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Deciduous Enamel Defects in Prehistoric Americans From Dickson Mounds: Prenatal and Postnatal Stress

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ABSTRACT The month of onset, duration, and incidence of dental enamel hypoplasia and hypocalcification was determined in sub-adults from the Dickson Mounds (Illinois) skeletal series (A.D. 950–1300). The onset of enamel defects occurred predominantly during the intrauterine period, suggesting maternal stress. There are marked differences in survivorship and the duration of enamel disruption in those affected prenatally and postnatally. The relationship between these data and studies of adult dentition is examined.

KEY WORDS Fetus, Neonate, Dental defects, Stress, Methodology

Hypoplastic defects in dental enamel have been used as indicators of ideopathic stress in both living and archaeological populations (El-Najjar et al., 1978). Although they appear to have an inspecific etiology, inclusive of a broad range of nutritional deficiencies (Infant 1974; Shaw et al., 1973; Levy et al., 1970) and diseases (Sarnat et al., 1941; Sweeney et al., 1968; Lindermann 1958; Nikiforuk et al., 1981), hypoplasias and hypocalcification have several advantages relative to other indicators of systemic pathology. Enamel does not remodel after recovery from injury, leaving defects as an indelible longitudinal record of the developmental age at which physiological stress occurred. These defects also provide data on the duration and periodicity of the pathologies that produce physiological changes leading to disturbances in enamel development.

The normal development of dental enamel is regular and sequential (Massler et al., 1941; Schour et al., 1940). Development begins with the cuspal of incisal aspect of the rudimentary dental organ, after which ameloblasts appose successive "rings" (transverse bands) of enamel downward toward the cemento-enamel junction (cervical line bordering with the root) where enamel calcification terminates. Each transverse band consists of microscopic enamel rods produced by ameloblasts moving centrafugally from the dentino-enamel junction toward what will become the crown surface. Rows of these rods form bands (divided by incremental striations) at a constant diurnal rate (4 μm thick/day) (Brand et al, 1977, 214; Bhaskar 1980, 56–57; Goose et al., 1982, 156). Each transverse band or segment of enamel represents a period in the individual's growth and development. Because each type of tooth develops during a different period of months or years, the ages during which transverse sections were apposed can be determined once adjustments are made for the peculiar period of development covered by each type of tooth. Deciduous dental enamel develops during a 16-month period beginning with the incisors by the 5th fetal month and ending during the 10th to 12th postnatal month when the second molars are complete. Secondary dentition begins to calcify at birth with M^1 and ends at 12–16 years with the completion of M^3 (Shaw and Sweeney 1973)[1].

Enamel hypoplasias are transverse linear lesions (which occur as a groove or serial pitting) caused by arrested calcium deposition during the initial phase of enamel development (apposition of the enamel matrix) and involve only the transverse bands of enamel being laid down during metabolic stress. Defective enamel segments or transverse bands occur when disease and/or malnutrition in-

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[1]Studies of odontogenisis (see Logan and Kronfeld, 1933; Schour et al., 1940; Ooe, 1981) show somewhat varied chronologies, which probably reflect methodological and population differences. We have chosen to use the chronologies of Shaw and Sweeney (1976) as the standard in this study, but professionally agreed upon standards are clearly needed.

terfere with the availability of calcium or effect the disorientation, degeneration, or atrophy of ameloblasts during enamel development.

Enamel hypocalcification occurs during the second phase of enamel development (calcification or mineralization). The enamel matrix contains approximately 20% protein, 5–10% calcium salts, and water. During calcification its water content is vastly reduced to 3% as is the organic component (1%); these are replaced by a 96% inorganic calcium-phosphate-carbonate component (Nikiforuk et al., 1981). Hypocalcification produces an opaque (chalky white or yellow-brown) transverse band of enamel resulting from the incorporation of extrinsic pigments. The hypocalcified enamel segment usually involves deeper enamel, leaving a hard outer enamel "skin" intact without the indentation characteristic of arrested matrix apposition (Spounge, 1973:28–29).

