Biologic Width around Dental Implants: An Updated Review

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Abstract

Soft tissue-implant interface is an important anatomical feature contributing to the long-term success of dental implants. Based on the available evidence, different factors may influence biological width around implants including the surgical technique, implant loading, implant surface properties, abutment materials, implant position, and width of the peri-implant mucosa. The purpose of the present review was critical evaluation of the available data, regarding the factors that may influence the biologic width around implants and their subsequent effect on clinical performance of implants. Available literature on this subject published primarily in English from 1921 to 2014, was found by searching several electronic databases and by hand searching relevant journals as well. Totally, 70 relevant articles were selected for this narrative review. The structure of peri-implant mucosa has many similarities, as well as differences with its periodontal counterpart. Most studies report larger values for peri-implant biologic width compared to that of natural teeth. This literature review yielded contradictory data regarding the dimensions of the biologic width when different influential factors were taken into account.

Keywords: Biologic width, Connective tissue, Dental implants, Junctional epithelium, Oral mucosa.

Historical perspectives

The “epithelial attachment” around teeth was first described in 1921 by Gottlieb (1). The “gingival crevice” or sulcus was later defined(2), followed by description of the connective tissue as three-dimensionally oriented fibers firmly connecting tooth structures to the adjacent gingiva (3). Marfino, Orban and Wentz (4), were the first to demonstrate that the attachment of gingiva to tooth is composed of gingival connective tissue attachment and junctional epithelium. In 1959, Sicher investigated the morphology of epithelial and connective tissue attachments to the teeth, described as the dentogingival junction (5). In 1961, Gargiulo et al. (6) quantified the vertical components of this structure in human cadavers and coined the term
“biologic width”. Biologic width is normally composed of 0.97mm junctional epithelium (JE) and 1.07mm connective tissue attachment (CTA). Accordingly, the biologic width is acknowledged 2.04 mm, reflecting the sum of the epithelial and connective tissue measurements. In addition, sulcus depth (SD) was normally observed to be 0.69mm. These findings were substantiated by Vacek (7). After detailed assessment of 171 cadaver tooth surfaces, the mean measurements for sulcus depth, epithelial attachment and connective tissue attachment were found to be 1.34 mm, 1.14, and 0.77 mm, respectively. Vacek also realized that the connective tissue attachment was the most stable measurement, with the least degree of variance. On the other hand, significant variations were observed in epithelial attachment ranging from 1.0 mm to 9.0 mm.

Peri-implant tissues have many similarities, as well as some anatomical differences with periodontal attachment apparatus. The differences include lack of a periodontal ligament around implants, different orientation of connective tissue fibers and vascular distribution (8). Peri-implant biologic width has been investigated and measured in histological animal studies as well as clinical human studies. The purpose of this review was to draw comparisons and contrasts between biologic width around implants and biologic width around teeth and evaluate factors that may influence the peri-implant biologic width.

Structure and biological dimensions

Listgarten et al. (9) in a comprehensive review article stated that biologic width around implants is composed of three distinct zones: sulcular epithelium, junctional epithelium, and connective tissue. Junctional epithelium around implants is derived from the oral epithelium, while the junctional epithelium around teeth originates from the reduced enamel epithelium (10); however, the structures appear morphologically similar (11-14).

Junctional epithelium facing the implant or abutment surfaces is thin in its apical portion (40µm mean width), consisting of only a few cell layers (stratum basale and stratum granulosum) (15). The first animal studies by Berglundh et al. (8) confirmed that the peri-implant mucosa established a cuff-like barrier adhering to the surface of the titanium abutment. The peri-implant mucosa, similar to gingiva, has a well-keratinized oral epithelium that is contiguous with the junctional epithelium that faces the titanium surface. The structure of the peri-implant junctional epithelium is similar to that of natural dentition, with the exception that it is shorter and thinner (8, 15–19).

Berglundh et al. (18) in a canine study of non-submerged mandibular implants reported that epithelial proliferation begins around 1–2 weeks post-operatively, with a mature epithelial barrier establishing after 6–8 weeks. Fibroblasts are the dominant cell type at the connective tissue/implant interface at two weeks post-operatively, but their density decreases by week 4. After 4–6 weeks of healing, the collagen fibers are well organized. Hence, it was concluded that 6-8 weeks is required for the formation of a mature soft tissue attachment following surgery.

