

Defective Enamel Histology of Prehistoric Teeth from Illinois

JEROME C. ROSE

*Department of Anthropology, University of Arkansas, Fayetteville,
Arkansas 72701*

KEY WORDS Enamel histology · Pathological bands · Prehistoric American Indians

ABSTRACT Histological enamel defects have been used as indicators of childhood morbidity and nutritional inadequacy. However, the usefulness of these defects has been hampered by a lack of clear criteria for differentiating normal and defective enamel. This report demonstrates that the criteria of abnormal prism structure can accurately differentiate defective enamel (i.e., pathological bands) from normal enamel. In addition, pathological bands can be divided into three distinct subtypes: distorted structure bands, black spot pathological bands, and structureless pathological bands.

It has been assumed that the patterning of pathological bands and enamel hypoplasia is the same for all populations. Comparisons between populations show that each population has its own unique pattern. It has also been assumed that striae of Retzius, pathological bands, and enamel hypoplasias represent three grades of severity of the same phenomenon. Correlations between these three features demonstrate instead that this patterning is possibly influenced by the morphology of the teeth.

Extensive research has shown that macroscopic linear enamel hypoplasia, a dental defect common to both human and nonhuman primates, is significantly correlated with morbidity and nutritional inadequacy (Baume and Meyer, '66; Giro, '47; Grahnen and Edlund, '67; Herman and McDonald, '63; Sarnat and Schour, '41, '42). Disturbances in enamel prism formation are also associated with episodes of morbidity and nutritional disturbances in man (Massler et al., '41; Watson et al., '64). Because of their relationship with disease, together with their chronological specificity and antiquity, these defects have been used in paleopathological studies to assess stress and disease experience (Falin, '61; Moorrees, '57; Sognaes, '55, '56).

Despite this research, paleopathological studies have been hindered by the absence of consistent criteria for differentiating normal and defective enamel. This lack of agreement as to what constitutes defective enamel has prevented the collection of

data which permit comparisons between studies.

Three major light microscope techniques have been used to detect defects in enamel: (1) assays of enamel pigmentation (Molar and Ward, '75); (2) polarized light analysis (Gustafson and Gustafson, '67); and (3) microscopic identification of abnormal enamel prism structure (Wilson and Shroff, '70). Whether or not these methods yield compatible results has never been systematically tested.

Enamel hypoplasia, abnormal prism structure, and pronounced striae of Retzius have all been used as morbidity indicators. In fact, these features have been occasionally combined to produce single measures of morbidity even though their interrelationships have never been adequately tested (Massler et al., '41). Also, although their chronological distributions within specific populations is not known, the frequencies of these features have been compared between populations (Falin, '61;

TABLE 1

One-half year age intervals	Enamel units	Striae			Pathological bands			Hypoplasias		
	Number	Number	Percent	Mean	Number	Percent	Mean	Number	Percent	Mean
0.5-0.9	22	8	0.3	0.36	1	4.3	0.045	0	0.0	0.000
1.0-1.4	45	45	1.6	1.00	1	4.3	0.022	0	0.0	0.000
1.5-1.9	63	136	5.0	2.16	4	17.4	0.063	0	0.0	0.000
2.0-2.4	72	357	13.1	4.96	6	26.1	0.083	0	0.0	0.000
2.5-2.9	89	600	22.0	6.74	4	17.4	0.045	3	11.5	0.034
3.0-3.4	98	751	27.5	7.66	5	21.7	0.051	14	53.8	0.143
3.5-3.9	99	501	18.4	5.06	1	4.3	0.010	7	26.9	0.071
4.0-4.4	98	331	12.1	3.38	1	4.3	0.010	2	7.7	0.020
Total	586	2729			23			26		

Table one presents the number of enamel units observed for each one-half year period of canine development, as well as the incidence of striae of Retzius, pathological bands, and hypoplasias. The three enamel features are also recorded as a percentage of the total observations and the mean incidence for each one-half year age interval.

