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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

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Abstract

Background—The question of the importance of keratinized mucosa around dental implants for the prevention of peri-implant disease could not be answered in the relevant literature so far.

Objective—To investigate the influence of peri-implant keratinized mucosa on long-term peri-implant soft-tissue health and stability over a period of 5 years.

Material and methods—A total of 386 mandibular dental implants were placed in 73 completely edentulous patients, and subsequently restored with fixed full-arch prostheses. At prosthesis delivery (baseline) and after 3, 6, 12, 18, 24, 36, 48 and 60 months, modified plaque index (mPlI), modified sulcus bleeding index (mBI), distance between implant shoulder and mucosal margin (DIM) and width of peri-implant keratinized mucosa (KM) were recorded. Statistical analysis included multivariate logistic regression, multivariate ordinal logistic regression, generalized estimating equations and Bonferroni's correction.

Results—Fifty-eight patients with 307 implants completed the 5-year study. Statistically significantly higher plaque accumulation on lingual sites (mean mPlI 0.67, SD 0.85), bleeding

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tendencies on lingual sites (mean mBI 0.22, SD 0.53) and larger soft-tissue recession on buccal sites (mean DIM − 0.69 mm, SD 1.11 mm) were found when the width of KM was <2 mm, compared to sites with ≥2 mm of KM (mean mPII 0.40, SD 0.68, $P = 0.001$; mean mBI 0.13, SD 0.41, $P < 0.01$; mean DIM − 0.08 mm, SD 0.86 mm, $P < 0.001$). The width of keratinized mucosa had no effect on bleeding tendency or plaque accumulation on buccal sites ($P > 0.05$).

Conclusion—In patients exercising good oral hygiene and receiving regular implant maintenance therapy, implants with a reduced width of < 2 mm of peri-implant keratinized mucosa were more prone to lingual plaque accumulation and bleeding as well as buccal soft-tissue recession over a period of 5 years.

Keywords

dental implant; keratinized mucosa; long-term effects; plaque; recession; soft tissue

The question of whether a sufficient amount of keratinized tissue around natural teeth or dental implants is necessary for long-term periodontal or peri-implant health, as well as the question of what has to be considered ‘sufficient,’ are still controversial (Lang & Loe 1972; Krekeler et al. 1985; Wennstrom 1987; Wennstrom et al. 1994; Heckmann et al. 2004).

More than 35 years ago, Lang & Loe (1972) found better periodontal health in teeth, which yielded at least 2 mm of keratinized gingiva with at least 1 mm being attached, compared to teeth with < 2 mm of keratinized gingiva. Although the latter findings have been challenged by subsequent studies (Miyasato et al. 1977; Wennstrom & Lindhe 1983a; Kennedy et al. 1985; Wennstrom 1987), a width of 2 mm of keratinized gingiva has been considered clinically desirable to provide an appropriate soft-tissue seal around natural teeth since then.

Taking into account the considerable differences between the soft-tissue interface of dental implants and that of natural teeth, the questions arise, if peri-implant keratinized mucosa is necessary or at least beneficial for peri-implant soft-tissue health, and if the same 2 mm threshold recommended for natural teeth also applies to dental implants.

If the amount of keratinized mucosa has an impact on peri-implant health, special measures would be required in patients with insufficient amounts of keratinized mucosa during maintenance therapy including specific oral hygiene instructions and closer maintenance intervals. Moreover, surgical procedures aiming at widening the keratinized tissue and increasing the vestibular depth may be indicated.

Although it has been demonstrated that dental implants are successful in the treatment of edentulous jaws with fixed or removable prostheses (Ferrigno et al. 2002), augmentation of keratinized mucosa or vestibular depth may especially be required in the edentulous mandible, in which moderate to severe ridge resorption may have lead to reduced vestibular depth and a lack of keratinized mucosa.

Numerous techniques to do so have been described, including free gingival grafts from the palate with or without combination of a vestibuloplasty (Edlan 1973; Buser 1987; Simons & Baima 1994; Han et al. 1995). Depending on the technique applied, some of these

procedures may pose a significant challenge for the patient, especially when elderly or medically compromised.

Accordingly, it would be of great importance both for clinicians and patients to better understand the significance of peri-implant keratinized tissue for long-term soft-tissue health and stability, in order to better assess the need for additional surgical procedures or special considerations during maintenance therapy. However, as of today, the literature is inconclusive, contradictory and lacks long-term human data.

Therefore, the purpose of this study was to investigate the necessity of peri-implant keratinized mucosa as a prerequisite for long-term soft-tissue health and stability over a period of 5 years.

