Preface: an Editors' guide to reading the book

To our immense pleasure, the first edition of this book has found its way all over the world. In the genesis of this second edition we have enlisted the help of two associate editors, Vibeke Baelum and Bente Nyvad, who have helped us at the planning stage and by contributing extensively to the book. Most of the old text has been updated and there are 10 new chapters. Our band of 30 international authors has grown to 49.

A textbook reflects the way in which the authors interpret scientific data on a given subject, but we do not pretend that this is the 'truth' about the complex disease called 'dental caries'. There are extensive data available on today's internet and the stream of information will continue to grow. This is an enormous challenge to clinical students and practitioners. How can sense be made of the bombardment of information? The authors have been asked to present their respective subtopics carefully, so that it is not just a compilation of data, but selected data critically brought together in order to explain why dental caries presents itself in the individual and in populations in the way it does in today's world.

This preface aims to give a sequential, bird's-eye view of our efforts and map your journey through these pages by highlighting features that we, the editors, consider important. The aim of this book is to present the dental student and the dental practitioner with an update on the available knowledge about dental caries, and the consequences of this to its diagnosis, and how most appropriately and cost-effectively to control caries progression. Clinical decision-making and the balance between non-operative and operative treatments become even more important parts of daily life in clinical practice. An understanding of the caries process is needed to estimate the prognosis of treatment procedures and the possibility of assessing the risk of disease development in individuals and populations.

This book will demonstrate that in real life the processes involved in dental caries are highly complex. In an ideal world there would be a perfect model that could relate all the potential determinants to caries outcome. It will appear throughout the book that most of the determinants that influence caries can, at best, be measured only as proxy variables. The most we can hope for, therefore, is to develop probabilistic models that relate determinants to risk of caries progression. However, even under such circumstances, caries would remain unpredictable. Such inputs as:

- variable exposure to fluoride
- times, lengths, frequencies and types of sugar consumption
- quality of tooth cleaning
- fluctuations in salivary flow rates and composition
- quality and composition of biofilms
- the behavior of the individual
- the societal context of the individual

are themselves highly variable. It is likely that this variability and unpredictability of the inputs may play a crucial role in the way in which the caries process develops. But all these factors make up the fascination and challenge of our profession.

It is our hope that this book will prepare the reader to become a less dogmatic and more knowledgeable health professional who strives to control dental caries in the most cost-effective way.

Part I. The disease and its diagnosis

Chapter 1 defines caries as a localized chemical dissolution of a tooth surface resulting from metabolic events taking place in a biofilm (dental plaque) covering the affected area. These metabolic events are the *carious process*. The interaction between the microbial deposits and the hard tissues of the teeth may result in the *caries lesion* that is the sign or symptom of the process. Most of the components of the caries process, such as biofilm, diet and saliva, can be interfered with. They act at the tooth surface, but another set of determinants acts at the level of the individual. These include the person's behavior, knowledge, attitudes and education, and they may be much more difficult to modify.

Dental caries can be considered on a number of levels: the tooth surface, the individual and the population. This should be remembered throughout the book. There is a section on terminology, introducing the student to ways of classifying lesions, by their site on the tooth and their activity. This activity concept is critical to this book that is about *controlling lesion progression*, so that the ubiquitous natural process that is caries does not result in progressive tooth destruction.

Chapter 2 shows the student what caries lesions look like clinically on various tooth surfaces. In the past we thought that the clinical appearance of dental caries was known to every student, but teachers from around the world have asked us to show the spectrum which we consider to be important. So make yourselves familiar with the extensive variations in the clinical features. The theme of caries control is carried forward by showing lesions that are designated as 'active' and 'progressing' as well as those that are 'arrested'. There are also pictures of 'active lesions' being converted to 'inactive lesions' by non-operative treatments such as improved oral hygiene and fluoride application.

Having described the clinical manifestations of caries in Chapter 2, Chapter 3 goes on to describe histological manifestations. Lesions manifest themselves in different ways, depending on variations in anatomical structures. Understanding how anatomy influences clinical presentation is important in the diagnosis of caries. It is also important in appreciating how lesions progress and when a restoration might be required. It is anatomical features that influence when and how the tooth surface breaks to form a cavity and whether this hole can be cleaned by the patient. If the biofilm cannot be disturbed, the lesion cannot be controlled and is likely to progress.

The following four chapters all concern diagnosis, which is an essential resting place for the mind before making a treatment decision. Chapter 4 concentrates on what the eyes can see (visual), aided by gentle use of a probe (tactile). The chapter starts by warning the student that the lesion is the consequence of the metabolic activity of the biofilm. Thus, the dentist is looking at the reflection of the caries process, not the process itself. The authors then stress that the purpose of diagnosis is to direct the clinician to appropriate management. This explains why features such as cavitation and lesion activity are so important. A hole in a tooth may require repair if the patient cannot keep it clean; active lesions require active management, whereas arrested lesions do not. The student is warned that a good diagnostic test will be valid (measure what it claims to measure) and reliable (the measurement can be repeated and give the same result). Commonly used visual–tactile criteria are described and a systematic clinical approach is suggested and, very importantly, the results of this are linked to clinical management.

Sometimes vision is obscured, perhaps by an adjacent tooth, and radiographs may be needed to detect lesions. It should be noted, however, that a radiograph taken on a single occasion cannot determine lesion activity and it cannot say whether there is a hole in the tooth. Moreover, ionizing radiation should not be used as an excuse for slovenly clinical examination. Chapter 5 describes the use, indications and limitations of radiography in caries diagnosis, suggesting when radiographs are indicated.

In Chapter 6 several additional diagnostic measures are described. These methods are often quantitative and seek to improve on clinical–visual and radiographic examination. However, the methods will often involve expensive kit and must still be interpreted by the dentist, who must never pass the responsibility for diagnosis to a machine. The authors conclude that of the measures described only laser fluorescence and digital radiography are currently used in practice. The chapter is a salutary read for the geeks among us!

Part I concludes with a most thoughtful Chapter 7 that considers the foundations or building blocks for good diagnostic practice. What are we looking for and why? To what use will we put this information and what will be the consequences of error? We are warned that diagnosis is an error-prone exercise and that decisions are inevitably made under uncertainty. This is a chapter that should be read more than once! It argues for diagnostic methods that link directly to appropriate management options (for instance cavitation versus non-cavitation), an appreciation of error and a bias towards a less invasive management approach. This is probably the most important consideration in caries management.

Part II. Clinical caries epidemiology

Epidemiology is the study of health and disease in populations. Chapter 8 begins by explaining how caries is measured in these studies and questions whether such measures can be used to assess treatment needs at the population level. There would seem to be considerable difficulties in this approach. The chapter goes on to consider the distribution of caries and the influence of environment, particularly the social environment. Caries is just as much a disease of social deprivation as it is a problem of bad diet. These are critical concepts because they show the limitations within which a dental profession operates. The key to disease control lies in improving the broad social environment as well as the intraoral environment.

Chapter 9 expands on measurement issues in caries epidemiology. Examples demonstrate how different diagnostic thresholds influence just how much or how little of the total caries experience of an individual or population is captured. In particular, the term 'caries free' should be interpreted with caution because sometimes it may just mean 'cavity free', but certainly not free of a spectrum of early signs of caries lesions.

Part III. Dental caries in a biological context

Part III of the book focusses on the biofilm, saliva, and chemical interactions between the tooth and the oral fluids. Not only is this a part of the book that looks into the conditions prevailing in the oral cavity, but in most of the examples the authors deal with events taking place at the single tooth surface.

Chapter 10 concerns the biofilm, a community of resident microorganisms that grow on a surface and function together and whose ecology is influenced by saliva and diet. The development and structure of the biofilm are described and the importance of the microbial community is stressed; these organisms function in concert, not as individuals. The microbiology of caries is described and it is emphasized that no single organism, or group of organisms, may be held solely responsible for the initiation or progression of caries. Lesion progression is a result of a shift in the balance of the resident microflora driven by a

change in the local environmental conditions. Thus, changes in diet, saliva and oral hygiene are of extreme relevance to caries, and identifying what is driving deleterious changes is the key to control strategies, tailored to the individual patient.

Chapter 11 explores the very complex secretion, saliva, from a cariological point of view. This oral lubricant is not fully appreciated until its flow is diminished. It is a unique fluid film covering all mucous membranes of the oral cavity as well as tooth surfaces. Hence, its composition and relative velocity (flow rate) are of decisive importance for the microenvironment throughout all niches in the oral cavity.

The chemical interactions between the tooth and saliva (or rather the oral fluids) are considered in Chapter 12. The caries lesion is the result of loss of mineral from the dental tissues and this occurs over months and years. The metabolism in the biofilm results in fluctuating pH values at the interface between the apatite crystals and their immediate fluid surroundings. Thus, the equilibrium between the tooth mineral and the plaque fluid is constantly interfered with. This chapter explains the basic chemical reactions behind caries dissolution and the way the fluoride ion plays a role in lesion progression.

Erosion is a surface loss of tooth tissue in the absence of biofilm. The key to understanding whether we end up with a mineral loss beneath an apparently intact enamel surface (a caries lesion) or end up with a so-called erosion (surface etching) lies in understanding the concept of saturation of the oral fluids with respect to the minerals comprising the bulk of mineralized dental tissues. Chapter 13 explains the basic chemical differences between erosion and caries. Of particular importance, the chapter explains why fluoride should not be expected to be helpful in controlling this type of chemical dissolution of teeth. The chapter briefly describes causes of erosion, clinical appearances and management options.

Part IV. Non-operative therapy

Part IV of the book is about caries control using non-operative means or treatments. Chapter 14 questions what is meant by the word 'treatment'. Many interpret this word to be synonomous with filling teeth, but the biological thrust of the text thus far has been the concept of *caries control*, and thus the phrase non-operative treatment emerges.

For decades it has been claimed that a clean tooth never decays. Despite this, the relative role of oral hygiene in caries control is hotly debated and questioned by many as playing a key role. Chapter 15 therefore presents the evidence of the importance of mechanical plaque control at the level of the individual surface, the patient and the population.

Since the caries process takes place in the microbial biofilm, caries control by chemical or antimicrobial means may at first seem an attractive prospect. Chapter 16 reviews various antimicrobial approaches, but concludes that the ideal chemical agent for dental biofilm control is not yet available and, apart from fluoride, there is little evidence for a prophylactic effect in humans. This is due to the fact that the causative microorganisms are organized in complex biofilms. The organisms within biofilms communicate with each other and this communication may regulate pathogenic traits. Further understanding of these communication systems may lead to developments in antimicrobial therapy.

Chapter 17 follows a discussion of this antimicrobial approach by questioning whether caries control might involve immunization and gene therapy. In a nutshell, the answer is 'no, not at the moment'. Although work on vaccines goes back 50 years and much has been learnt, there are some significant problems in the approach. The multitude of microorganisms involved and the fact that they are commensals are particularly important. It seems doubtful whether vaccines will ever go to human clinical trials. Similarly, there has been much work on an immune response-based approach, but although much has been learnt about cariogenic bacteria, translation to a practical therapy in humans seems unlikely.

The presence of fluoride in the oral environment, together with the mother's educational background, explains about 50% of the caries reduction in contemporary child populations. Every dentist must have a profound knowledge about how fluoride acts in the control of caries lesion development and progression. Chapter 18 introduces the reader to how fluoride came into dentistry and how it may be used most appropriately today based on our current understanding of cariostatic mechanisms. Fluoride from any source ingested during tooth formation results in varying degrees of hypomineralization in enamel, the severity of which is a direct result of the fluoride dose. Therefore, the chapter also includes sections on this dose–response relationship, as well as how dental fluorosis manifests itself clinically and histologically. The chapter gradually reveals how the spectrum of various topical fluoride measures work together so as to obtain the most effective caries control.

Chapter 19 distills a vast literature on diet and caries and also includes a section on diet and erosion. Much of the evidence on diet and caries is now old history, and some of the experimental protocols would not stand up to contemporary scrutiny. Despite this, the volume of effort argues strongly for the importance of the relationship. That said, some aspects of the evidence are conflicting or maybe a little confusing; for instance, can starch be dismissed as blameless in the story? One of the most important questions addressed in this chapter is the relative role of dietary control in the postfluoride era. Another relevant question is what matters more, the total amount of sugar consumed or the frequency of intake. Fortunately, frequency and amount

are linked, so if we advise in this way, we may be covering both options. It is salutary to realize that human experiments on diet and caries are virtually impossible to design ethically. Thus, we must take every opportunity to evaluate current eating patterns and their likely role in dental health.

Part V. Operative intervention

Part V consists of chapters on operative treatment. Chapter 20 is entitled 'The role of operative treatment in caries control' and to some this very title will be an anathema because they contend that operative dentistry has no role in caries control; all it can do is replace, rather inadequately perhaps, damaged tooth tissue. Perhaps this attitude comes as an overreaction to an unfortunate attitude that appeared prevalent in operative dentistry in the middle of the twentieth century. The Editors were at dental school during this period, and cariology and the management of caries seemed to have no place in the departments of adult dentistry when we were students. Caries was presented as a disease of children, managed preventively in this age group, but in adults caries was 'treated' by filling holes in teeth. This attitude, once inculcated, dies hard and there will still be departments of operative dentistry where the science of disease processes is not the bedrock of the teaching.

Chapter 21 is about caries removal. It was challenging to write because the evidence for the current operative paradigm of removing infected tissue before tooth restoration seems scant. Indeed, what evidence there is seems to indicate that current practice may even be detrimental to the pulp–dentin complex by interfering too soon and too vigorously in active lesions before the natural defense reactions of sclerotic and reparative dentin have had a chance to work.

The argument presented is that it may not be necessary to remove 'infected' demineralized tissue to arrest the caries process. This argument makes total sense if it is accepted that the process takes place in the biomass and the infected caries lesion is merely a reflection of this process. Perhaps the bacteria in the demineralized tissue are merely opportunistic squatters rather than major players in the game once the overlying biomass, designated as plaque, has been removed.

However, this suggestion, although possibly logical biologically, is contentious. At present there is too little research on which to base decisions. In other words, an evidence base for practice is missing. The practitioner must therefore rely on 'current practice' as the only evidence available. There is an urgent need to design randomized clinical trials where varying amounts of infected tissue are removed and the results followed longitudinally.

Chapter 22 deals with tooth restoration and puts the emphasis on achieving cavity seal to protect the pulp–dentin complex. Materials science has made enormous strides since G.V. Black spent time working on amalgam. This remarkable dentist addressed the problems of operative dentistry with total logic. First, he studied the disease, clinically and microscopically. Then he applied this knowledge to preventing the problem by plaque removal and designing cavities to try to place their margins in areas where plaque did not stagnate. He then made restorations to the highest technical standards possible, given the limitations of the equipment and materials of the day. The approach is exemplary and it is the approach taken in this chapter 100 years later.

The available materials are described and the emphasis is placed firmly on adhesive materials that support tooth tissue, give a good cavity seal when handled correctly and are tooth colored.

The fastidious clinicians take up the story, showing, mainly pictorially, ways in which restorations may be placed. Notice the concentration on technical perfection. After all, if the aim of restoration is to make the tooth cleanable, perfect junctions between tooth and filling are important. The dental student should be inspired by the technical prowess demonstrated here. You too can achieve this provided you demand that your teachers are constructively critical and prepared to pick up a handpiece, an instrument, and show how your efforts can be improved. So only those who can achieve the highest quality should be allowed to teach restorative care.

Chapter 23 is about the atraumatic restorative technique (ART). This was originally developed in response to the need to find a method of preserving decayed teeth in people of all ages, in developing countries. The restorative material is generally a chemically polymerized, adhesive, glass-ionomer cement. Evidence is presented to show the success of the technique in occlusal restorations, but a somewhat lower success rate in the load bearing approximal situation. The technique is used alongside non-operative treatment. The chapter demystifies the subject. ART is not a second rate restorative technique for low-income countries, but a biologically based and rational approach to caries removal and restoration that is applicable anywhere.

