9 Sources of variation in estimated ages at formation of linear enamel hypoplasias

ALAN H. GOODMAN AND RHAN-JU SONG

Introduction

Precise understanding of the pattern and timing of dental development is key to a number of basic and applied research areas. If one assumes that 'dental age' corresponds closely to chronological age, dental age is often used as a proxy for chronological age in contemporaneous and bioarchaeological studies of health and nutrition. In order to better understand the evolution of events associated with human tooth formation, one needs to know the degree of variation in tooth formation within, and among, human populations and primate species. The importance of understanding tooth crown development timing is also fundamental to studies of linear enamel hypoplasias (LEHs). The purpose of this chapter is to explore some of the issues involved in estimating an individual's age at formation of LEHs.

Enamel hypoplasias are a class of developmental defect of enamel that is morphologically distinguished by a macroscopically observable area of decreased enamel thickness (Sarnat and Schour 1941; Goodman *et al.* 1980; Fig. 9.1). These developmental defects are direct consequences of a temporary disruption to the ameloblasts during their primary function of secreting enamel matrix (Shawashy and Yaeger 1986). Because of their permanence and direct aetiological link to disrupted ameloblastic function, enamel hypoplasias have been intensively studied as retrospective evidence of ameloblastic disruption. Moreover, because secretory phase ameloblasts are sensitive to a wide variety of 'stressors' or 'insults' such as undernutrition, trauma, infection and temperature extremes, a resulting enamel hypoplasia may ultimately be related to these underlying conditions and processes (Kreshover 1960; Cutress and Suckling 1982).

A wealth of studies use enamel hypoplasias as a mirror onto overall levels of physiological perturbation and environmental causes of that perturbation during tooth crown development (Goodman and Rose 1990, 1991). In clinical research, for example, enamel hypoplasias have been compared between individuals with prenatal brain damage and a control group in order to determine whether the brain damage might be related to some yet undiscovered prenatal growth disruption (Judes *et al.* 1985). Similar clinical research has been undertaken in comparing low and normal birth weight individuals (Seow and Perham 1990), nutritionally supplemented and non-supplemented individuals (Goodman *et al.* 1991), and individuals with and without a history of infectious disease (Hall 1989). Finally, particularly intense research has taken place in bioarchaeology, in which the frequency of enamel defects has been used to discern levels of physiological disruption in different past groups. For example, Cohen and Armelagos's (1984) volume *Paleopathology at the Origins of Agriculture* includes over 15 reports on enamel hypoplasia in early agricultural groups. The number of studies of enamel developmental defects, the majority of which are applications such as these, is now around 1000.

Most enamel hypoplasias run linearly around part, or all, of tooth

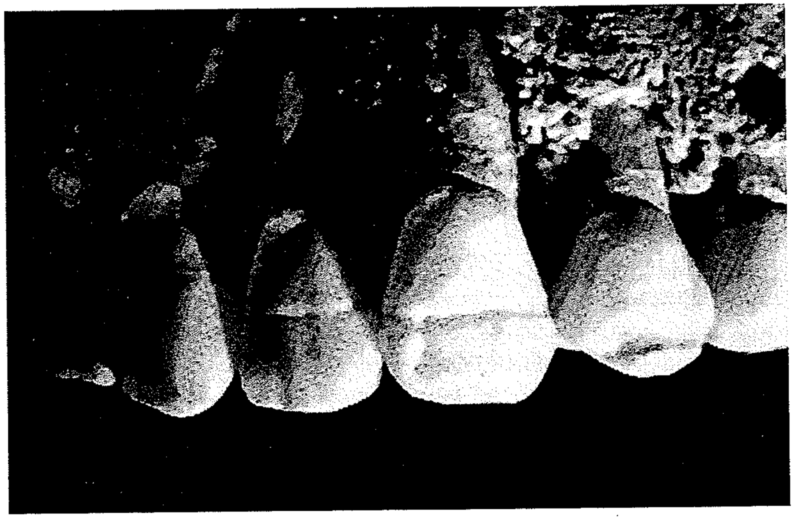

Fig. 9.1. Photographic example of chronological enamel hypoplasias on anterior maxillary permanent teeth. Note that the position of the hypoplasias varies on the different tooth crowns, presumably due to variation in the time of initial crown development and the rate of subsequent development. The lateral incisor and canine LEH is further toward the occlusal surface than is the defect on the premolar, probably because these teeth began development before the premolar.

crowns, roughly parallel to the cemento-enamel junction, and perpendicular to the long axis of teeth (Fig. 9.1). This linear form reflects the chronological development of tooth crowns, also seen in histological section by the location of striae of Retzius and surface perikymata (Hillson and Bond 1997). Because of this characteristic of most LEHs, they were once referred to as chronological enamel hypoplasia (Sarnat and Schour 1941).

Because there are so few methods to retrospectively assess the age of individuals at the time of a physiological event, understanding an individual's age at LEH development is critical. For example, in the above-noted study of the relationship between brain damage and LEH, knowing whether the enamel defect developed before birth, and if so exactly how much before birth, could be critical to pinpointing the trimester, month, week or day during which the defect formed. In bioarchaeological studies, as well as other population studies, it is common to reconstruct the distribution of LEH by age at development. Many studies of the last two decades have shown peak periods of LEH in permanent incisors and canines during the second to fourth year of life, leading to inferences about the degree of weaning or postweaning stress (Corruccini *et al.* 1985; Goodman *et al.* 1987). However, imprecision in estimating the age at formation muddles our ability to test this assertion.

In this chapter we outline and explore factors that may affect the accuracy of estimates of age at LEH formation. We first present a brief historical review of methods for estimation of age at formation of defects and the construction, from these data, of patterns of LEH formation in populations. We then categorise possible sources of variation in estimation of age at formation of an LEH. Finally, we focus on three factors: tooth size variation, the length of time in which appositional or cuspal enamel is formed (and, thus, not amenable to macroscopic observation), and the choice of developmental standard. These factors were chosen because their effects can be estimated and they may be the three most important sources of variation. The consequences of these factors are illustrated with applications to LEH data from the Classic Period Maya from Altun Ha, Belize (see Song 1997) and a historical sample, the Hamann–Todd osteological collection from greater Cleveland, Ohio (see Goodman 1988).

A brief history of methods for estimating age at LEH formation

The timing of LEH has been central to the larger history of research into dental development. Smith (1991), in her exceptional review of the development of tooth chronologies, notes that hypoplastic banding of teeth probably 'inspired' many early pictorial charts of tooth formation. This observation is clear from a perusal of early works on tooth development. For example, Logan and Kronfeld (1933) and Kronfeld (1935) reproduced a picture of an individual with an enamel hypoplasia assumed to have developed in the first year. Their point in so doing was to show that the upper lateral incisors commence development after the other anterior teeth: the upper central incisors are the only anterior teeth without hypoplastic involvement on the incisal surface. Just as the degree to which anatomical information on development provided valid information on development timing, it was assumed that the location of enamel hypoplasias did the same. The location of enamel hypoplasias was especially important for noting relative development of teeth at the time of a common event. The relative position of the 'birth line' in histological section is an excellent example of this. In a sense, this work has provided inspiration for recent histological methods of timing dental events (Bromage and Dean 1985; Macho and Wood 1995).

While dental texts noting the timing of tooth formation were common by the 18th century, the first extensive table of tooth formation events is generally credited to Legros and Magitot (1880, 1881), soon followed by Black (1883) and Berten (1895). All of these authors produced tables and graphs that illustrated the development of the different teeth by zones (the more occlusal/incisal zones developing earlier) to the later developing cervical zones (Smith 1991). In general, methods are not well articulated and sample sizes are not made explicit. One is left to assume that the charts were based on small sample sizes, with a great deal of extrapolation between ages.

