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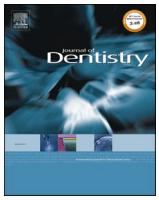

The influence of implant depth, abutment height and mucosal phenotype on peri-implant bone levels: A 2-year clinical trial

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ABSTRACT

Objectives: To evaluate the bone changes around equicrestal and subcrestal implants, analyzing the effect of abutment height [short abutments (SA < 2 mm) and long abutments (LA > 2 mm)] and the three components of the peri-implant soft-tissue phenotype.
Methods: Twenty-six patients received 71 implants that were placed according to supracrestal tissue height (STH) in an equicrestal ($n = 17$), shallow subcrestal ≈1 mm ($n = 33$), or deep subcrestal ≈2 mm ($n = 21$) position. After 3 months of healing, rehabilitation was completed using metal-ceramic crowns on multi-unit abutments of 1.5 mm, 2.5 mm, or 3.5 mm in height, depending on the prosthetic space and STH. Longitudinal clinical parameters (STH, mucosal thickness, and keratinized mucosa width) and radiographic data [bone remodelling and marginal bone loss (MBL)] were collected at 3, 6, 12, and 24 months postsurgery.
Results: The gain in STH was significantly greater around the implants placed in a subcrestal ≈2 mm position. After 2 years, the mean change in bone remodelling in the SA group was significantly greater than in the LA group. According to the multiple linear regression, bone remodelling depends primarily on abutment height (β = -0.43), followed by crestal position (β = 0.34), and keratinized mucosa width (β = -0.22), while MBL depends on abutment height (β = -0.37), and the patient's age (β = -0.36).
Conclusions: Implants placed in an equicrestal or subcrestal ≈1 mm position with LA undergo less bone remodelling, while the lowest level of MBL occurs in subcrestal ≈2 mm implants with LA. Differing soft-tissue thicknesses combined with the use of either SA or LA produced significant intergroup differences in bone remodelling and MBL.
Clinical significance: Abutment height is the most powerful predictor variable affecting bone remodelling and MBL. Depending on the dimensions of the peri-implant soft-tissue phenotype, placing the implants subcrestally may also be a viable option to decrease bone remodelling and, consequently, reduce MBL.
Clinical trial registration: identification number: NCT05670340.

Abbreviations

STH	Supracrestal tissue height
MT	Mucosal thickness
KMW	keratinized mucosa width
MBL	Marginal bone loss
SC≈1	Shallow subcrestal
SC≈2	Deep subcrestal
SA	Short abutment
LA	Long abutment

1. Introduction

Dental implants are a valuable resource for restoring dental functionality and aesthetics requiring several conditions for their long-term success, among the most important of which are stable peri-implant soft and hard tissues [1,2]. Early marginal bone loss (EMBL) of <0.5 mm in the first 6 months after prosthetic loading is currently considered

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indicative of success in implantology, whereas radiographic bone loss measuring >0.5 mm contributes to more severe peri-implant pathology [3,4].

These changes in marginal bone level are influenced by: surgical factors, such as surgical trauma or overheating during site preparation [5,6], the apico-coronal position of the implant shoulder [7], and use of a non-submerged or submerged technique [8]; biological factors, such as the formation of the biological space [9]; and prosthetic factors, of which prosthetic abutment height [10,11], the number of dis-connections/reconnections [12] and vertical soft tissue thickness [13] are particularly important.

In this context, a growing number of studies are investigating the three components of the peri-implant soft-tissue phenotype (morphology and dimensions), which not only impact aesthetics but also appear to influence hard-tissue stability [14–17]. Additionally, evidence from research and clinical experience supports the subcrestal placement of implants as a clinical strategy to promote increased supracrestal tissue height (STH) [18]. This gain in STH enables clinicians to select an adequate abutment height, considering that implants with prosthetic abutments <2 mm have been shown to lead to greater marginal bone loss (MBL) than implants with abutments ≥2 mm [11].

Over a minimum of 2 years, this clinical study aimed to radiographically evaluate bone remodelling and MBL around conical-connection implants placed in equicrestal or subcrestal positions, while also analyzing the effects of abutment height (short <2 mm versus long >2 mm) and peri-implant soft-tissue phenotype, the latter comprising keratinized mucosa width (KMW) mucosal thickness (MT) and supracrestal tissue height (STH). The null hypothesis was that none of the following factors would have any impact on bone loss (bone remodelling and MBL) associated with implants placed at different depths after at least 2 years of follow up: the three components of peri-implant soft-tissue phenotype (KMW, MT and STH), abutment height, or apico-coronal implant position with respect to the bone crest.

2. Material and methods

2.1. Study design

This clinical study with at least 24 months of follow up was conducted after evaluation and approval by the University of Bioethics Committee (reference number 473/2020). The study protocol was registered on clinicaltrials.gov (NCT05670340) and carried out in accordance with the Declaration of Helsinki on ethical principles for medical research involving human subjects (as revised in 2013). The CONSORT (Consolidated Standards of Reporting Trials) guidelines were followed throughout the study. A summary of the study timeline is provided in Fig. 1.

2.2. Patient selection

Research subjects were consecutively recruited, in line with the admission protocols of the School of Medicine's dental clinic at the University of . The target population included partially edentulous patients who needed at least two adjacent implants for restoration of either the posterior maxilla or mandible. All patients signed a specific informed consent form before participating in the project. After informed consent had been obtained, the following inclusion criteria were applied. Eligible patients:

(1) were aged over 18 years; (2) needed rehabilitation including at least two adjacent implants on healed bone crest (more than four months after tooth extraction) in the posterior region (premolars and molars); and (3) brushed their teeth ≥ twice per day. The exclusion criteria were as follows: (1) patients with medical conditions contraindicating implant surgery; (2) patients with immune disorders, pregnant women and nursing infants; (3) heavy smokers (≥20 cigarettes/day); (4) patients requiring bone or soft-tissue augmentation; and (5) patients whose anatomy would prevent correct three-dimensional positioning of a prosthetically driven screw-retained implant.

2.3. Sample size estimation

For the sample size calculation focusing on the major outcome variables (bone remodelling/bone loss), we assumed a pooled standard deviation of 0.3 mm, and an expected means ranging between 0.9 mm and 0.3 mm for the distinct groups. Based on that data dispersion, the study would require a subsample size of at least four implants to achieve a power of 80 % and a level of significance of 5 % (two-sided), in order to detect a true difference in means between the test and the reference group of 0.6 mm (i.e. 0.9 - 0.3 mm). These calculations were made using the online statistical calculator, Statulator (http://statulator.com/SampleSize/ss2M.html).

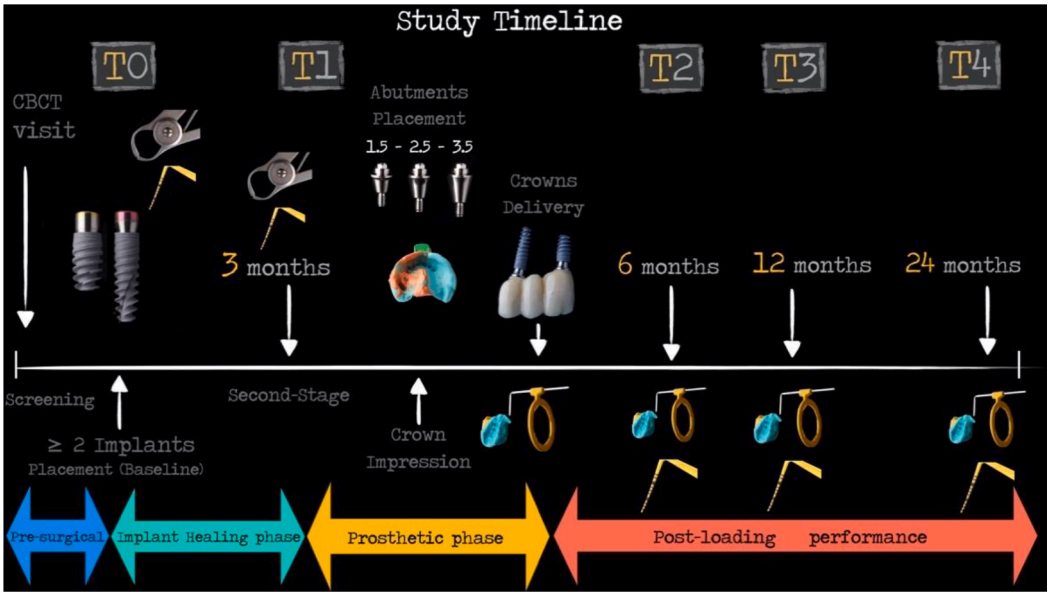

Fig. 1. Flow chart of time points for data collection.

