The structure of dental plaque

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Dental plaque has been defined as "the nonmineralized microbial accumulation that adheres tenaciously to tooth surface, restorations, and prosthetic appliances, shows structural organization with predominance of filamentous forms, is composed of an organic matrix derived from salivary glycoproteins and extracellular microbial products, and cannot be removed by rinsing or water spray" (102). The structure of dental plaque refers to the manner in which the elements of dental plaque, predominantly bacteria, are organized and interrelated.

Although this definition may suggest a vision of a rather static assembly of microorganisms contentedly sticking to a solid surface, nothing could be further from the truth. Indeed, the origin of dental plaque, its development and adaptation to changing environmental conditions are all governed by a dynamic, ever-changing equilibrium between the oral microbiota and a multitude of factors that differentially promote or inhibit the survival of its microbial constituents. For the sake of this discussion, the definition of dental plaque will be expanded to also include microorganisms that may be only loosely attached to assorted surfaces in the oral cavity, including firmly adherent bacteria.

Development and maturation of the microbiota of dental plaque

The earliest microbiota to colonize the mouth of the newborn infant is derived from the mother's genital tract, oral cavity and skin. Since the newborn infant has no teeth, the earliest microbial colonizers will be those that are able to adhere to the available surfaces: those lined by epithelium (83). Streptococcus salivarius, which is such an organism, becomes established within one day of birth (23). At the time of delivery, the child's mouth also becomes infected with Lactobacillus jensenii and Lactobacillus acidophilus (22). However, these and some gram-negative anaerobic species (60) remain at low levels and do not persist, presumably because of the absence of teeth, which normally favor their retention. Lactobacillus does not reappear in the mouth until after age 2, usually as Lactobacillus casei, the dominant oral species.

Streptococcus sanguis, an organism that preferentially colonizes tooth surfaces, can be recovered shortly after the teeth erupt (23). Streptococcus mutans, which favors the same ecological niche, was absent from the mouths of 91 predentate infants but was detected in 9 of 40 infants with only erupted primary incisors (9) and half of a group of 2-year-olds (57). It has been shown that, if mothers are treated so as to reduce their levels of S. mutans, the rate of transmission of S. mutans to their infants can be reduced by as much as one-third (57).

Actinomyces naeslundii, a predominant species of dental plaque, is recovered from 40% of young children. Actinomyces viscosus which is absent in infants, gradually increases in prevalence as the child grows older, with half the children colonized by age 7 (36). Veillonella species follow a similar pattern of increasing prevalence with increasing age (86).

Although most infants harbor a predominantly gram-positive, facultative microbiota, anaerobes can also be recovered (59), particularly after tooth emergence. As many as 61% of children aged 5–7 years harbor black-pigmenting gram-negative anaerobes (7, 30, 41, 84), as well as spirochetes (30, 84, 90). The proportion of these organisms and other strict anaerobes increases in adolescence and adulthood (106, 147) but may show a great deal of site-to-site variability (14), with local factors such as pH, eH and assorted bacterial interactions playing an important role (89). Some variability is also due to such factors as the state of health of the dentition (Table 1) and periodontal tissues (5, 6, 17, 31, 33), the medical status of the patients (8, 18, 19, 43, 87) and their racial origin (21, 120).

With the loss of teeth in older adults, some ecological niches such as tooth surfaces and gingival sulci that favor the retention of certain species are lost. As these specialized environments disappear there is a marked reduction, if not a complete elimination, of such species as Actinobacillus actino-
Table 1. Microbiology of the periodontal region: representative species in periodontal health and disease

