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The eruption of permanent incisors and first molars in prematurely born children

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SUMMARY The purpose of this work was to examine whether the clinical eruption of the permanent first molars and incisors is affected by pre-term birth. The data applied to 328 prematurely born white and black children and 1804 control children who participated in the Collaborative Perinatal Project (USA) in the early 1960s and 1970s. The dental examinations had been performed in a cross-sectional manner at ages varying from 6 to 12 years in 95 per cent of cases (range 5–14 years). Dental casts were placed in matched pairs by sex, race and conceptional age for comparison of the four clinical stages of eruption of the permanent incisors and first molars between the pre-term and control children.

The results showed significantly ($P < 0.05$) earlier tooth eruption in the pre-term children. The findings suggest that the eruption process is earlier in those teeth that go through a sensitive period circumnatally, being at the pre-eruptive stages and entering the first phases of mineralization under the influence of various neonatal systemic factors and accelerated growth period (catch-up growth) with related unknown mechanisms, which may influence the eruption of the permanent incisors and first molars in prematurely born children.

Introduction

The incidence of pre-term birth varies greatly between populations, being on average in the range 4–15 per cent (Papiernik *et al*., 1985). According to the nomenclature of the World Health Organization (1977), a delivery is pre-term when occurring at a gestational age of less than 37 completed weeks. There are ethnic differences in the rate of pre-term deliveries, which is highest among the Afro-American population of the USA (Savitz *et al*., 1991). Neonatal mortality in very low birthweight infants (birthweight less than 1500 g) has fallen from 50 to 15–20 per cent in the 30 years up to the early 1990s due to improved neonatal treatment, modern perinatal technology, improved socio-economic and educational levels, etc., but morbidity rates are still high (Valkama, 2000). A number of aetiological factors for premature birth exist, many of which are associated with maternal and foetal diseases, but often the cause remains obscure (Yu, 1989), and inborn factors of the child may be of importance. The immaturity of many organs makes a prematurely born infant more prone to neonatal complications and systemic derangements, and there is also interference with the developing teeth, especially those that are at a critical stage at the time of the insult. Permanent incisors and first molars are at the pre-mineralization and pre-eruptive stages and enter the first phases of mineralization before or soon after birth, when systemic factors may influence tooth development. The prevalence of dental defects is significantly higher in prematurely born children than in their full-term counterparts, including enamel hypoplasias, which are reported to exist in both the primary and permanent dentition (Seow, 1996, 1997) and are thought to be associated with a number of systemic derangements typical of pre-term birth, notably hypocalcaemia (Seow *et al*., 1984), metabolic and nutritional disorders, neonatal infections (Funakoshi *et al*., 1981) and respiratory distress syndrome (Johnsen *et al*., 1984). Pre-term delivery is a risk factor for optimal foetal growth, although pre-term infants have been reported to show catch-up growth, i.e. greater than usual rates of growth over a certain period (Elliman *et al*., 1992). The mechanism of this accelerated growth period has not been ascertained, however, and factors influencing general growth and development also seem to concern catch-up growth.

The timing of tooth eruption in prematurely born children has been found to be delayed (Fadavi *et al*., 1992; Viscardi *et al*., 1994), although it has also been reported that maturation of both the primary and permanent dentition does not differ appreciably between pre- and full-term children (Backström *et al*., 2000). The chronological teething age may be delayed in pre-term children, but not when corrected ages are used (Golden *et al*., 1981; Seow *et al*., 1988). Factors thought to be related to delayed tooth eruption are short gestation (Golden *et al*., 1981; Seow *et al*., 1988; Fadavi *et al*., 1992), low birthweight (Trupkin, 1974) and neonatal factors, including complications of prematurity, systemic disorders, duration of oral intubation, average weight gain/day, etc. (Viscardi *et al*., 1994). The nutritional status and growth of the child (post-natal weight gain)

are also important factors affecting the emergence of the primary dentition (Infante and Owen, 1973; Delgado *et al*., 1975).

