Tooth crown size and morphology in Turner syndrome

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The aims of this investigation were to analyze permanent tooth crown size and morphology and to perform symmetry analysis between corresponding teeth on the right and the left side. The material comprised 32 Turner syndrome patients aged 7–16.7 years. As controls served 33 normal girls 10.2–16.7 years old. The mesiodistal diameter was significantly reduced for every tooth measured except for the maxillary canine. The buccolingual dimension was reduced only for some teeth. Eleven morphologic traits were defined. Ten of these were identified in the Turner patients; seven occurred in the controls as well, but at a lower frequency. Some of the traits have not been described earlier for this group of patients. There was a significant difference between Turner and control patients both in the number of patients with bilateral tooth asymmetries and in the number of corresponding tooth pairs in the maxilla with bilateral asymmetry. No significant differences were found between the 45X patients and the other karyotypes. Maxillary central incisors showed a surprisingly high relative frequency (38.5%) of bilateral asymmetry. □ *Anatomy; tooth; X-chromosome*

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Total or partial loss of an X chromosome as expressed in the Turner syndrome will influence both the size and the shape of certain craniofacial structures (1–3). The degree of sex chromosome deficiency seems to determine the morphologic manifestations. Generally, symptoms are less severe in cases of mosaics and deletions than in complete monosomy X (4). Other authors suggest that the syndrome as caused by the i(Xq) and X0 variants is most severe (5).

Studies on tooth crown size seem to confirm that the different karyotypes are differently affected. Reduced size of the tooth crown is found in monosomy X and in mosaic and isochromosome of the long arm of X karyotypes, probably as a function of a reduced thickness of the enamel layer (6). However, the pattern and extent of the reductions have been shown to differ slightly for the various karyotypes (7–10).

The effect of sex chromosome aberrations on dental morphology is less explored. For Turner patients the reports available all deal with the 45X karyotype. Kirveskari & Alvesalo (11) have described simplified crown morphology and reduced expression of molar crown traits. Mayhall & Alvesalo (12) observed reduction of cusp areas and volume in the maxillary first molar. Townsend et al. (7) reported, after a preliminary analysis of dental morphology in 121 45X women, that no striking crown abnormalities were present.

Studies of normal populations and of persons with sex chromosome aberrations indicate further that two X chromosomes give a better control of symmetry between corresponding teeth on the right and left side (13, 14). The effect of sex chromosome aneuploidy and structural aberrations of the X chromosome, as seen in the Turner syndrome, on bilateral tooth asymmetries is investigated for the X0 karyotype only (10).

Since the data on tooth morphology in patients with the different karyotypes constituting Turner syndrome are scanty and several observations indicate that sex chromosome aberrations influence tooth morphology in various manners, we decided to study a group of Turner patients. The present investigation aims 1) to evaluate permanent tooth crown size and morphology, 2) to perform a symmetry analysis between

the teeth of the right and left side, and 3) to evaluate how these variables are influenced by the karyotype.

Materials and methods

This investigation is part of a systematic study of Turner patients whose intention is to evaluate growth and development before, during, and after therapy with growth hormone and estrogen. The karyotype was determined by chromosome analysis of peripheral lymphocytes. The karyotyping, the hormone therapy, and the study of general variables were performed by the Department of Pediatrics, University of Bergen.

The present material consisted of 32 girls with Turner syndrome from different parts of Norway. Their karyotypes were 45X (n = 23); 45X/46XX (n = 3); 46X,i(Xq) (n = 3); 45X/46X,i(Xq) (n = 1); 45X/46XY (n = 1); and 45X/46,r(Xq) (n = 1). At the Department of Orthodontics, University of Bergen, they were examined twice. At the first examination their mean age was 12.1 years (range, 7–16.7 years). Mean age at the second examination was 14 years. At both examinations plaster casts and five intraoral slides were made. In two patients only slides were obtained since lack of cooperation precluded impressions. As controls served 33 girls without any known genetic or hormonal disorder. Their mean age was 12.7 years (range, 10.2–16.4 years). One set of plaster casts and five intraoral slides were available.

Recording of tooth size

Mesiodistal and buccolingual crown diameters were measured on the plaster casts by one investigator (M. Midtbø), using a specially modified sliding caliper with 0.1-mm gradation, as described by Moorrees et al. (15). According to Moorrees, the measurements should be performed at the contact points to record the greatest mesiodistal width. Owing to altered morphology of some teeth in the Turner group the contact points were missing or located gingivally. The measurements were then performed at the greatest mesiodistal width close to the gingival margin. Teeth not fully erupted or damaged by trauma, restorations, caries, or hypoplasia were excluded. Owing to the eruption state of the second molars they were excluded from this part of the study. The measurements were repeated after 6 weeks. The mean of the two measurements was used in the further calculations.