The part ends with Chapter 24, considering the longevity of restorations. It is stressed that restorations have a limited lifetime and many fail owing to clinically diagnosed recurrent caries. Longitudinal randomized clinical trials and cross-sectional studies noting dentists' pragmatic decisions to replace restorations can both be used to assess longevity. Once a tooth has been restored, the filling is likely to be replaced several times in the patient's life and this repeated restoration can compromise the survival of the tooth. A tooth surface should not be restored unless it is unlikely that the lesion can be arrested. The durability of restorations should be maximized by optimal choice and use of restorative materials, prevention of recurrent disease and

judicious refurbishment to postpone replacement for as long as possible. The perspicacious student will notice the authors of Chapters 23 and 24 disagree on their interpretation of the literature on the longevity of glass ionomer relative to analgam restorations.

Part VI. Caries control and prediction

The part of the book that concerns caries control and prediction starts with Chapter 25, that summarizes the thought processes and ethics behind the concept of caries control. It emphasizes the need to base caries management decisions on biological knowledge rather than technical solutions. The Editors winced at the quote of the long-standing joke around science centers that 'the dental students are the only professional students on campus that can't locate the library'. We are uncomfortable because we know they can't locate the library at our schools because the dedicated dental libraries have been closed! The chapter emphasizes again the limited role of technical dentistry in the control of the biological process that is caries. It also argues that diagnosis should be linked to relevant treatment strategies and these should be based on the best evidence available; hence the need for a library.

Chapter 26 concerns health education and behavior, a subject of enormous importance in caries control because many non-operative treatments rely on patient compliance. The chapter outlines the theory of oral health promotion and education. There are useful practical tips for influencing behavior. However, the Editors are struck by the lack of research available in this field related to dentistry and therefore the lack of evidence base in this area. This seems surprising because studies of behavior, and its possible modification, seem salient to health in general, let alone the narrow field of caries control.

Chapter 27 on caries control for the individual patient is written by three dentists who relished the challenge of writing down what they actually do for patients based on the evidence presented in this book. We hope this chapter will be useful and understandable to ancillary dental workers and junior students as well as dentists. The authors argue it is important to identify patients at risk to caries progression and itemize important biological factors. They also caution that social factors, which may be impossible for the dentist to modify, can have an overriding influence. The non-operative treatments of plaque control, use of fluoride and dietary modification are dealt with in a practical way. Caries control in children and adolescents, patients with dry mouths and people who cannot care for themselves, is covered individually.

The reader may emerge from Chapter 27 with a warm, rosy glow at the thought of what might be achieved in the surgery setting, but beware the blast of cold reality that follows in Chapter 28. Now a group of community dentists considers caries control for populations and this chapter is uncomfortable reading for the wet-fingered dentist. We are reminded of the recent caries decline, but any self-congratulatory smile is wiped off our faces by the evidence showing chairside dentistry can take little credit for this success. To bring about a reduction in caries levels in populations a focus beyond the purely biological to the societal setting is required. A focus on making healthier choices easier and unhealthy ones more difficult is required (e.g. it should be usual for toothpaste to be fluoridated).

Two fundamentally different approaches to prevention are discussed: a high-risk strategy that targets efforts at those considered to be high risk, versus a whole-population strategy that targets everyone. The arguments for the whole population approach are persuasive. Finally, and perhaps most interesting and persuasive of all, is the common risk factor approach to prevention. Hygiene, diet and tobacco cessation are relevant to many diseases, so that in future dentists may find themselves promoting health in general, rather than dental health in particular.

Over the years the Editors have noticed some apathy from students studying dental public health. It can be seen as a waste of time, a distraction from the clinic. We can only conclude that in some schools the subject may be badly taught. We hope that students will be inspired by this chapter and its links to Chapters 8, 26, 29, 30 and 32.

Chapter 29 concerns caries prediction. Is it possible, on an individual patient basis, to predict who will and who will not develop progressing caries lesions? The answer to the question is intensely practical. If it is possible to predict, caries control strategies should be targeted at those at risk (the high-risk strategy). If it is not possible to predict, and the problem is still a common one, a whole-population strategy should be adopted. The chapter presents the evidence showing that clinical examination, together with a proper dental history, are the most important sources of information on which to base the decision. However, prediction prior to lesion formation is not reliable. Thus caries control should be based on a whole population or a directed population strategy. Clinical dentists, in focussing on the control of lesions currently present with self-care strategies, will also help to prevent the onset of future caries. This chapter links with the previous two showing how an individual patient and a population approach can combine to facilitate health.

This section on caries control and prediction ends with Chapter 30, considering economic issues. Economics is defined as a set of principles that allow decisions to be based on the efficient allocation of resources. One of the difficulties in writing this chapter is thrown into sharp relief in the opening sentences. The authors claim that the USA spends on health nearly half of what the whole world spends on health care. Read that sentence again please and consider for a moment. How can one possibly compare the

economic issues pertaining to such a high-income country with those in a low-income economy? A few themes emerge, however. The cost of restorative treatment seems almost obscene when considered against the average wage of some populations. Indeed, any dentist-delivered program, in economic terms, may be unacceptable. In contrast, community water fluoridation schemes are cheap, but to run them a central water supply is required. Fluoridated toothpaste may be no more expensive than its non-fluoride counterpart, so efforts to encourage improved oral hygiene using fluoridated dentifrices would seem the obvious way to go. However, it is not just as simple as providing 'free' paste and brushes. This also has a cost. Caries lesions are concentrated in socially deprived people. These economic considerations should make uncomfortable reading for the socially aware dental student.

Part VII. Dentistry in the twenty-first century

The final two chapters are very challenging and a must for any student because they lift the essentials from the minute details of the preceding chapters into a global view on:

- clinical decision making and
- the consequences of our knowledge for the future of dentistry if we are to serve the interest of the population.

Chapter 31 squares up to the variation in clinical decision making related to caries. It meticulously unpicks the problem to explain the reasons behind variation in both lesion detection and management options. It then lays out the consequences of the variation, and by this time the reader could be forgiven for being somewhat depressed. Fortunately, the cavalry comes over the hill in the last part of the chapter. There are real possibilities to reduce the variation by using systematically reviewed, scientific evidence. But how often are these reviews available?

Chapter 32 reflects on the role of chairside dentistry in the management of caries and periodontal diseases. It examines epidemiological data from high-income, middle income and low-income populations. It shows, uncomfortably, that the traditional chairside, dentist-to-patient approach to oral health-care delivery is both very expensive and inefficient. More scaling, more fillings, do not result in more functioning teeth. For a low-income society to follow the example of the high-income nations and devote resources to training dentists would be as unproductive as it is impractical. Furthermore, for high-income nations to train more dentists would be an expensive mistake. The key to oral health is desperately simple: a whole-population approach to improve oral hygiene with a fluoride dentifrice and encouraging abstinence from tobacco use. This chapter will raise the blood pressure of many but, when they have calmed down, they should reflect that the authors used the evidence available to reach these conclusions. The chapter ends with recommending how the dental team might be composed in the future if we are to serve the majority of this world's populations as cost-effectively as can be done based on the available evidence.

O. Fejerskov & E.A.M. Kidd
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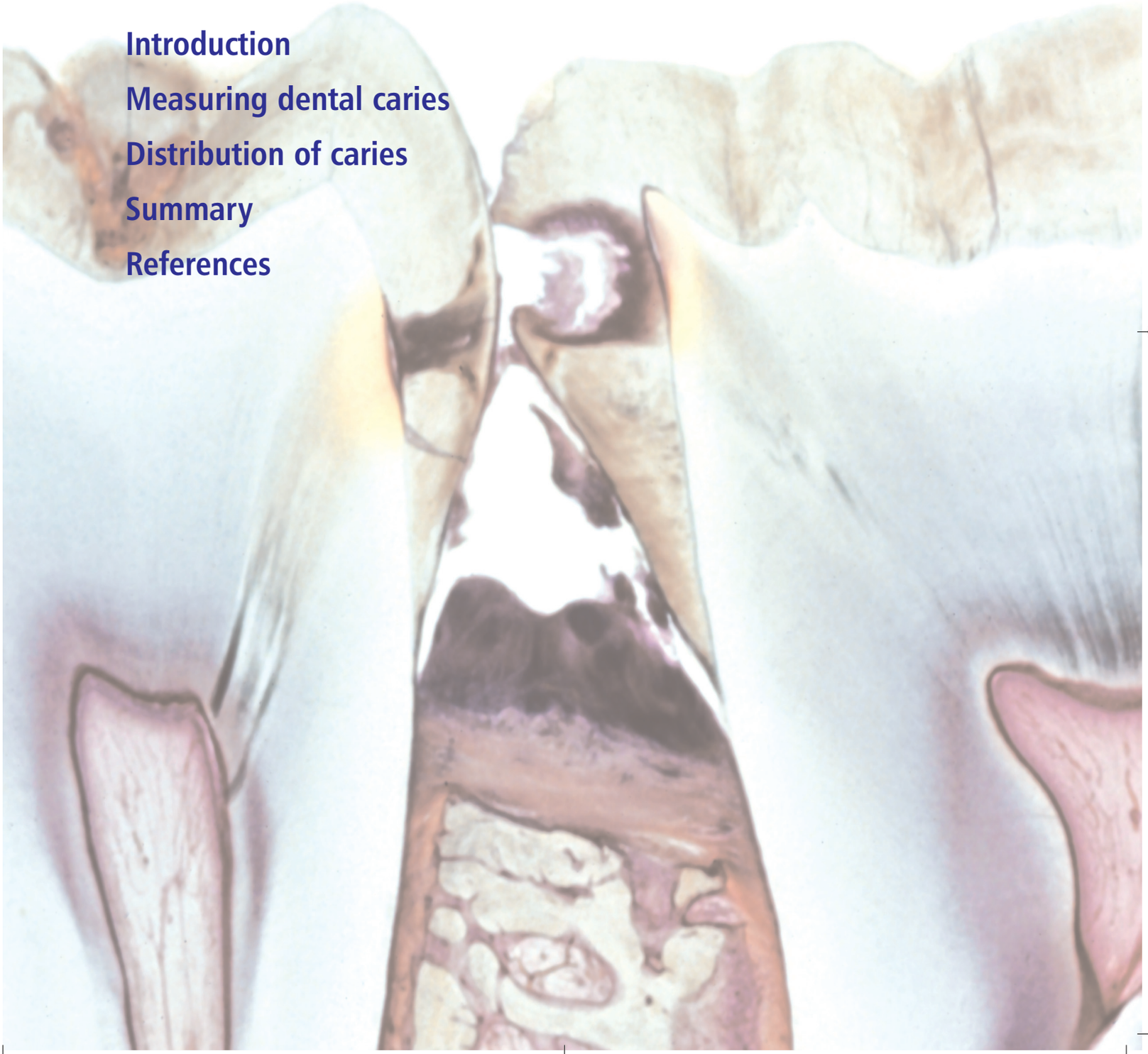
Part II
Clinical caries epidemiology

8

The epidemiology of dental caries

B.A. Burt, V. Baelum and O. Fejerskov



Introduction

Epidemiology is the study of health and disease in populations, and of how these states are influenced by heredity, biology, physical environment, social environment and human behavior. It differs from clinical studies in that epidemiology's focus is on groups of people, often whole populations, rather than on individuals or patients. The goal of epidemiological study is to identify the risk of disease that follows certain exposures, so that appropriate preventive interventions may be carried out at the public health and individual levels. To achieve this goal, epidemiological study uses a number of different research designs. All of them, however, include people with and without the disease in question, and with and without exposure to the correlates of interest. While research designs can become quite involved, Figure 8.1 shows the simple matrix that is the core of all epidemiological investigations.

Many factors are considered to be part of the causal chain in dental caries: bacteria, diet, plaque deposits, saliva quantity and quality, enamel quality, genetic history and tooth morphology have all been studied as possible risk factors for caries (Chapter 29). A risk factor is defined as:

> *An environmental, behavioral, or biologic factor confirmed by temporal sequence, usually in longitudinal studies, which if present directly increases the probability of a disease occurring, and if absent or removed reduces the probability. Risk factors are part of the causal chain, or expose the host to the causal chain. Once disease occurs, removal of the risk factor may not result in a cure (Beck, 1998).*

Epidemiology's role is to identify the risk factors for disease. As stated in the definition above, determining whether exposure to a potential risk factor leads to a particular disease requires longitudinal study. Where there is evidence to suggest that a particular exposure is a risk factor, but the relationship cannot be confirmed through prospective study, the exposure is referred to as a risk indicator.

Major intraoral entities that are part of the causal chain (e.g. oral microflora, specific dental plaques, and saliva quality and quantity) are dealt with in detail elsewhere in this book and so will not be part of this chapter. Instead, the distribution of dental caries in populations, and the factors that influence that distribution, are examined here. A major theme is that severe caries today is increasingly being recognized as a disease that closely follows a social gradient, so this chapter is broadened to emphasize the important role of the social environment in caries distribution. After a brief look at some of the issues in caries measurement, the relationships between caries experience and national income levels (as a broad measure of social resources) are examined. Then, following a brief consideration of how caries distribution is related to those individual attributes that cannot be changed, e.g. age, race, gender and genetic predisposition, the relationship between caries and socioeconomic status (an individual measure) and social determinants (a community measure) is explored. These community-based factors, sometimes called neighborhood characteristics, are now recognized as having a strong influence on caries extent and severity.

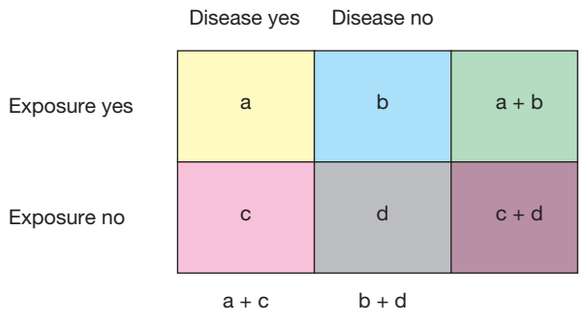


Figure 8.1 The simple matrix that is the core of all epidemiological investigations.

Caries is an ancient disease

Dental caries has been making people miserable since at least the time when humans began to develop agriculture. From palentological remains from the Iron Age, it appears that carious lesions in young people sometimes began in occlusal fissures, but developed no further because attrition progressed faster than caries. This pattern of development can still be seen today, e.g. in some African populations, where the rate of progression on approximal surfaces may also be so low that these lesions are ground away. Most lesions found in human remains from the Iron Age were cervical or root caries; coronal caries was relatively uncommon at this time, although it became more common during the time of the Roman Empire. Roman remains give evidence that some teeth with large coronal cavities had obviously been treated. The moderate caries experience found in Britain during the Anglo-Saxon period (sixth to seventh centuries) had changed little by the end of the Middle Ages (Moore & Corbett, 1971, 1973).

Increased consumption of processed food and greater availability of sugar were probably chiefly responsible for the development of the modern pattern of caries. Import duties on sugar in Britain were relaxed in 1845 and completely removed by 1875, a period during which the severity of caries increased greatly (Corbett & Moore, 1976; Lennon *et al.*, 1974). By the end of the nineteenth century, dental caries was well established as an epidemic disease of massive proportions in most of the economically developed countries (Burt, 1978). The severity of the caries epidemic in the late nineteenth century led directly to the establishment of public dental services, which first appeared in the Scandinavian countries.

Measuring dental caries

To study caries and its distribution in populations, it must be possible to measure it validly and reliably, and then put those measurements together in some systematic fashion so that caries distribution in one group can be compared with that in another. Since the disease of dental caries occurs on a continuum, from the earliest demineralization to cavitation, it is clearly important to have clear rules, or criteria, for the conditions under which caries is judged to be present. (Chapter 9 shows the impact of using differing measurement criteria on prevalence and severity data). Valid and reliable measurement is the basis of any science, including epidemiology, where an index (a numerical scale with upper and lower limits, with scores on the scale that correspond to specific criteria) is usually required to obtain a precise expression of disease distribution in a group. The properties of an ideal index are listed in Box 8.1. Index scores usually give no picture of clinical conditions (e.g. what does a plaque index score of 1.2 look like?), and in the past were often statistically mistreated when averages were computed from ordinal scales, but they have value when compared with the index scores from other groups measured in a similar way.

While various indexes for measuring caries were suggested during the 1920s to the early 1930s, it was only with Dean's studies of naturally occurring fluoridated water in the 1930s that a practical method was developed and used. Dean and his colleagues (Dean *et al.*, 1942) counted the numbers of teeth in the mouth with obvious caries (i.e. cavities). Filled teeth and teeth missing due to caries were added in, so that the index score included all teeth that had been attacked by caries. The first description of what is now known as the DMF index came from extensive studies of dental caries among children in Hagerstown, Maryland, USA, in the 1930s (Klein *et al.*, 1938). After that, the DMF index became the most used of all dental indexes.

