ETIOPATHOGENESIS OF PERIODONTAL DISEASE

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- Is Human foetus inside the uterus **sterile** ?

- Within **2 weeks**, a nearly mature microbiota is established in the gut of the newborn baby.

- **After weaning (>2 years),** the entire human microbiota is formed and comprises a very complex collection of hundreds of different types of bacteria with approximately **10^{14} microbial cells**.

- From this moment on, our body contains **1.3 to 10 times** more bacteria than human cells.

- It has been estimated that, for a normal, healthy human being, the bacterial population comprises **2 kg of the total body weight**. This is fascinating if one realizes that the average human brain weighs only about 1.4 kg.

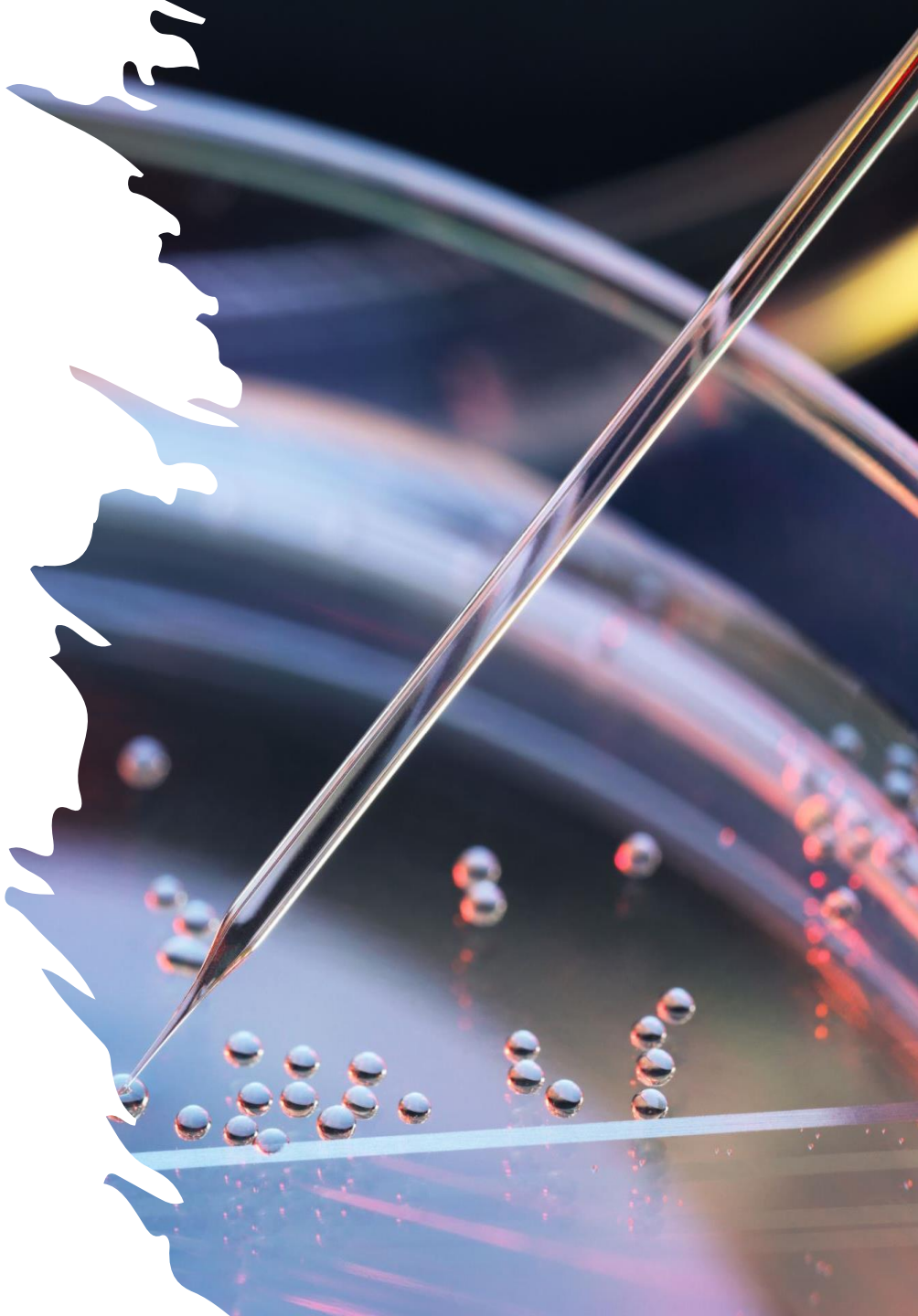
INTRODUCTION

- Within hours after birth sterile oral cavity is colonized by numbers of **mainly facultative and aerobic bacteria**
- From **2nd day, anaerobic bacteria** can be detected
- First and most dominant oral microbes in the oral cavity of newborn infants: ***Streptococcus salivarius and Streptococcus mitis***
- *Veillonella spp., Neisseria* spp., *Actinomyces spp. and Staphylococcus* spp. *are also among the* ***first colonizers of the oral cavity***

- It is estimated that the oral bacterial microbiome of adults encompasses approximately **750 commonly occurring species**, roughly half of which can be present at any time in any individual.

- A shift in microbial community leads to an **imbalance, or dysbiosis,** in the microbiota.

- This **shift** in the microbial community can be largely independent of the acquisition of new members of the microbiota and instead reflects changes in the abundance of either individual organisms or consortia of organism's resident within the **subgingival bioflm (previously referred to as plaque).**



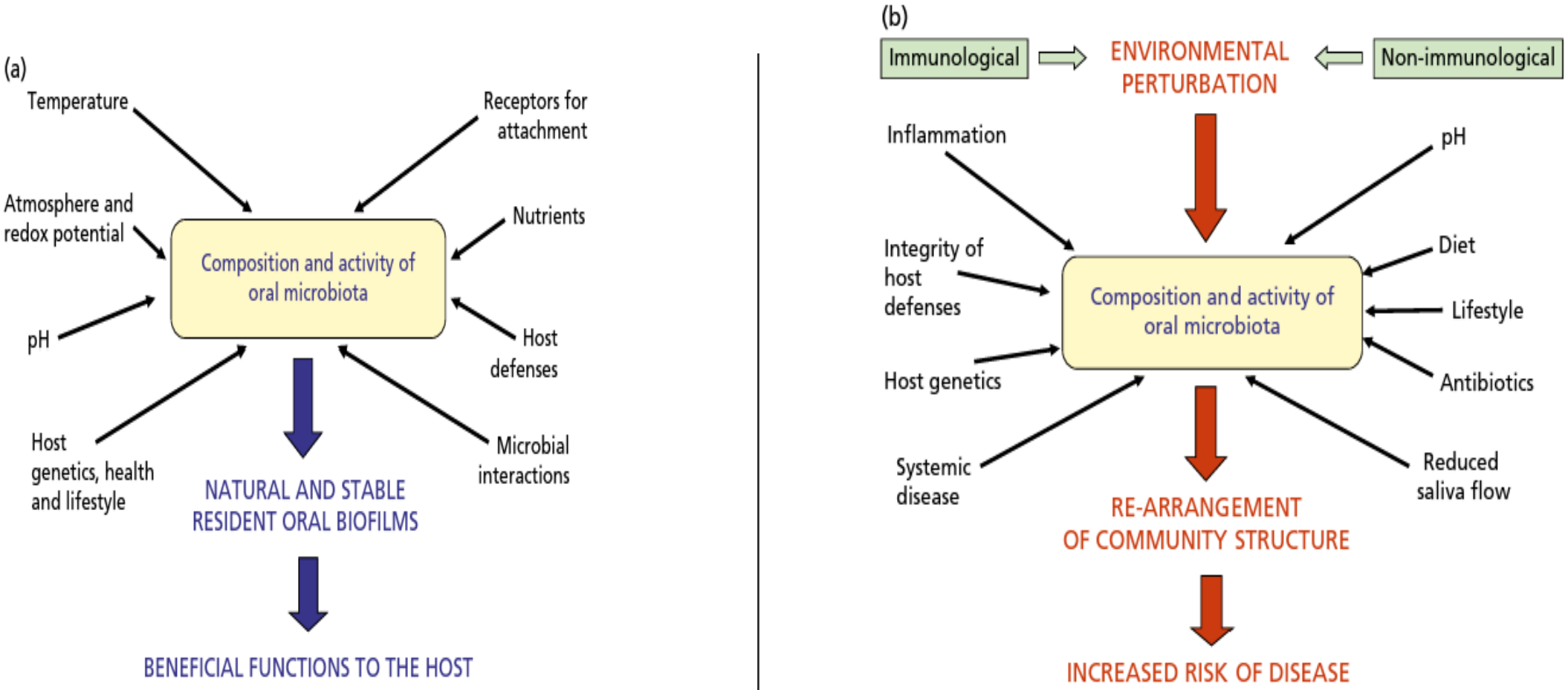


Fig. 8-1 Host factors that influence the microbial composition, activity and stability of the resident oral microbiota. (a) A number of host factors help to determine the composition and activity of the natural and beneficial oral microbiota. (b). A perturbation in a key environmental factor can disrupt the natural stability (microbial homeostasis) of the resident microbiota at a site and result in a re-arrangement of the composition and activity of the resident microbial community; such a change might predispose the site to disease. (Source: Adapted from Marsh et al. 2011.)

From an ecologic viewpoint, the **oral cavity, which communicates with the pharynx**, should be considered an "**open growth system"** with an uninterrupted ingestion and removal of microorganisms and their nutrients.

Any microorganisms that are **unable to adhere** to a surface within the **mouth are washed away in the flow of saliva and swallowed**.

The composition and diversity of the microbial community is therefore **impacted by physical gradients of temperature, humidity, nutrient content, salivary flow, oxygen tension and pH, as well as shear forces due to mastication.**

Frequent exposure to dietary sugars can significantly affect the dynamics of bacterial adhesion and accumulation.

- The constant lubrication of the oral cavity by saliva allows microbes to disperse and reach distant sites within the oral cavity, with **saliva** effectively acting as a **transport medium**.

- Although saliva has **antimicrobial** properties, it has also been reported to **promote growth of a health-associated microbiota.**

- The **primary source of nutrients for the oral microbiome** is provided by the host, and include the proteins and glycoproteins present in **saliva and gingival crevicular fluid (GCF).**

- The mouth is maintained at a temperature of around **35–37 °C**, which is suitable for the growth of a broad range of microbes, though temperature **does increase at subgingival sites during inflammation**, which can favour the growth and metabolism of some putative periodontal pathogens.

- **pH is a major determinant** of bacterial distribution and metabolism in the mouth.

- The **buffering activity of saliva plays a major role** in maintaining the intraoral pH at around neutrality, which favours the growth of the resident oral microbiome.

- The pH in dental biofilms falls rapidly to below **pH 5.0** following the intake of **dietary sugars** due to the production of **acidic fermentation products** (Marsh et al . 2016b).

- Many **health- associated bacteria** can tolerate brief conditions of **low pH** but are inhibited or **killed by more frequent or prolonged exposures to acidic** conditions (Svensater et al . 1997).

- The mouth is richly endowed with components of both the innate (e.g. lysozyme, lactoferrin, sialoperoxidase, host defence peptides, etc.) and adaptive (**secretory IgA, IgG, complement, neutrophils, etc**.) immune response (Marsh et al . 2016a, b)

The oral microbiome

Sedghi L, DiMassa V, Harrington A, Lynch SV, Kapila YL. The oral microbiome: Role of key organisms and complex networks in oral health and disease. Periodontol 2000. 2021;87:107–131