Dental morphology

On the basis of a pilot study of the material and a search of the literature, 11 different traits or irregularities were selected (Table 1). The nomenclature and description of the traits were partly from Kraus et al. (16), who studied the dentition of mentally retarded children; from Cohen et al. (17), who studied trisomy G patients; and from Carlsen's textbook on dental morphology (18). The scorings were performed twice by one investigator (M. Midtbø). Both sets of casts for each Turner patient were used in the scorings. Thereafter all positive registrations

Table 1. Traits or irregularities scored in 33 Turner patients

1.	**Cervicoincisal convergence of the approximal surfaces,** in relation both to each other and to the crown axis.
2.	**Wedge shape of the maxillary incisors.** Cervicoincisally and mesiodistally, the facial surface was plane, sometimes even concave, and without cervical prominence. The lingual surface was nearly plane, without marginal ridges and marked cingulum.
3.	**Shovel-shaped incisors.**
4.	**Altered mamelon pattern.**
5.	**Atypical buccal cusps on maxillary canines and premolars.** Accessory cusp.
6.	**Conical premolars,** defined by cervicoincisal convergence of the approximal and buccolingual surfaces.
7.	**Reduced lingual cusp height of the first mandibular premolar,** often combined with buccolingual compression of the occlusal surface.
8.	**Reduced distolingual cusp** (hypocone), distobuccal cusp (hypoconid).
9.	**Missing distolingual cusp** (hypocone), distobuccal cusp (hypoconid).
10.	**Carabelli's trait.** Only teeth with no sign of Carabelli's trait were registered.
11.	**Nippled cusps.**

and all borderline cases were discussed and scored as present or not by the two observers jointly (M. Midtbø, A. Halse). The joint decisions were used in the calculations. Teeth with large restorations or damaged by trauma were excluded. The number of teeth scored for morphologic traits was somewhat higher than the number of teeth measured, owing to the state of eruption and the location of minor restorations. Mandibular second molars were not sufficiently erupted for registration. Some maxillary second molars could be scored. One Turner patient had hypodontia of the left mandibular second molar.

Symmetry analysis

The symmetry was recorded by two variables: a) The mesiodistal diameters of corresponding teeth from the left and right sides were compared. Differences in mesiodistal width of 0.3 mm or more were recorded as asymmetry. b) Morphologic asymmetries between corresponding teeth on the right and left sides were recorded on the basis of the earlier described scorings. Some pairs of teeth had asymmetry in both mesiodistal width and morphology. The investigators were unaware of the karyotyping of the Turner patients during the registration.

Statistics

The error of measurement was calculated from the formula $\tau = \sqrt{\varepsilon d^2/2n}$ (19), where d is the difference between the first and the second measurement for each variable. The error was small for all teeth measured, averaging 0.06 mm. Possible skewness in the distribution pattern of the measured tooth widths was examined on histograms of the recorded values. The form of the histograms indicated no skewness. Differences in mesiodistal and buccolingual widths between the individual teeth on the right and the left side were tested by means of Student's t test. As no difference was found, only the right side was used.

The buccolingual and mesiodistal diameters of the teeth were compared with the control material and between the 45X karyotype and the other karyotypes with Student's t test.

The following variables were compared between Turner and control patients and between the 45X karyotype and the other karyotypes by means of the chi-square test: a) the number of teeth with traits; b) the number of patients with bilateral tooth asymmetries; and c) the number of pairs of corresponding teeth with bilateral asymmetry.

Two patients with incomplete records were not included.

All calculations were performed by a computer program (20).

Results

Tooth size

Table 2 shows the mesiodistal and buccolingual dimensions for the 45X patients, the isochrome and mosaic karyotype patients, and normal control girls.

45X patients

The mesiodistal diameter was significantly reduced for every tooth measured except the maxillary canine. The buccolingual dimension was significantly reduced only for second premolars, first molars, and the maxillary first premolar.

Mesiodistally the maxillary canine showed the smallest size reduction (2.7%). The first molars and second premolars were reduced 8.0–11.3%, the greatest difference being found for the mandibular first molar. The buccolingual dimensions showed smaller differences but a similar pattern. Average size reductions for 45X patients compared with normal control girls were 6.6% and 4.3% for the mesiodistal and buccolingual dimensions, respectively.

Isochromosome and mosaic karyotypes

Isochromosome and mosaic karyotypes showed significant reduction in the mesiodistal dimension for all teeth except the maxillary canine and the mandibular central

Table 2. Mesiodistal and buccolingual dimensions in 21 45X girls, 9 isochromosome and mosaic karyotypes (46X,i(Xq) ($n = 3$), 45X/46XX ($n = 3$), 45X/46XY ($n = 1$), 45X/46X,i(Xq) ($n = 1$), 45X/46X,r(Xq) ($n = 1$)) and 33 normal control girls. Mean value, standard deviation (SD), and coefficient of variation (CV). Asterisks indicate significant differences of 45X versus controls and isochromosome and mosaic karyotypes versus controls. Isochromosome and mosaic karyotypes versus 45X showed no significant differences

