Caries Management—Science and Clinical Practice

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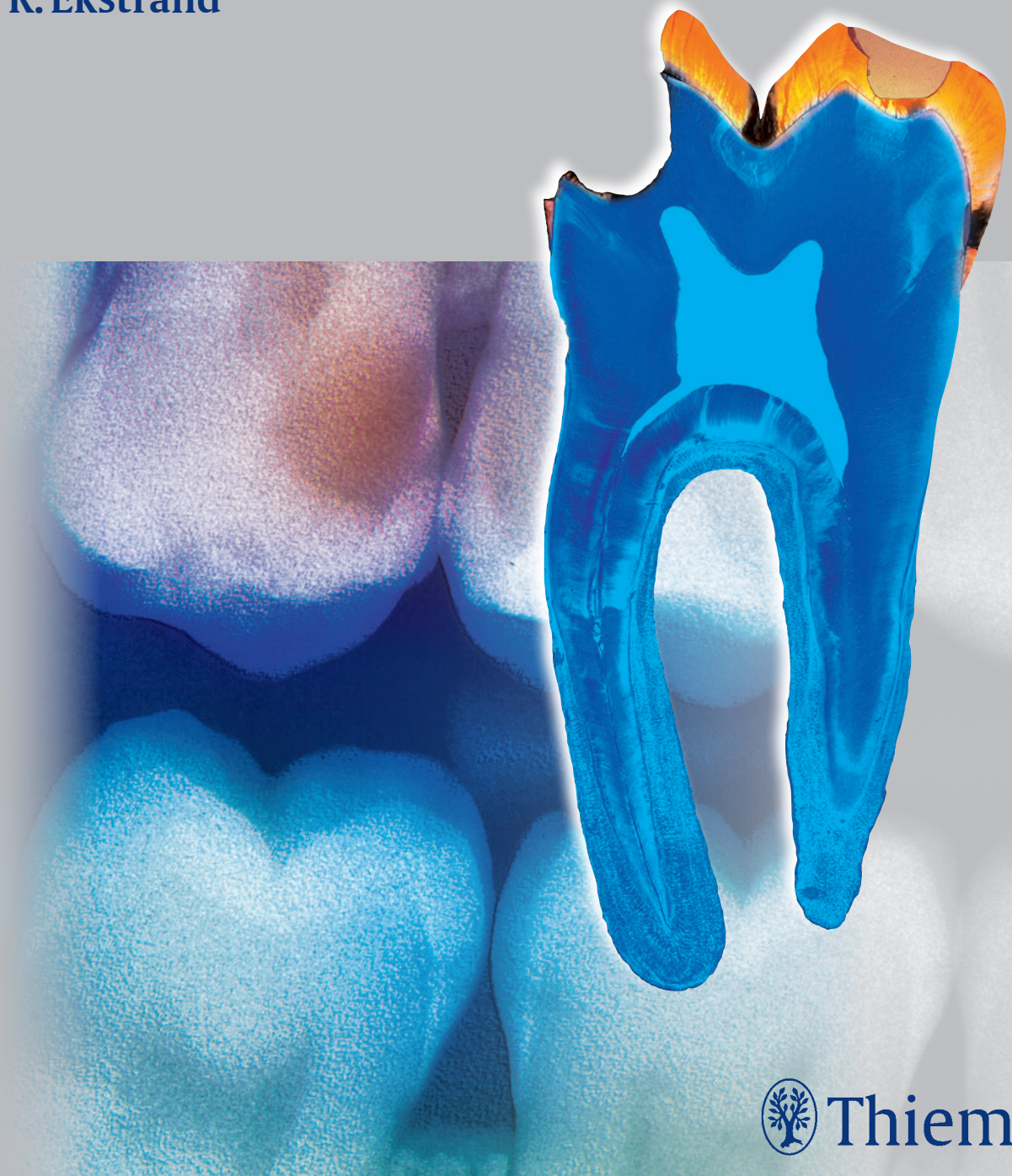

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Preface

Dental caries is still one of the most common diseases in human beings, causing smaller or larger problems to millions of people around the world every day. As dental professionals we have to help people to understand the disease to enable them to prevent caries by themselves (self-management). When more severe decay is diagnosed, we, together with the patient, should manage the caries disease in a minimally interventional way.

This introductory chapter will present some general thoughts about caries and where we come from in cariology. Moreover, we will explain why we think it is important to have another book about cariology and will describe the concept of the book, which is presented in two main parts: science and practice.

Caries—Important but Preventable

Dental caries is the term used for pathoanatomical changes of the dental hard tissues. These changes are caused by acids that are created in the dental plaque (biofilm) covering the affected tooth surface, when certain microorganisms ferment sugars, which in turn demineralize the dental hard tissues. Thus, the disease, which professionals perceive as changes of the dental hard tissues, in fact reflects activities within the overlying dental biofilm. If these unfavorable biofilm activities are occurring frequently, the signs of the caries process on the dental hard tissues will become more easily detectable. Nonetheless, the caries "scar" starts with signs that are only visible with high magnification in the laboratory but end up with clinically visible alterations of the tooth surface integrity. Thus, caries is a term which actually covers changes in the dental hard tissue from the time the first mineral ion leaves the tissue to when no mineral is left. This development takes several years, fortunately, giving the dental professional and the patient time to act. In the clinically nonvisible stages we can adopt a risk-related approach to intervene noninvasively; in the early visible stages of the disease, we can intervene noninvasively or microinvasively. Later stages of the disease need invasive intervention that aims to preserve the tooth as much as possible.

Where We Come From…

Numerous individuals or groups of scientists have contributed to our understanding of caries over time. In the following we have selected a few of the many contributors and taken the liberty to sketch their faces and make a small note about their contribution. We have not included those who are still among us. The figure captions will give the reader a good idea of the history and development of cariology including adhesive dentistry.

Do We Need Another Book about Cariology?

Nowadays the dental professional has to face an overwhelming amount of **information** concerning dental caries and its clinical management, which is derived from various "traditional" sources such as pre- and postgraduate courses at dental schools and from continuing educational programs. In addition, the Internet updates current knowledge not only for dental professionals, but also for their patients. As with everything else, when a variety of goods is on offer, the choice becomes more difficult!

From a researcher's perspective this also holds true for the increasing variety of scientific journals that provide us with evidence on related issues for dental caries and allied topics such as tooth wear. Thus, the choice and assessment of scientific information are becoming more difficult compared with former years, although this process has been formalized and professionalized in the form of **evidence-based dentistry**. Here, systematic reviews or even meta-analyses about a certain topic should help to inform the professional, being based on relevant science. Nonetheless, this systematic approach is not always feasible, either because there is not much clinical evidence available or the subject matter is quite complex. For the dental practitioner systematic reviews might even be too impracticable to provide clinical guidance in the daily grind.

In this area of conflict a textbook may be of help. Although, it cannot and need not be as objective as a scientific paper, the format of a book is capable of summing up the most relevant points in a readable manner, and is thus still an important tool in teaching. This is what we have aimed for, together with over 20 other authors from more than 10 countries, who are all experts in their respective fields of cariology.

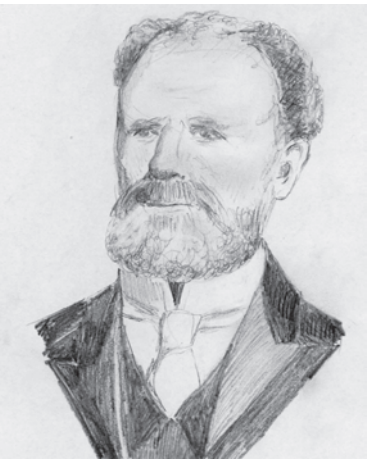
Proposal to Read the Book

The **target groups** for this book are those studying or working within dentistry: dental hygienists, dental students, graduates, and dentists whether working in the public dental service or in private practice. Dental assistants who would have their working arena extended within the field of cariology may also benefit from reading parts of this book.

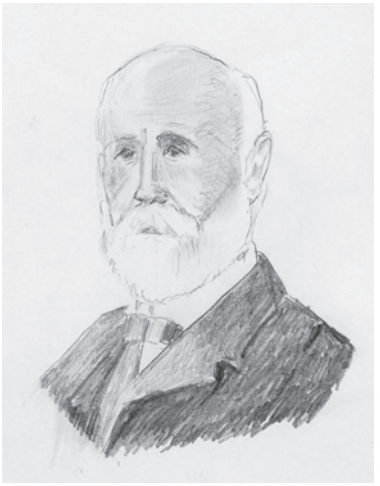
The book is divided into two parts: science and clinical practice. The **science part** is divided into five main subparts. Starting from oral ecology merging to etiology and (clinical) pathogenesis of caries and noncarious defects, the first subpart (Chapters 1–4) is rounded off by a more philosophical approach on how caries can be seen from a "modelling aspect." The second subpart (Chapters 5–9) is about clinical and radiographic detection of caries and assessment at the tooth surface level, as well as taking into account the individual level, meaning caries risk assessment. After a brief introduction to epidemiological

A. Van Leeuwenhoek, Holland
As far back as the 1650s Van Leeuwenhoek observed small animals in dental plaque, by using simple microscopes which he had made himself.

