Taurodontism – a minor diagnostic criterion in Laurence-Moon/Bardet-Biedl syndromes

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Abstract

The prevalence of taurodont molars and the degree of taurodontism in the mandibular permanent first and second molars were studied in 39 Norwegian individuals diagnosed with Laurence-Moon and/or Bardet-Biedl syndromes (LM/BBS). Four individuals were excluded due to missing mandibular molars. Among the 35 participants with mandibular molars present, one or more taurodont teeth were found in 83 %. In the population investigated, the second molars had the highest (72 %) and first molars the lowest (58 %) prevalence of taurodontism.

Keywords:
Taurodontism; Laurence-Moon; Bardet-Biedl; Mandibular molars; LM/BBS

Authors:

Lars-Fredrik Gjølstad, Master of dentistry student.

Katira Kakenova Akzholova, Master of dentistry student.

Supervisors:

Kari Storhaug, Dr.Odont, TAKO-centre[1], Oslo.

Els-Marie Andersson, PhD, TAKO-centre, Oslo and
Department of Orthodontics, faculty of Dentistry, University of Oslo.

Stefan Axelsson, Dr.Odont, TAKO-centre, Oslo.

[1] TAKO-centre, National Resource Centre of Oral Health in Rare Medical Conditions, Lovisenberg Diakonale Hospital, Oslo, Norway.

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Summary

The aim of the study was to assess the prevalence of taurodontism in Norwegian individuals diagnosed with Laurence-Moon/Bardet-Biedl Syndrome (LM/BBS).

Laurence-Moon and Bardet-Biedl syndrome are two syndromes which until recently have been considered as one condition. They have recently been established as two syndromes with very similar phenotypes.

Taurodontism is a disturbance of odontogenesis. It is a trait with unclear aetiology, and is proposed to be a result of disrupted developmental homeostasis involving delayed invagination of Hertwig's epithelial root sheath. Taurodontism appears to be a multifactorial trait; some reports suggest that taurodontism may be genetically transmitted.

Among 35 individuals with LM/BBS, taurodontism were found in 29 (83 %). The prevalence of taurodontism at an individual level (one or more taurodont teeth present in the mandibula per participant) is estimated to 83 % in the Laurence-Moon/Bardet-Biedl syndrome population in Norway, 1984 – 2007.

We suggest that taurodontism should be included as a minor diagnostic criterion for the Laurence-Moon and/or Bardet-Biedl syndromes.

Introduction

In 1865, John Zachariah Laurence and Robert Charles Moon described a syndrome comprising retinal degeneration, hypogonadism, mental retardation, and spastic paraplegia (Laurence and Moon, 1866). Over 50 years later Georges Louis Bardet and Artur Biedl independently described a similar syndrome with the additional features of polydactyly and obesity (Bardet, 1920; Biedl, 1922). Shortly thereafter it was concluded that the disorder described by these authors represented a single condition. Subsequently, the disorder was referred to in the scientific and medical literature as Laurence-Moon-Biedl syndrome (1988; Anadoliiska and Roussinov, 1993; Haskova *et al.*, 2009). Laurence-Moon-Bardet-Biedl-syndrome (LM/BBS) has traditionally been described by retinal dystrophy, polydactyly, obesity, mental retardation, spastic paraparesis, hypogonadism and renal abnormalities (Harnett *et al.*, 1988; Green *et al.*, 1989). LM/BBS have some overlapping features with other entities such as McKusick-Kaufman and Alstrom's syndromes. These signs are retinitis pigmentosa, mental retardation, obesity, and hypogonadism (Harnett *et al.*, 1988; Green *et al.*, 1989; Ingster-Moati *et al.*, 2000). LM/BBS also has features in common with Prader-Willi syndrome such as mental retardation, obesity, hypogonadism and diabetes mellitus (Dunn, 1968; Klein and Ammann, 1969). More recently, LM/BBS syndrome has been considered to be two distinct disorders; Laurence-Moon syndrome (LMS: OMIM 245800) and Bardet-Biedl syndrome (BBS: OMIM 209900) (Kobrin *et al.*, 1990; Iannello *et al.*, 2002; Sheffield, 2004). The two syndromes have a similar phenotype, which includes retinal dystrophy, obesity, and hypogonadism (Schachat and Maumenee, 1982; Harnett *et al.*, 1988; Moore *et al.*, 2005), but BBS is now a well-established distinct clinical entity similar to, but different from LMS, mainly by the absence of spastic paraparesis and presence of polydactyly (Kobrin *et al.*, 1990; Elbedour *et al.*, 1994). In LMS, polydactyly is rare and spastic paraparesis dominates, whereas neurologic complications are very unusual in BBS (Iannello et al., 2002; Sheffield, 2004). However, Moore et al (2005) concluded that the features found in a Newfoundland population did not support the notion that BBS and LMS are distinctly different.

BBS is a complex syndrome which varies both within and between families and diagnosis is often difficult (Beales *et al.*, 1997; Riise *et al.*, 1997). Green (1989) suggested that the cardinal manifestations of BBS should be considered to comprise retinal dystrophy, dystrophic extremities, obesity, hypogenitalism (in men only) and renal disease (Green *et al.*, 1989). Obesity among individuals with BSS is proposed to be of neuroendocrine origin (Marion *et al.*, 2011). The complications of BBS are severe, and almost all patients become blind before the age of 20 years (Riise *et al.*, 1997). Intelligence can be nearly normal, and testing has often not been corrected for the visual deficit (Green *et al.*, 1989; Lofterod *et al.*, 1990; Riise *et al.*, 1997). In a study by Beales (1999) it was reported that postaxial polydactyly was present in 69% of patients at birth, obesity begins to develop at around 2-3 years and retinal degeneration had not become apparent until a mean age of 8.5 years (Beales *et al.*, 1999). Patients with BBS should be screened for renal abnormality, and undergo ophthalmological and electrophysiological examinations essential for confirmation and correct diagnosis (Bui Quoc *et al.*, 2005).

BBS is a multisystemic, autosomal recessive disorder, typified by developmental and progressive degenerative defects (Zaghloul and Katsanis, 2009). It is now known that BBS has locus heterogeneity, with causative mutations identified in as many as 14 genes (Green *et al.*, 1989; Nishimura *et al.*, 2001; Billingsley *et al.*, 2010; Sapp *et al.*, 2010; Feuillan *et al.*, 2011). A combination of genetic, in vitro, and in vivo studies have highlighted ciliary dysfunction as a primary cause of BBS pathology, which has in turn contributed to the improved understanding of the functions of the primary cilium in humans and other vertebrates (Gunay-Aygun, 2009; Zaghloul and Katsanis, 2009). There is no definite treatment. However early diagnosis and symptomatic, supportive and rehabilitative measures can reduce the disability according to Abdulla et al (2009).

There is much more research around genetic markers for BBS than for LMS. A search for Laurence-Moon in the NCBI Gene database April 2011 resulted in no hits, where Bardet-Biedl resulted in 324 hits.

