The Peri-Implant Sulcus

Geoffrey R. Bauman, DDS, MS/John W. Rapley, DDS, MS/William W. Hallmon, DMD, MS/Michael Mills, DMD, MS

A review of the peri-implant sulcus, including the histology of the sulcular epithelium, epithelial attachment, and the gingival connective tissue, is presented. The peri-implant junctional epithelial attachment is mediated by hemidesmosomes, as in the periodontal tissue. There is some controversy on the possible attachment of connective tissue fibers to the implant, but current studies indicate a parallel orientation with no insertion of the peri-implant connective tissue fibers. This difference in connective tissue attachment may affect the peri-implant tissue's susceptibility to disease. (Int J Oral Maxillofac Implants 1993;8:273—280.)

Key words: epithelial attachment, gingival connective tissue, histology, peri-implant sulcus, sulcular epithelium

Are the peri-implant and periodontal soft tissues clinically comparable? Reports of peri-implant soft tissue histology have provided evidence of an equivalent sulcular epithelium, some affectation of a permucosal epithelial seal, and similar gingival fiber groups. However, useful data on long-term clinical results are needed to determine whether the peri-implant tissues are as similar in function as they are in histology. Various studies provide information on similarities and differences between peri-implant soft tissues and those associated with the natural dentition. Some claim a critical contribution of the permucosal seal to the success of the implant. Others have argued that the seal appears to function similarly to the attachment at natural teeth as long as the implant is relatively immobile and the implant surface and structural characteristics allow adequate plaque control.

Brånemark and Albrektsson found that mobile transcutaneous titanium implants were encircled by inflammation and/or soft tissue pockets, whereas corresponding nonmobile implants remained free of soft tissue problems. They postulated that mobility of the implant causes stress concentration at the attachment epithelium which may result in failure of the unit. Jansen and De Groot found that percutaneous implants in rabbits and guinea pigs became marsupialized unless immobilized by insertion into bone, or in close proximity to it. These findings support the opinions of Schroeder et al, who believe immobility of the soft tissue-implant junction is an important factor in the permucosal seal of implants.

The need for attached gingiva encircling the implant has been the subject of some discussion. Schoo and Van der Velden found no difference with regard to peri-implant soft tissue type in resistance to plaque accumulation, gingivitis...
prevalence, or appearance of recession. Zarb and Symington\textsuperscript{11} suggest that an attached gingival collar may be more capable of resisting the mechanical trauma of tooth brushing and the lateral pull of muscle attachments in severely resorbed ridges, although they acknowledge that keratinized gingiva is not a prerequisite for success. They also found that there was less peri-implant pocketing at implants surrounded by attached gingiva. Schroeder et al\textsuperscript{9} suggested that a keratinized gingival collar was more likely to be associated with a fibrous type of "attachment" to the implant (with textured surface implants) which can stabilize the peri-implant permucosal tissues against trauma. Others also advocate an attached gingival zone around implants\textsuperscript{12,13} to prevent traumatization by plaque-control procedures when bone resorption occurs, possibly exposing the most coronal thread of screw implants.

**Histology**

Light and electron microscopic studies depict similarities between the peri-implant and natural tooth sulcus in animal models\textsuperscript{2,3,14} and humans.\textsuperscript{4,15} Kurashina et al\textsuperscript{2} described noninflamed and inflamed peri-sulcular tissue at 27 dense hydroxyapatite (HA) implants in dogs, which closely parallels that observed about natural teeth in the same animal model:

1. **Noninflamed:** In the connective tissue of the gingiva, a limited infiltration of inflammatory cells was noted. This field of inflammatory cells was the same area as that in the gingiva of neighboring teeth. Outside this area, numerous bundles of collagen fibers were seen and many of these fibers terminated perpendicularly to the interface with the implants, resulting in a saw-toothed pattern (Sharpey's fibers?). The epithelium on a lower level, adjoining the implant surface, was 2 to 5 cells thick. There was no cell differentiation between the subsequent superficial layers, no keratinization, and few or no papillae.

2. **Inflamed:** There were multiple bone resorptions at the alveolar bone crest. In some sections, islands of bone were seen lying at the interface with the implant, just above the alveolar bone. At the supraalveolar level the gingival connective tissue showed a large field of inflammatory cells and disappearance of collagen fibers. Epithelial downgrowth lined the implant sulcus. The lowest level was always above the alveolar bone.

