Oral Diseases (2008) 14, 158–162. doi:10.1111/j.1601-0825.2007.01366.x

http://www.blackwellmunksgaard.com

ORIGINAL ARTICLE

The frequency of occurrence of abnormal frenal attachment of lips and enamel defects in Turner syndrome

A Kusiak[1], J Sadlak-Nowicka[2], J Limon[3], B Kochańska[1]

Departments of [1]Conservative Dentistry, [2]Periodontology and Oral Mucosa Diseases, and [3]Biology and Genetics, Medical University of Gdańsk, Gdańsk, Poland

OBJECTIVE: The aim of the work was to register the frequency of occurrence of abnormal frenal attachment of lips and enamel defects and find the correlation between these anomalies and three types of Turner syndrome.

MATERIALS AND METHODS: Fifty patients (aged 20–40 years) were clinically and cytogenetically diagnosed and divided into three groups, according to karyotype: 45,X (17 cases), with structural aberrations of chromosome X (12 cases) and with mosaic karyotype (21 cases). The control group consisted of 51 healthy woman aged 21–40 years. Subjects were screened for developmental anomalies in the labial frenula and enamel defects in three groups of Turner syndrome.

RESULTS: Some significant anomalies of soft and hard tissues were found in studied patients: abnormal frenal attachments (42% of cases), enamel opacities (58% of cases) and enamel hypoplasia (38% of cases). Differences in the occurrence of these anomaly in all group with Turner syndrome in comparison with the control group were significantly different. Enamel defects were prevalent in the patients with karyotype 45,X and patients with structural aberrations of chromosome X in comparison with the mosaic karyotype.

CONCLUSION: The results of the present study have shown, that abnormal attachment of lips and enamel defects were more frequent in Turner syndrome patients than in the control group. Enamel defects were correlated with the karyotypes of Turner syndrome and abnormal attachment of lips was not correlated with the karyotypes of Turner syndrome.

Oral Diseases (2008) **14**, 158–162

Correspondence: Aida Kusiak, DDS, PhD, Department of Conservative Dentistry, Medical University of Gdańsk, ul. Orzeszkowej 18, 80 - 208 Gdańsk, Poland. Tel: +48 58 349 21 23, Fax: +48 58 349 21 02, E-mail: akusiak@amg.gda.pl
Received 9 March 2006; revised 27 April 2006, 13 July 2006, 21 September 2006, 5 December 2006; accepted 12 Dcember 2006

Keywords: Turner syndrome; genetic diseases; dental anomalies

Introduction

Turner syndrome is a sex chromosomal disorder associated with a female phenotype. The pathophysiology of Turner syndrome is not understood. Many attempts have been made to correlate the type of X chromosome anomaly such as totally missing chromosome X (45X), aberrations of chromosome X (partial deletions of short and long arms, isochromosomes) and various mosaicisms in group of Turner syndrome individuals with their clinical features (Kusiak *et al*, 2000, 2005). The classical abnormalities of Turner syndrome include many somatic anomalies, such as short stature, infantile external genitalia, webbed neck, cubitus valgus, low hairline, shield-like chest, anomalies in the structure of some internal organs and others (Turner, 1938; Horowitz and Morishima, 1974; Palmer and Reichmann, 1976; Arulanantham *et al*, 1980; Jaspers and Witkop, 1980; Goldman *et al*, 1982; Hall and Gilchrist, 1990; Lippe, 1991; Pelz *et al*, 1991; Temtamy *et al*, 1992; Robinson and de la Chapelle, 1996; Lopez *et al*, 2002). Some oral abnormalities were also observed, such as malocclusion (Harju *et al*, 1989; Laine *et al*, 1992; Szilagyi *et al*, 2000), early development of permanent teeth (Filipsson *et al*, 1965), high-arched palate (Horowitz and Morishima, 1974), small teeth (Townsend *et al*, 1984; Varrela *et al*, 1988; Mayhall *et al*, 1991; Mayhall and Alvesalo, 1992; Midtbo and Halse, 1994a,b; Townsend and Alvesalo, 1995; Kusiak *et al*, 2000; Szilagyi *et al*, 2000; Zilberman *et al*, 2000), crown hypoplasia (Lopez *et al*, 2002), abnormalities in intercuspal distance (Lopez *et al*, 2002) and abnormality of root morphology of mandibule were also observed (Varrela, 1990, 1992; Lopez *et al*, 2002; Kusiak *et al*, 2005).

