Enamel hypoplasias and physiological stress in the Sima de los Huesos Middle Pleistocene hominins

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ABSTRACT

This study presents an analysis of linear enamel hypoplasias (LEH) and plane-form defects (PFD) in the hominine dental sample from the Sima de los Huesos (SH) Middle Pleistocene site in Atapuerca (Spain). The SH sample comprises 475 teeth, 467 permanent and 8 deciduous, belonging to a minimum of 28 individuals. The method for recording PFD and LEH is discussed as well as the definition of LEH.

The prevalence of LEH and PFD in SH permanent dentition (unilateral total count) is 4.6% (13/280). Only one deciduous tooth (lower dc) showed an enamel disruption. The prevalence by individual ranges from 18.7% to 30%.

The likeliest explanation for the relatively low LEH and PFD prevalence in the SH sample suggest that the SH population exhibited a low level of developmental stress.

The age at occurrence of LEH and PFD was determined by counting the number of perikymata between each lesion and the cervix of the tooth. Assuming a periodicity of 9 days for the incremental lines, the majority of LEH in the SH sample occurred during the 3rd year of life and may be related to the metabolic stress associated with weaning.

Hypoplasias are enamel surface alterations produced by a disruption in enamel formation processes generally attributed to stress that occurs during childhood. This subject has been extensively discussed in the anthropology literature because it is an informative source about *developmental stress* suffered by past populations (Skinner, 1996). The study of dental enamel defects, allows us to estimate childhood morbidity prevalence. By this means, we can evaluate the timing of stress episodes underlying the enamel defects – infections, illness, fevers and dietary deficiencies among others in modern and archaeological populations.

Hypoplasias have been recorded in fossil hominins as well as in the greater and lesser apes (see Guatelli-Steinberg, 2001; Lukacs, 2001a,b; Schwartz et al., 2001; Skinner, 1996; Ogilvie et al., 1989). The majority of studies have employed the classic method of determining the presence or absence of enamel defects solely by macroscopic inspection. Enamel hypoplasias were defined as developmental defects on the surface of the enamel, usually macroscopically distinguished as an area of decreased enamel thickness (Sarnat and Schour, 1941; Goodman et al., 1980; Goodman and Song, 1999). The timing of such hypoplasias formation was estimated in relation to crown height, as proposed by Goodman et al. (1980).

It is now known that enamel dental defects are a consequence of disruption in the enamel formation processes. Hillson and Bond (1997) describe the relationship between enamel hypoplasias and the internal enamel structure. Perikymata are the external manifestation of the enamel incremental lines - the striae of Retzius -which commonly have a formation periodicity of 8 - 11 days, with a modal value of 9 days. Counting the number of perkitymata between hypoplasias and the cervix allows the calculation of the timing of hypoplastic defects (Reid and Dean, 2000) in a more precise manner than relying on crown height (Hillson and Bond, 1997; Reid and Dean, 2000). Accordingly we have counted hypoplasias in the context of an analysis of perikymata.

The present study aims to evaluate the prevalence of enamel hypoplasias in the Middle Pleistocene hominine sample from Sima de los Huesos, Atapuerca (Spain). Although the SH fossil teeth have previously been studied, (Bermúdez de Castro and Pérez, 1995), two factors that call for the re-analysis. First, due to fieldwork in the intervening years, the sample has practically doubled. Second, the method to be used to record hypoplasias and their developmental age should yield more accurate results. Within the limitations derived from the use of different methodologies, our results will be compared with those obtained for Neanderthals populations (Ogilvie et al., 1989; Skinner, 1996). The significance of the prevalence and severity of the enamel defects will be discussed, and the general pattern of SH hominine growth, in terms of developmental stress, will be examined.

The Sima de los Huesos (SH) site

SH is a Middle Pleistocene site located inside the Cueva Mayor-Cueva del Silo karst system in Sierra de Atapuerca, Spain. Although the first SH human remains were recovered nearly a quarter of a century ago (Aguirre et al., 1976), the great majority of the sample derives from later systematic excavations since 1984. As of 2001, the bone breccias of the SH site have yielded a large hominine sample of more than 4,000 fossils.

Previous radiometric and paleomagnetic analyses, as well as biochronological data, pointed an age interval of 200 to 320 kyrs (OIS 7 to 9) for the SH hominins (Bischoff et al., 1997; Cuenca-Bescós et al., 1997; Parés et al., 2000). Recent radiometric study (U-series) of a 14 cm-thick in-situ speleothem overlying the mud-breccia containing the SH hominine bones has provided a minimum age of 350 kyrs (Bischoff et al., 2003). Estimations of the speleothem growth rate, correlation of the SH fauna (micro- and macromammals) with other Atapuerca sites (e.g., TD6, TD8, TD10, and TD11 Gran Dolina levels), and the normal

magnetization of the SH fossilifereous mud give an interval of 400 to 500 kyrs (OIS 12 to 14) for these hominins (Bischoff et al., 2003).

SH hominins have been assigned to the species *Homo heidelbergensis* (Arsuaga et al., 1997a). All human bones were deposited during the same sedimentation period (Episode II) (Arsuaga et al., 1997b) and, because of their relative morphological homogeneity, they probably belong to a single contemporary population (Arsuaga et al., 1997c; Bermúdez de Castro, 1988; Carretero et al., 1997; Rosas, 1997). Phylogenetically, SH hominins have been considered the direct ancestors of Neanderthals (Arsuaga et al., 1993; Arsuaga et al., 1997 a,b,c; Rosas and Bermúdez de Castro, 1998; Bermúdez de Castro, 2002).

