Mucosa Thickness and Peri-implant Crestal Bone Stability: A Clinical and Histologic Prospective Cohort Trial

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Purpose: *To correlate soft tissue thickness and peri-implant bone remodeling of platform-switching implants.* ***Materials and Methods:*** *This comparative prospective trial evaluated, for up to 3 years after implant loading, the influence of soft tissue thickness on changes in peri-implant marginal hard tissue levels. Any patient who was partially edentate in the mandible and required at least two adjacent implant-supported restorations was recruited at the University of Valencia in Spain. A 3-mm tissue punch biopsy, which corresponded to a diameter slightly smaller than the coronal diameter of the implants, was performed using a circular mucotome. Afterward, implants with a length of 10 to 13 mm and a diameter of 3.8 mm were inserted. Outcome measures were implant and prosthesis survival rates, marginal hard tissue changes, any complications, and results of morphologic and histomorphometric analyses. Correlation between mucosa width components (epithelium, connective tissue, and epithelium and connective tissue) and radiographic bone loss at 1 and 3 years after loading was performed at the patient level. Statistical significance was set at* $P \leq .05$. ***Results:*** *A total of 26 samples in 26 patients with 68 implants were analyzed. The specimens were divided into two groups: group 1 (16 patients, 40 implants), with thin mucosa (≤ 2 mm), and group 2 (10 patients, 28 implants), with thick mucosa (> 2 mm). Two dropouts (two specimens) were recorded at the 3-year follow-up. None of the implants or definitive prostheses failed during the healing period, resulting in an overall implant and prosthesis cumulative survival rate of 100%. No major biologic or mechanical complications were recorded. The mean (standard deviation, SD) epithelium thickness was 430.33 (250.21) µm; the mean (SD) connective tissue thickness was 1,324.31 (653.46) µm, and the mean (SD) mucosa thickness was 1,751.29 (759.53) µm. Comparisons of radiographic bone loss between group 1 and group 2 failed to show any statistically significant differences at the 1-year (*$P = .290$*) or 3-year (*$P = .090$*) follow-up examinations.* ***Conclusion:*** *The initial mucosa thickness surrounding a bone-level platform-switching implant seems not to influence the pattern of physiologic marginal bone loss.* Int J Oral Maxillofac Implants 2017 (7 pages). doi: 10.11607/jomi.5349

Keywords: *dental implants, marginal bone loss, mucosa thickness, platform switching*

Prospective studies using matched-abutment implants reported initial physiologic bone remodeling of up to 2.0 mm 1 year after abutment connection.[1] Different factors were described as contributing to this event, including crestal module design and biologic width establishment.[2–4]

Several randomized clinical trials and a systematic review found that implants with platform switching exhibited less bone loss compared with a traditional matching implant-abutment connection,[2,4–6] while few studies found no differences.[7] The microgap at the implant-abutment interface yields a chronic inflammatory response. Hence, bone remodeling may be expected to reestablish the biologic width.[3] The biologic width around teeth is defined as the soft tissue dimension comprising the junctional epithelium and the connective tissue attachment.[8] Dental implants present analogous histologic structures.[9,10] These structures measured about 3 to 4 mm around implants, with the sulcular epithelium ranging between 1.5 and 2 mm and the connective tissue between 1 and 2 mm.[3,9] Varying gingival phenotypes have been described in the literature, and they mainly have

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been divided into two groups: thick and thin gingival biotypes.[10,11] Claffey and Shanley[12] proposed that a thin gingival biotype might be correlated to periodontal diseases. Similarly, in implant-supported restorations, the presence of thick soft tissues was considered a crucial factor for long-term success.[13]

Animal and human studies have shown that peri-implant mucosa width might affect the peri-implant hard tissue levels. Berglundh and Lindhe[14] found that thin soft tissues around implants might be related to marginal hard tissue–level changes during the formation of biologic width. In a controlled study, Linkevicius et al[15] confirmed the hypothesis suggested in a previous animal study and reported that soft tissue thickness of 2 mm or less may produce more bone resorption compared with implants placed in thick tissues, regardless of implant depth. The same authors also reported that although the platform-switching concept was proven to be an effective strategy to reduce peri-implant bone resorption, it does not preserve bone when implants are inserted in thin tissues.[16] A recent systematic review concluded that implants placed in thicker soft tissues might present less marginal bone remodeling.[17] However, the study highlighted the influence of confounding factors, such as platform switching, type of retention, and type of surgical approach.

