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1.1 WHAT IS CARIES?

Dental caries is a process that may take place on any tooth surface in the oral cavity where dental plaque is allowed to develop over a period of time.

Plaque formation is a natural, physiological process which will be described in more detail in the next section. Plaque is an example of a **biofilm**, which means it is not a haphazard collection of bacteria but a community of micro-organisms attached to a surface. This community works together, having a collective physiology. The bacteria in the biofilm are always metabolically active. Some of the bacteria are capable of fermenting a suitable dietary carbohydrate substrate (such as the sugars sucrose and glucose), to produce acid, causing the plaque pH to fall to below 5 within 1–3 minutes. Repeated falls in pH may in time result in **demineralization** of the tooth surface. However, the acid produced is neutralized by saliva, so the pH increases and mineral may be regained. This is called **remineralization**. The cumulative results of the de- and remineralization processes may be a net loss of mineral and a carious lesion that can be seen. Alternatively, the changes may be so slight that a carious lesion never becomes apparent (Figure 1.1).[1]

From this description it becomes obvious that the carious process is an ubiquitous, natural process. The formation of the biofilm and its metabolic activity cannot be prevented, but disease progression can be controlled so that a clinically visible lesion never forms: alternatively, the process can be arrested and even advanced carious lesions may become inactive. However, the other side of the coin is that progression of the lesion into dentine can ultimately result in bacterial invasion and death of the pulp and spread of infection into the periapical tissues, causing pain.

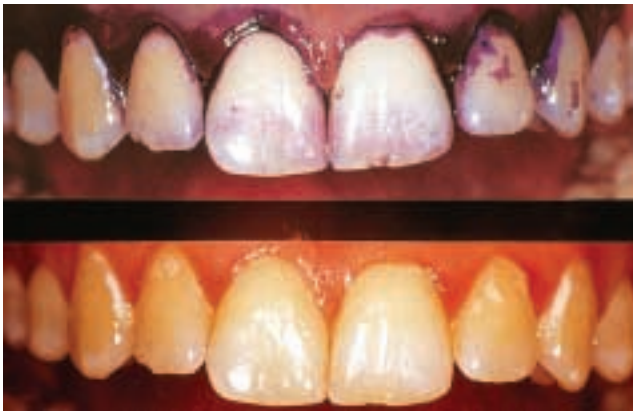


Figure 1.1. The upper anterior teeth of a young adult. In the upper picture, a disclosing agent reveals the plaque, while in the lower picture the plaque has been removed. White spot lesions are visible on the canines, but not on other tooth surfaces, although plaque is present.

1.2 THE CARIOUS PROCESS AND THE CARIOUS LESION

It is probably unfortunate that the word 'caries' is used to denote both the carious process and the carious lesion which forms as a result of that process. The process occurs in the biofilm at the tooth or cavity surface; the interaction of the biofilm with the dental tissues results in the lesion in the tooth. The metabolic activity in the biofilm cannot be seen, but the lesion, which is its reflection or consequence, can be seen. Thus the dentist is working on a reflection, and there is a danger that the dentist might forget that the 'action' is in the biofilm.

Please stand in front of a mirror and look at your reflection. Do you like what you see, or could it be improved by some makeup, a shave, a new haircut, new clothes? You are of course concentrating on the real you and it probably would not occur to you to pick up a brick and smash the mirror! But if you now go into the clinic you will see dentists filling holes in teeth, and in a way they are smashing the mirror unless they have **also** concentrated on teaching the patient to modify the metabolic activity in the biofilm.

1.3 DENTAL PLAQUE[2]

It is thought-provoking that the human body is composed of some 10^{14} cells, but only about 10% of these are mammalian; the remainder are resident microflora. Although a newborn baby's mouth is sterile, it soon acquires microbes, usually from the mother via saliva. More than 300 species of microorganisms have been identified in the mouth.

Dental plaque is an adherent deposit of bacteria and their products, which forms on all tooth surfaces and is the cause of caries. As already mentioned, plaque is a biofilm—a community of microorganisms attached to a surface. The populations of bacteria interact and the properties of the community are more than the sum of the constituent species. The organisms are organized into a three-dimensional structure enclosed in a matrix of extracellular material derived from the cells themselves and their environment.

Dental plaque formation can be described in sequential stages:

- Formation of **pellicle**: an acellular, proteinaceous film, derived from saliva, which forms on a 'naked' tooth surface.
- Within 0–4 hours, single bacterial cells colonize the pellicle. A large proportion of these are streptococci *(S. sanguis, S. oralis, S. mitis)*. There are also *Acintomyces* species and Gram-negative bacteria. Only about 2% of the initial streptococci are mutans streptococci, and this is of interest because these organisms are particularly associated with the initiation of the carious process.
- Over the next 4–24 hours the attached bacteria grow, leading to the formation of distinct **microcolonies**.
- In 1–14 days the **Streptococcus**-dominated plaque changes to a plaque dominated by **Actinomyces**. Thus the population shifts; this is called

microbial succession. The bacterial species become more diverse and the microcolonies continue to grow.
- In 2 weeks the plaque is mature but there are considerable site-to-site variations in its composition. Each site can be considered as unique and these local variations may explain why lesions progress in some sites but not others in the same mouth.