Molnar and Ward, '75; Sognaes, '56). Similarly, the influence of the morphology of different classes of teeth on the chronological distributions of these features is unknown. Yet, frequency data collected from different classes of teeth have been combined in populational comparisons (Falin, '61; Molnar and Ward, '75).

This report attempts to rectify these problems in the following ways: the concordance among the three observational techniques is tested; the chronological distributions of the enamel features is compared between populations; and the interrelationships among the enamel features within the mandibular canine is described. To accomplish these objectives, 105 individual permanent canines from a prehistoric cemetery are analyzed.

MATERIALS AND METHODS

One hundred and five individual permanent mandibular canines, representing 41 adult males, 46 adult females, and 18 juveniles were randomly selected from the Dickson Mounds cemetery. Each canine was cleaned in alcohol, measured, photographed, and embedded in Bioplastic (Ward's Natural Science Establishment, Inc.). The cutting and grinding procedures were modified from Bohatirchuk ('57) and Sognaes ('47). A longitudinal buccal-lingual slice 1-mm thick was cut from the center of each canine, and affixed to a petrographic slide with double sided

scotch tape. The slices were ground flat with a series of abrasive papers (i.e., Buehler Ltd. Carbimet 240, 320, 400, 600) on a motorized brass wheel. Polishing was initiated with 0000 emery paper and kerosene and completed with a microcloth (Buehler Ltd.) and Alpha polishing alumina (0.03 μ m). The specimens were re-mounted, polished surface down, with epoxy, and the above grinding and polishing procedure was repeated, producing a 100-150 μ m section. The specimens were etched in 1 N hydrochloric acid for 15 seconds, which enables the observer to focus only on the surface of each section. A low magnification (1.2 \times) photograph of each section was used to mark the location of enamel structures. Using the procedure described by Massler et al. ('41) the dentino-enamel junctions were divided into eight equal segments, on the photographs, each approximating one-half year of growth.

The number of striae of Retzius in each half-year enamel unit was recorded for each canine. These observations were all made using a 4 \times objective and repeated until consistent results were obtained. The intensity of pigmentation of each striae was graded as either light (i.e., faint, just observable), medium (i.e., easily observable), or dark (i.e., heavy opaque stain). The number of abnormal enamel prisms within each striae was recorded. Polarized light was used to estimate mineralization (after Gustafson, '59), and the striae were clas-

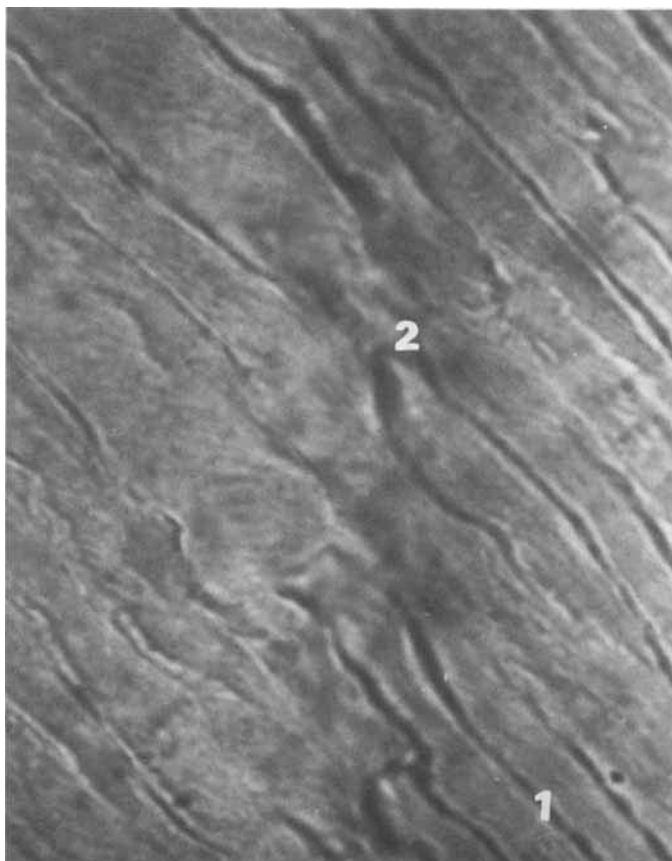


Fig. 1 Distorted structure band, (1) indicates normal prism boundary, (2) indicates distorted prism boundary. $\times 1,000$.

sified as hypocalcified, normal, or hypercalcified.