Although enamel hypocalcification is morphologically and physiologically distinct, it is closely related to hypoplasia and often appears in adjacent enamel in deciduous teeth (Blakey 1981). As Levy et al. (1970:617) report, unlike dentin and bone, enamel matrix formation and calcification are closely linked.

Timing charts (Massler et al., 1941; Swardstedt, 1966; Goodman et al., 1980a) have been an effective means of plotting the ages at which enamel defects occurred in clinical and archaeological populations. Our investigation involved a more detailed method, by which onset and the duration of the stress period were measured using a mathematical computation of data at each procedural step.

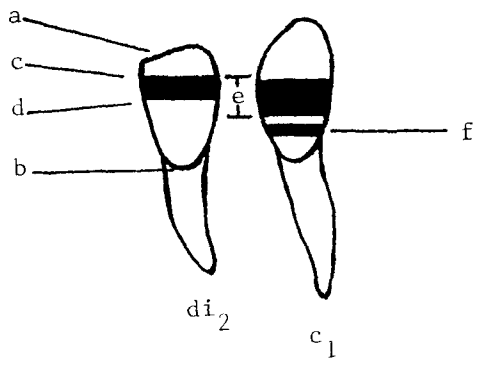

Fig. 1. a = occlusal surface where enamel apposition begins; b = cervical line where enamel development terminates; c = onset of hypocalcified enamel in di_2; d = conclusion of enamel hypocalcification in di_2; e = continuous segments of hypocalcified enamel in di_2 and c_1, representing the period and duration of a primary stress episode in the individual; and f = a discontinuous and secondary episode.

MATERIALS

The sample comprised 50 sub-adults (1–12 years of age) from Dickson Mounds. Dickson Mounds is a burial and habitation site near the junction of the Illinois and Spoon Rivers (about 4.8 km southeast of Lewiston, IL). Harn (1971) has described the environment and the potential exploitation of resources by inhabitants.

Dickson was continuously inhabited during three cultural phases: Late Woodland (A.D. 950–1050), Mississippian Acculturated Late Woodland (A.D. 1050–1200), and Middle Mississippian (A.D. 1200–1300). The Late Woodland hunter-gatherer populations occupied seasonal camps and exploited a broad range of flora and fauna. The Mississippian Acculturated Late Woodland experienced a gradual change in subsistence with the introduction of maize. The Middle Mississippian Period can be characterized by maize agriculture, increased population density, increased sedentarism, social stratification (Harn, 1975), and expansion of trade networks.

At other midwestern American archaeological sites, Cook and Buikstra (1979) found a significant increase in early prenatal stained hypocalcification in the Late Woodland sample compared to the Middle Woodland. Unpigmented hypocalcification with onset late in fetal development was, however, found to be more frequent in the Middle than in the Late Woodland horizon. There were no significant differences in the incidence of hypoplastic lesions, circular caries (related to lesions), or postnatal hypocalcification between cultural groups.

Sciulli (1977) studied deciduous dentition from the Glacial Kame hunter-gatherers (2000 to 1600 B.C.), Adena hunter-gatherers with auxiliary horticulture (1500 B.C., to 300 A.D.), Hopewell agriculturalists (100 B.C. to 400 A.D.), and post-Hopewellian intensive agriculturalists (1100 to 1600 A.D.) in Ohio. The combined Glacial Kame-Adena-Hopewell series had approximately one-third the frequency of defects evident in the post-Hopewell sample. Prenatal stress was common among agriculturalists.

Goodman et al. (1980a), using secondary dentition from Illinois skeletons, have shown an increase in the frequency of individuals with hypoplasias during the cultural evolution of Dickson Mounds. Frequencies increased from 45% (Late Woodland hunter-gatherers) to 60% (Mississippian Acculturated Late Woodland horticulturalists) to 80% among Middle Mississippian agriculturalists. Their frequencies for combined cultures (62% in males and 66% in females) are simi-

lar to our findings in deciduous dentition. They have hypothesized that increased reliance upon protein-deficient maize, population pressure and growth contributed to malnutrition and infectious disease with the intensification of agriculture. Some evidence for seasonality (possibily related to agricultural cycles) in the periodicity of hypoplasias was found (Huss-Ashmore et al., 1982; Goodman et al., 1980a).