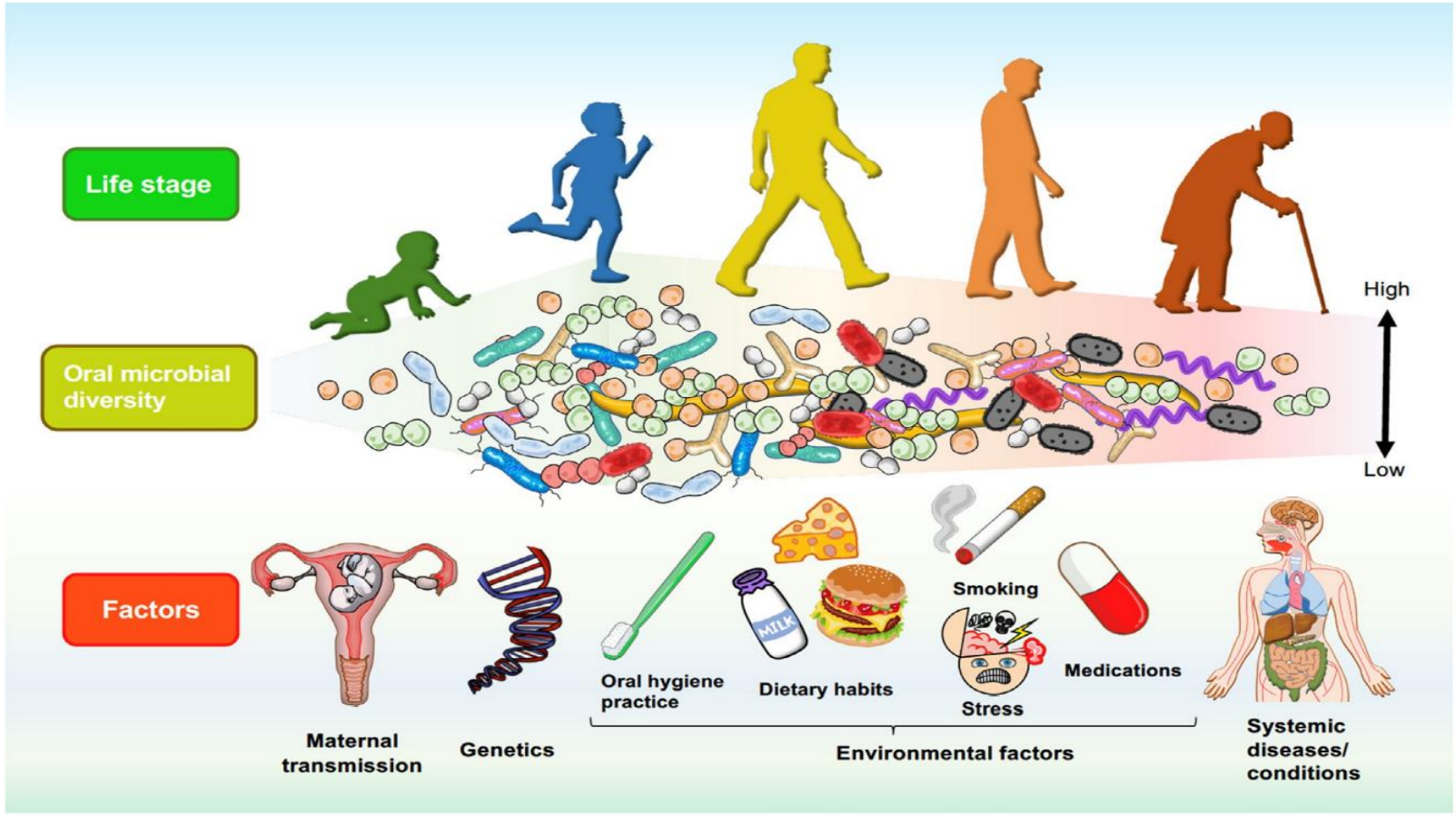


FIGURE 1 The Oral Microbiome: From First Encounters to Lifelong Encounters. *Prenatal*. The prenatal oral cavity is thought to be sterile until birth, with colonization occurring soon after delivery. The composition of the oral microbiota in infants has been shown to correlate with mode of delivery. However, infants share an oral microbiota similar to that of their mothers, suggesting that the infant oral microbiota may derive from hematogenous or intrauterine transmission from the mother. Detection of oral microbes of maternal origin among several intrauterine locations, as well as associations with adverse pregnancy outcomes, demonstrate the role of the maternal oral microbiome in prenatal health and suggests in utero colonization. *Early life*. Microbial colonization begins shortly following birth through vertical transmission from the mother, transmission from the diet, and transmission from infant-to-human interactions. Microbial diversity increases upon eruption of primary teeth as this process permits the expansion of microbial niches in the oral cavity. Eruption of primary teeth also results in deviation from the maternal oral microbiota. As children age, their oral microbiotas begin to stabilize. *Adult life*. The oral microbiota continues to be shaped throughout life by genetic and environmental factors. Environmental factors that influence the composition and function of the oral microbiome include diet, stress, oral hygiene practices, drinking alcohol, and smoking. Genetic factors are linked to conserved phylogenetic and functional microbial signatures related to development of dental caries and heritable predisposition to periodontal disease. *Aging and systemic disease*. Oral microbiome diversity has been shown to decrease with age. The phylogeny and functional signatures of the oral microbiome are

The Oral Cavity From a Microbe's Perspective

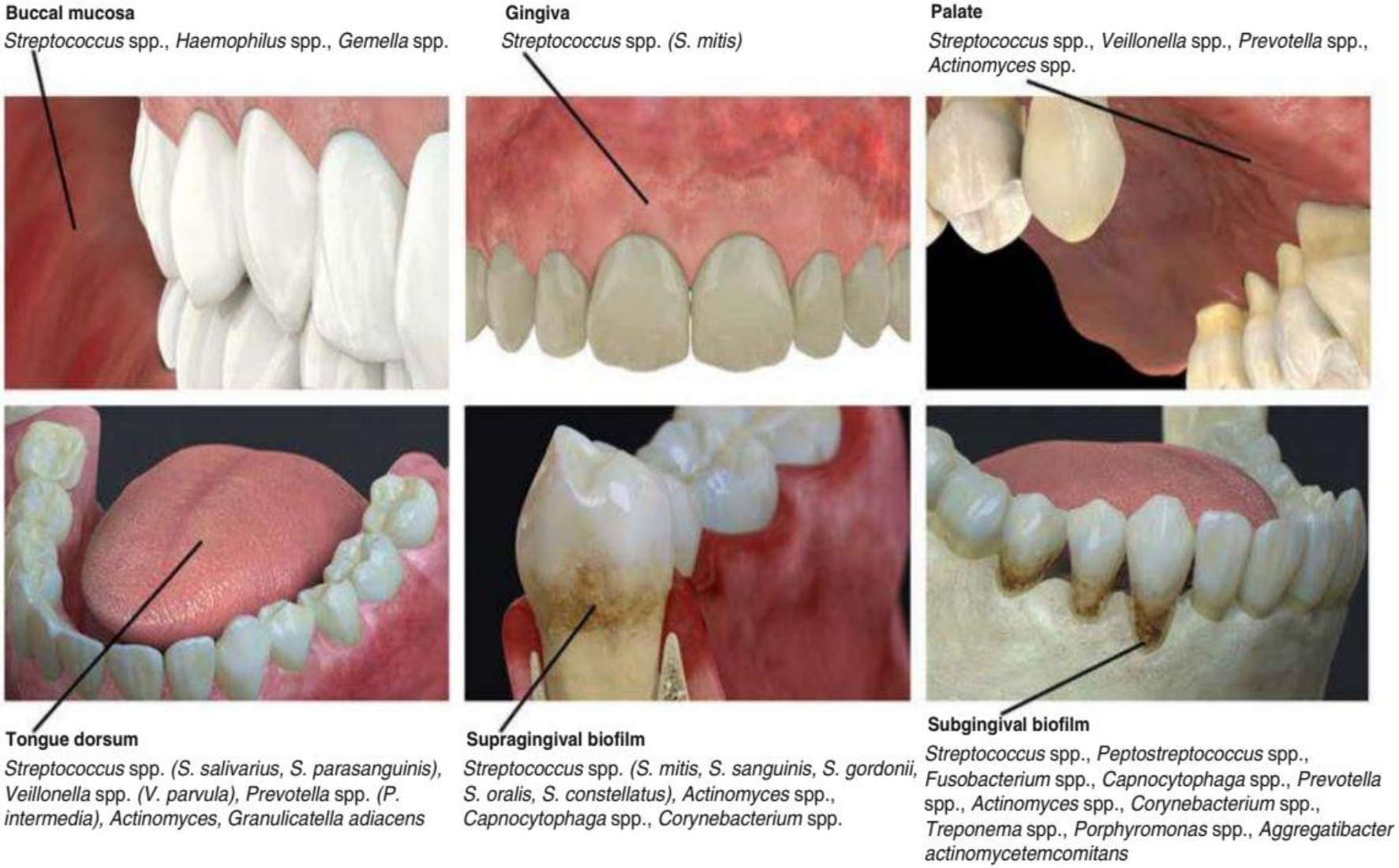
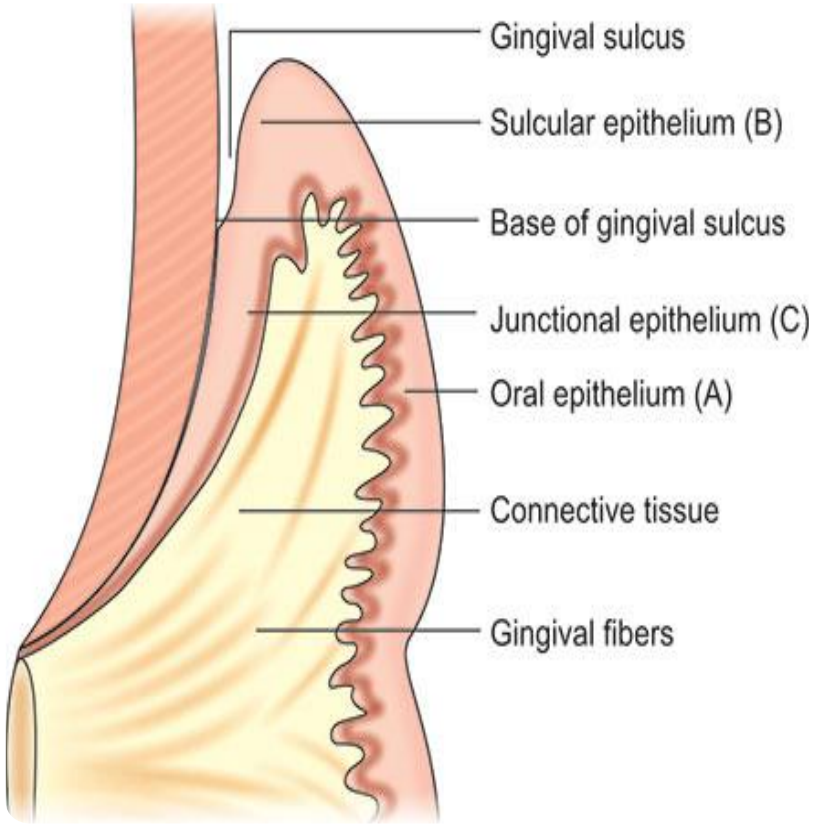


Fig. 10.1 Different intra-oral ecological niches with the most prevalent bacterial species.



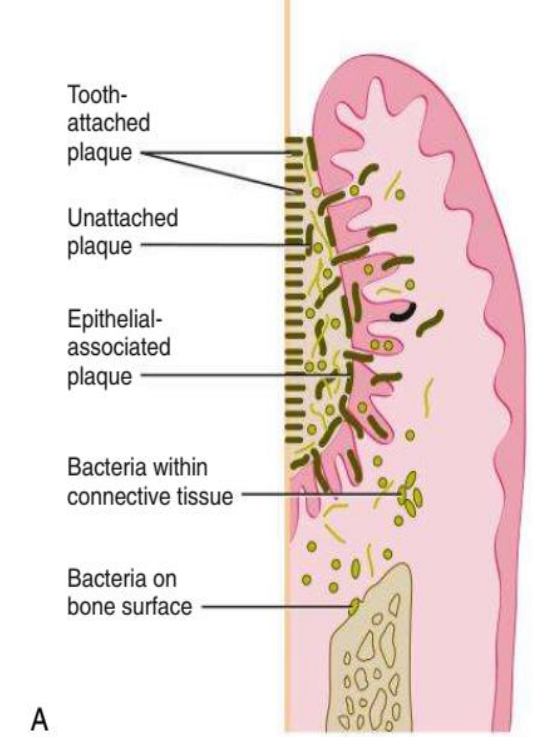
From a microbiological viewpoint, **teeth and implants** are unique for **two reasons**:

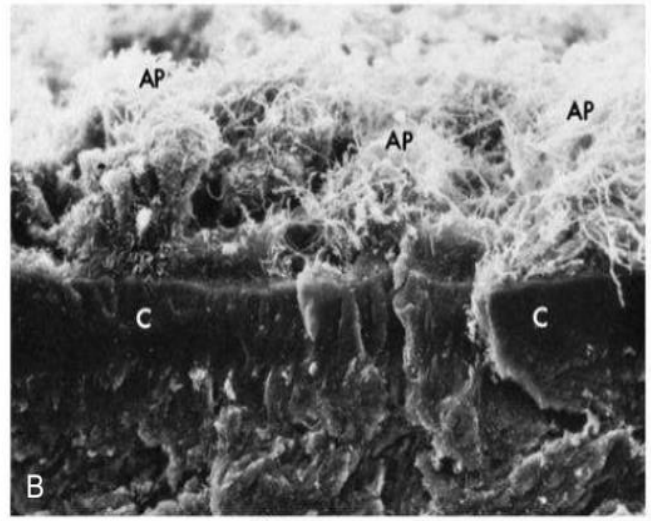
(1) They provide a hard, **non-shedding** surface that allows for the development of extensive structured bacterial deposits

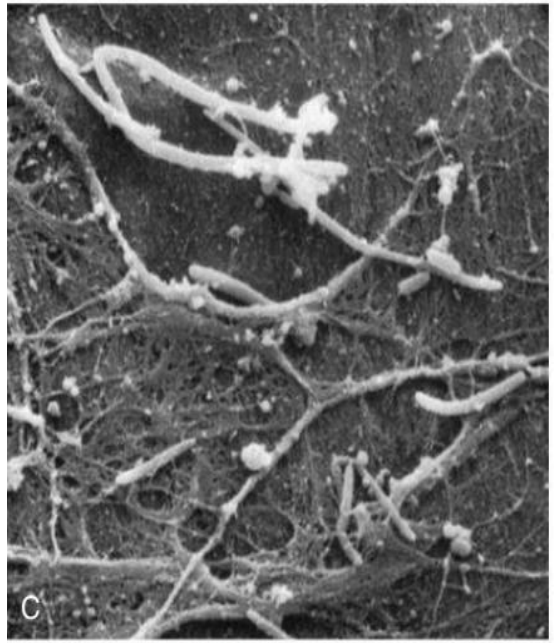
(2) They form a unique ectodermal interruption. A special seal of epithelium (**junctional epithelium**) and connective tissue is present between the external environment and the internal parts of the body.

