RECENT CONTRIBUTIONS TO THE STUDY OF ENAMEL DEVELOPMENTAL DEFECTS
Intertooth and Intratooth Variability in the Occurrence of Developmental Enamel Defects

Keith Condon* - Jerome C. Rose**

* Department of Cell Biology & Neuroscience, University of Texas Southwestern Medical Center, 5323 Harry Hines Blvd., Dallas, Texas 73235 (U.S.A.).
** Department of Anthropology, 330 Old Main, University of Arkansas, Fayetteville, Arkansas 72701 (U.S.A.).

Abstract.

The distribution of Wilson bands (abnormal striae of Retzius) and enamel surface defects (ESDs) is studied in histological sections of mandibular canine and first premolar pairs. In both teeth Wilson bands are found in all regions of the crown whereas ESDs are limited to the cervical two-thirds. Between teeth, every developmental enamel defect present in the premolar has a corresponding structure in the canine. Canine defects without premolar counterparts are concentrated in the cuspal two-thirds. These results suggest that susceptibility to developmental enamel defect formation varies both within and between tooth types.

Introduction.

Experimental, clinical and epidemiological studies over the last half century have demonstrated developmental enamel defects to be relatively sensitive and non-specific indicators of systemic stress (Goodman and Rose, 1990). Due to the absence of alteration to enamel after its deposition (aside from physical abrasion or caries formation) and the ability to determine chronological age at formation, the use of enamel defects as indicators of stress during childhood development has become increasingly popular in recent paleopathological research (e.g. Rudney, 1983; Goodman & al., 1984; Blakey & Armelagos, 1985; Corruccini & al., 1985; Rose & al., 1985; Goodman & Armelagos, 1988; Hutchinson & Clark, 1988; Lanphear, 1990; Van Gerven & al., 1990; Wright, 1990). Both macroscopic enamel surface defects (such as hypoplasia and hypocalcification) and microscopic defects (such as Wilson bands or abnormal striae of Retzius) have been used to reconstruct the pattern of stress episodes in historical and prehistoric populations.

The use of developmental enamel defects in reconstructing patterns of stress during development is not without methodological problems (Goodman & Rose, 1990). A primary issue is the choice of tooth for analysis. Theoretically all teeth whose enamel crowns are developing at the time of a systemic insult should bear a corresponding enamel defect. Thus, studies analyzing different
teeth should yield comparable results as long as the tooth crowns examined developed over the same period of time (e.g. between 3-6 years of age). However, such does not appear to be the case. For example, Goodman and Armelagos (1985a,b) in a macroscopic examination of enamel hypoplasia found that simultaneously developing teeth do not exhibit identical patterns of defects. Other investigators have noted the restriction of hypoplasia to specific regions of the enamel crown (Rose, 1977; Goodman & al, 1980; Cook, 1981; Hutchinson & Clark, 1988). These observations suggest that variation in enamel hypoplasia formation may occur both between different tooth types (intertooth variation) and between different regions of the enamel crown (intratooth variation). Whether or not similar patterns of variation occur in the expression of Wilson bands is unknown. Similarly, it is possible that previous macroscopic examinations of variability in enamel surface defect expression may have missed more subtle forms of enamel organ insult which are visible histologically. To address these questions, this study examines intertooth and intratooth variation in the histological expression of Wilson bands and enamel surface defects in mandibular canine and first premolar pairs. We will show that both teeth exhibit intratooth variation in the expression of enamel surface defects but not of Wilson bands. In terms of intertooth variation, the mandibular canine exhibits a greater frequency of both Wilson bands and enamel surface defects. Moreover, the enamel surface defects of the canine are always of equal or greater severity than their premolar counterparts.

Preliminary report of these results has appeared in abstract (Condon, 1981).

Materials and Methods.

Mandibular canine and first premolar pairs were selected from 30 individuals from the Libben site, a Late Woodland cemetery (ca. A.D. 800-1100; Lovejoy & al., 1977) in northern Ohio. Since the goal of this study was to examine variation in enamel defect expression, individuals exhibiting a high frequency of developmental enamel defects were chosen for analysis from a random sample of 146 pairs selected from the entire Libben collection. The mandibular canine was studied because of its frequent use in epidemiological studies (e.g. Rose, 1977; Lanphear, 1990; Van Gerven & al., 1990; Wright 1990) owing to its apparent susceptibility to enamel defect formation (Goodman & al., 1980). The mandibular first premolar was chosen for its similar histological structure and largely simultaneous period of development with the canine. Roentgenographic studies of tooth development in two modern populations (Moorees & al., 1963; Fanning & Brown, 1971; Anderson & al., 1975) show that except for the earliest development in the canine and the final development of the premolar, the crowns of the two teeth form simultaneously. Therefore, any developmental enamel defect resulting from systemic insult in one tooth should be present in the other except in the extreme cusp tip of the canine and the extreme cervical region of the premolar.