	45X				Isochromosomes and mosaics				Controls			
	n	*x*	SD	CV	*n*	*x*	SD	CV	*n*	*x*	SD	CV
Mesiodistal maxilla												
I^1	20	8.20**	0.63	7.7	9	7.95**	0.50	6.3	30	8.72	0.53	6.1
I^2	21	6.35*	0.53	8.3	9	6.13*	0.58	9.5	33	6.69	0.66	9.9
C	20	7.53	0.36	4.9	7	7.52	0.44	5.9	24	7.74	0.36	4.7
PM^1	16	6.42***	0.30	4.7	8	6.34*	0.55	8.7	29	6.95	0.48	6.9
PM^2	15	5.97***	0.42	7.0	7	5.80**	0.46	7.9	23	6.60	0.39	5.9
M^1	13	9.18***	0.28	3.1	8	9.51*	0.59	6.2	25	10.15	0.45	4.4
Mesiodistal mandible												
I_1	21	4.93***	0.26	5.3	9	5.16	0.29	5.6	30	5.36	0.28	5.2
I_2	19	5.66*	0.34	6.0	9	5.56*	0.38	6.8	32	5.89	0.34	5.8
C	20	6.45*	0.38	5.9	9	6.37*	0.37	5.8	33	6.71	0.32	4.8
PM_1	20	6.73*	0.31	4.6	8	6.46**	0.43	6.7	30	7.00	0.46	6.6
PM_2	20	6.47***	0.41	6.3	8	6.29**	0.50	7.9	28	7.03	0.39	5.5
M_1	14	9.84***	0.30	3.0	7	10.06*	0.89	8.8	27	11.09	0.50	4.5
Buccolingual maxilla												
I^1	18	6.76	0.61	9.0	8	6.98	0.39	5.6	32	7.07	0.60	8.5
I^2	17	5.85	0.65	11.1	8	6.28	0.56	8.9	30	6.21	0.64	10.3
C	16	7.75	0.49	6.3	6	7.94	0.74	9.3	19	7.83	0.61	7.8
PM^1	18	8.69**	0.44	5.1	8	8.57*	0.64	7.5	31	9.20	0.63	6.8
PM^2	16	8.82**	0.44	5.0	7	8.40**	0.63	7.5	27	9.21	0.51	5.5
M^1	18	10.59***	0.41	3.9	7	10.83	0.91	8.4	32	11.36	0.46	4.0
Buccolingual mandible												
I_1	18	5.79	0.33	5.7	7	5.79	0.36	6.2	33	5.90	0.36	6.1
I_2	18	5.95	0.33	5.5	7	6.06	0.48	7.9	29	6.19	0.33	5.3
C	19	6.95	0.52	7.5	5	7.15	0.59	8.2	26	7.06	0.55	7.8
PM_1	18	7.49	0.53	7.1	6	7.43	0.84	11.3	31	7.69	0.49	6.4
PM_2	19	8.03*	0.53	6.6	8	7.86*	0.48	6.1	29	8.39	0.59	7.0
M_1	16	9.56***	0.47	4.9	8	9.73**	0.61	6.3	30	10.51	0.39	3.7

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

incisor; average reduction was 7.4%. The buccolingual differences were generally small, and some teeth even showed slightly larger diameters than those of normal control girls. Significant reductions were found only for second premolars, the maxillary first premolar, and the mandibular first molar.

Isochromosome and mosaic karyotypes compared with the 45X group

The differences between the 45X girls and the isochromosome and mosaic karyotypes were non-significant for both mesiodistal (0.9%) and buccolingual (1.1%) measurements.

In all three samples the coefficient of variation showed the greatest value for the maxillary lateral incisor. Generally, the variability of tooth size was greatest in the isochromosome and mosaic group. For the 45X girls the greater variability was mainly related to mesiodistal tooth width. In buccolingual tooth width the coefficient of variation showed the same value in both groups.