- The accumulation and metabolism of bacteria on these hard surfaces are considered the **primary causes of caries, gingivitis, periodontitis, peri-implantitis, and, sometimes, bad breath.**

- In periodontitis patients, periodontal pockets form, creating a subgingival niche that increases the root surface area for microbial colonization, **resulting in a subgingival microbial biofilm.**







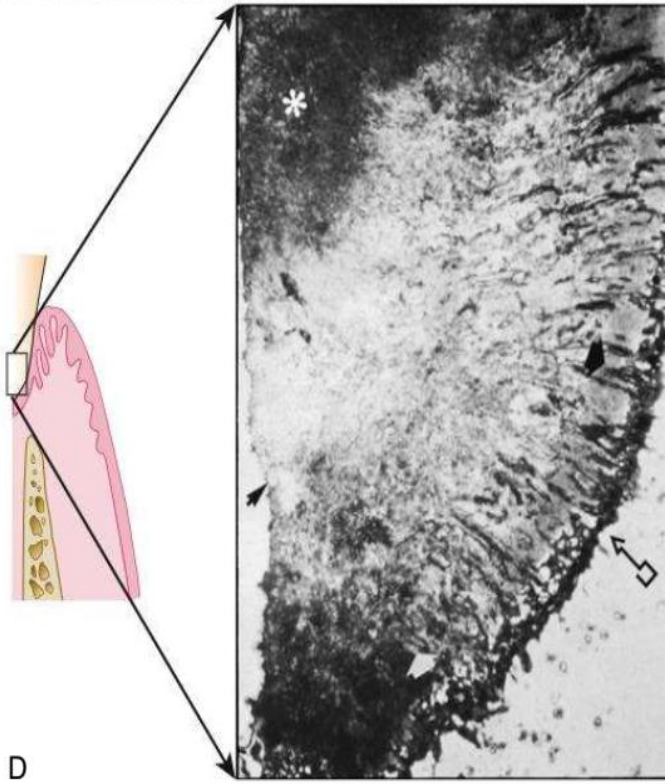


Fig. 10.2 Subgingival plaque. (A) Diagram depicting the plaque–bacteria association between the tooth surface and the periodontal tissues. (B) Scanning electron photomicrograph of a cross-section of cementum *(C)* with attached subgingival plaque *(AP)*. The area shown is within a periodontal pocket. (C) Scanning electron micrograph of cocci and filaments associated with the surface of pocket epithelium in a case of marginal gingivitis. ×3000. (D) *Left*, Diagrammatic representation of the histologic structure of subgingival plaque. *Right*, Histologic section of subgingival plaque. *Arrow with box*, **Sulcular epithelium**. *White arrow*, Predominantly gram-negative unattached zone. *Black arrow*, Tooth surface. *Asterisk*, Predominantly gram-positive attached zone. (B, Courtesy Dr. J. Sottosanti, La Jolla, California.)

- Bacterial species identifed in all sites investigated by Aas and coworkers belonged to the genera **Gemella, Granulicatella, Streptococcus, and Veillonella**.

- The most commonly found species in all subjects and all intra-oral sites investigated was **S. mitis**, which is considered a **pioneer species, or primary colonizer**.

- **Streptococci** are thought to be universally present in all sites within the mouth, but the distribution is thought to vary in different ecological niches of the oral cavity.

- It has been suggested that teeth are the primary habitat for periodontal pathogens because soon after a **full-mouth tooth extraction** in patients with severe periodontitis, key pathogens such as **Aggregatibacter actinomycetemcomitans and P. gingivalis disappeared** from the oral cavity, as determined by bacterial culturing techniques.

- **Prevotella intermedia** and other **black-pigmented Prevotella spp. remained**, but at lower detection frequencies and numbers.

The development and composition of the oral microbiome

- The mother is the main source of the oral microbiome in the newborn baby.
- The microbial composition of dental biofilms varies at distinct sites on a tooth (fissures, approximal surfaces, gingival crevice) due to inherent differences in their anatomy and biology (Papaioannou et al . 2009; Marsh et al . 2016b)
- **Fissures** are influenced by saliva and the diet and support a relatively sparse microbiota consisting of mainly saccharolytic Gram- positive bacteria, such as streptococci, while obligately anaerobic, and especially Gram- negative species, are rarely recovered.

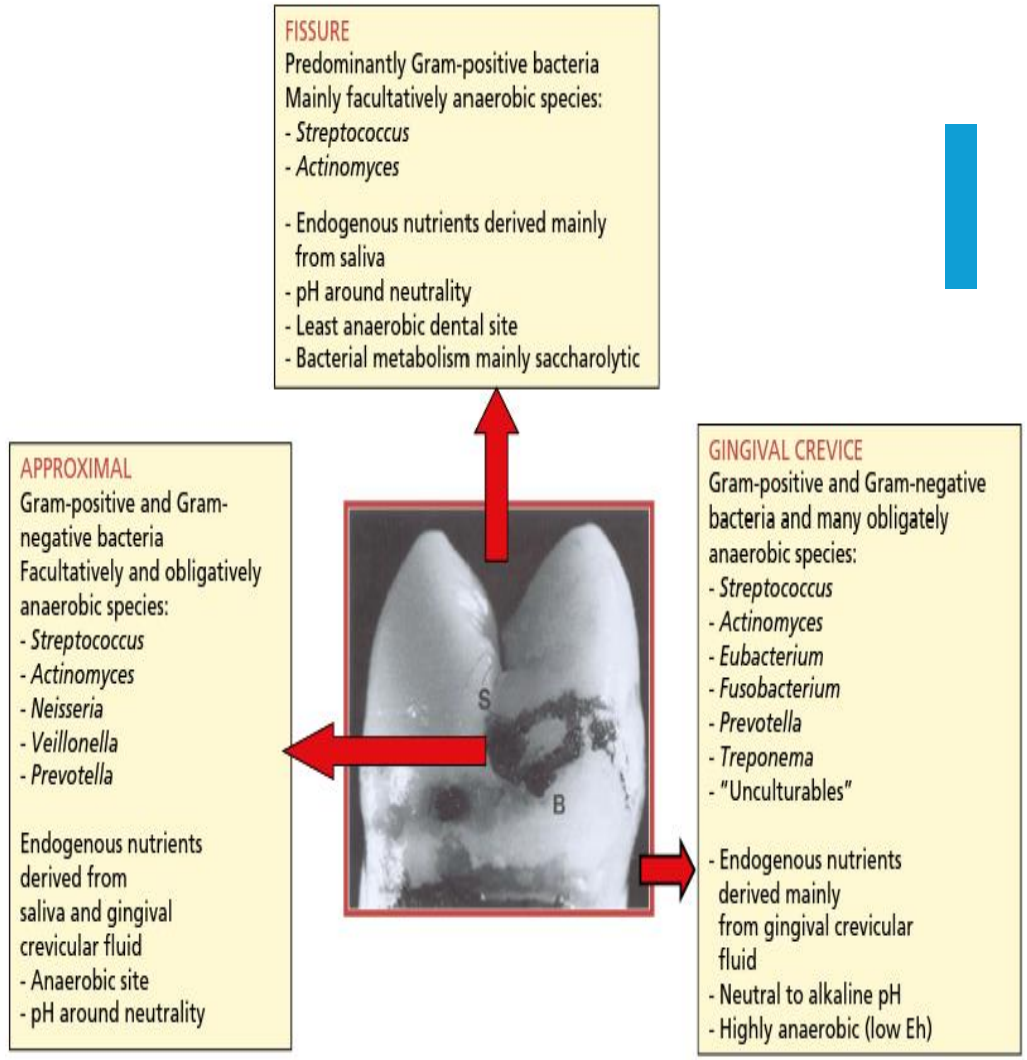
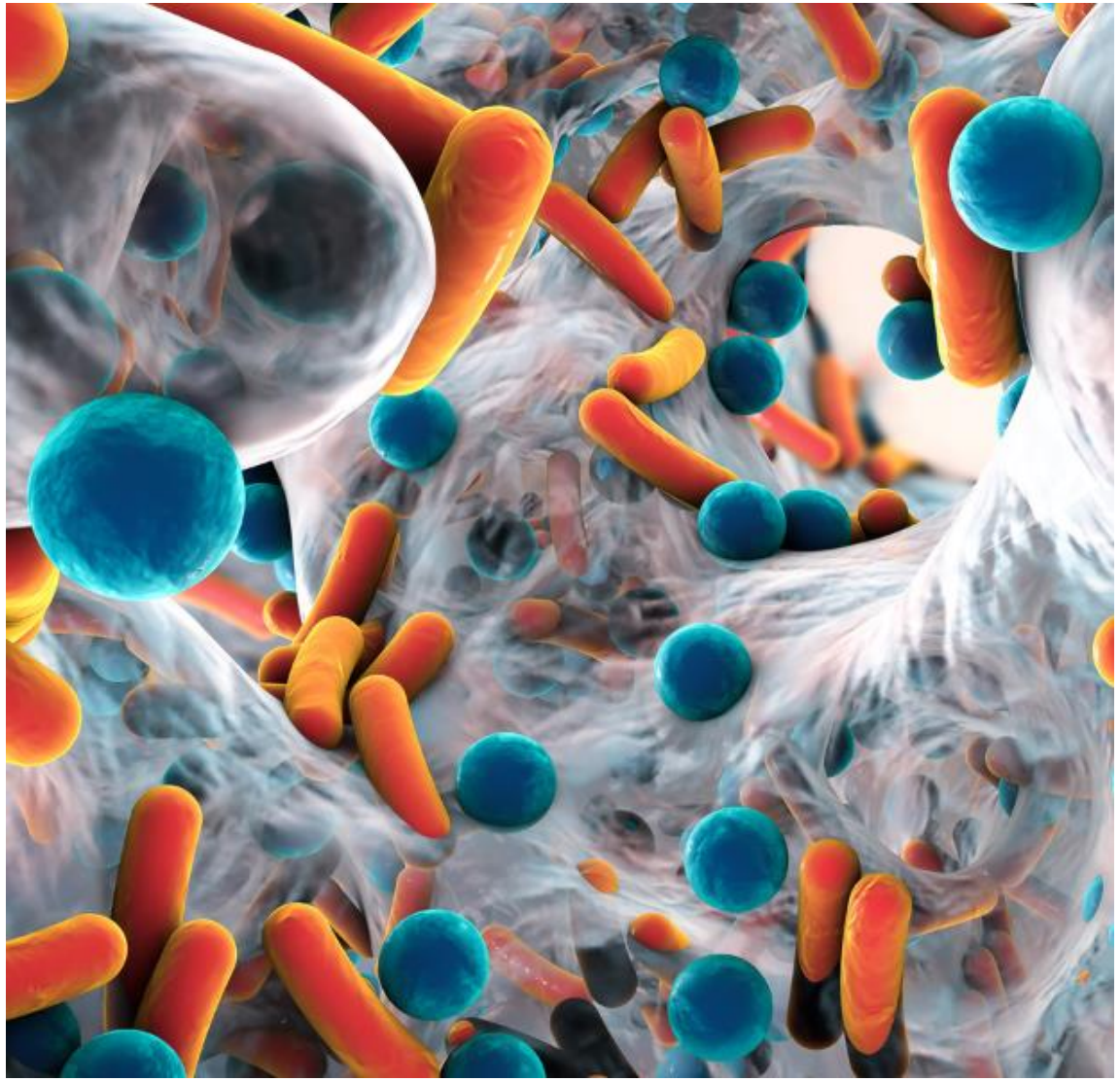


Fig. 8-2 Predominant groups of bacteria found at, and the key features of, distinct sites on the tooth surface.

Dental Biofilms

- Microbial populations on the surfaces of teeth are excellent examples of biofilm communities.
- The architecture of a dental biofilm has many features in common with other biofilms.
- It is **heterogeneous in structure**, with clear evidence of open fluid- filled channels running through the biofilm mass.