There are differences in the development of the permanent dentition and in the order of tooth mineralization and emergence between the sexes and between ethnic groups (Haavikko, 1970; Alvesalo, 1971, Harris and McKee, 1990, Heikkinen, 1996). Girls precede boys in reaching the crown mineralization stages (Harris and McKee, 1990) and in root development and tooth eruption (Haavikko, 1970), while blacks are found to achieve the mineralization stages significantly earlier than whites (Harris and McKee, 1990). In general it is assumed that the determinants of the timing of tooth eruption are more genetic than environmental (Hatton, 1950), although tooth eruption includes local controlling factors such as crowding and premature or delayed loss of primary teeth. It has been suggested that in addition to the substantial genetic point in tooth eruption, pre- and post-natal factors may also affect the eruption process. In the study of Friedlander and Bailit (1969) it was pointed out that an early critical stage in tooth development may exist after which most systemic environmental influences cease to have any effect on tooth eruption and the empirical evidence suggests that this critical time probably occurs pre-natally (Main, 1966; Glasstone, 1967). In teeth that develop later, the permanent canines and first premolars, the susceptible stage probably occurs during the first post-natal years, while in the second premolars and molars it is 2 or 3 years later (Heikkinen *et al*., 1995). There are also differences in tooth eruption between the jaws, the mandibular teeth usually erupting earlier than the corresponding maxillary teeth (Nanda, 1960).

With knowledge of the timing and sequence of odontogenesis, it is possible to study developmental events affecting the maturation of the dentition. The aim of this study was to investigate whether clinical eruption of the permanent incisors and first molars is affected by pre-term birth and associated factors.

Subjects and methods

The subjects were 328 pre-term and 1804 control children, a total of 2132. The pre-term group consisted of 60 white children (40 boys and 20 girls) and 268 black children (140 boys and 128 girls), while there were 803 white children (408 boys and 395 girls) and 1001 black children (477 boys and 524 girls) in the control group. The limit for prematurity was placed at 36 gestational weeks in whites and 35 gestational weeks in blacks in order to maintain practical proportions of pre-term/control children in the statistical comparisons. The mean gestational age was 33.7 weeks in the pre-term white boys and 40.4 weeks in their controls, with corresponding figures of 34.6 and 40.6 weeks for the white girls, 31.7 and 39.8 weeks for the black boys and 32.2 and 39.9 weeks for the black girls. The average length of gestation was approximately 9 days shorter for blacks than for whites (Hardy *et al*., 1979; Heikkinen, 1996). The ethnic differences seem to be clearly documented, the duration of pregnancy appearing to be 1 week shorter among black women (Papiernik *et al*., 1990).

The children represented some of the 60 000 pregnancies that comprised the material for the Collaborative Perinatal Study carried out by the National Institute of Neurological Disorders and Stroke (NINDS) in the 1960s. Medical background data, including anamnestic information of the mother from the first registration of the pregnancy and post-natal longitudinal growth data up to the seventh year of age, were obtained and approximately 1500 variables were included (Hardy *et al*., 1979). The gestational age was the time elapsing between the first day of the last menstrual period (LMP) and the date of delivery, counted in days and then transposed to weeks and rounded off to the nearest week. The date of the LMP was ascertained by a special interviewer and the duration of gestation was also based on history and physical findings and an estimate given by an obstetrician and confirmed at each pre-natal visit. LMP data are reliable and useful despite possible errors in dating and obtaining the information (Cushner and Mellits, 1971). The data concerning the type of delivery, including birthweight, birth length, head circumference, etc. were obtained within 1 hour of delivery by the observer of labour and delivery, using calibrated scales (Hardy *et al*., 1979).

The dental examinations were performed in six collaborating medical centres in the 1970s (Buffalo, NY; Richmond, VA; Portland, OR; Philadelphia, PA; Providence, RI; Johns Hopkins, MD) in a cross-sectional manner at ages varying from 6 to 12 years in 95 per cent of cases (range 5–14 years). Alginate impressions were taken at each co-operating centre and plaster casts made as soon as practicable (Hunter and Priest, 1960). All the casts were checked and compared with oral photographs taken from each child at the University of Wisconsin. Teeth with attrition, decay, fillings, orthodontic appliances, etc. on the surfaces were not studied.