The canines were divided into six subsamples using the criteria of sex (i.e., male, female, and unsexed juveniles), and archaeological period (i.e., early and late) based on grave furniture and stratigraphy. The frequencies of enamel one-half year units, striae, pathological bands, and hypoplasias were tabulated and compared between each of the six subsamples. As no significant difference ($p > 0.05$) was found for any feature between any two subsamples, they were combined for analysis.

RESULTS

Evaluation of the correspondence of the three observational techniques showed no correlation between the intensity of pig-

mentation and the quality of calcification and only a slight association between pigmentation and abnormal structure. The dark bands had a higher mean percentage of distorted prisms (73.8%) than both medium (60.6%) and light (37.8%) bands. However, because polarized light analysis showed no correlation between quality of calcification and abnormal prism structure, this report is confined to an analysis of abnormal prism structure, for which there are no frequency data reported.

In longitudinal sections, the striae of Retzius are observed as bands of brown stain running obliquely from the dentino-enamel junction to the surface of the tooth. These bands seen at low power are the result of the superposition through the thickness of the enamel of optically dense areas



Fig. 2 Black spot pathological band, (1) indicates black structure, (2) indicates normal prism. $\times 400$.

localized along the prism borders. The edges of longitudinally sectioned prisms are straight with minor fluctuations resulting from the undulation of the prisms in and out of the plane of section.

There are 2,729 striae distributed among 586 one-half year enamel units observed in this study, with 99.2% classified as having normal prism structure. The mean frequency of striae within each one-half year age interval (table 1) gradually increases from the 0.5-0.9 to 3.0-3.5 intervals and then decreases.

Pathological bands (after Wilson and Shroff, '70), which comprise 0.8% ($N = 23$) of all striae observed in this study, are divided into three distinct subtypes: distorted structure bands, black spot patho-

logical bands, and structureless pathological bands. Distorted structure bands represent 78.2% ($N = 18$) of all the pathological bands and are recognized as a striae of Retizus with distortion of the prism borders outside the normal range of variation (fig. 1). This complex pattern of crossing and intertwining prism boundaries is indicative of disturbed ameloblast metabolism (Kreshover, '40).

Black spot pathological bands comprise 17.4% ($N = 4$) of observed pathological bands (fig. 2), and exhibit the same structural disturbances as distorted structure bands with the additional feature of amorphous black spaces distributed through the thickness of the enamel along the striae. These irregularly shaped structures range



Fig. 3 Structureless pathological band, (1) indicates normal prism and direction (2) indicates structureless area. $\times 400$.

between 2.5 and 7.5 μm in diameter, and are located in the boundary area between adjacent prisms. Observation with the scanning electron microscope shows these structures to be cavities located between the prisms.

Structureless pathological bands represent 4.4% ($N = 1$) of the pathological bands in this study (fig. 3), and are characterized by an area with no normal structure along the striae. Where structure is observed it runs perpendicular to the normal prism direction. This disturbed enamel is tentatively interpreted as an area of abnormal apatite crystalline orientation (Miyoshi et al., '72).

Because of the low incidence of observed pathological bands, the three sub-

types have been combined for analysis. Their frequencies are reported as a percentage of observed half year age intervals. Their greatest concentration occurs between one and a half and three and one-half years in the middle two quarters of the canine crown.

