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SURFACE CHARACTERSTICS AND PERIIMPLANTMUCOSITIS: A REVIEW

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ABSTRACT

Developing different modalities for the replacement of lost natural teeth has been an elusive goal for centuries. Dental implants have increasingly emerged as an important treatment option for the restoration of both partially and complete edentulous persons. However, exposure of implants in the oral cavity presents a unique surface that can interact with native host bacteria leading to plaque formation and consequently peri-implant diseases. Different implant material promote selective adherence during early plaque formation. This article discusses the influence of different implant materials and surface characteristics of implant influencing the accumulation of plaque and periimplant diseases.

Key words: Periimplantitis, surface characterstics, microbiology.

INTRODUCTION

Developing artificial replacement for missing teeth has an elusive goal for more than 1500 years. [1]Studies of oral microflora of infants have shown that the hard surface of and gingival crevices of erupting teeth provide new habitats for previously undetectable microorganisms. A similar effect upon the microflora can be expected from the insertion of implants in edentulous areas.[2] Dental implants represent an increasingly important treatment modality for both partially edentulous patients and complete edentulous patients. Among the periimplant diseases, periimplant mucositis is a reversible inflammatory reaction of mucosa. Stability of dental implants depends upon integration of surrounding tissues.

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Bacterial adhesions and colonization are considered to play key role in the pathogenesis of infection related to biomaterials. Exposure of implants in the oral cavity presents a unique surface that can interact with native host bacteria leading to plaque formation. [3] The colonization of implants by oral microorganisms might be of importance for their clinical success or failure.

The interaction of host flora with teeth involves a highly selective process related to specific inter action between tooth bound salivary pellicle and bacterial surface adhesions. Alteration in either salivary pellicle or the bacterial surface can modify the initial bacterial attachment and therefore may alter the potential to develop plaque derived periodontal disease. [1, 4, 5]

It has been reported that different implant material promote selective adherence during early plaque formation. In vivo studies showed that *Actinomyces* species and *Streptococci* were the predominant colonizers preparing the environment for late colonizers that require more demanding growth conditions. Bacteria like Fusobacterium, Capnocytophaga and Prevotella species that bind to streptococci are also involved in

periodontal infection. Therefore it is important that the implant surface (around the transmucosal portion) is such that it reduces the number of initially adhering bacteria, minimizing plaque formation and subsequent inflammation to soft tissues. Surface characteristics of implant materials appeared to influence plaque formation in vitro. [6] Parameters like surface free energy and especially surface roughness were found to have significant impact on this process. Surface roughness was suggested to be more important than surface free energy. [7, 8, 9] Therefore, an implant surface ideal to resist bacterial colonization should be mainly smooth to allow the formation of an epithelial seal that prevents plaque accumulation.

While a rough transmucosal part of an implant will enhance plaque formation, the bony and connective tissue interface requires a porous or microtextured surface to promote tissue in growth. In a clinical study on titanium abutments, it was concluded that a certain threshold roughness (around R_a of 0.2 μm) might be most suitable to obtain a stable soft tissue sealing around transmucosal abutments. A titanium surface which is too smooth will therefore prevent cell attachment. However, an increase in surface roughness of the transmucosal portion above the R_a of 0.2 μm will facilitate early plaque formation. A smoothening below a threshold R_a of 0.2 μm showed no further significant changes, either in the total amount or in the periodontal pathogenicity of adhering bacteria. [10, 11]Therefore, an ideal transmucosal implant surface should not only minimize bacterial adhesion, but at the same time allow epithelial and connective tissue attachment.

In the past it was found that the biocompatibility of metal implants could be strongly enhanced by hard ceramic coatings separating body fluids from the metal. [12] In several studies, hard coatings were used to reduce plaque formation on implant[13, 14]or metal parts of partial denture. [15,16]Results of in vivo experiments using two different titanium hard coatings recommended the use of an osteophilic titanium-zirconium-oxide coating for the endosseous part of an implant. For the supragingival part a titanium-niobium-oxinitride coating was suggested which is extremely wear resistent and reduces bacterial adhesion. [11]

Properties of hard coatings, such as titanium nitride (TiN), are presently in the focus of interest, particularly with respect to their performance on tools for cutting, punching or shaping, as well as on machine parts and decorative coatings on consumer goods. Coating of metallic dental prostheses and instruments with TiN is applied to improve corrosion resistance and shear strength. Furthermore, it is preferred because of its golden colour. [13, 17]The use of an appropriate coating technique allows universal control of the required surface properties, resulting in reproducible thin hard coatings on almost any part of an implant. Sputtering can be used to produce dense, homogeneous corrosion-protective TiN coatings free of pinholes and cracks, if the sputtering parameters are optimized.[17, 18]The physical vapour deposition (PVD) process can also be used to deposit multilayer coatings.[15]