MATERIALS AND METHODS

In a previous study of enamel hypoplasias in the SH hominine dental sample (Bermúdez de Castro and Pérez, 1995) it was estimated that the teeth represented a minimum of 29 individuals. Because of a notable increase in mandible, maxilla and tooth specimens recovered from recent excavations, a new MNI determination was required. Overall, the majority of the new teeth are assigned to individuals previously identified. We observed that teeth previously assigned to individuals VII and XIII belong to the same individual, and some teeth have been re-assigned to different individuals identified in the previous study. For instance, isolated teeth such as AT-197 (right upper I1) and AT-282 (right lower I2) are no longer assigned to individual XXII. The new MNI is 28. The list of the teeth and individuals included in this study is listed in Appendix A. Individuals V, XIII and XXI were excluded from this study because their teeth are too worn to determine whether they have hypoplasias or not.

The current SH dental sample includes 475 teeth, 467 permanent and 8 deciduous. Ninety-seven teeth are not assigned to any individual. The distribution by upper and lower jaws of the permanent teeth is as follows: 78 *in situ* mandibular teeth, 34 *in situ* maxillary teeth, 198 isolated mandibular teeth and 157 isolated maxillary teeth.

Linear enamel hypoplasias (LEH: lines and grooves, Sarnat and Schour, 194; furrow-form defect, Hillson and Bond, 1997) and plane-form defects (PFD) were recorded (Hillson and Bond, 1997). In the present study, pits were not included since their association with perikymata is very difficult to determine from the analysis of the enamel surface alone (Hillson and Bond, 1997). In order to identify LEH and PFD in the SH dental sample, all teeth were examined several times by three different observers using a low-power binocular microscope Leica Wild MZ8 (0.63 x5). A fourth author checked the determinations. In addition, teeth were replicated using the addition-curing Coltene President in its variants putty and light (Beynon, 1987). Epoxy resin casts (RBS, T2L Chimie) were made from each silicone replica. Casts and original teeth were examined in reflected light with a Wild M8 stereomicroscope. Analysis was completed with the observation by SEM of casts that had been previously sputter-coated with gold.

Most previous studies of hypoplasia have identified enamel defects solely by macroscopic inspection. However, we believe that the examination of hypoplasia should include microscopic observation, as we have done in our study. An examination based exclusively in macroscopic inspection can lead to recording as hypoplasias such artifacts as changes in enamel coloration or inflexions of the enamel surface.

Recording hypoplasias: some improvements

The Developmental Defects Enamel Index (DDE Index) defines hypoplasia as “a defect involving the surface of the enamel and associated with a reduced localized thickness of

enamel", and it states that, "developmental defects of enamel are deviations from the normal appearance of tooth enamel resulting from enamel organ dysfunction" (Commission on Oral Health, Research and Epidemiology, 1992:419). From this statement, it is obvious that hypoplasias should be seen as a disruption in the process of crown formation. However, the DDE Index also indicates that many authors prefer a descriptive interpretation of hypoplasias, and even the DDE Index itself takes this position when describing linear hypoplasias (grooves) as a "partial or complete absence of enamel over a considerable area of dentine" (Commission on Oral Health, Research and Epidemiology, 1992:419). Hypoplasias have been interpreted from a descriptive point of view in almost all works on modern and archaeological populations. The relationship to crown formation was considered only for the estimation of the timing of the stress episode during growth. However, our knowledge of stress in populations would be improved in scope and precision if the study of hypoplasias is integrated with a thorough understanding of the crown formation process. Hillson and Bond (1997) have explained the relationship existing between hypoplasias and the underlying enamel structure. The striae of Retzius are enamel incremental lines which form commonly every 8-11 days, which vary between individuals, with a modal value of 9 days; represent successive steps in the advancement of enamel organ function. The striae of Retzius contact the enamel surface in the imbricational part of the crown and form the perikymata. In normal enamel, the perikymata are visible as wave-like forms closer to the occlusal/incisal aspect of the crown, and appear more ridge-like in the cervical region, becoming more closely spaced. Any dysfunction in the process of enamel matrix secretion, i.e., the proximate cause of hypoplasias, will be revealed by a change in the distribution of the striae of Retzius and, in turn, a modification in the appearance of the perikymata.

Hypoplasias are thought to arise from episodic events that disrupt physiology sufficiently to affect the production of enamel matrix. They are systemic, one disruption

producing hypoplasias in all teeth growing at the time of the disruption. Because teeth are not all at the same stage of development, the hypoplastic event will appear at different levels in tooth crowns. However, the manifestation of hypoplasia is not the same in all tooth crowns affected. The variation may be related either to differences in "susceptibility" between tooth classes (Goodman and Armelagos, 1985a,b) or to crown growth geometry (Hillson and Bond, 1997). A clearly identifiable hypoplasia in one tooth class may correspond to a weakly defined change in the perikymata spacing in another tooth class. This situation necessitates a definition of the minimum level of expression considered to be true hypoplasia. The definition of what is considered to be defective or normal is still a problematic point, and is one of the major concerns in Hillson and Bond's discussion (1997). At this point, we question whether a difference in distance between adjacent perikymata should be considered as hypoplasia. Microscopic inspection of enamel surface reveals that the distance between adjacent perikymata does not decrease in a strictly regular manner, that is, the spacing is not always smaller towards the cervix. Some variation in the perikymata pattern must be considered as normal biological variability. It is also noteworthy that changes in the distance between perikymata in relation to LEH (grooves) are not always more marked than in normal variation (Fig. 2; see figure 6 in Hillson and Bond, 1997). The identification of two distinct regions in a LEH groove (Hillson and Bond, 1997), one at the occlusal margin where the perikymata are more widely spaced and one at the cervical margin where the perikymata are closer, is not always evident. Therefore, we believe that a single variation in the distance between adjacent perikymata cannot be taken on its own to suggest an enamel formation dysfunction, since it would fall within the normal range of variability (Fig. 1). We understand linear hypoplasias as representations of a dysfunction in enamel organ function, manifested as a localized reduction of enamel thickness that may or may not be accompanied by a marked alteration in the distance between adjacent perikymata. According to this definition, the alteration of the

