Dental Implant Infections

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Dental implants provide a restorative tool to support crowns, bridge abutments, and removable dentures. Osseointegrated implants are titanium posts that are surgically implanted in alveolar bone. A tight immobile bond (osseointegration) forms between bone and titanium, and prosthetic and restorative fixtures are attached to the implants. Titanium implants differ from natural teeth, which may make them more susceptible to mechanical stress. A small proportion of implants are not successful and may fail due to infection. The microbiota of implants is similar to that of teeth in similar clinical states. Implants that fail because of mechanical stress are colonized by species associated with healthy teeth. Infected implants are colonized by subgingival species, including *Porphyromonas gingivalis, Bacteroides forsythus, Fusobacterium nucleatum, Campylobacter gracilis, Streptococcus intermedius,* and *Peptostreptococcus micros.* Different patients may be colonized by different microbial complexes, indicating that optimal treatment should be directed to the specific infection.

Osseointegrated dental implants are a major tool in prosthetic dentistry and are used to support many different configurations of tooth replacements ranging from single teeth to full dentures [1]. Although most implants are extremely successful, with survival rates of up to 98% for implants placed in controlled clinical settings [2], implants can fail; two principal reasons for this failure are mechanical stress or bacterial infection [3]. Microbial colonization associated with implants, like that associated with periodontal infections, is influenced by differences in the indigenous microbiota of individuals and by clinically different sites within individuals.

Dental Implant Design, Placement, and Restoration

Osseointegrated dental implants are manufactured from surgical grade titanium; they have a cylindrical post or open cylinder (basket) design and may be threaded or nonthreaded. Implants are inserted in alveolar bone so that the implant is immobile after placement [4]. During healing, a direct bone to implant bond called osseointegration is formed [5], a process that takes 3–6 months. The osseointegrated anchorage resembles the osseous joint of ankylosed teeth to alveolar bone, which may form following tooth reimplantation, rather than the periodontal ligament of connective tissue that attaches normally anchored natural teeth.

There are several stages in the procedure from inserting implants into alveolar bone and putting them in to function as a support for a prosthesis. Implants are surgically placed in bone, which, for one-stage implants, leaves part of the implant in direct contact with the oral cavity. For two-stage implants, the titanium implant is covered with oral attached mucosal tissue during surgery, allowed to heal, then reexposed surgically 3–6 months later [6]. An abutment, reaching through the mucosa into the oral cavity, is attached to the implant at this time. After 2–3 weeks of soft-tissue healing, the prosthodontic work is started, consequently leading to masticatory loading of the implant.

During the time of implant healing, the space may be covered by a temporary crown, by a bridge, or by a removable denture. The final dental restoration is fixed to the implant post with cement or may be removable and held in place with a screw. Whereas earlier implants were used mainly in edentulous patients to support full dentures, they are now increasingly used in partially edentulous subjects to replace single or multiple missing teeth. Single tooth replacements may be a crown similar to that used over natural teeth. To support multiple missing teeth, an intricate abutment apparatus is placed on implants. Because of differences of attachment of implant (osseointegration) and teeth (periodontal ligament) to bone, bridges on implants are generally fixed only to implants.

Comparisons of Teeth and Implants

Although they may function as prosthetic replacements for teeth, titanium implants are not teeth. Their arrival into the oral cavity, their connection to supporting alveolar bone, and the connective tissues involved differ markedly from natural teeth [7]. Teeth usually erupt into the gingivally healthy environment of childhood and adolescence. The gingival or periodontal environment changes with time and may become diseased with increasing loss of periodontal support. In contrast, implants are generally placed in the totally different environment of adult gingival or periodontal tissues and microbiota. The initially

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sterile and clean titanium surface of an implant offers a new surface in the oral cavity for adherence and colonization.

The connection between teeth and implant with supporting tissues differs markedly [8]. In contrast with the perpendicular fibers of the periodontal ligament sling around teeth, supra-crestal connective tissue fibers run parallel to implants [7, 9]. It is unclear whether this difference provides a faster route for infection around implants than around teeth. The hard tissue join of osseointegration appears to make implants more vulnerable to mechanical stresses than teeth, perhaps magnifying the effects of minimal levels of inflammation. In contrast, some of these differences seem to favor implants. For example, while teeth and implants in place both maintain alveolar bone, re-placement of a lost tooth with an implant appears to be more successful than reimplanting teeth [10].

