Critical Peri-implant Buccal Bone Wall Thickness Revisited: An Experimental Study in the Beagle Dog

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Abstract

**Purpose:** There is a lack of knowledge concerning the critical buccal bone thickness required for securing favorable functional and esthetic outcomes, conditioned to the dimensional changes after implant placement. A preclinical study was therefore carried out to identify the critical buccal bone wall thickness for minimizing bone resorption during physiologic and pathologic bone remodeling. **Materials and Methods:** A randomized, two-arm in vivo study in healthy beagle dogs was carried out. The first group of dogs was sacrificed 8 weeks after implant placement for histomorphometric examination of postsurgical resorption of the buccal bone wall. The second group of dogs was monitored during three ligature-induced peri-implantitis episodes and a spontaneous progression episode. Morphometric and clinical variables were defined for the study of physiologic and pathologic buccal and lingual bone loss. **Results:** Seventy-two implants were placed in healed mandibular ridges of 12 beagle dogs. Two groups were defined: 36 implants were placed in sites with a thin buccal bone wall (< 1.5 mm), and 36 were placed in sites with a thick buccal bone wall (≥ 1.5 mm). No implants failed during the study period. For the great majority of the histomorphometric parameters, a critical buccal bone wall thickness of at least 1.5 mm seemed to be essential for maintaining the buccal bone wall during physiologic and pathologic bone resorption. Suppuration (+) and mucosal recession (-) were more often associated with implants placed in sites with a thin buccal bone wall. **Conclusion:** A critical buccal bone wall thickness of 1.5 mm at implant placement is advised, since a thicker peri-implant buccal bone wall (> 1.5 mm) is exposed to significantly less physiologic and pathologic bone loss compared with a thinner buccal bone wall (< 1.5 mm). Int J Oral Maxillofac Implants 2019. doi: 10.11607/jomi.7657

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Introduction

Optimal three-dimensional (3D) implant placement has been regarded as a key element for ensuring satisfactory long-term esthetic and functional outcomes (1-4). Bone quantity and density clearly determine primary implant stability, which ultimately leads to osseointegration. Several techniques have been proposed to increase the width and height of the alveolar ridge. Such techniques are suggested to be particularly important in posterior implant sites, where more volume support (i.e., involving a wider dental implant) is needed to withstand the occlusal forces (5), and also in the anterior maxilla, where augmented bone contour is sought in order to satisfy the aesthetic demands. Interestingly, an early report indicated that not only adequate bone structure for harboring the implant is required, but also a buccal bone wall thickness of > 1.8 mm, since expected bone loss after implant placement may compromise implant outcomes (1). Likewise, Barone et al. reported more favorable peri-implant hard and soft tissue stability in the presence of ≥ 1 mm of buccal bone wall (6). In agreement with this, Covani et al. observed that in adequate alveolar bony crests, the magnitude of bone resorption after implant placement in a healed socket was roughly 3 mm (7). In contrast, recent data have demonstrated adequate peri-implant crestal bone levels with implants placed in limited buccal-oral alveolar bone (≤ 4.5 mm) during three years of loading (8).

It is of paramount importance to conceive the architecture of the buccal bone wall following implant bed preparation as a structure comprised of an external lining of cortical bone and an internal wall composed of cancellous bone. This is essential to understand the 3D dimensional changes that occur following implant surgery, which constitutes an example of trauma to the hard and soft tissues. Early findings in the field of Periodontology illustrated the substantial hard and soft tissue histological, clinical and radiographic changes that occur during repair after osseous surgery (9-13). In the past 15 years, tissue alterations in post-extraction sockets have been extensively examined in preclinical (14-17) and clinical studies (18, 19). These studies led to improved understanding of tissue biology, since the interruption of blood supply provided from the bundle bone seems to play a pivotal role in postsurgical bone resorption. Such
Catabolic changes lead to greater osteoclastic activity, in particular on the buccal aspect of the extraction socket, due to a predominantly thin facial bone wall (15, 16).