The importance of a thick biotype was confirmed in a recent study by Puisys and Linkevicius.[18] In addition, Favero et al[19] demonstrated that thickening thin soft tissues could produce minimal bone level changes, similar to that with implants inserted in a native thick biotype. However, no histologic analysis was performed, and soft tissue thickness was visually measured at the vestibular aspect after crestal incision with a 1.0-mm-marked periodontal probe. Thus, histologic analysis of thin tissues around platform-switching implants and their correlation with clinical peri-implant bone resorption remain to be established.

The aim of this histologic prospective cohort study was to assess the impact of the soft tissue thickness on marginal hard tissue–level changes around implants with a platform-switching design. The null hypothesis was that thin soft tissues are associated with more bone resorption around implants on function compared with thick tissues. This study was reported according to the STROBE statement.[20]

MATERIALS AND METHODS

Patients were selected and treated at the Department of Oral Surgery and Implantology Unit, Stomatology Department, Faculty of Medicine and Dentistry, University of Valencia in Spain, between January 2012 and January 2013. The study was performed according to the Declaration of Helsinki after approval was received from the institutional review board of the University of Valencia.

All patients 18 years or older with missing teeth in the mandible who required at least two adjacent implant-supported restorations and were able to sign an informed consent form were eligible for this study. Inclusion criteria were a partially edentulous mandible according to Kennedy Class I, II, and III; sites healed for at least 6 months; and adequate bone volume to insert implants without an augmentation procedure (horizontal width > 7 mm). Exclusion criteria were active periodontitis or peri-implantitis, absence of sufficient keratinized tissue for harvesting 3-mm biopsy specimen, smoking habit, pregnant and lactating patients, history of bisphosphonate therapy, malignant diseases or other diseases treated with radiotherapy or chemotherapeutic agents (chemotherapy) during the past 2 years, history of head and neck radiation treatment, and alcohol or drug abuse. Patients were informed about the protocol before being enrolled in the study.

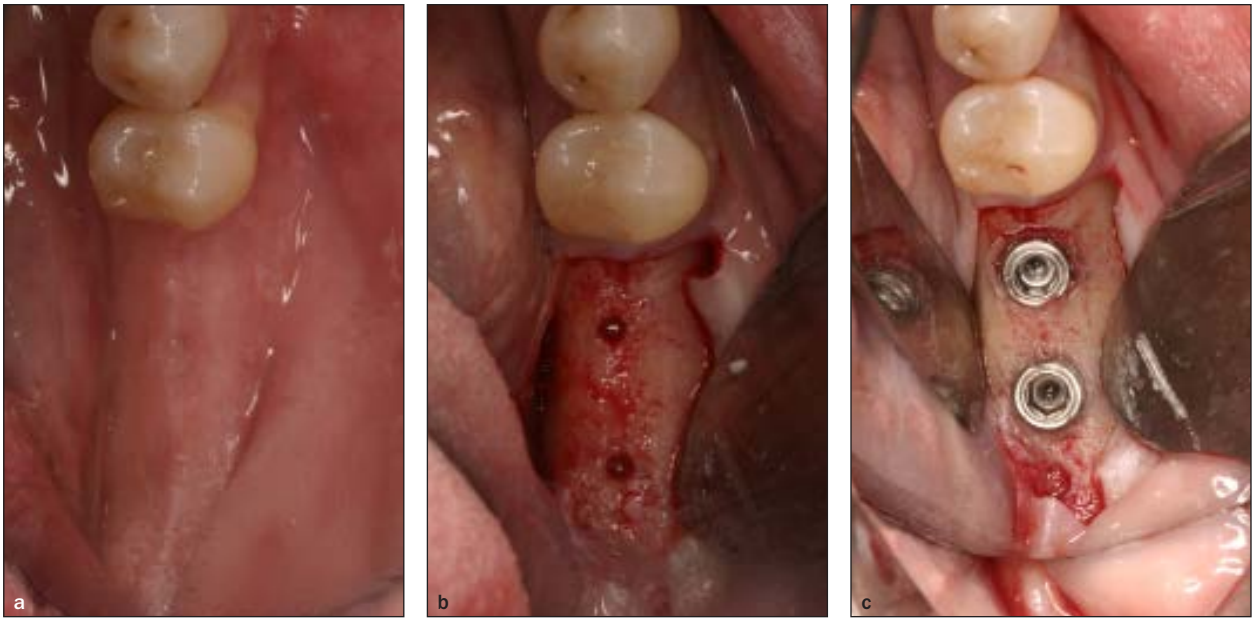
Treatment

Before surgery, patients underwent scaling, root planing, and oral hygiene instruction. Prophylactic antibiotic therapy was prescribed. Patients were treated under local anesthesia (articaine with adrenaline 1:100,000).