Although these early works correctly identified general patterns of development, variation among these standards in the estimated chronological age of events concerned Logan and Kronfeld (1933). For example, Black (1883) estimated that second molars began calcification around the sixth year, whereas Legros and Magitot (1880, 1881) estimated that calcification began around the third year. In an effort to add greater precision and certainty to the age at tooth formation, Logan and Kronfeld (1933) initially studied 25 individuals. Of these, 19 died before 2 years of age and only three were older than 4.5 years at death. Later on, an additional five individuals were added. Of greatest concern is the low number of individuals over the age of 2 years, and especially older than 4.5 years. In addition, like all studies based on postmortem examination, results may be biased by being based on individuals who died, possibly from conditions that may also affect the rate of tooth development.[1] Thus, while Logan and Kronfeld's research is an improvement over the prior generation of research, it remains limited. Through its influence on the standard of Massler and co-workers (1941), it is the basis for nearly all research into the timing of LEHs.

In two publications in 1941, among the two most widely cited in dental research, dental timing and enamel hypoplastic chronologies were again intimately related. In the paper on developmental timing, Massler *et al.* (1941) extrapolated from Logan and Kronfeld (1933). Concurrently written, the classic enamel hypoplasia paper of Sarnat and Schour (1941) was the first to explicitly present a group pattern of enamel defects based on translating the location of defects on tooth crowns to ages at formation. Sarnat and Schour (1941) estimated the age at formation of enamel hypoplasias for 60 individuals with hypoplasias from the Chicago area. They recorded the age at development of defects to monthly periods, extrapolating from the Massler *et al.* (1941) standard. According to these authors, most defects occurred before the first year of life and were of a few months' duration.

Despite the authors' obvious enthusiasm for using enamel hypoplasias as kymographic records of stress, little follow-up research was done over the next quarter century. However, on the basis of the fact that their work is widely cited in dental texts (Goodman 1988), this lack of replicability seems to be more due to a general acceptance of the universality of their results than lack of enthusiasm for their work.

An additional factor that may have contributed to the non-replicability of the Sarnat and Schour (1941) study is lack of details regarding their method of translating from LEH location on tooth crowns to age at formation. In 1966, Swärdstedt completed a Swedish dental thesis on the pattern of enamel hypoplastic defects found at Westerhus, a Mediaeval population from Jämtland/Mid-Sweden. In addition to being one of the first applications of enamel hypoplasias to bioarchaeology, this study clarified the method of translating enamel hypoplasia locations to age at formation. Swärdstedt (1966) explicitly diagrammed the location of enamel

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[1] Long-term illness and undernutrition may slow dental eruption and also to some degree dental formation rates. In the practice of estimating ages at LEH formation, research is done both on samples who died, possibly due to long-term illness and undernutrition, and living individuals. Thus the possibility of error is introduced; however, it is difficult to estimate its magnitude. In one regard, the older, postmortem-based standards may be more appropriate for archaeological populations, and radiographic standards may be more appropriate for living populations. This idea, however, is further complicated in LEH studies because these are often based on survivors of early stress.

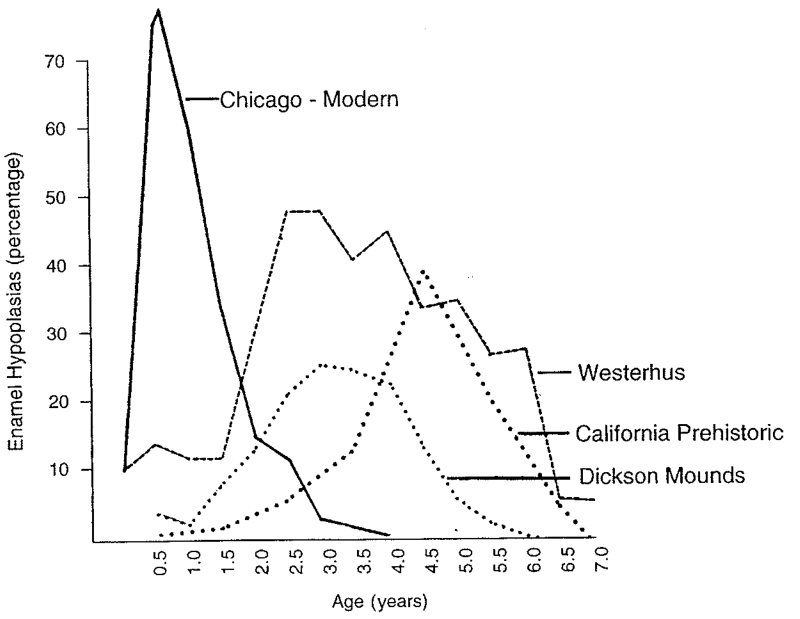

Fig. 9.2. Comparison of the chronological pattern of LEH in different populations. The Chicago-Modern sample is from Sarnat and Schour (1941). The peak in LEH is within the first year. Westerhus is from Swärdstedt (1966), California Prehistoric is from Schultz and McHenry (1975) and Dickson Mounds (Lewiston, Illinois, USA) is from Goodman *et al.* (1980). These four populations are among the first studied and represent some of the variation that is seen with different methods and different standards of use.

defects against an age at formation (Fig. 9.2). Like Sarnat and Schour (1941), he used the Massler *et al.* (1941) ages at beginning and end of crown formation as 'anchors'. He then divided tooth crowns into half-year developmental zones. Thus, a tooth such as a lower central incisor, which develops between birth and 4 years, was divided into eight zones. Because severe occlusal attrition is often found on archaeological teeth, Swärdstedt measured the locations of LEHs from the cemento–enamel junction (CEJ).

Half-year zones varied in width across teeth as a function of the number of zones per tooth and crown height, with crown heights based on previously published data. Interestingly, zones within teeth are not of similar width. Although this point is not discussed by Swärdstedt (1966), and its rationale is hard to reconstruct from the initial work of Massler *et al.* (1941), it appears that Swärdstedt may have divided crowns into anatomical thirds. Since some teeth, such as the lower central incisor, had a number of zones that did not divide evenly into thirds, tooth thirds have unequal numbers of zones.[2]

Although the frequency of defects varied in by cultural period and social class, Swärdstedt (1966) found that age patterns were constant across groups with a peak in LEH formation estimated to be around three to four developmental years (Fig. 9.3). Obviously, this chronology varies greatly from the prior chronology of Sarnat and Schour (1941).

In their study of the chronological distribution of enamel defects at Dickson Mounds, Lewiston, Illinois (AD 950–1300), Goodman and colleagues (1980) redrew the Swärdstedt chart and further clarified his methodology. The Dickson Mounds pattern, with a peak in defect formation at around 30 months of age, was similar to what Swärdstedt found, although a bit earlier, and countered the supposition of Sarnat and Schour (1941) that their pattern – most defects occurring within the first year of life – was a universal one.

The assumption of differential growth velocity within teeth is implied by a variable number of half-year developmental periods within tooth thirds in what we will refer to as the 'chart method' or the 'Swärdstedt method'. This assumption, however, appears to have little correspondence to the primary data (Goodman and Rose 1990). To correct for these limitations, a series of regression equations were formulated to translate age at development of an LEH from its location, starting and ending age at crown calcification and crown height (Table 9.1; Goodman and Rose 1990). The basic forms of this linear (constant velocity) regression equation is:

$$\text{age at formation} = -\,[(1/\text{velocity}) \times \text{distance of LEH from CEJ}] + \text{age at crown completion}$$

In this equation velocity is in mm/year and is thus related to tooth crown height and the total time of tooth development. All ages are in years and should be expressed as developmental ages. The 'velocity term' is negative and inverse (years/mm).