The mode of attachment of junctional epithelium to the implant surface has been demonstrated to be similar to that of teeth, which is by means of a basal lamina and hemidesmosomes (20). These findings have been verified both in vitro (23), and in vivo in rodents (27), canines (12, 15), non-human primates (21, 22) and humans (24, 25, 26). Contradictory findings were reported in an ultra-structural study by Shioya et al, who failed to observe hemidesmosomes and basal lamina adjacent to the implant surfaces (28).

A number of studies have investigated the composition of peri-implant tissues. Berglundh et al. (8) using an experimental model, observed collagen fibers to dominate peri-implant connective tissue, with fewer fibroblasts and vascular structures than normally seen in the gingiva around teeth. Importantly, collagen fibers were arranged parallel to the titanium surface in contrast to the orientation of gingival fibers, which tended to be arranged perpendicular to the cementum surface of the tooth root. Elsewhere in the marginal gingiva other fiber groups were arranged in a variety of different patterns (8, 29). In a recent study, Shioya et al. (28) reported a zone of dense collagen fibers, surrounded by loose connective tissue, consisting of a 3-dimensional network of collagen fibers running in different directions. A number of animal and human histologic studies have indicated that peri-implant collagen fiber bundles, while arranged in varying directions, are functionally oriented (30, 31). In contrast, Schierano et al. (32) in a human histologic study of nine retrieved abutments from seven patients demonstrated primarily horizontally and vertically directed connective tissue fibers around implants.

The nature of the collagen fiber contacted with implant surfaces, as well as the anatomic details of peri-implant tissues have been studied by a number of investigators. Buser et al. (11) noted that connective tissue fibers are in direct contact with the implant surface although without true attachments. The direct connective tissue contacted to the implant surface was approximately 50 to 100 µm wide, consisting of dense, avascular circular fibers. In the adjacent outer zone, the connective tissue appeared less dense, with horizontal and vertical collagen fibers and a large number of blood vessels. Berglundh et al. (33) indicated that the zone of connective tissue near the junctional epithelium had a
number of blood vessels, but the vessels were smaller in
diameter and sparser than those found around teeth.

In a dog model, Moon et al. (19) demonstrated the
presence of only a few blood vessels in peri-implant
tissues, while noting numerous fibroblasts orientated
with their long axes parallel to the implant surface
(Astra Tech Implants). This attachment tissue was
composed of approximately 80% collagen, 13% fibroblasts, 3% blood vessels and 3% residual tissue,
resembling scar tissue. Lateral to this region, fewer
fibroblasts with more collagen fibers and more vascular
structures were observed which were divided into two
zones: the inner avascular zone (0-40 μm) with more
fibroblasts, and the outer zone (40–200 μm) with dense
collagen and substantial numbers of vascular structures.
It appears from these and other similar findings that the
connective tissue attachment between titanium surfaces
and connective tissue is established and maintained by
fibroblasts.

The peri-implant junctional epithelium appears
similar to that found in the natural dentition. However,
since there is no peri-implant cementum layer, most
supracrestal connective-tissue fibers are oriented in
parallel alignment to the implant surface. The presence
of an avascular zone (50 to 100 μm) of dense,
supracrestal circular connective-tissue fibers that are in
direct contact with the implant surface has been
confirmed through histologic examination (34).

Berghlund et al. (33) compared the vascular system
of the periodontal and peri-implant tissues in beagle
dogs. The vascular supply to the gingiva originates from
two different sources, large supraperiosteal blood
to the gingiva originates from
two different sources, large supraperiosteal blood
to the gingiva originates from
two different sources, large supraperiosteal blood
to the gingiva originates from
two different sources, large supraperiosteal blood
vessels and the vascular plexus of the periodontal
ligament. In contrast, the vascular system of the peri-
implant mucosa of dogs appears to be derived solely
from large supra-periosteal blood vessels lateral to the
alveolar ridge. Interestingly though, scanning electron
microscopic study in rats by Selliseth et al. (35)
revealed that capillary loops in the connective tissue
under the peri-implant junctional and sulcular
epithelium appear to be anatomically similar to those
found in normal periodontium.

Factors influencing peri-implant biologic
width

a) Surface topography

Albrektsson and Wennerberg (36) have classified
surface topography of implants into three general
categories according to mean roughness (SA). The
lowest degree of surface roughness is minimally rough
with SA values of 0.5-1 μm. Moderately rough implants
have SA of 1-2μm and rough ones have Sa greater than
2 μm. Buser et al. (11) investigated the soft tissue
dimensions around three different titanium surfaces,
Table 1: Studies about the effect of implant and abutment materials on peri-implant biologic width.