Tissue biology in healed implant sites is much less understood, and preclinical studies are sparse. There is a potential risk that a thin buccal bone wall at implant placement will undergo marked resorption during healing, leading to significant vertical bone loss and causing a peri-implant bone defect in the crestal area prior to functional loading. This entails exposure of the micro-rough implant surface to the peri-implant sulcus in the supracrestal area. Micro-rough titanium implant surfaces are predominantly used today in implant treatments, since these have better anchorage in bone and offer shorter healing periods when compared to the original smooth, machined titanium surface implants (20-22). On the other hand, micro-rough implant surfaces, due to their topographical characteristics, are more prone to contamination by putative microorganisms (23). In consequence, faster tissue breakdown and less efficient disease resolution after treatment (24) is expected in modified surfaces when the implant is exposed to biofilm through the peri-implant sulcus. This was recently confirmed in a clinical study showing that small buccal bone defects can indeed progress to greater vertical bone loss and to more marginal bone loss compared to implants inserted in an intact circumferential bony housing without dehiscences (25).

While there has been a substantial increase in research on the implications of systemic factors upon peri-implant tissue stability, little is still known about local factors such as the thickness of the buccal bone wall at implant placement. Hence, the purpose of the present preclinical study in Beagle dogs was to examine the influence of peri-implant buccal bone thickness upon: (a) postsurgical physiological bone loss following an 8-week healing period; and (b) pathological bone loss caused by a ligature-induced peri-implantitis model followed by a spontaneous progression phase.
Material and Methods

The present study was approved by the Ethics Committee of the Health and Social Policy Council (Government of Extremadura, Spain [#2017209030001787]) in compliance with the pertinent local and European Union standards (REGA ES 100370001499). Furthermore, the study followed the ARRIVE guidelines (26).

Experimental design

Based on an a priori power analysis considering the dog as the independent variable (27), a randomized, two-arm *in vivo* study in healthy Beagle dogs was designed. The first group of dogs were sacrificed 8 weeks after implant placement for histomorphometric examination of postsurgical resorption of the buccal bone wall. The second group of dogs were monitored during three ligature-induced peri-implantitis episodes and a spontaneous progression episode to explore the influence of critical buccal bone thickness in relation to pathological bone loss. A surgeon (AM) accredited by the American Board of Periodontics carried out all the surgeries. Housing conditions as well as the protocols carried out for pre-anesthesia, local anesthesia and postoperative pain control medication have been reported elsewhere (28, 29).

Fluorescence labeling (calcein 10 mg/kg body weight; product number: C0875; Sigma-Aldrich, MO, USA) was performed at implant placement. This fluorescent dye allowed us to determine the exact location of the calcified tissue and of first bone-implant contact at the end of physiological bone remodeling. The animals received an intramuscular injection of the dye in isotonic 1.4% sodium bicarbonate solution once filtered (20 mg/ml).

Tooth extraction and implant placement protocols

The protocols referred to tooth extraction and implant placement have been detailed elsewhere (28, 29). Briefly, each dog underwent atraumatic mandibular premolar and molar extractions (PM3, PM4, M1) in the hemiarches. Implant placement was carried out 8 weeks later.
Following flap elevation through a crestal incision, a round burr was used to flatten the edentulous ridge to secure a wide ridge. Six implants were inserted per animal (3 per hemimandible). Two different implants based on their coronal design were tested: rough up to the top (R) (TiCare Inhex Mini 3.3 x 8 mm) and hybrid (H) (TiCare Inhex Mini 3.3 x 8 mm) with 1.5 mm of machined surface at the coronal portion. All implants were placed according to the recommendations of the manufacturer and were seated in an equicrestal position. A cover screw was then placed. The flaps were adapted around the healing abutments with interrupted sutures to achieve primary wound closure (Figure 2).

Ligature-induced peri-implantitis

The detailed ligature-induced peri-implantitis protocol can be found elsewhere (28, 29). Briefly, after 8 weeks of healing, silk ligatures (3/0) were placed looping the apical portion of the implant-supported healing abutments and changed three weeks apart for a total of three events (T1-T3). Finally, all ligatures were removed after three weeks with sustained deprivation of oral hygiene (T4).

Clinical assessment

Clinical assessment was carried out with a North Carolina probe (Hu-Friedy, Chicago, IL, USA) to evaluate four different clinical parameters: probing depth (PD), bleeding on probing (BOP), mucosal recession (MR) and suppuration (SUP), as described elsewhere (28, 29).