Although this 'rationalises' the methodology and makes clear the assumptions upon which it is based, the assumptions themselves are not challenged. The Massler chart method has been used in ageing LEHs in

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[2] Swärdstedt's allocation of half-year developmental zones to tooth crown thirds is more complicated still. For some teeth with a number of half-year developmental periods evenly divisible by 3, Swärdstedt still assigned a variable number of half-year periods to different thirds. For the upper central incisor and canine, the incisal third 'received', at the expense of the middle third, an extra half-year developmental period. Conversely, the lower canine's cervical third received an extra half-year period at the expense of its incisal third.

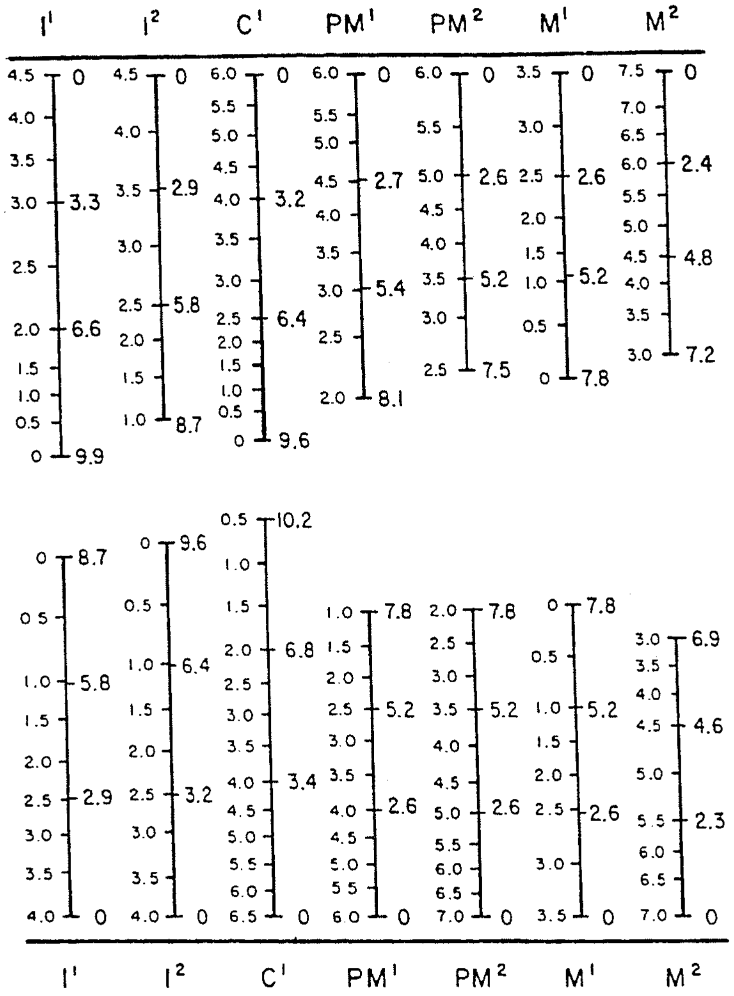

Fig. 9.3. Swärdstedt chart for estimating the age at development of enamel hypoplasias. The numbers to the right of each line are distances from the cemento-enamel junction to the LEH (in mm). The numbers to the left of each line are the corresponding ages at formation of an LEH in years. The crown heights are from Swärdstedt (1966) and the beginning and ending of crown development are adopted from Massler *et al.* (1941; also see Fig. 9.2). Super- and subscripts denote upper and lower jaws, respectively. (From Goodman *et al.* 1980, modified from Swärdstedt 1966.)

Table 9.1. *Regression equations to translate age at development of an LEH from its location, starting and ending age at crown calcification and crown height*

Tooth	Formulae[a]
Maxillary teeth	
I1	Age = − (0.454 × *Ht*) + 4.5
I2	Age = − (0.402 × *Ht*) + 4.5
C	Age = − (0.625 × *Ht*) + 6.0
P1	Age = − (0.494 × *Ht*) + 6.0
P2	Age = − (0.467 × *Ht*) + 6.0
M1	Age = − (0.448 × *Ht*) + 3.5
M2	Age = − (0.625 × *Ht*) + 7.5
Mandibular teeth	
I1	Age = − (0.460 × *Ht*) + 4.0
I2	Age = − (0.417 × *Ht*) + 4.0
C	Age = − (0.588 × *Ht*) + 6.5
P1	Age = − (0.641 × *Ht*) + 6.0
P2	Age = − (0.641 × *Ht*) + 7.0
M1	Age = − (0.449 × *Ht*) + 3.5
M2	Age = − (0.580 × *Ht*) + 7.0

[a] Age, age in years; Ht, distance of the LEH in mm from CEJ. Regression equations are based on mean crown heights of Swärdstedt (1966) and the crown formation standard of Massler *et al.* (1941).

Based in part on work of Murray and Murray (1989).

nearly all bioarchaeological studies to date.[3] In the following section we outline many of the assumptions behind it.

Sources of variation in estimation of age at formation of LEH

A wide variety of factors may cause variation between the estimated and actual age at formation of a LEH. We have organised these factors into five categories: measurement error, within- and among-population variation in developmental timing, within- and among-population variation in crown heights, interpretation of tooth histology and development, and choice of developmental standard (Table 9.2).

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[3] As we discuss below, a few authors have tried various corrections to the general method. For example, Hodges and Wilkinson (1990) have demonstrated the importance of population variation in crown heights and Song (1997) and Wright (1994, 1997) have both corrected for hidden cuspal enamel (discussed on pp. 221–224) and have 'shortened' the time of development of one or more canines.

Table 9.2. *Possible sources of variation and types of error in estimating the age at formation of linear enamel hypoplasias. Sources of error and variation focused upon in this chapter are shown in bold print*

Measurement error
Instrumentation and operator errors
Obliteration of CEJ anchor point
Measurement from center versus borders of LEH
Variation in developmental timing
Population variation in developmental timing
Sex differences in developmental timing
Within-population individual timing variation
Crown height variation
Population variation in crown height
Sex differences in crown height
Within population individual size variation
Correction related to enamel histology and developmental pattern
Changes in velocity of enamel surface extension
Buried cuspal enamel
Choice and interpretation of developmental standard

Measurement error refers to any variation in measured location of an enamel defect compared to the actual location (with location typically expressed as distance between the CEJ and the defect). While of obvious importance, especially when a defect is to be matched to a specific life event, this error is likely to be relatively minor compared to others noted below. In our experience, a typical error in measurement is of the order of 0.1–0.2 mm, which equals about a month of developmental time in permanent teeth and about a week for faster-developing deciduous teeth. Furthermore, at least part of this error is random and, therefore, may have little effect on population parameters.

Within- and among-population variation in tooth crown calcification is also likely and underexplored (Tompkins 1996). This variation may be characterised as overall advancement or delay, or variation in pattern of relative dental development (some teeth advanced or delayed relative to other teeth). Overall advancement in dental eruption has been proposed for females versus males (Demirjian 1986; Moorrees *et al.* 1963) better nourished individuals (Garn *et al.* 1965) and in African populations (Evelyth and Tanner 1976). The most easily interpreted results are of females vs. males. The extent of these differences may be around 2–3% (Gleiser and Hunt 1955; Garn *et al.* 1958; Demirjian and Levesque 1980;

Smith 1991). The cause of the difference may in part be due to the larger average size of teeth in males. If so, then the difference is not a sex difference per se (Moss and Moss-Salentijn 1977; also see Blakey and Armelagos 1985). While these population differences may be kept in mind when considering the accuracy of estimated ages at formation of enamel defects, at this time they are too small and too poorly understood to be systematically evaluated. More precise data are required before we can adequately address the importance of population variation in developmental timing.

