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Peri-implant health

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Abstract

Objective: The aim is to define clinical and histologic characteristics of peri-implant tissues in health and describe the mucosa–implant interface.

Importance: An understanding of the characteristics of healthy peri-implant tissues facilitates the recognition of disease (i.e., departure from health).

Findings: The healthy peri-implant mucosa is, at the microscopic level, comprised of a core of connective tissue covered by either a keratinized (masticatory mucosa) or non-keratinized epithelium (lining mucosa). The peri-implant mucosa averages about 3 to 4 mm high, and presents with an epithelium (about 2 mm long) facing the implant surface. Small clusters of inflammatory cells are usually present in the connective tissue lateral to the barrier epithelium. Most of the intrabony part of the implant appears to be in contact with mineralized bone (about 60%), while the remaining portion faces bone marrow, vascular structures, or fibrous tissue. During healing following implant installation, bone modeling occurs that may result in some reduction of the marginal bone level.

Conclusions: The characteristics of the peri-implant tissues in health are properly identified in the literature, including tissue dimensions and composition. Deviation from the features of health may be used by the clinician (and researcher) to identify disease, including peri-implant mucositis and peri-implantitis.

KEYWORDS

connective tissue biology, diagnosis, implantology, osseointegration

Peri-implant tissues are those that occur around osseointegrated dental implants. They are divided into soft and hard tissue compartments. The soft tissue compartment is denoted "peri-implant mucosa" and is formed during the wound healing process that follows implant/abutment placement.[1] The hard tissue compartment forms a contact relationship to the implant surface to secure implant stability.[2] Due to their histologic and anatomic features, peri-implant tissues carry out two basic functions: the mucosa protects the underlining bone, while the bone supports the implant. Indeed, the destruction of peri-implant tissues can jeopardize the implant success and survival,[3] and the understanding of the characteristics of healthy peri-implant tissues allows the recognition of disease. Thus, the aim of the present review was to define clinical and histologic characteristics of peri-implant tissues in health and describe the mucosa–implant interface.

A search in MEDLINE-PubMed was used to retrieve the evidence to support the present review. The following key words were used for the literature search: dental implants (Mesh) AND biological width OR mucosa OR soft tissue OR attachment OR keratinized mucosa OR peri-implant mucosa OR probing depth OR microbiota OR collagen fibers OR epithelium OR adhesion OR seal OR bone OR osseointegration AND humans OR animals. The two main reasons for exclusion of studies were: 1) not published in English, and 2) lack of detailed clinical, histologic, or microbiologic description of healthy peri-implant tissues.

PERI-IMPLANT MUCOSA

Most information regarding the structural features of the peri-implant mucosa is derived from animal studies using dog models.[4–15] In such studies implants were placed in the edentulous ridge (alternatively, the fresh extraction socket), the outer osseous part of which was covered with masticatory mucosa. It was also shown that the healed peri-implant mucosa on the buccal aspect averaged about 3 to 4 mm high when measured from the mucosal margin to the crest of the peri-implant bone. In addition, this mucosa contains a core of connective tissue, mainly comprised of collagen fibers and matrix elements (85%), comparatively few fibroblasts (3%), and vascular units (5%). The outer (oral) surface of the connective tissue is covered by an often orthokeratinized epithelium. The portion of the peri-implant mucosa that is facing the implant (abutment) contains two distinct parts, a "coronal" portion that is lined by a thin barrier epithelium (similar to the junctional epithelium of the gingiva) and sulcular epithelium, and a more "apical" segment in which the connective tissue appears to be in direct contact with the implant surface. This apical portion of the peri-implant mucosa is designated zone of connective tissue adhesion.

In the connective tissue immediately lateral to the barrier and sulcular epithelium, a delicate plexus of vascular structures, similar to the dentogingival vascular plexus,[16] is consistently present,[17] while the connective tissue adhesion zone appears to harbor only limited amounts of vascular structures. At implants placed into masticatory mucosa, the main collagen fiber bundles are anchored in the crestal bone and extend in a marginal direction parallel to the surface of the metal device. It is assumed that circular fibers may also be present in this type of peri-implant mucosa.