<table>
<thead>
<tr>
<th>Study</th>
<th>Model</th>
<th>Implant/abutment surface</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cochran et al (2014)</td>
<td>Dogs</td>
<td>Loaded implants with machined and roughened (SLActive) collars</td>
<td>The connective tissue contact were not significantly affected by the type of implants; but that the junctional epithelium and biologic width dimensions were larger around the implants with the machined collars. The amount of inflammation was not different between the two implant types. Slightly more bone formation and more mature collagen formation were detected around the implants with the roughened collars compared to the implants with machined collars.</td>
</tr>
<tr>
<td>Nevin et al (2010)</td>
<td>Dogs</td>
<td>Machined, laser microchannel surface abutments</td>
<td>compared with machine surfaces, the presence of a 0.7 mm laser ablated micro-channeled zone was associated with increased fibroblastic activity on the abutment-grooved surface, resulting in a denser interlacing complex of connective tissue fibers oriented perpendicular to the abutment surface.</td>
</tr>
<tr>
<td>Welander et al (2008)</td>
<td>Dogs</td>
<td>Ti, ZrO2, Au/Pt-alloy abutments</td>
<td>At Au/Pt-alloy abutment sites in comparison with Ti and ZrO2 : 1-apical migration of the barrier epithelium along with marginal bone loss occurred between the second to fifth months of healing. 2-the connective tissue zone (~80 µm wide) contained less collagen and fewer fibroblasts and larger fractions of leukocytes. 3-Soft tissue healing appeared to be less stable.</td>
</tr>
<tr>
<td>Weiner et al (2008)</td>
<td>Dogs</td>
<td>Laser micro textured, machined collars</td>
<td>The controls had more soft tissue down-growth, greater osteoclastic activity, and increased saucerization compared with sites near the laser micro textured experimental implants.</td>
</tr>
<tr>
<td>Abrahamsson et al (2007)</td>
<td>Dogs</td>
<td>CPT or gold alloy implants with Four different combinations of metal in coronal, central and apical zones (Ti/Ti/Ti, Ti/Au/Au, Au/Au/Au, Au/Ti/Ti).</td>
<td>No peri-implant marginal soft tissue dimensional differences between any of the Ti or Au designed implants.</td>
</tr>
<tr>
<td>Schwarz F et al (2007)</td>
<td>Dogs</td>
<td>SLA, modSLA implants</td>
<td>The formation of well-organized collagen fibers and abundant blood vessels in a newly formed loose connective tissue lateral to modSLA implants. While some fibers were oriented in a parallel alignment, others were extended and attached perpendicularly to the implant surface. In contrast, SLA implants appeared to be associated with a dense connective tissue area with parallel-running collagen fibers and sparse blood vessels.</td>
</tr>
<tr>
<td>Glauser et al (2005)</td>
<td>Human</td>
<td>One-piece mini-implants made of CPT with either oxidized, acid-etched, or machined surfaces</td>
<td>A biologic width is approximately 4.0 to 4.5 mm, consisting of an epithelial and a supracrestal connective tissue barrier around the experimental one-piece mini-implants that was similar to that described in animal studies. The oxidized and acid-etched implants experienced less epithelial down-growth and longer connective tissue barriers than machined implants.</td>
</tr>
<tr>
<td>Kohal et al (2004)</td>
<td>Monkeys</td>
<td>Zirconia and titanium abutments</td>
<td>9 months after implant placement, no significant differences between the responses to the two abutment materials.</td>
</tr>
<tr>
<td>Abrahamsson et al (2002)</td>
<td>Dogs</td>
<td>Dual thermal acid-etched surface, turned surface abutments</td>
<td>After 6 months of healing, the peri-implant mucosal attachment of the two types of abutments was similar in both linear dimensions and connective tissue composition.</td>
</tr>
<tr>
<td>Abrahamsson et al (1998)</td>
<td>Dogs</td>
<td>CPT, highly sintered Al2O3, gold and porcelain fused to gold abutments</td>
<td>After 6 months of healing, at CPT or Al2O3 abutment sites, a mucosal attachment had been formed consisting of an epithelial and a connective tissue portion that were approximately 2 mm and 1–1.5 mm in height, respectively. Abutments made of gold alloy did not allow the formation of a proper soft tissue abutment attachment, resulting in soft tissue marginal recession and bone resorption.</td>
</tr>
</tbody>
</table>