In the following section, we focus on three sources of variation in timing of LEH development. One of these factors, tooth crown size variation, has previously been studied by Hodges and Wilkinson (1990). We review their results, extend their analysis, and then focus on two additional sources of variation: buried cuspal enamel and choice of developmental standard.

Buried cuspal enamel

Enamel matrix formation begins occlusally at the dentine–enamel border. As ameloblasts retreat toward the eventual surface of the tooth, cervically located ameloblasts begin forming the enamel matrix. The synchronised and combined action of these secretory ameloblasts forms a developing front of enamel, shaped like a loop in cross-section (Fig. 9.4). The striae of Retzius in this incipient enamel form partial circles (or loops) which begin and end in the inner enamel. Because these striae never reach the surface of the tooth, any disruption in enamel formation at the time of their formation may only be detected histologically, and not by surface observation.

The question for LEH analyses concerns the length of time in which domed enamel is formed, or 'How much time passes between initial crown formation and the first striae that reach the outer (sleeve) enamel?' At the present time, the few histological studies that address this have produced variable estimates. Early research by Bromage and Dean (1985) ascribed a period of 6 months for appositional enamel in permanent incisors. However, in reviewing the findings since then, primarily in the work of Dean and Beynon (1991), Bullion (1987), Bromage and Dean (1985), and FitzGerald (1995), this half-year period appears to be an underestimation (Table 9.3).

According to Bullion (1987), on average about 150 striae characterise incisors, over 180 striae are in permanent canine enamel, and molars average 120–150 striae (also see Hillson and Bond 1997). From these totals, the first 20–35 striae (approximately 15–20%) are 'hidden' in incisors and canines, while significantly more, the first 50–80 striae (approximately 30–50%), are not detectable on molar surfaces (Hillson 1996; Hillson and Bond 1997). The results of histological analyses indicate that the majority of appositional times, the time of formation of dome enamel, fall between the range 8–12 months (Table 9.3).

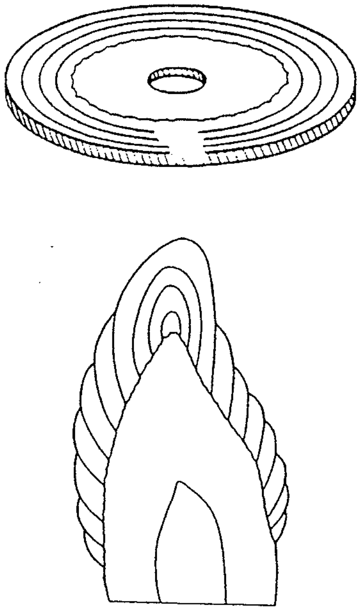

Fig. 9.4. Illustration of cuspal enamel (reproduced from Aiello and Dean 1990, with permission).

We are aware of only two LEH studies that have corrected for hidden period of appositional enamel (Wright 1994, 1997; Song 1997). Both studied ancient Maya samples. In her work, Wright estimated 1 year of non-visible 'buried' enamel for all tooth types. Song incorporated times from the survey table (Table 9.3; also see FitzGerald 1995: Table 12.5) to arrive at estimations ranging between 7 months (lower I1) to 10 and 12 months for all other teeth (Song 1997: Table 6.2.2).

What is the effect of correcting for this initial period of hidden development? To illustrate this correction, dental data from the Hamann–Todd human osteological collection were selected for study (Goodman 1988). The Hamann–Todd collection is made up of ethnically diverse individuals who died in the Cleveland area (northern Ohio) around the end of the 19th century and first quarter of the 20th century. Because no-one claimed them

Table 9.3. *A survey of estimated appositional enamel formation times from the literature* (*see FitzGerald 1995*)

Tooth source	Sample size	Appositional period (in months)
Maxillary I1		
Boyde (1963)	1	10.4
Bullion (1987) – mod.	3	9.0
Bullion (1987) – arch.	1	8.7
FitzGerald (1995)	2	11.4
Maxillary I2		
Bullion (1987) – mod.	4	11.6
Bullion (1987) – arch.	1	10.4
FitzGerald (1995)	2	12.8
Maxillary C		
Bullion (1987) – mod.	4	8.6
Dean and Beynon (1991)	1	12.3
FitzGerald (1995)	2	17.3
Mandibular I1		
Bromage and Dean (1985)	10	6.0
Bullion (1987) – mod.	6	7.5
Bullion (1987) – arch.	1	8.1
FitzGerald (1995)	3	6.3
Mandibular I2		
FitzGerald (1995)	*3*	*10.3*
Mandibular C		
Bullion (1987) – mod.	2	9.2
Bullion (1987) – arch.	1	9.5
FitzGerald (1995)	3	16.1

mod., modern; arch., archaeological.

and they underwent postmortem examination, individuals are generally considered to have been from the lower socioeconomic strata of society. Our data include the locations of moderate to severe hypoplasias on unworn mandibular canines and maxillary central incisors, the most frequently hypoplastic teeth. A total of 79 hypoplasias were found on the canines and 60 on the incisor.

On the basis of the regression equations, we plotted the estimated age at formation of an LEH on the y-axis vs. its location from the CEJ on the x-axis (Fig. 9.5). Formation ends at the CEJ at 4.5 years, on the basis of the development standard of Massler *et al.* (1941), and starts at the cusp,

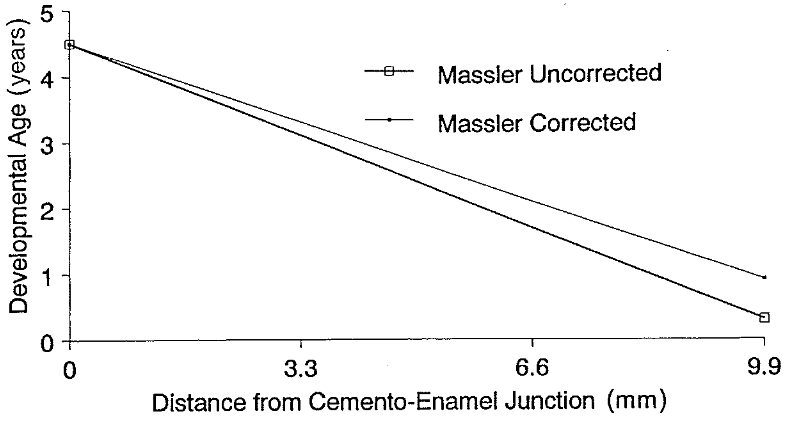

Fig. 9.5. The effect of a 6 month correction for buried cuspal enamel on the estimated age at development of an LEH on the upper central incisor.

9.9 mm from the CEJ, on the basis of the crown heights used initially by Swärdstedt (1966).

The effect of correcting for buried cuspal enamel is to change the length of time that the external crown forms and, thus, to change the 'velocity' term in the equation. Defects toward the cusp will be aged relatively older, while those toward the CEJ will be similarly aged. For example, using the conservative estimate of 6 months of hidden enamel, as originally proposed by Bromage and Dean (1985), yields a regression line that is 6 months advanced from the uncorrected line at the incisal tip. As the CEJ anchor point has not changed, the distance (in years) between the corrected and uncorrected regression lines decrease as they approach the CEJ (Fig. 9.5). In other words, the correction affects LEH ageing by a maximum of 6 months incisally, and then has a progressive decrease in consequence.

