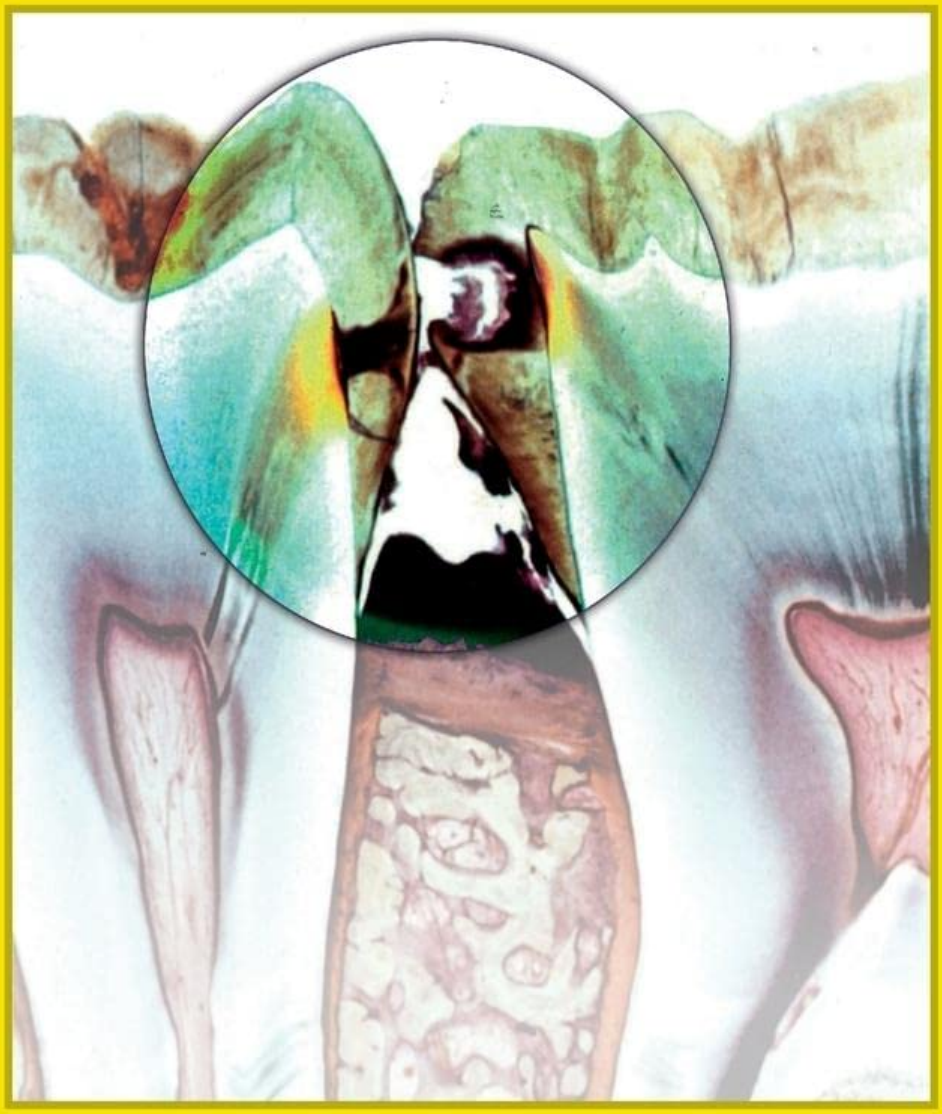
Dental Caries

The Disease and Its Clinical Management

Third Edition



Edited by
Ole Fejerskov, Bente Nyvad and Edwina Kidd

WILEY Blackwell

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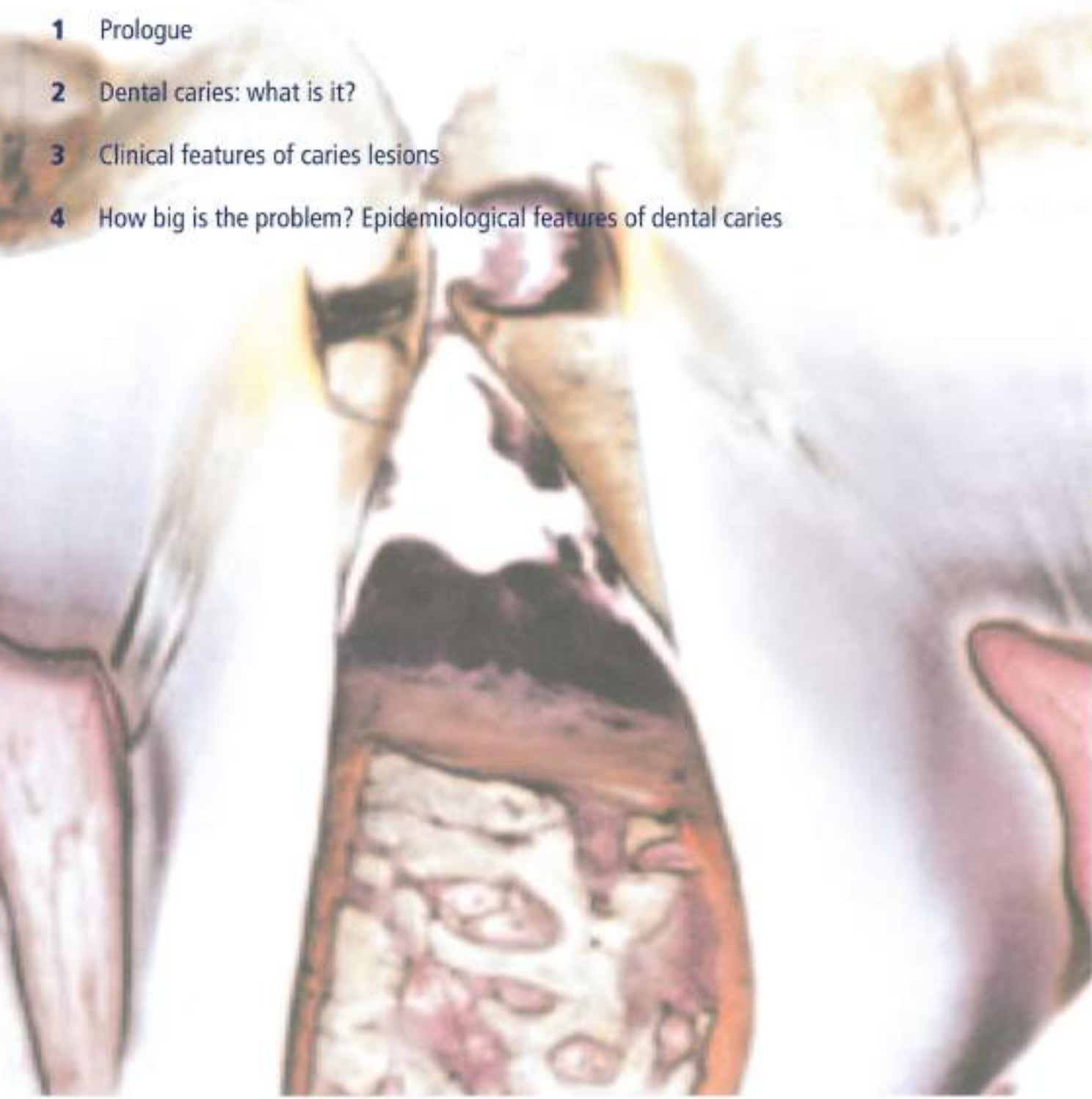
Part I
Dental caries: what is it and how widespread is it globally?



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Prologue

O. Fejerskov, B. Nyvad, and E.A.M. Kidd

Introduction

Dental caries is ubiquitous – it is omnipresent in all populations and is as old as mankind. The caries incidence rate varies extensively between and within populations. With increasing age, signs and symptoms of dental caries accumulate, and in most adult populations the caries prevalence approaches 100%. Prevention and operative treatment of caries lesions and its sequelae occupy the majority of the dental profession lifelong around the world, and the cost of dental health care is a major societal burden.