Polished longitudinal bucco-lingual thin sections (100-150 μm) were made using standard metallurgical techniques (detailed in Rose, 1977). The sections
were etched in 1N HCl for 15 seconds, rinsed and dried. Observations were made under bright field microscopy using a diffuse lighting technique (Wilson and Shroff, 1970). This technique permits evaluation of surface topography which is useful in the identification of Wilson bands (Rose, 1983; Goodman & Rose, 1990).

Accentuated striae of Retzius visible at x100 magnification were scored as Wilson bands if the following criteria were met: 1) the band occurred on both the labial/buccal and lingual sides of the section; 2) the band extended from near the dentino-enamel junction (DEJ) to the crown surface; and 3) at high magnification (x400 or greater) the prisms extending from the DEJ bent into or out of the plane of section or stopped along the length of the band (indicating a change in prism direction along the defect). Previous definitions of Wilson Bands have required assessment of enamel structure along the band as being either atypical, abnormal, distorted or unusual (Wilson and Shroff, 1970; Rose, 1977; Rudney, 1983). The criteria used here were devised in an attempt to render less subjective the identification of Wilson bands and therefore decrease rates of inter-and intra-observer error (Rudney, 1980). The validity of these criteria in identifying Wilson bands in this sample has been subsequently confirmed by scanning electron microscopy (Marks, 1988).

The majority of enamel surface defects observed in this sample exhibited a continuum of morphological expression. For purposes of comparison and analysis, four types were defined based upon histological features (see Results). Enamel surface defects were scored only on the labial/buccal side of the section.

The mandibular canine and first premolar pairs were scored separately. Defects were recorded as distances in millimeters from the cemento-enamel junction (CEJ) along the labial/buccal DEJ. Wilson bands were recorded as single points at their intersection with the DEJ (e.g., 3.2 mm). Enamel surface defects were recorded as intervals (e.g., 4.1-1.7 mm) by tracing the striae of Retzius associated with the beginning and end of the surface defect back to the DEJ.

Corresponding defects on the tooth pairs were matched by the following method. The median growth periods for the enamel crown of the mandibular canine and first premolar were plotted on 1/4 inch graph paper with each interval representing 0.25 years. These data are from serial roentgenographs of a modern Ohio population (Fanning and Brown, 1971). The growth periods were then divided into units equal to the modal length of complete (unworn) labial/buccal side lengths of the DEJ observed in a larger sample of each tooth type from the Libben population (7.8 mm for the mandibular first premolar and 11.5 mm for the mandibular canine; no difference in DEJ length was noted between the sexes). The scales standardize the rate of enamel extension (ameloblast differentiation rate) along the DEJ per unit of time thus making comparison between teeth of different sizes possible. For each tooth the method assumes a constant rate of ameloblast proliferation along the DEJ as the enamel organ increases in size. Based on this assumption, construction of the scales shows that ameloblast proliferation occurs at
a faster rate per unit of time in the larger mandibular canine than in the smaller first premolar (Figure 1).

The defects in the corresponding pairs were matched by marking the position of the defects upon the appropriate growth scales and then sliding the scales back and forth until a best match was obtained (Figure 1). The technique is analogous to that used in dendrochronology (cf. Figure 10; Stokes and Smiley, 1968).

Finally, for purposes of interpretation and comparison, the results are summarized by crown region. The mandibular canine and first premolar crowns were divided into cervical, mid-coronal and occlusal regions based upon morphological assessment. The relationship between crown region and defect position (measured along the DEJ) is illustrated in Figure 2.

**Results.**

**Developmental enamel defect morphology.**

At low magnification (x100) Wilson bands appear as ridges, troughs or opaque bands parallel to the striae of Retzius (Figure 3). The form of the band can change along its length in concert with changes in orientation of the prisms in the adjoining enamel. At higher magnification (x400; Figure 4) the prisms extending from the DEJ can be seen to stop or bend into or out of the plane of section along the band’s border. Prisms
along the edge of the band and/or within it often exhibit a distorted morphology (Rose, 1977; Wright, 1990).