Applying the 6 month correction to the Hamann–Todd sample pushes back the mean age of an LEH by less than 3 months. For the upper central incisor, this correction pushes back the mean age at development from 2.51 to 2.73 years, and, for the lower canine, this correction pushes back the mean age at development from 4.10 to 4.30 years. As anticipated, the effect of the correction is asymmetrical, greater for earlier developing defects and least for later developing defects. Aged by the uncorrected method, the earliest incisor defect is aged to 0.64 years, compared to 1.07 years with the correction. Conversely, the latest defect for the incisor is aged at 3.91 and 3.97 years, respectively, by the two methods.

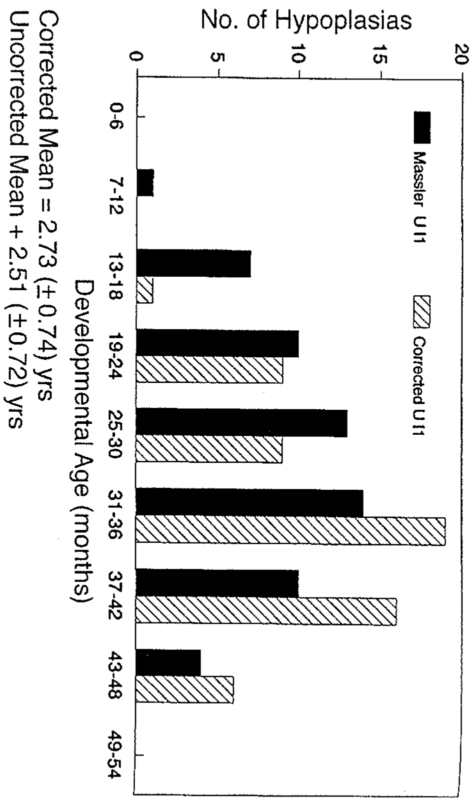

Fig. 9.6. The chronology of LEH by half-year developmental periods for the Hamann–Todd population with and without a 6 month correction for buried cuspal enamel.

The effect that the buried cuspal enamel correction has on the half-year distribution of enamel defects suggests a slight shift to older defects (Fig. 9.6). With the uncorrected method, eight defects are aged at 18 months or earlier, whereas only one defect is so aged by the corrected method. However, the peak half-year period is the same, 31–36 months of developmental age (Fig. 9.6).

A second test of correcting for hidden cuspal enamel involves re-examining the estimated mean age at development of enamel defects found in the Maya of Altun Ha, Belize (Song 1997; Table 9.4). Table 9.4 presents the results with and without correcting for hidden cuspal enamel. Corrections are for either 0.8 or 1.0 years, a rounded approximation of the averages from Table 9.3. The effect of the correction is to increase the mean age at formation of an LEH from a minimum of 0.24 years to 0.37 years (approximately 3 months to 4.5 months).

The change in estimated age at LEH formation based on hidden cuspal enamel are directional and biologically justified. We recommend that the correction should be implemented in future studies. Remaining questions concern the size of the correction and whether it should be used both with standards based on postmortem examination and those based on radiographic appearance of calcification.

Table 9.4. *The effect of correcting for hidden cuspal enamel on the estimated age (in years) of formation of LEH for the Ancient Maya of Altun Ha, utilising mean crown height standards of Swärdstedt (1966) and developmental timing of Massler et al. (1941)*

Tooth	Uncorrected: Equation A (no cuspal, Swärd, Massler)[a]	Equation A (Mean LEH age)	Cuspal enamel time (yrs) (Song)	Ext. dev. time (yrs) (Massler w/o cuspal)	Equation C (Cuspal, Swärd, Massler)	Equation C (Mean LEH age)	Diff. (yrs)
Upper							
I1	– 0.455x + 4.5	2.83	0.8	3.7	– 0.374x + 4.5	3.13	0.3
I2	– 0.402x + 4.5	3.19	1.0	2.5	– 0.287x + 4.5	3.56	0.37
C	– 0.625x + 6.0	3.78	1.0	5.0	– 0.521x + 6.0	4.15	0.37
P3	– 0.494x + 6.0	4.56	0.8	3.2	–0.395x + 6.0	4.85	0.29
P4	– 0.467x + 6.0	4.51	0.8	2.7	– 0.360x + 6.0	4.86	0.35
M1	– 0.449x + 3.5	2.44	0.8	2.7	– 0.371x + 3.5	2.68	0.24
M2	– 0.625x + 7.5	5.94	0.8	3.7	– 0.496x + 7.5	6.21	0.27
Lower							
I1	– 0.460x + 4.0	2.32	0.6	3.4	– 0.391x + 4.0	2.57	0.25
I2	– 0.417x + 4.0	2.47	0.8	3.2	– 0.333x + 4.0	2.77	0.3
C	– 0.588x + 6.5	4.31	1.0	5.0	– 0.490x + 6.5	4.67	0.36
P3	– 0.641x + 6.0	3.79	0.8	4.2	– 0.538x + 6.0	4.15	0.36
P4	– 0.641x + 7.0	5.35	0.8	4.2	– 0.538x + 7.0	5.62	0.27
M1	– 0.449x + 3.5	2.37	0.8	2.7	– 0.346x + 3.5	2.63	0.26
M2	– 0.580x + 7.0	5.28	0.8	3.2	– 0.464x + 7.0	5.63	0.35

cuspal, correction for cuspal enamel; w/o cuspal; without this correction; *x*, crown height in mm; Swärd, Swärdstedt (1966) standard; Massler, Massler *et al.* (1941) standard; Song, according to Song (1997); Diff., difference in mean age of formation (equation C – equation A).

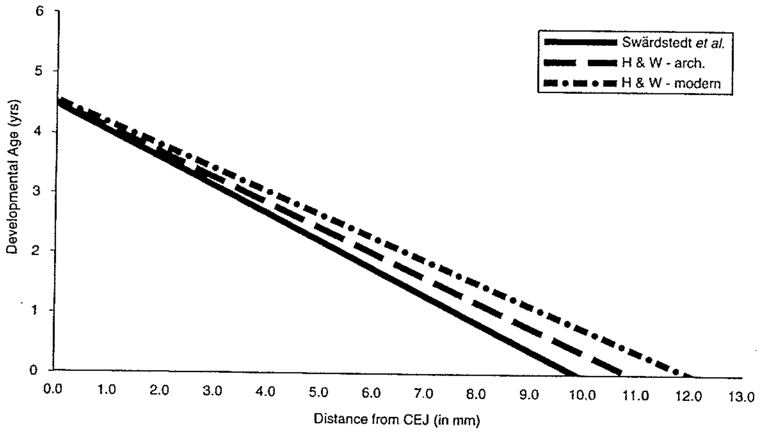

Fig. 9.7. The effect of correcting for larger upper central incisors on the estimated age at formation of an LEH. Three regression lines are shown. The darkest (bottom) line is based on the Swärdstedt tooth size, the intermediate line on tooth size based on the archaeological sample of Hodges and Wilkinson (1990) and the upper line on the modern sample tooth size of Hodges and Wilkinson (1990).

Crown height variation

The regression equations (Table 9.1) are also based on crown heights that were originally used in the Swärdstedt study. These are referenced to a study by Krogh-Poulsen (1950). Unfortunately, these crown heights have consistently been found to be small, and thus their use in the regression equation may have consequence, too, for the estimated age at formation of LEH. In the following, two types of variation are discussed: variation within populations and variation among populations.

Hodges and Wilkinson (1990) measured crown sizes for two samples: individuals with hypoplasias from prehistoric and historic skeletal collections from the eastern USA, and recently extracted teeth from donors living near urban areas in the northeastern and midwestern USA. They found that the maxillary central incisor increased from the 9.9 mm estimate in the original Swärdstedt method, to 10.86 mm in the archaeological sample and 11.99 in the contemporaneous sample. The change in mandibular canine is slightly less dramatic: from 10.2 mm in the original method to 10.92 mm and 11.78 mm in the archaeological and contemporaneous samples, respectively.

