Clinical and morphological aspects of the implant/soft tissue interface

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Purpose: This paper is aimed to describe clinical and morphological aspects of the soft tissue implant/soft tissue interface and to provide guidelines for assessment of peri-implant mucosal health.

Materials and Methods: Critical appraisal of the current literature regarding the interface between dental implants and the peri-implant mucosa is performed.

Results: The articles reviewed agree that smooth titanium surfaces are highly compatible with oral soft tissues. The biological seal that is formed by the mucosa surrounding dental implants is established to provide protection against microbial invasion. Implant design may influence the location of the biological seal, but not the formation of the seal. Non-keratinized peri-implant mucosa does not seem to predispose peri-implant disease. Keartinized mucosa seems to be desirable for hygiene procedures and esthetics, but not necessary for implant success. Assessment of peri-implant mucosal health has shown to be a predictable indicator of peri-implant disease.

Conclusion: Studies have consistently shown that biologic seal is formed between the oral cavity and the underlying tissues after implant placement, providing protection against the microbial challenge. However, future studies are needed in order to clarify the importance of the biological seal for implant success.

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Clinical Significance: Since dental implant penetrate into the oral cavity, the interface between dental implants and oral soft tissues allows the formation of a biologic seal. This biologic seal has the ability to protect the underneath structures by closing the pathway of communication between the oral environment and the alveolar bone. Assessment of the integrity of this interface aids in early detection of peri-implant pathologies, and may prevent future implant bone loss. In addition, the implant/soft tissue interface may provide esthetics that implant supported restoration needs.

Key words: biological width, dental implants, mucositis, peri-implant mucosa.

INTRODUCTION

The replacement of missing teeth by means of endosseous dental implants has become an important part of dentistry. Over the last two decades, research has validated the success of osseointegrated implants as a

viable replacement for partial and complete edentulism. Although techniques and materials have been developed which are capable of a high degree of clinical success, the ultimate long-term success of implants is dependent upon the efforts of both the patient and dentist in maintaining the health of the peri-implant tissues.

Dental implants have two distinct interfaces with oral tissues. First, there is the peri-mucosal interface where soft tissue meets the implant, creating a biological seal. Secondly, the endosseous interface exists where alveolar bone is in close proximity with the titanium surface, providing stability and rigidity to the implant. Since the soft tissues reform around dental implants as a result of a healing process following implant placement, they are called peri-implant mucosa, not gingiva.

The aim of this paper is to review the literature regarding the interface between the implant and peri-implant mucosa, which is divided into epithelium and connective tissue.

EPITHELIUM

Epithelial tissues have the capacity to proliferate and to move on surfaces. Following implant/abutment placement, epithelial migration from adjacent soft tissues begins. The epithelium moves in apico-coronal direction as soon as it reaches the implant surface, giving rise to a junctional epithelium about 2 mm long.[1-3] The formation of a junctional epithelium in the implant/mucosal interface can be considered the first barrier of defense against oral microflora. Figs. 1a and 1b illustrate the relationship between implant and surrounding soft tissue.

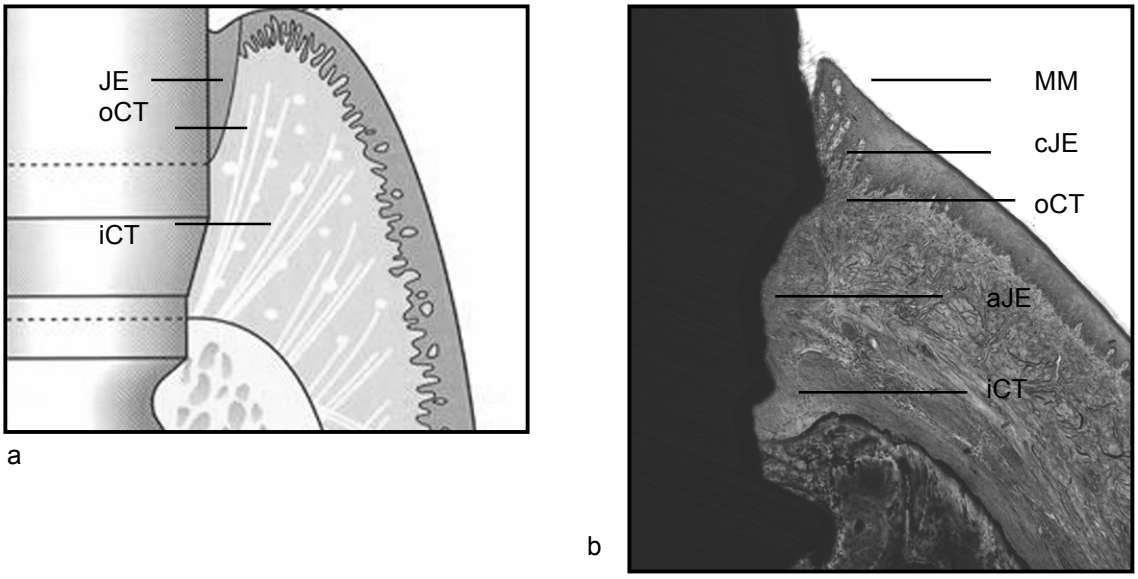

Fig. 1. a. Diagram illustrates the relationship between implant and surrounding soft tissue interface. b. Histological sections of the implant/peri-implant mucosal interface.