2.4. Surgical treatment protocol

A detailed description of the surgical interventions, study devices and methods has been provided by [16]. In summary, after making the incisions and raising the buccal flap, a periodontal probe (PCP-UNC 15, Hu-friedy) was used to measure STH and KMW, and MT was measured using a dental caliper (Amazon; 2020). The osteotomies were performed by following the manufacturer's recommended drilling sequence. The researcher placed Nobel Active® implants with a 12-degree conical connection (Nobel Biocare, Kloten), choosing between three different lengths (8.5 mm, 10 mm or 13 mm) and diameters (3.5 mm, 4.3 mm or 5 mm), according to local bone availability and anatomical variations. These implants were placed at distinct apico-coronal positions, with reference to the distance between the implant shoulder and the buccal cortical bone, and were divided by placement depth into three groups: equicrestal, shallow subcrestal ≈1 mm (SC≈1), and deep subcrestal ≈2 mm (SC≈2) (Fig. 2). This placement distribution was designed to ensure at least 3 mm distance between the implant shoulder and the mucosal margin of the lingual/palatal flap [19,20]. To enable statistical inferences to be made about the effects of the peri-implant phenotype, each implant was also grouped by the amount of peri-implant mucosa present, categorizing STH ≤3.5 mm as short and >3.5 mm as tall, and classing MT <1 mm as thin and >1 mm as thick. As a longitudinal variable, KMW was categorized as narrow (≤2 mm) or wide or (>2 mm) at each follow-up point.

The 3- and 5-mm transgingival healing abutments were then placed to enable a one-stage submerged technique to be used with a conventional loading protocol [21]. Surgical sites were closed with simple interrupted sutures, using non-absorbable polyamide monofilament suture (5–0 Seralon, Serag-Wiessner). After flap closure, standardized periapical radiographs were taken.

Patients received anti-inflammatory medication (Enantyun; 25 mg tid) and were instructed to use a spray with 0.12 % chlorhexidine + 0.05 % cetylpyridinium chloride twice per day for 7 days. Sutures were removed after 7 days and follow-ups were performed at 1 and 2 months postsurgery.

2.5. Prosthetic treatment protocol

At 3 months postsurgery (T1), the healing abutment was removed for the first time, and the STH, KMW and MT parameters were measured again (Fig. 3). STH measurements were made from the implant shoulder to the buccal mucosal margin, using a periodontal probe with 1 mm markings. The STH at T1 and the interocclusal prosthetic space determined the abutment height selected, bearing in mind that a screwed structure needed to fit in, with at least 3 mm distance between the head of the prosthetic abutment and the antagonist [22,23]. Multi-unit Abutment Plus (Nobel Biocare AB) abutments of 1.5 mm, 2.5 mm or 3.5 mm height were placed, and standard open-tray impressions were taken on the multi-unit abutment with splinted impression copings.

One week later, the passive fit of the underlying structure was radiographically verified and, after 2 or 3 weeks, the final metal-ceramic screw-retained crowns were attached to the multi-unit abutments. The crowns were screwed onto the abutments with a torque of 20Ncm. The screw access channel was closed using Teflon and a photopolymerizable composite resin.

To analyze the effect of abutment height on bone remodelling and MBL, abutments were classified as short (SA) when under 2 mm in height (1.5 mm abutments) or long (LA) when over 2 mm (2.5 mm or 3.5 mm abutments), using the cut-off point provided by Galindo et al. [4, 22].

2.6. Variables

All data were collected by a researcher trained in standardized clinical examination (NQ), including cross-sectional and longitudinal data. Cross-sectional data were obtained on sociodemographic (sex and age), behavioural (smoking habits/ number of cigarettes) and clinical variables (implant diameter/length/location, and implant-platform depth with respect to the buccal cortical bone). Longitudinal data were collected on gingiva-related and bone-related variables. The variables were also divided into two groups by follow-up schedule. STH and MT were measured on the day of implant placement (T0) and 3 months postsurgery (T1), whereas KMW, bone remodelling and marginal bone loss were measured on the day of implant placement (T0), then at 3 months (T1), 6 months (T2), 12 months (T3) and 24 months (T4) postsurgery.

2.7. Radiographic analysis

The study devices and methodology have been described in detail in [16]. In summary, standardized periapical digital radiographs were taken (EzSensor Classic™ 1.5, Vatech) then analyzed using imaging software (Gesden VixWin platinum™, Kavo Dental, S.L.). Immediately

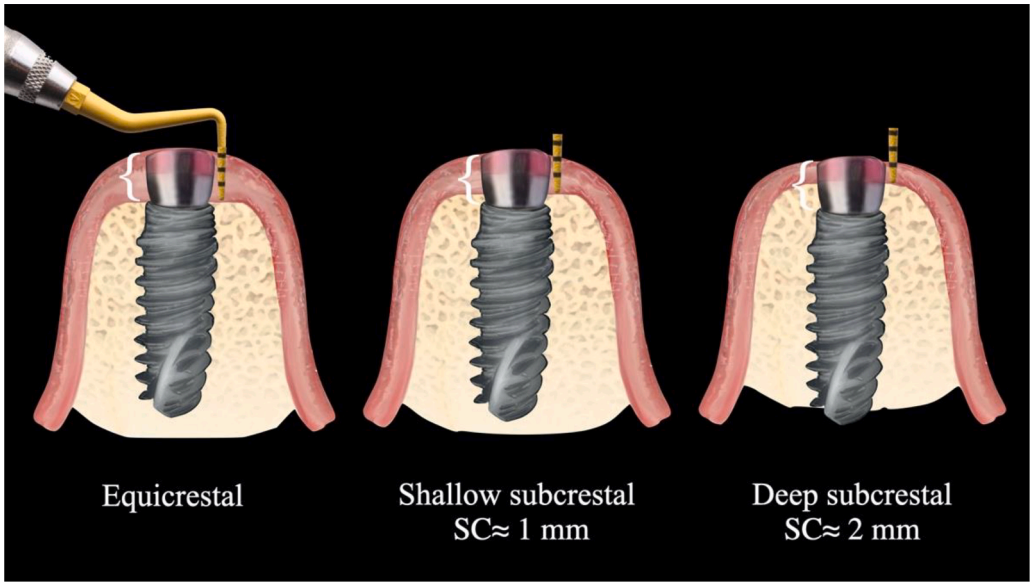

Fig. 2. Diagram showing the distance between the implant shoulder (IS) and the bone crest (BC), using the initial STH as a baseline.

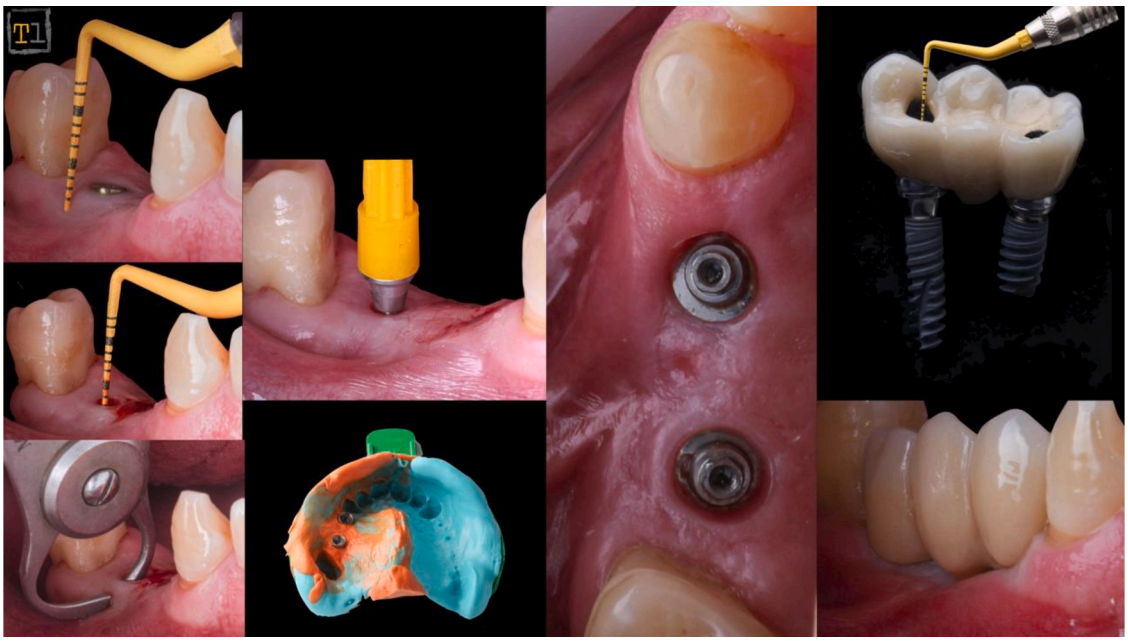

Fig. 3. Prosthetic phase (T1). Measurement of the peri-implant mucosa parameters (KMW, STH and MT), selection of the multi-unit abutment, impressions, and crown delivery.

after implant placement (T0), a baseline radiograph was taken. Radiographic evaluation was repeated: at 3 months postsurgery, upon crown delivery (T1); then at 6 months (T2), 12 months (T3), and 24 months (T4) postsurgery (Figs. 4 and 5). The following radiographic reference points (Fig. 4) were first recorded on the mesial and distal of each implant, to enable radiographic assessment of peri-implant bone changes: the most coronal level of the bone crest (BC), implant shoulder position (IS), and the most coronal level of the first radiographically visible bone to implant contact (fBIC). **Bone remodelling** (IS–BC) was calculated by measuring the vertical distance (mm) between the IS and the most coronal part of the mesial and distal BC. **Marginal bone loss** (IS–fBIC) was calculated by measuring the vertical distance (mm) between the IS and the fBIC on the mesial and distal sites. In the subsequent analyses, the final value used was the average of these mesial and distal measurements. The same researcher (JM) made each measurement twice for each implant, with a minimum interval between measurements of 15 days, in order to verify intra-examiner reliability by confirming that the intraclass correlation coefficient was $\geq$0.9 for the average of the radiographic parameters.

