DENTAL IMPLANT INFECTIONS AND DECONTAMINATION

A.C. Mongra

Department of Biomedical Engineering, Adesh Institute of Engineering & Technology (Punjab Technical University) Faridkot -151203
E-mail of Corresponding Author: acmongra@rediffmail.com

Abstract
There are no controls legislated over the operating environment in dental clinics. Despite this and the contaminated oral surgical field through which they are placed, success rates are reported as being as high as 90-95 %. Despite the high success rate of implant dentistry in recent years, implant failures due to peri-implant diseases do exist. Implant surfaces have significant role in osseointegration potential of the implant. The ability of bacteria to adhere titanium implant surfaces has been confirmed in various studies. Elimination of bacteria from the implant surface is necessary in order to terminate the source of infection and disrupt the formation of biofilm. Biomaterial therapies using fibers, gels, and beads to deliver antibiotics have been used in the treatment of Peri-implantitis. Future strategies include the development of surfaces that become antibacterial in response to infection and improvements in the permucosal seal. Research is still needed to identify strategies to prevent bacterial attachment and enhance normal cell/tissue attachment to implant surfaces. There is also possibility of development of recombinant protein using r-DNA technology and using the application of tissue engineering in development of coating of biomaterials using for dental implants. In the present study general microbial status of healthy implants, infected implants, along with normal microflora present in the mouth have been covered as per the reports of different methods of isolation used, the mechanisms of attachment of microbes through biofilm formation and how to minimize the forces of adhesion to the surface of dental implant material has been covered to enable the exploring the alternative approach of biomedical engineering with respect to the understanding of characteristics of microbiota (morphology and biochemical aspects) and compatibility surface characteristics of biomaterials with respect to the osseointegration and biofilm formation.

Keywords: Dental implant infection, Osseointegration, Surface forces, Biofilm, Biomaterials, Decontamination, Microflora of teeth, Dental implant

1. Introduction
Commercially pure titanium or titanium alloy materials are the common constituents of dental implants. However, alternative materials include ceramics such as aluminum oxide and other alloys gold and nickel chrome-vanadium. Implant surfaces may be modified by plasma spraying, anodizing etching, or sandblasting to increase the surface area and Osseo integration potential of the implant. Dental implants are predominantly placed in primary care settings, commonly in general dental practice under local anesthesia. There are however, no controls legislated over the operating environment in dental clinics. Despite this and the contaminated oral surgical field through which they are placed, success rates are reported as being as high as 90-95 %. Despite the high success rate of implant dentistry in recent years, implant failures due to peri-implant diseases do exist. The ability of bacteria to adhere titanium implant surfaces has been confirmed in various studies. Elimination of bacteria from the implant surface is necessary in order to terminate the source of infection and disrupt the formation of biofilm. Biomaterial therapies using fibers, gels, and beads to deliver antibiotics have been used in the treatment of Peri-implantitis though clinical efficacy is not well documented. Guided tissue regeneration membranes e.g., collagen, poly-lactic/glycolic acid, chitosan, ePTFE loaded with antimicrobials have shown success in reosseointegrating infected implants in animal models but have not been proven in humans. Experimental approaches include the development of anti-bioadhesion coatings, coating surfaces with antimicrobial agents e.g., vancomycin, Ag, Zn or antimicrobial releasing
coatings e.g., calcium phosphate, polylactic acid, chitosan. Future strategies include the development of surfaces that become antibacterial in response to infection and improvements in the permucosal seal. and using the application of tissue engineering in development of coating of biomaterials using for dental implants. In the present study, the distribution pattern of micro biota of healthy implants, failing implants, normal diseases sites gingivitis and healthy sites gingivally was reviewed along with the bacterial adhesion processes and methods of decontamination of dental has been covered.

2. Peri-implant Diseases
Peri-implantitis is an inflammatory reaction with the loss of supporting bone in the tissues surrounding a functioning implant. Peri-implant diseases are commonly recognized, peri-implant mucositis and peri-implantitis, both describing an inflammatory response around the peri-implant tissue. Peri-implant mucositis is a term used to describe the inflammatory reaction around the peri-implant tissue without any radiographic loss of bone. On the other hand, peri-implantitis, one of the major causes of implant failure, is an inflammatory response around osseointegrated implants, resulting in loss of bone around an implant in function.