perikymata spacing alone is not enough to identify hypoplasia. Our observations of LEH and PFD in the teeth from Sima de los Huesos are based on this definition.

The minimum level of alteration of the enamel surface that should be considered as an enamel defect, however, remains arbitrary. Some apparent reductions in the enamel thickness are due to the curvature of the enamel surface. Even when enamel thickness reduction is present, some depressions in the enamel surface are very shallow and their attribution to hypoplasia is questionable. The same problem is found with changes in distance between perikymata, which appear together with shallow depressions of the enamel surface. This apparent discordance invites us to be cautious when recording LEH defects. However, a distinction between normality and LEH, although arbitrary, should be established. In our study, we consider the enamel surface disruptions seen on individual XXIV's lower canine (Fig. 3) as the minimum limit of LEH expression.

Estimation of timing of hypoplasia formation

An important aspect of hypoplasia studies is to determine at which point in time during growth the stress responsible for hypoplasia occurred. As mentioned above, almost all works on hypoplasia have established the timing of hypoplasias by relating crown height to crown formation time (Goodman and Rose, 1991). However, this method is founded on two unsupported assumptions: that crown height is equivalent to total crown formation time, and that crown formation occurs at a steady rate. The first assumption would imply that a hypoplasia located at mid-crown would have formed at the mid-point of crown formation, and also that crown formation starts at the cusp tip and finishes at the cervix. However, this is not correct. When the cusp tip is completely formed, crown formation has already been in process for some time. Enamel located between the dentine horn and the cusp tip corresponds to the appositional enamel of the crown. The time frame for enamel apposition is different depending

on the tooth class, being shorter for incisors and canines - less than 20% of total crown formation time - and longer for molars in modern humans -more than 30% of the total formation time (Reid et al., 1998). In *Paranthropus aethiopicus*, the time span for appositional enamel formation reaches the highest value -more than 60% of the total time of molar crown formation (Ramirez Rozzi, 1993, 1998). The second assumption is related to the fact that hypoplasias can only be observed in the imbricational enamel, i.e. the part of the crown where the striae of Retzius contact the enamel surface and form perikymata. Perikymata do not have a constant distance between them, being more closely spaced towards the cervix (Dean and Reid, 2001). This means that the quantity of enamel formed at a given time (the interval between striae) is less towards the end of crown formation. Therefore, imbricational crown formation does not occur at a steady rate, but slows down as the cervical region is formed. A hypoplasia at mid-crown height was not formed at the exact midpoint of the imbricational crown formation time, it was formed sometime before. One way to resolve these two critical points is to establish the timing of hypoplasia by counting the perikymata (Reid and Dean, 2000).

In our study, counting the perikymata on teeth was carried out on those specimens in the SH sample with identified hypoplasia. To facilitate counting, each tooth replica was positioned with the buccal face orthogonal to the optical axis of the microscope. Buccal enamel was then divided into 10 equal divisions (10th percentiles) from the first formed enamel at the cusp to the last formed at the cervix (Reid and Dean, 2000; Dean and Reid, 2001). For that purpose, a vernier micrometer eyepiece connected to a digital ocular measure linked to a calculator-meter-printer RZD-DO (Leica) was used. Perikymata counts were made in each of the divisions from the hypoplasia to the cervix. It has been accepted that when hypoplasia involves only one perikymata, the affected one is the perikymata located at the greatest depth of the groove. If the hypoplasia involves more than one incremental line, the

stress occurred when the perikymata in the incisal/occlusal aspect of the groove were being formed (Hillson and Bond, 1997). The interval between adjacent perikymata varies commonly from 8 to 11 days between individuals, with a modal value of 9 days for Plio-Pleistocene hominine species, Pleistocene *Homo* species and modern humans (Fitzgerald, 1998; Dean and Reid, 2001). The age at onset and completion of crown formation for each tooth class is known for modern humans (e.g. Reid et al., 1998; Simpson and Kunos, 1998) but not for *H. heidelbergensis*. The only way to know crown formation time and the age at onset and completion of crown formation in each tooth type is to take histological sections of teeth from a complete dentition (Reid et al. 1998). At moment, it is impossible to carry out this methodology in fossil hominid teeth from Atapuerca. However, the study of some of the enamel structures can help to estimate the length of crown formation time in fossil hominid species (Dean 1987, Dean et al. 2001), specially the perikymata spacing pattern (Dean et al. 1986, Dean & Reid 2001).