Enamel hypoplasia is macroscopically observed as a rough transverse groove with pitting of the enamel surface. It is here defined microscopically by a deficiency of enamel thickness, convergence of the striae of Retzius, and an absence of observable prism structure along the enamel surface (fig. 4). The incidence of hypoplasias is presented as the proportion of affected enamel units over observed units for each one-half year age interval. All hypoplasias

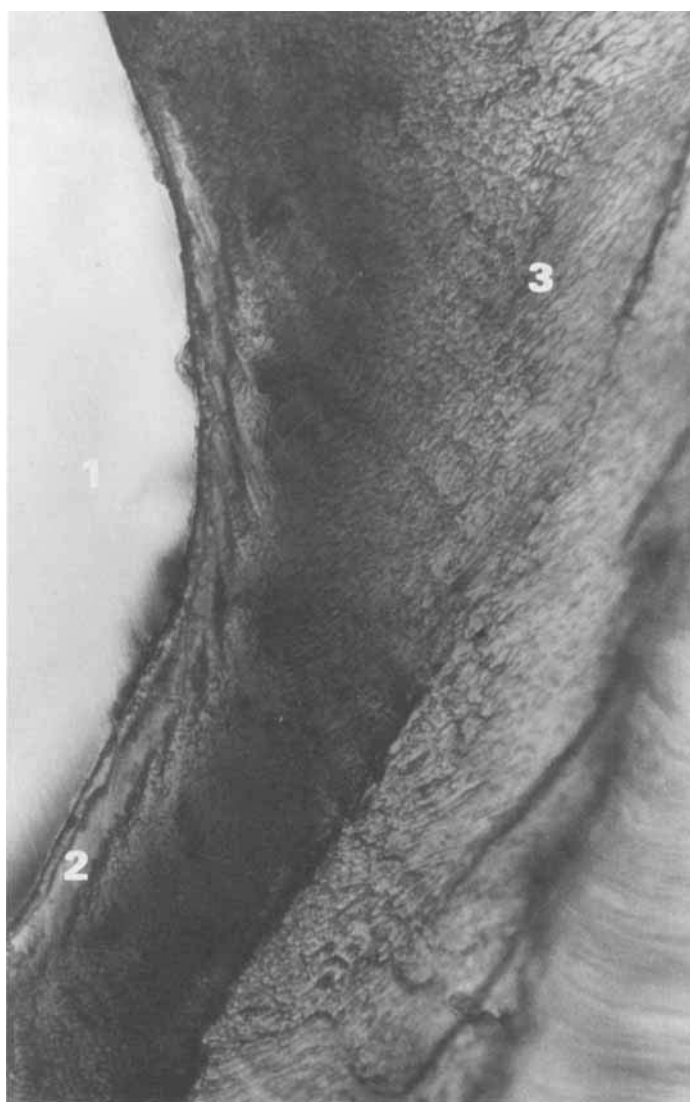


Fig. 4 Microscopic enamel hypoplasia, (1) indicates area of enamel deficiency, (2) featureless enamel, and (3) normal enamel. $\times 40$.

are confined to the cervical half of the canine crown with a mode between three and three and one-half years.

DISCUSSION

It is necessary to analyze the interrelationship and chronological distribution of hypoplasias, pathological bands, and striae in order to understand the complex factors producing these features. Massler et al.

(41) published the chronological distribution of histological enamel structures for American whites, utilizing the frequencies of striae of Retzius and enamel hypoplasias in all tooth classes to evaluate the quality of enamel calcification. In the Massler study the infancy period (0-1.0 years) is characterized by high frequencies of striae of Retzius and the occurrence of 70.0% of the hypoplasias; early childhood (1.0-3.0

years) by fewer striae of Retzius and 5.0-10.0% of the hypoplasias; the late childhood (3.0-6.0 years) by an increase in striae of Retzius with hypoplasias remaining at 5.0-10.0%.