The aim of the work was to register the frequency of occurrence of abnormal frenal attachment of lips and

enamel defects and find the correlation between these anomalies with the karyotypes of Turner syndrome.

Material and methods

Fifty patients, aged 20–40 years (27.4 ± 4.2) with Turner syndrome were studied. The diagnoses of both Turner syndrome and karyotype were made at the Department of Biology and Genetics, Medical University of Gdańsk. Karyotype was determined by chromosome analysis of peripheral lymphocytes. Patients were divided into three groups according to their karyotypes:

- 45,X ($n = 17$);
- aberrations of chromosome X ($n = 12$): 46,X,del(Xq) ($n = 4$), 46,X,i(Xq) ($n = 5$), 46,X,inv(Xp) ($n = 2$), 46,X,r(X) ($n = 1$) and
- mosaic karyotype ($n = 21$): mos45,X/46,XX ($n = 4$); mos45,X/46,XY/47,XYY ($n = 4$); mos45,X/46,Xi(Xq) ($n = 3$); mos45,X/46,XX/47,XXX ($n = 2$); mos45,X/46,X,t(XX) ($n = 2$); mos46,XYinv(Yp)/45,Xinv(Xq) ($n = 1$); mos45,X/46,XinfY ($n = 1$); mos46,X(Xq)/45,X ($n = 1$); mos46,X,del(Xp)/47,Xdel(Xp)2X ($n = 1$); mos47,XXX/45,X ($n = 1$); mos45,X/46,X,r(Y) ($n = 1$).

The control group consisted of 51 healthy woman aged 21–40 years (31.5 ± 5.2) who underwent dental treatment at the Department of Conservative Dentistry, Medical University of Gdańsk, Poland. Ethical aspects of the research followed the Word Medical Association Declaration of Helsinki.

On evaluating the occurrence of soft tissues anomalies, attention was paid to the frenal attachment of lower and upper lips. The lip's frenula were evaluated on the basis of Placek's classification (Mirko *et al*, 1974):

- Type 1 Frenula attached to alveolar mucosa ('mucosa-like')
- Type 2 Frenula attached to attached gingiva ('gingiva-like')
- Type 3 Frenula attached to interdental papilla ('papilla-like')
- Type 4 High frenula attachment ('penetrating papilla-like')

The teeth have been cleaned from debris, but not dried. Normal dental light providing illumination was used during the examinations of the teeth and one examiner carried out the examinations. All patients with Turner syndrome and the control group lived in an urban area north of Poland where the natural water contains 0.2 ppm fluor. None of them had fluoration treatment during their childhood nor adolescence. The type, number and location of defects were classified using the Developmental Defects of Enamel (DDE Index) (FDI Commission on Oral Health Research and Epidemiology, 1982). The DDE Index included three types of defects: opacities (white/cream, yellow/brown), hypoplasia (pits, horizontal grooves, vertical grooves, missing enamel) and discoloured enamel. Anomalies can occur as single or multiple well-demarcated areas or as diffuse patches or fine lines. Defect localisation was observed on buccal, lingual and occlusal surfaces. For documentational purposes, images of the cases were taken by DSC-F717 digital camera (Sony Corporation, Johor Bahru, Malaysia).

The results were statistically analysed by chi-squared test with Yates's adjustment, by means of which differences in the frequency of occurrence of the examined parameters in the patients with Turner syndrome and in the control group were compared.

Results

Numerous abnormalities within abnormal frenal attachment of lips and enamel defects in Turner syndrome patients were found.