Although the majority of enamel surface defects observed in this sample exhibit a morphological continuum, they are classified into 4 types (I-IV) based on the histological criteria of Gustafson (1959). Type I, II and III enamel surface defects represent an ordinal scale of increasing severity of enamel deformation whereas type IV defects are histologically distinct. For comparative purposes a section of normal enamel is shown in Figure 5. Note that the enamel surface is regular (even). The striae of Retzius are only slightly more opaque than the inter-striae enamel, are parallel to one another and extend rectilinally to the crown surface. The surface contour of type I enamel surface defects (ESD-I; Figure 6) can be even or slightly undulating. The associated striae of Retzius are accentuated (i.e. increased opacity) and can be seen to curve and converge in a cuspal direction at the crown surface. The enamel around and between such striae is often optically opaque. These characteristics are typical of developmental hypocalcifications (Gustafson, 1959; Kostlan & Plackova, 1962; Spouge, 1973). However, since the degree of mineralization of the defects in this study has not been assessed, the general designation ESD-I will be employed.

Type II enamel surface defects (ESD-II; Figure 7) appear as shallow pan-like depressions of varying length (0.3 mm and greater) in the enamel contour. The striae of Retzius underlying the deficiency are accentuated and converge at the crown surface. Occasionally, small sharp depressions occur within the larger pan-like depression. This class of defects appears to correspond to Sarnat and
Figure 3. A Wilson band in a mandibular first premolar. 
Seen at low magnification the defect (arrow) in this region of 
the crown has a trough-like appearance. Scale bar, 50 µm.

Figure 4. Wilson band morphology at high magnification. 
shown is the same defect (asterisk) as in Figure 2. The 
prisms on the dentin side of the defect stop along its margin 
(arrow). The prisms within the band appear oriented roughly 
perpendicular to those on 
either side of the defect, 
indicating a change in prism direction during enamel deposition. Scale bar, 25 µm.

Figure 5. A segment of mandibular canine exhibiting normal surface enamel. The 
enamel surface contour is regular, the striae of Retzius 
(example indicated by arrows) are parallel to one another and 
are only slightly more optically 
opaque than the inter-striae 
enamel. Scale bar, 100 µm.
Figure 6. A segment of mandibular canine enamel exhibiting a type I ESD. The surface contour is irregular with slight undulations. The striae of Retzius are slightly accentuated and converge toward one another at the crown surface (arrows). The inter-striae enamel is optically opaque. Scale bar, $\mu$m.

Figure 7. A segment of mandibular canine enamel exhibiting a type II ESD. The pan-like deficiency of enamel begins at upper left (arrow) and continues out of the frame. The striae of Retzius associated with the defect are accentuated and converge at the crown surface. The inter-striae enamel is also optically opaque. Scale bar, 100 $\mu$m.

Figure 8. A segment of mandibular canine enamel exhibiting a type III ESD. The defect appears as a deep concavity in the enamel surface contour. In this example, the hypoplasia is covered by calculus (c). The striae of Retzius are markedly accentuated and exhibit a contorted morphology along their length. An area of prismless enamel lies along the surface at the base of the defect (asterisk). Scale bar, 100 $\mu$m.
Schour's (1941) macroscopically defined single zone type hypoplasia.

Type III enamel surface defects (ESD-III; Figure 8) are marked deficiencies of enamel appearing as more abrupt, deeper concavities in the enamel surface contour. The underlying striae of Retzius are extremely accentuated and exhibit marked convergence. Areas of prismless enamel may occur at the base of the concavity (Rose, 1977). Typically these defects occur as part of a series and thus appear to correspond to Sarnat and Schour's (1941) multiple zone type hypoplasia.

The final type of enamel surface defect (ESD-IV; Figure 9) appears distinct from the others in that it is not part of the same morphological continuum. [Type I, II and III hypoplasia represent an ordinal scale of increasing enamel deformation]. Type IV enamel surface defects are characterized by a gross deficiency of enamel creating a large concavity in the enamel surface contour. Unlike other enamel surface defects, however, the associated striae appear normal, exhibiting neither accentuation nor convergence. A single case of this type was observed in this sample.

Intratooth Distribution of Wilson bands.

