

Analysis of crown widths in subjects with congenitally missing maxillary lateral incisors

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SUMMARY The aim of the study was to test the hypothesis that the gene defect causing congenital absence of maxillary lateral incisors also causes narrowing of the dentition. A total of 81 patients with one or two congenitally missing lateral incisors were retrieved; 52 (64.2 per cent) patients presented bilateral agenesis, whereas 29 (35.8 per cent) had unilateral agenesis. The control group consisted of 90 consecutively treated patients. The largest mesiodistal crown dimension for all teeth, except for the maxillary second and third molars, was measured on plaster casts using a digital caliper to the nearest 10th of a millimetre. Statistical testing was performed using the analysis of variance model ($P < 0.05$) to test for differences in the mesiodistal dimension between the sample and the control group. Significance has been assessed using a P -value threshold level of 5 per cent. Agenesis of maxillary lateral incisors was found to be a significant predictor of tooth size. Patients who were missing maxillary lateral incisors had smaller teeth compared to control subjects, except for the maxillary right and left first molars. This finding was true for both unilateral and bilateral lateral incisor agenesis. Interaction between maxillary lateral incisor agenesis and gender was not significant. Patients with congenitally missing lateral incisors have narrower teeth than patients without any dental anomalies, except for maxillary first molars. A higher prevalence of microdontic contralateral incisors was found in patients with unilateral agenesis with respect to the control group.

Introduction

Tooth agenesis is one of the most common congenital anomalies occurring in the permanent dentition. Genetic and environmental factors have been implicated with congenital absence of teeth, and recent studies have demonstrated the predominant role of genetics in the aetiology of this condition (Arte, 2001; Nieminen, 2001). The phenotypic expression of this genetic defect, in which six or less teeth are congenitally missing, is termed hypodontia, whereas oligodontia applies to more severe conditions (more than six missing teeth; Arte, 2001; Schalk-Van der Weide and Bosman, 1996).

The prevalence of hypodontia has been investigated thoroughly, and it ranges from 2.2 to 10.1 per cent (excluding third molars) and narrows to an interval of 5–8 per cent in Caucasians (Arte, 2001; Polder *et al.*, 2004). Among subjects affected by hypodontia, agenesis of the mandibular second premolars is the most frequent followed by the maxillary second premolars and lateral incisors; however, differences in the incidence of agenesis have been reported (Muller *et al.*, 1970; Arte, 2001; Polder *et al.*, 2004).

Furthermore, non-syndromic hypodontia has been associated with the presence of other dental anomalies, calling for a common genetic aetiology. Many researchers have reported that tooth agenesis is associated with palatally displaced maxillary canine, infraocclusion of primary molars,

ectopic eruption of molars, delay of tooth development, enamel hypoplasia, and abnormal morphology (peg-shaped lateral incisors and reduced dimensions; Lai and Seow, 1989; Garib *et al.*, 2010; Baccetti, 1998; Bjerklin *et al.*, 1992).

When a reduction in tooth size, also termed microdontia, has been associated with tooth agenesis, a direct proportion between the severity of the mesiodistal dimension decrease and the number of missing teeth has been demonstrated (Ooshima *et al.*, 1996; Schalk-Van der Weide and Bosman, 1996; Brook *et al.*, 2002; Altug-Atac and Erdem, 2007).

However, few studies have attempted to investigate the correlation between reduction in tooth size and the specific type of agenesis (Garib *et al.*, 2009), and little information is available on the association between agenesis of maxillary lateral incisors and mesiodistal dimensional decrease of the dentition (Garib *et al.*, 2010).

Clinical experience suggests that in patients with one or two congenitally missing lateral incisors, insufficient space for implant replacement may exist after orthodontic treatment even in the presence of good occlusion (Kokich, 2009). One possible explanation for this occurrence relates to the reduced tooth size in patients affected by the gene defects that cause congenitally missing lateral incisors. If the teeth were narrower than in the normal population, in presence of good posterior occlusion and an ideal incisal relationship, the space available for implant placement

and/or the prosthetic restoration would be small (Kokich, 2009). In addition, clinical inferences supporting this clinical observation have been noted by Rosa and Zachrisson, when canine substitution is used to manage patients with congenitally missing maxillary lateral incisors (Rosa and Zachrisson, 2007; Rosa and Zachrisson, 2010). In fact, in their conclusions, they stated that widening the maxillary central incisors with composite restorations could be necessary to improve the patient's incisor display.