The excellent biocompatibility of titanium surfaces mainly results from its surface properties. While problems in osseous healing of implants appear to be largely solved, biomolecular pellicle adsorption and subsequent accumulation and metabolism of bacteria on these surfaces is still a main reason for the induction of inflammatory processes. Many *in vitro* and *in vivo* studies showed that parameters like surface free energy and especially surface roughness have a significant impact on plaque formation. [8, 9, 19]

Several studies have shown that titanium surfaces are very reactive. [20, 21] Titanium is covered by a surface oxide approximately 2 to 5 nm thick. [22] This oxide (mainly titanium dioxide) has amphoteric character and supports cationic and anionic exchange adsorption. At the interface between titanium oxides and saliva covalent, ionic or hydrogen bonding can contribute to the adsorption of biopolymermolecules, thus providing a very reactive surface.

It has been suggested earlier by smaller clinical studies that physical modifications (such as hard coatings) may have an influence on bacterial adherence. [13,23] Another clinical study demonstrated that coating of the metal parts of partial dentures with TiN resulted in a reduction of plaque formation. [16] Yet another experimental clinical study evaluating plaque adhesion to titanium, ceramics and prosthetic materials showed that the highest plaque accumulation was found on polished titanium whereas the accumulation on zirconium oxide ceramic and aluminium oxide ceramic was almost fifty percent lower. [24] It appears that bacterial adherence on ceramic material or coatings with ceramic-like character (as hard coatings) is lower as compared to titanium alloys. [25]

It was shown that reproducible surface coatings may have indeed a strong effect on bacterial colonization. ZrN(Zirconium nitride) in particular appears highly suitable to reduce plaque formation. The thermically oxidized titanium surface used as another modification

was probably the most cost-effective surface treatment. Thermic oxidation resulted in reduction of bacterial colonization, although less effective than coating with ZrN. [26]

In a study using controlled electrochemical oxidation, it could be demonstrated that a thicker oxide layer on titanium – which is also the case after using thermal oxidation seems to reduce plaque adhesion. [24] The reduction of oxygen gaps at the titanium surface resulting in a more apolar surface structure was discussed as a possible reason..

Even though thermic oxidation is a cost-saving method and resulted in reduction of bacterial adherence, the surface softness facilitates surface roughening on abutments during oral hygiene measures. [27, 28]However, the use of titanium hard coatings for implant abutments might prevent surface roughening during professional oral hygiene procedures. Due to the hardness of the coatings used and the multilayer technique of the sputter process it appears unlikely that prophylactic measures (e.g. the use of scalers) or chemicals (e.g. fluoride) could alter surface characteristics.

Results of experiments performed on modified titanium discs coated with saliva revealed that the number of adherent *S. mutans* was much lower than for *S. sanguis*, which is in contrast to the uncoated discs. Compared to the uncoated titanium discs, the number of adhered bacteria on all modified and saliva coated discs was distinctly lower for both bacterial strains. [29, 5, 30]Pellicle coating results in a general reduction in the number of adhering bacteria, irrespective of the substratum free energy. Adsorption of salivary components to a surface, the principal part in pellicle formation, is likely to be specific to that surface.

More bacterial colonies were counted for *S. sanguis* than for *S. mutans*, which is in contrast to bacterial counts on uncoated surfaces. One explanation is that *S. sanguis* has a very hydrophobic surface and there are many molecules in saliva, and thus in the pellicle, that could serve as hydrophobic receptors. [31] A higher number of available binding sites might be the reason for these findings. In addition, surface free energy is altered by saliva coating. [32] However, this effect needs further investigation.

The composition of a titanium pellicle differs from enamel pellicle in that cystatins and low-molecular weight mucin were not detected but, in contrast to the enamel pellicle, a high-molecular weight proline-rich glycoprotein may be a prominent component. In consequence, these differences in pellicle composition might explain significant differences in the initial adhesion rate of some specific bacteria to the surfaces. [33, 34, 35]

CONCLUSION

TiN and ZrN-coating of titanium surfaces resulted in a clear reduction of bacterial adherence. Their use as a coating for the part of an implant penetrating the soft tissue and as implant abutments might reduce plaque formation and in this way mucosal inflammation.

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