The majority of dental restorations are made because of dental caries. Caries and failed restorative care are the main causes for tooth loss in all contemporary populations. So, it is hopefully obvious that there are good reasons for advancing an international textbook on *Dental Caries: The Disease and Its Clinical Management*.

It has been a great pleasure for us that the first two editions of this book have found their way all over the world. In the genesis of this third edition, we have realized that Edwina Kidd has been emerita professor for several years and soon Ole Fejerskov will follow, so it is time for a new generation to take over. Therefore, Professor Bente Nyvad is now a full member of the editorial team.

From the first to the second edition, the book 'put on weight,' becoming much more extensive. However, we have no wish for it to become 'morbidly obese' by continuing to add to the length of text and topics. Rather, we have aimed in this third edition to make a 'slimmer' volume that hopefully makes it more readable for the main target: dental students and practitioners in public health and dental practice. We have invited 34 international colleagues to join us, many of whom are new, to ensure both continuity and novelty in lines of thinking throughout the book.

The role of cariology in restorative dentistry

The content of the book reflects our wish to make the basic knowledge about the bio-physiology of the oral cavity and dental caries applicable in daily clinical practice. When G.V. Black published his comprehensive textbook in 1908 he emphasized that clinical diagnosis and treatment decisions should have a sound biological rationale. Although it became appreciated by the middle of the 20th century that dentistry is a biomedical speciality, the technical advances with high-speed drilling and so on distorted the true application of biological knowledge in optimal treatment of dental caries.

Dental caries became synonymous with 'a cavity' in the tooth – and the automatic reaction was that the treatment should be 'drill and fill.' In the growing field of caries epidemiology, dental caries was recorded as DMF teeth/surfaces, where D stood for decay and decay meant a cavity. The knowledge about etiology and pathogenesis of caries was

Dental Caries: The Disease and Its Clinical Management, Third Edition. Edited by Ole Fejerskov, Bente Nyvad, and Edwina Kidd.

often taught in dental curricula in departments of microbiology, pathology, and physiology, as well as in the growing disciplines of the 1950s and 1960s of dental public health and departments of pediatrics and preventive dentistry – but the clinical relevance of 'cariology' to restorative clinical departments was minimal, and knowledge to be applied at the chair-side fragmented. To some extent this was understandable, because in a clinical department the students were supposed to produce fillings, and crowns and bridges. *So the appreciation of the need for a concomitant disease control as part of any long-term successful restorative treatment was limited.*

The Keyes so-called triad stressed the components as being: (1) the tooth, (2) the diet, and (3) the microflora.

1. **The tooth.** In the last half of the 20th century much focus in caries research was on improving the 'resistance' of the tooth. Logically, there was an enormous interest in the role of fluoride in the prevention and control of dental caries. There were many attempts to introduce artificial water fluoridation worldwide in order to repeat the very impressive caries reduction that Trendley Dean and collaborators had documented in the USA. However, it proved difficult in many populations to introduce systemic fluoride programs, and by 1980 it became evident that the mechanism of action of fluoride in caries control was not a result of improving enamel resistance. Thus, the topical use of fluoride started to play a key role, particularly fluoride added to toothpaste.
2. **The diet.** The role of sugar in caries development had become evident by the middle of the 20th century,and much effort was invested in trying to reduce the intake, not least in children – with limited success. Total sugar consumption per capita stayed fairly constant and sugar substitutes were introduced. Then the dramatic caries decline became well documented, in particular in the Nordic countries, apparently without a significant reduction in total sugar consumption. So the relative role of diet was reconsidered.
3. **The microflora.** It was well known that 'a clean tooth never decays' – but the consequence of this statement was seldom fully appreciated. When rodent experiments around 1960 clearly indicated that dental caries was 'an infectious and transmittable disease,' extensive research focused on identifying one responsible microorganism: the caries pathogen. In the past, *Lactobacillus acidophilus* had been seen as the main bacterium causing caries, but the focus shifted towards mutans streptococci. This even led to attempts to introduce a caries vaccine against *Stretococcus mutans*! Extensive research was carried out without appreciating that there is a significant difference between the behavior of bacteria in a planktonic (free-floating) phase and the concept of oral ecology, where the single microorganism is part of a complex oral microflora composed of more than 1000 different species. This was the focus around the turn of the century, where, gradually, dental plaque came to be considered an oral biofilm and dental caries seen as a biofilm-induced demineralization of the dental hard tissues.