The purpose of this study is to test the hypothesis that the gene defect causing congenital absence of maxillary lateral incisors also causes narrowing of the dentition. Therefore, the null hypothesis is that patients with one or two congenitally missing maxillary lateral incisors and patients without any dental anomalies have no differences in tooth widths.

Materials and methods

Clinical files of consecutively treated patients from three orthodontic practices were reviewed (A. Davide Mirabella, Marco Rosa, Renato Cocconi), and patients with one or two congenitally missing lateral incisors (CMLI) were selected for the study. A total of 81 cases for group CMLI were retrieved; 52 (64.2 per cent) patients presented bilateral agenesis, whereas 29 (35.8 per cent) had unilateral agenesis. The control group consisted of 90 patients presented for orthodontic consultation in one orthodontic practice (ADM). The subjects ranged in age from 14 to 21 years, with a female/male ratio of 2.5:1.

The inclusion criteria for the sample were complete eruption of the teeth to be measured, availability of good quality dental casts and pre-treatment panoramic radiographs, absence of any syndromic conditions, and absence of any other dental agenesis. Presence of caries, fractures, or interproximal restorations; history of previous orthodontic treatment; or ectopic eruption of canines were regarded as exclusion criteria. The age of 14 years was considered as the threshold to diagnose agenesis of third molars (Garn and Lewis, 1962).

The control group was selected as follows: all patients that would fulfil the inclusion criteria were included in the sample. With respect to sample size adequacy, assuming standard one-way analysis of variance (ANOVA) assumption valid, using a reference power level of $\beta = 90$ per cent and $\alpha = 5$ per cent, three groups and $n = 171$ patients (57 per group on average), it can be stated that our sample size is able to detect an effect size delta of at least 0.274 (where $\text{delta} = \text{largest mean} - \text{smallest mean}/\text{withing groups standard deviation}$). Gender representation was assured by including the same number of consecutive male or female control patients with respect to the gender distribution in the sample group. Then, tooth widths were measured, and 18 patients with a Bolton Index (Bolton, 1958) beyond 1 SD were disregarded.

The descriptive statistics of the sample and distribution of anomalies are described in Table 1. The largest mesiodistal

Table 1 Description of the groups, gender distribution, and anomalies distribution.

Gender	Controls	CMLI (12–22)	CMLI (12)	CMLI (22)
Female	64	39	16	5
Male	26	13	5	3
Total (%)	90	52 (64.2)	21 (25.9)	8 (9.9)

crown dimension for all teeth, except for the maxillary second and third molars, was measured on plaster casts using a digital caliper (Ortho-Pli, Philadelphia, PA, USA). One person made all measurements to the nearest 10th of a millimetre. Each tooth was measured twice, and the measurement was recorded on a spreadsheet (Excel, Microsoft, Redmond, WA, USA) as the tooth dimension.

Gender and group (no agenesis, left agenesis, right agenesis, and bilateral agenesis) were recorded for each patient. Presence of microdontic maxillary contralateral incisors among the unilateral congenitally missing maxillary incisor patients was recorded. A lateral incisor was defined as microdontic, when its mesiodistal measurement was at least 3.5 SD smaller than the mean width of the lateral incisor in the control group (Ooshima *et al.*, 1996).

Analysis of data

Descriptive statistics were performed on the sample and control groups. An ANOVA analysis was performed on each tooth (excepting teeth 12 and 22), using the group as the independent factor to test for differences in the mesiodistal dimension. Gender was also considered in the ANOVA model in order to control for effect, and the presence of interaction between group and gender was verified. The replicate measure design of the experimental structure was considered in the ANOVA model as a blocking variable. The software used for statistical calculation was R 2.12 x64.

Statistical significance was assessed using a *P*-value threshold of 0.05. The reliability of the study has been assessed by means of a paired sample *t*-test and the Dahlberg coefficient calculation (Dahlberg, 1940) were performed on all tooth measurements to assess the measurement error due to replicability. No reproducibility assessment could be performed as long as the measures were recorded by one operator only.