A 3-mm tissue punch biopsy corresponding to a diameter slightly smaller than the coronal diameter of the implants was performed using a circular mucotome. The small disk of full-thickness mucosa was gently raised above the incision using an atraumatic surgical elevator (2/4 molt surgical curette, Hu-Friedy). The soft tissue specimen was then immediately fixed in 4% buffered paraformaldehyde for 6 hours and collected, according to a standardized soft tissue protocol.[21] An independent examiner not otherwise involved in the study collected all soft tissue specimens. After full-thickness flap elevation, implants with a length of 10 to 13 mm and a diameter of 3.8 mm (Premium SP, Sweden & Martina) were inserted. The same experienced, blinded clinician (L.C.) placed all implants slightly below the crestal bone level, according to the manufacturer's surgical indications (Fig 1). Once the healing screw (0.35 mm of platform switching) was inserted, suturing was performed. Two weeks later, the sutures were removed, and 2 months after that, definitive restorations (0.35 mm of platform switching) were cemented. Splinted restorations were used to better distribute occlusal loading. The occlusion was adjusted to prevent any premature contacts. Patients were recalled every 4 months for hygiene maintenance.

Radiographic Measurements

Marginal bone–level changes were measured from the abutment connection less than 0.5 mm to account for the smooth implant collar of the platform-switching



Figs 1a to 1c Clinical sequence at the time of implant insertion.

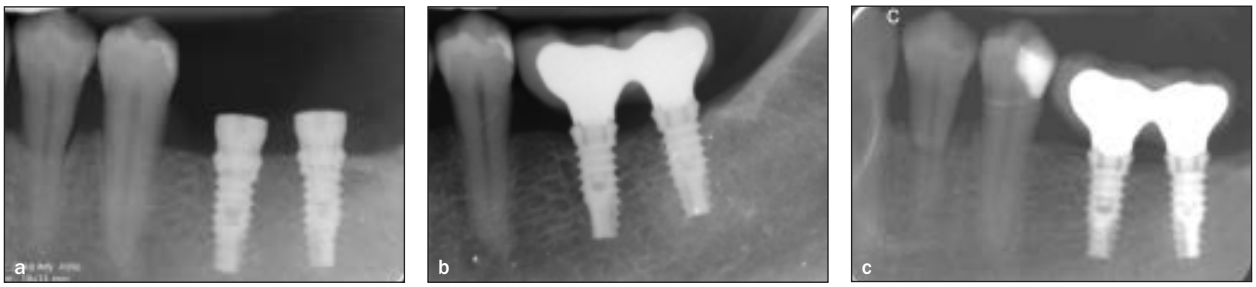


Fig 2 Radiographic analysis at *(a)* T_0 (baseline), *(b)* T_1 (1-year follow-up), and *(c)* T_2 (3-year follow-up).

implants as a reference. Radiographs were obtained at baseline (T_0, 3 months after implant insertion) and at clinical follow-up visits 1 (T_1) and 3 (T_2) years after loading (Fig 2). A digital film holder was used to ensure a reproducible radiographic analysis. Following the method of Canullo et al,[2] all readable radiographs were displayed by means of dedicated image analysis software (Autocad 2006, version Z 54.10, Autodesk) calibrated for every image using the known distance (pitch) between two consecutive implant threads. Measurements of the mesial and distal bone levels adjacent to each implant were made to the nearest 0.05 mm and averaged at the patient level. According to the study protocol, two adjacent implants were measured to avoid any bias due to eventual presence of bone peaks between adjacent implants or teeth. Two independent blinded examiners (MT, EX) performed all bone-level measurements. The mean value was used for the statistical analysis, and the κ index between investigators was calculated.

Morphologic Analysis

Cross sections of the punch biopsy sample embedded in polymethyl methacrylate were obtained using a tungsten carbide knife on an Autocut 1150 microtome (Reichert-Jung). Sections were stained with toluidine blue, trichrome Gomori, trichrome Masson, trichrome Ladewig, or Verhoeff stain.[21] All section slides were digitized using a brightfield and fluorescence slide scanner (Leica SNC400 F, Leica Biosystems) and observed at 5× magnification. Morphologic analysis (epithelium thickness, connective tissue thickness, mucosa thickness) was performed using dedicated software for image analysis (ImageJ, version 1.46, US National Institutes of Health) (Fig 2). The measurements (25 for epithelium thickness, 25 for connective tissue thickness, and 25 for mucosa thickness), in millimeters, were averaged, following the methodology proposed by Laliberté et al.[22] The samples were divided into two groups based on mucosa thickness (epithelium and connective tissue), according to the

