biology and anaesthesia, for which he has received national and international recognition. A particular interest of his is the training of postgraduate students. He has successfully supervised some 60 doctoral and magistral candidates and has trained approximately 800 researchers in research methodology.

Dental caries is the world's most prevalent disease, affecting up to 80% of the population.

Dental caries

Dental caries — in laypersons' terms, tooth decay — is an ugly, but generally non-fatal condition (Fig. 1). It has been reported in the skulls of Australopithecine hominids who lived in South Africa 500 000 years ago, as well as in ancient Neanderthal skulls.[1] Today, it remains the commonest human disease. The reason for this is simple. In contrast to most diseases in which healing may occur, once dental caries has appeared tooth damage remains forever so even a low incidence may be associated with a high prevalence.

What is this disease that affects so many of us and certainly at least 80% of the readers of this article? In essence it is a multifactorial condition that results in dissolution of the hard tissues of the teeth. A diagram (Fig. 2) that summarised the aetiology of dental caries was suggested by Keyes[2] and consisted of three intersecting circles, each representing a major causative factor — teeth, bacteria and foods. It was postulated that when all three factors were present under the right conditions, dental caries was the result. This concept, although convenient, is too simplistic. It is possible for all three

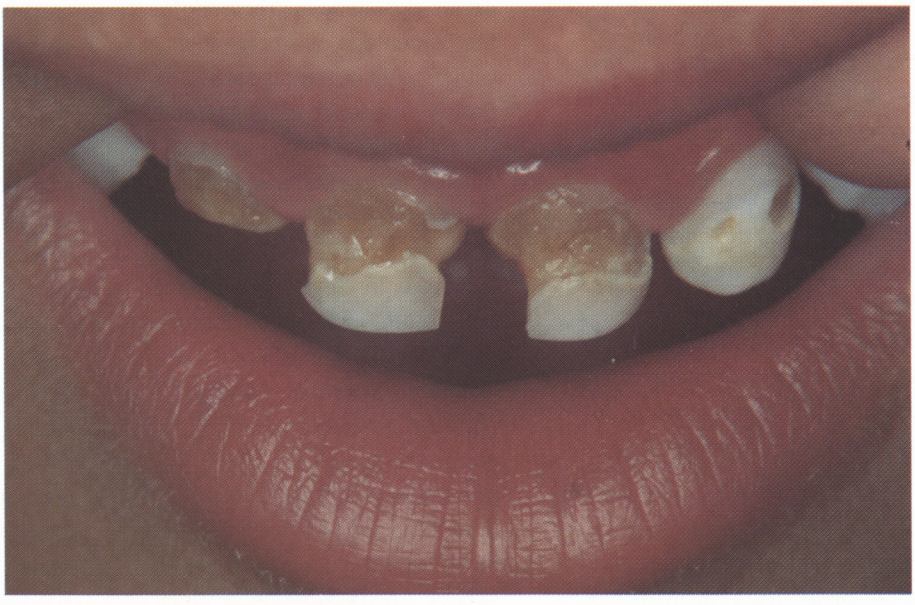

Fig. 1. Dental caries in the primary dentition of a 5-year-old.

factors to be present yet caries not to occur. Time is clearly a factor. This introduces another intersecting circle and I believe host resistance is paramount. My concept is shown in Fig. 3. Variation in host resistance would explain why some 60 - 80% of caries in children is seen in only 20% of the population.[3]

The chemical process of caries development consists of metabolism of foods by bacteria in plaque to produce organic acids such as acetic, formic and lactic. These acids decalcify inorganic tooth substance to expose the organic component, after which proteolytic enzymes continue the tissue destruction. Enamel, the outer layer of a tooth, consists of approximately 95% inorganic salts, 1% organic material and 4% water (by weight) while the underlying dentine is about 70% inorganic, 20% organic and 10% water (by weight). Early decalcification may be completely reversed but not the proteolytic digestion. This elegant decalcification-proteolysis theory (initially termed the chemico-parasitic theory) was proposed in 1890 by Miller,[4] an American dentist and researcher working in Berlin, Germany. Changes to details of his theory have been made but his fundamental observations remain.