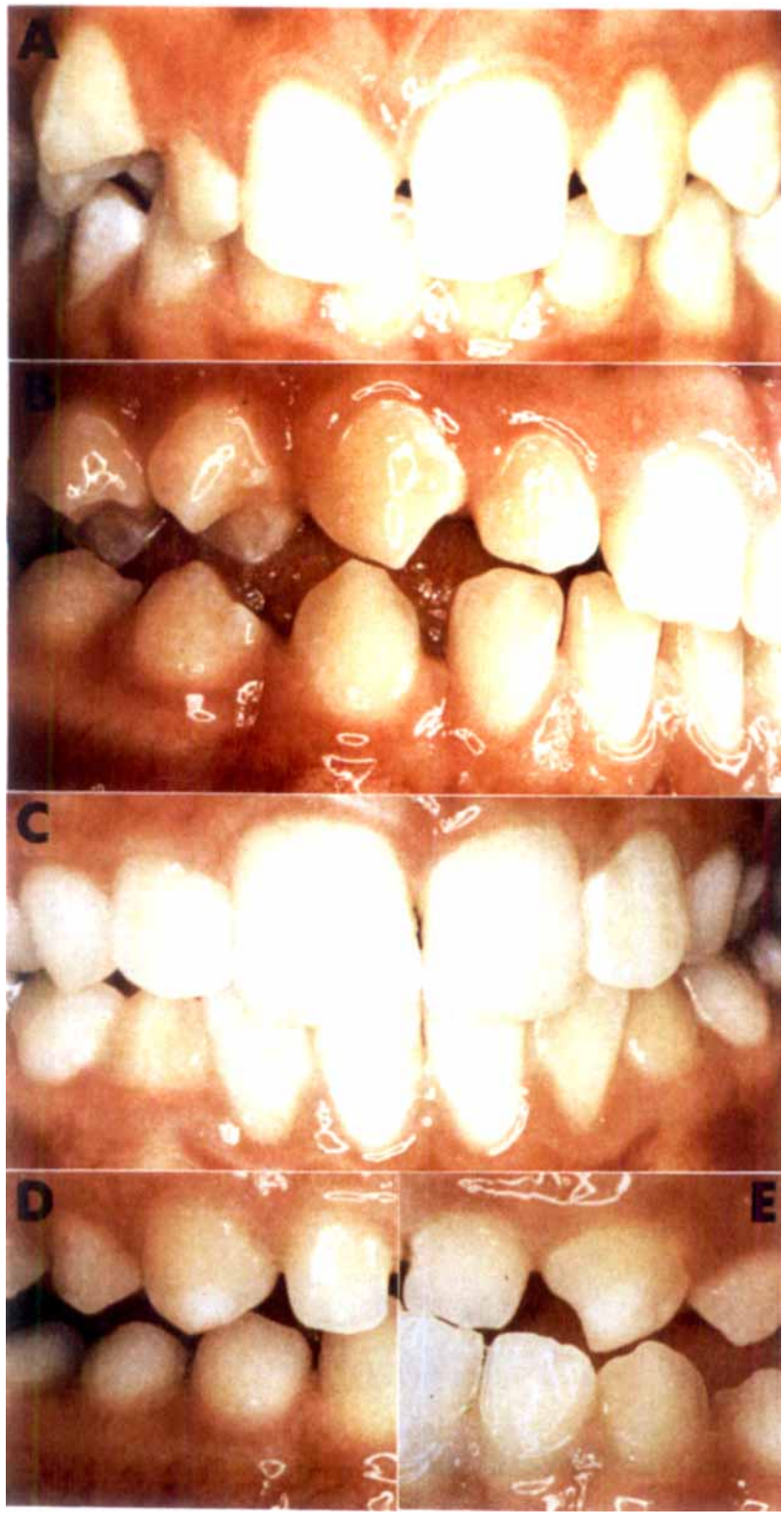

Fig. 1A. Cervicoincisal convergence of the maxillary central incisors.
1B. Wedge shape of the maxillary central incisor. Altered mamelon pattern of the maxillary lateral incisor. Atypical buccal cusps on maxillary canine and premolars; note also accessory cusps on 14, 15, 44, and 45.
1C. Asymmetry in mesiodistal width of the maxillary central incisors (11 > 21; difference, 0.6 mm).
1D and E. A patient, karyotype 45X/46XX, with several asymmetries. The lateral incisors differ both in mesiodistal width and morphology, the canines only in morphology.

Dental morphology

In the dentition of all registered Turner patients one or more of the described traits were identified. The number of affected teeth per patient ranged from 2 to 18. The dentition of some patients is illustrated in Figs. 1 and 2.

Traits were observed with equal frequencies on the right and the left side. Table 3 shows the relative frequencies of each trait on the basis of tooth type for the Turner and control samples on the right side. Some teeth were affected by two or three traits. Maxillary teeth were affected more often than mandibular teeth. The difference was most pronounced for the incisors.

Cervicoincisal convergence of the approximal surfaces occurred in 26.3% of the maxillary incisors (Fig. 1A); 41.9% of the central incisors and 10.7% of the lateral incisors were affected.

Wedge shape affected the maxillary central and lateral incisors in 58.1% and 51.7% of the cases (Figs. 1B and 2C).

Shovel shape was not observed for any tooth in the Turner material.

Altered mamelon pattern was mainly observed in the lateral incisors, where it occurred with a frequency of 37.9%. For the central incisors the frequency was 6.5% (Fig. 1B).

Atypical buccal cusps occurred on average in 24.7% of the maxillary canines and premolars. In the premolars the buccal cusp was peg-shaped centrally with a smaller mesially positioned accessory cusp (Fig. 1B). In the canine the mesial aspect of the cusp showed a cupulate defect, as illustrated in Figs. 1B and E.

Table 3. Relative frequencies of each trait distributed, in accordance with type of tooth

		Turner		Control	
Trait	Tooth affected	No. of teeth	Relative frequency	No. of teeth	Relative frequency
1	I^1_{max}	31	41.9	31	3.2
	I^2_{max}	29	10.7	33	3.0
2	I^1_{max}	31	58.1	31	0
	I^2_{max}	29	51.7	33	0
3	I^1_{max}	31	0	31	3.2
	I^2_{max}	29	0	33	6.1
4	I^1_{max}	31	6.5	31	0
	I^2_{max}	29	37.9	33	12.1
5	C_{max}	28	25.0	24	0
	PM^1_{max}	25	40.0	30	0
	PM^2_{max}	22	9.1	28	0
6	PM^1_{max}	25	12.0	30	0
	PM^2_{max}	22	72.7	28	0
	PM^1_{mand}	28	0	31	3.2
	PM^2_{mand}	28	57.1	29	0
7	PM^1_{mand}	28	60.7	32	0
8	M^1_{max}	28	10.7	32	0
	M^1_{mand}	30	36.6	32	12.5
9	M^1_{max}	28	10.7	32	0
	M^1_{mand}	30	36.6	32	9.3
	M^2_{max}	18	72.2	19	36.8
10	M^1_{max}	28	67.8	32	37.5
11	C_{max}	28	7.1	24	0
	PM^1_{max}	25	20.0	30	0
	PM^2_{max}	22	18.2	28	0
	C_{mand}	30	16.7	30	0
	PM^1_{mand}	28	17.9	32	0
	PM^2_{mand}	28	10.7	29	0

Conical premolars were frequently registered (Fig. 2A). Highest frequency was found for maxillary second premolars (72.7%). None of the mandibular first premolars were affected.

