Abstract

Low birthweight (LBW) infants are susceptible to several developmental problems (e.g., pulmonary diseases, hyperbilirubinemia, hypocalcemia) with potentially long-lasting effects that slow growth during infancy and childhood. Dental age (DA), judged from stages of permanent tooth mineralization, was scored in 4- to 7-year-old LBW African-American children (N = 66; x = 5.5 years) to test whether they were delayed due to LBW and its consequences. Data were matched in a case-control fashion to African-American children with normal birth-weights (N = 76). Only the early-forming teeth (incisors, first molars) were delayed significantly in their formation. Children with the lowest height-for-age centiles were the most delayed dentally which suggests that height status would improve as dental age caught up with chronological age (CA). Older children were more delayed because there is a proportionately greater opportunity for DA to diverge from CA as children grow older. Since only those teeth undergoing rapid differentiation neonatally were affected systematically, it was speculated that perinatal insults may have an enduring impact on developing primordia, while leaving later-forming teeth unaffected. (Pediatr Dent 15:30–35, 1993)

Delayed tooth formation in low birthweight African-American children

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Introduction

Low birth-weight (<2,500 g, 5 lb 8 oz) can have long-lasting, adverse effects on a child's development, including enamel defects and altered palate morphology.[1-6] LBW can affect growth in at least two ways. Small size at birth can mean that the child will never overcome the initial size deficit, which can affect body size and proportions, craniofacial dimensions, and, possibly, tooth size-arch size relationships. The other complication involves the serious illnesses often suffered by the small infant. Since prematurity is the major cause of LBW,[7-8] the organs typically are immature, and perinatal asphyxia, respiratory distress, apnea, mechanical ventilation, and chronic pulmonary disease are commonplace. These complications, along with hyperbilirubinemia and hypocalcemia, often result in seriously ill, LBW infants. One concern is whether these neonatal problems are transient — and masked by subsequent, compensatory growth — or whether they produce persistent problems manifested as small size and diminished rates of growth.

Previous reports in the dental literature have focused on two oral consequences of LBW, enamel opacities and hypoplasias, and abnormal palate morphology. Enamel defects are much more common in LBW children, affecting both the primary and permanent dentitions. Primary teeth are also at risk[9-11], which suggests that some of the growth disorders seen in LBW children have a prenatal onset. Most defects on the permanent teeth are on the early forming anterior teeth, suggesting that neonatal complications of LBW cause the enamel disturbances, but it also may involve trauma to the developing teeth from laryngoscopy accompanying placement of orotracheal and orogastric tubes.[4, 12, 13]

Tooth formation is a useful measure of the rate of physiologic development ("dental age") compared to a child's chronological development.[14] In this study, dental age was used to assess growth rates in LBW African-American children. African-Americans are the largest minority group in the United States, and prematurity and low birthweight are disproportionately common in this segment of the population.[8]

Materials and Methods

In this retrospective study, only infants weighing less than 2,000 g (4.4 lbs) were selected for examination; this cut point corresponds to the lightest 1–2% of all live births in the United States.[15] Hospital records were obtained on LBW children who would be between about 4 and 7 years of age at our examination. All LBW children (N = 66) were African-Americans born at the Newborn Center, Regional Medical Center at Memphis, Tennessee. Mean age at the oral examination was 5.5 years (SD = 1.50). While birthweight was the selection criterion, these children almost all were born prematurely (<37 weeks): average gestational age was 31.8 weeks (SD = 2.8) with a range of 24 to 40 weeks.

A case-control study design was used. Controls (N = 76) were full-term, normal birth-weight African-Americans from the same hospital, matched for the same age and gender distributions as the LBW series (N = 66). The procedures, possible discomforts or risks, and possible benefits were explained fully to each subject, and informed consent was obtained prior to investigation.

A panoramic film was exposed on each subject as part of a broader evaluation of their oral health status. The development of the crown and/or root of each permanent tooth was scored using the 14 stages described by Moorrees

and coworkers.[14] Films were randomized, and the scorers were blind as to which group a film belonged. All films were scored by two of the authors, and differences were resolved by discussion, or the tooth was eliminated as unscorable. Teeth on both the left and right sides of the arches were scored as an internal check and to test for increased bilateral asymmetry in the LBW group.

Tooth formation proceeds at a significantly faster rate in African-Americans than Caucasian- Americans,[16,17] so the race-specific standards of Harris and McKee[18] were used to assign dental ages. A gender-specific "dental age" was computed for each tooth by taking its stage of formation and assigning the chronological age at which the grade normally occurs. For example, if an African-American female had grade 6 for the maxillary canine (i.e., crown complete but no root formation), the tooth was assigned an age of 4.7 years, which is the average age at which this occurred in the Harris-McKee standards. All 32 permanent teeth were scored for each subject, so far as possible, but third molars were forming so infrequently at this age interval that they are not reported here. The difference between the subject's dental age and chronological age, DA-CA, was determined for all teeth; negative values indicate that the rate of tooth formation was delayed, while positive values suggest an accelerated rate of formation.