Implant Failure

The first 2 years after implant placement appear to be the most critical in determining whether any implant will be suc-cessful. The nature and consequences of infection around im-plants depend, in part, on the site affected along the implant. Infection at the gingival margin appears analogous to gingivitis around teeth; further along the implant where the attachment is to soft tissues close to the alveolar bone crest, infection appears analogous to periodontitis. At a third, deeper area at the level of direct titanium bone join, infection resembles oste-itis. The deeper infection in bone adjacent to dental implants does not have a direct equivalent in periodontitis, in which case the bone is usually resorbed at a site remote from the periodontal pocket. This difference between teeth and implants is probably related to their different attachment to surrounding tissue.

The impact of implant failure can be greater than that of natural tooth loss because of rapid loss of peri-implant bone [11] and because of the impact on the supported crown, bridge, or denture. The degree of infection around an implant can be severe, requiring hospital admission in certain cases [12]. Implants are placed with the expectation of being either re-stored with a crown, which may be a bridge abutment, or to support an overdenture. Thus, the ability to assess the success of an implant before placement would be at least as useful as the ability to assess the longevity of teeth.

It is difficult to estimate the number of symptomatic or failed implants since in many cases they are treated or removed and not documented. In cases where implants fail early because osseointegration does not occur, they are usually removed. One report cited a 15% implant failure rate [13]. Implants that fail after demonstrated osseointegration are considered to be ''late failures.'' These late failures may occur at any time but are usually within the first 2 years [14].

Several characteristics have been associated with, and are suspected risk factors for, implant failure [15]. Many of these factors are anatomical or mechanical; these factors include in-sufficient alveolar bone height or density [16, 17] or poor posi-tioning of the implant such that it is at a mechanical disadvan-tage or cannot be easily restored. Modern technology has addressed many of these factors. Methods are now available for computer analysis of radiographs to accurately assess the suitability and amount of bony support and to determine the proper positioning of implants [18]. The major mechanical problem after loading is attributed to ''occlusal trauma'' to implants from overloading. This trauma leads to loosening or fracturing of the implant components or an abnormal loss of supporting alveolar bone [19].

Other suspected risk factors for implant failure are directly related to infection and include a history of periodontal or endodontic infection. Infection or trauma to the bone while preparing the implant sockets appears to be the cause of early implant losses [20]. Tobacco smoking has also been considered a risk factor for dental implant failure [15, 21].

Clinical Assessment of Dental Implants

Failing implants are frequently characterized by loss of sup-porting bone, which is assessed by periapical radiographs. Fail-ing implants may have a probeable pocket around the implant and may be associated with increased implant mobility. Patients with failing implants may have significant spontaneous pain; pain on twisting (torque), clenching, percussion, or palpation; signs of local inflammation including bleeding and tenderness to probing; and/or peri-implant redness and swelling [22, 23].

Microbiota of Dental Implants

The sequence of microbial colonization of implants is similar to that for teeth in the same oral cavity. In microbiological studies of the peri-implant microbiota, darkfield microscopy, nonselective and selective culture, and DNA probe assays were used. Species that colonize dental implants have included spe-cies that characterize gingivally healthy sites and gingivitis sites (e.g., *Streptococcus sanguis, Actinomyces viscosus,* and *Actinomyces odontolyticus*) as well as putative periodontal pathogens (e.g., *Porphyromonas gingivalis, Prevotella inter-media, Prevotella melaninogenica,* and *Fusobacterium* species [24–27]). Other species that are infrequently isolated from periodontal samples include staphylococci, enteric rods, pseu-domonads, enterococci, and yeasts; these species have also been reported from infected implants [13, 25, 28, 29]. During longitudinal monitoring of individual sites in both an experi-mental gingivitis and periodontitis model in dogs [30], and in partially edentulous humans who were receiving implants [31], similar groups of species were detected on teeth and implants.

The peri-implant microbiota differs depending on whether the individual is edentulous or partially edentulous. In particu-lar, *P. gingivalis* was rarely isolated from edentulous individu-als [32], which makes an interesting analogy to the paucity of *P. gingivalis* isolated from patients with pericoronitis [33] and

those in the early stages of periodontitis [34], suggesting that in both cases the deeper periodontal pocket niches favored by *P. gingivalis* were missing.

The microbiota of healthy and diseased dental implants appear to differ depending on the suspected etiology of implant symptoms. The peri-implant microbiota of implants with symptoms associated with occlusal trauma was predominated by streptococci and was similar to the microbiota of gingivally healthy sites [23]. This situation appears to have a parallel in initial periodontitis, where some sites show loss of periodontal attachment with recession and are colonized by species associated with healthy teeth [34a]. Implants that were failing and that had an infectious etiology were colonized by putative periodontal pathogens including spirochetes, *Peptostreptococcus micros*, *Fusobacterium* species, enteric gram-negative rods, and yeasts; these pathogens were found in high proportions of the microflora cultured. No microbiological differences were found between pure titanium and hydroxyapatite-coated implants [25] or between one- and two-stage implants [35].