<table>
<thead>
<tr>
<th>Health</th>
<th>Juvenile periodontitis</th>
<th>Rapidly progressive periodontitis</th>
<th>Refractory periodontitis</th>
<th>Adult periodontitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Streptococcus sanguis</td>
<td>Actinobacillus actinomycetemcomitans</td>
<td>Actinobacillus actinomycetemcomitans</td>
<td>Actinobacillus actinomycetemcomitans</td>
<td>Campylobacter rectus</td>
</tr>
<tr>
<td>Streptococcus mitis</td>
<td>Other bacterial species (?)</td>
<td>Porphyromonas gingivalis</td>
<td>Porphyromonas gingivalis</td>
<td>Bacteroides forsythus</td>
</tr>
<tr>
<td>Veillonella parvula</td>
<td>Actinomyces viscosus</td>
<td>Campylobacter rectus</td>
<td>Prevotella intermedia</td>
<td>Peptostreptococcus micros</td>
</tr>
<tr>
<td>Actinomyces naeslundii</td>
<td>Rothia dentocariosa</td>
<td>Eikenella corrodens</td>
<td>Bacteroides forsythus</td>
<td>Enteric rods</td>
</tr>
<tr>
<td>Actinomyces viscosus</td>
<td></td>
<td></td>
<td>Fusobacterium species</td>
<td>Candida species</td>
</tr>
<tr>
<td>Veillonella parvula</td>
<td></td>
<td></td>
<td>Selenomonas species</td>
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<tr>
<td>Streptococcus mitis</td>
<td></td>
<td></td>
<td>Eubacterium species</td>
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<tr>
<td>Veillonella parvula</td>
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mycetemcomitans, Lactobacilli, S. mutans, S. sanguis, and spirochetes. By contrast, species that do not depend on the presence of teeth, for example Candida, continue to thrive (2, 58, 133).

Methods to study the morphological features of dental plaque

Much of the above information was derived from cultural studies of supragingival dental plaque in subjects of various ages. At the same time, studies were ongoing to clarify the morphological characteristics of dental plaque. Some of these studies examined dental plaque on natural teeth; others collected dental plaque on tooth sections or artificial substrates placed in the oral cavity for defined periods of time. Plaque morphology has been examined by light and transmission electron microscopy of sections through the plaque layer (38, 39, 47, 51, 70, 82, 101, 122, 124, 125, 141). Other studies analyzed plaque by light microscopy of whole or dispersed plaque (50, 95, 112, 113, 116), or scanning electron microscopy of plaque deposits (24, 42, 53, 98, 99, 101, 115) or plaque deposit replicas (61,119).

The results of these and other investigations demonstrated that dental plaque is composed of a large variety of bacterial morphotypes with some significant heterogeneity in the appearance of the microbial deposits. Because of the lack of standardization, it has been difficult to relate the findings from one study to another. Furthermore, most of the studies were not designed to investigate the dynamics of plaque formation at the cellular level but rather to provide a cross-sectional view of plaque structure in one or more subjects at a particular point in time. However, in at least one study (79), data were obtained that provided some new insight into the process of dental plaque formation and maturation. A related study also demonstrated that detectable morphological differences could be observed among dental plaques from mouths with differing states of periodontal health (70). These studies are reviewed in greater detail later.

Microbiota of dental implants

With the increasing use of dental implants to replace missing teeth, investigators also have been exploring the oral microbiota associated with dental implants.
Early studies focused on differences in the composition of the microbiota associated with stable and failing implants. The reports generally indicated that healthy implants have a microbiota similar to that of healthy teeth, while failing implants have a microbiota similar to that found around teeth with periodontitis (1, 91, 93, 110, 111, 117). It is likely, however, that around failing implants the composition of the microbiota may be related to the nature of the implant failure. Thus if a microbiota is found resembling that of adult periodontitis, the failure is probably infectious in nature (infectious peri-implantitis), since in failures due to traumatic injury the microbiota is similar to that around stable implants or periodontally healthy teeth (114). These dramatic differences between failures due to trauma and failures due to infection presumably apply only to cases that fall clearly either into one or the other category. The possibility exists that an implant failure initially due to trauma may become secondarily infected, with an accompanying shift in the composition of the microbiota. Resolving the infection may not necessarily solve the primary clinical problem, which is occlusal in nature.

It is noteworthy that spirochetes and Porphyromonas gingivalis do not colonize healthy implants in an edentulous mouth as readily as they do implants in partially edentulous patients (92, 97, 105, 107). The frequently reported absence of spirochetes around healthy implants in edentulous mouths coupled with their presence in low numbers around healthy fixtures in dentulous patients suggests that diseased teeth may serve as a source of infection for dental implants (107). Should this be the case, controlling periodontal infections prior to implant placement ought to improve the long-term survival rate of dental implants.

