Etiology and Pathogenesis of Periodontal Disease
Alexandrina L. Dumitrescu

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With Contributions by

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Springer
The important thing is not to stop questioning. Curiosity has its own reason for existing.

Albert Einstein, 1879–1955
In his 1968 book, *General Systems Theory: Foundations, Development, Applications*, Ludwig von Bertalanffy observed that “… science is split into innumerable disciplines continually generating new subdisciplines. In consequence, the physicist, the biologist, the psychologist and the social scientist are, so to speak, encapsulated in their private universes, and it is difficult to get word from one cocoon to the other….”

The same might have been true about subdisciplines within the fields of medicine and dentistry as well. But times are changing. The concept that oral diseases and disorders reflect and affect overall health has been gaining wide acceptance, especially over the past decade. As an illustration, a quick search of PubMed’s electronic database of biomedical journals yields approximately 225 research and review articles published any time before 1980 that mention both periodontal diseases and cardiovascular diseases; in just the last 10 years, that number has nearly quadrupled.

We are seeing an ever-increasing amount of research that links periodontal disease to an astonishingly large and diverse set of systemic health outcomes other than cardiovascular diseases. These include low birth weight, osteoporosis, diabetes, cognitive decline, obesity, and others. We are gaining a better understanding of the roles that pathogenic bacteria, the intra-oral media in which they thrive, and the local and systemic immunologic responses they elicit play in the etiologies of periodontal and systemic diseases. We are also gaining insights into the nature of interactions between periodontal disease and other intra-oral conditions and treatments. Since many of these associations are bidirectional, uncovering the true cause and effect relationships presents methodological challenges, and so it is not surprising to find conflicting reports and opinions.

This volume represents a truly comprehensive update and critical review of the complex interrelationships of periodontal diseases with our total health and well-being. Individual chapter topics cover disease microbiology and etiology; genetic, chronic systemic disease, and psychological factors; effects of periodontal disease and treatments on restorative and endodontic outcomes, and the impacts of malocclusion and orthodontic intervention. As these chapters illustrate, we are talking to one another, and this collection of papers will serve as an important resource for researchers and providers interested in the causes, prevention, and treatment of periodontal disease.

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The past decade has witnessed a remarkable growth of knowledge concerning the etiology and pathogenesis of periodontal disease. Biologic processes, including the characteristics of the biofilm and of the host inflammatory and immune responses, tend to vary among individuals, despite producing a similar clinical picture or diagnostic category. Studies on the microbiota associated with periodontal disease have revealed a wide variety in the composition of the subgingival microflora. Other factors that may influence the biologic phenotype and clinical expression of disease include unique environmental exposures, psychological (behavioral) factors, as well as differences in genetic and possibly epigenetic composition.

A strong relationship between periodontal health or disease and systemic health or disease was also revealed. This means a two-way relationship in which periodontal disease in an individual may be a powerful influence on an individual’s systemic health or disease as well as the most customary understood role that systemic disease may have in influencing an individual’s periodontal health or disease. There is increasing evidence that individuals with periodontal disease may be at increased risk for adverse medical outcomes: mortality, cardiovascular disease, metabolic syndrome, diabetes mellitus, adverse pregnancy outcomes, respiratory disease, rheumatoid arthritis, renal disease, cancer, inflammatory bowel disease, Alzheimer disease, and osteoporosis.

In this book we propose an holistic view, by delineating the multiple systemic and local factors that contribute to the clinical presentation of periodontal disease in a specific individual: dental plaque, calculus, microbial composition, immune response, systemic diseases, behavioral determinants, genetic variants, and local factors that should allow a more accurate diagnosis of periodontal disease, prognosis, provide insight into the customized treatment for the periodontal patient, as well as the identification of individuals of high risk.

As Socransky et al. stated in 1987, the task of defining the etiological agents of periodontal disease is a cyclical process with continual re-evaluation and refinement. This book, dedicated to the science and practice of periodontology as a contribution to understand, treat, and prevent this disease, would be of interest to periodontists, undergraduate and postgraduate dental students, dental educators, and researchers.

Tromsø, Norway

Alexandrina L. Dumitrescu
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Dental plaque is a unique ecosystem. It is a microbial biofilm, a diverse microbial community found on the tooth surface embedded in a matrix of polymers of bacterial and salivary origin. The biofilm is a thin basal layer on the substratum, in contact with, and occasionally penetrating, the acquired enamel pellicle, and with columnar, mushroom-shaped multibacterial extensions into the lumen of the solution, separated by regions (“channels”), seemingly empty or filled with extracellular polysaccharide. The bacteria in a biofilm communicate with each other by sending out chemical signals. These chemical signals trigger the bacteria to produce potentially harmful proteins and enzymes. Several plaque models, as well as dental calculus localization, composition, morphology, formation, assessment, association with periodontal disease pathology, and anticalculus agents, are presented.

1.1 Dental Plaque

Dental plaque is a unique ecosystem. Several hundred bacterial species inhabit the human oral cavity (Tanner et al. 1998), and these multiple bacterial species form a community of dental plaque (Kolenbrander 2000; Okuda et al. 2004). Bacteria in periodontal pockets use gingival crevicular fluid as the nutrient source of carbon and nitrogen, as well as essential growth factors, such as minerals and vitamins. These bacteria then proliferate and communicate by signals to each other (Carlsson 2000; Palmer et al. 2001a; Okuda et al. 2004). In order to maintain the ecosystem, various anaerobes anchor to each other by forming aggregated bacterial masses (Kigure et al. 1995; Okuda et al. 2004). The regulation of bacterial gene expression in response to changes in cell density is known as quorum sensing. Quorum-sensing bacteria synthesize and secrete extracellular signaling molecules called autoinducers, which accumulate in the environment as the population increases (Okuda et al. 2004). Gram-positive bacteria generally communicate via small diffusible peptides, while many gram-negative bacteria secrete acyl homoserine lactones (AHLs) (Whitehead et al. 2001), which vary in structure depending on the species of bacteria that produce them. AHLs are involved in quorum sensing whereby cells are able to modulate gene expression in response to increases in cell density. Another system involves the synthesis of autoinducer-2 (AI-2); its structure is unknown, but a gene product, LuxS, is required (Federle and Bassler 2003; Winzer et al. 2003). This system may be involved in cross-communication among both gram-positive and gram-negative bacteria, as homologues of LuxS are widespread within the microbial world (Marsh 2004). Several strains of Prevotella intermedia, Fusobacterium nucleatum, and Porphyromonas gingivalis (formerly Bacteroides gingivalis) were found to produce such activities (Frias et al. 2001; Wu et al. 2009). It was also revealed that the signals produced by subgingival bacteria induce both intra- and inter-species responses in the mixed-species microbial communities that exist in the oral cavity (Okuda et al. 2004).
1.1.1 Dental Plaque as a Microbial Biofilm

Dental plaque can be defined as “matrix-enclosed bacterial populations adherent to each other and/or to surfaces or interfaces” (Costerton 1995).

Dental plaque is a microbial biofilm, a diverse microbial community found on the tooth surface embedded in a matrix of polymers of bacterial and salivary origin (Bradshaw and Marsh 1999). Bradshaw and Marsh (1999) showed the biofilm as a thin basal layer on the substratum, in contact with, and occasionally penetrating, the acquired enamel pellicle, and with columnar, mushroom-shaped multibacterial extensions into the lumen of the solution, separated by regions (“channels”) seemingly empty or filled with extracellular polysaccharide (TenCate 2006; Costerton and Lewandowski 1997; Costerton et al. 1994, 1995). The bacteria in a biofilm communicate with each other by sending out chemical signals. These chemical signals trigger the bacteria to produce potentially harmful proteins and enzymes (Overman 2000) (Fig. 1.1).

The biofilms are considered as etiological communities that evolved to permit the survival of the community as a whole and having a molecular organization, physiochemical properties and growth characteristics. Organization of micro-organisms within biofilms confers, on the component species, properties that are not evident with the individual species grown independently or as planktonic populations in liquid media (Gilbert et al. 1997). The basic biofilm properties are (Overman 2000):

- Cooperating community of various types of microorganisms: Microorganisms are arranged in microcolonies.
- Microcolonies are surrounded by a protective matrix.
- Within the microcolonies are differing environments.
- Microorganisms have a primitive communication system.
- Microorganisms in the biofilm are resistant to antibiotics, antimicrobials, and host response.