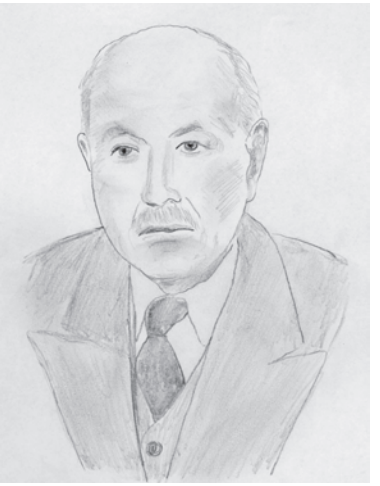
P. Fauchard, France
Around 1710, Fauchard asserted that sugar-derived acids like tartaric acid were responsible for dental decay. He also introduced dental fillings as treatment for dental caries.



W.D. Miller, USA
In the 1870s Miller observed that a multitude of microorganisms could produce acid. He suggested *the chemoparasitic caries theory*, which is still valid today.



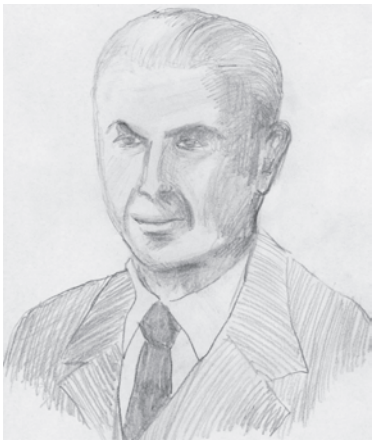
G.V. Black, USA
From the 1860s onward Black organized, among other things, Black's classification system for caries lesions (Class I, II, III, VI, V) and principles of tooth preparations for fillings.



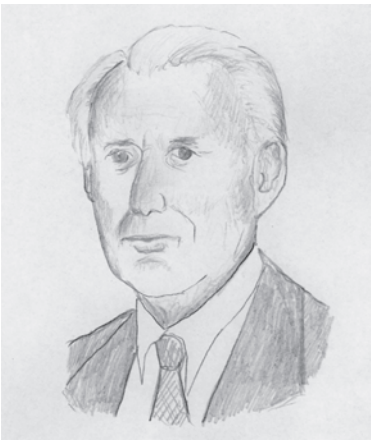
F.S. McKay, USA
In the 1930s McKay described the phenomenon of Colorado stained teeth, which later became synonymous with dental fluorosis.

H.T. Dean, USA
In the 1930s and 40s Dean observed an inverse relationship between dental fluorosis and dental caries.

matters on the topics of the book, the second subpart concludes with a proposal of how to transfer the knowledge about the caries process and its clinical assessment into clinical action. The noninvasive strategies (biofilm, diet, and mineralization modification) of how to deal with the caries process are described in the third subpart (Chapters 10–13) and possible ways of implementation in individualized and community-based dentistry are presented. The fourth subpart (Chapters 14–19) of the scientific section deals with microinvasive and minimally invasive caries treatment. This includes adhesion technology, sealing and infiltration, caries removal, and tooth-coloured direct restorations. The fifth subpart (Chapters 20–22) focuses on decision-making in treating caries in general as well as on special aspects of the presented concept in children. The scientific part concludes with some thoughts on future aspects in cariology.



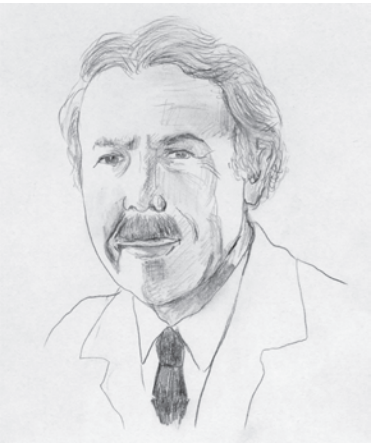
H. Klein, USA
In the late 1930s, Klein and co-workers introduced the DMF index for recording caries in the United States, where D corresponds to decayed teeth/surfaces, M to missing teeth/surfaces due to caries, and F to filled teeth/surfaces due to caries.



B. Krasse, Sweden
In the 1950s, Krasse and co-workers showed that the caries increment in mentally handicapped people (Vipeholm caries study) increased if sugar was consumed between meals in a form that was retained in the mouth for a long time. In contrast, no caries increment was seen if the diet did not contain sugar.

M.G. Buonocore, USA
In the mid-1950s Buonocore introduced a method for increasing the adhesion of acrylic filling materials to enamel surfaces, which was necessary for realizing the concept of sealing in caries.

R. Bowen, USA
In the 1950s and 60s Bowen devised Bowen's resin, a forerunner for the majority of the composite materials that dentists have used for fillings ever since.



P.H. Keyes, USA
In the 1960s Keyes described the etiology of caries by means of three overlapping circles.

A. Thylstrup, Denmark
In Denmark during the 1980s, Thylstrup and co-workers disagreed with the principle of caries resistance as being due to embedment of fluoride in the dental hard tissue, but instead explained that the effect of fluoride on caries was related to its presence in small concentrations in the plaque fluid.

D. Bratthal, Sweden
During the 1980s and 90s Bratthal introduced the caries risk assessment program, CARIOGRAM.

The **clinical practice** part describes step-by-step clinical processes as well as clinical cases, for which treatment decisions are reflected on and the treatment outcomes shown.

As the target readership for this book is very broad, the different groups within the dental profession will probably read the book differently. The best advice we can give a reader before tackling a chapter is to read the introduction, the headings, the fact boxes, and the concluding summary. Then it is time for detailed study of the chapter. Enjoy!

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List of Abbreviations

ACP	amorphous calcium phosphate
APF	acidulated phosphate–fluoride
API	approximal plaque index
BEWE	basic erosive wear examination
BMP	bone morphogenic protein
BW	bitewing
CAR	caries adjacent to restorations
CCOG	Calgary–Cambridge Observation Guide
CHAP	carbonate modified hydroxyapatite
CHX	chlorhexidene
CPP	Casein phosphopeptides
Dd	Diagnodent value
DDE	developmental defects of dental enamel
DMF(T,S)	decayed, missing, filled (teeth, surface)
dmft	decayed, missing, filled teeth (primary dentition)
DVT	digital volume tomograph
EDJ	enamel–dentin junction
EPS	extracellular polysaccharides
FAP	fluorapatite
FHAP	fluoride hydroxy apatite
FOTI	fiberoptic transillumination
GPDM	glycerophosphoric acid dimethacrylate
HAP	hydroxyapatite
HEMA	2-hydroxyethyl methacrylate
ICDAS	International Caries Detection and Assessment System
IPS	intracellular polysaccharides
MDP	methacryloyloxy decyl dihydrogenphosphate
MFP	monofluorophosphate
MHAP	magnesium modified hydroxyapatite
MIH	molar–incisor hypomineralization
mPBI	modified periodontal bleeding index
MTCP	magnesium containing β-tricalcium phosphate
MWH	magnesium whitlockite
NNT	number needed to treat
PF	prevented fraction
PSI	periodontal screening index
QHI	Quigley Hein index
RCI	root caries index
RI	refractive index
ROC	receiver operating characteristics
SEM	scanning electron microscopy
SSFR	stimulated saliva flow rate
TACT	tuned aperture computed radiography
TEM	transverse electron microscopy
TFI	Thylstrup–Fejerskov index
TMR	transverse microradiography
TSIF	tooth surface index of fluorosis

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Part 1 Caries—Science

Ecology of the Oral Cavity

Kim R. Ekstrand, Domenick T. Zero

1

In simplified terms, dental caries develops because certain bacteria in the oral cavity ferment carbohydrates (sugars) into organic acids,[1] which in the case of lactic acid may result in dissolution of dental hard tissue.[2] However, in reality the etiology and pathogenesis of caries are much more complex and will be comprehensively discussed in the following chapters. All oral tissues, especially the dental hard tissues, microorganisms, and the saliva interact not only in the physiology of the oral cavity, but also in the caries process. Therefore it is important to know their composition, structure, and functions to understand the caries process.

This chapter will deal with basic knowledge about the oral cavity focusing on the teeth, saliva, and oral microbiology, primarily from the perspective of caries disease. The subsequent chapters will build further on this knowledge. Age-related changes in dental hard tissue as well as in the salivary glands will also be touched on, as will related diseases and conditions other than caries.

In particular this chapter will cover:

- the structure of teeth,
- the functions of saliva,
- changes in the dental hard tissues and saliva with aging,
- dental plaque and its role in caries, and
- the interaction between tooth structure, saliva, and plaque in the oral cavity.

Teeth

The structure of the coronal part of the teeth is as follows.[3] The **enamel** is the outermost layer covering the **dentin,** which in turn covers the **pulp** (**Fig. 1.1**). In the roots the outer layer consists of **cementum**, covering the dentin, which covers the pulp.

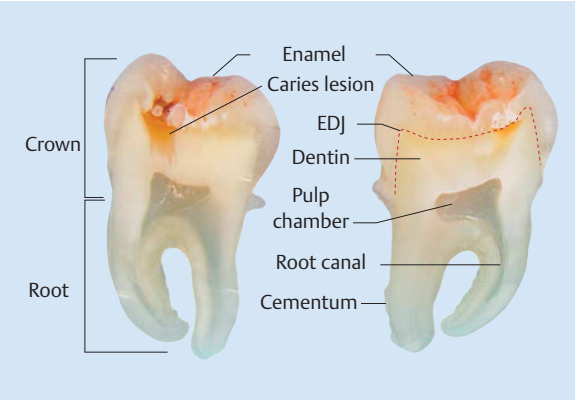


Fig. 1.1 Hemi-sectioned molar showing the major components of the tooth. The dentin forms the bulk of the tooth and encloses the pulp chamber and root canals. The enamel covers the dentin in the coronal part of the tooth and the cementum covers the dentin in the roots. EDJ: enamel–dentin junction.