The eruption of permanent teeth was determined from normal dental casts by one observer (LA), a member of the genetic odontometric study team at the University of Wisconsin (1972–1974). The tooth antimeres for each child were compared in terms of a four-grade eruption scale, each tooth separately. The eruption stages were: tooth not erupted (0), occlusal surface recently emerged (1), tooth crown half erupted (2), and eruption of tooth complete or nearly complete (3). Defective teeth that could not be readily assigned to an eruption stage were excluded from the analysis. The clinical eruption of each tooth lasts for about 6–12 months in this part of

the life span, and each stage of eruption is easily distinguishable clinically for an experienced observer. The reliability of staging was determined by performing duplicate blind determinations in 200 cases. Interobserver reproducibility of the stages by one observer (TH) was moderately good, with an average of 85 per cent. When pairing the dental casts, the total data were used in order to achieve the maximum number of antimeric pairs in spite of the fact that teeth in the category 0, not erupted, can occasionally be unilaterally missing in varying proportions. It was not possible to ascertain hypodontia by radiographs in these series (Heikkinen *et al*., 2001).

The age of the child in the study was taken as that at which the dental impression had been obtained, and the chronological age was then transposed to the conceptional (calculated) age by reference to the information on the gestational age in both the pre-term and control groups. Pairs were matched for sex, race and age ± 2 months, and the pre-term child was compared with the control child in terms of the four-grade eruption scale for each of the eight incisors and the four first molars, considering each tooth separately. The proportions expressing the binary distribution of antimeric tooth pairs into advanced eruption or equal eruption, were calculated for each tooth. The hypothesis of binomial frequencies for discordant pairs was tested using the method of McNemar (1947).

Results

The results showed significantly earlier eruption of the first permanent molars and permanent incisors in the prematurely born children as compared with the full-term children (Tables 1–4). Eruption was generally earlier for all permanent teeth and for all four pre-term groups, white boys, white girls, black boys and black girls, although differences appeared between the groups in the maxillary and mandibular teeth, and between the teeth on the left and right sides.

Eruption was earlier for all permanent teeth of the prematurely born black boys, significant results being found for the upper first permanent molar on the right side ($P < 0.04$), the upper central incisors ($P < 0.005$) and for all four permanent lateral incisors ($P < 0.05$) (Table 3). Eruption of the upper first permanent molars ($P < 0.05$), the upper central permanent incisors ($P < 0.005$) and all four lateral permanent incisors ($P < 0.05$) was also significantly advanced in the pre-term black girls (Table 4). There were no cases where the eruption had been delayed in pre-term black boys and girls.

In the pre-term white boys, significantly advanced eruption was found for the upper and lower central permanent incisors on the left side ($P < 0.05$ in each case; Table 1), while in the pre-term white girls the eruption of the upper lateral permanent incisor on the right side was significantly earlier ($P < 0.05$; Table 2). The white pre-term boys and girls formed smaller groups than the black children, and the relatively small sample size must be taken into consideration when evaluating their results.

Discussion

Both genetic and environmental factors acting during odontogenesis influence the tooth eruption process. The environment, pre-natal and maternal factors, diseases, nutrition, socio-economic status, climate, etc. may

Table 1 Comparison of permanent tooth eruption in discordant pairs of pre-term and control white boys matched by conceptional age.