CPT = commercially pure titanium; SLA = sandblasted, large grit and acid-etched; mod = modified; TEM = transmission electron microscopic
c) Surgical protocol

A number of studies have examined the potential role of surgical protocol on peri-implant soft tissue healing. The effect of one- versus two-stage protocol on soft tissue healing of three different implant systems (Astra Tech Implants, Bränemark and Bonefit-ITI) was investigated and compared.15 The histologic results demonstrated similar dimension and composition of epithelial and connective tissue components of biologic width with 1- or 2-stage procedures for all three implant systems. Similar findings have been reported in canine studies (16, 49-51). The current consensus appears to suggest that surgical protocol, especially one- versus two-stage procedures, has little effect on peri-implant soft tissue healing.

d) Loading time

A number of investigators has examined the effects of loading protocols on biologic width. Cochran et al. (52) evaluated biologic width dimensions around non-submerged loaded and non-loaded implants testing two different surfaces (SLA and TPS) in a canine model. Biologic width dimensions for the unloaded implants after three months of healing were 0.49 mm for sulcus depth, 1.16 mm for junctional epithelium, and 1.36 mm for connective tissue. The corresponding measurements in the loaded group were 0.5 mm, 1.44 mm, and 1.01 mm respectively. Results were similar after 12 months of loading, confirming that the biologic width around implants dimensionally resembles biologic width around teeth. In addition, the dimensions of its constituents appear to be independent of loading time. These results have also been confirmed by Pontes et al. (53) (Conexao System), demonstrating that loading times had no influence on soft tissue healing. Similar findings were confirmed by Hermann et al. (51) who compared non-loaded with loaded implants (ITI Implant System) and submerged with non-submerged healing at different time intervals. Histometric measurements revealed significant changes within individual tissue compartments (SD, JE, CTC) over time. However, over a 15-month healing period, the total biologic width remained constant. According to Hermann et al, no statistically significant differences were detected among groups during the study period. Similar results were also reported by Siar et al. (54) comparing immediate versus delayed implant loading at 18 sites in six monkeys after three months of follow-up. The overall mean value of the biologic width was 3.9 mm in the immediate group and 3.8 mm in the delayed group. The authors concluded that there were no statistically significant differences in the dimensions and compositions between the two groups.

In a study by Bakaeen et al. (55) the dimensions of peri-implant soft tissues around immediately- and early-loaded one-piece implants were compared with those of conventionally loaded one-piece implants. Forty-eight titanium sandblasted/acid-etched (SLA) implants were placed in four foxhounds. The implants were placed 3 months (group A), 21 days (group B), ten days (group C), and two days (group D) before restoration. Histometric analysis included dimensional measurements of the sulcus depth, junctional epithelium, the connective tissue seal, and gingival recession. There were no statistically significant differences among the four groups.

Finally, in a systematic review of marginal soft tissue around implants subjected to immediate loading or immediate restoration, Glauser et al. (56) reported the occurrence of soft tissue healing comparable to that in conventionally loaded implants. Therefore, available evidence suggests that the loading time has little effect on the biologic width.

e) Implant macro-design and microgap position

The effects of implant macro-design, especially one- versus two-piece, and the position of the microgap on the fate of biologic width have been widely studied. A variety of implant designs are available, including one-piece implants with contiguous endosseous, transmucosal and abutment segments, and two-piece implants with separate endosseous and abutment segments. Among two-piece implants, some are at bone-level, i.e. the endosseous portion ends at bone crest and the transmucosal portion joins the abutment. Alternatively, the transmucosal portion can be contiguous with the endosseous segment. At each of these designs, the microgap between the implant and abutment is positioned at different levels. In bone level positioned implants, the microgap is potentially near the bone crest, whereas in transmucosal fixtures the microgap is above the bone level. Findings of related studies are summarized in (table 2) (15, 49, 53, 57-69).