The length of time of crown formation is influenced by the extension rate of enamel, that is, by the number of ameloblasts that are recruited each day. It reflects the advance of the enamel-matrix front. In the inner layers of the enamel, the extension rate can be estimated by the angle of the slope of the striae of Retzius at their intersection with the enamel-dentine junction: the more acute the slope, the higher the extension rate. At the enamel surface, the extension rate of enamel can be deduced from the spacing between the perikymata (Dean et al. 1986, Dean & Reid 2001). In modern humans, the extension rate becomes lower from the cusp tip towards the cervix. At the enamel surface, the decrease in the extension rate of enamel is reflected by the reduction of the space between perikymata. This defines a characteristic perikymata packing pattern (number and distribution) in modern humans: perikymata are more widely spaced near the cusp tip, becoming closer toward the cervix (Dean & Reid, 2001). Thus, changes in the extension rate during crown formation can be

estimated from the number and the spacing variation of perikymata. Different perikymata spacing patterns are found in great apes, *Australopithecus*, *Paranthropus*, and modern humans, which is in accordance with differences in crown formation time between these groups (Dean & Reid 2001). Fossil *Homo* species show different perikymata spacing patterns; for example, *H. rudolfensis* and *H. erectus* have a resemblance to *Australopithecus* and *Paranthropus*, while *H. habilis* plots within the modern human range (Dean & Reid 2001). "The shifts in perikymata spacing and distribution seem to be associated with changes to the timing of crown formation rather than to changes in tooth height" (Dean & Reid 2001:214).

Homo heidelbergensis from Sima de los Huesos presents a perikymata spacing pattern in the anterior teeth and premolars similar to that in modern humans (Bermúdez de Castro et al. in press; Ramirez Rozzi and Bermúdez de Castro, 2001). As noted above, crown formation time can only be known from the study of sections of teeth. However, since the extension rate of enamel influences crown formation time and the perikymata spacing pattern seems to be associated with crown formation time, a similar perikymata pattern in *H. heidelbergensis* and modern humans may suggest analogous crown formation times.

Bermúdez de Castro and colleagues have suggested that the pattern of dental development in *H. heidelbergensis* from SH was similar to that in modern humans (Bermúdez de Castro et al., 1999, 2002). Based on the assumption of a similar crown formation time and a similar pattern of dental development, we estimated ages of onset and completion of crown formation in *H. heidelbergensis* similar to those in modern humans. To obtain an estimate of the timing of formation of each hypoplasia, the time elapsed from the formation of the lesion (calculated from the counting of perikymata) to crown completion was subtracted from the age at the completion of crown formation.

RESULTS

Table 1 lists maxillary and mandibular teeth exhibiting enamel defects by individual, as well as isolated teeth. The estimated developmental age for each defect is also presented. Table 2 presents the prevalence of LEH and PFD for each permanent tooth class. We employed the unilateral count method, that is, we regularly recorded defects in the right antimere of each tooth pair, referring to the left antimere only when the right antimere was missing. Table 3 shows an estimation of the minimum number of individuals affected by at least one tooth affected with an hypoplastic defect.

For permanent teeth, the most affected tooth class is the lower incisor (3 cases). Lower teeth are significantly more often affected than upper teeth, with 11/13 (84.6%) and 2/13 (15.4%) of 13 specimens affected, respectively. For deciduous teeth, only one eight (12.5%) presents a hypoplasia.

A minimum of 25 individuals has been considered in our study (including one child, individual IX). Of the 25, 7 have at least one hypoplastic defect (28%). Three of them (42.9%) are males, two (28.6%) are females, and two are of undetermined sex (Bermúdez de Castro et al., 2001). Six isolated teeth show hypoplasias. Of these, 5 (AT-2384, AT-3242, AT-197, AT-282, and AT-608) probably belong to the same individual, with AT-2384 and AT-3242 fitting in a mandible fragment (AT-506). In the total number of tooth specimens, the prevalence of hypoplasia is 5.64% (25/443). For permanent teeth only, 5.52 % (24/435) show a minimum of one enamel defect. When antimeres are excluded, this figure drops to 4.64% (13/280). In 10 cases the enamel defects were present in both right and left antimeres.

Regarding the degree of hypoplasia expression in affected individuals, the limits established to differentiate normality from defects (Fig. 3) indicate that hypoplasias occurred in some but not all teeth of a given individual. For instance, the lower canines of individual XVIII show hypoplasias whereas the upper canines present shallow depressions that do not

meet the established criteria for hypoplasias, though they correspond in developmental age to the lower tooth defect. A similar case is found in individual XXII, where the lower I1 and I2 present hypoplasias but the enamel surface depression in the upper right I2 cannot be classified as hypoplasia. Thus, the degree of expression varies in our sample, from surface depressions (Fig. 3) to the deep groove in AT-1144 (Fig. 4).

Variation is also observed in the duration of the stress episodes that produced hypoplastic lesions. In the right LP4 from individual XXVII, the hypoplasia comprises 7 perikymata, but in individual XXIV, in 3 out of the 4 teeth with hypoplasia the defective enamel only comprises one perikymata. It is noteworthy that in those 3 teeth, the perikymata formed immediately previous to the hypoplastic lesion is elevated over the normal level of the enamel surface, probably due to an over-production of enamel, thus accentuating the hypoplasia groove. Only 2 teeth, from individual XXIV and XXVII, present two hypoplasias on the buccal face. In individual XXIV, both hypoplasias are very close in time, separated only by 11 perikymata, and could be interpreted as two manifestations of the same perturbation. It is important to note that all hypoplasias were identified in lower teeth, except in individual XIX and in one isolated tooth. We postulate that the lower dentition may be more susceptible than the upper dentition to tooth formation dysfunctions. In individual XIX, the PFD affects only the buccal face of the upper M3 (Fig. 5).