This study extends the analysis of dental defects at Dickson Mounds to include deciduous dentition. The sample of 50 sub-adults selected includes skeletons from all cutural periods. Because of the small sub-sample sizes for skeletons of known cultural affiliation, this study focuses on age-specific incidence of physiological stress and mortality trends in fetuses and children, generally, at the Dickson Mounds habitation site.

The sample was selected according to the following criteria: complete crown enamel development and preservation was required of all teeth studied; to include individuals not having defects, at least four non-affected teeth were required; one affected tooth was required to include an individual as showing a defect.

If four unaffected teeth were present, we could be reasonably certain that a defect had not occurred in missing teeth and that the individual was indeed unaffected. Yet, a single affected tooth seemed sufficient to establish the presence of a physiological disturbance. These separate criteria might appear to reduce the number of non-affected individuals eligible for selection because of the greater number of teeth (or standard of preservation) required. That does not appear to have occurred in this study. Only three non-affected individuals were excluded for having fewer than four teeth. Moreover, had a greater number of teeth been required to include individuals with defects, the chance of under-representing these would have been considerable, owing to enhanced caries susceptibility among hypoplastics (Cook and Buikstra, 1979) and subsequent tooth loss.

Teeth were evaluated with the aid of a magnifying glass and probe. Measurements were taken with a sliding (helios) needle-tipped caliper and repeated to determine consistency. The distance from the cervical line (inferior border of completely developed enamel) to the center of the defect was measured if affected enamel was no more than 1.5 mm in width. Because width should correspond to duration, wider defects were measured to their superior and inferior borders (earliest and latest defective band, respectively). In this way the period of defective enamel formation on each tooth was recorded from onset to conclusion. This method is particularly appropriate for measuring hypocalcification, which often spans several developmental months on a single tooth.

METHOD

The first step in determining the timings used in this study is to determine the vertical width of the transverse segment of enamel that develops during a single month. Deciduous canines, for example, usually develop between the 6th prenatal and 9th postnatal month for a period of 13 months (Shaw and Sweeney, 1973). Dividing the canine crown height by 13 gives the fraction of enamel that develops in one month (the monthly enamel segment). This is similar to the method of Rose et al. (1978).

The next step is to determine the timing or age of occurrence of the defective enamel segment. The distance measured from the cervical line to the defect(s) is divided by the width of a monthly segment for that tooth. This becomes an expression of the number of months between the occurrence of the defect and the last month of enamel development. Finally, the number of months is subtracted from the standard developmental month during which enamel is complete for that tooth (the 9th postnatal month for canines), giving the month of occurrence (Table 1). By the same process, the distance to superior and inferior aspects of wide defects gives the age at onset and conclusion of enamel disruption in a given tooth.

All teeth were utilized in this study, which includes defects that occurred during the entire 16-month period of deciduous enamel development. Duration was defined as the period of continuous enamel pathology. Every tooth in a skeleton may not have been defective for the full term of stress in that individual. If different teeth had overlapping periods of pathology that extended toward earlier and later periods, the duration of stress in that individual consisted of the continuous period represented by different teeth in which the timing of defects overlapped. Secondary onset of stress is defined as an additional episode or discontinuous defect that had no overlap with an earlier period of defective enamel (Fig. 1). Secondary onset defects are only included in the analysis of age-related incidence (Fig. 2).

The major problem to be considered in the development of this technique is whether timings should be based upon 1-month enamel segments derived from crown height averages for each type of tooth in the sample, or whether timings for each individual should

TABLE 1. Periods of enamel development in deciduous maxillary teeth[1]

Tooth	Beginning of enamel development (Prenatal months)	Completion of enamel development (Postnatal months)	Duration of enamel development (months)
Central incisors	5th	4th	9
Lateral incisors	5th	5th	10
Canines	6th	9th	13
First molar	5th	6th	11
Second molar	6th	10–12 (11th)	14–16 (15)

[1]This sequence applies approximately to mandibular dentition with only slight variations (adapted from Shaw and Sweeney, 1973).