Risk factors for peri-implantitis consist of a history of periodontitis, dental plaque, poor oral hygiene, smoking, alcohol consumption and diabetes. A clinical diagnosis indicates inflammatory signs including bleeding on probing with or without suppuration and a peri-implant pocket depth $\geq 5$ mm. Several authors reported high rates of implant failures due to peri-implantitis. Esposito et al., 1997 found implant removal rates due to peri-implantitis ranged from 8-50%.

Peri-implantitis is conditioned by the status of the tissue surrounding the implant, implant design, degree of roughness, and the poor alignment of implant components, external morphology and excessive mechanical load.

3. Microbiota associated with PI
In the failing implant site, increased proportion of gram negative anaerobic rods, black pigmented Bacteroides and Fusobacterium spp., spirochetes, fusiform bacteria, and motile and curved rods were found. As for control sites successful implants in the same patient, coccoid cells were the predominant morphotype. If this predominates for significant time periods then peri-implantitis and eventual implant failure may result.

Table 1 showing the microbiota of infected & healthy implant, gingivitis sites & gingivally healthy sites.

| Table 1 Colonization pattern of microbiota of periodontal, dental implants and infected implants |
|---|---|---|---|---|
| Frequently occurring --species in. Microbiota of disease, healthy and dental implants | Infrequently occurring --species in Microbiota of periodontal and infected implants | Species colonize the implants that were failing | Dominant species characterizing symptomatic implants | Microbiota of healthy implants included health associated species |
| Gingivitis sites | Gingivally healthy sites | Dental implants | Periodontal samples | Infected implant samples |
| *Streptococcus sanguis,* *Actinomyces vincentii,* and *Actinomyces odontolyticus* as well as putative periodontal pathogens e.g., *Porphyromonas gingivalis,* *Prevotella inter-media,* *Prevotella melaninogena,* and *Fusobacterium species* | *staphylococci,* *enteric rods,* *pseudomonads,* *enterococci,* and *yeasts* | *Putative periodontal pathogens including spirochetes,* *Peptostreptococcus micros,* *Fusobacterium species,* enteric gram-negative rods, and yeasts | *gram-negative species* *Bacteroides forsythus,* *Fusobacterium nucleatum* subspecies *vincentii,* *Campylobacter gracilis,* *P. gingivalis,* *Gram-positive species* *Streptococcus inte2 rmedius* and *P. micros* |
| 3. *sanguis,* *Streptococcus orallis,* and *Streptococcus gordontii* and gingivi-tis-associated species such as *Actinomyces naeslundii* and *Capnocytophaga gingivalis.* Overall, the microbiota of the peri-implants and the periodontal infections was found similar. |
After the insertion of titanium implants, rapid colonization of bacteria has been observed at the peri-implant sulcus. Some microbiological studies have shown that implants affected by PI tend to harbor microbiota encompassing periodontal pathogen species, including Porphyromonas gingivalis, Tannerella forsythia, Aggrega-tibacter actinomycete mcomitans, Prevotella intermedia, and Fusobacterium species. Leonhardt et al. also reported that less common oral species, such as staphylococci, enteric species, and yeasts, were recovered from failing implants. The peri-implant microbiota differs depending on whether the individual is edentulous or partially edentulous. In particular, P. gingivalis was rarely isolated from edentulous individuals, which makes an interesting analogy to the paucity of P. gingivalis isolated from patients with pericoronitis and those in the early stages of periodontitis suggesting that in both cases the deeper periodontal pocket niches favored by P. gingivalis were missing.

The microbiota of healthy and diseased dental implants appears 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. 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. Implants that were failing and that had an infectious etiology were colonized by putative peri-odontal 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 or between one- and two-stage implants.

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 gingivi-tis-associated species such as Actinomyces naeslundii and Cap-nocytaphaga gingivalis. Overall, the microbiota of the peri-implants and the periodontal infections is similar.