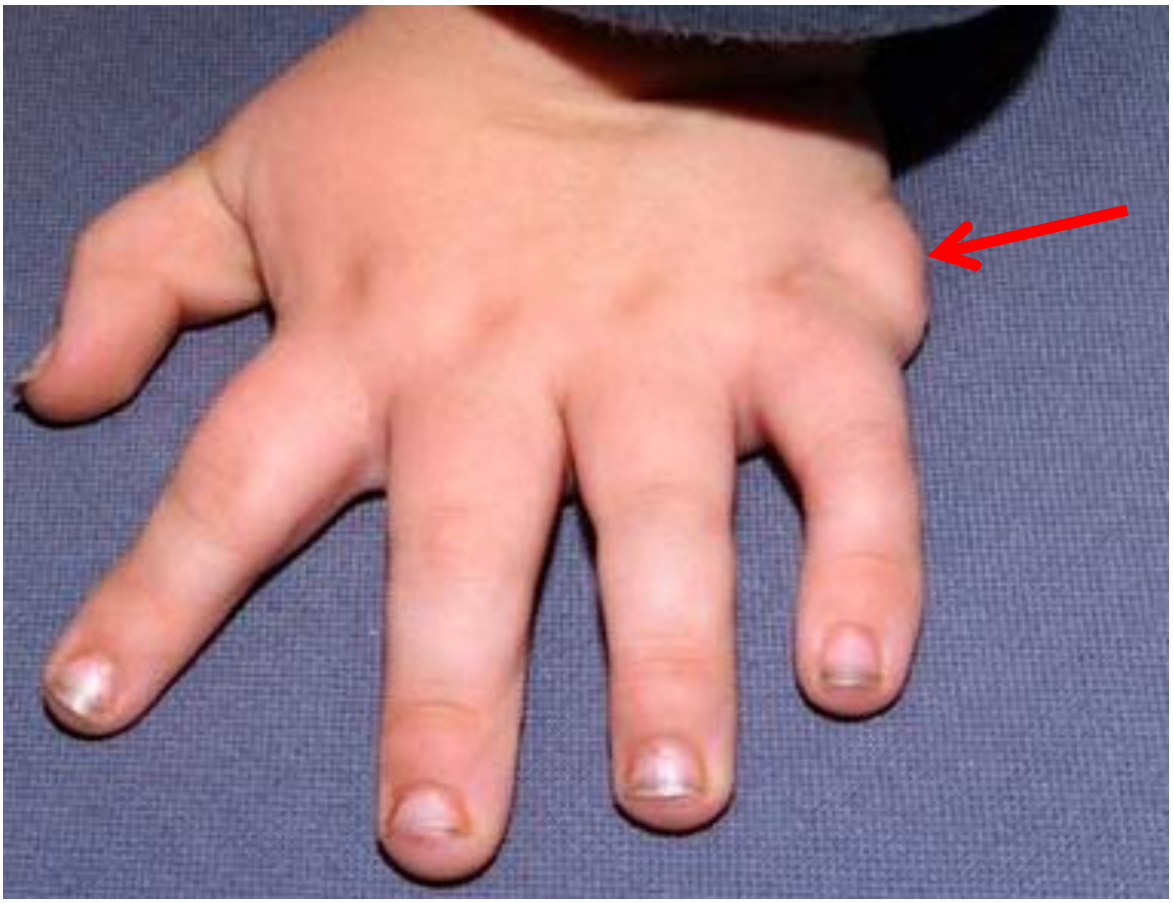


Figure 1: Picture showing postaxial polydactyly (arrow) – from the archive of the TAKO-centre.

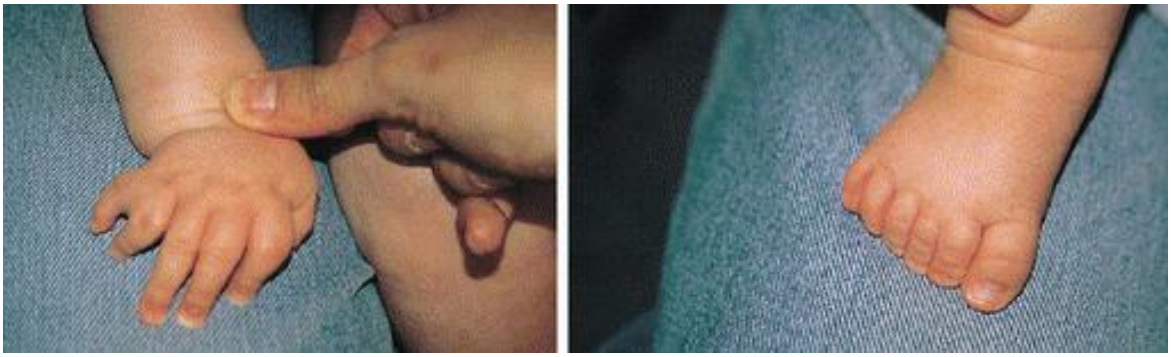


Figure 2: Postaxial polydactyly in a hand and foot from the same child with BBS (Tobin and Beales, 2007).

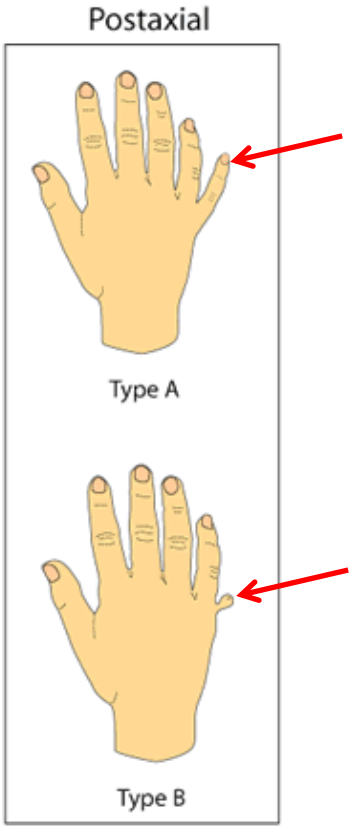


Figure 3: Different types of postaxial polydactyly (Uni.Florida).

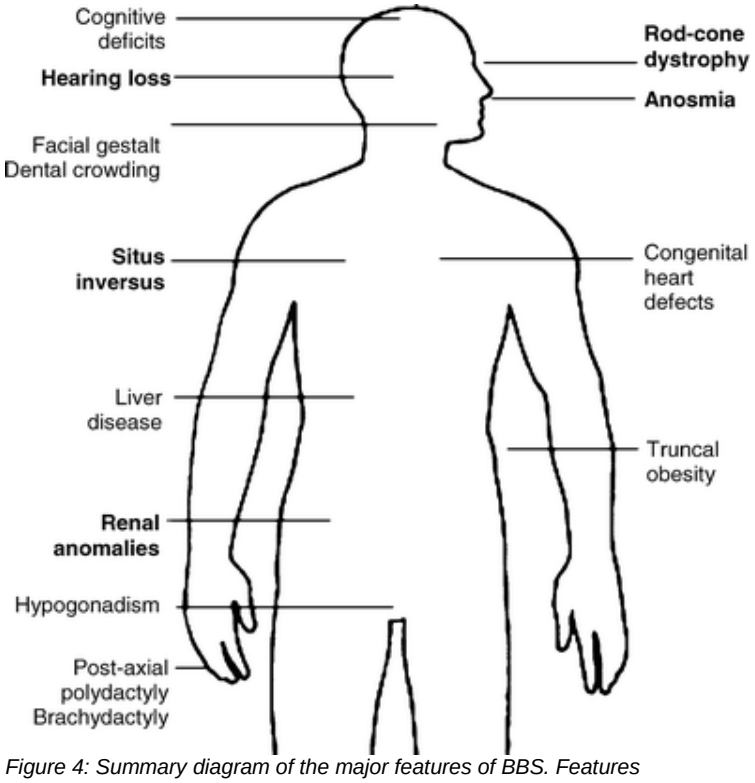


Figure 4: Summary diagram of the major features of BBS. Features directly attributable to defects in ciliary function are marked in bold (Tobin and Beales, 2007).

Dental anomalies in BBS where first documented in 1960 when Magnusson found delayed eruption and anatomical anomalies (Magnusson, 1960). The most significant findings were hypodontia, small teeth, enamel hypoplasia, short roots, and a thickened mandibular body (Magnusson, 1960; Kobrin *et al.*, 1990; Lofterod *et al.*, 1990; Borgstrom *et al.*, 1996). Dental problems appear to be common; in a study by Beales, among 109 9-year old individuals with BBS, 27% had malocclusion or crowding of the teeth, or had teeth extracted. In the study they observed enamel hypoplasia with yellow discolouration, crowding of teeth, and mild micrognathia. Eighty-nine per cent had a high arched palate (Beales *et al.*, 1999).

Estimated prevalence of Bardet-Biedl syndrome ranges from one in 160 000 in northern European populations to as high as one in 13 500 in Kuwait and Newfoundland (Zaghloul and Katsanis, 2009).

Dental morphological traits are of particular importance in the study of phylogenetic relationships and population affinities (Constant and Grine, 2001). One of the most important abnormalities in tooth morphology is taurodontism. Since the literature offers little information on dental aberrations in LMS and BBS, more detailed data are needed.