1.3.1 Pathogenic properties of cariogenic bacteria

There are a number of organisms, normally present in plaque, which can cause caries. These cariogenic bacteria can:
- transport sugars and convert them to acid (**acidogenic**)
- produce extracellular and intracellular polysaccharides which contribute to the plaque matrix; intracellular polysaccharides can be used for energy production and converted to acid when sugars are not available
- thrive at low pH (**aciduric**).

1.3.2 Which plaque bacteria cause caries?

There are a number of possibilities, each of which has consequences:
- The **specific plaque hypothesis** proposed that only a few organisms out of the diverse collection in the plaque flora were actively involved in the disease. Preventive measures targeting specific bacteria (e.g. immunization) would be a logical consequence of this hypothesis.
- The **non-specific plaque hypothesis** considered the carious process to be caused by the overall activity of the total plaque microflora. A consequence of this approach is that all plaque should be disturbed by mechanical plaque control (toothbrushing).
- The **ecological plaque hypothesis** proposes that the organisms associated with disease may be present at sound sites. Demineralization will result from a shift in the balance of these resident microflora driven by a change in the local environment. Frequent sugar intake (or decreased sugar clearance if salivary secretion is low) encourages the growth of acidogenic and aciduric species, thus predisposing a site to caries. The consequence of this hypothesis is that both mechanical cleaning and some restriction of sugar intake are important in controlling caries progression.

1.3.3 Where does caries occur?

Bacterial plaque is the essential precusor of caries and for this reason sites on the tooth surface which encourage plaque retention and stagnation are particularly prone to progression of lesions. These sites are:
- enamel in pits and fissures on occlusal surfaces of molars and premolars (Figure 1.2), buccal pits of molars, and palatal pits of maxillary incisors
- approximal enamel smooth surfaces just cervical to the contact point (Figure 1.3)

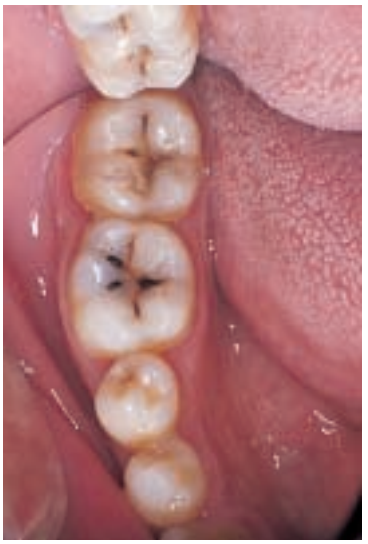


Figure 1.2. Occlusal caries in molars showing stained fissures. Cavities were present.

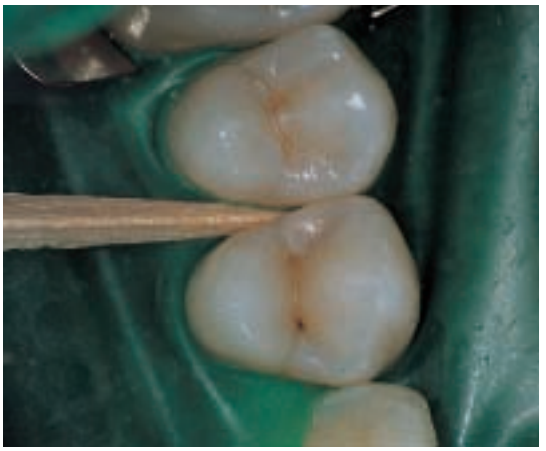


Figure 1.3. A carious lesion is present on the distal aspect of the upper first premolar. The lesion is shining up through the marginal ridge which shows a pinkish-grey discolouration.

- the enamel of the cervical margin of the tooth just coronal to the gingival margin (Figure 1.4a–c)
- in patients where periodontal disease has resulted in gingival recession, the area of plaque stagnation is on the exposed root surface (Figure 1.5)
- the margins of restorations, particularly those that are deficient or overhanging
- tooth surfaces adjacent to dentures (Figure 1.5) and bridges which make cleaning more difficult, thus encouraging plaque stagnation.