**Sulcular Epithelium**

The sulcular epithelium surrounding dental implants in animal models has been compared to the sulcular epithelium of natural teeth in the same animal.\textsuperscript{2,16,17} Steflik et al\textsuperscript{17} provided a description of the crevicular epithelium around 36 sapphire implants in dogs:

The tissue adjacent to the implant consists of the free gingival margin composed of collagenous stroma, covered by stratified squamous epithelium. The external surface of this free margin is keratinized epithelia in the dog. As the epithelium progresses from the superior aspect of the margin crest down internally along the
implant, the epithelia becomes non-keratinized and is termed the sulcular gingiva as it lines the gingival sulcus. SEM of block implant specimens displayed a normal appearing free gingival margin surrounding the implant. The transition from the keratinized squamous epithelium of the external free gingival margin over the gingival crest and a change to non-keratinized sulcular epithelium on the internal surface was vividly demonstrated. The width of the sulcular epithelium narrowed as it progressed to the lower recesses of the sulcus. SEM of the plasma etched EPON sections displayed excellent retention of sulcular epithelial morphology. The outermost epithelial cells appear more flattened than the basal cells. Intercellular connections were prominent with scattered leukocytes found in small intercellular cysts at the peripheral margin. At the sulcular margin, minor incursions of bacteria were present. The microorganisms were generally confined to spaces similar to crypts occupied by leukocytes.

This description of the peri-implant sulcus in the dog is similar to the nondiseased periodontal sulcus in the dog and in humans.

"Epithelial Attachment" Histology
The epithelial attachment histology around natural teeth has been well described in the literature. At the base of the gingival crevice of natural teeth, cells of the attachment epithelium have been characterized as being larger, with wider intercellular spaces and fewer desmosomes than the cells lining the gingival crevice. At the tooth surface, a basal lamina is secreted by the junctional epithelial (JE) cells, which is composed of three distinct layers: the lamina lucida, lamina densa, and sublamina lucida. The JE cell membrane approximating the basal lamina contains electron-dense plaques (hemidesmosomes), which have been shown to be associated with epithelial attachment to underlying connective tissue or to substrate. In addition, various proteinaceous cuticles are often interposed between the basal lamina and tooth surface. Most studies advocating the presence of an epithelial attachment apparatus at implants have cited the presence of one or more of these components as evidence.

Significant debate has transpired over the histology at the dental implant-permucosal penetration site. Listgarten and Lai were the first to report hemidesmosomes associated with implant material (resin). James and Schultz, who were also early proponents of the hemidesmosome-implant relationship, observed hemidesmosomes associated with vitallium implants in monkeys. However, many investigators refused to accept the early reports of hemidesmosomes in association with implants. Older histologic sectioning techniques made identification of hemidesmosomes difficult and were probably responsible for the negative findings in some reports. However, after the development of new histologic techniques such as cryofracture and oxygen plasma surface etching of poly(methyl methacrylate), hemidesmosomes became more accurately defined.
Hemidesmosomes have been detected in vivo in various living models in association with several implant materials: titanium in man, 15,29 sapphire in dogs, 3,17 and vitallium in monkeys. 23,28 Schroeder et al 9 reported functional hemidesmosomes and basal lamina on titanium-sprayed (textured surface) implants in monkeys, stating that:

As the implant surface particles protruded into the basal lamina, the intracytoplasmic tonofilaments, which are usually parallel to the cell axis, were rearranged to a position perpendicular to the surface of the inward protruding implant possibly indicating that functional loads imparted to the implant are partially taken up by the epithelial cells.

In vitro studies have reported hemidesmosomal attachment in different species and various materials including resin, 22 HA and polystyrene (guinea pig epithelium), 30 and titanium (porcine periodontal ligament epithelium). 31 Many of these studies provided excellent electron microphotographs of the hemidesmosome-implant phenomenon. 17,23,28-32 Regardless of whether the hemidesmosome truly functions or is present in sufficient quantity to be effective, there is general agreement that some type of adhesion exists at the epithelium-implant junction (Fig 2).

Other in vitro attachment apparatuses that have been noted between epithelial cells and titanium, carbon apatite, and gold include extracellular matrix contacts (gaps of 100 nm between the cells and substrata that are filled with extracellular substance) and focal contacts (15-nm gap between cell and substratum, with microfilament bundles in the cytoplasm at these sites). 30

Van Steenberghe 33 believes that too much attention has been placed on the presence of hemidesmosomes contacting the implant surface. He states, "Although this has undoubtedly been documented both in vitro and in vivo, this morphologic structure only indicates a close contact and can be correlated to some adhesion force.... More fundamental is the question of why migration of epithelial cells does not seem to occur in the osseointegrated implants modum Brånemark." Gould 34 reasoned that contact inhibition from the underlying connective tissue was responsible. Van Steenberghe 33 agreed with this theory of contact inhibition but felt that the contact inhibition was achieved from the mature collagen seal at bone level or a surface irregularity of the implant.

