AMERICAN JOURNAL OF PHYSICAL ANTHROPOLOGY 136:351–356 (2008)

Brief Communication: Physiological Stress in the Florida Archaic—Enamel Hypoplasia and Patterns of Developmental Insult in Early North American Hunter-Gatherers

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KEY WORDS bioarchaeology; hunter-gatherers; LEH; Windover

ABSTRACT We examined the prevalence and developmental timing of linear enamel hypoplasias (LEHs) in an early Archaic Floridian population from Windover (8,120–6,980 ^{14}C years B.P. uncorrected). Using digital images, mandibular and maxillary canines were analyzed for defect prevalence and timing of insults. Although overall prevalence was very weakly correlated with earlier defect timing, there were significant differences in defect prevalence that varied by sex and tooth type. The mean LEH count in male mandibular canines was far higher than in male maxillary canines or in female mandibular or maxillary canines. We examined defect timing as a possible predictor of the sex differences in LEH prevalence. There were no significant sex differences in the developmental timing of the earliest defects in either tooth class. Developmental timing is not responsible for the sex differences seen in defect prevalence in mandibular canines. Am J Phys Anthropol 136:351–356, 2008.

Dental enamel hypoplasias result from a disruption in dental enamel production reflecting an interruption in ameloblastic matrix formation. These defects and the nonremodeled nature of teeth provide a window into the "metabolic memory" of the developmental process. Histological studies reveal that disturbances in enamel formation commonly result in increased spacing in perikymata grooves. These disturbances manifest as lines, furrows, rings, small holes, or pits (Rose, 1973; Shellis, 1984; Buikstra and Ubelaker, 1994; Hillson, 1996). This study documents and interprets patterns of linear enamel hypoplasias (LEH) in the Windover series. We examine sex and demographic differences in LEH distribution, and test the hypothesis that developmental timing directly affects the prevalence of LEHs. The skeletal sample from Windover, Brevard County, Florida (8BR246) provides a unique opportunity to explore questions of health, disease, and demography in the Early Archaic of the southeastern United States.

Enamel hypoplasias can result from systemic stress (Skinner and Goodman, 1992). Most researchers agree on possible causes of LEH, and some propose that the severity of LEH indicates elevated childhood stressors and less favorable living conditions. Others (c.f. Arcini, 1999) argue that these stress markers demonstrate that the individual survived the stress, indicating either that the individual's resistance to the stressor or that their living conditions must have been more favorable than those who died in childhood. Studies of enamel hypoplasia frequency in contemporary populations support an association between prevalence of hypoplastic defects and general living conditions (Goodman and Armelagos, 1985; Goodman and Rose, 1991). Many researchers (Goodman and Armelagos, 1988; Duray, 1996; Malville, 1997) find that higher rates of enamel hypoplasia are strongly correlated with lower mean age of death.

Defect prevalence depends on factors beyond that of individual stress. The most important factors appear to be tooth type, location on the tooth, and sex. The majority of researchers agree that the anterior dentition shows the highest hypoplasia prevalence but that there is variability across tooth type (Hillson, 1996; Malville, 1997). Generally, maxillary incisors are most hypoplastic, followed by canines, lower lateral incisors, and second molars (Goodman and Armelagos, 1985).

As each tooth type has a slightly different developmental track, correlating defects with developmental events in an individual's life involves modeling the formation of each tooth type. Among-tooth variation in hypoplastic rates is often explained by developmental timing. Earlier forming teeth such as central incisors are generally more hypoplastic. Goodman and Armelagos (1985) suggest that both time of development and developmental stability of the tooth type affect LEH prevalence. Less developmentally stable teeth (less tightly genetically controlled) are less resistant to ameloblast disruption and will have more hypoplasias than their simultaneously developing, more stable counterparts.

Studies of tooth crowns by developmental stages demonstrate that the time of crown development is not the only determinant of hypoplasias. Teeth developing at the same time do not always demonstrate defects to the same degree (Goodman and Armelagos, 1985). Despite

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Received 22 March 2007; accepted 10 January 2008

DOI 10.1002/ajpa.20816

Published online 6 March 2008 in Wiley InterScience (www.interscience.wiley.com).

established developmental chronologies for crown formation, some general trends in the distribution of hypoplastic defects remain unexplained (Buikstra and Cook, 1980).