But even at this stage many questioned the role of tooth cleaning in caries control!

This somewhat simplified overview of the major trends in dental caries research over the last 50 years is important because it will hopefully help new readers to understand how the very different concepts and 'paradigms' of today are influenced by historical tradition. The way in which scientific literature is selected and interpreted and its results introduced into diagnosis, prognostic assessments, treatments decisions, prevention, and public health strategies will profoundly influence how successful the dental profession will be in controlling dental caries and maintaining a functioning dentition in every patient from cradle to grave.

The content of this textbook

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 carefully present their respective subtopics 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.

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 nonoperative 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 deterministic model that could relate all the potential determinants perfectly 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, and
- 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.

The *content* of the book is organized according to our wish to link theory with clinical performance. In other words, making prevention, diagnosis, and restorative procedures evidence based.

In Chapters 2–4 we define what dental caries is and how it most often manifests itself on different tooth surfaces. Then we ask the question 'How big is the caries problem in different parts of the world?' and present basic epidemiological tools.

Part II, 'The caries lesion and it biological determinants' (Chapters 5–9), basically covers the aspects of the Keyes triad. Then this knowledge is applied in Part III, 'Diagnosis' (Chapters 10–12), and Part IV, 'Controlling dental caries' (Chapters13–18). When caries lesions require 'Operative intervention' (Part V, Chapters 19–21), biological knowledge is essential to provide the most careful intervention and not to make unnecessary replacement of restorations. Finally, Part VI, 'From chair-side to population caries control' (Chapters 22–25), brings the principles of dealing with the single tooth surface or the individual patient forward by approaching caries control in whole populations of different kinds. Moreover, this part addresses the very important questions about risk assessment versus prediction. In the final chapter we ask the question: If we were to apply the knowledge we have today on how to control dental caries most cost-effectively in various parts of the world and maintain a functional dentition lifelong, what recommendations would we then formulate?

We hope you will enjoy the book – and interact with us, whether agreeing or disagreeing. As Charles Darwin said: 'All observations must be for or against some view to be of any service.'

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Dental caries: what is it?

O. Fejerskov, B. Nyvad, and E.A.M. Kidd

The disease

The term *dental caries* is used to describe the results – the signs and symptoms – of a localized chemical dissolution of the tooth surface caused by metabolic events taking place in the biofilm (dental plaque) covering the affected area. The destruction can affect enamel, dentin and cementum. The lesions may manifest themselves clinically in a variety of ways, as will be dealt with in Chapter 3.

In principle, dental caries lesions may develop at any tooth site in the oral cavity where a biofilm develops and remains for a period of time. It is therefore a misconception to talk about more- or less-susceptible surfaces as this may erroneously give rise to the belief that certain parts of a tooth are more 'resistant' or 'less susceptible' to developing caries lesions due to variations in the chemical and structural composition [1, 3].

This is not to say that all tooth surfaces within the oral cavity of an individual develop caries lesions at the same rate. Dental caries lesions develop at relatively 'protected sites' in the dentition where biofilms (dental plaque) are allowed to accumulate and mature over time. Such sites include pits, grooves, and fissures in occlusal surfaces, especially during eruption, approximal surfaces cervical to the contact point/area, and along the gingival margin. Obviously, insertion of foreign bodies to the dentition (e.g., fillings with inappropriate margins, dentures, orthodontic bands) may also result in such 'protected' sites. These areas are relatively protected from mechanical influence from the tongue, the cheeks, abrasive foods, and, not least, tooth-brushing. Thus, these are the sites where lesion development is more likely to occur because the biofilm is allowed to stagnate there for prolonged periods of time.