Tooth Development and Tooth Emergence

Human beings have two sets of teeth: those belonging to the primary dentition, and those belonging to the permanent dentition. The conditions influencing the start of mineralization of the individual teeth, when the crowns are formed, the time for eruption, and when the roots are fully formed were mapped during the first half of the last century.[4] Teeth start to develop late in embryonic development. The first tooth type to erupt is most commonly a primary incisor in the lower jaw, which normally happens when the child is 6–8 months old (**Fig. 1.2a**). All teeth in the primary dentition are fully erupted when the child is about 2½ years old,[6] and approximal contact between first and second primary molars is seen about 1 year later.[7]

The first permanent teeth to erupt are either the central incisors or the first molar teeth; this happens in about 90% of children between 5 and 6 years of age.[8] The last permanent tooth to erupt is the third molar, which happens at the age of around 18 years. Thus, during a period of 18 years, different teeth erupt into the oral cavity, and between the ages of 5–6 and 12 years the child has a mixed dentition consisting of primary as well as permanent teeth (**Fig. 1.2b**).

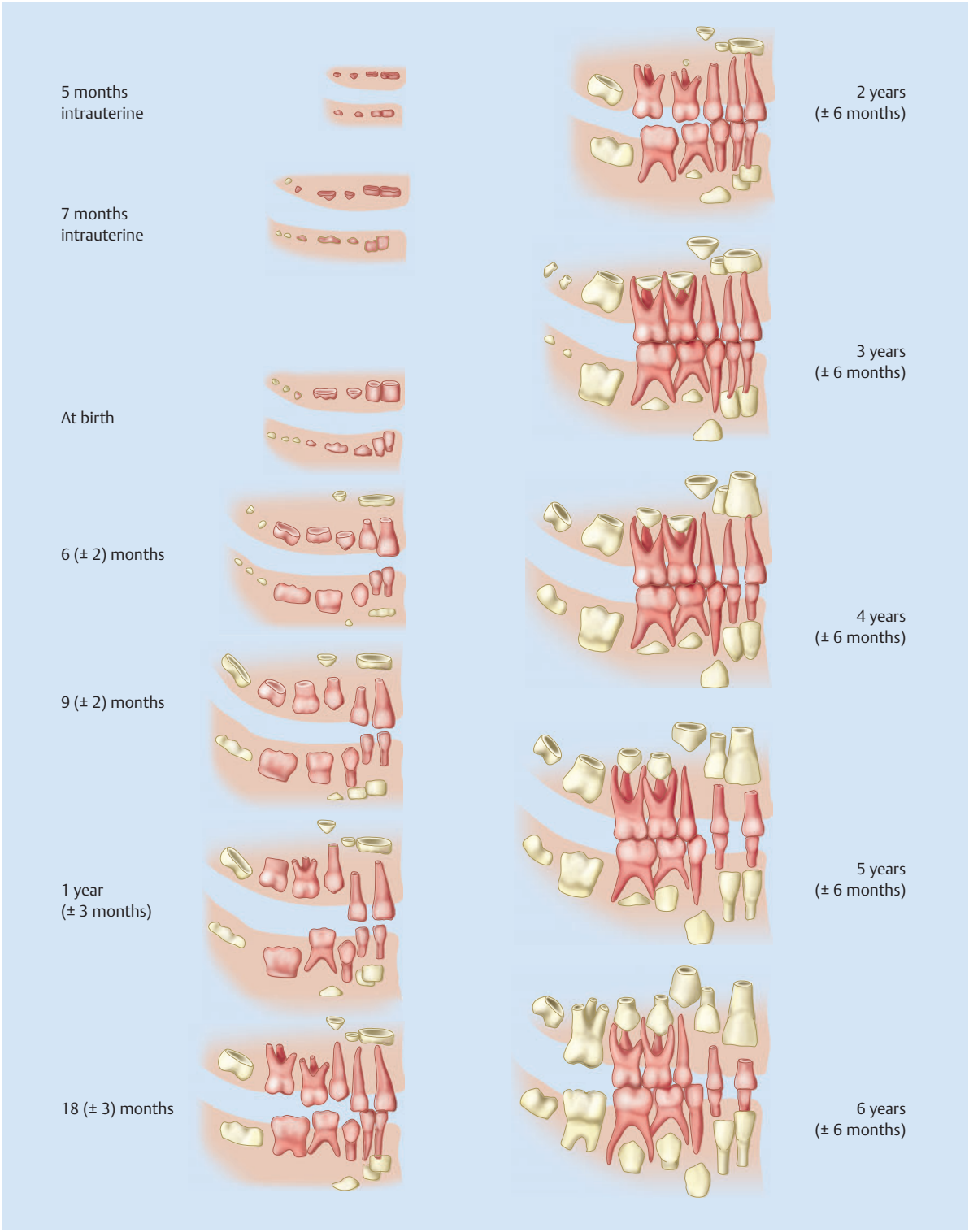
Macromorphological Terms

Professionals know where caries develops: in the primary dentition it develops mainly on the approximal and occlusal surfaces and occasionally on smooth surfaces along the gingival margin; in the permanent dentition it develops primarily on the occlusal surfaces, foramen cecum, and later, on approximal surfaces. In the elderly, caries also develops on root surfaces. The following paragraph will describe macromorphological terms related to these caries-prone sites of the teeth.

Occlusal Surfaces

In a simple model, Carlsen (1987) suggested dividing the crowns of teeth into **lobes**—from one (e.g., incisors) to five (e.g., some molars) in number.[3] Often molar teeth have five lobes, each with an essential cusp. Three of them (**Fig. 1.3a**) are the facial lobes, namely the mesiofacial, centrofacial, and distofacial lobes, which are separated on the occlusal surface by the mesiofacial and distofacial interlobal grooves. These interlobal grooves run down to the facial surface. In particular, the mesiofacial interlobal groove can end cervically in a (sometimes deep) tract called the **foramen cecum**.

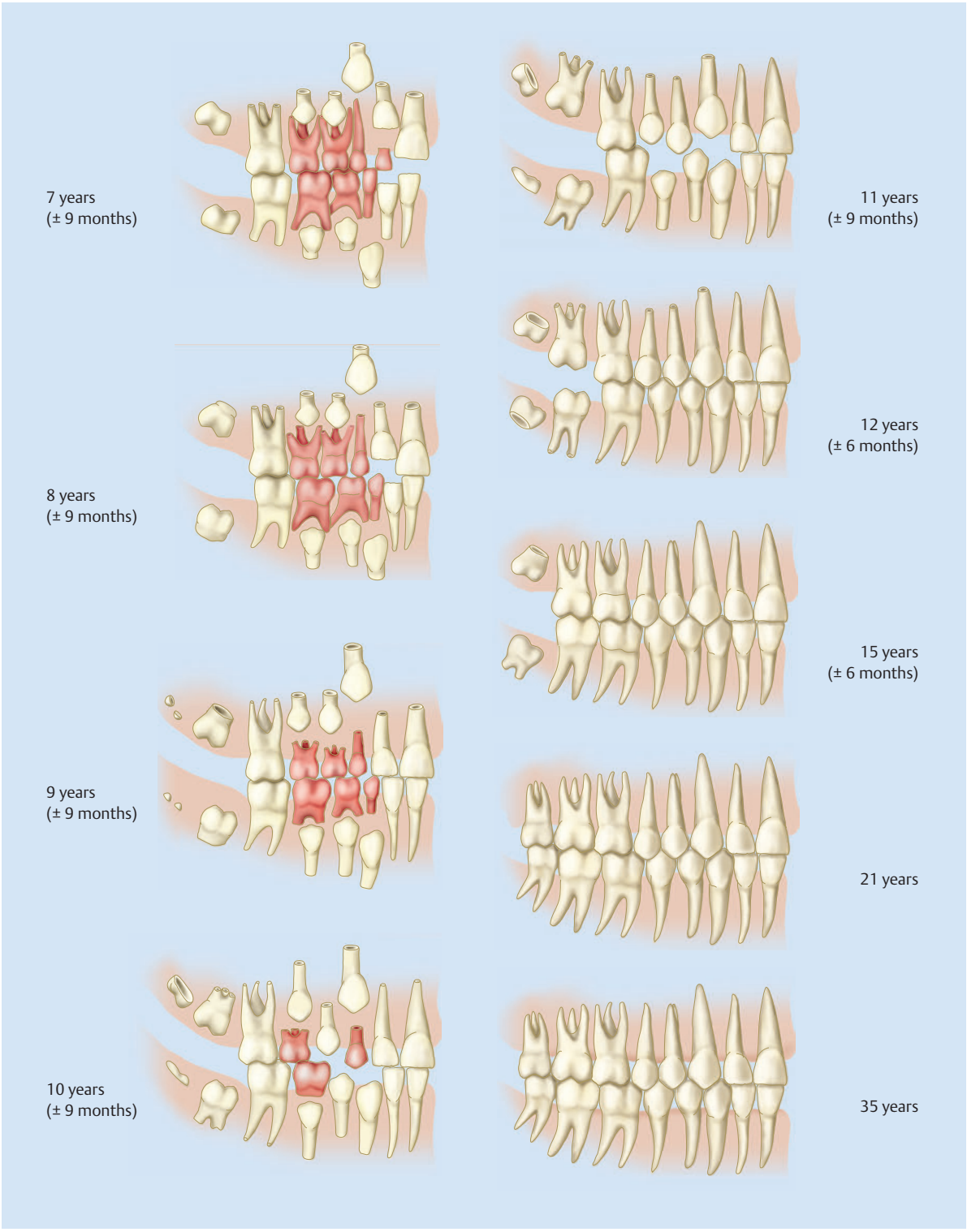
The remaining two lobes are placed lingually: the mesiolingual and distolingual lobes separated on the occlusal surface by the lingual interlobal groove. The facial lobes are separated from the lingual lobes by the mesial and distal interlobal grooves. Where the interlobal grooves meet, a tract called the **fossa** arises. Thus molar teeth often have at least three fossae: the mesial, central, and distal