Results

Results are presented in Table 2. Agenesis of maxillary lateral incisors was found to be a significant predictor of tooth size. Patients who were missing maxillary lateral incisors had smaller teeth compared to control subjects. This finding was true for both unilateral and bilateral lateral incisor agenesis.

Table 2 Mean values and SD of mesiodistal dimension of the teeth in the control and sample groups and its relevant statistical significance.

Tooth type	Controls, mean (mm; SD)	Bilateral agenesis, mean (mm; SD)	Unilateral agenesis, mean (mm; SD)	P-value
11	8.84 (0.54)	8.39 (0.46)	8.32 (0.42)	***
21	8.84 (0.54)	8.44 (0.45)	8.29 (0.5)	***
12	6.87 (0.53)		5.34 (0.93)	***
22	6.86 (0.54)		5.2 (0.94)	***
13	7.81 (0.4)	7.52 (0.43)	7.48 (0.44)	***
23	7.82 (0.39)	7.48 (0.44)	7.48 (0.55)	***
14	6.98 (0.44)	6.73 (0.38)	6.65 (0.43)	***
24	7.06 (0.44)	6.8 (0.37)	6.62 (0.41)	***
15	6.67 (0.45)	6.43 (0.38)	6.37 (0.36)	***
25	6.71 (0.46)	6.45 (0.39)	6.31 (0.4)	***
16	9.92 (0.54)	9.79 (0.52)	9.79 (0.56)	n.s.
26	9.96 (0.54)	9.87 (0.53)	9.8 (0.55)	n.s.
31	5.44 (0.37)	5.08 (0.37)	4.98 (0.36)	***
41	5.44 (0.38)	5.04 (0.36)	4.98 (0.34)	***
32	6.02 (0.38)	5.54 (0.37)	5.53 (0.42)	***
42	6 (0.4)	5.55 (0.39)	5.61 (0.39)	***
33	6.74 (0.42)	6.42 (0.4)	6.51 (0.37)	***
43	6.76 (0.4)	6.43 (0.38)	6.48 (0.45)	***
34	7.09 (0.43)	6.73 (0.43)	6.78 (0.34)	***
44	7.11 (0.43)	6.78 (0.38)	6.75 (0.35)	***
35	7.16 (0.46)	6.82 (0.44)	6.84 (0.41)	***
45	7.17 (0.47)	6.83 (0.41)	6.86 (0.44)	***
36	10.56 (0.63)	10.3 (0.53)	10.31 (0.6)	*
46	10.56 (0.64)	10.29 (0.57)	10.29 (0.62)	*

*** $P < 0.001$; ** $P < 0.01$; * $P < 0.05$.

Table 3 Tooth width, mean differences, and standard error mean differences between control and combined agenesis group and corresponding independent sample *t*-test *P*-values.

Tooth type	Controls, mm	Agenesis, mm	Difference, mm	SE mean difference	P-value
11	8.84 (0.54)	8.37 (0.45)	-0.48	0.08	***
21	8.84 (0.54)	8.39 (0.47)	-0.46	0.08	***
12	6.87 (0.53)				
22	6.86 (0.54)				
13	7.81 (0.4)	7.5 (0.43)	-0.31	0.06	***
23	7.82 (0.39)	7.48 (0.48)	-0.34	0.07	***
14	6.98 (0.44)	6.7 (0.4)	-0.28	0.06	***
24	7.06 (0.44)	6.74 (0.39)	-0.32	0.06	***
15	6.67 (0.45)	6.41 (0.38)	-0.26	0.06	***
25	6.71 (0.46)	6.40 (0.39)	-0.31	0.07	***
16	9.92 (0.54)	9.79 (0.53)	-0.13	0.08	*
26	9.96 (0.54)	9.85 (0.53)	-0.11	0.08	n.s.
31	5.44 (0.37)	5.05 (0.37)	-0.4	0.06	***
41	5.44 (0.38)	5.02 (0.35)	-0.42	0.06	***
32	6.02 (0.38)	5.53 (0.39)	-0.49	0.06	***
42	6 (0.4)	5.57 (0.39)	-0.42	0.06	***
33	6.74 (0.42)	6.45 (0.39)	-0.29	0.06	***
43	6.76 (0.4)	6.45 (0.4)	-0.31	0.06	***
34	7.09 (0.43)	6.75 (0.4)	-0.34	0.06	***
44	7.11 (0.43)	6.77 (0.4)	-0.34	0.06	***
35	7.16 (0.46)	6.82 (0.43)	-0.33	0.07	***
45	7.17 (0.47)	6.84 (0.42)	-0.33	0.07	***
36	10.56 (0.63)	10.3 (0.55)	-0.26	0.09	***
46	10.56 (0.64)	10.29 (0.58)	-0.27	0.09	***