Diagnosis of dental caries is based on the changes brought about by the decalcification and proteolysis, e.g. change in colour

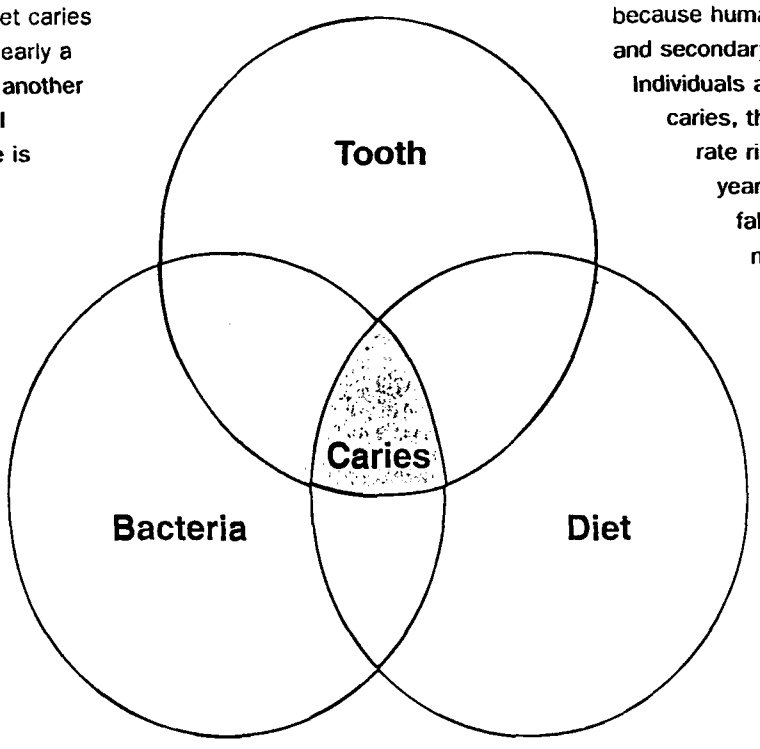

Fig. 2. Triad of causative factors for dental caries.

(white in enamel, darker yellow in dentine), dark areas on transillumination, softening on probing or radiolucent areas on radiographs. Measurement of caries is broadly by two methods. Caries prevalence is recorded as the percentage of a population with or without the disease. Caries experience is measured by the total number of decayed, missing (extracted) and filled teeth (dmft/DMFT) or tooth surfaces (dmfs/DMFS) in an individual. In the primary dentition dmft (teeth, maximum = 20) or dmfs (surfaces, maximum = 88) scores are recorded. For the permanent dentition the equivalent scores are DMFT (maximum = 32) and DMFS (maximum = 148); often the third molars are excluded from the scores. Caries experience has a bimodal distribution (Fig. 4) because humans have primary and secondary dentitions. Individuals are born free of caries, thereafter the caries rate rises until about 5 years of age when it falls due to the natural shedding of healthy and diseased primary teeth. As the permanent teeth erupt into the mouth the rate of dental caries increases once more, the greatest rate being until the age of about 15 years. After this the rate of caries development becomes less steep for reasons not yet clarified.

Principles of treatment of established dental caries are much as they were at the turn of the century — remove the diseased tissue and replace with a tooth substitute. What has changed considerably are the details of the treatment. Removal of diseased tissue with high-speed instruments, local anaesthesia, inhalation of nitrous oxide/oxygen mixtures, hypnosis and so on has substantially reduced the pain of treatment. Less tooth tissue is removed today and filling materials are easier to use, stronger and more aesthetic. Emphasis is on retention of teeth, so extractions are far less common and preventive programmes (discussed elsewhere in this issue) may reduce both caries prevalence and experience. The extent of treatment in a

community may be easily measured through expressing the extraction (m or M) and filling (f or F) components of the dmf and DMF scores as a percentage of the total score.