Taurodontism

The term is derived from the Greek word 'tauros', meaning 'bull', and 'odontos', referring to 'tooth' (Jaspers and Witkop, 1980). The opposite is the cynodont – derived from the Greek word 'kyno' meaning 'dog' – referring to 'doglike' teeth (Keith, 1913) – which is the same as normal teeth. The first report of taurodontism in modern man's dentition was published in the beginning of the 20th century by among others M. de Terra (1903), Gorjanovic-Kramberger (1906, 1907, and 1908), Adloff (1907) (Lysell, 1965) and in 1909 by Pickerill – who used the term "radicular dentinoma" to describe the condition (Pickerill, 1909; Jaspers and Witkop, 1980). The trait was first brought to light in fossil remains at Krapina, Croatia in 1899, when Professor Gorjanovic-Kramberger started excavations of 200 fragments of human skeletons representing at least 10 individuals of the Neanderthal race. The trait was found in most molars, but it was not a general trait (Lysell, 1965).

The term taurodontism was proposed by Arthur Keith to describe this unusual tooth form (Keith, 1913; Lysell, 1965; Blumberg *et al.*, 1971). Since its inception, the term has been clouded by controversy and confusion (Blumberg *et al.*, 1971).

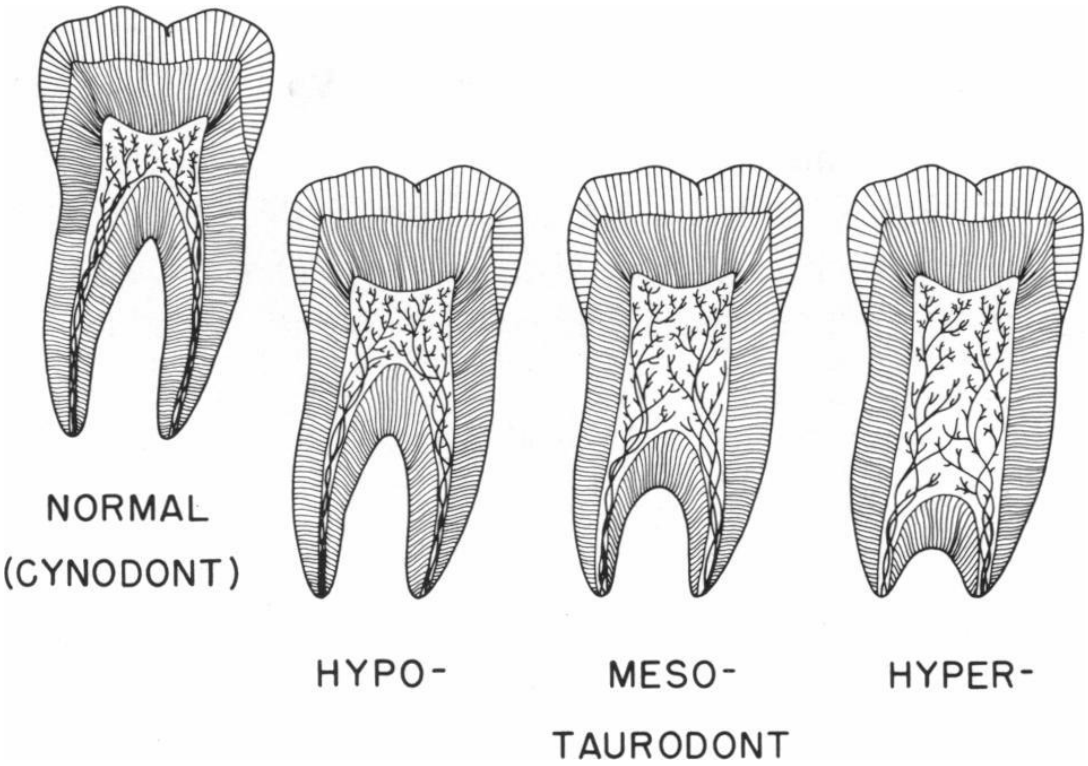


Figure 5: Illustration of the differences of cynodont/normal teeth and different grades of taurodont teeth (Jaspers and Witkop, 1980).

It is proposed that taurodontism is more advantageous than cynodontism in people with heavy masticatory habits, for example Neanderthals and Inuit (Eskimos) (Pedersen, 1949), who prepared skins for protection from the cold by chewing them (Coon, 1963) or in populations in which teeth were used as tools (Witkop Jr., 1976). Despite this theory, no evidence of taurodontism has been found in prehistoric American Indians, a group who must also have used their teeth extensively as tools (Sciulli, 1977).

The reported prevalence of taurodont molars and premolars ranges from 0.25-11.3% (Haskova et al., 2009).

Aetiology

The aetiology of taurodontism is still unclear, but it has been proposed that taurodontism is a result of disrupted developmental homeostasis and involves a delayed invagination of Hertwig's epithelial root sheath (Kan *et al.*, 2010). The trait is characterized by vertically elongated pulp chambers, apical displacement of the pulpal floor and bifurcation or trifurcation of the roots (Keith, 1913; Brkic and Filipovic, 1991; Rao and Arathi, 2006).

Taurodontism appears to be a multifactorial trait; some reports suggest that taurodontism may be genetically transmitted (Fischer, 1963; Witkop Jr., 1971; Goldstein and Gottlieb, 1973) and could be associated with an increased number of X-chromosomes (Gage, 1978). However, other researchers have found no simple genetic association but have noticed a trend for X-chromosomal aneuploidy amongst patients with more severe forms of the trait (Jaspers and Witkop, 1980). These chromosomal abnormalities may disrupt the development of the tooth form; however, a specific genetic abnormality cannot be ascribed to taurodontism (Neville *et al.*, 2002). Blumberg (1971) who biometrically studied the trait ascribed taurodontism to a polygenic system and described the anomaly as a continuous trait without discrete modes of expression (Blumberg *et al.*, 1971). Autosomal transmission of the trait has also been observed (Mangion, 1962). Whilst genetic transmission can be demonstrated in most cases, other external factors can also damage developing dental structures. Amongst these are infection (osteomyelitis) (Reichart, 1975), disrupted developmental homeostasis (Witkop, 1988), high-dose chemotherapy (Greenberg, 2003) and a history of bone marrow transplantation (Vaughan, 2005).

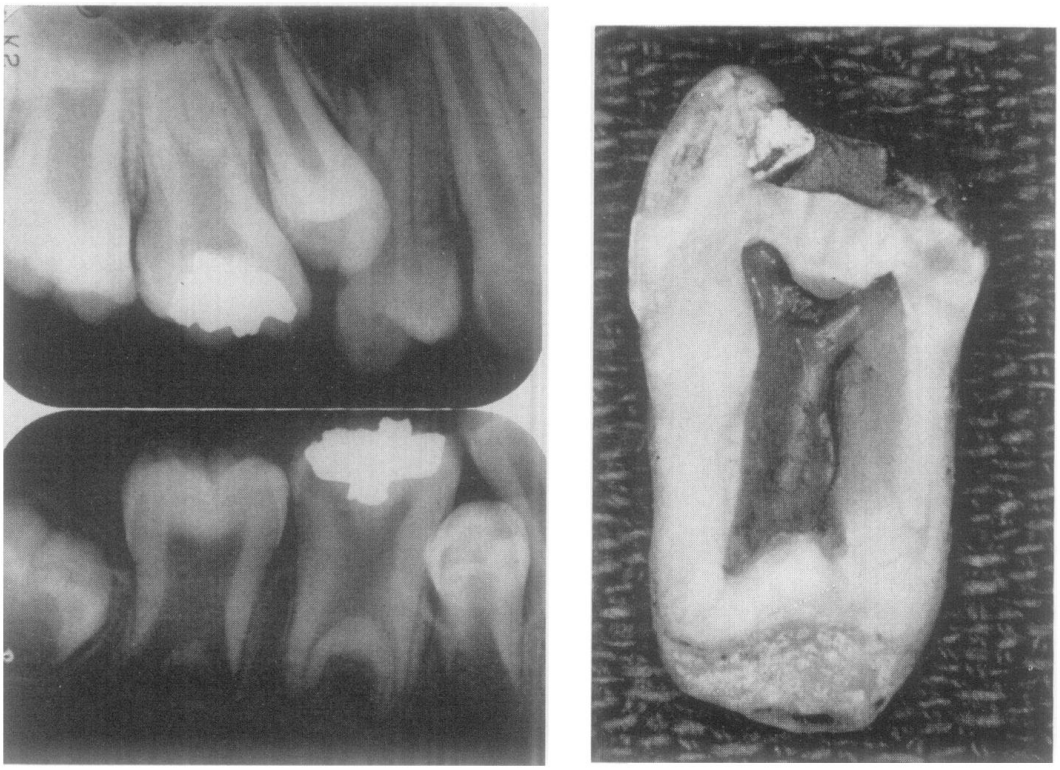


Figure 6: Left: Maxillary and mandibular x-rays showing excessive roominess of pulp chambers, characteristic of taurodontism. Right: Gross view of mandibular right first molar (Hamner et al., 1964).

