



PERGAMON

Archives of Oral Biology 45 (2000) 217–225

Archives
of
Oral
Biology

www.elsevier.com/locate/archoralbio

Crown components of mandibular molar teeth in 45,X females (Turner syndrome)

U. Zilberman^{a,*}, P. Smith^a, L. Alvesalo^b

^aLaboratory of Physical Anthropology, Department of Anatomy and Embryology, Faculty of Dental Medicine, Hebrew University-Hadassah, POB 12272, 91120, Jerusalem, Israel

^bDepartment of Oral Development and Orthodontics, Institute of Dentistry, University of Oulu, Aapistie 3, 90220, Oulu, Finland

Accepted 28 September 1999

Abstract

This study was designed to determine the possible effect of one X-chromosome constitution on components of the human permanent and primary molar teeth. Enamel, dentine, pulp and crown dimensions were measured on radiographs of first and second permanent and second primary mandibular molars of 49 Finnish 45,X females (Turner syndrome), their 46 first-degree male and female relatives and 50 non-related males and females. In permanent first and second molars of the 45,X females, crown width and the dimensions of tooth components were less than those of normal females and males. Reduction in size affected first more than second molars, and in both teeth the enamel was relatively as well as absolutely thinner than in the controls. No differences were found in tooth components between normal relatives and unrelated controls. These data agree with previous studies which have demonstrated that the X chromosome promotes enamel apposition and that both X chromosomes in normal females are active in amelogenesis, while the Y chromosome influences both dentine and enamel growth. The relative reduction in “dentine” or the estimated mesiodistal width of the tooth germ in the 45,X females indicates that their tooth development is affected at an early stage of morphogenesis. Taken together with the results already reported for anterior teeth, the present results suggest that there is an inverse correlation between the duration of crown formation and the severity of size reduction. © 2000 Elsevier Science Ltd. All rights reserved.

Keywords: Turner syndrome; Tooth; Enamel; Dentine

1. Introduction

Turner syndrome is the eponym used to describe the clinical features of females with 45,X chromosome constitution (females with only one X chromosome) and its variants. Its incidence is estimated as 1:3000 live female births. The typical affected newborn presents

with marked dorsal lymphoedema of the hands and feet, and with lymphoedema or loose folds of skin over the posterior aspect of the neck. Characteristic features of girls and women are short stature, webbing of the neck, “shield” chest with broad-spaced nipples, multiple pigmented naevi, short 4th metacarpals, hypoplastic nails, coarctation of the aorta, amenorrhoea, failure of breast development and juvenile external genitalia. The ovaries are replaced by bilateral streaks of fibrous stroma which are usually devoid of developing ova (Berkow, 1977). All permanent teeth in 45,X

* Corresponding author. Tel.: +972-2-6758577; fax: +972-2-6757951.

females are reduced in size, with mesiodistal diameters affected more than buccolingual (Kari et al., 1980; Townsend et al., 1984, 1988). The enamel layer in upper permanent first incisors and canines as measured from radiographs is thinner than that of normal relatives (Alvesalo and Tammissalo, 1981). In permanent upper first molars, decreased basal area, cusp volume, intercuspal distances and sharper cusps have been noted in 45,X females (Mayhall and Alvesalo, 1995).

Garn et al. (1964, 1965) have suggested X-linkage for tooth crown size and dental development, and also proposed Y-chromosome involvement in tooth crown growth. Alvesalo (1971) concluded, from a correlative study of normal relatives, that both the X and Y chromosomes carry genes that affect tooth size, but the influence of the Y-chromosome genes differs from that of X-chromosome genes. Subsequent studies on individuals with sex-chromosome anomalies have further showed that the X-chromosome genes regulate enamel apposition while cellular division demarcated by the dentine–enamel junctions and also enamel growth are influenced by the Y-chromosome genes (Alvesalo, 1985, 1997; Alvesalo and Tammissalo, 1981; Alvesalo et al., 1985, 1987, 1991). Recent discoveries at the molecular level provide additional confirmation for these results on enamel growth. The gene for amelogenin has been sequenced from both sex chromosomes by Nakahori and associates (1991). Its location has been mapped to the short arm of the X chromosome and is now considered to be the source of the defect in X-linked amelogenesis imperfecta (Lench and Winter, 1995). In the Y chromosome the gene for amelogenin is probably located on the long arm, although the short arm has also been suggested as possible location (Lau et al., 1989; Nakahori et al., 1991).