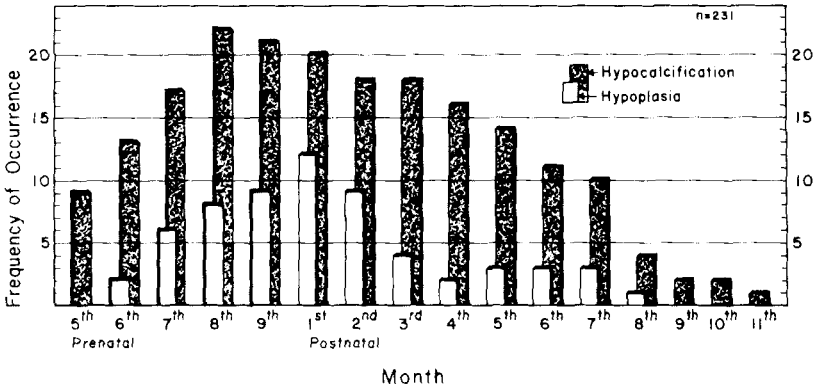

Fig. 2. Incidence of occurrence of defective enamel shown as the number of individuals affected each month.

be based on the crown heights of the individual's teeth. Each involves biases that will be considered here. When individual crown heights of varied size are used and all canines, for example, are assumed to develop for 13 months, the widths of monthly segments vary among individuals with crown height variation. All defect timings will lie within the standard or fixed 13-month developmental period.

However, there is evidence that the period of enamel development does not remain constant, but that the widths of monthly segments remain the same, and the length of development varies with crown height. Male dentition is larger than in females largely because male crown development begins earlier and ends later than in females. Experiments with fluoride intake in rats show that those with high fluoride intake have smaller crowns that develop for a shorter period than rats that were not given fluoride (Moss et al., 1976).

Adjustments for this relationship between length of development and crown height can be made by basing timings on average crown heights for the population, but only at the expense of objectivity in the assessment of age of defect occurrence. The first procedural step involves determining the width of standard monthly segments by dividing the *average* crown height by the standard or *average* period of enamel development (again, in canines the standard period is 13 months). By this procedure, the width of a transverse segment of enamel that develops during 1 month is considered the same in all individuals.

However, in individuals whose teeth are larger than average, some defects may be plotted during a developmental period beyond the usual period of development so that, in effect, the length of the developmental period fluctuates with crown height variation. When a defect appears in a canine enamel segment that is higher on the crown than the average crown height, it will be plotted

earlier than the 6th prenatal month once the number of monthly segments between the cervical line and defect are subtracted from the standard terminal developmental month. Adjustments can be made to distribute the excess period of development over earlier and later periods of development or to consider the excess enamel development as having taken place only in later and not earlier months. Each adjustment changes the timing of defects in teeth of above average size. There is currently, however, no objective means of determining whether the longer period of development involved earlier, later, or earlier and later development. This speculative aspect presents a substantial problem in defect timing using averages.

In canines we find that when using the width of 1-month segments based on average and individual (tooth-specific) crown heights in hypoplastic teeth, defect timing differences are slight. In a sample of 22 measurements of the canines of 11 individuals, the mean difference in defect timing is 0.32 month, a mode of 0.00 month, standard deviation of 0.38 month, and a range from 0.00 to 0.92 months. Therefore, tooth-specific timing in canines is usually within 10 days of timings based upon a sample average with a range of not more than 1 month (earlier or later) or 0.5 month (dispersed over earlier and later periods). Standard deviation in canine height in this sample is 0.41 (0.40 in c^1 and 0.46 in c_1) as compared with those in other teeth, which range from 0.26 in di_1 to 0.56 in di^1.