Material and methods

Patient enrollment and clinical procedures

The data for the present study was acquired from a 5-year prospective multi-center trial of the ITI Dental Implant System, which included five clinical centers: the University of Connecticut Health Center (Farmington, CT, USA), the University of Texas Health Science Center (San Antonio, TX, USA), Baylor University (Dallas, TX, USA), the Harvard School of Dental Medicine (Boston, MA, USA) and a private practice in Birmingham, United Kingdom. After approval by the Institutional Review Boards patients were consecutively enrolled according to the following study protocol.

Patients had to be edentulous and desire a fixed mandibular implant prosthesis. Before study enrollment, patients read and signed the informed consent form. Age, gender and smoking status were recorded and the following exclusion criteria were defined for study enrollment:

- Smoking (> 10 cigarettes/day)
- Chewing tobacco
- Alcohol or drug abuse
- Severe bruxing or clenching
- Lack of motivation or compliance
- Psychiatric disorders
- Current pregnancy
- Uncontrolled diabetes
- Metabolic bone disorders
- History of renal failure
- Compromised immune system, HIV
- Current steroid therapy
- Current chemotherapy

- Coumadin therapy
- Hematological disorders
- History of leukocyte dysfunction/deficiencies
- High endocarditis risk
- Previous bone augmentation at surgical site
- History of head and neck radiation
- Physical handicaps interfering with oral hygiene measures
- Investigational drug/device ≤30 days before implant placement.

For the planned implant recipient sites, adequate oral hygiene, the absence of local inflammation or mucosal diseases such as lichen planus, no history of local radiation therapy and an adequate bone volume were required.

All implants (ITI Solid Screw Implants, TPS Surface, Straumann, Basel, Switzerland) were placed in a non-submerged manner (Fig. 1a). After 4–7 months of healing, all patients were treated with the same prosthodontic treatment protocol. Full-arch, screw-retained hybrid-type prostheses consisting of semi-precious metal alloy frames, denture teeth and denture base resin (Branemark et al. 1987; Weingart & ten Bruggenkate 2000) were fabricated and delivered (Fig. 1b) using octa abutments and gold copings for bridges (Straumann). Implant maintenance, which included debridement and oral hygiene instructions, was administered at every visit.

Clinical evaluation parameters

The following clinical parameters were measured at prosthesis delivery (baseline) and 3, 6, 12, 18, 24, 36, 48 and 60 months after prosthesis insertion by clinicians, who had been calibrated before the start of patient enrollment.

Plaque accumulation was assessed using the *modified plaque index (mPII)* (Mombelli et al. 1987). Measurements were taken on four implant sites (mesial, distal, buccal, lingual).

Bleeding tendencies of the peri-implant mucosa, as a measure of soft-tissue inflammation, were evaluated using the *modified sulcus bleeding index (mBI)* (Mombelli et al. 1987), measured on four implant sites (mesial, distal, buccal, lingual).

The level of the mucosal margin was evaluated in relation to the implant shoulder and the *distance between the implant shoulder to the peri-implant mucosa (DIM)* was measured in mm on the mid-facial aspect only (Weber et al. 2000). Positive values were recorded, when the mucosal margin was located coronal to the implant shoulder, while negative values were marked, when the mucosal margin had receded apical to the implant shoulder. All measurements were rounded to the nearest millimeter.

The *width of peri-implant keratinized mucosa (KM)* was measured in millimeters on the mid-facial and mid-lingual aspects. Differences in color, texture and mobility between the keratinized mucosa and the lining mucosa served as markers for the detection of the muco-

gingival junction. KM was then measured as the distance between the gingival margin and the muco-gingival junction, and measurements were rounded to the nearest millimeter.

Statistical analysis

The implant was chosen as the statistical unit. The significance level α was set to 0.05.

To evaluate the influence of KM on soft-tissue outcome, the exposure variable KM was categorized into sites with < 2 mm of KM and sites with at least 2 mm of KM. Longitudinal trends were displayed in time-dependant curves, describing the medians or means of the respective outcome over time for buccal and lingual sites separately. Plaque and bleeding indices were modeled as binary outcomes while DIM was modeled as a normally distributed continuous outcome. The effect of KM was evaluated as a binary variable (KM < 2 mm vs. KM ≥2 mm) on both facial and lingual aspects. To compare soft-tissue outcomes between the two exposure groups at each time point, multivariate logistic regression analyses were used on both mPlI and mBI outcomes, and multiple ordinal logistic regression models (OLR) (Hosmer & Lemeshow 2000) were applied on DIM outcomes, all adjusting for clustering by individual and controlling for age, gender and smoking status. The proportional odds assumption was evaluated for each OLR model and in the cases where the assumption was not met separate binary logistic regression models were conducted. The trend over time was analyzed using generalized estimating equations using the logit link function to model the log odds for binary plaque and bleeding indices and the identity link function for the normally distributed DIM. Hence, for binary plaque and bleeding indices over time, the odds ratio (OR) and the corresponding 95% confidence interval (CI) were reported. All models were adjusted for clustering of subject and implants and accounted for correlations between repeated measures over time. Potential effect modifications of KM by time in each model were evaluated by the inclusion of an interaction term. Significance of the interaction was evaluated by a likelihood ratio test ($\alpha = 0.05$) comparing the main effects only model to the model with an interaction term.