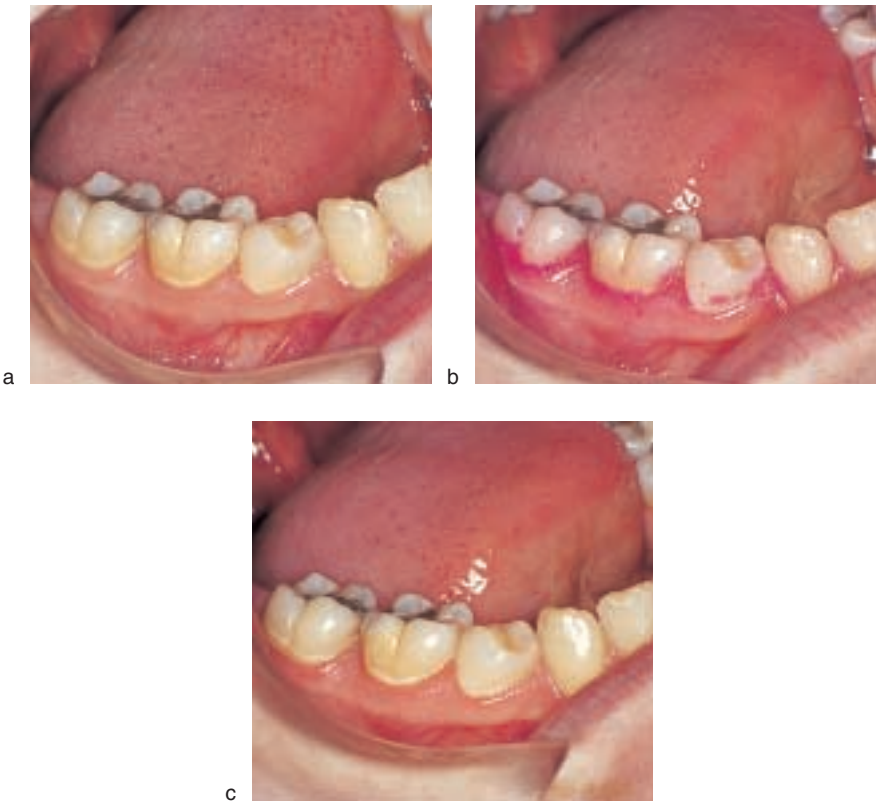


Figure 1.4. Caries of the enamel at the cervical margin of the lower molars: (a) The white spot lesions covered with plaque. (b) A red dye has been used to stain the plaque so that the patient can see the plaque clearly. (c) The patient has now removed the stained plaque with a toothbrush: the white spot lesions are now very obvious. Note they have formed in an area of plaque stagnation and this can been shown to the patient to demonstrate the importance of plaque removal.

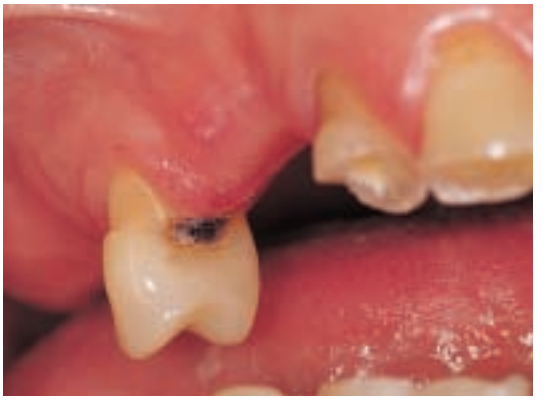


Figure 1.5. Caries on the exposed root surface of the mesial aspect of the upper premolar. Note the lesion is in an area of plaque stagnation adjacent to a removable denture. Dentine is also exposed buccally, but this has been cleaned and abraded by the toothbrush and is caries-free.

1.3.4 Is dental caries an infectious, preventable, disease?

No, perhaps it is neither. Although it is caused by bacteria, these are commensal organisms, not extraneous infecting invaders. The carious process cannot be prevented, because the activity in the biofilm is an ubiquitous, natural process. However, the progression of lesions can be controlled. These statements are contentious and may provoke strong reaction and interesting discussion from your teachers!

1.4 THE ROLE OF DIETARY CARBOHYDRATE

It is necessary for fermentable carbohydrates and plaque to be present on the tooth surface for a minimum length of time for acid to form and cause demineralization of dental enamel. These carbohydrates provide the plaque bacteria with the substrate for acid production and the synthesis of extracelluar polysaccharides. However, carbohydrates are not all equally cariogenic. Complex carbohydrates such as starch are relatively harmless because they are not completely digested in the mouth, but carbohydrates of low molecular weight (sugars) diffuse readily into plaque and are metabolized quickly by the bacteria. Thus, many sugar-containing foods and drinks cause a rapid drop in plaque pH to a level which can cause demineralization of dental enamel. The plaque remains acid for some time, taking 30–60 minutes to return to its normal pH (in the region of 7). The gradual return of pH to baseline values is a result of acids diffusing out of the plaque and buffers in the plaque and salivary film overlying it, exerting a neutralizing effect. Repeated and frequent consumption of sugar will keep plaque pH depressed and cause demineralization of the teeth.

The change in plaque pH may be represented graphically over a period of time following a glucose rinse (Figure 1.6). Such a graph is called a 'Stephan

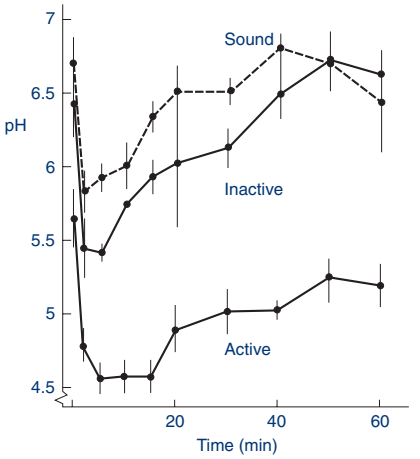


Figure 1.6. Stephan response curves obtained from sound occlusal surfaces, inactive occlusal carious lesions and deep, active occlusal carious cavities following a sucrose rinse in a group of 14-year-olds. Bars indicate standard errors.[3] (Reproduced by kind permission of Professor Fejerskov).

curve' after the person who first described it in 1944. Once a cavity, or hole, forms in the tooth, the plaque within it becomes even more efficient at producing acid. Lower pH values are recorded in plaque within cavities than in plaque on inactive lesions or sound surfaces in the same individuals.[3]

The synthesis of extracellular polysaccharides from sucrose is more rapid than from glucose, fructose, or lactose. Consequently, sucrose is the most cariogenic sugar, although the other sugars are also harmful. Since sucrose is also the sugar most commonly eaten, it is a very important cause of dental caries.