We have chosen to use tooth-specific or individual crown heights as the basis for timing the age of occurrence of enamel defects in this study for the following reasons: (1) because of the degree of speculation required in timing defects in larger than average teeth; (2) for consistency with most prior research in which timings also do not exceed the standard developmental periods; and (3) because the timing differences between the two methods appear to be slight. This method gives a close approximation of age of occurrence for the purposes of questions addressed in our study. The fact that defects on extremely large and small crowns may be plotted with a bias toward earlier and later developmental months, respectively, should be considered when interpreting these data. The validity of this approach is born out to a considerable degree by the results, which show predictable age-related patterns in defect incidence, duration, and mortality effects. With better data on the relationship between crown height and length of enamel development, methods utilizing uniform monthly segments may be an appropriate choice particularly in populations where differences in crown height are great. Either method may only be possible with deciduous dentition where occlusal attrition is minimal and crown height can be accurately measured.

RESULTS

Nearly two-thirds (64%/n = 32) of the sample had defective enamel. Of these, 93.8% (n = 30) show evidence of hypocalcified enamel. Hypoplasia occurred among 56.3% (n = 18) of affected individuals and 88.9% (n = 16) of those with hypoplasia also had hypocalcified enamel. Hypocalcification occurred alone more often (31%/n = 10) than hypoplasia.

We have used all available teeth in this study. Some researchers prefer to study canines only (or in conjunction with incisors) because of the sensitivity of canine enamel to metabolic stress (Huss-Ashmore et al., 1982). The percentage of individuals with defective canines (61%/n = 11) of all individuals with canines (n = 18) is similar to results using all available teeth.

Defect incidence is shown by age of occurrence in Figure 2. Individuals with defects spanning several months are plotted as having had one episode during each month in which enamel was defective. The incidence of metabolic stress represented by hypoplasia increases over the course of the prenatal period (as do fetal nutritional demands), peaks neonatally, and declines rapidly during the postnatal period. Hypocalcification incidence is highest during the 8th prenatal month and remains high for the first few months after birth. This figure shows that although most individuals had prenatal onset, chronic effects converge in the late prenatal and neonatal months when more individuals are stressed concurrently than at any other age. The pattern is similar in canines, although an additional peak occurs in the 7th postnatal month.

The prevalence of prenatal and maternal stress is reflected more strikingly by the pattern of onset. Although some individuals had more than one episode, only the onset of the first or primary episode is presented below. Over 28% (n = 9) of individuals with defects had primary onset by the 5th month in utero. Onset occurred prenatally in over 71% (n = 23); 78.1% (n = 25) had onset by the end of the first postnatal month; and all but two

affected individuals had onset by the end of the second month.

When only canines are used (which do not record the earliest month of enamel development) prenatal onset occurred in 4 of the 11 individuals with defective canines. Thus, for those who died by the age of 12, the prenatal and neonatal periods were highly stressful.

Further analysis revealed distinct differences in prenatal and neonatal morbidity (duration) and mortality (survivorship) effects. Four categories of defect type were compared for the two onset groups: (1) non-affected, (2) hypocalcified alone, (3) hypoplasia alone, and (4) cases in which there was a combination of hypoplasia and hypocalcification. The age at death of each affected individual by age at primary onset is shown in Figure 3. Those with neonatal onset generally live longer and show more variation in survival. Prenatal onset cases tend not to live as long and show less variability in age at death. Duration and variation in duration (Fig. 4) also differ between the two groups. Early prenatal onset is accompanied by longer and more variable morbidity. The pattern of stress duration in the last two prenatal months is similar to that of the postnatal onset group (less than or equal to 2 months). Yet, age at death and variation in survivorship are consistent with the prenatal period. The late "prenatal" combination of prenatal pattern (early mortality) and neonatal pattern (short duration or acute stress) may represent prematurity. We can presently offer no conclusion. Premature birth is, however, an important contributor to the etiology of hypoplasia in another study (Sweeney et al., 1969).

Survivorship curves demonstrate the need to distinguish between prenatal and perinatal stress before attempting to show a relationship between *defect type* and mortality. Prenatal and postnatal onset groups are combined in Figure 5. While non-affected cases survive the longest, those with hypoplasias appear to outlive substantially those with hypocalcification alone. This can be misleading. The extended survivorship of those with hypoplastic lesions in Figure 5 is a product of extended lifespan in the neonatal onset group in which there is a disproportionate number of hypoplasia cases. Survivorship for those with prenatal onset only (including possible prematuri) is shown in Figure 6. The survivorship of those with both defect types is virtually the same. Survivorship in individuals with enamel defects closely follows that for children with porotic hyperostosis and infectious diseases at Dickson Mounds (Bickerton, 1979).