This knowledge is very important, and it is 100 years ago that Black [1] stated:

> *...the beginning of caries of the teeth occurs at such points as will favour such lodgement or attachment in which the microorganisms will not be subject to such frequent dislodgement as would prevent a fairly continuous growth. This is the cause of the localisation of the beginnings of caries on particular parts of the surface of the tooth.*

Dental caries lesions, furthermore, do not develop at the same rate in all parts of the mouth. Thus, openings of the major salivary glands represent areas with a special salivary composition that favors a relative protection towards chemical dissolution because of buffering capacity and chemical composition of the secretory product (see Chapter 6).

Dental caries lesions result from a shift in the ecology and metabolic activity of the biofilm (Chapter 7) whereby an imbalance in the equilibrium between tooth mineral and biofilm fluid has developed. It is important to appreciate that an oral biofilm, which forms and grows ubiquitously on solid surfaces in the oral cavity, does not

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necessarily result in the development of *clinically visible* caries lesions when grown on dental hard tissues. Thus, a biofilm grown on tooth enamel inserted in palatal devices (so-called in-situ models) has to be 'protected' from abrasion from the tongue movement. Similarly, in-situ models for studying caries lesion development under controlled conditions have preferably to be located so that microbial stagnation areas are created. But the biofilm is a prerequisite for caries lesions to occur. The biofilm is characterized by continued microbial activity, resulting in metabolic events in the form of continuous, minute pH fluctuations. The metabolism may be dramatically enhanced by changing the nutritional conditions (e.g., by adding fermentable carbohydrates) and the outcome of the metabolism can be recorded as pH fluctuations. Any shift in pH will influence the chemical composition of the biofilm fluid and the relative degree of saturation of this fluid with respect to the minerals that are important for maintaining the chemical composition of the tooth surface (see Chapter 9). From the very moment of eruption into the oral cavity, the tooth surface apatite will continue to be subject to these chemical modifications on innumerable occasions. Most of these modifications are so subtle that they can only be recorded at the nano-level. Surfaces that are frequently covered by biofilm (e.g., a cervical enamel surface) will gradually accumulate fluoride in the very surface layers (outermost 100μm) (see Chapter 9, Fig. 9.11). Thus, the enamel surface is in a state of dynamic equilibrium with its surrounding environment. *When the cumulative result of the numerous pH fluctuations over months or years is a net loss of calcium and phosphate of an extent that makes the enamel sufficiently porous to be seen in the clinic, we may diagnose it as 'a white spot' lesion* (see Chapters 3 and 5). It is important to appreciate, however, that, although the metabolic events may result in detectable caries lesion formation, most sequences of metabolic events tend to cancel each other, which is why the metabolic events should be considered intrinsic to biofilm physiology (Chapter 7). The caries lesions arise when there is a drift in the metabolic events; that is, when the pH drops result in a net loss of mineral. Thereby, *the dental caries lesions are a result of an imbalance in physiologic equilibrium between tooth mineral and biofilm fluid.*

These considerations lead to some important points.