In order to identify the minimal amount of keratinized mucosa, which would be beneficial for long-term soft-tissue conditions, the means of soft-tissue measurements of all nine clinical examinations were computed for different widths of keratinized mucosa and the results displayed in bar charts. An estimate of the number of implants contributing to each category of keratinized mucosa was provided by computing the median score for each implant over time. Differences between categories of KM by each outcome were calculated using GEE to allow for adjustments for the correlation between individuals and follow-up. To assess differences in the outcome, pairwise comparisons were calculated for each outcome on buccal and lingual sites separately, with adjustments for multiple testing using a Bonferroni correction.

Results

A total of 386 implants were placed in the edentulous mandibles of 73 patients (35 male, 38 female) with a mean age of 58 years (SD 9.6 years; range 34–78 years). Twenty-five percent of patients were light smokers (≤10 cigarettes/day) (Table 1).

Fifteen patients (21% of the study population) were lost to follow-up during the 5-year study period. Four patients died, seven failed to attend the follow-up examinations at various points throughout the study and could not be reached despite several attempts to contact them, and four patients were lost to follow-up due to relocation to another city, lack of transportation or unspecified reasons. A total of 58 patients with 307 implants finished the 5-year follow-up period.

Plaque accumulation on buccal sites did not reveal any differences between sites with < 2 mm of KM (mean mPlI 0.24, SD 0.54) and sites with at least 2 mm of KM (mean mPlI 0.25, SD 0.56) ($P = 0.38$). Plaque accumulation on lingual sites, in contrast, was statistically significantly different between sites with < 2 mm of lingual KM (mean mPlI 0.67, SD 0.85) and sites with at least 2 mm of lingual KM (mean mPlI 0.40, SD 0.68) (OR = 0.53; 95% CI: 0.46–0.62, $P = 0.001$) (Fig. 2a).

Bleeding scores on the buccal side did not differ between sites with < 2 mm of KM (mean mBI 0.05, SD 0.24) and those with at least 2 mm of KM (mean mBI 0.07, SD 0.32) throughout the study period ($P = 0.13$). On lingual sites, however, higher mBI scores were significantly associated with reduced widths in KM over time. The presence of at least 2 mm of lingual KM (mean mBI 0.13, SD 0.41) significantly reduced the OR for bleeding by 40% (OR = 0.60, 95% CI: 0.48–0.74) compared to sites with < 2 mm of lingual KM (mean mBI 0.22, SD 0.53).

As the DIM was only measured on buccal sites, the influence of keratinized mucosa on DIM, as a measure of peri-implant recession, could only be analyzed on these aspects. A statistically significant difference in DIM measurements was found between sites with < 2 mm of buccal KM (mean DIM − 0.69 mm, SD 1.11 mm) and sites with at least 2 mm of buccal KM (mean DIM − 0.08 mm, SD 0.86 mm) ($P < 0.001$) (Fig. 2c). A significant interaction with time was observed (P-value for interaction < 0.001), i.e. the effect of KM on mean DIM over time was different between the two groups, suggesting that sites with < 2 mm of KM exhibited significantly greater increases in mucosal recession over time compared to sites with at least 2 mm of KM. In addressing the clinical question how much keratinized mucosa around an implant is desirable or necessary for long-term soft-tissue health and stability, sites with various widths of keratinized mucosa were compared according to their clinical outcome.

No clear trends between mPlI scores and the width of keratinized mucosa could be observed on buccal sites. On lingual sites, however, a trend of generally increasing mean mPlI scores with decreasing widths of lingual KM was evident (Fig. 3a). A noticeable and statistically significant change in mean mPlI values occurred between 1 and 2 mm of KM ($P < 0.01$). Another statistically significant, but less pronounced change occurred between sites with 3 mm and sites with at least 4 mm of lingual KM ($P = 0.04$).

In terms of mBI scores, a trend of increasing bleeding tendencies with decreasing widths of keratinized mucosa was once more only observed on lingual sites (Fig. 3b). The most pronounced change in mean mBI values was seen between 0 and 1 mm of KM ($P < 0.001$).

On buccal surfaces, there was no association between the amount of keratinized tissue and bleeding tendencies.

Another distinct association could be found between the width of keratinized mucosa and DIM measurements on buccal sites. A tendency towards increasing soft-tissue recession was evident as the amount of buccal KM decreased (Fig. 3c). Changes in outcome primarily occurred between 1 and 2 mm of buccal KM ($P < 0.001$). Another statistically significant change could be seen between 3 mm and at least 4 mm of buccal KM ($P < 0.001$). The mean difference between sites without buccal KM (mean DIM − 0.71 mm, SD 1.08 mm) and sites with at least 4 mm of buccal KM (mean DIM 0.16 mm, SD 0.96 mm) was 0.87 mm.