DISCUSSION

Sarnat and Schour (1941) in one of the earliest studies of living populations found two-thirds of hypoplasias had occurred during in-

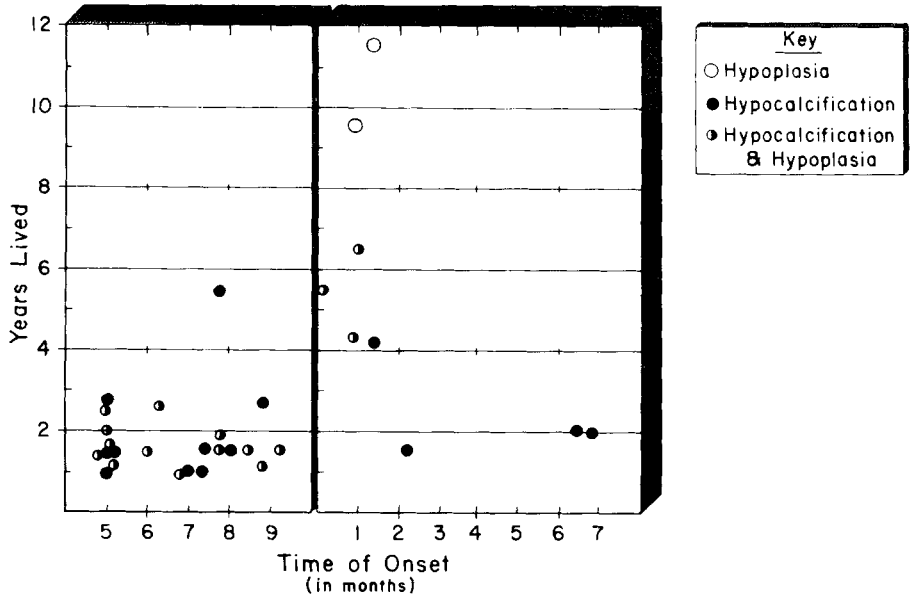

Fig. 3. Variation in age at death in relation to month of stress onset and defect type (n = 32).

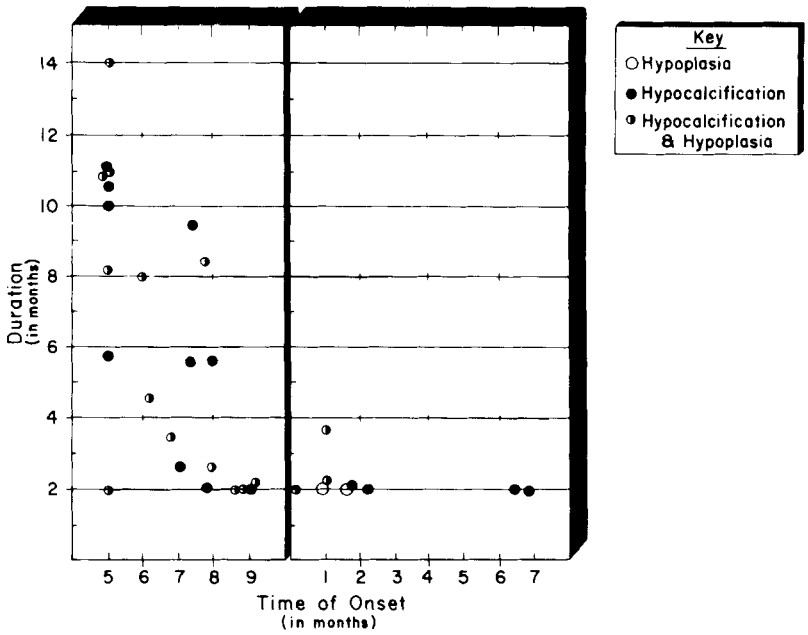

Fig. 4. Variation in the duration of enamel disruption in relation to the month of stress onset and defect type (n = 32). Episodes spanning 1.5 mm or less in vertical enamel width have been approximated at 2 months or less in duration.