In Table 1, it can be seen that stress occurrence is clustered between the ages of 0 and 6 years (third molars excluded), with the third year of life being the most physiologically stressful.

In individual XXIV, the hypoplasia on the left lower canine has a developmental age of 3.84 years and seems to be the result of an enamel organ dysfunction different from the one affecting the incisors, whose mean developmental age is 2.73 yrs. However, it is possible that the seemingly different timing in defects of the lower canines and incisors in this individual

derives from the application of modern human standards for lower canine crown offset to *Homo heidelbergensis* species. The study of the perikymata pattern in *H. heidelbergensis* from Sima de los Huesos suggests that anterior teeth crown formation time in this fossil species was similar to that in modern humans in all teeth except the upper I1 and the lower canine (Ramirez Rozzi and Bermúdez de Castro, 2001; Bermúdez de Castro et al., in press). Indeed, the upper I1 and the lower canines present different perikymata spacing patterns in *H. heidelbergensis*, which would suggest a shorter crown formation time than in modern humans. A recent analysis of dentitions in a sample of South African Black individuals suggests a shorter canine crown formation time than that for European individuals, probably indicating a difference between ethnic groups (Reid, pers. comm.). Therefore, if lower canine crown formation time was shorter, it is likely that this tooth offset occurred at an earlier age in *H. heidelbergensis*. Hence, it is possible that the disruption underlying the canine hypoplasia happened at an earlier time during growth, matching the age of the incisors' defects.

In the 3 isolated lower incisors (AT-2384, AT-282 and AT-608) belonging to one individual, the hypoplasias' timing is very close, around 1.85 years. Nevertheless, the hypoplasia in the upper I1 (AT-197) of this same individual seems to have been formed later (2.88 years). As stated before, upper I1s have a shorter crown formation time in *H. heidelbergensis* than in modern humans (Ramírez Rozzi and Bermúdez de Castro 2001, Bermudez de Castro et al. in press) and probably an earlier offset has occurred, as with the lower canine described above. Thus, the timing of the AT-197 hypoplasia could be earlier and in accordance with the age obtained for the remaining isolated teeth. In individual XVIII, the only teeth presenting hypoplasia are the lower canines. The estimated age of occurrence is about 4.5 years, although considering the possibly shorter crown formation time in lower canines as well as its earlier offset, the hypoplasia could have occurred at an earlier age. A small difference between the timing of defects is found in individual XXII: for the incisors it is

around 2.2 years, whereas for the lower M1 it is around 2.8 years. This difference may mean that two different causes at different developmental ages disrupted dental formation. However, the possibility that the same disruption produced the hypoplasias in both the incisors and the M1 cannot be discarded. In that case, a shorter M1 crown formation time in *H. heidelbergensis* compared to modern humans could be hypothesized.

DISCUSSION

This study presents data from a European population from the Middle Pleistocene. Except for a previous study of the same sample (Bermúdez de Castro and Pérez, 1995), there are no other published data on DEH prevalence in *Homo* populations from Lower and Middle Pleistocene. Most of the SH hominids are represented by at least one complete half-arcade of dentition, making this the most complete sample recovered from a Pleistocene site.

The incorporation of observations on microstructure variations with data on macroscopic enamel defects significantly improves the precision and reliability of identification of developmental defects (King et al., 2002). It also permits the detection of artifacts that resemble hypoplasias on the macroscopic level, and provides increased accuracy in the estimation of hypoplasia chronology. Finally, it ensures a better understanding of systemic stress throughout the entire period of growth.

Comparing the results obtained in this study with the former one (Bermúdez de Castro and Pérez, 1995) we can observe that the prevalence of hypoplasia in the SH permanent dentition has decreased to less than the half (12.8 % *vs.* 5.52%). In the first study, the hypoplasia prevalence *per* individual was less than the 40%. With the new information available, we have obtained a prevalence *per* individual in the SH-sample between the 28% (7/25) and the 30% (8/26, if we consider that the isolated teeth belong to a different individual

which has not been identified yet). In the former study it was stated that the most affected teeth were upper and lower canines whereas in this new study the most affected teeth are lower incisors. Finally, in the previous study it was determined that the mean age of hypoplasias occurrence, between birth and 7 years, was 3.5 years (N=18, SD=1.3). In the present study the mean age of occurrence is 2.9 years (N=25, SD=1.29). The differences between these two values are not statistically significant (t=1.57, df=41, p=0.123).

Hypoplasia in the deciduous dentition is associated with physiological stress in the early stages of ontogenetic development (Lukacs, 2001a,b), and the canines are most commonly affected in modern humans and other primates (Skinner, 1996; Lukacs, 2001 a,b; Lukacs, 2001b; Lukacs and Nelson, 2001). In our sample of 8 deciduous teeth, only one canine was affected; however, since it was the only canine in the sample we cannot conclude on this basis whether perinatal stress was high or low in the SH population. However, it seems likely that individual IX suffered an episode of systemic stress associated with malnutrition or some other neonatal health problem, frequently linked to low bioavailability of vitamin A (Skinner, 1996).