Discussion

In the natural dentition, the keratinized gingiva includes the free and the attached gingiva and extends from the gingival margin to the muco-gingival junction (Ainamo & Loe 1966).

The question of whether or not the amount of keratinized gingiva around natural teeth has an impact on periodontal health and whether areas diagnosed as having little or no attached gingiva should consequently be treated accordingly has been a matter of controversy until today (Hallmon et al. 1996).

Lang & Loe (1972) reported a correlation between the width of keratinized and attached gingiva and periodontal health. While over 80% of the surfaces with at least 2 mm of keratinized tissue and at least 1 mm of attached gingiva were clinically healthy, all surfaces with < 2 mm of keratinized gingiva and < 1 mm of attached gingiva exhibited various amounts of clinical inflammation. The authors found it conceivable that a movable gingival margin would facilitate the introduction of microorganisms into the gingival crevice. It was concluded that a width of 2 mm of keratinized tissue, with at least 1 mm being attached, is adequate to maintain gingival health.

Stetler & Bissada (1987) compared the tissue response around teeth with and without sub-gingival margins in association with narrow (≤2 mm) or wide (> 2 mm) zones of keratinized gingiva. Higher gingival index scores were observed when sub-gingival restorative margins were present in areas with a narrow band of keratinized gingiva. The authors suggest that augmentation of keratinized tissue may be warranted, if sub-gingival restorations were to be placed in areas of minimal keratinized gingiva and less than optimal plaque control.

Moreover, Tenenbaum (1982) found a statistically significant correlation between the width of attached gingiva and the incidence of gingival recession.

Other studies, however, could not confirm the importance of keratinized gingiva for periodontal health and stability, and hence have not supported routine soft-tissue grafting of sites with minimal or no keratinized or attached gingiva.

Miyasato et al. (1977) could not find any differences in gingival health between sites with or without keratinized tissue. In addition, after temporary withdrawal of oral hygiene regimens, sites with minimal keratinized and no attached gingiva were not more prone to develop

plaque-induced inflammatory changes than areas with attached gingiva and a greater band of keratinized tissue.

A study by Wennstrom & Lindhe (1983b) in beagle dogs indicated that the inflammatory response to bacterial plaque is not related to the presence or absence of attached gingiva.

In further studies, Wennstrom and colleagues investigated the influence of attached gingiva on the occurrence of gingival recession. After the entire zone of attached gingiva had been surgically removed from the test sites, the authors found that the amount of gingival recession remained stable over time independent of the presence or absence of attached gingiva (Wennstrom & Lindhe 1983a). Another investigation confirmed, that the lack of an 'adequate' zone of attached gingiva did not result in an increased incidence of soft-tissue recession in patients maintaining good oral hygiene (Wennstrom 1987).

Kennedy et al. (1985) also emphasized the importance of oral hygiene and demonstrated that minimal to zero attached gingiva could be maintained in a state of health, if adequate plaque control was provided. However, sites with little or no attached gingiva in non-maintained patients demonstrated proceeding recessions with a frequency of 20%, whereas no further recessions were noted in teeth with wide zones of attached gingiva.

Although soft tissues around teeth and implants have many features in common, there are several structural differences that may play a role in the responses to stimuli. While the junctional epithelium seems to end at a similar distance coronal to the osseous crest at teeth and around implants, i.e. between 1 and 1.5 mm, the orientation of supra-crestal collagen fibers differs between teeth and implants. The absence of root cementum for anchorage of gingival fibers leads to a rather parallel fiber orientation around implants, whereas collagen fibers perpendicularly attach to the tooth surface (Berglundh et al. 1991). The question whether this feature makes dental implants more prone to peri-implant inflammation and peri-implant soft and hard tissue breakdown in cases of insufficient amounts of keratinized tissue has been of great interest to both clinicians and researchers, but has yet to be answered. Although not substantiated in the literature, it often has been assumed that the lack of an adequate zone of keratinized mucosa impedes proper oral hygiene measures and provides insufficient protection from mechanical trauma during tooth brushing or mastication as well as from bacterial plaque load (Wennstrom et al. 1994).

Nevertheless, several clinical studies in humans have revealed that the absence of marginal keratinized tissue is compatible with peri-implant soft-tissue health, if adequate levels of plaque control are maintained (Wennstrom et al. 1994; Bengazi et al. 1996; Heckmann et al. 2004).

Wennstrom et al. (1994) conducted a retrospective analysis of 171 implants in 39 patients analyzing the influence of the presence or absence of keratinized mucosa on plaque accumulation and health status of marginal tissues. The results failed to reveal an influence of keratinized mucosa or marginal soft-tissue mobility on plaque index and bleeding on probing.