Standing height (stature) and body weight were recorded for each subject using conventional anthropometric techniques. To permit pooling of individuals of various ages and genders, height of each subject was transformed to its centile using the *NCHS Growth Curves for Children Birth-18 Years*.[15] Interpolations to the nearest centile were computer-generated. If stature is affected by LBW, then it might follow that the subject's weight also would be affected. Consequently, we followed Roche's approach of expressing body weight as a function of height.[15]

One method of assessing the rate of tooth formation would have been to compare the LBW series to the published standards, since these norms were derived from patient records at the same graduate pediatric dental clinic. As shown, though, a more informative method was to compare the LBW group with the full-term controls. This accounts for minor cohort and regional differences and sampling fluctuations in the published norms.

Age at examination had a significant influence on the difference between chronological and dental age (DA-CA). To correct for this, multiple linear regression was used to standardize the DA-CA values for subject's age (Table 1). Two variables were used, age and age squared, since this combination often was significantly better than age alone in accounting for the regression of age on DA-CA. Factorial analysis of variance then was performed on the residuals having standardized for age at examination. The same results would be obtained using analysis of covariance with age as the covariate.

Table 1. Standardized regression coefficients predicting the difference between dental and chronologic ages (DA-CA) from subject's age using multiple linear regression.

Tooth	*Age at Examination*	*Age² at Examination*
Maxilla		
Central incisor	–0.65	0.41
Lateral incisor	–2.45*	2.01*
Canine	–2.01*	1.71*
First premolar	0.35	–0.71
Second premolar	1.14*	–1.33*
First molar	–1.03*	–0.01
Second molar	0.11	–0.51
Mandible		
Central incisor	–0.42	0.04
Lateral incisor	–1.46*	0.99*
Canine	–1.38*	0.94*
First premolar	0.10	–0.52
Second premolar	0.85	–1.11*
First molar	0.12	–0.35
Second molar	–0.90	0.60

* $P < 0.05$ based on partial F-ratios associated with these standardized regression coefficients.

Results

Height and Weight

Stature and weight-for-height were assessed as comprehensive measures of a child's growth. At the average examination age of 5.5 years, the low birth-weight (LBW) group was significantly shorter than the matched control group ($t = 2.1$; df = 139; $P < 0.05$), suggesting incomplete compensatory growth (Fig 1). In contrast, body weight corrected for height was statistically equivalent in the two groups ($t = 1.0$; df = 139; $P = 0.34$).

We also tested whether those children with the greatest discrepancy between dental and chronological ages were smaller for age (i.e., shorter and/or lighter). All of the correlation coefficients were positive for stature (Table 2), and several (8/14) achieved statistical significance. Particularly for the earlier forming permanent teeth, those who were most delayed in their dental development also tended to have lower-than-average height centiles.

In contrast, tests using weight-for-height were invariably nonsignificant (Table 2), so while growth in stature tended to be delayed when dental age lagged behind chronological age, weight for height did not. It should be noted here that this variable is not the raw weight of the child; it has been standardized against the child's stature.[15]

Dental Development

Age at examination had a significant influence on the magnitude of the difference between dental and chrono-

logical ages. DA-CA tended to be greater in older children. This is related to the relative, proportionate opportunity for DA to diverge from CA as a child becomes older;[19] DA-CA increases as a proportion of age. This does, however, require that DA-CA be evaluated in light of the patient's age.

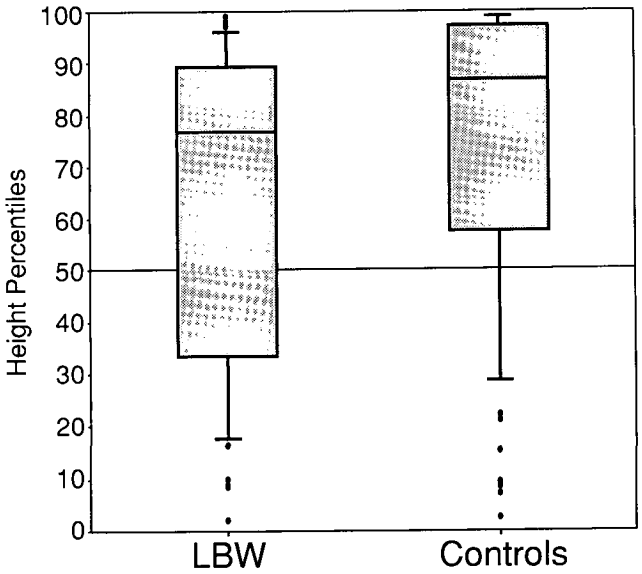

Fig 1. Box plots showing the difference in distributions for stature in the LBW and term control groups. The horizontal lines, from bottom top, define the 10th, 25th, 50th, 75th, and 90th percentiles for each group. Individuals outside the 10th and 90th centiles are shown as solid circles. The LBW group had a significantly lower average stature for age ($t = 2.1$, $P < 0.05$).

