REVIEW ARTICLE

Influence of Implant Surface Characteristics on Microbiota: A Review

Gowtham K[1], Sakthi Devi[2], Vineela K Reddy[3], Saravana Kumar R[4], Pratebha Balu[5]

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Abstract

Dental implants have a broad array of applications, from single tooth replacement to complete oral rehabilitation with a prosthesis. Microorganisms colonize the implant's surface when it is exposed to the oral cavity. A successful implant treatment, however, is dependent on the absence of inflammation in the peri-implant tissues. The relationship of implant surface characteristics to the microbiota plays a major role in the success of an implant. The initial adherence of bacteria to intraoral surfaces is determined by the surface's physiochemical properties, such as the surface free energy (SFE) of the bacteria, the solid phase, and the surrounding liquid, as well as the surface integrity and composition of the solid medium. The surface characteristics at the micro or nanometer level, hydrophilicity, and biochemical bonding are responsible for implant success. The surface can be modified by either addition or subtraction procedures like acid etching, sandblasting, plasma spraying, hydroxyapatite coating, etc. By modifying the characteristics of the surface, biocompatibility can be improved, faster osseointegration can be provoked. The roughed surface has good osseointegration due to clot stability whereas, on the contrary, they are more prone to biofilm formation. Biofilm has been associated with almost all periodontal diseases, therefore knowledge of microbes around the implant is necessary for diagnosis and treatment. This review focuses on the influence of implant surface characteristics such as surface roughness, surface free energy, and surface composition on oral microbiota.

Keywords: Implant, Microbiota, Peri-implantitis, Surface characteristics.

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Introduction

Dental implants have a wide array of applications, from single tooth replacement to complete oral rehabilitation with a prosthesis. The primary goal of implant placement is to maintain stable osseointegration.[1] The stable anchorage of an implant attained through direct bone-to-implant contact is referred to as osseointegration. Factors responsible for osseointegration include biocompatibility, implant surface, surgical technique, and undisturbed healing phase. Implant surface characteristics (microtopography) have long been recognized as having an essential role in molecular interactions, cellular response, and osseointegration by influencing the ability to heal.[2] Soft tissue health is easily maintained when the portion of the implant emerging from the bone has a smooth surface, while a rough surface increases the bone-implant contact (BIC) percent during the initial process of healing. The bone-implant interface can be controlled by the selection and modification of the biomaterial.[3]

Biofilms on the surface of dental implants are the primary source of pathogens for peri-implantitis. In fact, biofilms have been associated with almost 65% of infectious diseases. The relationship of implant surface characteristics to the microbiota plays a major role in the success of an implant. According to the consensus report of the sixth European workshop on periodontology, implant surface characteristics are one of the risk factors for peri-implantitis.[1] The three major factors known to determine initial bacterial adherence on implant surfaces are surface material composition, surface roughness, and surface free energy.[4] It can happen when the coronal portion of the implant, which was primarily designed to aid osseointegration, becomes exposed to the oral environment due to peri-implantitis.[5]

[1,2,4,5]Department of Periodontology, Indira Gandhi Institute of Dental Sciences, Puducherry, India

[3]Department of Periodontology and Oral Implantology, Indira Gandhi Institute of Dental Sciences, Puducherry, India

Corresponding Author: Gowtham K, Department of Periodontology, Indira Gandhi Institute of Dental Sciences, Puducherry, India, Phone: +91 7358662884, e-mail: drgowtham167@gmail.com

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Surface Characteristics and Microbiota

Biofilm-associated infections are the major cause of implant failure; additionally, bacterial infections are characterized by bacterial colonization and biofilm formation on dental implants; therefore, it is essential to develop implant surfaces that reduce bacterial adherence and biofilm formation. Biofilms that form on implant surfaces cause inflammation and infection.[6] Reducing initial bacterial adhesion to the surface may aid in restricting their formation. The three most important factors determining bacterial attachment to the implant surface are considered to be surface roughness, SFE, and surface material composition.[7] It was investigated that the microbial adherence and colonization of a polyspecies biofilm on seven different titanium surfaces and concluded that surface roughness had a significant influence on biofilm development, wettability did not appear to influence biofilm formation, and the modified sandblasting acid-etching (SLA) surface exhibited the greatest trend for bacterial colonization[8] (Tables 1 and 2). According to Teughels et al., the surface roughness and chemical composition of the implant surface has a significant effect on plaque formation. Rougher surfaces and surfaces with a high surface free energy, such as titanium, have been shown to accumulate and retain more plaque.[9] Furthermore, initial bacterial adherence occurs primarily in areas with high wettability (a property of titanium) and where bacteria are guarded against shear forces (for example, in grooves and pits).[10]