Results of mentioned studies in (table 2), seem to indicate that the dimensions and composition of the biologic width are not significantly influenced by the type of implant (i.e. one- versus two-piece implants) or the surgical protocol (i.e. one- versus two-stage). Limited evidence suggests, however that more deeply placed implants lead to a longer biologic width.
Table 2. Studies about the effect of implant macro-design and microgap position on peri-implant biologic width

<table>
<thead>
<tr>
<th>Study</th>
<th>Model</th>
<th>Implant macro-design and microgap position</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alves et al (2015)</td>
<td>Dogs</td>
<td>Platform switching abutment at crestal bone level 1) in the test group, all prosthetic procedures were carried out direct to multi-base abutment without disconnecting it 2) in the control group, the multi-base abutment was connected/ disconnected five times during prosthetic procedures</td>
<td>S-BIC distances both lingually and buccally between test and control groups was similar. Only buccal aBE-BC parameter presented statistically significant differences between test and control groups. Test group presented 0.57 mm less recession than control groups, being this difference statistically significant between the two groups.</td>
</tr>
<tr>
<td>Judgar et al (2014)</td>
<td>Human</td>
<td>Unloaded one- and two-piece implants</td>
<td>After 4 months of healing, marginal bone loss, gaps, and fibrous tissue were not detected around two types of implants. The biologic width dimension ranged between 2.55 ± 0.16 and 3.26 ± 0.15 to one- and two-piece implants, respectively. This difference was influenced by the connective tissue attachment, while the dimensions of sulcus depth and junctional epithelium were similar between two groups.</td>
</tr>
<tr>
<td>Farronato et al (2012)</td>
<td>Pigs</td>
<td>Test: three implants, each with a 0.25 mm implant/abutment mismatch, were placed either flush with (group1), 1 mm below (group2), or 1 mm above the bone crest (group3). Control: one conventionally restored implant without platform switching was placed at the bone level. The implants were randomly inserted flapless into the mandible.</td>
<td>Control implants presented a mean BW of 3.20mm (±0.33), with a CTA of 1.29 mm (±0.53) and a JE of 1.91mm (±0.71). Differences between the groups were related mainly to the length of the JE. The JE was significantly longer in the control sites (1.91 mm) than in the test groups (0.84 mm). However, no other differences among the groups were detected. If the implants are positioned at the level of the alveolar crest, the platform-switching technique may have a minor effect on the length of the JE (0.84 vs. 1.91 mm), while the CTA remains relatively constant.</td>
</tr>
<tr>
<td>Baffone et al (2012)</td>
<td>Dogs</td>
<td>Implants with matching and 0.85mm non-matching abutments</td>
<td>When abutments are mismatched to be smaller than the implant, more coronal levels of bone-to-implant contact are obtained and the BW reduces.</td>
</tr>
<tr>
<td>Canullo et al (2011)</td>
<td>Human</td>
<td>Implants with matching and non-matching abutments ranging from 0.25 to 0.85mm</td>
<td>No significant differences between groups in ICT, MVD and collagen content. Forty-eight months after restoration, platform switching and traditional platform implants had similar histological peri-implant soft tissue profiles.</td>
</tr>
<tr>
<td>Welander et al (2009)</td>
<td>dogs</td>
<td>Two-part implants (Osseospeed) 2 mm apical to the ridge crest In the test implants, abutment surface modifications (TiOblast) extended to the implant margin, including the shoulder portion of the implant. Regular abutments with a turned surface (Zebrat) were connected to the control implants</td>
<td>Four months later, histometric results showed similar dimensions of the JE and CTA for both the test and control sites. The CTA of the peri-implant mucosa that was facing the test abutments, however, contained a greater density of collagen and a smaller proportion of fibroblasts than seen at the control sites.</td>
</tr>
<tr>
<td>Pontes et al (2008)</td>
<td>dogs</td>
<td>at crestal bone level 1 mm below the crestal bone 2 mm below the crestal bone</td>
<td>The dimensions of CTA were larger when implants were placed subcrestally. In contrast, JE dimensions were not significantly affected by the position of the implant relative to the bone crest.</td>
</tr>
</tbody>
</table>
**Luongo et al. (2008)** Implants with non-matched narrower abutments The inflammatory connective tissue infiltrate initiated at the microgap located approximately 0.35 mm apical and coronal to the implant/abutment interface

**Lazzara and Porter (2006)** Implants with matching and non-matching abutments Implants with non-matching abutments (i.e. the implant platform was wider than the abutment) showed limited marginal bone resorption, when compared with implants restored by matching abutments.