Table 1 shows the frequencies of the abnormal frenal attachment and enamel defects in Turner syndrome patients with various aberrations of chromosome X and the control group. Nineteen patients had labial frenula with abnormal attachment as well as enamel defects; 31 patients had only one anomaly (two patients had abnormal attachment and 29 had enamel defects). No cases of Turner syndrome without anomaly were detected. Analysis of the oral soft tissues showed that abnormal frenal attachments had evident influence on the aetiopathogenesis recession of gingiva and gingivitis (Figure 1). Abnormal frenal attachment (lower and upper lips) was found in 42% of the patients with

Table 1 Anomalies of labial frenula and enamel defects in Turner syndrome patients

Karyotype	*Labial frenula with abnormal attachment N* (%)	*Enamel opacities N* (%)	*Enamel hypoplasia N* (%)
45,X ($n = 17$)	7 (41)[a]	12 (71)[f]	8 (47)[k]
Structural aberrations of chromosome X ($n = 12$)	5 (42)[b]	8 (67)[g]	6 (50)[l]
Mosaics ($n = 21$)	9 (43)[c]	9 (43)[h]	5 (23)[m]
Total ($n = 50$)	21 (42)[d]	29 (58)[i]	19 (38)[n]
Control group ($n = 51$)	4 (8)[e]	10 (20)[j]	1 (2)[z]

n, number of patients; *N*, number of patients with anomaly.
$P < 0.05$ a-e, b-e, c-e, d-e, f-j, g-j, h-j, i-j, f-h, g-h, k-z, l-z, m-z, k-m, l-m, n-z.

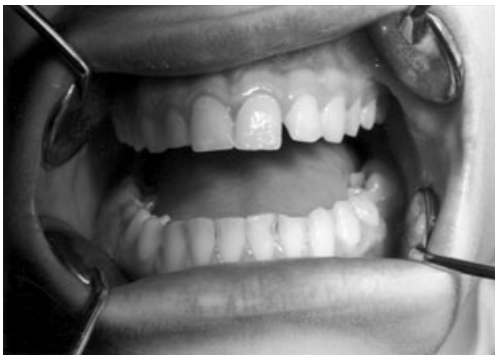

Figure 1 Patient (H.U. age 40) with Turner syndrome with structural aberration of chromosome X labial frenula with attachment type 4, recession of gingiva- teeth 11, 23, enamel opacities teeth 13, 12, 11, 21, 22, 23, 24, 25, 26, 35, 34, 33,32, 21, 41, 42, 43, non-carious lesions teeth 13, 11, 23, 24, 25, 35, 34, 33, 32

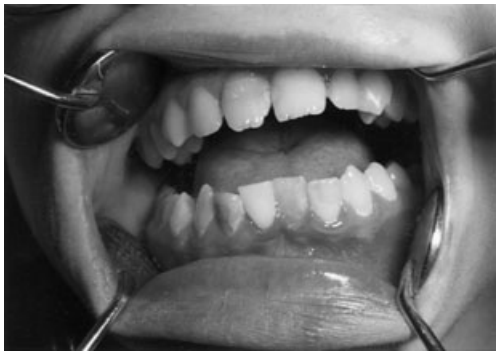

Figure 2 Patient (I.M. age 38) with mosaic karyotype 46XX/45X – enamel opacities buccal surfaces, incisors and canines of maxilla and mandibula

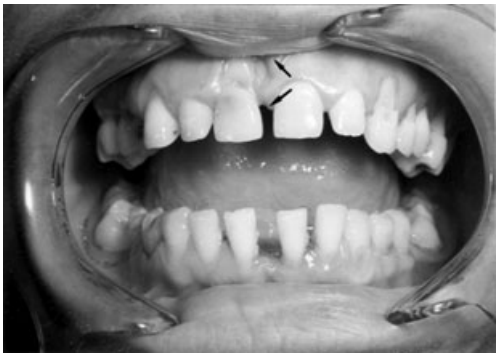

Figure 3 Patient (R.N. age 24) with Turner syndrome 45,X – enamel hypoplasia of buccal surfaces teeth 12, 11, 21, 22, 23, 33, 32, 42, 43 Tooth 41 with prosthetic crown. Gingivitis surrounding teeth 33, 32, 31, 41, 42, 43

Turner syndrome (type 2 'gingiva-like'- eight patients, type 3 ' papilla-like' - eight patients and type 4 'penetrating papilla-like'- five patients) and in 8% of control subjects only (type 2 'gingiva-like'- lower lips in two patients and upper lips in two patients). Differences in frequency of these anomalies in patients with Turner syndrome and the control group were significant ($P < 0.05$). There were not statistically significant differences between 45,X and patients with structural aberrations of chromosome X and mosaics (Table 1).