1.5 ENVIRONMENT OF THE TOOTH: SALIVA AND FLUORIDE

Under normal conditions the tooth is continually bathed in saliva. Saliva is supersaturated with calcium and phosphate ions and capable of remineralizing the very early stages of lesion formation, particularly when the fluoride ion is present. Fluoride slows down the progression of lesions.

When salivary flow is diminished or absent, there is increased food retention. Since salivary buffering capacity has been lost, an acid environment is encouraged and persists longer. This in turn encourages aciduric bacteria which relish the acid conditions and continue to metabolize carbohydrate in the low-pH environment. The stage is set for uncontrolled carious attack.

1.6 CLASSIFICATION OF DENTAL CARIES

Carious lesions can be classified in different ways; this section introduces and defines this terminology.

Lesions can be classified according to their anatomical site. Thus lesions may be found in **pits and fissures** or on **smooth surfaces**. Lesions may start on enamel (**enamel caries**) or on exposed root cementum and dentine (**root caries**).

Primary caries denotes lesions on unrestored surfaces. Lesions developing adjacent to fillings are referred to as either **recurrent or secondary caries**. **Residual caries** is demineralized tissue left in place before a filling is placed.

Carious lesions may also be classified according to their activity. A progressive lesion is described as an **active carious lesion** (Figure 1.1) whereas a lesion that may have formed earlier and then stopped is referred to as an **arrested** or **inactive carious lesion** (Figure 1.7, 1.8). This concept of activity is very important as it impinges directly on management because active lesions require active management. However, the distinction between active and arrested may not be straightforward. There will be a continuum of

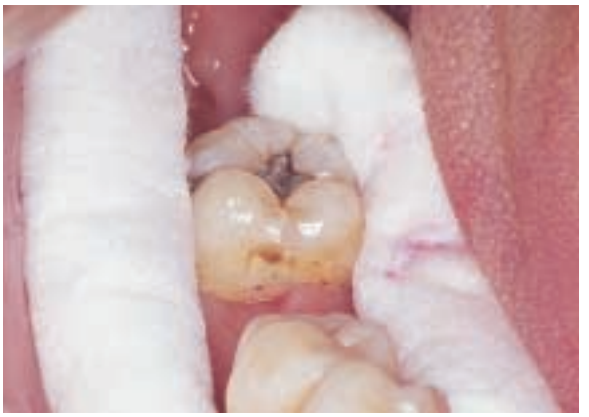


Figure 1.7. Arrested caries on the mesial aspect of the lower second molar. The lesion probably stopped progressing after extraction of the lower first molar.

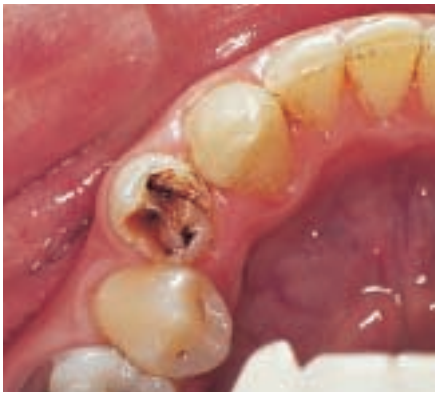


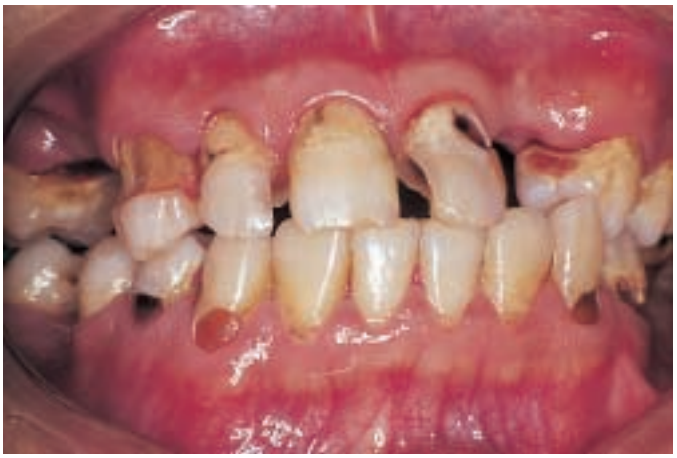
Figure 1.8. An arrested carious lesion in the lower first premolar. The lesion was well into dentine, but the tissue was hard and shiny. Note it is plaque-free. The tooth had been in this state for at least 10 years.

changes between active and arrested, and part of a lesion may be active while another part is arrested. This concept is totally logical because the lesion merely reflects the ecological balance in the overlying biofilm.

Different teeth and surfaces are involved, depending on the area of plaque stagnation and the severity of the carious challenge. Thus, with a very mild challenge only the most vulnerable teeth and surfaces are attacked, such as the cervical margin of the teeth or the occlusal pits and fissures of permanent molars. A moderate challenge may also involve the approximal surfaces of posterior teeth. A severe challenge will cause the anterior teeth, which normally remain caries-free, also to become carious.