Studies of tooth size in individuals with chromosomal anomalies have indicated that the severity of growth defects observed may be related to their time of development and location in the jaw (Townsend et al., 1988). However, there is little evidence on the extent to which morphogenesis rather than ameloblast function is affected. Crown size is determined both by cellular proliferation as defined by the dentine–enamel junction, and by the thickness of enamel deposited by the ameloblasts (Ten Cate, 1998). An estimate of the relative contribution of these two components on tooth size can be obtained from measurements of dentine and enamel thickness in radiographs (Zilberman et al., 1992). When taken by experienced personnel these provide an easily reproducible, two-dimensional representation of the maximum diameters of the crown, enamel, dentine and pulp cavity, whose accuracy can easily be verified by repeated radiographs (Stroud et al., 1994, 1998; Zilberman et al., 1992). The main problem lies in possible modification of enamel thickness through attrition or dentine thickness through contin-

ued apposition in later life. This can be estimated from the fact that such modifications are correlated with age and function and associated with a decrease in enamel and increase in dentine thickness (Ten Cate, 1998). In the present study, standard bitewing radiographs taken for clinical diagnostic purposes were used to investigate the timing, extent and possible growth defects in the mandibular primary and permanent molars of 45,X females.

2. Materials and methods

2.1. Participants

The study group consisted of 49 Finnish females with 45,X chromosome constitution (group I). The control groups were their 46 first-degree male and female relatives (group II) and 50 male and female unrelated individuals (group III). The diagnoses of 45,X females had been confirmed cytogenetically. All persons in this study were participants in the Kvantti project directed by Lassi Alvesalo from the Institute of Dentistry, University of Oulu.

2.2. Measurements

Crown components of mandibular primary second molars and permanent first and second molars were measured from standardized bitewing radio-

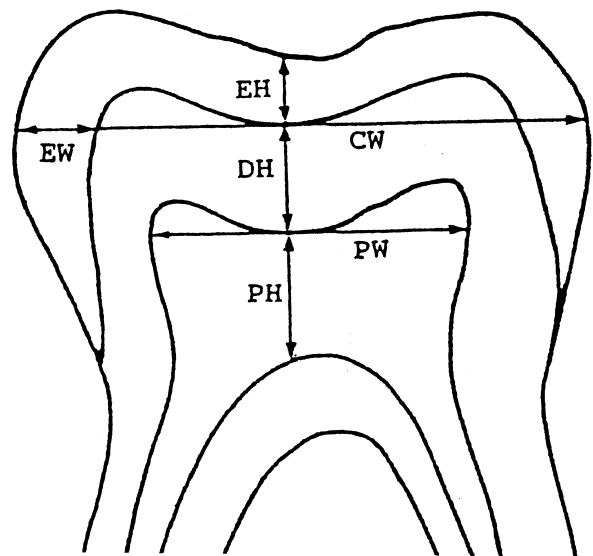


Fig. 1. Measurements of crown components: EH, enamel height; DH, dentine height; PH, pulp height; EW, enamel width; PW, pulp width; CW, crown width.

graphs taken for routine dental evaluations. The measurements were made with a digital caliper on a light table using the method described by Zilberman et al. (1992). Six measurements were taken on each tooth if possible (Fig. 1). Enamel height, dentine height and pulp height were measured on the same line, parallel to the long axis of the tooth, 1 mm mesial to the central fossa. Pulp width was measured perpendicular to the long axis of the tooth on the widest part of the pulp. Enamel width and maximal crown width were measured on the same line, on the widest mesiodistal length of the tooth, perpendicular to the long axis of the tooth. All measurements were expressed also as a ratio of mesiodistal crown width. The teeth examined showed no marked attrition. In order to increase samples sizes, teeth with very small occlusal restorations were also included in the study, but in them only enamel, pulp and crown widths were measured. The same examiner (U.Z.) performed all measurements. The enamel thickness measured from radiographs is in fact the superposition of enamel layers from buccal to lingual. Sperber (1985, p. 444) measured enamel thickness of unworn teeth of Australopithecines from radiographs and stated that

the limitations of accuracy of this indirect form of measurement of a minute dimension are well recognized from variations arising from distortion by magnification to different X-ray angulations casting variable thickness of enamel shadows; but since a standardized procedure of radiography was practiced, comparison of similar derived shadows were felt to be valid

The mesial enamel width is similar to the maximal mesial enamel thickness measured from tooth slices (Zilberman et al., 1992). The occlusal enamel thickness measured from radiographs was in the same range of occlusal enamel dimensions obtained from tooth slices and naturally broken teeth of the same population (Zilberman et al., 1990). Pulp chamber dimensions measured from dental radiographs are in fact the maximal true dimensions of the pulp (Puddhikarant and Rapp, 1983). Alvesalo and associates (Alvesalo and Tammissalo, 1981; Alvesalo et al., 1985, 1987, 1991) measured enamel width and dentine size from radiographs. Despite the limitation of this method, radiographs when carefully taken do provide standardized, easily reproducible, images of the internal structure of the tooth. As such they have long been used for measurement of tooth structure in dentistry and research (Dean, 1985; Smith et al., 1986, 1989; Sperber, 1985; Stroud et al., 1994, 1998; Wood et al., 1988).