Assuming that larger teeth do not take longer to develop than shorter teeth of the same type produces a regression equation with a slower velocity but the same starting point. Showed graphically (Fig. 9.7) with the Hodges and Wilkinson incisor crown heights, it is clear that the majority of the variation occurs with earlier developing defects. The consequence of this correction is precisely similar in form to the hidden cuspal enamel correction.

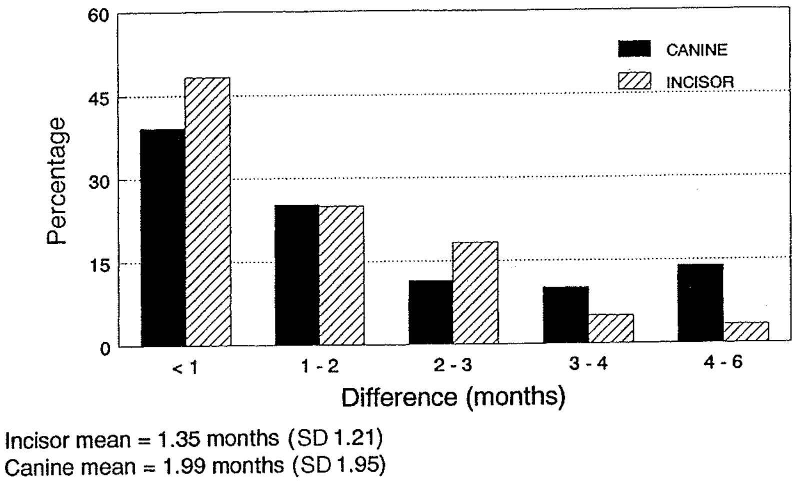

Fig. 9.8. The variation introduced in estimated ages of individual LEHs when the individual tooth size is substituted for the population's mean tooth size.

Hodges and Wilkinson (1990) found that using the population's own crown heights may significantly change the estimated age at formation of an LEH. Interestingly, their half-year peak age at formation of an enamel defect does not change for the archaeological sample, and changes by just a half-year for the larger-toothed, contemporaneous sample.

Hodges and Wilkinson (1990) also corrected for individual crown heights and found that this has little consequence on population parameters over the population correction. We concur that correcting for individual crown heights has little consequence for estimated age at formation of an LEH. With the Hamann–Todd sample, we examined the extent of variation between the individual and population crown height estimations (Fig. 9.8). The mean difference was 1.35 months for the upper central incisor and 1.99 months for the lower canine. As we expected, the consequence on the population distribution of LEH is negligible.

Similar to the crown heights found by Hodges and Wilkinson (1990), the Altun Ha crown heights are consistently larger than those originally used by Swärdstedt (Table 9.5). Only one crown height was actually smaller, the maxillary first molar, and many crowns were over 1.0 mm larger, including

Table 9.5. *The effect of correcting for mean population crown height on the estimated age (in years) of formation of LEH for the Ancient Maya of Altun Ha*

Tooth	Uncorrected equation A (no cuspal, Swärd, Massler)	Equation A (mean LEH age)	Swärd (Mean height)	AH (Mean height)	Crown Height diff.	Equation B (no cuspal, AH Massler)	(Mean LEH age)	Diff. (yrs)
Upper								
I1	– 0.455x + 4.5	2.83	9.9	11.77	1.87	– 0.314x + 4.5	3.1	0.27
I2	– 0.402x + 4.5	3.19	8.7	10.44	1.74	– 0.239x + 4.5	3.41	0.22
C	– 0.625x + 6.0	3.78	9.6	11.51	1.91	– 0.434x + 6.0	4.15	0.37
P3	– 0.494x + 6.0	4.56	8.1	8.56	0.46	– 0.374x + 6.0	4.64	0.08
P4	– 0.467x + 6.0	4.51	7.5	8.11	0.61	– 0.333x + 3.5	4.63	0.12
M1	– 0.449x + 3.5	2.44	7.8	7.27	– 0.53	– 0.371x + 3.5	2.36	– 0.08
M2	– 0.625x + 7.5	5.94	7.2	7.46	0.26	– 0.496x + 7.5	5.99	0.05
Lower								
I1	– 0.460x + 4.0	2.32	8.7	9.99	1.29	– 0.340x + 4.0	2.54	0.22
I2	– 0.417x + 4.0	2.47	9.6	9.91	0.31	– 0.323x + 4.0	2.51	0.04
C	– 0.588x + 6.5	4.31	10.2	12.03	1.83	– 0.416x + 6.5	4.64	0.33
P3	– 0.641x + 6.0	3.79	7.8	8.75	0.95	– 0.480x + 6.0	4.04	0.25
P4	– 0.641x + 7.0	5.35	7.8	8.62	0.82	– 0.487x + 7.0	5.51	0.16
M1	– 0.449x + 3.5	2.37	7.8	8.35	0.55	– 0.323x + 3.5	2.45	0.08
M2	– 0.580x + 7.0	5.28	6.9	7.39	0.49	– 0.433x + 7.0	5.4	0.12

cuspal, correction for cuspal enamel; w/o cuspal; without this correction; x, crown height in mm; Swärd, Swärdstedt (1966) standard; Massler, Massler *et al.* (1941) standard; AH, Altun Ha; Diff., difference in mean age of formation (equation B – equation A).

Table 9.6. *The effect of correcting for mean population crown height length and hidden cuspal enamel on the estimated age (in years) of formation of LEH for the Ancient Maya of Altun Ha*

Tooth	Uncorrected equation A (no cuspal, Swärd, Massler)	Uncorrected equation A (Mean LEH age)	Corrected equation A (cuspal, AH, Massler)	Corrected (Mean LEH age)	Diff. (yrs)
Upper					
I1	– 0.455x + 4.5	2.83	– 0.314x + 4.5	3.35	0.52
I2	– 0.402x + 4.5	3.19	– 0.239x + 4.5	3.72	0.53
C	– 0.625x + 6.0	3.78	– 0.434x + 6.0	4.49	0.68
P3	– 0.494x + 6.0	4.56	– 0.374x + 6.0	4.91	0.35
P4	– 0.467x + 6.0	4.51	– 0.333x + 6.0	4.94	0.43
M1	– 0.449x + 3.5	2.44	– 0.371x + 3.5	2.62	0.18
M2	– 0.625x + 7.5	5.94	– 0.496x + 7.5	6.26	0.32
Lower					
I1	– 0.460x + 4.0	2.32	– 0.340x + 4.0	2.76	0.44
I2	– 0.417x + 4.0	2.47	– 0.323x + 4.0	2.81	0.34
C	– 0.588x + 6.5	4.31	– 0.416x + 6.5	4.95	0.64
P3	– 0.641x + 6.0	3.79	– 0.480x + 6.0	4.35	0.56
P4	– 0.641x + 7.0	5.35	– 0.487x + 7.0	5.75	0.4
M1	– 0.449x + 3.5	2.37	– 0.323x + 3.5	2.69	0.32
M2	– 0.580x + 7.0	5.28	– 0.433x + 7.0	5.72	0.44

cuspal, correction for cuspal enamel; w/o cuspal; without this correction; x, crown height in mm; Swärd, Swärdstedt (1966) standard; Massler, Massler *et al.* (1941) standard; AH, Altun Ha; Diff., difference in mean age of formation (corrected – uncorrected).

five out of six of the anterior teeth. A longer crown has the effect of increasing the estimated age at formation of an LEH. The effect of using the overall greater population crown heights is to change the average age at formation of an LEH from – 0.08 years (upper first molar) to + 0.37 years (upper canine). Most corrections are less than 2 months, but interestingly the largest corrections are for canines.