Pathogenesis

Taurodontism is caused by the failure of Hertwig's epithelial sheath diaphragm to invaginate at the proper horizontal level (Hamner *et al.*, 1964; Barker, 1976; Terezhalmy *et al.*, 2001). Interference in the induction has also been proposed as a possible aetiology (Llamas and Jimenez-Planas, 1993).

Classification of taurodontism

Keith made no attempt to define the phenomenon when he described the trait in 1913 (Lysell, 1965). The first to make such an attempt was probably Shaw in 1928, who divided taurodont teeth into three groups by their external appearance; hypo-, meso- and hyper-taurodont referring to the pre-historic findings in Bantu-Boskop hybrids, the Heidelberg-man and the Krapina findings respectively (Lysell, 1965; Blumberg *et al.*, 1971). Shaw (1928) also suggested that the second molar should be considered as the "standard tooth" for determining the degree of taurodontism (Lysell, 1965). Several indices have been presented for classifying taurodontism, such as Shifman and Chanannel (1978) and Seow & Lai (1989), among others before them (Lysell, 1965; Jafarzadeh *et al.*, 2008). Differences of opinion exist as to how much displacement and/or morphologic change constitutes taurodontism, and most authors prefer a subjective rather than objective diagnosis of taurodontism. (Jafarzadeh *et al.*, 2008). When classifying taurodontic teeth, it has been suggested that it is necessary to consider not only the size of the pulp chamber and the size of roots but also the position of the body of the tooth in relation to the alveolar margin. The body of all taurodonts is located below the alveolar margin, opposed to cynodont of modern man which generally exhibits a body that is above the alveolar margin; the closest to the tooth form is the hypotaurodont of the Bantu-Boskop hybrid (Lysell, 1965).

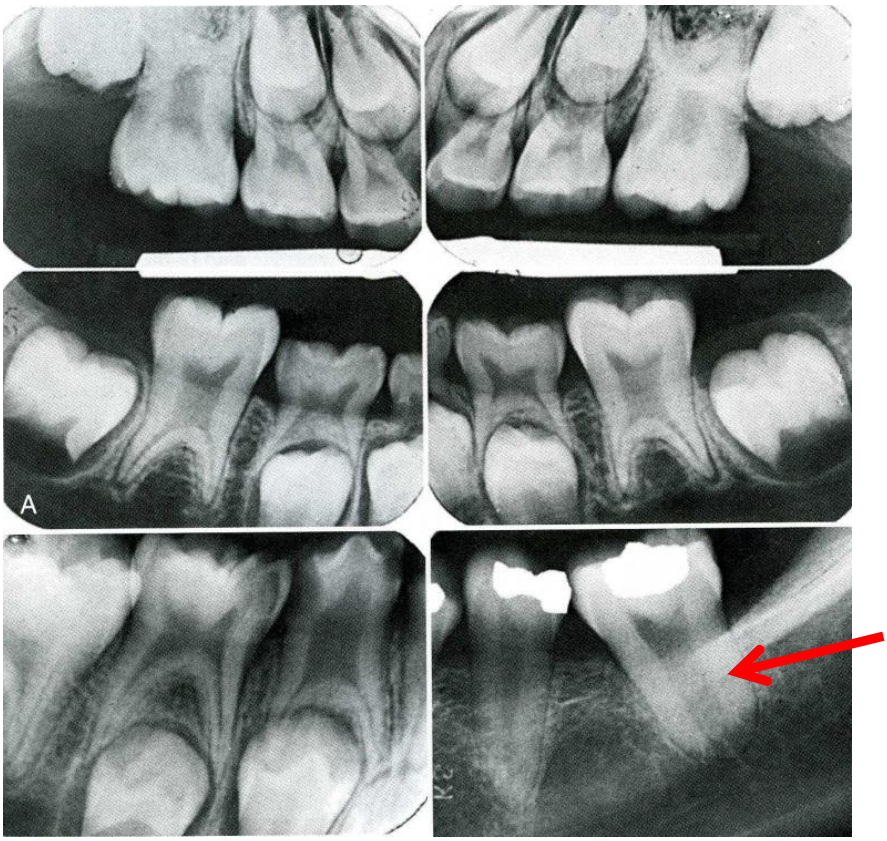


Figure 7: Taurodont teeth in both dentitions. Notice the pulp chamber of first mandibular molar on the left side, in the lower right radiograph, where no pulpal floor is visible (Rao and Arathi, 2006).

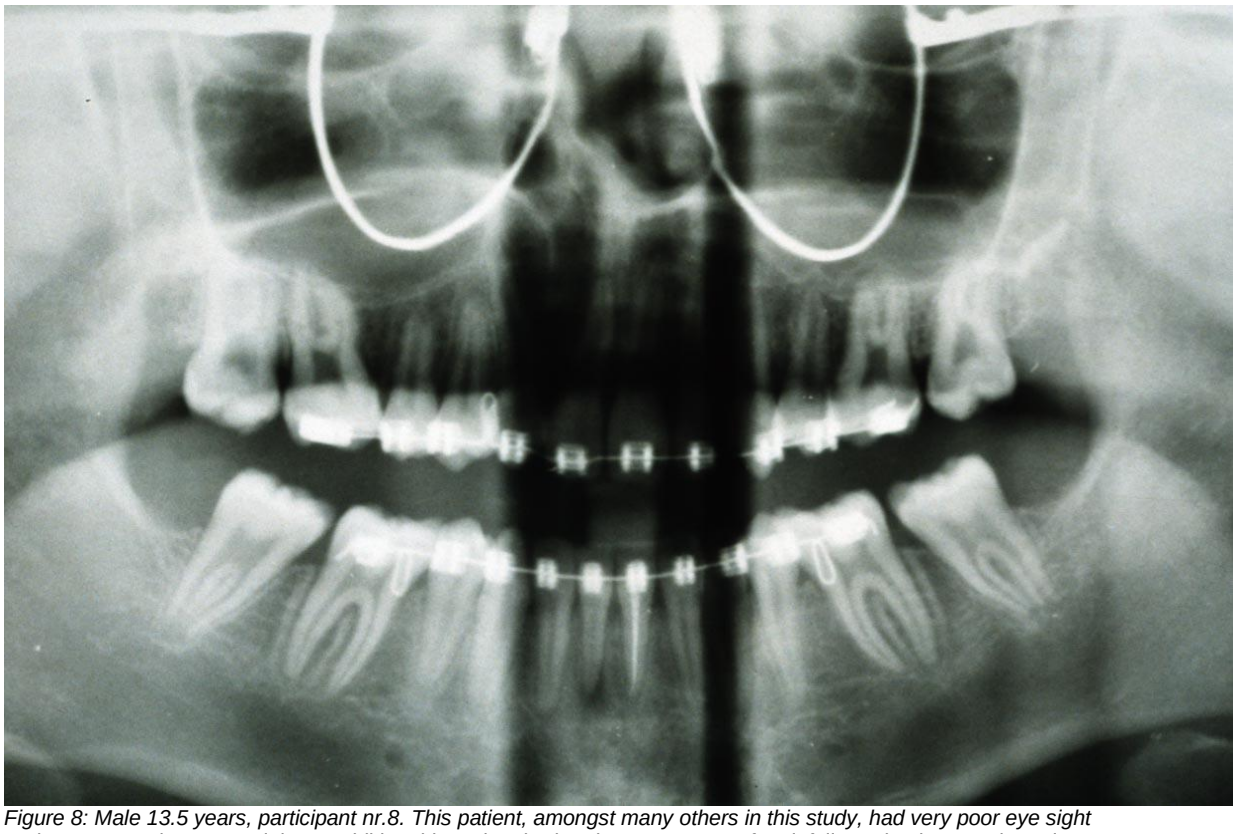


Figure 8: Male 13.5 years, participant nr.8. This patient, amongst many others in this study, had very poor eye sight and was somewhat overweight. In addition this patient had undergone surgery for cleft lip and palate, as the only one in this material (From the archive of the TAKO-centre).