**Tenenbaum et al. (2003)** Ankylos gap-free implant system, after abutment placement without functional loading and without plaque control The connective tissue between the most apical junctional epithelial cells and the alveolar crest to be characterized by collagen fibers running from the periosteum and the alveolar crest towards the oral epithelium, and, in front of the cone-shaped abutment, by a narrow zone of extracellular matrix containing a few collagen fibers. Compared with results obtained by other studies using different implant types (Astra, Bränemark, ITI), the Ankylos implant demonstrated greater length and width of connective tissue contact as well as a shorter JE.

**Abrahamsson et al. (2003)** The two healing abutments were removed 2 weeks later, and one Uni-abutment and one prepable abutment were placed. A single abutment reconnection proved to induce no marginal bone remodeling (Astra Tech Implant System) resulting in a transmucosal attachment of adequate quality and dimensions.

**Todescan et al. (2002)** At crestal bone level 1mm below the crestal bone 1mm above the crestal bone The dimensions of CTA were larger when implants were placed subcrestally. In contrast, JE dimensions were not significantly affected by the position of the implant relative to the bone crest.

**Herman et al. (2001)** One/two-piece and submerged/nonsubmerged unloaded titanium implants No statistically significant differences in the soft tissue dimensions were observed when comparing two-piece implants placed with submerged technique to those placed in a non-submerged approach, although the mucosal margin was located more coronally for one-piece compared to two-piece titanium implants.

| Dogs | One-piece (Bonefit) / two-piece (Astra Tech and Brånemark) implants | After 9 months follow-up, the BW for the non-submerged group was 3.50 mm, and 3.11 to 3.42 mm for the submerged group. The histological results demonstrated similar epithelial and connective tissue dimensions and composition. |

**Abrahamsson et al. (1997)** The dis- and subsequent reconnections of the abutment component of the implant (Brånemark System). The contralateral abutment remained undisturbed. Abutment manipulation jeopardized the mucosal barrier and induced an apical shift of the CTA. Therefore, while normal dimensions of the hard and soft tissues were observed in the control group, the abutment manipulation at test sites resulted in apical migration of the soft tissue barrier, leading to a mean marginal bone loss of 1.5 mm

**Abrahamsson et al. (1996)** The presence of inflammatory connective tissue infiltrate approximately 0.75 mm apical and coronal to the implant/abutment interface.

**Ericsson et al. (1996)** Implants with non-matched narrower abutments

S-BIC = distance from multibase abutment shoulder to the first bone implant contact; aBE-BC = distance from the apical end of the barrier epithelium to the first bone implant contact; BW = biologic width; JE = junctional epithelium; CTA = connective tissue attachment; ICT = inflammatory connective tissue infiltrate area; MVD = microvascular density.
Immediate Implant Placement Following Tooth Extraction

Buccal versus lingual aspects

Immediate implant placement following tooth extraction is now an accepted treatment protocol. Although most studies examining peri-implant biologic width focus on delayed implant placement, a number of studies have examined the nature of the biologic width following immediate implant insertion post tooth removal. These findings are summarized in (table 3) (8, 70-74).

Table 3. Studies about the effect of immediate versus delayed Implant Placement (Buccal versus lingual aspects) on peri-implant biologic width.