Reduced lingual cusp height, often combined with buccolingual compression of the occlusal surface, was found in 60.7% of the mandibular premolars. Occasionally, a grove or furrow also appeared, extending from the mesial part of the lingual surface to the middle buccal surface (Figs. 1B and 2B).

Reduced distobuccal cusp or hypoconid was found for 36.6% of the mandibular first molars. Of the first maxillary molars, 10.7% showed reduced hypocone.

Missing distobuccal cusp or hypoconid occurred in mandibular first molars (36.6%). In maxillary ones 10.7% lacked the hypocone. Thirty-seven maxillary second molars were registered; of these, 27 were missing the distolingual cusp.

No sign of Carabelli's trait was observed in 67.8% of the maxillary first molars.

Nippled cusps were observed in canines and premolars with a frequency ranging from 7.1% to 20.0%. The maxillary canine showed the lowest prevalence and the maxillary first premolar the highest one (Fig. 2D).

Traits found in the control material

As shown in Table 3, only seven of the irregularities were present in the control material and always in a lower frequency. Shovel shape of the maxillary incisors occurred only in the control material.

Karyotype and distribution of traits

There was a significant difference ($p < 0.000$) between Turner and control patients in the number of teeth with traits in both the mandible and the maxilla. The relative frequencies of teeth with traits distributed on the basis of the karyotypes are shown in Table 4. Highest relative frequency occurred in 45X patients, who showed 67.1% affected teeth in the maxilla. For the other karyotypes the sample size was small, varying from one to three persons in each group, so the results have to be considered with caution. There were no significant differences between the 45X patients and the other karyotypes.

Symmetry analysis

Totally, 30 pairs of teeth with asymmetries in mesiodistal tooth widths of 0.3 mm or more (range, 0.3–1.2 mm) were registered in the Turner group; of these, 5 pairs also showed morpholgic asymmetries. Ten pairs of teeth showed only morphologic asymmetries related to the earlier described traits.

A most interesting finding was the high number of asymmetric pairs of maxillary central incisors. Ten of 26 pairs showed asymmetry: 9 pairs in mesiodistal tooth widths

Table 4. Relative frequencies of teeth with traits distributed in accordance with karyotype

Karyotype	No. of patients	No. of teeth		Teeth with traits		Relative frequencies (%)	
		Max.	Mand.	Max.	Mand.	Max.	Mand.
45X	21	225	235	151	81	67.1	34.5
45X/46XX	3	38	36	21	8	55.3	22.2
46X,i(Xq)	3	27	30	14	12	51.9	40.0
45X/46X,i(Xq)	1	8	12	4	3	50.0	25.0
45X/46X,r(Xq)	1	14	12	7	6	50.0	50.0
45X/46XY	1	12	12	6	7	50.0	58.3
Turner	30	324	337	203	117	62.6	34.7
Control	33	349	375	42	16	12.0	4.3

Maxilla: $x = 189.87$; $p < 0.000$; mandible: $x = 109.75$; $p < 0.000$.

and 1 pair in morphology (Figs. 1C and 2C). The difference in mesiodistal width ranged from 0.3 to 0.8 mm.

Asymmetry was frequent also for the mandibular central incisor (12.9%). Maxillary lateral incisors had high but equal frequencies of asymmetries in both groups (24.1–28.6%).

Asymmetric pairs of maxillary canines and first molars were found only in the Turner group. Maxillary canines showed asymmetry in cusp morphology (Figs. 1D and E), whereas first molars showed both dimensional and morphologic asymmetry.

Karyotype and asymmetry

One to four asymmetric pairs of teeth were found in 23 of 31 examined Turner patients. Table 5 lists the karyotype, the number of patients with and without tooth asymmetries, and the number of asymmetric pairs of teeth compared with control patients. Maxillary teeth were affected more often than mandibular ones. One patient with 45X/46XX karyotype showing asymmetry of maxillary laterals and canines is illustrated in Figs. 1D and E.

There was a significant difference between Turner and control patients both in the number of persons with asymmetry ($p < 0.01$) and in the number of asymmetrical tooth pairs in the maxilla ($p < 0.001$). In the mandible there were no significant differences.

None of the patients with isochromosome karyotype 46X,i(Xq) had asymmetric pairs of teeth. Within all the other karyotypes asymmetries were observed. There were no significant differences between the 45X patients and the other karyotypes.

Discussion

The present investigation showed reduced tooth crown size and altered tooth morphology in 32 young Turner patients. Several morphologic traits and bilateral asymmetries not earlier reported for this syndrome were observed.

Our findings on tooth size agree with those of Townsend et al. (7) and Varrela et al. (9), who investigated 45X and 45X/46XX women. There are few similar studies on morphology. To a certain extent our results are in accordance with those of Kirveskari & Alvesalo (11). This indicates that reduced tooth crown size and certain morphologic traits are parts of the Turner syndrome.