Albouy et al. concluded in 2009 that reducing the roughness of intraoral hard surfaces below the threshold level of 2 µm will result in a delay in supra and subgingival plaque maturation.[11] Too smooth surface, on the contrary, may interfere with the stability of soft tissue attachment. At a surface roughness of 0.2 m, a good balance between both aspects (bacterial adhesion and soft tissue sealing) appears to be accomplished. In 2013, Al-Ahamad et al. observed that the influence of surface roughness on biofilm formation diminishes as the biofilm matures.[12] Wennerberg et al. in 2003 investigated the early inflammatory response to mucosa-penetrating abutments with varying surface topography. Clinical and histological evaluations failed to show a link between surface roughness and peri-implant mucositis at the end of the 4-week test period. In 2004, a 3-year follow-up report of a comparative study of ITIs (Waldenburg, Switzerland) dental implants (titanium plasma-sprayed surface) and Branemarks (Nobel Biocare AB, Gothenborg, Sweden) system implants (turned surface) in the treatment of the partially edentulous maxilla was published. The authors compared the outcomes of fixed partial prostheses assisted by these implants in terms of survival rates, changes in marginal bone level, aesthetic results, and peri-implantitis frequency. There were statistically significant differences in the implant systems, with the rough

Table 1: Summary of the reviewed articles evaluating the influence of surface free energy on biofilm formation

Authors	*Experimental condition*	*Sample morphology*	*Sample material*	*Range of surface roughness (R_a)*	*Influence of surface free energy for biofilm formation*
Sardin et al., 2004	*In vitro*	Ø 11 mm disk	Casting alloys, Ceramic, Titanium	0.33–0.13 µm	Not significant
Al Radha et al., 2012	*In vitro*	Ø 5 mm Disc Ø 6 mm disk	Titanium, Zirconia	0.043–0.15 µm	Major
Pereni et al., 2006	*In vitro*	30 × 30 × 1 mm square	Stainless steel, Silicone	0.08–0.25 µm	Major
Almaguer Flores et al., 2012	*In vitro*	Ø 15 × 1 mm disc	Titanium	Pretreatment titanium—<0.2 µm Acid etched—<0.8 µm SLA or hydrophilic SLA—3.2 µm	Positive correlation
Salihoglu et al., 2011	*In vitro* (human)	Implant abutment	Titanium, Zirconia	N/A	Not significant
Quirynen et al., 1994	*In vitro* (human)	Implant abutment	Titanium	0.81–0.82 µm	Major (supragingival) Not significant (sub-gingival)
Weerkamp et al., 1988	*In vitro*	4 × 4 mm square	Human teeth	N/A	Moderate

Table 2: Summary of the reviewed articles evaluating the influence of surface composition on biofilm formation

Authors	*Experimental condition*	*Sample morphology*	*Compared sample material*	*Range of surface roughness (R_a)*	*Influence of surface free energy for biofilm formation*
Lima et al., 2008	*In vitro*	Ø 10 × 2 mm disc	Titanium, Zirconia	N/A	Not significant
Lee et al., 2011	*In vitro*	Ø 12 mm disc	Resin, Titanium, Zirconia	0.059–0.179 µm	More attachment on resin. Similar between titanium and zirconia
Scarano et al., 2004	*In vitro* (human)	Disc	Titanium, Zirconia	0.73–0.76 µm	Less attachment on zirconia
Rasperini et al., 1998	*In vitro* (human)	4 × 3 × 1 mm rectangular form	Titanium, Novel ceramic	0.6–0.7 µm	Not significant
Van Brakel et al., 2011	*In vitro* (human)	Implant abutment	Titanium, Zirconia	0.21–0.236 µm	Not significant
Bremer et al., 1994	*In vitro* (human)	3 × 3 × 1.5 mm square	Glass ceramic, Lithium disilicate glass ceramic, Zirconia, HIP zirconia, HIP zirconia with 25% aluminia	0.04 µm	Lesser attachment on zirconia

surface implants having more peri-implantitis. Peri-implantitis was observed in seven ITIs implants, one of which failed completely after 12 months and another after 3 years.[13] In 2018, Bevilacqua et al. observed that biofilm formed *in vitro* is more influenced by surface topography than biofilm formed by complex biofilm communities in the mouth, where the cooperation of a range of bacterial species, as well as the presence of a wide range of nutrients and conditions, allows bacteria to optimize substrate colonization.[14] It was observed in 1993 that roughened abutments (Ra-0.81 m) contained 20 times more bacteria than smooth abutments (Ra-0.35 m) subgingivally, with a higher ratio of spirochetes and motile organisms. When the Ra value was reduced below 0.2 μm, no further changes in the amount of adhering bacteria were detected.[4] In 2011, Albouy et al. investigated the effect of surgical treatment of peri-implantitis without systemic antibiotics on various types of implants [turned (Biomet 3i), TiOblast (Astra Tech AB), SLA (Straumann AG), and TiUnite] (Nobel Biocare AB). The results show that radiographic bone gain occurred at implants with turned, TiOblast, and SLA surfaces, whereas additional bone loss was found after treatment at TiUnite implants, and he concluded that implant surface characteristics influence treatment outcome.[15]

Conclusion

Surface roughness is typically considered to be more essential in biofilm formation than surface free energy. When compared to implants with smooth surfaces in a similar biofilm structure, implants with rough surfaces accumulate more bacterial biomass and a significantly greater number of pathogenic bacteria (*Fusobacterium nucleatum* and *Aggregatibacter actinomycetemcomitans*). Therefore, the rougher the surface, the more plaque accumulation. As a result, implant surfaces that inhibit attachment of early colonizers are needed to destabilize both late colonization and infectious biofilm formation.

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