

# Biologic Width Around Implants. An Evidence-Based Review

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## SUMMARY

**Purpose.** The concept of biologic width forms a basis for successful peri-implant soft tissue integration around titanium implants. Therefore, the objectives of this review are to determine and critically evaluate the present knowledge about biologic width around implants and to establish future research trends.

**Materials and Methods.** The literature was selected through several electronic databases, as well as a manual search in the major dental implant, prosthetic and periodontal journals. The reviewed data was published in English from 1980 to December 2007. Questions for systematic review were formulated. Abstracts, chapters from books, and unpublished materials were excluded, as they do not meet criteria for evidence-based studies. Articles were prioritized according to the value of different study types on the same issue. In vitro studies and literature reviews were excluded. The included publications were clinical, human histology and animal studies.

**Results.** In total, 75 articles were obtained. After two rounds of evaluation and criteria application 54 papers remained for final appraisal, namely 2 clinical papers, 8 human histology and 44 animal studies were analysed. Twenty-one full-text articles were excluded.

**Conclusions.** Evidence analysis shows that the present knowledge about biologic width around implants is mainly derived from animal studies and that clinical controlled human studies are insufficient.

**Key words:** biologic width, crestal bone loss, implant, abutment, peri-implant soft tissues.

## INTRODUCTION

It has been well documented in literature that bone supporting two-piece implants undergo crestal bone loss after the connection of the abutment and delivery of prosthesis in single tooth replacements [1, 2], partially edentate [3, 4] and completely edentulous patients [5, 6]. Albrektsson et al in 1986 established success criteria for implant treatment that included 1.5 mm loss of crestal bone in the first year of implant function [7].

While the reasons for early crestal bone loss have been extensively discussed in last decade, stability of crestal bone still remains a controversial issue. Overload [8], microgap [9], polished implant neck [10, 11], and infection [12] are some factors implicated in early peri-implant bone loss.

For a long time overload was considered to be the

main reason for crestal bone level changes, but recent studies have questioned the role of loading in aetiology of early crestal bone loss [13, 14, 15]. Microgap (the implant-abutment interface) has been shown to be a factor, if placed at bone level or subcrestally [9, 16], but such changes can be neutralized by positioning implant about 2 mm supracrestally [17]. A polished implant collar may provoke crestal bone loss associated with "nonload" factor, but, similarly to microgap, bone loss can be avoided by leaving smooth implant neck above the bone level [11]. One further factor that should be considered may be biologic width, i.e. the distance between the margin of peri-implant mucosa and underlying bone crest [18], which has not been as extensively studied as the other reasons for crestal bone loss.

The term biologic width was based on the work of Gargulio et al in 1961 who described the dimensions and relationship of dentogingival junction in human cadavers [19]. It has been hypothesised that a similar relationship of bone to overlying soft tissue exists around implants and changes in this relationship may be one of the reasons for the early crest bone loss [20].

There is a number of literature reviews published on biologic width around implants, all of them following

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the traditional narrative approach [21, 22, 23, 24]. The traditional review is informative and can provide a general perspective of the topic, but it is susceptible to bias in the selection of the publications to review [25]. It has been suggested, that a systematic critical review is the best method to extract the evidence from the literature [26]. However, there is a lack of critical review of the literature about biologic width around implants. The objective of this paper is twofold: (1) to evaluate up-to-date evidence from different type of studies of biologic width around implants; and (2) to establish future research trends.

## MATERIAL AND METHODS

Literature was selected through a search of PubMed, Embase and Cochrane Central Register of Controlled Trials electronic databases. The keywords used for search were *biologic width*, *peri-implant soft tissues*, *crestal bone loss*, *microgap*, *peri-implant seal*, *implant* and *abutment*. The search was restricted to English language articles, published from 1980 to December 2007.

Additionally, a manual search in the major dental implant, prosthetic and periodontal journals and books was performed. The issues from 1990 were searched in following journals: *Clinical Oral Implant Research*, *Journal of Clinical Periodontology*, *International Journal of Oral and Maxillofacial Implants*, *International Journal of Periodontics and Restorative Dentistry*, *International Journal Of Prosthodontics*, *Journal of Periodontology*, *Journal of Prosthetic Dentistry*, *Periodontology 2000*, *Implant Dentistry*, *Journal of Oral Implantology*, *Journal of Periodontal Research* and *Clinical Implant Dentistry Related Research*.

