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Bacterial adhesion mechanisms on dental implant surfaces and the influencing factors

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ABSTRACT

Bacterial adhesion on dental implants may cause peri-implant disease including peri-implant mucositis and peri-implantitis. Peri-implantitis may lead to bone resorption and eventually loss of the implant. Therefore, the factors which influence bacterial adhesion are critical and revealed by many studies. The purpose of this review is to summarize the current knowledge of factors influencing the bacteria adhesion, including local factors of implant surface topography, abutment, cement and oral environment factors of saliva and protein. In addition, the corresponding strategies of surface modifications, coatings and challenges for implant materials as prevention and treatment approaches for bacteria adhesion on implant will also be discussed. We expect to give an overall picture of the bacteria adhesion on implant, and provide future perspectives, such as laser therapy, photocatalysis, plasma and bioelectric effect to inspire researchers to explore on this issue.

Contents

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1. Introduction

Dental implants have been used for several decades, with undeniable benefits for patient care and are now considered as the appropriate strategy for the replacement for missing teeth [1]. After the dental implant has been placed, complex processes take place in the wounded tissue and the non-vital, mineralized cortical bone has to be remodelled. In 1981, Albrektsson and Brånemark defined osseointegration as “a direct functional and structural connection between living bone and the surface of a load carrying implant” [2]. Observing the interface between the bone and the titanium surface with light and transmission electron microscopy, it was revealed that osseointegration is present at a visible and ultrastructural level [3].

The 5- or 10-year survival rates reported for dental implants are encouraging, ranging from 82% to over 95% [1,4]. However, the sole consideration of survival, defined by implant osseointegration (with or without peri-implantitis, or associated issues such as aesthetics) is not sufficiently representative of the global clinical picture. When considering success instead of survival, the rates decrease significantly (from 93.9% to 73.5%), becoming lower than endodontically treated teeth [1]. The complications associated with this lower success, namely peri-implant mucositis and peri-implantitis, are now increasingly reported. It was reported in 5–11 year observations that peri-implant mucositis affects 40–90% of implants in 80% of subjects, while around 20% of implants develop peri-implantitis [5–9].

Peri-implant mucositis and peri-implantitis are triggered by the presence of biofilms at the implant surface (Fig. 1). Mucositis is defined as an inflammation of the soft tissue surrounding dental implants, as evidenced by change in mucosal colour and contour, and bleeding upon gentle probing (< 0.25 N) [10]. The condition is not accompanied by bone loss around the implants, and is reversible. Characterised by the predominance of plasma and polymorphonuclear cells in the soft tissue around the implant [11,12], mucositis may result in the proliferation of the sulcular epithelium and the degeneration of connective tissue, followed by the destruction of the mucosal seal. Once this seal is lost, the sub-gingival implant surface can be progressively colonized by pathogenic bacteria, which may be followed by inflammatory resorption of the surrounding alveolar bone [13]. Such bone loss can be observed in the radiographic images, and confirmed clinically by the presence of a peri-implant pocket [14].

The frequency and/or severity of such complications depend on a large variety of factors, either before, during and after the treatment procedure. Implant failures might be divided into early and late types [15,16]. Early failure occurs when the implants fail during the process of osseointegration, whereas late failure refers to issues occurring only after occlusal loading [15]. Peri-implant mucositis and peri-implantitis are inflammatory responses of gingival and alveolar bone tissues triggered by the colonization of different pathogenic microorganisms on the implant surface and their organization in biofilm, and may be considered as the most common cause for late failures [14,17,18].

A microbial biofilm is defined as a “complex, functional community of one or more species of microbes, encased in an exopolysaccharide matrix and attached to one another or to a solid surface” [19]. Its formation is a rather complex process as well as an essential step in the development and evolution of the pathologic process [20–22]. It can also be affected by many factors: surface characteristics of implant; bacterial types and properties; serum proteins and oral environment [23]. The process of bacterial adhesion to a surface can be divided into two phases, including an initial, instantaneous, and reversible physical phase (phase one), followed by a time-dependent and irreversible molecular and cellular phase (phase two) [23–25]. In brief, following initial attachment, bacteria start to colonize and grow on the implant surface. Multilayered cellular clusters are formed due to cell proliferation, intercellular adhesion and production of an extracellular polymeric matrix [26,27]. Subsequently, such a three-dimensional architecture develops into maturation. After that, some bacteria start to detach from the implant surface and disperse into the body fluids, leading to the spreading of biofilm across surfaces [28].

Microbiological studies in healthy peri-implant tissues demonstrated the presence of large proportions of coccoid cells, with a low proportion of anaerobic and aerobic species, a small number of Gram-negative species, and a low detection of periodontopathogenic bacteria [29–31]. Gram-positive aerobic bacteria such as *Streptococcus mitis*, *Streptococcus sanguis* and *Streptococcus oralis* were observed on dental implant surfaces surrounded by healthy oral environment [32].

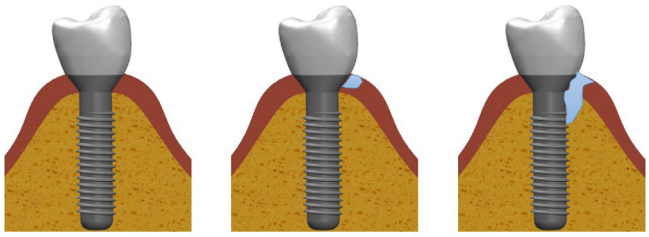

Fig. 1. Progressive periodontal scenarios with the presence of biofilm at the implant surface.