Moon et al.[18] analyzed under electron scanning microscope the zone of connective tissue adhesion confined to a 200-μm wide zone of the connective tissue facing the implant. The findings demonstrated that the adhesion includes two distinct layers: one inner layer, about 40 μm wide, which harbors large amounts of fibroblasts (32% of volume) that appear to be in intimate contact with the surface of the implant; and one outer layer, about 160 μm wide, that is dominated by collagen fibers (83%), smaller amounts of fibroblasts (11%), and larger volumes of vascular structures (3%).[18]

Valid histologic information is not currently available regarding the peri-implant mucosa when implants are placed in non-keratinized lining or alveolar mucosa.

MORPHOGENESIS OF THE MUCOSAL ADHESION

The formation of the mucosal adhesion was studied in a dog model.[1] One-piece implant devices were placed in the edentulous mandible of dogs, and healing was monitored using light microscopic examination of biopsies sampled at different intervals during a 3-month period. In the initial phase of the wound between the implant and cut connective tissue, a fibrin clot/coagulum formed that was infiltrated with mainly neutrophils and limited amounts of macrophages. The number of inflammatory cells subsequently subsided, and the wound surface became characterized by its dense layer of fibroblasts that appeared to be in intimate contact with the implant surface. In the 2nd to 3rd week of healing, the density of fibroblasts was reduced, the amount of collagen and matrix components increased, and epithelial cells, extending from the oral epithelium, had started to occupy marginal parts of the connective tissue wound. Collagen fibers in the previous wound area became organized in bundles after about 4 weeks. After 6 to 8 weeks the mucosal adhesion appeared mature, and the interface zone at tissue–implant was comprised of a combined epithelial and connective tissue adhesion to the implant surface. Since the build-up of the soft tissue adhesion did not change much after the first month, it is suggested that a homeostasis had been reached at this interval.[1]

DIMENSION OF THE PERI-IMPLANT MUCOSA

Animal studies

The dimension of the peri-implant mucosa, often called the biological width or dimension,[5] was examined in biopsies mainly obtained from studies in dogs.[19–26] Such measurements disclosed that a certain width of soft tissue may be required to cover the peri-implant bone. The studies referred to the length of the epithelium (from the peri-implant mucosa margin to the apical portion of the junctional epithelial) as about 2 mm, while the height of the zone of connective tissue adhesion exhibited more variation (between 1 and 2 mm). The experiments in the animal model included the study of different variables such as material used for the fabrication of the implant and/or the abutment, surgical placement protocol, implants/abutments with different surface texture,[5,19–23] as well as so-called implants with a "platform switching" implant/abutment design.[24–26] The results obtained documented that while abutments made of gold alloy and dental porcelain failed to establish appropriate soft tissue adhesion,[23] other variables had apparently limited effect on the dimensions of the peri-implant mucosa.

It should be noted, however, that although animal models may provide valuable data valid for proof-of-principle issues, they may not completely recreate the anatomic, physiologic, biomechanical/functional, or pathologic environment of the clinical conditions in humans.[27]

Human studies

Studies on the morphogenesis and morphology of the mucosa at implants in humans used block biopsies obtained from mini-implants or from soft tissue dissection techniques from conventional or specially designed abutments.[22,28–32] Tomasi et al.[31,32] presented a *de novo* biopsy technique and reported on the morphogenesis of the peri-implant mucosa at single implant sites in human volunteers. Soft tissue biopsies were sampled after 2, 4, 8, and 12 weeks of healing

following abutment connection. They reported that after 2 weeks large areas of the severed connective tissue were infiltrated with inflammatory cells, while after 4 weeks the infiltrated areas were smaller and a short barrier epithelium had formed in the interface zone. Sections representing later phases of observation exhibited continued healing of the connective tissue wound and the formation of a well-defined barrier and sulcular epithelium in the marginal portion of the soft tissue samples. The height of the peri-implant mucosa, measured along the profile of the soft tissue, increased during the healing phase from 2.7 mm at 2 weeks to between 3.0 and 3.5 mm after 4, 8, and 12 weeks. In the corresponding intervals the length of the epithelium varied between 2.2 and 2.0 mm, while the zone of connective tissue adhesion varied between 1.7 and 1.1 mm.

In summary, results from the available studies in man and from animal experiments are consistent and document that the peri-implant mucosa is about 3 to 4 mm high with an epithelium that is about 2 mm long.