In order to be precise in data collection and to obtain all available information, references to all articles on biologic width were examined. In addition, congresses, courses and workshop materials were also assessed. Within the context of the aim of this review, following questions were formulated:

- What is the structure of biologic width around implants?
- What is the function of biologic width?
- What is the influence of mucosal thickness on biologic width formation?
- Does abutment connection/disconnection have influence on biologic width?

Full-text papers were sorted according to the nature of publication – experimental publications, reviews, hypothetical articles, technical notes, etc.

Experimental publications were prioritized according to the value of different study types on the same issue – in vitro studies (6<sup>th</sup> level), animal studies (5<sup>th</sup> level), his-

tological human studies (4<sup>th</sup> level), case series (3<sup>rd</sup> level), clinical studies (2<sup>nd</sup> level) and long term clinical studies (1<sup>st</sup> level) [27].

In order to determine which studies would be included in the review, several criteria were used depending on the type of the study. Evidence-based selection criteria have been published for clinical studies; however similar criteria are not available for animal studies [28, 29, 30]. In default of standard criteria, the following inclusion/exclusion criteria were formulated for animal studies: (1) the number, type, age of tested animals should be clearly mentioned in the study; (2) the number of implants tested should not be fewer than four per animal [31]; (3) the study should include trials with titanium or titanium alloy endosseous implants used in oral cavity.

Human histological studies were reviewed for the presence of (1) a clear outcome, and (2) examination of titanium implants. Clinical studies were included if they reported (1) a clear outcome of the study, (2) had a control group of titanium abutments or one-piece implants, and (3) the study included at least a 12-month follow-up analysis.

## RESULTS

The search identified 75 full-text articles, related to biologic width around implants. Unpublished materials (congress, workshop materials and personal communication) were excluded since they do not meet the criteria for evidence-based studies. Standard reviews and hypothetical articles were excluded due to possible bias. In vitro studies were excluded as they have low clinical relevance [32, 33].

Therefore, (1) animal; (2) human histology and (3) clinical studies were included in this critical analysis.

After the application of the inclusion/exclusion criteria, 54 articles were reviewed:

- 2 clinical studies;
- 8 human histological studies;
- 44 animal histological studies.

### What is the structure of biologic width around implants?

The included studies can be found in the Table 1.

#### *Animal studies*

Biologic width around titanium implants is well investigated in animal studies. Experiments in dogs focused on examining vertical extension and composition of tissues that form the biologic width. Included literature consisted of studies with teeth as a control [34], uncontrolled descriptive study [35], comparative studies between submerged and non-submerged implants [36, 37, 38, 39, 40], comparison between one- and two-piece implants [40]. Another series of studies tested the influence of loading

time on parameters of peri-implant seal [41, 42, 43]. One study looked at the influence of location of microgap to bone crest on extension of BW around implants [44].

Ten studies showed that biologic width around implants consists of sulcular and junctional epithelium and an underlying connective tissue zone [34, 35, 36, 37, 38, 39, 40, 41, 42, 43, 44]. Morphological structure of the epithelial part was investigated by Kawahara et al [45] in the study with 3 monkeys and 6 titanium blade implants and by Abrahamsson et al [36] in the study with 5 dogs and 30 titanium screw-type implants. They showed that the apical part of the epithelium is very thin and attaches to implant surface with hemidesmosome-like structures. Other studies elaborated on the connective tissue zone. The connective tissue appeared to be similar to scar-like tissue and had direct contact with implant surface, but without any attachment [46, 47]. Direct connective tis-

sue contact to implant surface was characterised by the absence of blood vessels and the abundance of fibroblasts interposed between collagen fibers. More lateral to this area there was a zone of fewer fibroblasts, more and larger collagen fibers and numerous blood vessels.

Circular collagen fiber network in horizontal sections around implant neck was found in the study by Ruggeri et al with 4 monkeys and 32 implants [48].