In our laboratory we compared healthy and symptomatic dental implants using nonselective culture. Failing implants were identified either by increases in probing depth or suppuration or by recent increased bone loss assessed from periapical radiographs. When examined clinically, the symptomatic implants had deeper probing depths, bled more frequently on probing, and had hotter peri-implant temperature readings than healthy implants.

The dominant species characterizing symptomatic implants (figure 1) were the gram-negative species *Bacteroides forsythus* (6 of 12 sites), *Fusobacterium nucleatum* subspecies *vincentii* (4 of 12 sites), and *Campylobacter gracilis* (7 of 12 sites). Only one site harbored *P. gingivalis*. Gram-positive species isolated from the symptomatic implants included *Streptococcus intermedius* (7 of 12 sites) and *P. micros*.

Healthy implants did have ''gingivitis,'' as was indicated by positive plaque and redness scores. The microbiota of healthy implants included health-associated species such as *S. sanguis, Streptococcus oralis,* and *Streptococcus gordonii* and gingivitis-associated species such as *Actinomyces naeslundii* and *Capnocytophaga gingivalis* [34]. Overall, the microbiota of the peri-implants and the periodontal infections was similar, as had been described previously [24–27].

Although cultural methods have some advantages for studying the microbiota around implants by detecting species in small samples and by identifying unexpected or new species, they are labor intensive and time-consuming. A new method for microbial analysis of the peri-implant microbiota that overcomes some of these shortcomings is the use of DNA probes in a checkerboard assay [36].

DNA probe technology has revolutionized the analysis of subgingival bacterial samples and allows rapid detection of multiple species in one assay procedure. This methodology can handle many more samples per unit time than can cultural methods while still routinely assaying up to 30–40 species.
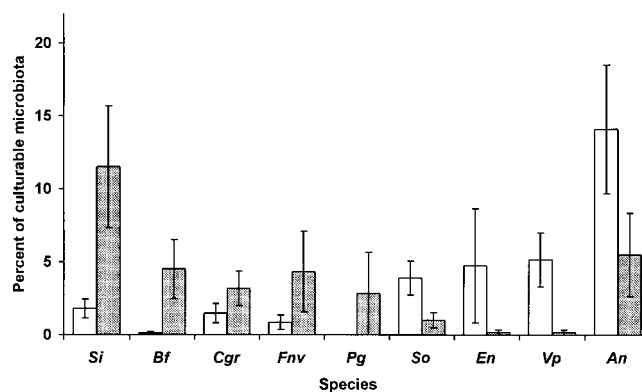

Figure 1. Predominant culturable microbiota of successful (☐) and failing (▩) dental implants. Nineteen healthy implants from 12 individuals were sampled. Twelve symptomatic implants from 12 different individuals were sampled. A healthy and a symptomatic implant were sampled from three individuals. The microbiota of healthy implants and symptomatic implants was markedly different for these individuals. Data represent mean percentage (±SEM) of the total culturable microbiota represented by each species. *Si = Streptococcus intermedius; Bf = Bacteroides forsythus; Cgr = Campylobacter gracilis; Fnv = Fusobacterium nucleatum* subspecies *vincentii; Pg = Porphyromonas gingivalis; So = Streptococcus oralis; En = Eubacterium nodatum; Vp = Veillonella parvula;* and *An = Actinomyces naeslundii.* Symptomatic implants were colonized by higher proportions of *S. intermedius, B. forsythus,* and *F. nucleatum,* whereas healthy implants were colonized by higher proportions of *A. naeslundii, V. parvula,* and *E. nodatum.*

This approach allows a comprehensive microbial profile to be obtained from many more sites per individual and for more individuals. Target species for the probe assay can be selected on the basis of the results of nonselective cultural studies, and thus these findings can be expanded to a much larger population of individuals and sites.

