

# Biofilm On Dental Implants-A Review

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**Abstract:** Mouth provides a congenial environment for the growth of the microorganisms as compared to any other part of the human body by exhibiting an ideal nonshedding surface. Dental plaque happens to be a diverse community of the microorganisms found on the tooth surface. Periodontal disease and the peri-implant disease are specific infections that are originating from these resident microbial species when the balance between the host and the microbial pathogenicity gets disrupted. As more implants are nowadays being placed, clinicians may encounter more complications. Therefore, understanding the etiology is warranted to establish adequate diagnosis and provide proper treatment. This review discusses the biofilms in relation to the peri-implant region, factors affecting its presence, and the associated treatment to manage this complex microbial colony.

**Keywords:** implant, biofilm, microorganisms, peri-implant diseases, surface roughness

## 1. Introduction

Biofilm is a microbial-derived sessile community characterized by cells that are irreversibly attached to a substratum or interface to each other, embedded in a matrix of extracellular polymeric substances produced by microbes [1]. Biofilms formed on the tooth surfaces are known as dental plaque. Biofilms in the oral cavity consist of complex microbial communities found in a matrix of polymers, primarily of bacterial and salivary origin [2]. Bacteria from the dental plaque are the major etiologic causes for caries, gingivitis, periodontitis, peri-implantitis, and stomatitis. Well-developed biofilms on dental implant surfaces and prosthetic restorations become the main source of microbes causing peri-implantitis [3].

Dental implants are made from titanium because of its excellent surface properties and biocompatibility. During the transmucosal healing stage of titanium dental implants, the adsorption of salivary pellicle, bacterial accumulation and biofilm formation produce an inflammatory process [4]. The composition of the biofilm and the speed of formation of biofilm and the surface energy, roughness, chemical characteristics of the implant, the abutment materials, and the prosthetic components, influence the formation of biofilms. Therefore, the purpose of this article is to review the formation of biofilm on teeth and implant surfaces, including the pathogenesis, the determining implant-related factors and the treatment.

## 2. Biofilm and Tooth

The formation of the microbial complex in the oral cavity is a multistage journey [5]. Bacteria obtains its nutrients from saliva. The thin film covering the tooth called as pellicle and is derived from the salivary proteins and covers the enamel. Proteins and the glycoproteins promote the adhesion and coaggregation of the oral bacteria. The bacterial adherence to the pellicle is achieved by the special surface molecules (adhesins) chiefly lectins present on the bacterial cell surface. Further multilayered bacterial colonies are formed from intercellular bacterial adhesion and secretion of the extracellular polysaccharides, e.g., levans, dextrans. This is

followed by growth-dependent accumulation by cell-to-cell adhesion to form multilayered cell clusters in the polymer matrix. The first step is reversible adhesion which is mediated by electrostatic and hydrophobic forces.

The second step is irreversible adhesion which is caused by a time-dependent shift to a higher binding affinity state [6]. Division of the attached bacterial cells produces microcolonies. Confluent growth results in the formation of plaque biofilm, which increases in complexity with time.

The microbial load in the saliva is about  $10^8$  [7] bacteria per milliliter [8]. The bacterial cells colonize the tooth surface within 4 hours of the pellicle formation. The initial colonizers are the *Streptococci* (*S. viridens*, *S. mitis*, *S. oralis*). Secondary colonizers predominantly comprising of the *Actinomyces* species, *S. mutans*, *S. sobrinus* bind to the bacteria. The bacteria multiply and co-aggregate with the other species. *Fusobacterium nucleatum* has the ability to aggregate with several bacteria and they form an important link in the dental biofilms bridging the early and the late colonizers [9]. The oral bacteria receive their nutrient supply from saliva, gingival crevicular fluid, sugar rich food metabolic products of other bacteria and food debris. Metabolic products and evulsed cell wall components (lipopolysaccharides, vesicles) activate the host response. Specialized cell to cell communication is shown by the bacteria that coordinate the gene expression and is passed on as signals. Bacteria sense the changes in the local environment and receive the information of the adjacent population. Communication within the biofilms is mediated through the metabolic exchange, genetic exchange, and the quorum sensing [10]. Quorum sensing is genetically governed chemical communication among bacteria which influences several functions of the bacteria, e.g., virulence, acid tolerance, and the biofilm formation. Two specific signaling molecules called as Competence Stimulating Peptides (CST) and AI-2 (autoinducer-2) are produced by the oral bacteria [11]. The biofilm acts as a barrier against host immunity and the antimicrobial agents. The anaerobic microflora occupies the subgingival environment gradually as the plaque starts maturing. Supragingival plaque leads to gingivitis and the subgingival microbial colonies progress the gingivitis to an established form of periodontitis.