- *Validity*. The index must measure what it is intended to measure, so it should correspond with clinical stages of the disease under study at each point.
- *Reliability*. The index should be able to measure consistently at different times and under a variety of conditions. The term *reliability* is virtually synonymous with *reproducibility*, *repeatability* and *consistency*, meaning the ability of the same or different examiners to interpret and use the index in the same way.
- *Clarity, simplicity and objectivity*. The criteria should be clear and unambiguous, with mutually exclusive categories. Ideally, it should be readily memorized by an examiner after some practice.
- *Quantifiability*. The index must be amenable to statistical analysis, so that the status of a group can be expressed by a distribution, mean, median or other statistical measures.
- *Sensitivity*. The index should be able to detect reasonably small shifts, in either direction, in the condition.
- *Acceptability*. The use of the index should not be painful or demeaning to the subject.

Box 8.1 Properties of an ideal index

The DMF index

As originally described, D was for decayed teeth, M for teeth missing due to caries and F for teeth that had been previously filled. Filled teeth were assumed to have been unequivocally decayed before restoration. The index could be applied to teeth as a whole (designated as DMFT), or applied to all surfaces of the teeth (DMFS). The DMFT score for any one individual can range from 0 to 32, in whole numbers, while the mean DMFT score for a group can have decimal values. The index can be modified to deal with such factors as filled teeth that have redecayed, crowns, bridge pontics and any other particular attribute required for study. It can also be applied with varying criteria for what constitutes caries. The original intention to score D only when there was cavitation has largely given way to scoring systems which record caries at all stages from the earliest enamel caries through to cavitation. An example of the latter is the International Caries Detection and Assessment System (ICDAS), which records caries on a six-point ordinal scale (Pitts, 2004). Another example is a scale that distinguishes between caries progress in active and inactive lesions (Nyvad *et al.*, 1999). As a result, DMF scores where caries is measured over its full range of development are higher than those where only cavitation, i.e. a later stage of caries progression, is used as the criterion for decayed teeth.

The DMF index has been used widely since its introduction in 1938 because it meets a number of the criteria for an ideal index (Box 8.1). For example, it is simple, versatile, statistically manageable and reliable when examiners have been trained. However, the DMF does have its limitations, and the main ones are listed in Box 8.2.

The limitations of the DMF index today frequently relate to modern preventive and restorative technology. For example, there were no sealants or adhesive resins when the DMF index was first developed. There are two reasonable approaches for adapting the DMF to deal with sealants. One says that the sealed tooth is not restored in the usual sense and should therefore be considered sound. The other says that it has required hands-on, one-to-one dental attention, and so should be considered a filled tooth. Probably the best way to deal with sealed teeth is to put them in a category by themselves, S for sealed. The DMFS index would then become DMFSS. Depending on the study's purpose, the S teeth can be left separate, included with F or regarded as sound.

The DMF index today is really outdated as a measure of caries incidence and severity, and may actually be more valid as a measure of treatment received. It is philosophically questionable to use an index for a disease that is so dependent upon the treatment judgments of many practitioners,

The DMF index has received remarkably little challenge over some 70 years of life, probably because it is conceptually simple and versatile in practice. But it was developed for use in children a long time ago, and accordingly it shows its age in a few areas. The principal limitations of the DMF index are:

- DMF values are not related to the number of teeth at risk. A DMF score for an individual is a simple count of those teeth that in the examiner's judgment have been affected by caries; it has no denominator. A DMF score thus does not directly give an indication of the intensity of the attack in any one individual. A 7-year-old child with a DMF score of 3.0 may have only nine permanent teeth in the mouth; thus, one-third of these teeth have already been attacked by caries in a short space of time. An adult may have a DMF score of 8.0 from a full complement of 32 teeth; thus, over a longer period only one-quarter of the teeth have been affected. DMF scores therefore have little meaning unless age is also stated.
- The DMF index gives equal weight to missing, untreated decayed or well-restored teeth. Common sense suggests that this philosophical basis is faulty for many purposes.
- The DMF index is invalid when teeth have been lost for reasons other than caries. Teeth can be lost for periodontal reasons in older adults, and for orthodontic reasons in teenagers. Decision rules, which go along with criteria, are required to determine how to deal with these instances.
- The DMF index can overestimate caries experience in teeth with 'preventive restorations'. In an epidemiological survey, such teeth must be included in the F component of DMF, although had they not been filled in the first place they might have been diagnosed as sound teeth. DMF scores will thus be inflated (Bader *et al*., 1993). Composite restorations judged to have been placed only for cosmetic reasons likewise should not be included in DMF counts.
- Composite and resin restorations not only may have been placed on non-carious teeth, but are often hard for an examiner to detect, thus leading to underestimation.
- DMF scores cannot be compared from one group to another without considering the criteria by which caries was considered present or absent. There is no universal criterion for what is a decayed tooth. Comparing one group where caries was recorded across the full disease continuum to one which only recorded caries at cavitation is clearly invalid.
- DMF data are of little use for estimating treatment needs.
- DMF cannot account for sealed teeth. Sealants did not exist in 1938, so are obviously not included in the description of the index. Sealants and other composite restorations for cosmetic purposes have to be dealt with separately.

Box 8.2 Limitations of the DMF index

and the combination of previous treatment (i.e. the M and F components) with current treatment need (the D component) is not used elsewhere in public health surveillance. An objective measure of caries activity (e.g. a marker of active disease) would be preferable to clinical judgment for many purposes, but because valid markers for caries are elusive, scoring caries activity is still based on clinical acumen (Nyvad *et al*., 1999). On the credit side, DMF has been used for generations and still has some use in monitoring trends over time. Its mix of treated and untreated caries measures also gives it some value in health services research. Until a more objective measure is developed and accepted then some modification of DMF will continue to be the principal index used to express the caries status of a population.

Other measures of caries

Other methods of measuring dental caries, using different philosophical bases, have been suggested from time to time. One is Grainger's hierarchy, an ordinal scale designed to simplify the recording of the caries status of a population, which uses five zones of severity of the carious attack (Grainger, 1967). This method was based on a landmark paper of Klein and Palmer (1941), which presented a caries-susceptibility order for the teeth, an ordering which has changed little down the years (Macek *et al*., 2003). Several studies confirmed the validity of the hierarchy (Katz & Meskin, 1976; Kingman, 1979; Poulsen & Horowitz, 1974). The Grainger hierarchy could be useful in public health surveillance, but it has received little further use.

'Composite' indicators have been suggested that attempt to measure health rather than disease by statistically weighting healthy restored teeth differently from missing or decayed teeth (Sheiham *et al*., 1987). The first of these is the FS-T, which sums the sound and well-restored teeth. The second is T-Health, which seeks to measure the amount of healthy dental tissue and ascribes descending numerical weights for a sound healthy tooth, a well-restored tooth and a decayed tooth. These are conceptually sound approaches to measuring dental health and function (rather than disease), and they probably deserve more attention than they have received.

An offshoot of the present-day skewed distribution of caries is the significant caries index (SiC) (Bratthall, 2000; WHO, 2005). The SiC is not a new index, but rather is a form of data presentation to help give a better picture of caries distribution in the population. It is the mean DMF score for the third of the population that is most affected by caries, intended to be used alongside the mean DMF of the whole population to give a more complete summary of its caries distribution. The more skewed the distribution, the greater the gap between the mean DMF and the SiC.

Criteria for diagnosing coronal caries

There is no global consensus on the criteria for diagnosing dental caries, despite a vast quantity of words on the subject. Apart from the inherent problem of diagnosing a borderline lesion, the major philosophical issue comes with scoring the early carious lesion which has not yet become cavitated. These lesions appear as discolored fissures without loss of substance, as a 'white spot' on visible smooth surfaces, or radiographically as an early interproximal shadow. The issue is that not all non-cavitated lesions progress to dentinal lesions requiring restorative treatment, and active non-cavitated lesions should receive non-operative treatment to prevent any further caries progression. With non-operative treatment (or sometimes even without it), a good

proportion of them will remain static or even remineralize (Pitts, 1993). These lesions are thus reversible, as opposed to dentinal lesions, which are usually considered irreversible. Because there are usually more non-cavitated than cavitated lesions at any one time in both high-caries and low-caries populations (Pitts & Fyffe, 1988; Bjarnason *et al.*, 1992; Ismail *et al.*, 1992; Machiulskiene *et al.*, 1998), the decision of whether to include or exclude them, and how to express them if included, can make a substantial difference in the oral health profiles obtained (Chapter 9). This is illustrated in Fig. 8.2, where in surveys in Kenya the carious lesions were recorded as both cavitated and enamel-only (i.e. non-cavitated). There is a marked difference in any caries profile depending on whether non-cavitated lesions are included or not.

Examples of these two broad approaches to diagnostic criteria for dental caries are shown in Box 8.3. European investigators have long recorded caries on a scale that extends through the full range of disease from the earliest detectable non-cavitated lesion through to pulpal involvement (Backer Dirks *et al.*, 1961). The criteria in Box 8.3 were first published by the World Health Organization (WHO) in 1979 (WHO, 1979), and will be referred to as the D_1–D_3 scale. (There is a D4 for pulpal involvement, but that recording is seldom contentious.) More recently, clinical researchers in Europe have expanded on this concept to produce a scale with up to 10 points, combining increasing depths of lesion development with clinical signs of activity or inactivity (Machiulskiene *et al.*, 1999; Nyvad *et al.*, 1999; Pitts, 2004). However, investigators in North America, Britain and other English-speaking countries have until recently used visual–tactile means to record caries as a dichotomous condition, meaning that caries was recorded only as present or absent. (This is referred to here as the dichotomous scale.) In the dichotomous recording, caries was only noted when it reached the level of dentinal involvement (Horowitz, 1972), i.e. the D_3 level. The D_1–D_3 scale requires the teeth to be dried and receive a longer, more meticulous examination, although with well-trained examiners this can be done even under fairly primitive field conditions.

A scoring system based on the D_1–D_3 scale is a necessity in cariology research studies, for it permits identification of lesion initiation, progression and regression. Research questions on the conditions under which early lesions progress, regress or remain static can only be answered with a measurement scale of this nature. Its use demands meticulous examiner training, since because D_1 lesions are capable of remineralizing back to sound enamel it becomes difficult to differentiate examiner error from natural phenomena. This may influence the assessment of absolute changes, although in the absence of bias it should not affect the contrast between groups. There is less consensus on whether the D_1–D_3 scale should be used in large-scale surveillance surveys, for arguments can be made both ways. Surveillance surveys are conducted at multiyear intervals to address the broad questions of whether disease levels are increasing or decreasing so that appropriate public policy can be formulated. For comparisons over time, it is clear that disease-measurement criteria need to be similar, and generally the simpler the system the better the examiner reliability. However, measuring caries only when cavitation can be

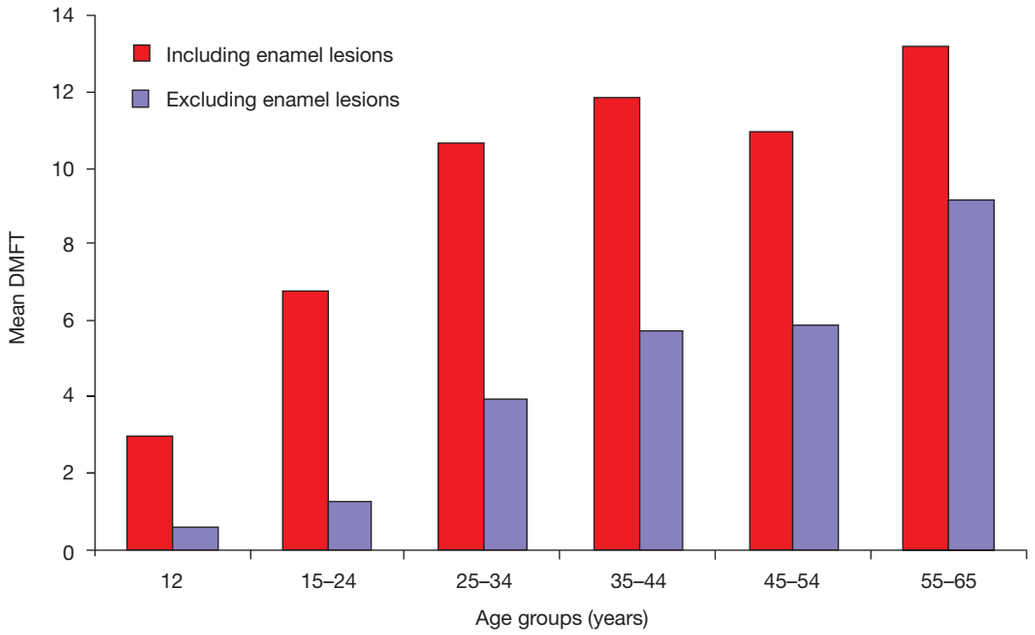


Figure 8.2 Mean DMFT scores by age in a Kenyan population, recorded for inclusion and exclusion of enamel caries (non-cavitated). (Source: Manji *et al.*, 1989.)

Diagnosis through the full range of caries; the D_1–D_3 scale

0. *Surface sound*. No evidence of treated or untreated clinical caries (slight staining allowed in an otherwise sound fissure).

D_1. *Initial caries*. No clinically detectable loss of substance. For pits and fissures, there may be significant staining, discoloration or rough spots in the enamel that do not catch the explorer, but loss of substance cannot be positively diagnosed. For smooth surfaces, these may be white, opaque areas with loss of luster.

D_2. *Enamel caries*. Demonstrable loss of tooth substance in pits or fissures, or on smooth surfaces, but no softened floor or wall or undermined enamel. The texture of the material within the cavity may be chalky or crumbly, but there is no evidence that cavitation has penetrated the dentin.

D_3. *Caries of dentin*. Detectably softened floor, undermined enamel or a softened wall, or the tooth has a temporary filling. On approximal surfaces, the explorer point must enter a lesion with certainty.

D_4. *Pulpal involvement*. Deep cavity with probable pulpal involvement. Pulp should not be probed. (Usually included with D3 in data analysis.)

Diagnosis at the dentinal lesion stage only; the dichotomous scale

Pits and fissures on the occlusal, vestibular and lingual surfaces are carious when the explorer 'catches' after insertion with moderate to firm pressure and when the 'catch' is accompanied by one or more of the following signs of decay:

- softness at the base of the area
- opacity adjacent to the area[a] provides evidence of undermining or demineralization
- softened enamel adjacent to the area that may be scraped away by the explorer.

[a] These areas should be diagnosed as sound when there is apparent evidence of demineralization, but no evidence of softness.

Source: Pitts & Fyffe (1988), Horowitz (1972) and WHO (1979).

Box 8.3 Typical criteria for diagnosing caries through the full range of lesion development (the D_1–D_3 scale), shown to contrast with the criteria for diagnosis at the dentinal-lesion stage only (the dichotomous scale)

detected underestimates the true extent of caries. Some see measuring caries only when cavitation is present as serving to perpetuate the old-fashioned 'drill–fill–bill' approach to tooth repair, while others see the opposite, that the same undesirable outcome will come from recording early demineralized lesions that should receive non-operative treatment. This discussion is likely to continue.

Measuring caries treatment needs

Assessment of the caries treatment needs of a group, at first glance, appears to be nothing more than the D segment of a mean DMF score assessed from a survey. This approach, however, has been shown not to work, for the reasons listed in Box 8.4.

Because surveys are usually conducted in field conditions that are less than ideal, relative to the dental office, it would be expected that surveys detect fewer carious lesions than

- Criteria used to diagnose caries in a survey usually are not the same as those used by practitioners in forming a patient's treatment plan. For example, a practitioner has to judge whether a minor lesion will develop into a major lesion over time, and whether a lesion in a primary tooth can safely remain untreated for the life of the tooth. A survey scores a tooth by how it appears at the present time.
- Patients' own perceived needs, level of interest in their dental conditions and ability or willingness to pay, all influence the level of treatment carried out. These factors are not part of survey data.
- Treatment philosophies change with expanding knowledge and technological developments; a treatment that is standard today may not be so tomorrow.