Rampant caries is the name given to multiple active carious lesions occurring in the same patient, frequently involving surfaces of teeth that are usually caries-free. It may be seen in the permanent dentition of teenagers and is usually due to poor oral hygiene and taking frequent cariogenic snacks and sweet drinks between meals (Figure 1.9a–c). It is also seen in mouths

a



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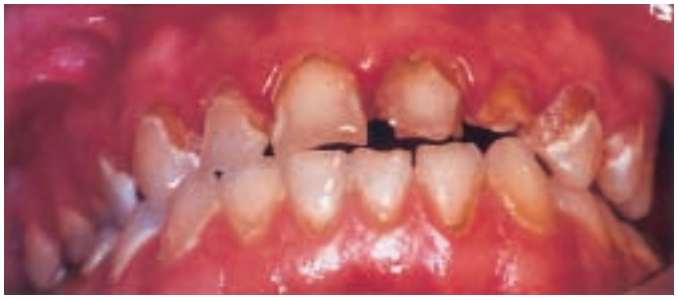

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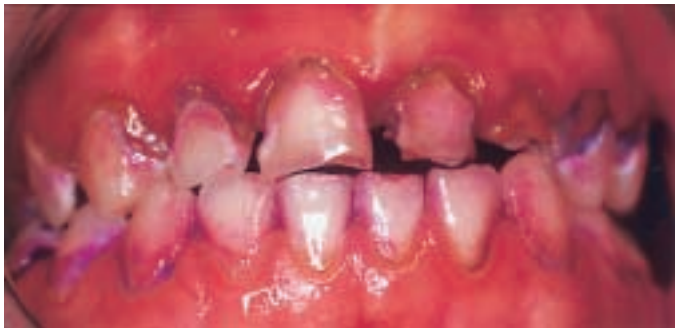

Figure 1.9. Rampant caries in young men: (a) Note these teeth look clean. This patient is now making strenuous attempts to remove plaque with a toothbrush. These lesions are on their way to arrest. Compare this with Figure 1.8. (b) Despite help with oral hygiene, this patient is not keeping these teeth clean. (c) The teeth are now disclosed and the plaque deposits are obvious. In addition, all this man's drinks are fizzy and sweet. This shows the devastating result of a combination of poor oral hygiene and a high-sugar diet.

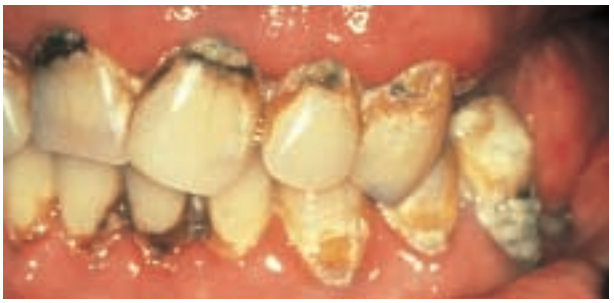

Figure 1.10. Radiation caries. This patient has been irradiated in the region of the salivary glands for the treatment of a malignant tumour. Heavy plaque deposits are obvious over the lesions.

where there is a sudden marked reduction in salivary flow (hyposalivation) (Figure 1.10). Radiation in the region of the salivary glands, used in the treatment of malignant tumours, is the most common cause of an acute reduction in salivary flow.

Early childhood caries is a term used to describe dental caries presenting in the primary dentition of young children.

Bottle caries or nursing caries are names used to describe a particular form of rampant caries in the primary dentition of infants and young children. The problem is found in an infant or toddler who falls asleep sucking a bottle (called a nursing bottle) which has been filled with sweetened fluids (including milk). Alternatively, nursing caries may be found in infants using a pacifier dipped in sweetener or in children who have a prolonged demand breast-feeding habit. The frequency of sugar intake combined with a low salivary flow at night are important in the development of this form of rampant caries. The clinical pattern is characteristic, with the four maxillary deciduous incisors most severely affected (Figure 1.11).

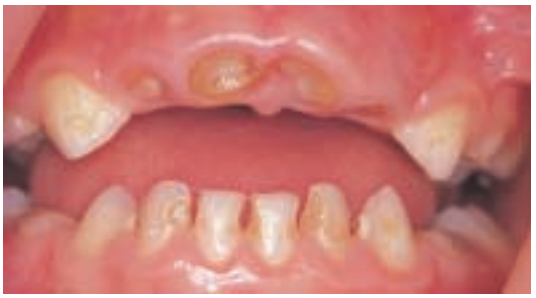

Figure 1.11. Rampant caries of deciduous teeth. The child continually sucked a dummy filled with rosehip syrup.

1.7 EPIDEMIOLOGY OF DENTAL CARIES

Epidemiology is the study of health and disease states in populations rather than individuals. The epidemiologist defines the frequency and severity of health problems in relation to such factors as age, sex, geography, race, economic status, nutrition, and diet. It is a bird's-eye view of a problem which attempts to delineate its magnitude, study its cause, and assess the efficacy of preventive and management strategies. Epidemiological surveys are of great importance to politicians because they should indicate areas of need where public money may be spent appropriately.

1.7.1 Measuring caries activity

Epidemiologists are interested in both the prevalence and the incidence of a disease. **Prevalence** is the proportion of a population affected by a disease or condition at a particular time. **Incidence** is a measurement of the rate at which a disease progresses. In order to measure incidence, therefore, two examinations are required—one at the beginning and one at the end of a given time period. The incidence of the condition is then the increase or decrease in the number of new cases occurring in a population within that time period.

Before incidence and prevalence can be recorded, a quantitative measurement is required that will reflect accurately the extent of the disease in a population. In the case of dental caries, the measurements of disease that are used are:

- the number of decayed teeth with untreated carious lesions (D)
- the number of teeth which have been extracted and are therefore missing (M)
- the number of filled teeth (F).