A second analysis using the Tukey *post hoc* comparisons verified the strength of the tooth width difference between controls and those subjects with unilateral and bilateral agenesis. The mean width difference between unilateral agenesis and bilateral agenesis was not significant.

After controlling for gender and repeated measures effects, agenesis of maxillary lateral incisors has been found to affect all tooth width measurements except for the maxillary right and left first molars. Interaction between maxillary lateral incisor agenesis and gender was not significant.

When the agenesis groups were combined, the mean difference in tooth size between the control group and the agenesis group varied from 0.11 to 0.49 mm (Table 3). Among the 29 patients with unilateral incisor agenesis, 13 (44.8 per cent) patients had a microdontic contralateral maxillary incisor that was narrower than 4.99 mm in width.

A paired sample *t*-test was performed to assess the measurement error due to replicability on all teeth measurements and showed a non-significant difference for all tooth measures. The range of Dahlberg values for all 24 measurements was from 0.062 mm (tooth #11) to 0.134 mm (tooth #46).

Discussion

Patients with one or two congenitally missing lateral incisors and no other associated dental anomalies have smaller teeth than controls, except for maxillary first molars.

The data presented in this study agrees with previous findings from other researchers who have shown an association between tooth agenesis and reduction in tooth width (Baum and Cohen, 1971; Ooshima *et al.*, 1996; Schalk-Van der Weide and Bosman, 1996; Brook *et al.*, 2002; McKeown *et al.*, 2002; Altug-Atac and Erdem, 2007). Previous researchers have focused on patients with multiple congenitally missing teeth, and a close relationship between the degree of agenesis and the reduction in tooth width has been demonstrated (Schalk-Van der Weide and Bosman, 1996; Brook *et al.*, 2002; McKeown *et al.*, 2002). However, little data are available on tooth widths in patients with congenitally missing lateral incisors as a single dental anomaly.

In our sample, the average difference in the largest mesiodistal width between the control group and the agenesis group ranged from 0.011 mm (tooth #26) to 0.049 mm (tooth #42). Olivadoti *et al.* (2009) performed a morpho-dimensional analysis of the maxillary central incisor crown in a group of patients with unilateral and bilateral agenesis of maxillary lateral incisors. They found a significant average reduction of .038 mm in the mesiodistal width measured at the contact point of the central incisors that had a rectangular shape. Yaqoob *et al.* (2011) found that the mean between-group discrepancy was 0.42 and 0.33 mm per tooth in the upper and lower anterior segments,

respectively. In our sample, the average difference in the largest mesiodistal maxillary central incisor width was 0.47 mm, and it was 0.43 mm for the lower incisors.

Similarly, Brook *et al.* (2002) found a significant decrease of the mesiodistal width for all teeth in mild hypodontia (one or two congenitally missing teeth) patients as compared to controls. The difference ranged from 0.6 to 0.91 mm, and it was slightly larger than the difference observed in our research. However, differences in measurement methodology and the presence of agenesis of teeth other than the maxillary lateral incisors could possibly explain such a difference. Conversely, Yamada *et al.* (2010) found that agenesis of one or two teeth is associated to presence of larger remaining teeth. It has been claimed that a compensatory interaction of the teeth adjacent to the missing teeth is responsible for this increase in size.