Volume 6 Issue 5, May 2017

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### 3. Biofilm and Implant

Evidence of the first human biofilm-related peri-implant infection comes from the study on plaque samples collected from apical most part of 17 diseased implants [12]. Biofilm formation on dental implants and the teeth follow the similar pattern of microbial colonization [13]. The clean tooth surfaces are likely to have remnants of unattached microbiota that can multiply and provide a favorable surface for the attachment of the late colonizers [14]. Implant surfaces lack the desired indigenous microbiota and demand the early colonizers to set the stage for the complex communities to develop [15]. The pellicle starts forming on the implant surface in about 30 minutes after the implant is exposed in the oral cavity [16]. The acquired pellicle on the dental implants due to their lower albumin absorption capacity causes low plaque formation around implants. The gram-positive cocci, rods, and actinomyces species are the early colonizers [17]. The periodontal pathogens colonizing are the causative microorganisms responsible for peri-implantitis and periodontitis [18]. The attachment of the microorganisms to the hard surfaces, depend on their interactions with the surface components and certain specific characteristics of the interacting surfaces in terms of their wettability/hydrophobicity and surface free energy (SFE).

### 4. Biofilm at the Implant – Abutment Interface

Dental implant consists of an implant-abutment junction (IAJ). The joint/gap between the implant and abutment is called “microgap”. Two important microbiologic entities in the implant crestal region was identified by Ericsson et al.: (a) Plaque-associated inflammatory cell infiltrate and (b) implant-associated inflammatory cell infiltrate [19]. The microgap has been reported to be as high as 40-60  $\mu\text{m}$  [20]. It allows micromovement during function [21] and permits microleakage of fluids congenial for bacterial growth. When the implants are in contact with plasma or saliva, the proteins can direct the attraction or repulsion of bacteria present on external layers. The salivary protein that gets adsorbed to titanium in vivo and in vitro is albumin [22,23] and albumin adsorption to titanium occurs through calcium ( $\text{Ca}^{+2}$ ) bridges [24]. The negative charge from titanium dioxide will attract the positive ions,  $\text{Ca}^{+2}$  and thus increases the adhesion of some bacteria species.

### 5. Surface Characteristics of Implants

Osseointegration of dental implants is related with increased surface roughness of the dental implant [25,26]. Conversely, a higher surface roughness with a Ra value  $>0.2\mu$  increases biofilm formation [27,28] and thus contributes to spontaneous progression of periimplantitis lesions [29,30]. A study performed to study the attachment of oral bacteria on titanium disks with different surface morphologies (smooth, grooved, or rough) [31] concluded that most bacterial attachment was observed on the rough titanium surfaces, whereas smooth surfaces showed poor attachment. Another in vitro study to evaluate the effects of modified titanium surfaces [32] exhibited that rough or hydrophobic surfaces showed higher degrees of bacterial colonization. Another study examining the bacterial colonization on titanium

implant surfaces modified with titanium nitride (TiN) or zirconium nitride (ZrN) [33] showed that hard coatings such as TiN or ZrN on dental implants can reduce the number of initially adhering bacteria, thereby minimizing plaque biofilm formation and subsequent inflammation of the peri-implant tissues. An *in vivo* study done on the titanium discs to evaluate the effect of the surface roughness and the microbial colonization concluded that a titanium surface with a roughness inhibits the colonization and maturation of the plaque [34]. SFE is defined as the interaction between the forces of adhesion and the forces of cohesion that determine the property of wetting [35]. An *in vivo* study was undertaken on the supra and subgingival microbial plaque samples in patients with two-stage abutments, titanium versus Fluoropolyethylene propylene coated abutment. The results revealed that SFE of the implant and the abutment material have a vital role in the colonization of the bacteria [36].