This measurement is known as the **DMF index** and is an arithmetic index of the cumulative caries attack in a population. DMF(T) is used to denote decayed, missing, and filled teeth; DMF(S) denotes decayed, missing, and filled surfaces in permanent teeth and therefore takes into account the number of surfaces attacked on each tooth. The similar indices for the primary dentition are def(t) and def(s) where e denotes extracted teeth (to differentiate from loss due to natural exfoliation) and f denotes filled teeth or surfaces.

1.7.2 Practical problems with DMF and def indices

There are some potential problems in the use of these indices. In young children missing deciduous teeth may have been lost as a result of natural exfoliation, and these must be differentiated from teeth lost due to caries. Permanent teeth are lost for reasons other than caries, such as trauma, extrac-

tion for orthodontic purposes and periodontal disease, or to facilitate the construction of dentures. For this reason missing teeth may be omitted from the indices and only decayed and filled surfaces included.

Epidemiologists take enormous trouble to achieve standardization of examination and recording techniques. They will practice and check their diagnoses during a clinical trial to try to ensure reproducibility. Despite this, even a trained and experienced worker will not be completely consistent on the same day, let alone consistent with others in studies spanning years.

In many populations there is a large filled component to the indices, and the dentists who have done the fillings are not standardized in their diagnosis of disease. Dentists do not practice and check their diagnostic reproducibility in the same way as epidemiologists. In addition, there is likely to be variation between dentists in their recording of disease. Epidemiologists carrying out national surveys may be limited in their access to clinical facilities because these surveys are not necessarily carried out in a dental surgery. Thus, access to good lighting, the ability to clean and dry teeth and the opportunity to examine radiographs may not be available. Unless radiographs are required for clinical care, it would be unethical to use ionizing radiation.

1.7.3 The relevance of diagnostic thresholds[4]

The recording of caries in epidemiological surveys is usually carried out at the 'caries into dentine' level of diagnosis. Enamel lesions are not recorded, which means that epidemiological surveys inevitably underestimate the caries problem. This may be very important because the earlier stages of lesion formation, which are not recorded, should be managed by non-operative preventive treatments so that the progression of lesions is controlled. The later stages (cavities) may also require restorations, in addition to preventive treatments. However, if only these are recorded, and those without cavities are described as 'caries-free', the politicians who commission the surveys in the first place may get a false impression of the dental care needed by the population.

This has indeed happened. In the early 1990s politicians (including dental politicians) in some developed countries gained the impression that because many children were described as 'caries-free', there was a danger of over-producing dentists. As a consequence of this unfortunate terminology and a lack of understanding of the carious process, some dental schools closed. However, it is now realized that in many people the carious process is delayed and thus lesions may present as cavities as the person grows older. In addition, the improvement in the caries status means there will be fewer extractions and thus many more teeth requiring dental care. For these reasons, more dental personnel are now needed. It must also be remembered that the arithmetic means of DMF(T) are meaningless at the level of the individual patient.

1.7.4 Caries prevalence[5]

Dental caries is ubiquitous in modern humans, and is the main cause of tooth loss in people of all ages. For most of the twentieth century caries was seen as a disease of economically developed countries, with a low prevalence in the developing world. By the late twentieth century this pattern was changing in two ways:

- There was evidence of a rise in caries experience in some developing countries. To give an example, studies in the 1990s show dental caries as a major problem in the former socialist countries of eastern Europe. These countries can be considered 'developing' in the economic sense, and the use of fluoride toothpastes and toothbrushes there is still low.
- By the late 1970s a marked reduction in caries experience among children and young adults was obvious in developed countries (Figure 1.12) although in 1983 there were considerable differences between countries.[6]

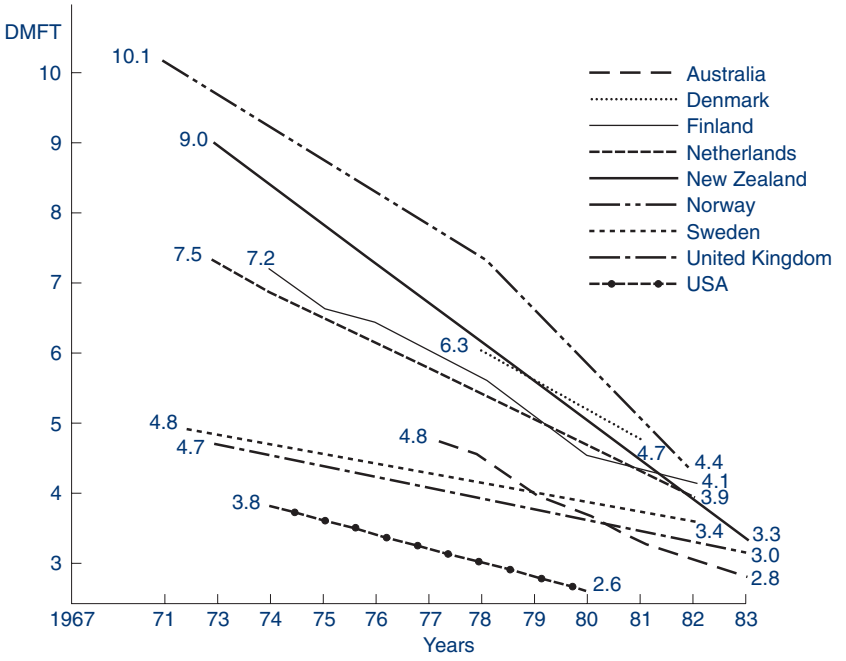

Figure 1.12, DMFT data from 12-year-old children of many countries demonstrating a decline in caries prevalence between 1967 and 1983. Note the considerable inter-country differences (data from the *WHO Global Oral Data Bank* (Renson *et al.*, 1986)[6]

