### RESEARCH



# Influence of keratinized mucosa on peri-implant diseases: a retrospective cohort study in humans

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## Abstract

**Objective** The present study aimed to assess the relationship between keratinized mucosa width and peri-implant diseases, namely peri-implant mucositis and peri-implantitis.

**Materials and methods** Ninety-one dental implants in function for  $\geq 6$  months from 40 partially or completely edentulous non-smoker subjects (24 females and 16 males) were evaluated clinically and radiographically. The width of keratinized mucosa, probing depth, plaque index, bleeding on probing, and marginal bone levels were assessed. Keratinized mucosa width was categorized as  $\geq 2$  mm or < 2 mm.

**Results** There was no statistically significant association between keratinized buccal mucosa width and peri-implant mucositis or peri-implantitis ( $p \ge 0.37$ ). In the regression analysis, peri-implantitis was associated with longer implant function time (RR: 2.55, 95% CI: 1.25–11.81, p=0.02) and implants in the maxilla (RR: 3.15, 95% CI: 1.61–14.93, p=0.003). Mucositis was not associated with any of the factors analyzed.

**Conclusion** In conclusion, in the present sample, keratinized buccal mucosa width was not associated with peri-implant diseases, suggesting that a band of keratinized mucosa may not be necessary to maintain peri-implant health. Prospective studies are required to better understand its role in the maintenance of peri-implant health.

Keywords Keratinized mucosa · Peri-implant tissues · Dental implants · Peri-implantitis

# Introduction

Peri-implant diseases are classified as peri-implant mucositis and peri-implantitis [1]. Peri-implant mucositis is characterized by bleeding on probing and visual signs of inflammation [2]. Peri-implantitis is a plaque-associated pathologic condition occurring in the tissue around dental implants,

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characterized by inflammation in the peri-implant mucosa and progressive loss of supporting bone [3].

The oral mucosa surrounding natural teeth and dental implants is classified into two types: the masticatory mucosa, including the gingiva and soft tissue covering the alveolar process and hard palate, and the lining mucosa. The masticatory mucosa is covered by a parakeratinized stratified squamous epithelium interdigitating with dense fibrous connective tissue (lamina propria). The adjacent lining mucosa is covered by a nonkeratinized stratified squamous epithelium interfacing with an underlying loose connective tissue containing numerous elastic fibers [4, 5].

Studies have suggested that the lack of an adequate amount of keratinized mucosa hinders appropriate oral biofilm control and may provide insufficient protection of the implant-supporting tissues against friction forces [6–8]. However, this issue is a matter of controversy. While some studies have shown that keratinized mucosa is required to maintain peri-implant health [9–15], others did not find such association [16–19] or reported a negligible correlation [20]. Accordingly, the amount of keratinized mucosa has little influence on soft tissue inflammation when proper plaque control is achieved. Suboptimal oral hygiene due to access difficulty for plaque control in the areas of minimal keratinized mucosa may cause greater tissue damage [11, 15, 21].

Considering the controversies, the present study aimed to assess the relationship between keratinized mucosa width and peri-implant diseases.

# **Material and methods**

The Institutional Ethics Committee approved the study protocol, and all subjects provided informed consent (protocol 05/454).

## Study design, setting and participants

This retrospective cohort study involved a convenience sample of subjects selected from the pool of consecutive patients treated at the private practice of one of the authors (VLP). Forty partially or completely edentulous volunteers (24 females and 16 males) aged 21 to 82 years, who had received 91 dental implants, were evaluated.

Peri-implantitis and peri-implant mucositis were the outcomes, and lack of keratinized mucosa was the exposure variable. Potential confounders were sex, age, duration of clinical function of the implant, implant region, type of prosthesis, and dental plaque index.

The inclusion criteria were: 1) implants in function for at least six months; 2) at least six months of use of an implantsupported prosthesis; 3) good systemic health condition, 4) not using any medication;5) no smoking habits; 6) absence of occlusal alterations such as occlusal overload, premature contacts, occlusal interference, occlusal instability, anterior guidance, altered vertical dimension, and bruxism.