Buccal/lingual alveolar bone thickness

The Aurora Tracking System†† was used to assess the distance between the implants and the buccal/lingual bony plates (28). The tracking principles and methodology have been extensively described elsewhere (28, 29).

Histomorphometric analysis

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Details on the histomorphometric analysis can be found elsewhere (28, 29). The fluorescence images were first captured at magnifications up to x40 with a blue filter at a wavelength of 495–520 nm (U-MNB2, Olympus). Bone growth was identified by very intense green fluorescence. The slides were then stained (30), and morphometric analysis was performed by a previously calibrated and blinded examiner (intra-examiner agreement: > 90%) under x40 magnification of the original magnification, using specific software (ImageJ, National Institutes of Health).

The following measurements were made to evaluate physiological and pathological bone loss (Figure 1):

- Bone-to-implant contact (BIC, as %), defined as the bone surface in direct contact with the implant divided by total implant length, starting from the shoulder.
- Internal buccal bone loss (BL-I, in mm), defined as linear bone loss from implant shoulder to the most coronal point of the BIC.
- External buccal bone loss (BL-E, in mm), defined as linear bone loss from implant shoulder to the most coronal and external point of the buccal bone wall.
- Internal lingual bone loss (LL-I, in mm), defined as linear bone loss from implant shoulder to the most coronal point of the BIC.
- External lingual bone loss (LL-E, in mm), defined as linear bone loss from implant shoulder to the most coronal and external point of the lingual bone wall.
- Presence of fluorescence (FL, expressed as +/-) and its length (FL, in mm) along the implant surface in the peri-implant defect area.

**Radiographic assessment**

As explained elsewhere (28), the images were obtained by computed tomography (CT)** under general anesthesia immediately after each clinical examination and assessed with radiological planning software (Osirix MD, Berne, Switzerland).

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** Phillips Medical System, Eindhoven, The Netherlands
**Statistical analysis**

A linear regression model was estimated by generalized estimation equations (GEEs) to explore the histomorphometric variables and groups according to buccal bone thickness (thin buccal bone wall [tbb] versus thick buccal bone wall [TBB]). Wald's chi² statistic yielded the 95% confidence interval. Likewise, a linear regression model was estimated by GEE to explore the odds for positive immunofluorescence. The Mann-Whitney U-test was carried out to analyze the effect of the morphological variables or length of immunofluorescence positivity in both study groups. The level of statistical significance was established at $\alpha = 0.05$.

The receiving operator characteristic (ROC) curve was calculated to test the sensitivity and specificity of critical buccal bone thickness in assessing the magnitude of physiological and pathological bone remodeling. Binary logistic regression models with a dependent variable (group: TBB versus tbb) and independent variables (morphometric variables) were estimated to assess prognostic accuracy.

**Results**

Overall, 72 implants were placed in healed mandibular ridges of 12 Beagle dogs. Two groups were defined: 36 implants were placed in sites with a thin buccal bone wall (tbb < 1.5 mm), and 36 were placed in sites with a thick buccal bone wall (TBB $\geq$ 1.5 mm). There were no adverse events during the study period, and healing proved uneventful after each surgical phase.

**Critical buccal bone wall thickness and implant survival rate**

No implants failed during the study period. The influence of critical buccal bone wall thickness upon implant survival was therefore null.

**Critical buccal bone wall thickness for physiological peri-implant bone loss**

The influence of critical buccal bone wall thickness in relation to physiological bone loss was studied in the dogs sacrificed 8 weeks after implant placement ($n_{\text{dogs}} = 6$; $n_{\text{implants}} = 36$). As
hypothesized, the dispersion plots showed the critical threshold for the prevention of excessive postsurgical buccal bone loss to be 1.5 mm (Figure 4). Strong statistical significance was demonstrated for all parameters except BIC (p=0.977) (Figure 5).