In a 10-year retrospective study of 46 implants supporting mandibular overdentures in severely atrophied edentulous jaws, Heckmann et al. (2004) found no significant difference in mBI scores between sites with or without keratinized mucosa.

Buser et al. (1990) recommended the placement of implants within a band of keratinized mucosa, but also mentioned that the need of keratinized mucosa for the maintenance of peri-implant health is still subject to debate. In their study, over 90% of the evaluated one-stage ITI implants were surrounded by keratinized mucosa, which the authors attributed to the one-stage surgical procedure, in which the wound margins are adapted to the implant post, hereby preserving the pre-existing keratinized tissues. In comparison, clinical studies on two-stage implants resulted in the presence of keratinized mucosa in only 20–65% of evaluated sites, indicating that keratinized mucosa may often be sacrificed during the reopening procedure (Adell et al. 1986; Cox & Zarb 1987).

Evaluating one-stage ITI implants supporting mandibular overdentures, Mericske-Stern et al. (1994) found that a wide zone of keratinized mucosa generally did not result in improved peri-implant parameters, compared to sites with a narrow band or the complete absence of keratinized tissue. However, buccal sites exhibited reduced bleeding tendencies over a period of 5 years in areas with at least 2 mm of keratinized mucosa. The authors concluded that healthy peri-implant conditions around one-stage implant-supported overdentures can be maintained over a period of 5 years, irrespective of the presence or absence of keratinized mucosa.

Reviewing the relevant literature, it becomes obvious that a clear statement about the role of peri-implant keratinized mucosa in preventing peri-implant inflammation, excessive plaque accumulation and soft-tissue recession can only be made with great difficulties due to contradicting results and the limited long-term human data.

The 5-year longitudinal data presented in the study at hand clearly points to an impact of the width of keratinized mucosa on plaque accumulation, bleeding and recession. This observation occurred despite a thorough maintenance program and good to excellent oral hygiene, which was indicated by the favorable clinical results with generally low bleeding and plaque indices as well as little mucosal recession throughout the 5-year observation period.

A clear trend toward higher plaque scores with decreasing amounts of KM could be observed on lingual sites, where sites with < 2 mm of KM exhibited significantly higher plaque scores compared to sites with at least 2 mm of KM. In consequence, lingual sites with insufficient amounts of KM also exhibited increased bleeding tendencies as a sign of plaque-induced inflammation. This trend was not observed on buccal surfaces. Although these findings point toward a correlation between plaque accumulation and/or peri-implant inflammation and the width of keratinized mucosa on lingual sites, it has to be mentioned that the cause–effect relationship between less KM and higher plaque or bleeding scores on lingual sites may be influenced by the fact, that sites with less lingual KM may also be associated with a shallower floor of the mouth, making oral hygiene access difficult. As oral hygiene access is generally better on buccal aspects, the depth of the vestibular fold may not

have had an impact on plaque accumulation on buccal sites. Additionally, oral hygiene access on the lingual side is in general more difficult than on the buccal.

Another clear association was found between the width of keratinized mucosa and buccal soft-tissue recession, measured as the DIM. Sites with less keratinized tissue generally exhibited higher amounts of peri-implant recession. Once more, the presence of at least 2 mm of keratinized mucosa proved advantageous. Differences were already present at the time of baseline measurements, which were taken after the implants had healed and the prostheses were inserted. While the level of the gingival margin remained stable over the 5-year period at sites with at least 2 mm of keratinized mucosa, it generally receded at sites with < 2 mm of peri-implant keratinized tissue, particularly during the first 12 months after prosthesis delivery. As already suggested by Bengazi et al. (1996), different remodeling processes in keratinized and non-keratinized tissues or in the underlying bone over time, but especially during the initial healing phase and the first 12 months after prosthesis delivery, could explain these findings. In that regard, Chang et al. (2006) could not find greater amounts of bone loss in implants with < 2 mm of keratinized mucosa compared to implants with broader bands of keratinized tissue. Further radiographic studies are needed to investigate peri-implant bone levels in relation to the width of keratinized mucosa.

Conclusions

In patients exercising good oral hygiene and receiving regular implant maintenance therapy, the existence of at least 2 mm of keratinized mucosa was beneficial for reduced lingual peri-implant plaque accumulation and bleeding, as well as buccal soft-tissue recession at implants supporting full-arch mandibular fixed prostheses. Hence, lingual sites with insufficient keratinized mucosa should be of specific concern during maintenance therapy to detect early inflammatory changes. An increased soft-tissue recession over time has to be expected at implants with insufficient keratinized mucosa.