Occurrence of developmental lesions within hard tissue's were more frequent (Table 1). It is evident that these alterations occurred more often within patients with Turner syndrome than in the control group. Enamel opacities in the form of white/cream spots were found mainly in 45,X (71% of cases) and structural aberrations of chromosome X (67% of cases). Frequency of enamel opacities was less in patients with mosaics (43% of cases) (Figure 2). Differences in frequency of this anomaly in patients with Turner syndrome and patients in control group were significant ($P < 0.05$). Defects were observed on labial or buccal surfaces of incisors, canines, premolars and molars as single well-demarcated areas both in Turner syndrome patients and those in the control group. No cases of single teeth with enamel defects in Turner syndrome were found. In 18 patients of Turner syndrome (two cases of 45,X, seven cases of structural aberrations of chromosome X and nine cases of mosaics) were found enamel opacities of incisors, canines and premolars. In 11 of 50 patients with Turner syndrome (10 patients with karyotype 45,X and in one patient with structural aberrations of chromosome X) enamel opacities of all teeth were observed. In nine of 10 patients in the control group were found enamel opacities of incisors, canines and premolars. Enamel opacities of all teeth were observed in one case of the control group. There were statistically significant differences between 45,X and these patients with structural aberrations of chromosome X and others where expressed mosaics. ($P < 0.05$).

Enamel hypoplasia was observed in 38% of Turner syndrome patients and only in 2% of the control group (Table 1). This anomaly as pits, horizontal grooves or vertical grooves on labial or buccal surfaces of incisors, canines and premolars was observed in two cases of 45,X, five cases of structural aberrations of chromosome X and four cases of mosaics (Figure 3). Enamel hypoplasia of all teeth was observed in eight of 50 patients with Turner syndrome: six cases of 45,X, one case of structural aberration of chromosome X and one case of mosaic karyotype. Only one patient with enamel hypoplasia as pits on labial incisors was found in control group. Differences in the frequency of this anomaly in patients with Turner syndrome and patients in control group were also significant ($P < 0.05$). There were statistically significant differences between 45,X and these patients with structural aberrations of chromosome X and mosaics ($P < 0.05$). Enamel hypoplasia was found mostly in patients with karyotype with structural aberrations of chromosome X (50% of cases) and patients with karyotype 45X (47% of cases).

Discussion

Our study has shown that all patients with Turner syndrome had oral anomalies, which were rarely observed by other authors, especially descriptions concerning of soft tissues of the oral cavity. These occurred more often than in the control group. Labial frenula with abnormal attachment and other malformations in maxillofacial region were observed in orofacialdigital syndrome I by Gunbay (Gunbay *et al*, 1996). In Ellis–van Creveld syndrome (chondroectodermal dysplasia) some oral features eg. abnormal frenal attachment, congenital missing incisors, malocclusion and others were described (Hattab *et al*, 1998; De Felice *et al*, 2001). These development changes are well known essential factors in the origin of the gingival recession and gingivitis which require early corrective treatment (Addy *et al*, 1987; Peacock, 1998; Fowler and Breault, 2000; Jimenez *et al*, 2002).

