The peri-implant mucosa – sanguine dreams and harsh reality

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Introduction
Dental implants are inserted into alveolar bone to initiate osseointegration, with the clinician’s expectation that the peri-implant mucosa will heal to the abutment surface and subsequently provide the primary functions of the gingiva: protection and stability. The reality is a different one and has clinical implications.

In addition to contributing to the anchorage of the tooth in the jaw, the primary function of the marginal periodontium, or gingiva, around our natural teeth is protection; that is, to provide a seal against the contaminated environment of the oral cavity, to stabilize the position of the tooth in the alveolar bone, to withstand the frictional forces of mastication, and to defend against foreign invaders at the interface between the teeth and the soft tissues.

Viewed under powerful microscopes, the structural framework that makes it possible for the periodontium to accomplish these tasks displays a variety of spectacular structures. On close examination we can see how the gingiva is protected and sealed by a highly specialized tissue — the so-called junctional epithelium — and the way the connective tissue of the gingiva is anchored in the mineralized cementum of the root via strong collagenous fibers.

The junctional epithelium
The peri-implant mucosa surrounding successful endosseous implants is in many ways analogous to that surrounding the natural dentition. The lamina propria extends out from the alveolar bone coronally, covered by a keratinized oral epithelium.

In healthy situations, a shallow sulcus forms, lined by a sulcular epithelium, which is contiguous with the junctional epithelium.

Of the tissues in contact with the implant, the junctional epithelium most closely matches the structures around a

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tooth. It forms an epithelial sleeve around the tooth collar and preserves thereby the continuity of the epithelial coating of the oral cavity. It is located at a strategically important interface between the contaminated oral cavity and marginal periodontium, which needs protection from bacteria and their byproducts (for review see Bosshardt and Lang, 2005).

Its unique structure and its antibacterial peripheral defense mechanisms provide a seal, given the attachment of the peri-implant mucosa to the abutment and the control of the life-long constant microbiological challenge (Schüpbach and Glauser, 2007).

**The connective tissue**

Despite these similarities, important differences exist in the way the connective tissue interfaces with the surface of the abutments or, in case of marginal bone loss, with the implant collar. Strong dento-gingival collagenous fibers are directed perpendicular / oblique to the tooth surface with firm anchorage in the mineralized root cementum. Thus the connective tissue forms a barrier inhibiting – in a healthy situation – the further downgrowth of the junctional epithelium, and supports the latter in the task of mucosa attachment.

In terms of architecture and function, evolution has delivered a remarkably good solution for the attachment and protection of the tissues around our teeth – an almost perfect solution, in fact, as we know that this area of the body is challenged as long as we live by the presence of bacteria, which may destroy this sophisticated attachment system via one or several periodontal diseases.

Professionals who provide implants continue to dream, however, of a perfectly analogous counterpart to the natural connective tissue (as far as architecture and function around the abutments are concerned). The reality is unfortunately quite different. Functionally oriented collagenous fibers adhering to or anchored in the surface texture of an abutment are conspicuous by their absence.

The interface between connective tissue and implant surface looks, at least at small magnifications, perfectly smooth. That means that the connective tissue is adapted only to the abutment surface and cannot provide surface adherence for the junctional epithelium. Thus, the sealing of the peri-implant mucosa and protection of the underlying soft tissues and bone are based on the junctional epithelium only, which makes for a much more vulnerable situation than in the situation ordinarily found around natural dentition.

**Stability of the peri-implant mucosa**

The structurally most important elements providing stability to the gingival around our natural teeth are dense, collagenous fiber bundles, which form the extremely complex architecture of the supracrestal fiber apparatus (Feneis H, 1952).

The most important fibers for stability are functionally oriented fibers anchored in root cementum and the circumferentially running fiber bundles that form a circular “o-ring” around the tooth. Functionally oriented fibers are absent around abutments, but a recent study showed the presence of strong circular fibers (Schüpbach et al, in preparation). In absence of Sharpey's fibers, the stability of the peri-implant mucosa is mainly based on fiber bundles arranged in this “o-ring” configuration.

**Conclusions**

If we agree on the importance of the “o-ring” for the stability in the peri-implant mucosa, we can conclude that the design of the implant/abutment complex is likely to be of importance. A back-tapered implant collar, for instance, and a diverted abutment design provide more volume for an “o-ring”.

For doctors it is very important to be aware that punching out peri-implant soft tissue, particularly in the front area and...
The “O-ring.” Stability provided by circular fibers arranged around an abutment in an o-ring configuration (left). They correspond to the circular fibers of the supracrestal fibers around a tooth (right). (Rendering on right adapted from Feneis, 1952 and Schröder, 1992).

in the presence of a thin gingiva biotype, could result in the complete removal of the “o-ring,” leading to insufficient stability of the peri-implant mucosa. There is a range of alternative clinical procedures that can be carried out instead, among them short incisions to place the thin band of keratinized mucosa around the implant, or the preparation of a mini-flap. (Jeong et al, 2008)

References


This article is a reprint from Nobel Biocare News Vol. 15, No.1, 2013. © Nobel Biocare Services AG, 2013.