In a study of dental asymmetry Townsend et al. (10) reported no differences between 45X females and normal controls, which is inconsistent with our findings.

It is generally accepted that dental crown

Table 5. Number of patients and tooth pairs with bilateral asymmetry in morphology and mesiodistal width distributed in accordance with karyotype

Karotype	No. of patients		Pairs of teeth		Pairs of teeth with asymmetry	
	Without asymmetry	With asymmetry	Max.	Mand.	Max.	Mand.
45X	5	16	109	110	15	6
45X/46XX		3	19	18	7	0
46X,i(Xq)	3		13	15	0	0
45X/46X,i(Xq)		1	2	6	2	2
45X/46X,r(Xq)		1	7	6	2	2
45X/46XY		1	6	5	2	1
Turner	8	22	156	160	28	11
Control	20	13	167	167	9	5

Patients with asymmetry: $x = 7.73$; $p < 0.01$; pairs of teeth with asymmetry in the maxilla: $x = 12.54$; $p < 0.001$; pairs of teeth with asymmetry in the mandible: $x = 2.64$, NS.

morphology cannot be altered after full mineralization has been reached. The critical period of odontogenesis is therefore between the initial developmental stage and the mineralization stage of the dental crown. This period will vary both in time of onset and duration for the different teeth, extending from about 28 days in utero to approximately 16 years postnatally (21). In our material of Turner patients size reductions and morphologic traits were observed for all types of teeth, indicating that the etiology is found in the general condition of X chromosome aberration rather than in a particular embryologic event.

Disturbed or restricted growth is the most predominant characteristic of girls with the Turner syndrome (22–24), besides various somatic abnormalities. According to Lubin et al. (25), there seems to be a generalized growth retardation process affecting all tissues in the body. The mechanism whereby complete or partial X chromosome monosomy results in so many anatomic abnormalities is still unknown. Lippe (26) concluded that almost all the abnormalities observed in Turner syndrome patients involve mesenchymal tissue. She has therefore proposed a hypothesis to explain this phenomenon specifically—namely that 'a major defect in Turner syndrome is disordered mesenchymal tissue growth'.

Does this hypothesis of disordered mesenchymal tissue growth fit with our findings on dental morphology?

Huggings et al. (27) demonstrated as early as in 1934 that the dental mesoderm controls the tooth form. Kollard & Baird (28–30) found that the shape of a particular tooth is determined not by the epithelial enamel organ but by the dental mesoderm, and that the dental mesoderm can produce the expression of genes for enamel synthesis in ectopic, non-dental epithelium. Berkovitz & Moxham (31) and Berkovitz & Shellis (32) conclude that the dental papilla is important in the induction of the enamel organ and in the determination of crown form, but the nature of the induction process itself remains obscure. The morphologic pattern or basic form and relative size of the future tooth is established by morphodifferentiation—that is, differential growth (33). Disturbances in morphodifferentiation may affect the form and size of the tooth without impairing the function of the individual ameloblast or odontoblast. The result may be a malformed tooth with structurally normal enamel and dentin. This may be the type of morphologic malformation found in Turner syndrome patients. On the basis of these findings it seems probable that the observed alterations in dental size and morphology may be caused by disordered mesenchymal tissue growth, thus confirming the earlier hypothesis of Lippe (26).

As shown by Alvesalo & Tammisalo (6), reduced enamel thickness contributes to the reduced tooth size. Normally, the enamel layer is thinnest near the cementoenamel junction and shows increasing thickness towards the contact points. The tooth measurements in this investigation and those of Townsend et al. (7) and Varrela et al. (9) were done by the method of Moorrees et al. (15) on plaster models. According to the method the measurements should be performed at the contact points corresponding to the greatest mesiodistal width. This investigation showed that cervicoincisal convergence of the approximal surfaces was a prevalent trait both for the maxillary central incisors and for the premolars. Because of the convergence the contact points were missing or located more gingivally (Figs. 1A and 2A). Accordingly, the measurements were also performed more gingivally for Turner than for control patients. Owing to this deviation an underestimation of the 'true' difference in mesiodistal width between Turner and control patients may have occurred.

The present investigation showed that all mesiodistal crown diameters except that of the maxillary canine were significantly reduced. In the buccolingual direction only some teeth showed significant reductions, and these reductions were always smaller than the former ones. The 45X patients and the other karyotypes showed slightly different patterns of reduction. Similar findings were also made by Varrela et al. (9) and Townsend et al. (7). These findings are consistent with the effect of reduced thickness of the enamel layer (6) and indicate that

the extent of X chromosome deficiency is the cause of the observed differences between the karyotypes. The size reductions confirmed further the altered tooth form; the Turner patients had more rounded or conical teeth most prevalent for incisors, premolars, and first molars.

Ten morphologic traits were observed in our material of Turner patients; seven were found also in the control material. Some of the traits observed have not been reported earlier for this group of patients. Those were wedge shape of the incisors, as illustrated in Figs. 1B and 2C, altered mamelon pattern (Fig. 1B), atypical buccal cusps on maxillary canine and premolars (Figs. 1B and E), and nippled cusps (Fig. 2D). Some of the specific traits also occurred with high frequencies (Table 3). Altogether this gives the dentition a characteristic appearance, which in our opinion is specific for Turner syndrome patients.