In our studied lesions within the enamel, mainly the forms of opacities and hypoplasia, were often observed. These anomalies were observed most commonly in the 45,X group and structural aberrations of chromosome X. Lopez reported that about 78% patients with Turner syndrome had hypoplasia (Lopez *et al*, 2002). Defects of enamel in the other somatic diseases are often seen. Aine

et al (1990) reported dental enamel defects in adult patients with celiac disease. Unspecific enamel lesions were found in 80% cases with celiac diseases and in 18% cases in control group. Dental anomalies associated with hereditary disorders as amelogenesis imperfecta (Bäckman, 1988; Ooya *et al*, 1988; Collins *et al*, 1999), Morquio syndrome (Kinirons and Nelson, 1990; Rolling *et al*, 1999), epidermolisis bullosa (Wright *et al*, 1993) and tricho-dento-osseous syndrome (Jorgenson and Warson, 1973; Seow, 1993). Within the general population these anomalies are not often met. Umesi Koleoso (2004) reported adult population with enamel anomalies, 7% subjects presented enamel hypoplasia and 16% enamel opacities. Epidemiological study of idiopathic enamel hypomineralisation in permanent teeth of Swedish children showed enamel hypomineralisation from 4.4% to 15% of cases (Koch *et al*, 1987). Many authors reported that chromosome X controls of tooth size, shape, thickness of enamel and root morphology (Varrela *et al*, 1988; Mayhall *et al*, 1991; Mayhall and Alvesalo, 1992; Midtbo and Halse, 1994a; Kusiak *et al*, 2000). The absence of chromosome X is claimed to have a stronger influence on that condition. It also influences tooth crown size by many authors (Alvesalo and Tammisalo, 1981; Townsend *et al*, 1984; Varrela *et al*, 1988; Mayhall *et al*, 1991; Mayhall and Alvesalo, 1992; Midtbo and Halse, 1994a; Kusiak *et al*, 2000; Zilberman *et al*, 2000). Lopez *et al* (2002) reported less values of blood calcium and phosphorus determinations for most of patients with Turner syndrome which probably influence the hypoplastic appearance of Turner syndrome teeth. Furthermore, the study of Lau on the amelogenin gene responsible for amelogenesis inperfecta and other craniofacial diseases is relevant (Lau *et al*, 1990). From the studies conducted it transpires that the highest number of abnormal frenal attachment were observed in Turner syndrome when compared with control group. There were no statistically significant differences between 45X, these patients with structural aberrations of chromosome X and mosaics. Enamel defects occurred more often in Turner syndrome than in the control group. These defects were correlated with the type of chromosome X anomaly in Turner syndrome patients. There were statistically significant differences between 45X and patients with structural aberrations of chromosome X in comparison with the mosaic karyotype. Patients with mosaic karyotypes including normal cells are less likely to develop congenital abnormalities of the oral cavity.

Treatment of patients with Turner syndrome is necessary, especially 45X and these patients with structural aberrations of chromosome X by dental doctors of aesthetic dentistry and periodontology.

References

Addy M, Dummer PH, Hunter ML, Kingdon A, Shaw WC (1987). A study of the association of fraenal attachment, lip coverage, and vestibular deph with plaque and gingivitis. *J Periodontol* **58:** 752–757.

Aine L, Maki M, Collin P, Keyrilainen O (1990). Dental enamel defects in celiac disease. *J Oral Pathol Med* **19:** 241–245.

Alvesalo L, Tammisalo E (1981). Enamel thickness in 45,X females' permanent teeth. *Am J Hum Genet* **33:** 464–469.

Arulanantham K, Kramer MS, Gryboski JD (1980). The association of inflammatory bowel disease and X chromosomal abnormality. *Pediatrics* **66:** 63–67.

Bäckman B (1988). Amelogenesis imperfecta- clinical manifestation in 51 families in a northern Swedish country. *Scand J Dent Res* **96:** 505–516.

Collins MA, Mauriello SM, Tyndall DA, Wright JT, Hill AGC (1999). Dental anomalies associated with amelogenesis imperfecta. *Oral Surg Oral Med Oral Path Oral Radiol Endod* **88:** 358–364.

De Felice C, Toti P, Di Maggio G, Parrini S, Bagnoli F (2001). Absence of the the inferior and lingual frenula in Ehlers–Danlos syndrome. *Lancet* **12:** 1500–1502.

FDI Commission on Oral Health Research and Epidemiology (1982). An epidemiological index of developmental defects of enamel (DDE Index). *Int Dent J* **32:** 158–167.