Tooth	Pairs (*n*)	Premature advanced	Equal eruption (*n*)	Advanced eruption		McNemar's test	*P* value
				Premature (*n*)	Controls (*n*)		
1 M1	36	(+)	23	7	6	0.077	0.8
2 M1	36	(+)	22	8	6	0.286	0.6
3 M1	36	(+)	23	7	6	0.077	0.8
4 M1	36	(+)	25	7	4	0.818	0.4
1 I1	36	(+)	20	11	5	2.250	0.13
2 I1	36	+*	19	13	4	4.765	0.029*
3 I1	36	+*	24	10	2	5.333	0.021*
4 I1	36	(+)	22	10	4	2.571	0.11
1 I2	36	(+)	24	7	5	0.333	0.6
2 I2	36	(–)	25	5	6	0.091	0.8
3 I2	36	(+)	22	9	5	1.143	0.3
4 I2	36	(+)	22	9	5	1.143	0.3

1 M1, upper right first permanent molar; 2 M1, upper left first permanent molar; 3 M1, lower left first permanent molar; 4 M1, lower right first permanent molar; I1, central permanent incisor; I2, lateral permanent incisor, etc.
*$P < 0.05$; **$P < 0.01$.
+, significantly advanced eruption in premature children; (+), advanced eruption in premature children, but not significant; (–), delayed eruption in premature children, but not significant.

Table 2 Comparison of permanent tooth eruption in discordant pairs of pre-term and control white girls matched by conceptional age.

Tooth	Pairs (*n*)	Premature advanced	Equal eruption (*n*)	Advanced eruption		McNemar's test	*P* value
				Premature (*n*)	Controls (*n*)		
1 M1	18	(+)	11	4	3	0.143	0.7
2 M1	18	(+)	11	5	2	1.286	0.3
3 M1	18	(+)	13	3	2	0.200	0.7
4 M1	18	(–)	13	2	3	0.200	0.7
1 I1	18	(+)	11	6	1	3.571	0.059
2 I1	18	(+)	12	5	1	2.667	0.10
3 I1	18	(+)	15	2	1	0.333	0.6
4 I1	18	(+)	15	2	1	0.333	0.6
1 I2	18	+*	7	9	2	4.455	0.035*
2 I2	18	(+)	8	8	2	3.600	0.058
3 I2	18	(+)	9	6	3	1.000	0.3
4 I2	18	(+)	7	7	4	0.818	0.4

1 M1, upper right first permanent molar; 2 M1, upper left first permanent molar; 3 M1, lower left first permanent molar; 4 M1, lower right first permanent molar; I1, central permanent incisor; I2, lateral permanent incisor, etc.
*$P < 0.05$.
+, significantly advanced eruption in premature children; (+), advanced eruption in premature children, but not significant; (–), delayed eruption in premature children, but not significant.

Table 3 Comparison of permanent tooth eruption in discordant pairs of pre-term and control black boys matched by conceptional age.

Tooth	Pairs (*n*)	Premature advanced	Equal eruption (*n*)	Advanced eruption		McNemar's test	*P* value
				Premature (*n*)	Controls (*n*)		
1 M1	116	+*	63	34	19	4.245	0.039*
2 M1	116	(+)	68	30	18	3.000	0.083
3 M1	116	(+)	85	19	12	1.581	0.2
4 M1	116	(+)	82	21	13	1.882	0.17
1 I1	116	+**	73	31	12	8.395	0.004**
2 I1	116	+**	71	33	12	9.800	0.002**
3 I1	116	(+)	101	10	5	1.667	0.2
4 I1	116	(+)	101	10	5	1.667	0.2
1 I2	116	+*	62	35	19	4.741	0.029*
2 I2	116	+**	59	39	18	7.737	0.005**
3 I2	116	+**	67	34	15	7.367	0.007**
4 I2	116	+**	68	34	14	8.333	0.004**

1 M1, upper right first permanent molar; 2 M1, upper left first permanent molar; 3 M1, lower left first permanent molar; 4 M1, lower right first permanent molar; I1, central permanent incisor; I2, lateral permanent incisor, etc.
*$P < 0.05$; **$P < 0.01$.
+, significantly advanced eruption in premature children; (+), advanced eruption in premature children, but not significant; (–), delayed eruption in premature children, but not significant.

influence the timing of permanent tooth eruption (Nanda, 1960; Friedlander and Bailit, 1969; Rantakallio and Mäkinen, 1983; Heikkinen *et al*., 1995). On the other hand, the chronology of the eruption of the primary teeth is thought to be more extensively genetically determined than that of the permanent dentition (Hatton, 1950; Backström *et al*., 2000).