The reasons for the decline in caries prevalence are not entirely understood, but experts consider the regular use of fluoridated toothpastes, preferably twice a day, to be the most important single factor.[7]

Data from studies indicate that the decline in caries took various courses. For instance, in Norway it appears the decline started several years before the widespread use of fluoride toothpaste. In the Netherlands, water fluoridation, beginning in 1953, led to a reduction in caries prevalence but the decline soon became independent of water fluoridation, which was discontinued in 1973.[8]

1.7.5 The position in the UK

Children

Figure 1.13 shows time trends in caries experience of children in England and Wales between 1973 and 1993. The end of the decline first became evident in the primary dentition (5-year-old data) with a levelling out becoming apparent in 12- and 14-year-old children in the early 1990s.[9]

Preliminary results of the 2003 Children's Dental Health in the UK survey have been released as this edition goes to press. There is little change between 1993 and 2003 in the obvious decay experience in 5-year-olds, but there has been a decrease in the average number of filled teeth in this age group. The picture for permanent teeth in older children is more positive with further reductions in obvious decay experience in 12- and 15-year-old children.

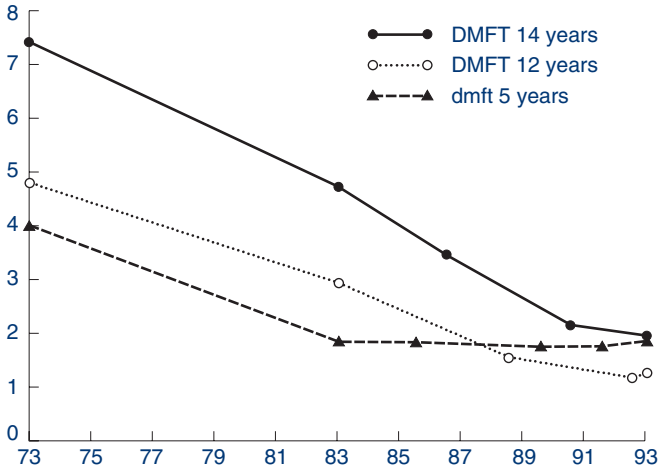

Figure 1.13. Time trends in caries experience of children in England and Wales between 1973 and 1993. (Reproduced by kind permission of the *International Dental Journal*.[9])

The reduction in caries experience has not occurred evenly across all tooth surfaces. As caries prevalence falls, the least susceptible sites (smooth and approximal surfaces) reduce by the greatest proportion, while the most susceptible sites (occlusal surfaces) reduce by the smallest proportion.

There are large regional inequalities in dental health, with people in Northern Ireland, Scotland and the north of England having the worst caries status (Figure 1.14).[10] In addition, caries is much worse in areas of social

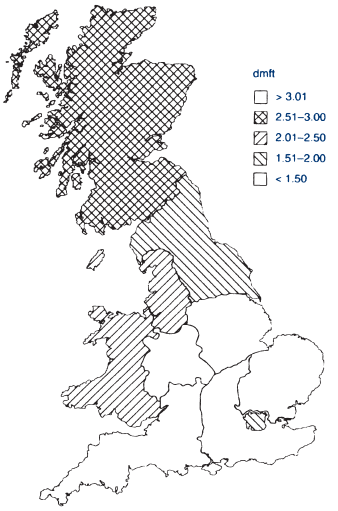

Figure 1.14. Dental caries experience (dmft) of 5-year-old children in Great Britain (BASCD coordinated National Health Service Dental Epidemiological Programme survey of 5-year-old children, 1999/2000). (Reproduced by kind permission of Blackwell Munksgaard).

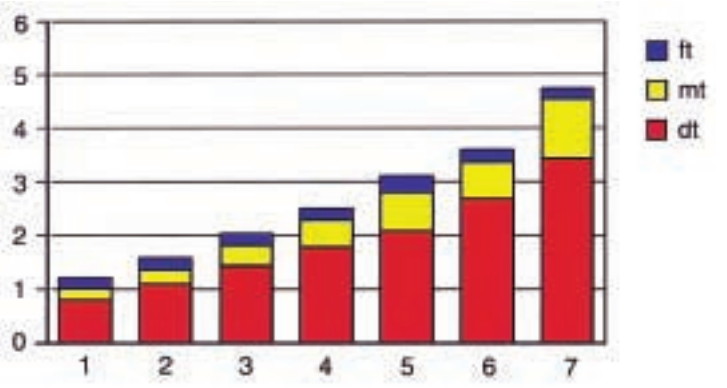

Figure 1.15. Mean number of decayed, missing and filled teeth by 'deprivation category' (DEPCAT score) in Scottish school children aged 5 years.[11] (Reproduced from Sweeney, P. C., Nugent, Z. L., and Pitts, N. B. (1999) Deprivation and dental caries status of 5-year-old children in Scotland. *Community Dent. Oral Epidemiol.* **27**, 152-9 by kind permission of Blackwell Munksgaard).

deprivation and Figure 1.15 shows the dmft levels for 5-year-old children in Scotland plotted against DEPCAT status (a measure of social deprivation based on postcodes).[11] A clear link between caries levels and DEPCAT scores, which reflect socio-economic conditions, is obvious. It can also be seen that many decayed teeth remain unfilled; presumably because many practitioners are unwilling to spend time restoring deciduous teeth.