- **Nutrients reach the sessile (attached) microcolonies by diffusion through water channels to microbial microcolonies.**



- The process of dental biofilm formation can be divided into several phases:

  (1) The formation of the pellicle on the tooth surface

  (2) The initial adhesion/attachment of bacteria

  (3) Colonization/biofilm maturation

- The development of dental biofilms follows a well-established sequence of events where initial colonizers, predominantly streptococci, act as a foundation to establish an environment suitable for **later colonization by potentially more pathogenic species**

- Initially, **bacteria can be held reversibly near to the surface by weak**, long- range, physicochemical forces between the electrical charge on the molecules on the pellicle- coated surface and those on the microbial cell.

(1) The formation of the pellicle on the tooth surface

- **All surfaces** in the oral cavity, including hard and soft tissues, are coated with a layer of organic material known as the ***acquired pellicle.***

- **Microorganisms rarely colonize clean enamel.**

- Within seconds of eruption, or following cleaning, tooth surfaces become coated with a conditioning film (**acquired pellicle**) of molecules (biologically active proteins, phosphoproteins, and glycoproteins) **derived mainly from saliva (but also from GCF and bacteria)** (Hannig et al . 2005).

- ***The pellicle on tooth surfaces*** consists of more than 180 peptides, proteins, and glycoproteins, including keratins, mucins, proline-rich proteins, and other molecules that can function as adhesion sites (receptors) for bacteria.

2-Initial Adhesion/Attachment of Bacteria

- The interactions between microbial cell surface **"adhesin"** molecules and salivary pellicle receptors determine if a bacterial cell will stay on the surface.

- Only a relatively small proportion of oral bacteria possess adhesins that interact with receptors in the host pellicle. These species are considered the **"primary colonizers" of tooth surfaces**. The primary colonizers provide new binding sites for adhesion by other oral bacteria.

- Initial colonizers such as streptococci produce **lactic acid** which can be utilized as a carbon and energy source by Veillonella species.

- **Veillonella spp.** are amongst the most prevalent bacterial species in dental biofilms and are thought to have a similar role to oral fusobacteria (such as **Fusobacterium nucleatum** in that they are able to act as bridging organisms that are able to support the colonization and growth of later colonizers, **by their ability to bind to both early and late colonizers**

Colonization of teeth by bacteria occur in three phases

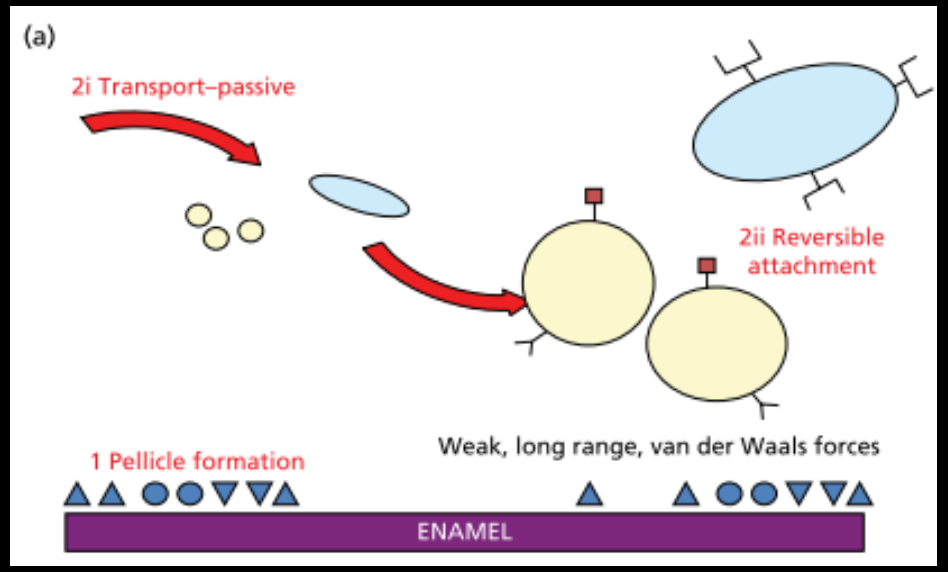
- **Phase 1 is transport to the tooth surface-** Brownian motion (average displacement, 40 μm/hour)
- **Phase 2 is initial reversible adhesion-** Van der Waals attractive forces and electrostatic repulsive forces
- **Phase 3 is strong attachment -** Proteins, Glycoproteins, or polysaccharides

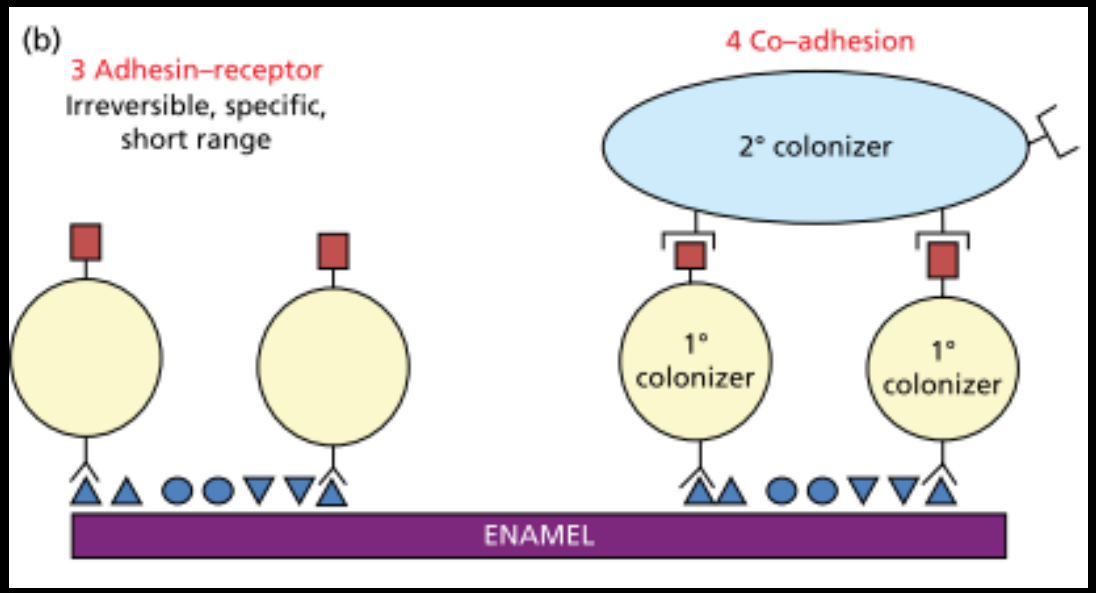
3- Colonization and Plaque Maturation

TABLE 10.1 Overview of Primary and Secondary Colonizers in Dental Plaque

Primary colonizers	*Streptococcus gordonii* *Streptococcus intermedius* *Streptococcus mitis* *Streptococcus oralis* *Streptococcus sanguinis* *Actinomyces gerencseriae* *Actinomyces israelii* *Actinomyces naeslundii* *Actinomyces oris* *Aggregatibacter actinomycetemcomitans serotype a* *Capnocytophaga gingivalis* *Capnocytophaga ochracea* *Capnocytophaga sputigena* *Eikenella corrodens* *Actinomyces odontolyticus* *Veillonella parvula*
Secondary colonizers	*Campylobacter gracilis* *Campylobacter rectus* *Campylobacter showae* *Eubacterium nodatum* *Aggregatibacter actinomycetemcomitans serotype b* *Fusobacterium nucleatum* spp. *nucleatum* *Fusobacterium nucleatum* spp. *vincentii* *Fusobacterium nucleatum* spp. *polymorphum* *Fusobacterium periodonticum* *Parvimonas micra* *Prevotella intermedia* *Prevotella loescheii* *Prevotella nigrescens* *Streptococcus constellatus* *Tannerella forsythia* *Porphyromonas gingivalis* *Treponema denticola*

- The primary colonizing bacteria adhered to the tooth surface provide new receptors for attachment by other bacteria, in a process known as **“coadhesion” or “coaggregation”.**
- The transition from early supragingival dental biofilm to more mature biofilm developing beneath the gingival margin involves a shift in the microbial population from primarily **gram-positive** organisms to high numbers of **gram-negative** bacteria.
- A key organism in dental biofilm development is **Fusobacterium nucleatum** . This species can co- adhere to most oral bacteria and acts as an **important bridging organism between early and later colonizing species.**





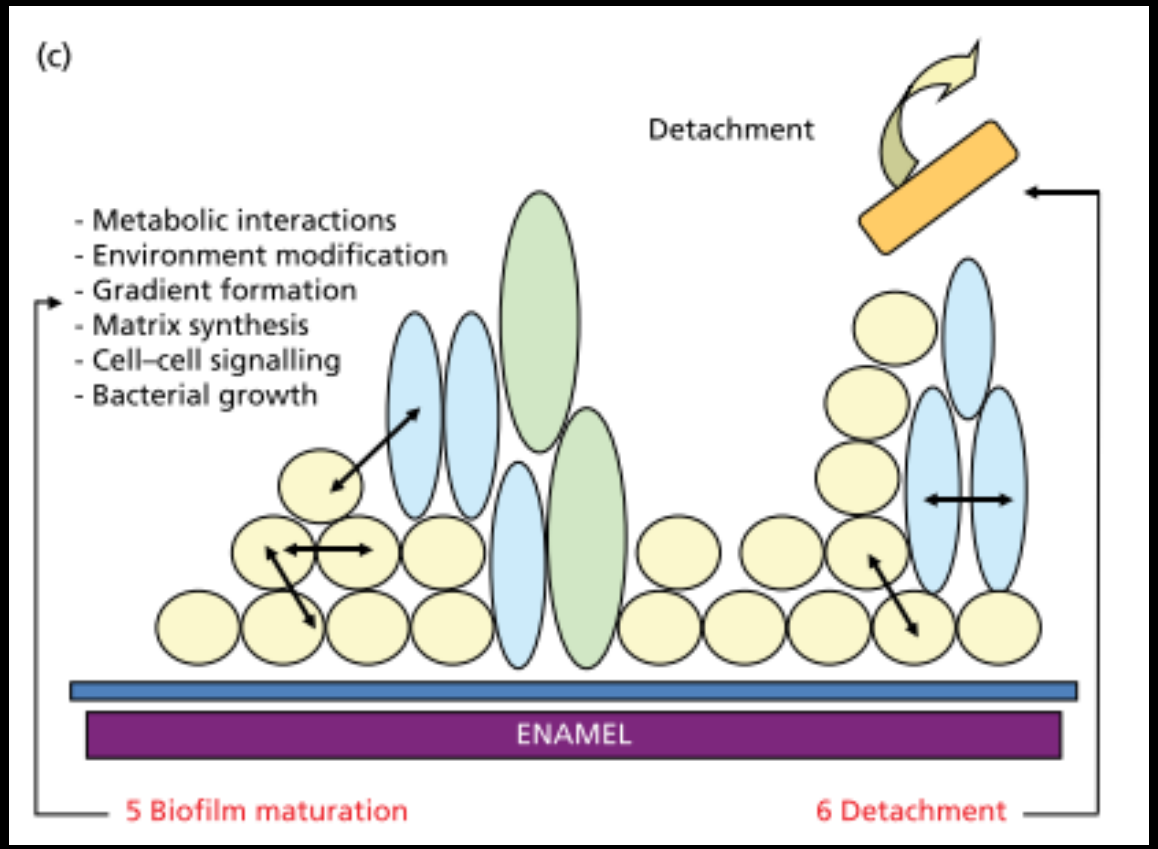
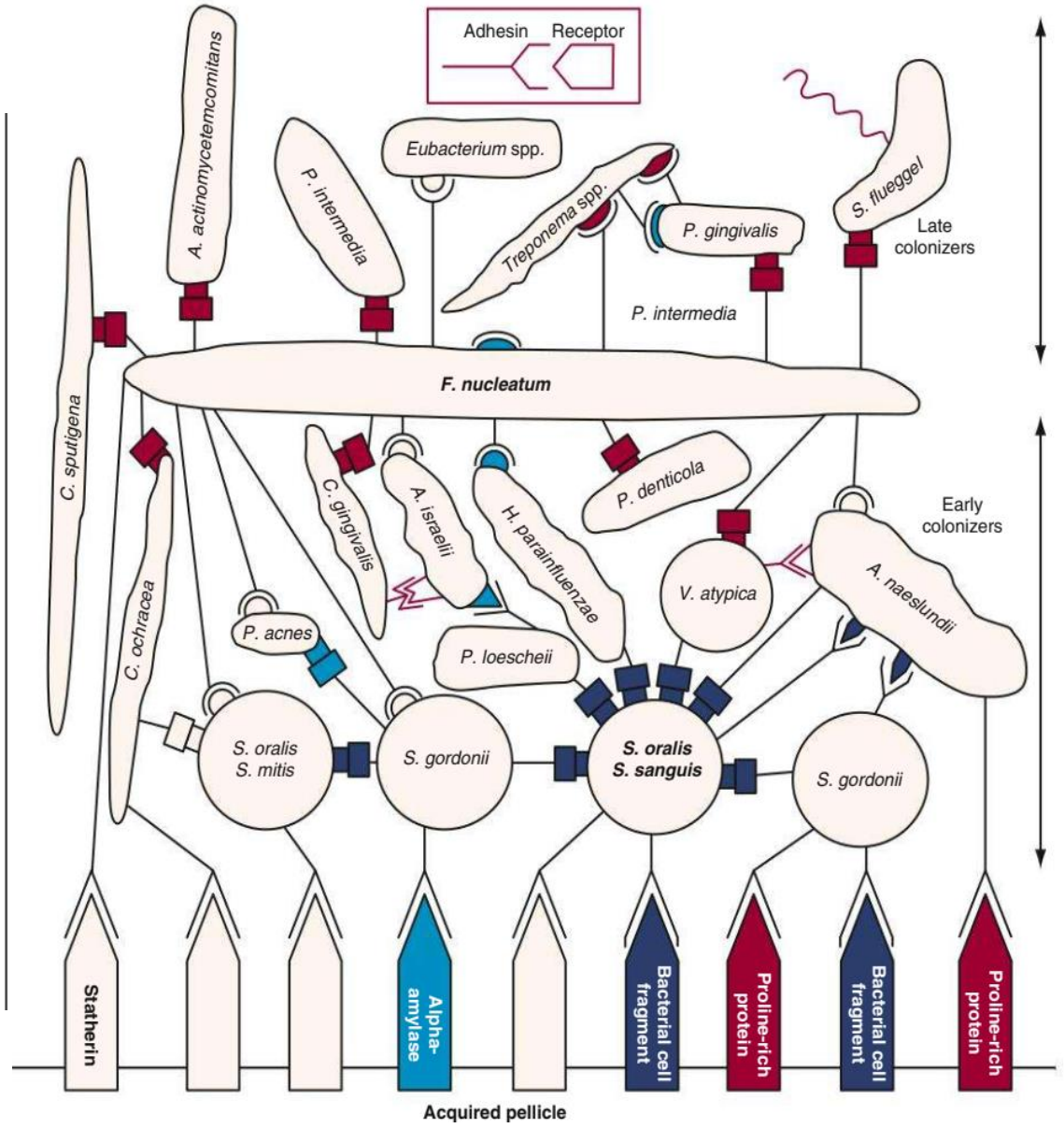
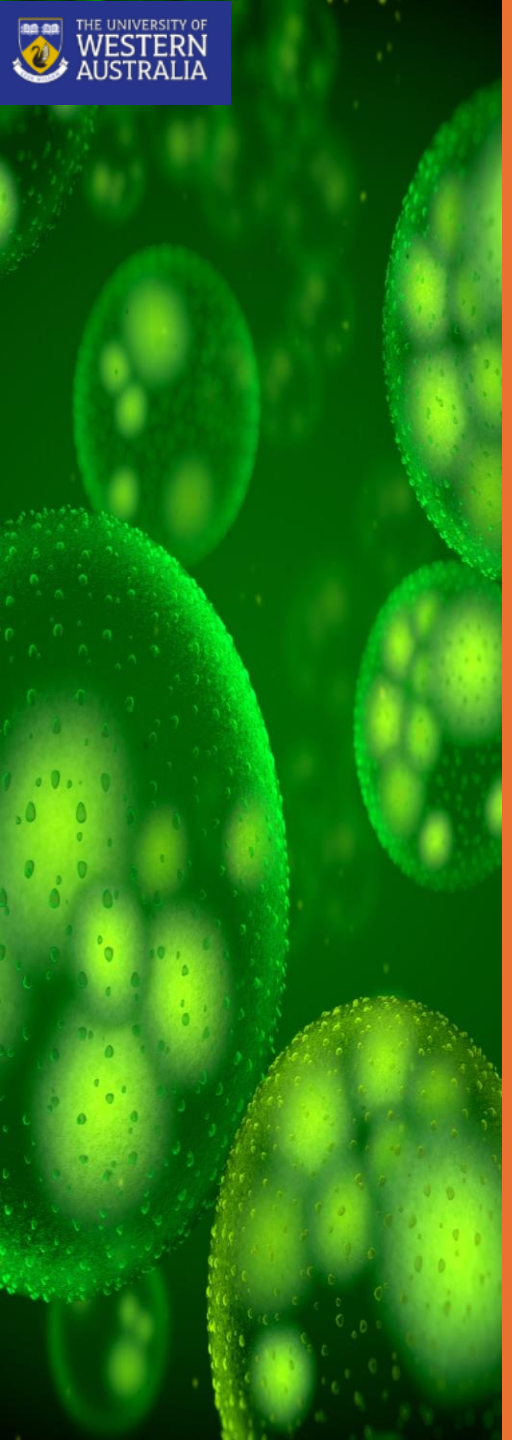


