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Variance components of gingival thickness

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Objectives: Distinct periodontal phenotypes have been identified by cluster analysis, which is an explorative method with very low external validity. The aim of the present study was to investigate variance components of facial gingival thickness in young adults with mild gingivitis.

Material and methods: Thirty-three non-smoking females, 18–23 years of age, with mild or moderate plaque-induced gingivitis participated. Gingival thickness was measured at every tooth present by use of ultrasound technology to the next 0.1 mm with a lowest measurement of 0.5 mm. Periodontal probing depth and clinical attachment level were measured with a pressure-controlled probe. Gingival bleeding index was assessed after probing on a 0–2 scale, where 1 was slight, and 2 was profuse bleeding on probing. The Silness-Löe plaque index was recorded. Multilevel variance components and random intercept models were built.

Results: A 2-level (subject, tooth) variance component model of gingival thickness without any explanatory variable revealed an intercept (mean) of 0.93 ± 0.02 mm. Subject variation of gingival thickness amounted to 4.2% of the total variance. Addition of tooth- and subject-related covariates to the model revealed, after adjusting for tooth type, an association with periodontal probing depth (estimated coefficient 0.067 ± 0.025), and considerable association with average bleeding index (-0.395 ± 0.149) and plaque index (0.125 ± 0.048). Variation at the tooth level was drastically reduced; subject variation amounted to 5.2%.

Conclusion: Gingival thickness is mainly associated with tooth-related variables. Bleeding tendency is higher if gingiva is thin. Subject variability related to periodontal phenotype may add to the total variance, however, to a very low extent.

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It has been speculated for a long time that gingival dimensions may determine initiation and course of both recession and plaque-induced periodontal diseases (1). Gingival dimensions width and thickness show great intra- and interindividual variation, which is associated with tooth type and shape, and is certainly also genetically determined (2). Recently, distinct gingival phenotypes have been identified on a subject level (3), and their existence later confirmed in an independent, periodontally healthy population of young adults by using cluster analysis (4). In particular, a rather small subpopulation of about 30% was identified (4), which was characterized by narrow (about 3.5 mm) and thin (0.6–0.8 mm) facial gingiva at maxillary incisors and canines, as well as comparably slender teeth. This finding may have clinical significance, because individuals with a thin phenotype had slightly more recession than subjects with wide and thick gingival tissues. Most interestingly, masticatory mucosa was rather thin in any other region of the oral cavity, in particular that of the hard palate (4), rendering harvesting connective tissue for surgical root coverage more difficult in these individuals.

Cluster analysis is an explorative method to split a set of objects into a selected number of groups by maximizing between-cluster variation relative to within-cluster variation. External validity of cluster analysis is questionable. It is a first-stage technique that does not incorporate the machinery that allows one to evaluate the statistical significance and reliability of the patterns observed (5). Due to the typical hierarchical structure of the data

acquired in dental studies, in recent years (6) researchers have paid considerable attention to multilevel modeling (7). Some of its advantages are correct consideration of data clustering and non-independence of observations, and the possibility of detailed analysis of the covariance matrix.

The aim of the present study was to determine, in more detail, subject variation of gingival thickness in a population of young adults with healthy gingiva or plaque-induced gingivitis by using multilevel modeling. The second aim was to study the influence of clinical variables at both the tooth and the subject level.

Materials and methods

Volunteers

Thirty-three female, fifth- and sixth-year dental students participated. They were between 19 and 23 years of age (mean ± SD age 22.0 ± 0.9 years) and systemically healthy. In particular, the following exclusion criteria applied: (i) any indication for antibiotic prophylaxis; (ii) pregnancy or lactation; (iii) any medication with a possible effect on thickness of gingival tissues; (iv) any non-plaque induced-gingival disease (8); (v) destructive periodontal disease with a possible exception of localized gingival recession; (vi) extensive tooth restoration or tooth replacement; (vii) smoking. The participants had a minimum of 23 erupted teeth (mean 28.8 ± 2.7), and, with a few exceptions (extracted first molars due to caries), teeth had been extracted for orthodontic reasons. After briefing on the aim of this study as well as procedures planned, volunteers gave a written consent to participate. If large amounts of supragingival calculus were present, a prophylaxis session was provided 1 week prior to clinical examination.