Interestingly, micro-organisms not usually associated with periodontitis or dental abscesses such as staphylococci, coliforms and Candida spp. are commonly isolated from peri-implant lesions in some studies. Staphylococci are present within the oral cavity and their isolation from peri-implant infection is significant as both Staphylococcus aureus and coagulase-negative staphylococci are frequently recognized for infections associated with metallic biomaterials and indwelling medical infections in general. More recently, Staphylo-coccus aureus has been demonstrated to have the ability to adhere to titanium surfaces. This may be significant in the colonisation of dental implants and subsequent infections. These findings indicate the complexity of the microbiota in PI and the species responsible for PI remain unclear. It is also possible that unknown bacteria are involved in the lesions. As pockets around the remaining teeth may act as a bacterial reservoir, the composition of the peri-implant microbiota is likely to be similar to that around teeth. However, few studies have evaluated the differences in bacterial composition between dental implants and remaining teeth in the same subjects. In a recent study, molecular techniques such as oligonucleotide probes, polymerase chain reaction PCR, and checkerboard DNA hybridization have been applied to identify the bacteria in PI. However, these approaches only detect specific target bacteria and are not practical for identifying the true diversity of potential pathogens in the pockets of PI. In contrast, PCR amplification of conserved regions of the 16S ribosomal RNA. rRNA gene followed by clone library construction has been used to comprehensively identify various microbiota. This approach allows the detection of almost every species in a given sample and is able to indicate the presence of previously uncultivated and unknown bacteria. A total of 335 sequences from eight samples were subjected to sequence analysis revealed 112 species; 51 46% were uncultivated phylotypes, of which 22 were novel. The total numbers of bacterial species identified at the sites of PI, periodontitis, and periodontally healthy implants were 77, 57, and 12, respectively. This type of method of using the 16 r DNA gene clone library for detecting the Bacterial phyla and genera has merit over the traditional methods of culturing the microbes for identification as shown in Table 2. This study reflects that the bacterial population as well as number of bacteria phyla and genera are more at the site of dental implant failure, and require the necessary steps to check the
bacterial population and density at the initial stage before going to implant the teeth. PI biofilms showed a more complex microbiota when compared to periodontitis and periodontally healthy implants, and were mainly composed of gram-negative anaerobic bacteria. Previously established periodontopathic bacteria showed low prevalence and several bacteria were identified as candidate of pathogens in PI, although it is unclear whether the importance of these species is higher when compared to established periodontopathic bacteria.

Table 2 Microbiota of failing implants as per reports of various authors using different methods of detection of bacteria