### **Data collection**

All available implants in each patient were examined. Using a periodontal probe, one trained examiner performed all clinical evaluations at 3 points on each implant's buccal and lingual surfaces (PCP-UNC 15 periodontal probe, Hu-Friedy, Chicago, IL, USA). The following parameters were measured to the nearest millimeter: 1) mucosal recession, measured as the distance from the implant shoulder to the mucosal margin; 2) probing depth, measured as the distance from the mucosal margin to the bottom of the peri-implant sulcus; 3) clinical attachment level, measured as the distance from the implant shoulder to the bottom of the sulcus; 4) keratinized mucosa width, measured as the distance from the soft tissue margin to the mucogingival junction, with the location of the latter being determined by a visual method [22]. The presence or absence of plaque and bleeding on probing [23] was evaluated by moving the tip of a probe along the mucosal margin on the abutments' mesial, buccal, distal, and lingual surfaces.

The peri-implant mucosal condition was also assessed visually and by palpation. Mucosal manifestations were defined as the presence of redness, hyperplasia, suppuration, swelling, and/or pain on palpation. The type of prosthesis and function time of the implant were recorded.

### Analysis of radiographs

Radiographs were obtained during the consultation using the following parameters: 70 kVp, 8 mA, and 0.8 ms (Dabi Atlante, Ribeirão Preto, Sao Paulo, Brazil). The radiographs were taken using the long-cone paralleling technique. The films were manually developed according to the manufacturer's instructions under standardized development time conditions, fixing time, and temperature using fresh solutions. The radiographs were then digitized (HP Scanjet 7400C Series Scanner – XPA, Hewlett- Packard Development Company, Palo Alto, CA, USA) at 512×480 pixels and 8 bits. Peri-implant radiolucency and marginal bone levels were examined on the digitized radiographs.

The distance between the top of the implant shoulder (abutment interface) and the first visible bone-implant contact was measured on the mesial and distal sides of the implants using the Axiovision 4.4 software (Carl Zeiss, Göttingen, Germany). Two independent examiners performed the radiographic measurements. All measurements were reviewed for consistency, and the examiners reassessed inconsistent readings. The differences between readings were within 0.5 mm for all measurements. The mean measurement of the two examiners was computed. Radiographic dimensional distortion was corrected as a function of the known true dimension of the implant and the radiographic dimension.

# Criteria for classification of peri-implant health, peri-implant mucositis and peri-implantitis

Peri-implant health was defined clinically by an absence of visual signs of inflammation and bleeding on probing and normal bone support. Peri-implant mucositis was defined as the presence of inflammation (presence of bleeding and/ or suppuration on gentle probing accompanied by local swelling, redness, shininess, and/or soreness) without bone loss beyond crestal bone level changes of 2 mm. Peri-implantitis was defined as a radiographic bone loss  $\geq$  3 mm and/or probing depth  $\geq$  6 mm in at least one implant site, along with bleeding and/or suppuration on probing [24].

### **Statistical analyses**

Missing data were excluded from the analysis. The implant was considered the statistical unit. The exposure variable, keratinized mucosa width was categorized as  $\geq 2$  mm or < 2 mm. The independent variables were categorized as follows: sex (male or female); median age ( $\geq 44$  years and < 44 year); median implant function time ( $\geq 18$  months and < 18 months); type of fixed prosthesis (screwed or cemented); implant region (mandible or maxilla); dental plaque (present or absent).

The following categories were compared: peri-implant mucositis x peri-implant health, and peri-implantitis x peri-implant health. The presence of peri-implantitis or mucositis was the dependent variable. The associations of peri-implant mucositis or peri-implantitis with the exposure variable and independent variables were assessed using the chi-squared test or Fisher's exact test for n > 5 and  $n \le 5$ , respectively. Next, backward stepwise logistic regression was performed to assess the associations between peri-implantitis and the independent variables. Only variables that showed a p-value  $\le 0.20$  in the chi-squared analysis were included in the model. P values, risk ratios (RR) and 95% confidence intervals (CI) were computed.

Data analysis was performed using the SPSS v. 13.0 program (SPSS Inc., Chicago, IL, USA).