1.1.1.1 Inter-species communication: explaining the biological effects of bacterial biofilms

Communication among the different species within biofilms appears to be the key to understanding how plaque can act as a single unit, and how specific bacteria emerge and impair the balance with the host. Physical (coaggregation and coadhesion), metabolic and physiological (gene expression and cell-cell signaling) interactions yield a positive cooperation among different species within the biofilm: the metabolic products of some organisms may promote the further growth of other bacteria or prevent the survival of others. A key role in the cooperative processes is
played by \textit{Fusobacterium nucleatum}, which is able to form the needed “bridge” between early, i.e., \textit{Streptococci} spp., and late colonizers, especially obligate anaerobes. In the absence of \textit{Fusobacterium nucleatum}, \textit{Porphyromonas gingivalis} cannot aggregate with the microbiota already present, such as the facultative aerobes \textit{Actinomyces naeslundii}, \textit{Neisseria subflava}, \textit{Streptococcus mutans}, \textit{Streptococcus oralis} and \textit{Streptococcus sanguinis} (formerly \textit{Streptococcus salivarius}). The presence of \textit{Fusobacterium nucleatum}, on the other hand, enables anaerobes to grow, even in the aerated environment of the oral cavity. Other microorganisms are also able to link otherwise noncommunicating bacteria (i.e., \textit{Streptococcus sanguinis} forms a “corn cob” complex together with \textit{Corynebacterium matruchotii} (formerly \textit{Bacterionema matruchotii}) and \textit{Fusobacterium nucleatum}), and this may represent the basic event leading to biofilm initiation and development (Sbordone and Bortolaia 2003).

The pattern of colonization and coaggregation is often unidirectional, which is proof that some bacteria need to have the environment prepared by other microbiota in order to colonize. \textit{Porphyromonas gingivalis} can adhere to oral \textit{streptococcus} spp. and \textit{Actinomyces naeslundii}, forming small coaggregates resistant to removal, if the substratum has been previously exposed to \textit{Streptococcus gordonii}. Lacking \textit{Streptococcus gordonii}, only few \textit{Porphyromonas gingivalis} cells manage to attach and are easily removed (Sbordone and Bortolaia 2003).

Using DNA probes and checkerboard DNA-DNA hybridization analysis, Socransky et al. (1998) have been able to provide a clear explanation of this colonization pattern and the positive cooperation among subgingival microbiota. They describe how bacteria tend to be grouped in clusters according to nutritional and atmospheric requirements, with the exception of \textit{Actinomyces viscosus}, \textit{Selenomonas noxia} and \textit{Aggregatibacter actinomycetemcomitans} (formerly \textit{Actinobacillus actinomycetemcomitans}) serotype b which do not belong to any group (Sbordone and Bortolaia 2003). The red cluster consisted of \textit{Porphyromonas gingivalis Tannereilla forsythia} (formerly \textit{Bacteroides forsythus}) and \textit{Treponema denticola}. The orange cluster consisted of \textit{Fusobacterium nucleatum} subsp., \textit{Prevotella intermedia} and \textit{Prevotella nigrescens}, \textit{Peptostreptococcus micros}, \textit{Campylobacter rectus}, \textit{Campylobacter showae}, \textit{Campylobacter gracilis}, \textit{Eubacterium nodatum}, and \textit{Streptococcus constellatus}. The three \textit{Capnocytophaga} sp., \textit{Campylobacter concisus}, \textit{Eikenella corrodens} and \textit{Aggregatibacter actinomycetemcomitans} serotype a formed the green cluster, while a group of streptococci made up the yellow cluster. \textit{Streptococcus mitis}, \textit{Streptococcus sanguinis} and \textit{Streptococcus oralis} were the most closely related within this group. \textit{Actinomyces odontolyticus} and \textit{Veillonella parvula} formed the purple cluster. Two \textit{Actinomyces naeslundii} genospecies (formerly \textit{Actinomyces viscosus}), \textit{Selenomonas noxia} and \textit{Aggregatibacter actinomycetemcomitans} serotype b, did not cluster with other species (Socransky et al. 1998) (Fig. 1.2).

The authors describe how each cluster appeared to influence the others. The species within complexes are closely associated to one another: Most periodontal sites harbor either all or none of the species belonging to the same complex, while individual species or pairs of species are detected less frequently than expected, reinforcing the hypothesis of the community theory rather than the germ theory. Precise interrelations are established between complexes as well. Microbiota belonging to the red cluster are seldom detected in the absence of the orange complex, and the higher the detected amounts of orange complex bacteria, the greater is the colonization by red complex members. Yellow and green clusters show a similar preference for each other and a weaker relation with the orange and red complexes, while the purple complex shows loose relations with all the other clusters. Such relations can be explained by mechanisms of antagonism, synergism and environmental selection (Socransky et al. 1998; Sbordone and Bortolaia 2003).

Clinically, yellow and green complexes are associated with shallow pockets (probing depth <3 mm), while the orange and red ones are related to increasing periodontal indices and more advanced lesions. \textit{P}gingivalis, \textit{B} forsythus and \textit{T}denticola are detected in deeper pockets (probing depth >4 mm) and bleeding on probing-positive sites. Given the consecutive colonization of orange and red clusters, altering the former might prevent the emergence of the latter, though it is quite difficult to interfere with the colonization mechanisms and relations among the species as they are yet to be completely understood (Sbordone and Bortolaia 2003).

Specific microbial complexes in \textit{supragingival plaque}, which were similar to those found in subgingival plaque samples with a few minor differences, were recently described by Haffajee et al. (2008a–c) (Table 1.1). Red complex community was formed containing the three species, previously identified as the red...
complex in subgingival plaque namely, *Tannerella forsythia*, *Porphyromonas gingivalis*, and *Treponema denticola*. *Eubacterium nodatum* was also part of this complex and *Treponema socranskii* was loosely associated with these four species. A number of species previously identified in subgingival plaque as orange complex species were also detected as part of an orange complex in supragingival plaque. These included *Campylobacter showae*, *Campylobacter rectus*, *Fusobacterium nucleatum* subsp. *nucleatum*, *Fusobacterium nucleatum* subsp. *vincentii*, *Fusobacterium periodonticum*, *Fusobacterium nucleatum* subsp. *polymorphum*, *Campylobacter gracilis*, *Prevotella intermedia*, and *Prevotella nigrescens*. These taxa were joined by *Gemella morbillorum*, *Capnocytophaga ochracea*, *Selenomonas noxia*, and *Prevotella melaninogenica*. Yellow complex was primarily formed of the *streptococcus* sp. *Streptococcus mitis*, *Streptococcus oralis*, *Streptococcus gordonii*, *Streptococcus sanguinis* and, somewhat separately, of *Streptococcus anginosus*, *Streptococcus intermedius*, and *Streptococcus constellatus*. These species were joined by *Leptotrichia buccalis*, *Propionibacterium acnes*, *Eubacterium saburreum*, *Peptostreptococcus micros*, and *Aggregatibacter actinomycetemcomitans*. A tight cluster of *actinomyces* sp, including *Actinomyces israelii*, *Actinomyces naeslundii* one, *Actinomyces odontolyticus*, *Actinomyces gerencseriae*, and *Actinomyces naeslundii* two was formed. A green complex consisting of *Capnocytophaga sputigena*, *Eikenella corrodens*, and *Capnocytophaga gingivalis*, as well as a loose purple complex consisting of *Neisseria mucosa* and *Veillonella parvula* was formed (Haffajee et al. 2008a) (Fig. 1.3). While plaque mass was associated with differences in proportions of many species in supragingival biofilms, tooth location also was strongly associated with species proportions in both univariate and multivariate analyses (Haffajee et al. 2008b, c).

The relationship between the microbial composition of supragingival plaque samples and the clinical measures of inflammation was quite strong, and on probing, many species were found to be significantly elevated in mean counts at sites that exhibited gingival redness or bleeding. The species that were most in number, adjacent to the inflamed sites, were members of the orange and red complexes. This relationship of orange and red complex species with inflammation was in accordance with the findings related to subgingival biofilms. There was a strong relationship of supragingival counts to

**Fig. 1.2** Diagram of the association among subgingival species. The base of the pyramid is comprised of species thought to colonize the tooth surface and proliferate at an early stage. The orange complex becomes numerically more dominant later, and is thought to bridge the early colonizers and the red complex species which become numerically more dominant at late stages in plaque development (Socransky and Haffajee 2002; with permission from Wiley-Blackwell Publishing).
measures of pocket depth and attachment level. When counts of species in the different pocket depth categories were examined for sites that did or did not exhibit inflammation, the increased levels of orange and red complex species were still observed at the sites with deep pockets irrespective of the level of inflammation (Haffajee et al. 2008a). (Table 1.1).