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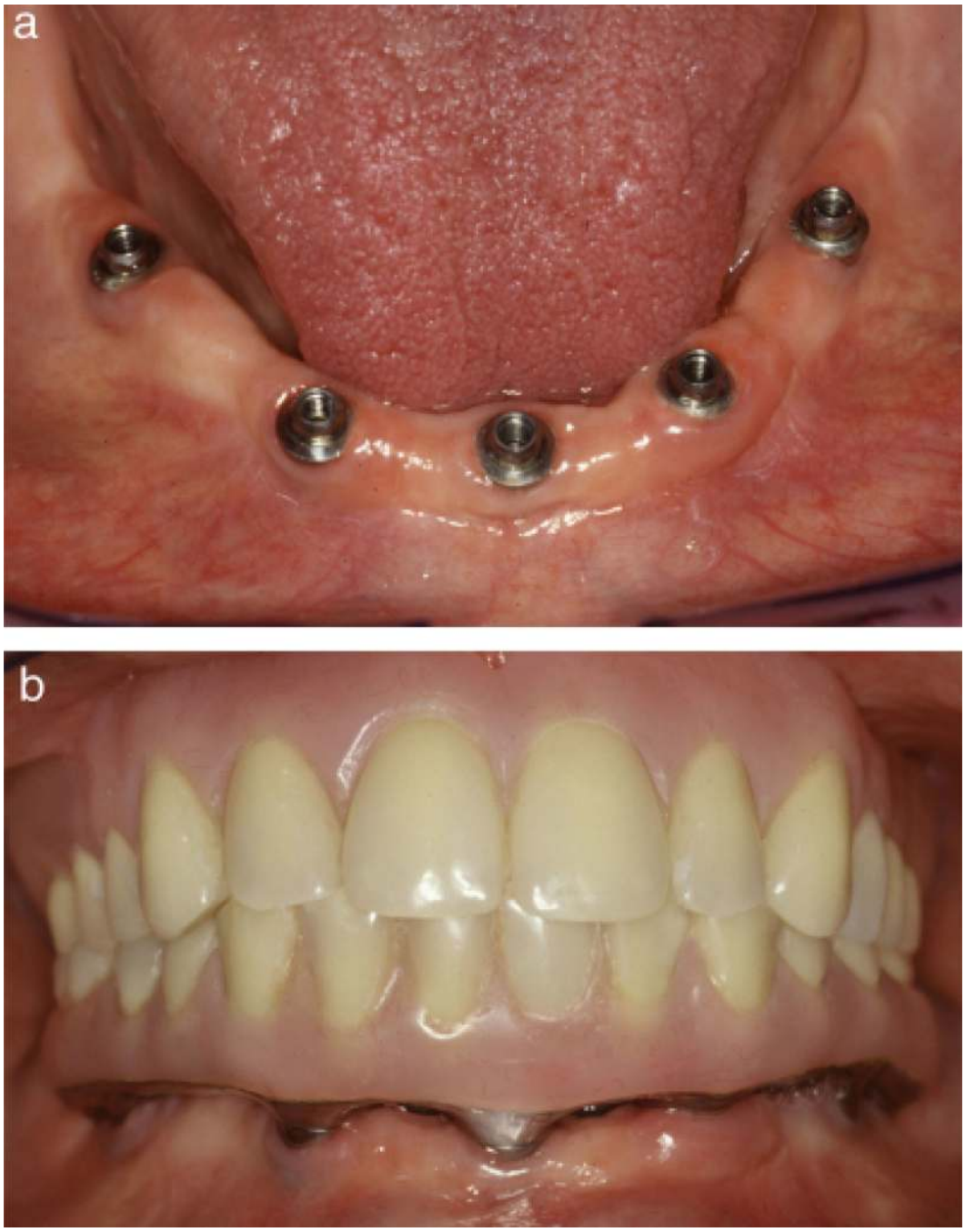

Fig. 1.

Prosthodontic treatment protocol. (a) All patients were treated with five to six one-stage dental implants placed in the symphysis of edentulous mandibles. (b) Semi-precious metal frameworks and acrylic resin prostheses with distal cantilevers were fabricated and screw-retained 4–7 months after implant placement.