Sex differences in rates of hypoplastic defects are highly variable (Guatelli-Steinberg and Lukacs, 1999). Several studies (Lanphear, 1990; Duray, 1996; Malville, 1997) have reported no significant sex differences in defect distribution, while others (Palubeckaite et al., 2002) indicate higher incidence of hypoplastic defects in males. Although many studies report sex differences in LEH prevalence, few have analyzed sex differences in defect timing. Some authors express concerns over interpretation of developmental timing of hypoplastic defects when reference is not made to microscopic anatomic structures (Hillson and Bond, 1997).

MATERIALS AND METHODS

Ninety-five individuals had scorable canines (73 mandibular and 63 maxillary). Thirty-nine individuals had both maxillary and mandibular canines. This sample includes an approximately equal number of males (n = 24) and females (n = 27) though the mean female age was 5 years lower (44) than the mean male age (49), and there are nearly twice as many females (n = 12) as males (n = 7) in the 40 and under age group. Figure 1 shows the distribution of the sample by sex in each age cohort.

Sex assessment was based on nonmetric pelvic and cranial traits and metric analysis of femoral and humeral head dimensions following methodology outlined by Buikstra and Ubelaker (1994). Aging was based on a combination of pubic symphysis assessment coupled with Windover specific population attrition standards (Buikstra and Ubelaker, 1994). In cases of disagreement in estimated age at death, attrition was preferentially used. There appears to have been no systematic age, gender, or socially stratified burial treatment (Doran and Dickel, 1988; Doran, 2002).

Canines were chosen as an indicator of overall metabolic stress for this population. Some studies score both incisors and canines (Reid and Dean, 2000; Palubeckaite et al., 2002; King et al., 2005), but we analyzed only canines exclusively since their defect prevalence is usually higher and they have a slightly longer developmental time frame for crown completion relative to other permanent teeth (Reid and Dean, 2000). One mandibular canine and one maxillary canine were used to assess LEH in an individual (rather than averaging measurements from both the right and left teeth in a tooth class). Left canines were preferentially used for this analysis; however when no left canine was available for an individual, right canines were used. Crown height was measured with digital calipers from the cementoenamel junction (CEJ) to the apex on a vertical plane bisecting the labial surface of the tooth. Teeth with excessive wear (less than 25% of the crown remaining) were excluded from analysis. Where dental calculus prevented an accurate measurement of crown height, measurements were taken from the bottom of the calculus to the top of the crown.

Photographs were taken of maxillary and mandibular canines using a Nikon 990 Coolpix in macro mode. Macro mode provides finer detail and the object fills approximately 75% of the image frame. A metric scale

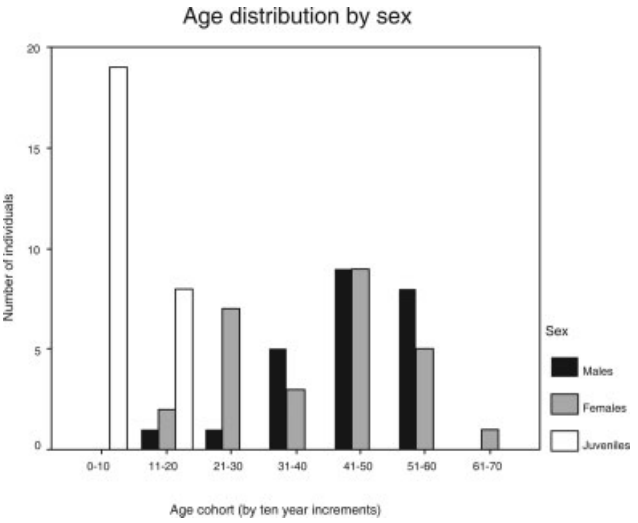

Fig. 1. Age distribution by sex.

was placed in the plane of the labial tooth surface in each photograph. The diminished focal length presents some difficulty with depth of focus outside of one plane, so multiple photographs were taken to provide defect clarity with minimal distortion from tooth surface curvature.