A consequence of biofilm growth that has profound implications for their control in the environment and in medicine is a markedly enhanced resistance to chemical antimicrobial agents and antibiotics (Marsh 2004). Mechanisms associated with such resistance in biofilms are thought to be related to the following: (1) modified nutrient environments and suppressed growth rates within the biofilm; (2) direct interactions between the exopolymer matrices and their constituents, and antimicrobials, affecting diffusion and availability; and (3) the development of biofilm/attachment-specific phenotypes that can result in reduced sensitivity to inhibitors. Growth on a surface may also result in the drug target being modified or not expressed in a biofilm, or the organism may use alternative metabolic strategies. Bacteria grow only slowly under nutrient depleted conditions in an established biofilm and, as a consequence, are much less susceptible to change than faster-dividing cells. The structure of a biofilm may restrict the penetration of the antimicrobial agent; charged inhibitors can bind to oppositely charged polymers that make up the biofilm matrix (diffusion–reaction theory). The agent may also adsorb to and inhibit the organisms at the surface of the biofilm, leaving the cells in the depths of the biofilm relatively unaffected. The matrix in biofilms can also bind and retain neutralizing enzymes (e.g., β-lactamase) at concentrations that could inactivate an antibiotic or inhibitor. In addition, it has also been proposed that the environment in the depths of a biofilm may be unfavourable for the optimal action of some drugs (Gilbert et al. 1997; Marsh 2004 Stewart and Costerton 2001).

Although it has been shown that bacterial species residing in biofilms are much more resistant to antibiotics than the same species in a planktonic state, antibiotics that have been used frequently in the treatment of periodontal infections (Teles et al. 2006). van Winkelhoff et al. (1996) and Slots and Ting (2002) have revealed that systemically administered antibiotics provided a clear clinical benefit in terms of mean periodontal attachment...
level “gain” post-therapy, when compared with groups not receiving these agents. Meta-analyses performed by Herrera et al. (2002) and Haffajee et al. (2003) indicated that adjunctive systemically administered antibiotics can provide a clinical benefit in the treatment of periodontal infections. However, it must be pointed out that not every study found that adjunctive systemically administered antibiotics provided a benefit to the subject in terms of clinical or microbial outcomes beyond control mechanical debridement therapies (Teles et al. 2006).

The supra- and subgingival habitats present distinct opportunities for colonization by bacterial species. The supra- and subgingival biofilms form a continuum, at least on the tooth surface. Thus, major changes in the supragingival environment are likely to bring about shifts in the subgingival microbiota. It is thought that supragingival plaque control decreases inflammation and gingival crevicular fluid flow, resulting in less nutrition for the subgingival organisms. Removal of supragingival biofilm may also directly affect the contiguous subgingival plaque because the supragingival bacteria may provide nutrients for the subgingival plaque. Several microbial changes in the subgingival microbiota have been reported as a result of supragingival instrumentation, including a reduction in the total number of subgingival microorganisms, a reduction in the levels of spirochetes, an increase in the proportion of gram-positive organisms, a reduction in the frequency of detection of Porphyromonas gingivalis, Aggregatibacter actinomycetemcomitans and Fusobacterium nucleatum, and a decrease in the levels of subgingival species, such as Prevotella intermedia, Fusobacterium nucleatum subsp. nucleatum, F. n. subsp. vincentii, Fusobacterium periodonticum, Fusobacterium nucleatum subsp. polymorphum, Campylobacter gracilis, Prevotella intermedia, Prevotella nigrescens, Gemella morbillorum, Capnocytophaga ochracea, Selenomonas noxia, Prevotella melaninogenica.

Table 1.1 Microbial complexes in subgingival and supragingival plaque (according to Socransky et al. 1998 and to Haffajee et al. 2008a)

<table>
<thead>
<tr>
<th>Cluster</th>
<th>Subgingival plaque</th>
<th>Supragingival plaque</th>
</tr>
</thead>
<tbody>
<tr>
<td>Purple</td>
<td>Veillonella parva, Actinomyces odontolyticus</td>
<td>Veillonella parva, Neisseria mucosa</td>
</tr>
<tr>
<td>Green</td>
<td>Eikenella corrodens, Capnocytophaga gingivalis, Capnocytophaga sp.</td>
<td>Capnocytophaga sp.</td>
</tr>
<tr>
<td>Orange</td>
<td>Aggregatibacter actinomycetemcomitans serotype a</td>
<td>Capnocytophaga sp.</td>
</tr>
<tr>
<td>Red</td>
<td>Porphyromonas gingivalis, Tannerella forsythia, Treponema denticola, Actinomyces viscosus, Selenomonas noxia, Aggregatibacter actinomycetemcomitans serotype b</td>
<td>Porphyromonas gingivalis, Tannerella forsythia, Treponema denticola, Eubacterium nodatum, Treponema socranski</td>
</tr>
</tbody>
</table>

Table 1.1: Etiology of Periodontal Disease: Dental Plaque and Calculus
exposed root surfaces. The immediate effect of scaling and root planing is an enormous disruption of the subgingival biofilm. Curettes, when used as a sampling method, can remove up to 90% of the subgingival plaque. It was suggested that the impact of scaling and root planing on the subgingival microbiota can last beyond the first 3 months posttherapy, in spite of a lack of subgingival reinstrumentation (Teles et al. 2006).

The proposed benefits of periodontal surgery over scaling and root planing include better access for cleaning of the root surfaces, pocket reduction (or elimination) and exposure of root surfaces for proper cleaning by the patients. Of all periodontal therapies, surgery is the procedure that most drastically alters the periodontal pocket environment. Hence, it is logical to anticipate that these techniques, particularly those aiming at total pocket elimination, will result in dramatic changes in the subgingival microbiota. Additional beneficial changes in the subgingival microbiota after surgery, when compared with scaling and root planing alone, were reported (Teles et al. 2006).

Despite the best efforts by clinicians and patients, certain subgingival sites will become recolonized by periodontal pathogens after active therapy. There are several potential sources for the reinforcement of the gingival crevice, including: regrowth of residual cells present in the depths of the pocket; neighboring supra- and subgingival biofilms still colonized by the species in question; other intra-oral sites; and exogenous sources, through vertical or horizontal transmission. A key role of supragingival plaque control in retarding the resurgence of pathogens within the pockets after mechanical therapy was clearly established, suggesting a clear involvement of supragingival plaque as a major source of reinfecting organisms. In fact, the observation that periodontal pathogens, including members of the red complex (Porphyromonas gingivalis, Tannerella forsythia and Treponema denticola), can colonize supragingival biofilms of both, subjects with periodontitis and healthy individuals (although at lower levels and at a reduced prevalence), highlights the importance of this habitat as a source of reinfecting microorganisms. The alternative explanation consists of the fact that the pathogenic species could have remained in the depths of the healed pockets, and been provided with an enhanced source of nutrients from the gingival crevice fluid, resulting from the inflammation triggered by early colonizers of the supragingival microbiota. It has long been hypothesized that the sulci of neighboring teeth, or even teeth from distant quadrants, can foster the recolonization of treated sites (Teles et al. 2006).

Traditionally, the oral cavity of a periodontal subject is treated in several sessions during which antinfective therapy is applied to different areas of the mouth, divided into quadrants or sextants. Based on the idea that the microbiota of nontreated sites could compromise the healing of treated quadrants, a new therapeutic approach was devised, based on the principle of full-mouth disinfection. In this approach, the full dentition receives scaling and root planing within 24h, in order to minimize reinfection of treated sites by pathogens present on untreated teeth. As other oral surfaces (saliva, tonsils, oral mucosa, tongue) also harbor periodontal pathogens, the therapy, additionally involved the disinfection of these surfaces using chlorhexidine. This technique resulted in superior pocket depth reduction and clinical attachment level gain when compared with the typical weekly/bi-weekly quadrant treatment regimen for scaling and root planing. Reported microbiological improvements included a decreased percentage of spirochetes and motile rods, greater reduction in the levels of pathogenic species such as Porphyromonas gingivalis, Prevotella intermedia, Fusobacterium nucleatum, Peptostreptococcus micros and Campylobacter rectus, and diminished levels of “black-pigmented bacteroides” (Teles et al. 2006).