*Human histological studies*

The search identified 4 histological human studies, describing the structure of biologic width around implants. The most informative is a recent publication by Glauser et al. Five patients received a total of 12 experimental one-piece mini-implants – equal number of an oxidized and acid-etched or a machined surface. The total height of peri-implant tissues was calculated to be from 4 to 4.5 mm. The peri-implant sulcus varied from

**Table 1.** Included studies describing the structure of biologic width around implants

Publication	Study	Sample size and species	Follow-up	Results
Berglundh et al [34]	Animal controlled histology	5 dogs, 5 implants and teeth	9 months	BW extension – 3.80 mm around implants and 3.17 mm around teeth.
Tenenbaum et al [35]	Animal histology	6 dogs, 12 implants	9 months	Total extension of BW was 4.00 mm on buccal and 4.92 mm on lingual sites
Abrahamsson et al [36]	Animal histology	5 dogs, 30 implants	9 months	Non-submerged implants BW – 3.50 mm, submerged - 3.11 to 3.42 mm
Weber et al [37]	Animal histology	6 dogs, 38 implants	4.5 months	No statistical difference between submerged and non-submerged implants
Ericsson et al [38]	Animal histology	5 dogs, 30 implants	6 months	No statistical difference between submerged and non-submerged implants
Abrahamsson et al [39]	Animal histology	6 dogs, 18 implants	9 months	Submerged 3.00 mm, non-submerged 3.15 mm. No statistical difference
Hermann et al [40]	Animal histology	5 dogs, 59 implants	6 months	No difference between one- and two-piece implants
Hermann et al [41]	Animal histology	6 dogs, 69 implants	3 – 12 months	Unloaded group – 3.01 mm, loaded – 2.94 to 3.08 mm. No statistical difference.
Cochran et al [42]	Animal histology	6 dogs, 69 implants	3 – 12 months	No statistical difference between loaded and unloaded groups
Siar et al [43]	Animal histology	6 monkeys, 18 implants	3 months of loading	Immediate loading group – 3.9 mm, delayed loading – 3.78 mm. No statistical difference.
Todescan et al [44]	Animal histology	4 dogs, 24 implants	6 months	Longer BW in deeper placed implants
Kawahara et al [45]	Animal histology	3 monkeys, 6 blade implants	9 months	Morphometric evaluation of JE attachment zone.
Buser et al [46]	Animal histology	6 dogs, 24 implants	3 months	Similar composition of CT around implants with different surface roughness.
Moon et al [47]	Animal histology	6 dogs, 36 implants	9 months	CT divided into 2 zones: central, 40µm wide and lateral zone - 160µm. Scar-like tissue.
Ruggeri et al [48]	Animal histology	4 monkeys, 32 implants	14 months	Circular fiber network around implant neck in horizontal sections.
Glauser et al [49]	Human histology	5 patients, 12 implants	2 months	BW was found to be 4.0 – 4.5 mm. SD 0.2 – 0.5 mm, JE 1.4 - 2.9 mm, CT 0.7 – 2.6 mm.
Arvidson et al [50]	Human histology	10 patients, 10 implants	At least 36 months	JE attachment to implant via hemidesmosome-like structures
Scierano et al [51]	Human histology	7 patients, 9 abutments	At least 12 months	Horizontal and vertical alignment of CT fibers around implant abutments.
Liljenberg et al [52]	Human histology	9 patients, 18 implants	12 months	Inflammatory cells found in peri-implant mucosa.
Kan et al [53]	Clinical study	45 patients, 45 implants with crowns	Mean 32 months	Facial extension was 3.63 mm, medial – 6.17 mm and distal – 5.93 mm.

0.2 – 0.5 mm, junctional epithelium was limited to 1.4 – 2.9 mm and connective tissue had apical extension from 0.7 – 2.6 mm [49].

Arvidson et al evaluated the peri-implant seal of Brånemark titanium implants in 10 patients by taking soft tissue biopsies. The attachment of junctional epithelium to implant surface via hemidesmosome-like structures was noted [50].

Schierano et al investigated the direction of collagen fibers from 9 retrieved abutments with adjacent peri-implant mucosa in 7 patients. They reported that fibers align themselves circularly and horizontally around

the abutment [51]. Liljenberg et al measured the thickness of peri-implant soft tissues biopsies from 9 partially edentulous patients. The mean mucosa thickness was calculated to be 1.87 mm [52]. There seems to be clear evidence that the soft tissues histologically are capable of creating a seal around the implant neck.

