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# Lip piercing: prevalence of associated gingival recession and contributing factors. A cross-sectional study

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*Background and Objective:* Body piercings have increased tremendously in popularity in recent years. For oral piercing, late complications include gingival trauma and localized periodontitis. The purpose of this cross-sectional study was to investigate the prevalence and contributing factors of gingival recession associated with labial piercing.

*Material and Methods:* The test group included 50 subjects with lower-lip studs. Nonpierced controls were matched according to gender, age and smoking status. Clinical examination included plaque and bleeding indices, probing depth, recession, clinical attachment level, width of keratinized gingiva, periodontal biotype, insertion of frenula, evaluation of hard tissues, trauma of occlusion, characteristics of the stud, radiographs and photographs.

*Results:* Gingival recessions were noted on teeth opposite the stud in 68% of the test group compared with 4% of the controls. Localized periodontitis was recorded in 4% of test subjects. Time since piercing and the position of the stud in relation to the cemento–enamel junction were significantly associated with the prevalence of buccal recessions. There were no significant associations between piercing and abnormal tooth wear.

*Conclusion:* The prevalence of gingival recessions is associated with labial piercing. The position of the intra-oral disc and time since piercing are associated with a greater prevalence of gingival recession. A narrow width of keratinized gingiva is associated with increased buccal recessions.

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Body piercing and other body modifications have increased tremendously in popularity in recent years and have started to be practised across many social and age groups (1). In a 2001 survey of 481 United States college students, the prevalence of body piercing was 51% (2). The number of individuals with body piercing in western societies is still rising and has not yet reached its peak (1). Oral piercing mostly involves the lips, tongue and cheeks. For oral piercing, a variety of complications have been reported (3). These complications can be categorized as acute (or early) and late (or chronic) (4). Early complications include pain, swelling, difficulties in mastication, swallowing and speech, bacterial infection and prolonged bleeding (4). Late complications include chipped and fractured teeth, gingival trauma, localized periodontitis, persistent difficulties in oral

functions and swallowing of the device (4). The increasing popularity of labial piercing has prompted several case reports and studies documenting associated periodontal complications (5–13). However, studies with representative numbers of cases, and studies assessing potentially significant risk factors, are limited.

The present cross-sectional study aimed to assess the prevalence and severity of periodontal and dental complications, in association with lower-lip studs, in a population obtained from a nondental setting. In addition, possible cofactors for the development of recessions were evaluated.

## Material and methods

### Study subjects

One-hundred subjects were actively recruited on the Campus of the University of Vienna and through an Austrian student website (http://www.unijobs.at). The inclusion criterion was peri-oral piercing of the lower lip in the labio-mental groove below the vermilion border. The following exclusion criteria were applied: need for antibiotic prophylaxis; pregnancy and lactation; medication with an effect on gingival tissues; nonplaque-induced gingival disease; prior diagnosis of periodontitis; and missing teeth in the lower front. Test and control subjects were matched according to gender, age ($\pm$ 1 years) and smoking status. Smoking status, defined as the total number of packs of cigarettes consumed in a lifetime, was calculated as the number of cigarettes consumed on average per day, multiplied by number of days of habit and divided by 20 (one pack) (14). Patients were divided into three groups according to their smoking habit: nonsmokers (i.e. never smoked); light smokers (1–912 packs); and moderate/heavy smokers ($>$ 912 packs) (14).

### Questionnaire

Participants were asked to complete a questionnaire to determine demographic data, smoking habit, characteristics of the labret worn (time since piercing, material – plastic or metal) and postpiercing complications. Also included were questions about previous orthodontic treatment, frequency of tooth brushing, medication and systemic disease. If tooth chipping was found during clinical examination, subjects were asked to provide information on the circumstances under which the chipping occurred.