Oral implants provide a unique opportunity for the observation of initial subgingival colonization patterns, since it is started with a “pristine” bacteria-free surface/pocket. Quirynen et al. (2005) recorded the development of the “initial” subgingival biofilm on implants with shallow (<3 mm) and moderate (>3 mm) pockets, to estimate the time needed before a complex subgingival flora could be established with the supragingival area as the single source. The undisturbed subgingival microbiota of neighboring teeth in the same individuals served as controls. Checkerboard DNA-DNA hybridization and culture data revealed a complex microbiota (including several pathogenic species) in the pristine pockets within a week, with a minimal increase in counts up to 4 weeks. The reason for the rapid recolonization in the “pristine” environment is not clear. It is possible that the blood coagulum at the fresh implant sites may favor the colonization and growth of oral species in a fashion similar to that which might occur after mechanical debridement of periodontal pockets. Alternatively, the large number of organisms in saliva and on the oral soft tissues, particularly the tongue, and the rapid multiplication rate of bacteria may be sufficient for many species to establish
and reach sizeable numbers in the absence of the competing microbiota (Quirynen et al. 2005).

Surprisingly, it was recently revealed that periodontal pathogens such as *Aggregatibacter actinomycetemcomitans* and *Porphyromonas gingivalis* are also clearly present in the samples from edentulous subjects. Microbial profiles in samples from the soft tissue surfaces differed among site locations. Samples from the dorsum of the tongue exhibited the highest bacterial counts followed by the “attached gingiva” and the lateral surfaces of the tongue, while the lowest mean counts were found in samples from the buccal mucosa and labial vestibules. Using cluster analysis of the proportions of the test species, three clusters were formed. The first cluster comprised saliva, supragingival plaque, and the lateral and dorsal surfaces of the tongue. The second cluster comprised the other six soft tissue surfaces. Species on the denture palate formed a third cluster (Sachdeo et al. 2008).

1.1.2 Microbial Composition of Dental Plaque in Relation to Periodontal Health or Disease

1.1.2.1 Periodontal Pathogens Associated with Health

Healthy gingivae have been associated with a very simple supragingival plaque composition: few (1–20) layers of predominantly gram-positive cocci (*streptococcus* spp.: *Streptococcus mutans*, *Streptococcus mitis*, *Streptococcus sanguinis*, *Streptococcus oralis*, *Rothia dentocariosa*, *Staphilococcus epidermidis*), followed by some gram-positive rods and filaments (*actinomyces* spp: *Actinomyces viscosus*, *Actinomyces israelis*, *Actinomyces gerencseriae*, *corynebacterium* spp.) and very few gram-negative cocci (*Veillonella parvula*, *neisseria* spp.). The latter are aerobic or facultative aerobic bacteria, able to adhere to the nonexfoliating hard surfaces; initial adhesion is promoted by surface free energy, roughness and hydrophilia, and is mediated by long- and short-range forces (Sbordone and Bortolaia 2003). The species involved vary depending on local environmental characteristics, but the colonization pattern is always the same (Sbordone and Bortolaia 2003). The severe forms of gingivitis have been associated with subgingival occurrence of the black-pigmented *Porphyromonas gingivalis* (White and Mayrand 1981).

In pregnancy gingivitis, an association has been observed between high levels of *Prevotella intermedia* and elevations in systemic levels of estradiol and progesterone (Kornman and Loesche 1980).

Microbial studies in acute necrotizing ulcerative gingivitis (ANUG) indicates high levels of *Prevotella intermedia* and *Treponema pallidum* – related spirochetes. *Spirochetes* are found to penetrate necrotic tissue as well as apparently unaffected connective tissue (Loesche et al. 1982; Riviere et al. 1991).
1.1.2.3 Periodontal Pathogens Associated with Periodontitis

The etiologic role of bacteria in periodontal disease is clearly established (Socransky 1977). According to the nonspecific plaque hypothesis, it appears that different combinations of indigenous bacteria, rather than just a single species, can produce the pathogenic potential necessary to cause progression from gingivitis to destructive periodontitis (Theilade 1986). Microbiological studies have revealed that some of the infections in periodontal pockets are multibacterial (Rodenburg et al. 1990; Moore et al. 1991; Söder et al. 1993; Colombo et al. 1998). On the other hand, according to the specific plaque hypothesis, one or several bacterial species cause the initiation and progression of destructive periodontal disease (Slots 1979; Socransky 1979; Socransky and Haffajee 1992; Loesche 1982) (Fig. 1.4). However, it can be found that not only an increase in the total microbial load (10^5–10^8 microorganisms), but, with a high probability, certain species, such as Aggregatibacter actinomycetemcomitans, Porphyromonas gingivalis, Prevotella intermedia and Treponema denticola, are also major etiological agents in destructive periodontal disease (Van der Weijden et al. 1994). Aggregatibacter actinomycetemcomitans and Porphyromonas gingivalis fulfill, at least partly, the modified Koch’s criteria for defining a periodontal pathogen (Haffajee and Socransky 1994).

Plaque accumulation leads to gingivitis, but the shift to periodontitis depends on both host factors and the selection of virulent bacteria. Periodontitis is not a single disease, but rather a collection of pathologies with similar patterns and symptoms. Though many classifications have been proposed, during the 1999 International Workshop for Classification of Periodontal Diseases and Conditions, the previously accepted terms “early-onset periodontitis” and “adult periodontitis” were replaced by “aggressive periodontitis” and “chronic periodontitis.” Thus, age and microbiological features no longer represent the primary classification criteria, but rather, clinical behavior and laboratory findings are used to distinguish the two forms (Sbordone and Bartolaia 2003).

The common features of localized and generalized forms of Aggressive Periodontitis are (Armitage 1999):

- Healthy patients, except for the presence of periodontitis.
- Rapid attachment loss and bone destruction.
- Familial aggregation.
- Secondary features that are generally, but not universally present are:
  - Amounts of microbial deposits inconsistent with the severity of periodontal tissue destruction.
  - Elevated proportions of Aggregatibacter actinomycetemcomitans and, in some populations, Porphyromonas gingivalis.
  - Phagocyte abnormalities.
  - Hyper-responsive macrophage phenotype, including elevated levels of PGE2 and IL-1β.
  - Progression of attachment loss and bone loss may be self-arresting.

Generally the term “chronic periodontitis” replace the term “adult periodontitis” (Armitage 1999). The chronic periodontitis is defined as an infectious disease resulting in inflammation within the supporting tissues of the teeth, progressive attachment, and bone loss. It is

Fig. 1.4 A hypothesized relationship between the addition of species during microbial succession leading to the development of gingival inflammation. In turn, the increased inflammation would result in increased growth of colonizing species (Socransky and Haffajee 2005) (with permission from Wiley-Blackwell Publishing)
characterized by pocket formation and/or gingival recession. It is recognized as the most frequently occurring form of periodontitis. Its onset may be at any age, but is most common in adults. The prevalence and severity of the disease increase with age. It may affect a variable number of teeth and it has variable rates of progression.

The microbiota of slight chronic periodontitis in adults and adolescents has been associated with *Porphyromonas gingivalis* and *Tannerella forsythia*, using rapid immunofluorescence (Riviére et al. 1996; Clercugh et al. 1997; Hamlet et al. 2004), PCR (Tanner et al. 2007) and DNA probe methods (Tran et al. 2001). In a longitudinal study to detect progressing slight periodontitis, a combination of anaerobic culture and DNA hybridization assays associated *Tannerella forsythia*, *Campylobacter rectus*, *Selenomonas noxia* and *Prevotella intermedia* with inter-proximal progressing slight (initial) chronic periodontitis, compared with health or gingivitis (Tanner et al. 1998, 2006).

The major species associated with moderate and advanced chronic adult periodontitis were originally detected using cultivation-based methods and include *Porphyromonas gingivalis*, *Prevotella intermedia*, *Tannerella forsythia*, *Treponema denticola* and *Aggregatibacter actinomycetemcomitans* (Moore et al. 1991; Kagga et al. 1995; Haffajee et al. 1998; Mombelli et al. 1998; Machtei et al. 1999; van Winkelhoff et al. 2000). Peri-implant infection in edentulous subjects (Mombelli and Mericske-Stern 1990; van Winkelhoff et al. 2000). Peri-implantitis can be considered the “twin-sister” of periodontitis, even though some important differences between natural teeth and dental implants must clearly be borne in mind, the most important being that implants are not surrounded by a periodontal ligament and therefore, present different biomechanics and defensive cell recruitment (Sbordone and Bartolaia 2003).