a

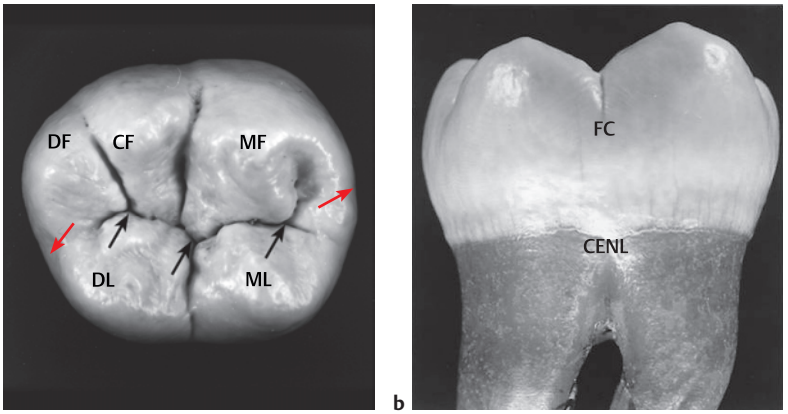
Fig. 1.2a,b Development and growth patterns of the teeth in both dentitions.[5]

Fig. 1.2b ▷



b

Fig. 1.2 (Continued)



a b

Fig. 1.3a,b
a Occlusal aspect of a permanent first molar. MF, CF, and DF are the mesiofacial, centrofacial, and distofacial lobes. The ML and DL are the mesiolingual and distolingual lobes. Black arrows point to fossae areas and red arrows point to margino-segmental grooves.
b Lingual aspect of a first permanent molar. The cervical enamel line/cemento-enamel junction separates the crown from the root. FC: location of foramen cecum.

fossae (**Fig. 1.3a**). On each lobe there are also several **intersegmental grooves**. On the marginal ridge, particularly in molars, grooves termed **margino-segmental grooves** run downward along the approximal surface. Premolars have normally two lobes, one buccal and one lingual, separated by the mesiodistal interlobal groove.

The total number of grooves, intersegmental grooves, and fossae on the occlusal surface are termed the "**groove–fossa system**," replacing the classical term "**pits and fissure system**." To build a bridge between the two classification systems, it has been suggested that the groove–fossae systems can be fissurelike or groovelike, where "fissurelike" is defined as an area where the bottom of the groove–fossa system is not clinically visible. On the occlusal surface, caries most often develops in wide fissures and in the fossae areas.[9]

Approximal Surfaces

On approximal surfaces at least three macromorphological features can influence the development of caries and must be taken into consideration:

- The width and location of the approximal contact area. That is, approximal surfaces on tooth types with narrow contact points (front teeth) have less caries than approximal surfaces of tooth types with wide approximal surface contact areas (molar teeth).[10,11]
- The curvature of the approximal surfaces. Certain molars in both dentitions show a degree of concavity on the approximal surfaces.[3]
- The margino-segmental grooves (**Fig. 1.3a**) may contribute to an uneven contact with the adjacent tooth, and the grooves can be both fissurelike and groovelike.

The Cervical Enamel Line and the Roots

The cervical enamel line (**Fig. 1.3b**) is also termed the cemento-enamel junction and is the boundary line between the anatomical crown and the anatomical root complex.[3] In patients with healthy gingiva, the line/junction is at the same level as the marginal gingiva. This line/junction is irregular and rough, so microorganisms can adhere easily to this area of the tooth.

Apart from some grooves on the roots of particular teeth, there are no macromorphological structures which promote caries development in the roots. Rather, the gingiva around the neck of the tooth promotes stagnation of microorganisms, eventually developing into plaque. In the case of gingival recession, new plaque stagnation areas are formed where root caries can develop.

NOTE

Caries usually develops in specific locations in the teeth: these are the occlusal surfaces, the approximal surfaces, and along the gingival margin.

Enamel

The enamel is formed by **ameloblasts** in three consecutive steps. Initially, the ameloblasts secrete proteins in such a way that the final form of the tooth is developed; simultaneously, a part of the protein is replaced by mineral. This is the **secretory phase** of amelogenesis.[12] The majority of the protein is, however, replaced by mineral during the **maturation stage** of amelogenesis, which takes place over several years. The amelogenesis ends at the time for emergence of the tooth when the reduced ameloblast fuses with the epithelium cells. More details can be found in Mjör and Fejerskov.[12]

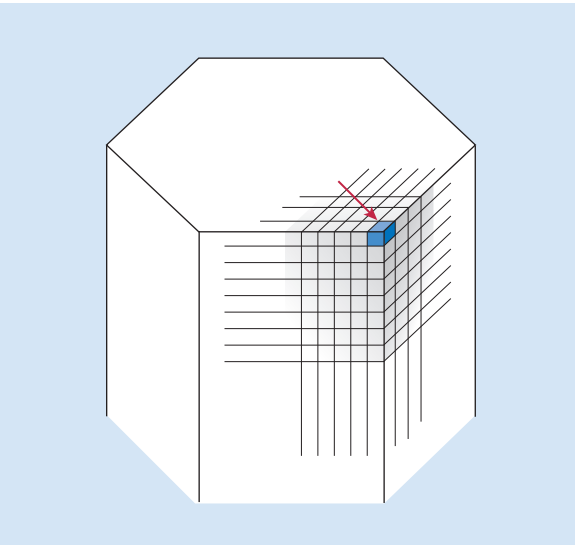


Fig. 1.4 Illustration showing the structure of a hydroxyapatite crystal. The smallest repeating entity of the crystal in enamel (arrow) has in its purest form the formula $Ca_5(PO_4)_3(OH)$.

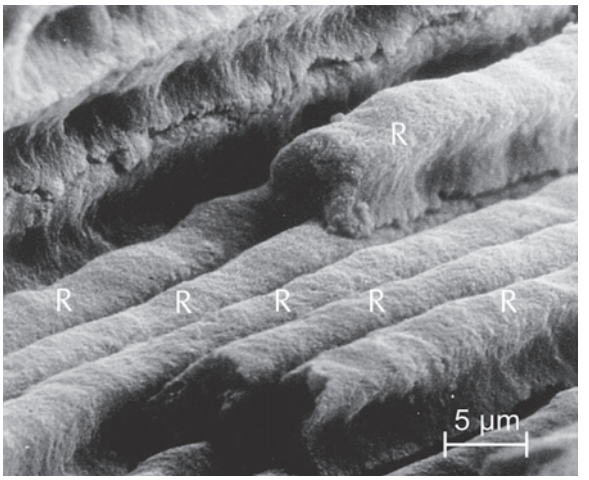


Fig. 1.5 Scanning electron microscopic images of enamel rods (R) built up by crystals.

Chemical Composition and Structure of Apatite Crystals

The inorganic content of mature enamel amounts to 96%–97% by weight; the remainder is organic material and water. On the basis of volume around 86% is mineral, 12% is water, and 2% is organic material.[12]

Owing to its hardness, enamel is difficult to cut for histological examinations used to study its structure. Therefore different approaches have been considered to describe its nature. One way to do this is at the crystalline level. In material science, a crystal is a solid substance in which the atoms, molecules, or ions are arranged in an orderly repeating pattern extending in all three dimensions. The crystals made by the ameloblasts consist of calcium phosphate, and the smallest repeating entity of the crystals in enamel has, in its purest form, the formula $Ca_5(PO_4)_3(OH)$, which is termed **hydroxyapatite** (HAP) (**Fig. 1.4**). The crystals are approximately hexagonal in cross-section, with a diameter of ca. 40 nm. The length of the crystals is difficult to assess, but today it is assumed that the length is between 100 nm and 1000 nm.[13]

At the chemical level, several substitutions of the ions in HAP can and do occur (resulting in impure forms of HAP)—for example, substitution with fluoride giving **fluoride hydroxyapatite** (FHAP); with carbonate, **carbonate-modified hydroxyapatite** (CHAP); and with magnesium, **magnesium-modified hydroxyapatite** (MHAP). **Fluorapatite** is a crystal where (nearly) all of the OH^- ions in HAP are replaced by fluoride, and which has a lower solubility than HAP; this, however, is not that common in human enamel.[14] More commonly, the OH^- ions are only partially replaced by fluoride, and FHAP is formed. These crystals also have a lower solubility than HAP, which again has lower solubility than CHAP.[15–17] These chemical conditions have great influence on the caries process and will be highlighted in Chapters 2 and 3.