Figure 2 shows the microbiota of three patients with failing implants. Just before the symptomatic implants were removed, samples were taken using either steel scalers for subgingival sites or graphite scalers for peri-implant sites. Samples were placed in 100 μl of buffer (Tris-EDTA), and an equal volume of 0.1N NaOH was added within 30 minutes to stabilize the sample DNA [36]. The microbiota of mesial sites of all teeth or implants present was analyzed with use of the checkerboard DNA probe assay. The three patients had different microbial profiles. Subject 1 had low levels of species, except for *S. gordonii* and *S. intermedius,* that were similar to those reported for some patients with refractory periodontitis [37]. Subject 2 had elevated levels of *P. gingivalis, B. forsythus, P. intermedia,* and *Prevotella nigrescens.* Subject 3 had a profile characterized by higher levels of *F. nucleatum* subspecies *vincentii, S. intermedius,* and *Campylobacter rectus.* These results illustrate that different oral microbiota can be associated with infections in different individuals. It is possible that implant infections, like periodontal infections, may be influenced by the microbiota of the individual before infection. This hypothesis would sug-
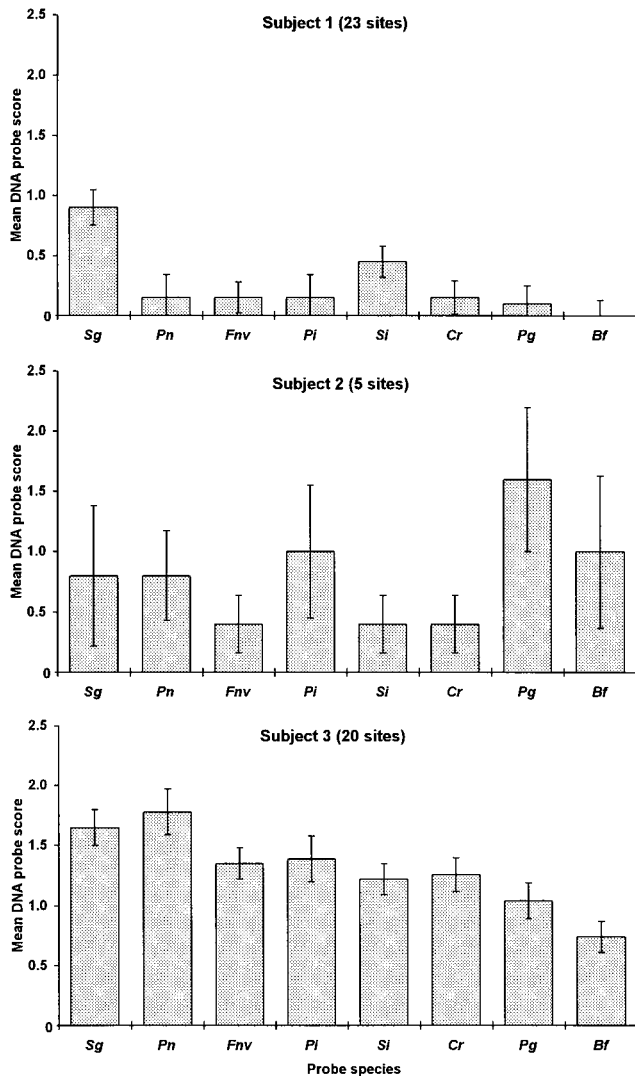
Figure 2. Oral microbiota of three patients whose dental implant was failing because of infection. The mesial sites of all standing teeth and implants were sampled for each subject, and samples were analyzed with use of the checkerboard DNA probe assay. Data represent mean DNA probe level ($\pm$SEM) of each species, where a level of 1.0 is equivalent to a microbial count of $\geqslant 10^4$ and $< 10^5$ per sample and 2.0 is equivalent to $\geqslant 10^5$ and $< 10^6$ per sample. *Sg = Streptococcus gordonii; Pn = Prevotella nigrescens; Fnv = Fusobacterium nucleatum* subspecies *vincentii; Pi = Prevotella intermedia; Si = Streptococcus intermedius; Cr = Campylobacter rectus; Pg = Porphyromonas gingivalis;* and *Bf = Bacteroides forsythus.*

gest that pretreatment screening of the microbiota of implant patients may be valuable for detecting, and then eliminating, microbial risk factors for implant infections.

Summary

Although implants can support natural-looking teeth, they differ from teeth in several significant ways. In healthy individ-

uals, implants can appear more robust than teeth because of the firm ankyloic-type support of osseointegration, the tight connection of gingival tissue, and a sparse microbiota. Most implants that are placed are successful and healthy, and the technology is an attractive option for replacing lost teeth.

For those implants that fail and become diseased, however, the progression of failure can be rapid. Because they are supporting functioning prostheses, ranging from a single crown to bridges or full dentures, the impact of implant failure on the patient can be considerable. As with periodontal diseases, implants can be infected with a range of different species that may require different approaches for successful treatment. As more dental implants are placed, dental and clinical practitioners will increasingly encounter patients with implant infections and will be required to treat these infections [38].

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