De Lange et al 35 reported that the competency of the epithelial seal was related to the thickness of underlying connective tissue around HA implants in dogs. They found connective tissue attachment to HA implants and noted a direct relationship between increased thickness of the implant associated connective tissue attachment and adequacy of the seal at the pseudoepithelial attachment. De Lange et al 35 suggested that the tenuous relationship of the epithelial attachment in natural teeth should be assumed to be at least as weak in implants, and that a connective tissue attachment is desirable regardless of the adhesive qualities of the epithelium at the
Efforts to discourage epithelial migration led to experimentation with circumferential grooves at the cervical area of test implants in animals. Chehroudi et al, Brunette et al, and others have reported the effect of horizontal grooves on fibroblast orientation and epithelial migration. The in vitro studies showed that fibroblasts will orient with the direction of a groove (0.5- to 3.0-cm deep) in titanium or epoxy. This effect seems to limit epithelial migration past the groove. This was confirmed in vivo in cat crania on epoxy and titanium implants with 17-µm-wide × 10-µm-deep grooves separated by flat 22-µm-wide ridges. Epithelial migration was prevented past the groove in experimental specimens but not in smooth-surface controls. Another study by Chehroudi et al reported that epithelium is excluded from the grooves by the fibroblasts which can exist in grooves or on smooth surfaces.

Whatever the mechanism of epithelial attachment, it appears to have some similarity to that of the natural dentition because uncontrolled plaque accumulation in the sulcular area of implants is associated with downgrowth of the "pseudoepithelial attachment." A study by Koth et al compared the epithelial attachment of implants (unspecified type) receiving oral hygiene to those without oral hygiene in dogs. At 3 months, attachment was still present at both implants; however, at the no-hygiene sites, the attachment apparatus appeared compromised with fewer hemidesmosomes and intercellular desmosomes and a "looser" appearance. If unchecked, complete marsupialization, avulsion, and/or infection may occur.

Whether hemidesmosomes at epithelial-implant junctions are comparable to their counterpart in the natural dentition is yet unresolved. However, because the evidence for implant-epithelial adhesion predominates in the literature, one is led to believe in its existence and probable function (Table 1).

**Gingival Connective Tissue Histology**

Although van Steenberghe has remarked that much needs to be learned of the nature of the gingival connective tissue associated with dental implants, many reports have described the similarity of the peri-implant gingival connective tissue to that of the natural tooth regarding the amount and character of inflammation, the component tissues, fiber structure and alignment, and even evidence of attachment to the implant. James and coworkers and Armitage et al detailed the gingival fiber groups within the peri-implant connective tissue (Fig 2), which resembled those seen in teeth:

The most striking and probably the most critical difference between periodontal and peri-implant tissues is the absence of Sharpey's fibers extending into the implant. This results in a compromised pergingival defense mechanism, which must rely primarily on the adhesive quality of the junctional epithelium. There is no fiber
"backup" system as that enjoyed by the natural dentition. And further, fibers resembling the gingival ligament may be observed in the pergingival area, originating from the peri-implant ligament, the palatal submucosa, buccal lamina propria, and the bony crest. These fibers anastomose with the circumferential fibers, which extend around the implant post in the free gingiva in a manner similar to the circumferential fibers of the dental gingiva. It would appear that this fiber system provides the architecture for the gingival crest noted as an elevation around the neck of pergingival implants. Fibers of the alveolar crest have been observed extending into the implant junctional epithelium. Since it is recognized that collagen orients in the direction of tension, and in light of the evidence favoring adhesion of the junctional epithelium to the implant surface, this fiber attachment would appear to be functional.

Most studies claiming histologic evidence of a collagenous attachment have been those examining porous or textured surface implants, especially HA and plasma-sprayed titanium. However, evidence of attachment to nontextured surface implants (Brånemark type) has also been reported. Much of the evidence is based on observations of collagen fibers approaching the implant at right angles, suggesting a "functional attachment." Connective tissue fibers have appeared to enter the porous surface, suggesting a mechanical attachment to the implant. A report by Schroeder et al provides excellent micrographic evidence of fibrous adherence to the surface of titanium plasma-sprayed implants. The strength of this "attachment" is undetermined. They asserted that separation of the implant from gingival connective tissue resulted in the avulsion of titanium particles from the implant surface. Albrektsson et al also presented scanning electron microscopic evidence of collagenous filaments traversing from bone to implant that appeared adherent to the surface of the implant (Brånemark): "The mechanism of attachment seemed to be the same as that for Sharpey's Fibers to bone, i.e., gluing by the amorphous coating formed by the ground substance." Reports of HA implants in dogs described a 2-mm supracrestal formation of mineralized tissue associated with the implant surface into which the gingival fibers appear to insert. Whether this phenomenon can occur in humans is unknown.

Some evidence may appear to indicate a fibrous attachment to a variety of implant surfaces and materials, but the frequency and importance of this event is still in question. The direction of collagen fibers associated with implants in many studies, especially those implants without a textured or porous surface, has been reported as parallel to the long axis of the implant.