The present canine data, on the other hand, show an entirely different pattern. The comparable infancy period (0.5-1.0 years) has the lowest frequency of striae and no evidence of hypoplasia. Instead of decreasing during early childhood, the frequency of striae gradually increases. The proportion of hypoplasias (11.5%) is not greatly different, but they are all found during the last half year interval (i.e., 2.5-3.0 years). The frequency of striae peaks early in the later childhood period and gradually declines, but, as in the modern sample, the mean frequency of striae remains higher than the previous period. Another important difference is that whereas hypoplasias are rare during late childhood in the modern sample, 88.5% of the prehistoric hypoplasias occur during this period.

In summary, there are two major differences in the patterning of these enamel features. In the modern samples, approximately two thirds of the hypoplasias are in the cuspal half of the canine, while hypoplasias are only found in the cervical half in the prehistoric sample. The modern striae are bimodally distributed with peaks during infancy and late childhood, whereas in the prehistoric sample they are distributed in a simple curve with a mode between three and three and one-half years. If Sarnat and Schour ('41) are correct in attributing the pattern of enamel features to genetically controlled growth patterns, then all populations should display similar patterns. Thus, it seems logical that cultural factors such as the age of weaning, weaning diet, and other determinants of childhood disease and nutritional patterns are intimately involved in producing these differences in the chronological distribution of enamel features.

Theoretically, the occurrence of striae of Retzius, pathological bands, and enamel hypoplasias should be intercorrelated.

Massler et al. ('41) utilize both the frequency of striae and hypoplasias to estimate the quality of enamel calcification, and thus imply a relationship between the two. Kreshover's ('40) study of the histopathology of hypoplasia indicates that pathological bands and hypoplasias represent increasing grades of severity of ameloblast injury. Kronfeld ('39) also indicates a relationship between pathological bands and hypoplasia when he describes lines of injury extending from the hypoplastic lesion into the deeper enamel.

Examination of the age distributions of striae and pathological bands reveals major differences. Utilizing Spearman's rank correlation coefficient (Siegal, '56), the two distributions have a nonsignificant ($p > 0.05$) correlation of 0.303. Consequently, the metabolic disturbances which produce pathological bands are not strongly influenced by events determining the distribution of the striae of Retzius. If, as Osborn ('71; Gaunt et al., '71) implies, the distribution of striae is influenced by the morphology of the teeth, then morphology does not strongly influence the distribution of pathological bands.

Similarly, despite their theoretical association, the distributions of hypoplasias and pathological bands are different. In fact, only one hypoplasia in this study is associated with a pathological band, and the correlation is small (0.270) and non-significant ($p > 0.05$). This result indicates that some unknown factor determines whether a metabolic disturbance produces a hypoplasia or pathological band.

Similarities in the distributions of striae and hypoplasias suggest that this unknown factor may be related to the structure of the teeth. However, the significant ($p > 0.05$) correlation of 0.857 between the distributions of striae and hypoplasias does not explain why hypoplasias are only found in the cervical half of the canine. The distribution of macroscopically observed hypoplasias recorded from all the teeth of each individual in the cemetery indicates that hypoplasias do occur on other teeth during the first half of canine formation

(i.e., 0.5-2.5 years), but not on the canine itself. One of several possible explanations is that ameloblasts producing the thinner cervical enamel are more sensitive to disturbances which cause hypoplasia. Another possibility is that as the time differential between initial enamel formation in the canine and the commencement of amelogenesis in the lower portion of the canine crown increases, the ameloblasts are more susceptible to damage. These alternatives cannot be tested in a retrospective analysis.

In conclusion, these results indicate that differences between populations do exist in the distribution of enamel features, but that frequency comparisons should be made with extreme caution. Striae, pathological bands, and hypoplasias, since their distributions are not highly correlated, should not at the present time be combined indiscriminately in interpopulational analysis. In addition, as the distributions of striae and hypoplasias may possibly be influenced by the structure of the teeth, comparisons should be made only between samples composed of identical tooth types.