Adults

National surveys of adult dental health, carried out in UK every 10 years, show steady and substantial improvements with the most dramatic improvements being in young adults. The bulk of filled teeth is now in older adults. Northern Ireland, Scotland and the north of England remain the parts of the UK with the poorest dental health.[12]

Older people

Caries is the commonest cause of tooth loss in all ages but edentulousness has decreased in UK adults. In 1968, 37% of the population over 16 had no teeth but by 1998 this had decreased to 13%. This means that people are coming dentate to old age, and caries in elderly people can be a particular problem because:

- oral hygiene may be poor if people are not able to brush or forget to do so
- salivary flow may be reduced by medications
- diet may change, with more sugar consumed.

The dental state of older people in residential homes is a disgrace. The clients are there because they can no longer look after themselves, and yet carers often do not clean mouths. It is unacceptable to ignore such an intimate part of the body—it eats, its speaks, it smiles, it kisses—and our profession must face this challenge (Figure 1.16).[13]

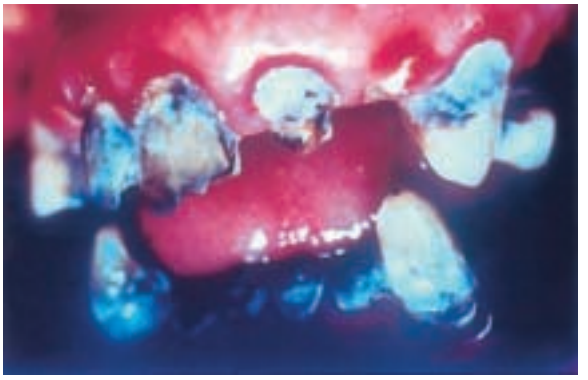

Figure 1.16. Gross caries in a client in a residential home. (Reproduced by kind permission of Dr Debra Simons).

1.8 MODIFYING THE CARIOUS PROCESS

Caries is a multifactorial disease. The cause is pH fluctuations in the bacterial plaque, but these in turn may be influenced by such factors oral hygiene, diet, fluoride and salivary flow. In addition a number of other variables are important such as social class, income, education, knowledge, attitudes and behaviour.

Figure 1.17 is a diagrammatic representation of the carious process. It makes the point that the process does not have to progress. When the destructive forces outweigh the reparative powers of saliva, the process will progress. Conversely, if the reparative forces outweigh the destructive forces, the process will arrest. Early diagnosis is important because, once carious lesions have cavitated, only operative intervention can replace the tissue. Fillings do not prevent caries, because new lesions can develop adjacent to restorations. If fillings are to last, preventive non-operative treatments must go hand-in-hand with operative treatment.

The basis of preventive, non-operative treatment is modification of one or more of the factors involved in the carious process. Since the process usually takes months or years to destroy the tooth, time is on the patient's side.

The dentist can help the patient modify the carious process in a number of ways:

- **Oral hygiene instruction.** Since the process is the metabolic activity in the biofilm, plaque removal using a fluoride toothpaste is very important (Chapter 4).

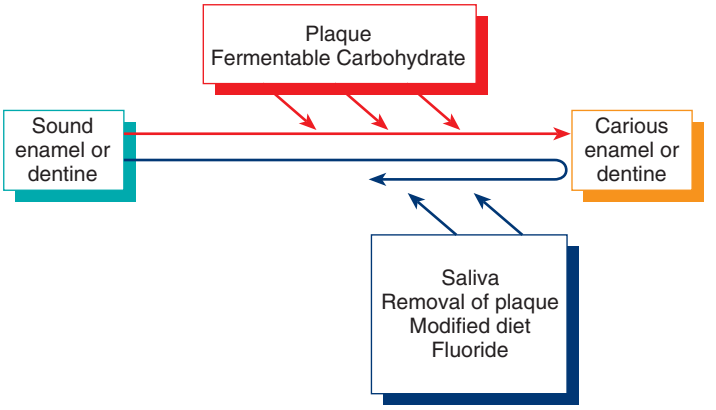

Figure 1.17. A diagrammatic representation of the carious process as an alternating process of destruction and repair. Sound enamel or dentine will become carious in time if plaque bacteria are given the substrate they need to produce acid. However, progression of lesions can be arrested by improving plaque control, modifying diet, and using fluoride appropriately.

- **Dietary advice.** Relatively simple measures, such as reducing the frequency of consumption of sugar and confining it to meal times, are usually sufficient (Chapter 5).
- **Appropriate use of fluoride.** Fluoride used in toothpaste, water, or mouthwashes and applied topically will delay progression of the lesion (Chapter 6).
- **Operative treatments.** Holes in teeth that are not cleansable are likely to progress. The role of operative dentistry in caries management is to facilitate plaque control (Chapter 9).

It is salutary to note that all the non-operative treatments require the patient's active cooperation. An important role for the dental profession, therefore, is to provide patients with knowledge so they understand their essential role in this control. In addition, patients need to be persuaded to accept responsibility for their own mouths (Chapter 8).

Further reading and references

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