2.3. Data analysis

All data were transferred to the WAX computer of the Hebrew University and statistically analysed using the SAS package (1989). The paired Student *t*-test was used to determine the differences between antimere teeth. The two-tailed Student *t*-test was used to determine the differences between 45,X females and their female and male relatives, between 45,X females and non-related females and males, and between the related and non-related groups.

In order to test the reliability of measurements taken from radiographs, seven teeth from the 45,X group and eight teeth from group II were remeasured after 48 h. The range of variation found between measurements of the same tooth was 0.3–8.2% and averaged 4.15%. Considering this variability, it was decided to use a *p*-value of <0.01 to ensure statistical significance of each pair of measurements.

3. Results

Tables 1 and 2 show the mean values of the crown components of the teeth studied and their value calculated as a ratio to crown width in 45,X females (group I), healthy relatives (group II) and unaffected non-related individuals (group III). Case analysis (when only one tooth was chosen per individual) and tooth analysis showed similar results, thus tooth analysis was used in order to maximize sample sizes. Because some measurements could not be taken on all teeth, the number of observations for different components of the teeth varies.

Crown width in first permanent molars of 45,X females was significantly smaller than that of males and females in groups II and III ($p < 0.01$), while values obtained for external crown dimensions in the related and non-related control groups were similar. The first permanent mandibular molars of the 45,X group had very thin enamel, both on the occlusal table (enamel height) and on the mesial surface (enamel width), compared to males or females in groups II and III, and the differences were statistically significant ($p < 0.01$). Dentine thickness in the 45,X group was similar to that of females in group II and significantly less than that of males from group II and III and females from group III. Pulp height was similar in all groups, but pulp width in 45,X females was significantly less than that of males and females in groups II and III. When expressed as a ratio to crown width, occlusal and mesial enamel in 45,X females was significantly thinner than that of normal females and males, but the ratios of dentine height and pulp dimensions were similar in all groups.

Table 1

Tooth components in mandibular molars of 45,X females^a. EH, enamel height; DH, dentine height; PH, pulp height; EW, enamel width; PW, pulp width; CW, crown width

	dm2															
	M1						M2									
	45,X		RELATIVES		CONTROL		45,X		RELATIVES		CONTROL					
Mean	N	F	M	F	M	F	M	F	M	F	M					
EH	1.34 ^{abcd}	23	1.7	1.55	1.8	1.61	1.47 ^{abcd}	1.84	1.77	1.91	1.91	1.15	1.21	0.94	1.11	1.15
SD	0.14		0.24	0.16	0.2	0.19	0.18	0.21	0.16	0.17	0.24	0.25	0.13	0.15	0.1	2
Min	1.11		1.4	1.24	1.44	1.36	1.16	1.51	1.53	1.64	1.64	0.74	1.05	0.73	0.96	
Max	1.7		2.02	1.78	2.04	1.78	1.82	2.2	2.02	2.12	2.16	1.39	1.38	1.1	1.25	
DH	2.85 ^{bcd}	23	2.84 ^b	3.6	3.4	3.7	2.91 ^{abcd}	3.31	3.47	3.25	3.95	2.43	2.55	3.12	2.87	2.95
SD	0.45		0.23	0.67	0.3	0.47	0.34	0.4	0.42	0.47	0.35	0.24	0.39	0.34	0.42	2
Min	2.14		2.52	2.36	2.89	3.12	2.3	2.7	2.68	2.43	2.43	2.02	2.02	2.68	2.1	
Max	3.87		3.14	4.51	3.87	4.5	3.46	4.18	4.04	3.8	4.25	2.63	2.9	3.61	3.57	
PH	1.24	22	1.28	1.43	1.57	1.09	1.43 ^a	2.18	1.68	2.02	2.12	0.94	1.59	0.86	0.81	0.86
SD	0.55		0.62	0.59	0.16	0.47	0.48	0.48	0.61	0.84	0.93	0.14	0.57	0.31	0.12	2
Min	0.58		0.53	0.53	0.92	0.63	0.67	1.31	0.85	0.99	0.72	0.8	0.96	0.46	0.63	
Max	2.36		2.54	2.24	2.12	1.88	2.67	3.35	2.61	3.22	3.13	1.1	2.21	1.17	1	
EW	1.15 ^{abcd}	77	1.5	1.45	1.49	1.48	1.16 ^{abcd}	1.5	1.44	1.49	1.44	0.84	1.01	1	1	0.89
SD	0.15		0.18	0.17	0.17	0.16	0.15	0.15	0.23	0.21	0.14	0.09	0.13	0.08	0.13	2
Min	0.87		1.12	1.18	1.15	1.25	0.84	1.17	0.99	1.16	1.22	0.72	0.87	0.94	0.8	
Max	1.46		1.97	1.75	1.74	1.87	1.53	1.8	1.82	2.05	1.74	0.99	1.2	1.13	1.18	
PW	4.14 ^{abcd}	77	4.47	4.55	4.38	4.52	4.02	4.1	4.31	4	4.16	5.04	4.68	5.02	5.01	5.17
SD	0.37		0.5	0.59	0.47	0.53	0.4	0.37	0.39	0.46	0.39	0.52	0.14	0.47	0.25	4
Min	3.29		3.65	3.76	3.57	3.54	2.98	3.53	3.61	3.32	3.44	4.24	4.44	4.69	4.72	4.57
Max	4.98		5.78	5.64	5.17	5.39	4.84	5.32	4.81	5.06	4.8	5.86	4.77	5.83	5.43	5.5
CW	10.28 ^{abcd}	77	11.48	11.55	11.43	11.62	10.34 ^{abcd}	11.02	11.45	11.09	11.48	9.7	9.99	10.29	10.42	10.76
SD	0.46		0.58	0.69	0.59	0.55	0.53	0.36	0.77	0.81	0.69	0.58	0.84	0.71	0.54	4
Min	9.22		10.51	10.05	10.58	10.66	9.27	10.46	10.06	9.38	10.38	9.07	9.3	9.56	9.73	10.44
Max	11.27		12.74	12.8	12.84	12.57	11.2	11.65	12.75	12.62	12.6	10.44	11.06	11.28	11.1	10.92