Since a variety of materials are used in the manufacturing of dental implants, some studies have examined the influence of materials on plaque formation in situ (46, 54, 96, 108, 109, 146). It appears that materials can affect the rate of early plaque formation, that is, within the first few hours of exposing the fresh surface to the intraoral environment. After 4 hours in the mouth, some differences were noted in the rate of plaque formation on disks of assorted materials, including titanium and hydroxyapatite, that were glued to the gingiva. Quirynen et al. (108) and Weerkamp et al. (146) observed a direct correlation between plaque accumulation on a variety of substrates and increasing surface free energy. However, surface roughness emerged as a more important factor than surface free energy in promoting early plaque accumulation. After 48 hours, there was no longer any detectable difference in the plaque collected on the different materials by Nakazato et al. (96) These observations were supported by Quirynen et al. (109), who detected significant differences between rough and smooth surfaces for as long as 6 days. An earlier suggestion that titanium possesses some antimicrobial properties has been refuted (54).

Use of artificial substrates to study dental plaque formation

The effect of certain materials on plaque formation has also been of interest to other investigators attempting to identify suitable experimental substrates for studying plaque development in vivo. Berthold et al. (10) noted a similar plaque morphology on enamel and polished epoxy resin after 2-3 days of plaque formation. However, the morphology differed from that of plaque collected on sand-blasted Mylar strips and sand-blasted epoxy resin. Plaque thickness tended to be greater on the roughened surfaces, a finding that is compatible with Nakazato's (96) observation that roughness is more important than surface free energy in promoting early plaque formation on assorted implant materials.

Lie (62, 65) produced veneers composed of a mixture of epoxy resin and hydroxyapatite that could be glued to the buccal surfaces of posterior teeth in human volunteers for various periods of time. His short-term findings indicated that organic pellicles tended to adsorb to the hydroxyapatite particles earlier and form more well-defined adherent layers than on the epoxy resin in which the hydroxyapatite particles were embedded (66, 67). Plaque formation also tended to be faster on hydroxyapatite than epoxy resin. However, the differences in rates of plaque formation and maturation had disappeared by 48 hours (65). Colonization began in pits and fissures from which the bacteria spread as monolayers over the adjacent surfaces. Scanning electron microscopy revealed a predominance in early plaque of coccoid cells, with some rods and a few filaments.

Structural features of developing dental plaque

Listgarten et al. (79) recruited human volunteers in need of full-crown restorations to participate in a
study of dental plaque development. A series of identical epoxy resin crowns were fabricated for each subject. These crowns were worn for different time periods, according to a predetermined schedule, to provide plaque samples of different maturities from the same anatomic site in each volunteer. The periods of plaque formation varied from 1 day to 2 months. The experimental design allowed the investigators to obtain sections through undisturbed dental plaque that could be studied both at the light and transmission electron microscopic level. The resulting data provided for the first time information on the chronological changes that take place in supragingival dental plaque from a specific location in the mouth of several human subjects.

The following is a summary of the main structural features that characterize developing human dental plaque. It is based in part on results from the epoxy resin crown model as well as other reports in the literature that have investigated various stages in the plaque formation process (13, 15, 16, 137, 138). Since the morphology of developing dental plaque on tooth surfaces and smooth epoxy resin surface appears to be similar, the description that follows, although referring to the tooth surface, is based on data from both natural teeth as well as artificial substrates. Where differences are known to exist, these will be pointed out.

Formation of supragingival dental plaque

Within minutes after a tooth surface is freshly cleaned, an acquired pellicle, composed primarily of salivary proteins, is adsorbed to the exposed hydroxyapatite crystallites (4, 20, 69, 100, 128). Morphologically similar coatings are produced on various other substrates, albeit at different rates (11, 63). Their chemical composition may be affected by the physical and chemical nature of the surfaces and the distribution of surface charges (64, 134). Because of the speed with which acquired pellicles are formed, it is unlikely that bacterial colonization will begin on any surface without prior formation of an organic coating (Fig. 1).