**Critical buccal bone wall thickness for bone loss induced by peri-implant infection**

The influence of critical buccal bone wall thickness in relation to pathological bone loss was studied in the dogs sacrificed after three episodes of ligature-induced peri-implantitis followed by an episode of spontaneous progression (n_dogs= 6; n_implants= 36). The dispersion plots likewise showed the critical threshold for the prevention of excessive pathological buccal bone loss to be 1.5 mm (Figure 4). Strong statistical significance was demonstrated for all the parameters except LL-I, which showed a tendency towards greater bone loss in the TBB group (p=0.10) (Figure 5).

**Critical buccal bone wall thickness and peri-implant clinical parameters**

The following parameters referred to tbb (< 1.5 mm) reached statistical significance: BOP-B (p=0.010), SUP-MB (p=0.02) and mean SUP (p=0.01). Moreover, mean MR(-) (p=0.001), MR-MB(-) (p=0.007) and MR-B(-) (p=0.007) were significantly associated to TBB (> 1.5 mm). In other words, tbb was correlated to greater clinical MR(+) values (Figure 6).

**Critical buccal bone wall thickness and fluorescence images**

The incidence of FL(+) on the buccal aspect was 16.7% for tbb and 30.6% for TBB. On the lingual side, the incidence was 33.3% for tbb and 19.4% for TBB. Thus, at buccal level, TBB was associated to a three-fold higher incidence of FL(+) versus tbb (odds ratio [OR] = 3.14; p=0.005). In contrast, on the lingual aspect, the incidence of FL(+) in the TBB group was one-third of that recorded in the tbb group (OR=0.32; p=0.06). The comparative analysis of TBB versus tbb showed strong statistical significance on the buccal aspect (p=0.001), while a tendency towards statistical significance was observed on the lingual aspect (p=0.06). However, no statistical significance was recorded on analyzing FL(+) length (Figure 3).
Critical buccal bone wall thickness and radiographic marginal bone loss

As published elsewhere (28), radiographic peri-implant bone loss was substantially greater at the buccal sites than at the lingual sites in each study period, reaching a difference of ~1 mm at the last assessment timepoint (T4).

Critical buccal bone thickness and implant design

In relation to physiological bone resorption, H implants showed greater lingual loss (LL-I) in the tbb group (p=0.01), though no significant correlations were observed with respect to the rest of the histomorphometric variables. In relation to pathological bone resorption, no influence on the part of implant design was observed.

Sensitivity and specificity of critical buccal bone thickness in predicting bone loss

Regarding the prediction of physiological bone resorption, the highest sensitivity and specificity values were 100% and 94.1%, respectively for BL-I when critical buccal bone thickness was set at 1.5 mm with an area under the curve (AUC) of 0.99. Likewise, regarding the prediction of pathological bone resorption, the highest sensitivity and specificity values were 77.8% and 88.9%, respectively for BL-I (AUC=0.81) (Supplementary Figure 1).

Discussion

Main findings

The critical bone dimensions for implant placement are of great interest in implant dentistry, since a strong impact upon long-term tissue stability has been observed. Given the burden of peri-implant diseases, the question to be addressed is: What is the buccal bone wall thickness needed to minimize postsurgical bone loss during healing and hence guarantee embedding of the micro-rough implant surface in bone over the long term? Findings from the present study indicate that a buccal bone wall measuring < 1.5 mm in thickness is not enough to maintain the wall during a healing period of 8 weeks. In this regard, all the investigated morphometric
variables yielded strong statistical significance (p<0.001) favoring less vertical and horizontal buccal bone changes in thicker (> 1.5 mm) buccal bone walls, leading to significantly less frequent exposure of the micro-rough implant surface. In the second group of animals, involving ligature-induced peri-implantitis followed by a spontaneous progression phase, the “critical buccal bone thickness” of > 1.5 mm was able to minimize the progression and severity of peri-implantitis in the presence of biofilm.