The individual crystals are arranged in rods (or prisms) (**Fig. 1.5**) extending from the enamel–dentin junction to the surface, with an average diameter of about 4–5 µm. The crystals in the rods all align in the same direction except at the periphery, where the crystals change direction from those in the core of the rod. Thus, the space between the crystals or intercrystalline spaces (also called the pore volume which is filled with air, water, or proteins) is larger at the periphery of the rod than at the core. As the periphery of one rod meets other peripheries of other rods, the pore volume between rods is relatively large and much larger than in the core of the rod (**Fig. 1.6**). This is important for caries formation as acid and other products more easily penetrate through areas of enlarged pore volume (see also Chapter 3).

Due to this uniform structure of the enamel with tightly packed crystals, **light** penetrates through the enamel and is reflected or absorbed in the dentin. Well mineralized, permanent enamel is translucent, and it is the underlying dentin which, eventually, gives the tooth its color (**Fig. 1.7**). If the **pore volume** in the enamel increases, the light is scattered and reflected in the enamel which results in a white color. Primary teeth (see **Fig. 1.7**), which show a greater pore volume than the erupting permanent enamel, appear therefore whiter than permanent teeth.

Macroscopically/clinically the enamel generally looks smooth and even (**Figs. 1.3, 1.7**); however, at high magnification the surface enamel is full of **developmental defects** such as pits, cracks, and fissures[18,19] as well as normal anatomical features such as Tomes' process pits corresponding to the head of the ameloblasts (**Fig. 1.8**). Thus, there are numbers of surface irregularities on enamel where the microorganism can shelter.

In some parts of the surface enamel, and particularly in teeth of the primary dentition, the enamel is covered by crystals which are not organized as rods, but the direc-

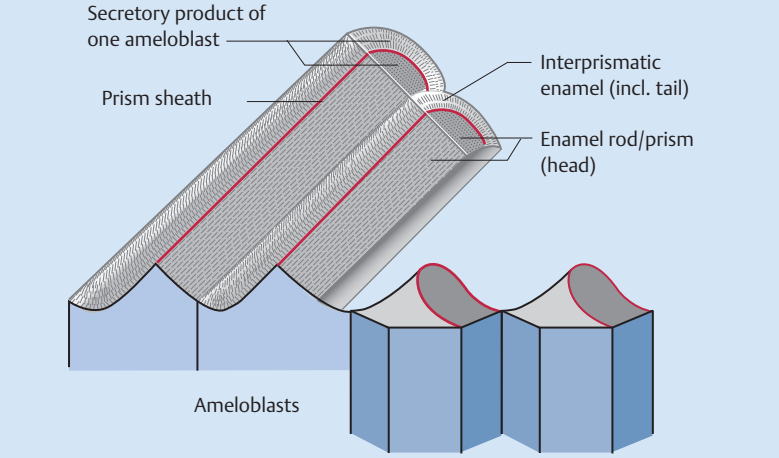


Fig. 1.6 Schematic illustration of three-dimensional arrangement of enamel crystallites within the rods (prisms) resulting from their formation by ameloblasts. Note the in the prism periphery, the crystal orientation changes abruptly, resulting in enlarged intercrystalline spaces in the prism boundaries.[45]

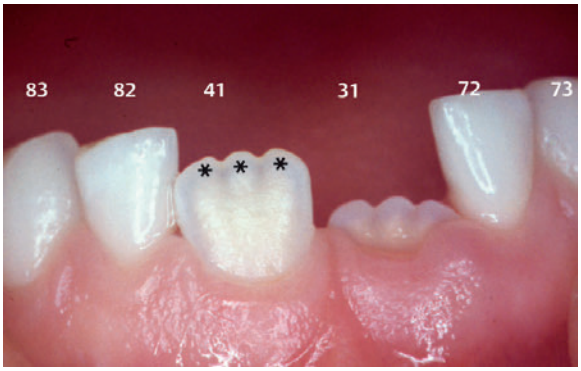


Fig. 1.7 Mandibular front teeth of a 6-year-old child, with erupting permanent first incisors. They appear more yellowish than the adjacent more opaque deciduous teeth, owing to the color of the underlining dentin. * protuberances.

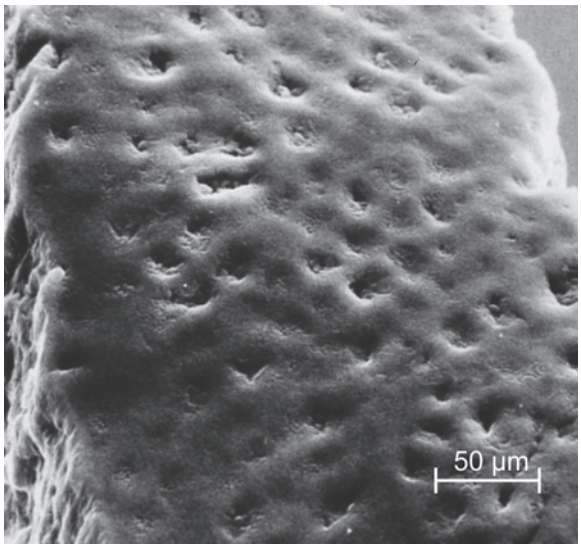


Fig. 1.8 Scanning electron microscopic view of an enamel surface showing developmental defects as Tome's process pits, large enough for microorganisms to shelter in them.

tions of the individual crystals are oriented perpendicular to the surface. This layer is called aprismatic enamel[12] and can present problems when etching enamel for sealing/bonding procedures (see below).

NOTE

Enamel is the hardest tissue in the human body; however, it is still soluble in acid with a pH below 5.5. The inorganic content of enamel is hydroxyapatite (HAP), fluoride hydroxyapatite (FHAP), carbonate-modified hydroxyapatite (CHAP), and magnesium-modified hydroxyapatite (MHAP). FHAP is less soluble than HAP, which is less soluble than CHAP or MHAP.

The Dentin–Pulp Organ

The dentin and the pulp (see **Fig. 1.1**) are closely related developmentally and functionally. The odontoblasts, which are the cells responsible for the formation of the dentin, are separated from the pulp cells only by a cell-free zone.

In contrast to the enamel, dentin continues to be formed after crown formation is complete. This is called secondary dentin formation, which over time results in reduction of the size of the pulp chamber.

The dentin consists of about 70 wt% inorganic material, 18 wt% organic material, and 12 wt% water.[12] As in the enamel, the inorganic material consists of HAP crystals (20 nm in length, <20 nm in width, and 3.5 nm in thick-

ness) which are smaller than those in enamel. As in enamel, the ions in dentin HAP can also be substituted by other ions, for example, fluoride. About 90% of the organic material consists of collagen. The structure of dentin includes dentinal tubules holding the odontoblast process, surrounded by the periodontoblastic spaces, the peritubular dentin, and the intertubular dentin. The mineral content varies in these different parts of the dentin, with the highest mineral level in the peritubular dentin. Dentin is a vital tissue that reacts to a stimulus such as caries by further dentin formation, in particular tubular sclerosis but also reparative dentin (see Chapter 3).

The pulp consists of 25 wt% organic material and 75 wt% water. The organic content is connective tissue cells (fibroblasts), fibers (collagenous in nature), and ground substances (proteoglycans and fibronectin).[12] Arterioles and venules enter and leave the pulp through the apical foramen and accessory root canals. The pulp is richly vascular; however, this changes with age. The nerves follow the course of the blood vessels and often a triad of artery, vein, and nerves is found scattered around the pulp. Extensions of nerve fibers in the pulp are seen along with the odontoblast process in the dentin.

Sensations in the pulp and in the dentin are limited to pain reactions irrespective of the factor initiating the reaction. Pulpal pain is usually dull, throbbing and lasts for some time, dentinal pain is sharp, stabbing, and short-lived.

The Cementum

Cementum made by cementoblasts is the least mineralized of the three dental hard tissues, consisting of about 65 wt% HAP/FHAP or other impure forms of HAP. As with dentin, the majority of the organic matrix (~23%) is composed of collagen. Cementum is a part of the attachment apparatus of the tooth to the alveolar bone. Cementum plays no major role in caries disease as it is often abraded at predilection sites in elderly patients.

NOTE

In contrast to enamel, dentin is a vital tissue, with less inorganic content, and is therefore more soluble in acid than enamel. Cementum often abrades before caries initiates.

Saliva

Saliva Production, Salivary Glands

Saliva is produced mainly by three large pairs of glands: the parotid glands, the submandibular glands, and the sublingual glands (**Fig. 1.9**). The amount of saliva secreted per day is **0.7–1.5L**.[20] Without stimulation, an average of

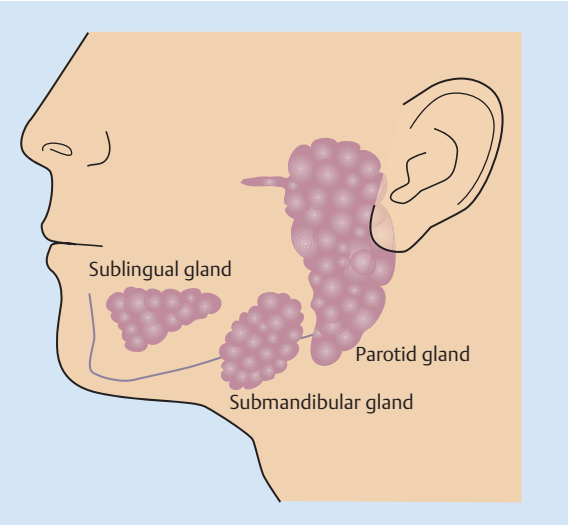


Fig. 1.9 Overview showing the location and names of the three large glands producing >90% of the daily production of saliva.

