# Peri-implant tissues health and its association to the gingival phenotype

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**Abstract**

Peri-implant tissue is an adaptation of the masticatory mucosa to the different implant systems placed in the oral cavity. The lack of root cement to anchor gingival fibers to the surface of the implant is responsible for the parallel direction of the fibers around it. The absence of connective attachment between the mucosa and the implant may suggest a deficiency of the structural defenses in the region and may be associated with the more rapid progression of peri-implantitis than of periodontitis. Several studies have evaluated the importance of epithelial connections to form an adequate seal around implants. Other discussions have focused on the evaluation of whether peri-implant gingival health may be correlated with the presence of a specific amount (height and thickness) of keratinized mucosa. This study evaluated the association of the structural role of the soft tissue and the effect of gingiva phenotype on peri-implant health. The studies that were reviewed stressed the importance of a good biological seal around the implant system, the protective function that the structures of this tissue provide to the bone-implant interface, and the discussion about the need to have a band of keratinized mucosa around tooth implants to ensure a better prognosis. Current studies point to the need to conduct further investigations to evaluate the effect of the clinical characteristics of soft peri-implant tissues so that peri-implant health may be ensured and preserved.

**Keywords:** Mucositis. Peri-implantitis. Keratinized mucosa.

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## Introduction

Some factors associated with amount and quality of bones, soft tissues (keratinized mucosa), host response, type of treatment applied to implant surface and biomechanical events (occlusal overload) are very important and directly associated with implant success or failure.[1]

The masticatory mucosa, composed of gingiva and palate mucosa, is covered by keratinized squamous epithelium and dense fibrous connective tissues (lamina propria). In the gingiva, the lamina propria is attached directly to the alveolar bone through the periosteum and, in the supracrestal portion, to the tooth root.[2] In implants, the connective tissue fibers run parallel to the implant surface and form a collar.

Although the soft tissues around teeth and implant have aspects in common, the direction of their connective tissue fibers is different. The string of soft tissue around the implants is a critical barrier that ensures the protection of bone adjacent to the implant.[3] The preservation and stability of the bone walls around the implant depend on the formation of a functional barrier on the abutment/implant interface (transmucosal), important for implant protection against bacterial invasion.[4]

In contrast, some authors report that the keratinized mucosa around implants is not necessary in patients that have a good oral hygiene.[3,5,6]

Although not conclusive, some authors have suggested that the lack of an adequate zone of masticatory mucosa may be a barrier to good hygiene and may provide poor protection to the support teeth and the implant against injuries caused by frictional forces during mastication and brushing and against the accumulation of bacterial plaque.

This study compared findings in the literature about the actual effect of peri-implant phenotype and its correlation which gingival health and long-term implant prognosis in relation to inflammation.

## Literature review

### The effect of bacterial plaque on inflammation

For several years, periodontal disease (PD) was believed to be the only entity caused by the accumulation of bacterial plaque that led to gingivitis, which, if not treated, may lead to bone loss and, consequently, periodontitis. Dentistry students underwent a model of experimental gingivitis that included an initial period of intensive plaque control, a period of plaque induction, with controlled onset and progression of the disease, and a final period of new plaque control. All individuals had rapid plaque accumulation and different changes in microbiota, followed by gingival inflammation. The study clearly demonstrated that gingivitis in human beings may be produced by bacterial plaque and may be controlled after plaque removal.[7]

The literature about this topic shows a correlation between the presence of bacterial plaque and periodontal disease (PD), although plaque does not necessarily result in PD. Longitudinal studies showed that individuals without good oral hygiene standards had different patterns of bone loss, or no bone loss.[8] This demonstrated that, for the development of PD, other factors must be present in addition to bacterial plaque.

Some individuals have intrinsic characteristics that trigger the most severe form of the disease, such as environmental factors, smoking, systemic diseases or genetic changes.