There are ethnic and sexual differences in the order of emergence and mineralization of teeth, and in the order of tooth development between individuals (Sofaer *et al.*, 1971; Harris and McKee, 1990). Girls precede boys and blacks precede whites in tooth development and eruption (Harris and McKee, 1990), but the standards of tooth emergence vary greatly between populations and may be altered by changing environmental conditions (Nanda, 1960). Differences also exist in tooth eruption between the jaws, the mandibular teeth usually erupting earlier than the corresponding maxillary ones (Nanda, 1960). The ethnic and sexual differences in the timing of permanent tooth eruption are thought to be greater than those in the eruption of primary dentition and it has been suggested that there might be stimulating

Table 4 Comparison of permanent tooth eruption in discordant pairs of pre-term and control black girls matched by conceptional age.

Tooth	Pairs (*n*)	Premature advanced	Equal eruption (*n*)	Advanced eruption		McNemar's test	*P* value
				Premature (*n*)	Controls (*n*)		
1 M1	112	+*	83	20	9	4.172	0.041*
2 M1	112	+*	83	20	9	4.172	0.041*
3 M1	112	(+)	92	14	6	3.200	0.074
4 M1	112	(+)	95	11	6	1.471	0.2
1 I1	112	+**	97	13	2	8.067	0.005**
2 I1	112	+**	98	12	2	7.143	0.008**
3 I1	112		108	4	–	–	–
4 I1	112	(+)	107	4	1	1.800	0.18
1 I2	112	+*	71	28	13	5.488	0.019*
2 I2	112	+*	70	28	14	4.667	0.031*
3 I2	112	+*	82	22	8	6.533	0.011*
4 I2	112	+*	85	20	7	6.259	0.012*

1 M1, upper right first permanent molar; 2 M1, upper left first permanent molar; 3 M1, lower left first permanent molar; 4 M1, lower right first permanent molar; I1, central permanent incisor; I2, lateral permanent incisor, etc.
*$P < 0.05$; **$P < 0.01$.
+, significantly advanced eruption in premature children; (+), advanced eruption in premature children, but not significant; (–), delayed eruption in premature children, but not significant.

pressures in mastication favouring the tooth eruption process (Friedlander and Bailit, 1969).

Various theories have been put forward to explain the tooth eruption process, which is largely unknown. Root growth, dentine formation, proliferation of the dental pulp, the periodontal ligament, the connection between the enamel organ and the oral epithelium and the role of the dental follicle, including its innervation and blood supply, are considered to be essential in the process of tooth eruption (Magnusson, 1968; Cahill and Marks, 1980; Sutton and Graze, 1985). The dental follicle is required for tooth eruption and its role may in part be one of local control of alveolar bone formation and resorption in alveolar tooth eruption (Cahill and Marks, 1980). The presence of the dental follicle is necessary for the tooth to erupt, as shown by Cahill and Marks (1980) and Marks and Cahill (1984), and it has been proposed that a molecular signal initiates the cellular events of eruption, culminating in alveolar bone resorption to form an eruption pathway. Possible molecular signals that may initiate and regulate tooth eruption at the cellular level are epidermal growth factor (Cohen, 1962), transforming growth factor-alpha (Tam, 1985) and colony-stimulating factor-1 (Iizuka *et al*., 1992), which stimulate and accelerate the eruption. Cellular events in the follicle just before or at the onset of eruption include an influx of monocytes and an increase in the number of osteoclasts in the bony crypt to achieve localized bone resorption (Wise *et al*., 1985, 1994; Wise, 1998). The blood vessel thrust theory is a hypothesis regarding the forces producing the eruption of teeth, that emphasizes the blood flow through the vessels of the dental pulp, while the tissues surrounding the tooth produce hydrodynamic and hydrostatic forces towards the tooth within the blood vessels that may partly cause movement of the tooth through the bone (Sutton and Graze, 1985).