2.8. Statistical analysis

The data were tested for normality using the Kolmogorov-Smirnov and Shapiro-Wilk tests, according to the sample size of the subgroups.

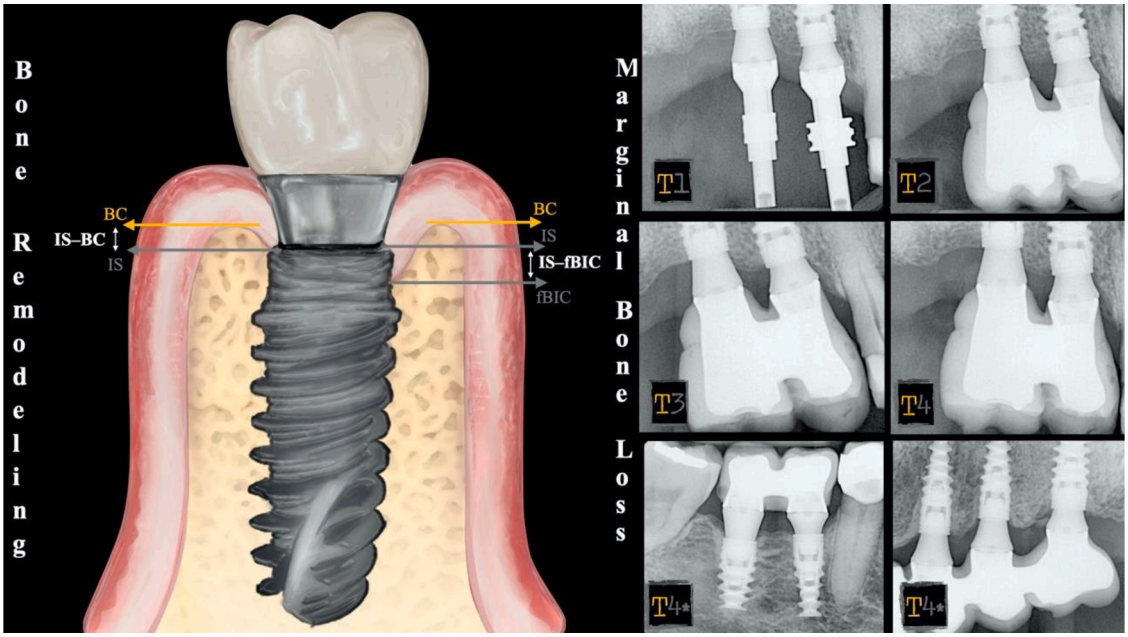

Fig. 4. Distances (IS–BC and IS–fBIC) and reference points used for calculation of bone remodelling and MBL. Clinical case (T1–T4) radiograph of a patient at the time of impression taking; (T2) radiograph after 6 months; (T3) radiograph after 1 year, and (T4) radiograph after 2 years. (T4*) Check-up of several patients after at least 2 years of follow up.

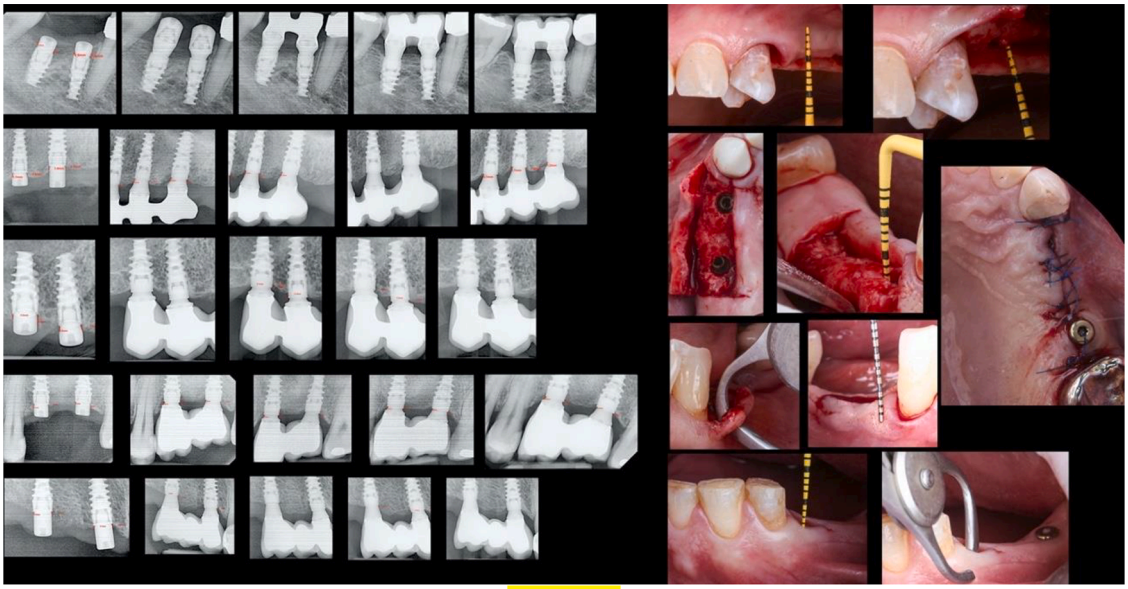

Fig. 5. Different radiographs where bone remodelling and MBL were evaluated during the two years of follow-up (T1–T4). Clinical measurements showing how the depth of implant placement were recorded and measurements of the peri-implant soft tissue phenotype (T1 and T2).

Irrespective of the normality and homoscedasticity result, the mean and standard deviation (SD) were used to describe the sample's quantitative variables (STH, MT, KMW, bone remodelling and MBL) rather than the median and interquartile range. However, the significance levels presented in the tables were obtained using parametric tests (the Student's *t*-test and analysis of variance/ ANOVA) or non-parametric tests (the Kruskal-Wallis and Mann-Whitney U tests) to compare the effect of the dependent variables (bone remodelling and MBL) on the crestal position of the implant, abutment height, and peri-implant mucosal phenotype, according to the result of the normality tests.

A stepwise linear regression was used to analyze the predictor variables, using bone remodelling and marginal bone loss after 2 years as dependent variables, after introducing the potential modulating factors (age, sex, smoking habits, implant width, location in the arch, KMW, MT, STH, depth of the implant shoulder with respect to the buccal cortical bone, and abutment height). Statistical analysis was conducted using SPSS v.26, with $p < 0.05$ as the threshold for determining statistical significance.

3. Results

Full data sets were collected for 71 implants in 26 patients (13 men and 13 women; mean age: 62.0 ± 11.5 years) (Table 1). All patients completed the follow-up evaluations, and all implants were available for at least a 24-month analysis, resulting in a 100 % survival rate. A total of 43 implants were placed in the maxilla (60.6 %) and 28 in the mandible (39.4 %). The distribution of implant depth with respect to the buccal cortical bone was as follows: 17 implants (23.9 %) in an equicrestal position, 33 implants (46.5 %) in a SC≈1 mm position and 21 implants (29.6 %) in a SC≈2 mm position (Table 1). Regarding abutment height, 25 abutments of 1.5 mm height (35.2 %), 31 abutments of 2.5 mm height (43.7 %), and 15 abutments of 3.5 mm (21.1 %) were placed. This variable was divided into two categories: 25 abutments <2 mm height (35.2 %), and 46 abutments >2 mm height (64.8 %).

The descriptive data on peri-implant mucosal phenotype at each of the five follow-up points are shown in Table 2 and Fig. 6. At the beginning of the study (T0), the mean STH was 2.9 ± 1.2 mm. At T1, the mean STH was 3.7 ± 1.2 mm. Therefore, the mean increase in STH between T0 and T1 was 0.8 ± 1.4 mm. Table 3 shows the analysis of mean increase in STH for each crestal-position group between the surgical (T0) and the prosthetic (T1) stages. An increase in STH was observed in both subcrestal groups, with statistically significant intragroup differences found for the SC≈1 ($p = 0.001$) and SC≈2 ($p = 0.001$) groups. The intergroup comparison of STH gain (T1–T0) showed significant differences ($p = 0.003$). A deeper implant position therefore helps create a significantly greater increase in STH.

For the analysis of mucosal thickness, two groups were created at T0 (Table 2): a group with a thin MT phenotype <1 mm (44 implants; 62.0 %), and another with a thick MT phenotype ≥1 mm (27 implants; 38.0 %). Between T0 and T1, the mean change in MT was 0.15 ± 0.53 mm, and statistically significant differences were found ($p = 0.02$).

Finally, a mean KMW of 4.1 ± 1.6 mm was recorded at T0, but the keratinized mucosa band had decreased in width at the prosthetic phase (T1) by an average of 0.74 ± 1.33 mm (95 % CI: 0.42 to 1.05 mm), and this KMW reduction was significant ($p < 0.001$). After 24 months of follow up (T0–T4), a decrease in KMW of 1.6 ± 1.8 mm was recorded (CI 95 %: 1.21 to 2.08 mm), and this reduction was also very significant ($p < 0.001$). In conclusion, a loss of KMW was observed over the entire follow-up period, the most significant decrease having occurred from T0 to T1 and T1 to T2, with KMW stabilizing from T2 onwards (Fig. 6).