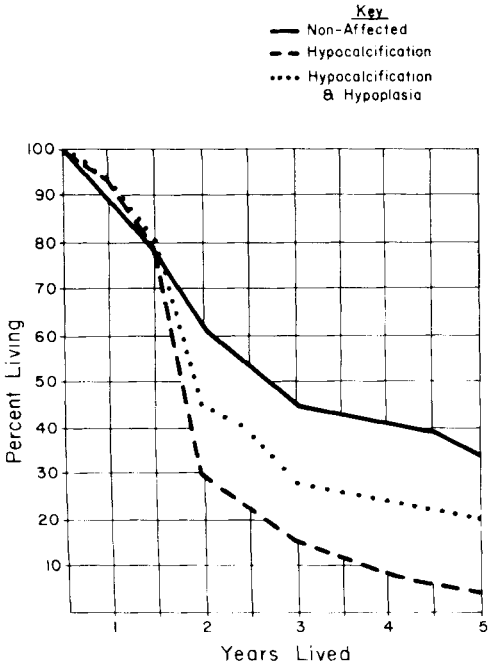

Fig. 5. Survivorship of prenatal and postnatal onset groups (combined) compared with the non-affected group (n = 50). The hypocalcification and hypoplasia sub-sample shown here includes those with combined defects and the only two individuals with hypoplasia alone.

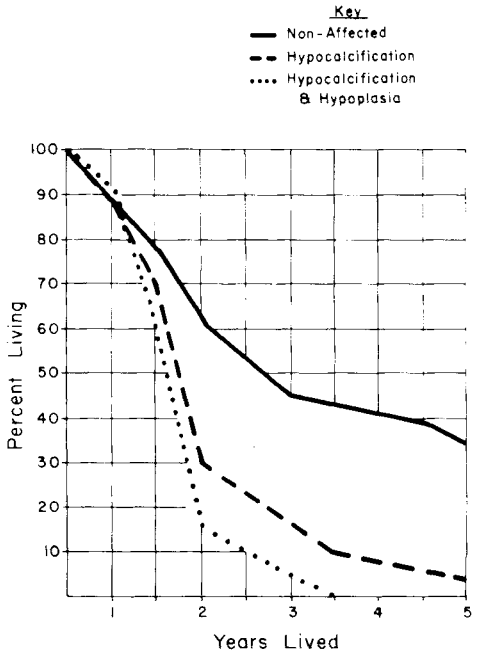

Fig. 6. Survivorship of prenatal onset groups compared with the non-affected group (n = 41).

fancy. A clinical study by Sweeney et al. (1969) showed hypoplasias in 42.5% of rural Guatemalan children. Eighty percent of children from Santa Maria Cauque who were born prematurely had infections in their first month. Seventy percent of these children had hypoplastic lesions (1969). Sweeney et al. attribute this incidence to low cord blood levels of vitamin A in children of malnourished mothers acting synergistically with infections. Massler et al. (1941), using a sample of children, also find the highest frequency of hypoplasias and striae of Retzius (enamel microdefects) during infancy. These clinical studies of living children suggest a higher incidence of dental defects during the first year than has been found in skeletal studies using adult dentition as a record of infant and childhood stress.

Hypoplastic studies using secondary dentition in archaeological populations have shown the highest frequencies of hypoplastic defects between the ages of 4 and 5 years in California Indians (Schultz and McHenry, 1975) and between 2 and 4 years at Dickson Mounds (Goodman et al., 1980b) and at the Westerhaus site studied by Swardstedt (1966). Incidence in the first year is lowest in these studies. Less than 5% of the Dickson Mounds and California adult series had defects in infancy (Huss-Ashmore, 1982).