#### *Clinical studies*

The vertical extension of soft peri-implant tissues was examined by Kan et al in a study of single anterior implants in 45 humans. In each patient implant soft tissues were probed to the bone on mesial, mid-facial and distal aspects. The mean dimension of biologic width

**Table 2.** Included studies describing the function of biologic width around implants

Publication	Study	Sample size and species	Follow-up	Results
Kawahara et al [45]	Animal histology	3 monkeys 6 blade implants	9 months	Migration of leukocytes through junctional epithelium
Bergludh et al [54]	Animal histology	5 dogs 15 implants 5 teeth	3 weeks of plague	No bone loss, increased rate of leukocytes migration (1.9% vs. 0.9%).
Lindhe et al [55]	Animal histology	5 dogs 15 implants	4 months	Mean 3.0 mm of crestal bone loss
Marinello et al [56]	Animal histology	5 dogs 20 implants	1-1.5 months	25% of original bone height was lost
Zitzmann et al [57]	Animal histology	5 dogs 22 implants	2 months	Mean bone loss was 4.10 mm
Ericsson et al [58]	Animal histology	5 dogs 30 implants	1.5 - 2 months	20% of implant length bone loss
Zechner et al [59]	Animal histology	8 dogs 48 implants	8 months	Bone loss and increased gingival probing depths around all ligatured implants.
Shibli et al [60]	Animal histology	6 dogs 36 implants	0 – 2 months	Bone loss from 1.62 mm to 2.09 mm around implants with different surfaces.
Hayek et al [61]	Animal histology	9 dogs 18 implants	8 months	All ligatured implants developed peri-implantitis.
Gotfredsen et al [62]	Animal histology	5 dogs 30 implants	4 months	Approximately 40% of initial bone support was lost.
Warrer et al [63]	Animal histology	5 monkeys 30 implants	9 months	All implants had attachment loss. BIC varied from 54% – 65% of total implant length.
Shou et al [64]	Animal histology	8 monkeys 32 cylindrical implants	0-7 weeks	Increase of probing depth, gingival with bleeding score and bone loss around ligatured implants.
Shou et al [65]	Animal histology	8 monkeys 64 implants	9-18 months	Bone loss of 4-6 mm around all implants.
Shou et al [66]	Animal histology	8 monkeys 32 implants	8 months	Bone loss of 2-4 mm prevailed within peri-implantitis group.
Ericsson et al [67]	Animal histology	5 dogs 15 implants 15 teeth	3 months	Spread of infiltrate (ICT) was 1.3 mm at implants and 0.9 mm at teeth. No bone loss, inflammation.
Abrahamsson et al [68]	Animal histology	5 dogs 30 implants	5 months	Clinical signs of inflammation, ICT size about 1.6-2.0 mm, bone loss 0.64 mm.
Ericsson et al [69]	Animal histology	5 dogs 15 implants	9 months	Inflammation, ICT – 1.8 mm, bone loss – 1.4 mm.
Watzak et al [70]	Animal histology	9 implants 54 implants	1.5 years	Inflammation, bone loss 0.6-0.9 mm.
Sanz et al [71]	Human histology	12 patients 12 implants	9 months	Significantly higher migration of inflammatory cells to JE.
Zitzmann et al [72]	Human histology	12 patients 24 implants	3 weeks	Increase of inflammation markers in JE – 5.0% infected sites vs. 3.5 % healthy sites.
Bullon et al [73]	Human histology	5 patients 5 implants	No	Increase of T lymphocytes
Chavier and Coubles [74]	Human histology	8 patients 32 implants	2 years	Type I collagen was dominant in CT biopsies.

was recorded to be 6.17 mm at mesial, 3.63 mm at mid-facial and 5.93 mm at distal sites of implants [53].

**What is the function of biologic width around implants?**

It has been suggested that soft tissue around implants form biological structures similar to BW around teeth and may serve as a protective mechanism for underlying bone. Included studies can be found in the Table 2.