In spite of technological advances in treatment and the desire of the dental profession to treat all established caries, individuals affected must have the motivation to seek or to accept treatment and the means to pay, whether payment be by the individual, the state or another body. Treatment percentages remain low in many communities. For example, my colleagues and I have found well over 60% of carious teeth in 5-year-olds in Germiston in 1994 to have had no treatment at all.

Sugars

For dental caries to occur foods must be metabolised by plaque bacteria. Hippocrates associated the development of dental caries with the eating of figs[5] while many others implicated sugars. During World War II a group of Swedish dental researchers carried out the Vipeholm Study. In this, some 400 adult inmates in a mental institution were fed sugar (sucrose) in various quantities, frequencies and forms over a 5-year period.[6] From the study it was concluded that sucrose eaten with meals had little effect on caries rate, there was a moderate increase in caries in a group eating chocolates 4 times a day between meals, while groups eating snacks of caramels (22 per day) or toffees (8 or 24 per day) between meals had high increases in caries. Strangely, some individuals did not develop caries at all even when eating 24 toffees per day. This study was well designed and conducted and was valuable at the time it was done. By modern standards the methodology is unethical and had major flaws such as eating of toffees of 50 mm diameter (to avoid early swallowing) by individuals with poor oral hygiene and older than the high-risk ages. Based on the Vipeholm Study and several smaller investigations in children's homes, sucrose was accorded major blame in caries development. On this, as well as the decrease in caries rate seen during World War II, is based the concept that a decrease in sugar (sucrose) intake would decrease caries. Since sucrose is usually the main dietary sugar eaten, this has logically been accorded most blame. It is not a specific property of the sucrose, however, since it is now well known that all sugars and, to a lesser extent sugar alcohols and starches, may be metabolised into acids, thereby contributing to dental caries.

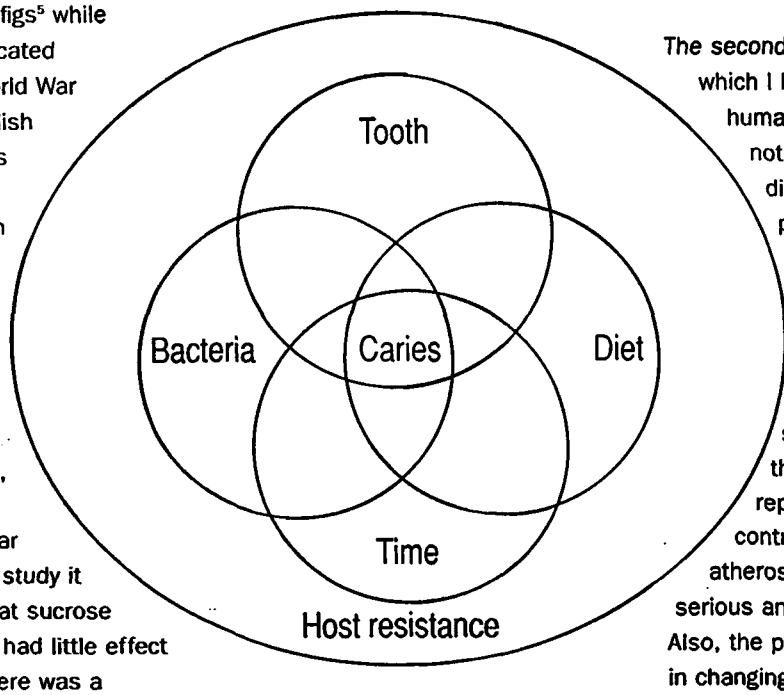

Fig. 3. Modern concept of interacting causative factors.

Currently there are two schools of thought regarding sugar intake. The first, exemplified by the COMA report,[7] desires that refined sugar intake be reduced to not more than 60 g per day or 10% of energy intake. The hypothesis for this is that there will be decreases in dental caries prevalence. The reductions in sugar intake may be achieved through motivating individuals to reduce their own refined sugar intake or through government policies to generally reduce intake.