Filipsson R, Lindsten J, Almqvist S (1965). Time of eruption of the permanent teeth, cephalometric and tooth measurement and sulphation factor activity in 45 patients with Turner's Syndrome with different types of X chromosome aberrations. *Acta Odontol Scand* **48:** 91–113.

Fowler EB, Breault LG (2000). Early creeping attachment after frenectomy: a case report. *Gen Dent* **48:** 591–593.

Goldman B, Polani PE, Daker MG, Angell RR (1982). Clinical and cytogenetic aspects of X-chromosome deletions. *Clin Genet* **21:** 36–52.

Gunbay S, Zeytinoglu B, Ozkinay F, Ozkinay C, Oncag A (1996). Orofaciodigital syndrome I: a case report. *Clin Pediatr Dent* **20:** 329–332.

Hall JG, Gilchrist DM (1990). Turner syndrome and its variants. *Pediatr Clin North Am* **37:** 1421–1440.

Harju M, Laine T, Alvesalo L (1989). Occlusal anomalies in 45,X/46,XX-and 46,Xi(Xq) –women (Turner syndrome). *Scand J Dent Res* **97:** 387–391.

Hattab FN, Yassin OM, Sasa IS (1998). Oral manifestations of Ellis-van Creveld syndrome: report two siblings with unusual dental anomalies. *Clin Pediatr Dent* **22:** 159–165.

Horowitz SL, Morishima A (1974). Palatal abnormalities in the syndrome of gonadal dysgenesis and its variants and in Noonan's syndrome. *Oral Surg* **38:** 839–844.

Jaspers MT, Witkop CJ Jr (1980). An isolated trait associated with syndromes and X – chromosomal aneupolidy. *Am J Hum Genet* **32:** 396–413.

Jimenez Y, Bagan JV, Milian MA, Gavalda C, Seculty C (2002). Lichen sclerosus et atrophicus manifestating with localized loss of periodontal attachment. *Oral Dis* **8:** 310–313.

Jorgenson RJ, Warson RW (1973). Dental abnormalities in the tricho-dento-osseous syndrome. *Oral Surg Oral Med Oral Path* **36:** 693–700.

Kinirons MJ, Nelson J (1990). Dental findings in mucopolysaccharidosis type IV A (Morquio's diseases type IVA). *Oral Surg Oral Med Oral Pathol* **70:** 177–179.

Koch G, Hallonsten AL, Ludvigsson N, Hansson BO, Holst A (1987). Epidemiologic study of idiopathic enamel hypomineralization in permanent teeth of Swedish children. *Community Dent Oral Epidemiol* **15:** 279–285.

Kusiak A, Sadlak-Nowicka J, Iliszko M, Limon J (2000). Anomaly of morphological construction of permanent teeth in Turner syndrome with various aberration of chromosome X. *Czas Stom* **LIII:** 608–614 (abstract in English).

Kusiak A, Sadlak-Nowicka J, Limon J, Kochańska B (2005). Root morphology of mandibular premolars in 40 patients with Turner syndrome. *Int Endod J* **38:** 822–826.

Laine T, Alvesalo L, Lammi S (1992). A study in 47,XYY men of the expression of sex chromosome anomalies in dental occlusion. *Archs Oral Biol* **7:** 923–928.

Lau EC, Slavkin HC, Snead ML (1990). Analysis of human enamel genes: insights into genetics disorders of enamel. *Cleft Palate Craniofac J* **27:** 121–130.

Lippe B (1991). Turner syndrome. *Endocrinol Metab Clin North Am* **20:** 121–152.

Lopez ME, Bazan C, Lorca IA, Chervonagura A (2002). Oral and clinical characteristics of a group of patients with Turner syndrome. *Oral Surg Oral Med. Oral Pathol Oral Radiol Endod* **94:** 196–204.

Mayhall JT, Alvesalo L (1992). Dental morphology of 45,XO human females: molar cusp area, volume, shape and linear measurements. *Arch Oral Biol* **37:** 1939–1943.