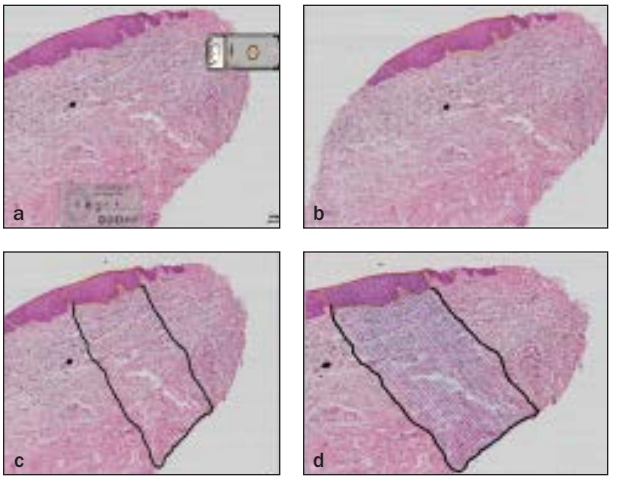


Fig 3 Morphologic analysis to measure the thickness of epithelium, connective tissue, and mucosa using software for image analysis (ImageJ, version 1.46, US National Institutes of Health). *(a)* Scanned image at magnification ×5. *(b)* Epithelial area selected for measure; *(c)* connective tissue area selected for measure; *(d)* mucosa thickness analysis (25 measurements each for epithelium thickness, connective tissue thickness, and mucosa thickness).

classification proposed by Linkevicius et al:[15,16] Group 1 consisted of samples with thin mucosa (≤ 2 mm) and group 2 consisted of samples with thick mucosa (> 2 mm). A well-trained examiner, not previously involved in this study, performed all morphologic measurements.

Statistical Analysis

The authors analyzed the data using a preestablished plan. The patient was the statistical unit of the analyses. The Fisher exact test was used to compare differences between groups in the proportion of patients with prosthesis failures, implant failures, and complications.

The Kolmogorov-Smirnov normality test and Levene variance homogeneity test were applied; the data showed a skewed distribution and were analyzed using a nonparametric ranking test.

Pearson's correlation coefficient was used to evaluate the correlation between mucosal tissue composition and radiographic bone loss after 1 and 3 years of loading. The Student *t* test for two independent samples was used for quantitative variables, in each case determining whether variances were homogenous. *P* value was set at .05 for all statistical comparisons.

RESULTS

A total of 26 samples (26 patients with 68 implants) were analyzed. Seventy-five measurements each were made for morphologic analysis of epithelium thickness, connective tissue thickness, and mucosa thickness using dedicated software for image analysis (ImageJ, version 1.46) (Fig 3). According to the method of Linkevicius et al,[15,16] the specimens were divided into two groups: group 1 (n = 16), mucosa thickness ≤ 2 mm and group 2 (n = 10), mucosa thickness > 2 mm.

By the end of the study, two patients (two specimens) had dropped out. None of the implants or definitive prostheses failed during the healing period ($P = 1.000$), resulting in an overall implant and prosthetic cumulative survival rate of 100%. No major biologic or mechanical complications were observed ($P = 1.000$).

The mean (standard deviation [SD]) epithelium thickness was 430.33 (250.21) µm; the mean (SD) connective tissue thickness was 1,324.31 (653.46) µm; and the mean (SD) mucosa thickness was 1,751.29 (759.53) µm. After an initial mean (SD) marginal bone loss of 0.31 (0.46) mm, all implants were in stable condition at the 3-year follow-up examination, with a mean (SD) marginal bone loss of 0.39 (0.48) mm.

At the 1-year follow-up examination, no or very weak correlation was found between connective tissue ($r = 0.003$; $P = .989$), epithelium ($r = –0.247$; $P = .224$), and mucosa (epithelium and connective tissue) thickness ($r = –0.090$; $P = .663$) (Fig 4) and radiographic bone loss. Three years after loading, a weak correlation was found for all comparisons ($r = –0.277$, –0.206, and –0.286, respectively) (Fig 5). Tables 1 and 2 present these data.

No statistically significant differences in radiographic bone loss were found between group 1 and group 2 at the 1-year ($P = .414$) or 3-year ($P = .107$) follow-up examinations. Data are presented in Table 3.

DISCUSSION

The aim of this prospective cohort study was to examine the impact of soft tissue width on peri-implant marginal hard tissue–level changes for up to 3 years after loading. The results showed no statistically significant association between peri-implant marginal bone loss and soft tissue width. Therefore, the null hypothesis that a thin biotype produces major bone resorption around an implant on function was rejected in favor of the alternative hypothesis that bone resorption for a thin biotype does not differ from that for a thick biotype.