What, then, is the consequence of simultaneously correcting for both hidden cuspal enamel and population crown heights? Using the Altun Ha data, one is able to see that the consequences are nearly additive (Table 9.6). The estimated mean age at formation of an LEH increases from 0.18 years to 0.68 years. The average adjustment tends to be between 4 and 6 months and both these corrections are most important in the ageing of early defects.

Choice of developmental standard

Mainly due to historical precedent, nearly all estimated ages at formation of LEHs are based on the tooth development diagram of Massler and co-workers (1941). As noted above, this standard is based on small sample sizes and individuals who died during tooth development. More recent developmental standards are based on a radiographic study of the degree of dental development, often with repeat radiographs of the same individuals. With the exception of the small study by Trodden (1982) that is based on native Canadian children, all of the modern growth studies are based on populations of European descent.

The key questions are, first, how much effect a change to a more recent developmental standard might have on the chronology of enamel hypoplasias and, secondly, which standard, if any, is most justified? As with skeletal growth research, can one standard be chosen as a universal standard for all studies?

Since the 1950s, over 10 studies have published data on the radiographic appearance of permanent crown development. All of them are generally lacking data on initial crown formation, especially for the earlier developing anterior teeth. These studies do, however, provide estimates of mean ages at termination of crown formation. To illustrate the importance of the choice of developmental standard in estimating the age at formation of LEH, we focus on variation among developmental standards in age at completion of crown calcification.

On the basis of a sampling of the most frequently referenced standards, sexes combined and averaged, the range of variation for upper central incisor completion extends from 3.3 to 5.5 years (Table 9.7). The Massler *et al.* (1941) incisor mean, at 4.5 years, is toward the centre of the distribution. The earliest-forming estimate is from the Finnish study by Haavikko (1974), at 3.3 years, and the latest two are from Moorrees, Fanning and Hunt (MFH) (Moorrees *et al.* 1963) for Boston area children and Trodden (1982) for her small sample of Canadian Indian children, at 5.1 and 5.5 years, respectively.

The lower canine standards are even more variable. While there is a general lag in canine development compared to the incisor, the standards are inconsistent in relative degree of delay. In other words, the relative development of the canine vs. the incisor shifts from standard to standard. The MFH canine develops relatively early compared to the incisor. At the other extreme, the Massler *et al.* (1941) standard includes a relatively early-developing incisor and the last-developing canine.

Applying different standards for age at crown completion to the Hamann–Todd data yields some highly variable ages at formation of LEHs (Table 9.8). The Haavikko (1974) standard yields a mean age LEH formation of 2.07 years for maxillary central incisor, whereas the Massler *et al.* (1941) standard is nearly 8 months later at 2.73 years, and the MFH standard is a full year later. Contrary to the hidden cuspal enamel and crown length corrections, the consequence of this standard change is minimal for earlier-developing defects and increases linearly with later developing defects.

The variation introduced by changing standards is even greater for the canine, and of further concern, because of the extreme position of the widely used MFH standard and the Massler (1941) standard (Table 9.9). For the Hamann–Todd data, the MFH standard yields a mean age at formation of LEH on lower canines of 2.70 years. From this earlier standard, the mean ages increase quickly to around 4.0 years for the Gustafson and Koch (1974) 'summary standard' and 4.3 years for the Massler *et al.* (1941) standard.

Table 9.7. *Comparison of ages (in years) at ending of crown formation of the upper central incisor and lower canine in different developmental standards*

Standards (Ref.)	Upper central incisor	Mandibular canine
Haavikko (1974)	3.3	4.3
Anderson *et al.* (1976)	3.7	4.5
Daito *et al.* (1990)	3.8	4.4
Nolla (1960)	4.5	5.8
Gustafson and Koch (1974)	4.5	6.0
Massler *et al.* (1941)	4.5	6.5
Moorrees *et al.* (1963)	5.1	3.8
Trodden (1982)	5.5	5.2

Table 9.8. *Central tendencies of estimated ages (in years) at formation of LEH on maxillary central incisors from Hamann–Todd using different developmental standards*

Standard (Ref.)	Mean	Median	Maximum	Minimum
Haavikko (1974)	2.07	2.06	2.93	0.90
Anderson *et al.* (1976)	2.26	2.25	3.24	0.95
Fass (1969)	2.51	2.50	3.63	1.02
Nielsen and Ravn (1976)	2.57	2.56	3.72	1.03
Massler *et al.* (1941)	2.73	2.72	3.97	1.07
Moorrees *et al.* (1963)	3.09	3.08	4.53	1.18

Table 9.9. *Central tendencies of estimated ages (in years) at formation of LEH on mandibular canines using different developmental standards*

Standard (Ref.)	Mean	Median	Maximum	Minimum
Moorrees *et al.* (1963)	2.70	2.81	3.55	1.28
Haavikko (1974)	2.95	3.07	3.93	1.34
Daito *et al.* (1990)	3.05	3.18	4.09	1.39
Anderson *et al.* (1976)	3.07	3.20	4.11	1.34
Nolla (1960)	3.88	4.06	5.32	1.47
Gustafson and Koch (1974)	4.00	4.19	5.51	1.49
Massler *et al.* (1941)	4.30	4.51	6.05	1.54

Figure 9.9 is a plot of the age distribution of LEHs on the Hamann–Todd canines by half-year developmental periods based on the Massler *et al.* (1941) and MFH estimated ages at crown completion. There is almost no overlap between the distributions: the MFH standard peaks between about 25 and 42 months, whereas the Massler standard yields a peak a full 2 years later.

Implications and recommendations

In this chapter we have endeavoured to determine the consequence of different sources of variation on estimated ages at formation of LEH. It should be clear that the sources of variation focused upon are just three of many possible sources of variation. A major goal of anthropology is to understand this variation.

Whereas more work is required to clarify the precise size of the correction, we feel that some corrections are now justified. These include using population crown heights, correcting for hidden cuspal enamel and moving forward the age at completion of canine crown formation from the extreme ages of the Massler *et al.* (1941) chronology.

Crown height

At present, there appears to be little consequence of correcting for individual variation in crown heights. However, correcting for population variation in crown heights does have a measurable consequence. Where possible, we recommend that LEH regression equations should be based on mean population crown heights. The correction we have experimented with in this chapter is to slow the rate of enamel formation. This is a simple correction that has consequences for earlier-developing defects. Whether it is biologically justified is the more important question, and unfortunately is not yet answered with absolute certainty. It may prove true that larger teeth simply grow for a longer period of time, rather than have greater growth velocity, as the current correction employs. If this alternative hypothesis proves true, then a different type of correction will need to be employed.

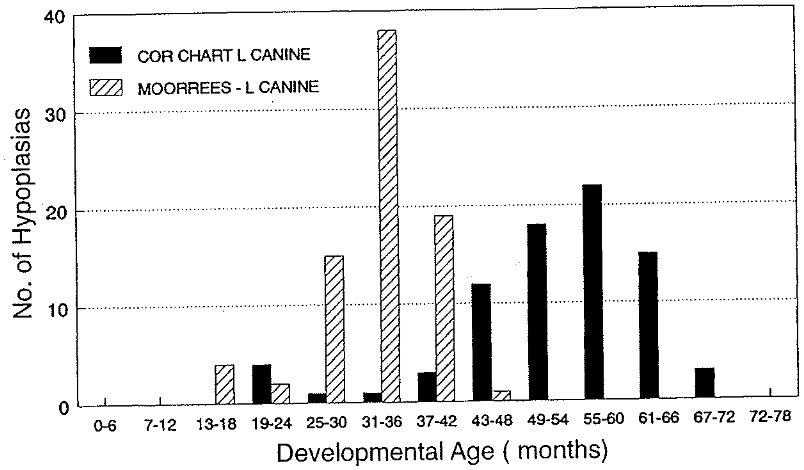

Fig. 9.9. Comparison of the chronology of LEH on lower canines with two different standards: Massler *et al.* (1941) (solid) and Moorrees *et al.* (1963) (hatched).