Studies of archaeological populations in which deciduous dentition is used reveal more infant stress than do studies of secondary dentition. In this study, the incidence of hypocalcification increased during gestation, decreasing to 40% (n = 20) of all individuals in the neonatal period, and declines incrementally after. The age pattern for stained hypocalcification in the Middle Woodland in the study by Cook et al. (1979) is similar to the pattern at Dickson Mounds. Their age-specific incidence for hypoplasias, however, shows a different trend.

Sciulli (1977) in a small post-Hopewell series found a pattern in age-specific incidence for individuals with hypoplasias similar to the one found here. Twenty-three percent (n = 3) of individuals were affected prenatally, decreasing to 15% (n = 2) in the first four postnatal months without subsequent incidence. The hunter-gatherer or mixed subsistence groups in that study showed no prenatal defects; 7% (n = 2) were affected during the first four months, declining to 3% (n = 1) between the 4th and 10th months. Our data on hypoplasias in Figure 2 show the highest incidence (24%/n = 12) neonatally with rapid decline to 4% (n = 2) by the 4th postnatal month. These frequencies of infant stress are high compared to those found in adult skeletal series.

CONCLUSIONS

The greatest change in mortality occurs at 1–2 years in sub-adults with defective enamel. Interestingly, the ages at which the most rapid increase in the incidence of defects was shown in the adult series at Dickson Mounds is concurrent (1–3 years) with the ages of accelerated mortality in children (Huss-Ashmore et al., 1982). The years during which the highest incidence was recorded using adult dentition (2–4 years of age) encompasses the ages during which the majority of children with deciduous defects have died.

The more obvious nutritional stressors between the ages of 1 and 4 years should be considered. Prolonged lactation and delayed weaning up to 3–4 years are not uncommon in malnourished populations. Prolonged lactation/nursing becomes inadequate to the increasing protein requirements of children over time (Harrison et al., 1970:429, 438–9). The weanling diet that follows (maize gruel) may also have been protein poor. These conditions would have contributed to protracted malnutrition and immune susceptibility to disease (Scrimshaw et al., 1968).

Those with prenatal onset appear to have been at higher mortality risk during this period than those with postnatal onset. Nearly all of our sample with prenatal onset died between the ages of 1 and 3 years, while most of those with postnatal onset survived past their 4th year. Although we do not know the prenatal experience of surviving adults, the low frequency of infancy-derived defects in adult skeletons suggests that those who survived to adulthood experienced primary onset predominantly during the period of prolonged lactation/nursing, weanling, and post-weanling events. Therefore, those stressors that caused a high incidence of defects in survivors may have been sufficient to cause mortality in those who had been stressed previously, especially those with prenatal onset stress.

This interpretation explains the differences in the frequencies of infant stress recorded in deciduous and adult dentition. Most of those stressed during infancy are not surviving long enough to erupt secondary dentition nor to enter the adult population. Studies of secondary dentition, as Schulz and

McHenry (1975) have suggested, under-represent infancy-derived enamel defects.

Methodological differences should also be considered. The "best tooth" method (in which only secondary canines and incisors are examined) has been shown to be an effective strategy for studying frequencies of hypoplastic lesions in adults, providing a record of 95% of lesions found using all available teeth (Goodman et al., 1980a). The first tooth to develop, secondary incisors, however, does not begin crown development until the 3–4 postnatal month. In this study, most infant hypoplastic episodes had subsided prior to the 4th month. The first permanent molar, which begins development at birth, should be included in studies of infant stress when possible.

In summary, dental enamel defects are the only skeletal indicators that currently provide retrospective data on previous age of occurrence and duration of morbidity. This study shows that infants, fetuses, and mothers were frequently under stress sufficient to produce macro-enamel defects, presumably linked to malnutrition and disease. Fetal onset morbidity was usually chronic, highly variable, and often continued into infancy. Primary neonatal onset cases were usually more acute and were associated with longer and more variable survival. The health of mothers appears to have had the most marked effect on the health of children by placing them at risk. Comparisons with rates of pathologies in women of childbearing ages at Dickson Mounds would seem a logical avenue for future research. By inference from demographic trends, prolonged lactation/nursing and poor weanling diets acted as secondary contributors to early childhood mortality.

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