The main limitation of this study was the recruitment of patients with a partially edentulous mandible according to Kennedy Class I, II, and III. Thus, the results may be generalized only to posterior mandibles. Another limitation is the small sample size, which could have hidden some differences between groups. Finally, soft tissue samples were obtained at implant placement, and they did not have exactly the same thickness as the final peri-implant soft tissues.

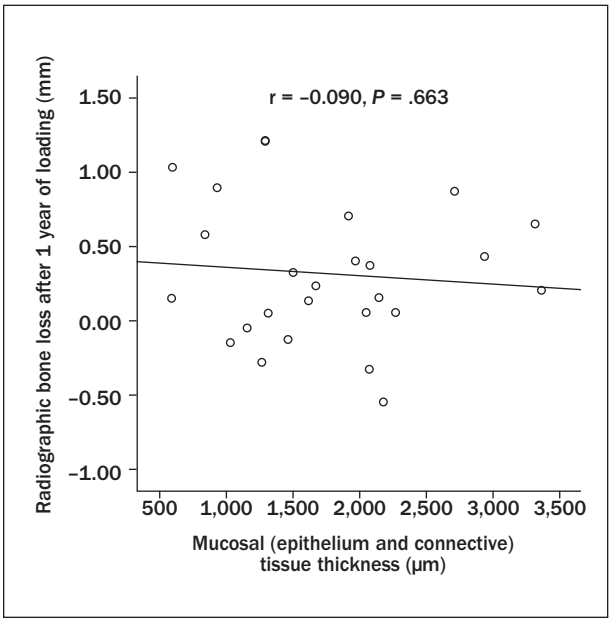


Fig 4 Correlation between mucosa thickness (epithelium and connective tissue) and radiographic bone loss after 1 year of loading.

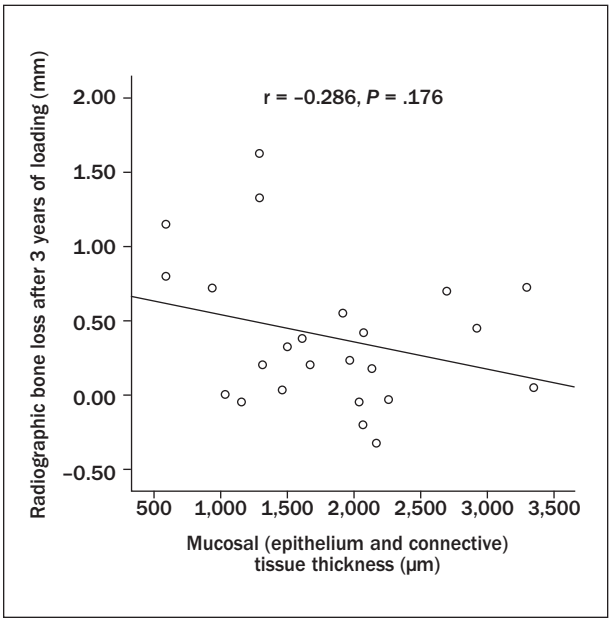


Fig 5 Correlation between mucosa thickness (epithelium and connective tissue) and radiographic bone loss after 3 years of loading.

Table 1 Correlation Between Epithelium Thickness, Connective Tissue Thickness, and Mucosa (Epithelium and Connective Tissue) Thickness and Radiographic Bone Loss (0.31 ± 0.46 mm) After 1 Year of Loading (Average Values of Mesial and Distal Surfaces)

Correlation (n = 26)	Mean ± SD (µm)	*r*	*P* value
Epithelium thickness	430.33 ± 250.21	–0.247	.224
Connective tissue thickness	1,324.78 ± 653.46	0.003	.989
Mucosa thickness	1,751.29 ± 759.53	–0.090	.663

Table 2 Correlation Between Epithelium Thickness, Connective Tissue Thickness, and Mucosa Thickness and Radiographic Bone Loss (0.39 ± 0.48 mm) After 3 Years of Loading (Average Values of Mesial and Distal Surfaces)[a]

Correlation (n = 26)	Mean ± SD (µm)	*r*	*P* value
Epithelium thickness	430.33 ± 250.21	–0.277	.190
Connective tissue thickness	1,324.78 ± 653.46	–0.206	.333
Mucosa thickness	1,751.29 ± 759.53	–0.286	.176

[a]Two patients (two specimens) dropped out at the 3-year follow-up examination.