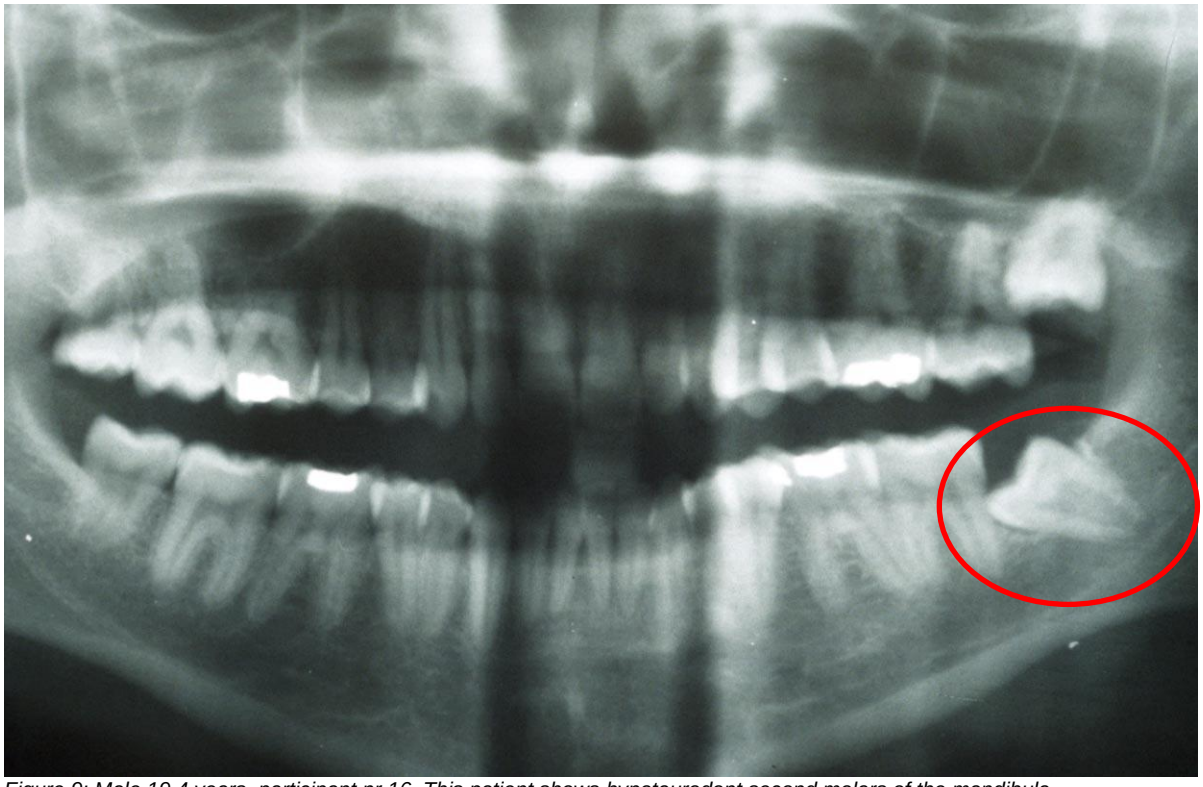


Figure 9: Male 19.4 years, participant nr.16. This patient shows hypotaurodont second molars of the mandibula bilaterally. 3rd molar left side shows an example of a hypertaurodont molar. (3rd molars were excluded from the material) (From the archive of the TAKO-centre).

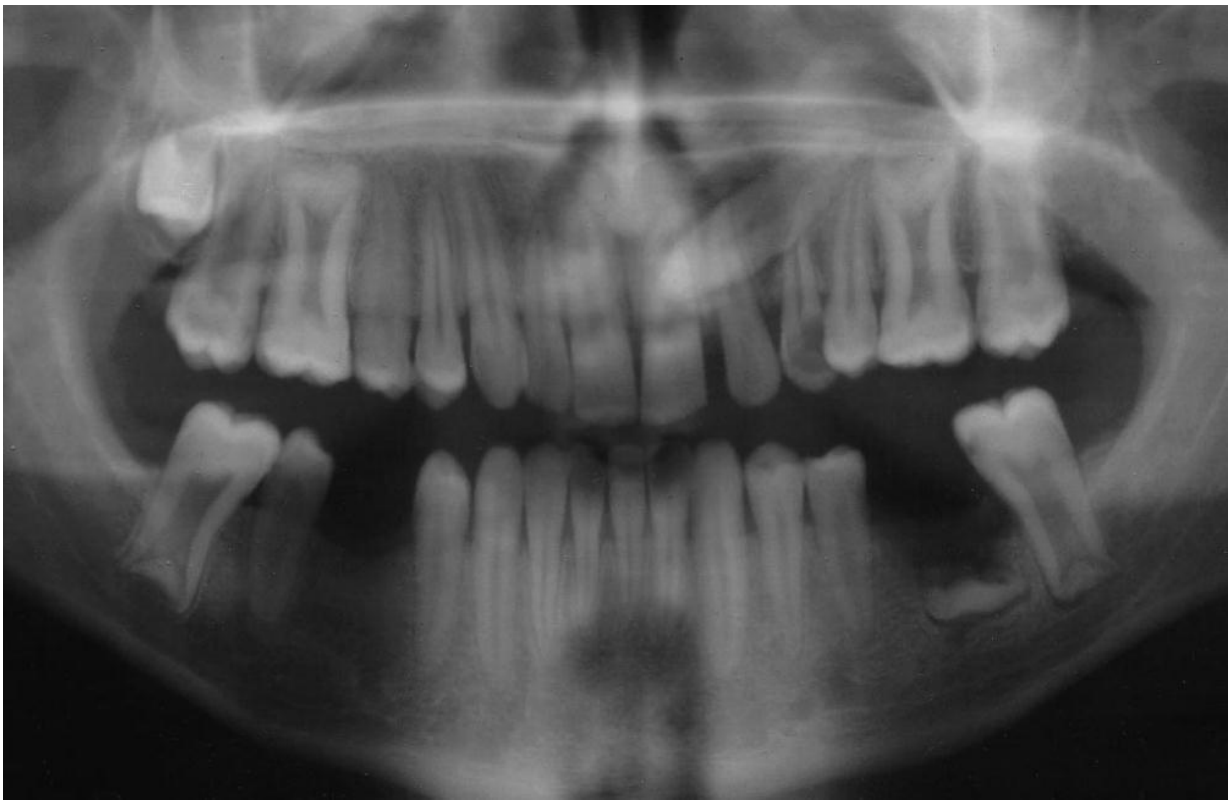


Figure 10: 18 years old solider with 6 hypertaurodont molars – reported by Sert and Bayrl (2004)