Several individuals display hypoplastic lines formed later in life. For example, the canines in individual XVIII suggest that he apparently suffered systemic stress during his fourth year of life. Since lower canine crown formation time in *Homo heidelbergensis* was probably shorter than in modern humans (Ramirez Rozzi and Bermúdez de Castro, 2001), it is possible that the stress occurred somewhat earlier. In the case of individual XXVII, lesions in the premolars indicate stress suffered during his fifth year, and individual XIX suffered a severe stress episode during the ninth year. This latter case displays the only affected M3, but since only molar teeth were recovered, the coverage of the entire growth period is incomplete.

Some studies of recent human groups have interpreted peak frequencies of hypoplasias at a certain age as an indicator of the time of weaning, which is known to vary according to the

subsistence strategy (Lanphear, 1990; Goodman et al., 1984; Corruccini et al., 1982). Weaning is considered to be the time of highest metabolic stress during early childhood in pre-industrial societies and societies with low socioeconomic levels (Moggi-Cecchi et al., 1994), and therefore a possible cause of high rates of hypoplasia. Olgivie et al. (1989) report a range of 2 - 5 years, with a peak around 4 years, for the majority of LEH formation observed in their Neanderthal sample, and they interpreted this pattern as reflecting the nutritional stresses associated with weaning. Skinner (1996) recorded hypoplasias formed during the whole developmental period, from birth to 13 years, in his sample of immature Neanderthals. He noted an age range of 3.5 - 6 years for the majority of cases of hypoplasia, with a peak at 3.5 years.

The differences between LEH and PFD prevalence presented here and the prevalence rates for SH presented earlier by Bermúdez de Castro and Pérez (1995) are attributed both to differences in size and composition of the analyzed samples and to differences in methodology. We deleted three individuals from the original sample, and our microstructure analysis removed nine specimens included in the list of hypoplastic teeth in 1995.

In comparative terms, the LEH and PFD prevalence in the SH hominins both by individual (28%) and by total number of teeth (5.5%) is significantly lower than prevalence rates published for Neanderthals and recent modern populations (El-Najjar et al., 1978; Lanphear, 1990; Mack and Coppa, 1992; Moggi-Cecchi et al., 1994; Webb, 1989; White, 1978).

The results from SH require careful interpretation within the ecological and cultural contexts of the Middle Pleistocene period in Europe. The low prevalence of LEH and PFD in this *H. heidelbergensis* sample would seem initially to indicate a successful adaptation of these individuals to their environment. However, a closer examination of the environmental conditions in which they lived reveal specific sources of stress that affect normal growth and

development, such as climate, demographic density and diet (King and Ulijaszek, 1999: 261). During the Middle Pleistocene, the Iberian Peninsula may have been a refuge for both humans and for large mammal communities from the more severe effects of glaciation to the north (van Der Made, 1999, and in press). At this time, we lack accurate data on the demographic density of *H. heidelbergensis*. However, based on comparisons of the SH sample with the first anatomically modern humans in Africa about 200 kyrs later (Relethford and Jorde, 1999), it seems logical to assume that the demographic density 400 kyrs ago was lower. In sum, we can hypothesize that climate and demographic density in Atapuerca during the Middle Pleistocene both favored good adaptation of these hominins to their environment, thus weakening the likelihood that severe nutritional stress was common.

However, as noted a decade ago by Wood and his collaborators (1992) in their discussion of 'the osteological paradox', the relationship between the record of developmental stress based on dental defects in sampled individuals and the corresponding health status of the total population remains ambiguous. Growing individuals may perish from acute stresses, such as lethal epidemic disease, before their bones or teeth record this severe systemic stress, whereas individuals who have completed growth would not register such stress episodes in that manner regardless of the mortality impact. As a result, it would be erroneous to conclude automatically that individuals who show a low level of stress-related developmental defects were healthier *per se* than individuals from the same population who show higher levels of defects, without a careful comparison of the age structure of the two groups: a higher level of defects might be linked essentially with longer survival. One of the crucial assumptions of palaeopathology is "absence of evidence can not be read as evidence of absence".

Trinkaus (1995) proposed that the mortality rate of immature individuals in Neanderthal populations might be similar to rates in recent human foraging groups, and it seems possible that both the childhood mortality rate (0-10 years) and the level of stress during

development were similar in Middle Pleistocene populations and in their successors, the Neanderthals. Considering this possibility, the explanation for a significantly higher LEH rate in Neanderthals could be lower resistance to physiological stress, which would be reflected in their enamel with a higher frequency of developmental defects. The other alternative is that levels of stress during development and the childhood mortality rate were both truly higher in Neanderthal populations.

CONCLUSIONS

In this paper we have introduced a new methodology to calculate the timing of hypoplasia formation, and comparisons with previous studies employing other methodologies must be made with caution. We note that the majority of hypoplasias occurred during the third year of life: for example, in individuals X and XXII the defects were formed around 2.5 years, the hypoplasias in individual XXIV and the individual represented for the isolated incisors were formed shortly before 3 years, and the hypoplasia in the isolated canine AT-1144 was formed at 2 years. Our data seems to suggest that the weaning process in the SH population may have begun earlier than in Neanderthals, around the third year of life. However, more data from different Middle Pleistocene populations are needed to verify this hypothesis.