Clinical examination

Clinical periodontal conditions were recorded at six sites of every tooth present. Periodontal probing depth and clinical attachment level were measured with a simple pressure-calibrated probe (ClickProbe 1395, KerrHawe, Bioggio, Switzerland) to the nearest mm. The probe tip diameter was 0.5 mm and probing force, according to the manufacturer, 0.25 N, yielding an approximate probing pressure of 1.27 MPa. After probing all facial sites of the first quadrant, gingival bleeding was assessed using a 0–2 scale bleeding index, where 1 was slight (single spot) and 2 profuse bleeding (the whole sulcus filled immediately with blood) on probing. Thereafter, facial sites of the second quadrant were measured and bleeding index recorded. Probing was continued at palatal sites and in the mandible. Gingival thickness was assessed with an ultrasonic measuring device (Krupp SDM®, Austenal Medizintechnik, Cologne, Germany) at midfacial sites of each tooth present (9). The device makes use of the pulse-echo principle. A piezo crystal is set oscillating by a pulse generator at a measurement frequency of 5×10^6 s^{-1} with an initial delay of 0.3 ± 0.2 ms. Ultrasonic pulses are transmitted through the sound-permeable mucosa at 1518 m/s and reflected in part at the surface of hard tissues alveolar bone or tooth. One-thousand signals are transmitted, received, and analyzed per second. The transducer probe has a diameter of 4 mm and is applied to the moistened measurement site with light pressure to produce acoustic coupling. By timing the received echo with respect to the transmission pulse, thickness of the soft tissue is determined within 2–3 s while transmitting an acoustic signal. The measurement is digitally displayed with a resolution of 0.1 mm and a minimum measurement of 0.5 mm. According to the manufacturer, the transducer probe of the device has to be disinfected with an aldehydic agent. Validity and reliability of the device has been tested intensively (10). Thereafter, presence or absence of calculus was recorded and plaque disclosed with a plaque revelator (D&C Red-28, Sultan Chemists Inc., Englewood, NJ, USA). The amount of plaque was estimated using criteria of the plaque index system (11).

Statistical analysis

Descriptive data are given as mean ± standard deviation of patient's averages and percentages. Multilevel modeling was applied using special software (*MLwiN* 2.0, Centre for Multilevel Modeling, Institute of Education, University of London, London, UK). Two-level (tooth, subject), variance components (without explanatory variables), and random intercept models with clinical site- and subject-level explanatory variables were built. Ordinal variables bleeding index and plaque index were encoded by design variables. Model assumptions were confirmed through analysis of residuals generated by the software. Calculus and clinical attachment loss were rarely found in this population and not considered in any model. In the final model, tooth type as encoded by design variables was also allowed to enter.

Results

An overview of clinical periodontal conditions of the study population is presented in Table 1. The volunteers had mild to moderate plaque-induced gingivitis with a few sites with increased periodontal probing depth of > 4 mm at partially erupted third molars (no loss of attachment). Any loss of clinical attachment was due to few facial areas with gingival recession. Mean bleeding index was 0.23 ± 12, and bleeding on probing was recorded at between 4 and 53% sites. Mean plaque index was 1.09 ± 0.39, and 64 ± 19% sites were covered by supragingival plaque, on average. Some traces of supragingival calculus were found at lower anterior teeth.

Mean facial gingival thickness was 0.93 ± 0.12 mm. Subjects' means ranged between 0.74 and 1.17 mm. Figure 1 shows the distribution of mean values for each tooth type. Facial gingival thickness varied within the oral cavity. Minimum values were found at first premolars (0.76–0.77 mm) and canines (0.75–0.78 mm) in the maxilla, and first premolars (0.68–0.73 mm) in the mandible. In the maxilla, facial gingiva at central incisors (0.95–1.03 mm), and second

Table 1. Overall clinical data of 33 volunteers. Subject's average values or percentages were used to calculate statistics

Variable	Mean	SD	Median	Range
N sites	172.4	16.2	174	138–192
PPD (mm)	1.81	0.27	1.78	1.30–2.37
Max_PPD (mm)	3.82	0.95	4.00	2.00–6.00
CAL (mm)	0.01	0.01	0.00	0.00–0.04
Max_CAL (mm)	0.64	1.08	0.00	0.00–3.00
BI (0–2)	0.23	0.12	0.24	0.04–0.55
BOP (%)	21.6	11.2	21.7	3.6–52.9
PI (0–3)	1.09	0.39	1.07	0.41–1.83
Plaque (%)	64.4	18.9	59.9	28.0–94.2
CLS (%)	2.8	0.3	1.9	0.0–13.2
GTH (mm)	0.93	0.12	0.93	0.74–1.17

SD, standard deviation; PPD, periodontal probing depth; Max_PPD, maximum PPD; CAL, clinical attachment level; Max_CAL, maximum CAL; BI, bleeding index; BOP, bleeding on probing; PI, plaque index; CLS, calculus; GTH, facial gingival thickness.