<table>
<thead>
<tr>
<th>Method of detection</th>
<th>Type of implant (no. of patients/implants)</th>
<th>Most prevalent microbes detected (% sites infected with bacteria)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PCR</td>
<td>Various 10/12 [39]</td>
<td>Porphyromonas gingivalis 67%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Campylobacter rectus 42%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Eikenella corrodens 42%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Treponema denticola 42%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Prevotella intermedia 33%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Tannerella forsythia 33%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Actinobacillus actinomycetemcomitans 17%</td>
</tr>
<tr>
<td>Culture/dark field microscopy</td>
<td>Titanium hollow cylinder implants 7/not stated [41]</td>
<td>Porphyromonas gingivalis 27%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Bacteroides spp., Fusobacterium spp., spirochetes, fusiiform bacilli, motile and curved rods % not stated</td>
</tr>
<tr>
<td>Culture</td>
<td>Bra˚nemark 37/1-4 per patient [15]</td>
<td>Prevotella intermedia/P. nigrescens 60%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Actinobacillus actinomycetemcomitans 60%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Staphylococci, coliforms, Candida spp. 55%</td>
</tr>
<tr>
<td>Culture</td>
<td>IMZ 12/18 [37]</td>
<td>Bacteroides spp. 89%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Actinobacillus actinomycetemcomitans 89%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fusobacterium nucleatum 22%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Capnocytophaga spp.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>27.8% Eikenella corrodens 17%</td>
</tr>
<tr>
<td>16S rRNA gene clone library</td>
<td>Various not stated 77 species PI sites; 57 species periodontitis sites; 12 species periodontally healthy implants [34]</td>
<td>Actinobacteria Actinobaculum, Actinomyces, Atopobium, Propionibacterium, Rothia % not stated Firmicutes Catonella, Dialister, Eubacterium, Gemella, Granulicatella, Lachnospiraceae, Lactobacillus, Mogibacterium Parvimonas, Peptostreptococces, Pseudoramibacter, Selenomonas, Solobacterium, Streptococcus, Veillonella % not stated Proteobacteria Campylobacter, Cardiobacterium, Desulfobulbus, Eikenella, Hemophilus, Lautropia, Neisseria Terrahaemophilus % not stated Bacteroidetes Bacteroidetes, Capnocytophaga, Porphyromonas, Prevotella % not stated Fusobacteria Fusobacterium, Leptotrichia % not stated Tenericutes Mycoplasma % not stated Synergistetes Synergistetes % not stated TM7, Chloroflexi % not stated</td>
</tr>
<tr>
<td>Checkerboard DNA-DNA hybridization technique</td>
<td>Not stated 21/28 [36]</td>
<td>P. nigrescens, P. micros</td>
</tr>
</tbody>
</table>
4. Factors involved in peri-implantitis.
Each manufacturer has its own processing method, whose details may not be disclosed to the public. And although the principles of manufacturing implants may be the same, minor details may differ from company to company. Therefore these studies may not reflect the true characteristics. The composition of the bacterial environment as well as the ability of bacteria to adhere to the implant surface appears to be two risk factors of peri-implantitis. The ability of bacteria to adhere titanium implant surfaces has been confirmed in various studies. Bacteria ranging from Streptococcus sanguis, Actinomyces viscosus, Porphyromonas gingivalis, and Actinobacillus actinomycetemcomitans have been reported to have the abilities to adhere to titanium surfaces. Cimasoni and McBride 1987 also documented the successful adherence of Treponema denticola on modified hydroxyapatite of implants.

In comparison of natural tooth and the implant, the natural tooth is endowed with certain specific protective mechanisms such as junctional epithelium, connective tissue and cells belonging to the immune system. The epithelium and the interface between the supra-alveolar connective tissue and the titanium surface of an implant differ from the interface of the dental-gingival unit. Like the connective tissue attachment, the epithelium presents a hemidesmosomal attachment to the implant surface; the difference lies in the fact that the epithelial fibres are predominantly longitudinal to the surface of the implant and not perpendicular, as in the case of a natural tooth.

The design of the implant is an important factor in the onset and development of peri-implantitis. A specific system of implants is described according to its macroscopic morphology, its micro surface and the quality of the alignments of its different components. The implant’s superficial roughness favors bacterial plaque adherence when the surface is exposed to the oral environment, although there is no correlation between the type of surface and the selection of aggressive colonizing bacterial species. Poor alignment of the components that comprise an implant prosthesis system may foster the retention of bacterial plaque, as well as enabling microorganisms to pass inside the trans epithelial abutment.

As Binon et al described in their study, this is possible because on average, there is a difference of between 20 and 49 micra between the components of the different types of implants currently on the market. This space provides a point of entry for microorganisms of the oral flora measuring less than 10 micra. The external orphology of the titanium implant seems to be less relevant provided that it has been properly placed. However, the influence of the macroscopic design should be taken into account in terms of the pattern of stress transmission to the bone, which can lead to excessive mechanical stress at certain points, particularly at the junction between the bone and the cervical collar of the implant. Bone loss at this biomechanically weak spot increases the likelihood of bone defect formation at this level and subsequently becoming contaminated. Another reported cause of peri-implantitis is the corrosion that can occur when a non-noble metal structure is connected to a titanium implant. In these cases, increased amounts of macrophages have been observed in the tissues surrounding the implant; which would favor the initial bony reabsorption due to non-infectious causes.