JE: Junctional epithelium; **oCT:** Outer connective tissue; **iCT:** Inner connective tissue; **MM:** Mucosal margin; **cJE:** Coronal junctional epithelium; **aJE:** Apical juctional epithelium; **iCT:** Inner connective tissue.

Donley et al. suggested that junctional epithelium cells adjacent to a titanium implant to form a hemidesmosomal type of attachment to the implant surface similar to that of a natural tooth.[4] On the other hand, Meffert et al. suggested that the junctional hemidesmosomal arrangement may not be predictable in a metallic system.[5] Research has shown that once epithelial cells have reached the implant surface, their attachment occurs directly via a basal lamina (<200 nm) and hemidesmosomes.[6-8] Kawahara et al. hypothesized another form of attachment, where a glycoprotein layer of approximately 200 nm forms between the cell wall, without direct epithelium/implant contact.[9,10] Lekholm and Adell, however, have discussed the fact that the seal is probably viable and adequate in function based on the fact that there were minimal histologic inflammatory reactions in the underlying connective tissues.[11,12] The surface topography has also shown to play a role in the soft tissue attachment to titanium surfaces, since polished surfaces have shown higher compatibility to fibroblasts when compared to rough surfaces.[13-18]

A prerequisite to a successful dental implant should be obtaining a peri-implant mucosal seal to the implant surface.[19,20] Failure to achieve or maintain this seal results in the apical migration of the epithelium into the bone/implant interface and possibly caused partially or completely encapsulation of the endosseous implant.[21,22] In a natural dentition, the junctional epithelium provides a seal at the base of the sulcus against the penetration of chemical and bacterial substances. If the seal is disrupted and/or the fibers apical to the epithelium are destroyed, the epithelium migrates apically, forming a periodontal pocket after cleavage of the soft tissue from the radicular surface. The importance of the biologic seal should be emphasized due to the absence of cementum and fiber insertion into the implant surface which may cause formation of a "peri-implant pocket" extending into the osseous structures. The characteristics of the soft tissues adjacent to teeth and implants were listed in Table 1.

Table 1. Soft tissues adjacent to teeth and implants.

	Attachment	Orientation of collagen fibers	Source of blood supply	Biological width/Seal
Teeth	Basal lamina and hemidesmosomes	Perpendicular	Periosteum, PDL	JE: 0.97 mm[70] CT: 1.07 mm[70]
Implants	Basal lamina and hemidesmosomes	Parallel	Periosteum	JE: 1.88 mm[1] CT: 1.05 mm[1]

PDL: Periodontal ligament; JE: Junctional epithelium; CT: Connective tissue.

CONNECTIVE TISSUE

For the connective tissue attachment to the implant, collagen fibers form a tight cuff around the implant abutment. The length of the connective tissue attachment ranges from 1.3 to 1.8 mm,[1,23] and is also dependent on the design of the implant (one- or two-part implant). There have been reports in the literature that the use of a plasma spray surface promotes connective tissue adherence with fibers inserted

functionally at 90° into the plasma sprayed surface of the implant.[15,24] However, most reports have shown that connective tissue fibers are not inserted into the implant surface. Dense bundles of thick collagen fibers are oriented only longitudinally due to a lack of fiber insertion.[25,26]

The connective tissue of the peri-implant mucosa can be subdivided into two distinct zones. The outer zone located under the junctional epithelium, composed of collagen Types I and III, is responsible for the transformation of collagen. The inner supracrestal connective tissue zone, composed mainly of Type I collagen, is responsible for mechanical resistance and stability of the peri-implant mucosa.[25,27]

The connective tissue surrounding dental implants possess fewer capillaries as compared to the connective tissue surrounding natural teeth.[28] The blood supply of the peri-implant mucosa is composed of terminal branches of larger vessels originating from the periosteum of the alveolar bone, while the vasculature of the gingiva and the supracrestal connective tissue at teeth is derived from the supraperiosteal vessels lateral of the alveolar process and the vessels of the periodontal ligament.[28] The peri-implant mucosa has also shown to exhibit an inflammatory response, similar to gingival tissues, characterized by increased proportions of T- and B-cells in the inner connective tissue. However, the host response is less pronounced than in gingival tissues.[29-31]