More factors can be associated with biological failures of oral implants: medical status of the patient, smoking, bone quality, bone grafting, irradiation therapy, parafunctions, operator experience, degree of surgical trauma, bacterial contamination, lack of preoperative antibiotics, immediate loading, nonsubmerged procedure, number of implants supporting a prosthesis, implant surface characteristics and design (Esposito et al. 1998a, b).

The colonization of the implant sulcus is different in partially edentulous patients in comparison to fully edentulous patients. Early bacterial colonization of peri-implant pockets in edentulous subjects is characterized by an increase of facultative anaerobic streptococci, whereas gram-negative strict anaerobic rods are usually isolated infrequently in low proportions (Mombelli et al. 1988; van Winkelhoff et al. 2000). Long-term results on colonization of the peri-implant area showed a decrease in the proportions of facultative streptococci and an increase in the percentage of gram-positive facultative rods and gram-negative strict anaerobic rods, e.g., *fusobacterium spp.* and *prevotella spp.* (Mombelli and Mericske-Stern 1990; van Winkelhoff et al. 2000). Peri-implant infection in edentulous subjects is associated with bacteria that are found in adult periodontitis, however, with the exception of *Porphyromonas gingivalis* and *Aggregatibacter actinomycetemcomitans* (Mombelli et al. 1987; van Winkelhoff et al. 2000).

In contrast to fully edentulous patients, colonization of peri-implant pockets in partially edentulous patients is characterized by rapid appearance of spirochetes. Samples from partially edentulous subjects also contained more black-pigmenting gram-negative
anaerobes than samples from fully edentulous subjects (Mombelli 2002). Takanashi et al. (2004) investigated the colonization by black-pigmented anaerobic bacteria that occurs between the time before fixture installation and 6 months after inserting superstructures in implant treatment in partial edentulous cases. Dental plaque was serially collected from around the natural teeth and implants in 12 patients in whom a dental implant was indicated, and Porphyromonas gingivalis and Prevotella intermedia were detected using polymerase chain reaction. One month after connecting the abutment, the detection rate of Porphyromonas gingivalis per site from around the implants was 63.7% and that of Prevotella intermedia was 50.8%. Six months after superstructure setting, the detection rate per site of Porphyromonas gingivalis from around the implants was 56.8% and that of Prevotella intermedia was 41.1%. When chromosomal DNA segmentation patterns in the isolated Porphyromonas gingivalis and Prevotella intermedia were compared using pulsed field gel electrophoresis (PFGE), the patterns in the natural teeth were in accordance with those in the implants in three of four cases (75.0%) in Porphyromonas gingivalis, and all cases in Prevotella intermedia. Similar findings were obtained by Koka et al. (1993), Kohavi et al. (1994) and Leonhardt et al. (1993), suggesting that bacterial colonization around implants occurred early after the implant region was exposed to the intraoral cavity and that the bacteria were transmitted from the area around the natural teeth. Aggregatibacter actinomycetemcomitans and Actinomyces viscosus were, however, more frequent in the supragingival plaque of teeth than of implants.

The occurrence of peri-implantitis may be dependent on distinct individual susceptibility factors, e.g., immuno inflammatory factors, interacting with molecular processes that are similar to periodontitis. Hence, it is important to ascertain whether patients with an increased susceptibility to periodontitis would have an increased susceptibility to peri-implantitis and implant loss (i.e., decreased survival or success rate of implants), even in partially dentate patients who have been treated for periodontitis. This is relevant because periodontitis is one of the leading causes of tooth loss, and dental implants are increasingly used to replace missing teeth in such patients. Consequently, a history of past periodontitis may act as a prognostic factor for the future survival and success of dental implants (Ong et al. 2008).

Conversely, there are some studies that have shown successful osseo-integration in patients with different types of periodontitis (Nevins and Langer 1995; Ellegaard et al. 1997; Quirynen et al. 2001). However, in a long term study, Karoussis et al. (2003) demonstrated lower survival rates and more biological complications, than patients with implants replacing teeth lost due to reasons other than periodontitis, during a 10-year maintenance period. Three systematic reviews were performed to determine implant outcomes in partially dentate patients who have been treated for periodontitis compared with periodontally healthy patients. Van der Weijden et al. (2005) concluded that the outcome of implant therapy in periodontitis patients may be different compared with individuals without such a history in terms of loss of supporting bone and implant loss. Schou et al. (2006) revealed that the survival of the supra-structures and the implants was not significantly different in individuals with periodontitis-associated and nonperiodontitis-associated tooth loss. However, significantly increased incidence of peri-implantitis and significantly increased peri-implant marginal bone loss were revealed in individuals with periodontitis associated tooth loss. More recently, Ong et al. (2008) reported that there is some evidence, that patients treated for periodontitis may experience more implant loss and complications around implants than nonperiodontitis patients. Evidence was stronger for implant survival than implant success. This is probably caused by the presence of periodontal pockets that serve as a reservoir for these bacteria (van Winkelhoff et al. 2000).

Microbiological studies of dental implants with clinically healthy marginal peri-implant tissues (Lee et al. 1999; Hultin et al. 2002; Renvert et al. 2007) have demonstrated a scattered, sub-mucosal microbiota dominated by facultative gram-positive cocci and rods. In contrast, a peri-implant pocket of diseased implants seems to harbor a microbiota similar to that found in periodontal disease, such as Porphyromonas gingivalis, Prevotella intermedia, Prevotella nigrescens, Tannerella forsythia, Campylobacter rectus and Aggregatibacter actinomycetemcomitans, especially serotype b (Mombelli et al. 1987; Mombelli 2002; Tanner et al. 1997; Shibli et al. 2008; Quirynen et al. 2002). Organisms not primarily associated with periodontitis, such as staphylococcus spp., enterics and candida spp. have also been found in peri-implant infection (Leonhardt et al. 1999).
It seems that proper periodontal infection control may help to prevent early bacterial complications in implant dentistry. Infection control should involve suppression of commensal periodontal bacteria below certain thresholds and elimination of putative exogenous periodontal pathogens, i.e., *Porphyromonas gingivalis*. This may be of special importance in patients with a history of periodontitis. Microbiological testing in partially edentulous subjects with a history of periodontitis may be one measure to prevent peri-implantitis by employing appropriate antimicrobial therapy before placing the dental implants (van Winkelhoff et al. 2000).

Such findings have relevance for the planning of immediate postextraction implants, especially if tooth loss is determined by periodontal disease. A wait of at least one month after extraction was suggested to allow for the elimination of *Aggregatibacter actinomycetemcomitans* and *Porphyromonas gingivalis* from the extraction socket. The same rules apply when Guided Tissue Regeneration (GTR) and Guided Bone Regeneration (GBR) procedures are performed: membrane exposure and bacterial colonization impair the outcome in terms of tissue regeneration. Exposure is more likely in patients presenting periodontitis, peri-implantitis or residual deep pockets: the smallest degree of attachment and bone gain occur when *Porphyromonas gingivalis*, *Aggregatibacter actinomycetemcomitans*, *Prevotella intermedia*, *Tannerella forsythia* and *capnocitophaga* spp. are detected on the infected barriers. It can be concluded that implant and GTR/GBR procedures achieve the best results in those subjects that comply with domestic plaque control routines and maintenance protocol schedules (Sbordone and Bortolaia 2003).

### 1.1.3 Dental Plaque Formation

The formation of bacterial plaque is initiated by the adhesion of micro-organisms to the tooth surface, and is the first step in the development of periodontal infections (Newman et al. 1978).

Until now, no uniform theory has been developed to explain the fundamental mechanisms of cell adhesion. Moreover, it would be impossible and erroneous to conclude that one single mechanism dictates the adhesive tendency of microorganisms because the situation is too complex (Quirynen and Bollen 1995).

The process of plaque formation can be divided into several phases:

#### 1.1.3.1 Adsorption of Host and Bacterial Molecules to the Tooth Surface

This conditioning film (the acquired pellicle) forms immediately following eruption or cleaning and directly influences the pattern of initial microbial colonization (Marsh 2004). Dental pellicles mediate many of the interactions that take place at intraoral surfaces. The term pellicle is used to describe a thin, continuous membrane or cuticle, composed primarily of salivary components deposited on a cleaned tooth surface (Al-Hashimi and Levine 1989). All surfaces of the oral cavity, including all tissue surfaces (Bradway et al. 1989) as well as surfaces of teeth—enamel (Al-Hashimi and Levine 1989), cementum (Fisher et al. 1987) and fixed, and removable restorations (Edgerton and Levine 1992; Edgerton et al. 1996) are coated by dental pellicle.