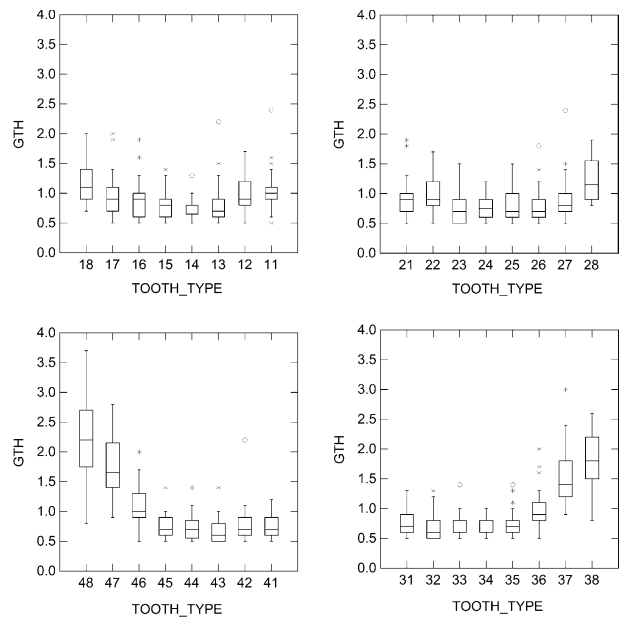

Fig. 1. Box plots illustrating measurements of gingival thickness (GTH) at different teeth in millimeters.

and third molars was rather thick (0.93–1.24 mm). In the mandible, maximum values in excess of 1.5 mm were found at second and third molars. Facial gingival thickness varied also among subjects. However, coefficients of variation were rather low at maxillary first premolars (0.24–0.26), mandibular canines (0.25–0.30), and central incisors(0.27).

Table 2 presents the results of a two-level (tooth, subject) variance components model, which indicates sufficient variation across both levels. This so-called null model revealed an intercept of 0.93 (confirming mean gingival thickness) with a standard error of 0.02 mm. Subject variation amounted to 0.008 making up 4.2% of the total variance. When tooth- and subject-related clinical covariates were allowed to enter the random intercept model (Table 3), gingival thickness was positively associated with periodontal probing depth. There was also a tendency of thinner gingiva at non-bleeding sites as compared to sites bleeding on probing, and sites with no or low amounts of supragingival plaque as compared to sites with plaque index of 2 or 3. At the subject level, in particular, average bleeding index and periodontal probing depth were negatively associated with gingival thickness. The random part of the model indicated that variation at both subject and tooth level was reduced. When tooth type was allowed to enter the model (Table 4), the influence of periodontal probing depth was drastically reduced, but remained significant. In these young individuals with shallow periodontal probing depth, thickness increased, on average, by 0.067 ± 0.025 mm per millimeter probing depth. With maxillary central incisors as reference, the model provides figures confirming tooth-related differences in gingival thickness as displayed in Fig. 1. At the subject level, average bleeding index

Table 2. Two-level variance components model (null model) of facial gingival thickness. Significant effects bold

	Estimate	95% confidence interval
Fixed effects		
Intercept	**0.931**	0.891; 0.971
Random effects (variances)		
Subject (σ^2_{u0})	**0.008**	0.001; 0.015
Tooth (σ^2_{e0})	**0.183**	0.166; 0.200

Table 3. Two-level random intercept model of facial gingival thickness. Significant effects bold

	Estimate	95% confidence interval
Fixed effects		
Intercept	**0.904**	0.618; 1.190
Tooth level		
PPD	**0.333**	0.280; 0.386
BI_0[a]	−0.070	−0.158; 0.018
PI_0[b]	**−0.109**	−0.177; 0.041
PI_1[c]	−0.101	−0.182; −0.020
Subject level		
Average PPD	−0.154	−0.312; 0.004
Average BI	**−0.343**	−0.677; −0.010
Average PI	0.057	−0.052; 0.164
Random effects (variances)		
Subject (σ^2_{u0})	**0.006**	0.001; 0.011
Tooth (σ^2_{e0})	**0.149**	0.135; 0.163

[a]Design variable encoding BI = 0, reference BI ≥ 1.
[b]Design variable encoding PI = 0, reference PI ≥ 2.
[c]Design variable encoding PI = 1, reference PI ≥ 2.
See Table 1 for further explanations.