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TABLE 1. *Occurrence and timing of hypoplasias*

Tooth		N° Pk	Pk aff	Time from hypoplasia to the cervix		Age OtCF	Aging Hypoplasias		
				In days X9	In years X9	In days	In days	In years	
Individual IX									
Right dc	AT-90	51	*	459	1.26	303	-156 ± 102	-0.43 ± .28	
Individual X									
Right LM1 LF	AT-414	30	6	270	0.74	1219	949 ± 60	2.6 ± .16	
Left LM1	AT-556	31	6	279	0.76	1219	940 ± 62	2.58 ± .17	
Individual XVIII									
Right LC	AT-2165	64	4	576	1.58	2266	1690 ± 128	4.63 ± .35	
Left LC	AT-410	69	5	621	1.7	2266	1645 ± 138	4.51 ± .38	
Individual XIX									
Right UM3	AT-816	45	*	405	1.11	3997	3592 ± 90	9.84 ± .25	
Left UM3	AT-826	47	*	423	1.16	3997	3574 ± 94	9.79 ± .26	
Individual XXII									
Left LI1	AT-3199	68	2	612	1.68	1399	787 ± 136	2.16 ± .37	
Right LI1	AT-3250	66	3	594	1.63	1399	805 ± 132	2.21 ± .36	X
Right LI2	AT-2753	90	6	810	2.22	1522	712 ± 180	1.95 ± .49	2.38 yrs
Left LI2	AT-3198	78	2	702	1.93	1522	820 ± 156	2.25 ± .42	SD
Left LM1	AT-605	21	6	189	0.52	1219	1030 ± 42	2.82 ± .11	0.38
Right LM1	AT-605	19	7	171	0.47	1219	1048 ± 38	2.87 ± .10	
Individual XXIV									
Right LI1	AT-596	53	3	477	1.31	1399	922 ± 106	2.53 ± .29	
Right LI2	AT-281	55	1	495	1.36	1522	1027 ± 110	2.81 ± .30	X
Left LI2	AT-2391	49	6	441	1.21	1522	1081 ± 98	2.96 ± .27	2.75 yrs
		60	1	540	1.48	1522	982 ± 120	2.69 ± .33	SD
Left LC	AT-2438	96	1	864	2.37	2266	1402 ± 192	3.84 ± .52	0.18

Table 1 continued on page 36

Table 1 continued from page 36

Individual XXVII								
Right LP4	AT-792	43	7	387	1.06	2230	1843 ± 86	5.05 ± .24
Left LP4	AT-792	18	1	168	0.44	2230	2062 ± 30	5.65 ± .10
		54	2	486	1.33	2230	1744 ± 108	4.78 ± .30
Isolated teeth								
Right LI1	AT-2384	75	3	675	1.85	1399	724 ±150	1.98 ± .41
Left LI1	AT-3242	71	2	639	1.75	1399	760 ± 142	2.08 ± .39
Right UI1	AT-197	87	2	783	2.14	1836	1053 ± 174	2.88 ± .48
Right LI2	AT-282	96	4	864	2.37	1522	658 ± 192	1.80 ± .52
Left LI2	AT-608	97	4	873	2.39	1522	649 ± 194	1.78 ± .53
Right LC	AT-1144	41	1	369	1.01	1399	1030 ± 82	2.82 ± .23

N° Pk: the number of perikymata between the hypoplasia and the cervix on the buccal face.

Pk aff: the number of perikymata affected by hypoplasia, including the deepest perikymata and those in the occlusal part of the groove.

Age OtCF: Age in days at the completion of crown formation. After Reid and Dean (2000) for permanent anterior teeth; Reid et al. (1998) for premolars and molars; Liversidge and Molleson (2001) for deciduous canines. The age estimates for hypoplasia formation were based on the assumption of a 9-day interval (± 2 days) between adjacent perikymata.

LF: counts made on the lingual face.

*: Plane-form defect. Nº Pk in these individuals was counted from the lower limit of the PFD.

Mean (X) and standard deviation (SD) were calculated for those individuals where hypoplasias are seen in more than the two antimeres teeth.

TABLE 2. *LEH prevalence by tooth class*

	Maxillary			Mandibular		
Tooth	N	Affected	%	N	Affected	%
I1	18	1	5.5	16	3	18.7
I2	14	0	0	17	3	17.6
C	16	0	0	21	2	9.5
P3	11	0	0	17	0	0
P4	12	0	0	21	1	4.8
M1	14	0	0	22	2	9.1
M2	15	0	0	23	0	0
M3	18	1	5.5	25	0	0

TABLE 3. Prevalence of individuals affected by enamel hypoplasia in some Pliestocene hominin samples

Sample	N individuals studied	N individuals affected	Percentage	Author
SH[1]	25	7	28.0 [2]	Present study
Swartkrans	143	44	30.6	White, 1978
Sterkfontein	66	8	12.1	White, 1978
Neanderthals	58	44	75.9	Ogilvie et al., 1989
Neanderthals[3]	59	22	37.3	Skinner, 1996

[1] Includes only LEH

[2] Isolated teeth AT-2384, AT-3242, AT-282, AT-608, AT-197 have not been assigned to any of the 7 individuals affected by hypoplasia. If these teeth belong to the remaining 18 non-affected individuals, the prevalence would rise to 23.0% (8/25). If they belong to a new individual, the frequency would be 30.7%

[3] This sample includes only immature individuals; it is not the same sample studied by Ogilvie et al. (1989)

Figure 1. AT-808, left lower canine. (A, B) In the upper half of the crown, two grooves are clearly visible (arrows). These grooves appear to be deep and might be interpreted as hypoplasias in a macroscopic analysis. However, when the enamel surface is analyzed under SEM, the grooves correspond to shallow depressions of the enamel surface (C). The white line (B) marks the place corresponding to the profile shown in C. In tooth sections, it is possible to see marked incremental lines in the enamel (Reid et al. 1998), which are probably responsible for this appearance. We consider that this shallow depression (C, arrows) does not reach the limit of expression defined as a hypoplasia (see figure 3). In the cervical half of the crown, there are changes in the enamel opacity that look like hypoplasias for the naked eye (A, bracket). However, microscopic inspection reveals no change on the enamel surface (D). Observe that the spacing between perikymata does not always decrease towards the cervix, but it exhibits a range of normal variability (E).