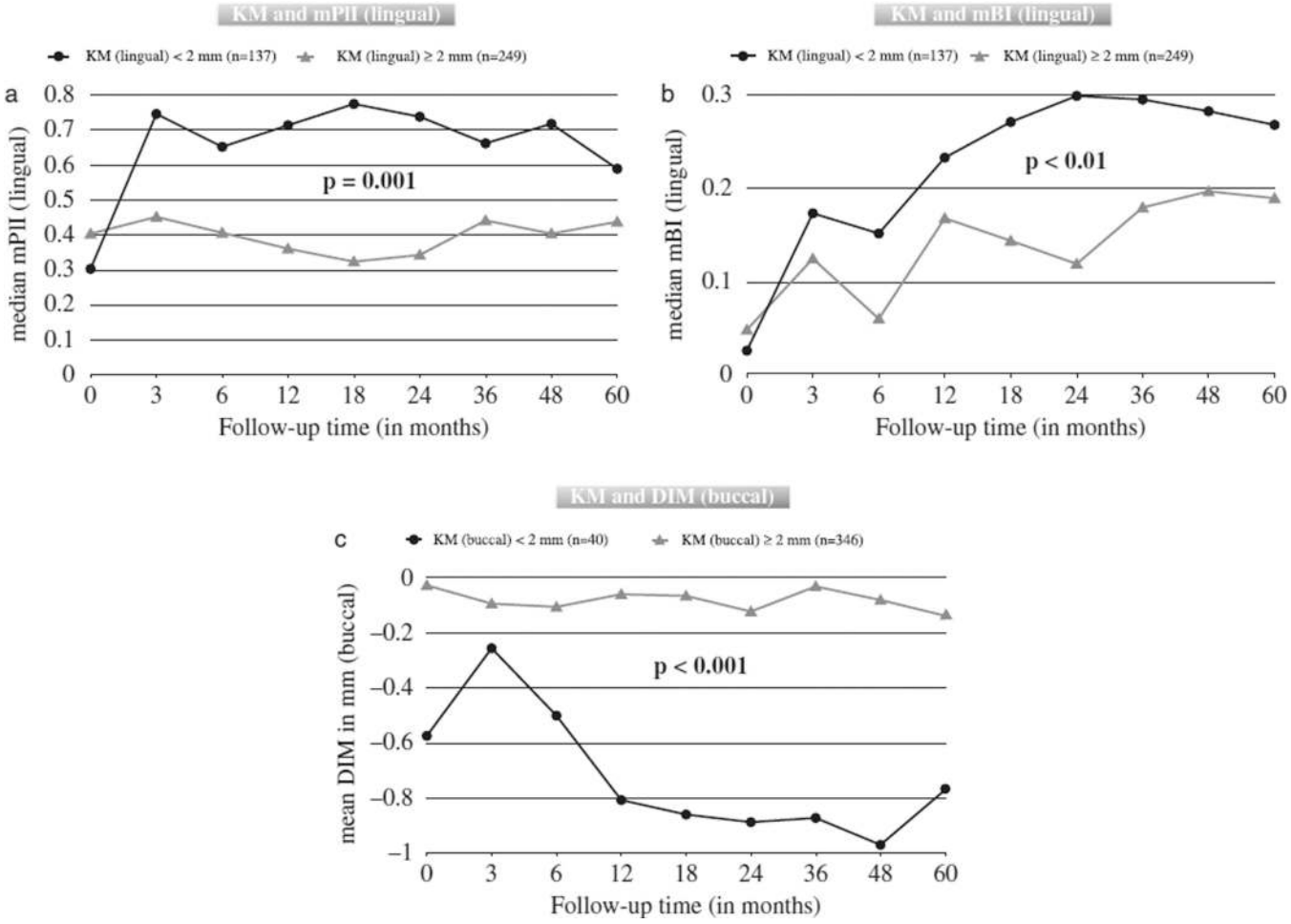

Fig. 2.

Clinical parameters affected by the width of keratinized mucosa. (a) Plaque accumulation (mPlI) at lingual sites: Lingual sites with < 2 mm of lingual KM presented with significantly more plaque accumulation compared to sites with at least 2 mm of lingual KM ($P = 0.001$). (b) Bleeding tendency (mBI) at lingual sites: Lingual sites with < 2 mm of lingual KM presented with significantly higher mBI scores compared to sites with at least 2 mm of lingual KM ($P < 0.01$). (c) Soft-tissue recession (DIM): Sites with < 2 mm of buccal KM presented with significantly greater soft-tissue recession compared to sites with at least 2 mm of buccal KM throughout the 5-year observation period ($P < 0.001$, DIM was measured at buccal sites only). mPlI, modified plaque index; KM, width of peri-implant keratinized mucosa; DIM, distance between implant shoulder and mucosal margin; mBI, modified sulcus bleeding index.