Box 8.4 Reasons why the DMF index is of limited value in measuring the treatment needs of a population

practitioners do. However, that begs the question of which assessment is 'correct'. Field surveys can miss early lesions, but practitioners can also overtreat. To add to the uncertainty, treatment plans for the same patients have been shown to vary drastically from one dentist to another (Espelid *et al.*, 1985; Bader *et al.*, 1993; Nuttall *et al.*, 1993).

The difficulty of determining treatment needs by survey was illustrated after a 1978 national dental survey in Britain. In the course of this survey, 720 dentate adults in Scotland agreed to permit their dental records to be followed over subsequent years. After 3 years, records showed that while 863 teeth in this group had been assessed as needing restorative care in the survey, 3108 actually had been restored. One might think that this finding could be explained by early lesions missed under the less-than-ideal survey conditions, but if that explanation is accepted then the next finding has no logic at all: of the 863 teeth classified as needing restorative treatment in the survey, only 271 (31%) were in the 3108 restored (Nuttall, 1983). This shows that the care carried out, rather than being an extension of the survey results, in fact bore no relation to them. This outcome is not easy to explain, but it would seem to illustrate the diverse approaches that dentists take towards diagnosing caries. It has been shown that dentists bring their own characteristics and biases to the task, and are largely guided by their inclinations towards particular intervention strategies. As a result, it is hardly surprising that there is substantial divergence between dentists in the nature of their diagnostic decisions (Bader & Shugars, 1997).

'Dental needs' in the USA were assessed by examiners in the first National Health and Examination Survey (NHANES I) of 1971–1974, and 65% of the population were judged as being in need of restorative care (US Public Health Service, NCHS, 1979). A similar assessment was made with the first national survey of schoolchildren in 1979/80, when 37% of schoolchildren were judged to be in need of restorative care (US Public Health Service, NIDR, 1982). These figures have received little use. In later national surveys (US Public Health Service, NIDR, 1987, 1989), treatment needs assessments were not carried out. The

most recent national surveillance survey in the USA, for which data were collected during 1999–2002, reported that 16.1% of children aged 12–15 years had at least one decayed tooth (enamel or dentinal cavitation level), but the issue of 'treatment needs' was not brought up (Beltrán-Aguilar *et al.*, 2005).

The WHO includes a subjective treatment-need assessment by the examiner as part of its Pathfinder survey method (WHO, 1997), although it is not known how well these estimates approximate treatment actually carried out. WHO also has developed a broad-based approach to determining needs in low-income countries through what it calls a situation analysis, an enhancement of Pathfinder survey data with information on population trends, school enrollment figures, per capita income and health-care resources (WHO, 1980).

Distribution of caries

Global distribution

Caries has historically been seen as a disease of the high-income countries, with a low prevalence in poorer countries. The most obvious reason for this pattern is usually considered to be diet: high consumption of refined carbohydrates and other processed foods in the high-income countries and hunting and subsistence farming in the low-income countries.* Some of the historic patterns of high attrition, little coronal caries and moderate prevalence of root caries described above, can still be found in some parts of the world, but they are fast disappearing as once-isolated populations increasingly adopt the cariogenic diets and cultural lifestyles of the high-income world.

There is good evidence that this historical pattern was clearly changing by the later years of the twentieth century. First, there was evidence that caries experience in some low-income countries had risen in the years after World War II (1939–1945) (Møller *et al.*, 1978), although this change was by no means universal, with some populations, notably in Africa, remaining relatively unaffected (Chironga & Manji, 1989; Mosha & Robison, 1989; Manji & Fejerskov, 1990; Baelum *et al.*, 1991; Mosha & Scheutz, 1992; Mosha *et al.*, 1994). The second change is the marked reduction in caries experience among children and young adults in high-income countries, a trend that first became evident in the late 1970s (Burt, 1978). This change affects the oral conditions of the whole population in due course as today's younger cohorts progress through the lifespan.

WHO maintains the Global Oral Health Data Bank, a collection of surveillance data from most of the world's countries. As in any surveillance system where a data-collection protocol is used in a multitude of different situations by different people, there are likely to be some inconsistencies in these data. Still, the databank provides an invaluable profile of broad trends in oral health. Figure 8.3 uses figures from the Global Oral Health Data Bank and from the World Bank to show the trends in DMFT scores in 11 high-income countries from 1979–1987 and from 1993–2002. For most of these countries the decline in caries levels has been substantial, but again it is not universal because both Korea and Kuwait have seen a rise in DMFT scores. This could be because preventive measures have lagged behind rapidly growing affluence (and hence easier access to cariogenic diets) in these countries.

Figure 8.4 shows the same display for selected upper middle- and lower middle-income countries, those without the same resources as the countries in Fig. 8.3, and here the pattern is different. Only Cuba, which has had a school dental service for years, and Estonia, where caries levels were very high, have shown a substantial drop in caries patterns over the past 20–25 years. Of the others, four have shown a minor decline, and four have had an increase. They do show a pattern: nations with better developed public health prevention programs generally have shown most success with caries reduction. However, distinct differences in caries experience exist from one country to another and from region to region within a country. Intercountry differences are illustrated by the results of the second International Collaborative Study (ICS II) in the 1990s (Chen *et al.*, 1997), shown in Fig. 8.5. The 13–14-year-old children examined were from selected communities rather than from nationally representative population samples, so there is a possibility of selection bias in these data. The highest caries levels are seen in Eastern European countries and Japan. Differences in caries experience between countries are not always easy to explain, but can often be seen to stem from sample selection and variable caries criteria.

Secular variations in caries extent and severity

Several reports from both national data and local surveys by the early 1980s suggested that the previously high DMF scores in high-income countries were diminishing (Hunter, 1979; McEniery & Davies, 1979; Sardo Infirri & Barmes, 1979; Hugoson *et al.*, 1980; Anderson *et al.*, 1981; Glass, 1981; Hughes & Rozier, 1981; Bryan *et al.*, 1982; Fejerskov *et al.*, 1982; Stookey *et al.*, 1985). In the USA, as one example, this decline in caries prevalence and severity was confirmed by results from the National Dental Caries Prevalence Survey in US School Children of 1979/80 (US Public Health Service, NIDR, 1981). This survey found that mean DMF scores among children aged 5–17 years were some 32% lower than those in the first National Health and

* The World Bank classifies countries as high-income, high middle-income, low middle-income and low-income. These terms replace such words as developed/developing and industrialized/non-industrialized. See the World Bank website: http://www.worldbank.org/data/countryclass/classgroups.htm

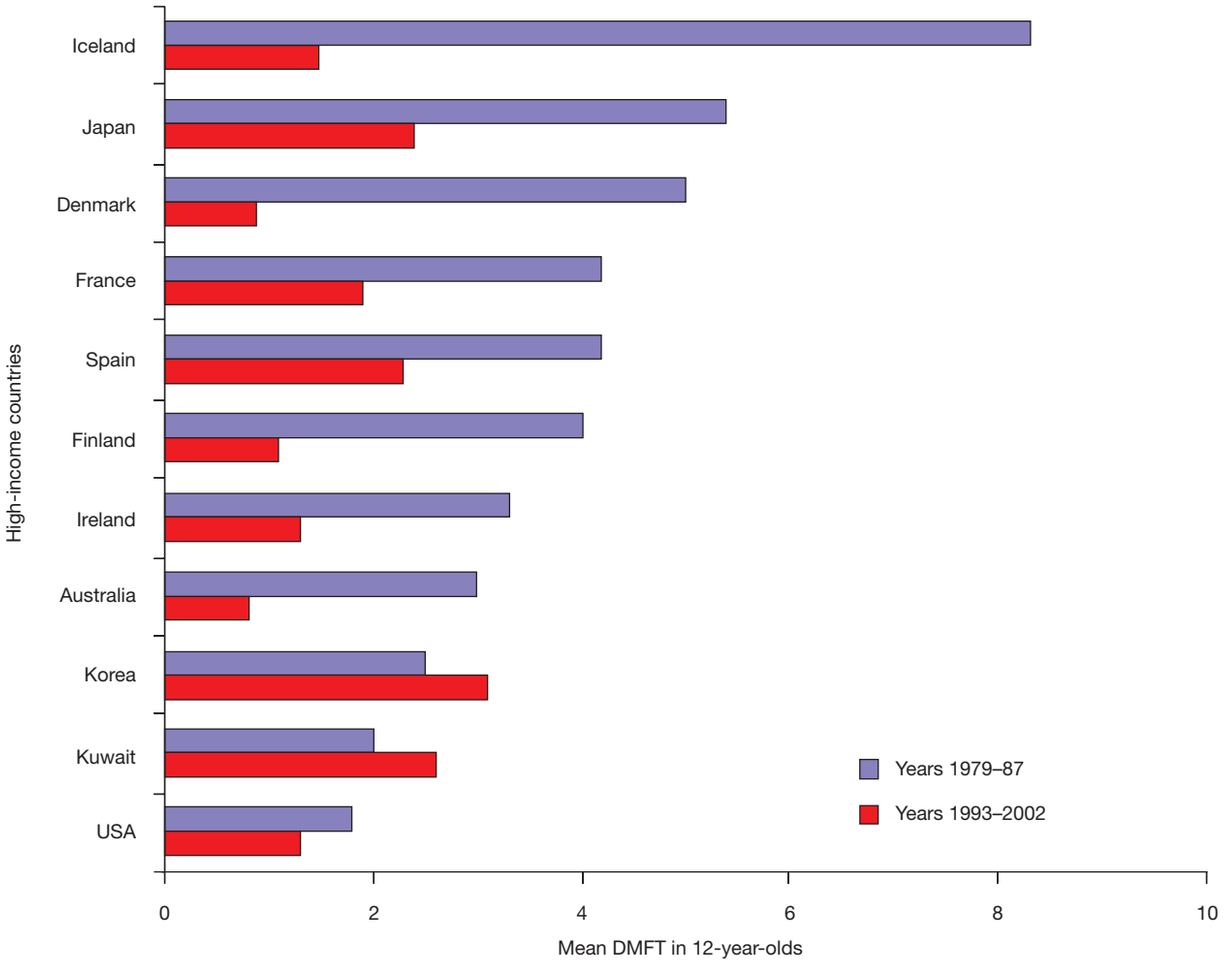


Figure 8.3 Caries then and now: DMFT scores for 12-year-old children from various surveys in high-income countries (as defined by the World Bank) during the period 1979–1987, and again during the period 1993–2002. (Source: World Health Organization, *Caries for 12-year-olds by country/area*.)

Nutrition Examination Survey of 1971–1974 (US Public Health Service, NCHS, 1981). The next national survey of US schoolchildren in 1986/87 found that the decline was continuing (US Public Health Service, NIDR, 1989), with mean DMF scores for 5–17-year-olds again 36% lower than those from seven years earlier, and this trend continued through the national survey of 1988–1994 (US Department of Health and Human Services, 1997).

The caries decline in the permanent dentition continues to the present day, and is apparent in just about all of the high-income countries. As an example from the USA, Fig. 8.6 shows the average DMFT scores among children of three different age groups in the two most recent national surveys, only some 8–11 years apart. Another example comes from Australia, where the city of Tamworth was the site of a series of dental surveys over a period of 25 years. The first survey was a baseline for Tamworth's commencement of water fluoridation in 1963, and the surveys of schoolchildren continued periodically until 1988. Figure 8.7, in which the data are all for 15-year-old children, shows the trend of constantly diminishing caries experience in Tamworth over a period of increasing use of fluoride.

The DMF index, as described above, is a measure of average caries severity in terms of the number of teeth affected by caries. Another measure, more readily understood by other health professionals and the public, is prevalence, defined as the number of disease cases at a given time (while incidence is the number of new disease cases over a specified time). It is shown in Chapter 9 that caries is a process, thus making it difficult to say exactly when caries starts. Chapter 9 also makes the point that there really is no such thing as 'caries free'. However, public health workers need to be able to identify those children who need operative or non-operative treatment, and a convenient marker for that is 'free of obvious caries', i.e. open cavities or tissue loss in fissures that are readily detectible. So 'prevalence of caries', as the term is used in public health surveillance and programming, means the proportion of a population that

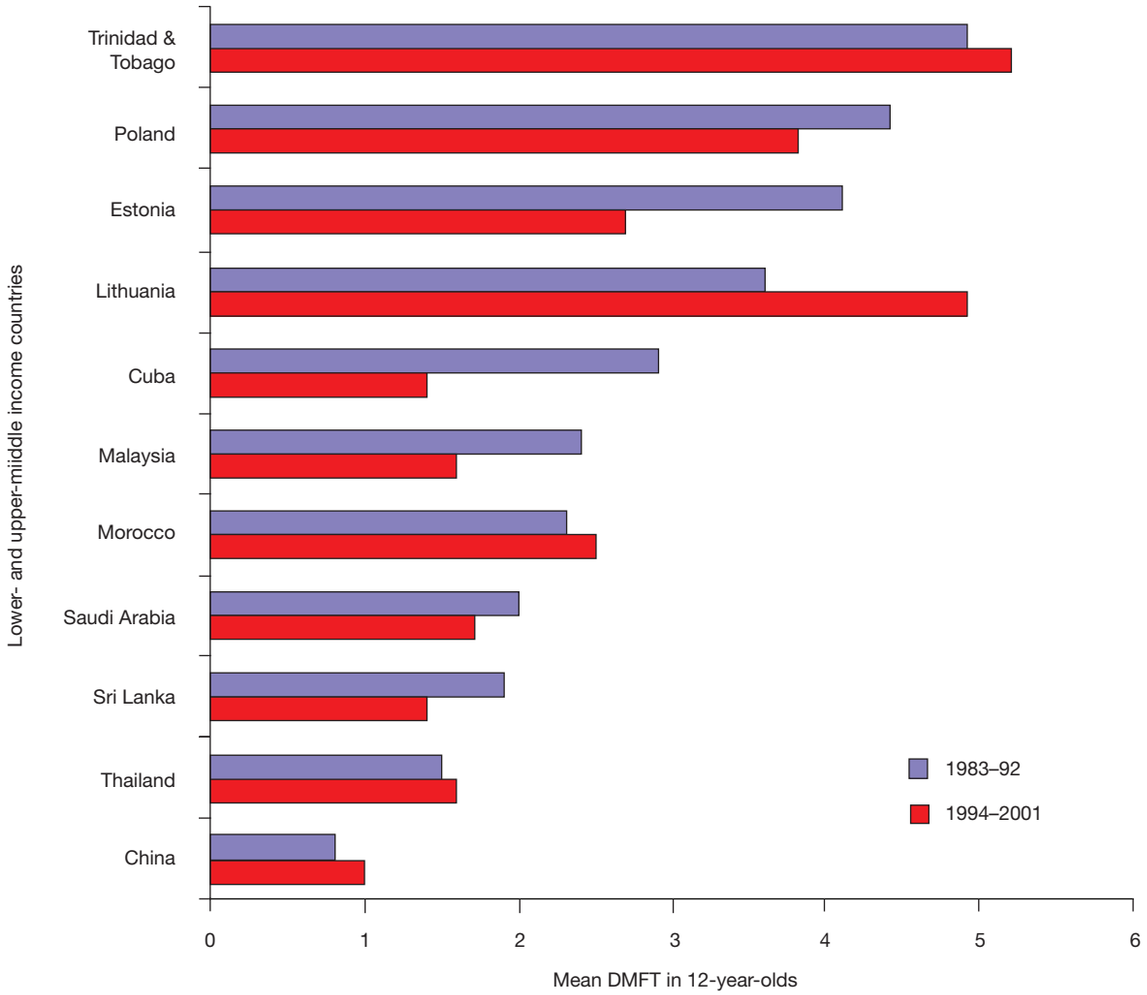


Figure 8.4 Caries then and now: DMFT scores for 12-year-old children from various surveys in upper middle- and lower middle-income countries (as defined by the World Bank) during the period 1983–92, and again during the period 1994–2001. (Source: World Health Organization, *Caries for 12-year-olds by country/area*.)

has at least one obvious lesion (or a restoration or extraction due to caries). Looking at such data from the Danish School Dental Service over the period from 1988 to 2005 (Fig. 8.8), it can be seen that the prevalence of caries has dropped steadily over the years, both in the primary dentition (5-year-olds) and in the permanent dentition (15-year-olds).