5. Colonization/plaque maturation
The bacteria start growing and form biofilm after they have attached firmly onto the surface. There is negative aspect of roughened surface. Roughened surfaces produce a sheltering effect for bacterial adhesion protecting them from shearing forces, and allowing them the stability and time to easily transform their adhesion from a reversible bond to an irreversible bond onto the substrate. In addition, rough surfaces are capable of accumulating increased thickness/area...
of plaque and number of colony forming units of bacteria. As time progresses with undisturbed matured plaque, rough surfaces tend to harbor more motile organisms and spirochetes. Many studies have shown this effect of rough surfaces on bacterial adhesion in multiple surfaces including restorative material, teeth and titanium. Quirynen et al. 1990 demonstrated the effect of surface roughness by applying two strips of material glued on human teeth, one rough Ra = 2µm and one smooth Ra = 170.1µm. Only a quarter of the smooth region displayed plaque accumulation while the rough region was completely covered by biofilm after three days cessation of oral hygiene. Rimondini et al. 1997 demonstrated higher bacterial count in rough surfaces in an in vivo study involving titanium discs with various roughnesses after suspension of oral hygiene of 24 hours. In addition, more long and short rods were noted in the rougher surfaces when compared to the smooth ones. Tanner and colleagues 2005 tested four different materials with Ra values ranging from 0.05 to 0.51 µm bonded to the buccal surface of a molar. After 24 hours intra-orally they found the roughest surface with highest colony forming units of total facultative bacteria and plaque formation. Subgingivally, Waerhaug 1956 demonstrated roughening of subgingival enamel in both dogs and monkey induced more deposit of plaque and calculus, and also resulted in more inflammation.

From a microbiological standpoint, rough surfaces appear to harbor more oral bacteria. Rans et al. in 1991 reported a higher percentage of P. micros P. micra in healthy hydroxyapatite-coated fixtures 17.4% than in healthy pure titanium fixtures 9.8%. Quirynen et al. 1993 also reported rough surface abutments harbor twenty five times more bacteria subgingivally when compared to smoothed abutments. The majority of studies indicate that a rough surface in general creates a friendlier environment for microbial adhesion. Nevertheless, data on newly marketed implant surfaces are surprisingly lacking with regard to the infectious aspect of peri-implantitis.

6. Removal of bacteria on Implant Surfaces decontamination techniques

6.1 High-pressure sodium bicarbonate device: Jovanovic et al. suggested in 1993 a “decontamination protocol” which consists of exposure of titanium surfaces to a high-pressure sodium bicarbonate device for one minute under aseptic conditions. Pereira da Silva et al. in 2005 tested this method of decontamination and found no viable bacteria in all three titanium groups. Augthun’s study 1998 also reported an air abrasive system to be the most effective decontamination method and resulted in no damage to the underlying titanium surface. However, Chairay et al. 1997 demonstrated altered morphology of machined implants after administration of air powder abrasion. This difference was possibly due to differences in duration of application, as Augthun’s group only applied the air abrasive for 60 seconds. Besides altering implant surfaces, additional concerns such as possible retained particles after administration and application of compressed air intra-orally may raise some concerns toward this treatment modality.

6.2 Laser therapy: Laser therapy had been suggested for the decontamination of implant surface. However, studies show that not all types of lasers are suitable for this purpose. Subsequent damage to the implant surface has been implicated in many cases. For instance, the Nd:YAG laser was reported to cause pitting on implant surfaces in certain settings and also resulted in melting, loss of porosity and surface alteration of plasma coated implants. In addition, Block et al. reported failure of sterilization by the Nd:YAG laser after contamination by spores of Bacillus subtilis. In Duarte et al. 2009, the Er:YAG laser was not effective in removing S. sanguinis. Stübing et al. 2010 demonstrated surface alterations of different degrees based on various energy settings of Er:YAG laser on both surfaces. No visible surface alterations were seen when irradiated by CO2 and diode lasers. The Er:YAG treated SLA surfaces appeared to decrease in roughness due to melting of surface peaks, while smooth surfaces increased in roughness as a results of developing cracks after irradiation. CO2 and diode laser treated surfaces were not tested for surface roughness due to lack of visible alteration. Deppe et al. 2001 reported thermal changes as well as surface melting and alteration by using CO2 lasers in different settings. No data was reported on the efficacy of bacteria removal even though the in vivo part of the study appeared successful. The application of laser and its negative effects remains uncertain and further research will be needed in this area before any solid conclusions can be drawn.