Table 1 shows the distribution by millimeter (mm) unit and crown region of the 38 Wilson bands identified in the mandibular canine. These micro-defects occur in all three regions of the crown although the three occlusal defects are found close to the mid-coronal/occlusal border. Due to attrition, not all regions of the crown are observable in all individuals, therefore the raw frequencies by region are not equal to the total sample size. To control for the effects of attrition, the number of Wilson bands per region is divided by the total number of mm units observable for that region to derive a relative frequency of occurrence. The relative frequencies of Wilson bands for the occlusal, mid-coronal and cervical regions are 0.07, 0.16 and 0.14 defects/mm, respectively. Thus, the occlusal regions shows a lower frequency of Wilson bands relative to the mid-coronal and cervical regions.
Table 1. Distribution of Wilson bands in the mandibular canine by crown region and millimeter unit and their correspondence to defects in the mandibular first premolar.

<table>
<thead>
<tr>
<th>corresponding premolar defect?</th>
<th>Location of canine Wilson band</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mm unit: 1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
<td>8</td>
<td>9</td>
<td>10</td>
<td>11</td>
<td>12</td>
</tr>
<tr>
<td>yes</td>
<td>1</td>
<td>3</td>
<td>6</td>
<td>5</td>
<td>5</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>no</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>4</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>not observable</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>totals:</td>
<td>21 (57%)</td>
<td>13 (35%)</td>
<td>3 (8%)</td>
<td>38 (100%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The distribution of Wilson bands (N = 25) in premolars is summarized in Table 2. As in the canine, these micro-defects occur in all three crown regions. Again, the relative frequency is less in the occlusal region (0.04) than in the mid-coronal (0.15) and cervical regions (0.16).

**Intratooth Correspondence of Wilson Bands.**

Table 1 shows the correspondence of canine Wilson bands to premolar Wilson bands by crown region. Twenty-five (66%) canine Wilson bands have a matching defect on the premolar, nine (24%) do not and the corresponding area of premolar enamel is missing due to attrition in four cases (10%). Removing the missing cases and summarizing by crown region, 95% (all but one) of the canine Wilson bands in the cervical region has a corresponding premolar defect, with the single exception occurring close to the cervical/mid-coronal border (4.7 mm). In the combined mid-coronal/occlusal regions, only 4 of 12 (33%) canine Wilson bands have a corresponding premolar defect. If comparison is made from the first premolar to the canine (Table 2), every Wilson band present in the premolar has a corresponding microdefect on the mandibular canine regardless of crown location.

**Intratooth Distribution of Enamel Surface Defect.**

Fifty-nine enamel surface defects are identified in the mandibular canines (Table 3). The majority (84%) occur in the cervical region and the remaining 16% are found in the mid-coronal area. No occlusal region defects are observed. Controlling for the effects of attrition gives relative frequencies of 0.12 and 0.34 defects/mm in the mid-coronal and cervical regions, respectively. Type I ESDs are the most frequent (43%) followed by approximately equal frequencies of type II (29%) and type III (26%) surface defects. A single class IV ESD is present. Type II and III defects are observed only in the cervical region.
Table 2. Distribution of Wilson band in the mandibular first premolar by crown region and millimeter unit and their correspondence to defects in the mandibular canine.

<table>
<thead>
<tr>
<th>Corresponding canine defect?</th>
<th>Location of premolar Wilson band</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mm unit: cervical</td>
</tr>
<tr>
<td>yes</td>
<td>1</td>
</tr>
<tr>
<td>no</td>
<td>0</td>
</tr>
<tr>
<td>not observable</td>
<td>0</td>
</tr>
<tr>
<td>totals</td>
<td>14 (56%)</td>
</tr>
</tbody>
</table>

Table 3. Distribution of enamel surface defects (ESDs) in the mandibular canine by crown region.

<table>
<thead>
<tr>
<th>ESD type</th>
<th>cervical</th>
<th>mid-coronal</th>
<th>occlusal</th>
<th>totals</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>18 (30%)</td>
<td>8 (14%)</td>
<td>0 (0%)</td>
<td>26 (44%)</td>
</tr>
<tr>
<td>II</td>
<td>17 (29%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>17 (29%)</td>
</tr>
<tr>
<td>III</td>
<td>15 (25%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>15 (25%)</td>
</tr>
<tr>
<td>IV</td>
<td>0 (0%)</td>
<td>1 (2%)</td>
<td>0 (0%)</td>
<td>1 (2%)</td>
</tr>
<tr>
<td>totals:</td>
<td>50 (84%)</td>
<td>9 (16%)</td>
<td>0 (0%)</td>
<td>59 (100%)</td>
</tr>
</tbody>
</table>

whereas type I are present in both the cervical and mid-coronal regions.