Maternal smoking is a risk factor for intrauterine growth retardation and pre-term delivery (Hartikainen-Sorri and Sorri, 1989), and earlier eruption of the primary and permanent teeth has been found in the children of such mothers (Rantakallio and Mäkinen, 1983; Heikkinen *et al*., 1995). An association between birthweight and advanced tooth eruption has been suggested by Bailit *et al.* (1968) and Bailit and Sung (1968), in that heavier children at birth have significantly earlier permanent tooth eruption. Investigations into the role of nutrition and vitamin supplementation in the eruption process suggest that early vitamin D intake has no effect on the maturation of the primary dentition in pre-term children (Backström *et al*., 2000), although children who receive a higher vitamin D dose in the neonatal period have a more mature permanent dentition. It has also been suggested that climate may affect dental maturation, as populations living in tropical climates are found to be dentally more advanced (Friedlander and Bailit, 1969). Tooth emergence has also been assumed to be affected by socio-economic factors, advanced tooth eruption being found among children with superior socio-economic circumstances (Lee *et al.*, 1965).

Low birthweight and premature infants are predisposed to various developmental problems that may potentially affect their growth in early childhood, but it is not certain whether the emergence of the dentition is affected by pre-term birth, low birthweight or both. Harris *et al.*

(1993) found that the development of early forming teeth (permanent incisors and first molars) was significantly delayed in low birthweight children (birthweight less than 2500 g) and individuals with the poorest height-for-age had the greatest delay in tooth formation, but only teeth undergoing rapid differentiation neonatally were systematically affected. Gestational age should be taken into account when estimating the eruption of the dentition in premature infants, because the 'delay' in dental development may be due to the early birth. According to other investigations, premature infants may develop their first teeth at a later chronological age than full-term infants, but no difference has been found between pre- and full-term children in the eruption of the primary teeth based on biological age (Falkner, 1957; Golden *et al*., 1981; Seow *et al*., 1988). Individual differences may exist, in that healthy pre-term infants may have their first tooth erupt at the usual chronological age, but premature infants with prolonged neonatal illness, mechanical ventilation and inadequate nutrition may have delayed tooth emergence even when corrected ages are used (Viscardi *et al*., 1994).

A close correlation has been found between the general growth of the child and the eruption of the primary dentition (Wedgwood and Holt, 1968; Infante and Owen, 1973; Fadavi *et al*., 1992). Dental mineralization has been found to be more related to height and weight than to skeletal mineralization in both sexes, but the stages in tooth development in males are related more to height than weight (Anderson *et al*., 1975). Pre-term infants usually experience a period of catch-up growth in early childhood, even up to 8 years of age among very low birthweight children (Hack *et al*., 1996). Thus, Fadavi *et al.* (1992) observed a delayed tooth eruption pattern in low birthweight, pre-term infants under 24 months of age, whereas after that they appeared to catch up and the eruption pattern did not differ from that in full-term children after 2 years of age. The eruption of the primary dentition was more closely related to the child's growth in height than to chronological age. The effect of catch-up growth was also indicated by Seow (1996), whose pre-term low birthweight children showed a delay of approximately 3 months in dental maturation under 6 years of age, but no significant differences over 9 years of age.

During the early 1960s, when the children in this sample were born, neonatal intensive medical care was not so advanced as today, so that the pre-term cases may differ in some way from prematurely born children in later periods. Neonatal mortality and morbidity rates were higher, and individual characteristics were of considerable importance for the survival of prematurely born neonates. There may also be genetic differences between populations, variation in the magnitude of environmental stress and variation in individual responses to environmental disturbances and prematurity.

Conclusions

The findings suggest that the eruption process is earlier in prematurely born children in the teeth that go through a sensitive period circumnatally (permanent incisors and first molars). Various post-natal factors and an accelerated growth period (catch-up growth) with related unknown factors may influence the eruption of the teeth.

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