BIOLOGICAL WIDTH/SEAL

Animal and human studies have shown that an adequate biological width/seal can only be achieved if there is a supracrestal smooth titanium surface of at least 3 mm long in the apico-coronal direction.[1,21,32-35] The junctional epithelium occupies approximately 2 mm of this surface, while the rest of it is occupied by the connective tissue. Berglundh and Lindhe observed that the values for two-part implants were 2.0 mm for JE, and 1.3 to 1.8 mm for CT.[23] Cochran et al. observed the values for a biological width/seal around one-part implants before and after loading period of 12 months. Mean values for 3-month unloaded implants were 0.49 mm for sulcus depth (SD), 1.16 mm for the junctional epithelium attachment (JE), and 1.36 mm for the connective tissue attachment (CT). These dimensions differed from the 3-month loaded implants that showed 0.50 mm for SD, 1.44 mm for JE, and 1.01 mm for CT. After the 12-month loading period, these values were 0.16 mm for SD, 1.88 mm for JE, and 1.05 mm for CT.[1] It can be speculated that the biological width/seal increases approximately 1 mm after implant loading, possibly due to crestal bone resorption.

THE INFLUENCE OF IMPLANT DESIGN

Since a biological seal is formed by epithelial and connective tissue attachments to the implant surface, the length of titanium surface interfaced with the peri-implant mucosa may influence the location of the biological seal. One-part implants seem to provide a more predictable surface for soft tissue attachment, since the length of the smooth titanium surface is stable, and the implant/abutment connection is positioned more coronally.[36] Soft tissue attachment in two-part implants are dependent on the location of the restorative margin that must respect the dimensions necessary for the formation of a biological seal/width.

If the required dimensions are not respected, an apical migration of the biological seal/width can be expected with subsequent exposure of the rough implant surface to the implant/mucosal interface.

In two-part implant, the microgap present at the implant/abutment interface may result in an inflammatory infiltrate due to presence of microorganisms that can populate the implant/abutment junction.[22] The inflammatory infiltrate is a protective host defense mechanism that limits microbial invasion, preventing further deterioration of implant supporting tissues. Ericsson et al. showed that an apical migration of the junctional epithelium occurs after abutment connection in two-part implant, allowing a biological seal to be formed.[34] Weber et al. compared the formation of the biological seal around one- and two-part implants and demonstrated that a biological seal is formed irrespective of the implant design. However, two-part implants resulted in a longer junctional epithelium attachment, which was located more apically. The distance from the crestal bone was also shorter in comparison to one-part implants. No differences were found in the length of connective tissue attachment.[37] Similar findings was also reported by Hermann et al.[38]

KERATINIZED VS. NON-KERATINIZED PERI-IMPLANT MUCOSA

The need of the keratinized mucosa surrounding dental implants in order to maintain peri-implant mucosal health is controversial, since healthy tissues can be noted not only on the presence but also on the absence of keratinized peri-implant mucosa. Becker et al. speculated that keratinized peri-implant mucosa is not a necessary prerequisite for peri-implant health and that movable mucosa around the trans-epithelial extension of an endosseous implant is not necessarily a vulnerable situation.[39] However, Warrer et al. used an animal model and demonstrated that implants surrounded by non-keratinized mucosa may exhibit more crestal bone loss and mucosal recession, when compared to implants surrounded by a keratinized mucosa, under similar levels of plaque accumulation.[40] Similar observation was also demonstrated by Strub et al.[41]

Adell et al. reported based on 15-year results of 2,768 implants placed in 371 subjects, that even when a movable mucosa was found surrounding implants, a clinically healthy mucosa was noted.[42] Wennstrom and Lindhe evaluated the prospective and retrospective studies of Branemark to associate implant failure with nonkeratinized peri-implant mucosa. An overall 90% success rate in terms of fixture survival and in which the marginal tissue was keratinized gingiva in only 67% and 51% of the facial and lingual surfaces respectively demonstrated that the absence of a keratinized peri-implant mucosa does not predispose implant failure.[3,43] On the other hand, Kirsch and Ackermann published 10-year clinical results of 2,284 implants, with a failure rate of 2.2%. The study suggested that the main cause of failure was insufficient width of keratinized peri-implant mucosa.[44] The difference in the two studies may be attributed to the difference that the retrospective and prospective studies of Branemark were in fully edentulous cases, while the majority of the implants in Kirsch's study were in partially edentulous mouths.