Fig. 10.10 Diagrammatic representation of initial plaque formation. Early colonizers bind to receptors in the pellicle. Each adherent cell becomes in turn the nascent surface and bridge for additional species (secondary colonizers). The complementary sets of adhesin receptor symbols (example in box) represent the various kinds of coaggregations as well as the interactions with molecules in the pellicle. The symbol with a stem (adhesin) represents a cellular component that is heat-inactivated (cell suspension heated to 85°C for 30 minutes) and sensitive to protease treatment. The cell type that exhibits the complementary symbol (receptor) is insensitive to either treatment. The symbols with a rectangular shape represent lactose-inhibitable coaggregations; the others are lactose noninhibitable. *A. actinomycetemcomitans, Aggregatibacter actinomycetemcomitans; A. israelii, Actinomyces israelii; A. naeslundii, Actinomyces naeslundii; C. gingivalis, Capnocytophaga gingivalis; C. ochracea, Capnocytophaga ochracea; C. sputigena, Capnocytophaga sputigena; F. nucleatum, Fusobacterium nucleatum; H. parainfluenzae, Haemophilus parainfluenzae; P. acnes, Propionibacterium acnes; P. denticola, Prevotella denticola; P. gingivalis, Porphyromonas gingivalis; P. loescheii, Prevotella loescheii; S. flueggel, Selenomonas flueggei; S. gordonii, Streptococcus gordonii; S. mitis, Streptococcus mitis; S. oralis, Streptococcus oralis; S. sanguis, Streptococcus sanguis; V. atypica, Veillonella atypica.* (Adapted from Kolenbrander PE, London J. Adhere today, here tomorrow: oral bacterial adherence. *J Bacteriol.* 1993;175:3247.)





Communication Between Biofilm Bacteria

- Bacterial cells do not exist in isolation. In a biofilm, capacity to communicate with each other.

- One example of this is **“quorum sensing”**, in which signaling molecule that accumulates in the local en triggers a response such as a change in the expressi genes once they reach a critical threshold concentr

There are 2 types of signaling molecules of plaque b

- ***Peptides*** released **by gram-positive** organisms dur
- In Streptococcus mutans , quorum sensing is media **competence stimulating peptide (CSP)** (Li et al . 2
- **“Universal”** signal molecule ***autoinducer 2 (AI-2).***

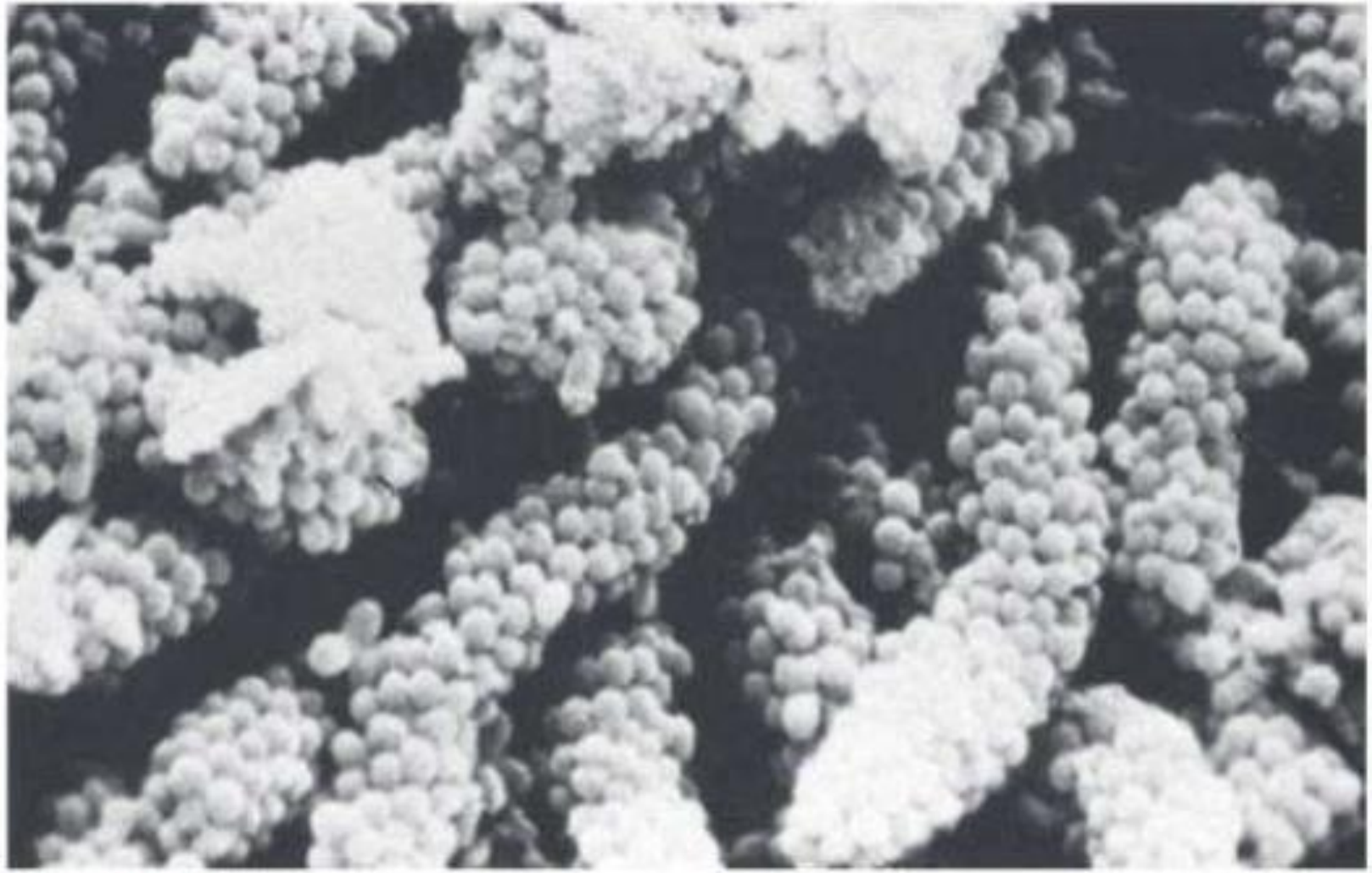


Fig. 10.11 Long-standing supragingival plaque near the gingival margin demonstrating a "corn cob" arrangement. A central gram-negative filamentous core supports the outer coccal cells, which are firmly attached by interbacterial adherence or coaggregation.

Table 1. The evolution of plaque hypotheses

Dates	Hypothesis	Main assumption
19th century	Golden age of microbiology	Specific pathogens are associated with systemic conditions (no oral pathogens found)
Early to mid-20th century	Non-specific plaque hypothesis	Periodontal disease is not caused by specific oral pathogens
1976	Specific plaque hypothesis	SEM technology may permit the identification of specific oral pathogens
1986	(back to) Non-specific plaque hypothesis	Overall activity of microbes could lead to disease through differences in virulence
1994	Ecological plaque hypothesis	Disease results from a microbial imbalance caused by ecological stress
2012	Keystone pathogen hypothesis	Certain low-abundance microbial pathogens cause inflammation by interfering with host immune response
2019	IMPEDE Model (Inflammation-Mediated-Polymicrobial-Emergence and Dysbiotic-Exacerbation")	Inflammation is the driver of the ultimate dysbiosis that leads to periodontitis rather than the pathogenic microbes themselves