Figure 2. AT-410, left lower canine. The groove corresponds to a hypoplasia (A, B arrow). The defect corresponds to the period of formation of five perikymata. Closer inspection (C) reveals that the distance between the perikymata included in the hypoplasia (arrows) is the same as in the earlier perikymata (upper lines) but greater than in the most cervical hypoplastic perikymata (lower lines).

Figure 3. AT-2438, left lower canine. A groove is clearly visible on the buccal face (A, B). When the groove is observed in lateral view (C), it can be identified a depression of the enamel surface. Depression of the enamel surface is a continuous feature in the manifestation of hypoplasias, but the limit below which a depression cannot be considered hypoplasia remains arbitrary. However, according to our definition of the minimum limit, we consider

the depression in AT-2438 to be an example of the minimum change of enamel surface necessary to be interpreted as hypoplasia (see figure 1). The white line in B marks the location place corresponding to the profile shown in C.

Figure 4. AT-1144, lower canine showing a marked LEH.

Figure 5. AT-826, left upper M3. The plane-form defect observed in the upper M3 of individual XIX (A, B) does not surround the cusps, but is only present on one face of one cusp, in this case the metacone. The limit of the hypoplastic enamel shows a different direction from that of the perikymata (C). The perikymata were counted from the lower limit of the plane-form defect.

APPENDIX

List of teeth assigned to the SH individuals included in this study

Individual	Teeth
I	Lower right: I1, I2, C, P4, M1, M2, M3
	Lower left : I1, I2, C, P4, M1, M2, M3
II	Lower right: I1, I2, C, P3, P4, M1, M2
	Lower left: I1, I2, C, P3, P4, M1, M2
	Upper right: I1
	Upper left: I1, I2
III	Lower right: I1, I2, C, P3, P4, M1, M2
	Lower left: I1, I2, C, P3, P4, M1, M2
	Upper right: P4, M1
	Upper left: P4, M1, M2
IV	Lower right: P3, P4, M2, M3
	Lower left: P3, P4, M1, M2, M3
	Upper right: M1
	Upper left: M1, M2
VI	Lower left: P3, P4, M1, M2, M3

VII Lower right: I1, I2, C, P3, P4, M1, M2

Lower left: I1, I2, C, P3, P4, M1, M3

Upper right: I1, C, P3, P4, M1, M2

Upper left: I1, I2, C, P3, P4, M1, M2, M3

VIII Upper left: P4, M1, M2

IX Lower right: dc

X Lower right: C, P4, M1

Lower left: M1, M2

XI Lower right: I2, P3, P4, M1, M2

Lower left: I2, C, M1, M2

XII Lower right: I2, C, P3, P4, M1, M2, M3

Upper right: M1, M3

Upper left: P4, M1, M2, M3

XIV Lower right: M1, M2

Lower left: M1, M2

XV Lower right: I2, C, P4, M1, M2, M3

Lower left: I1, I2, C, P4,

XVI Lower right: I2, C, P3, P4, M1, M2

Lower left: I1, I2, C, P3, P4, M1, M2, M3

Upper right: I1, I2, P3, P4, M1, M2, m3

Upper left: I1, I2, C, P3, P4

XVII Upper right: P3, P4, M1

XVIII Lower right: dm2, I1, I2, C, P3, P4, M1, M2, M3

Lower left: dm2, I1, I2, C, P3, P4, M1, M2, M3

Upper right: dm2, I1, I2, C, P3, P4, M1, M2, M3

Upper left: dm2, I1, I2, C, P3, P4, M1, M2, M3

XIX Lower right: M2, M3

Lower left: M1, M2, M3

Upper right: M1, M2, M3

Upper left: M1, M2, M3

XX Lower right: I2, C, P3, P4, M1

Lower left: I1, C, P3, P4, M2

Upper right: I1, I2, C, P3, M1, M2

Upper left: I1, I2, C, P3, P4, M1, M2

XXII Lower right: I1, I2, C, P3, P4, M1, M2, M3

Lower left: I1, I2, P3, P4, M1, M2, M3

Upper right: I2, C, M1, M2, M3

Upper left: M3

XXIII Lower right: I1, I2, C, P3, P4, M1, M2, M3

Lower left. I2, C, P3, P4, M1, M2, M3

XXIV Lower right: I1, I2, P3, M1, M2, M3

Lower left: I2, C, P3, M1, M2, M3

Upper right: C, M1

XXV Lower right: dm2, I1, I2, C, P3, P4, M1, M2, M3

Lower left: dm2, I1, I2, C, P3, P4, M1

XXVI Lower right: M1, M2, M3

Lower left: M1, M2

Upper right: M1, M2, M3

Upper left: P3, M1, M2, M3

XXVII Lower right: P3, P4, M1, M2

Lower left: P3, P4, M1, M2, M3

Upper right: C, P3, P4, M1, M2

Upper left: C

XXXI Lower right: I1, M1, M2, M3

Lower left: I1, I2, C, M1, M2, M3

Upper right: I1, C

Upper left: I1, I2, C, M3