*Animal studies*

Migration of leukocytes through junctional epithelium towards bacterial plaque was reported in an animal experiment with monkeys [45]. Accumulation of these cells in the presence of infection may demonstrate the possible defence mechanism of biologic width. In a dog experiment soft tissues around implants after uninterrupted plaque accumulation were characterized by an increased rate of migration of leukocytes through the junctional epithelium, as compared to not infected control implants (1.9% vs 0.9%) [54].

The evidence of the protective peri-implant seal abilities may be found in animal studies, which use induced peri-implantitis model. Lindhe and co-workers in an experiment with 5 dogs (15 implants), induced peri-implantitis using ligatures and within 4 months, had about 3 mm of bone height loss around the implants [55]. Seven subsequent experiments with dogs [56, 57, 58, 59, 60, 61, 62] and four with monkeys [63, 64, 65, 66] confirmed that the combination of plaque accumulation and biologic width injury can result in crestal bone loss around implants.

In contrast, a number of studies in which implants were exposed to undisturbed plaque formation without ligature placement for different periods of time, ranging from 3 weeks to 1.5 years [54, 67, 68, 69, 70], reported no or only minimal bone loss in the presence of soft tissue inflammation. It would seem that the ligature may disrupt the epithelial attachment causing the bone loss.

**Table 3.** Included studies describing influence of mucosa thickness

Publication	Study	Sample size and up species	Follow-up	Results
Berglundh and Lindhe [18]	Animal histology	5 dogs 30 implants	9 months	Bone resorption and angular defects around implants with <2 mm mucosa thickness.
Berglundh et al [76]	Animal histology	20 dogs 80 implants	0-12 weeks	BW formation included crestal bone loss, however no precise measurements.

*Human histological studies*

The function of junctional epithelium was investigated by Sanz et al. Comparative histological study of healthy and infected implant sites in 12 patients revealed that biopsies from implant infection group showed significant higher transmigration of inflammatory cells in sulcular epithelium [71]. Zitzmann et al investigated the reaction of peri-implant mucosa to plaque accumulation for three weeks in 12 partially edentulous patients. In each patient two implants sites were selected and soft tissue biopsies obtained. There was significant increase in density of PMN elastase<sup>+</sup> -cells (inflammation markers) within the junctional epithelium after 21s day of plaque accumulation – 5.0% in comparison to 3.5% in healthy implant soft tissues [72]. A case-controlled study showed significant increase of T lymphocytes in sulcular epithelium in peri-implantitis biopsies, compared with healthy peri-implant tissue [73].

Chavier and Couble focused their study on connective tissues around implants. The biopsies were obtained from healthy keratinized soft peri-implant tissues of 32 implants in 8 patients and analysed for structure and function of the connective tissue. Type I collagen was found to be the dominant fiber [74].

No clinical trial articles on the issue were found.

**What is the influence of mucosa thickness on biologic width around implant formation?**

It has been hypothesized that a certain width of the peri-implant mucosa is required to enable a proper epithelial – connective tissue attachment and, if this soft tissue dimension is not satisfied, bone resorption may occur to ensure the establishment of attachment with an appropriate biologic width [75]. Included studies can be found in the Table 3.

*Animal studies*

Berglundh and Lindhe in a controlled experiment with 5 dogs (30 implants) tested the influence of mucosa thickness on biologic width formation around implants [18]. At

**Table 4.** Included studies describing influence of abutment manipulation

Publication	Study	Sample size and species	Follo w-up	Results
Abrahams-son et al [77]	Animal histology	5 dogs 10 implants	9 months	Bone loss in test group – 1.49 mm and 0.78 mm in control group.
Abrahams-son et al [78]	Animal histology	6 dogs 36 implants	12 months	Bone loss at test group – 0.7 mm and in control group – 1.1mm.
Watson et al [79]	Clinical retrospective study	117 patients 430 abutments	3 years	Mean levels of marginal bone were not higher.

the second stage surgery in test implants, peri-implant mucosa was thinned to about 2 mm, while control implants had healing abutment connected without tissue thickness alteration. The histology showed that in the test implants bone resorption was consistently observed after soft tissue healing, while the total biologic width was not statistically significant between the test and control implants. The process of biologic width formation around implants was described by Berglundh et al in a dog study. The authors observed that the morphogenesis of peri-implant mucosa involved loss of marginal bone [76].