Pellicles contain salivary components, constituents from gingival crevicular fluid, microbial, and cellular sources (Scannapieco 1995). Enamel pellicle formation is driven by a combination of physical forces (ionic, hydrophobic, hydrogen loading and van der Waals) between molecules in saliva and the tooth surface (Scannapieco et al. 1995).

To study the acquired enamel pellicle, it is convenient to examine the freshly extracted teeth (Listgarten 1976) or by placing plastic strips or epoxy crowns in the oral cavity as analogs to the tooth (Brecx et al. 1981; Scannapieco 1995).

1.1.3.2 Passive Transport of Oral Bacteria to the Tooth Surface

Weak, long-range physicochemical interactions between the microbial cell surface and the pellicle-coated tooth create a weak area of net attraction that facilitates reversible adhesion. Subsequently, strong, short-range interactions between specific molecules on the bacterial cell surface (adhesions) and complementary receptors in the pellicle can result in irreversible attachment and can explain microbial tropisms for surfaces. Some of the adhesions that have been identified on subgingival species include fimbriae (Cisar et al. 1984; Sandberg et al. 1988) and cell-associated proteins (Socransky and Haffajee 1992). Adhesions are often lectins which bind to saccharide receptors, but some adhesions are thought to bind to proteinaceous receptors (Gibbons 1989). Receptors on tissue surfaces include galactosyl residues, sialic acid residues (Murray et al. 1986), proline-rich proteins or statherin and Type I and IV collagen (Socransky and Haffajee 1992). Oral bacteria generally possess more than one type of adhesion on their cell surface and can participate in multiple interactions both with host molecules and similar receptors on other bacteria (coadhesion) (Marsh 2004; Quirynen and Bollen 1995).

1.1.3.3 Coadhesion of Later Colonizers to Already Attached Early Colonizers

This stage also involves specific interbacterial adhesion-receptor interactions (often involving lectins) and leads to an increase in the diversity of the biofilm and to the formation of unusual morphological structures, such as corn-cobs and rosettes (Marsh 2004; Kolenbrander 2000). Coaggregation (interactions between the suspended micro-organisms in a fluid phase) between oral microbial pairs as well as its role in the sequential colonization of the tooth surface has been studied extensively (Kolenbrander et al. 1994; Cisar et al. 1997). However, coadhesion (interactions between suspended and already-adhering microorganism to a surface) may well be equally important (Bos et al. 1996). Bacteria engage in a range of antagonistic and synergistic biochemical interactions (Marsh and Bradshaw 1995). The efficiency of metabolic interactions among bacteria in food chains may be enhanced if they are brought into close physical contact. Likewise, the coadhesion of obligate anaerobic bacteria to oxygen-consuming species can ensure their survival in overt aerobic oral environments (Marsh 2004).

The analysis of the coaggregation profiles of hundreds of subgingival isolates has provided evidence that coaggregation might be important for subsequent plaque development. Certain streptococi (for example, Streptococcus oralis), which bear receptors are coaggregation partners of members of several genera. Early colonizing partners of receptor-bearing streptococci include Streptococcus gordonii, Actinomyces naeslundii, Eikenella corrodens, Veillonella atypica, Prevotella loescheii and Haemophilus parainfluenzae, as well as Capnocytophaga ochracea. It is worth noting that these coaggregating partners of the initial colonizing Streptococcus oralis, Streptococcus sanguinis and Streptococcus mitis are almost all gram-negative, which correlates with the 40-year-old reports of a temporal shift from gram-positive to gram-negative bacterial flora. The dominant species in initial dental plaque were Streptococcus oralis that are receptor-bearing cells, indicating that receptor-bearing streptococci are an abundant surface readily available for recognition by gram-negative bacteria expressing complementary adhesions which recognize receptor polysaccharides. Possibly, receptor polysaccharides on the early colonizing streptococci are a prerequisite for the shift from gram-positive to gram-negative flora accompanying the shift from health to gingivitis (Kolenbrander et al. 2006) (Fig. 1.5).

1.1.3.4 Multiplication of the Attached Micro-Organisms

Cell division leads to confluent growth and, eventually, a three-dimensional spatially and functionally organized, mixed-culture biofilm. Polymer production results in the formation of a complex extracellular matrix made up of soluble and insoluble glucans, fructans and heteropolymers. Such a matrix is a common feature of biofilms and makes a significant contribution to the known structural integrity and general resistance of biofilms; the matrix can be biologically active and retain nutrients, water and key enzymes within the biofilm. Endogenous substrates (derived from saliva or gingival crevicular fluid) are the main source of nutrients for oral bacteria, but their catabolism requires the concerted and sequential action of groups of microbes.
Fig. 1.5 Spatiotemporal model of oral bacterial colonization, showing recognition of salivary pellicle receptors by early colonizing bacteria, and coaggregations between early colonizers, fusobacteria, and late colonizers of the tooth surface. Each coaggregation depicted is known to occur in a pairwise test. Collectively, these interactions are proposed to represent the development of dental plaque. Starting at the bottom, primary colonizers bind via adhesions (round-tipped black line symbols) to complementary salivary receptors (blue–green vertical round-topped columns) in the acquired pellicle coating the tooth surface. Secondary colonizers bind to previously bound bacteria. Sequential binding results in the appearance of nascent surfaces that bridge with the next coaggregating partner cell. Several kinds of coaggregations are shown as complementary sets of symbols of different shapes. One set is depicted in the box at the top. Proposed adhesins (symbols with a stem) represent cell-surface components that are heat inactivated and protease sensitive; their complementary receptors (symbols without a stem) are unaffected by heat or protease. Identical symbols represent components that are functionally similar but may not be structurally identical. Rectangular symbols represent lactose-inhibitable coaggregations. Other symbols represent components that have no known inhibitor. The bacterial species shown are Actinobacillus actinomycetemcomitans, Actinomyces israelii, Actinomyces naeslundii, Capnocytophaga gingivalis, C. ochracea, C. sputigena, Eikenella corrodens, eubacterium spp., Fusobacterium nucleatum, Haemophilus parainfluenzae, Porphyromonas gingivalis, Prevotella intermedia, P. loescheii, Propionibacterium acnes, Selenomonas flueggei, Streptococcus gordonii, S. mitis, S. oralis, S. sanguinis, treponema spp., and Veillonella atypica (Kolenbrander et al. 2006) (with permission from Wiley-Blackwell Publishing)
with complementary enzyme profiles, i.e., plaque functions as a true microbial community (Marsh 2004).

### 1.1.3.5 Active Detachment

Once established, the resident plaque microflora remains relatively stable over time and is of benefit to the host. The resident microflora of all sites plays a critical role in the normal development of the physiology of the host and also reduces the chance of infection by acting as a barrier to colonization by exogenous (and often pathogenic) species (“colonization resistance”). Mechanisms contributing to colonization resistance include more effective competition for nutrients and attachment sites, the production of inhibitory factors, and creation of unfavorable growth conditions by the resident microflora. Thus, treatment should attempt to control rather than eliminate the plaque microflora (Marsh 2004).

### 1.1.4 Impact of Surface Characteristics and/or Surface Topography on Biofilm Development

Bacterial accumulation on dental materials is determined by various surface characteristics. The adhesion of bacteria is significantly affected by high surface roughness values because of a reduction of shear forces on initially attaching bacteria. The impact of surface roughness on the biofilm formation can be explained by several factors:

- The initial adhesion of bacteria preferably starts at locations where they are sheltered against shear forces, so that they find time to change from reversible to irreversible attachment.
- Roughening of the surface increases the area available for adhesion by a factor 2–3.
- Rough surfaces are difficult to clean, resulting in a rapid regrowth of the biofilm by the multiplication of remaining species, rather than by recolonization (Teughels et al. 2006).

Materials with high surface free energy values are known to increase adhesion of bacteria (An and Friedman 1998; Taylor et al. 1998). Furthermore, the bacterial adhesion process is influenced by the chemical composition of the material, surface hydrophobicity, and the zeta potential. (An and Friedman 1998; Taylor et al. 1998; Quirynen and Bollen 1995; Carlen et al. 2001).

### 1.1.5 Bacterial Colonization on Tooth Surfaces and Dental Materials

Differences in the amount of adherent plaque are observed in various materials (Siegrist et al. 1991) and tissues (Nyvad and Fejerskov 1987; Carrassi et al. 1989).