^a $P < 0.01$ compared to relatives — females.^b $P < 0.01$ compared to relatives — males.^c $P < 0.01$ compared to control — females.^d $P < 0.01$ compared to control — males.

Table 2
Ratios of tooth components in mandibular molars of 45,X females^a. EH, enamel height; CW, crown width; DH, dentine height; PH, pulp height; EW, enamel width; PW, pulp width.

	M1												M2												dm2			
	45,X				RELATIVES				CONTROL				45,X				RELATIVES				CONTROL				RELATIVES		CONTROL	
	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M
EH/CW	Mean	0.13 ^{ac}	0.15	0.13	0.16	0.14	0.14	0.15 ^{ac}	0.17	0.14	0.18	0.16	0.12	0.12	0.09	0.11	0.11	0.11	0.11	0.11	0.11	0.11	0.11	0.11	0.11	0.11	0.11	0.11
	N	22	6	12	8	6	6	21	16	5	5	5	6	6	5	8	8	5	5	5	5	5	5	8	8	8	8	2
	SD	0.015	0.02	0.02	0.016	0.016	0.016	0.018	0.019	0.012	0.02	0.009	0.08	0.08	0.02	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
	Min	0.1	0.13	0.1	0.12	0.11	0.11	0.11	0.13	0.13	0.17	0.15	0.15	0.15	0.11	0.07	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09
	Max	0.16	0.19	0.17	0.18	0.15	0.15	0.18	0.2	0.16	0.21	0.18	0.18	0.18	0.14	0.12	0.13	0.13	0.14	0.14	0.14	0.14	0.14	0.13	0.13	0.13	0.13	0.27
DH/CW	Mean	0.28	0.25	0.31	0.3	0.32	0.29 ^d	0.3	0.3	0.3	0.32	0.36	0.25	0.25	0.3	0.27	0.27	0.27	0.27	0.27	0.27	0.27	0.27	0.27	0.27	0.27	0.27	
	N	22	6	12	8	7	21	16	16	5	5	5	6	6	5	8	8	5	5	5	5	5	8	8	8	8	2	
	SD	0.05	0.02	0.06	0.031	0.049	0.03	0.04	0.04	0.04	0.017	0.02	0.03	0.03	0.05	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	0.03	
	Min	0.2	0.24	0.23	0.25	0.26	0.23	0.23	0.24	0.28	0.25	0.34	0.22	0.22	0.24	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21	0.21
	Max	0.37	0.29	0.4	0.34	0.39	0.35	0.38	0.38	0.31	0.41	0.38	0.29	0.3	0.36	0.33	0.33	0.33	0.33	0.33	0.33	0.33	0.33	0.33	0.33	0.33	0.33	0.33
PCH/CW	Mean	0.12	0.11	0.13	0.14	0.09	0.14 ^a	0.2	0.2	0.16	0.19	0.19	0.1	0.16	0.08	0.08	0.08	0.08	0.08	0.08	0.08	0.08	0.08	0.08	0.08	0.08	0.08	
	N	22	12	15	8	7	20	19	19	6	5	5	6	6	5	8	8	5	5	5	5	5	8	8	8	8	2	
	SD	0.06	0.05	0.06	0.04	0.039	0.04	0.04	0.04	0.07	0.1	0.09	0.02	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04	
	Min	0.06	0.06	0.05	0.08	0.05	0.06	0.06	0.12	0.07	0.09	0.06	0.08	0.1	0.04	0.06	0.06	0.06	0.06	0.06	0.06	0.06	0.06	0.06	0.06	0.06	0.06	
	Max	0.24	0.2	0.21	0.19	0.16	0.23	0.23	0.31	0.23	0.34	0.3	0.12	0.21	0.12	0.12	0.12	0.12	0.12	0.12	0.12	0.12	0.12	0.12	0.12	0.12	0.12	
EW/CW	Mean	0.11 ^{abcd}	0.13	0.13	0.13	0.13	0.11 ^{abcd}	0.14	0.14	0.13	0.13	0.13	0.09	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.08	
	N	77	34	31	22	19	49	27	27	12	18	20	7	7	5	9	9	5	5	5	5	5	9	9	9	4		
	SD	0.01	0.013	0.013	0.014	0.011	0.015	0.015	0.015	0.015	0.016	0.015	0.07	0.07	0.003	0.01	0.01	0.015	0.015	0.015	0.015	0.015	0.015	0.015	0.015	0.009		
	Min	0.08	0.1	0.1	0.11	0.11	0.08	0.11	0.11	0.11	0.11	0.1	0.08	0.08	0.09	0.08	0.08	0.09	0.09	0.09	0.09	0.09	0.09	0.08	0.08	0.08	0.07	
	Max	0.14	0.16	0.15	0.16	0.15	0.14	0.14	0.17	0.15	0.17	0.15	0.1	0.1	0.13	0.11	0.11	0.13	0.13	0.13	0.13	0.13	0.11	0.11	0.11	0.11	0.09	
PW/CW	Mean	0.4	0.39	0.39	0.39	0.39	0.39	0.39	0.37	0.38	0.36	0.36	0.52	0.47	0.49	0.48	0.48	0.48	0.48	0.48	0.48	0.48	0.48	0.48	0.48	0.48		
	N	77	33	31	22	18	48	27	27	12	18	19	7	5	5	9	9	5	5	5	5	5	9	9	9	4		
	SD	0.03	0.03	0.04	0.03	0.04	0.03	0.03	0.03	0.03	0.04	0.02	0.03	0.04	0.02	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.03	0.03	0.03	0.03		
	Min	0.31	0.33	0.33	0.33	0.31	0.31	0.31	0.31	0.31	0.31	0.33	0.47	0.43	0.46	0.44	0.44	0.43	0.43	0.43	0.43	0.43	0.44	0.44	0.44	0.44	0.44	
	Max	0.47	0.46	0.46	0.46	0.46	0.46	0.45	0.43	0.46	0.46	0.43	0.57	0.52	0.52	0.52	0.52	0.52	0.52	0.52	0.52	0.52	0.53	0.53	0.53	0.53	0.5	