LITERATURE CITED

- Baume, L. J., and J. Meyer 1966 Dental dysplasia related to malnutrition, with special reference to melanodontia and odontoclasia. *J. Dent. Res.*, 45: 726-741.
- Bohatirchuk, F. P. 1957 Stain historadiography. *Stain Tech.*, 32: 67-74.
- Falin, L. I. 1961 Histological and histochemical studies of human teeth of the Bronze and Stone Ages. *Arch. Oral Biol.*, 5: 5-13.
- Gaunt, W. A., J. W. Osborn and A. R. Ten Cate 1971 *Advanced Dental Histology*. John Wright and Sons Ltd., Bristol.
- Giro, C. M. 1947 Enamel hypoplasia in human teeth: an examination of its causes. *J. Am. Dent. Ass.*, 34: 310-317.
- Grahnen, H., and K. Edlund 1967 Maternal diabetes and changes in the hard tissues of primary teeth. *Odontologisk Revy.*, 18: 157-162.
- Gustafson, A. G. 1959 A morphological investigation of certain variations in the structure and mineralization of human dental enamel. *Odontologisk Tidskrift*, 67: 361-472.
- Gustafson, G., and A. G. Gustafson 1967 Micro-anatomy and histochemistry of enamel. In: *Structural and Chemical Organization of the Teeth*. A. Miles, ed. Academic Press, New York.
- Herman, S., and R. McDonald 1963 Enamel hypoplasia in cerebral palsied children. *J. Dent. Child.*, 30: 46-49.
- Kreshover, S. 1940 Histopathologic studies of abnormal enamel formation in human teeth. *Am. J. Orth. Surg.*, 26: 1083-1101.
- Kronfeld, R. 1939 *Histopathology of the Teeth and their Surrounding Structures*. Lea and Febiger, Philadelphia.
- Massler, M., I. Schour and H. G. Poncher 1941 Developmental pattern of the child as reflected in the calcification pattern of the teeth. *Am. J. Dis. Child.*, 62: 33-67.
- Miyoshi, S., T. Nakata and S. Nishijima 1972 Scanning electron microscopy of prismless enamel of human teeth. *Arch. Oral Biol.*, 17: 359-362.
- Molnar, S., and S. C. Ward 1975 Mineral metabolism and microstructural defects in primate teeth. *Am. J. Phys. Anthrop.*, 43: 3-17.
- Moorrees, C. F. A. 1957 *The Aleut Dentition: A Correlative Study of Dental Characteristics in an Eskimoid People*. Harvard University Press, Cambridge.
- Osborn, J. W. 1971 A relationship between the striae of Retzius and prism directions in the transverse plane of the human tooth. *Arch. Oral Biol.*, 16: 1061-1070.
- Sarnat, B. G., and I. Schour 1941 Enamel hypoplasia (chronologic enamel aplasia) in relation to systemic disease: a chronologic, morphologic and etiologic classification. *J. Am. Dent. Ass.*, 28: 1989-2000.
- 1942 Enamel hypoplasia (chronologic enamel aplasia) in relation to systemic disease: a chronologic, morphologic and etiologic classification. *J. Am. Dent. Ass.*, 29: 67-75.
- Siegel, S. 1956 *Non-parametric Statistics for the Behavioral Sciences*. McGraw-Hill, New York.
- Sognnaes, R. F. 1947 Preparation of thin "serial" ground sections of whole teeth and jaws and other highly calcified and brittle structures. *Anat. Rec.*, 99: 133-144.
- 1955 Post-mortem microscopic defects in the teeth of ancient man. *Arch. Path.*, 59: 559-570.
- 1956 Histological evidence of developmental lesions in teeth originating from Palaeolithic, Prehistoric, and Ancient Man. *Am. J. Path.*, 32: 547-577.
- Watson, A., M. Massler and M. Perlstein 1964 Tooth ring analysis in cerebral palsy. *Am. J. Dent.*, 107: 370-384.
- Wilson, D. F., and F. R. Shroff 1970 The nature of the striae of Retzius as seen with the optical microscope. *Aust. Dent. J.*, 15: 162-171.