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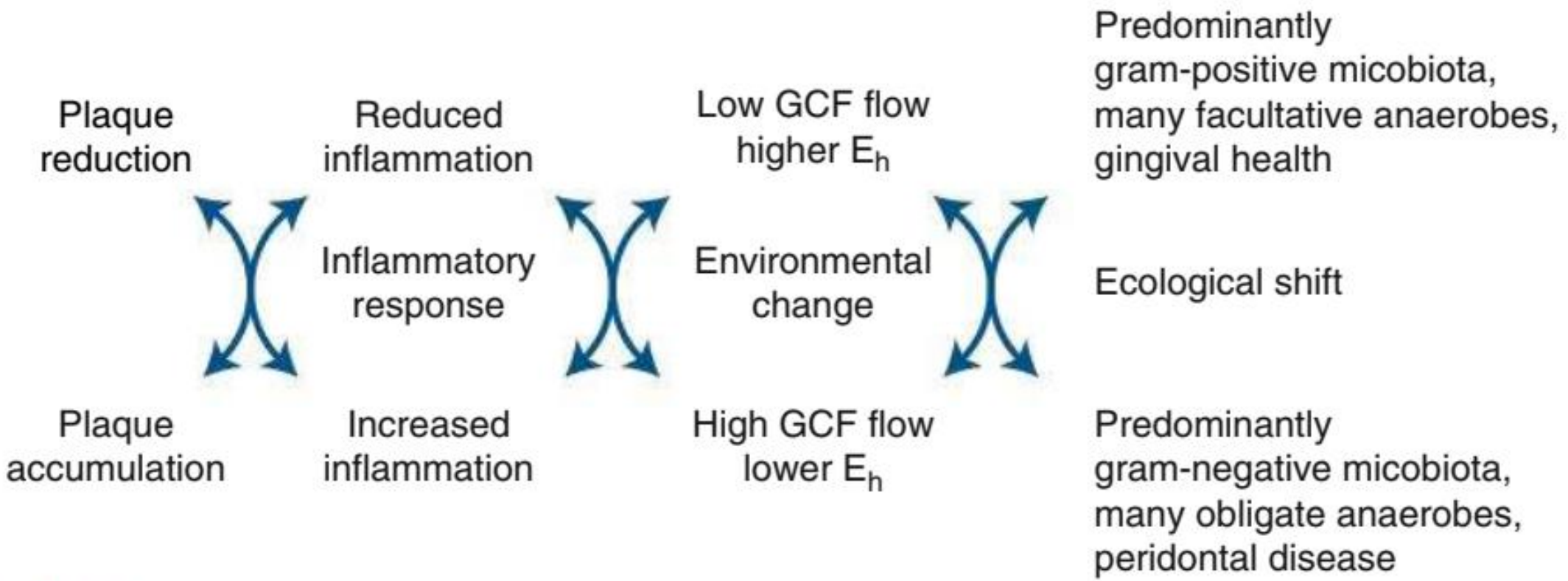


Fig. 10.25 Ecologic plaque hypothesis in relation to periodontal diseases: gingivitis and periodontitis. The accumulation of plaque causes the inflammation of adjacent tissues (gingivitis) and other environmental changes that favor the growth of gram-negative anaerobes and proteolytic species, including periodontal pathogens. The increased proportions of such species result in the destruction of periodontal tissues (i.e., periodontitis). E_h, Redox potential; *GCF*, gingival crevicular fluid. (Adapted from Marsh PD. Microbial ecology of dental plaque and its significance in health and disease. *Adv Dent Res*. 1994;8:263.)

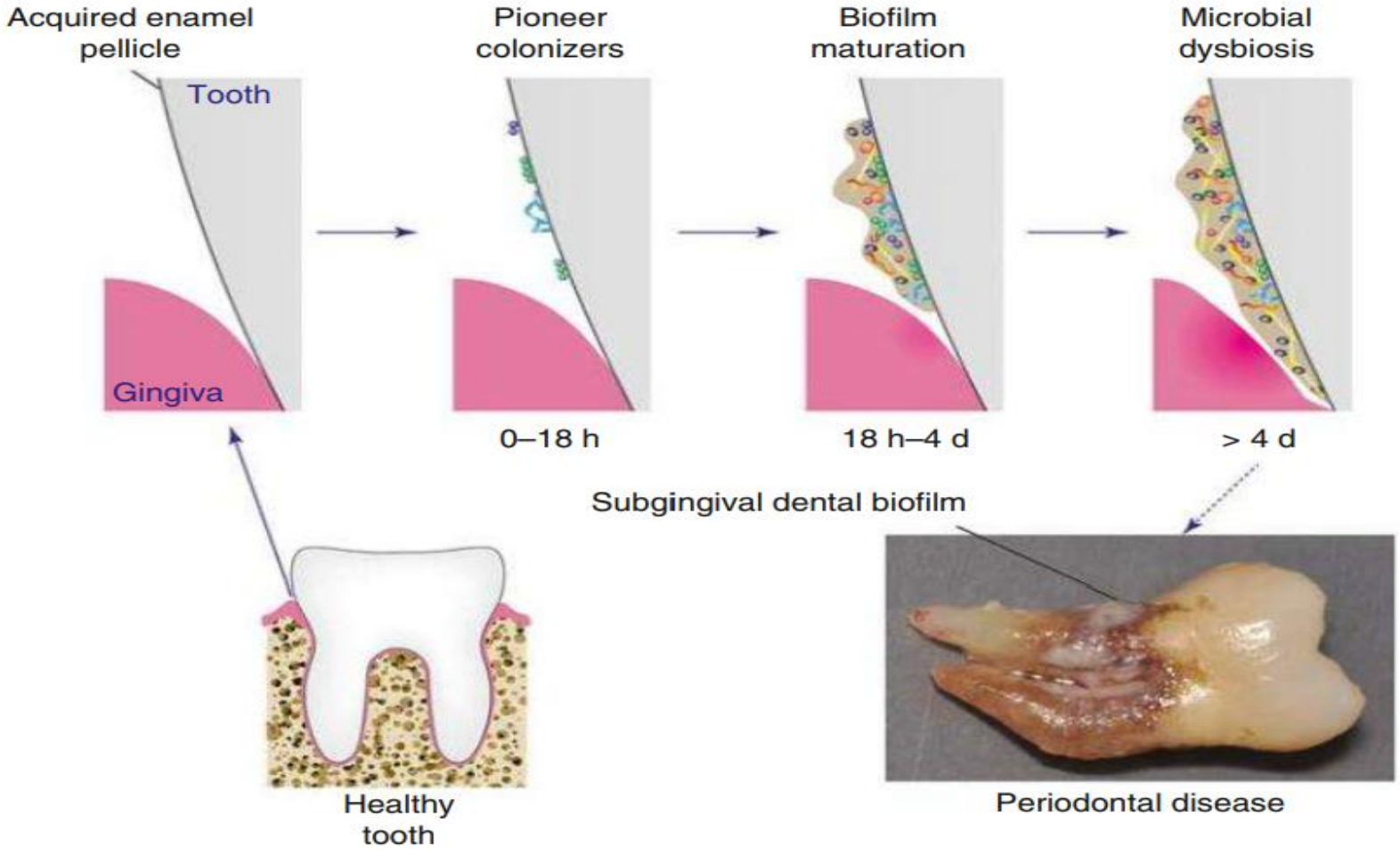
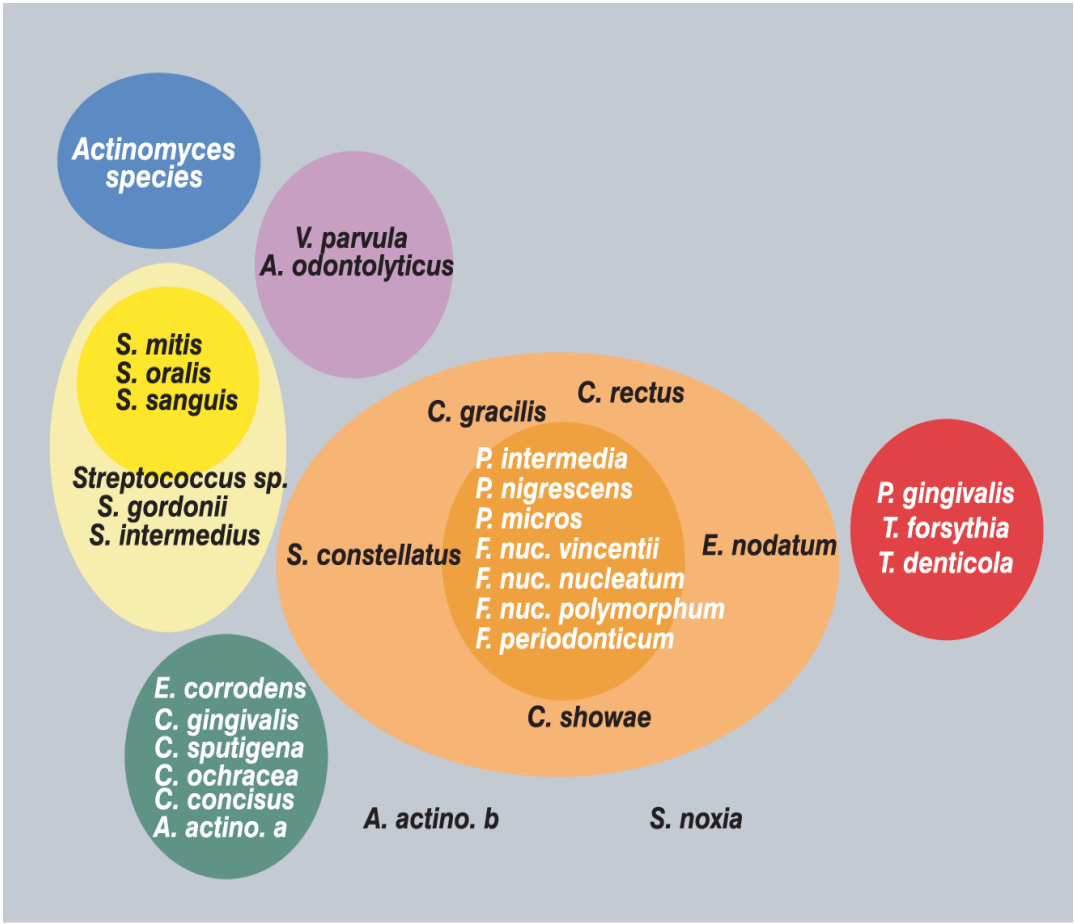


Fig. 10.7 Accumulation of dental biofilm and progression to disease. Good oral hygiene maintains low levels of dental biofilm at the gum margin. The acquired enamel pellicle, a layer of protein and glycoprotein largely derived from saliva, is not removed during tooth cleaning and forms attachment sites for pioneer colonizing bacteria. Initial attachment starts within minutes. Without further oral hygiene, the dental biofilm thickens and an extracellular matrix accumulates ("biofilm maturation") after around 18 h. This mature biofilm reorganizes over the next few days, without dramatically increasing in thickness. Interactions between the biofilm and gingival tissue triggers inflammation, indicated by reddening of the gingiva. Ultimately, this can lead to microbial dysbiosis and the accumulation of significant biofilm below the gingival margin which leads to chronic inflammation and loss of tissue surrounding the tooth. Ultimately, the biofilm-laden tooth becomes mobile and requires extraction. A tooth extracted for periodontal disease is shown.

Fig. 10.24 Associations among subgingival species. The data were derived from 13,261 subgingival plaque samples taken from the mesial aspect of each tooth in 185 adult subjects. Each sample was individually analyzed for the presence of 40 subgingival species with the use of checkerboard DNA–DNA hybridization. Associations were sought among species via cluster analysis and community ordination techniques. The complexes to the left consist of species that are thought to colonize the tooth surface and to proliferate at an early stage. The orange complex becomes numerically dominant later; it is thought to bridge the early colonizers and the red complex species, which become numerically more dominant during the later stages of plaque development. *A. actinomycetemcomitans, Aggregatibacter actinomycetemcomitans; A. odontolytica, Actinomyces odontolytica; C. concisus, Campylobacter concisus; C. gingivalis, Capnocytophaga gingivalis; C. gracilis, Campylobacter gracilis; C. ochracea, Capnocytophaga ochracea; C. rectus, Campylobacter rectus; C. showae, Campylobacter showae; C. sputigena, Capnocytophaga sputigena; E. corrodens, Eikenella corrodens; F. nuc., Fusobacterium nucleatum; F. periodonticum, Fusobacterium periodonticum; P. gingivalis, Porphyromonas gingivalis; P. micra, Parvimonas micra; P. nigrescens, Prevotella nigrescens; S. constellatus, Streptococcus constellatus; S. mitis, Streptococcus mitis; S. noxia, Selenomonas noxia; S. oralis, Streptococcus oralis; S. sanguis, Streptococcus sanguis; T. denticola, Treponema denticola; T. forsythia, Tannerella forsythia; V. parvula, Veillonella parvula.* (Adapted from Socransky SS, Haffajee AD, Cugini MA, et al. Microbial complexes in subgingival plaque. *J Clin Periodontol.* 1998;25:134.)



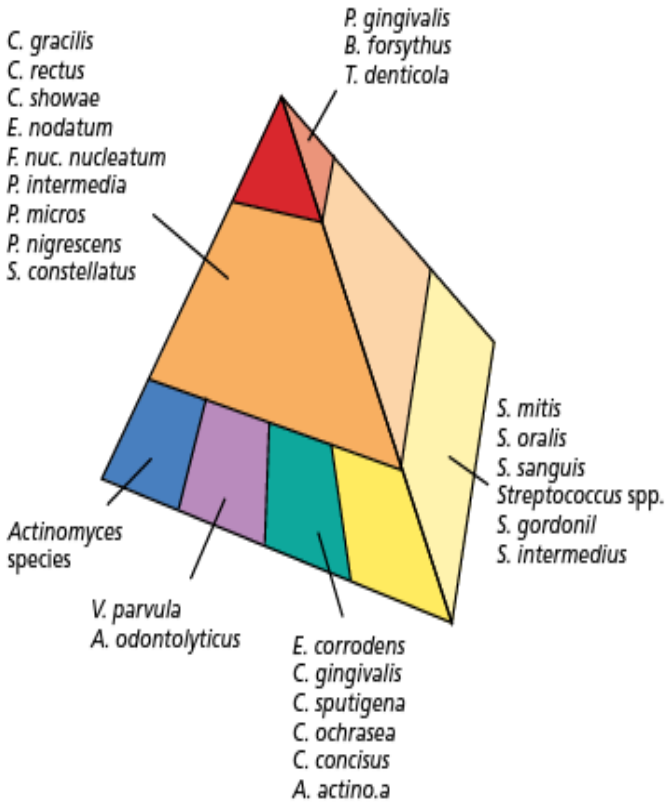


Fig. 9-4 The association among subgingival species. The different colors in the pyramid represent different bacterial complexes which are frequently detected in association with one another. The base of the pyramid represents the early stage of plaque development whereas the apex contains those organisms thought to be the last species to become established in the microbiota. The red complex of bacteria are those organisms frequently associated with sites of periodontal disease. (Source: Reprinted from Socransky & Haffajee 2002, with permission.)