The second school of thought, to which I belong, says that human epidemiology has not shown sufficient differences in caries prevalences between low and high sugar consumers to justify such reductions. A further consideration is that the refined sugars removed from the diet are likely to be replaced by fats, thereby contributing to atherosclerosis, a more serious and often fatal disease. Also, the priorities of an individual in changing a sugar intake pattern and the government reduction policy are low, particularly among

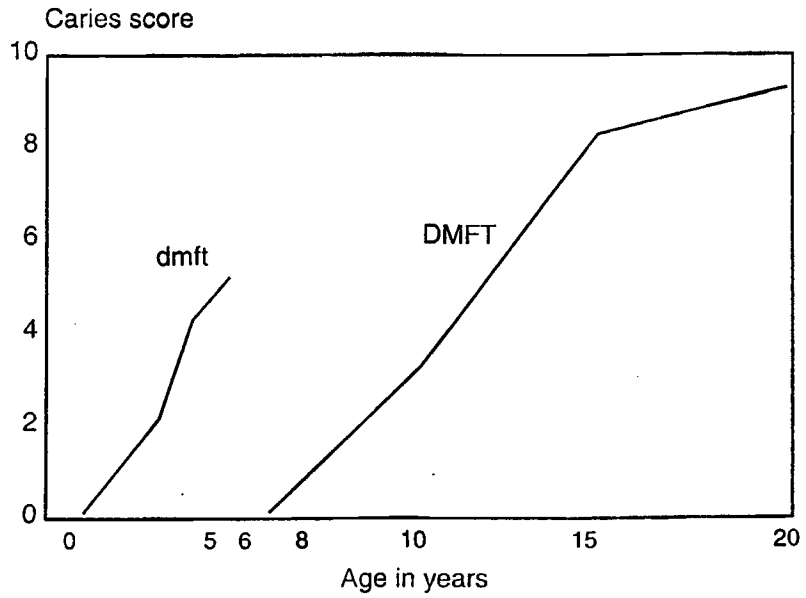

Fig. 4. Bimodal distribution of caries experience (dmft and DMFT scores).

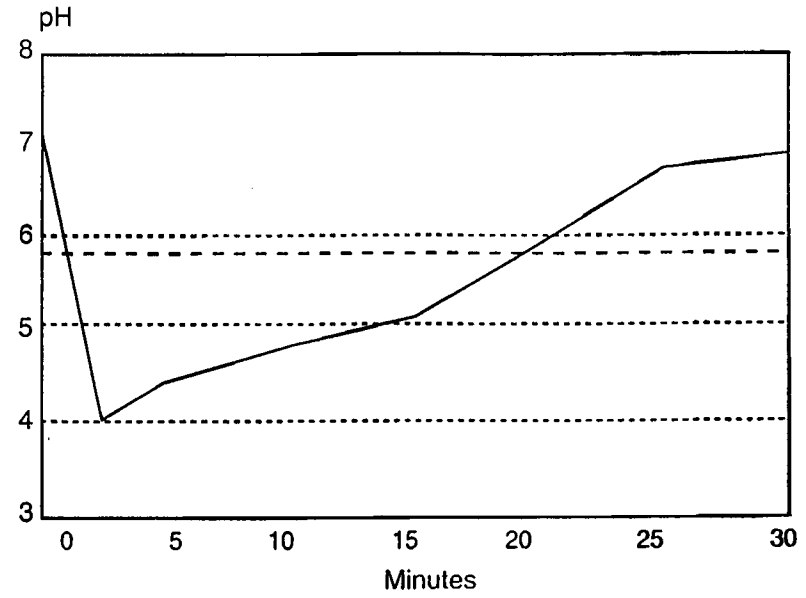

Fig. 5. Stephan curve to illustrate pH changes in plaque after a sucrose rinse. The critical level for decalcification (pH 5,7) is the horizontal line.

poorer communities who use sugar intake as a cheap source of energy. This was recently emphasised by Burt and Szpunar,[8] who believe that government sugar reduction policies are expensive and ineffective. They prefer that dietary advice for dental health should be linked to policies which educate the public on sensible food choices for healthy living — a choice of unrefined carbohydrate, moderate protein and low fat.