Kirveskari & Alvesalo (11) reported 'morphological dental reduction' after having examined 45X women for four traits: shovel shape, hypocone reduction, Carabelli's trait, and lower molar cusp number. As shown in Table 3, our findings partly agreed. Reduced or missing hypoconid occurred as often as in 42 of 59 mandibular molars, which is also reflected in the observed reduction in mesiodistal width of 11.3%. Carabelli's trait was found in a lower frequency, and none of the maxillary incisors showed true shovel shape. In contrast, 54.9% had wedge shape characterized by smaller cingulum and absence of marginal ridges on the lingual surface. As suggested, the etiology of the altered tooth morphology may be found in the general growth disturbance of Turner syndrome. Particularly, the reduced enamel layer (6) seems decisive for the tooth form, and it appears that many of the morphologic characteristics given by a normal enamel layer are also lost with the reduction.

Jensen (34) observed significantly more spacing, especially in the upper front, in Turner patients than in controls. It is obvious that the high frequency of conical tooth morphology contributes to the spacing. This spacing may therefore not only be a true expression of excess of space in the maxillary arch but also demonstrate the high frequency of conical tooth morphology.

The frequency of traits or irregularities was higher in the maxilla than in the mandible, especially for incisors and canines. Similar findings were made by Kraus et al. (16) for mentally retarded subjects and by Cohen et al. (17) for trisomy G patients.

The distribution of traits showed no significant differences between the 45X patients and the other karyotypes. However, for some of the karyotypes the sample size was small, so the results should be considered with caution.

Townsend et al. (10) found no differences in dental asymmetry between 45X patients and the controls, whereas Down syndrome patients showed increased values. Cohen et al. (17) stated, after examining the dentition of trisomy G patients, that bilateral symmetry was found within each arch both in the control and in the mongoloid samples. There are some reports of asymmetry in Turner syndrome patients. Eberle et al. (35) observed facial asymmetries, and Gorlin et al. (36) asymmetry of the facial bones and the gingiva. The present investigation showed that both the number of persons with bilateral asymmetries and the number of asymmetric pairs of teeth in the maxilla were significantly higher in Turner than in control patients. No differences were found in the mandible. Our findings indicate that there is an association between the Turner syndrome and asymmetry and supports the hypothesis of reduced growth control for this group of patients.

The distribution of asymmetry on the basis of the type of tooth surprisingly showed that the maxillary central incisors had the highest relative frequency. Normally, the first tooth in each tooth group is the morphologically most stable. Garn et al. (37, 38) found that asymmetry was related to tooth size and was most pronounced for the more distal tooth in each class. The same findings were made by Townsend et al. (10) in 45X patients.

The high incidence of asymmetric incisors indicates that the teeth formed early are more affected than those formed later. Similar findings have also been made for tooth size reduction (9). It may be speculated

whether this has to do with the timing or level of X-chromosome inactivation.

It is known from Garn et al. (39) that asymmetry is slightly more prevalent in boys than in girls, most probably because the paired X chromosome in females gives a more effective dimensional control. This finding might explain the increased number of asymmetric pairs of teeth in women with X-chromosome aberrations. Anticipating that the degree of X-chromosome aberration is decisive for the asymmetries, an increased number of asymmetries in the 45X karyotype should be expected. Our results showed no difference between 45X karyotype and the other karyotypes. However, larger sample sizes are needed before conclusions can be drawn.