0.25mL per minute is produced, while in stimulated conditions an average **0.7mL per minute** is produced. The saliva covers all surfaces in the mouth with a thin film. The parotid gland secretes thin, watery saliva rich in amylase (an enzyme that breaks down starch into sugar). The submandibular glands secrete viscous, slimy saliva rich in mucin (a protein lubricant that also protects body surfaces). The sublingual glands produce viscous saliva. Without stimulation, two-thirds of the total saliva is secreted by the submandibular glands. Some 50% of stimulated saliva is secreted by the parotid glands and 35% comes from the submandibular glands. On viewing reflected light, one will notice that the floor of the mouth is always wet. About 10% of the daily volume of saliva comes from the minor salivary glands in the tongue, lips, and palate.

Function of Saliva

More than 99% of saliva is water, the rest is electrolytes and organic components including proteins, glycoproteins, and enzymes. The functions of saliva concerning caries are related to all three types of constituent.

The **water** in the saliva contributes to the following:

- Rinsing effect of the mouth (clearance rate)
- Solubilization of food substances
- Facilitation of bolus formation
- Facilitation of food and bacterial clearance
- Dilution of detritus
- Lubrication of oral soft tissues
- Facilitation of mastication, swallowing, and speech

The **electrolytes** have the following functions:

- Maintaining supersaturated calcium and phosphate concentrations in saliva with regard to HAP
- Neutralization of acid by buffering actions

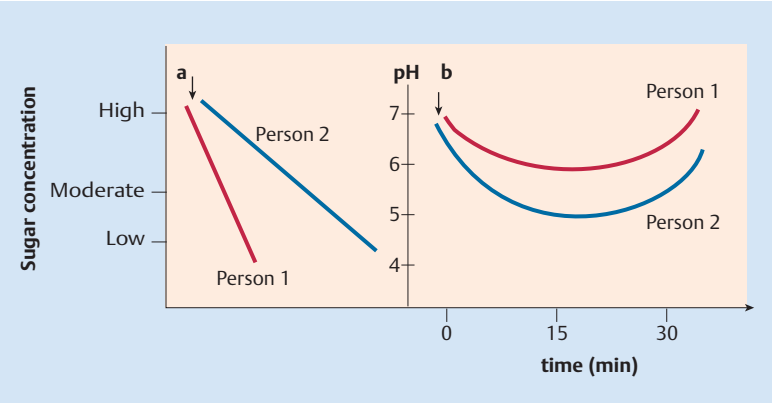


Fig. 1.10a, b
a Simplified illustration of sugar clearance of two persons after sugar intake (arrows); person 1 eliminates sugar faster than person 2.
b Consequently person 2 has a lower pH for a much longer time than person 1. Under such circumstances person 2 will likely develop caries faster than person 1.

The **organic components** have the following functions:
- Participating in enamel pellicle formation
- Mucosal coating
- Antimicrobial defense
- Digestive actions

Clearance rate. Oral clearance can be defined as the dilution and elimination of substances in the oral cavity, which can be fast or slow.[21] **Figure 1.10a** illustrates diagrammatically two persons with different saliva clearance rates,[21] person 1 with a fast clearance and person 2 with a slower clearance. The curves in **Fig. 1.10b** are the corresponding pH variations in plaque (see definition below) following the elimination of the sugar lump. This figure aims to show that a slow clearance may result in a pH drop in the saliva and/or plaque that will be lower and remain so for a longer period, which may be more harmful to the teeth than a faster clearance. It is the salivary flow rate and volumes of saliva in the mouth before and after swallowing that affect the clearance rate. Thus, stimulating saliva secretion by using chewing gum will increase the clearance rate.

Electrolytes. From the caries disease angle, the most important electrolytes are calcium, inorganic phosphate, bicarbonate, and fluoride. The concentration of the various salivary electrolytes is strongly dependent on the salivary flow rate[22,23] (**Fig. 1.11**). It appears that when the flow rate increases, the concentration of the electrolytes increases, apart from inorganic phosphate. The pH of unstimulated and stimulated saliva is between 6 and 7. At this level the relevant ions in saliva are supersaturated, which actually should result in precipitation of the electrolytes resulting in development of mineral on the tooth surface. Why this is not a common phenomenon is explained below.

Buffers. Saliva also has systems which buffer acids from the sugar-fermenting oral microorganisms. A buffer in this context is a substance which, to a certain degree, resists changes in pH. In the development of caries disease the two following buffer systems are important:
- The phosphate system
- The bicarbonate system

The form of phosphate in saliva is influenced by its pH. At pH 7.5–6.0 most of the phosphate is present as dihydrogen ($H_2PO_4^-$) and monohydrogen (HPO_4^-) phosphate, which exchange H^+ ions according to the following reaction:

$$H^+ + HPO_4^{2-} \rightleftarrows H_2PO_4^-$$

When the pH value decreases, that is, the H^+ concentration increases, hydrogen phosphate binds a hydrogen ion and changes to a dihydrogen phosphate ion. Thus, if there is sufficient monohydrogen species to react with H^+, the pH will not drop further.

The bicarbonate system works at a lower pH than the phosphate system (around 6) and takes up H^+ according to the following reaction:

$$H^+ + HCO_3^- \rightleftarrows H_2CO_3 \rightleftarrows CO_2 + H_2O$$

This system works best with stimulated saliva, because the concentration of HCO_3^- increases with increasing flow rate (**Fig. 1.11**).[22–24] The release of carbon dioxide gas (CO_2) from saliva further boosts the buffering capacity of the system as the reaction shifts toward the right.

The organic components of saliva. Table 1.1 presents the most important proteins and enzymes in the saliva and their known functions. It appears that several of them—lysozymes, agglutinins, and antibodies—have a strong antimicrobial function. Of note among the phosphoproteins found in saliva is **statherin**, which is rich in the amino acid tyrosine and is indirectly very important in the caries process. As mentioned above, neutral pH saliva is supersaturated with respect to the ions of HAP, which is the main inorganic component of tooth enamel. Phosphoproteins contain sequences of phosphorin that bind calcium very strongly, thereby maintaining the supersaturated state and at the same time preventing random crystallization from occurring.[25] Statherin is so far the only salivary protein currently known to inhibit both the primary and secondary precipitation of HAP in the supersaturated environment of the saliva. As statherin and

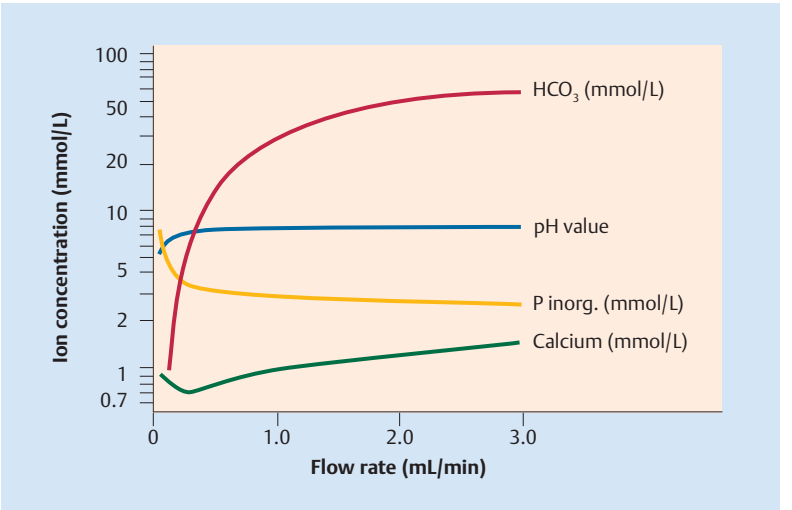


Fig. 1.11 Concentrations of important salivary electrolytes depend on the salivary flow rate (modified from Dawes 2004).[23]

Table 1.1 Organic components in the saliva and their possible roles

Organic component	Function
Amylase	Degradation of starch
Lysozyme	Antimicrobial activity by destruction of bacterial cell membranes
Lactoferrin	Antimicrobial activity by high affinity for iron
Peroxidase	Antimicrobial activity and protection against H_2O_2
Agglutinin	Antimicrobial activity by agglutination of bacteria to large aggregates
Statherin	Inhibits spontaneous precipitation
Antibodies	IgA/IgG, IgM inhibition of adhesion, enhancement of phagocytosis

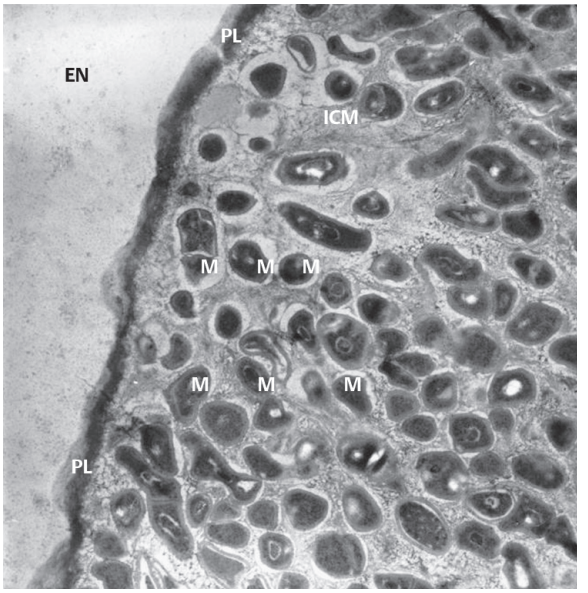


Fig. 1.12 Transmission electron microscopic examination of dental plaque consisting of microorganisms (M) and intercellular substances (ICM) lying next to the pellicle (PL) which in turn is lying next to the enamel (EN), which in this case has been removed artificially.

other inhibitors are proteins, they are subject to microbiological degradation, in particular caused by acids in the plaque.