A similar pattern of expression is seen for the 50 enamel surface defects present in the first premolar (Table 4). Fourteen percent of the premolar surface defects are found in the mid-coronal region with the remaining 86% located cervically, with relative frequencies of 0.14 and 0.46 defects/mm, respectively. As in the canine, no enamel surface defects are found in the occlusal region. Type I ESDs comprise 92% of the total and one type IV and three type II ESDs (6%) comprise the remainder.

_intertooth Correspondence of Enamel Surface Defects._

The correspondence of enamel surface defects between the mandibular canine and first premolar shows a pattern similar to that of Wilson bands. Eighty-five percent (Table 5) of the canine surface defects have a corresponding structure on the premolar. Summarized by crown region, 90% of the cervical defects..
### Table 4. Distribution of enamel defects (ESDs) in the mandibular first premolar by crown region.

<table>
<thead>
<tr>
<th>ESD type</th>
<th>cervical</th>
<th>mid-coronal</th>
<th>occlusal</th>
<th>totals</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>39 (78%)</td>
<td>7 (14%)</td>
<td>0 (0%)</td>
<td>46 (92%)</td>
</tr>
<tr>
<td>II</td>
<td>3 (6%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>3 (6%)</td>
</tr>
<tr>
<td>III</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>IV</td>
<td>0 (0%)</td>
<td>1 (2%)</td>
<td>0 (0%)</td>
<td>1 (2%)</td>
</tr>
<tr>
<td>totals:</td>
<td>42 (84%)</td>
<td>8 (16%)</td>
<td>0 (0%)</td>
<td>50 (100%)</td>
</tr>
</tbody>
</table>

### Table 5. Correspondence of enamel surface defects (ESDs) in the mandibular first premolar.

<table>
<thead>
<tr>
<th>canine ESD</th>
<th>corresponding premolar ESD</th>
<th>location of canine ESD</th>
<th>total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>cervical</td>
<td>mid-coronal</td>
<td>occlusal</td>
</tr>
<tr>
<td>I</td>
<td>I</td>
<td>13</td>
<td>4</td>
</tr>
<tr>
<td>II</td>
<td>0</td>
<td>0</td>
<td>0</td>
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<tr>
<td>III</td>
<td>0</td>
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</tr>
<tr>
<td>II</td>
<td>I</td>
<td>16</td>
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<tr>
<td>II</td>
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<tr>
<td>IV</td>
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<td>1</td>
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<td></td>
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</tbody>
</table>

* no corresponding ESD on the premolar.
Table 6. Correspondence of enamel surface defects (ESDs) in the mandibular first premolar to structures in the canine.

<table>
<thead>
<tr>
<th>premolar ESD</th>
<th>corresponding canine ESD</th>
<th>location of premolar ESD</th>
<th>total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>cervical</td>
<td>mid-coronal</td>
</tr>
<tr>
<td>I</td>
<td>I</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>II</td>
<td>II</td>
<td>11</td>
<td>5</td>
</tr>
<tr>
<td>III</td>
<td>III</td>
<td>15</td>
<td>0</td>
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<tr>
<td></td>
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<tr>
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<td>III</td>
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</tr>
<tr>
<td>IV</td>
<td>IV</td>
<td>0</td>
<td>1</td>
</tr>
</tbody>
</table>

50 (100%)

* no corresponding ESD on the canine.

have corresponding premolar defects whereas only 50% of the mid-coronal defects have matching structures. All nine (15%) canine defects without a corresponding premolar structure are type I ESDs and 8 of these occur partially or completely within the mid-coronal region.

Type I, II and III ESDs represent an ordinal scale of increasing severity of enamel deformation. For all canine defects with a corresponding premolar structure, the canine ESD is of equal or greater severity. The single type IV ESD in the canine corresponds to a type IV defect in the premolar.

If comparison is made from the premolar to the canine, every premolar enamel surface defect has a corresponding canine defect regardless of crown location (Table 6). Further, all premolar ESDs correspond to a canine defect of equal or greater severity.

Evidence supporting the validity of the matching technique.