Crown heights taken with calipers were used to validate the accuracy of the image analysis software (Scion, a PC friendly software modeled after the National Institute of Health Image program for Macs; www.nist.gov/lispix/imlab/labs.html), and to identify any possible image distortion. A zero correlation test revealed the mandibular control sample (n = 42) and the digitally analyzed crown heights have a 0.95 correlation coefficient (t = 19.341, and P = 0.000). The maxillary canine control sample is even more strongly correlated with crown height (n = 34, r = 0.961, t = 19.702 and P = 0.000). The physical caliper measurements of the canines are statistically equal to the Scion software measurements of the digital images. Distortion appears to be essentially eliminated by exact and consistent placement of the scale in the measurement plane. Scion proved to be reliable in measuring digital images.

Each photograph was then scored for hypoplastic defects in Microsoft Paint (see Fig. 2).

Scored images were imported into Scion Image software for analysis. Because of significant attrition in the Windover sample, the stage of development for each defect was determined by measuring the distance from the CEJ to the bottom of each defect. Developmental chronology was determined using the estimate by Reid and Dean (2000), which necessitates estimation of complete, unworn crown height for every tooth. An estimate of completeness for each canine was based on surrounding dentition and other canines within the population.

Estimation of crown completion was then used to generate an estimated complete crown height for each individual tooth. This introduces age bias, as older individuals exhibit greater attrition. In adult teeth the median percent complete was 75%. The mean was 75% complete for mandibular canines, and 72% complete for maxillary canines with the first 25% of many teeth unavailable for analysis due to attrition. We feel the estimated crown

Fig. 2. Image scored for LEHs (pits noted). Black lines indicate LEH, pits noted in white.

height for each tooth is more appropriate than relying on a standardized crown height either from a modern population or from a mean completed crown height from Windover. Reid and Dean (2000) then correlated age of development by percent of total crown completion, and we used this to establish a relative age of metabolic insult for each defect. Determining the developmental timing of each defect allows the comparison of particular windows of time in each individual's life on a population basis. This may indicate changes within the population over time, or susceptibility of children at developmental milestones.

Reid and Dean (2000) estimate cusp enamel completion at 1.7 years for maxillary canines and 0.98 years for mandibular canines. Estimated crown completion for maxillary canines is 5.3 years of age, and 6.2 years for mandibular canines. These estimates are based on a modern sample but that is preferable to older standards because they do not assume equal developmental time for each increment or decile (10%). However, the variation in canine crown development within a population can be as great as 1.0 to 1.5 years (Massler et al., 1941; Ubelaker, 1989). There are few population-specific standards of detailed developmental chronologies of tooth formation, however standards have been developed for some nonwestern populations (Reid and Dean, 2006).

Despite precision in mapping the location of defects, there is still subjectivity in the identification of defects. Intraobserver error was tested on a random subset of 10 maxillary and 12 mandibular canines. The canines were rescored 6 months after the original analysis, and paired *t*-tests on LEH counts were statistically equal for both tooth types. The maxillary mean difference was 0.100 ($P = 0.678$) and the mandibular mean difference was 0.083 ($P = 0.723$). Interobserver error was measured on a sample of 10 maxillary and 10 mandibular canines. The maxillary canine mean difference was −0.100 ($P = 0.678$) and the mandibular difference was 0.400 ($P = 0.104$). Crown height was also measured for interobserver error. Neither maxillary (mean = −0.168, $P = 0.562$) nor mandibular canines (mean = −0.174, $P = 0.194$) had significant difference in measurement by different scorers. The use of magnified computer images clearly allows easier and more consistent LEH identification; however, this method may result in higher defect counts than unaided observations might produce. Based on our experience, this increase is probably within the range of interobserver variability.

RESULTS

Combining mandibular and maxillary canine defect counts, there were no significant sex differences in LEH prevalence. Males have only a slightly higher prevalence with an overall mean of 2.7 per person while the female mean is 2.6. This small difference is misleading, however, as it obscures a larger sex difference in LEH by tooth type.