One of the causes of osseointegrated implant failure is bacterial infection.[1]

Implantology recognizes the existence of groups of individuals that have increased risks to osseointegration and applies the concepts developed by Periodontics.[9]

The process of infection of the peri-implant sulcus first leads to the formation of peri-implant mucositis, which may be defined as an inflammation of peri-implant soft tissues without bone loss. In some situations, mucositis may progress and turn into peri-implantitis, which is peri-implant inflammation with bone loss. Both processes are associated with bacteria that are pathogenic for the periodontium.[10]

An experimental study with beagles was conducted to evaluate inflammatory changes of the peri-implant mucosa and compare it with periodontal changes. After 3 months of plaque accumulation, clinical examinations showed that the peri-implant gingiva was edematous, red and bleeding at probing, and that the lesion in the peri-implant mucosa grew and extended more apically than in the gingival tissue. This study showed that mucositis may occur due to plaque accumulation, and that the peri-implant mucosa was less efficacious than gingiva in preventing plaque-associated lesions.[11]

In contrast, as study of experimental mucositis in human beings collected biopsies of periodontal and peri-implant gingival tissues of 12 people after a time of plaque control and then after 21 days of no oral hygiene. The authors found that plaque formation was associated with clinical signs of inflammation and more lesions to soft tissues with variable rates of cell markers. However, there were no significant differences in the location of teeth and implants in both the first sample and after 21 days.[6]

A study with monkeys showed that, in the presence of inflammation, peri-implant tissues were more susceptible to probing, and the tip of the probe reached a point closer to the bone than in inflamed periodontal tissues. These results suggest a greater fragility of the peri-implant tissue when associated with marginal inflammation than of periodontal tissues in the same clinical condition.[12]

In a review study that collected clinical, radiographic and biochemical factors to control peri-implant conditions, the parameters used by the authors to evaluate peri-implant health and disease severity were presence of plaque, macroscopic aspect of the mucosa, depth of peri-implant probing, presence and width of keratinized mucosa, analysis of fluid of the peri-implant sulcus, suppuration, mobility, discomfort and radiographic follow-up. The authors showed that, when oral hygiene was satisfactory, the characteristics of the mucosa have little influence on long-term implant success. However, they admitted that inadequate oral hygiene may lead to an increase of tissue loss around the implant in the area of alveolar mucosa when compared with regions of keratinized tissues. The authors also found that oral hygiene procedures are more easily performed when there is an appropriate band of keratinized mucosa.[13]

## Biological periodontal and peri-implant distances

The first study about biological distance evaluated the dimensions and associations of the dentogingival junction in autopsies of human specimens. That study established that there is a proportional dimensional association within a region of +2.73 mm, from the level of the alveolar bone crest to the level of the gingival

margin, and including the connective attachment, the junctional epithelium and the sulcus epithelium. In their study, 325 measurements were made in clinically normal specimens. The authors found a great consistency in the dimensions of several components:

a) Sulcus depth was 0.69 mm;
b) Junctional epithelium covered 0.97 mm;
c) Mean connective attachment was 1.07 mm.

The most consistent finding was recorded for connective attachments, whose mean measure was 1.07 mm, ranging from 1.06 to 1.08 mm. The mean combined value of connective attachment and junctional epithelium was 2.04 mm, and this was classified as the "biological distance".[14]

The epithelium in the sulcus has been described as the extension of the oral gingival epithelium whose coronary limit is the height of the free marginal gingiva, and its apical limit, the surface of the junctional epithelium.[15]

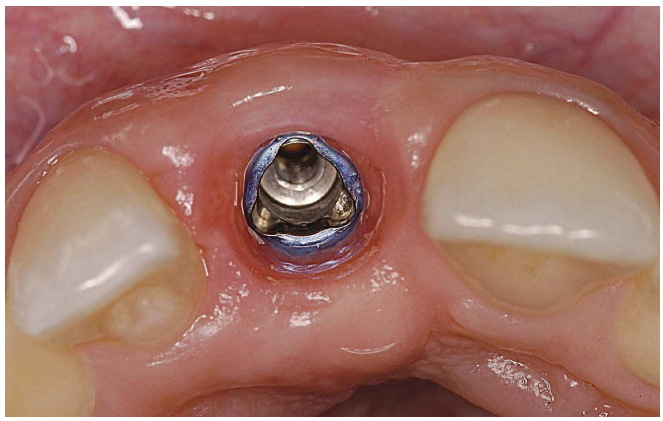

**Figure 1** - Peri-implant tissue with pink color, firm consistency and good thickness of keratinized mucosa.

The junctional epithelium is the tissue that joins the tooth in one side and the oral sulcus epithelium or connective tissue in the other, and forms the basis of the clinical gingival sulcus. Its structure and function are significantly different from those of the gingival epithelium. In several aspects, the junctional epithelium clearly seems to be a unique biological system.[16]