6.3 Rotating brush with pumice and cotton soaked in saline: Persson et al. 1999 conducted
an in vivo study on beagle dogs utilizing a decontamination method of a rotating brush with pumice and cotton soaked in saline. The rotating brush was used to polish the surface until a “gray and frosty” appearance was noted and cotton dipped in saline was used to clean the surface of implant until no visible plaque was noted. The histological parameter of “re-osseointegration” did not reflect any advantage of either treatment; microbial parameters were not tested in this study.

6.4 Curette: The most direct method of physically removing bacteria, plaque or calculus from a surface in the oral cavity is the use of the curette. However the potential damaging effect of a curette to the titanium surface is of great concern. Augthun et al. 1998 found that the usage of a metal curette, diamond polishing device, and ultrasonic scalers all resulted in damage to the implant surface. Mengel et al. 1998 advised the use of a plastic curette, prophyl tips and air abrasive systems because the metal curettes, sonic and ultrasonic devices with universal tips cause pronounced traces or instrumentation and remove substantial substance from titanium. Due to the potential negative effect of the above mentioned implant decontamination methods, more conservative methods have been explored. In addition, no direct proof of complete removal of bacteria was provided. It is unclear whether the systemic antibiotics, degranulation during flap surgery or the actual surface cleaning of saline decontaminated and promoted the implant site healing.

Schou et al. 2003 tested four methods of decontamination: 1 air-powder abrasive, 2 air-powder abrasive follow by citric acid, 3 gauze soaked in saline following citric acid and 4 gauze soaked in 0.1% chlorhexidine and saline alternatively. Following decontamination of implant surfaces, peri-implantitis defects were treated with autogenous particulate bone fill and covered by e-PTFE membrane. Positive results were obtained and the authors recommended the decontamination of soaked gauze with chlorhexidine and saline. Once again the bacterial aspect of results was not discussed and many variables were included in the study. Rinsing of saline and chlorhexidine was described by Hammerle et al. 1995 Sites of peri-implantitis were treated with flap debridement along with rinsing of sterile and 0.2% chlorhexidine digluconate.

Dennision et al. 1994 conducted an experiment on the removal of bacterial endotoxin by means of burnishing implants with cotton pellet prepared with water, citric acid, or 0.12% CHX and air-powder abrasive. Three types of implants were used: machined, plasma sprayed and hydroxyapatite coated implants. An air abrasion system appeared to be effective in decontamination of P.gingivalis endotoxin in all implant types. Both air abrasion and citric acid were effective in decontamination of hydroxyapatite coated implants. For machined surface implants, all treatments including cotton pellet soaked in water can be effective in removal of endotoxin. One can speculate from this study that the application of citric acid or CHX did not provide additional benefit. In addition, the use of acid in decontamination of titanium surface could be alarming because acids are used for surface modification of titanium. Water rinsing is a non-invasive and inexpensive way to remove biofilm on teeth. Studies on the efficacy of water rinsing on different titanium surfaces have not been conducted. If proven effective, rinsing titanium implants with sterile saline water with an easily accessible syringe device could be a simple and inexpensive way of treatment if proven to be effective.

7. Treating peri-implants:
At present, there is no reliable evidence for the most successful method of treating peri-implantitis. Despite a variety of therapeutic options, infected implants are difficult to treat and usually require removal. Some clinicians advise systemic antibiotics for the treatment of failing implants and a variety of drug regimens are described. Oral agents such as doxycycline, clindamycin, co-amoxiclav, penicillin V, amoxicil-lin and a combination of amoxicillin and metroni-dazole have been recommended. Nevertheless, no double-blind, randomised, placebo-controlled trial has been undertaken. Microbiological study of periimplantitis conducted by the Barcelona School of Dentistry that determined that the antibiotic therapy proven to be most efficacious in the antibiogram was the association of amoxycillin and clavulanic acid.

Conclusion
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. Data on failure and complications of dental implants should be collected and reported in a systematic fashion. This would enable a more detailed analysis of the microbiology, treatment outcomes and assist in the formulation of biomedical engineered materials of dental implants to minimize the risk factor of infections or to completely avoid the implant infections.

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