PERI-IMPLANT TISSUES IN CLINICAL HEALTH

The gingiva and the peri-implant mucosa and their adhesion (seal) are consistently challenged by the oral environment, including the steady exposure to microorganisms in the biofilm present on the tooth and implant surfaces.[22,32–37] In the clinically normal peri-implant mucosa (and gingiva), the continuous host response includes both vascular and cellular events. Thus, distinct vascular structures occur in the connective tissue lateral to the epithelium, as well as small clusters of inflammatory cells (T- lymphocyte and B-lymphocyte). Macrophages seem to be present along the entire interface zone, while polymorphonuclear leukocytes occur mainly in the connective tissue immediately lateral to the epithelium.[32]

PROBING PERI-IMPLANT TISSUES

For many years it was incorrectly assumed that the tip of the periodontal probe in a probing depth (PD) measurement identified the apical base of the dento-gingival epithelium.[38] Later research documented, however, that this was not the case. At healthy sites the tip of the probe failed to reach the apical portion of the epithelial barrier, while at diseased sites the probe found the apical base of the inflammatory cell infiltrate. Hence, PD measurements assess the depth of probe penetration or the resistance offered by the soft tissue.[39–47]

The influence of the condition (health, disease) of the peri-implant mucosa on the outcome of the probing measurement was studied in animal models.[48–50] Lang et al.[49] reported that at sites with healthy mucosa or mucositis, the tip of the probe identified the apical border of the barrier epithelium with an error of approximately 0.2 mm, while at sites with peri-implantitis, the measurement error was much greater at 1.5 mm. Abrahamsson and Soldini,[50] in a subsequent study, stated that the probe penetration into the healthy soft tissues at the buccal surface of teeth and implants in dogs was alike and similar to the length of the junctional/barrier epithelium. It was assumed that probing the implant–mucosa interface would sever the soft tissue seal and jeopardize the integrity of the adhesion. This issue was examined in a dog study[51] that documented that already after 5 to 7 days following clinical probing, the soft tissue seal had regenerated to its full extent.

BONE SOUNDING

Bone sounding or transmucosal sounding (TS) is a measurement that is used to determine the height of the entire soft tissue cuff at various groups of teeth and implants. The dimensions of the peri-implant mucosa and the gingiva at adjacent tooth sites was studied by clinical measurements performed mainly in partially edentulous subjects who had been treated with implant-supported single-crown restorations. In such studies the brand of the periodontal probe used for the assessments was identified; PD as well as TS measurements were used to describe some features of the soft tissue.

Results from such studies[52–60] demonstrated that the PD was greater at proximal than at facial/buccal surfaces at both tooth and implant sites and greater at implant than at tooth sites. This shows that the soft tissue cuff around implants exhibits less resistance to probing than the gingiva at adjacent teeth. There are reasons to suggest that the lack of root cementum on the implant surface as well as the difference in the orientation of the collagen fibers in the two types of soft tissue may be associated with the variation observed in the "resistance to probing."

The TS measurements disclosed that the peri-implant mucosa was in most cases 1.0 to 1.5 mm higher than the corresponding gingiva at both buccal/facial and proximal sites. It was further demonstrated that patients with a "flat-thick" periodontal phenotype[61,62] exhibited greater peri-implant mucosa dimensions than subjects that belonged to the "scalloped-thin" biotype.[57,63] In addition, the height of the papilla between an implant-supported restoration and a natural tooth was reported to be ≤5 mm[52,56,64,65] and related to the connective tissue adhesion level at the adjacent approximal tooth surfaces.[57,66] The corresponding dimension between two adjacent implant restorations averaged 3 mm[64,67] and apparently was dependent on the outline of the crest of the supporting bone.

KERATINIZED MUCOSA (KM)

KM is a term used to describe the masticatory mucosa that is present at many, but not all, implant sites. KM extends from the margin of the peri-implant mucosa to the movable lining (oral) mucosa. KM is comprised of a lamina propria (fibrous connective tissue that contains fibroblasts and equal amounts of type I and type III collagen) that is covered by an orthokeratinized squamous epithelium. The width of the KM at the facial/buccal side of teeth is, as a rule, about 1 mm

greater than at contralateral implant sites.[54,59,60] It is suggested that loss of crestal bone following tooth extraction is the main reason for dimunition of the KM. The thickness of facial KM, determined with a probe at the base of the PD, is greater at implants than at teeth (2.0 mm vs 1.1 mm, respectively).[54]

The need for a minimum amount of keratinized mucosa to maintain peri-implant tissue health is apparently a controversial issue.[68–72] Several studies failed to associate the lack of a minimum amount of KM with mucosal inflammation,[73–80] while other studies suggested that plaque build-up and marginal inflammation were more frequent at implant sites with < 2 mm of KM.[81–85]