The gingival connective tissue may be dense and fibrous, with a complex functional orientation developed gradually during tooth eruption, which is later modified due to functional demands. The structural orientation of this tissue is appropriate to support the physical stresses of mastication and deglutition. The fibers are intertwined, and several are not even attached to the tooth surface. The function of the fibers is to stabilize the gingiva in relation to the alveolar process and the tooth and, secondarily, to stabilize the tooth to the bone. The circumferential distribution of the fibers (circular ligament) keeps the junctional epithelium in close contact with the tooth and helps to keep the epithelium sealed to the tooth, while interdental fibers help to stabilize the teeth.[17]

The evaluation of clinically healthy soft tissues around teeth and implants reveals that both are pink and consistent. The two types of tissue also have several microscopic characteristics in common, such as keratinized oral epithelium in continuation of junctional epithelium in teeth and implants and a measure of about 2 mm. Epithelium is separated from alveolar bone by a high area of connective tissue of about 1 mm.[18]

Therefore, a biological space of 3 to 4 mm above the bone is necessary and defined by about 2 mm of an epithelial component and 1 to 1.5 mm of connective tissue. A 2-year longitudinal analysis of Branemark implants to evaluate changes in the position of the margin of peri-implant soft tissues found that the mean value of recession was greater for implants with a small band of

keratinized mucosa only in the first 6 months, but the examination at 18 months revealed that the regions with little keratinized mucosa at baseline had a lower mean recession value than the regions with a greater amount of keratinized mucosa. According to the authors, this tissue change is not associated with inflammation. Areas with a greater probing depth had greater recession because of remodeling to stabilize the biological dimensions of supraosseous soft tissues. We may conclude that both soft tissues and recession are not significantly affected by the amount or mobility of marginal tissue, which confirms studies that found that the alveolar mucosa has the capacity of protecting the bone surrounding the implant, similarly to the masticatory mucosa.[3]

A study with dogs, whose internal portion of soft tissue surrounding the implant was removed to decrease its biological distance from the implant, demonstrated that there was bone resorption in those areas to ensure adequate fixation of soft tissue around the implants and reestablish the junctional epithelium, which suggests that a certain mucosa thickness is necessary to prevent bone remodeling around implants.[19]

In confirmation of the study described above, the biological distance of 3 different implant systems was histologically evaluated, and results showed that their morphological characteristics were similar. The biological distance was 3.03 to 3.15 mm, the distance of the junctional epithelium from the mucosa margin ranged from 1.6 to 4.3 mm, and the height of the connective tissue ranged from 1 to 1.5 mm. There was dense collagen with lithe vascular structure and dispersed inflammatory cells. Their conclusion was that the mucosa adjacent to the alveolar crest follows the same pattern. Sites with angular bone defects also have a thin mucosa. Therefore, to promote an adequate attachment of epithelial and connective tissue, a minimal amount of peri-implant mucosa is necessary.[21]

In addition, the composition of connective tissue between the mucosa and titanium implants was analyzed by authors that divided it, for analysis, into two portions within the adjacent connective tissue. The one closer to the implant characteristically had few blood vessels and abundant fibroblasts, and the one more lateral, had more vessels and fibers and fewer fibroblasts.

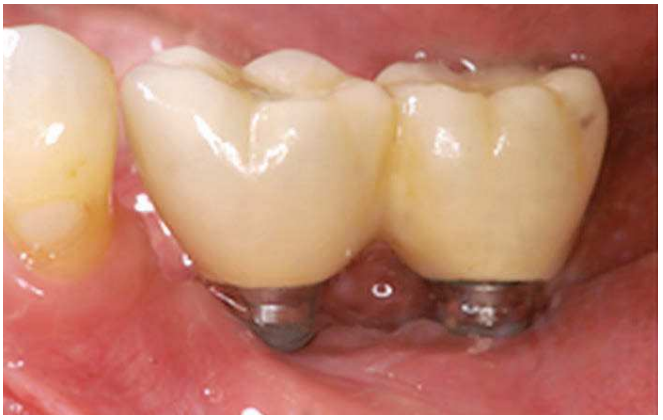

**Figure 2** - Illustration of two implants in the left posterior inferior region, showing soft tissue with absence of keratinized mucosa and presenting gingival recession.