The seemingly sudden decline in caries among children in high-income nations (which was perhaps not all that sudden because there are data to suggest that it might have started in the 1960s: Fejerskov *et al.*, 1982; Burt, 1985) was documented at a 1982 conference in Boston, the proceedings of which were published in a special issue of the *Journal of Dental Research* in November 1982. The decline in caries in the permanent dentition among children of the high-income nations has continued since then (Kumar *et al.*, 1991; Bjarnason *et al.*, 1993; Athanassouli *et al.*, 1994; Downer, 1994; Spencer, 1994; Marthaler *et al.*, 1996; Truin *et al.*, 1998; Carvalho *et al.*, 2001). The main caries problem in the high-income countries today is not so much overall caries levels, but rather the disparities in disease experience and treatment between different socioeconomic and racial/ethnic groups.

Regarding caries trends in the primary dentition, there is evidence that the caries decline paralleled that seen in the permanent dentition until around the late 1980s to early 1990s, when the decline seemed to level out (Hargreaves *et al.*, 1987; Brown *et al.*, 1995; Kaste *et al.*, 1996; Marthaler *et al.*, 1996; Poulsen, 1996; Speechley & Johnston, 1996; Truin *et al.*, 1998; Poulsen & Scheutz, 1999). Data from the Danish School Dental Service shown in Fig. 8.9 and covering the period from 1988 to 2005, however, suggest that another decline was evident in the early 1990s, followed by a plateauing from then to the present. (Figure 8.9 also

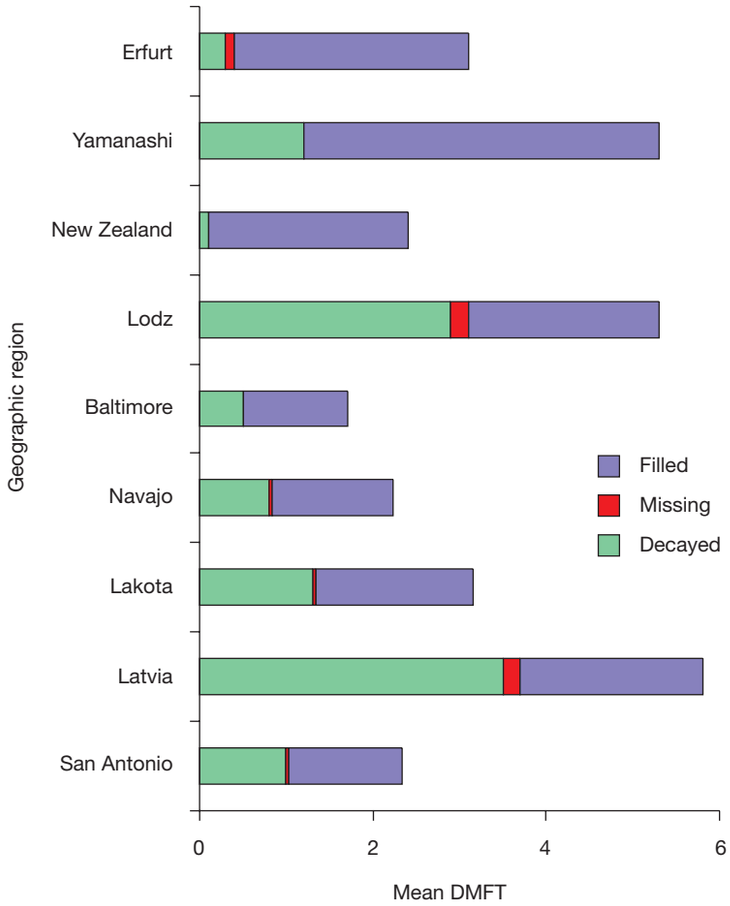


Figure 8.5 Mean DMFT scores in children aged 13–14 years in nine communities in six countries. (Source: Second International Collaborative Study, Chen *et al*., 1997.)

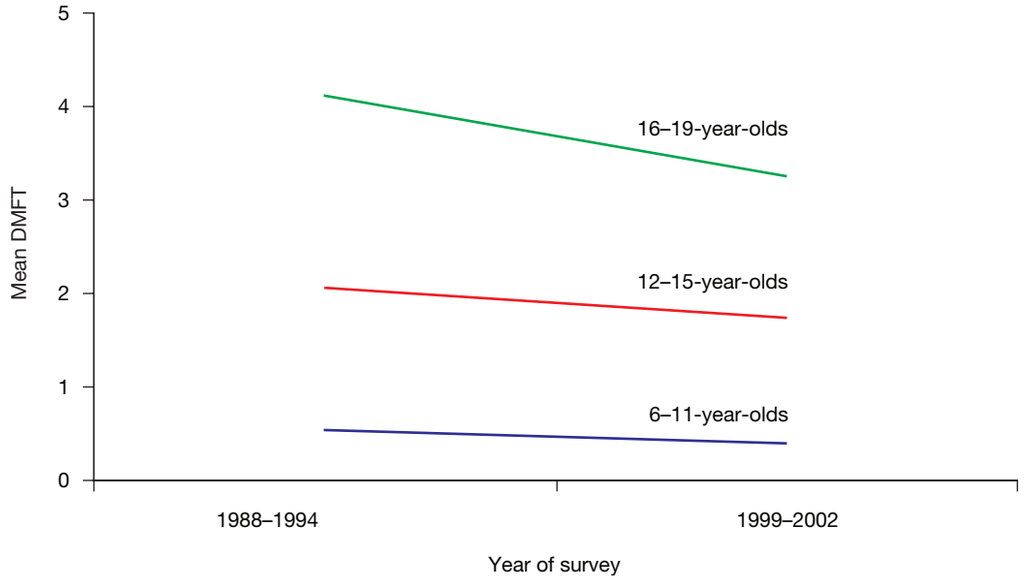


Figure 8.6 Decline in DMFT scores in three children's age groups between two national surveys, USA, 1988–94 and 1999–2002. (Source: Beltrán-Aguilar *et al*., 2005.)

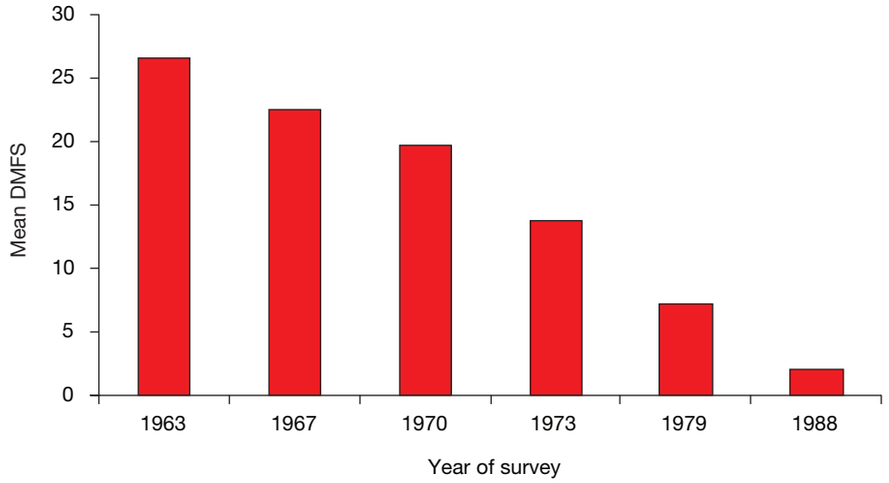


Figure 8.7 Decline in mean DMFS scores in six cohorts of 15-year-old children in Tamworth, Australia, over a 25-year period from 1963 to 1988. Tamworth has fluoridated its drinking water since 1963. (Source: Barnard, 2005.)

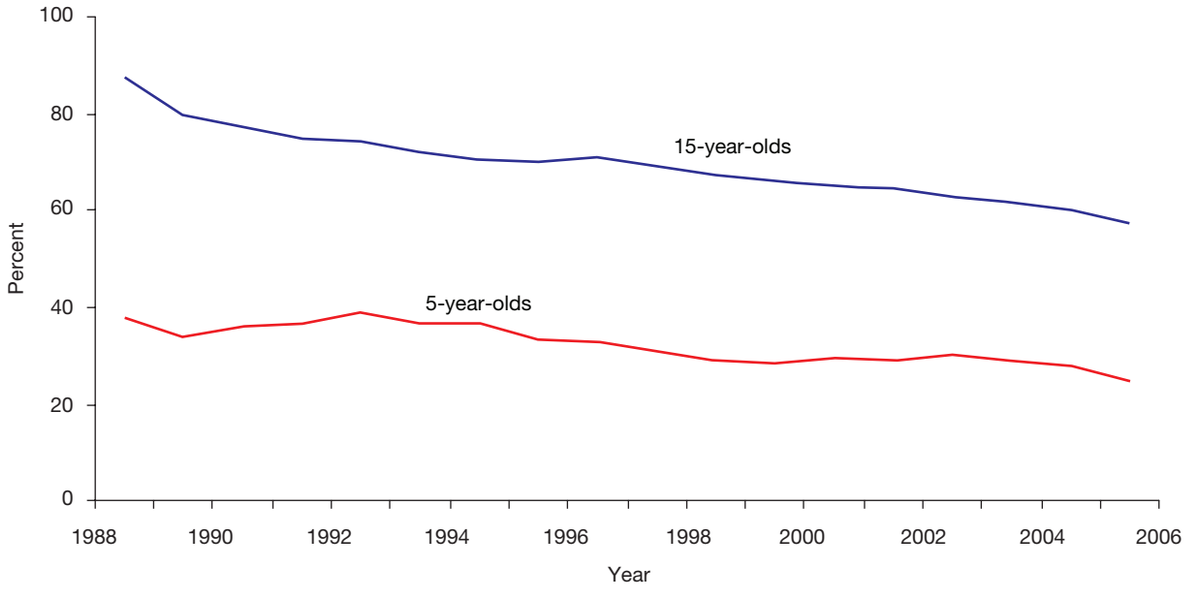


Figure 8.8 Prevalence of caries in 5-year-old Danish schoolchildren (primary dentition), and in 15-year-old schoolchildren (permanent dentition), 1988–2005. (Prevalence defined as the presence of at least one obvious cavity, or restoration or extraction due to caries.) (Source: Danish National Board of Health, Copenhagen, 2006.)

demonstrates again how including or excluding non-cavitated lesions in the recording affects the outcome.)

The reduction in caries has not occurred evenly across all kinds of tooth surfaces, for it has been proportionately greater in free smooth surfaces and proximal surfaces than in pit-and-fissure surfaces (Bohannon, 1983; Stamm, 1984). In a 3-year longitudinal study in Michigan in the early 1980s, 81% of all new lesions were on occlusal surfaces and in the pits and fissures of buccal and lingual molar surfaces, with no lesions at all on free smooth surfaces (Burt *et al.*, 1988). The net result is that while the total number of new carious lesions has been dropping, an increasing proportion of them is made up of pit-and-fissure lesions.

> *As caries prevalence falls, the least susceptible sites (proximal and smooth surfaces) reduce by the greatest proportion, while the most susceptible sites (occlusal) reduce by the smallest proportion (McDonald & Sheiham, 1992).*

History has many examples of diseases that have waxed and waned without a firm understanding of why, and caries is one of these. No clear reasons for the caries decline have been identified, although most researchers see the various

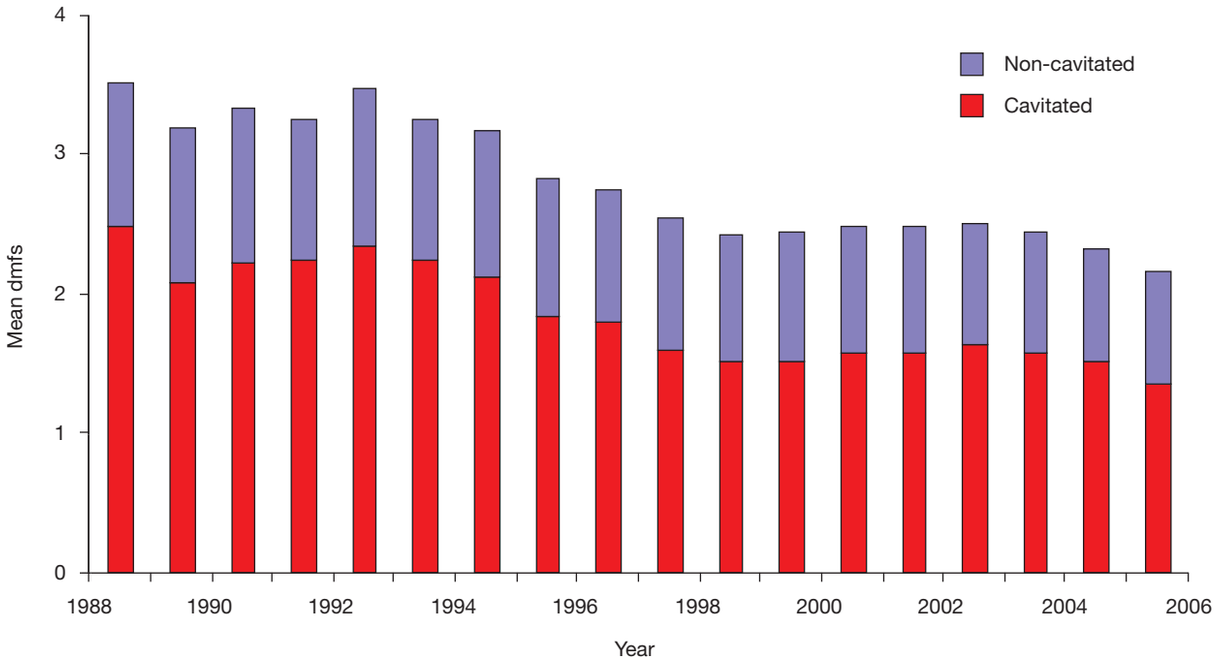


Figure 8.9 Trends in caries experience in the primary dentition in Danish 5-year-old children between 1988 and 2005. (Source: Danish National Board of Health, Copenhagen, 2006.)

uses of fluoride as the main reason (Bratthall *et al.*, 1996). However, fluoride alone does not explain more than about 50% of the reduction (Marthaler *et al.*, 1996), and we still do not have a clear understanding of the relative strength of caries risk factors. Sugar consumption, in the USA at least, has increased rather than diminished, and there is no good evidence concerning the roles of better oral hygiene, changes in the bacterial ecology of the oral cavity and the widespread use of pediatric antibiotics on oral bacteria. Many see a likely impact, though as yet unquantified, from raised living standards that come with indoor plumbing, and elevated social norms concerning laundry, personal hygiene and grooming. Better oral hygiene can easily be seen as part of more meticulous personal hygiene and grooming rather than a conscious act to improve oral health. As with the cyclical nature of other diseases over time, however, it is quite likely that there are factors operating that have not been identified (see discussion later in this chapter on the effect of the social environment).

The uneven distribution of caries

For many years, the results of national surveys were presented only as mean DMF values, usually with only a standard deviation to indicate the distribution. While means are valid and useful, they 'compress' extreme values, meaning those with no caries and those with a lot, into an average figure which sometimes can be misleading. A break from this convention in the USA came with the results of a major preventive study in the mid-1980s, which drew attention to the fact that while average caries experience in children was lower than the researchers had originally expected, there was still a significant minority with severe caries (Graves *et al.*, 1986). This type of distribution is illustrated in Fig. 8.10, which shows data from the US national surveys of schoolchildren in 1979–1980 and 1988–1994. Rather than mean DMF scores, this graph illustrates the distributional changes and shows a classic skewed distribution. It is evident that in the more recent survey the proportion of children with low DMF scores had increased, while the proportion with high scores (i.e. severe caries) had decreased. Even so, the shape of the distribution remained much the same: highly skewed toward zero or few DMFS teeth, but with a persistent 'tail', meaning children at the severe end of the scale. It is these children in the tails who are considered to be at high risk, and who thus absorb a lot of attention from public health services. It is generally accepted today that in any population some 60–70% of all carious lesions are found in 15–25% of the children. Whether these children should be targeted for special preventive treatment or not remains a subject for active debate (see Chapters 28 and 29).