3.1. Radiographic results (changes in interproximal bone level)

Table 4 shows the mean values and changes in bone remodelling and MBL at each follow-up point in each treatment group (equicrestal with SA and LA, SC≈1 mm with SA and LA, and SC≈2 mm with SA and LA). Our analysis of how implant depth and abutment height influenced supra-platform bone remodelling (Table 4) showed a degree of remodelling at all follow-up points, albeit of a greater magnitude in the first 6 months. Logically, the mean distance between the implant shoulder and bone crest (IS-BC) at implant placement (T0) was significantly smaller for the implants placed in equicrestal and shallow subcrestal positions ($p < 0.001$; 0.75 mm ± 0.14 and 0.73 ± 0.12 mm, respectively) than the implants in a deep subcrestal position (1.72 ± 0.59 mm).

When intergroup comparisons in bone remodelling were made at 3 follow-up points (T0–T1, T0–T3, and T0–T4), significant differences were found ($F = 2.55$, $p = 0.04$; $F = 3.92$, $p = 0.004$, and $F = 6.06$, $P < 0.001$, respectively). Therefore, it seems clear that placing implants at a greater depth leads to increased bone remodelling, however, abutment height plays an important role in this remodelling. The greatest change in bone remodelling was observed between surgery (T0) and crown delivery (T1), when there was more radiographic bone remodelling in

Table 1
Baseline description of the patient ($n = 26$) and implant ($n = 71$) samples.

Patient-related variables ($n = 26$)	Groups	(n,%)
Age (years)	<50 years	4 (15.4 %)
	50–65 years	11 (42.3 %)
	>65 years	11 (42.3 %)
Average age (Mean ± s.d.)	62.0 ± 11.5 years	
Gender (%)	Women	13 (50 %)
	Men	13 (50 %)
Smoking habits	Non-smokers	23 (88.5 %)
	Smokers	3 (11.5 %)
Average no of cigarettes: smokers (Mean ± s.d.)	5.0 ± 4.4	
Implant-related variables ($n = 71$)	**Groups**	**(n,%)**
Crestal level of the implant with respect to the buccal cortical bone (mm)	Equicrestal	17 (23.9 %)
	Shallow subcrestal ≈1mm	33 (46.5 %)
	Deep subcrestal ≈2mm	21 (29.6 %)
Abutment height (mm)	1.5 mm	25 (35.2 %)
	2.5mm	31 (43.7 %)
	3.5mm	15 (21.1 %)
Location in the dental arch	Maxilla	43 (60.6 %)
	Mandible	28 (39.4 %)
Implant length (mm)	8.5mm	9 (12.7 %)
	10mm	29 (40.8 %)
	11.5mm	30 (42.3 %)
	13mm	3 (4.2 %)
Implant width (Ø)	3.5 (Ø)	18 (25.4 %)
	4.3 (Ø)	51 (71.8 %)
	5.0 (Ø)	2 (2.8 %)

all the short-abutment (SA) groups. These intragroup changes were statistically significant for the equicrestal implant group with SA, and the SC≈1 mm group with SA (0.46 mm ± 0.33 mm, $p = 0.02$; and 0.44 mm ± 0.33 mm, $p < 0.001$, respectively).

Two years (T0–T4) postoperatively, there was least bone remodelling in the equicrestal implant group with LA (0.28 ± 0.44 mm), and this result was significantly lower than in the shallow subcrestal implants with SA (−0.49 mm, $p = 0.02$), and the deep subcrestal implants with SA (−0.63 mm, $p = 0.01$). The most bone remodelling after 24 months occurred in the SC≈2 mm with SA group (0.91 mm ± 0.26 mm), and this result was significantly higher than in the equicrestal implants with LA and SC≈1 mm implants with LA (0.63 ± 0.18, $p = 0.01$; 0.62 ± 0.17, $p = 0.006$).

After 2 years of follow-up, there had been an average MBL of 0.10 ± 0.18 mm around the implants ($n = 71$) but, when analyzed by subgroups (Table 4), the greatest changes were recorded in the three groups of implants rehabilitated with a SA. The intergroup comparisons showed statistically significant differences at all time points (T1–T0, T2–T0, T3–T0, and T4–T0). The largest change was observed in the SC≈1 mm with SA group (0.30 ± 0.27 mm), and there were statistically significant differences between this result and the SC≈1 mm with LA (0.25 ± 0.05 mm; $p = 0.001$) and the SC≈2 mm with LA (0.30 ± 0.06; $p < 0.001$) groups. The lowest mean MBL was recorded for the SC≈2 mm with LA

Table 2
Baseline and changes in soft-tissue parameters at each follow-up point ($n = 71$).

Timeline	Variable	Groups	N (%)	Mean ± SD
(T0) Implant placement	Supracrestal tissue height (mm)	≤2mm	26 (36.6 %)	2.9 ± 1.2
		2.1–3.5mm	26 (36.6 %)	
		>3.5mm	19 (26.8 %)	
	Mucosal thickness (mm)	Thin: <1 mm	44 (62.0 %)	1.0 ± 0.3
		Thick: ≥1mm	27 (38.0 %)	
	Keratinized mucosa width (mm)	Narrow: ≤2mm	14 (19.7 %)	4.1 ± 1.6
		Wide: >2mm	57 (80.3 %)	
(T1) Prosthetic phase	Supracrestal tissue height (mm)	≤2mm	11 (15.5 %)	3.7 ± 1.2
		2.1–3.5mm	18 (25.4 %)	
		>3.5mm	42 (59.2 %)	
	Change in supracrestal tissue height T1–T0	0.8 ± 1.4 (95 % CI: 0.51 to 1.16 mm)		
	Mucosal thickness (mm)	Thin: <1 mm	29 (40.8 %)	1.1 ± 0.5
		Thick: ≥1 mm	42 (59.2 %)	
	Change in mucosal thickness T1–T0	0.15 ± 0.53 (95 % CI: 0.02 to 0.28)		
	Keratinized mucosa width (mm)	Narrow: ≤2mm	20 (28.2 %)	3.3 ± 1.4
		Wide: >2mm	51 (71.8 %)	
	Change in keratinized mucosa width T1–T0 (mm)	−0.74 ± 1.33 (95 % CI: −1.05 to −0.42)		
(T2) 6 mths after implant placement	Keratinized mucosa width (mm)	Narrow: ≤2mm	36 (50.7 %)	2.5 ± 1.2
		Wide: >2mm	35 (49.3 %)	
	Change in keratinized mucosa width T2–T0 (mm)	−1.59 ± 1.62 (95 % CI: −1.98 to −1.21 mm)		
(T3) 12 mths after implant placement	Keratinized mucosa width (mm)	Narrow: ≤2mm	42 (59.2 %)	2.4 ± 1.3
		Wide: >2mm	29 (40.8 %)	
	Change in keratinized mucosa width T3–T0 (mm)	−1.7 ± 1.6 (95 % CI: −2.06 to −1.31 mm)		
(T4) 24 mths after implant placement	Keratinized mucosa width (mm)	Narrow: ≤2mm	42 (59.2 %)	2.4 ± 1.3
		Wide: >2mm	29 (40.8 %)	
	Change in keratinized mucosa width T4–T0 (mm)	−1.6 ± 1.8 (95 % CI: −2.08 to −1.21 mm)		

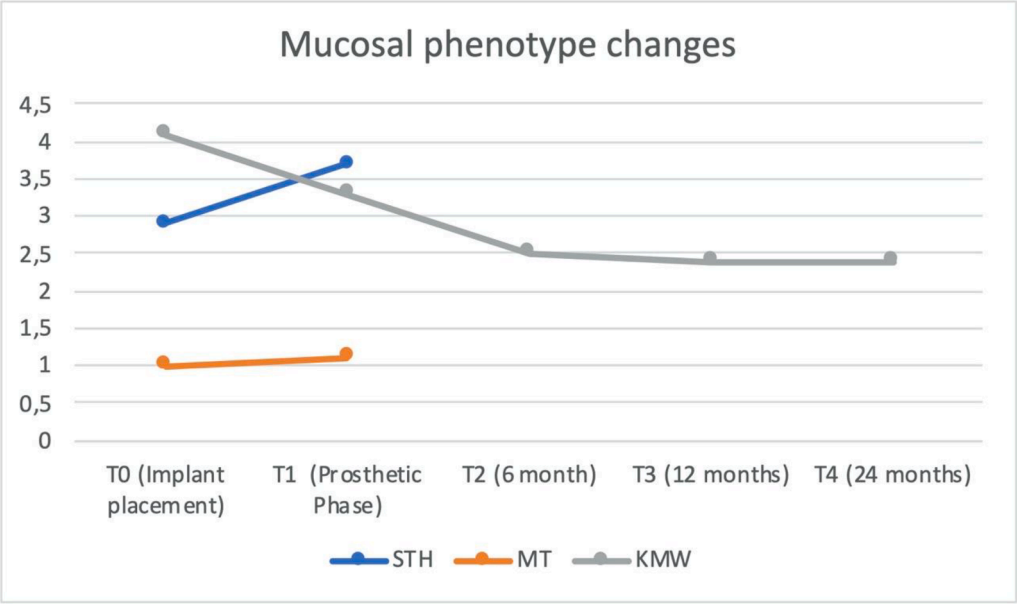

Fig. 6. Changes in the components of the soft-tissue phenotype (STH, MT, KMW) over time.