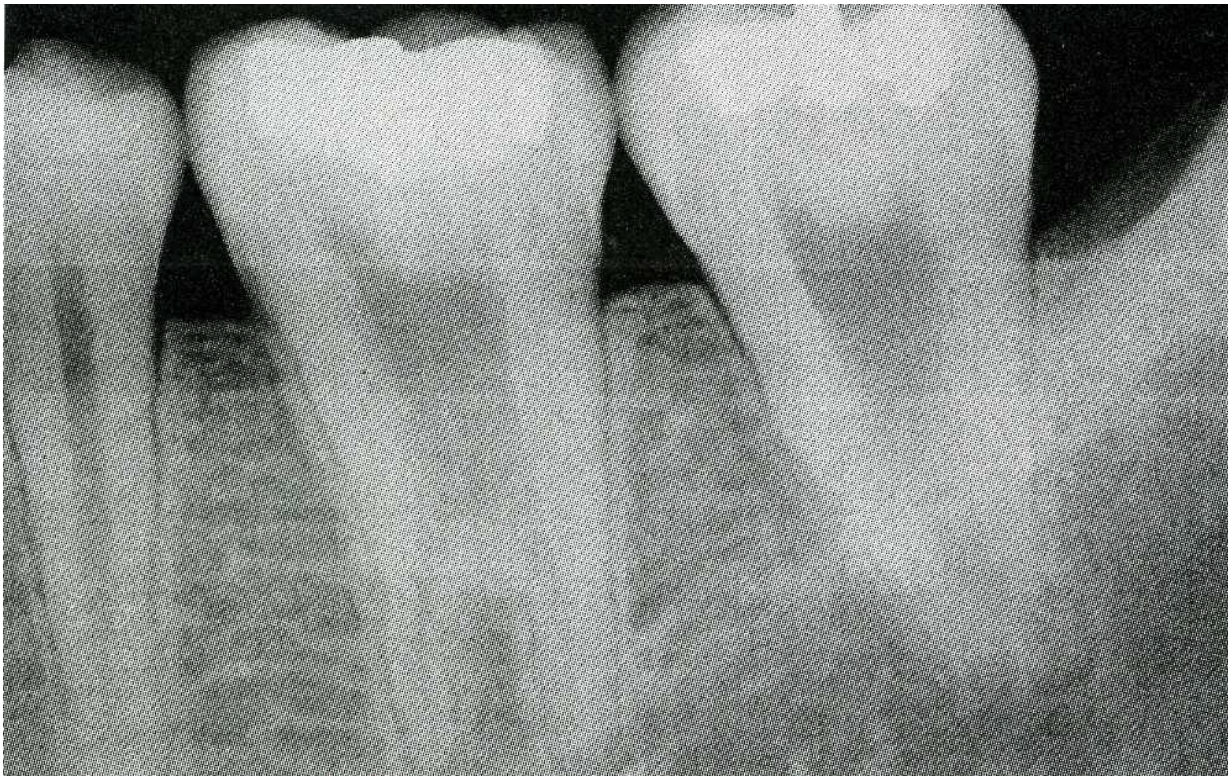


Figure 11: An illustration of taurodont teeth in periapical radiograph (Constant and Grine, 2001).

In establishing the degree of taurodontism in an individual, one must note that the degree of taurodontism tends to increase from the first to the third molar (Lysell, 1965). Taurodontism in the mandibular molars in LM/BBS were in this study diagnosed according to Seow and Lai (1989).

Aim

The aim of the study was to assess the prevalence of taurodontism in Norwegian individuals diagnosed with Laurence-Moon syndrome (LMS) and/or Bardet-Biedl Syndrome (BBS).

The hypothesis of this study was that taurodontism is more common in Laurence-Moon/Bardet-Biedl syndrome patients than in individuals without these syndromes.

Participants

The 39 participants (18 males, 21 females) constituted the whole population of known LM/BBS patients in Norway at the time of examination. The 39 participants were diagnosed with LM/BB syndrome – examined by an experienced geneticist at Frambu Health Centre prior to the diagnosis. The mean age was 25.9 years with an age range of 8,0 to 61.2 years at the time the orthopantomogram (OPG) was taken. The OPGs were collected over a period from 1984-1992 during family courses for LM/BB syndromes at Frambu Health Centre, and after 1993 during consultations at the TAKO-centre (National Resource Centre for Oral Health in Rare Medical Conditions). The measuring on orthopantomograms for diagnosing taurodontism was carried out at the TAKO-centre.

Criteria for inclusion in this study:

- Patients clinically diagnosed with LM/BB syndromes.

Criteria for exclusion from this study:

- All mandibular molars missing – since it is impossible to determine whether the teeth were taurodont or cynodont. 4 participants were excluded from the study for this reason.

Methods

Taurodontism of the mandibular 1st and 2nd permanent molars was assessed by measuring and calculating the CB/R ratio (crown-body/root ratio) on radiographic orthopantomograms according to Seow and Lai (1989).

	Cynodont	Hypotaurodont	Mesotaurodont	Hypertaurodont
CB/R-ratio	<1.10	1.10 – 1.29	1.30 – 2.00	>2.00

Table 1: Limit values for diagnosing taurodont teeth according to Seow and Lai (1989).

Prior to measuring, one of the authors (KKA) was calibrated against another author (SA) for the procedure. Landmark identification and measurements were likewise done by the former once during the year 2009, and then all of the data were verified by the latter. Divergence between the two occurred in two cases, and these two were then brought up for discussion between these two authors.
All registrations and calculations were later proofread by the main author (LFG).

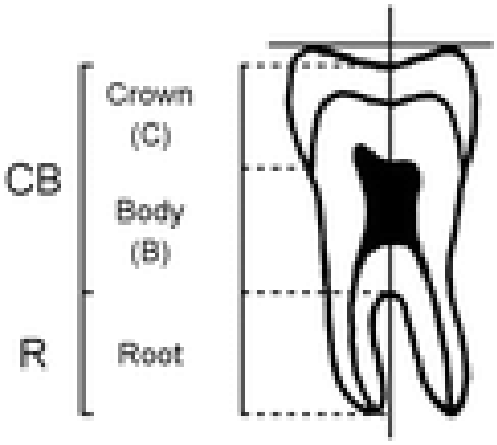


Figure 12: Assessment of the tooth morphology was based on measurements of Crown (C), Body (B) and Root (R). The CB/R ratio was then calculated, and the teeth were then diagnosed as taurodont according to Table 1 (Seow and Lai, 1989).

We chose to classify the mandibular molars according to Seow and Lai (1989), because their method offers a straightforward assessment of the tooth itself, not

taking into account the displacement of the molar in the mandibula, which may change with age.

Results

Mandibular molars with taurodontism in LM/BBS participants:

In our study, the prevalence of taurodontism in mandibular molars was 83 % among the 35 participants with LM/BBS. The mandibular molar most affected was the second molar on the left side (78 %), and the second molars were affected more often (72 %) than the first molars (58 %).

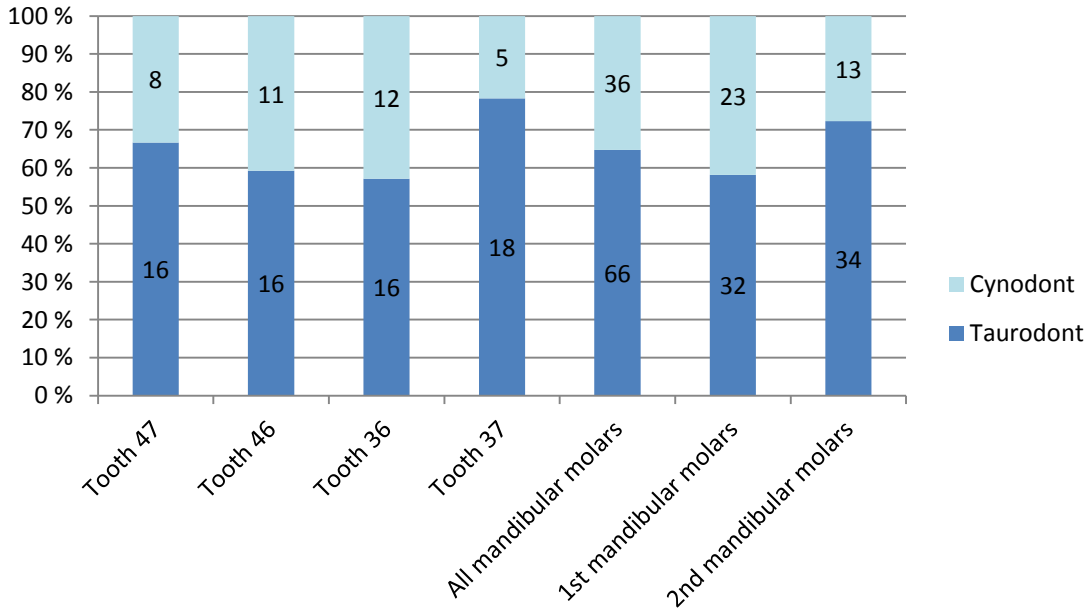


Table 2: The findings of taurodont teeth of the mandibular molars from the radiographic survey at the TAKO-centre 2009. Molars missing in the mandibula were excluded from the statistics, since they could not be diagnosed.