^a P < 0.01 compared to relatives — females.
^b P < 0.01 compared to relatives — males.
^c P < 0.01 compared to control — females.
^d P < 0.01 compared to control — males.

In the second permanent molars, means for crown width in the 45,X females were larger than those of the first permanent molar, but still significantly less than crown widths in groups II and III. This despite the fact that, in both these groups, crown width in the second permanent molar was less than that of first permanent molar. Enamel height and pulp height of second permanent molars in groups II and III were greater than that of the first permanent molar.

In the 45,X females, enamel thickness, both occlusally and mesially, in the second permanent molars, was also significantly less than that of males and females from groups II and III. Dentine height in the 45,X group was also significantly reduced. Means for pulp height and width were smaller in the 45,X group than in controls, but the differences were not statistically significant. The relative occlusal enamel thickness of the 45,X group (the ratio of enamel height to crown width) was similar to that of the control males and significantly less than that of control females ($p < 0.01$). Mesial enamel ratio was reduced in the 45,X group and the differences from those of males and females in groups II and III were statistically significant ($p < 0.01$).

As only a few primary second molars were present in all groups, statistical tests were not applied to those data. It appears that differences between the groups were smaller than in the permanent teeth. Tooth size in the 45,X group was less than that of groups II and III as was mesial enamel thickness and dentine thick-

ness. However, pulp dimensions and occlusal enamel thickness were similar in all groups and tooth component ratios in the 45,X females were similar to those of females and males in groups II and III.

The permanent first molar appeared to be the tooth most affected in 45,X females. It was smaller than the second permanent molar, whereas in both sexes of the control groups (groups II and III) it was larger than the second molar. Enamel thickness on the occlusal table of the M2 was thicker than that of M1 in all groups (I, II and III), but enamel thickness on the mesial surface was similar in both molars. Dentine height was similar in both molars, but pulp height in the M2 was greater than that of M1, although pulp width was reduced in all three groups.