Table 3
Effect of implant position on the gain in supracrestal tissue height between the T0 and T1 follow-up points. Intergroup comparison using the Kruskal-Wallis test (K-W) and intragroup comparison using the paired Wilcoxon test.

Implant position with respect to the bone crest						
Variable	Clinical phases Groups (n)	Equicrestal ^{A} ($n = 17$) Mean ± SD	Shallow subcrestal ^{B} ≈ 1 mm ($n = 33$) Mean ± SD	Deep subcrestal ^{C} ≈ 2 mm ($n = 21$) Mean ± SD	Kruskal Wallis	p-value
Supracrestal tissue height	(T0) Implant placement	3.35 ± 1.47	2.91 ± 1.15	2.55 ± 0.93	3.47	0.18
	(T1) Prosthetic phase	3.41 ± 1.24	3.67 ± 1.10	4.14 ± 1.24	3.11	0.21
	T1–T0 comparison (Wilcoxon; p-value)	0.06 ± 1.16 ^{C} ($P = 0.81$)	0.76 ± 1.12 ($P = 0.001$)	1.60 ± 1.54 ^{A} ($P = 0.001$)	11.46	0.003

A,B,C: the superscript letters after the means indicate statistically significant comparisons after the Bonferroni post hoc tests ($p < 0.05$) with these columns.

(0.007 ± 0.03 mm).

Table 5 shows the mean values and changes in bone remodelling at each follow-up point in each treatment group (short STH with SA and LA; tall STH with SA and LA; thin MT with SA and LA; thick MT with SA and LA; narrow KMW with SA and LA, and wide KMW with SA and LA). When the effect of STH on bone remodelling was analyzed, more remodelling was observed in the SA group than in the group where a LA was used. From 1 year postsurgery (T0–T3) to the end of the follow-up period (T0–T4), only slight changes in bone remodelling were recorded. At 2 years postsurgery, a lower level of bone remodelling was observed in the tall STH with LA group (0.42 ± 0.47 mm) than in the short STH with SA group (0.76 ± 0.25 mm), and this difference was statistically significant (0.35 ± 0.11 mm; $p = 0.01$).

Analysis of the relationship between bone remodelling and MT (Table 5) showed that, after 2 years (T0–T4), the most remodelling occurred in the group with MT < 1 mm when a SA was used. In fact, the MT <1 mm with SA group underwent significantly greater bone remodelling than the MT < 1 mm with LA group (0.35 mm ± 0.12 mm; $p = 0.02$) or the MT ≥ 1 mm with LA group (0.48 mm ± 0.12 mm; $p = 0.001$). According to the trend observed, the greater the MT, the less bone remodelling occurs and, within each group, use of a LA results in less bone remodelling than use of a SA. Regarding the relationship between bone remodelling and KMW (Table 5), greater levels of bone remodelling were observed with KMW ≤2 mm and use of a SA.

Table 6 shows the mean values and changes in MBL at each observation point in each treatment group (short STH with SA and LA; tall STH with SA and LA; thin MT with SA and LA; thick MT with SA and LA; narrow KMW with SA and LA, and wide KMW with SA and LA). Analysis of the effect of STH on MBL (Table 6) showed that, at 2 years postsurgery, a lower level of MBL was observed (−0.15 ± 0.05 mm; $p = 0.001$) in the tall STH with LA group (0.05 ± 0.11 mm) than in the short STH with SA group (0.20 ± 0.23 mm).

Regarding the relationship between MBL and MT (Table 6), greater MBL was observed in the group of implants rehabilitated with a SA. At 2 years postoperatively, the MT <1 mm with LA group (0.02 ± 0.08 mm) had undergone significantly less MBL than the MT <1 mm with SA group (0.16 ± 0.05 mm; $p = 0.002$) or the MT >1 mm with SA group (0.26 ± 0.07 mm; $p = 0.002$). When the relationship between MBL and the width of the keratinized mucosa band was evaluated (Table 6), significant intergroup differences were found at all follow-up points (T2–T0; T3–T0, and T4–T0). After 2 years, the greatest levels of MBL were observed with KMW ≤2 mm and use of a SA (0.21 ± 0.24 mm). This bone loss was significantly greater than that recorded in the KMW ≤2 mm with LA group (0.13 ± 0.05 mm; $p = 0.04$) and the KMW >2 mm with LA group (0.19 ± 0.05 mm; $p < 0.001$).

A stepwise linear regression model was created (Table 7) in which bone remodelling and MBL were each analyzed as dependent variables. The following predictor variables were used: age, sex, implant diameter, location in the arch, KMW, MT, STH, depth of the implant shoulder with respect to the buccal cortical bone, and height of the multi-unit abutment. A set of three variables, and another set of two, were found to be significantly predictive of bone remodelling ($F = 10.76$; $p < 0.001$) and of MBL, respectively ($F = 14.46$; $p < 0.001$). Therefore, 33 % of the bone remodelling could be predicted if the abutment height, crestal position

Table 4
Effect of the crestal position of the implant on peri-implant bone remodelling and loss at each follow-up point. Intergroup comparison using ANOVA and K-W, and intragroup comparison using the paired Student's *t*-test and Wilcoxon test.

Implant position with respect to the bone crest									
Variable	Timeline	Equicrestal ($n = 17$)		Shallow subcrestal ≈1 mm ($n = 33$)		Deep subcrestal ≈2 mm ($n = 21$)		ANOVA F	p-value
	Groups (n)	Short abutment [A] ($n = 6$) Mean ± SD	Long abutment [B] ($n = 11$)	Short abutment [C] ($n = 13$) Mean ± SD	Long abutment [D] ($n = 20$)	Short abutment [E] ($n = 6$) Mean ± SD	Long abutment [F] ($n = 15$)		
BONE REMODELLING	(T0) Implant placement	0.73 ± 0.37 [E,F]	1.10 ± 0.40 [F]	0.89 ± 0.21 [E,F]	1.05 ± 0.37 [F]	1.55 ± 0.56 [A,C]	1.78 ± 0.60 [A,B,C,D]	**9.66**	**<0.001**
	(T1) Crown placement	0.28 ± 0.28 [F]	0.99 ± 0.73	0.45 ± 0.35 [F]	0.96 ± 0.49	1.12 ± 0.45	1.48 ± 0.57 [A,C]	**7.81**	**<0.001**
	(T2) 6 mths after implant placement	0.13 ± 0.30 [B,F]	0.91 ± 0.65 [A,C]	0.23 ± 0.25[B,D,F]	0.81 ± 0.47 [C]	0.93 ± 0.57	1.26 ± 0.50 [A,C]	**8.96**	**<0.001**
	(T3) 12 mths after implant placement	0.13 ± 0.26 [F]	0.82 ± 0.64 [C]	0.18 ± 0.23[B,D,E,F]	0.77 ± 0.50 [C]	0.94 ± 0.61[C]	1.16 ± 0.55 [AC]	**7.57**	**<0.001**
	(T4) 24 mths after implant placement	0.12 ± 0.19 [F]	0.82 ± 0.67 [C]	0.12 ± 0.19 [B,D,F]	0.76 ± 0.46 [C]	0.64 ± 0.54	1.10 ± 0.48 [A,C]	**8.48**	**<0.001**
	T0–T1 comparison (Student's T; p-value)	0.46 ± 0.33 ($P = 0.02$)	0.10 ± 0.45 ($P = 0.52$)	0.44 ± 0.33 ($P = <0.001$)	0.09 ± 0.27 ($P = 0.15$)	0.43 ± 0.39 ($P = 0.10$)	0.30 ± 0.44 ($P = 0.004$)	**2.55**	**0.04**
	T0–T3 comparison (Student's T; p-value)	0.61 ± 0.22 ($P = 0.001$)	0.28 ± 0.40 ($P = 0.02$)	0.71 ± 0.28 [D] ($P = <0.001$)	0.28 ± 0.34 [C] ($P = <0.001$)	0.61 ± 0.37 ($P = 0.01$)	0.63 ± 0.43 ($P = <0.001$)	**3.92**	**0.004**
	T0–T4 comparison (Student's T; p-value)	0.62 ± 0.30 ($P = 0.004$) [‡]	0.28 ± 0.44 [C,E] ($P = 0.02$)	0.77 ± 0.21[B,D] ($P = <0.001$)	0.29 ± 0.36[C,E,F] ($P = 0.001$)	0.91 ± 0.26[B,D] ($P = 0.002$)	0.68 ± 0.44 [D] ($P = <0.001$)	**6.06**	**<0.001**
								K-W	**p-value**
BONE LOSS	(T0) Implant placement	0.00 ± 0.0	0.00 ± 0.0	0.00 ± 0.0	0.00 ± 0.0	0.00 ± 0.0	0.00 ± 0.0	**0**	**1**
	(T1) Crown placement	0.04 ± 0.07	0.02 ± 0.05	0.10 ± 0.15 [D,F]	0.0 ± 0.0 [C]	0.0 ± 0.0	0.0 ± 0.0 [C]	**16.5**	**0.006**
	(T2) 6 mths after implant placement	0.08 ± 0.09	0.04 ± 0.07	0.18 ± 0.17 [D,F]	0.01 ± 0.05 [C]	0.03 ± 0.06	0.0 ± 0.0 [C]	**21.7**	**0.001**
	(T3) 12 mths after implant placement	0.07 ± 0.08	0.06 ± 0.12	0.18 ± 0.17 [D,F]	0.04 ± 0.11 [C]	0.05 ± 0.12	0.0 ± 0.0 [C]	**21.4**	**0.001**
	(T4) 24 mths after implant placement	0.13 ± 0.06 [F]	0.10 ± 0.14	0.30 ± 0.27 [D,F]	0.05 ± 0.14 [C]	0.05 ± 0.12	0.007 ± 0.03 [A,C]	**27.7**	**<0.001**
	T1–T0 comparison (Wilcoxon; p-value)	0.04 ± 0.07 ($p = 0.18$)	0.02 ± 0.05 ($p = 0.18$)	0.10 ± 0.15 [D,F] ($p = 0.04$)	0.0 ± 0.0 [C] ($P = 1$)	0.0 ± 0.0 ($P = 1$)	0.0 ± 0.0 [C] ($P = 1$)	**16.5**	**0.006**
	T2–T0 comparison (Wilcoxon; p-value)	0.08 ± 0.09 ($p = 0.10$)	0.04 ± 0.07 ($p = 0.10$)	0.18 ± 0.17 [D,F] ($p = 0.01$)	0.01 ± 0.05 [C] ($p = 0.18$)	0.03 ± 0.06 ($p = 0.32$)	0.0 ± 0.0 [C] ($P = 1$)	**21.7**	**0.001**
	T3–T0 comparison (Wilcoxon; p-value)	0.07 ± 0.08 ($p = 0.10$)	0.06 ± 0.12 ($p = 0.11$)	0.18 ± 0.17 [D,F] ($p = 0.008$)	0.04 ± 0.11 [C] ($p = 0.18$)	0.05 ± 0.12 ($p = 0.32$)	0.0 ± 0.0 [C] ($P = 1$)	**21.4**	**0.001**
	T4–T0 comparison (Wilcoxon; p-value)	0.13 ± 0.06 [F] ($p = 0.03$)	0.10 ± 0.14 ($p = 0.06$)	0.30 ± 0.27 [D,F] ($p = 0.005$)	0.05 ± 0.14 [C] ($p = 0.10$)	0.05 ± 0.12 ($p = 0.32$)	0.007 ± 0.03 [A,C] ($p = 0.32$)	**27.7**	**<0.001**