The first bacteria to colonize the surface are mostly gram-positive, facultative cocci, mainly of the *Streptococcus* species, and coccobacilli, mainly *Actinomyces*. *Veillonella*, a genus of gram-negative anaerobic cocci, is also an early colonizer. Initial colonization is characterized by a transient, reversible attachment to the tooth. In time, the attachment becomes stronger and less readily ruptured. The attachment is mediated through sticky proteo-
Fig. 1. One-day-old supragingival plaque on epoxy resin crown. The bacterial deposit (B) is attached to an electron-dense pellicle (P) which lines the surface of the epoxy crown (E). ×24,000; bar=1 μm.

Fig. 2. One-week-old supragingival plaque on epoxy resin crown. The bulk of this deposit consists of columnar colonies of coccoid bacteria (C) growing from the epon surface (E) outward. Some filamentous bacteria (F) have colonized the plaque surface. ×1200.

Fig. 3. One-week-old supragingival plaque on epoxy crown. Three different coccoid bacterial morphotypes (labelled A through C) have formed columnar microcolonies that compete for space as they grow away from the crown surface (toward top of figure). ×5400; bar=1 μm.

to 2 months old as well as in supragingival plaque of unknown age obtained from extracted teeth (38, 39, 51, 70, 79, 122). It can be considered as the typical, relatively stable structure of mature, supragingival dental plaque.

Calculus

Mineralization of supragingival as well as subgingival plaque gives rise to calculus. Mineralization generally proceeds in an incremental pattern from the
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inner zones of the dental plaque outward. This pattern of mineralization may give rise to Liesegang rings, concentric rings reflecting successive phases of mineralization, which are particularly noticeable in old calculus deposits. For a comprehensive review of calculus formation, see Schroeder (121). As new layers of plaque become mineralized from the deeper layers outward, the layering pattern throughout the deposit becomes emphasized, giving the erroneous impression that the entire deposit grew by apposition of bacteria on the surface of the underlying calculus. Although calculus may grow in an incremental fashion through the progressive mineralization of the overlying plaque, this growth pattern differs markedly from that of dental plaque. As described earlier, increased thickness of plaque deposits, particularly in the early stages, primarily depends on bacterial proliferation and competitive growth of microbial colonies within the plaque mass.

Formation of subgingival dental plaque

The undisturbed growth of supragingival plaque gradually results in soft tissue alterations in the adjacent gingiva. Beginning within a few days of undisturbed plaque formation, the gingival margin begins
to show typical inflammatory changes, including redness and swelling. The latter changes result in the creation of a deepened gingival sulcus (pseudo-pocket), which provides a relatively anaerobic environment for the development of an anaerobic microbiota. Anaerobic bacteria that colonize this subgingival region include motile rods and spirochetes. They are able to increase their mass by contributing to the deepening of the sulcus, thereby increasing the volume of their ecological niche. Because many of the subgingival microorganisms are motile, the structural organization of this microbial population is quite different from that seen supragingivally.

A relatively thin layer of adherent bacteria covers the tooth surface. Rods and filaments tend to be arranged in a palisading pattern, with the long axis of the cells perpendicular to the tooth surface. Unique bacterial aggregates, resembling test-tube brushes, can be found attached to the adhering plaque and extending into the space between the bacterial layer and the adjacent soft tissue wall (Fig. 5). The "bristles" of these test-tube brush formations are gram-negative filamentous bacteria, some of which may be flagellated. The axial portion of the test-tube brush consists of a single or several long filaments held together by an amorphous extracellular matrix. The bulk of the subgingival microbiota consists of a complex mixture of predominantly anaerobic bacteria that surround and cover the test-tube brush formations. The lack of well-defined microbial colonies in this environment may be due to the high degree of motility of the resident microbiota. The peripheral region of the subgingival microbiota is composed of a high concentration of spirochetes (Fig. 6, 7) that are in direct contact with the gingival tissue wall as well as the apical lining of the sulcus or pocket. Sometime a layer of leukocytes, mostly neutrophils that have migrated out of the junctional epithelium, separates the bacterial mass from the sulcular or pocket epithelium.