Two-way analyses of variance on the DA-CA residuals standardized for subject's age (Table 3) showed that, in most cases (11/14), there was no significant difference between the LBW and control groups (Fig 2). In each of the three instances where a significant difference did occur — maxillary central incisor and first molar and mandibular first molar — the LBW group was *delayed* relative to controls.

Left-right asymmetry was assessed to determine whether the consequences of LBW decreased developmental control. Asymmetry varied by tooth type from 0 to 10%, but, based on Fisher's exact test, there was no occurrence (0/14) where asymmetry was significantly higher in one group than the other.

Discussion

Prematurity (i.e., gestational age less than 37 weeks) is the most frequent cause of low birth-weight.[20] LBW infants are at a disadvantage because their organ systems are immature, which puts them at risk for respiratory distress syndrome with hyaline membrane disease, hyperbilirubinemia, hypocalcemia, anemia, and other disorders that adversely affect health and growth.

Tooth formation is delayed in LBW children, but not in a systemic or uniform manner (Fig 2). Indeed, in most instances, no difference was found between the LBW and control groups, and it is noteworthy that those teeth exhibiting a difference are among the earliest to form, namely the incisors and first molars. Incisors and first molars erupt during what van der Linden and Duterloo[21] have termed the *first transition*. This raises a pertinent issue. One might anticipate that the youngest children in this cross-sectional study are the ones whose permanent incisors and first molars are undergoing rapid formation and that they are contributing to these significant delays in tooth formation. As shown in Fig 3, this is not the case; instead, the *older* LBW children are most delayed.

One speculative explanation is that those tooth primordia that develop perinatally — when growth of the LBW in-

Table 2. Correlation coefficents (r) between DA-minus-CA and the subject's height centile and weight-for-height centile

Tooth	*Height*		*Weight-for-Height*	
	r	*F-ratio**	*r*	*F-ratio*
Maxilla				
Central incisor	0.33	14.8†	0.06	0.5
Lateral incisor	0.33	14.8†	0.10	1.4
Canine	0.17	3.8†	0.07	0.7
First premolar	0.03	0.1	0.12	1.7
Second premolar	0.03	0.1	0.01	0.0
First molar	0.28	10.7†	0.03	0.1
Second molar	0.04	0.1	0.14	2.3
Mandible				
Central incisor	0.27	9.6†	0.13	2.3
Lateral incisor	0.23	7.0†	0.11	1.5
Canine	0.01	0.0	0.04	0.2
First premolar	0.04	0.2	0.01	0.0
Second premolar	0.03	0.1	0.10	1.3
First molar	0.34	16.8†	0.06	0.5
Second molar	0.17	3.6†	0.06	0.4

*The F-ratio tested whether r was significantly different from zero; degrees of freedom were 1 and 129.
† $P < 0.05$.

fant is most compromised because of morbidity — are smaller, less well vascularized, and receive fewer nutrients than later-forming elements. Teeth forming during the critical perinatal period might never overcome these initial insults. While there is no direct evidence for such a scenario, it is compatible with our findings[22] that the permanent incisors and first molars have the smallest mesiodistal crown diameters relative to controls. Analogously, it is those early forming permanent teeth undergoing crown formation (specifically, amelogenesis) perinatally that are most susceptible to enamel dysplasias.[11, 23] The temporal link is that those structures forming at critical periods of development when an insult occurs are most likely to exhibit irreparable size diminutions.[24] Late-forming teeth, such as the premolars, develop when the child is growing more normally generally following whatever compensatory growth is going to occur,[25] and their primordia would be unaffected.

Table 3. F-ratios from two-way analyses of variance testing for differences in DA-CA* between the low birth-weight series and controls and by gender

Tooth	*LBW vs Control*	*Gender*	*Interaction*
Maxilla			
Central incisor	6.2†	2.9	1.1
Lateral incisor	0.0	0.0	2.4
Canine	0.0	1.2	0.0
First premolar	0.3	11.2†	0.0
Second premolar	0.3	3.2	0.7
First molar	8.1†	1.1	1.6
Second molar	0.3	3.4	0.6
Mandible			
Central incisor	2.5	2.2	0.4
Lateral incisor	0.0	1.2	0.0
Canine	3.7	2.0	0.3
First premolar	3.4	3.6	1.4
Second premolar	1.1	9.9†	1.9
First molar	6.1†	4.6†	0.0
Second molar	0.1	2.0	0.2

* Based on residuals of dental age minus chronologic age after standardizing for subject's age examination (see text). "Interaction" tests for non-additivity between group and gender.