- Certain bacterial species have been proposed to be protective or beneficial to the host, including ***S. sanguis, Veillonella parvula***, and ***C. Ochraceus***

- ***S. sanguis*** produce of H_2O_2 by; H_2O_2 is known to be lethal to cells of *A. actinomycetemcomitans*.

- **Aggregatibacter actinomycetemcomitans** , the bacterium associated with rapidly progressive disease (molar Incisor pattern) in individuals of West African descent, **does not cluster with** the most disease-associated **red complex organisms.**

- The species most associated with periodontitis were **P. gingivalis, T. denticola, T. forsythia** (the three red complex species), and **Filifacor alocis**, a newly identified pathogen.

- **Orange complex** and **few green complex** bacteria are Gingivitis associated bacteria.

TABLE 10.2 Differences Between Tooth Deposits

Materia Alba	Dental Plaque	Calculus
• White, cheeselike accumulation	• Resilient clear to yellow-grayish substance	• Hard deposit that forms via the mineralization of dental plaque
• Soft accumulation of salivary proteins, some bacteria, many desquamated epithelial cells, and occasional disintegrating food debris	• Primarily composed of bacteria in a matrix of salivary glycoproteins and extracellular polysaccharides	• Generally covered by a layer of unmineralized dental plaque
• Lacks an organized structure and is therefore not as complex as dental plaque	• Considered to be a biofilm	
• Easily displaced with a water spray	• Impossible to remove by rinsing or with the use of sprays	

Dental calculus

- Dental calculus or tartar represents **mineralized bacterial biofilm**, although calculus formation can be induced in germ- free animals because of precipitation of mineral salts **originating from saliva.**

- As the mineral content of dental biofilms increases, the biofilm mass becomes calcified to form calculus **Calculus** is frequently found in areas of the dentition adjacent to salivary ducts (e.g., the lingual surface of the mandibular incisors and canines, the buccal surface of the maxillary first molars), and this reflects.

- **The high concentration of minerals available from saliva in those regions**.

- The inorganic components of subgingival dental biofilms are derived from crevicular fluid (a serum transudate).

- It should be noted that calculus continually harbors a viable bacterial biofilm.

Clinical appearance and distribution

- Supragingival calculus appears as a **creamy- whitish to dark yellow** or even brownish mass of moderate hardness.

- The degree of calculus formation is not only dependent on the amount of bacterial biofilm present, but also on the secretion of the salivary glands.

- Hence, supragingival calculus is predominantly found adjacent to the excretion ducts of the major salivary glands, such as the **lingual aspect of the mandibular anterior teeth** for the submandibular glands and the **buccal aspect of the maxillary first molars**, whereas the parotid gland ducts open into the oral vestibule.

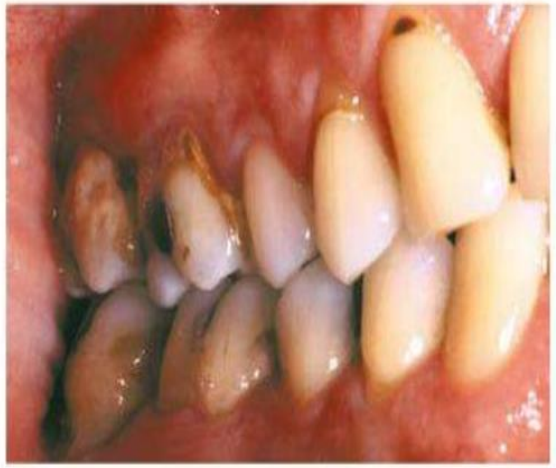


Fig. 24.1 Supragingival calculus is depicted on the buccal surfaces of maxillary molars adjacent to the orifice for the parotid duct.

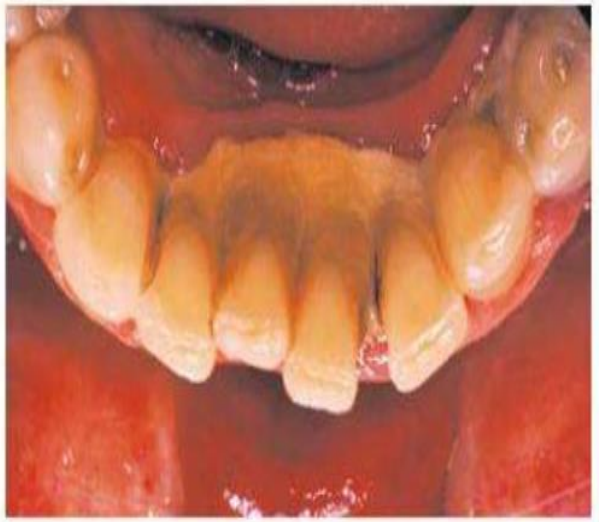
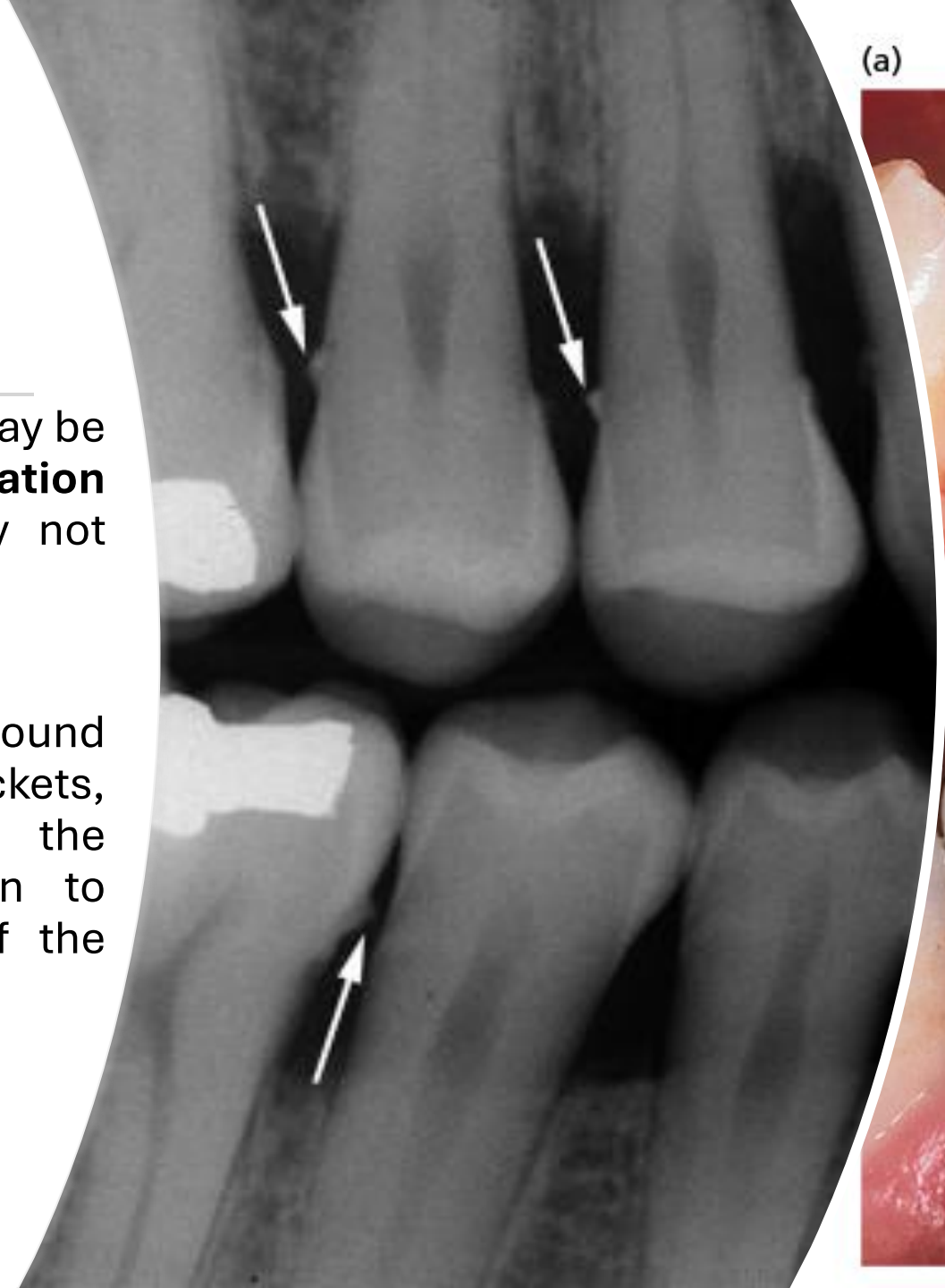


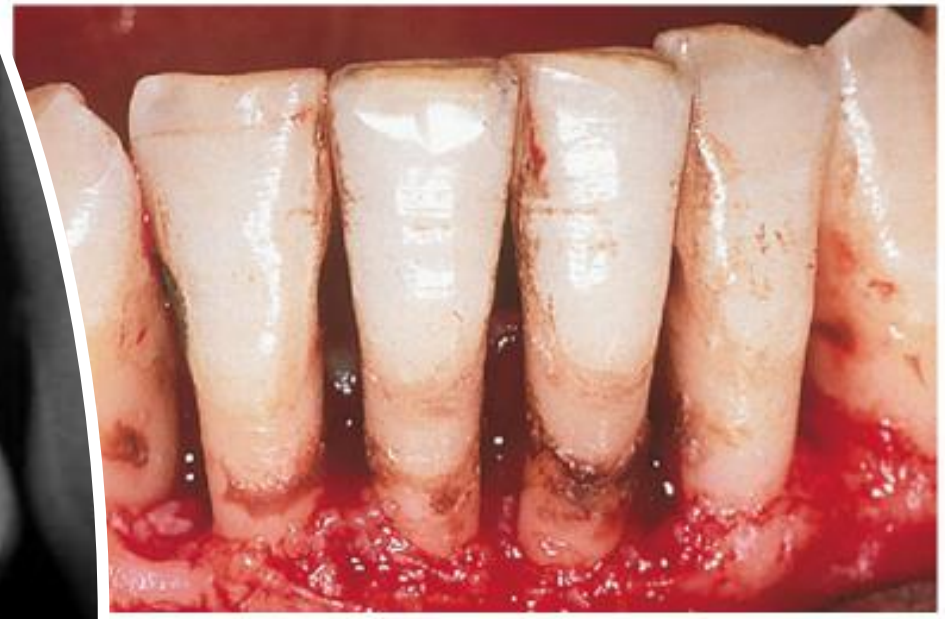
Fig. 24.2 Extensive supragingival calculus is present on the lingual surfaces of the lower anterior teeth.

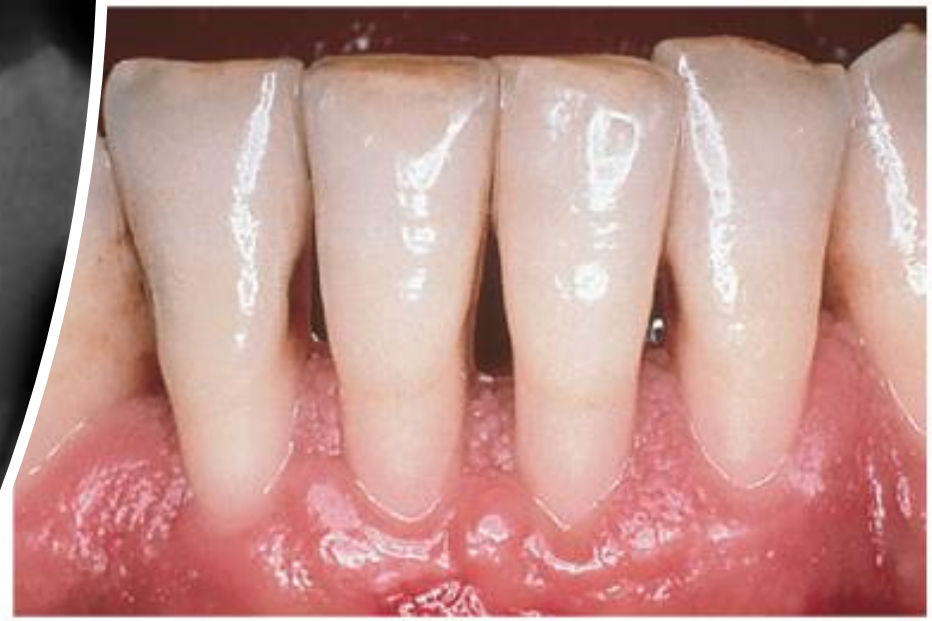
- Subgingivally, calculus may be found by **tactile exploration** only, since it is usually not visible to the naked eye.