In order to evaluate the relative severity of the dental changes in 45,X females, the percentage reduction in total tooth size and tooth components was calculated incorporating the data from Alvesalo and Tammissalo (1981) for anterior teeth, as follows (Table 3):

$$\begin{aligned} \text{Tooth size reduction} &= (\text{mean crown width in 45, X} \\ &\quad - \text{mean crown width in controls}) \\ &\quad \times 100 / \text{mean crown width in controls.} \end{aligned}$$

In 45,X females, tooth size reduction was least in the upper permanent canines (2.3% compared to control females and 7.1% compared to control males), followed by the upper central incisors (4 and 9.2%) and

Table 3

The percentage reduction of tooth crown size and enamel and dentine thickness in 45,X females compared to normal population^a

Tooth	Relatives		Control		Relatives		Control	
	Males (%)	Females (%)	Males (%)	Females (%)	Males (%)	Females (%)	Males (%)	Females (%)
(a) Reduction in crown mesio-distal diameter					(b) Reduction at DEJ: mesio-distal			
Upper I1	7.0	4.0	9.2	4.9	3.8	0	7.6	1.55
Upper C	5.3%	2.4	7.1	2.3	3.0	0	6.3	0
Lower M1	11.0	10.5	11.5	10.1	7.8	5.8	7.8	6.7
Lower M2	9.2	6.2	10.0	6.8	6.6	0	6.7	0
(c) Reduction in enamel thickness								
1. Mesial and distal surfaces								
Upper I1	20.4	21.2	16.6	19.0				
Upper C	11.4	14.0	10.1	12.5				
2. Enamel height and width								
Lower M1								
(EH)	13.5	21.2	16.8	23.6				
(EW)	20.7	23.4	22.3	22.8				
Lower M2								
(EH)	17.0	20.1	23.1	23.1				
(EW)	19.5	22.7	19.5	22.1				

^a The results for upper I1 and C were calculated from Alvesalo and Tammissalo (1981), where enamel thickness = mesial plus distal enamel and dentine thickness = mesio-distal dentine width.

second mandibular molars (6.2 and 10%), and was greatest in the first mandibular molars (10.1 and 11.5%). For all teeth the size reduction in 45,X teeth was greater when compared to normal males than to normal females. The percentage reduction in enamel thickness was minimal in upper canines and showed similar values on the mesial aspect of upper incisors and mandibular molars. The percentage reductions in dentine thickness in the upper incisors and canines were similar to each other and smaller than in mandibular molars. The mesiodistal reduction of the first permanent mandibular molars was also compared to the results published for the maxillary first molars of 45,XO females measured from dental casts (Mayhall and Alvesalo, 1992). The reduction in the upper permanent M1 of 45,X females was 5.8% compared to normal females and 9.2% compared to normal males, similar to the reduction observed in second permanent mandibular molars, but less than that observed in the first mandibular molars.

4. Discussion

We show that tooth crown size and its components in Finnish 45,X females differ both from those of first-degree unaffected relatives and from the general population. We also show that tooth size and components in unaffected relatives of 45,X females are no different from those of the general population sampled. Our results for the controls conform to those carried out on other populations of European origin using both ground sections (Macho and Berner, 1993; Shillinburg and Grace, 1973) and radiographs (Stroud et al., 1998). The results showed that in normal modern small-toothed populations, enamel thickness in second molars is greater than that of first molars, despite the size difference between the two teeth. In order to maximize sample size, no correction was made for age or enamel attrition in our study. If, however, age or attrition were important factors affecting enamel thickness, then this should be reflected in increased dentine height and reduced pulp dimensions in affected individuals (Ten Cate, 1998). This was not the case in our study, where the 45,X females with thinner enamel also showed reduced dentine thickness and pulp dimensions. Moreover, the estimated width of the molars at the dentine–enamel junction, calculated as crown width—twice enamel width, is also significantly less in the 45,X group. These results indicate that in females with 45,X constitution both cell division and function are affected, resulting in smaller tooth germs and reduced enamel. They agree with the results from previous studies, which demonstrate a reduction in crown size, enamel and dentine in 45,X females (Alvesalo and

Tammissalo, 1981; Townsend et al., 1988). The insult to enamel growth and cell division also provides a developmental basis for the observed change in cuspal shape in 45,X females reported by Mayhall and Alvesalo (1992).

We also found that the first molars were affected more than the second molars in 45,X females; this may be related to differences at the onset and duration of crown formation of the two teeth, and/or to their position in the developing jaws. Comparison of our findings with those of an earlier study on tooth components in anterior teeth (Alvesalo and Tammissalo, 1981) suggests that the main factor may be the differences in timing of crown formation. The percentage difference calculated between tooth components in 45,X females and that of non-affected relatives and non-related controls is greater for the M1 and central incisor and least in the M2 and canine. This indicates that the influence of the “missing” X chromosome may be more pronounced in those permanent teeth that develop earlier and faster. The differences observed between the incisors and first permanent molars may reflect the difference in the duration of crown formation of these two teeth. Despite their overlapping developmental periods, mineralization of the first lower molar crown is completed more rapidly than that of the central incisor. Mean values quoted are from birth (mineralization begins) up to 2.6–2.7 years (enamel completed) for M1 and 14 weeks in utero to 3.3–3.4 years for the central incisor, that is 14 months’ difference. (Moorrees et al., 1963; Kraus and Jordan, 1965; Ten Cate, 1998). There may be other explanations, such as a greater susceptibility of teeth in the lower jaw to growth insults, because of their more localized blood supply, or lack of space in the small underdeveloped mandible.