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References

1. Jensen BL. Craniofacial morphology in Turner syndrome. J Craniofac Genet Dev Biol 1985;5:327–40.
2. Laine T, Alvesalo L, Lammi S. Palatal dimension in 45,X-females. J Craniofac Genet Dev Biol 1985; 5:239–46.
3. Laine T, Alvesalo L. Size of the alveolar arch of the mandible in relation to that of the maxilla in 45,X females. J Dent Res 1986;65:1432–4.
4. Bühler EM. A synopsis of the human Y chromosome. Hum Genet 1980;55:145–75.
5. Therman E, Denniston C, Sarto GE, Ulber M. X chromosome constitution and the human female phenotype. Hum Genet 1980;54:133–43.
6. Alvesalo L, Tammisalo E. Enamel thickness in 45,X females' permanent teeth. Am J Hum Genet 1981; 33:464–9.
7. Townsend C, Jensen BL, Alvesalo L. Reduced tooth size in 45X (Turner syndrome) females. Am J Phys Anthropol 1984;65:367–71.
8. Mayhall J, Alvesalo L, Townsend G. Tooth crown size in 46,X,i (XQ) females (abstract). J Dent Res 1987;66(Spec Iss):Abstract 22.
9. Varrela J, Townsend G, Alvesalo L. Tooth crown size in human females with 45,X/46,XX chromosomes. Arch Oral Biol 1988;33:291–4.
10. Townsend G, Alvesalo L, Jensen BL, Kari M. Patterns of tooth size in human chromosomal aneuploidies. In: Russel DE, Santoro J-P, Sigogneau-Russell D, editors. Teeth revisited. Proceedings of the VIIth International Symposium on Dental Morphology. Mus Nat Hist C:53. Paris: Museum of Natural History, 1988:25–45.
11. Kirveskari P, Alvesalo L. Dental morphology in Turner's syndrome (45,X females). In: Kurten B, editor. Teeth: form, function, and evolution. New York: Columbia University Press, 1982:298–303.
12. Mayhall JT, Alvesalo L. Dental morphology of 45,XO human females: molar cusp area, volume, shape and linear measurements. Arch Oral Biol 1992;12:1039–43.
13. Alvesalo L. The influence of sex-chromosome genes on tooth size in man [thesis]. Proc Finn Dent Soc 1971;67:7–52.
14. Garn SM, Lewis AB, Kerewsky RS. Buccolingual size asymmetry and its developmental meaning. Angle Orthod 1967;37:186–93.
15. Moorrees CFA, Thomsen SØ, Jensen E, Yen PKJ. Mesiodistal crown diameters of the deciduous and permanent teeth in individuals. J Dent Res 1957; 86:39–47.
16. Kraus BS, Clark GR, Oka SW. Mental retardation and abnormalities of the dentition. Am J Ment Defic 1968;72:905–17.
17. Cohen MM, Blitzer FJ, Arvystas MG, Bonneau RH. Abnormalities of the permanent dentition in trisomy G. J Dent Res 1970;49:1386–93.
18. Carlsen O. Dental morphology. Copenhagen: Munksgaard, 1987.
19. Dahlberg G. Statistical methods for medical and biological students. London: Allen and Unwin, 1940.
20. Ryan BF, Joiner BL, Ryan TA. Minitab handbook. 2nd ed. Boston: Duxbury Press, 1985.
21. Nery EB, Kraus BS, Croup M. Dental organ formation: a chronologic and topographic sequence. J Dent Child 1975:42:467–73.
22. Lindsten J, Filipsson R, Hall K, Leikrans S, Gustavson K-H, Ryman N. Body height and dental development in patients with Turner's syndrome. Helv Paediat Acta 1974;34 Suppl:33–46.
23. Park E. Body shape in Turner's syndrome. Hum Biol 1977;49:215–23.
24. Pelz L, Timm D, Eyermann E, Hinkel GK, Kirchner M, Verron G. Body height in Turner's syndrome. Clin Genet 1982;22;62–6.
25. Lubin MB, Gruber HE, Rimoin DL, Lachman RS. Skeletal abnormalities in the Turner syndrome. In: Rosenfeld RG, Grumbach MM, editors. Turner syndrome. New York and Basel: Marcel Dekker Inc, 1990.
26. Lippe BM. Physical and anatomical abnormalities in Turner syndrome. In: Rosenfeld RG, Grumbach MM, editors. Turner syndrome. New York and Basel: Marcel Dekker Inc, 1990.
27. Huggins CB, McCarrou HR, Dahlberg AA. Transplantation of tooth germ elements and the experimental heterotopic formation of dentine and enamel. J Exp Med 1934;60:199–210.
28. Kollar EJ, Baird GR. The influence of the dental papilla on the development of tooth shape in embryonic mouse tooth germs. J Embryol Exp Morphol 1969; 21:131–48.
29. Kollar EJ, Baird GR. Tissue interactions in embry-

onic mouse tooth germs. I. Reorganization of the dental epithelium during tooth-germ reconstruction. J Embryol Exp Morphol 1970;24:159–71.
30. Kollar EJ, Baird GR. Tissue interactions in embryonic mouse tooth germs. II. The inductive role of the dental papilla. J Embryol Exp Morphol 1970; 24:173–86.
31. Berkovitz BKB, Moxham B. Development of dentition. Early stages of tooth development. In: Osborn JW, editor. Dental anatomy and embryology. Vol. 1, Book 2. Oxford: Blackwell Scientific Publications, 1981: 253–5.
32. Berkovitz BKB, Shellis P. Development of dentition. Dentine and pulp formation. In: Osborn JW, editor. Dental anatomy and embryology. Vol. 1, Book 2. Oxford: Blackwell Scientific Publications, 1981:260.
33. Bhaskar SN, editor. Orban's oral histology and embryology. 10th ed. St Louis: The C V Mosby Company, 1986:40–4,169.
34. Jensen BL. Kranio-facial morfologi og skeletal modenhet ved Turner's syndrom [thesis]. Copenhagen: University of Copenhagen, 1974.
35. Eberle P, Ammermann M, Wolf H. Zur Variabilität des weiblichen Turner-syndroms. Arch Gynak 1973; 213:202–55.
36. Gorlin RJ, Redman RS, Shapiro BL. Effect of X-chromosome aneuploidy on jaw growth. J Dent Rest 1965;44:269–82.
37. Garn SM, Lewis AB, Walenga AJ. Maximum-confidence values for the human mesiodistal crown dimension of human teeth. Arch Oral Biol 1968; 13:841–4.
38. Garn SM, Lewis AB, Kerewsky RS. The meaning of bilateral asymmetry in the permanent dentition. Angle Orthod 1966;36:55–62.
39. Garn SM, Lewis AB, Swindler DR, Kerewsky RS. Genetic control of sexual dimorphism in tooth size. J Dent Res 1967;46:963–72.

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