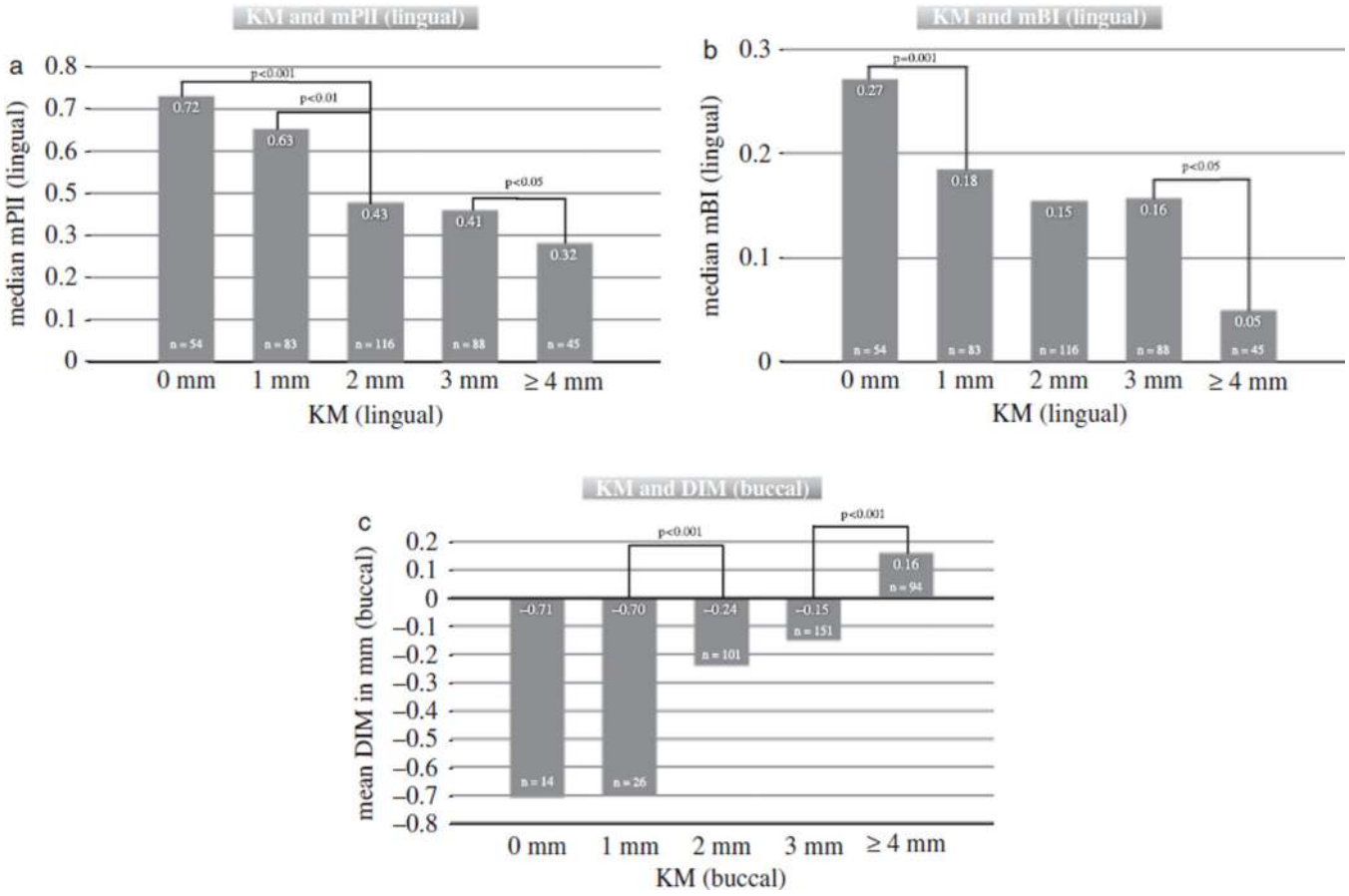

Fig. 3.

Outcome parameters according to different widths of KM. (a) Increasing mPlI scores on lingual sites with decreasing widths of KM can be seen. Statistically significant changes in outcome primarily occurred between 1 and 2 mm of lingual KM ($P < 0.01$ Bonferroni adjusted; n-median exposure level over follow-up). (b) Increasing mBI scores on lingual sites with decreasing widths of KM are evident. Statistically significant changes in outcome primarily occurred between 0 and 1 mm of lingual KM ($P < 0.001$ Bonferroni adjusted; n-median exposure level over follow-up). (c) Increasing soft-tissue recession on buccal sites with decreasing widths of buccal KM are evident. Changes in outcome primarily occurred between 1 and 2 mm of buccal KM ($P < 0.001$ Bonferroni adjusted; n-median exposure level over follow-up). mPlI, modified plaque index; KM, width of peri-implant keratinized mucosa; mBI, modified sulcus bleeding index.

Table 1

Study population

	Patients		Implants
	n	*%*	*N*
Gender			
Male	35	48	191
Female	38	52	195
Total	73	100	386
Smoking			
No	55	75	284
Yes	18	25	102
Age (mean)			
Male	57.3 years (SD 9.0, minimum 43, maximum 78)		
Female	58.7 years (SD 10.1, minimum 34, maximum 75)		
Total	58.0 years (SD 9.6, minimum 34, maximum 78)		

A total of 73 patients were treated with 386 implants in five clinical centers. Light smokers (≤10 cigarettes/day) were included in the study.