### Clinical examination

Clinical periodontal conditions were recorded at six sites around the lower front teeth. Probing depth was measured with a pressure-calibrated probe (ClickProbe 1395; KerrHawe, Bioggio, Switzerland) to the nearest mm. The probe tip diameter was 0.5 mm, and the probing force, according to the manufacturer, was 0.25 N. After probing all facial sites, bleeding on probing (15) was recorded dichotomously. Thereafter, probing was continued at lingual sites. Full-mouth periodontal probing was carried out if the probing depth was greater than 3 mm. The presence or absence of plaque was measured using the plaque control record (16). The amount of recession in the occluso-apical direction was measured from the cemento–enamel junction to the free gingival margin at six sites per tooth. The amount of recession in the mesio-distal direction was measured horizontally at the level of the cemento–enamel junction, between the mesial and distal aspect of the gingival margins of the tooth. Clinical attachment level was calculated by adding the amount of recession in the occluso-apical direction and the probing depth. Periodontitis was defined clinically as clinical attachment level $\geq$6 mm and one or more sites with probing depth $\geq$5 mm (17). Clinical attachment level was verified radiologically as reduced interproximal bone level (distance from the cemento–enamel junction to the alveolar crest $>$ 2 mm) (18). The amount of keratinized tissue was measured at six sites per tooth, from the mucogingival junction to the free gingival margin; the width of attached gingiva was calculated by subtracting the probing depth measurement from the amount of keratinized gingiva. The periodontal biotype (thin, scalloped periodontium/thick, flat periodontium) was determined according to Olsson & Lindhe (19). The absence or presence of a frenulum attachment at the gingival margin was recorded. Subgingival margin discrepancies of restorations (20), and violation of the biologic width (21), were documented as traumatic restorations. Trauma from occlusion has often been discussed as an etiological factor of gingival recessions (22,23). One common clinical sign of occlusal trauma is fremitus (24). Therefore, fremitus was evaluated by palpating the buccal aspects of the mandibular front teeth as the patient went into central occlusion (24). The extent of any abnormal tooth wear was determined according to Imfeld's definition of abrasion (25), and was recorded for lesions deeper than 1 mm. The following characteristics of the piercing were evaluated in a resting lower lip position: teeth in contact with the retainer; and height of the stud at the opposing tooth in relation to the cemento–enamel junction (coronal to the cemento–enamel junction, on the cemento–enamel junction, apical to the cemento–enamel junction). In addition, radiographs and photographs of the lower front teeth were taken. One examiner performed all clinical examinations.

### Ethical considerations

The study protocol was reviewed and approved by the Research Ethical Committee, Medical University of Vienna, Austria. All participating subjects signed an informed consent form. At the conclusion of the clinical examination, participants obtained appropriate compensation and were informed about their oral status and any diagnosed mucogingival lesions. Patients with diagnosed pathological conditions were offered appropriate treatment.

### Data management and statistical analysis

The primary outcome measure was prevalence of buccal recession. Secon-

dary outcome measures were: amount of buccal recession in occluso-apical and mesio-distal directions; prevalence of abnormal toothwear; contributing factors; and additional periodontal measurements.

Statistical calculation was carried out using SAS version 8 (SAS Institute Inc., Cary, NC, USA) and SPSS version 13 (SPSS Inc., Chicago, IL, USA). Unless stated otherwise, results are expressed as means ± standard deviation. Standard descriptive statistics were used to summarize the variables studied. Variations in plaque control record, bleeding on probing and keratinized gingiva between test and control groups were assessed by the unpaired *t*-test. For analysis of the primary outcome measure, prevalence of gingival recession and gingival recession on the labial aspect of mandibular incisors served as the binary dependent variable, coded as 1 if present, or 0 if absent. Differences in the prevalence of buccal recessions between test and control groups were assessed by the McNemar test. Correlation analysis of buccal recessions and different parameters was evaluated using the appropriate statistical tests. Differences in mean scores of the amount of buccal recession in occluso-apical and mesio-distal directions between test and control groups were assessed by the Student's *t*-test. For correlation analysis of the amount of buccal recession in the occluso-apical direction, a continuous measure of recession was constructed. If more than one tooth was affected, the greatest amount of recession in the occluso-apical direction per patient was used. Correlation analysis of severity of buccal recessions and different parameters was evaluated using the appropriate statistical tests.

## Results

### Demographic data

One-hundred subjects (88 female, 12 male) were enrolled and completed the study. All participants were Caucasians, 14–28 years of age (mean age ± standard deviation = 21.76 ± 2.66); 18% had never smoked, 28% were light smokers (1–912 packs lifetime exposure) and 54% were moderate/heavy smokers (> 912 packs lifetime exposure). Test and control subjects were matched according to gender, age (± 1) and smoking status. Demographic data and smoking status are shown in Table 1. The average time since piercing on the examination day was 39.4 ± 3.5 mo (range 3 mo to 9 years, median 36 mo). One participant reported that his piercing had been carried out by a doctor, two had performed the lip piercing themselves and 47 had received their piercing at piercing studios.