<table>
<thead>
<tr>
<th>Study</th>
<th>Model</th>
<th>Implant system</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vignoletti et al (2009)</td>
<td>Dogs</td>
<td>Four different implant systems immediately in fresh extraction sockets (3i, Astra Tech, Thommen, ITI Implant Systems)</td>
<td>The BW, 6 weeks after immediate implant placement averaged between 3.5-4.1 mm and 2.8-3.2 mm at the buccal and lingual aspects, respectively. On the buccal aspect, the JE and the CTA dimensions measured between 2.0 - 2.7 mm and 1.0 -1.8 mm, respectively. The corresponding lingual values were 1.6 – 2.0 mm and 0.9 - 1.4 mm respectively. There is no difference in soft tissue healing when comparing the four different implant systems. The JE length in all four systems was longer than the reported value for delayed implant sites.</td>
</tr>
<tr>
<td>Araújo et al (2006)</td>
<td>Dogs</td>
<td>Immediate implant placement (Straumann Implant System)</td>
<td>At one month, BW dimensions varied from 3.3 mm at buccal to 3.5 mm at lingual surfaces. At 3 months post immediate implant placement, substantial BW dimensional changes had occurred (4.2 ± 0.8 mm at buccal and 2.7 ± 0.2 mm at lingual). Most of the dimensional changes were secondary to changes in the CTA of the BW (in this case 1.9 ± 0.6 mm at the buccal and 0.6 ± 0.2 mm at the lingual surfaces).</td>
</tr>
<tr>
<td>Araújo et al (2005)</td>
<td>Dogs</td>
<td>Straumann Implant System into the distal sockets of third and fourth mandibular premolars</td>
<td>The composition and dimensions of peri-implant soft tissue following immediate placement of implants were identical to those found following delayed implant insertion.</td>
</tr>
<tr>
<td>Rimondini et al (2005)</td>
<td>Mini-pigs</td>
<td>Immediate implant placement (3i osseotite Implant System)</td>
<td>After 2 months of healing, the BW dimensions were found to be larger in immediately inserted implants following tooth extraction.</td>
</tr>
<tr>
<td>Schultes et al (2001)</td>
<td>Dogs</td>
<td>Delayed or immediate implant placement (SIS Implant System)</td>
<td>After 2 months of healing, the BW dimensions were found to be larger in immediately inserted implants following tooth extraction.</td>
</tr>
<tr>
<td>Berglundh et al (1991)</td>
<td>Dogs</td>
<td>Traditional, non-immediately, two-stage implants (Bränemark Implant System)</td>
<td>After 3 months of healing, the buccal and lingual dimensions of the BW were 3.9 ± 0.5 mm and 2.6 ± 0.4 mm, respectively. This difference in dimensions was secondary to the CTA dimensional changes which were 1.8 ± 0.8 mm at buccal and 0.7 ± 0.2 mm at lingual surfaces. Greater buccal marginal bone loss was the likely reason for the buccal/lingual differences in connective tissue length.</td>
</tr>
</tbody>
</table>

$BW =$ biologic width; $JE =$ junctional epithelium ; $CTA =$ connective tissue attachment
g) Mucosal thickness

An important question is whether a minimum width of the peri-implant mucosa is required to maintain health and stability of peri-implant tissues. To that end, Berglundh and Lindhe reduced peri-implant mucosa in an experimental canine model surgically (75). When the connective tissue side of flaps around implants (Branemark System) was thinned to 2mm or less at the time of abutment connection, increased bone resorption was observed (75). The influence of soft tissue thickness on peri-implant bone remodeling has been investigated by correlating the abutment cuff height, as a surrogate measure for mucosal thickness, with peri-implant marginal bone loss (76). Results demonstrated increased marginal bone loss around implants with shorter abutments, reflecting thin mucosa, possibly attributed to the need for re-establishing biologic width.

Platform switching has been proposed as a macro-design feature of implants to minimize peri-implant marginal bone loss. The outcome of a randomized trial has reported that platform switching decreases bone loss by 30% (76, 77). However, platform switching appears to be effective only when adequate mucosal thickness (mucosa thicker than 4.22 mm) is present (76, 77).

A promising concept evolving from the quality (thickness) and quantity (width) of keratinized mucosa is that the volume of the soft tissue over the crestal bone is the main factor establishing the biologic width and preventing crestal bone resorption around the teeth and implants. (78, 79) The volume of the supracrestal gingival tissue was initially evaluated by sonar, followed by elevation of a mucoperiosteal flap and direct measurement. Results showed that the distance from the crestal alveolar bone margin to the crest of the gingival margin correlates positively with the increase in thickness and width of keratinized gingiva.

A connective tissue circular ligament is present around the neck of implants. Whether or not accepting the assumption that the fibers may be perpendicularly distributed around implants (48), a promising hypothesis is to raise an activated osteoperiosteal flap over a 1.0 mm supracrestal area around an implant in order to allow deposition of new bone in this area; yet separated from the area of osseointegration by a 1.0 mm ring of smooth implant surface. The expected result would be the deposition of new bone over the 1.0 mm area separated from the area of osseointegration by the 1.0 mm ring of smooth implant surface. This way, this new bone joins the implant and allows for the development of Sharpey’s fibers from the peri-implant soft tissue, thereby reproducing the area of connective tissue attachment similar to what is seen around a natural tooth.