LM/BBS participants with one or more taurodont molars in the mandibula:

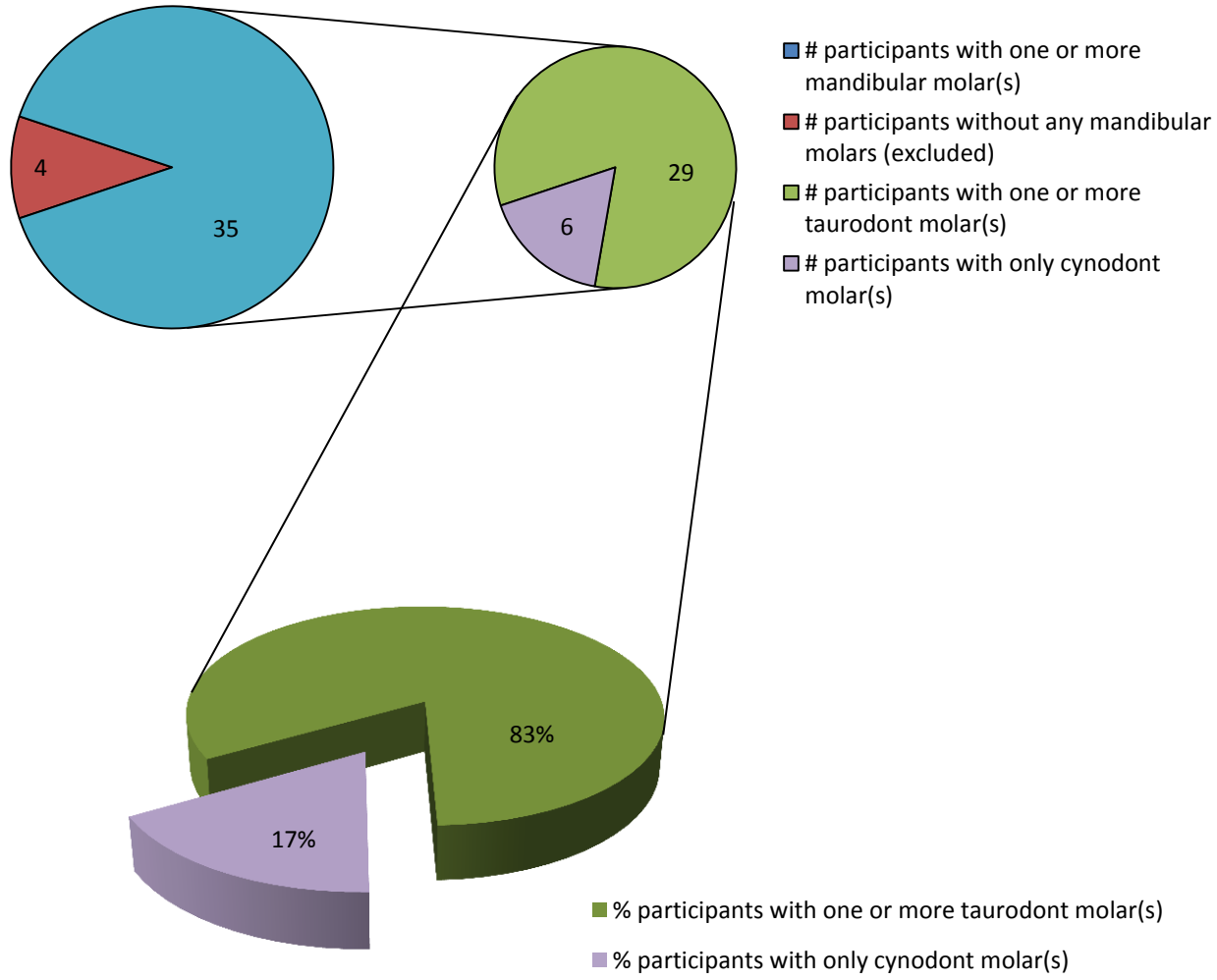


Table 3: The prevalence of taurodontism at individual level in the participants with LM/BBS (Data from the radiographic survey at the TAKO-centre 2009).

Discussion

The 39 participants constituted the whole population of known LM/BBS patients in Norway at the time of examination. Since none of the participants had undergone a genetic screening, we cannot determine whether the patients had Bardet-Biedl syndrome or Laurence-Moon syndrome, making the data somewhat uncertain. However, all were diagnosed by an experienced clinical geneticist based on accepted diagnostic criteria at that time. Of the 102 molars of the mandibula examined, 66 presented as taurodont teeth, giving the LM/BBS population at tooth level, a prevalence of 65 %.
At an individual level, the prevalence of taurodontism (one or more taurodont teeth present in the mandibula per participant) is estimated to 83 %, which is much higher than the prevalence of taurodontism of 0.25-11.3% in the general population (Haskova *et al.*, 2009).

It is highly likely that taurodontism is a trait that should be considered, amongst other dental anomalies, in patients with Laurence-Moon and/or Bardet-Biedl syndromes.

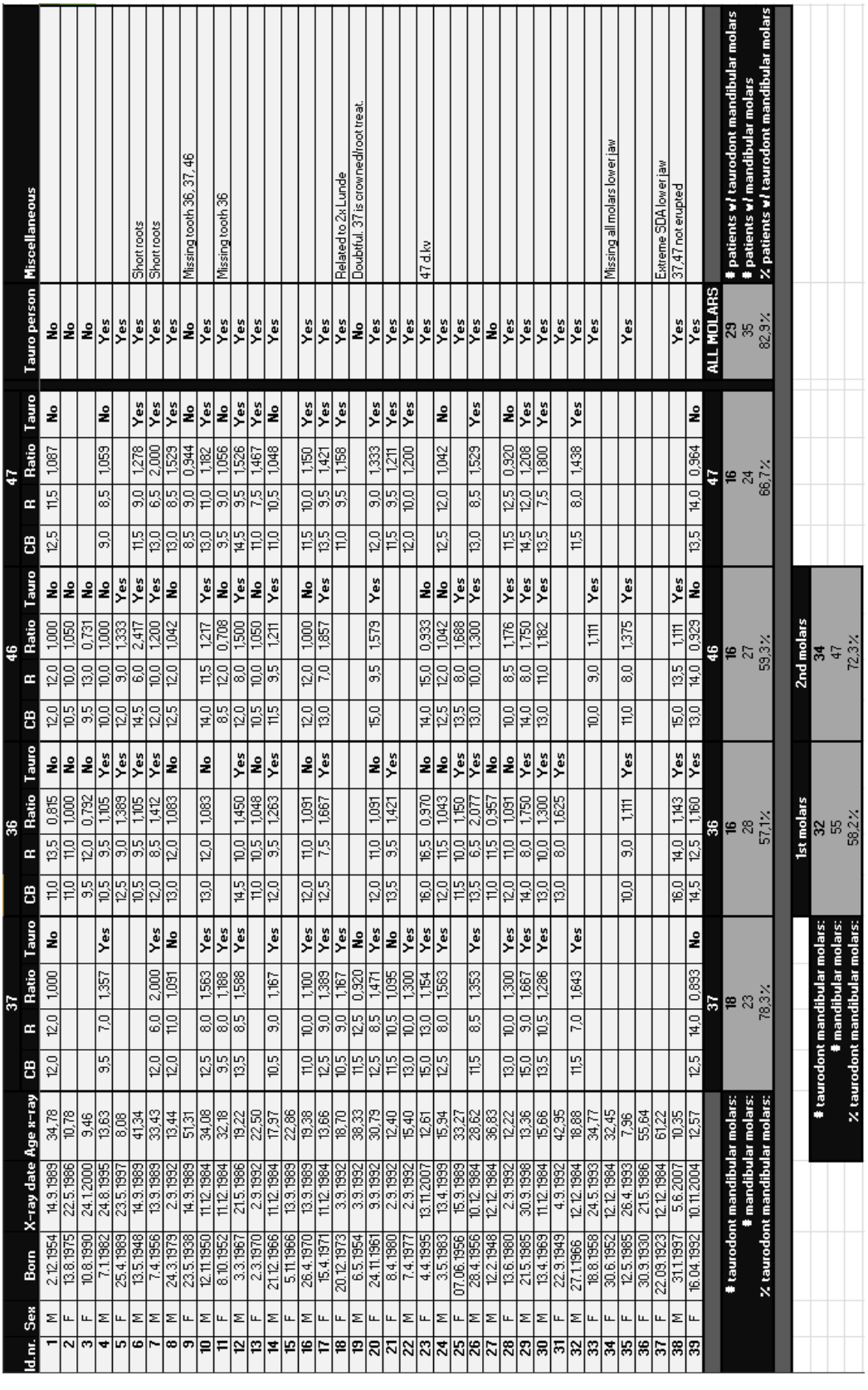
It is recommended that all individuals over the age of 12 years with the two syndromes are referred to a dental clinic for orthopantomograms and a clinical examination to determine presence of taurodontism.