† $P < 0.05$ (two-tail tests).

This study also found that overall growth, namely standing height, was delayed in the LBW group (Fig 1). At these examinations (ca. 5.5 years), it was too early to determine whether shorter stature was a permanent consequence of abnormally small size at birth or a transient condition. The observation that those individuals with the smallest height centiles also tended to have the greatest delay in tooth formation suggests the latter, that the long-term prognosis for catch-up growth is good.

Fig 2 shows that, in fact, both series of children have medians significantly above the 50th percentile. This is because the NCHS standards[15] are based predominantly on Caucasian-American children, and African-American

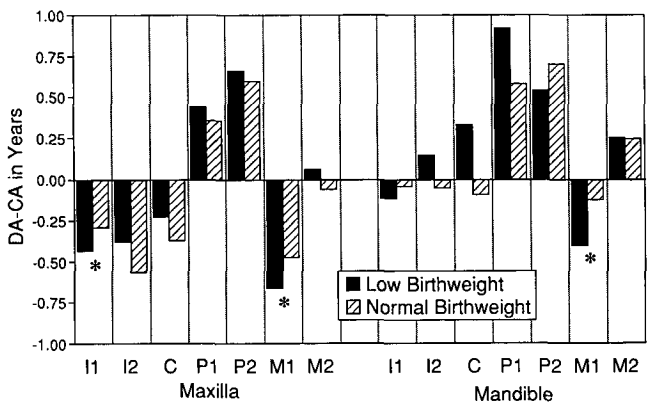

Fig 2. Histogram of the mean differences between dental and chronologic ages (DA-CA) for each tooth. The three comparisons flagged with an asterisk achieved statistical significance, each with the LBW group having delayed dental development.

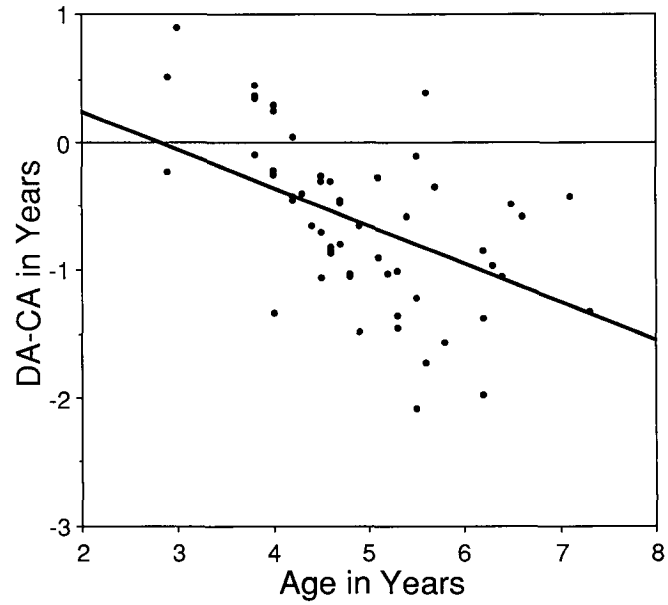

Fig 3. Plot of the age at examination against DA-CA for the maxillary first molar in the LBW group. The greatest disparities between DA and CA occur in the older children. The heavy diagonal line is the least squares best fit; linear regression accounted for 31% of the variation between the two variables.

children are taller and leaner than Caucasian-Americans through early adolescence.[26,27]

The present status and prognosis of the LBW child is much better now that tertiary care centers are in place than in the past.[25] There is much more attention on recognition and prevention of perinatal/neonatal complications.[7] This results in less of an initial growth delay, fewer neonatal complications, and greater likelihood that catch-up growth will mask any growth delays at birth. Clinically this implies that the number and severity of enamel dysplasias should decline as pre- and perinatal care improves, and that persistent effects on childhood growth—including delays of bone age and dental age—should be increasingly subtle. Fitzhardinge[28] and Hack et al.[25] have documented improving status of LBW children as gauged from stature, and this present study confirms that tooth formation is not affected substantially. Only select, early-forming teeth exhibit significant delays in formation.

Summary

Tooth-specific dental ages were compared in a series of low birth-weight (LBW) African-American children (4–7 years of age) and matched term, normal birth-weight controls.

1. Only development of the early-forming teeth (incisors, first molars) differed significantly between the two groups, with the LBW group being significantly delayed. Later-forming teeth were unaffected.
2. In this age span, the *older* children showed greater absolute delays, probably because of the increasing opportunity for dental age to lag behind chronological as a proportion of chronological age.
3. Stature of the LBW children also lagged behind (i.e., incomplete compensatory growth), but those children with the poorest height-for-age tended to exhibit the greatest delays in dental age, so the prognosis for catch up growth seems favorable.

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