Although the absence of a keratinized mucosa does not seem to predispose implant failure, keratinized tissue may facilitate personal home care and maintenance procedures. Connective tissue grafts, free gingival grafts, and other techniques, have been described and have achieved predictable results for

augmentation of keratinized mucosa around dental implants.[45-52]

ASSESSMENT OF SOFT TISSUE HEALTH

Experimental evidence during the last decade supported the concept of a host-parasite imbalance being the major responsible reason for implant failures after loading[53,54]. As in the case of teeth, the imbalance in the host-parasite equilibrium can manifest itself in a series of inflammatory changes, which lead to two distinct clinical syndromes: 1) Mucositis is a reversible inflammatory lesion confined to the superficial peri-implant mucosal tissues, and 2) Peri-implantitis is a lesion involving the peri-implant soft tissues as well as the marginal portion of the bone-implant interface.

As is true for gingivitis and periodontitis, the relationship between peri-implant mucositis and peri-implantitis is not known with respect to the natural history, evolution and progression.[55] Endosseous dental implants rarely fail beyond the first year after restoration.[56,57] However, it has been suggested that conventional periodontal therapy should be instituted if inflammation develops around an implant.[58,59] Problems limited to the peri-implant mucosa and not involving the supporting bone have been defined as "ailing implants" and, more recently, as biological complications.[36,54]

Probing studies in dental implants are dependent on the emergence profile of the final restoration. Assessment of probing depth around overcontoured crowns is largely irrelevant unless the superstructure is removed, allowing probing parallel to the long axis of the fixture. Peri-implant probing depth, mucosal recession, bleeding upon probing, and peri-implant exudation have been suggested as useful diagnostic tools for biological complications.[60-62] Probing studies in dental implants are also dependent on the presence or absence of inflammation since it has been shown that a probe is able to penetrate into the connective tissue and reaching close proximity to the alveolar bone even in presence of mild to moderate mucositis.[63] Successful implants usually allow approximately 3 mm of probe penetration, while deeper probing depths are often associated with a marked difference in the composition of the peri-implant microflora.[1, 62]

While the overall prevalence of peri-implantitis ranges from 5-10%, the prevalence of peri-implant mucositis seems to be higher. Smedberg et al. reported that radiological diagnosed marginal bone defects in 6% of implants after 2 years in function; 28% of the patients were diagnosed as having peri-implant mucositis.[64] However, the classic study of Adell and coworkers reported that the mean values of peri-implant mucositis and plaque were 7.6 and 13.7% respectively.[42]

The microbiological findings related to healthy and failing implants are similar as those for healthy and periodontally compromised teeth.[60,62] Increased levels of subgingival *Porphyromonas gingivalis, Prevotella intermedia,* and *Fusobacterium nucleatum* may lead dental implants to fail. On the other hand, failing implants with a traumatic etiology may have a microflora comprised predominantly of streptococci which is consistent with periodontal health.[65] The microbiota present in the oral cavity can influence bacterial colonization around implants since implants placed in partially edentulous periodontal patients had the same microflora found in their periodontal pockets.[66,67] Salcetti et al. found increased levels of

PGE_2, IL-1ß, and PDGF in failing implant sites demonstrating that not only the microbiota but also the host response is similar to those of periodontally diseased teeth.[68]

Sbordone et al. evaluated implants placed in partially edentulous patients with previous history of moderate to severe chronic periodontitis. They found that the treated periodontal sites were stable at 3 years, with nearly 90% of the sites demonstrating CAL<1 mm. Despite the similarity of the microbiota of the samples from implant and natural tooth sites, no evidence of recurrent periodontal attachment loss or implant failure was noted.[69] These findings support the recommendation that patients with implants should be evaluated at regular visits for periodontal maintenance procedures, and any clinical signs and symptoms of peri-implant mucositis or peri-implantitis should be recorded and treated.

CONCLUSIONS

The use of dental implants in the treatment of partially or fully edentulous patients has become an important addition in oral/dental rehabilitation. The literature to date concurs that coated, non-coated, or plasma-sprayed implants all have the capacity to achieve osseointegration, if appropriate surgical techniques are involved.

The fact that implants penetrate the oral mucosa and the mucosa is able to seal around the implant, leads to the assumption that the peri-implant mucosa fulfills the function of acting as a barrier to protect the anchorage to the alveolar bone. The biological seal has shown to be present irrespective of the implant design, but a smooth titanium surface is preferable for soft tissue attachment.

Research has shown that insufficient plaque removal may lead to peri-implant disease with or without bone loss. However, it is unclear how important this cause is as a cause of implant failure, compared with other factors, such as inadequate bone healing, unfavorable quantity and quality of bone, or (bio) mechanical and functional problems. It is also not understood if the presence or absence of keratinized peri-implant mucosa is needed to slow down or prevent tissue breakdown. The current knowledge has shown that the keratinized mucosa maybe desirable, but not necessary.

Future studies in these areas are necessary and will certainly provide a better understanding in this field.

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