*Table 1.* Subjects' demographic background and smoking habit

| | Control group ($n = 50$) | Test group ($n = 50$) |
|---|---|---|
| Mean age ± SD (years) | 21.74 ± 2.671 | 21.76 ± 2.669 |
| Gender | | |
| Male, *n* (%) | 6 (12%) | 6 (12%) |
| Female, *n* (%) | 44 (88%) | 44 (88%) |
| Smokers (lifetime exposure) | | |
| Nonsmokers, *n* (%) | 9 (18%) | 9 (18%) |
| Light smokers, *n* (%) | 14 (28%) | 14 (28%) |
| Moderate/heavy smokers, *n* (%) | 27 (54%) | 27 (54%) |

### Questionnaire

All subjects reported wearing metal closures for 2–6 wk immediately post piercing. Thirteen patients reported wearing plastic closures following the healing period, whereas 37 patients reported wearing a metal closure. Early postpiercing complications were reported by 86% ($n = 43$) of the subjects (data not shown). Twenty-four (48%) test subjects reported recession of gingiva as a late complication. Previous orthodontic treatment was reported by 50% ($n = 25$) of the test group and 58% ($n = 29$) of the control group ($p = 0.232$). Average tooth brushing time per day (tooth brushing frequency × average time of brushing) was 8.04 ± 3.43 min/d for controls and 6.38 ± 3.13 min/d for test subjects ($p = 0.894$).

### Periodontal conditions

Periodontal conditions of the lower front teeth are shown in Table 2. The mean probing depth of the test teeth (range 1–5 mm) was not significantly different from that of the control teeth (range 1–3 mm) ($p = 0.963$). The average plaque control record and average bleeding on probing did not differ significantly between the two groups (plaque control record $p = 0.703$; bleeding on probing $p = 0.234$). Localized periodontitis limited to the teeth directly opposite the labret was noted clinically and radiographically in 4% of the test subjects ($n = 2$). Full-mouth periodontal probing did not reveal further clinical attachment loss. Buccal recession in one or more sites in the area directly opposite the labret was noted in 68% of test subjects as compared with 4% in the same gingival area of controls ($p < 0.0001$). Average values (teeth 33–43) of the amount of buccal recessions in the occluso-apical direction were significantly higher in the test group (0.53 ± 0.60 mm; range 0–5 mm) than in controls (0.03 ± 0.13 mm; range 0–2 mm) ($p < 0.0001$). Also, average values (teeth 33–43) of the amount of buccal recessions in the mesio-distal direction were significantly higher in the test group (0.53 ± 0.60 mm; range 0–5 mm) than in the controls (0.03 ± 0.13 mm; range 0–2 mm) ($p < 0.0001$). The average width of keratinized gingiva was significantly higher in the test group than in the controls ($p < 0.0001$). The distribution of periodontal biotype was not statistically different between the two groups ($p = 0.119$).

### Abnormal tooth wear

Among 100 subjects examined, none showed abnormal tooth wear, according to Imfeld's definition of abrasion (18). Eight per cent of the test group had tooth chipping on one tooth and/

*Table 2.* Periodontal conditions and abnormal tooth wear of the mandibular anterior dentition (teeth 33–43)

| Variable | Test group | Control group | *p*-value | Statistical test |
|---|---|---|---|---|
| PCR (%), mean ± SD | 70.6 ± 3.3 | 68.5 ± 4.5 | 0.703 | Unpaired *t*-test |
| BOP (%), mean ± SD | 11.3 ± 2.5 | 7.3 ± 2.2 | 0.234 | Unpaired *t*-test |
| Prevalence of recession, *n* (%) | 34 (68) | 2 (4) | < 0.0001 | McNemar test |
| Prevalence of LP, *n* (%) | 2 (4) | 0 (0) | [a] | |
| PD (mm), mean ± SD | 1.59 ± 0.37 | 1.57 ± 0.38 | 0.963 | Unpaired *t*-test |
| CAL (mm), mean ± SD | 1.70 ± 0.73 | 1.98 ± 0.86 | 0.751 | Unpaired *t*-test |
| Amount of recession in the occluso-apical direction (mm), mean ± SD | 0.53 ± 0.60 | 0.03 ± 0.13 | < 0.0001 | Paired *t*-test |
| Amount of recession in the mesio-distal direction (mm), mean ± SD | 0.46 ± 0.46 | 0.06 ± 0.03 | < 0.0001 | Paired *t*-test |
| Width of keratinized gingiva (mm), mean ± SD | 3.76 ± 0.76 | 3.18 ± 0.85 | < 0.001 | Unpaired *t*-test |
| Thin, scalloped periodontal biotype, mean ± SD | 32 (64) | 25 (50) | 0.119 | Fisher's exact test |
| Tooth chipping, *n* (%) | 4 (8) | 3 (6) | 0.576 | Fisher's exact test |
| Cracks, *n* (%) | 14 (28) | 12 (24) | 0.325 | Fisher's exact test |