Table 3 Comparison of Radiographic Bone Loss After 1 and 3 Years of Loading (Average Values of Mesial and Distal Surfaces) Between Study Groups

	Bone loss (mm), mean (range)		
	Group 1[a] (n = 16)	Group 2[b] (n = 10)	*P* value
After 1 year of loading	0.27 (–0.28 to 1.21)	0.17 (–0.55 to 0.87)	.414
After 3 years of loading	0.35 (–0.05 to 1.63)[c]	0.11 (–0.33 to 0.73)	.107

[a]Group 1: Thin mucosa (≤ 2 mm).
[b]Group 2: Thick mucosa (> 2 mm).
[c]Two patients (two specimens) dropped out at the 3-year follow-up examination.

The results of this study disagree with those of previous studies on the topic,[15,16,18] as well as with a recent systematic review.[17] A possible explanation for these differences is the small sample size and different technique used to measure the thickness of the mucosa. Most researchers report using a periodontal probe after partial flap deflection,[15,16,18] which allows for measurements to be made to the nearest millimeter. However, this method is affected by possible bias consisting of nonstandardized periodontal probe inclination, flap incision line angulation, and flap mobility. Furthermore, this method allows measurement of only the tissue thickness (quantity), with no information regarding its quality. In the present study, the soft tissue thickness was histologically measured. The major advantages of this method are that it provides accurate objective measurements of the tissue thickness, as well as distinguishes between epithelium and connective tissue components, which offers some idea about the quality of the tissues. The biopsy sample was obtained by means of a tissue punch that allowed a wider portion of the soft tissue to be included. The histologic sample was prepared at the time of implant placement, once the incision line was performed but before the flap was raised, thus avoiding any bias due to flap mobility and cutting angle that may affect the measurements. Furthermore, the surgeon was blinded, avoiding any bias caused by the knowledge of the soft tissue thickness immediately before implant placement.

Peri-implant bone resorption occurs mainly during the first year of loading and seems to occur irrespective of any effort to prevent it.[4,23] Peri-implant bone resorption is mediated by the bone inflammatory response. Although this topic is controversial, the inflammatory response seems to depend on the interaction between the patient's individual inflammatory pattern and the local environment (presence of inflammatory cells in the bone).[24] Crestal tissue quality, quantity, and composition have been linked to marginal bone changes and risk of inflammatory complications.[24] At the same time, surgical and prosthetic procedures could affect the expression of this phenotype.[24] In fact, although etiologic factors explaining crestal bone loss have not been completely elucidated, several factors have been described: surgical trauma,[4] microbiologic contamination at the implant-abutment microgap,[10] and the implant-abutment design.[4,25,26] Designs and surfaces are critical factors for determining the amount of peri-implant bone loss.[27–29]

In cases of cemented restorations, subgingival resin cement remnants might represent an additional factor regarding the peri-implant inflammatory response.[30]

Implant-abutment connections are based on a design that permits the abutment and implant to fit together, creating a microgap visible both microscopically and macroscopically. Micromovements at the implant-abutment interface have been described as direct and indirect factors affecting peri-implant bone stability.[31] This microgap not only allows for movement of bacteria and toxins to and from the abutment-implant external interface, but also allows for micromovements of the abutment within the implant.[32] Furthermore, micromovements can create abutment screw loosening and a "micropumping effect" that expels additional bacteria and toxins at the implant–soft tissue interface, including increased inflammatory cells at the osseous crest, causing soft tissue detachment and crestal bone loss.[32] Moreover, Stanford and Brand,[33] applying Frost's bone mechanostat theory to the bone-implant interface,[34,35] suggested that longitudinal implant stability was associated with the correlation between occlusal loading and strain-driven bone remodeling. Peri-implant hard tissue may adjust its mechanical strength and architectural connectivity by means of this interaction.[33,36]

Another possible factor involved in marginal bone loss is the stability of peri-implant soft tissues. Several studies[37–41] describe the need for keratinized tissue around the implant neck as a control factor for peri-implant bone loss.

According to Linkevicius et al[15,16,18] and De Almo et al,[17] soft tissue thickness around the implant neck seems to be correlated with peri-implant marginal bone loss, but this finding was not supported by the results of the present study. As mentioned earlier, some important differences in techniques used to measure the soft tissue thickness (histologic analysis instead of clinical method, use of blinded examiner) might explain the different study results. Moreover, in the present study, the role of soft tissue thickness was investigated when placing platform-switched implants with internal connections in the presence of at least 2 mm of stable keratinized gingiva around the implant neck. In addition, implants were submerged slightly below the crestal bone to avoid eventual exposure of the implant collar after the surgical phase, according to Vervaeke et al.[42]

In the presence of these factors, it is possible that soft tissue vertical thickness plays a minor role, but similar to other etiologic factors involved in marginal bone loss, the role of soft tissue thickness should be further investigated in clinical trials with larger sample sizes.