Age and gender

Mean DMF scores increase with age. Caries used to be considered just a childhood disease, a perception from those days of high caries severity when most susceptible surfaces were usually affected by adolescence. With younger people now reaching adulthood with many surfaces free of caries,

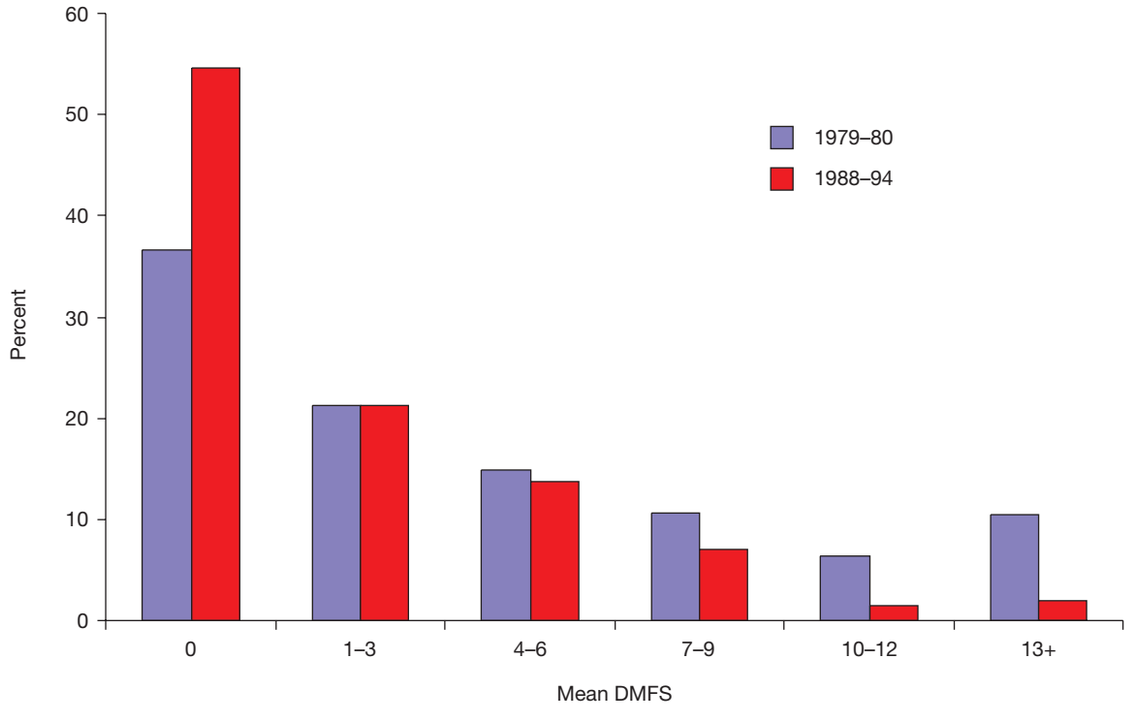


Figure 8.10 Distribution of mean DMFS values in US schoolchildren, aged 5–17 years, in a national survey in 1979–1980 and in another national survey in 1988–1994. (Source: US Public Health Service, National Institute of Dental Research, 1981; US Department of Health and Human Services, National Center for Health Statistics, 1997.)

the carious attack is spread out more throughout life. Adults of all ages can, and do, develop new coronal lesions (Hand *et al.*, 1988; Drake *et al.*, 1994; Luan *et al.*, 2000), and caries has to be viewed as a lifetime disease. Even the skewed disease distribution seen in youth can be seen among the elderly (McGuire *et al.*, 1993). In populations where caries experience is severe the disease starts early in life and is common in the young. A more even occurrence of new lesions throughout life is characteristic of a lower community attack rate.

Root caries

One important offshoot of the age–caries relationship is root caries, defined as caries that begins on cemental root surfaces exposed to the oral environment, and hence when bacterial plaque can accumulate around these exposed roots. As mentioned above, root caries has been with humankind since our earliest days. Even so, awareness of root caries in the high-income countries only grew around the early 1980s with the realization that older adults were keeping more teeth than they used to. Root caries is highly prevalent among older people in high-income countries (Salonen *et al.*, 1989; Beck, 1993; Lo & Schwarz, 1994; Hawkins *et al.*, 1998; Gilbert *et al.*, 2001; Chalmers *et al.*, 2002; Morse *et al.*, 2002), although it too has declined in recent years, just as coronal caries has. Figure 8.11 shows the decline in root lesions in the USA, despite greater tooth retention, between the two most recent national surveillance surveys.

Root caries, by definition, is strongly associated with the loss of periodontal attachment (Locker & Leake, 1993; Slade *et al.*, 1993; Whelton *et al.*, 1993; Lawrence *et al.*, 1995; Ringelberg *et al.*, 1996). Other correlates associated with root caries are primarily socioeconomic: years of education, number of remaining teeth, use of dental services, oral hygiene levels and preventive behavior (Vehkalahti & Paunio, 1988; DePaola *et al.*, 1989; Beck, 1993;). Another important risk factor is the use of multiple medications among the elderly (Kitamura *et al.*, 1986), a common practice in institutions, and one that can promote xerostomia. Xerostomia has long been known as a major risk factor for caries among people of any age, and is particularly prevalent among those who have received radiation treatment for cancer. Other risk factors identified in a representative British sample of people aged 65 or more were poor oral hygiene, wearing partial dentures, sucking candies in a dry mouth and living in an institution (Steele *et al.*, 2001). Root caries is less prevalent in high-fluoride areas than in low-fluoride communities (Burt *et al.*, 1986; Stamm *et al.*, 1990), smokers exhibit more root caries than non-smokers, and prevalence tends to be inversely related to the number of teeth remaining (Beck, 1993; Locker & Leake, 1993).

Root caries seems to be a particular problem among older people of lower socioeconomic status, those who have

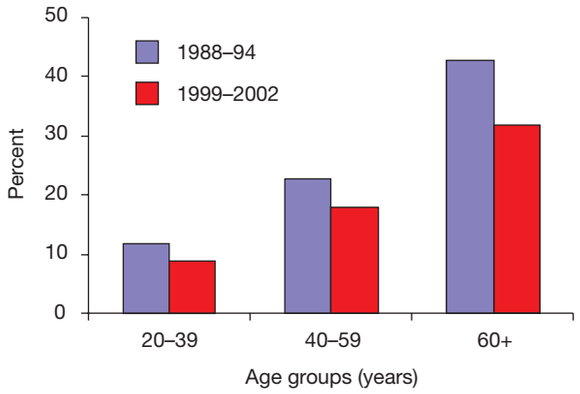


Figure 8.11 Prevalence of carious or restored root lesions in US adults in two national surveys, one in 1988–1994 and the other in 1999–2002. (Source: Beltrán-Aguilar *et al*., 2005.)

lost some teeth, who do not maintain good oral hygiene and do not visit the dentist regularly. Because of our aging populations and increasing retention of teeth, it could be that the dimensions of the root caries problem will continue growing in the years ahead. However, the decline shown in Fig. 8.11 adds strength to the argument that root caries, while not exactly going away, will continue to diminish in the population.

Regarding gender, women usually have higher DMF scores than do men of the same age, although this finding is not universal. When observed in children, the difference has been attributed to the earlier eruption of teeth in girls, but this explanation is hard to support when the differences are seen in older age groups. In those instances a treatment factor is more likely because in national survey data men usually have more untreated decayed surfaces than women, while women have more restored teeth. Women visit the dentist more frequently, so this observation is perhaps to be expected. In a national survey in the USA, girls aged 12–17 years had the same mean number of decayed and missing surfaces as their male counterparts, but 25% more filled surfaces (Kaste *et al*., 1996). One cannot conclude from these figures that women are more susceptible to caries than are men; a combination of earlier tooth eruption plus a treatment factor is a more likely explanation for the observed differences.

Race and ethnicity

Global variations in caries experience result from environmental rather than from inherent racial attributes. To illustrate that point, there is evidence that some racial groups, once thought to be resistant to caries, quickly developed the disease when they migrated to areas with different cultural and dietary patterns (Beal, 1973; Russell, 1966). In the United States, to choose one example of a multiracial and multiethnic society, most surveys before the 1970s found that whites had higher DMF scores than African–Americans, although the latter usually had more decayed teeth. One of the early national surveys in 1960–1962 showed that whites had higher DMF scores than did African–American adults of the same age group, a difference that remained even when the groups were standardized for income and education (US Public Health Service, NCHS, 1967). This difference was still evident in a national survey from the 1970s (US Public Health Service, NCHS, 1981). By the time of the 1988–1994 national survey, however, there was little difference in total DMF scores between whites and African–Americans, although whites still had a higher filled component and lower scores for decayed and missing surfaces. DMF values for Mexican–Americans were in between. This turnaround probably reflects improving access to care for African–Americans, although it still reflects socioeconomic and cultural contrasts between the groups.

Even though overall caries experience in the permanent dentition continues to decline in the USA, disparities between racial and ethnic groups in the prevalence and severity of dental caries still remain in the twenty-first century (Beltrán-Aguilar *et al*., 2005). However, the overall pattern is that there is no evidence to support inherent differences in caries susceptibility between racial and ethnic groups. Far more important are socioeconomic differences and contrasting social environments, meaning differences in education, available income and access to health care. More difficult to measure are long-held cultural beliefs that affect values and behavior related to dental health. However, no one doubts that these factors are present and influencing caries incidence.

Familial and genetic patterns

Familial tendencies ('bad teeth run in families') are seen by many dentists and have been recorded (Klein & Palmer, 1938; Klein & Shimizu, 1945; Ringelberg *et al*., 1974). However, these studies have not identified whether such tendencies have a true genetic basis, or whether they stem from bacterial transmission or continuing familial dietary or behavioral traits. Husband–wife similarities clearly have no genetic origin, and intrafamilial transmission of cariogenic flora, especially from mother to infant, is considered by some to be a primary way for cariogenic bacteria to become established in children (Kohler *et al*., 1983; Caufield *et al*., 2000). The lack of a demonstrable genetic influence by race, discussed above, weakens the case for genetic inheritance of susceptibility or resistance to caries, although it is interesting that Klein, years ago, concluded that the similarities within families involved 'strong familial vectors which very likely have a genetic basis, perhaps sex-linked' (Klein, 1946).

With the explosion of research discoveries of genetic influences in many diseases, dental caries is being looked at in a different light. It is plausible that host attributes that could affect an individual's caries experience, such as salivary flow and composition, tooth morphology and arch

width, are genetically determined, and the genetics of the cariogenic bacteria themselves may have an effect. However, the ready preventability of caries, indicated by the caries decline, strongly counters (if not refutes) the idea of any genetic component worth mentioning. At present, no genetic role in caries experience has been demonstrated.

Socioeconomic status

Socioeconomic status (SES), or social class, is intended to be a broad summary of an individual's attitudes, values and behavior as determined by such factors as education, income, occupation or place of residence. Attitudes toward health are often part of the set of values that follow from an individual's social standing in the community, and can help to explain some of the observed variance in health measures. Obtaining a valid measure of SES, however, is always a problem because of the construct's complexity. The most commonly used measures are income and years of formal education, despite acknowledged shortcomings with the latter measure (Hadden, 1996).

SES is used in research as an attribute specific to the individual, rather than to the community. It is usually inversely related to the occurrence of many diseases, and to characteristics thought to affect health (Marmot & Wilkinson, 1999). The reasons for disparities in health status between various SES strata can often seem obvious, but that is not always the case (Link & Phelan, 1996). For example, higher infant mortality in lower SES strata can be partly explained by the facts that higher SES women have easier access to prenatal care, can better afford such care, and have more time to obtain it, a deeper educational base to permit better understanding of the condition and probably less fatalistic attitudes, and perhaps some other factors. However, even after all these likely variables have been factored into explaining the differences, there is still a considerable gap which defies explanation (Fuchs, 1974). Measurements used in science cannot always pick up all the subtleties embedded in SES.

These subtleties have also been seen in dental health. In one Finnish study, for instance, differences in caries experience between children in the higher and lower social classes still remained after accounting for the reported frequency of tooth brushing, consumption of sugars and ingestion of fluoride tablets (Milen, 1987). These are all individual behaviors that would be more common in higher SES strata. Another instance comes from Sweden, where even with the extensive Swedish welfare system a social gradient in oral health is still evident (Flinck *et al.*, 1999; Kallestal & Wall, 2002).

As part of his landmark research in caries epidemiology during the 1930s and 1940s, Klein observed that overall DMF values did not vary much between SES groups, but aspects of treatment certainly did (Klein & Palmer, 1940). Lower SES groups had higher values for D and M, lower for F. In the first national survey of US children in 1963–1965, white children in the higher SES strata actually had higher DMF scores than did white children in the lower strata, but African–American children showed the opposite profile (US Public Health Service, NCHS, 1971). In the white children, the F component ballooned so much with increasing SES that it lifted the whole DMF index. By contrast, the F component in the African–American children did not change with SES, with the net result that their DMF scores diminished with increasing SES. As mentioned earlier, these results from 1963–1965 showed that a 'treatment effect' was artificially inflating the DMF data in the white children.

In today's lower overall caries experience, however, the position has been reversed. During the period when the caries decline was first recognized, it was soon found that the higher SES groups enjoyed the sharpest decline in caries experience (Graves *et al.*, 1986), so that the DMF values of children in higher SES strata are now generally well below those of children in lower SES strata. This is illustrated in Fig. 8.12, which shows the components of the DMFS index for 15-year-old children in low, medium and high SES groups from a national survey in the USA during 1988–1994. Figure 8.13 illustrates two features of the caries decline. The first, the secular decline in caries, has already been illustrated (Figs 8.6–8.10) and is seen again with the reductions over time in each poverty-status group in Fig. 8.13, showing that, even over this fairly short period of 8–11 years, caries levels across all age and socioeconomic groups have continued to decline. The second aspect illustrated here is how caries levels are related to SES. The children are grouped by poverty status as defined by the US federal government (a socioeconomic measure used to determine eligibility for government programs), and it can be seen that children in the higher SES groups (i.e. those at ⩾200% of

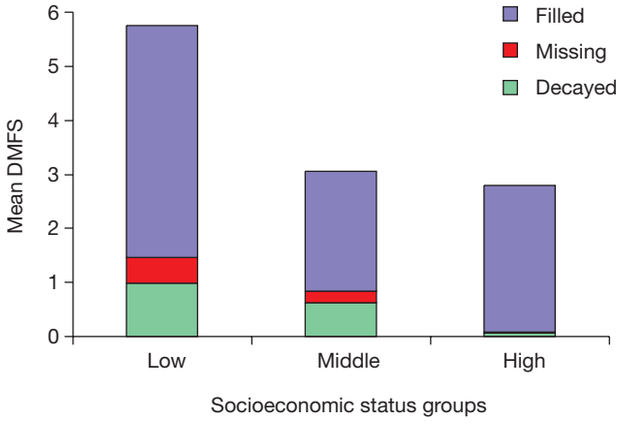


Figure 8.12 Mean DMFS scores for 15-year-old children at three socioeconomic levels in the United States, 1988–1994. (Source: US Department of Health and Human Services, 1997. Reprinted with permission of Elsevier from Burt & Eklünd, *The dentist, dental practice and the community*. Elsevier, Philadelphia, PA; p. 244.)

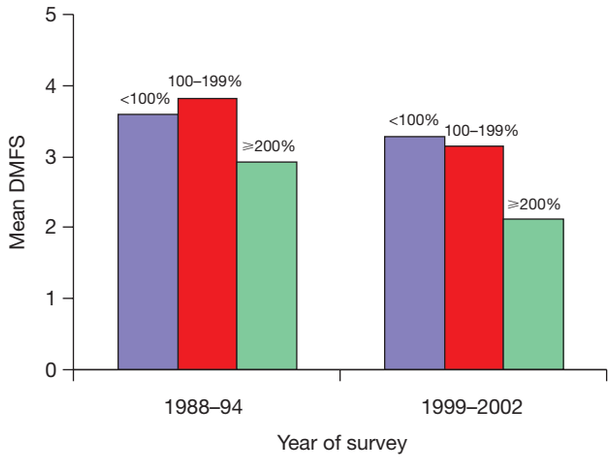


Figure 8.13 Decline in mean DMFS scores in children aged 6–19 years between 1988–1994 and 1999–2002, as seen in three levels of poverty status as defined by the US federal government. (US government's poverty guidelines: http://aspe.cos.dhhs.gov/poverty/06poverty.shtml; accessed 14 March 2006.) The green bars indicate the highest SES groups.

the poverty level) have lower caries scores than do children below the poverty level or only just above it.