The adequate volume of the supracrestal peri-implant keratinized tissue and the immobility of the implant-bone union are the main factors responsible for the peri-implant sulcus homeostasis and should be the ultimate goal to be achieved along with the stronger attachment of connective soft tissue fibers to the implant.

h) Maxilla versus Mandible

The question whether placement of implants in either the maxilla or mandible can have differential effects on the biologic width was investigated recently by Romanos et al. (80) In their study, a total of 12 implants were placed either in the maxilla or mandible of a patient with a history of smoking. Ten months post implant placement the patient died and the implants were removed en bloc and examined histologically. Distinct dimensional differences were noted. In the maxilla, the biologic width was approximately 6.5 mm versus 4.8 mm in the mandible. Importantly, in the maxilla the connective tissue component was significantly greater than in the mandible. However, no dimensional differences were found in junctional epithelial length for implants placed in either the maxilla or mandible.

i) Flap vs. flapless techniques

A recent study by Blanco et al. (81) examined whether inserting implants via flap or flapless techniques had an effect on the biologic width. In their study on five beagle dogs, four implants were placed in the mandible of each dog immediately following tooth extraction. For each dog, flaps were raised on one side (control) while no flaps were raised in the other (test). After 3 months of healing, the dogs were sacrificed. Histometric analysis revealed the followings: 1) Flapless surgery junctional epithelium: 2.54 mm buccal and 2.11 mm lingual; 2) Flapped surgery junctional epithelium: 2.59 mm buccal and 2.07 mm lingual; 3) Flapless surgery connective tissue: 0.68 mm buccal and 0.54 mm lingual; and 4) Flapped surgery connective tissue: 1.09 mm buccal and 0.91 mm lingual. None of the differences between the groups were statistically significant.

Conclusion

Peri-implant soft tissue healing, including establishment of a physiologic peri-implant biologic width, is important for long-term implant function. In order to establish a functional biologic soft tissue seal, a minimum dimension of biologic width is required. When this minimum dimension is absent, crestal bone resorption is likely to occur, to allow space for establishment of a biologic width. In this review, multiple factors with varying impacts on peri-implant soft tissue healing were discussed. For some factors,
their effects on the establishment of a healthy biologic width are readily evident; for others, this relationship is less clear. The following is a brief summary of the important factors in the establishment of a physiologic peri-implant biologic width, the less important factors, as well as unresolved questions requiring further investigation.

- The peri-implant biologic width is similar to the biologic width around natural teeth and appears to serve similar protective barrier functions. However, the peri-implant biologic width is larger than the biologic width around teeth, primarily due to a longer junctional epithelium. In addition, unlike teeth with inserting periodontal ligament fibers into cementum, peri-implant connective tissue fibers are generally parallel to the implant surface, forming a tight adhesive attachment to the implant surface.

- Evidence appears to point to a hemidesmosomal attachment of epithelial cells to the implant surface.

- Implant surface topography and roughness may affect connective tissue fiber orientation. Fibers forming on minimally rough implant surfaces appear mostly parallel to the implant surface, while textured surfaces, including laser-modified surfaces, seem to promote the formation of perpendicular connective tissue fibers.

- Ambiguity exists regarding the possible effects of surface roughness on biologic width, with some studies suggesting smaller dimensions of biologic width with minimally rough implant surfaces while others reporting no difference related to surface roughness.

- Implant and abutment materials appear to have varying effects on the composition and size of the peri-implant biologic width, as well as the type of attachment formed between the implant and the surrounding soft tissue.

- Position of the implant shoulder relative to the bony crest may have an impact on biologic width, namely the more deeply placed the implant, the longer the resultant biologic width due primarily to an increase in the connective tissue component. The evidence, while suggestive, is not conclusive.

- The importance of the microgap relative to the dimensional components of the biologic width seems to parallel implant position, namely the deeper the microgap from the bony crest, the longer the biologic width.

- The inflammatory infiltrate within the connective tissue zone surrounding the microgap appears to induce peri-implant crestal bone resorption, resulting in a longer biologic width.

- Surgical technique, such as one- versus two-stage protocols or flap versus flapless surgery appears to have little influence on peri-implant soft tissue healing.

- Differences in implant loading times seem to have little effect on the compositional and dimensional properties of peri-implant biologic width.

- Immediate placement of implants into sockets following tooth extraction appears to have a differential impact on the development of peri-implant biologic width when compared to delayed implant placement. Most studies have documented a greater facial biologic width dimension, particularly attributed to increased connective tissue component with immediate implant placement.

- Data regarding the effect of maxillary or mandibular implant placement on biologic width is not sufficient. However, based on a cadaver study it appears that a longer biologic width, primarily secondary to an increased connective tissue component, may occur with maxillary implant placement.

References


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