Table 4. Two-level random intercept model of facial gingival thickness adjusted for tooth type. Significant effects bold

	Estimate	95% confidence interval
Fixed effects		
Intercept	**0.673**	0.409; 0.937
Tooth level		
PPD	**0.067**	0.018; 0.116
BI_0[a]	−0.047	−0.117; 0.023
PI_0[b]	0.010	−0.050; 0.070
PI_1[c]	0.012	−0.055; 0.079
Tooth type[d]		
ULI	−0.005	−0.108; 0.098
UC	**−0.205**	−0.309; −0.101
UP1	**−0.222**	−0.331; −0.113
UP2	**−0.178**	−0.283; −0.073
UM1	**−0.150**	−0.257; −0.043
UM2	−0.055	−0.165; 0.055
UM3	**0.158**	0.021; 0.295
LCI	**−0.259**	−0.363; −0.147
LLI	**−0.242**	−0.346; −0.138
LC	**−0.259**	−0.363; −0.154
LP1	**−0.248**	−0.393; −0.175
LP2	**−0.226**	−0.331; −0.121
LM1	0.084	−0.020; 0.188
LM2	**0.635**	0.525; 0.745
LM3	0.923	0.773; 1.073
Subject level		
Average PPD	0.120	− 0.019; 0.259
Average BI	**−0.395**	− 0.687; −0.103
Average PI	**0.125**	0.032; 0.218
Random effects (variances)		
Subject (σ^2_{u0})	**0.005**	0.001; 0.009
Tooth (σ^2_{e0})	**0.091**	0.083; 0.099

[a]Design variable encoding BI = 0, reference BI ≥ 1.
[b]Design variable encoding PI = 0, reference PI ≥ 2.
[c]Design variable encoding PI = 1, reference PI ≥ 2.
[d]Upper (U) and lower (L) incisors (I), canines (C), premolars (P), and molars (M); reference tooth: upper central incisor.
See Table 1 for further explanation.

was negatively associated with gingival thickness (−0.395 ± 0.149), whereas average plaque index was positively associated (0.125 ± 0.049). Tooth variation was largely reduced in this model, whereas subject variation amounted to 5.2% of the total variance (Table 4).

Discussion

In previous studies (3, 4), cluster analysis was employed in the analysis of data derived from two independent populations of young adults to confirm the long-claimed existence of periodontal phenotypes. Periodontal phenotypes had been described (12) as either *thick* (flat, thick and wide gingiva, interestingly associated with a square form of maxillary incisors), or *thin* (scalloped, thin and narrow gingiva, associated with slender tooth form of maxillary anterior teeth). The periodontal phenotypes affect thickness of other parts of masticatory mucosa as well (4). Clinical examples of periodontal phenotypes have been published by several authors (1, 3, 13). However, as a matter of fact they do not reflect the majority of individuals grouped together by cluster analysis but rather represent extremes in respective clusters. There is great overlap among clusters of phenotypes pointing to considerable genetic variability. Moreover, the underlying interdental bone, which can also be categorized as being either flat, scalloped or pronounced scalloped, did not correlate with tooth shapes (14). From a clinical point of view, it turned out to be difficult to assign cases to respective groups. The most probable reason for this is low external validity and more or less artificial results of cluster analysis. Since clusters must be formed, irrespective of the real structure of the data, results are prone to overinterpretation of intracluster similarities, even in a purely descriptive sense (5). In our previous studies (4), Generalized Estimating Equation models were employed to consider the correlated structure of data. When dealing with hierarchical data, these 'marginal' models start with the formulation of the covariance structure, but not necessarily based upon the multilevel structure of the data (7). They provide estimates with acceptable properties only for the fixed parameters in the model and treat the existence of any random parameters as unavoidable 'nuisance' without giving explicit estimates for them. If interest lies only in the fixed parameters, marginal models may be useful. In the present study, a major aim was getting more information on factual subject variation of gingival thickness. To avoid any possible influence of smoking, and as gingival thickness is also related to gender (9, 15), the study was conducted in a group of young adult, non-smoking females. Higher subject variation might be expected in a more heterogeneous population including both sexes and, for example, different ethnic background.