The pattern of microbial colonization in vivo is determined by the surface structure of the tooth; on enamel surfaces the first bacteria appeared in pits and surface irregularities followed by proliferation along the perikymata, while on root surfaces bacterial colonization is characterized by a haphazard distribution (Nyvad and Fejerskov 1987). It was also observed that within the initial 24-h period, root surfaces were more heavily colonized than were enamel surfaces (Nyvad and Fejerskov 1987).

Different types of soft mucosal and hard dental surfaces may constitute various prerequisites for bacterial colonization (Gibbons 1989).

There are also more ecological differences in the supra- and subgingival environment which are of importance when bacterial adhesion is considered. Supragingly, bacteria can adhere to the enamel surface or, to a lower extent, to the desquamating oral epithelium. Subgingingly, more niches are available for bacterial survival: adhesion to the root cementum, adhesion to the desquamating pocket epithelium, swimming in the crevicular fluid, invasion in the soft tissue or invasion into the hard tissue via the dentine tubules (Quirynen 1994).

The ultrastructural pattern of early plaque formation was studied on various dental materials: amalgam, casting alloys, titanium, ceramics, glass polyalkenoate cement, composite resins, unfilled resins, and bovine enamel (Hannig 1999). Because only less pronounced variations could be detected in the ultrastructural appearance of the early plaque formed on the different material surfaces, it was concluded that early plaque formation on solid surfaces is influenced predominantly by the oral environment rather than by material-dependent parameters. These findings may be ascribed to the presence of the pellicle layer, which apparently
masks any difference among materials, with regard to surface properties and biocompatibility.

Similar results were obtained by Leonhardt et al. (1995) who evaluated qualitative and quantitative differences in bacterial colonization on titanium, hydroxyl-apatite, and amalgam surfaces in vivo. No significant differences among the materials regarding colonization of investigated bacteria were found during the study period.

The different composition of materials only slightly affects plaque colonization. The amount of the early plaque colonization seems to be related more to the roughness degree than to material composition (Siegrist et al. 1991).

Materials used for dental restorations may also have antibacterial properties per se. Several studies have shown that amalgam alloys have a bacteriostatic effect (Glassman and Miller 1984). Titanium has been shown to inhibit plaque growth in vitro, particularly in the early stages, probably due to the antimicrobial effect of metal ion release (Joshi and Eley 1988).

Fixed or removable orthodontic appliances also impede the maintenance of oral hygiene, resulting in plaque accumulation (Batoni et al. 2001; Jordan and LeBlanc 2002). Plaque retention surrounding orthodontic appliances leads to enamel demineralization caused by organic acids produced by bacteria in the dental plaque (Arends and Christofferson 1986; O’Reilly and Featherstone 1987). Recently, fluoride-releasing elastomeric modules (Wiltshire 1999; Banks et al. 2000; Mattick et al. 2001) and chlorhexidine varnish (Beyth et al. 2003) were suggested for reducing plaque accumulation and decalcification (Türkkahraman et al. 2005).

Fixed orthodontic appliances create new retention areas, which are suitable for bacterial colonization and lead to an increase in the absolute number and percentage of Streptococcus mutans and lactobacilli (Türkkahraman et al. 2005; Forsberg et al. 1991; Balenseifen and Madonia 1970; Corbett et al. 1981; Mattingly et al. 1983; Scheie et al. 1984; Diamanti-Kipioti et al. 1987; Lundström and Krasse 1987; Sinclair et al. 1987; Svanberg et al. 1987; Rosenbloom and Tinanoff 1991; Chang et al. 1999). As revealed by Türkkaahraman et al. (2005) and Faltermeyer et al. (2008), a lot of studies have evaluated the effect of fixed orthodontic appliances on microbial flora and periodontal status (Balenseifen and Madonia 1970; Corbett et al. 1981; Scheie et al. 1984; Sinclair et al. 1987; Rosenbloom and Tinanoff 1991; Chang et al. 1999; Pender 1986; Huser et al. 1990; Glans et al. 2003), but their sample sizes were relatively low, and generally no additional periodontal evaluation was performed.

Osseo-integrated titanium dental implants have been proven to provide highly reliable restoration of function in totally and partially edentulous patients (Adell et al. 1990). One of the most important causes of dental implant failure appears to be bacterial plaque colonization (Mombelli and Lang 1994). The microbiology of failing implants seems to be similar to that of the natural dentition in advanced stages of adult periodontitis (Becker et al. 1990).

Bacterial colonization seems to be promoted by surface-free (Van Dijk et al. 1987), roughness (Quirynen and Bollen 1995) and the presence of specific molecules adsorbed from the saliva onto the titanium surface (Wolinsky et al. 1989).

The titanium surface with Ra < 0.088 mm and Rz < 1.027 mm strongly inhibits accumulation and maturation of plaque at the 24-h time period (Rimondini et al. 1997).

The sequential appearance of microbial morphotypes during maturation of supra- and subgingival plaque on natural tooth structure (enamel and cementum surfaces) and implant materials (titanium and plasma spray-coated titanium and hydroxyl-apatite) showed similar results regardless of the surface (Gatewood et al. 1993). In both supra and subgingival plaque, depending on time interval, cocci, rods of various lengths, filamentous organisms, fusiforms, spirochetes and corn cob formation were observed.

It has to be noted that the microflora of implants in partially and totally edentulous mouths differ (Bauman et al. 1992). It is also suggested that implants in edentulous mouth have less chance of peri-implant infection than those in the partially edentulous mouth (Gatewood et al. 1993).

Bacterial colonization of both bio-resorbable and non-resorbable periodontal membranes used in guided tissue regeneration surgery has been extensively reported in the literature (Zucchelli et al. 1997, 1998; Nowzari et al. 1995; Tempro and Nalbandian 1993; Selvig et al. 1990; DeSanctis et al. 1996; Grevstad and Leknes 1993).

A negative relationship was observed between the amount of microorganisms present on the membrane surfaces and the clinical attachment gain following surgical procedure (Nowzari et al. 1995). It is conceivable that early bacterial accumulation on membrane materials can prevent fibrin organization on the membrane and thus reduce its integration with connective
tissue and consequently the outcome of surgery (Zucchelli et al. 1997). The exposure of bio-absorbable membranes may also reduce the capability of the body to resorb the material (DeSanctis et al. 1996).

Systemic antibiotics and local application of chlorhexidine do not prevent bacterial colonization of exposed membranes (DeSanctis et al. 1996).

Quantitative differences in early plaque accumulation on various membranes (expanded polytetrafluoroethylene, polyglactin 910 and polylactic acid) seem to be related to the textural and structural characteristics of the surface, which is not adequately represented by the surface Ra value measured with a profilometric instrument (Zucchelli et al. 1998). The 4-h results indicated a statistically significant difference in the proportion of bacteria-positive fields among the three membranes; a greater amount of bacteria was demonstrated on the ePTFE membrane compared to the other two membranes. At 24 h, the difference in the proportion of bacteria-positive fields was statistically significant; a lesser amount of bacterial plaque was present on the polylactic acid membrane compared to the ePTFE and polyglactin 910 membranes. No difference in the proportion of rod/bacteria-positive fields was demonstrated among the three membranes at either 4 or 24 h. It was concluded that quantitative differences in early plaque accumulation on various membranes seem to be related to the textural and structural characteristics of the surface. (Zucchelli et al. 1998).

### 1.1.6 Assessment of Dental Plaque

A summary of most used dental plaque indices is presented in Table 1.2 (Fig. 1.6).

### 1.1.7 Bacterial Plaque Models

Various supragingival plaque biofilm models have been employed for the study of plaque formation, structure and antimicrobial susceptibility. Guggenheim et al. (2001, 2004) have described a defined multispecies model designed to mimic the composition of the supragingival plaque and used this model to study the structure and antimicrobial susceptibility. Several investigators have utilized in-mouth splints in healthy subjects in which supragingival plaque formed over time on the splints (Auschill et al. 2001; Wood et al. 2000; Zaura-Arite et al. 2001). Wimpenny et al. (1999) have described several different laboratory biofilm models that make use of a constant depth film fermenter using a plaque inoculum. The constant depth film fermenter models have been used to study the structure (Netuschil et al. 1998; Pratten et al. 2000; Wood et al. 2000) and spatial distribution (Auschill et al. 2001; Hope et al. 2002) of viable and nonviable supragingival plaque bacteria.