- The dissolution (demineralization) when pH drops below a certain level in the biofilm and the redeposition (remineralization) of minerals when pH goes up (see Chapter 9) takes place in the enamel surface at the interface between the biofilm and the tooth surface. These processes occur numerous times during a day and can be modified extensively. If, for example, the biofilm is partly or totally removed, mineral loss may be arrested or even reversed towards mineral gain because saliva is supersaturated with respect to the enamel apatite. This will result in arrest of disease progression – and may even result in some redeposition of minerals in the very surface of the tooth.
- Any factor that influences the metabolic processes, such as the composition (e.g., content of buffering proteins) and thickness of the biofilm, the salivary secretion rate and composition (Chapter 6), the diet (Chapter 8), and the fluoride ion concentration in the oral fluids (Chapters 9 and 13), will contribute to the likelihood of a net loss of mineral – and the *rate* at which this occurs. Figure 2.1 indicates how the many biological determinants of the caries process may act at the level of the individual tooth surface (inner circle). At the individual/population level (outer circle) the behavior, education, knowledge, and attitudes will have a strong influence on some of the biological determinants (quality of oral hygiene, choice of foods, use of fluorides, salivary flow from chewing gum, etc.)
- At any given point in time the net mineral loss or gain is part of a continuous spectrum of events. The absence of a clinically detectable caries lesion does not necessarily

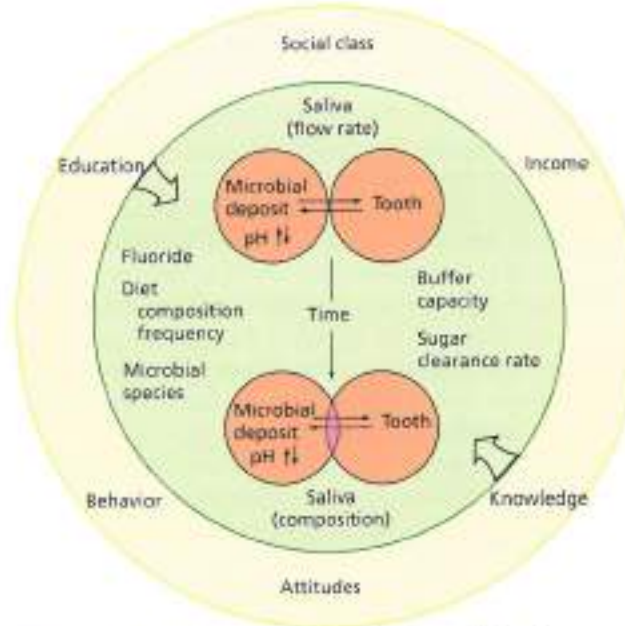

Figure 2.1 Schematic illustration of the determinants of the carious process. Those that act at the tooth surface level are found in the inner circle. With time, an ecological shift in the composition and metabolic activity of the biofilm ('microbial deposit') may result in an imbalance in the equilibrium between biofilm fluid and the mineral of the tooth. Thus, a net loss of mineral results in formation of a caries lesion (overlap of the two small circles). In the outer ring are listed more distant determinants that influence these processes at the individual and population levels. Adapted from [2]. Reproduced with permission of the University of North Carolina School of Dentistry.

mean that no mineral loss has occurred (Chapter 5); it only means that it could not be discerned clinically. If this concept of a continuum is appreciated it will immediately be understood why diagnosis of various stages of lesion progression is a question of defining certain 'cut-off' points (Chapter 4).

Terminology

Caries lesions may be classified in a number of ways. Unless the student is familiar with this terminology it can be difficult to understand what is written. This section introduces and defines various terms that will trip off the writers' pens in subsequent chapters.

Carious lesions can be classified according to their *anatomical site*. Remember, there is nothing chemically special about these sites. Thus, lesions may commonly be found in *pits and fissures* or on *smooth surfaces*. Smooth surface lesions may start on enamel (*enamel caries*) or on the exposed root cementum and dentin (*root caries*).

Primary caries is used to differentiate lesions on natural, intact tooth surfaces from those that develop adjacent to a filling, which are commonly referred to as *recurrent or secondary caries*. These two latter terms are synonyms, but in this textbook we will use the term *recurrent caries* throughout. Recurrent caries is simply a lesion developing at a tooth surface adjacent to a filling. As such, its etiology is similar to primary caries.

Residual caries (as the term implies) is demineralized tissue that has been left behind before a filling is placed.