Brook *et al.* (2002) stated that the degree of difference in tooth size was related to the degree of hypodontia. We could not confirm this hypothesis as in our research, the mean tooth width difference between unilateral agenesis and bilateral agenesis was not significant. Among the 29 patients with unilateral incisor agenesis, 13 (44.8 per cent) patients had microdontic contralateral maxillary incisors. Garib *et al.* (2010) found a similar prevalence of a microdontic antimere in their sample and pointed out that augmentation of the contralateral incisor in unilateral agenesis patients should be incorporated in a comprehensive treatment plan.

In our sample, there was no difference in the upper first molar mesiodistal width between the sample and the control group. Brook (2009) stated that dental anomalies are caused by complex interaction between genetic, epigenetic, and environmental factors acting during tooth development with different phenotypic expression. Accordingly, factors determining agenesis of lateral incisors may have affected tooth width for all teeth except for upper first molars.

Clinical inferences can also be withdrawn from our findings. In his personal communication, Kokich (2009) stated that a possible clinical challenge in patients with congenitally missing lateral incisors is to open the proper amount of space for implant replacement. Clinical experience and research evidence suggest that a minimum of 6.0 mm of space should be opened for a successful implant supported restoration (Tarnow, 2000). In the presence of a good posterior occlusion and an ideal incisor relationship, patients with narrow natural teeth would require a proportionate but smaller space opened for implant replacement. Therefore, an insufficient amount of interproximal space between the implant and the adjacent teeth could jeopardize the interproximal papilla health and morphology.

A possible treatment strategy for this problem could be to open a wider space for the implant, but an intra-arch and inter-arch tooth size discrepancy could be introduced, and restorative widening and or thickening of the maxillary anterior teeth and restorative widening mandibular anterior teeth could be required. Another possible option is to reduce

the width of the posterior teeth in order to move the maxillary canine distally to increase the amount of space for the implant (Kokich, 2009). According to the findings of our research, maxillary molar mesiodistal dimension could be efficiently reduced in patients with congenitally missing lateral incisors since molar width in patients with maxillary lateral incisor agenesis is comparable to normal patients.

In a series of articles, Rosa and Zachrisson advocated orthodontic space closure combined with restoration as the treatment of choice in patients with congenitally missing lateral incisors (Rosa and Zachrisson, 2001, 2007, 2010). In their latest update, they recommended that clinicians should evaluate the necessity not to grind the mesialized canine to a smaller lateral incisor but eventually restore it as a 'big' lateral incisor and restore the first premolars to replace the canines. The central incisors should be also restored (i.e. the six anterior teeth) to achieve a result that provides the look of a healthy natural dentition (Rosa and Zachrisson, 2010). Their indication to restore the six maxillary anterior teeth was intended to improve function and esthetics and was guided by their clinical judgement. Our research findings provide a rationale for such procedures: patients with maxillary lateral incisor agenesis have smaller teeth that are in need of restorations if appropriate absolute and relative mesiodistal dimensions are to be achieved for optimal esthetics. However, it should be pointed out that widening of the maxillary anterior teeth will result in a tooth-size discrepancy (maxillary excess) and increased overjet since we found reduced tooth widths of both maxillary and mandibular teeth in patients with maxillary lateral incisor agenesis. Therefore, clinical procedures such as enlargement of mandibular anterior teeth or thickening of maxillary restorations, as proposed by Rosa and Zachrisson (2010), may have to be implemented to achieve ideal functional and esthetic outcomes (Rosa and Zachrisson, 2010).

In our CMLI group, 39 (48.1 per cent) patients had at least one central incisor that was smaller than the average control central incisor by more than 1 SD. About 7 per cent of the patients had at least one central incisor that was smaller than the control central incisor by more than 2 SDs. These data would provide the rationale for increasing the widths of the maxillary anterior teeth in patients with maxillary lateral incisor agenesis. Nevertheless, an appropriate and individualized interdisciplinary treatment plan is recommended.

Conclusions

- Patients with congenitally missing lateral incisors have narrower teeth than patients without any dental anomalies, except for maxillary first molars
- There is no difference in the amount of mesiodistal width reduction between patients with unilateral and bilateral agenesis

- Patients with unilateral agenesis have a higher prevalence of microdontic contralateral incisors
- Restoration of the maxillary anterior teeth could be recommended in order to improve the overall esthetic outcome in both treatment alternatives: space-opening and canine substitution.

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