Pellicle

The pellicle is a thin, bacteria-free layer covering the teeth (**Fig. 1.12**). It is formed by the adsorption of salivary proteins, for example, glycoproteins, which have high affinity for the mineral in the surface of the tooth.[26] The positively charged HAP crystals will attract negatively charged organic components from the saliva. If the pellicle is removed, for example, by the dentist during a professional cleaning, it will start forming again within seconds. The thickness of the pellicle varies in different areas of the teeth, generally ranging from 1µm to 10µm. However, it can be thicker and it can become discolored due to the staining from foods and/or tobacco.

The pellicle plays an important role in protecting the dental hard tissue against mechanical and chemical damage: mechanically, so it is not worn away, and chemically because the pellicle serves as a permselective diffusion barrier,[27] limiting what can pass through it, including plaque acids.

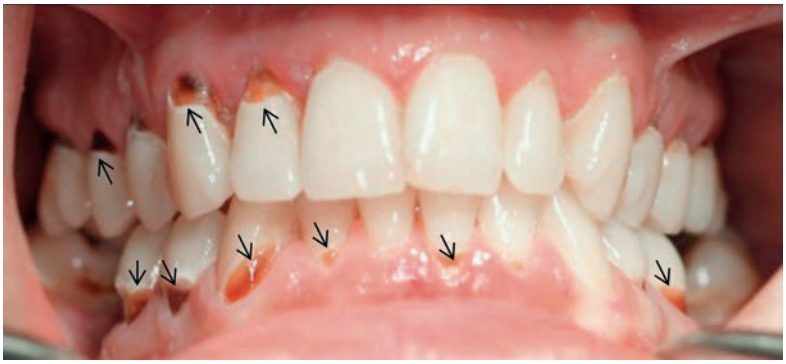


Fig. 1.13 Example of a patient who suffers from hyposalivation (unstimulated flow rate of 0.05 mL/min) and xerostomia due to the use of antidepressants. The oral mucosa is dry and caries (arrows) is seen located primarily along the gingival margin.

Hyposalivation

Hyposalivation is a diagnosis made when the unstimulated salivary flow rate is less than 0.1 mL/min and/or when the stimulated flow rate is less than 0.7 mL/min.[28]

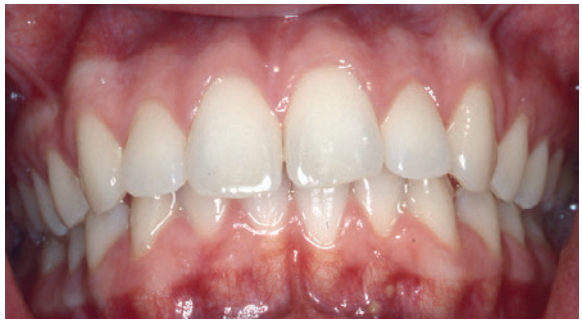
The following conditions can influence the flow rate and lead to hyposalivation:

- Medications—for example, antidepressants, diuretics, antihistamines, antihypertensives, antiemetics, narcotics
- Radiation
- Autoimmune diseases, AIDS, diabetes mellitus
- Menopause
- Eating disorders
- Salivary gland stones

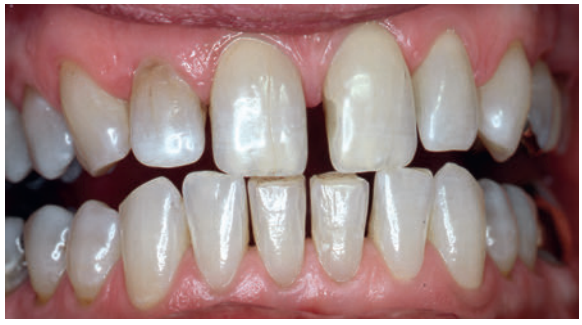
Xerostomia is the subjective feeling (symptom) of a sensation of oral dryness, which often impairs oral function and even the overall quality of life. A salivary flow rate below 0.16 mL/min increases the risk of developing caries[29] (**Fig. 1.13**), which is related to the reasons mentioned above (low clearance rate, less supersaturation with respect to important electrolytes).

NOTE

Saliva is the liquid of the oral cavity and reduces dissolution of the dental hard tissue by its clearance ability, by means of its content of electrolytes, and its content of antimicrobials. Hyposalivation therefore increases the risk of caries development.



a



b

Fig. 1.14a, b Dentition of a young (**a**) and old (**b**) person. Wear is a natural aging process that only turns pathologic if it is excessive for the respective age and results in clinical symptoms.

Changes in Teeth and Saliva with Aging

Most tissues in the human body have a physiological turnover of their components. The rate of turnover varies from tissue to tissue; in the pulp tissue the turnover is considered to be high, while it is limited for the dentin and cementum. Tooth enamel is a tissue with no biological turnover after it is formed. Alteration of enamel during a lifetime is thus physico-chemically related. Wear will cause loss of incisal protuberances, perikymata, and imbrication lines resulting in a flattening of the teeth with age (cf. **Fig. 1.14a, b**). At the crystal level, old enamel has a higher content of fluoride,[30] the reason for which will be covered in Chapters 2 and 12.

At least two age-dependent changes take place in the dentin; namely physiological dentin formation and gradual obturation of the dental tubules. The former is referred to as **secondary dentin** formation, to differentiate it from primary dentin formation which occurs until the tooth is fully formed, while the latter is referred to as **dentin**, or **tubular sclerosis**. The changes in the dentin during a lifetime have some clinical and cosmetic implications[31]; thus the diminishing size of the pulp chamber due to the secondary dentin formation may prevent pulp reaction

and pulp exposure, but may also complicate pulp treatment. Tubular sclerosis results in a reduction in the sensitivity and permeability of dentin, although the latter may prevent ingress of toxic agents deeper into the dentin. The sum effect of changes in the dentin (condensation) influences the color of the teeth, thus owing to the translucence of the enamel, the color of older teeth is more yellow than younger teeth (**Fig. 1.14b**).

The most striking age-related change in the cementum is that its width nearly triples with age. To the best knowledge of the authors this has no clinical implication.

The pulp changes with age—in general from a cell-rich and fiber-poor tissue to a cell-poor and fiber-rich tissue.[31] These changes are important from a clinical point of view, as the reactivity of an old pulp is different from the young one. This must be taken into consideration when choosing between different treatment options.

As described above, more than 99% of saliva is water, thus less than 1% is electrolytes and organic components. What happens to these components with age? Most data[32,33] indicate that there are changes in the structure of the salivary glands due to age, but it seems that these changes are not sufficient to significantly influence the three components (water, electrolytes, and organics) in such a way that the tendency for developing caries increases. Instead of relating the increasing prevalence and incidence of caries seen in elderly people to age-related disorders of the salivary glands, we should rather consider age as a possible contributory factor to increasing patient vulnerability.[33]

Dental professionals should be able to differentiate between signs of natural aging/wear and signs of pathological processes. However, it should be kept in mind that the transition between "natural aging" and "disease" is mostly fluid, and the definition of what is "disease" is often controversial.

Dental Plaque or Dental Biofilm?

Definition. Dental plaque is a general term for the complex microbial community found on the tooth surface embedded in a matrix of polymers of bacterial and salivary origin.[34] The term "**dental plaque**" has been used by the dental profession since G.V. Black (see Preface) defined it at the end of the 19th century. Professionals use it clinically for describing **visible accumulations of microorganisms on teeth**. More recently the term "**dental biofilm**" has been used to describe dental plaque. Biofilms are defined as "3-D accumulations of interacting microorganisms attached to a surface, embedded in a matrix of extracellular polymers."[35] Biofilms are also found on other, water-covered surfaces, for example, the waterlines in dental units and in aquariums. Throughout this book the authors will use both terms for visible accumulations of microorganisms on the teeth.

NOTE

In the context of caries, dental plaque or dental biofilm is the same—meaning visible accumulation of microorganisms mixed with intercellular substance on the teeth.

Classifying Oral Microorganisms

The Dutchman Antonie van Leeuwenhoek was the first to discover small organisms in dental plaque by means of simple microscopy. Actually what he saw was microorganisms of differing morphology—some small and round, some quite long, some lying still, and some moving. Since then, microorganisms in the oral cavity have been examined using simple and more complex light microscopes; sometimes the microorganisms are colored, other times not so (e.g. **gram+** or **gram−**) (**Table 1.2**). The microorganisms have also been examined by means of electron microscopy, cultivation on different media, and more recently by means of genetic methods. Today, more than 700 different species have been identified in oral biofilms. The composition of species varies between individuals and various tooth sites, and even within different locations of the plaque.