The bottom of the sulcus or pocket is formed by the coronal, desquamative surface of the junctional epithelium, which is attached to the tooth surface on one side and to the gingival connective tissue on the other. This portion of the junctional epithelium is subject to bacterial as well as mechanical injuries, which may result in enlarged intercellular spaces and vertical tears in the epithelium. These alterations in the integrity of the junctional epithelium allow a gradual apical colonization of the tooth surface by coccoid cells and rods. Irregularities in the root surface, such as those caused by localized root resorption, may shelter plaque microorganisms and contribute to their retention at such sites. The tendency for bacteria to colonize tooth surfaces freshly exposed because of disruptions in the junctional epithelium leads to a gradual deepening of the sulcus or pocket.

Thus, a distinctive subgingival microbiota, predominantly composed of gram-negative, anaerobic bacteria, including a number of motile species, becomes established in the gingival sulcus between 3-12 weeks after the beginning of supragingival plaque formation. The establishment of this subgingival microbiota is dependent on a series of interrelated events: the successive colonization of the tooth surface by different bacterial populations. Each of these microbial populations appears to facilitate the colonization of this region by the next wave of bacterial settlers, with the ultimate establishment in the subgingival region of a predominantly anaerobic, gram-negative microbiota. Most bacterial species currently suspected of being periodontal pathogens are anaerobic, gram-negative species whose main ecological niche is the subgingival region. In this protected environment they are in an excellent position to participate in the destruction of the periodontal tissues, with the resulting maintenance and expansion of their subgingival habitat.
Fissure plaque

The microbial colonization of occlusal fissures is of interest because of the relationship of the fissure microbiota to the pathogenesis of dental caries. In order to obtain a dynamic picture of the colonization process in humans, Löe et al. (82) developed an artificial fissure made of Mylar foil, which they embedded in the occlusal surface of human mandibular molars for periods of up to 2 months (55, 139, 142). In the first week, the fissure first contained mostly food debris, later replaced by gram-positive cocci, mostly streptococci, short rods and yeast cells. Later, lactobacilli appeared and increased in number with time (136). Some filaments appeared after 3 weeks. Some foci of mineralization were observed at about the same time. After 2 months, the contents consisted mostly of gram-positive cocci and short rods, many of which had undergone lysis, and some plant wall material. No fusiform or spirillar forms were observed at any time (55, 139, 142). This type of plaque did not exhibit the dynamic changes observed on smooth tooth surfaces (139).

Relationship of plaque structure to clinical status

The qualitative differences in the microbial populations observed over time in developing dental plaque on epoxy resin crowns have an interesting parallel in the natural dentition. Natural teeth were extracted from patients with a diagnosis of health, gingivitis or periodontitis (70). As the severity of the periodontal condition increased, the composition of the associated microbiota tended to replicate that observed during plaque development on the resin crowns.

The dental plaque associated with the periodontally healthy tooth was predominantly that of early, supragingival plaque. It was characterized by a predominantly coccoid microbiota, including many gram-positive species. Cultural studies of such plaques indicate that this microbiota is composed predominantly of facultative bacteria. The gingivitis-associated microbiota was characterized by a marked increase in the microbial mass as well as a relative increase in the proportion of gram-negative bacteria, motile rods and filaments. In adult periodontitis, an abundant, complex microbiota was observed in the periodontal pockets. This bacterial population was predominantly gram-negative, included test-tube brush formations similar to those noted in the late maturation stages of developing dental plaque on epoxy resin crowns and a large proportion of spirochetes preferentially distributed on the periphery, or tissue side, of the microbial mass. Cultural studies of such plaques indicate that this microbiota is predominantly anaerobic.

In juvenile periodontitis, the dental plaque appeared less voluminous and less complex than in adult periodontitis. Its morphological features did not have a counterpart in the stages observed during plaque development on epoxy resin crowns. Frequently, the exposed root surface within the pockets was covered with a lobulated dental cuticle of varying thickness. Although in our study (70) this lobulated cuticle was typically observed in deep pockets of patients with juvenile periodontitis, Friedman et al. (40) also reported similar cuticles in adult periodontitis. Closely related to the cuticle were large, gram-negative coccoid cells, later identified by Berthold et al. (12) as A. actinomycetemcomitans, and some palisading thin, gram-negative fusiform bacteria resembling Capnocytophaga species.