CONCLUSIONS

Within the limits of the present study, the initial mucosal thickness surrounding a bone-level platform-switching implant does not seem to influence the pattern of physiologic marginal bone loss.

ACKNOWLEDGMENTS

The authors report no conflicts of interest related to this study.

REFERENCES

1. Albrektsson T, Zarb G, Worthington P, Eriksson AR. The long-term efficacy of currently used dental implants: A review and proposed criteria of success. Int J Oral Maxillofac Implants 1986;1:11–25.
2. Canullo L, Fedele GR, Iannello G, Jepsen S. Platform switching and marginal bone-level alterations: The results of a randomized-controlled trial. Clin Oral Implants Res 2010;21:115–121.
3. Cochran DL, Nevins M. Biologic width: A physiologically and politically resilient structure. Int J Periodontics Restorative Dent 2012;32:371–373.
4. Pozzi A, Agliardi E, Tallarico M, Barlattani A. Clinical and radiological outcomes of two implants with different prosthetic interfaces and neck configurations: Randomized, controlled, split-mouth clinical trial. Clin Implant Dent Relat Res 2014;16:96–106.
5. Atieh MA, Ibrahim HM, Atieh AH. Platform switching for marginal bone preservation around dental implants: A systematic review and meta-analysis. J Periodontol 2010;81:1350–1366.
6. Strietzel FP, Neumann K, Hertel M. Impact of platform switching on marginal peri-implant bone-level changes. A systematic review and meta-analysis. Clin Oral Implants Res 2015;26:342–358.
7. Meloni SM, Jovanovic SA, Lolli FM, et al. Platform switching vs regular platform implants: Nine-month post-loading results from a randomised controlled trial. Eur J Oral Implantol 2014;7:257–265.
8. Gargiulo AW, Wentz FM, Orban B. Dimensions and relations of the dentogingival junction in humans. J Periodontol 1961;32:261–267.
9. Hermann JS, Buser D, Schenk RK, Higginbottom FL, Cochran DL. Biologic width around titanium implants. A physiologically formed and stable dimension over time. Clin Oral Implants Res 2000;11:1–11.
10. Broggini N, McManus LM, Hermann JS, et al. Peri-implant inflammation defined by the implant-abutment interface. J Dent Res 2006;85:473–478.
11. Müller HP, Eger T. Gingival phenotypes in young male adults. J Clin Periodontol 1997;24:65–71.
12. Claffey N, Shanley D. Relationship of gingival thickness and bleeding to loss of probing attachment in shallow sites following nonsurgical periodontal therapy. J Clin Periodontol 1986;13:654–657.
13. Maia LP, Reino DM, Novaes Junior AB, et al. Influence of periodontal biotype on buccal bone remodeling after tooth extraction using the flapless approach with a xenograft: A histomorphometric and fluorescence study in small dogs. Clin Implant Dent Relat Res 2015;17(suppl):s221–s235.
14. Berglundh T, Lindhe J. Dimension of the periimplant mucosa. Biological width revisited. J Clin Periodontol 1996;23:971–973.
15. Linkevicius T, Apse P, Grybauskas S, Puisys A. The influence of soft tissue thickness on crestal bone changes around implants: A 1-year prospective controlled clinical trial. Int J Oral Maxillofac Implants 2009;24:712–719.
16. Linkevicius T, Apse P, Grybauskas S, Puisys A. Influence of thin mucosal tissues on crestal bone stability around implants with platform switching: A 1-year pilot study. J Oral Maxillofac Surg 2010;68:2272–2277.
17. Suárez-López Del Amo F, Lin G-H, Monje A, Galindo-Moreno P, Wang HL. Influence of soft tissue thickness on peri-implant marginal bone loss: A systematic review and meta-analysis. J Periodontol 2016;87:690–699.
18. Puisys A, Linkevicius T. The influence of mucosal tissue thickening on crestal bone stability around bone-level implants. A prospective controlled clinical trial. Clin Oral Implants Res 2015;26:123–129.
19. Favero V, Lang NP, Ross F, Favero R, Baffone D, Botticelli D. Peri-implant tissues morphometry at SLActive surfaces. An experimental study in the dog. Clin Oral Implants Res 2016;27:993–998.
20. von Elm E, Altman DG, Egger M, et al. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: Guidelines for reporting observational studies. Int J Surg 2014;12:1495–1499.