The validity of the enamel defect matching technique is supported by the following observation. If the point of development of the first premolar crown corresponding to the time of completion of canine crown formation is determined for each individual (obtained after the corresponding defects are matched by drawing a line from the CEJ end of the canine scale down across the premolar scale; see Figure 1, point C), the chronological distribution of these points falls within a fairly small time period. Twenty-eight of the thirty individuals in the sample completed canine crown development within an 0.4 year span of each other and the remaining two individual are within 0.8 years. This entire range of values (0.8 years) is less than one standard deviation for canine crown completion in a
roentgenographic study of modern populations (+/-0.5 years; Moorees & al., 1963). The consistent termination of development of the canine crown relative to the premolar supports the notion that corresponding defects were identified and could be matched between teeth.

Discussion.

The distributions of developmental enamel defects both within and between the crowns of the mandibular canine and first premolar show clear and consistent patterns of variation. Within the crowns of these two teeth enamel surface defects were never observed in the occlusal region, were infrequent in the mid-coronal region and most common in the cervical region. These results suggest that the more cuspal regions of both teeth may be less susceptible to enamel surface defect formation and are consistent with previous reports of reduced frequencies of enamel hypoplasias in the cuspal regions of several tooth types (Rose 1977; Goodman & al., 1980; Cook, 1981; Hutchinson & Clark, 1988). In contrast, Wilson bands were observed in all three crown regions of both the mandibular canine and first premolar although the occlusal region exhibited a lower relative frequency than the mid-coronal and cervical regions. The absence of enamel surface defects and the possibly reduced frequency of Wilson bands in the occlusal region of both the canine and premolar in this study strongly suggest that susceptibility to enamel defect formation varies with the stage of crown development. However, an alternative explanation for the intratooth distributions seen in this study would be that the stressors responsible for developmental enamel defects, particularly ESDs, may not have been present in this population during the time corresponding to amelogenesis of the occlusal regions. The absence of data from teeth whose cervical development corresponds synchronously to the occlusal development of the canine and premolar (e.g. the central maxillary incisor) prevents exclusion of this alternative hypothesis. However, comparison of Wilson band frequencies to both infectious lesions of the skeleton and age specific mortality in the Libben population suggests that individual experiencing stress during the first year of life capable of causing a defect died and were not available to be included within this predominantly adult sample (Boyd, 1978).

In regard to intertooth variation, all developmental defects present in the mandibular first premolar had a corresponding structure in the mandibular canine whereas several canine defects were found without corresponding premolar structures. As in the case of intratooth variation, defects in the canine without a corresponding premolar defect showed a clear relationship to crown region. The vast majority of Wilson bands and ESDs in the cervical region (95% and 90%, respectively) had corresponding defects on the premolar. In contrast, in more cuspal regions the correspondence decreased to 33% and 50% for Wilson bands and ESDs, respectively. Thus it appears that the cervical regions of the two teeth have comparable susceptibility to defect formation, but that the mid-coronal and occlusal regions of canine are more susceptible than the corresponding areas of the premolar. In regards to enamel surface defects, this susceptibility is expressed not only quan-
tatively in terms of a greater frequency of defects, but also qualitatively given that the canine defect was always of equal or greater severity than that of the premolar.

The patterns of intratooth and intertype variation observed here are similar to that reported by Goodman and Armelagos (1985a,b) in their macroscopic analysis of enamel hypoplasia. Examining complete dentitions (excluding third molars) from thirty individuals, Goodman and Armelagos (1985a,b) found the greatest frequencies of hypoplasias to occur in the cervical and mid-coronal region of all tooth types and the lowest frequency to occur in the occlusal region. Since these investigators found hypoplasias in tooth types whose cervical development corresponded to areas of non-hypoplastic development in the occlusal region of other teeth, they concluded that the occlusal regions of all tooth types (including the mandibular canine and first premolar) are relatively intratable to hypoplasia formation. In addition to intratooth variation, these investigators found significant differences in the frequency of defects among different tooth types developing at the same time. In agreement with the present study the mandibular canine exhibited a greater frequency of defects (approximately threefold) than the first premolar despite the synchronous development of these teeth.