A,B,C,D,E,F: the superscript letters after the means indicate statistically significant comparisons after the Bonferroni post hoc tests ($p < 0.05$) with these columns. Abbreviations: SD, standard deviation; K-W, Kruskal-Wallis.

Table 5
Effect of the peri-implant soft-tissue phenotype on bone remodelling at each follow-up point.

Supracrestal tissue height							
	Groups (n)	Short ≤3.5 mm (*n* = 29)	Tall >3.5 mm (*n* = 42)	F	p-value		
		Short Abutment (*n* = 21) [A]	Long Abutment (*n* = 8) [B]	Short Abutment (*n* = 4) [C]	Long Abutment (*n* = 38) [D]		
BONE REMODELLING		Mean ± SD	Mean ± SD				
	(T1) Crown placement	0.54 ± 0.48 [D]	0.84 ± 0.39	0.69 ± 0.48	1.20 ± 0.65 [A]	6.32	0.001
	(T2) 6 mths after implant placement	0.32 ± 0.43 [D]	0.68 ± 0.44	0.65 ± 0.64	1.05± 0.56 [A]	9.03	<0.001
	(T3) 12 mths after implant placement	0.30 ± 0.43 [D]	0.65 ± 0.45	0.63 ± 0.74	0.96 ± 0.58 [A]	7.14	<0.001
	(T4) 24 mths after implant placement	0.20 ± 0.33 [D]	0.58 ± 0.44	0.50 ± 0.50	0.95 ± 0.53 [A]	11.88	<0.001
	T0–T3 comparison (Student T; p-value)	0.66 ± 0.26 (*P*=<0.001)	0.32 ± 0.22 (*P* = 0.004)	0.65 ± 0.42 (*P* = 0.05)	0.41± 0.44 (*P*=<0.001)	2.89	0.04
	T0–T4 comparison (Student's T; p-value)	0.76 ± 0.25 [D] (*P*=<0.001)	0.39 ± 0.27 (*P* = 0.005)	0.78 ± 0.31 (*P* = 0.02)	0.42 ± 0.47 [A] (*P*=<0.001)	4.42	0.007
Mucosal thickness							
BONE REMODELLING	Groups (n)	**Thin <1 mm (*n* = 44)**	**Thick ≥1 mm (*n* = 27)**	**F**	**p-value**		
		Short Abutment (*n* = 19) [A]	**Long Abutment (*n* = 25) [B]**	**Short Abutment (*n* = 6) [C]**	**Long Abutment (*n* = 21) [D]**		
	(T1) Crown placement	0.63 ± 0.50 [B]	1.31 ± 0.66 [A,C]	0.35 ± 0.33 [B]	0.93 ± 0.51	7.84	<0.001
	(T2) 6 mths after implant placement	0.44 ± 0.50 [B]	1.13 ± 0.58 [A,C]	0.17 ± 0.29 [B]	0.80 ± 0.47*	9.54	<0.001
	(T3) 12 mths after implant placement	0.42 ± 0.52 [B]	1.05 ± 0.60 [A,C]	0.13 ± 0.26 [B]	0.74 ± 0.48*	7.96	<0.001
	(T4) 24 mths after implant placement	0.28 ± 0.40 [B,D]	0.98 ± 0.57 [A,C]	0.14 ± 0.25 [B,D]	0.77 ± 0.47 [A,C]	10.46	<0.001
	T0–T3 comparison (Student T; p-value)	0.68 ± 0.30 (*P*=<0.001)	0.40 ± 0.47 (*P*=<0.001)	0.60 ± 0.22 (*P* = 0.001)	0.38± 0.35 (*P*=<0.001)	2.84	0.04
	T0–T4 comparison (Student's T; p-value)	0.82 ± 0.23 [B,D] (*P*=<0.001)	0.47 ± 0.47 [A] (*P*=<0.001)	0.58 ± 0.26 (*P* = 0.003)	0.35 ± 0.40 [A] (*P* = 0.001)	5.60	0.002
Keratinized mucosa width T2							
BONE REMODELLING	Groups (n)	**Narrow ≤2 mm (*n* = 36)**	**Wide >2 mm (*n* = 35)**	**F**	**p-value**		
		Short Abutment (*n* = 16) [A]	**Long Abutment (*n* = 20) [B]**	**Short Abutment (*n* = 9) [C]**	**Long Abutment (*n* = 26) [D]**		
	T0–T2 comparison (Student T; p-value)	0.59 ± 0.22 [D] (*P*=<0.001)	0.42 ± 0.38 (*P*=<0.001)	0.71 ± 0.29 [D] (*P*=<0.001)	0.24 ± 0.38 [A,C] (*P* = 0.004)	5.96	0.001
	Keratinized mucosa width T3						
		Narrow ≤2 mm (*n* = 42)	**Wide >2 mm (*n* = 29)**	**F**	**p-value**		
		Short Abutment (*n* = 18)	**Long Abutment (*n* = 24)**	**Short Abutment (*n* = 7)**	**Long Abutment (*n* = 22)**		
	T0–T3 comparison (Student T; p-value)	0.70 ± 0.24 [D] (*P*=<0.001)	0.48 ± 0.40 (*P*=<0.001)	0.56 ± 0.38 (*P* = 0.008)	0.30 ± 0.41 [A] (*P* = 0.003)	4.0	0.01
	Keratinized mucosa width T4						
		Narrow ≤2 mm (*n* = 42)	**Wide >2 mm (*n* = 29)**	**F**	**p-value**		
		Short Abutment (*n* = 19)	**Long Abutment (*n* = 23)**	**Short Abutment (*n* = 6)**	**Long Abutment (*n* = 23)**		
	T0–T4 comparison (Student T; p-value)	0.80 ± 0.24 [B,D] (*P*=<0.001)	0.47 ± 0.33 [A] (*P*=<0.001)	0.65 ± 0.29 (*P* = 0.003)	0.36 ± 0.53 [A] (*P* = 0.004)	5.02	0.003

A, B, C, D: the superscript letters after the means indicate statistically significant comparisons after the Bonferroni post hoc tests ($p < 0.05$) with these columns. Abbreviation: SD, standard deviation.

of the implant with respect to the bone, and KMW are known. This model indicates that bone remodelling is determined primarily by abutment height (standardized $\beta = -0.43$; p=<0.001), since the longer the abutment used, the less bone remodelling occurs (95 % CI: from −0.55 mm to −0.19 mm). The next most influential variable is the crestal position of the implant with respect to the bone (standardized $\beta =$ 0.34), since the deeper the implant is placed subcrestally, the more bone remodelling occurs (95 % CI: 0.008 mm to 0.031 mm). Finally, KMW (standardized $\beta = -0.22$; $p = 0.04$) influences bone remodelling, since KMW of over 2 mm around the implants results in a lower level of bone remodelling (95 % CI: −0.036 mm to −0.001 mm) (Table 7 and Fig. 7). Thirty per cent of MBL can be predicted when the abutment height and the patient's age are known. MBL depends primarily on abutment height (standardized $\beta = -0.37$; $p = 0.001$). Therefore, the longer the abutments placed, the less MBL occurs (95 % CI: −0.22 to −0.06 mm). The second most influential variable is patient age (standardized $\beta = -0.36$), whereby with increasing age, the level of MBL decreases (95 % CI: −0.008 to −0.002 mm) (Table 7 and Fig. 7).