BONE TISSUE AROUND IMPLANTS

Bone tissue in the edentulous ridge

In a study involving partially edentulous subjects, hard tissue biopsies were sampled from the maxilla and the mandible with the use of trephine drills.[86] The bone tissue was found to include a blend of mainly lamellar bone (46%) and bone marrow (23%) with less amounts of fibrous (12%) and osteoid (4%) tissue. Bone marrow was the dominant tissue element in the anterior maxilla, while dense lamellar bone characterized the anterior portion of the mandible. The cortical cap was consistently comprised of lamellar bone and was wider in the mandible than in the maxilla (1.8 mm vs 0.8 mm, respectively) and substantially more narrow in the anterior maxilla than in the anterior mandible.

Osseointegration

The term osseointegration was coined by Brånemark et al.[87] and was described as bone-to-implant contact on the light microscopic level. Later, Albrektsson and Sennerby[2] defined osseointegration as, "a direct functional and structural connection between living bone and the surface of a load-carrying implant."

In animal experiments[88,89] the process of hard tissue healing around implants made of c.p.titanium was described. The individual device had the shape of a solid screw with a modified surface configuration and U-shaped invaginations (wound chambers) that allowed the ingrowth of bone. The wound chambers were first occupied with a coagulum that after 4 days had been replaced with granulation tissue that contained inflammatory cells and also numerous mesenchymal cells and newly formed vessels. After about 1 week of healing, fingerlike projections of woven bone occurred around vascular structures in the center of the chambers and also in direct contact with small areas of the implant. After 2 to 4 weeks the chambers were filled with woven bone extending from the old bone to reach the surface of the titanium device. In the 6- to 12-week interval the woven bone was replaced with lamellar bone and marrow and bone-to-implant contact had been established. At the end of the experiment about 60% of the moderately rough implant surface was occupied with mineralized bone and the marginal bone-to-implant contact was located about 0.3 mm from the abutment/implant level. Additional preclinical studies[90,91] have confirmed that rough surfaces enhance early bone formation and bone-to-implant contact. Findings from studies in man[92–97] confirmed the animal results by documenting that the amount of direct bone (mineralized tissue)-to-implant contact was about 60% of the circumference of the implanted device after a healing period of 6 weeks to 3 months.

Crestal bone-level change

Following implant installation and loading, modeling of the bone occurs, and during this process some crestal bone height is lost. Studies in animals have demonstrated the location of the implant–abutment interface (microgap) determines the amount of this initial marginal bone loss.[26,98–100] Thus, the crestal bone reduction that occurs in this healing phase apparently varies between brands and seems to be related to the design of the implant system used.[101–112] After this initial period about 75% of implants experience no additional bone loss but osseointegration takes place. Most implant sites that exhibit crestal bone loss of > 1 mm appear to be associated with soft tissue inflammation although some sites may have an apparently healthy peri-implant mucosa.[3]

MAJOR DIFFERENCES BETWEEN HEALTHY PERI-IMPLANT AND PERIODONTAL TISSUES

The implant device lacks tooth characteristic structures such as root cementum, periodontal ligament, and bundle bone (alveolar bone proper).[113] The dento-alveolar and the dento-gingival fiber bundles connect the soft tissues with the tooth (root cementum), while no such fiber bundles are apparent in the peri-implant tissues. At periodontally healthy sites, the margin of the gingiva follows the outline of the cemento-enamel junction, while at a corresponding implant site the mucosal margin follows the contour of the crestal bone (multiple implants) or relates to the connective tissue adhesion at adjacent teeth (single implants). The tooth is mobile within its socket, while the implant is rigidly anchored (ankylosed) to the surrounding host bone.

CONCLUSIONS

The healthy peri-implant mucosa is comprised of a core of connective tissue covered by either a keratinized or non-keratinized epithelium. Most of the intrabony part of the implant is in contact with mineralized bone, while the remaining portion faces bone marrow, vascular structures, or fibrous tissue. The characteristics of peri-implant tissues in health are properly identified in the literature. According to the available definitions[114] of peri-implant mucositis and peri-implantitis, the absence of signs of clinical inflammation is necessary for concluding that a site has peri-implant health.

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The authors report no conflicts of interest related to this review paper.

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