# Results

Forty volunteers with 91 implants were included. Table 1 shows the general characteristics of the sample. In summary, most subjects were women and  $\geq$  44 years old. Regarding implant characteristics, 62.6% of the implants were located

Table 1 General characteristics of the sample

Variable	Category	n	%
Sex	Female	24	60
	Male	16	40
Age (years)	<44	40	44.0
	≥44	51	56.0
Function time (months)	<18	58	63.7
	≥18	33	36.3
Prosthesis type	Cemented	34	37.4
	Screwed	57	62.6
Dental plaque	Absent	5	5.5
	Present	86	94.5
Implant region	Mandible	57	62.6
	Maxilla	34	37.4
Keratinized buccal mucosa	$\geq 2 \text{ mm}$	63	69.2
	<2 mm	28	30.8

in the mandible, 37.4% were found in the maxilla, 62.6% of the prosthesis were screwed, and 37.4% were cemented. The presence of a biofilm was observed in 94.5% of the implants.

A band of keratinized mucosa  $\geq 2$  mm was observed in 69.2% of the sample and <2 mm in 30.8%. There was no statistically significant association between keratinized buccal mucosa width and peri-implant mucositis or peri-implantitis (p  $\geq$  0.37) (Table 2). Mucositis was not associated with any of the factors analyzed (p  $\geq$  0.06). In the regression analysis, peri-implantitis was associated with longer implant function time (RR: 2.55, 95% CI: 1.25–11.81, p=0.02) and implants located in the maxilla (RR: 3.15, 95% CI: 1.61–14.93, p=0.003) (Table 3).

# Discussion

This study evaluated the relationship between keratinized mucosa width and peri-implant diseases. The results revealed that keratinized buccal mucosa was not associated with peri-implant mucositis or peri-implantitis. However, peri-implantitis was associated with longer implant function time and implants in the maxilla. In contrast, mucositis was not associated with any of the factors analyzed.

Other studies have also reported the lack of association between keratinized buccal mucosa width and periimplant diseases [5, 16-19]. Furthermore, areas devoid of attached gingiva were not less resistant to the development of inflammation than areas with a wide zone of attached gingiva when exposed to plaque accumulation [6-8]. However, some authors suggested that circumferential sealing effects through dense connective tissue may be a prerequisite for the long-term success of a dental implant and that it is more difficult to perform appropriate plaque control in areas not surrounded by keratinized mucosa, with higher rates of peri-implant diseases in implants lacking or surrounded by an inadequate width of healthy keratinized mucosa [9–15]. Other risk factors have been associated with peri-implant diseases [25], a fact that may explain the findings of the present study.

Studies suggest that the presence of keratinized mucosa to maintain peri-implant health does not seem essential in situations of adequate self-performed biofilm control around implants [2]. However, plaque control was inadequate in the present sample since 94.25% of the implants had visible dental plaque. Despite this poor dental plaque control, peri-implant mucositis or peri-implantitis was not associated with dental plaque in the present sample, in agreement with a recent study [26]. In contrast, another study found an association between dental plaque and peri-implant disease [27, 28]. These contrasting results may be attributed to the following factors: a single assessment of plaque accumulation which may not necessarily reflect long-term plaque control;

Table 2 Relationship between the independent variables and the presence of peri-implant mucositis or peri-implantitis