Plaque

Dental plaque is the mixture of bacteria, saliva and food debris that collects on teeth and gums. It is a distinct micro-ecosystem, the composition of which varies with time. A newly cleaned tooth surface is covered by a granular deposit known as pellicle which gradually increases in thickness. In the first 8 hours small groups of micro-organisms (mainly cocci) settle in natural depressions on the tooth surface. Between 8 and 12 hours later there is a rapid increase in the number of micro-organisms which spread across the surface as a monolayer. After 24 hours the whole surface is covered and multilayers begin to appear. During the second day the micro-organism accumulation is invaded by filamentous organisms which are orientated perpendicularly to the surface. The numbers and thickness of the organisms increase so that within 3 weeks there is a dense layer of cocci covered by a looser outer filamentous layer. In the first 24 hours streptococci (*Streptococcus mitior, S. sanguis* and *S. mutans*) and actinomycetes are the main organisms. Adhesion of the organisms to the tooth is by a complex but effective mechanism which is best broken down by mechanical cleaning. The mechanism of plaque build-up described above indicates that teeth must be cleaned at least every 24 hours.

Within plaque, acid formation due to metabolism of dietary substrate, particularly sugars, is rapid. After a substrate enters plaque the pH in plaque will drop within 2 - 3 minutes from a resting value of above 7,0 to a level determined by the substrate, number of organisms, salivary flow etc. This is followed by a recovery phase back to resting level as buffer mechanisms in plaque and saliva act. The typical

response is known as a Stephan curve (Fig. 5). It is accepted that a pH of less than 5,7 is needed to decalcify tooth substance. The longer the pH is below this level, the greater is the decalcification. Also, the more frequently the pH drops the greater will be the decalcification (Fig. 6). Incidentally, pH drop in plaque may also occur when acid foods such as fruit juices enter plaque.

Plaque accumulates all over the tooth but may be conveniently described to be predominantly in pits and fissures, in interstitial areas (between the teeth) and on the smooth surfaces of teeth. Mechanical removal of plaque is an aid to dental caries reduction. This may be done professionally, for example as in Sweden by dentists or oral hygienists, with marked reduction and even elimination of caries in communities of children. Individuals may remove plaque with effective toothbrushing (smooth surfaces, pits and fissures) and flossing (interstitial surfaces). This must be done conscientiously for several minutes, at least once a day — preferably before retiring to bed. Smooth surfaces are more easily cleaned than interstitial surfaces and pits and fissures; chemical sealants can be placed over these pits and fissures to reduce plaque accumulation and to aid cleaning.

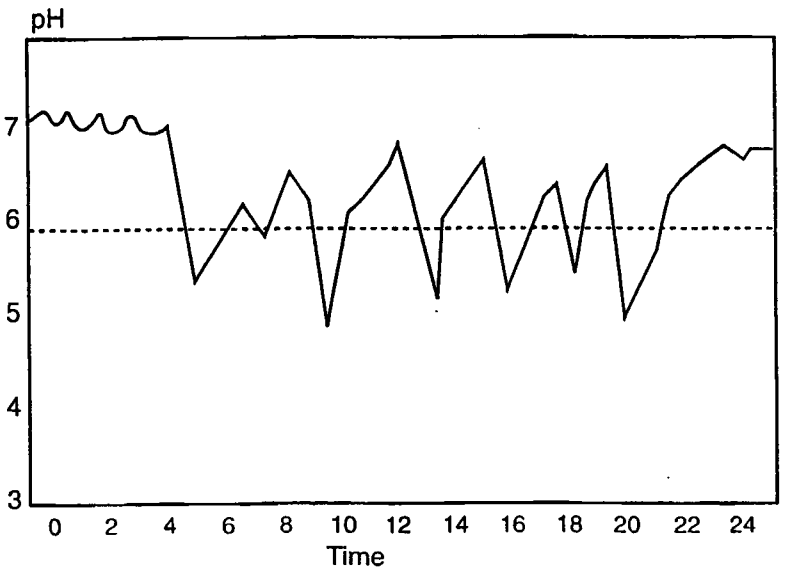

Fig. 6. Repeated pH decreases below pH 5,7 during a 24-hour period.