The morphological differences in the composition of the supragingival and subgingival microbiotas in humans, originally documented in sections of epoxy resin crowns (119) as well as dental plaque sections of extracted teeth with different periodontal conditions (70), could also be confirmed with technically simpler methods, such as dark-field and phase-contrast microscopy. By using dark-field microscopy for differential counting of bacterial morphotypes in bacterial scrapings from the subgingival region, Listgarten & Hellédén (75) were able to show statistically significant differences in mean percentages of various morphotypes between healthy and diseased sites. Healthy sites had a predominantly coccoid microbiota, with few if any spirochetes and motile rods as compared with diseased sites in patients with adult periodontitis. Furthermore, after mechanical debridement of periodontal pockets, Mousquès et al. (94) demonstrated that bacterial recolonization to pretreatment levels averaged 6 weeks, a time interval compatible with the chronological development of dental plaque in the epoxy resin crown model (119).

Their findings support the concept that plaque maturation in a diseased site is a process requiring the orderly succession of bacterial populations until one is left that is ultimately dominated by the anaerobic, gram-negative microbiota ordinarily found in adult periodontitis pockets. Routine professional cleanings coupled with daily oral hygiene prevent
such a microbiota from developing, by regularly removing recently formed dental plaque, thereby interfering with its maturation and ability to provide a hospitable environment for a number of periodontal pathogens.

Our improved understanding of the dynamic nature of plaque formation has helped clarify the biological basis underlying the effectiveness of plaque control on the clinical status of the periodontal structures. Even though mechanical and some forms of chemical plaque control are nonspecific methods of reducing plaque mass, they also affect the qualitative nature of dental plaque. This is accomplished through regular interference with the normal plaque maturation process, which leads to the establishment of an anaerobic environment favorable to the proliferation of most periodontal pathogens. Of course, there are exceptions to this general observation. One example is that of A. actinomycetemcomitans infections, which may persist despite repeated mechanical debridement. This may be due, in part, to the ability of A. actinomycetemcomitans, a facultative bacterium, to colonize the dentition without the prior establishment of an anaerobic environment. It has also been suggested that the persistence of A. actinomycetemcomitans may be due to its ability to invade and persist within periodontal tissues (25, 27). Similarly, other periodontal pathogens may be able to survive mechanical debridement and lead to the production of periodontal diseases that have been described as refractory to standard treatment.

Some reports indicate that, in relatively short-term studies, supragingival plaque control has no detectable influence on established subgingival plaque in deep pockets, that is, with probing depths of 7 mm or greater (56, 75). However, studies that included shallower pockets or extended for one or more years suggest that supragingival plaque control not only results in beneficial clinical improvement but also affects the composition of subgingival plaque (29, 88, 131). Yet, even in long-term studies, pockets with probing depths of 7 mm or greater were not significantly affected by supragingival plaque control alone (29). The apparent discrepancy between these results may be due to the cumulative influence of a relatively minor effect of supragingival plaque control on the subgingival microbiota. Waerhaug (145) demonstrated that toothbrushing was able to disrupt subgingival plaque up to 0.9 mm from the gingival margin. This effect may not be sufficient to cause immediate changes in the composition of the subgingival plaque of deep pockets. In time, however, the cumulative effect of brushing and periodontal prophylaxis may lead to a gradual change in subgingival plaque composition of pockets shallower than 7 mm. This qualitative shift may be mediated not only through a direct effect of mechanical debridement but also indirectly through an altered immune response (34, 35), and environmental changes, such as reduced gingival inflammation and decreased pocket depth, that follow improved oral hygiene.

Clinical relevance of plaque structure and composition

There is increasing evidence that the amount and composition of dental plaque are directly related to the health status of the periodontal tissues (68). It is also obvious that evaluation of plaque structure and composition by means of histological sections is not practical in a clinical setting. Therefore, indirect means of obtaining this information are needed if the information is to be applied to the management of patients with periodontal problems.