Until recently, there has been great controversy as to whether the implant must be osseointegrated to be considered successful. Several authors still consider fibrous integration to represent the physiologic, if not structural, analog of the periodontal ligament at endosseous implants. Although some studies report success with fibrointegration, the contemporary view is more in favor of osseointegration as the
Based on this premise, the peri-implant ligament will not be discussed as a component of the soft tissue-implant junction.

**Clinical Significance**

A question often arises after evaluating differences and similarities between periodontal and peri-implant tissue attachments: Is there a greater susceptibility to plaque-induced disease in either the peri-implant or the periodontal tissue? Various answers have been suggested. One view is that the peri-implant tissues react to the plaque bacteria with resultant chronic inflammation the same as do periodontal tissues. In one study, both tissues reacted similarly but the peri-implant tissue had a higher percentage of inflammatory infiltrate with a higher number of plasma and mononuclear cells. Many authors have shown similar microflora around both healthy teeth and stable implants, and a similar pathogenic microflora around both periodontally diseased teeth and failing implants. One study that showed similar reactions to plaque between the periodontal and peri-implant tissue was by Berglundh et al. When the implants and teeth were exposed to plaque accumulation, an infiltrate progressed apically along with the apical spread of the subgingival microflora in both the peri-implant and periodontal tissues.

Another view is that the peri-implant tissues are more resistant to disease. In an animal investigation, monkeys were partially edentulated with subsequent implant placement. After healing, periodontal and peri-implant disease was induced by the use of ligatures, and subsequent bone loss was evaluated. The authors found more periodontal than peri-implant bone loss and concluded that there was greater resistance to disease by the implants. One explanation may be the lack of a cemental layer that could absorb the endotoxin produced by the microflora and thus decrease chronic inflammation.

The final viewpoint is that the peri-implant tissues are more susceptible to disease. One investigation that demonstrated this involved five beagle dogs that had been partially edentulated with subsequent implant placement. After healing, ligatures were placed around both implants and remaining teeth; they were removed after 6 weeks, and then 1 month later biopsy specimens were taken. Results from the clinical and histologic examination indicated more pronounced peri-implant tissue destruction. There was a larger peri-implant lesion, and the peri-implant lesions, unlike the periodontal lesions, extended into the bone marrow. The peri-implant bone loss averaged 3.2 mm versus 1.1 mm of periodontal bone loss. One possible explanation is that the lack of a cemental surface with inserting collagen fibers enabled a more rapid downgrowth of plaque at the implants. Also, the parallel fiber orientation in the peri-implant tissue may have favored a more rapid spread of the lesion; the progression of the lesion into the bone marrow may be an inability of the implant tissue to heal after subgingival infection. After consideration of the differences between the periodontal connective tissue attachment and the
peri-implant connective tissue cuff-like barrier, it would appear there is a basis for the premise that the peri-implant tissue is more susceptible to plaque-associated disease.

It is difficult, if not impossible, to completely transfer findings in the animal model to the human clinical environment. However, many basic wound-healing principles and the progression of disease factors were initially evaluated in the animal model and found to be similar in humans. Also, many other surgical principles were derived from the animal model and translated to humans. Therefore, it is prudent to take heed from these findings and to determine whether there are any preventive or interceptive therapies that could enhance implant longevity in the animal model that may be transferred to humans.

Clinical parameters for the peri-implant tissues will be similar to those that are monitored to detect tissue changes in the periodontal tissue resulting from plaque-induced inflammation. These include gingival changes in color, contour, and consistency; probing depths, including bleeding upon probing; and radiographic exposures to detect changes in the proximal bone levels. Future parameters may include chairside microbiologic monitoring. The criteria of a progressing loss of attachment that signifies the change from a sulcus to that of a pocket will be the same in both the periodontal and peri-implant tissues.

Conclusion
The peri-implant and periodontal soft tissues are histologically similar but not identical. The difference in the presence or absence of a connective tissue fiber attachment between the tooth and implant is critical and may alter the peri-implant tissue's susceptibility to disease. The question of whether the peri-implant tissues are as resistant to long-term breakdown must now be proven by longitudinal implant studies.

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**Fig. 1** Epithelial and perpendicular connective tissue fiber attachment at the soft tissue-tooth interface. Tooth (T), bone (B), epithelial attachment (E), connective tissue (C), and sulcular epithelium (S).
Fig. 2 Soft tissue-implant interface. Right inset is of nonperpendicular connective tissue fibers and left insets are of epithelial cells and hemidesmosomal attachments. Implant (IM), bone (S), connective tissue (c), epithelial attachment (E), sulcular epithelium (S), epithelial cells (EC), and hemidesmosomes (H).