**INTRODUCTION**

The colonization of the oral cavity by microbiota starts immediately after birth and around 500 different species are found colonizing the oral cavity. Usually an individual can harbor around 150 different species in the oral cavity. Oral cavity is said to have wide array of species but not all cause disease. Normally there exists a balance between the host and the microbiota. The disease occurs when this harmony is disrupted. For a bacterium to flourish in dental biofilm they should be able to adhere to the tooth surface and should have potential to survive in anaerobic environment like gingival sulcus and tooth crevice. These are usually referred to as plaque. It is defined “as structured, resilient, yellow greyish substance that adheres tenaciously to the intraoral hard surfaces, including removable and fixed restorations.”

This microbial collaboration increases their pathogenicity besides they also withstand the host defences and antimicrobial agents.

Dental implant in recent years are becoming a trendy material for tooth replacement because of their durability and biocompatibility. Exposure of the dental implants in oral cavity to wide array of species is the major cause for development of biofilm on dental implants. This can lead to early and/or late implant failure. This article will review in detail the formation, composition, structure and treatment of biofilm on dental implant.

**HISTORICAL PERSPECTIVE**

The dental plaque biofilm was introduced someday around 17th century by Anton Von Leeuwenhoek, from tooth scrapings where he noticed a substantial number of microorganisms. The term “biofilm” was coined by Bill Costerton in 1978. Donlan and Costerton in 2002 explained biofilm as “a microbially derived sessile community characterized by cells that are irreversibly attached to a substratum or interface or to each other, embedded in a matrix of extracellular polymeric substances that they have produced, and exhibit an altered phenotype with respect to growth rate and gene transcription.”

**NATURAL TEETH AND DENTAL IMPLANT:**

<table>
<thead>
<tr>
<th>Natural Teeth</th>
<th>Dental Implants</th>
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<tbody>
<tr>
<td>The gingival margin follows the outline of the cementoenamel junction.</td>
<td>The mucosal margin follows the contour of crestal bone or relates to the connective tissue adhesion to the adjacent teeth</td>
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<tr>
<td>Decreased interproximal probing depths.</td>
<td>Increased interproximal probing depths.</td>
</tr>
<tr>
<td>Interproximal papillae larger</td>
<td>Interproximal papillae shorter</td>
</tr>
<tr>
<td>Mobile within socket</td>
<td>Immobile within socket</td>
</tr>
</tbody>
</table>
ASSOCIATION BETWEEN TITANIUM AND DENTAL BIOFILM

Titanium dioxide layer, which protects it against corrosion, always coats titanium implant surfaces but at times, this layer might be disrupted leading to titanium corrosion. This titanium corrosion is shown to produce late bone loss. Fukushima et al. highlighted the role of Streptococcus mutans in increasing the titanium corrosion. Besides, a study by Pozhitkov et al. showed the tendency of titanium corrosion products in altering the subgingival microbiome. Hence, it is always mandatory to use titanium oxide coated implant.

BIOFILM FORMATION ON DENTAL IMPLANTS

Biofilm on dental implants practically corresponds to that on teeth, with the same type of microorganism in both health and disease. The formation of biofilm on dental implants is a multidisciplinary process, which begins with the formation of salivary pellicle through a dynamic process via the selective adsorption of the surrounding macromolecule. Then comes the initial attachment of bacteria, when the negatively charged bacterial cell wall and positively charged salivary pellicle comes in contact due to various indiscriminate movements. Following this secondary attachment occurs, where the bacterial biofilm thickness increase in size either due to accumulation of early colonizers or due to attachment of late colonizers. During this stage cell-cell interconnection known as coaggregation. Coaggregation initially was identified by Gibbons and Nygaard in 1970. They stated it as a biological relationship among genetically distinct species and it is also shown to ease the functional organization of biofilm. Finally comes the maturation, where the plaque attains maximum stability.

Studies proclaim that bacterial colonization on dental implants and teeth are similar except that periimplant sulci exhibit a less diverse microbial community. Healthy implant sites were found to have Streptococcus, Capnocytophaga, Veillonellaparvula, Peptostreptococcus micros, and Fusobacteriumnucleatum and also few periodontal species including Porphyromonasgingivalis, Bacteroidesforsythus, Prevotella intermedia, Prevotellamigrella, and Treponema denticola with dark stains on their surface. The microbiota of crowned and uncrowned implants is found to be similar other than the presence of elevating amount of Streptococcus oralis, Prevotella intermedia and Selemonas noxia. Assenza et al. hypothesized the cement retained and conical implant abutment connection are far superior than the screw retained implant abutment connections in withstanding the bacterial penetration.

MICROBIOTA IN FULLY EDENTULOUS PATIENT

Emrani et al., stated that aggressive periodontitis patient who underwent complete extraction followed by prosthetic rehabilitation with dental implants had reestablishment of disease causing microbes within 6-7 months and then developed periimplatitis thereafter. Whereas the study done by Nakou et al. states that microorganism growth was dependent on the periimplant niche rather than the past history of microorganism.