21. Farina V, Zaffe D. Changes in thickness of mucosa adjacent to implants using tissue matrix allograft: A clinical and histologic evaluation. Int J Oral Maxillofac Implants 2015;30:909–917.
22. Laliberté F, Laliberté MF, Lécart S, Bousquet J, Klossec JM, Mounedji N. Clinical and pathologic methods to assess the long-term safety of nasal corticosteroids. French Triamcinolone Acetonide Study Group. Allergy 2000;55:718–722.
23. Brägger U. Use of radiographs in evaluating success, stability and failure in implant dentistry. Periodontol 2000 1998;17:77–88.
24. Albrektsson T, Dahlin C, Jemt T, Sennerby L, Turri A, Wennerberg A. Is marginal bone loss around oral implants the result of a provoked foreign body reaction? Clin Implant Dent Relat Res 2014;16:155–165.
25. Fickl S, Zuhr O, Stein JM, Hürzeler MB. Peri-implant bone level around implants with platform-switched abutments. Int J Oral Maxillofac Implants 2010;25:577–581.
26. Degidi M, Nardi D, Piattelli A. One abutment at one time: Non-removal of an immediate abutment and its effect on bone healing around subcrestal tapered implants. Clin Oral Implants Res 2011;22:1303–1307.
27. Hürzeler M, Fickl S, Zuhr O, Wachtel HC. Peri-implant bone level around implants with platform-switched abutments: Preliminary data from a prospective study. J Oral Maxillofac Surg 2007;65(suppl):s33–s39.
28. Oh TJ, Yoon J, Misch CE, Wang HL. The causes of early implant bone loss: Myth or science? J Periodontol 2002;73:322–333.
29. Calvo-Guirado JL, Ortiz-Ruiz AJ, Negri B, López-Marí L, Rodriguez-Barba C, Schlottig F. Histological and histomorphometric evaluation of immediate implant placement on a dog model with a new implant surface treatment. Clin Oral Implants Res 2010;21:308–315.
30. Wadhwani C, Piñeyro A, Hess T, Zhang H, Chung KH. Effect of implant abutment modification on the extrusion of excess cement at the crown-abutment margin for cement-retained implant restorations. Int J Oral Maxillofac Implants 2011;26:1241–1246.
31. Hermann F, Lerner H, Palti A. Factors influencing the preservation of the periimplant marginal bone. Implant Dent 2007;16:165–175.
32. Doundoulakis JH. The abutment/implant interface and its role in retention of crestal bone: A new paradigm. New York County Dental Society Dentists' Quarterly 2009;16:6–7.
33. Stanford CM, Brand RA. Toward an understanding of implant occlusion and strain adaptive bone modeling and remodeling. J Prosthet Dent 1999;81:553–561.
34. Frost HM. Bone "mass" and the "mechanostat": A proposal. Anat Rec 1987;219:1–9.
35. Frost HM. A 2003 update of bone physiology and Wolff's law for clinicians. Angle Orthod 2004;74:3–15.
36. Stanford CM, Schneider GB. Functional behaviour of bone around dental Implants. Gerodontology 2004;21:71–77.
37. Chung DM, Oh TJ, Shotwell JL, Misch CE, Wang HL. Significance of keratinized mucosa in maintenance of dental implants with different surfaces. J Periodontol 2006;77:1410–1420.
38. Bouri A Jr, Bissada N, Al-Zahrani MS, Faddoul F, Nouneh I. Width of keratinized gingiva and the health status of the supporting tissues around dental implants. Int J Oral Maxillofac Implants 2008;23:323–326.
39. Adibrad M, Shahabuei M, Sahabi M. Significance of the width of keratinized mucosa on the health status of the supporting tissue around implants supporting overdentures. J Oral Implantol 2009;35:232–237.
40. Kim YK, Kim SG, Oh HK, et al. Evaluation of peri-implant tissue in nonsubmerged dental implants: A multicenter retrospective study. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2009;108:189–195.
41. Schrott AR, Jimenez M, Hwang JW, Fiorellini J, Weber HP. Five-year evaluation of the influence of keratinized mucosa on peri-implant soft-tissue health and stability around implants supporting full-arch mandibular fixed prostheses. Clin Oral Implants Res 2009;20:1170–1177.
42. Vervaeke S, Dierens M, Besseler J, De Bruyn H. The influence of initial soft tissue thickness on peri-implant bone remodeling. Clin Implant Dent Relat Res 2014;16:238–247.