Variable	Peri-implantitis				Mucositis					
	Yes	No	RR	95% CI	p-value	Yes	No	RR	95% CI	p-value
	n (%)					n (%)				
Age (years)			0.36	0.08-0.73	0.01 <sup>a</sup>			1.24	0.74-4.11	0.21 <sup>a</sup>
<44	15 (39.5)	23 (60.5)				22 (55)	18 (45)			
≥44	6 (14)	37 (86)				34 (68)	16 (32)			
Sex			0.76	0.23-2.04	0.50 <sup>a</sup>			1.17	0.60-3.94	0.36 <sup>a</sup>
Female	15 (28.3)	38 (71.7)				36 (59)	25 (41)			
Male	6 (21.4)	22 (78.6)				20 (69)	09 (31)			
Function time			2.47	1.27-10.27	0.01 <sup>a</sup>			1.36	0.94-6.32	0.06 <sup>a</sup>
<18 months	10 (17.9)	46 (82.1)				32 (55.2)	26 (44.8)			
$\geq 18$ months	11 (44.0)	14 (56.0)				24 (75.0)	8 (25.0)			
Prosthesis			1.38	0.54-4.33	0.42 <sup>a</sup>			1.27	0.57-3.28	0.49 <sup>a</sup>
Cemented	7 (21.2)	26 (78.8)				19 (57.6)	14 (42.4)			
Screwed	14 (29.2)	34 (70.8)				37 (64.9)	20 (35.1)			
Dental plaque			1.38	1.20-1.58	0.57 <sup>b</sup>			1.59	0.41-16.5	0.36 <sup>b</sup>
Absent	0 (0.00)	4 (100)				2 (40)	3 (60.0)			
Present	21 (27.3)	56 (72.7)				54 (63.5)	31 (36.5)			
Implant region			3.07	1.61-13.50	0.003 <sup>a</sup>			1.12	0.55-3.32	0.51 <sup>a</sup>
Mandible	7 (14.3)	42 (85.7)				34 (59.6)	23 (40.4)			
Maxilla	14 (43.8)	18 (56.3)				22 (66.7)	11 (33.3)			
Keratinized mucosa			0.70	0.33-1.50	0.37 <sup>a</sup>			0.91	0.73-1.30	0.84 <sup>a</sup>
$\geq 2 \text{ mm}$	14 (23.3)	46 (76.7)				39 (62.9)	23 (37.1)			
<2 mm	7 (33.3)	14 (66.7)				17 (60.7)	11 (39.3)			

<sup>a</sup> Chi-squared test; <sup>b</sup> Fisher's exact test

*RR* Risk ratio, *CI* confidence interval, *n* number of subjects with the condition in the group

 
 Table 3
 Logistic regression analysis of the relationship between periimplantitis and putative risk factors

Variables*	Risk ratio (95% CI)	p-value
Implant region		0.003
Mandible	1	
Maxilla	3.15 (1.61–14.93)	
Function time		0.02
<18 months	1	
$\geq$ 18 months	2.55(1.25-11.81)	

\*Age, Implant region and Function time were included in the model

differences in the plaque indices used; differences in the case definitions of peri-implantitis, and the inclusion of patients with a short follow-up time after implant placement.

In this study, peri-implantitis was associated with longer implant function time and implants in the maxilla. This finding agrees with a previous study [29] that showed a mean marginal bone loss ranging from 0.17–0.82 mm/year and concluded that peri-implantitis increases over time. Regarding the implant region, a previous study [30] found

that peri-implantitis occurs more frequently in the maxilla compared to the mandible, in agreement with the present study. This finding might be explained by the lower density of maxillary bone, the anatomical and morphological structure of the maxilla, and the reduced bone volume due to the high degree of alveolar ridge resorption that may be critical for the success of dental implants [31]. In the present study, peri-implant mucositis was not associated with any of the factors analyzed, in agreement with a previous study [29]. A meta-analysis [32] found an association of mucositis with immediately placed implants but not with follow-up period, implant/subject ratio, implant surface, type of prosthesis (fixed vs. removable), smoking habits, or periodontitis. Periimplant mucositis has also been associated with biofilm accumulation, smoking, and radiation therapy [2]. However, mucositis was not associated with biofilm accumulation in the present study, probably due to the small sample size, especially considering there were only five implants without dental biofilm.

The limitations of the present study include its retrospective design, the short function time of 64% of the implants, the lack of radiographs at the moment of prosthesis installation and the small sample size. It has been showed that the majority of peri-implantitis develops after 3 years of follow up [33]. Prospective studies are needed to elucidate the relationship between keratinized mucosa width and peri-implant health.

# Conclusion

In conclusion, in the present sample, keratinized buccal mucosa width was not associated with peri-implant diseases, suggesting that keratinized mucosa width may not be necessary for maintaining peri-implant health. Prospective studies are essential to understand better the role of keratinized mucosa in maintaining peri-implant health.

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**Data availability** The datasets generated during and/or analysed during the current study are available from the corresponding author on reasonable request.

### Declarations

**Ethics approval** This study was performed in line with the ethical principles of human research and approved by The Institutional Ethics Committee San Leopold Mandic Dental Research Center (protocol 05/454).

**Consent to participate** Informed consent was obtained from all individual participants included in the study.

#### Consent to publish Not applicable.

**Competing interests** The authors have no relevant financial or non-financial interests to disclose.

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