STRUCTURE OF DENTAL IMPLANT BIOFILM

SEM analysis done after 12 hrs of implant placement (early colonizers) are shown to exhibit large amount of spindle shaped rods and short streptococcal strains suggesting Fusobacteriumnucleatum and Streptococcus oralis, respectively. Whereas the mature biofilm exhibits increased thickness of bacterial layer. CSLM revealed increased amount of bacteria on crests and sides of implants whereas the implant thread embrasures are free of microbial community. Early biofilm images taken after 48-96 hours of incubation depicted blending of solitary microorganisms whereas the images of mature biofilm that where taken after 120 hours showed large amount of dead bacteria on its surface.

IDEAL SURFACE CHARACTERISTICS OF DENTAL IMPLANT

Surface modifications are shown to impede the bacterial biofilm development on dental implant surface. Other factors that influences the bacterial colonisation includes surface roughness, surface free energy, surface area and hydrophobicity. Ideal surface roughness of dental implants should be 0.2µm, beyond this there is an increase in bacterial colonisation. Increased surface free energy and hydrophobicity are also shown to enhance biofilm formation.
DIAGNOSIS OF INITIAL BACTERIAL COLONISATION

With advancing diagnosing aids, there still remains a hindrance in efficient detection of biofilm species. Microbiological methods for detection of sample and biosensors at present seems to be the promising tool. Besides Surface Enhanced Raman spectroscopy are also shown to detect Salmonella, Listeria, E.coli, S.epidermidis, and Bacillus with high sensitivity using theranostic nanoparticles.

CONTEMPORARY THERAPEUTIC STRATEGY FOR DENTAL IMPLANT BIOFILM

Surface coatings of the dental implants is the emerging method of biofilm prevention. Most commonly used and widely accepted coatings are Nanohyperxypatite coating (NonoTite BIOMET3i). Other coatings available includes anodized nanotubular titanium alone or in combination with gold nanoparticles, Zinc oxide nanoparticles, plasma fibronectin, Pac-525, and antimicrobial peptide based coatings. Besides many herbocoeutrical based coatings like umbelliferon derivative, totarol and Brazilian red propolis are emerging and will make development of eco-friendly coatings in mere future.

Various options like bioacoustics therapy, antimicrobial photodynamic therapy, electromagnetic methods and ozone therapy are used to treat dental implant biofilm.

DISCUSSION

Biofilm is a well-organized cooperative community of microorganisms and is defined as a matrix enclosed bacterial population adherent to each other and/or to surfaces or interfaces. Biofilm associated implant failure is one of the most common cause. Exposure of the osseo-integrated implant in the oral cavity leads to the formation of acquired pellicle through selective adsorption of environmental macromolecules within 30 minutes of exposure. This acts as a substrate for bacterial colonization. The difference in biofilm formation on dental implants is mainly due to their surface characteristics, which is different from natural teeth. The failing implant is characterized by a greater proportion of red (P.gingivalis, Treponema denticola, and T. forsythia) and orange (Pintermedia and Fusobacterium nucleatum) complex, as well as Aggregatibacter actinomycetemcomitans and Eikenellacorrodens.

Few factors are shown to enhance the biofilm formation on dental implants, for instance, previous history of periodontitis, increased surface roughness, surface free energy of dental implant and even the presence of titanium in the oral cavity itself can cause enhanced biofilm formation on implant surface. A systematic review by Heitz-Mayfield LJ et al. demonstrated that the history of periodontitis represented an increased risk for implant failure, with a odds ratio ranging between 3.1 and 4.7. Increased surface roughness has been associated with increased level of osseointegration, but a higher surface roughness (Ra) value > 0.2 µ is seen to be causing increased biofilm formation and thus, contributes to spontaneous progression of peri-implantitis lesions. Presence of titanium in the oral cavity is also known to cause peri-implantitis. Dental implants have a layer of titanium dioxide coating that is responsible for the biocompatibility of titanium implants; when this coating is disrupted, it leads to titanium corrosion. A recent case control study by Safioti et al. with peri-implantitis and healthy controls found 8-fold increase in titanium corrosion products in the plaque around implants with peri-implantitis compared with healthy ones. Daubert et al. assessed the potential effects of titanium products on the periimplant microbiome using 16s rRNA analysis and the results demonstrated titanium dissolution products to act as a modifier of perimplant microbiome structure. These data support the utilization of techniques that disrupt the titanium oxide surface to prevent biofilm formation.

FUTURE OF BIOFILM

Developed of therapeutic strategy targeting the bacterial communication system may decrease the development of antibacterial resistance. Devising a non-invasive way of diagnosing the genetic cause of biofilm formation may pave way for development of efficient treatment strategies for dental implant biofilm. Innovations of implant materials along with advanced diagnosing approaches and treatment may prevent dental implant biofilm related infections.

CONCLUSION

Discovery of dental biofilm is a notable event in dentistry. Dental implant related biofilm is different from natural teeth in that the periimplant area have less diverse bacterial species compared to periodontal biofilm. Titanium corrosive particles alone or by modulating the host response may also influence the microbial growth pattern. Taking these into considerations it is essential to develop a concise treatment planning, since the traditional mechanical and chemical therapy done in periodontal pockets must be carefully carried out before initiating treatment. In spite of increasing knowledge on dental biofilm, diagnosis and treatment of biofilm continues to be a difficult therapeutic target.
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