As noted above, it is difficult for any one measure to capture all the nuances of SES. British studies have explored the issue by introducing broader measures of SES determinants, such as private housing and car ownership, that go beyond just education and income (Palmer & Pitter, 1988; Carmichael *et al.*, 1989; Gratrix & Holloway, 1994). These measures were all related to caries levels in the population. A better sense of coherence among poor parents of adolescent children, i.e. people who have a more structured life than do others in the same social circumstances, is related to lower caries levels among their children (Freire *et al.*, 2002). This is an intriguing area for further research. Caries levels are also related to the degree of neighborhood deprivation, where several area summary measures have been used (Jones *et al.*, 1997a; Jones & Worthington, 1999; Gray *et al.*, 2000). These broad measures of area deprivation lead directly into the role of social determinants.

Caries and the social environment

A useful definition of public health is '… assuring conditions in which people can be healthy' (IOM, 1988) (with emphasis on the word *can*). That covers everything from maintaining the stratospheric ozone layer to picking up the garbage, to providing recreational facilities, decent housing or dental care where needed. While it stresses the public responsibility for a healthy physical and social environment, it still leaves room for individuals to exercise personal choice in their health-related behavior and how they use health-care services. The term social determinants is related to this definition in that it refers to factors that affect health outcomes for everyone in the community, and whose presence or absence influences the environment 'in which people can be healthy'. Social determinants can include such factors as the quality of housing, extent of community services, availability of transport, prospects for employment, crime levels, and access to parks, open space and suitable recreational facilities.

There are substantial and well-documented differences in health status between people who reside in upscale areas and those who reside in more deprived areas. These contrasts have been documented in a number of countries and in a variety of cultures, although they have been best studied in the high-income countries (Kaplan, 1996). The data show with remarkable consistency that people who live in the more affluent areas are invariably in better health than those from poorer areas, and this observation has endured well over time (Syme & Berkman, 1976; Kaplan *et al.*, 1987; Marmot *et al.*, 1987; Haan *et al.*, 1989). This finding is not dependent on how health status is measured, for it has been documented in terms of overall mortality, heart disease, diabetes or even subjective perceptions of ill-health (Pappas, 1994; Blane, 1995; Kaplan *et al.*, 1996; Link & Phelan, 1996; Goodman, 1999).

While there are numerous individual behavioral determinants involved in this profile (e.g. smoking, use of alcohol, quality of diet, regular exercise), there is also evidence that social determinants, i.e. those risk factors that apply to the whole community rather than to specific individuals, play a key role in determining health outcomes. When any of these basic community necessities are deficient or absent, the quality of life is diminished. When they are mostly deficient, as is not uncommon in rundown parts of major cities, quality of life is diminished to the degree that a high level of emotional stress ensues from the demands of day-to-day coping with the cumulative burden of living in deprived circumstances. Poor social circumstances are linked to disease by way of material, psychosocial and behavioral pathways. Social and environmental disadvantages can lead directly to poor health behavior and the subsequent biological disturbances that lead directly to ill-health. This argument therefore holds that social stresses in themselves can negatively affect health (Brunner & Marmot, 1999). As one example, the gap in both mortality rates and cardiovascular disease levels between Western European countries and those that were formerly part of the Soviet bloc were accentuated sharply around the time of the break-up of the Soviet Union. This phenomenon has been attributed to the high degree of social stress that accompanied the political break-up (Bobak & Marmot, 1996).

Dental research in this area has not developed to the extent it has in medical research, but there is some indication that the same relations between social determinants and oral health are generally evident. Parents' employment status and attitudes have been identified as determinants of the dental health of young children in Belfast (Freeman

et al., 1997) and work stress was related to oral health among workers in Brazil (Marcenes & Sheiham, 1992). British studies, using a variety of measures to characterize the nature and extent of social deprivation in contrasting neighborhoods, have shown how the severity of dental caries is related both to the presence/absence of fluoridated drinking water (Chapter 18) and to the social deprivation level of the residential areas (Jones *et al.*, 1997b, c; Jones & Worthington, 1999; Riley *et al.*, 1999). The effects of social inequalities in youth can be enduring; both low birth weight and social deprivation in youth are related to caries levels at age 13 years (Nicolau *et al.*, 2003, 2005). Contrasts between social inequalities in youth are similarly related to caries severity in adolescence (Pattussi *et al.*, 2001).

These studies collectively demonstrate that dental caries today should be looked upon as a disease of poverty or deprivation as much as being a disease of diet, oral hygiene and other aspects of individual behavior. The great reductions in caries experience among more affluent people in the high-income countries stand in contrast to the much lower reductions in the more socially deprived groups. Further advances in closing these disparities will require sweeping changes in social circumstances as much as changes in individual behavior. Since diet is a prime example of a personal behavior affected by neighborhood characteristics, its relationship to caries deserves some special attention.

Diet and dental caries

Dietary practices are a complex mix of food availability, community and personal affluence, history, culture, marketing practices, and personal affluence, behavior and tastes. Dietary quality is a major determinant of caries levels in a community, and is a major factor in the social environment.

The word diet refers to the food and drink that passes through the mouth, whereas nutrition is concerned with the absorption and metabolism of nutrients from dietary sources. Although a link between malnutrition and caries would be intuitively expected, there is actually little evidence to support it. Studies of children in Peru have concluded that chronic and severe malnutrition during the first year of life is associated with increased caries years later, although this association is difficult to demonstrate because malnutrition delays eruption and exfoliation of the primary teeth (Alvarez, 1995). Chronic malnutrition among children in India has been shown to reduce salivary flow, which could be one reason for a causative link (Johansson *et al.*, 1992).

By contrast, diet has a clear influence on caries development. The relation between the intake of refined carbohydrates, especially sugars, and the prevalence and severity of caries is so strong that sugars are clearly a major etiological factor in the causation of caries. Added sugars are the primary culprit, although a limited degree of caries occurs in populations for whom the only sugars they consume are naturally occurring (Schamshula *et al.*, 1978).

While the evidence is strong that consumption of sugars is a major risk factor for caries, sugars are not the only food sources involved in the carious process. Cooked or milled starches can be broken down to low molecular weight carbohydrates by the salivary enzyme amylase and thus act as a substrate for cariogenic bacteria. It has long been asserted that sugar–starch mixtures are more cariogenic than sugars alone (Bibby, 1975), and there is some animal evidence to support that view (Firestone *et al.*, 1984). The issue may never be totally clarified in humans, but it is reasonable and prudent to view all sugar-containing food and drinks, as well as cooked or milled starches, as potentially cariogenic. By contrast, the large molecular weight carbohydrates in lightly cooked vegetables are considered non-cariogenic because so little breakdown of these foods occurs in the mouth (Lingstrom *et al.*, 2000).

The evolving understanding of diet and caries

The great exploratory voyages of the seventeenth and eighteenth centuries led to the discovery of peoples previously unknown to Europeans, such as the islanders of the South Pacific, who appeared to live an idyllic life free of the diseases that afflicted Europe at the time. The concept of the 'noble savage' (Dubos, 1959) thus developed during the latter part of the eighteenth century. An offshoot from this ideal was the belief that the apparent freedom from caries enjoyed by so-called 'primitive' races could be attributed to the 'natural' diet on which they existed. Eating hard, fibrous and unprocessed food, so the theory went, led to better development of the jaws and teeth and helped to clear food debris from the teeth. By contrast, Europeans were even then eating a lot of processed food, high in fermentable carbohydrates, which was thought to exercise the masticatory apparatus insufficiently and lead eventually to tooth decay. Against the background of these beliefs, Miller, in the late nineteenth century, put forward his chemoparasitic theory of the development of dental caries. Miller's theory, developed during the 'golden age of bacteriology', was based on the action of microorganisms upon fermentable carbohydrates that adhered to the tooth's surface (Miller, 1883). Modern research shows that Miller got the overall picture pretty right.

Theories about the preventive value of hard and fibrous foods became more widespread in the early twentieth century and became established dogma in many places. One such article of faith stated that accumulations of fermentable carbohydrates could be removed by eating hard and fibrous foods (Wallace, 1902), the so-called 'cleansing' or 'detersive' foods. Another view was that if a meal was finished with a salivary stimulant such as an apple, the mouth would be kept free of fermentation both by the physical cleansing effect of the fibrous food and also because of the salivary flow induced by it (Pickerill, 1923). However, 'protective' factors in a diet based on unprocessed foods have proven

hard to identify. High-fiber diets with a good proportion of vegetables and fruit are today recommended by all health authorities, and these dietary guidelines are seen as promoting good oral health as well as general health. Their value, however, is attributable less to the presence of hard and fibrous foods than to the relative absence of fermentable carbohydrates.

In this historical context, the best known attempt to conduct an experimental study on the effect of diet on dental decay in humans was the Vipeholm study, conducted in Sweden between 1945 and 1952 (Gustafsson *et al.*, 1954). The study was conducted in a mental institution (and by today's standards it would be considered unethical because the participants were unable to give their informed consent to potentially harmful exposures). Vipeholm's conclusions profoundly influenced the way in which the role of sugars in dental decay was viewed, despite its complicated and flawed study design. In brief, inmates of the institution were divided into groups with controlled consumption of refined sugars which varied in amount, frequency, physical form, and whether they were taken with or between meals. The extremes of intake were (a) no added sugars at all, to (b) daily between-meal consumption of 24 sticky toffees, each of which was too large to be swallowed and so had to be sucked and chewed. The differences in caries incidence between the groups were pronounced, although some of the Vipeholm conclusions can be challenged in the light of more recent research.

Sugars–caries relationships in today's low-caries environment

Many of the views on diet and caries are carry-overs from the pre-fluoride era, when caries was widespread and severe in high-income countries. In light of modern research protocols, design and analysis can be criticized in virtually all of them: all studies except for Vipeholm were cross-sectional, measurement of dietary intakes was often sloppy, and data analysis usually did not take likely confounders into account. In the period of caries decline, however, researchers have to think that the 'Vipeholm rules' have changed. Are all the children with no detectable caries seen today not consuming sugar, or are other factors having a major influence? Studies such as those concluding that oral hygiene is an important covariable in the sugar–caries relationship (Hausen *et al.*, 1981; Kleemola-Kujala & Rasanen, 1982; Sundin *et al.*, 1992) have served to question the validity of the Vipeholm findings.

Two prospective studies reported in the 1980s, one in Britain and one in the USA, measured diet and caries incidence concurrently and included more analytical detail than did any previous research. The British study followed 405 children with an average initial age of 11.5 years for 2 years (Rugg-Gunn *et al.*, 1984). The children, all from a low-fluoride area near Newcastle, completed five food diaries, each for a 3-day period, for a total of 15 days of recorded diet over the 2 years. Interviews with a dietitian followed each 3-day period to clarify uncertainties and to quantify amounts. The mean DMFS incidence of the group was 3.63 over the 2 years, with 57% of new lesions in pits and fissures, a lower caries increment than the authors had expected. Average consumption of all sugars was 118 g per day, providing 21% of energy intake. The results showed that caries increment was weakly but significantly correlated with total intake of sugars, but poorly correlated with frequency of intake. The authors stated that because of the lower-than-expected caries increment, more clear-cut results would have been likely if the study had been extended for another year.

The second study was based in the low-fluoride area of Coldwater, Michigan, USA. It followed 499 children, initially aged 11–15 years, for 3 years (Burt *et al.*, 1988). The majority completed four 24 h dietary recall interviews with a dietitian, although 27% completed more interviews. The boys in the study averaged 156 g of sugar intake per day from all sources, the girls 127 g, and sugars accounted for 26% of total energy intake. Both of these measures are higher than was found in the British group. Caries incidence, however, was lower than in the British group, averaging 2.9 DMF surfaces over the 3 years, of which 81% were pit-and-fissure lesions (buccal pits and lingual extensions as well as occlusal lesions). Nearly 30% of the group developed no caries at all over the 3 years, and only 51 children (10.2%) developed two or more proximal lesions during the study. Only among the latter 'high-risk' group was caries experience related to total intake of sugars, and that relationship was weak. No relationships between caries experience and frequency of consumption were found. The relative risk of caries from high sugar consumption relative to low sugar consumption was low (Burt & Szpunar, 1994); each additional 5 g of sugars ingested daily was associated with a 1% increase in the probability of developing caries (Szpunar *et al.*, 1995).

Despite some differences in study protocols, findings across these two independent studies were generally similar. Between them, the studies indicated that consumption of sugars is not a major risk factor for many children (i.e. those with no incident caries despite eating a lot of sugar), but it is for those who are still clearly susceptible to caries (broadly defined here as the minority who developed two or more proximal-surface lesions during the study). A systematic review of the present-day caries–sugars relationship (Burt & Pai, 2001) also concluded that the sugar–caries relationship was not as strong as generally supposed. This report identified 36 studies since 1980, all conducted in countries where there is widescale exposure to fluoride, which met the quality criteria for inclusion in the review. Eighteen of these studies found only a weak relationship between sugar consumption and incident caries, 16 found

a moderate relationship and only two identified a strong relationship. The evidence from these more recent studies suggests that sugars still play a modest role as a risk factor for caries, especially among more high-risk children, but that this relationship is by no means linear.

The much stressed role of frequency of consumption of sugars ('it's not how much you eat, it's how often you eat it') is clearly questioned by the results of the Newcastle and Michigan studies, as it has been by other studies in Sweden (Sundin *et al.*, 1983; Bergendal & Hamp, 1985; Stecksen-Blicks *et al.*, 1985). The importance of frequency of consumption was a major finding of the Vipeholm study, and it has been prominent in dental health education ever since. However, the importance of frequency in Vipeholm was principally based on the caries experience of the group which consumed 24 large toffees between meals each day, a frequency of consumption that was not even approached in either the British or the Michigan study. The results from the highly artificial circumstances and non-representative sample of the Vipeholm study thus may be misleading when generalized to the population at large. Even so, frequency and amount of fermentable carbohydrate are related, and this issue has come to light again with recent evidence on the role of soft drinks in caries development.

Caries and soft drinks

Sugar in liquid form is cariogenic; it served well to demonstrate demineralization in landmark experimental caries studies (Von der Fehr *et al.*, 1970). There is more recent evidence to show that soft drink consumption is related to caries: the more often soft drinks are consumed, the greater the extent and severity of caries (Ismail *et al.*, 1984; Jones *et al.*, 1999; Watt *et al.*, 2000; Marshall *et al.*, 2003). Soft drinks have also been implicated as part of the cause of the global epidemic of obesity in children (Mann, 2003), for it is now common to find soft drinks and juices replacing formula and milk in children up to 2 years of age (Marshall *et al.*, 2003). A study among low-income adults found that 54% of their total energy intake came from several types of soft drinks and juices, and that high consumption of soft drinks when linked to poor oral hygiene was associated with higher caries levels (Burt *et al.*, 2006). Soft drinks seem to have replaced confectionery as the prime source of sugar in several populations.

The subject therefore has serious health implications that go beyond dentistry, and is yet another example of a general public health problem having clear dental overtones. Soft drinks thus can be viewed as a 'common risk factor' in public health (Sheiham & Watt, 2000).

Summary

The epidemiology of caries has traditionally been expressed in terms of bad diet, poor oral hygiene, cariogenic bacteria in plaque, 'acid attacks' and demineralization, salivary flow and exposure to fluoride. Those factors and others are all part of how and why caries develops, but this is too narrow a view for full understanding of the disease. In recent years there has been a growing awareness that there is a wider social dimension to caries, just as there is with other diseases. The growth of social and lifecourse epidemiology has shown the importance of the social environment in caries, and how youthful influences can years later affect adult disease. A comprehensive view of caries epidemiology includes all environments, from those at the plaque–enamel interface to the social environment in which a person lives. Caries is a disease of social deprivation, just as it is a disease of bad diet (indeed, those two factors are frequently found together). The key to eventual control of caries thus lies in improving the broad social environment for affected populations just as much as it does in intervening to improve the intraoral environment.