BOP, bleeding on probing; CAL, clinical attachment level; LP, localized periodontitis; PCR, plaque control record; PD, probing depth; SD, standard deviation.
[a]Not determined.

or 28% had cracks on one or more mandibular front teeth, which was not statistically different from the control group (24% cracks, 6% tooth chipping). Incidence of tooth chipping and cracks did not correlate with the position of the intra-oral stud retainer.

### Contributing factors

Correlation analyses revealed the significant relationships shown in Table 3. The longer the time since piercing, the higher the prevalence of buccal recessions. The mean time since piercing was 39.4 ± 3.5 mo (range: 3 mo to 9 years; median 36 mo). Patients with their stud placed at the cemento–enamel junction had a significantly higher prevalence of buccal recessions on teeth directly opposite the labret. Nine stud retainers (18%) were positioned apical to the cemento–enamel junction, 30 (60%) were positioned on the cemento–enamel junction and 11 (22%) were positioned coronal to the cemento–enamel junction of opposing teeth. The material of the stud retainer showed no statistical correlation with the prevalence of buccal recessions. The amount of buccal recession in the occluso-apical direction was not correlated with time since piercing, position or material of the stud (data not shown). The distribution of buccal recessions occluso-apically, with respect to time since piercing, is shown in Fig. 1.

*Table 3.* Correlation analyses of prevalence of buccal recessions and associated factors

| Variable | *p*-value | Statistical test |
|---|---|---|
| Biologic parameters | | |
| Age | 0.272 | Unpaired *t*-test |
| Gender | 0.699 | Fisher's exact test |
| Smoking status | 0.353 | Spearman correlation coefficient |
| Periodontal biotype | 1.0 | Fisher's exact test |
| Width of KG | 0.093 | Double analysis of variation |
| Frenula attachment | [a] | |
| Occlusal trauma | [a] | |
| Other parameters | | |
| Orthodontic treatment | 0.639 | Unpaired *t*-test |
| Time of tooth brushing/day | 0.551 | Unpaired *t*-test |
| Characteristics of the study | | |
| Time since piercing | 0.013[b] | Spearman correlation coefficient |
| Material of the closure | 0.774 | Mann–Whitney *U*-test |
| Position in relation to CEJ (95% CI) | | Pearson-Clopper-Confidence interval |
| • stud coronal to CEJ | 0013 (0.165) | |
| • stud on CEJ | 0355 (0.645)[b] | |
| • stud apical to CEJ | 0033 (0.218) | |

CEJ, cemento–enamel junction; CI, confidence interval; KG, keratinized gingiva.
[a]Not determined, number too small.
[b]Statistically significant correlation with the prevalence of buccal recessions.

Comparing both groups, no significant correlations were found between prevalence of recession and the width of keratinized gingiva (mean scores), periodontal biotype, smoking status, gender, age, previous orthodontic treatment, or average tooth brushing time per day. Similarly, no significant correlations were found between the amount of buccal recession in the occluso-apical direction and periodontal biotype, smoking status, gender, age, previous orthodontic treatment, or average tooth brushing time per day (data not shown). However, a narrow width of keratinized gingiva (mean scores) was significantly associated with greater amounts of buccal recession in the occluso-apical dimension ($p = 0.01$; Pearson correlation coefficient). Twenty-three of a total of 200 mandibular incisors (11.5%) were affected by fremitus in the test group vs. three (6%) mandibular incisors in