**Understanding the biological mechanisms behind the findings**

The observed vertical bone loss around implants with a thin buccal bone wall (< 1.5 mm) during an 8-week healing period is mediated by avascular necrosis (31). In a healed ridge of the mandible, the alveolar process is predominantly composed of cortical bone at the outer side, whereas the central portion of the mandible is characterized by a more cancellous structure. The cortical bone receives a blood supply branched from the outside through blood vessels of the periosteal surface, and from the inside from the endosteum (32). When an implant is inserted with an open-flap procedure, as in the present study, the blood supply from both sources is disrupted. Elevation of the periosteum eliminates the periosteal blood supply from the outside. The same occurs from the inside, since insertion of the implant interrupts the endosteal blood supply. This phenomenon of avascular necrosis is well known in bone biology (33). Briefly, within 12 hours after disruption of the blood supply, the hematopoietic cells (which are particularly sensitive to low oxygen levels) die. This event is followed by the death of bone cells such as osteocytes and osteoblasts, leading to more noticeable osteoclast activity (34). In consequence, in the presence of an implant placed < 1.5 mm from the buccal bony flange, the blood supply might not be sufficient to repair the bone on the buccal aspect. In response, osteoclasts activated by the RANKL/RANK pathway and mediated by a transcription factor (nuclear factor of activated T cells) cause buccal bone resorption (33).

In addition, findings from this study indicate that a thicker buccal bone wall (> 1.5 mm) provides a protective mechanism against the progression of peri-implantitis. Interestingly, the
vast majority of the morphometric variables evidenced less advanced peri-implantitis lesions through vertical loss in thicker buccal bone walls (>1.5 mm). This was in concordance with the clinical parameters investigated after the induction and spontaneous progression of peri-implantitis. As commented, peri-implant diseases are conceived as infective disorders where, in the presence of a putative anaerobic environment in susceptible individuals, chronification of the condition can be expected. Given that the scenarios of thinner buccal bone walls presented with significantly greater buccal bone loss, a longer junctional epithelium is expected to be formed after abutment placement. Considering the weak fibrous hemidesmosome junctional attachments existing around dental implants, plaque and byproducts tend to migrate more apically, inducing more pronounced tissue breakdown.

Interestingly, it should be further highlighted that the dimensions of the lingual bone cannot be neglected. A tendency towards greater lingual bone loss in TBB due to the reduced lingual bone thickness was observed. This fact indicates that a critical lingual thickness may exist for minimizing bone loss during the physiological bone remodeling process.

**Agreements and disagreements with the findings of the present study**

Much is known about physiological bone resorption secondary to periodontal surgery (9, 11, 12), tooth extractions (15, 35) or implants placed in fresh alveolar sockets (36). In contrast, there is a lack of knowledge about bone resorption following implant insertion in healed ridges. It has been suggested that a buccal bone thickness of 1 mm (37) or 2 mm (38) is a crucial requirement for minimizing such resorption. Spay et al. postulated that the “critical buccal bone thickness” was 1.8 mm with the aim of minimizing buccal bone resorption and significantly decreasing the implant failure rate during healing (1). Cardaropoli et al. in turn demonstrated that a mean reduction of 0.4 mm in buccal bone thickness occurred during early healing, while further buccal bone loss amounting to 0.9 mm before crown delivery and followed by 0.7 mm at one year post-loading was to be expected (35). More recently, Yoda et al., in a cone-beam computed tomography (CBCT) study, found a minimum initial thickness of 1.5 mm to be
crucial for reducing horizontal bone resorption and for diminishing the mechanical-biological stimuli, which in maxillary anterior sites may have a further impact upon the aesthetic outcomes (39). Contrarily, a histomorphometric study in humans failed to show an association between buccal bone wall thickness > 1 mm and bone resorption. In fact, horizontal bone resorption was shown to be 0.3 mm and 1 mm when implants were placed in 1 mm and 2 mm of residual buccal bone, respectively (40). In agreement with the above, an experimental study in dogs demonstrated that after three months of healing, the top of the bony crest in the group with 1 mm of residual buccal bone thickness migrated apically 0.3 mm, whereas in the group with 2 mm of residual buccal bone thickness the bone crest was located at 0.57 mm (41). In line with such findings, Temmerman et al. supported the feasibility of medium term (3 years after functional loading) hard and soft tissue stability in crests of limited bone width (≤ 4.5 mm) (8). In contrast, the present study showed that the “critical buccal bone thickness” threshold can be defined as 1.5 mm, since thinner buccal bone thicknesses expose the implants to an increased risk of excessive physiological and pathological bone loss - thereby potentially jeopardizing the long-term clinical outcome.