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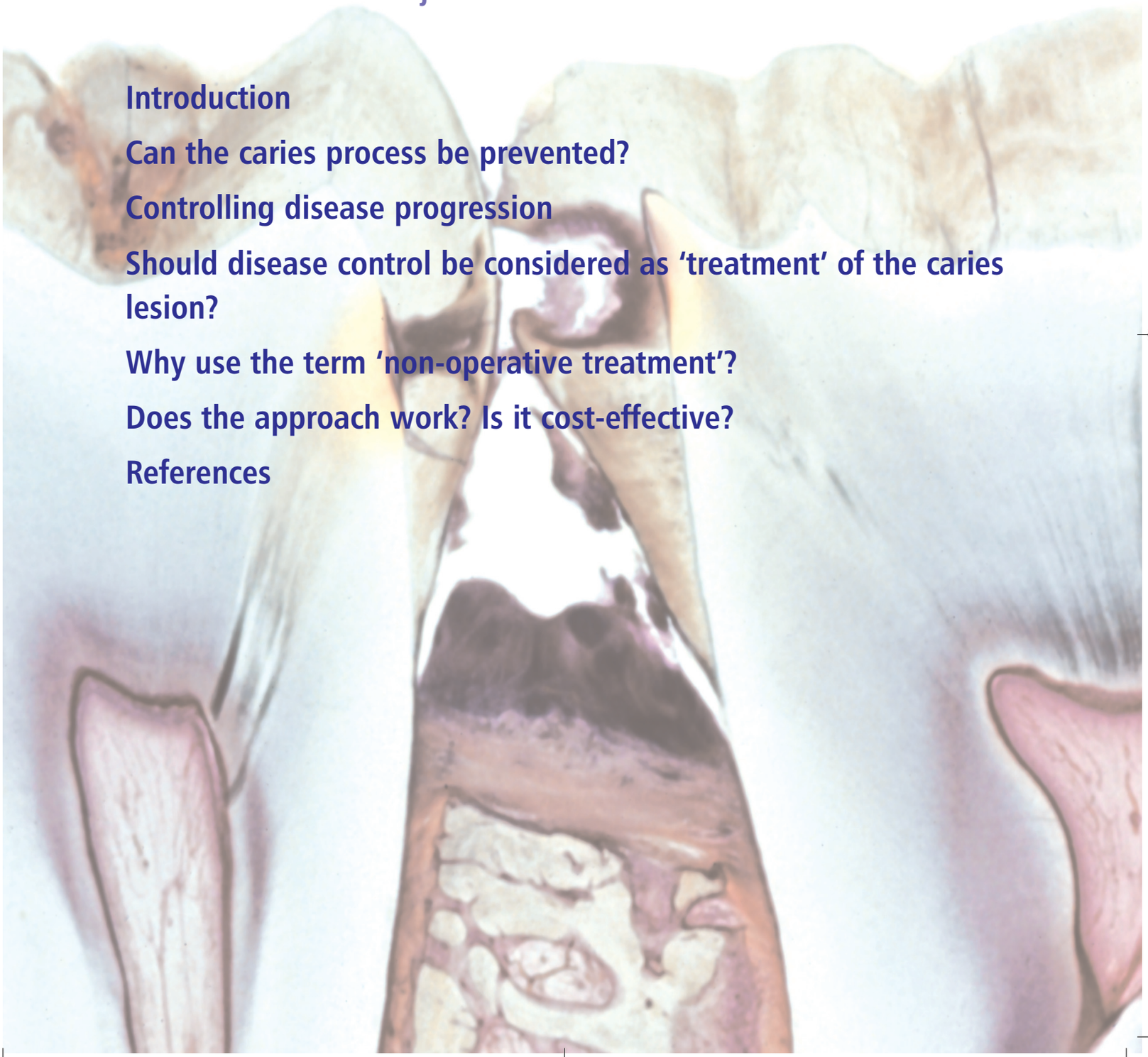
Part IV
Non-operative therapy

14

The control of disease progression: non-operative treatment

E.A.M. Kidd and O. Fejerskov



Introduction

From the previous chapters it will be apparent that although dental caries is considered an infectious dental disease, it should not be considered a result of an infection with one specific type of microorganism (once thought to be *Lactobacillus acidophilus* and later, and still by some, thought to be *Streptococcus mutans*). As presented in Chapter 10, the infectious agents are the indigenous flora of the oral cavity. Although there are some 500 different species present in the oral environment, they only present a possible insult to the dental hard tissues if they are allowed to form a biofilm on the tooth surfaces, i.e. dental plaque.

The bacterial metabolism in the microbial deposits that constitute the plaque biomass results in fluctuations in pH. These fluctuations influence the dynamic equilibrium between the mineral in the tooth and the degree of saturation in the plaque fluid with respect to the apatites. This causes dissolution and redistribution of mineral in the underlying dental hard tissues (see Chapter 12). A range of factors determines the extent to which and the rate at which the metabolic events may result in a net loss of mineral (development or progression of a lesion). These include the composition of the bacterial biofilm, the composition and flow rate of saliva, the presence of fermentable carbohydrates, and the concentrations of fluoride, calcium and phosphate in the oral fluids (Fejerskov & Manji, 1990) (see schematic illustration in Chapter 1, Fig. 1.1).

The interplay of these factors affects what happens in the interface between the biomass and the tooth surface, and therefore mineral dissolution and reprecipitation are dynamic processes, as described in Chapters 2, 3 and 12. If the caries challenge is high (the rate of dissolution exceeds the rate of redeposition) the clinical appearances will be different from those observed where the local environment favors redeposition of minerals. The rate of progression of the lesion reflects the activity of the biofilm. This means that a clinician can gauge the net activity of the biofilm by looking at the surface features of the caries lesion that is beneath it. Lesions may appear 'active' or 'arrested', as described in Chapter 2.

Can the caries process be prevented?

The formation of a biofilm on a tooth surface cannot be prevented in surface irregularities such as occlusal fissures, or in the gingival or approximal niches. In these areas occlusal function or attrition (friction) from cheeks, lips and tongue does not occur. All bacterial deposits, irrespective of their stage of maturation, are metabolically active. These metabolic activities will affect the tooth surface beneath and a plaque that is only a few days old will produce a classical Stephan response to sugar. If, over time, such regular pH fluctuations are able to result in a net loss of mineral, then the caries process results in a detectable lesion. The formation of cavities can be prevented by controlling the caries process, but metabolic fluctuations in the biofilm cannot be prevented. Thus, caries is a ubiquitous, natural process (Manji *et al*., 1991a; Fejerskov, 1997).

A tooth surface covered over long periods by a metabolically active biofilm will gradually be chemically modified. For instance, the biological apatite in newly erupted enamel contains a variety of impurities such as carbonate and magnesium which make the apatite more soluble (see Chapter 12). If chemical conditions with fluctuations in pH prevail, a gradual loss of magnesium and carbonate in surface enamel should be expected. This chemical change in surface enamel is therefore to be considered the earliest sign of the caries process. If, however, fluoride is available during this process, the fluoride will become incorporated in the biological apatite over time, to such an extent that the fluoride content in the clinically 'normal' cervical enamel surface (where plaque accumulates) increases significantly over a period of 6–7 years (Richards *et al*., 1977; Fig. 18.10). All this can happen without any clinically recordable changes in porosity or, for that matter, changes assessed by different microscopic methods. Thus, the metabolically active biofilm results in a permanent chemical modification of the tooth surface. As explained in Chapter 3, the process known as posteruptive enamel maturation may be considered a part of the caries process at a subclinical level.

Thus, accepting that biofilms constantly form and grow on any tooth surface, these regular demineralizations and remineralizations, which occur at random (Manji *et al*., 1991b), cannot be prevented, because they are a ubiquitous and natural process. Their effect on tooth surfaces over time can, however, be influenced and the metabolic processes can be modified. Thus, caries lesion development and progression can be controlled. Therefore, by controlling the metabolism in the microbial biomass, it is possible to prevent cavities from occurring. An already formed subsurface lesion, presenting itself as a thin, white opaque line or the extensive white-spot lesion, does not have to progress. Any lesion, at any stage of tissue destruction, noncavitated or cavitated, can become arrested. This statement is true irrespective of the age of the patient.

Controlling disease progression

Disease control concerns influencing biofilm formation and growth, or modifying the dissolution kinetics of the apatites, or both. The following may have a role to play:

- mechanical/chemical removal of plaque (oral hygiene)
- chemical (antimicrobial) modification of plaque
- use of fluorides
- dietary composition
- salivary composition and stimulation.

Each of these topics is discussed in this text, but it is salutary to realize that most of these measures depend on a degree of patient co-operation, and behavioral considerations are a very important part of this. Potentially, any non-medicated and physically normal individual can learn to control lesion development and progression. As such, dental caries, progressing to the stage of frank cavities in teeth, can be prevented in the majority of any population, and in most patients white-spot lesions may also be prevented or their appearance modified.

It is important to realize that on an individual patient basis there is great variation in the complex interplay between all known and unknown determinants involved in caries lesion development. Therefore, an assessment of the individual patient's relative risk is an important prerequisite to planning appropriate treatment.

Should disease control be considered as 'treatment' of the caries lesion?

Disease control measures, such as showing a patient how to remove plaque, applying topical fluoride and discussing their diet and whether it needs modification, have classically been described as 'prevention'. As already discussed, this may not be an accurate term because the caries process as such is not prevented; rather, the likely outcome (a lesion with a cavity) can be prevented or most often postponed (Haugejorden *et al.*, 1990). Unfortunately, 'preventive' on the one hand has been contrasted with 'treatment' on the other, with 'treatment' implying operative intervention. Sadly, operative intervention is seen by many patients, dentists and politicians as the way to manage the process. This is despite the fact that once a tooth has been subject to an operative treatment procedure, the likelihood of losing the tooth with age is higher than for a sound tooth and, in certain populations, may be as high as having a non-treated caries lesion (Luan *et al.*, 2000).

Many patients do not understand, or do not believe, that they will not necessarily need fillings. Some expect this management when they visit the dentist and may not question what can be done to prevent fillings being needed in the future. Moreover, fillings were considered by the profession as 'secondary prevention', adding to the belief that inserting a filling would not only restore form and function, but also prevent further damage. However, even the best performed restorative procedure does not result in a lifelong restoration (see Chapter 24). The dental profession is by tradition focussed on pain relief by the extraction or restoration of severely damaged teeth. For years dentists have been paid for filling teeth but sometimes not paid, or very poorly remunerated, for taking measures to prevent disease progression. Even in public dental services the efficiency of the dental staff has been measured in numbers of fillings inserted (or fissure sealants placed in recent years).

There are several examples of attitudes that may have to be reconsidered to render dentists more cost-effective. For instance, patient education in measures that prevent disease progression is often delegated to another member of the dental team, perhaps a dental hygienist. This individual has received a shorter training and has a lower salary than the dentist. Does this mean that this treatment is easier to perform or less important than operative treatment? What about turning the present situation on its head and training ancillary personnel to place fillings, leaving the dentist with diagnosis, treatment planning, decisions on whether to perform non-operative treatments and conventional filling, and patient education?

As another example, for years dental epidemiologists have recorded decayed teeth, in the DMFT/S recording, as being a reflection of 'the disease' (Pitts, 1997). Thus, decayed is considered synonymous with an 'untreated' cavity (i.e. a cavity in need of a filling). Moreover, some public health dentists have been content to tell their political masters that a certain percentage of their child population is 'caries free'. These figures are produced on the basis of the most peremptory clinical examination at the level of cavitation or caries into dentin. The politicians seize on the good news that caries prevalence and incidence are falling in young people and they erroneously believe that the disease has been eradicated. The fact that this is delayed disease progression (Haugejorden *et al.*, 1990) and that the disease may present at the cavitation level in older populations unless controlled, has not been made sufficiently clear.

In all fairness, the dental schools should also bear some responsibility for the current attitude to restorative dentistry. The school may fuel the 'treatment equals fillings' philosophy by giving points for restorative procedures but not rewarding non-operative treatments. A school has a responsibility to train competent operators and the amount of experience each student has must be recorded; however, a points system that only rewards operative procedures may engender the attitude that it is 'filling holes in teeth' that counts in monetary terms. Efficient cutting automatons may be hatched from the dental school egg confident of their own ability to treat caries lesions by restorations. This is an important message: restorations have a role to play in managing some caries lesions, and this role will be discussed in Chapter 20, but operative dentistry must go hand in hand with non-operative treatment to control further disease progression, otherwise restorations may not survive. To ignore non-operative treatment would be biologically illogical and ethically unacceptable. Disease control should be seen as 'treatment' of ongoing caries processes and hence of the lesions at different stages of development.

Why use the term 'non-operative treatment'?

The perceptive reader will have noticed the term 'non-operative treatment' creep into this chapter. It is used to encompass all those measures that attempt to control disease progression. The aim of this is to set non-operative treatment alongside operative treatment, implying that both 'treat' the disease process and both are time-consuming, skillful and worthy of payment.

Does the approach work? Is it cost-effective?

At this point it would be reassuring to direct the reader to a series of practice-based, randomized control trials showing the value of non-operative treatment in terms of reduced caries incidence. Unfortunately, this work is not particularly positive. These studies have targeted preventive efforts at individuals judged to be a high risk. Work focussing on advising preschool mothers in tooth brushing with fluoride toothpaste and sugar control achieved an 18% difference in the mean DMFT between the test and control groups. However, this was not a statistically significant difference, despite the test group having 4-monthly counselling over 2 years compared with a single session for the control group (Blinkhorn *et al.*, 2003).

Two Finnish studies (Seppä *et al.*, 1991; Hausen *et al.*, 2000) targeted 'high-risk' adolescents with intensified non-operative treatments, with modest results. Recent work from Sweden (Kallestal, 2005) also concentrated on 'high-risk' adolescents over 5 years and could find no significant differences in mean 5-year increment among four different programs. These were:

- information on tooth-brushing techniques
- prescription of fluoride lozenges
- semi-annual application of fluoride varnish
- quarterly appointments for oral hygiene and diet instruction and fluoride varnish application.

The results showed all programs to have a rather low efficacy.

Perhaps it is relevant that none of these studies had a true control group that received no advice on oral care. Inclusion of such a group would be unethical, but it would also probably be impossible in countries where all dentists are giving basic preventive advice. The studies are important because they appear to show that preventive interventions targeted only at 'high-risk' individuals may not be cost-effective, and this topic will be raised again in Chapters 28 and 29. Perhaps it is patient compliance that is the all-important factor, or maybe there is a ceiling through which further preventive effort cannot break. It must be remembered that there are no tests with sufficient predictive power to identify individuals at 'high risk' (Chapter 29). However, recent work from a child population with an overall low caries experience (Hausen *et al.*, 2007), targeting caries control at all children in an area with an active lesion, recorded a significantly reduced increment in dental decay. Indeed the prevented fraction was similar to studies on non-operative caries management conducted in areas of high or moderate levels of dental caries (Ekstrand *et al.*, 2000; Curnow *et al.*, 2002).

An attempt has been made to carry out an economic evaluation of preventive programs (Kallestal *et al.*, 2003). Five studies were considered worthy of inclusion, but the authors were frustrated in their attempt to draw a conclusion because the studies gave contradictory results, some negative, some positive.

It has also been pointed out (Seppä, 2001) that there will be relevant intercountry differences. In countries with a high caries rate, a low level of basic prevention and an unorganized dental care system, any preventive program seems effective, whereas in other countries, often described as economically developed, the effectiveness of preventive programs seems to have diminished. One aspect of prevention that seems indisputable is the importance of fluoride toothpaste as a cost-effective and feasible method of fluoride delivery and this will hold good in all countries, irrespective of the caries level and dental care systems.

The other side of the coin is concentrating on reparative treatment, and experiences from New Zealand are a cautionary tale indicating how wrong it may be to take this course (Hunter, 1998). Despite a comprehensive child and adolescent restorative program in New Zealand, there was a high rate of tooth extraction in adults. Teenagers had little untreated dental decay but many fillings. It is even tempting to think, with today's knowledge, that the high extraction rate was a direct result of the restorative program! When a policy change discouraging early operative intervention was made in the late 1970s, the rapid decline in DMFT was the result of the F component (and hence the M component) being dramatically reduced. In other words, the 'disease pattern' was partly a reflection of the treatment orientated concept. Sheiham (1997) discussed this concept and also pointed out that the literature indicates that dental care has a small impact on the incidence of dental caries in children, with social factors being more important.

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