No human histology or clinical studies about formation of biologic width or influence of mucosal thickness on bone resorption could be found.

### **Does abutment disconnection/connection (prosthetic manipulation) have influence on the stability of biologic width?**

Included studies can be found in the Table 4.

#### *Animal studies*

Abrahamsson et al [77] in a controlled histological study with 5 dogs (10 implants) proved that disconnection of healing abutment five times may cause crestal bone loss. Test implants showed significantly higher reduction of bone height than control implants - 1.49 mm and 0.78 mm. Clinically, the bleeding and ulceration of soft peri-implant tissues after the disconnection of the abutment was observed. In a later study in 6 dogs and 36 implants, Abrahamsson et al [78] found that single disconnection of healing abutment to prosthetic abutment did not cause any additional bone loss.

#### *Clinical studies*

Watson et al [79] in retrospective clinical study evaluated soft tissue condition and crestal bone loss around implants which had earlier healing abutments placed after second stage surgery. After a 3-year follow-up, it was concluded that there was no evidence to suggest that abutment exchange adversely affects the outcome of implant treatment. The shift from healing abutment to prosthetic analogue neither affected the survival rates of implants nor increased the marginal bone loss.

## **DISCUSSION**

The first unexpected finding was the insufficiency of clinical studies on biologic width around implants, as only 2 papers were identified. The requirements for systematic reviews state that randomised controlled trials are preferred because they provide the highest level of evidence [80]. However, in the absence of available randomised controlled clinical trials evidence is sought at less reliable levels.

The major part of the information about biologic width around implants is derived from animal studies. In

the light of evidence-based dentistry, the place of animal study is not clear. The similarity of physiology between animals and humans forms the reason for animal studies, and the results obtained may have a high degree of relevance for humans, although they can not be directly transferred to clinical situations. On the other hand, some researchers have postulated that animal studies are of low clinical relevance and even a simple case report may have more clinical validity than well controlled and randomised animal experiment [81]. However, not all experiments on biologic width can be repeated in humans, due to ethical reasons, leaving clinicians to rely on data from animal studies. It is agreed that animal experiments are more significant than *in vitro* studies; however, they provide a lower rank of evidence as compared to human histological or clinical trials [27].

In summary, it can be said that histological animal studies provide sufficient information to state that structure of biologic width around implants is composed of peri-implant sulcus, junctional epithelium and connective tissue zone. Human histology studies are in agreement with the outcome of animal experiments, listing the same component parts of the biological dimension [49, 50, 51, 52]. The results of dog studies indicate that the parameters of biologic width are very similar around one-piece and two-piece implants. Submerged and non-submerged implants, as studied by Weber et al [37], Ericsson et al [38], Abrahamsson et al [39] and Hermann et al [40], had a very similar soft tissue length; therefore, it can be concluded that surgical techniques do not influence formation, composition or extension of biologic width. It seems that conventional or immediate loading of implants does not influence the parameters of peri-implant seal, as it was observed in comparative studies with unloaded implants [43]. Only the position of implant/abutment interface (microgap) to bone level proved to affect the vertical extension of biologic width – the deeper implant is placed, the longer biological dimension is formed [44]. However, it must be noted that the majority of histological experiments were performed on dogs, although non-human primates are considered to better resemble human oral anatomy and histology than any other animal [82]. The literature search identified only two studies performed on monkeys, which investigated the structure of biologic width [43, 45].

In a human histological study the length of the peri-implant seal was found to be about 4-4.5 mm [49]. In contrast, Liljenberg et al [53] reported the same measurement to be 1.57 mm. However, the authors of the latter experiment admitted that such results may have occurred due to improper biopsy harvesting. The mean extension of biologic width around implants in primate studies was recorded to be 3.84 mm. In histological dog studies this distance was calculated to be around 4 mm.