Mayhall JT, Alvesalo L, Townsend GC (1991). Tooth crown size in 46,X,i(Xq) human females. *Arch Oral Biol* **36:** 411–414.

Midtbo M, Halse A (1994a). Tooth crown size and morphology in Turner syndrome. *Acta Odontol Scand* **52:** 7–19.

Midtbo M, Halse A (1994b). Root length, crown height, and root morphology in Turner syndrome. *Acta Odontol Scand* **52:** 303–314.

Mirko P, Miroslav S, Lubor M (1974). Significance of the labial frenum attachment in periodontal disease in man. Part II. An attempt to determine the resistance of periodontium. *J Periodontol* **45:** 895–897.

Ooya K, Nalbandian J, Noikura T (1988). Autosomal recessive rough hypoplastic amelogenesis imperfecta. A case report with clinical light microscopic, radiografic and electron microscopic observations. *Oral Surg Oral Med Oral Pathol* **65:** 449–458.

Palmer CG, Reichmann A (1976). Chromosomal and clinical findings in 110 females with Turner syndrome. *Hum Genet* **35:** 35–49.

Peacock ME (1998). Frenotomy and keratinized tissue augmentation. *Gen Dent* **46:** 194–196.

Pelz L, Köbschall H, Lubcke UG, Krüger G, Hinkel GK, Verron G (1991). Long-termal follow-up in females with Ullrich – Turner syndrome. *Clin Genet* **40:** 1–5.

Robinson A, de la Chapelle A (1996). Sex chromosome abnormalities. In: Rimoin DL, Connor JM, Pyeritz RE, eds. *Emery and Rimoin's principles and practice of medical genetics*. Churchill Livingstone: New York, pp. 973–981.

Rolling I, Clausen N, Nayvad B, Sindet-Pederson S (1999). Dental findings in three siblings with Morquio's syndrome. *Int J Paediatr Dent* **9:** 219–224.

Seow WK (1993). Taurodontism of the mandibular first permanent molar distinguishes between the tricho-dento-osseous (TDO) syndrome and amelogenesis imperfecta. *Clin Genet* **43:** 240–246.

Szilagyi A, Keszthelei G, Nagy G, Madlena M (2000). Oral manifestations of patients with Turner syndrome. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* **89:** 577–584.

Temtamy SA, Ghali I, Salam MA, Hussein FH, Ezz EH, Salah N (1992). Karyotype phenotype correlation in females with short stature. *Clin Genet* **41:** 147–151.

Townsend G, Alvesalo L (1995). Intercuspal distances of maxillary premolar teeth in 45,X (Turner Syndrome) females. In: Moggi–Cecchi J, ed. *Aspects of Dental Biology: Paleontology, Anthropology and Evolution*. Institute for the Study of Man: Florence, pp. 77–85.

Townsend G, Jensen BL, Alvesalo L (1984). Reduced tooth size in 45,XO (Turner syndrome)females. *Am J Phys Anthropol* **65:** 367–371.

Turner HH (1938). A syndrome of infantilism, congenital webbed neck, and cubitus valgus. *Endocrinology* **23:** 566–574.

Umesi Koleoso DC (2004). Dental fluorosis and other enamel disorders in 12-year-old Nigerian children. *J Comm Med Prim Health Care* **16:** 25–28.

Varrela J (1990). Root morphology of mandibular premolars in human 45,X females. *Arch Oral Biol* **35:** 109–112.

Varrela J (1992). Effect of 45, X/46,XX mosaics on toot morphology of mandibular premolars. *J Dent Res* **71:** 1604–1606.

Varrela J, Townsend G, Alvesalo L (1988). Tooth crown size in human females with 45,X/46,XX chromosomes. *Arch Oral Biol* **33:** 291–294.

Wright JT, Fine JD, Johnson L (1993). Hereditary epidermolysis bullosa: oral manifestations and dental management. *Pediatr Dent* **15:** 242–248.

Zilberman U, Smith P, Alvesalo L (2000). Crown components of mandibular molar teeth in 45,X female (Turner syndrome). *Arch Oral Biol* **45:** 217–225.