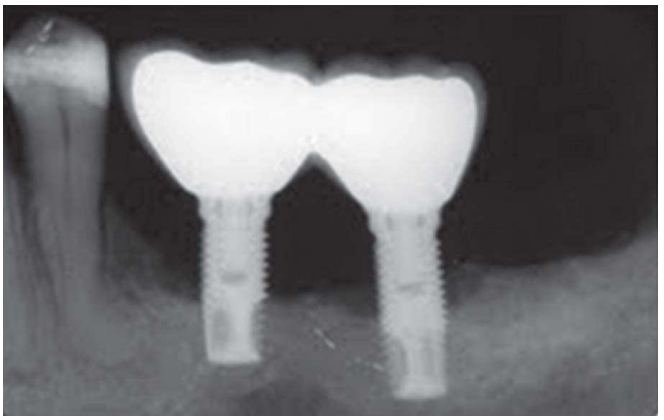

**Figure 3** - Radiographic image of Figure 2, showing excessive bone loss to the mid third of implants.

These findings suggest that a barrier rich in fibroblasts close to the titanium surface plays a role in maintaining adequate sealing between the oral environment and the peri-implant bone.[22]

Osseointegrated implants have few functional and anatomic barriers than natural teeth. Adhesion occurs only by means of the junctional epithelium. The presence of keratinized mucosa seems to promote such sealing.[23]

## The association between keratinized mucosa and mucositis

Although the masticatory mucosa adjacent to teeth is similar to that around implants, they have structural differences that may affect the health of the marginal tissues. While the final sulcus epithelium (junctional epithelium) seems to end at a similar distance to the bone crest (1 to 1.5 mm) both in teeth and in implants, the orientation of the supracrestal collagen fibers, however, is different. The lack of root cement to anchor gingival fibers to the surface of the implant results in a parallel orientation of the fibers around it, instead of the perpendicular orientation found around teeth.[18]

The formation of tissue defense in the host (granulation tissue) begins in the narrow layer of vascularized connective tissue below the junctional epithelium. If the surface of the implant is contaminated by bacteria, an inflammatory response will be triggered in the connective tissue. The bone on the implant bed cannot organize a defense against infection, in contrast with the periodontal ligament, which is rich in vessels that are found around a natural tooth. Therefore, the apical extension of the inflammatory