Table 1.3 shows the overall **biological classification hierarchy** of two of the most studied microorganisms relating to caries disease, namely *Streptococcus mutans* and *Lactobacillus acidophilus*. Both are Bacteria (Kingdom) and have a gram-positive cell wall structure (Firmicutes). *S. mutans* is a coccus and *L. acidophilus* a rod (Class), and the major metabolic end product of carbohydrate fermentation is lactic acid, making them *Lactobacillales* (Order); they belong to the families of Streptococcaceae and Lactobacillaceae, respectively. Microbes of the genus *Streptococcus* (**Table 1.3**)—which make up the majority of the microorganisms in the oral cavity and include the species *S. mutans*—are thus facultative anaerobic gram-positive cocci occurring in chains, which do not move or produce spores (**Table 1.2**). Microbes of the genus *Lactobacillus*, of which *L. acidophilus* is a member (**Table 1.3**), are mainly facultative anaerobic gram-positive rods, which do not move or produce spores (**Table 1.2**).

Differentiation within the individual species can be seen, for example, by means of the growth pattern on a range of selective and nonselective agar plates (**Table 1.2**). Concerning the streptococci, *S. mutans* can be differentiated from *S. sanguinis* by the pattern of colony formation when cultivated on Mitis Salivarius Agar. *S. mutans* appears as slimy granulated colonies, while *S. sanguinis* appears as small, firmly adhering colonies. Biochemical tests show in addition that *S. mutans* metabolizes sorbitol while *S. sanguinis* does not.

Table 1.2 Traditional way to classify oral microorganisms, with examples

Feature	Parameter value	Streptococci	Lactobacilli
Cell morphology	Cocci, rods, filaments, etc.	Coccoid	Rod
Gram (dye) coloring of microorganisms	Positive or negative	Positive	Positive
Cell arrangement	Single or chains	Chains	Random, but often in chains
Movements	yes/no	no	no
Spore	yes/no	no	no
Oxygen tolerance	Aerobe, facultative anaerobe and strict anaerobe	Facultative anaerobe	Facultative anaerobe
Catalase	Positive or negative	Negative	Negative
Carbohydrate metabolism	Homo- or hetero-fermentation	Both	Both

By the use of other techniques introduced in the 1980s such as serological and genetic testing methods (checkerboard DNA–DNA hybridization, polymerase chain reaction),[36,37] it has been suggested that *S. mutans* can be subdivided into subgroups, such as serotypes a–h, where the original *S. mutans* consists of serotypes c, e, and f. Serotypes d and g are called *S. sobrinus*. This differentiation is important because some serotypes produce more acid from sucrose than *S. mutans*.[38]

Colonization of the Mouth in the Newborn

When a child is born his or her mouth is usually sterile, but will very quickly become colonized by microorganisms, particularly from the mother, but also from other sources such as milk, food, water, etc. The first microorganisms to colonize the mouth of a newborn are termed **pioneers.**[34] Further development or microbial succession is dependent on the conditions offered to or changed by these pioneers, for example, nutrition and local pH. Eventually, a **climax community develops**, which is a stable, complex microbial community of great species diversity. In the period before the first teeth appear, the microflora consists mainly of Streptococcus and in particular *Streptococcus salivarius.* However, plaque development does not occur on oral soft tissues in the same way as on teeth, owing to the continual shedding of the outer cells harboring the microorganisms. When the first teeth appear, a change in the microflora is noted, as types which can adhere to dental hard tissues such as *S. mutans* and *S. sanguini* become established.

Table 1.3 Biological classification of the *Streptococcus mutans* and *Lactobacillus acidophilus*

	Streptococcus mutans	Lactobacillus acidophilus
Kingdom	Bacteria	Bacteria
Division/Phylum	Firmicutes	Firmicutes
Class	Bacilli (coccoid)	Bacilli (rod)
Order	Lactobacillales	Lactobacillales
Family	Streptococcaceae	Lactobacillaceae
Genus	Streptococcus	Lactobacillus
Species	*Mutans, salivarius*, etc.	*Acidophilus, casei*, etc.

Plaque: Development and Metabolic End Products

Professional cleaning as well as tooth brushing, if done properly, removes plaque and the pellicle on the teeth leaving the enamel naked. When saliva moistens the teeth a new pellicle will start to form. During the first couple of hours after the cleaning procedure microorganisms in the saliva will adhere to the **pellicle** on the teeth by means of weak biological as well as electrostatic forces such as van der Waal's interaction.[39] Such microorganisms are also called **pioneers**, as mentioned above, when a newborn child's mouth is colonized. The pioneers are mostly *Streptococcus sanguinis*, *S. oralis* and *S. mitis* biovar 1, but genera such as *Actinomyces*, *Haemophilus*, and *Neisseria* are also present.[40] The mechanism of this initial adherence of microorganisms to the pellicle is complex and not fully

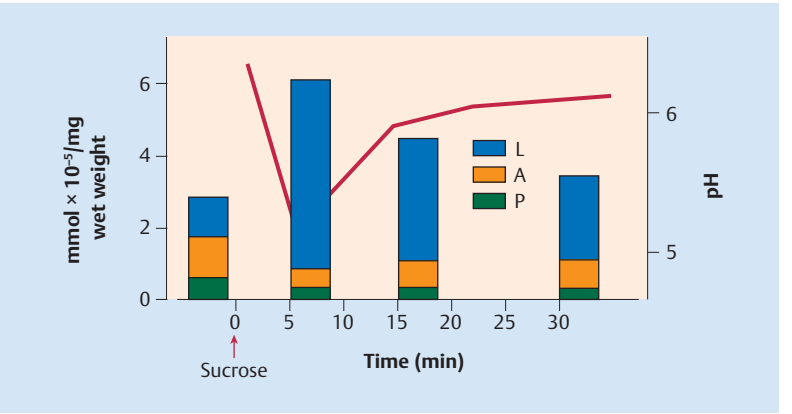


Fig. 1.15 Acidic metabolic end products and change of pH in dental plaque before and after intake of a lump of sugar [2].
L: lactic acid; A: acetic acid; P: propionic acid.

understood. However, it seems that microorganisms have a kind of recognition system in their cell membrane which fits to receptors in the pellicle.[39] In addition, microorganisms can shelter in development defects and in the groove–fossa system without physicochemical interactive forces.[18,19]

The microorganisms need energy for their survival and replication. They can use many different methods for obtaining energy, which is influenced by the substrate available in the mouth that comes from saliva and the host's diet. The pioneers accumulated on the teeth after 3–6 hours are arranged in a **monolayer**. The pioneers, which primarily are aerobes or facultative anaerobes, will most likely use oxygen from the surrounding salivary film, which enters via the cell membrane, and the tricarboxylic acid cycle of Krebs (see Ref. [1] or other biochemical textbooks) to get intracellular energy. The end products leaving the cells are CO_2 and water, which are not harmful to the teeth.

Through multiplication of the pioneers and arrival of newcomers, over the next few hours a rapid increase in the number of microorganisms accumulating on the teeth is seen (6–12 h). Thus, the monolayer of microorganisms is replaced by **multiple layers**.[40] As the thickness of the layers increases (at a certain stage it becomes visible, and thus, as plaque), the oxygen tension in the inner layer (against the tooth surface) will drop, and the microorganisms in that layer will shift their metabolism and become more facultative anaerobic or strictly anaerobic.

In the case of no access to dietary sources of nutrition, the microorganisms get energy primarily from the glycoproteins in the saliva. Under such conditions, the by-products of metabolism by microorganisms on the teeth are evenly distributed among lactic acid, acetic acid, and propionic acid (**Fig. 1.15**). The concentration and strength of these acids do not harm the teeth, mainly owing to the action of the buffering systems. In the case of access to fermentable carbohydrates, the pH will drop in the liquid phase of the plaque within 3 minutes, and it takes about 20–30 minutes for the pH to return to normal. The reason for the pH drop is that some microorganisms are able to convert the available sugar—which due to its very high concentration enters the cell membrane passively—via the glycolytic pathway and metabolize it to lactate[1] (**Fig. 1.16**). The fraction of lactate increases eightfold[2] during the first couple of minutes after starting to eat breakfast. This process requires that the microorganism has a system of constitutive enzymes, and in this case it is the **lactate dehydrogenase** that enables the microorganism to transform pyruvate to lactate, which then is released through the cell membrane (lactate gate) to the environment[1] (**Fig. 1.16**). During the metabolic process energy in the form of ATP is created. The microorganisms use the energy from ATP mainly for cell functions and replication. Microorganisms that do not possess lactate dehydrogenase may die, caused by substrate (sugar) killing. Some microorganisms can also synthesize intracellular polysaccharides to be used as "fuel" when there is no sugar in the surroundings to be metabolized.[1] Finally, some microorganisms also have constitutive enzymes, such as glucosyltransferases and fructosyltransferases, which can convert sucrose to glucans and fructans (extracellular polysaccharides), respectively. Glucans serve to glue the microorganisms together, and fructans are easily metabolized and can act as a reserve source of nutrients.[1]

NOTE

Microorganisms in cariogenic plaque have the following characteristics[35]:
- Anaerobe or facultative anaerobe
- Acidogenic (produce acid, mainly lactic acid)
- Aciduric (can survive under low pH conditions)
- Produce intracellular polysaccharides
- Produce extracellular polysaccharides

Plaque Stagnation Areas

Plaque development can only happen on areas of the teeth where there is no mechanical or chemical disturbance.[9,34,35,41] Examples of mechanical disturbance on the teeth are movements of the tongue and lips, and oral hygiene practices such as tooth brushing, flossing, etc.