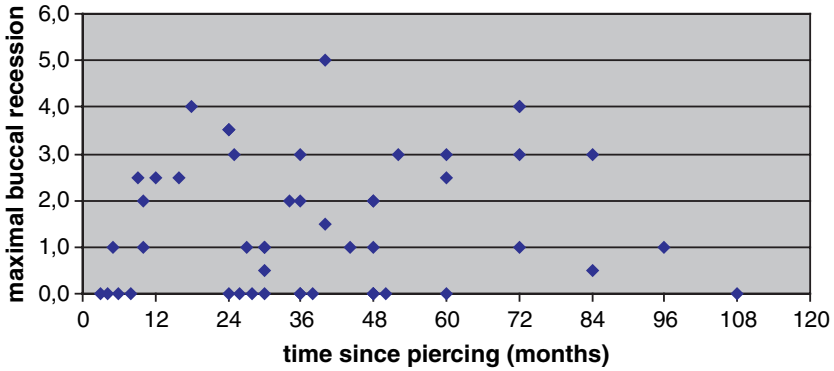


*Fig. 1.* Maximal buccal recession in the occluso-apical dimension vs. time since piercing.

the control group ($p = 0.325$; Fisher's exact test). On the contrary, test and control significantly differed with respect to lip–frenulum insertion. A lip–frenulum attachment was apparent at 13 of 100 (13%) first mandibular incisors in the test group as opposed to one (1%) first mandibular incisor in the control group ($p < 0.01$; chi-square test). None of the mandibular anterior teeth in the test or control group had any dental restorations.

## Discussion

Since 1997, several case reports and series (5–8,13) have reported on the occurrence of gingival recessions associated with labial piercing. All these piercings were similar: the lip studs were positioned in the labio-mental groove below the vermilion border, with an intra-oral metal disk adjacent to the mandibular incisors. In a self-assessment questionnaire given to a cohort with oral piercing, 12.5% admitted to gingival injuries inflicted by lip piercing ($n = 24$) (26). However, self-assessment questionnaires dealing with nonpainful gingival or dental trauma are of limited value as such damage often goes unnoticed by patients because of lack of dysfunction. Previously published clinical studies have shown that gingival recession was recorded in up to 80% of pierced subjects (9,10,12). As most of these studies included subjects with different kinds of intra-oral piercing, the number of subjects with labial piercing in these studies were very low. Therefore, the present cross-sectional study investigated the effect of lower lip studs on adjacent soft and hard oral tissues, clinically and radiographically, in a population obtained from a nondental setting. In addition, possible cofactors for recession development, and severity and special characteristics of the studs, were evaluated and related to clinical findings.

Most participants in our study were students who were recruited through a students' employment website and were therefore mainly interested in financial compensation. Only a few were interested in the medical information provided after the examination, and none was interested in treatment for buccal recessions or periodontitis. Consequently, we concluded that a possible selection bias for individuals suspecting damage to their teeth was low.

A recent epidemiologic study examining risk indicators for gingival recessions ($n = 1460$) showed that prevalence, extent and severity correlated with age (14). Individuals who were 25–50 years of age showed the highest level of recession (14). In addition, men aged ≥ 30 years showed significantly higher prevalence and extent of gingival recession than women (14), and, in a multivariable model, cigarette smoking (total number of packs of cigarettes consumed in a lifetime) was significantly associated with localized and generalized recessions (14). To minimize differences between test and control groups in these parameters in the present study, subjects were matched according to age (± 1 years), gender and smoking status. Eighty-eight per cent of the study population was female. This might be a result of the fact that the prevalence of body piercing is higher in women than in men. In a study examining the prevalence of body piercing in undergraduates in the USA, body piercing was present in 42% of men and 60% of women (2).

The results of our study suggest that subjects with labrets in the labio-mental groove have a significantly higher prevalence of buccal recessions in the mandibular anterior dentition (teeth 33–43) than unpierced subjects. Buccal recessions in one or more sites in the area directly opposite the labret was noted in 68% of test subjects compared with 4% in the same gingival area of controls ($p < 0.0001$). Additionally, average values for the amount of buccal recession in occluso-apical and mesio-distal directions were significantly higher in pierced individuals than in unpierced individuals ($p < 0.0001$). These data support the findings of a previous study, in which 68.13% of subjects with lip piercing showed recessions at teeth opposite the labret (11). In this particular study, only Miller's class 1 recession defects were detected in the nonpierced group, while 18.7% displayed Miller's class 2 and 3 in the pierced group (11).

In the present study, two patients showed localized periodontitis, limited to the area directly opposite the labret. Full-mouth periodontal probing did not reveal any further clinical attachment loss. To date, only a limited number of case reports have been published on the association of intra-oral piercing with localized periodontitis (27–29).