Attempts to obtain realistic subgingival plaque biofilms have been made by placing various insert materials into the periodontal pockets of periodontitis patients and then analyzing the bacterial components that colonized the inserts (Takeuchi et al. 2004; Wecke et al. 2000). Recently, Hope and Wilson (2006) have described the development of subgingival plaque on hydroxyapatite disks in a constant depth film fermenter. This model used a plaque inoculum and reached a steady state after 4 days. Although this is an excellent model for the study of subgingival plaque structure and viability, the apparatus for maintaining an anaerobic constant depth film fermenter is somewhat complex.

Numerous biofilm models have been described for the study of bacteria associated with the supragingival plaque. However, there are fewer models available for the study of subgingival plaque. One major challenge in understanding bacterial interactions in subgingival biofilms is the acquisition of undisturbed samples in which spatial relationships between bacteria are maintained, and for which the orientation within the oral cavity is known. So far, an analysis of the subgingival microbiota relied on sampling of bacteria either by paper points or by mechanical debridement. Both sampling procedures, however, disrupt the organization of biofilms. However, it has been indicated recently by Teles et al. (2008) that the use of curettes provided a reliable and reproducible method to obtain subgingival samples.

Biofilm formation has also been extensively studied using in vitro models, like flow chambers or chemostats. However, these studies might not necessarily reflect the situation in a periodontal pocket, and clearly have limitations regarding fastidious and so far uncultured microorganisms. The only method to study subgingival plaque, available so far, required the extraction of teeth (Wecke et al. 2000). Such samples have been useful in pioneering studies that map subgingival plaque structure on a macro scale using immunohistochemical approaches. Electron microscopy has also been applied to these samples to
Table 1.2 A summary of most used plaque indices

<table>
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<tr>
<th>Index, authors</th>
<th>Scoring criteria</th>
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| Simplified oral hygiene index – oral debris (Greene and Vermillion 1964) | The surface area covered by debris is estimated by running the side of an explorer along the tooth surface being examined. The following scoring system is used:  
  0 no debris or stain present  
  1 soft debris covering not more than one third of the tooth surface being examined or the presence of extrinsic stains without debris regardless of surface area covered  
  2 soft debris covering more than one third but not more than two thirds of the exposed tooth surface  
  3 soft debris covering more than two thirds of the exposed tooth surface |
| Plaque index (Ramfjord 1959) | The following teeth were selected as indicators of the periodontal condition within the dentition: maxillary right first molar 16, maxillary left central incisor 21, maxillary left first bicuspid 24, mandibular left first molar 36, mandibular right central incisor 41 and mandibular right first bicuspid 44. Record plaque after application of disclosing solution:  
  P0 no plaque present  
  P1 plaque present on some but not on all of the interproximal and gingival surfaces of the tooth  
  P2 plaque present on all interproximal and gingival surfaces, but covering less than one half of the entire clinical crown  
  P3 plaque extending over all interproximal and gingival surfaces but covering more than one half of the entire clinical crown |
| Plaque index (Quigley and Hein 1962) | The examiner made a quantitative estimate of the amount of stained plaque on the buccal, labial and lingual surfaces of the teeth as shown below:  
  0 no plaque present  
  1 flecks of stain at gingival margin  
  2 definite line of plaque at gingival margin  
  3 plaque extending on gingival third of surface  
  4 plaque extending on two thirds of surface  
  5 Plaque extending greater than two thirds of surface |
| Plaque index (Turesky et al. 1970) | Disclosed plaque is scored from 0 to 5 for each facial and lingual nonrestored surface only of all the teeth except third molars, as follows. An index for the entire mouth was determined by dividing the total all plaque scores by the number surfaces examined:  
  0 no plaque present  
  1 separate flecks of plaque at the cervical margin of the tooth  
  2 a thin continuous band of plaque (up to 1 mm) at the cervical margin of the tooth  
  3 a band of plaque wider than 1 mm but covering less than one-third of the crown of the tooth  
  4 plaque covering at least one-third but less than two-thirds of the crown of the tooth  
  5 plaque covering two-thirds or more of the crown of the tooth |
| Plaque index (Silness and Löe 1964) | The teeth which were examined were: maxillary right first molar 16, maxillary right lateral incisor 12, maxillary left first bicuspid 24, mandibular left first molar 36, mandibular left lateral incisor 32, and mandibular right first bicuspid 44. Assessment of soft deposits was made according to the plaque index system:  
  0 no plaque  
  1 a film of plaque adhering to the free gingival margin and adjacent area of the tooth. The plaque may be seen in situ only after application of disclosing solution or by using the probe on the tooth surface  
  2 moderate accumulation of soft deposits within the gingival pocket, or on the tooth and gingival margin which can be seen with the naked eye  
  3 abundance of soft matter within the gingival pocket and/or on the tooth and gingival margin |
### Table 1.2 (continued)

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<thead>
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<th>Index, authors</th>
<th>Scoring criteria</th>
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<tr>
<td><strong>1.1 Dental Plaque</strong></td>
<td>Each of the four surfaces of the teeth (buccal, lingual, mesial and distal) is given a score from 0 to 3, the plaque index for the area. The scores from the four areas of the tooth are added and divided by four in order to give the plaque index for the tooth. The indices for the teeth (incisors, premolars and molars) may be grouped to designate the index for the group of teeth. By adding the indices for the teeth and dividing by six, the index for the patient is obtained. The index for the patient is thus an average score of the number of areas examined</td>
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<td>Plaque control record (O’Leary et al. 1972)</td>
<td>This index was developed to give the therapist, hygienist or dental educator a simple method of recording the presence of plaque of individual tooth surfaces (mesial, distal, facial, lingual). The form also allows the patient to visualize his own progress in learning plaque control. After rinsing the stained solution, each stained surface is examined with an explorer for soft accumulations at the dentogingival junction. When found, they are recorded by making a dash in the appropriate spaces on the record form. No attempt is made to differentiate between varying amounts of plaque on the tooth surfaces. After all the teeth are examined and scored, an index can be derived by dividing the number of plaque-containing surfaces by the total number of available surfaces. The same procedure is carried out at subsequent appointments to determine the patient’s progress in learning and carrying out the prescribed oral hygiene procedures</td>
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<td>Navy plaque index (Grossman et al. 1973; Hancock et al. 1977)</td>
<td>This index is one part of the navy periodontal screening examination. This system of scoring stained plaque is applied on six teeth shown to be representative of the total mouth condition: 16, 21, 24, 36, 41 and 44 The tooth is separated into three major zones, the occlusal, the middle, and the gingival zone. By assigning all areas a score of one, more emphasis is placed on plaque adjacent the gingival tissues since the surface area is much smaller. The scoring is as follows: Area A, B, C score 1 a thin line of stained plaque approximately 1 mm or less adjacent to the gingival tissue, both facial and lingual Area D, E, F score 1 the stained plaque extends further into the gingival zone Area G and H score 1 the mesial and distal halves of the middle zone area, both facial and lingual Area I score 1 the occlusal area Score facial and lingual areas. The total score for each tooth is the sum total of all areas of stained plaque on that tooth</td>
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<td>Rustogi et al. modified navy plaque index (Rustogi et al. 1992)</td>
<td>Disclosed plaque is scored in each tooth area as present (scored as 1) or absent (scored as 0) and recorded in each of the nine areas of the buccal and lingual tooth surfaces. The index assesses the amount of plaque in the tooth area bounded by the tooth contact, the free gingival margin, and mesial or distal line angles. The use of this new index enables the examiner to evaluate and record both the gumline (or marginal areas) and interproximal areas of the tooth, thus giving these anatomical areas an increased importance</td>
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<td>New method of plaque scoring (NMPS) (Dababneh et al. 2002)</td>
<td>According to the NMPS, the visible facial or lingual surface of the tooth is divided horizontally into two major zones – the gingival one-third (zone A) and the remaining coronal two-thirds of the surface, T, which is further subdivided into three vertical thirds: mesial (zone B), distal (zone C) and middle (zone D). Each of zones A, B and C is given a score ranging from 0 to 3 depending on subjective evaluation of the proportional area, in thirds, of disclosed plaque on the relevant zone, i.e., 0 no plaque, 1 up to one-third coverage, 2 more than one-third and up to two-thirds coverage. 3 more than two-thirds coverage. The middle third zone, D, is scored on the basis of presence or absence of stained plaque as 1 or 0, respectively. This gives a score ranging from 0 to 10 per buccal or lingual surface. The aim of the NMPS was to emphasize the plaque scoring at the gingival and proximal regions of the tooth surface</td>
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