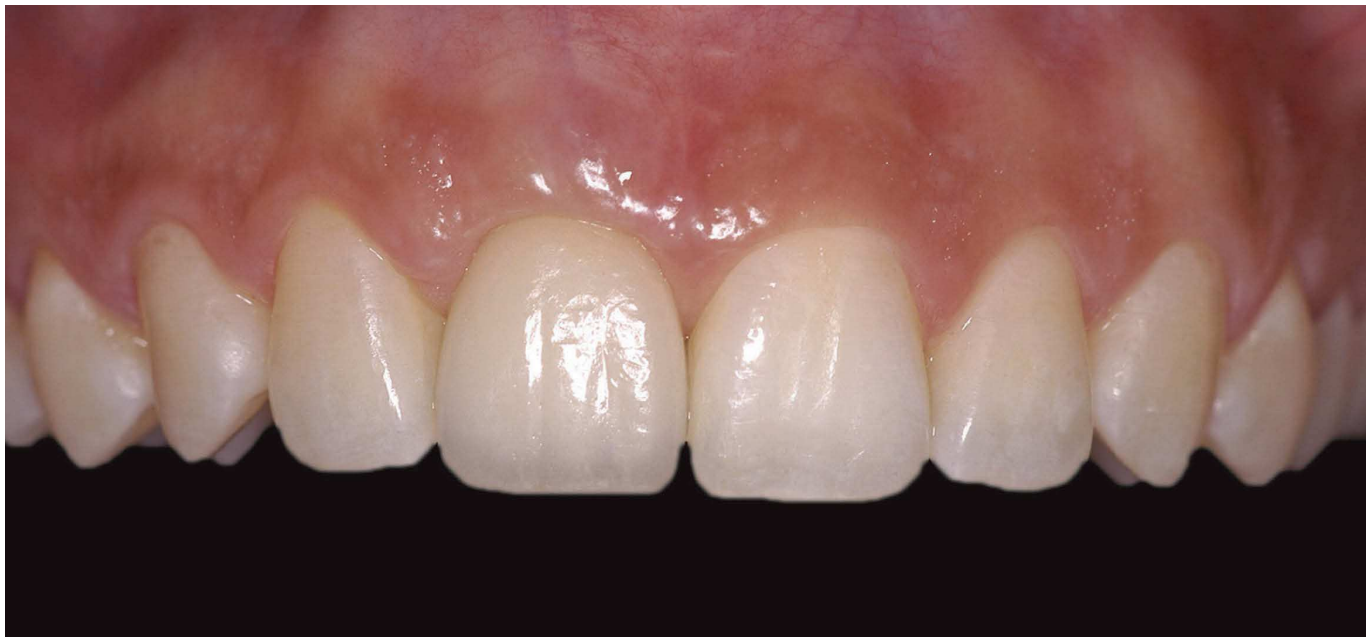

**Figure 4** - Illustration of peri-implant tissue with contours, appearance and texture similar to periodontal tissue.

infiltrate around the implants, more significant when found in the periodontium, seems to result from the morphological orientation of the supra-alveolar peri-implant fibers.[34] This is also the opinion of other authors, who found that the great difference between inflammatory response of the peri-implant and periodontal tissues is associated with the organization of the supra-alveolar fibers and the mobility of the gingival margin, which renders the implant more vulnerable to bacterial contamination.[24]

Therefore, although not based on conclusive studies, clinical findings suggest that the lack of an adequate area of masticatory mucosa may prevent the performance of appropriate oral hygiene procedures and grant insufficient protection against peri-implant infection in tissues that support implants.[5]

In teeth, minimal widths of keratinized tissues are compatible with gingival health. However, inflammation persists in areas with less than 2 mm of keratinized mucosa and, therefore, the width of the keratinized mucosa area should be 2 mm or more, and the gingiva should have an attachment of at least 1 mm.[25]

Another longitudinal study with teeth followed up 106 sites with buccal recession and probing depth of 3 mm or less. They concluded that, when there is gingival recession, the elimination of hygiene trauma is usually enough to prevent recession or loss of independent attachment, regardless of the width of the attached gingiva.[26]

However, it is still unclear whether a sufficient amount of keratinized tissue is necessary to preserve, in the long term, periodontal and peri-implant health, as well as how much tissue is sufficient.[27]

Some authors evaluated the association of the width of peri-implant soft tissue in 39 patients that received complete fixed prosthesis about 10 or more years before, or a partial denture at least 5 years before (total of 171 Branemark implants). They found that 24% of the sites did not have masticatory mucosa, and the measure of the keratinized mucosa in 13% of the implants was less than 2 mm. Analyses revealed that neither the width of the masticatory mucosa nor the mobility of the margin tissue had a significant influence on the pattern of bacterial plaque control or the health of the peri-implant mucosa according to the diagnosis made by bleeding at probing.[5]

In contrast, an experimental study evaluated the effect of the presence or absence of keratinized mucosa in the progression of peri-implantitis induced in monkeys. Five monkeys and a total of 30 transmucosal implants in mandibles, with or without keratinized mucosa, were included in the study. After healing for 3 months under optimal plaque control, all implants were submitted to plaque accumulation for 9 months. Loss of attachment was measured clinically and histometrically. Implants placed in areas without keratinized mucosa had a significantly greater loss of attachment and a greater gingival recession than those placed in areas with keratinized mucosa. The results of that study suggest that the absence of keratinized mucosa around endosseous tooth implants increases the susceptibility of the peri-implant region to tissue destruction induced by plaque.[2] These findings were confirmed by other studies, which suggested that the presence of keratinized mucosa around implants is strongly associated with optimal health of soft and hard tissues.[19,20,28]

At the same time, other longitudinal clinical studies have failed to confirm great differences in the progression of lesions around implants placed in sites with or without keratinized mucosa, and suggest that it may mask a health problem of the peri-implant mucosa.[3,5,6]