The studies of dental plaque by light and electron microscopy referenced earlier were crucial in demonstrating that plaque structure is intimately related to microbial composition. Specific bacterial species tend to occupy and thrive under specific environmental conditions and become organized into communities that give dental plaque its unique structural characteristics. Microscopic analysis of dispersed subgingival plaque samples, which provides a differential count of bacterial morphotypes, provided the first evidence that a correlation exists between differential morphotype counts and the clinical status of patients (3, 72, 75, 76, 85, 94, 103, 104, 129, 148). This method of monitoring dental plaque destroys the structural framework of the subgingival microbiota. However, the differential morphotype count does reflect the nature of the intact microbial community, albeit in a rather crude way.

Because the method is rapid, noninvasive and inexpensive, it has been used successfully to answer some basic questions regarding the relationship of plaque composition to certain clinical problems, including differentiation between periodontal health and disease. Microscopic monitoring played a key role in the early demonstration that mechanical debridement not only decreases plaque mass but also radically changes the composition of the subgingival microbiota for several weeks postoperatively (71, 78, 94). When mechanical debridement is supplemented with antibiotic therapy, spirochetes may not
Microscopic monitoring of bacterial morphotypes in patients with untreated periodontitis also provided some early insight into the differential distribution of bacteria between sites within subjects and between subjects (37). This information is essential for developing and optimizing sampling methods. Despite some variability in the distribution of bacterial morphotypes between the deepest pockets in individual patients, most of the variance originates from samples between subjects (37). These findings led to the conclusion that pooled samples of the deepest pockets might be the optimal method of sampling pockets for clinical applications. These conclusions have been generally confirmed and extended on the basis of studies that used other experimental designs and identification methods: immunofluorescence (26) or DNA probes (48, 49, 118). These studies have also demonstrated that the number of sampled sites required to identify the presence of a particular microorganism increases as the prevalence and recovery level of the organism decreases.

Despite the excellent correlations that have been described between certain periodontal conditions and the composition of their accompanying microbiota, few investigations have demonstrated that a given microbial population is indicative of future clinical events. Listgarten & Levin (76) reported that high proportions of spirochetes and/or motile rods were predictive of disease recurrence in a treated periodontitis population from whom maintenance visits were withheld or were provided at very infrequent intervals (80). However, this observation was not valid for patients with regularly scheduled trimonthly maintenance visits, either for spirochetes and/or motile rods (80) or for other suspected pathogens such as A. actinomycetemcomitans, Prevotella intermedia or P. gingivalis (81). Nor was it possible to demonstrate, in patients receiving regular prophylaxis, that sites with clinical evidence of breakdown were microbiologically distinct from those without discernible deterioration (77, 81). These results, which at first may appear contradictory, are most likely due to the disruptive influence of routine prophylaxis on plaque structure and composition, with the resulting loss of diagnostic reliability.

Today several methods are available to monitor the periodontal microbiota. With the emergence of certain species as likely pathogens, the emphasis is to monitor selected species, rather than the entire plaque population.

However, it is apparent that the mere presence of one or more pathogenic species in a subject is insufficient to produce significant deterioration in the periodontal status of this individual. This finding can be explained by the multiplicity of other factors that contribute to the clinical outcome, including host and environmental factors as well as local bacterial interactions that can modulate the virulence of otherwise pathogenic species (28, 73, 132, 135).

Nevertheless, in patients who have received standard periodontal therapy and exhibit evidence of satisfactory plaque control, microbiological testing may provide an additional and valuable diagnostic aid for the therapist who suspects a contributing periodontal infection and seeks some guidance with respect to antimicrobial treatment (74). Such testing may be of special value for older patients in a general practice, and more specifically for patients at high risk for periodontitis. It is of little value, however, as a screening test for the general population, particularly the younger age groups (32). As our knowledge of periodontal microbiology increases, it is likely that microbiological diagnosis will play an increasingly important role in the management of patients with infectious forms of periodontal disease.

References


46. Grisano AG. Biomaterial-centered infection: microbial

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