4. Discussion

The present study has verified that the initial supracrestal tissue height (STH–T0) is increased by adapting the subcrestal position of the implant. The deeper the implant placement, the greater the gain in STH,

Table 6
Effect of the peri-implant soft-tissue phenotype on marginal bone loss at each follow-up point.

Supracrestal tissue height							
B	Groups (n)	Short ≤3.5 mm ($n = 29$)		Tall >3.5 mm ($n = 42$)		K-W	p-value
O		Short abutment ($n = 21$)	Long abutment ($n = 8$)	Short abutment ($n = 4$)	Long abutment ($n = 38$)		
N		Mean ± SD		Mean ± SD			
E	(T1) Crown placement	0.05 ± 0.10	0.0 ± 0.0	0.10 ± 0.20	0.007 ± 0.03	8.53	0.04
L	(T2) 6 mths after implant placement	0.11 ± 0.15 ^{D}	0.01 ± 0.04	0.14 ± 0.19	0.01 ± 0.05 ^{A}	13.84	0.003
O	(T3) 12 mths after implant placement	0.11 ± 0.14 ^{D}	0.04 ± 0.12	0.19 ± 0.23	0.03 ± 0.09 ^{A}	13.22	0.004
S	(T4) 24 mths after implant placement	0.20 ± 0.23 ^{B,D}	0.05 ± 0.14 ^{A}	0.21 ± 0.27	0.05 ± 0.11 ^{A}	17.15	0.001
S	T3–T0 comparison	0.11 ± 0.14 ^{D}	0.04 ± 0.12	0.19 ± 0.23	0.03 ± 0.09 ^{A}	13.22	0.004
	(Wilcoxon; p-value)	($P = 0.003$)	($P = 0.32$)	($P = 0.18$)	($P = 0.07$)		
	T4–T0 comparison	0.20 ± 0.23 ^{B,D}	0.05 ± 0.14 ^{A}	0.21 ± 0.27	0.05 ± 0.11 ^{A}	17.15	0.001
	(Wilcoxon; p-value)	($P = 0.001$)	($P = 0.32$)	($P = 0.18$)	($P = 0.02$)		
Mucosal thickness							
		Thin <1 mm ($n = 44$)		Thick ≥1 mm ($n = 27$)		K-W	p-value
B	Groups (n)	Short	Long	Short	Long		
O		abutment ($n = 19$)	abutment ($n = 25$)	abutment ($n = 6$)	abutment ($n = 21$)		
N	(T1) Crown placement	0.05 ± 0.10	0.004 ± 0.20	0.08 ± 0.16	0.007 ± 0.03	8.62	0.04
E	(T2) 6 mths after implant placement	0.08 ± 0.11 ^{B}	0.006 ± 0.02 ^{A,C}	0.23 ± 0.20 ^{B,D}	0.02 ± 0.06 ^{C}	17.12	0.001
L	(T3) 12 mths after implant placement	0.09 ± 0.11^{B}	0.01 ± 0.07 ^{A,C}	0.23 ± 0.22 ^{B,D}	0.05 ± 0.12 ^{C}	16.24	0.001
O	(T4) 24 mths after implant placement	0.18 ± 0.23 ^{B}	0.02 ± 0.08 ^{A,C}	0.28 ± 0.23 ^{B,D}	0.08 ± 0.15 ^{C}	20.61	<0.001
S	T3–T0 comparison	0.09 ± 0.11^{B}	0.01 ± 0.07 ^{AC}	0.23 ± 0.22 ^{B}	0.05 ± 0.12	16.24	0.001
S	(Wilcoxon; p-value)	($P = 0.007$)	($P = 0.32$)	($P = 0.07$)	($P = 0.07$)		
	T4–T0 comparison	0.18 ± 0.23 ^{B}	0.02 ± 0.08 ^{A,C}	0.28 ± 0.23 ^{B,D}	0.08 ± 0.15 ^{C}	20.61	<0.001
	(Wilcoxon; p-value)	($P = 0.002$)	($P = 0.18$)	($P = 0.04$)	($P = 0.03$)		
Keratinized mucosa width T2							
	Groups (n)	Narrow ≤2 mm ($n = 36$)		Wide >2 mm ($n = 35$)		K-W	p-value
B		Short	Long	Short	Long		
O		abutment ($n = 16$)	abutment ($n = 20$)	abutment ($n = 9$)	abutment ($n = 26$)		
N	T2–T0 comparison	0.09 ± 0.15	0.02 ± 0.06 ^{C}	0.17 ± 0.14 ^{B,D}	0.01 ± 0.03 ^{C}	16.97	0.001
E	(Wilcoxon; p-value)	($P = 0.03$)	($P = 0.16$)	($P = 0.03$)	($P = 0.10$)		
L	Keratinized mucosa width T3						
O		Narrow ≤2 mm ($n = 42$)		Wide >2 mm ($n = 29$)		K-W	p-value
S		Short	Long	Short	Long		
S		abutment ($n = 18$)	abutment ($n = 24$)	abutment ($n = 7$)	abutment ($n = 22$)		
	T3–T0 comparison	0.13 ± 0.14 ^{D}	0.05 ± 0.12	0.12 ± 0.18	0.01 ± 0.05 ^{A}	14.26	0.003
	(Wilcoxon; p-value)	($P = 0.005$)	($P = 0.07$)	($P = 0.11$)	($P = 0.32$)		
	Keratinized mucosa width T4						
		Narrow ≤2 mm ($n = 42$)		Wide >2 mm ($n = 29$)		K-W	p-value
		Short	Long	Short	Long		
		abutment ($n = 19$)	abutment ($n = 23$)	abutment ($n = 6$)	abutment ($n = 23$)		
	T4–T0 comparison	0.21 ± 0.24 ^{B,D}	0.08 ± 0.16 ^{A}	0.18 ± 0.22	0.01 ± 0.05 ^{A}	18.91	<0.001
	(Wilcoxon; p-value)	($P = 0.001$)	($P = 0.03$)	($P = 0.07$)	($P = 0.18$)		

A, B, C, D: the superscript letters after the means indicate statistically significant comparisons after the Bonferroni post hoc tests ($p < 0.05$) with these columns. Abbreviations: SD, standard deviation. K-W, Kruskal-Wallis.

Table 7
Linear regression model of the relationship between marginal bone loss and study variables.

Dependent variables	Independent variables	β	Error	Standardized β	p-value	Lower CI 95 %	Upper CI 95 %
Bone remodelling ^{A}	**Constant**	0.60	0.09		<0.001	0.42	0.79
	Abutment height (<2 mm vs >2 mm)	−0.37	0.09	−0.43	<0.001	−0.55	−0.19
	Crestal position (Equicrestal, shallow subcrestal and deep subcrestal)	0.02	0.006	0.34	0.002	0.008	0.031
	Keratinized mucosa width (≤2 mm vs >2 mm)	−0.02	0.009	−0.22	0.04	−0.036	−0.001
Marginal bone loss ^{B}	**Constant**	0.52	0.10		<0.001	0.33	0.72
	Abutment height (<2 mm vs >2 mm)	−0.14	0.04	−0.37	0.001	−0.22	−0.06
	Age (years)	−0.005	0.002	−0.36	0.001	−0.008	−0.002

^{A}Coefficient of determination R2=0.33 $F = 10.76$; $P < 0.001$.
^{B}Coefficient of determination R2=0.30 $F = 14.46$; $P < 0.001$.

and this difference is significant for implants placed at subcrestal ≈1 mm and subcrestal ≈2 mm positions. The clinical study conducted by Vervaeke et al. already showed that biological width can be re-established more rapidly by adapting the vertical position of the implant to the soft tissue thickness available, thereby avoiding unwelcome bone loss [24]. However, Linkevicius et al. demonstrated gains in supracrestal tissue height of 1.8 mm ± 0.4 mm in implants placed in an equicrestal position combined with a coronal repositioning of the flaps around 2-mm healing abutments [18]. Similar gains have been obtained in the present study (1.60 mm ± 1.54 mm) in the group of implants placed in a SC≈2 mm position, without the delicate manipulation required by Linkevicius et al.'s technique or the MBL that occurred when this technique was applied to equicrestal implants.