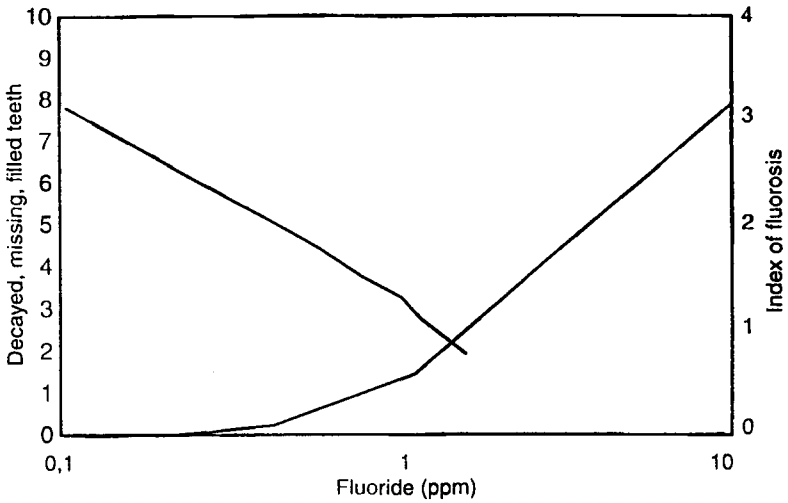

Fig. 7. Relationship between dental caries, fluorosis and fluoride concentration in the drinking water.[9]

Fluoride

In the field of caries prevention nothing has been as comprehensively researched as fluoride. At the end of the 19th century and in the first years of the 20th century, German, Danish and French dentists were advising the ingestion of fluoride tablets and pastes to combat caries. Thereafter, apart from an interest in the detrimental effects on teeth (termed dental fluorosis), the use of fluoride as a caries-inhibitory agent was dormant until the late 1930s.[9] Rather, there was concentration on the quality of tooth enamel. Mellanby postulated that it was the quality of tooth substance that made one more or less susceptible to the disease.[9] Enamel hypoplasia brought about by poor diet played an important role, according to Mellanby.

Some years later Dean was able to prove, from human epidemiological studies, that the fluoride content of drinking water strongly influenced caries prevalence and experience.[9] These were high in areas with

Table I. Fluoride administration methods[9]

Systemic	Topical
Public water fluoridation	Professional topical application
School water fluoridation	Fluoride mouthrinse
Fluoridised salt	Fluoride toothpaste
Fluoridised milk	
Fluoridised drops or tablets	

Table II. Ranking of fluoride therapies by cost-effectiveness

Rank	Procedure	Likely % caries reduction
1	Public water fluoridation	50
1	Weekly mouthrinse (0,2% NaF)	25
2	Professional application (0,2% NaF) (multiple chairs)	40
3	Fluoride tablet distribution	35
3	School water fluoridation	40
3	Professional application (1,23% F APF gel in tray) (annual)	40
4	Toothbrushing in school (5 x year, 0,6% F solution)	20
5	Toothbrushing at home (0,1% F toothpaste)	20
6	Daily self-application by tray (0,5% F APF gel)	80

APF = acidulated phosphate fluoride.[9]

very low fluoride in the drinking water and diminished as fluoride increased. However the higher the fluoride the more dental fluorosis was seen. Dean demonstrated that in temperate climates a fluoride concentration of 1 ppm reduced dental caries by about 50% and only 10% of teeth had mild mottling (Fig. 7). This optimum level in drinking water around the world has saved millions of teeth.

The enormous amount of research into the use of fluoride in dentistry has confirmed that, provided sufficient is available for incorporation into dental enamel, caries will be reduced by about 50%. Table I lists the various methods of fluoride administration. These may be systemic or topical, they include professional applications (e.g. gels), therapy by individuals or

community-administered schemes. Each has its own cost-effectiveness ratio. A ranking in descending order of cost-effectiveness[9] is shown in Table II.

As fluoride ingestion from all sources has increased so has fluorosis, the only detrimental effect of therapeutic doses, which has required a reduction in earlier recommended systemic doses to approximately half the original level, at least up to the age of 8 years when most of the permanent tooth crowns have been formed. A pamphlet that lists doses of fluoride tablets according to the local water content in South Africa is available at pharmacies.