Mandibular canines have a mean LEH count of 2.78, while maxillary canines have a mean LEH count of 2.00. These findings are equivalent to one more defect on the mandibular canine than on the maxillary canine in nearly 80% of the cases. The sample correlation coefficient is 0.629 ($P = 0.000$). Difference between LEH counts in the mandibular *versus* maxillary canines is tested by Pearson Chi-square test, resulting in a value of 21.541 ($P = 0.001$). Mandibular canines have more LEHs than maxillary canines. The higher prevalence for mandibular canines may be explained at least in part by the longer time recorded by crown formation of those teeth (0.9 years longer than maxillary canines). Within male tooth types the mean LEH counts reveal a difference of 3.2 mandibular to 1.9 maxillary (statistically significant with a 41% higher prevalence in mandibular defects). In females, the mean LEH counts (1.9) are the same in the mandibular and maxillary canines, despite the longer developmental timeframe for mandibular canines. Assessment of sex differences in visible attrition supports the sex differences seen in LEH prevalence. There was no sex difference in visible mandibular attrition ($P = 0.577$) or maxillary attrition ($P = 0.831$).

Prevalence of LEHs also appears to be correlated with age at death. Table 1 illustrates mean LEH counts by 10 year age cohorts. These data are for permanent dentition only, as the deciduous dentition is not developmentally comparable. The three youngest cohorts (0–10, 11–20, 21–30) have a mean of 2.7 defects, which is significantly higher than that observed in the three older cohorts (31–40, 41–50, 51–60) where the mean is 2.2 defects. Maxillary canines demonstrate a distinct and consistent decline in frequency after 20 years of age, while mandibular canine defect counts remain fairly constant in all individuals older than 20 years of age.

The correlation of LEH prevalence with age at death can be further illustrated by dividing the population into two groups: those who died before reaching 20 years of age, and those who lived past 20. Mean mandibular and maxillary LEH counts are higher in the group that failed to reach 20 years of age, though the difference is statistically significant only for maxillary canines ($P = 0.045$).

Individuals with three or more defects ($n = 11$) ranged in age from 10.0 to 49.0 years and had a mean age of 24.9 and a median age of 23.0 years. Individuals with two or fewer defects ($n = 12$) ranged in age from 16.0 to 54.0 and had a mean age of 34.7 and a median age of 32.0 years. These two groups demonstrate a 9.8 year difference in mean age of death. As would be expected, those with more than three defects on the mandibular canine and less than three defects on the maxillary canine have an intermediate mean age of death (29.9 years). Only one individual exhibited three or more max-

TABLE 1. LEH distribution by tooth type

Age (years)	1–10	11–20	21–30	31–40	41–50	51–60	61–70	Total
Mandibular N/no. LEHs	6/20	10/32	7/18	7/18	15/27	10/27	1/0	N = 66/142
Maxillary N/no. LEHs	4/8	11/28	6/14	7/15	12/6	6/6	0/NA	N = 54/77
Mandibular mean LEH	3.3	3.2	2.6	2.6	2.7	2.7	0	Mean = 2.4
Maxillary mean LEH	2.0	2.5	2.3	2.1	1.6	1.2	N/A	Mean = 2.0
Mean LEH by age group	2.7	2.9	2.5	2.4	2.2	2.0	0	Mean = 2.1

The first and second rows show the number of individuals in each age cohort over the total number of LEHs in that cohort.

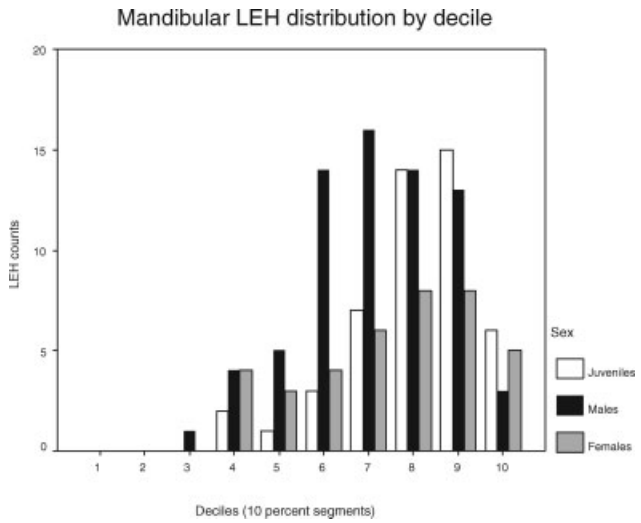

Fig. 3. Mandibular LEH distribution by sex.