Test and control groups differed significantly with respect to the presence of a frenula insertion. Frenula insertion was apparent at 13% of first mandibular incisors in the test group as opposed to 1% of first mandibular incisors in the control group ($p < 0.01$). The vestibular frenulum has been considered as a possible primary cause for gingival recession (30). However, it has also been proposed that the frenulum acts only as a secondary cause in the development of recessions (31). Therefore, further longitudinal studies are needed to clarify

the role of frenula insertions during the development of buccal recessions with labial piercing.

In general, the development of gingival recessions is related to multiple etiologic factors (22,23,30–39). With lip studs, gingival recession might be related to the mechanical trauma of the intra-oral retainer of the stud. The extent of mechanical trauma might be modified by the material of the intra-oral retainer. In our study, the prevalence of buccal recession was not associated with the material of the intra-oral retainer (13 were plastic and 37 were metal). This might be a result of the fact that all subjects wore a metal retainer for at least 2–6 wk immediately following the insertion. Clinical effects of the mechanical trauma might also depend on the intra-oral position of the retainer: coronal to the cemento–enamel junction, at the cemento–enamel junction (at the gingival margin), or apical to the cemento–enamel junction (coronal or apical to the mucogingival junction). Our results indicate that the position of the stud at the cemento–enamel junction is significantly associated with a higher prevalence of buccal recessions (Table 3). In contrast, a recent publication by Leichter & Monteith (11), using similar criteria as in our study, the position of the lip-stud, in relation to the cemento–enamel junction, was not correlated with buccal recessions. Our results also indicate that with an increase of time since piercing, the prevalence of buccal recessions will rise (Table 3). No correlation was found between the amount of recession in the occluso-apical direction and with time since piercing, position, as well as material of the stud. It might be possible that the recession does not change in the occluso-apical dimension because the retainer is stable in its position.

Other possible contributing factors for the development of recessions, such as gender, age, smoking status and previous orthodontic treatment, were also evaluated in our study, but no association with prevalence or severity of buccal recessions were found. Similarly, Leichter & Monteith (11) noted no correlation between these factors and buccal recessions. In addition, our study looked at the width of keratinized gingiva, periodontal biotype, frenulum attachment, occlusal trauma (fremitus) and average time of tooth brushing per day. These parameters have been described in the literature as possible etiologic factors of gingival recession (22,23,33–39). In the present study, no significant correlations with prevalence of buccal recessions were found, either in the test group or in the control group. However, the amount of buccal recession in the occluso-apical direction correlated with the width of keratinized gingiva, in test and control groups. Reduced width of keratinized gingiva has been discussed as a possible etiologic factor for recession development (38), and it has been suggested that sites with a narrow zone or a lack of keratinized gingiva may, in the presence of subgingival plaque, favour the apical displacement of the soft tissue margin (39). Interestingly, the mean scores (teeth 33–43) for width of keratinized tissue differed between the control and test groups. The mean scores were derived from buccal and lingual measurements, and therefore it can be concluded that independently from the present recession, test subjects had significantly more keratinized tissue than controls. However, it is not clear whether this is a reaction to the labret.

To date, medical complication rates following oral labial piercing are not available. Interestingly, in our study, 86% of pierced subjects reported early complications immediately after piercing. These involved mild pain, swelling, mild bleeding, mild infection, severe infection, loss of sensitivity, and problems with speaking or eating. In comparison to other studies evaluating postpiercing complications of several other body regions, this complication rate seems high. Mayers *et al.* (2), for example, reported the incidence of medical complications after body piercing to be 17%. These included bleeding, tissue trauma and bacterial infections. The rate of acute complications resulting from body piercing is determined by piercing site, material, practitioner experience, hygiene and follow-up (1). As most of our test subjects had their piercing carried out at piercing studios, insufficient hygiene and lack of follow-up might be responsible for the high early complication rate. Because of the cross-sectional design of the present study, early postpiercing complications could only be determined retrospectively using a self-assessment questionnaire and are therefore of limited value. Similarly, no statement can be made on when the buccal recession started to develop. Further longitudinal studies are needed to clarify these issues.

In conclusion, within the limitations of the current study, labial piercing was found to be a significant factor for the development of buccal recession in mandibular anterior teeth. Time since piercing and the position of the intra-oral disc at the cemento–enamel junction are associated with a greater prevalence of gingival recession. In addition, a narrow width of keratinized gingiva is associated with higher amounts of buccal recession in the occluso-apical and mesio-distal directions.

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