The 2017 World Workshop in Periodontology and Peri-Implant Diseases has identified implant positioning as a potential confounder for peri-implant diseases (42, 43). It seems reasonable to claim that implant position and design of the prosthesis should ease access for self-performed oral hygiene and professionally administered plaque removal. Moreover, data from this preclinical study suggest that when implants are placed too buccal (< 1.5 mm from the buccal flange), there is an increased risk of bone dehiscence during the early phases of postsurgical healing that could potentially lead to biofilm accumulation on the micro-rough surface (23) and consequently to more aggressive progression of peri-implantitis (24).

In this regard, it is advisable to avoid procedures that cannot guarantee adequate implant surface seating within the alveolar envelope, keeping a critical safety buccal bone thickness of > 1.5 mm, such as flapless surgery or inadequate three-dimensional implant placement. In the present
experimental study, fluorescent dye was incorporated to the progressing mineralization front through a chelation process. This indicated that FL(+) surfaces along the implant axis within the peri-implant defects were attributable to the ligature-induced peri-implantitis phenomenon, and not to excessive physiological bone resorption. Present findings showed TBB to be associated to three-fold greater FL(+) than tbb. Hence, this is consistent regarding excessive bone resorption during the early stages of healing in implants placed in sites with a thin buccal bone wall.

**Clinical implications and limitations**

When implants are inserted in a healed ridge, the buccal and lingual bone wall should be at least >1.5 mm in thickness in order to expect minimal dimensional changes that do not compromise the integrity of the alveolar ridge. In other words, the crest width should measure at least 3 mm more than the diameter of the implant to be inserted. Most often, the lack of bone volume occurs on the buccal aspect of the implant to be inserted. Accordingly, it is advised that simultaneously, the resulting bone defect should be laterally augmented using the principles of guided bone regeneration, in order to compensate the process of physiological bone resorption.

The findings of this experimental study shed light on the influence of buccal bone thickness upon the physiological and pathological events that lead to bone resorption. Nevertheless, the findings of the present study should be interpreted with caution, since the experimental model involved may differ from the scenario found in humans. Hence, the concept of “critical buccal bone thickness” should be validated in a prospective controlled human study. On the other hand, the findings are not applicable to all case scenarios. For instance, it has been reported that bone in the maxilla, in particular in atrophic sites, is comparatively more porous in architecture (44). In such clinical scenarios where there is a thinner cortical layer, the concept of critical buccal bone thickness for might be challenged. Furthermore, it is yet to be demonstrated whether the critical buccal bone thickness established in the present study can be applied in other approaches, including different drilling protocols, flapless interventions and implant macro-geometries. In this sense, it is important to note that in general terms, implant design did not
significantly influence histological bone resorption in the course of the present experimental study. Nevertheless, the potential of hybrid implants should be tested in controlled clinical trials in naturally occurring peri-implantitis, given the influence of surface micro-characteristics upon the course of disease under spontaneous progression (24, 45, 46).

Conclusions

The critical buccal bone thickness for minimizing physiological and pathological bone resorption after implant placement is 1.5 mm. In other words, a buccal bone thickness > 1.5 mm proves more effective in compensating the dimensional changes occurring after implant placement and the progression of peri-implantitis. However, the critical buccal bone thickness does not seem to influence implant survival rate.
References


Figure legends

Figure 1. Schematic representation of the morphometric variables.
Figure 2. Workflow representing the steps from tooth extraction to implant placement following a random buccolingual position.
**Figure 3.** Representative hematoxylin-eosin (HE) stained histological samples exemplifying physiological bone remodeling and fluorescence images displaying peri-implant bone loss attributable to ligature-induced peri-implantitis, particularly in the thin buccal bone wall group.
Figure 4. Plots of the morphometric variables showing 1.5 mm to be the critical buccal bone wall thickness (BBT) threshold required to minimize physiological and pathological bone resorption.
**Figure 5.** Bar graph of the morphometric parameters corresponding to postsurgical and peri-implantitis-induced bone loss in both study groups.
**Figure 6.** Comparison of the clinical parameters at timepoint T4 between thin versus thick buccal bone walls.
Supplementary Figure 1. Receiver operating characteristic (ROC) curve for physiological (A) and pathological bone loss (B).