As compared to biologic width around teeth, the same parameter around implants was longer nearly by the factor of 1.5 mm. Gargulio et al [19] found the dimension of biologic width around cadaver teeth to be 2.73 mm and Vacek et al – 3.25 mm respectively [83]. It is evident that the peri-implant seal around implants tends to be longer, than around teeth. However, the clinical importance of this difference is unknown. Clinical study by Kan et al recorded most extension of biologic width around implants – 6.17 mm at medial and 5.93 mm at distal sites of implants. These results were obtained by probing to bone level and may have been influenced by the emergence profile of the crowns on implants. Additionally, proximal sites frequently show deeper probing depths due to position of the bone crest. However, the mid-facial measurement was recorded to be 3.63 mm, which is very close to the width observed in animal and human histology studies.

The proceedings of the 3<sup>rd</sup> European Workshop on Periodontology and Implant Dentistry state that the function of the peri-implant seal is “to maintain homeostasis of the internal environment in response to challenges from external environment” [84]. Animal and human histology studies show that there is an increase of inflammatory cell migration through junctional epithelium, in response to bacterial presence [45, 54, 71, 71, 73]. These findings support the idea that junctional epithelium of biologic width around implants serves as a protective mechanism against bacterial invasion. This is in agreement with studies around teeth [85]. Studies which experimentally induced peri-implantitis may be another argument that junctional epithelium attachment protects bone. Mechanical damage of junctional epithelium by means of subgingival ligature placement resulted in the loss of protective abilities and constant bone loss around implants [55, 56, 57, 58, 59, 60, 61, 62, 63, 64, 65, 66]. In contrast, a number of articles show that the stable bone level around osseointegrated implant can be maintained even under the onset of plaque-induced inflammation if components of biologic width are not mechanically damaged [54, 67, 68, 69, 70].

One of the functions of the connective tissue zone is to support epithelial tissues and limit its migration apically. The dominance of Type I collagen fibers (strong and inelastic) in connective tissues confirms their supportive role. However, it must be pointed out that in Chavier and Coubles' study, biopsies were taken from keratinized mucosa and may differ from that of non-keratinized peri-implant mucosa.

It can be summarized that there is enough evidence from animal and human histology studies to state that the function of biologic width around implants is to protect underlying bone. However, clinical controlled randomised trials would be desirable, but difficult due to ethical reasons.

The hypothesis that tissue thickness and biologic width formation may influence crestal bone loss is supported by animal studies [75, 76]. A similar conclusion was made by Oakley et al in the study on the formation of biologic width around teeth after crown lengthening in primates [86]. After 3 months, a mean crestal bone loss of 0.6 mm was registered as the biologic width was regaining its dimension. In addition, Albrektsson et al noticed that implant sites with thin tissues were prone to form angular defects around fixtures after healing [87]. Clinically, thin tissues can be expected if thin gingival biotype is present [88], and crestal bone loss may be expected as a result of the biologic width establishing its minimal dimension. However, there are no clinical studies to support this hypothesis.

It was suggested that healing abutment disconnection as a part of prosthetic treatment results in disruption of the epithelial seal, causing bleeding and ulceration of the site. This mechanical disruption may be considered as an open wound or exposure of connective tissue which may result in inflammatory responses and epithelial migration. The reestablishment of biological width in more apical position may be the explanation for crestal bone loss. However, this hypothesis is based on animal study [77]. Moreover, another animal study did not confirm that abutment disconnection may be deleterious to the stability of peri-implant tissues. Such conclusion is in agreement with the retrospective clinical trial outcome which suggested that abutment manipulation did not cause any evident bone loss or mucosal health impairment around implants. However, control group and randomization were not used in this study; therefore, the results should be evaluated with caution.

## CONCLUSION

Within the limitations of this analysis and currently available evidence, it can be concluded that the structure and function of biologic width around implants are well documented in animal and human histological studies. However, it is not clear what influence abutment disconnection may have on peri-implant tissues, as animal experiments provide contrary findings. There is enough evidence to acknowledge that thin tissues can cause crestal bone loss in the process of biologic width formation, at least on the level of animal studies. On the other hand, clinical evidence is weak or absent. Data from animal studies should be very carefully interpreted, when applied to clinical cases, if reliable clinical evidence is. Therefore, it can be recommended to perform randomised controlled clinical trials to test abutment disconnection and tissue thickness influence on biologic width around implants.

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Received: 17 01 2008

Accepted for publishing: 26 03 2008