### 6. Design Features of Implant and Abutment Materials

Several design features of currently used implants present plaque-retentive areas that can harbor bacteria, which in turn facilitates the formation of plaque biofilm. Earlier a study [37] performed on retrieved failed implants to identify design characteristics showed that plaque biofilm formation and accumulation occurred along the implant–transmucosal abutment interfaces, transmucosal abutment–prosthesis interfaces, implant–prosthesis interfaces, and on the surfaces of the abutment, the implant, and the prosthesis. Microscopic gaps between the various components, a high degree of surface roughness of restorations and abutments, exposure of plasma-sprayed coatings and threaded surfaces of implants, and overcontouring of implant restorations contributed to plaque accumulation and provided an environment that facilitated bacterial colonization. Thus, the design features of implants and abutment materials can also contribute to biofilm formation.

### 7. Smoothness of Abutment Material

In addition to the design features of the abutment components, their surface smoothness is a crucial determinant of biofilm formation at the implant abutment junction. To evaluate the effect of the smoothness of abutment materials, an in vivo study [38] was done in which two titanium abutments (transmucosal part of the implant) were replaced by either an unused standard abutment or a roughened titanium abutment. The authors found that supragingivally, the rough abutments harbored significantly fewer coccoid microorganisms and subgingivally they harbored nearly 25 times more bacteria. These results reinforce the finding that rough surfaces of abutments facilitate bacterial colonization and plaque biofilm formation.

### 8. Prevention of Biofilm Formation

Management of the biofilms has a multilevel approach: (1) to prevent the microleakage at the IAJ to limit/eliminate the biofilm ingress; (2) treatment of the biofilm-related

infections. Implant biofilm can lead to infection at two levels: the mucosal level (peri-implant mucositis) that causes an inflammatory lesion residing in the mucosa and bone level (peri-implantitis) which is explained as inflammatory lesion affecting the supporting tissues [39]. Treatment of dental implant associated infections consists of an anti-infective protocol that can be achieved through mechanical debridement of the implant surface or treatment using local and systemic antibiotics. The selected treatment modality depends on the established diagnosis of peri-implant mucositis or periimplantitis. The successful outcome of the treatment is assessed using measures such as reduction of inflammation, probing depth, and pathogenic bacteria [40].

Decontamination of the implant surface is quite challenging. Nonsurgical mechanical therapy has been effective in reducing the microbial load with enhanced results when combined with the antimicrobial rinse in the peri-implant mucositis lesions [41]. Various systemic local drugs such as minocycline, tetracyclines, have shown successful results by decreasing the levels of the *P. gingivalis*, *T. forsythia*, *A. actinomycetemcomitans* [42]. In the past decades, laser therapy such as diode, CO<sub>2</sub>, and Er:YAG laser has gained popularity based on the rationale of surface decontamination, hemostatic properties, calculus removal, and bactericidal effects [43,44]. Photodynamic therapy, using low level lasers, has been used to decontaminate the infected implant surfaces. Photodynamic therapy and the regenerative periodontal treatment (autogenous bone graft) can help in regeneration of the peri-implant bone defects [45].

Previous results have shown that nanoscale coatings of ZnO on titanium implants may play an important role in decreasing the formation of biofilm and thereby the subsequent peri-implant pathology. Vancomycin-modified titanium surfaces are effective against in vitro bacterial colonization on implant surfaces [46]. Different implant materials, implant designs with different surface characteristics are currently available. Transgingival implants and abutments with an Ra below 0.088 µm decrease biofilm formation and maturation [47]. It can be understood that smoother implant and abutment surfaces inhibit biofilm formation. IAJ is a vulnerable area for biofilm-related infections. Innovative implant abutment designs have helped reducing the microleakage at the IAJ with the sequential decrease in the microbial growth at the microgap [48]. Platform switch, use of tapered implants decreases or eliminates this probable microbial ingress. Any micro-structured part that is exposed to the oral cavity should be highly polished to generate an anti-plaque adhering surface. The oral antimicrobial rinse (e.g., chlorhexidine) can be advised as a daily regime for implant patients. Ultrasonic and hand scaling should be used to avoid the risk of surface scratches on the abutment as caused with metal instruments.

## 9. Conclusion

An implant's surface characteristics such as roughness, surface free energy, and chemistry have a significant influence on the pathogenicity of the peri-implant

microbiota. In addition, the design features of dental implants and the composition of the biomaterials used to fabricate implants and abutment components play vital roles in bacterial colonization and biofilm formation. These factors influence biofilm formation at the implant-abutment junction and at the implant-soft tissue interface. Surface modifications of titanium implants have proved to be effective against early bacterial colonization.

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