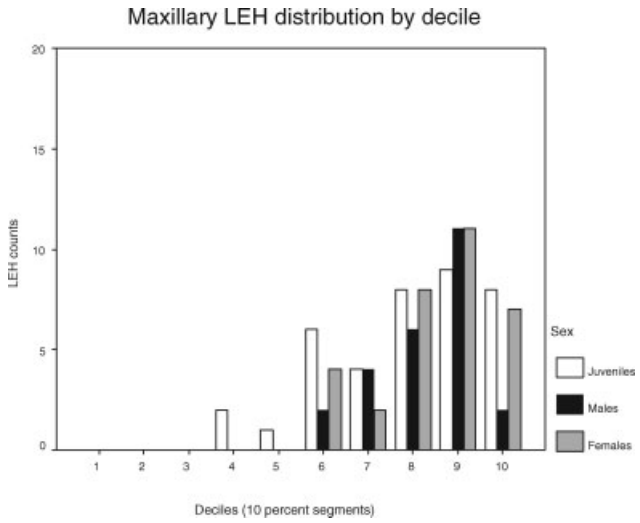

Fig. 4. Maxillary LEH distribution by sex.

illary defects and less than three mandibular defects. This individual died at approximately 18 years of age. Though the mean ages of death in the group with mandibular-maxillary variation show differences, the findings are not statistically significant for this subsample (P = 0.097).

The clustering of defects around a particular developmental time frame might indicate cultural or biological milestone events (i.e. birth or age at weaning). It should be mentioned again that in the adult dentition the earliest age interval is so often worn away that much of the data on the first years of life are lost. Reconstruction of health status must then begin with the last portion of the crown formation, since these data are the most prevalent. To examine the possibility that clustering of LEH events is due to attrition rather than a trend in developmental timing, a two sample t-test was performed on teeth that were less than 70% complete as compared to teeth that were more than 70% complete. This test showed no difference in LEH prevalence on either maxillary (P = 0.487) or mandibular canines (P = 0.926). If attrition was obscuring a significant pattern of early LEHs in this population, the group with less than 70% complete crowns (greater attrition) should demonstrate fewer defects than those with more complete crowns. Since the two groups are not statistically different we conclude it is unlikely that attrition is confounding these results.

The mean developmental timing of metabolic insult for the mandible is 3.58 years of age with a mean maxillary insult age of 2.85 years. Ages from 2 to 4 years are the most likely times for weaning given the median age of weaning among modern hunter-gatherers (Marlowe, 2005), though some researchers would extend this timeframe (Goodman and Armelagos, 1989). This timeframe encompasses a significant percent of the total crown formation interval, particularly because the first visible defects in life may be removed by attrition. At Windover, the mandibular developmental period with the most data (visible crown) ranges from approximately 2.0 to 6.2 years of age (approximately when the CEJ is formed). Mandibular defect distribution in this population ranges in timing from 2.7 to 4.97 years of age.

Males demonstrate the greatest variability of LEH prevalence within tooth type. They appear to have mandibular defects closer to the CEJ (developmentally closer to 6 years of age), whereas females demonstrate defects closer to the CEJ in maxillary canines. Figures 3 and 4 show defect timing by sex in mandibular and maxillary canines with the crown broken into 10 percent segments (deciles).

Developmental timing of LEH did not vary significantly between males and females. Neither mandibular nor maxillary canines showed significant differences in defect timing. In the mandible, mean male defect age was 4.27 years, the mean female defect age was 4.21 (P = 0.758), and the mean was 4.60 in unsexed juveniles. In the maxillary canines, the mean male defect age was 4.08 years, 4.32 years in females (P = 0.136), and 4.24 years in juveniles.