The mesiodistal measurements on the present radiographs of molar teeth and on radiographs of anterior teeth (Alvesalo and Tammissalo, 1981) show a similar pattern to those obtained from dental casts and reported by Alvesalo (1985). Townsend and associates (1988) showed that the Z-score of permanent canines in Turner syndrome was similar to that of the normal population, whilst the mandibular first permanent molars showed a high negative Z-score and were the teeth most affected. We do not have measurements on enamel thickness in upper permanent molars of 45,X females, but from former studies we know that lower first permanent mandibular molars show a greater reduction in total crown size than the upper permanent molars: a 10.1% reduction compared to normal females and a 11.5% reduction compared to normal males for lower first permanent molars examined here, compared to a 5.8 and 9.2% reduction, respectively, in upper first permanent molars (Mayhall and Alvesalo, 1995). This provides additional support for a pos-

itional as well as temporal effect on tooth development in 45,X females. The relative “immunity” of the permanent canine crown size might be due to its long period of development: mineralization begins at 20 weeks in utero and enamel is completed at 4.1–4.9 years (Ten Cate, 1998), during which there may be a compensatory activity of the only active X chromosome. It may be possible that when only one X chromosome affects enamel apposition, the magnitude of its effect is progressive and time related. In teeth with prolonged enamel formation the only X chromosome may cause a higher rate of enamel apposition per unit time during the later period of apposition. This possibility could be explored using histological slices of teeth from affected females, and the extension and apposition rate of ameloblasts between fast and slow crown-developing teeth should be compared. The most affected tooth in terms of mesiodistal crown width and relative thickness of enamel, as previously stated, is the first permanent molar, the tooth with the shortest period of crown formation, and the least affected is the upper canine.

The differences observed between the first and second permanent mandibular molars in groups II and III, i.e., thicker occlusal enamel on second molars, is in agreement with the findings of Macho and Berner (1993) from ground sections of upper permanent molars.

The influence of the “missing” X chromosome on components of the primary second molars appears to be minimal, a phenomenon observed in other chromosomal abnormalities such as Down’s syndrome (Townsend, 1983). However the results from the deciduous dentition should be considered with caution because of the small sample size.

Our results provide further evidence to show that in 45,X females morphogenesis and enamel apposition are affected in both posterior and anterior permanent teeth. It seems that earlier developing teeth with faster developing crowns are more affected and that the least affected tooth is the upper canine.

Acknowledgements

This study was supported by the University of Turku Foundation and the Academy of Finland.

References

- Alvesalo, L., 1971. The influence of sex chromosome genes on tooth size in man. *Proceedings of Finnish Dental Society* 67, 3–54.
- Alvesalo, L., 1985. Dental growth in 47,XXY males and in conditions with other sex-chromosome anomalies. In: Sandberg, A.A. (Ed.), *The Y Chromosome, Part B: Clinical Aspects of Y Chromosome Abnormalities*. Alan R. Liss, Inc, New York, pp. 277–300.
- Alvesalo, L., 1997. Sex chromosomes and human growth. A dental approach. *Human Genetics* 101, 1–5.
- Alvesalo, L., Tammisalo, E., 1981. Enamel thickness in 45,X females’ permanent teeth. *American Journal of Human Genetics* 33, 464–469.
- Alvesalo, L., Tammisalo, E., Hakola, P., 1985. Enamel thickness in 47,XYY males’ permanent teeth. *Annals of Human Biology* 12, 421–427.
- Alvesalo, L., Tammisalo, E., Therman, E., 1987. 47,XXX females, sex chromosomes, and tooth crown structure. *Human Genetics* 77, 345–348.
- Alvesalo, L., Tammisalo, E., Townsend, G.C., 1991. Upper central incisor and canine tooth crown size in 47,XXY males. *Journal of Dental Research* 70, 1057–1060.
- Berkow, R., 1977. *The Merck Manual of Diagnostic and Therapy*, 13th ed. Merck & Co, Inc, Rahway, NJ, p. 1107.
- Dean, M.C., 1985. Variation in the developing root cone angle of the permanent mandibular teeth of modern man and certain fossil hominids. *American Journal of Physical Anthropology* 68, 233–238.
- Garn, S.M., Lewis, A.B., Kerewsky, R.S., 1964. Sex difference in tooth size. *Journal of Dental Research* 43, 306.
- Garn, S.M., Lewis, A.B., Kerewsky, R.S., 1965. X-linked inheritance of tooth size. *Journal of Dental Research* 44, 439–441.
- Kari, M., Alvesalo, L., Manninen, K., 1980. Sizes of deciduous teeth in 45,X females. *Journal of Dental Research* 59, 1382–1385.
- Kraus, B.S., Jordan, R.E., 1965. *The Human Dentition Before Birth*. Lea & Febiger, Philadelphia, p. 109, 117.
- Lau, E.C., Mohandas, T.K., Shapiro, L.J., Slavkin, H.C., Snead, M.L., 1989. Human and mouse amelogenin gene loci are on the sex chromosomes. *Genomics* 4, 162–168.
- Lench, N.J., Winter, G.B., 1995. Characterisation of molecular defects in X-linked Amelogenesis Imperfecta (AIH1). *Human Mutation* 5, 251–259.
- Macho, G.A., Berner, M.E., 1993. Enamel thickness of human maxillary molars reconsidered. *American Journal of Physical Anthropology* 92, 189–200.
- Mayhall, J.T., Alvesalo, L., 1992. Dental morphology of 45,XO human females: molar cusp area, volume, shape and linear measurements. *Archives of Oral Biology* 37, 1039–1043.
- Mayhall, J.T., Alvesalo, L., 1995. The effect of the sex chromosomes on molar morphology. In: Moggi-Cecchi, J. (Ed.), *Aspects of Dental Biology: Paleontology, Anthropology and Evolution*. International Institute for the Study of Man, Florence, pp. 69–75.
- Moorrees, F.A., Fanning, E.A., Hunt, E.E., 1963. Age variation of formation stages for ten permanent teeth. *Journal of Dental Research* 42, 1490–1502.
- Nakahori, Y., Takenaka, O., Nakagome, Y., 1991. A human X–Y homologous region encodes “Amelogenin”. *Genomics* 9, 264–269.
- Puddhikarant, P., Rapp, R., 1983. Radiographic anatomy of pulpal chambers of primary molars. *Pediatric Dentistry* 5, 25–29.