Increasing scholarly attention has been paid to the impact of abutment height on peri-implant marginal bone preservation in recent years [10,22,25]. Our findings demonstrate that abutments under 2 mm in

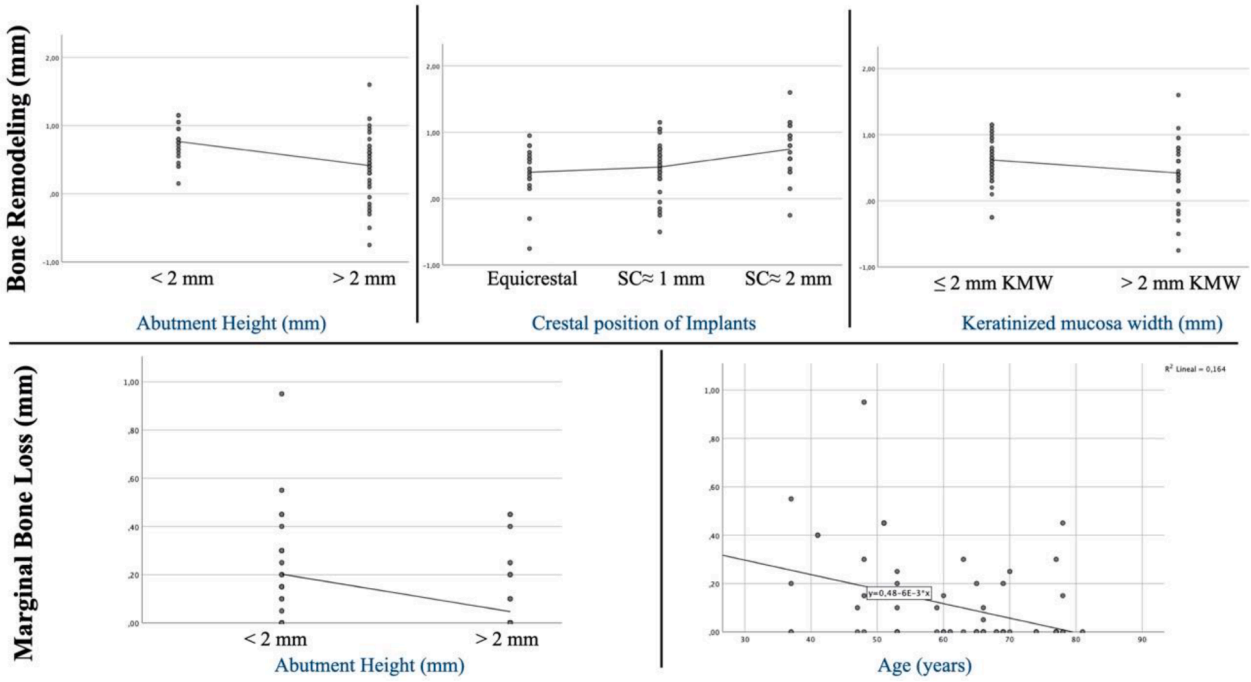

Fig. 7. Scatter plot of bone remodelling according (T0–T4) to abutment height, crestal position of implants and KMW, and of MBL (T4–T0) according to abutment height and age.

height are accompanied by greater bone remodelling and MBL, as shown in Table 4. This supports the conclusions of a recent systematic review with meta-analysis, which found that using a LA with bone-level implants is a treatment option to reduce early MBL [26]. This publication included a quantitative analysis of six studies, reporting greater levels of MBL at 1 year postsurgery in the short-abutment group than the long-abutment group (0.60 mm compared to 0.31 mm), meaning that the LA group produced 0.26 mm less MBL after 1 year. The impact of the short abutments on the MBL could be explained by two reasons. The first reason is that shorter the abutment the shorter STH and biological width would be protecting the first bone-to-implant contact [9,24]. The other reason is strictly physical, i.e. the shorter distance between the implant shoulder and the prosthetic interface, get the oral microbiota closer to the fBIC [9,27]. Another variable on which much of the literature has focused is the apico-coronal implant position with respect to the alveolar crest, as this seems to play a critical role in changes in peri-implant marginal bone levels [7]. This is what motivated the present authors to compare the bone changes (bone remodelling and MBL) that occur after implants are placed in equicrestal, SC≈1 mm, and SC≈2 mm positions, when sufficient supracrestal tissue height (≥3 mm STH) is provided for biological width to be established [9]. After 1 year of follow up, Pico et al. [2] concluded that the use of a LA (3 mm) combined with a subcrestal implant position (2 mm) in sites with thin mucosa resulted in lower MBL levels than the use of a SA (1 mm) in an equicrestal position (0.12 ± 0.33 mm compared to 0.95 ± 0.88 mm). Similar results were produced by the present study, where the use of a LA resulted in less bone loss (bone remodelling and MBL) in implants in all positions. However, at 24 months postsurgery, our results showed that equicrestal implants with a LA produce significantly less bone remodelling (0.28 ± 0.44 mm) than subcrestal implants (SC≈1 mm and SC≈ 2 mm) with a SA. The SC≈2 mm with LA group produced the lowest level of MBL at this follow-up point (0.007 ± 0.03 mm). One possible explanation for these results is that all implants were surrounded by at least 3 mm of STH at T1, on average (Table 3), providing more than the minimum level needed to maintain peri-implant health [28,29]. Furthermore, as it has explained above, the longer abutments the greater the distance of main source of bacterial contamination (prosthetic-abutment interface) of the first bone-to-impant-contact [10,11,30,31]. Additionally, in the present study, when determining the different apico-coronal implant positions (equicrestal, SC≈1, and SC≈2), the standard clinical reference point of the buccal cortical bone was used, given its more apical location than the lingual/palatal cortical bone [32,33]. Similar findings have been reported in a 1-year randomized clinical trial (RCT) [30] in which implants positioned 1.5 mm subcrestally with an abutment of 3 mm height were compared to those implants with a 1 mm abutment. The authors concluded that implants placed subcrestally with a 3 mm abutment produced significantly smaller marginal bone changes (0.40 mm versus 0.66 mm for the IS-BC distance, and 0.03 mm versus 0.19 mm for the IS-fBIC distance), and that supracrestal tissue height was not correlated with radiographic interproximal marginal bone loss. This RCT used a similar methodology to the present research, which produced very similar mean results at the 1-year follow up, both for bone remodelling (0.28 ± 0.34 mm in the SC≈1 mm with LA group compared to 0.71 ± 0.28 mm in the SC≈1 mm with SA group) and for MBL (Table 4). However, our study produced lower mean values for bone remodelling with abutments >2 mm compared to the aforementioned study. This may be explained by the fact that, unlike in Muñoz et al. (2021), all the implants placed at SC≈1 mm in the present research had an average STH of 3.67 ± 1.10 mm (Table 3), providing sufficient space for biological width to be established [9]. It should also be noted that this lower level of remodelling was obtained without following the one abutment one time protocol, although the abutment was placed in the T1 period with only a single disconnection.

The present study's findings on the peri-implant soft-tissue phenotype showed that, at the 2-year follow up (T0-T4), those implants surrounded by narrow KMW (≤2 mm) with a LA had produced significantly less bone remodelling than those rehabilitated with a SA (Table 5). The least bone remodelling was observed with implants surrounded by wide KMW (>2 mm) with use of a LA (0.36 ± 0.53 mm), as shown in Table 5. These results are in line with earlier systematic reviews that found implants placed in thick mucosa (≥2 mm) produced less MBL than those placed in thin mucosa (<2 mm) [13,34]. A consensus report concluded that a lack of keratinized mucosa or limited KMW (<2 mm) is associated with an increased prevalence of biofilm accumulation, soft-tissue

inflammation, mucosal recession, MBL, patient discomfort and peri-implant conditions such as mucositis or peri-implantitis [35]. However, in the present study, the changes in bone remodelling and MBL did not depend as much on STH or MT as on the abutment height, given that a more than adequate STH was always available (there were no groups with short STH).

The results of this clinical trial, after at least 2 years of follow up, should be interpreted with caution, taking into account the inherent limitations of the study design. Firstly, the crestal implant positions were not randomly allocated, because we aimed to ensure the provision of at least 3 mm of STH, as recommended by several authors [17,18,24]. Additionally, there are variables that were not analysed which may have a significant influence on peri-implant marginal bone changes, such as adequate oral hygiene [36], crown-to-implant ratio [37], and restorative angle of the prosthesis [38].

In this study we have focused on the outcomes when multiple restorations are screwed on either long or short implant abutments, but the prognostic value of the soft tissue phenotype has already been found among single-implant restorations in a recent study performed by the same research group [16]. Future studies should aim to include these variables and to perform a standardized digital volumetric analysis of the peri-implant soft-tissue phenotype, in order to analyze the interactions between the soft and hard tissues (peri-implant bone loss) with greater precision.

5. Conclusion

The findings described above enable us to reject the null hypothesis, and conclude that deeper implant placement leads to increased bone remodelling, which also depends on abutment height, the use of short abutments (<2 mm) resulting in more remodelling.

The combination of a deep subcrestal (≈2 mm) implant position and long abutments (>2 mm) results in the lowest level of marginal bone loss.

After 2 years, the implants placed in sites with a narrow band of keratinized mucosa (≤2 mm) produced more bone remodelling than those surrounded by a wide band (>2 mm), and this remodelling was inversely proportional to the abutment height.

CRediT authorship contribution statement

Norberto Quispe-López: Writing – review & editing, Writing – original draft, Software, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Yasmina Guadilla:** Writing – original draft, Visualization, Supervision. **Cristina Gómez-Polo:** Visualization, Software, Methodology. **Nansi López-Valverde:** Writing – review & editing, Supervision. **Javier Flores-Fraile:** Validation, Supervision, Data curation. **Javier Montero:** Writing – review & editing, Software, Methodology, Investigation, Formal analysis, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Ethical approval

This study was approved by the Bioethics Committee of the University of Salamanca (number and date of approval: 473/ June 22, 2020).

Data availability

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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