Juveniles did not demonstrate significant timing differences when compared to adults for maxillary canines (P = 0.760). The overall mean age of insult in juveniles was 4.60, while the mean age of insult for adults was 4.25. Analysis by tooth type revealed no significant differences between adult and juvenile defect timing for

maxillary canines. In contrast, mandibular timing differences were significant ($P = 0.047$). Juveniles demonstrated significantly later mandibular defect timing than adults. Mean ages of the first insult are nearly equal, at 3.58 for adults and 3.59 for juveniles. Individuals showing more LEHs also show earlier LEHs, though the correlation is weak ($r = -0.278$, $P = 0.040$). Correlations of defect timing by sex did not reveal any significant results.

DISCUSSION

Windover males exhibit slightly higher overall hypoplastic defect prevalence than females. This difference could be attributed to an underlying difference in developmental milestones between the sexes, greater biological susceptibility to stress in males, and cultural practices causing health status differences by sex. For males tooth type plays a role in the patterning of hypoplastic defects. The mean LEH count in male mandibular canines is far higher (3.3), but the level for maxillary canines is the same as it is for both female maxillary or mandibular canines (1.9). Mandibular canines are often reported as being more hypoplastic than maxillary canines (c.f. Cucina and Iscan, 1997), however reported sex differences in LEH prevalence by tooth type are highly variable. Further research on sex differences by tooth type may reveal trends linked to other factors.

Earlier age of defects in the total population was only weakly correlated with higher prevalence. The "damaged goods" hypothesis (Goodman and Armelagos, 1989) suggests that the thymolymphatic growth that is responsible for the development of the immune system can be damaged by early stress. The "damaged goods" hypothesis would predict higher overall defect counts in those with earlier defect timing due to the permanently compromised immune system of immunologically battered individuals. At Windover, we found that the earliest hypoplastic defect shows no significant sex differences in developmental age. Developmental timing is not responsible for the sex differences seen in overall defect prevalence. However, juveniles did demonstrate timing differences with adults, exhibiting later mean mandibular defect timing. These results lend little support for the "damaged goods hypothesis" in this population.

The fact that older individuals showed fewer LEHs supports the use of LEHs as an indicator of health status, despite the possible confounds presented by the Osteological Paradox (Wood et al., 1992). Individuals in Windover most affected by hypoplastic defects (three or more) had a mean age of death 10 years younger than those with two or fewer hypoplastic defects. These results are further supported by the prevalence of LEHs in those surviving past twenty years. The population dying before 20 years of age exhibited a higher mean prevalence of LEHs in both mandibular and maxillary canines than those who survived to 20 years or older.

The photographic methods used here are more time intensive and costly than gross methods of analysis such as the unaided observer scoring system used in the Western Hemisphere Health Database (Steckel et al., 2002). Even so, the benefits of the digital image methods are fourfold. First, these images can be archived. Second, they provide replicability and contestability of research in this and other domains (i.e. such as scoring other features). Third, they allow for the separation of one particular set of data from the rest of the osteological evidence, allowing for double-blind studies and a more solidly independent line of evidence into health status. Lastly, high resolution photographs allow for more standardized training and quality control in osteological scoring and research. On-line storage/distribution provides global access to the images in contrast to the traditional necessity of physically visiting each collection for comparative studies.

CONCLUSIONS

The aim of this study was to examine the effect of developmental timing of LEH on overall defect prevalence in an Archaic hunter-gatherer population. The "damaged goods" hypothesis would predict that earlier developmental timing of metabolic insults would result in higher overall incidence of LEHs. The "damaged goods" hypothesis is not supported by this analysis. It is possible that this hypothesis would be supported by comparing insults across a greater portion of the individual's life history (such as correlating LEHs with porotic hyperostosis). Developmental timing does not appear to be a significant factor in the LEH sex differences seen in this particular population. Mandibular canines are the most at-risk tooth class in males. Mandibular canines potentially record more insults due to the longer time of development, however this does not address the sex differences seen in overall defect counts in this tooth type. The sex differences seen in this population must then be attributable to greater male susceptibility for the mandibular canines (which is currently not well supported by other studies), or a sociocultural factor that impacts the male children more than the female children of this population.

ACKNOWLEDGMENTS

We would like to thank Clark Larsen and two anonymous reviewers for comments. We would also like to thank Bill Parkinson for the use of his camera.

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