Another correction, and one that we would recommend for studies in which it is difficult to estimate population crown lengths, is to use a standard with larger teeth. The clearest issue concerned with the factor of crown size is that the existing standard is based on small teeth (Fig. 9.2; Table 9.1). Most studies of which we are aware find anterior teeth to be on the order of 5–15% longer. How much to 'lengthen' the tooth crown size standard is a question we leave for further study.

Hidden cuspal enamel

Correcting for hidden (dome or cuspal) enamel is biologically justified and significantly increases the ages of early hypoplasias. Here, also, further research is needed to better gauge the length of time between initial enamel apposition and the first formation of surface enamel. The 6 month correction used in the Hamann–Todd study appears now to be an underestimate. Conversely, Wright (1997) employed a full year correction on all teeth, and this may be an overestimate for some tooth types (Hillson and Bond 1997). Somewhat intermediate are the corrections used by Song (1997), which are based on the currently measured means (Table 9.3; see also FitzGerald 1995). This correction may be honed further with more studies of the length of time between initial enamel apposition and the first formation of surface enamel. At present only a few dozen teeth have been studied.

Finally there may be cause to 'dampen' this correction if it is applied to a radiographic standard. The reasons for this suggestion are that radiographic appearance is based on calcification and also requires some unquantified degree of mineralisation for tooth crown development to be radiographically visible. Thus these method-introduced delays may, in a sense, already account for the hidden cuspal enamel.

Developmental standards for the incisor and canine

The Massler *et al.* (1941) upper central incisor standard is generally close to, but perhaps somewhat delayed in comparison with, other developmental standards. Conversely, the Massler *et al.* (1941) age at completion of the lower canine is extremely late in comparison with other standards. It may, therefore, be reasonable to adjust the Massler *et al.* (1941) canine forward. The question is 'how much to adjust?'

Wright (1994, 1997) and Song (1997) have recently adjusted forward from 6.5 to 4.5 years the age at completion of the crown of the lower canine. This adjustment better correlates the temporal pattern of LEHs found on the canine with those found on incisor teeth. This adjustment, however, puts the canine completion before that found in most standards. We, therefore, recommend an intermediate adjustment forward of 1 year to 5.5 years at completion of the lower canine crown. It is probably also advisable to similarly adjust forward the upper canine's age at crown formation by 1 year, from 6.0 to 5.0 years. This recommendation, however, should be viewed as only temporary.

LEH, peak stress and weaning

A number of researchers have suggested that the peak period of LEH formation on permanent teeth might reflect weaning stress (e.g. Corruccini *et al.* 1985). Goodman and co-workers (1987) found that the peak in LEH formation in a contemporary sample of Mexican children was only slightly delayed from the mean age at documented termination of breast-feeding. Most studies of ancient groups find a peak age of hypoplasia well into the fourth or fifth year and there is disagreement as to whether this peak might correspond to age at termination of breastfeeding. Although this age is rather late, recent work such as that of Wright and Schwarcz (1998) suggests that breastfeeding might continue for this long.

Furthermore, the corrections we suggest above further push back the mean age at formation of LEHs, and thus increases the 'gap' between ages at completion of weaning and the peak age at LEH formation. We therefore caution against interpreting LEH patterns as reflecting the age at completion of weaning without additional sources upon which to suggest this inference.

Deciduous teeth

Although we have not addressed deciduous tooth LEHs, the study has some clear implications for the primary dentition. Most studies report much smaller variations in the time of deciduous crown formation. The standards problem may therefore be less serious. However, less is known of crown height variation; correcting for population crown height is recommended too for deciduous teeth, although sample sizes will obviously be restricted due to the requirement for unworn tooth crowns.

On the basis of the angle of accentuated striae of Retzius, including neonatal lines, it is certain that a greater percentage of deciduous tooth enamel development occurs as dome vs. sleeve enamel. Thus, correcting for dome/cuspal enamel is also likely to have great consequence for deciduous tooth LEH chronologies. In particular, it is not yet known with certainty at what approximate age the first striae reach the external surface of teeth. Finally, the location of the neonatal line provides an important potential landmark (Skinner 1992).

Implications beyond studies of LEH timing

The variation shown by enamel development chronologies is an issue that plagues studies beyond enamel hypoplasias. Depending on what standard is used, hominids can be made to resemble pongids, or to be seen as having a very different developmental pattern. In a creative study of the pattern of enamel defects, Owsley and Jantz (1983) found systematic variation between the Arikara (native North American) developmental pattern and that of the Moorrees *et al.* (1963) standard. However, it is not clear at this point whether the variation they found is due to different population patterns of development or the choice of developmental standard. Whereas these authors found many differences in pattern of development compared to the standard, a different standard would have yielded an entirely different set of deviations.

A similar problem plagues hominid/pongid comparisons. Dean and Wood (1981: 116) have produced a chart that compares a generalised pattern of calcification of humans and pongids. Their human canine crown completes calcification at around the same time as the incisors, which is in agreement with some, but not all, of the standards we have reviewed (see Table 9.7). This interpretation sets off the lengthened age of canine crown formation in pongids vs. hominids. It is noteworthy that the Massler *et al.* (1941) chronology resembles the pongid pattern better than it does the human pattern. The point is that the degree of variation among human standards is so great that it may obscure phylogenetic comparison.

Conclusions

We have pointed out significant sources of variation in the estimated age at formation of enamel defects. Clearly, more research is needed into these and other sources of variation. However, we do not wish to suggest that there is no value in recording the position of enamel defects on tooth crowns and trying to estimate their age at development. Our intention is exactly the opposite. Precise location of LEHs and precise location of accentuated striae of Retzius are required to tap the potential of LEH analyses, and these data may also be key to understanding the relative pattern of development of different teeth.

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10 *Reconstructing patterns of growth disruption from enamel microstructure*

SCOTT W. SIMPSON

Introduction

The earliest years of life present the greatest risk of sickness and death. An understanding of the causes of age-specific morbidity and mortality in the youngest cohort can produce a very useful model of the patterns of adaptation in a population. Palaeoepidemiological studies rely on the hard tissues as the primary medium of analysis. In the study of past disease episodes, we must rely on individuals that lived with, or recovered from, some pathological state, thus retaining a record of this homeostatic imbalance. Accurate data of the timing, duration and frequency of metabolic disruptions are necessary to provide insight into their cause. Growth disruptions that occurred during the first years of life may leave a characteristic signature in the tissues developing at that time. Teeth are useful because they develop over a very characteristic schedule, are formed incrementally, do not remodel, and have unique metabolic demands during formation.

In this chapter, the temporal patterning and relationship between two types of structural defect in the enamel crown (pathological striae of Retzius and surface defects) are explored in a New World lineage. These data can be used to develop hypotheses of the frequency and types of disease that affected the study group. The population lived in the southeastern USA (Florida) between AD 1 and AD 1704. During this period, marked changes occurred within Native American population. In the prehistoric period (AD 1–AD 1565), the indigenes made a transition from gathering–hunting economic base to one increasingly focused on maize agriculture occurring around AD 600 (Larsen 1982). In the middle of the 16th century, this area was the focus of European (primarily Spanish) exploration and missionisation. Thus ultimately led to the displacement, extinction, or incorporation of the native groups. Within this area and during this time, two significant changes occurred: the adoption of intensive agriculture and Spanish missionisation. The implications of these