An important classification is whether a lesion is *cavitated* or *noncavitated*. *A cavity is a physical hole in the tooth*, and it may impinge directly on the management of the lesion (Chapters 16 and 18).

Caries lesions may also be classified according to their activity. This is a very important concept and one that impinges directly on management, although it will be evident from the text that the clinical distinction between *active* and *inactive* (arrested) lesions is sometimes difficult.

A lesion considered to be progressing (you anticipate that the lesion would have developed further at a subsequent examination if not interfered with) would be described as an *active caries lesion*. This distinction is based on a judgment of the features of the lesion in question in combination with an assessment of the oral health status of the patient. In contrast to this is a lesion that may have formed years previously and then stopped further progression. Such lesions are referred to as *arrested caries lesions* or *inactive caries* lesions.

You may also meet the terms *remineralized* or *chronic lesions* used to signify arrested lesion; but, as you will appreciate later, the term remineralization should be used with caution (Chapters 5 and 9). The distinction between active and inactive/arrested lesions may not be totally straightforward. Thus, there will be a continuum of transient changes from active to inactive/arrested and vice versa. A lesion (or occasionally part of a lesion!) may be rapidly progressing, slowly progressing, or not progressing at all. This will be entirely dependent on the ecological balance in the biofilm covering the site and the environmental challenge. Clinically, if in doubt, the dentist should always react as if they are dealing with an active lesion.

Despite the diagnostic difficulties, these distinctions are very important to the clinician because if a lesion is not active then no action is needed to control further progression. If, on the other hand, a lesion is considered active, steps should be taken to influence the metabolic activities and possible the ecological balance in the biofilm in favor of arrest rather than further demineralization.

At this point it is also sensible to discuss a possible confusion in terminology. The first sign of a carious lesion on enamel that can be detected with the naked eye is often called a *white spot lesion*. This appearance has also been described as an early, *initial* or *incipient lesion*. These terms are meant to say something about the stage of lesion development. However, a white spot lesion may have been present for many years in an arrested state, and to describe such a lesion as early would be inaccurate. A dictionary definition of incipient is 'beginning'; an initial stage. In other words, an initial lesion appears as a white, opaque change (a white spot) – but any white spot lesion is not incipient!

Rampant caries is the name given to multiple active carious lesions occurring in the same patient. This frequently involves surfaces of teeth that do not usually experience dental caries. These patients with rampant caries can be classified according to the assumed causality; for example, *bottle or nursing caries*, *early childhood caries* (ECC) when observed in children, and *bakers' caries*, *radiation caries*, and *drug-induced caries* when seen in adults. ECC is simply caries on teeth that are not clean, exposed to carbohydrates, and located in an area of the mouth where oral clearance is low (for details, see Chapter 16).

Hidden caries is a term used to describe lesions in dentin that are missed on a visual examination but are large enough and demineralized enough to be detected radiographically. It should be noted that whether a lesion is actually hidden from vision depends on how carefully the area has been cleaned and dried and whether an appropriate clinical examination has been performed.

Background literature

Baelum V, Fejerskov O. Caries diagnosis: 'a mental resting place on the way to intervention'? In: Fejerskov O, Kidd EAM, eds. *Dental caries. The disease and its clinical management*, 1st ed. Blackwell/Munksgaard, 2003: 101–10.

Fejerskov O. Changing paradigms in concepts on dental caries: consequences for oral health care. *Caries Res* 2004; **38**: 182–91.

References

1. Black GV. *Operative dentistry*. Vol. 1. *Pathology of the hard tissues of the teeth*. London: Claudius Ash, Sons & Co. Ltd, 1914.
2. Fejerskov O, Manji F. Risk assessment in dental caries. In: Bader J, ed. *Risk assessment in dentistry*. Chapel Hill, NC: University of North Carolina Dental Ecology, 1990: 215–17.
3. Weatherell JA, Robinson C, Hallsworth AS. The concept of enamel resistance – a critical review. In: Guggenheim B, ed. *Cariology today*. Basel: Karger, 1984: 223–30.