Other authors, however, conducted clinical studies to investigate the role of presence, or absence, of keratinized mucosa in the preservation of bone integrity around

implants that received different surface treatments (smooth x rough) in human beings. They examined 69 patients that had received implants three or more years before. The following parameters were evaluated: Bone loss, amount of attached gingiva, depth of probing, bleeding index, width of keratinized mucosa and attached mucosa. The implants were divided into 4 subgroups according to the band of keratinized mucosa. They found that gingival inflammation and plaque accumulation were statistically greater in implants with keratinized mucosa smaller than 2 mm. The analysis according to implant locations, divided into posterior and anterior sites, revealed an increase of gingival inflammation in posterior implants with a keratinized gingival band smaller than 2 mm. Bone loss of the anterior and posterior implants with a band of attached gingiva equal to or smaller than 2 mm wide was 0.04 mm and 0.14 mm. These values were statistically significant.[29]

To study the importance of peri-implant keratinized mucosa as a prerequisite for the health of soft tissue in the long term and its stability for 5 years, the following parameters were examined: Plaque accumulation, bleeding index, amount of mucosa margin and width of keratinized mucosa. There was no clear association between plaque accumulation and the width of keratinized mucosa in buccal regions. However, in the lingual region, plaque accumulation increased as the amount of keratinized mucosa decreased. The association of recession with width of keratinized mucosa revealed that recession is greater in areas with a smaller amount of keratinized mucosa.[27]

Another longitudinal study divided 276 implants into two groups. One group had keratinized mucosa equal to or greater than 2 mm, and the other, lower than 2 mm. The parameters evaluated were plaque and gingiva (Löe) index, depth of buccal probing, mucosa recession and marginal bone loss. Ninety implants were in the group that had keratinized mucosa smaller than 2 mm, and the other 186 implants were in the first group. The authors found that the plaque and gingiva indices had only a slight increase in implants with less than 2 mm of keratinized mucosa. In contrast, gingiva recession and marginal bone loss was greater and statistically significant in the group with keratinized mucosa. They concluded that in cases that require tissue maintenance for the long term, particularly in esthetic areas, keratinized mucosa should be present.[30]

To determine whether the width of keratinized mucosa of the implants has a significant effect on the health of soft and hard tissues around it, 200 implants were evaluated to define thickness of gingival tissue, width of keratinized mucosa, plaque index (PI) and gingival index (GI), depth of probing, implant mobility, radiographic bone level (RBL) and smoking. The implants were divided into group A, with 110 implants that had a keratinized mucosa equal to or greater than 2 mm, and group B, with 90 implants with less than 2 mm of keratinized mucosa. As a result, the study showed that the sites with less than 2 mm of keratinized mucosa had a greater accumulation of plaque and clinical signs of inflammation. Moreover, bleeding at probing and mean alveolar bone loss were also greater in areas with a keratinized mucosa smaller than 2 mm. They concluded, therefore, that there is an association between width of the keratinized mucosa and health of peri-implant tissues.[31]

Another study investigated the association of keratinized mucosa and the health of tissues that surrounded implants supporting overdentures. A total of 24 implants in the maxilla and 42 in the mandible were evaluated and divided into two groups: Group A = 36 implants with keratinized mucosa U 2 mm; and group B = 20 implants with keratinized mucosa < 2 mm. There were no statistically significant differences between the two groups in probing depth. The mean values of plaque and gingiva indices, bleeding at probing,

recession and periodontal insertion level were statistically greater in B than in A. Therefore, the study suggested that the absence of keratinized mucosa around implants that support overdentures is associated with a great accumulation of plaque, gingival inflammation, bleeding at probing and recession.[32]

The width of the keratinized mucosa was not significant to maintain health in case of adequate oral hygiene. However, a thin gingiva may be more susceptible to recession when exposed to orthodontic or prosthetic functional demands. The functional need of keratinized gingiva around implants does not seem to have been clearly defined, despite the fact that its esthetic benefit has been widely acknowledged.[33]

## Conclusions

- Studies indicate a consensus about the fact that the presence of an adequate band of keratinized mucosa promotes greater stability of peri-implant tissues and, therefore, greater recession in the regions where the gingiva is thin.
- There is no consensus about the effect of gingiva phenotype to maintain peri-implant health in the long term, although studies suggest that a band of keratinized tissue may facilitate oral hygiene and preserve adequate levels of attachment in the long run.
- Further prospective and longitudinal studies should evaluate the effect of the clinical characteristics of peri-implant soft tissues on the different implant systems currently used.

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