- Subgingival calculus is found in most periodontal pockets, usually extending from the cementoenamel junction to close to the bottom of the pocket.



(a)





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Effect of iatrogenic factors on periodontal health: An epidemiological study

Kankara Vinathi Reddy [a,*], Chembolu Nirupama [a], Pathakota Krishnajaneya Reddy [a], Pradeep Koppolu [b], Dalal H. Alotaibi [c]

Conclusion: This study clearly identified a higher prevalence, 50.8% of sub-gingival restorations causing gingivitis and has shown significant influence on periodontal status of the tooth.

Composition

Inorganic Content

- Dental calculus is primarily composed of inorganic components **(70% to 90%)** and the organic components constitute the rest.

The major inorganic proportions of calculus are approximately

- **76% calcium phosphate ($Ca_3[PO_4]_2$)**
- **3% calcium carbonate ($CaCO_3$)**
- **4% magnesium phosphate ($Mg_3[PO_4]_2$)**
- **2% carbon dioxide**
- Traces of other elements such as sodium, zinc, strontium, bromine, copper, manganese, tungsten, gold, aluminum, silicon, iron, and fluorine.

- The calcium concentration or content in plaque is **2 to 20 times higher than in saliva.**

- The time required to reach the maximal level has been reported between **10 weeks & 6 months**

TABLE 24.1 Calculus Versus Other Oral Hard Tissues

Structure	Inorganic Content (%)[a]
Dental calculus	70–90
Enamel	96
Dentin	45
Bone	60–70

[a]Organic components and water constitute the rest.

- Formation
- **Calculus** is mineralized dental plaque.
- The soft plaque is hardened by the precipitation of mineral salts, which usually starts between the **1st and 14th day of plaque formation**.
- Calcification has been reported to occur within as little as 4 to 8 hours.
- Calcifying plaques may become **50% mineralized in 2 days and 60% to 90% mineralized in 12 days.**

KEY FACT

Calculus by itself does not contribute directly to gingival inflammation. Like other retentive factors, such as open crown margin or an overhanging restoration, calculus retains dental plaque, which contributes to gingival inflammation.

Attachment to tooth surfaces and implants

- **Four** modes of attachment have been described.

(1) Attachment by means of an organic pellicle on cementum is depicted in Fig. 24.6

(2) Mechanical locking into surface irregularities, such as caries lesions or resorption lacunae, is illustrated in Fig. 24.8.

(3) Close adaptation of the undersurface of calculus to depressions or gently sloping mounds of the unaltered cementum surface is shown in Fig. 24.9.

(4) Penetration of bacterial calculus into cementum

The reason for firm attachment to the tooth surface is the fact that the **pellicle beneath the bacterial biofilm also calcifies.** This, in turn, results in an intimate contact with enamel, cementum or dentin crystals

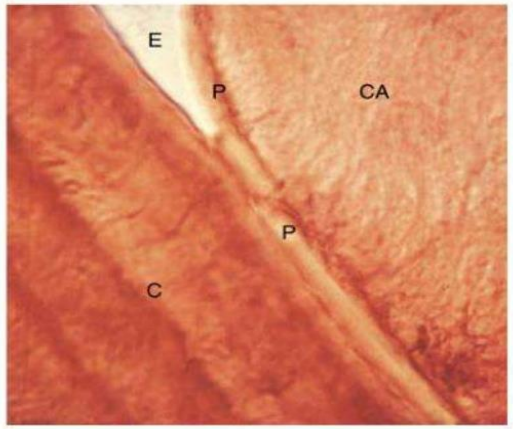

Fig. 24.6 Calculus attached to the pellicle on the enamel surface and the cementum. An enamel void *(E)* has been created during the preparation of the specimen. *C*, Cementum; *CA*, calculus; *P*, pellicle. (*Courtesy Dr. Erwin Schaffer, Minneapolis, MN.*)

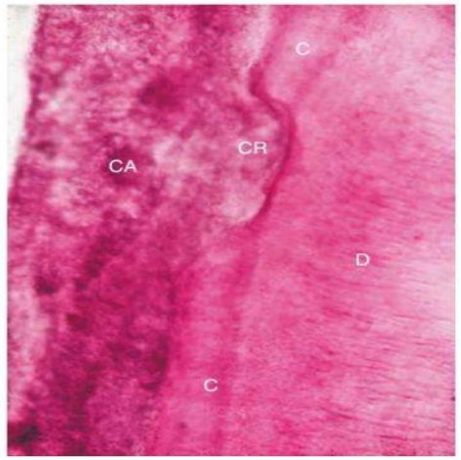

Fig. 24.8 Calculus *(CA)* attached to a cemental resorption area *(CR)* with cementum *(C)* adjacent to dentin *(D)*. (*Courtesy Dr. Erwin Schaffer, Minneapolis, MN.*)

Fig. 24.9 Undersurface of subgingival calculus *(C)* previously attached to the cementum surface *(S)*. Note the impression of cementum mounds in the calculus *(arrows)*. (*Courtesy Dr. John Sottosanti, La Jolla, CA.*)

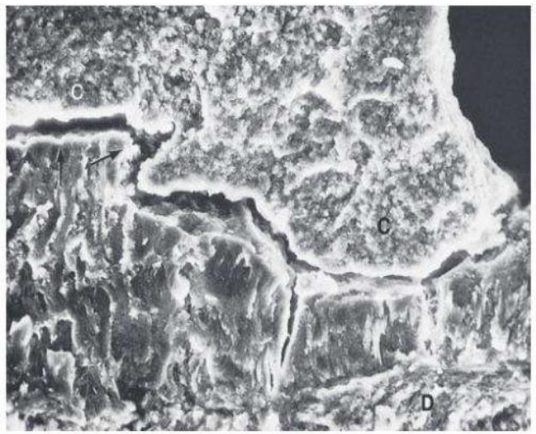

Fig. 24.10 Subgingival calculus *(C)* embedded beneath the cementum surface *(arrows)* and penetrating to the dentin *(D)*, thereby making removal difficult. (*Courtesy Dr. John Sottosanti, La Jolla, CA.*)

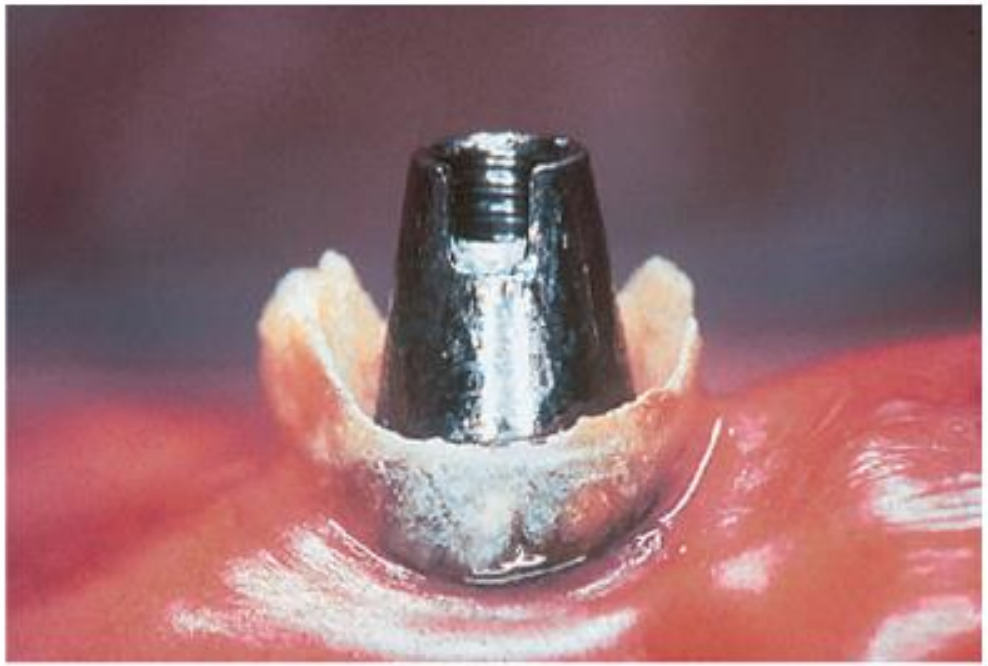

Fig. 8-22 Calculus deposit on an oral implant in a patient without regular maintenance care.

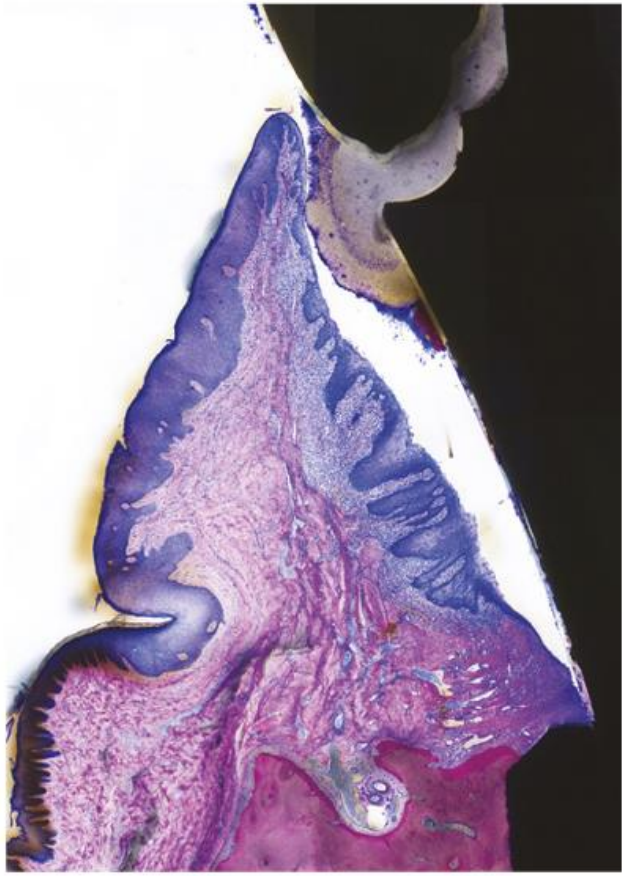

Fig. 8-23 Excess cement at the abutment–crown interface provides an ideal substrate for biofilm and calculus deposition and retention. Bacterial biofilm covers the entire surface of the cement, whereas calculus is present apical to the cement overhang. Detachment of the epithelium indicates pocket formation. The detachment of the apical-most portion of the epithelium, however, may represent an artifact due to histologic processing. Undecalcified ground section.

Conclusion

- Dental calculus represents mineralized bacterial biofilm. It is always covered by unmineralized viable bacterial biofilm, and hence, does not directly come into contact with the gingival tissues.

- Calculus, therefore, is a secondary etiologic factor for periodontitis. Its presence, however, makes adequate biofilm removal impossible and prevents patients from performing proper biofilm control.

- It is the most prominent biofilm- retentive factor that has to be removed as a basis for adequate periodontal and peri- implant therapies and prophylactic activities.

References

CHAPTER 24

The Role of Dental Calculus and Other Local Predisposing Factors

Vivek Thumbigere-Math | James E. Hinrichs

CHAPTER OUTLINE

Calculus
- Supragingival and Subgingival Calculus
- Prevalence
- Composition
- Attachment to the Tooth Surface
- Formation
- Etiologic Significance

- Materia Alba, Food Debris, and Dental Stains

Other Predisposing Factors
- Iatrogenic Factors
- Margins of Restorations
- Malocclusion

- Periodontal Complications Associated With Orthodontic Therapy
- Extraction of Impacted Third Molars
- Habits and Self-Inflicted Injuries
- Smokeless Tobacco
- Radiation Therapy

Chapter 8

Dental Biofilms and Calculus

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CHAPTER 10

Biofilm and Periodontal Microbiology

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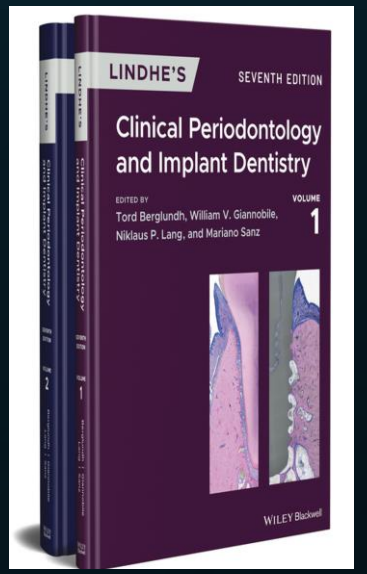

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