Conclusions

Our study suggests that taurodontism is far more common in Norwegian Laurence-Moon/Bardet-Biedl syndrome patients than in individuals without this syndrome.

We suggest that taurodontism should be included as a minor diagnostic criterion for the Laurence-Moon and/or Bardet-Biedl syndromes.

Data sheet



Id.nr.	Sex	Born	X-ray date	Age x-ray	37 CB	37 R	37 Ratio	37 Tauro	36 CB	36 R	36 Ratio	36 Tauro	46 CB	46 R	46 Ratio	46 Tauro	47 CB	47 R	47 Ratio	47 Tauro	Tauro person	Miscellaneous
1	M	2.12.1954	14.9.1989	34,78	12,0	12,0	1,000	No	11,0	13,5	0,815	No	12,0	12,0	1,000	No	12,5	11,5	1,087	No	No	
2	F	13.8.1975	22.5.1986	10,78					11,0	11,0	1,000	No	10,5	10,0	1,050	No					No	
3	F	10.8.1990	24.1.2000	9,46					9,5	12,0	0,792	No	9,5	13,0	0,731	No					No	
4	M	7.1.1982	24.8.1995	13,63	9,5	7,0	1,357	Yes	10,5	9,5	1,105	Yes	10,0	10,0	1,000	No	9,0	8,5	1,059	No	Yes	
5	F	25.4.1983	23.5.1997	8,08					12,5	9,0	1,389	Yes	12,0	9,0	1,333	Yes					Yes	
6	M	13.5.1948	14.9.1989	41,34					10,5	9,5	1,105	Yes	14,5	6,0	2,417	Yes	11,5	9,0	1,278	Yes	Yes	Short roots
7	M	7.4.1956	13.9.1989	33,43	12,0	6,0	2,000	Yes	12,0	8,5	1,412	Yes	12,0	10,0	1,200	Yes	13,0	6,5	2,000	Yes	Yes	Short roots
8	M	24.3.1979	2.9.1992	13,44	12,0	11,0	1,091	No	13,0	12,0	1,083	No	12,5	12,0	1,042	No	13,0	8,5	1,529	Yes	Yes	
9	F	23.5.1938	14.9.1989	51,31													8,5	9,0	0,944	No	No	Missing tooth 36, 37, 46
10	M	12.11.1950	11.12.1984	34,08	12,5	8,0	1,563	Yes	13,0	12,0	1,083	No	14,0	11,5	1,217	Yes	13,0	11,0	1,182	Yes	Yes	
11	F	8.10.1952	11.12.1984	32,18	9,5	8,0	1,188	Yes					8,5	12,0	0,708	No	9,5	9,0	1,056	No	Yes	Missing tooth 36
12	M	3.3.1967	21.5.1986	19,22	13,5	8,5	1,588	Yes	14,5	10,0	1,450	Yes	12,0	8,0	1,500	Yes	14,5	9,5	1,526	Yes	Yes	
13	F	2.3.1970	2.9.1992	22,50					11,0	10,5	1,048	No	10,0	10,0	1,050	No	11,0	7,5	1,467	Yes	Yes	
14	M	21.12.1966	11.12.1984	17,97	10,5	9,0	1,167	Yes	12,0	9,5	1,263	Yes	11,5	9,5	1,211	Yes	11,0	10,5	1,048	No	Yes	
15	F	5.11.1966	13.9.1989	22,86																		
16	M	26.4.1970	13.9.1989	19,38	11,0	10,0	1,100	Yes	12,0	11,0	1,091	No	12,0	12,0	1,000	No	11,5	10,0	1,150	Yes	Yes	
17	F	15.4.1971	11.12.1984	13,66	12,5	9,0	1,389	Yes	12,5	7,5	1,667	Yes	13,0	7,0	1,857	Yes	13,5	9,5	1,421	Yes	Yes	
18	F	20.12.1973	3.9.1992	18,70	10,5	9,0	1,167	Yes									11,0	9,5	1,158	Yes	Yes	Related to 2x Lunde
19	M	6.5.1954	3.9.1992	38,33	11,5	12,5	0,920	No													No	Doubtful. 37 is crown ed/root treat.
20	F	24.11.1961	9.9.1992	30,79	12,5	8,5	1,471	Yes	12,0	11,0	1,091	No	15,0	9,5	1,579	Yes	12,0	9,0	1,333	Yes	Yes	
21	F	8.4.1980	2.9.1992	12,40	11,5	10,5	1,095	No	13,5	9,5	1,421	Yes					11,5	9,5	1,211	Yes	Yes	
22	M	7.4.1977	2.9.1992	15,40	13,0	10,0	1,300	Yes									12,0	10,0	1,200	Yes	Yes	
23	F	4.4.1995	13.11.2007	12,61	15,0	13,0	1,154	Yes	16,0	16,5	0,970	No	14,0	15,0	0,933	No					Yes	47 d.kv
24	M	3.5.1983	13.4.1999	15,94	12,5	8,0	1,563	Yes	12,0	11,5	1,043	No	12,5	12,0	1,042	No	12,5	12,0	1,042	No	Yes	
25	F	07.06.1956	15.9.1989	33,27					11,5	10,0	1,150	Yes	13,5	8,0	1,688	Yes					Yes	
26	M	28.4.1956	10.12.1984	28,62	11,5	8,5	1,353	Yes	13,5	6,5	2,077	Yes	13,0	10,0	1,300	Yes	13,0	8,5	1,529	Yes	Yes	
27	M	12.2.1948	12.12.1984	36,83					11,0	11,5	0,957	No									No	
28	F	13.6.1980	2.9.1992	12,22	13,0	10,0	1,300	Yes	12,0	11,0	1,091	No	10,0	8,5	1,176	Yes	11,5	12,5	0,920	No	Yes	
29	M	21.5.1985	30.9.1998	13,36	15,0	9,0	1,667	Yes	14,0	8,0	1,750	Yes	14,0	8,0	1,750	Yes	14,5	12,0	1,208	Yes	Yes	
30	M	13.4.1969	11.12.1984	15,66	13,5	10,5	1,286	Yes	13,0	10,0	1,300	Yes	13,0	11,0	1,182	Yes	13,5	7,5	1,800	Yes	Yes	
31	F	22.9.1949	4.9.1992	42,95					13,0	8,0	1,625	Yes									Yes	
32	M	27.1.1966	12.12.1984	18,88	11,5	7,0	1,643	Yes									11,5	8,0	1,438	Yes	Yes	
33	F	18.8.1958	24.5.1993	34,77									10,0	9,0	1,111	Yes					Yes	
34	F	30.6.1952	12.12.1984	32,45																		Missing all molars lower jaw
35	F	12.5.1985	26.4.1993	7,36					10,0	9,0	1,111	Yes	11,0	8,0	1,375	Yes					Yes	
36	F	30.9.1930	21.5.1986	55,64																		
37	F	22.09.1923	12.12.1984	61,22																		Extreme SDA lower jaw
38	M	31.1.1937	5.6.2007	10,35					16,0	14,0	1,143	Yes	15,0	13,5	1,111	Yes					Yes	37,47 not erupted
39	F	16.04.1932	10.11.2004	12,57	12,5	14,0	0,893	No	14,5	12,5	1,160	Yes	13,0	14,0	0,929	No	13,5	14,0	0,964	No	Yes	

	37	36	46	47	ALL MOLARS	
# taurodont mandibular molars:	18	16	16	16	29	# patients w/ taurodont mandibular molars
# mandibular molars:	23	28	27	24	35	# patients w/ mandibular molars
% taurodont mandibular molars:	78,3%	57,1%	59,3%	66,7%	82,9%	% patients w/ taurodont mandibular molars

	1st molars	2nd molars
# taurodont mandibular molars:	32	34
# mandibular molars:	55	47
% taurodont mandibular molars:	58,2%	72,3%

Table 4: The data sheet with registrations per molar per participant (TAKO-centre, Oslo).

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