- SAS Institute Inc, 1989. SAS-Stat Users Guide, Version 6, 4th ed. Cary, NC.
- Shillenburg, H.T., Grace, C.S., 1973. Thickness of enamel and dentine. *Journal of Southern California Dental Association* 41, 33–52.
- Smith, P., Wax, Y., Adler, F., Silberman, U., Heinic, G., 1986. Post Pleistocene changes in tooth, root and jaw relationship. *American Journal of Physical Anthropology* 70, 339–348.
- Smith, P., Wax, Y., Adler, F., 1989. Population variation in tooth, jaw and root size: A radiographic study of two populations in a high-altitude environment. *American Journal of Physical Anthropology* 79, 197–206.
- Sperber, G.H., 1985. Comparative dental enamel thickness: a radiodontological study. In: Tobias, P.V. (Ed.), *Hominid Evolution: Past, Present and Future*. Alan R. Liss Inc, New York, pp. 443–454.
- Stroud, J.L., Buschang, P.H., Goaz, P.W., 1994. Sexual dimorphism in mesiodistal dentine and enamel thickness. *Dentomaxillofacial Radiology* 23, 169–171.
- Stroud, J.L., English, J., Buschang, P.H., 1998. Enamel thickness of the posterior dentition: its implications for non extraction treatment. *The Angle Orthodontist* 68, 141–146.
- Ten Cate, 1998. *Oral Histology: Developmental Structure and Function*, 5th ed. The C.V. Mosby Co, St Louis.
- Townsend, G., 1983. Tooth size in children and adults with trisomy 21 “Down’s Syndrome”. *Archives of Oral Biology* 28, 159–166.
- Townsend, G., Jensen, B.L., Alvesalo, L., 1984. Reduced teeth in 45,X (Turner syndrome) females. *American Journal of Physical Anthropology* 65, 367–371.
- Townsend, G., Alvesalo, L., Jensen, B., Kari, M. 1988. Patterns of tooth size in human Chromosomal aneuploidies. Russell, D.E., Santoro, J.P., Sigogneau-Russell, D. (Eds.) *Teeth Revisited: Proceedings of the VIIth International Symposium on Dental Morphology, Paris 1986. Mem. Mus. Natn. Hist. Nat. Paris, (serie C) 53: 25–45.*
- Wood, B.A., Abbott, S.A., Uytterschaut, H., 1988. Analysis of the dental morphology of Plio–Pleistocene hominids. IV. Mandibular postcanine root morphology. *Journal of Anatomy* 156, 107–139.
- Zilberman, U., Smith, P., Sperber, G.H., 1990. Components of Australopithecine teeth. A radiographic study. *Human Evolution* 5, 515–529.
- Zilberman, U., Skinner, M., Smith, P., 1992. Tooth components of mandibular deciduous molars of *Homo sapiens sapiens* and *Homo sapiens neanderthalensis*: a radiographic study. *American Journal of Physical Anthropology* 87, 245–254.