**Sources of variation in development enamel defect occurrence.**

In this study intertooth variation among enamel surface defects was both quantitative (i.e. the mandibular canine exhibited a greater frequency of defects) and qualitative in that the canine defects were always of equal or greater severity than their premolar counterpart. The factors responsible for variability in enamel defect formation have yet to be determined (Goodman & Rose, 1990). In their analysis of hypoplasia distribution, Goodman & Armelagos (1985a,b) proposed separate factors to account for intratooth and intertooth variation. A more parsimonious approach would be to identify a common factor(s) which could account for both intratooth and intertooth variation. One such possibility is that susceptibility to defect formation varies with the rate of enamel matrix deposition such that the slower the rate, the greater the susceptibility to defect formation. This hypothesis is suggested by Dean’s (1987) review of growth layer and incremental markers in enamel. Dean (1987) notes that in human incisors the distance between adjacent perikymata (the surface expression of striae of Retzius) decreases cervically, but that the number of cross- striations (presumed daily incremental lines within the prisms) between adjacent striae remains relatively constant. Thus, Dean infers that the volume (rate) of enamel matrix deposition per day varies along the length of the enamel organ from greatest cusply to least cervically. It should be pointed out that other than Dean’s (1987) work there is little conclusive evidence for variation in the rate of matrix formation (Goodman & Rose, 1990). The pattern of perikymata seen in the incisor is also characteristic of the mandibular canine (personal observation) and first premolar (Stern & Skobe, 1985). Thus, there appears to be a correlation between the pattern of intratooth variation (susceptibility to enamel defect
formation; least cuspsally, greatest cervically) and the rate of enamel deposition (greatest cuspsally, least cervically as indicated by perikymata studies). If this correlation is causal, then intertooth variation may simply be due to synchronous differences in the rate of enamel deposition between tooth types. For example, the difference between the mandibular canine and first premolar in susceptibility to defect formation is that for synchronously developing regions, the rate of enamel matrix deposition is greater in the premolar than in the canine. This hypothesis can be tested since it predicts that for simultaneously deposited enamel, those teeth showing a greater susceptibility to enamel surface defect formation should have absolutely smaller inter-striae widths (measured at the enamel surface; indicating a greater rate of enamel matrix deposition). Conversely, those teeth showing reduced susceptibility should have an absolutely greater inter-striae width. Unfortunately, since the majority of individuals in his sample possessed ESDs it was not possible to measure normal inter-striae widths to test the hypothesis.

The above hypothesis is consistent with Osborne's (1973) theory of amelogenesis which models the enamel organ as a hydrostatic structure. In terms of defect formation, the hydrostatic enamel organ will be most stable or rigid (and thus less susceptible to defect formation) when it is expanding at its greatest rate (i.e. when enamel matrix production is greatest) and correspondingly less stable at lower rates. The situation is analogous to the rigidity of the surface of an expanding balloon, the wall of a rapidly expanding balloon being less deformable than that of a slower expanding one.

An alternative explanation for the variation in sensitivity of the ameloblasts to stress is that ameloblasts which differentiate late in the development of the enamel organ are more sensitive or, alternatively, less physiologically robust than those differentiating earlier. In other words, the further from the cusp tip that an ameloblast originates, the less robust it will be and the more likely that systemic stress will result in a hypoplasia or Wilson band. Thus, we would expect to find more developmental defects in more cervical enamel, as has been shown in this study. In addition, teeth with long dentino-enamel junctions (i.e. tall crowned teeth such as the canine) will be overall more sensitive to systemic stress than those with shorter dentino-enamel junctions (i.e. premolar) as has been shown here. Testing this hypothesis and differentiating it from the previous one would require experimental developmental research not possible with an archeological collection.

**Implications for epidemiological studies.**

The existence of variability in developmental enamel defect expression both within and between tooth types has important methodological implications (Goodman & Rose, 1990). In epidemiological studies this variability requires careful consideration of both the type of enamel defect to be studied and the tooth type(s) examined. Improper selection of either will result in a stress profile unrepresentative of the total stress pattern. For example, use of the mandibular first premolar in lieu of the mandibular canine would underestimate the fre-
quency of stress episodes resulting in Wilson band formation during earlier ages of development. Similarly, if enamel hypoplasia (type II-IV ESDs) were selected for analysis, the mandibular first premolar would yield a frequency eight-fold less than the mandibular canine, and the defects would be distributed over a narrower chronological range. Finally, the results presented here suggest that the choice of a single tooth in enamel surface defects studies is justifiable only if analysis is limited to the age range corresponding to the tooth’s cervical development, i.e. it’s period of susceptibility to defect formation. Inclusion of ages corresponding to crown regions known to be resistant to defect formation would mis-characterize the stress profile of the population.

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