Box plots (Fig. 1) essentially confirmed previous findings (9, 15–18) of a distinct, tooth type-related pattern of facial gingival thickness as well as interindividual variation. Next, a variance components model without any further explanatory variables revealed that most of the variance of gingival thickness was due to tooth-related factors. Subject variation was significant (0.008 ± 0.003) but amounted to only 4.2% of the total variance. When tooth-related periodontal probing depth, bleeding on probing, and plaque index, as well as average periodontal probing depth, bleeding index, and plaque index were entered into the model, tooth- and subject-related variation decreased. Periodontal probing depth was associated with thicker gingiva, and lower plaque index scores with thinner gingiva. Average bleeding index was negatively associated with gingival thickness. Regarding periodontal probing depth, this is in accordance with observations made by several other authors (4, 9, 13), who reported that subjects with a *thick* periodontal phenotype had, on average, higher mean periodontal probing depth. The final two-level random intercept model (Table 4) showed that gingival thickness was in fact mainly influenced by tooth type. By adjusting for tooth type, variation at this level was largely decreased whereas subject variation only slightly changed.

Although the influence of local periodontal probing depth was greatly reduced in the final model, average bleeding index was still negatively associated with gingival thickness. For a long time, sulcular bleeding on probing has been regarded as a quite objective, valid, and reliable indicator of plaque-induced gingivitis (19). This view has been challenged over the years. In site-specific analyses, in which the correlated structure of the data in a given individual was correctly considered, it was observed that bleeding on

probing is only weakly associated with the presence of supragingival plaque (20, 21). Among other, still ill-defined, intrinsic and extrinsic factors which modify the bleeding status of gingiva, smoking (22, 23), intake of non-steroidal anti-inflammatory drugs (24), or genetic polymorphisms (25, 26) have called considerable attention in recent years. Bleeding after probing depends, to a large extent, on probing pressure (27). Therefore, standardization of probing force in assessing the inflammatory status of the gingiva is mandatory. In the present study, a simple, pressure-controlled probe was employed. Interestingly, a higher full-mouth bleeding score was associated with thinner gingiva. This relationship was demonstrated by considering variables operating at both the subject and tooth level simultaneously. Thus, thin and vulnerable gingiva tended to bleed more frequently than thicker periodontal tissues. In contrast, local bleeding on probing was not significantly affected by facial gingival thickness, which is in accordance with observations made in a previous study (28). The most probable reason is quite low bleeding frequency at this location in either study. The present findings might be in accordance with observations made in a recent gingivitis experiment (29). Although thickness of gingiva was not measured in that study, higher bleeding scores were observed in young adults with 'long-narrow' as compared to 'short-wide' shape of maxillary incisors. Incisor tooth shape has been regarded as an essential component of periodontal phenotypes by numerous authors; for review see (1).

Average plaque index was positively associated with gingival thickness. A higher mean plaque score certainly leads to generalized gingival inflammation, and swelling of marginal tissues might be measurable with the ultrasonic device. Although gingival edema had been introduced in a widely used index system for scoring the degree of gingivitis (30), quantitative data of gingival swelling have only recently been presented using 3-D laser scanning methodology (31). Although this method revealed promising results, measurements are very time-consuming. Over the years, ultrasonic diagnosis of periodontal tissues has attracted considerable attention. Very recently, a novel, highly accurate device for dermal ultrasonography was introduced, which can even produce images of some of the periodontal structures, such as gingiva and alveolar bone (32). Whether ultrasonic devices can ultimately differentiate non-inflamed from inflamed gingiva must be investigated in future studies.

Within its limitations, the present study provides new insights into the role of subject-related factors when considering thickness of gingival tissues. In general, most of the variation of gingival thickness seems to be due to tooth type. Subject variability related to periodontal phenotype may add to the total variance, however, to a very low extent. In fact, the clinical relevance of proposed periodontal phenotypes has to be questioned in view of the present findings. Instead, thickness of keratinized tissues should be considered locally if, for example, risk of recession development is assessed or flaps designed. Of great interest might be the observation that thin gingiva was associated with higher bleeding scores after standardized sulcus probing. In particular, the latter finding has considerable impact on the correct interpretation of a presumed cardinal symptom of gingival inflammation, namely bleeding on probing.

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