The mechanism of action of fluoride in decreasing caries is still not generally agreed upon. The three main hypotheses[9] are that:

- fluoride affects the morphology of teeth during tooth formation to make them more self-cleansing
- fluoride makes the apatite in teeth less soluble
- fluoride favours remineralisation.

A final point is that the World Health Organisation resolved in 1969 and endorsed in 1975 that the fluoridation of communal water supplies, where feasible, should be the cornerstone of any national programme of dental caries prevention.[9]

Future developments

Dental caries is an expensive disease to treat and efforts should rather be directed to prevention. It is also clear that the rate of development of the disease varies between individuals. Regular attendance at the dentist or dental therapist needs to be redefined in terms of risk assessment.[3,10] Individuals at high risk of the disease need to be seen more frequently to ensure adequate prevention, perhaps even once a month, those at low risk perhaps only yearly. Concentration of resources on those who need them most will produce the greatest cost-benefit.

The difficulty with this approach, as those of us working in the field have found, is to identify risk factors which vary from community to community. In South Africa, for example, race is associated with variation in caries rates. Our research has shown that the lowest caries rates are seen in rural black children and urban white children. The highest rates are seen in urban 'coloured'

and Indian communities — up to 4 times higher, while urban black children's caries scores lie between the two groups. Social class and education are also important. In high social classes caries rates are lower, especially if education is high too. In high socio-economic groups who have lower education the opposite pattern may be seen. A practical problem therefore is to identify risk factors applicable to us in South Africa.

Recommendations to patients

What should practitioners currently recommend to their patients — recommendations that are moral as well as practical and based on scientific evidence?

- Practice good oral hygiene at least once per day by using a fluoride containing toothpaste, preferably at night before retiring to bed.
- Use fluoride in an appropriate form and press for the introduction of water fluoridation or other community fluoride schemes.
- Limit the intake of refined carbohydrate to 5 exposures per day — 3 meals and 2 snacks.
- See the local dentist, dental therapist or oral hygienist regularly, at a frequency appropriate to the level of risk.

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IN A NUTSHELL

Dental caries is common. It increases from birth to 5 years in the primary dentition, then increases in the permanent dentition, the rate being fastest between 6 and 15 years.

In early dental caries, the stage of demineralisation is reversible but once the disease is established professional care is needed.

Dental plaque, consisting of bacteria, saliva and food debris, collects on the teeth and gums.

All refined carbohydrates may be metabolised to acids by plaque bacteria, these acids producing the demineralisation that precedes proteolysis of dental tissues.

Fluoride is a safe and effective anti-caries agent.

To prevent dental caries teeth should be well cleaned at least once every 24 hours using a fluoride toothpaste.

Community fluoride schemes should be operative.

Refined carbohydrate intake should be reduced and a method of individual risk assessment needs to be developed.

IN 'N NEUTEDOP

Tandkaries is algemeen. Dit neem toe vanaf geboorte tot 5 jaar in die primêre dentisie en neem dan weer toe in die permanente dentisie. Die koers is die hoogste tussen 6 en 15 jaar.

In vroeë tandkaries is die stadium van demineralisering omkeerbaar maar as die siekte eers gevestig is, is professionele sorg nodig.

Tandplaak, wat uit bakterieë, speeksel en voedseloorblyfsels bestaan, versamel op die tande en tandvleis.

Alle verfynde koolhidrate kan deur plaakbakterieë in sure gemetaboliseer word, en hierdie sure produseer die demineralisasie wat proteolise van tandweefsel voorafgaan.

Fluoried is 'n veilige en effektiewe middel teen karies.

Om tandkaries te voorkom moet tande ten minste een maal elke 24 uur goed met 'n fluoriedtandepasta skoongemaak word.

Fluoriedprogramme moet in die gemeenskap in werking wees.

Inname van verfynde koolhidrate moet verminder word en 'n metode van individuele risikobepaling moet ontwikkel word.